O.G. Goryacheva

SYMPTOMS AND SIGNS OF GASTROINTESTINAL DISEASES

Manual for Foreign Students

Federal State Budgetary Educational Institution of Higher Education
«Academician E.A. Vagner Perm State Medical
University» of the Ministry of Healthcare of the Russian Federation

SYMPTOMS AND SIGNS OF GASTROINTESTINAL DISEASES

Manual
for Foreign Students

Approved by Academician Council
of FSBEI HE academician
E.A. Vagner PSMU MOH Russia

Perm
2023

UDK 616.33/.34-07-036.1(075.8)
BBK 54.132+54.133я73Г71
G 71

G 71 **Goryacheva O.G.**
Symptoms and signs of gastrointestinal diseases: manual for Foreign Students. – Perm: FSBEI HE academician E.A. Vagner PSMU MOH Russia, 2023. – 316 p.

ISBN 978-5-7812-0681-0

The study of the pathology of the gastrointestinal tract is associated with the need to master many manual diagnostic techniques in the clinic, to study the main laboratory and instrumental parameters that allow for a clear differentiation of symptoms and syndromes.

The study guid presents the propaedeutics of diseases and conditions associated with the pathology of the esophagus, stomach and intestines in relation to the clinic of internal diseases. Methods of manual skills are presented in accordance with the program of the course of propaedeutics of internal diseases of medical universities. The author tried to include in the textbook all the traditional clinical methods for studying diseases of the gastrointestinal tract, taught in the Russian medical school. Recommended for students of medical universities studying in English.

Reviewers:

doctor of medical sciences, professor of the FSBEI HE Donetsk State Medical University named Maxim Gorky of the Ministry of Healthcare of the Russian Federation ***Vatutin N. T.***;

doctor of medical sciences, professor of the FSBEI HE First Moscow State Medical University named of I.M. Sechenov of the Ministry of Healthcare of the Russian Federation ***Napalkov D.A.***

Published by decision of Academic Concil
of FSBEI HE academician E.A. Vagner PSMU MOH Russia.
Protocol № 3 from 22.03.2023.

UDK 616.33/.34-07-036.1(075.8)
BBK 54.132+54.133я73Г71

ISBN 978-5-7812-0681-0

Федеральное государственное бюджетное образовательное учреждение высшего образования «Пермский государственный медицинский университет имени академика Е.А. Вагнера» Министерства здравоохранения Российской Федерации

О.Г. Горячева

СИМПТОМЫ И ПРИЗНАКИ ЗАБОЛЕВАНИЙ ЖЕЛУДОЧНО-КИШЕЧНОГО ТРАКТА

Утверждено ученым советом
ФГБОУ ВО ПГМУ им. академика Е.А. Вагнера
Минздрава России в качестве учебного пособия

Пермь
2023

Горячева Ольга Георгиевна – канд. мед. наук, доцент кафедры пропедевтики внутренних болезней № 2 ФГБОУ ВО ПГМУ им. академика Е.А. Вагнера Минздрава России

УДК 616.33/.34-07-036.1(075.8)
ББК 54.132+54.133я73Г71
Г 71

Г 71 **Горячева О.Г.**
Симптомы и признаки заболеваний желудочно-кишечного тракта: учеб. пособие. − Пермь: ФГБОУ ВО ПГМУ им. академика Е.А. Вагнера Минздрава России, 2023. – 316 с.

ISBN 978-5-7812-0681-0

Изучение патологии желудочно-кишечного тракта связано с необходимостью овладения многими методами физикального исследования, изучения основных лабораторных и инструментальных показателей, позволяющих четко дифференцировать симптомы и синдромы.

Представлена пропедевтика заболеваний и состояний, связанных с патологией пищевода, желудка и кишечника применительно к клинике внутренних болезней. Методы физикальных исследований представлены в соответствии с программой курса пропедевтики внутренних болезней медицинских вузов. Автор постарался включить в работу все традиционные клинические методы изучения заболеваний желудочно-кишечного тракта, преподаваемые в российской медицинской школе.

Рекомендуется для студентов медицинских вузов, обучающихся на английском языке.

Рецензенты:

д-р мед. наук, профессор ФГБОУ ВО «Донецкий государственный медицинский университет имени Максима Горького» Минздрава России **Ватутин Н.Т.**;

д-р мед. наук, профессор ФГАОУ ВО «Первый Московский государственный медицинский университет имени И.М. Сеченова Минздрава России **Напалков Д.А.**

Печатается по решению ученого совета ФГБОУ ВО ПГМУ
им. академика Е.А. Вагнера Минздрава России
Протокол № 3 от 22.03.2023 г.

УДК 616.33/.34-07-036.1(075.8)
ББК 54.132+54.133я73Г71

ISBN 978-5-7812-0681-0

CONTENT

ABBREVIATIONS

ALP	–	alkaline phosphatase
ALT	–	alanine aminotransferase
AST	–	aspartate aminotransferase
BP	–	blood pressure
CD	–	Crohn's disease
CRP	–	C-reactive protein
CT	–	computer tomography
DNA	–	deoxyribonucleic acid
ELISA	–	enzyme-linked immunosorbent assay
ESR	–	erythrocyte sedimentation rate
FEGDS	–	fibroesophagogastroduodenoscopy
GERD	–	gastroesophageal reflux disease
GGTP	–	gamma-glutamyl transpeptidase
GIT	–	gastrointestinal tract
HH	–	hiatus hernia
HRS	–	hepatorenal syndrome
LAP	–	leucine aminopeptidase
MRI	–	magnetic resonance imaging
NSAID	–	non-steroidal anti-inflammatory drugs
PCR	–	polymerase chain reaction
PH	–	potential of hydrogen
PU	–	peptic ulcer
RNA	–	ribonucleic acid
UC	–	ulcirative colitis
X-ray	–	Roentgen investigation

Dear readers!

Since 2018, I have been teaching internal medicine propaedeutics daily to foreign students studying in English. I have to teach students from India and Egypt more often. The teaching of our subject is connected, among other things, with the acquaintance of students with the discoveries of Russian and Soviet medicine, the founders of which are the authors of many world discoveries in examining patients with internal diseases in the clinic. The purpose of designing this textbook was not only to present the features of the examination of patients with diseases of the gastrointestinal tract, but also to focus on the features of clinical examination in the Russian school. I thank my students for inspiring me to write this manual.

Sincerely, Olga Goryacheva

CHAPTER 1.
SYMPTOMS AND SIGNS IN PATIENTS WITH ESOPHAGUS, STOMACH AND BOWEL DISEASES

Human digestive system is represented by a complex of organs that jointly perform many vital functions in the body (fig. 1).

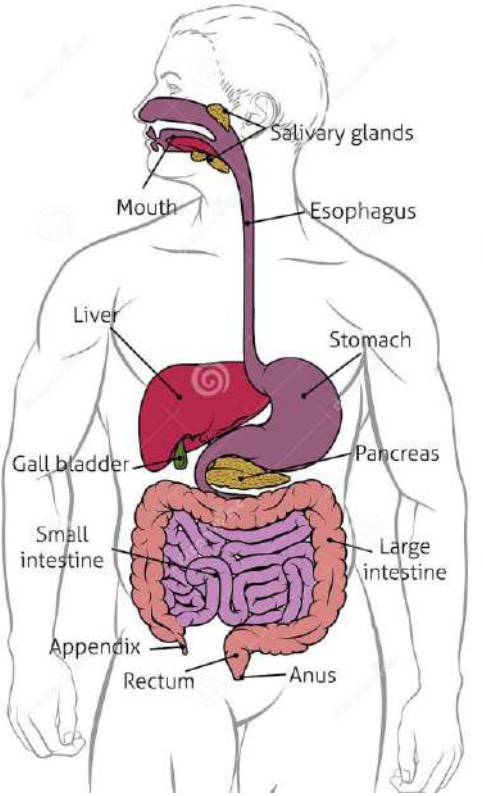


Figure 1. The main organs of digestive system

The main functions of the digestive system:

1) motor-evacuation function (mixing, transit and evacuation of food);

2) secretory − the formation of enzymes, biologically active fluids for parietal digestion (saliva, gastric juice, secretion of the small intestine);

3) splitting and absorption;

4) endocrine function − the production of pancreatic hormones, intestinal hormones;

5) immune function;

6) protective function (natural barrier, detoxification, disinfection).

The pathology of each organ of the digestive system is accompanied by characteristic symptoms. Patients with diseases of the gastrointestinal tract often complain of swallowing disorders, heartburn, belching, abdominal pain, decreased appetite, nausea and vomiting, weight loss, constipation or diarrhea, rectal bleeding and other complaints. It is important to understand how and why these complaints may occur.

Complaints about swallowing disorders (dysphagia). Impaired swallowing can be a symptom of damage to the oral cavity, pharynx, and esophagus at various levels. It is important to clarify with the patient about the peculiarities of the passage of food through the esophagus (free, difficult, painful, impossible), and also to clarify whether solid and liquid food passes through, whether the patient chews food well, whether he has pain while chewing.

Heartburn complaints. With complaints of heartburn, it is necessary to clarify the frequency, intensity, duration of their manifestations, to clarify the connection with food intake, with the type of food. It is important to assess the localization of the burning sensation, to ask if there is a connection with physical activity or stress, if shortness of breath occurs. Often, patients

who develop exertional angina take burning pains behind the breastbone for heartburn, and vice versa.

Complaints about belching. Belching is an unexpected evacuation of air from the esophagus, stomach, and the initial sections of the duodenum − may be empty, tasteless and odorless, or it may be bitter, sour, rotten egg, or recently eaten food. It is necessary to clarify how the eructation is facilitated, whether there is regurgitation at this moment.

Complaints of abdominal pain. The most common complaint of patients with diseases of the gastrointestinal tract is abdominal pain. The Mayo Clinic (USA) compiled a list of possible characteristics of abdominal pain: the pain is acute or appeared unexpectedly; burning pain; chronic or persistent; spastic; stupid; hungry; intense; intermittent or episodic; worsening or progressive; sharp; permanent. The physician should be sure to clarify and document the nature of the pain. Abdominal pain can be accompanied by swelling of the anterior abdominal wall, black or bloody stools, constipation, diarrhea, fever, intestinal paresis, nausea or vomiting, flatulence, throbbing, rumbling of the abdomen and weight loss. It is necessary to note the connection of abdominal pain with food intake, the dependence of the intensity of pain on the type and amount of food, the presence of pain on an empty stomach and pain at night. It is important to clarify the effect of changes in body position and certain movements on abdominal pain, whether they begin and end slowly or abruptly, whether they are relieved by vomiting.

According to the mechanism of occurrence of abdominal pain, 4 types are distinguished:

1) spastic − pain as a result of spasm of smooth muscles, wich often occur with intestinal colic;

2) distal − as a result of stretching of the hollow organs and their ligamentous apparatus;

3) peritoneal − as a result of the transition of inflammation to the parietal peritoneum or perforation of hollow organs;

4) vascular − as a result of acute circulatory disorders in organs (spasm, thrombosis), they can be the most intense.

The algorithm for describing abdominal pain includes 5 main points:

1) localization;

2) the nature of the pain;

3) connection with food intake;

4) intensity;

5) irradiation;

6) relieving factors (food intake, antacids, bowel movements, gas, etc.).

The manifestations of pain syndrome with the following characteristics are considered alarming:

1) the pain is sharp, persistent or recurrent, lasting more than 6 hours or getting worse;

2) the pain is accompanied by shortness of breath, fever, dizziness, vomiting;

3) the pain is accompanied by impaired urination, impaired discharge of gases, weakening of peristalsis;

4) pain radiates to the neck, chest, shoulder;

5) pain accompanies vomiting of blood;

6) pain accompanied by bloody or haematuric stools.

It is important to ask the patient about the presence of such unpleasant sensations in the abdomen as swelling, heaviness, rumbling.

Complaints about impaired appetite. The patient's appetite requires detailing: good, average, absent. Is there an aversion to food or a particular dish? In some diseases, there is a pathological increase in appetite (polyphagia), wolf hunger (bulimia), perversion of appetite (malacia, pica). Is there a feeling

of fullness after eating? Clarify about dry mouth, bitterness, acidity, metallic taste, lack of taste, salivation.

Complaints about bloating and distension of the abdomen. This condition can be accompanied by diffuse abdominal pain, increased gas production (meteorism) and increased gas excretion (flatulence). It should be clarified whether there was a fact of alcohol abuse, whether there was any previous jaundice, hematuria. Did the patient notice any disturbances in the bowel function (diarrhea, constipation), does he suffer from rheumatic heart disease? The information obtained in this case can serve as a basis for the detection of latent cirrhosis of the liver, colon tumors with metastasis to the peritoneum, congestive heart failure or nephrosis.

Complaints of nausea and vomiting. Nausea can bother the patient on an empty stomach or after eating. It is necessary to indicate the frequency of nausea, intensity, duration, dependence on the quality of food. Complaints about vomiting are subjected to similar detail − whether vomiting occurs on an empty stomach or after eating, how long after eating, what is the amount of vomit. Vomit is described in detail: taste (tasteless, bitter, sour, etc.), character (particles of undigested food, mucus, foam, sticky masses, the presence of pure blood, yellowish-green masses due to the admixture of bile, masses in the form coffee grounds, fecal vomit of yellow or dark brown color with a fecal odor).

Complaints about violations in the evacuation function of the bowel. The activity of the patient's intestines is described in detail. Is there a daily defecation and at what time, the number of bowel movements per day? If defecation is not daily, then in how many days? Do I have to do enemas and / or take laxatives? Does the patient have a feeling of incomplete bowel movement? Is there a change in constipation and diarrhea, does the consistency of the stool often change? Are there involuntary bowel movements?

Each observed patient with complaints about the state of the gastrointestinal tract is obligatory to the nature of the bowel movements: the stool is shaped, mushy, liquid, solid, in the form of sheep feces. The blooming feces are indicated − brown, dark brown, black, tarry, yellow, yellow-green, light yellow. This clarifies the presence of blood impurities (hematochezia) and mucus in the feces. If so, in what quantity. It is recommended to determine the nature of the stool in accordance with the Bristol Stool Scale (fig. 2), in accordance with the selection of 7 types of stool.

Figure 2. Bristol Stool Scale

The first type is a stool of the «sheep feces» type, very constipated and separated, dry; the second type is dry, decorated with sausage, moderately constipated; the third type is the shape of the sausage, with impressions (norm); fourth type − sausage shape, smooth; the fifth type − soft cakes with clear edges (with a high content of fibrin fibers); the sixth type is mushy (inflammatory), the seventh type is of a liquid consistency, without solid inclusions, unformed.

Diarrhea. Diarrhea is liquid, unformed stools (type 7 on the Bristol scale), combined with frequent bowel movements. In the pathogenesis of diarrhea, there may be violations of peristalsis, impaired absorption of fluid in the intestine, an increase in the pathological secretion of fluid in the intestine during inflammation. The causes of diarrhea can be acute and chronic inflammatory processes in the mucous membrane of the small intestine (enteritis, enterocolitis, sigmoiditis, proctitis), with various kinds of exogenous (arsenic, mercury) and endogenous (uremia, diabetes, gout) intoxications, endocrine disorders (adrenal dysfunction), with symptoms of hypersensitivity to certain food substances (allergy). Also, the cause of diarrhea can be inflammatory processes in the mucous membrane of the distal colon (colitis), a decrease in the secretory function of the stomach (achilia) with impaired digestion, and a decrease in the exocrine function of the pancreas.

Pathogenetically, there are ***4 types of diarrhea*** − secretory, hyperosmolar, hyper-exudative, hyperkinetic. Secretory diarrhea is observed in intestinal infections, terminal ileitis, short bowel syndrome, after cholecystectomy. Hyperosmolar diarrhea occurs in celiac disease, Whipple's disease, amyloidosis, lymphomas, primary lymphangiectasia, and general variable hypogammaglobulinemia. Hyperexudative diarrhea occurs in Crohn's disease. Hyperkinetic diarrhea occurs with irritable bowel syndrome, endocrine dyskinesia.

There are ***two forms of diarrhea*** − acute diarrhea and chronic diarrhea. Acute diarhea is a sudden increase in stool more than 3 times a day with a change in its consistency. The causes of acute diarrhea can be acute intestinal infections, parasitic invasions, foodborne toxic infections, poisoning with poisons (mercury, arsenic), drug poisoning, neuropsychiatric disorders, endogenous intoxication.

Chronic diarrhea is an increase in stool more than 3 times a day for more than 1 month. The causes of chronic diarrhea are inflammatory bowel diseases (ulcerative colitis, Crohn's disease), abuse of laxatives, malignant neoplasms, HIV infection, hyperthyroidism, enzyme diseases (gluten, lactase), functional disorders of motility (irritable bowel syndrome), radiation damage.

The dehydration is a serious complication of diarrhea. In this case, such a complaint as thirst appears. The clinical assessment of the degree of dehydration includes 3 degrees of severity. Mild dehydration − fluid deficit less than 5%, mild thirst is characteristic, normal blood pressure values, tachycardia is possible, mucous membranes are moist, urine output is slightly reduced. Moderate dehydration − fluid deficit 5−10 %, characterized by thirst, postural hypotension, moderate tachycardia, dry mucous membranes, oliguria. Severe dehydration − fluid deficiency of more than 10%, characterized by intense thirst, low blood pressure, tachycardia, parched mucous membranes, anuria. In the history of the disease, it should be noted whether there are false urges to defecate (tenesmus), burning and pain in the rectum during defecation, itching, prolapse of hemorrhoids, prolapse of the rectum.

Constipation (obstipatio). This term is understood as a long-term (more than 48 hours) retention of feces in the bowel; more often constipation is a consequence of the peculiarities of the diet. So, when eating plant foods, stools happen 2−3 times a day, and in cases where the meat diet predominates, it becomes rarer. Constipation can be organic and functional.

Organic constipation is usually associated with narrowing of the intestinal lumen (tumors, scars), developmental anomalies (dolichosigma, diverticulosis).

Functional constipation is divided into alimentary − the use of easily digestible food; neurogenic (reflex) − dysfunction of

the intramural nervous apparatus or the vagus nerve; inflammatory lesions of the colon (dysentery); toxic (exogenous intoxication with lead, morphine, cocaine); endocrine (with hypofunction of the thyroid gland, pituitary gland, etc.); caused by insufficiency of movements; due to weakness of the abdominal press.

It is important to clarify the nature of nutrition from early childhood; about past intoxications; hypersensitivity to certain foods; observance of meal times. Is the diet monotonous? Whether the patient smokes? Whether he drinks alcohol? Ask about past bowel diseases; about the presence of pathology of other organs; in connection with occupational hazards; about the connection of constipation with the frequent suppression of the urge to defecate for some reason.

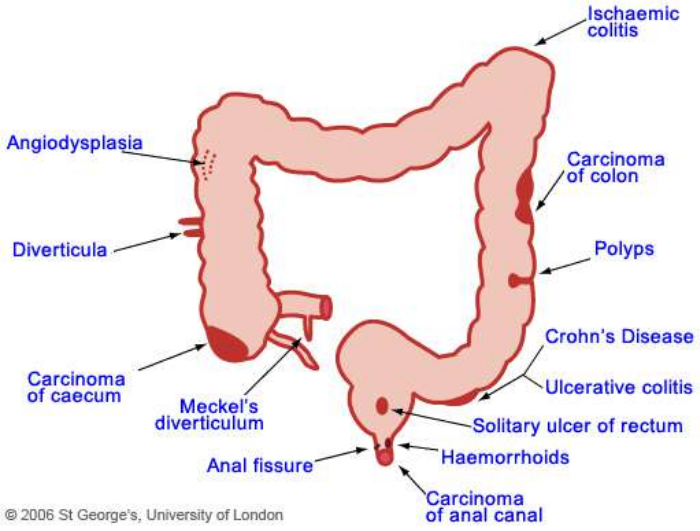


Figure 3. Possible reasons for gastrointestinal bleeding

Gastrointestinal bleeding can be an alarming complaint in patients with gastrointestinal disease. The main causes of bleeding are peptic ulcer of the stomach and duodenum, ruptures of varicose veins of the esophagus, tumors of the stomach and duodenum, erosion of the stomach, hemorrhagic gastritis, acute gastric ulcers with bleeding, ruptures of the mucous membrane of the cardiac part of the stomach (Mallory−Weiss syndrome). However, often patients do not tell the doctor about the presence of this symptom, because they do not realize its clinical significance (fig. 3).

Clinical manifestations of gastrointestinal bleeding are the following symptoms:

1) dizziness, weakness, cold sweat, pallor of the skin, tachycardia;

2) hypotension;

3) anemia, leukocytosis, reticulocytosis;

4) vomiting with blood or in the form of «coffee grounds» (hematemesis);

5) melena, or impurities of scarlet blood in the feces, or a positive fecal test for occult blood.

If an acute ulcer of the stomach and duodenum is suspected, it is important to remember that the causes of acute ulcers with ulcerative bleeding are:

1) taking non-steroidal anti-inflammatory drugs;

2) extensive burns (Curling's ulcers);

3) lesions of the central nervous system (Cushing's ulcer);

4) severe stress (myocardial infarction, surgery, sepsis, etc.);

5) old age («senile ulcers»);

6) peptic ulcer with a possible recurrence of bleeding ulcers, including those that arose against the background of active *Helicobacter pylori* gastritis.

Acute ulcers often occur in patients with chronic diseases, endogenous intoxications, after severe injuries and operations that were accompanied by stress factors: shock, collapse, hypovolemia, hypoxemia, renal and hepatic failure, etc.

Bleeding from the lower parts of the digestive tract is recognized in connection with the release of fresh red or reddish-brown blood through the rectum. For bleeding from the upper gastrointestinal tract, the presence of melena, a black tar-like stool, is characteristic. The main causes of bleeding from the lower gastrointestinal tract (in descending order of frequency) are diseases of the rectum, including hemorrhoids and anal fissures; colitis (ulcerative colitis, Crohn's disease, infections); diverticulosis, diverticulitis; intestinal polyposis; colon cancer; angiodysplasia of the colon.

Profuse bleeding from the intestine may be due to the collapse of the intestinal tumor, Crohn's disease, UC, necrosis of the intestinal wall. They can develop with some helminthiases, hemorrhoids and anal fissures. With gastric bleeding, the color of the blood in the vomit changes to brown, due to the interaction of hemoglobin with hydrochloric acid. During esophageal bleeding, the blood in the vomit is scarlet, and similar to bleeding of varicose veins − dark cherry. Bleeding in the upper intestines is manifested by black, tarry stools (melena) due to the formation of black iron sulfide. The blood released from the lower parts of the intestine is venous in nature. The dark stools can be with the use of black coffee, beets, black currants, carbolin, basic bismuth nitrate.

Hiccups − an involuntary strong short breath with a closed glottis, due to convulsive contraction of the diaphragm. Hiccups are associated with irritation of the respiratory center, phrenic or vagus nerve. Each contraction is followed by a sudden closure of vocal cords, which produces the characteristic «hic» sound (fig. 4).

Hiccups often occur in patients with hiatal hernia, esophagitis, cancer of the stomach's cardia or distal esophagus, peritonitis, peritoneal carcinomatosis, subphrenic abscess. Often repeated or persistent hiccups are associated with some kind of disease.

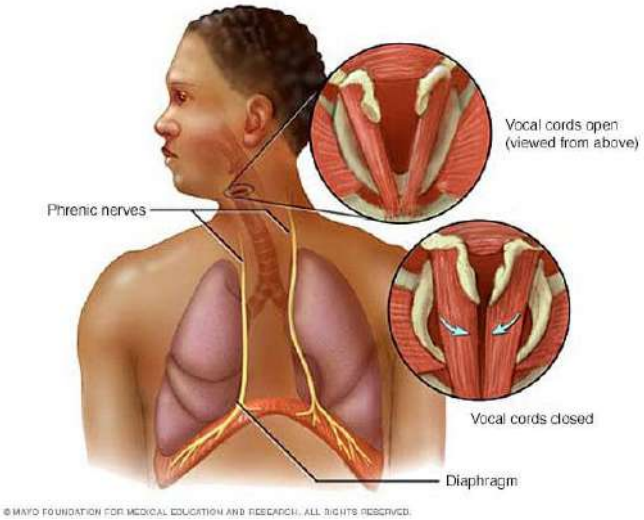


Figure 4. Mechanism of hiccups

There are three types of hiccups:

1) arising by direct irritation of the diaphragm (lower lobe pneumonia, peritonitis) or phrenic nerve (diseases of the esophagus, mediastinum, aortic aneurysm, etc.);

2) hiccups of central origin (with neurosis, stroke, encephalitis, meningitis, brain tumors, intoxications);

3) reflex hiccups − in diseases of the abdominal organs, abdominal injuries.

Dyspepsia is a term that includes most of the subjective manifestations of diseases of the digestive system. Dyspepsia is characterized by a feeling of pain in the abdomen, flatulence, «bursting» or a feeling of fullness in the stomach, heartburn, belching, dysphagia, nausea, vomiting, diarrhea, constipation, loss of appetite (anorexia) and other subjective appearances.

Conventionally, there are several variants of dyspepsia:

1) reflux-like − accompanied by heartburn, burning behind the sternum, esophageal dysphagia and belching;

2) ulcer-like − accompanied by periodic pain in the epigastrium (hungry early or late, nocturnal);

3) dyskinetic − characterized by a feeling of fullness in the abdomen, rumbling, transfusion, flatulence and, as a rule, stool disorders (diarrhea, constipation).

The diagnostic value of the symptoms of dyspepsia increases if the doctor manages to get the patient to accurately describe his condition (tab. 1).

Table 1

The main causes of dyspeptic disorders

Heartburn	**Reflux of gastric contents into the esophagus (reflux esophagitis, hiatal hernia, etc.)**
Esophageal vomiting (regurgitation)	Significant narrowing of the esophagus (strictures, tumors, etc.)
Belching	Insufficiency of the cardia with gastroesophageal or duodeno-gastric reflux
Nausea	Many causes, including gastrointestinal diseases
Feeling of fullness, pressure in the epigastrium	Many reasons, incl. as a result of tension on the intestinal wall and abdominal cavity (cirrhosis of the liver, tumors, cysts)
Meteorism	Excessive accumulation of gases in the intestines due to indigestion

Ending of table 1

Heartburn	Reflux of gastric contents into the esophagus (reflux esophagitis, hiatal hernia, etc.)
Esophageal dysphagia (feeling of a «lump» behind the sternum)	Esophageal dyskinesia (esophagitis, tumors, cicatricial strictures, external compression, aortic aneurysm, etc.)
Hiccup	Irritation of the respiratory center, phrenic or vagus nerves (hiatal hernia, cancer of the cardia or distal esophagus, peritonitis, subdiaphragmatic abscess, etc.)
Vomit	Slow gastric emptying (gastric ulcer, duodenal ulcer), duodenal obstruction (pancreatic cancer), peritoneal irritation (appendicitis, pancreatitis, cholecystitis, pyelonephritis)
Anorexia, nausea, vomiting, «rapid satiety»	Gastrostasis, bile reflux (gastric ulcer, stomach cancer)
«Stomach overflow»	Increased intraluminal pressure in the intestines (intestinal obstruction)

Anamnesis of the disease, anamnesis of life. It is desirable to describe the anamnesis in detail, characterizing the sequence, the intensity of the appearance of certain symptoms. When describing a patient's visit to a doctor or a patient's hospitalization in a hospital, it is necessary to indicate such important points as the available research results, the methods of treatment used and their effectiveness, take into account the patient's condition against the background of previous treatment, which included improvement or worsening of symptoms. It is important to clarify the facts of the presence or absence of intolerance to certain foods, to clarify the relationship of the main symptoms with food intake, stress, physical activity, defecation. The anamnesis of the

disease should not be broken into separate episodes that characterize the next hospitalization of the patient, sometimes not related to each other. It is necessary to present the data of the anamnesis sequentially, without skipping individual years of life. It should be noted how the patient previously felt after being discharged from the hospital, whether he followed the prescribed regimen and diet, whether he took any medications, and how this affected his well-being. Features of anamnesis for various diseases of the esophagus, stomach and intestines are reflected in the relevant sections.

CHAPTER 2.
STATUS PRAESENS OBJECTIVUS IN DISEASES OF THE ESOPHAGUS, STOMACH AND INTESTINES

EXAMINATION OF THE ABDOMEN

When examining the abdomen, it is important to describe its shape, participation in the act of breathing, the presence or absence of a forced posture, retraction of the muscles of the anterior abdominal wall, and the presence of visible pulsations. It is important to examine the contours of the abdomen both from a straight position and from the side. While collecting complaints and anamnesis, it is important to clarify the exact localization of the pain syndrome, to enable the patient to point to the painful area with his hand. The abdomen is conditionally divided into three levels − epigastrium, mesogastrium and hypogastrium. At each level, the right, left and central parts are distinguished, which also have their own names (fig. 5).

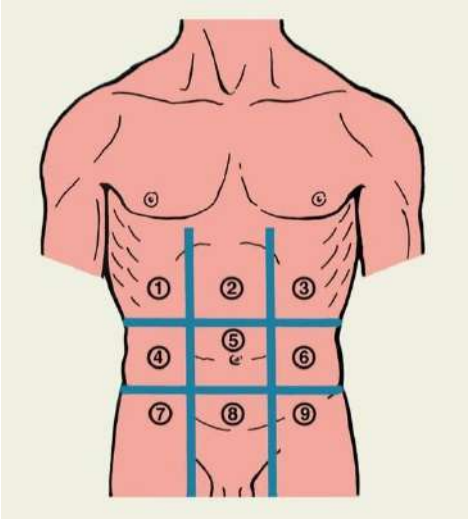


Figure 5. Areas of the abdomen, where 1 − right hypochondrium, 2 − epigastrium, 3 − left hypochondrium, 4 − right flank, 5 − mesogastrium, 6 − left flank, 7 − right iliac region, 8 − hypogastrium, 9 − left iliac region

Area 1 − **right hypochondrium**, contains projections of the right lobe of the liver, gallbladder, pyloric stomach, duodenal bulb, and head of the pancreas.

Area 2 − **epigastrium**, is the projection zone of the cardial part of the esophagus, the body of the stomach, the body of the pancreas, the right lobe of the liver.

Area 3 − **left hypochondrium**, contains projections of the spleen, greater curvature of the stomach and tail of the pancreas.

Area 4 − **right flank**, is the area of projection of the body of the duodenum, ascending colon, hepatic margin, right kidney and ureter.

Area 5 – **mesogastrium**, the projection zone of the small intestine, solar plexus, celiac trunk, transverse colon.

Area 6 – **left flank**, contains projections of the descending colon, the angle of the spleen, the left kidney, and the ureter.

Area 7 − **right iliac region**, the projection zone of the caecum, appendix, right ovary in women.

Area 8 − **hypogastrium**, the projection zone of the bladder, uterus, rectum, prostate.

Area 9 – **left iliac region**, contains projections of the sigmoid colon and the left ovary.

Examination of the abdominal organs must be carried out in a vertical and horizontal position. In a vertical position, the shape of the abdomen is assessed, which is largely determined by the person's belonging to a certain constitutional type. So, asthenics usually have a small belly with a small epigastric region, an acute epigastric angle. On the contrary, hypersthenics are characterized by a large belly, with a large epigastric region and an obtuse epigastric angle. Pay attention to the pecularities of the shape of the abdomen, whether there are retractions, protrusions of the abdomen forward (fig. 6). Uneven and asymmetrical protrusion of the abdomen is most often observed with an increase in internal organs or the development of neoplasms in the abdominal cavity,

as well as with the occurrence of large tumors and cysts of the small pelvis. The protrusion is observed with excessive development of the subcutaneous fat layer, obesity. In addition, a symmetrical protrusion may be due an excessive accumulation of gases in the intestines, as well as the presence of fluid in the abdominal cavity (ascites). Small ascites lead to protrusion of the abdomen in the lower section during a vertical examination of the patient.

The accumulation of a large amount of fluid in the abdominal cavity (ascites) causes a symmetrical increase in the size of the abdomen, and in the supine position, the fluid causes stretching of the lateral flanks of the abdomen − the «frog belly». With severe ascites, a protrusion of the navel is observed (fig. 6). With cirrhosis of the liver, the syndrome of portal hypertension often visualizes a network of subcutaneous tortuous, often nodularly altered vein anastomoses, which is called the «jellyfish head».

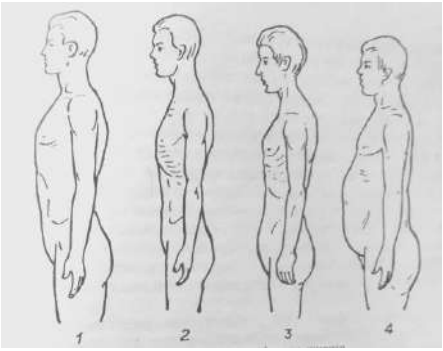


Figure 6. Various forms of the abdomen (according to A. Y.Gubergrits): 1 − normal stomach; 2 − retracted abdomen; 3 − lowered abdomen with splanchnoptosis; 4 − protruding abdomen

Naturally the increase in the size of the abdomen during pregnancy. Protrusion of the abdomen only in the lower section,

the so-called «pendulous abdomen», is observed with weak abdominal muscles, as well as in multiparous women. An atonic and very large, overfilled bladder can also be the cause of the protrusion of the abdomen in the lower floor. It is important to identify, when examining the abdomen, a pronounced venous network, which is often venous collaterals, formed due to difficulty in the outflow of blood through the system of the portal and inferior vena cava. At the same time, the saphenous veins meander like snakes, go from the navel to the chest. The phenomenon is called the head of a jellyfish (Caput medusa, Medusa head) (fig. 7).

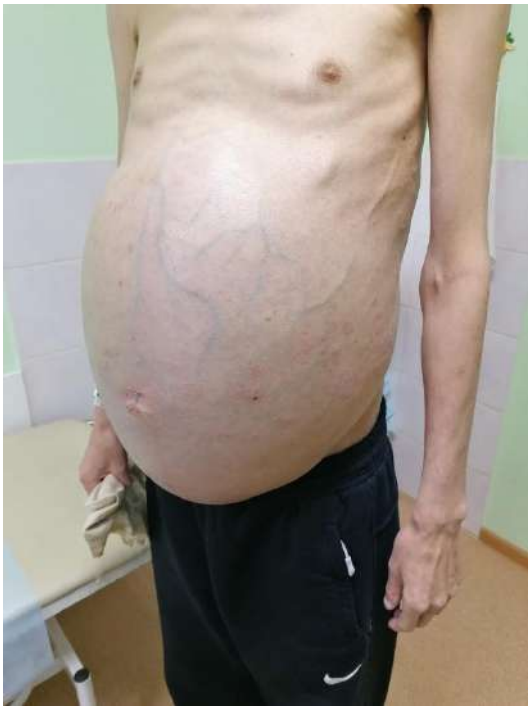


Figure 7. The abdomen, enlarged due to ascites, the network of dilated saphenous veins is well visualized

An inverted abdomen occurs primarily in general malnutrition, such as cancer cachexia, endocrine disorders, anorexia nervosa, and in patients with intestinal infections accompanied by debilitating diarrhea.

With a significant narrowing of the pylorus, it is possible to detect a protrusion in the upper floor of the abdomen, which, as a result of powerful gastric peristalsis, can move from left to right. Also, if there is an obstruction in any part of the large intestine (for example, compression by a tumor, compression by intraperitoneal adhesions, volvulus, etc.), bloating is observed along the intestine located above the obstruction, often with visible peristalsis. Identification of postoperative scars is of great diagnostic importance.

When examining the abdomen, it is necessary to evaluate the respiratory excursion of the anterior abdominal wall. Sometimes noticeable peristalsis of the stomach and intestines, pulsation of large vessels. The lag of the abdomen during the act of breathing in a limited area indicates the presence of local peritonitis (perivisceritis), which is of great diagnostic value.

Normally, in the lean patients there is a noticeable pulsation of the abdominal aorta. Pronounced pulsation in the epigastric region can be observed due to the contraction of the enlarged and hypertrophied right ventricle (cardiac shock) or pulsation of the abdominal aorta. At the same time, the pulsation caused by the right ventricle is better seen under the most xiphoid process, especially with a deep breath, in the standing position of the subject. The pulsation of the abdominal aorta is better seen a little lower, in the patient's supine position, especially on exhalation. Pulsation of the liver can be secondary (transmitted from another organ) and true. Transmitted pulsation is due to the transmission of heart contractions to the liver. In this case, the entire mass of the liver moves in one direction. True pulsation is expressed in the alternation of an increase and decrease in the volume of the liver

and is observed with insufficiency of the aortic valves, while the swelling of the liver coincides in time with the apex beat. With tricuspid valve insufficiency, a true venous pulsation of the liver is noted, which occurs due to blood regurgitation through an open opening from the right ventricle to the right atrium, and from the right atrium to the inferior vena cava and hepatic veins, which causes swelling of the liver. If you ask a patient lying on a couch to raise his head a little, it will cause symmetrical tension of the straight muscles of the abdominal wall.

In thin patients, under the right costal arch, the lower edge of the enlarged liver may be noticeable. The protrusion of epigastrium may be associated with a tumor of the stomach, pancreas or an aneurysm of the abdominal aorta. Under the xiphoid process, a pulsation of a hypertrophied right ventricle can be seen with an increase in the right heart, for example, in chronic cor pulmonale (pulsation sub scrabiculo cordis). Peristaltic intestinal waves become especially noticeable in intestinal obstruction («herring symptom» by I.I. Grekov). When straining, hernias of the white line of the abdomen and umbilical hernias become more noticeable. Postoperative scars also become a place where hernias can potentially occur. If postoperative scars are found in a patient with cramping abdominal pain, adhesive intestinal obstruction should be suspected.

Whitish long scars − striae on the skin of the abdomen may remain after childbirth, after resolution of massive ascites, treatment of obesity. Pink-purple striae are more common in Itsenko-Cushing's syndrome.

When examining the abdomen in the horizontal position of the patient, it is important that the couch must be rigid. The same changes are noted as in the vertical position. Against the background of bloating due to flatulence, increased intestinal peristalsis is better seen. In the complete absence of intestinal peristalsis in a horizontal position, it is necessary to exclude diffuse peritonitis. A characteristic symptom of a perforated

gastric or duodenal ulcer is the immobility of the anterior abdominal wall in the epigastrium.

ABDOMINAL PALPATION

Palpation of the abdomen is best done on an empty stomach, after emptying the intestines, in the position of the patient on his back, on his side and standing. The couch should be flat, moderately soft with a low headboard. It is better to place the head end of the couch in the opposite direction from the window and the light source, so that the patient's face and stomach are well lit and accessible to the doctor's visual control. The doctor's chair − right-handed − is located on the right side of the patient, at the level of his pelvis. The height of the chair should correspond to the level of the couch or bed. The doctor sits down parallel to the position of the patient, facing his face. The patient should breathe through the open mouth, evenly, calmly, mainly with the diaphragm, however, it is necessary to ensure that diaphragmatic breathing is not accompanied by tension in the abdominal muscles.

The doctor's hands should be warm. Cold hands are warmed by hot water, by the radiator or by rubbing the palms. Palpation with cold hands is extremely unpleasant for the patient, it causes a reflex spasm of the muscles of the abdominal wall, which makes it difficult to study, in addition, the tactile ability of cold hands is significantly reduced.

The main goals of superficial palpation of the abdomen:

1) assess the degree of participation of the abdominal wall in the act of breathing;

2) determine the tone of the abdominal wall, the degree of its tension;

3) exclude or detect hernial protrusions of the abdominal wall, hernial gates in the area of postoperative scars, divergence of the rectus abdominis muscles;

4) exclude or identify total or local pain;

5) exclude or identify tumors of the abdominal wall;

6) exclude or identify a significant increase in the abdominal organs;

7) exclude or identify large tumors of the abdominal cavity.

Based on the results of superficial palpation, one can roughly judge the nature of the pathological process, its localization, prevalence and severity. Before superficial palpation of the abdomen, and during its implementation, first of all, you need to pay attention to the condition of the skin and subcutaneous tissue. They are evaluated according to general rules: temperature, wetness, turgor, thickness of the fat fold, presence of pain. In healthy people, there is no significant palpatory difference in the condition of the skin and subcutaneous tissue of the abdomen with other parts of the body. The skin on the abdomen is soft, mobile, with the exception of the navel, where there is retraction. Here the skin is fused with the underlying tissues.

The subcutaneous fat of the abdomen, especially in women, is loose, more developed in the lower abdomen. After examining the condition of the skin and subcutaneous tissue, a palpatory assessment of the participation of the abdominal wall in the act of breathing is carried out. This complements the visual observations. To do this, the doctor's hand is sequentially superimposed on symmetrical sections of the abdominal wall, from the hypochondrium to the iliac regions, while assessing the amplitude of the abdominal wall oscillations and hand movements during each respiratory cycle. Usually, observing two cycles is sufficient. The depth of breathing is regulated by the doctor. Normally, the amplitude of oscillations of the abdominal wall in

symmetrical areas is the same, it is greater in the epigastrium and less in the hypogastrium. In right-handers with very developed abdominal muscles on the right, the respiratory movements of the abdominal wall may be less than on the left. Superficial palpation of the abdomen is carried out in two successive versions: superficial indicative and superficial comparative. Palpation begins from the bottom of the abdomen, comparing the left and right iliac regions, then the lateral and hypochondriac regions (fig. 8). Each time the arm is placed, the terminal phalanges should face laterally. Comparative superficial palpation of the abdomen has the following goals: to evaluate and compare the condition of the anterior abdominal wall in symmetrical parts of the abdomen on the left and right, as well as epigastrium, mesogastrium and hypogastrium with each other.

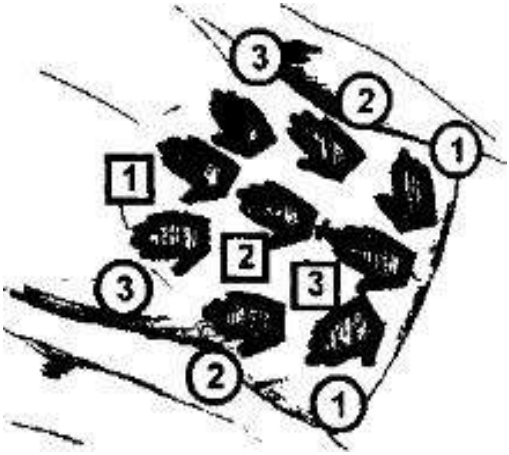
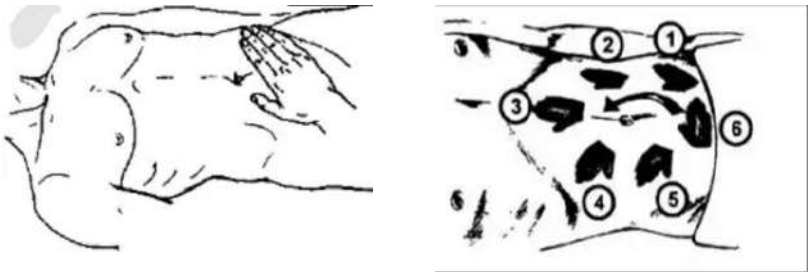


Figure 8. Scheme of comparative superficial palpation of the abdomen

After superficial comparative palpation, the method of superficial approximate palpation is used, with the help of which it is possible to determine soreness, irritation of the peritoneum

(Shchetkin-Blumberg symptom), divergence of the abdominal muscles, the presence of a white line on the abdomen, tension of the abdominal wall in the stomach area, the presence of muscular protection (defence musculare). During superficial palpation, soft terminal phalanges of the hands are used to apply light pressure (without immersion) to the abdominal areas, starting from the left inguinal region and ending with the epigastric region. In the presence of pain of a certain localization, this area is examined last (fig. 9).



A *B*

Figure 9: *A* – beginning of superficial palpation, correct hand position and direction; *B* – scheme of further superficial palpation with the correct setting of the hand

Normally, the anterior abdominal wall is soft, supple, painless on palpation. The abdominal press is well developed. In the presence of pain, its prevalence and the concomitant reaction of the muscles of the anterior abdominal wall are determined. To detect local pain, it is recommended to use light tapping with a bent finger on various parts of the abdominal wall (Mendel's symptom).

Then you need to ask the patient to raise his head, inhale and strain. At the same time, the doctor places the tips of the

closed and slightly bent fingers of the right hand along the anterior midline and feels the white line of the abdomen from the xiphoid process to the pubis (fig. 10). It is allowed to use both hands of the doctor side by side. Methods of deep palpation of the abdomen are covered in this manual in the sections on diseases of the stomach and intestines. The most important task of palpation is to identify tumors of the abdomen.

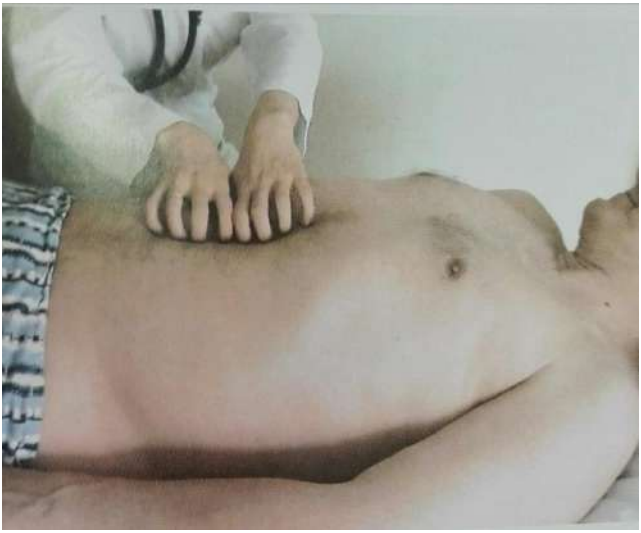


Figure 10. Palpation of the white line of the abdomen of the anterior abdominal wall in order to detect diastasis of the rectus abdominis muscles

Tumors of the anterior abdominal wall are superficially easily detected during examination, clearly palpated and fixed when the muscles of the anterior abdominal wall are tensed. When the muscles contract, the tumors do not disappear, as happens with intraperitoneal tumors. During respiratory excursions, tumors of the anterior abdominal wall move in the

anterior-posterior direction with the protrusion of the abdominal press during inhalation and sink during exhalation.

Retroperitoneal tumors are distinguished by close contact with the posterior wall of the abdominal cavity, are inactive during breathing and palpation, and are always covered by the intestines or stomach. An exception to mobility is small tumors of the kidneys and tail of the pancreas.

Tumors located intraperitoneally have great respiratory and passive mobility. The closer they are to the diaphragm, the more breathing they have. If inflammation of the peritoneum develops around an intraperitoneal tumor, then they lose their mobility and adhere tightly to the surrounding tissues**.**

When palpating the abdomen and detecting painful areas, it is important to understand which organs the patient may have diseases, as well as what nature these diseases can have. In the case of pain in the epigastrium, there can be a stomach ulcer, gastritis, duodenal ulcer. Pain in the mesogastric region around the navel − infectious gastroenteritis, Crohn's disease, lymphoma, intestinal obstruction. Pain in the epigastrium and right hypochondrium and right lumbar region − cholelithiasis, cholecystitis, cholangitis. Pain in the epigastrium, left hypochondrium, left lumbar region − pancreatitis, pancreatic cancer. Pain in the right hypochondrium − hepatitis, cirrhosis of the liver. Pain in the hypogastrium − infectious colitis, ulcerative colitis, Crohn's disease, intestinal tumors, irritable bowel syndrome.

PERCUSSION OF THE ABDOMEN

One of the methods of percussion of the abdomen − percussion according to Mendel − determines the sensitivity of the abdominal wall. Percussion is carried out with the middle finger of the right hand along the upper sections of both rectus abdominis muscles abruptly, while in pathological cases (gastric ulcer or duodenal ulcer), pain is noted at the site of impact, sometimes very sharp − due to the viscerosensory reflex due to increased sensitivity of the parietal sheet of the peritoneum in the place corresponding to the affected body. You will read about the features of abdominal percussion in various diseases in the following chapters.

Diagnosis of ascites

It is possible to verify the presence of free fluid in the abdominal cavity by invoking fluid fluctuation. For this purpose, the palmar surface of the left hand is tightly applied to the right lower abdomen of the patient standing facing the doctor. A light blow is applied with the right hand, preferably in the form of jerky clicks on the surface of the left lower abdomen. In the presence of fluid in the abdominal cavity, the left palm of the doctor feels a wave (fluctuation of the fluid). However, the similarity of the «wave» can be observed in obese individuals, with sagging abdominals. In this case, it is advisable to involve an assistant who sets the brush with the edge of the palm on the midline of the abdomen, between the hands of the doctor. This «diaphragm» delays the transmission of vibrations caused by the shaking of the abdominal wall, and does not affect the vibrations caused by fluid fluctuation (fig. 11).

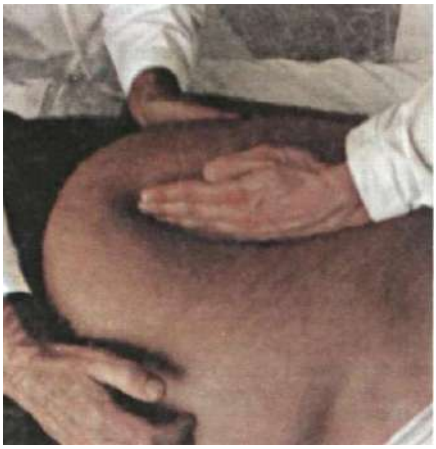


Figure 11. Determination of ascites by the method of fluctuation with the involvement of an assistant

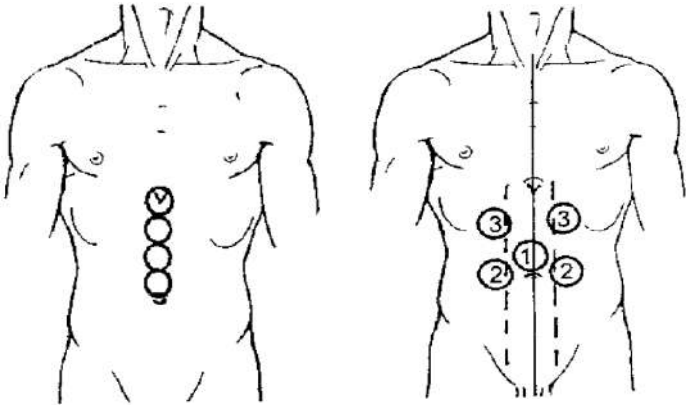
AUSCULTATION OF THE ABDOMEN

Auscultation of the abdomen also finds its use in diagnosis. It is possible to carry out auscultation of noises of intestinal peristalsis, which are normally always present. Systolic murmurs over the abdominal aorta and renal arteries, friction murmurs over the area of the liver and spleen, splashing murmurs can also be heard. With an increase in the right ventricle, its auscultation in the epigastric region may be available.

The place of listening to abdominal aortic murmurs is in the midline or slightly to the left of it above and below the umbilicus. A stethophonendoscope you need to put in place of the most perceptible pulsation of the abdominal aorta. In healthy people, tones and noises are not heard. Auscultation of the renal arteries is carried out at the following points:

1) in the depth of the umbilical region to the right and left of the navel while holding the breath after a deep exhalation;

2) over the transverse processes of the XI–XII thoracic and I–II lumbar vertebrae in the position of the patient on his side with holding his breath after a deep exhalation (fig. 12).



A *B*

Figure 12. Places of auscultation of murmurs of the abdominal aorta (*A*) and renal arteries (*B*): 1 – 2–3 cm above the navel along the white line; 2 –2–3 cm outward from the navel at the edges of the rectus abdominis muscles; 3 – at the outer edges of the rectus muscles at the level of the middle of the distance from the xiphoid process to the navel

In the phase of gastric digestion and the movement of chyme in the small intestine, a long periodic rumbling is heard. 5–7 hours after eating, rhythmic intestinal noises are heard over the caecum. With mechanical intestinal obstruction, peristalsis is heard in loud, large waves. With paralytic intestinal obstruction, peristalsis disappears, and when an ulcer perforates with secondary paralysis of the intestine, the so-called «deathly silence» occurs. In patients with fibrinous peritonitis, during respiratory movements over the sites of inflammation, a peritoneal friction noise, resembling a pleural friction noise, can be heard.

CHAPTER 3.
THE MAIN FEATURES OF THE CLINICAL PICTURE OF DISEASES OF THE ESOPHAGUS, THE VOLUME OF DIAGNOSTIC STUDIES

Esophagus − a tubular organ located between the pharynx and stomach, begins at the level of the VI cervical vertebra, descends into the posterior mediastinum and runs along the spine in the chest cavity, where it enters the abdominal cavity through the esophageal opening of the diaphragm. Anterior to the esophagus are the trachea and the aortic arch. The main function of the esophagus is the act of swallowing. The rate of passage of food through the esophagus depends on its consistency. Liquid food passes in 2−3 s, solid food in 6−8 s. The main complaints in diseases of the esophagus are dysphagia, pain, esophageal vomiting, regurgitation, salivation, putrid breath, heartburn and bleeding.

Dysphagia is the most common symptom of esophageal disease. The patient feels the delay of the lump of food, sometimes the lump stops and even pain and fullness. Pain behind the sternum can resemble angina pectoris (pressing, bursting), occur not only in connection with food intake, but also during stress. Dysphagia appears immediately after a foreign body enters the esophagus, and can also occur with a burn of the esophagus, which develops gradually with compression of the esophagus from the outside (for example, an aortic aneurysm, a mediastinal tumor, a package of enlarged lymph nodes, etc.). Thus, dysphagia may be due to functional narrowing of the esophagus and organic narrowing of the esophagus.

Functional narrowing is due to a reflex spasm of its muscles due to a violation of the innervation of the esophagus in neuroses. Sometimes solid food passes more freely than liquid food.

Functional dysphagia can occur with very fast food, excitement, with a frequency of several times a day to 1−2 times a month and disappear after taking myotropic antispasmodics.

Organic narrowing develops gradually, progressing with cancer, cicatricial stenosis of the esophagus, when the passage of solid food is difficult at first, and then liquid.

It is necessary for the patient to clarify which food intake causes discomfort when swallowing − liquid or solid. The short (less than 3 months) and progressive nature of dysphagia is characteristic feature of a malignant tumor. The presence of regurgitation and heartburn gives reason to suspect reflux esophagitis. Cough indicates the reflux of the contents of the esophagus into the tracheobonchial tree and may be the result of a broncho-esophageal fistula. Pain behind the sternum during swallowing (odynophagia) maybe a manifestation of achalasia, diffuse spasm of the esophagus, esophagitis. It is important to study nearby groups of lymph nodes to exclude a malignant tumor.

Pain − observed in acute inflammation of the mucous membrane of the esophagus (esophagitis), burns. Usually, pain is felt throughout the esophagus, both during the act of swallowing and outside it, and can radiate to the interscapular region. With achalasia cardia, pain occurs spontaneously, more often at night. The pain is intense, radiating to the back and up the esophagus, to the jaw, to the neck. Pain duration from several minutes to several hours. With hiatal hernia and gastroesophageal reflux, pain can radiate to the left side of the chest and mimic heart disease.

Esophageal vomiting − observed with a significant narrowing of the esophagus. From vomiting of gastric origin, esophageal vomiting is distinguished by a number of signs: it occurs without nausea; follows dysphagia; vomit consists of undigested, unchanged food; vomit does not contain pepsin and hydrochloric acid; vomit containing long-taken food has a putrid

odor and is observed in large diverticula of the esophagus and malignant neoplasm of the esophagus with decay.

Regurgitation is the return of part of the ingested food back into the mouth. Regurgitation occurs due to the inability to pass food through the narrowed portion of the esophagus. Regurgitation is habitual for persons suffering from neurosis as a result of a spasm of the cardia.

Salivation − observed with esophagitis, cicatricial narrowing of the esophagus, with cancerous stenosis as a result of the esophago-salivary reflex.

Putrid smell from the mouth − more often due to a cancerous tumor of the esophagus, or stagnation and decomposition of food with achalasia of the cardia.

Heartburn − a painful burning sensation behind the sternum associated with the throwing of gastric contents into the lower esophagus, occurs with gastroesophageal reflux disease (GERD).

Bleeding − may be due to an esophageal ulcer, damage to the esophagus by a foreign body, tumor decay, bleeding from the dilated veins of the esophagus.

Anamnesis in patient with esophagus disoders. When collecting an anamnesis of diseases of the esophagus, it is necessary to clarify the presence of previous injuries and diseases that could provoke a real clinical picture.

The most important facts of history in diseases of the esophagus:

1) burns of the esophagus, chemical or thermal, as the cause of cicatricial changes;

2) previous syphilis as a cause of syphilitic aortitis with the formation of an aortic aneurysm that compresses the esophagus;

3) recurring esophagospasm may be the cause of the formation of pulsion diverticula of the esophagus, which are round in shape and result from congenital weakness of the esophageal wall;

4) injuries of the esophagus.

The presence of cachexia may indicate a malignant lesion of the esophagus.

The main methods for examining the esophagus

X-ray of the esophagus with an aqueous suspension of barium sulfate is a very old and reliable examination method that allows you to see the esophagus throughout, analyze swallowing movements and the course of the food bolus. It makes it possible to identify defects in filling the folds of the esophagus with a contrast agent (fig. 13). Asymmetric narrowing of the lumen of the esophagus with uneven contours suggests the diagnosis of a tumor. Symmetrical uniform narrowing of the esophagus indicates its diffuse spasm.

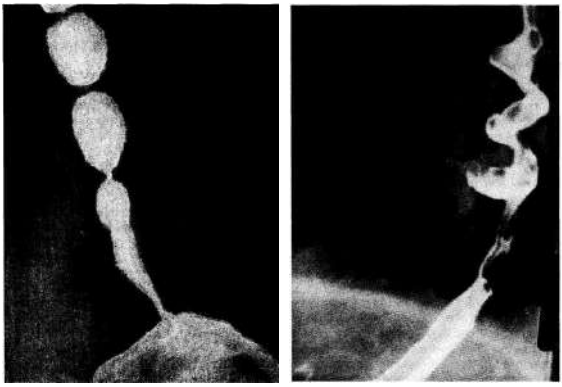


Figure 13. Radiographic pictures of the esophagus in diffuse esophagus spasm. The esophagus was contrasted with barium sulfate in both images

X-ray of the esophagus with contrast is usually followed by examination of the stomach and duodenum (duodenum).

Indications for the study:

1) suspicion of the presence of a tumor of the esophagus, stomach, duodenum, suspicion of the presence of an inflammatory process of the esophagus, stomach, duodenum;

2) the presence of clinical signs of functional changes in the esophagus, stomach, duodenum;

3) suspicion of the presence of diverticula of the esophagus, stomach, duodenum;

4) suspicion of the presence of anomalies of the esophagus, stomach, duodenum;

5) suspicion of the presence of a hernia of the esophageal opening of the diaphragm.

Contraindications for fluoroscopy of the esophagus with contrast:

1) fluoroscopy and radiography of the esophagus, stomach, duodenum cannot be combined on the same day with colonoscopy, fibrogastroduodenoscopy;

2) relative contraindications are pregnancy and severe general condition of the patient.

Pregnant X-ray examinations are carried out only according to strict clinical indications. Studies are recommended to be carried out no earlier than the second half of pregnancy, when the possibility of negative effects of X-ray radiation on the fetus is less. The study is possible only in emergency situations, when it comes to saving the life of the mother, and is also possible when deciding whether to terminate a pregnancy. The X-ray dose received by the fetus during two months of pregnancy should not exceed 1 mSv.

Preparation for the study: the study is recommended to be carried out in the first half of the day. On the eve of the study, no later than 19.00, a light dinner is allowed. On the day of the study, you can not take any food, medicine, drink water, smoke. For the study, it is necessary to take the conclusions of specialists regarding this disease, the results of ultrasound, endoscopic

studies, previous x-rays. The duration of the study is 15−30 minutes. With stenosis of the esophagus, stomach, duodenum, delayed images can be performed (after 1−24 hours).

Fibroesophagogastroduodenoscopy (FEGDS) with biopsy − allows you to visually assess focal and diffuse lesions of the esophagus, to obtain a piece of affected tissue for histological examination (fig. 14). Usually, an endoscopic examination of the esophagus is performed after an X-ray, but it can also be performed initially. The interval between barium fluoroscopy and endoscopy should be at least 12 hours. FEGDS can be performed according to planned and emergency indications.

Indications for planned FEGDS:

1) clarification of the diagnosis in the presence of a clinical picture of damage to the esophagus, stomach, duodenum, the initial section of the small intestine;

2) in order to clarify the diagnosis and biopsy of the mucous membrane;

3) all patients with anemia of unspecified etiology;

4) to assess the effectiveness of the treatment of diseases of the esophagus, stomach, duodenum, the initial section of the small intestine;

5) all patients going for planned surgical treatment to exclude possible sources of bleeding that can complicate the operation and the postoperative period;

6) therapeutic measures through the endoscope − bougienage of the esophagus, local treatment of ulcers, polyps;

7) determination of the secretory topography of the stomach (PH-metry or congo-mouth staining (can be performed before gastric resection)).

Figure 14. Fibroesophagogastroduodenoscopy procedure

There are present absolute and relative contraindications for elective FEGDS. Absolute contraindications are acute coronary syndrome, acute cerebrovascular accident, severe heart failure, severe respiratory failure, hemophilia. Relative contraindications are acute inflammatory diseases of the pharynx, tonsils, mediastinum, tracheobronchial tree; epilepsy with frequent seizures and mental illness (for such patients, FEGDS can only be performed under general anesthesia).

Indications and contraindications for examination in urgent situations are considered in a separate cases.

Indications for emergency FEGDS are:

1) bleeding from the upper gastrointestinal tract − to clarify the cause of bleeding, stop bleeding through a fiberscope;

2) foreign bodies of the esophagus − for diagnosis and removal;

3) anastomoses;

4) intraoperative gastroscopy.

Contraindications to emergency FEGDS: when the severity of the condition does not allow for surgical treatment; when the results of FEGDS do not affect the tactics of managing the patient; agonal or preagonal state of the patient. In patients with acute coronary syndrome or acute cerebrovascular accident, indications for emergency FEGDS are determined by a council of 3 medical specialists.

There is no requirement for any special preparation for a planned FEGDS. It is necessary that the patient does not take food later than 19:00 on the previous day. The study is performed under local anesthesia with solutions of dicaine, lidocaine or trimecaine (0.5−1 %). Especially for the procedure, only patients with stenosis of the esophagus and cardiospasm of stages 3−4 are prepared, they are washed out of the esophagus or stomach through a tube.

Cytological examination of esophageal lavage is a method for detecting atypical cells in esophageal tumors. The method is routinely used for suspected malignancy in patients with dysphagia. To do this, the esophagus is washed through the probe with an isotonic solution of sodium chloride. In the future, the entire volume of wash water is sent to the laboratory for cytological examination.

Daily pH-metry − a study of the functional reactions of the upper gastrointestinal tract, in particular, the study of the level of acidity directly in the esophagus, stomach or duodenum. In addition to the pH-level (acidity), impedancemetry can be carried out during the study − a measurement of the resistance level of the gastrointestinal tract environment. The method is used in the diagnosis and control of the treatment of acid-dependent diseases − gastroesophageal disease (GERD), gastritis. Daily pH-metry allows you to assess the daily production of gastric juice, taking into account the influence on the acidity of such external factors as smoking, physical activity, body position, medication.

Daily pH-metry allows clarifying the diagnosis of GERD, the frequency and severity of its manifestations, as well as the degree of aggressiveness of the effect of the contents thrown from the duodenum and stomach on the esophageal mucosa. The method allows to detect factors that provoke the formation of reflux in a particular patient, to evaluate the effectiveness of therapy. The modern possibilities of the method allow creating graphs of individual acidity rhythms with correction of the diet taking into account the data obtained. The method is simple, has minimal trauma, can be used both in the hospital and outpatient. The main indications for daily pH monitoring are apnea, heartburn, chest pain, cough, hoarseness and hoarseness, common recurrent dental caries, bad breath, subject to regular hygiene procedures. When clarifying the diagnosis of GERD, the study should be carried out in dynamics in a planned manner. An extraordinary study is carried out when extraesophageal symptoms appear (hoarseness of the voice, sore throat, pain in the sternum, paroxysmal suffocation, dental diseases, association with bronchial asthma); with refractory form of GERD; with individual selection of antisecretory treatment; as well as in the period before and after surgery, for example, during gastric resection or elimination of a hernia of the esophageal opening of the diaphragm − a disease in which the lower part of the esophagus or stomach is displaced relative to the diaphragm. Fig. 15 shows the procedure for conducting daily pH-metry.

Contraindications to the procedure are aortic aneurysm, severe maxillofacial trauma, gastric bleeding and 10 days after it stops, esophageal strictures, diverticula, esophageal burns, nasopharyngeal obstruction, severe coronary insufficiency and hypertension, blood clotting disorders.

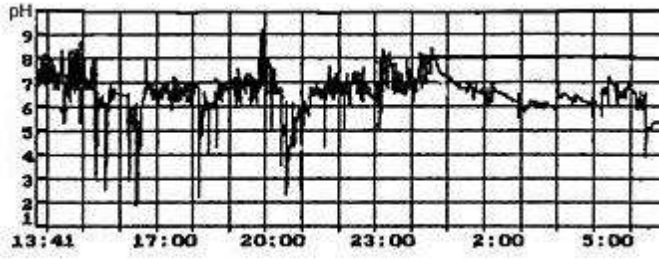


Figure 15. Carrying out daily monitoring of esophageal pH and a possible schedule of daily pH-metry

Computed manometry of the esophagus is an instrumental method for studying the motility of the esophagus, which allows you to track the work of contraction and relaxation of its muscles, which allows you to identify diseases of the esophagus such as achalasia, diffuse esophagospasm, scleroderma (fig. 16). Indications for manometry are dysphagia, chest pain not related to the heart, clarification of the type of esophageal achalasia, exclusion of contraindications for operations for hiatal hernia, clarification of the level of the lower esophageal sphincter.

The study is contraindicated in malignant neoplasms of the pharynx, larynx, esophagus, stomach, preventing the introduction of the probe; with varicose veins of the esophagus more than

5 mm; with ulcers of the esophagus and stomach with the threat of bleeding; with burns, diverticula, narrowing of the esophagus; with severe bleeding disorders; with severe forms of coronary artery disease; in mental illness and severe epilepsy.

Figure 16. Manometry of the esophagus

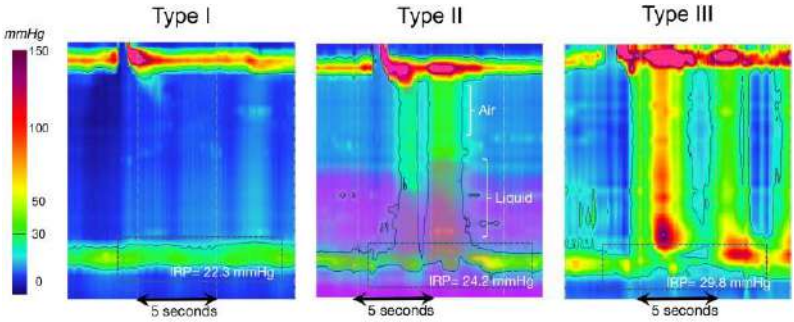


Figure 17. Esophageal manometry for different types of achalasia

Before the procedure, all patients must undergo FEGDS. Esophageal manometry very effectively allows diagnosing the type of achalasia, presenting the conclusion in the form of colorful holograms with a characteristic pattern (fig. 17).

The some diseases of esophagus

Gastroesophageal reflux disease (GERD) is an inflammatory process in the lower third of the esophagus caused by the action of gastric juice, bile, as well as pancreatic and intestinal enzymes on its mucous membrane. GERD can be a primary disease, or accompany hiatal hernia (HH). The main cause of GERD is gastroesophageal reflux due to decreased tone of the lower esophageal sphincter. With severe GERD, a stricture of the esophagus, its ulcers, as well as a malignant neoplasm can form.

A patient with GERD complains of heartburn, burning behind the sternum and their intensification after eating, in the supine position, with the torso tilted, physical exertion, and overeating. GERD is characterized by sour and bitter eructations, reflux of sour contents into the mouth, excessive salivation during sleep, and angina-like chest pain that can be relieved by nitrates, but it is important to remember that nitrates weaken the lower esophageal sphincter. Taking antacids relieves these symptoms. The examination includes FEGDS, intraesophageal pH-metry**.**

Hiatus hernia (HH) is a pathological displacement of the diaphragm through the esophageal opening into the posterior mediastinum of the abdominal esophagus, part of the stomach, and in severe cases, the entire stomach and other abdominal organs. The appearance of HH is facilitated by factors that increase intra-abdominal pressure: heavy physical labor, obesity, pregnancy, ascites, etc., as well as factors that reduce tissue elasticity and muscle tone (old age, myopathy) and endocrine diseases with severe metabolic disorders. Patients complain of heartburn, belching and other symptoms of gastroesophageal reflux (see above). The diagnosis is confirmed by X-ray of the esophagus and stomach with contrast.

Barrett's esophagus − the presence of gastric metaplasia of the esophageal mucosa above the cardia more than 2.5 cm (cells of the gastric mucosa grow into the esophagus). Complaints − as with RE. The outcome of the disease can be a malignant tumor − adenocarcinoma of the esophagus − up to 5 % of cases. Diagnostics includes FEGDS.

Ulcers of the esophagus − usually occur against the background of severe RE or Barrett's esophagus. Symptoms are similar to GERD with HH. To confirm the diagnosis, it is necessary to conduct FEGDS with targeted biopsy, histological and cytological examination of the biopsy.

Candidiasis of the esophagus is a fungal infection of the genus Candida in the esophageal mucosa. It often accompanies the course of HIV infection without antiretroviral therapy. Painful esophageal dysphagia with signs of damage to the oral mucosa by candidiasis is characteristic. The diagnosis is confirmed by endoscopic and cytological examination of targeted biopsy specimens.

Herpetic esophagitis is an acute viral lesion of the esophagus with the appearance of a characteristic blistering rash throughout, accompanied by a clinic of painful esophageal dysphagia. Diagnosis − FEGDS, biopsy, cytological examination. Often accompanies HIV-infected patients.

Esophageal strictures − narrowing of the esophagus due to prolonged, pronounced, untreated RE, as well as due to the use of NSAIDs, less often − the consequences of burns, radiation therapy. Complaints − dysphagia with heartburn belching. Diagnosis − X-ray examination with contrast, followed by FEGDS.

Esophageal cancer is a malignant tumor of the esophagus originating from epithelial tissue. In terms of prevalence, it occupies the 7th place among all malignant tumors and only in 30% of patients is detected in the operable stage. More often

occurs in the middle third of the esophagus, somewhat less often in the lower third, very rarely in the upper third. Complaints are manifestations of progressive esophageal dysphagia and weight loss. Over time, pain syndrome, hoarseness of voice and other symptoms join. The tumor grows intensively, spreading deep into the tissues of the esophagus, and then to the surrounding organs and tissues. The tumor quickly metastasizes. Intrathoracic lymph nodes (mediastinal lymphadenopathy) are often affected.

Achalasia cardia is a chronic neuromuscular disease of the esophagus, as a result of which timely opening of the lower esophageal sphincter does not occur, food overflows the esophagus, it is stretched with the formation of a giant esophagus (megaesophagus). The reason is not clear. Establishing the diagnosis requires the exclusion of esophageal cancer, gastric lymphoma, systemic scleroderma, and amyloidosis. Diagnosis includes FEGDS, esophageal manometry.

Diffuse spasm of the esophagus − episodic disorders of esophageal motility with spastic contraction of its wall, is a common cause of intermittent painful dysphagia. Spasm of the esophagus is felt as an attack of intense pain behind the sternum. The pain stops on its own after a few seconds or disappears after taking a sip of warm liquid. Diagnosis − only X-ray examination of the esophagus with barium. X-ray resembles a corkscrew – «corkscrew-shaped esophagus» (fig. 13).

Diverticula of the esophagus are protrusions of the wall of the esophagus that communicate with its lumen. They can be true, containing all layers of the esophageal wall, false – containing only the mucosa of the esophagus. Zenker's diverticulum – located on the back wall of the pharynx and esophagus, is characterized by esophageal dysphagia and regurgitation. Diagnosis – X-ray examination with barium. Diverticula in the middle part of the esophagus are often not felt sick, but only in

case of inflammation (diverticulitis) pain syndrome, dysphagia, regurgitation, subfebrile condition appears.

Foreign bodies of the esophagus – accidentally or intentionally swallowed objects. Elderly people, children, the mentally ill and drug couriers are more likely to swallow. Complaints are not always collected. There may be pain, excessive salivation, regurgitation. Diagnosis – examination of the oral cavity, FEGDS with extraction of foreign bodies. Packages of narcotic substances from drug couriers can cause obstruction of the esophagus and pylorus. Damage to the package causes resorption of the drug with the appearance of a poisoning clinic. Thus, heroin poisoning is characterized by constriction of the pupils, shortness of breath, hypoglycemia, pulmonary edema and coma. For diagnosis and treatment, urgent FEGDS is necessary.

Rupture of the esophagus – an attack of severe pain behind the sternum, often occurs after intense vomiting (spontaneous rupture), during endoscopic procedures, chest injuries. The diagnosis is established radiographically with barium contrasting of the esophagus. The disease begins spontaneously, the clinic grows intensively, the exclusion of myocardial infarction, exfoliating aortic aneurysm, perforated gastric ulcer, acute pancreatitis, spontaneous pneumothorax is required.

CHAPTER 4.
FEATURES FOR THE DIAGNOSIS OF CERTAIN DISEASES OF THE STOMACH AND DUODENUM

The stomach is an organ of the digestive system that borders the esophagus at the top and the duodenum at the bottom. The handicap and volume of the stomach are not constant. Conventionally, 4 parts of the stomach are distinguished – cardiac, body, fundus and pylorus (pyloric part) (fig. 18).

The stomach ensures the accumulation of food, its initial digestion and partial absorption, and most importantly, the evacuation of the contents into the duodenum. The duodenum is directly adjacent to the stomach and is the initial section of the small intestine. In a healthy person, the length of the duodenum is on average 30 cm, the shape of the intestine is often horseshoe-shaped. There are four parts of the duodenum: the upper (bulb), descending, lower horizontal and ascending. In the descending is the papilla of Fater (large papilla of the duodenum), which is the final part of the common bile and pancreatic ducts.

Diseases of the stomach and duodenum include various pathological processes that affect different parts of stomach and duodenum, different layers of the organ wall (mucous, submucosal, muscular, serous). Diseases of the stomach and duodenum can be of both functional and organic origin. Functional include various disorders of motor function (atony, aerophagia) and secretory functions (functional achylia, functional hypersecretion). Organic diseases include gastritis, peptic ulcer and tumors (benign and malignant).

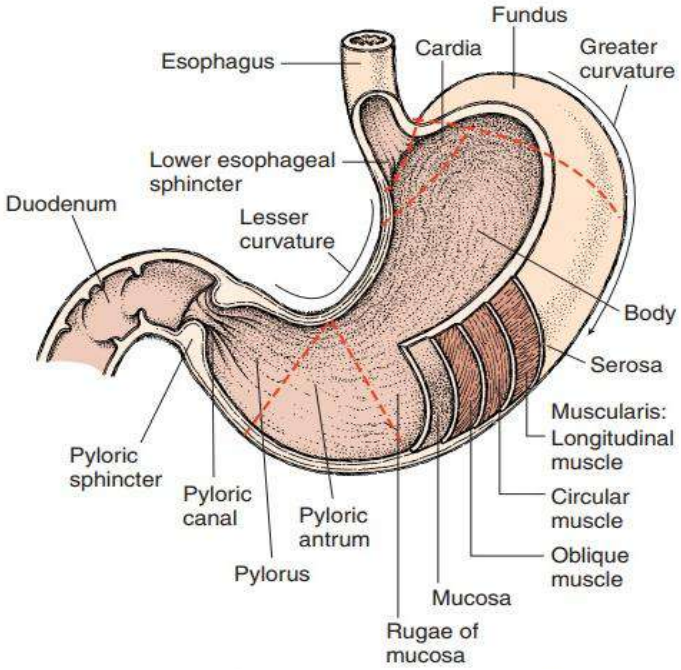


Figure 18. The structure of a stomach

The main complaints in diseases of the stomach and duodenum

Appetite disorders – increase (bulimia) and decrease (anorexia).

Anorexia is more common in acute gastritis and stomach cancer. Bulimia – with peptic ulcer, especially with the localization of the ulcer in the duodenum.

Abstinence from food due to fear of pain (sitophobia) – often in people with stomach ulcers, despite their increased appetite.

Perversion of appetite – addiction to inedible things (coal, chalk, kerosene, etc.) – can be observed in pregnant women, in those suffering from achlorhydria, and also accompany brain tumors and various endocrine disorders.

Aversion to meat – often occurs in patients with cancer of the stomach and other organs.

Perversion of taste – an unpleasant taste in the mouth and a dulling of taste sensations. Often this is associated with pathological processes in the oral cavity (carious teeth, chronic tonsillitis), and a coated tongue can also cause an unpleasant taste in the mouth.

A burp is a sudden, sometimes noisy release of air from the mouth that has accumulated in the esophagus or stomach. Belching can be only air (lat. eructatio), or it can be with a small amount of gastric contents entering the mouth along with air – belching food (lat. regurgitatio). Sour belching – combined with hypersecretion of gastric juice. May occur during an attack of pain in peptic ulcer disease. Bitter belching – appears when bile is thrown into the stomach from the duodenum, as well as with increased acidity of gastric juice. Belching with a putrid odor is characteristic of large dilatations of the stomach, hypochlorhydria, achilia with stagnation of gastric contents, as well as stomach cancer.

Heartburn (lat. pyrosos) – a burning sensation in the epigastric region and behind the sternum. Occurs with gastroesophageal reflux due to violations of the motor-tonic and evacuation functions of the stomach, duodenum, cardiac esophagus. Heartburn often occurs with increased acidity of gastric juice, sometimes during pregnancy.

Nausea (nausea) is a reflex act associated with irritation of the vagus nerve, manifested by a feeling of pressure in the epigastric region. Nausea is often accompanied by blanching of

the skin, general weakness, dizziness, sweating, salivation, lowering blood pressure, and cold extremities. Nausea often precedes vomiting, as it serves as the initial manifestation of irritation of the vomiting center**.**

Vomiting is the evacuation of the contents of the stomach and duodenum through the oral cavity. Vomiting can be considered as a symptom of stomach disease, only in the presence of other signs of this disease. Vomiting of gastric origin is caused by irritation of the receptors of the gastric mucosa by an inflammatory process (gastritis, peptic ulcer), or by food entering the stomach that affects the stomach receptors chemically (poor quality) or physically (too cold or hot, or abundant food) by, as well as in case of violation evacuation function of the stomach (spasm or stenosis of the pylorus). It is necessary to clarify the time of onset of vomiting, the relationship with food intake, pain, to inquire about the nature of the vomit and impurities**.**

Vomiting is the evacuation of the contents of the stomach and duodenum through the oral cavity. Vomiting can be considered as a symptom of stomach disease, only in the presence of other signs of this disease. Vomiting of gastric origin is caused by irritation of the receptors of the gastric mucosa by an inflammatory process (gastritis, peptic ulcer), or by food entering the stomach that affects the stomach receptors chemically (poor quality) or physically (too cold or hot, or abundant food) by, as well as in case of violation evacuation function of the stomach (spasm or stenosis of the pylorus). It is necessary to clarify the time of onset of vomiting, the relationship with food intake, pain, to inquire about the nature of the vomit and impurities.

Pain (dolor) in the epigastrium can be associated not only with diseases of the stomach, but also with diseases of the liver,

pancreas, with the presence of a hernia of the white line of the abdomen. In addition, pain in the epigastrium can occur through a viscero-visceral reflex in acute appendicitis, myocardial infarction, damage to the diaphragmatic pleura, pneumonia, etc. It is necessary to specify the exact location of the pain – show the patient with your hand.

The following points should be clarified with the patient:

1) the place of localization of pain (ask the patient to indicate the place of pain with his hand);

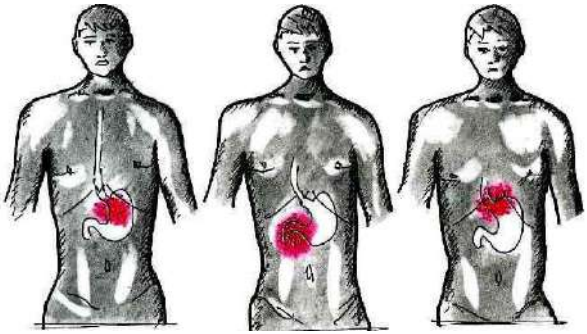
2) the nature of pain – paroxysmal, periodic (at certain hours), constant, seasonal (spring or autumn);

3) connection of pain with food intake, its quality, consistency;

4) irradiation of pain (in the back, shoulder blade, behind the sternum, left hypochondrium);

5) the nature of pain (decrease, increase, no change) after vomiting, eating, applying heat, antispasmodics, baking soda, the relationship of pain with physical stress, excitement.

Depending on the time of onset of pain after eating, they are divided into early (occur 30–40 minutes after eating), late (1–2 hours after eating), nocturnal and hungry, calming down after eating. Early pains are typical for the localization of an ulcer in the stomach, late, nocturnal or hungry pains – for ulcers located near the pylorus and in the duodenum. Localization of pain in peptic ulcer is more often noted in the midline between the xiphoid process and the navel. With a stomach ulcer, pain is felt above the navel, with a duodenal ulcer – to the right of the midline of the abdomen in the upper abdomen, with an ulcer of the cardiac section – at the xiphoid process (fig. 19).



a *b* *c*

Figure 19. Typical localization of pain in ulcers: *a* – body of the stomach; *b* – antrum, pylorus and duodenum; *c* – subcardial and cardiac parts of the stomach

Pain in peptic ulcer can radiate to the left nipple, behind the sternum, to the left shoulder blade, thoracic spine, but more often irradiation of pain indicates a complication of ulcerative disease – penetration into neighboring organs, the development of perivisceritis and solar syndrome, and may also indicate the presence of concomitant diseases – more often chronic cholecystitis and chronic pancreatitis. There is a natural connection of pain in peptic ulcer disease with the quality and quantity of food. Abundant, spicy, sour, salty, rough food always causes intense pain. Pain in peptic ulcer is characterized by seasonality – spring and autumn exacerbations. Periods of exacerbations are replaced by periods of remission. In the initial stage of the disease, the frequency of pain may not be clearly identified, then it becomes obvious, the intensity of pain increases. With the exception of hunger pains that are relieved by eating, the pains of peptic ulcers reach their maximum at the height of digestion.

With perigastritis, pain occurs immediately after taking a copious amount of food, regardless of its quality, with any physical exertion, a change in body position. The resulting

stretching of the stomach causes irritation of the nerve receptors and fibers embedded in the adhesions.

Pain syndrome with gastritis, duodenitis, peptic ulcer of the stomach and duodenum is different. So with antral non-atrophic gastritis, periodic pains occur, including hungry in the epigastrium. Pain is accompanied by symptoms of ulcer-like dyspepsia: heartburn, sometimes acid belching, constipation. Diffuse pangastritis is accompanied by a constant feeling of heaviness, fullness, fullness, frequent dull pains in the epigastrium, an unpleasant taste in the mouth, and a decrease in appetite. With duodenitis, the most common complaint is epigastric pain, which occurs 1–2 hours after eating. Pain is significantly reduced by taking antacids and food.

Gastric bleeding is an alarming syndrome that can manifest as bloody vomiting (haematemesis) or tarry stools (melaena). Under the influence of hydrochloric acid of the stomach, hydrochloric hematin is formed, therefore the contents of vomit containing blood resembles coffee grounds. However, in the presence of heavy bleeding associated with damage to a large vessel, vomit may contain a lot of scarlet blood (peptic ulcer, cancer, polyps, erosion of the stomach, etc.).

History (Anamnesis) of stomach disorders

1. We get acquainted with the nature of the patient's diet – is the rhythm of meals observed? What is the total and single amount of food eaten per day? Is the food chewed well?

2. We study the features of work and life: work in hazardous production, the possibility of occupational poisoning, alcohol abuse, smoking.

3. We clarify whether there has been a recent loss of body weight, anemia, the appearance of bloody vomiting, tarry stools?

4. We clarify whether there were any operations on the digestive tract?

5. Does the patient take drugs that irritate the gastric mucosa and also drugs that thin the blood?

6. We clarify whether there was intense stress on the eve of the development of symptoms?

Objective examination of patients with stomach diseases

A general examination may reveal cachexia (with stomach cancer, pyloric stenosis), pallor of the skin after bleeding.

When examining the oral cavity, pay attention to the absence of many teeth, which can cause a violation of the digestion of food. The presence of many carious teeth entails the entry of microbial flora into the stomach.

Examination of the gums allows you to clarify the diagnosis of gingivitis, periodontal disease, often combined with loosening or even loss of several teeth.

Superficial defects of the oral mucosa (aphthae) are found in aphthous stomatitis, and deeper defects in leukemia, agranulocytosis, and panmyelophthisis. Various rashes are often found on the oral mucosa. Fungal infections (thrush in the form of powdery-white spots of various sizes and black-brown candidal plaque) occur in HIV-infected patients (fig. 20), in debilitated patients after several courses of antibiotic therapy. Brown patchy pigmentation of the oral mucosa is characteristic of adrenal insufficiency.

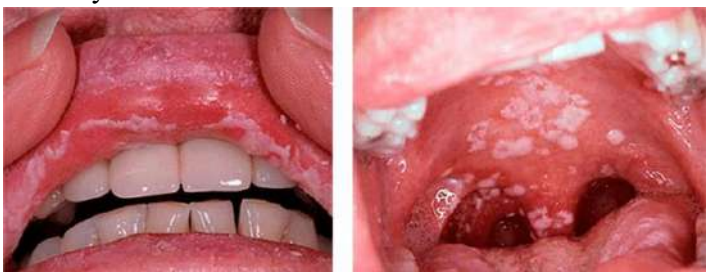


Figure 20. Candidal plaque on the oral mucosa in a patient with HIV infection

When examining the tongue, its shape and size, humidity, condition of the epithelium, and plaque are evaluated. An increase in the size of the tongue (macroglossia) has been described in a number of both hereditary (Down's syndrome, Gierke's glycogenosis) and acquired diseases (Quincke's edema, malignant tumors, acromegaly, myxedema, amyloidosis). Dryness of the tongue is characteristic of dehydration, uremia, severe febrile conditions. Inflammatory changes in the tongue (glossitis) occur as a result of a variety of reasons (tongue injury, viral or bacterial infection, hypovitaminosis) (fig. 21).

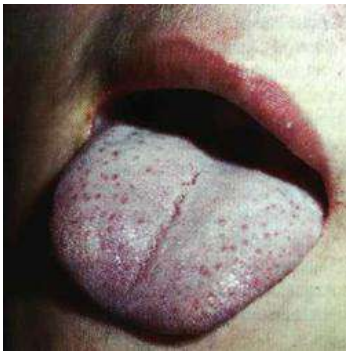


Figure 21. Dirty white (light gray) plaque on the tongue during exacerbation of chronic hyperacid gastritis

Atrophic glossitis is described in B_{12}-deficiency anemia, iron deficiency states (Plummer-Wilson syndrome), celiac disease, and chronic intestinal fistulas. «Geographical» or «furrowed» tongue is an advanced stage of atrophic glossitis (fig. 22).

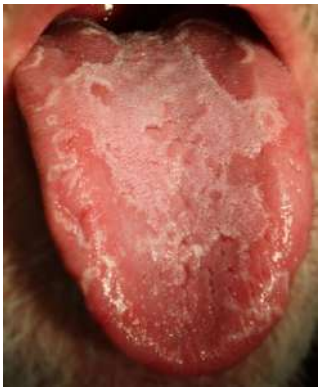


Figure 22. «Geographical» tongue in chronic atrophic gastritis

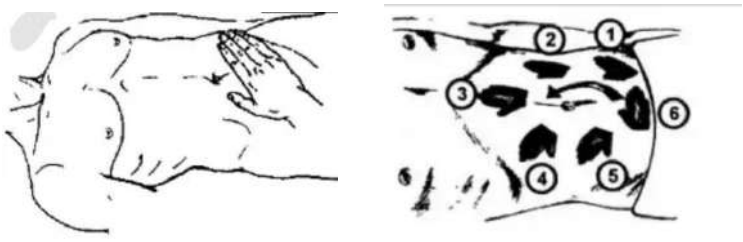
In several diseases, the tongue is coated with a gray-white coating, while in acute gastritis the tongue smells bad, in an acute abdomen it is dry, in stomach cancer and atrophic gastritis it has flattened papillae (atrophic tongue).

When examining the abdomen, it is necessary to evaluate the respiratory excursion of the anterior abdominal wall. Sometimes, on examination, peristalsis of the stomach and intestines, pulsation of large vessels are visible. The lag of the abdomen in the act of breathing in a limited area indicates the presence of local peritonitis (perivisceritis), which is of great diagnostic value.

Palpation in determination of stomach diseases

Superficial palpation of the abdomen begins after examination – with a superficial approximate palpation (fig. 22), with which you can determine pain in the epigastric region, irritation of the peritoneum (Shchetkin-Blumberg symptom), divergence of the abdominal muscles, the presence of a white line

of the abdomen, tension of the abdominal wall in the stomach, the presence muscular defense (defence musculare) (fig. 23).



A *B*

Figure 23. The beginning of the superficial palpation of the abdomen (*A*) and the algorithm for superficial palpation of the abdomen (*B*)

During superficial palpation, soft terminal phalanges of the hands are pressed (without immersion) on symmetrical parts of the abdomen, starting from the left inguinal region and ending with the epigastric region. In the presence of pain of a certain localization, this area is examined last.

Deep palpation of the abdomen is carried out according to the method of Obraztsov V.P. and Strazhesko N.D. The examiner, with four fingers folded together and slightly curved, pulls the skin of the abdomen up and carefully, on exhalation of the patient, plunges into the abdominal cavity, trying to reach the posterior abdominal wall. The stomach, being pressed against the posterior abdominal wall, slides under the fingers and «slips» out from under them. The method gives an idea of the shape and size of the stomach. Palpation of the stomach allows to detect tumors of the pylorus, greater curvature and the anterior wall of the stomach, since it is the greater curvature and the pylorus that are more accessible to palpation. Tumors of lesser curvature can be detected in the vertical position of the patient. Tumors of the cardiac part of the stomach are inaccessible to palpation.

Palpation of the stomach is carried out both in a vertical and horizontal position, since in a horizontal position it is not possible to palpate the lesser curvature and highly located tumors of the stomach. The greater curvature of the stomach is located on both sides of the midline of the abdomen 2−3 cm above the navel. The correctness of palpation of the greater curvature is confirmed by percussion and splash noise. Palpation of the greater curvature starts from the edge of the costal arch and ends 2−3 cm to the right of the midline and 2−4 cm above the navel in men, 1−2 cm in women.

The doctor's fingers are sequentially placed parallel to the studied edge of the curvature outside the body of the stomach. The skin of the abdominal wall shifts slightly forward, and the fingers sink deep into the exhalation. Having reached the back wall, they make a sliding movement outward. A large curvature, if it is palpable, is perceived as an elastic «threshold» 0,5−0,7 cm thick, painless. In clinical practice, more often the doctor is limited to palpation of the lower part of the greater curvature. The study is carried out on both sides of the midline 2−3 cm above the navel (fig. 24).

It should be borne in mind that the lower edge of the stomach can move up and down, depending on the degree of filling and the tone of the muscles of the stomach. Sometimes the transverse colon is taken as a large curvature. If the boundaries of the stomach, determined by the methods of percussion and auscultation, coincide with the results of palpation, then this is the edge of the stomach, but if the palpable edge is lower, then this is the transverse colon.

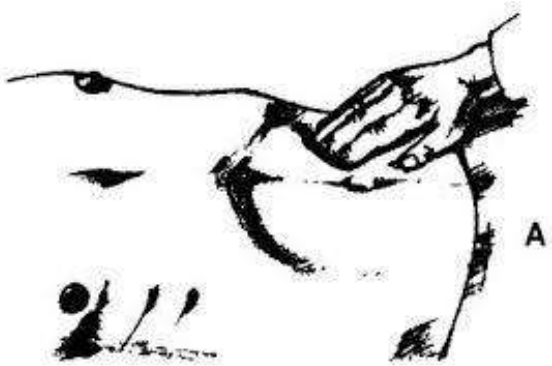


Figure 24. Palpation of the greater curvature of the stomach

Palpation of the pylorus is performed after palpation of the greater curvature of the stomach. The pylorus is located 3−7 cm above the navel and 1−2 cm to the right of the anterior midline of the abdomen. Sometimes the pylorus is covered by the left lobe of the liver and is inaccessible to palpation. The place of palpation of the pylorus is determined by the construction of a right angle between the median line and the transverse line drawn 3−4 cm above the navel, and sometimes up to 7 cm (fig. 25). After the skin is shifted forward by 1−2 cm, with each exhalation, the fingers plunge deeper. This is often difficult due to the resistance of the right rectus muscle. Having reached the back wall, a sliding movement is made across the axis of the pylorus. The pylorus, if palpable, looks like an elastic, smooth, thin cylinder as thick as a pencil. Every 40−50 seconds, it contracts and becomes dense, sometimes purring (symptom of «mouse squeak» according to V.P. Obraztsov). With pylorospasm, the pylorus is not palpable. It is recommended to start palpation 1−3 cm higher if palpation fails at the traditional site.

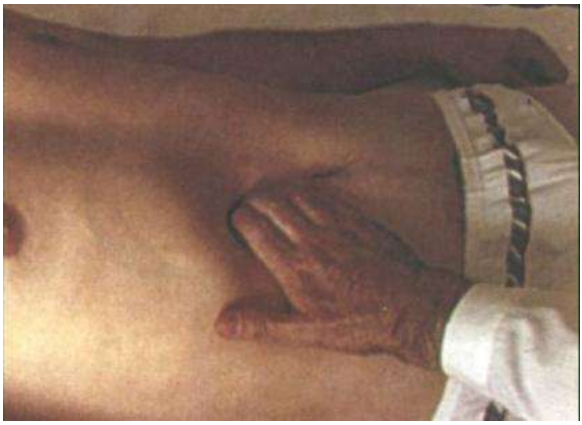


Figure 25. The site of the beginning of palpation of the pylorus

Percussion of the stomach is used to determine its lower border. By the method of quiet percussion, it is possible to establish the lower border of the stomach, revealing the different nature of gastric and intestinal tympanitis. Jerky blows with bent fingers on the epigastric region (Mendel's symptom) are performed in order to identify the involvement of the parietal sheet of the peritoneum in the pathological process. When complaining of the inability to eat a full meal, it is necessary to percussion determine Traube's semilunar space, the size of which can be significantly reduced. To determine the noise of the splash, the examiner with four bent fingers of the right hand, without tearing them off the anterior surface of the abdominal wall, produces jerky blows. The examiner's left hand fixes the abdominal muscles at the edge of the end of the sternum. Outside the lower border of the stomach, splashing noise is not heard. In healthy people, splashing noise is caused only after eating and is absent outside the lower border of the stomach. If it is determined 7−8 hours or more after the last meal, then this indicates a decrease in the evacuation capacity of the stomach. A splashing

noise to the right of the midline of the abdomen is detected with the expansion of the prepyloric part of the stomach (Vasilenko's symptom).

Auscultation of the stomach is used as the third way to determine the lower border of the stomach (fig. 26).

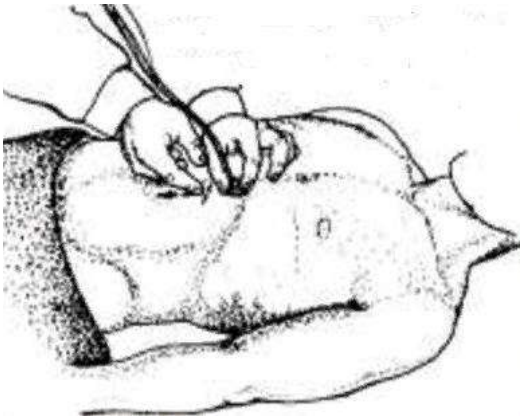


Figure 26. Determination of the lower edge of the stomach by auscultation

The doctor places the stethoscope membrane on the left rectus abdominis muscle with the left hand directly below the left costal arch. With the tip of the middle or index finger of the right hand, he makes light stroke-like movements in the direction transverse to the rectus abdominis muscles, starting from the stethoscope and moving away from it down. At the same time, a loud rustling sound is heard above the stomach, which disappears outside the stomach. Normally, the lower border of the stomach is 3−4 cm above the navel in men, 1−2 cm in women. Omission of the lower border of the stomach is observed with gastroptosis, a significant expansion of the stomach due to atony, pyloric stenosis.

Laboratory studies in diseases of the stomach have a low specificity. Diagnosis of *H.pylori* infection has gained great importance. There are the following methods: morphological, urease, respiratory, enzyme immunoassay and using PCR. Morphological − staining of bacteria in histological preparations of coolant according to Giemsa, Ghent, toluidine blue, staining of bacteria in smears-imprints of biopsy specimens of coolant according to Giemsa, Gram. Urease − determination of urease activity in the biopsy of the coolant by placing it in a liquid or gel-like medium containing a substrate, buffer, indicator. Respiratory − determination of 14C and 13C isotopes in the exhaled air, which are released as a result of the splitting of urea in the stomach of a patient under the action of urease of the bacterium *H. pylori*.

ELISA method is the determination of antibodies to *H.pylori*. PCR method − determination of *H.pylori* using PCR in feces.

With many diseases of the stomach, anemia of a different nature can be observed in the general blood test, more often − B12-deficient macrocytic, a little less often − folic acid deficiency. With bleeding − hypochromic anemia.

Instrumental diagnostic of diseases of the stomach. Step-by-step pH-metry allows you to register the pH of the stomach every centimeter, as the probe moves along the esophagus, stomach, duodenum, to identify acidification zones in them, the presence of refluxes, the severity of acid formation before and after parenteral administration of a synthetic analogue of gastrin − pentagastrin to assess the nature of secretory disorders, especially in achlorhydria

FEGDS in gastroenterological practice occupies a leading position among diagnostic methods. FEGDS allows you to assess the condition of the mucous membrane of the esophagus, stomach and duodenum: color, the smallest changes in the surface − tumors, polyps, erosion, ulcers. Allows you to assess the state of blood vessels, hemorrhages, to study the relief (character, height,

width of mucosal folds), to identify Barrett's esophagus, diverticula, refluxes, to control the healing of the damaged mucous membrane of these organs. The technique allows you to take a biopsy of the mucosa for histological examination.

X-ray and fluoroscopy of the esophagus and stomach with barium allows you to identify direct and indirect symptoms of peptic ulcer. Direct symptoms − ulcerative niches, indirect symptoms − cicatricial deformity, convergence of folds, impaired tone and motility of different departments.

Some diseases of the stomach and duodenum

Gastritis is an inflammation of the gastric mucosa in response to damage to it. There are three types of gastritis: acute, chronic and special (special) forms − granulomatous, eosinophilic, lymphocytic, hypertrophic and reactive. The most common cause of gastritis is infection of the gastric mucosa with a microbe − *Helicobacter pylori*. A little less common is non-microbial gastritis − autoimmune, alcoholic, post-resection, NSAID-induced, chemical. Often there are also mixed forms of gastritis.

Acute gastritis occurs as a result of a destructive lesion of the gastric mucosa. Accompanies the clinic of poisoning, trauma, liver and kidney failure. Among acute gastritis, acute hemorrhagic gastritis is the most dangerous.

Chronic gastritis is a chronic inflammation of the gastric mucosa with its cellular infiltration, atrophy of the glandular epithelium, intestinal metaplasia, disorders of the secretory, motor, and endocrine functions of the stomach. More than 50 % of the working population of our planet suffers from chronic gastritis.

Granulomatous gastritis is a rare sign of sarcoidosis, Crohn's disease, fungal infections, and tuberculosis. Diagnosis is based on

histological examination of the gastric mucosa. Dyspeptic disorders are possible, including vomiting, hematomesis.

Eosinophilic gastritis − very rare, due to vasculitis. Some patients have a history of allergies, eczema. The diagnosis is established histologically. Characterized by pronounced infiltration of all layers of the stomach wall with eosinophils, edema, plethora.

Hypertrophic gastritis (Menetrier's disease) is a change in the gastric mucosa in the form of giant folds resembling the convolutions of the brain. The cause of the disease is not completely known. Patients complain of weight loss, diarrhea, epigastric pain after eating, nausea, vomiting, anorexia may develop. The diagnosis is specified histologically, with the exclusion of other known diseases that cause hypertrophy of the gastric folds − gastric lymphoma, adenocarcinoma, Zollinger-Ellison syndrome, lymphocytic gastritis, eosinophilic gastritis, Kaposi's sarcoma, sarcoidosis, Crohn's disease, and others).

Lymphocytic gastritis is characterized by pronounced lymphocytic infiltration of the gastric mucosa with the formation of nodules, thickened folds. Complaints are the same as in Menetrier's disease.

Duodenitis − dystrophic, inflammatory and regenerative changes in the duodenal mucosa. There can be both without the phenomena of an atrophy, and atrophic variants. The most common cause is the defeat of *H. pylori*.

Patients complain of aching, less often cramping epigastric pain that occurs 1−2 hours after eating. The pain is significantly reduced or disappears after eating or antacids. The pain may be accompanied by hypersalivation, heaviness in the epigastrium, nausea. There is dyskinesia of the duodenum, the throwing of duodenal contents into the stomach (duodenogastric reflux), which is accompanied by bitter belching, sometimes vomiting with an admixture of bile, heartburn.

Bulbitis − inflammation of the bulb of the duodenum − is accompanied by symptoms of ulcerative dyspepsia of the duodenum. Distal duodenitis resembles the clinical signs of cholecystitis and pancreatitis. Exacerbation of chronic duodenitis is accompanied by general malaise, headaches, autonomic disorders in the form of sweating, drowsiness, increased intestinal motility 2−3 hours after eating. With a long break between meals, there may be symptoms of hypoglycemia − muscle weakness, trembling in the body, hunger, sweating. On palpation, some tension of the anterior abdominal wall in the epigastrium, moderate local pain is determined. Pain on palpation is significant only with periduodenitis or concomitant cholecystitis and pancreatitis.

Peptic ulcer of the stomach and duodenum. Peptic ulcer (PU) is a chronic relapsing disease, prone to progression, with involvement in the pathological process along with the stomach and duodenum of other organs and digestive systems, the development of complications that threaten the life of the patient. Peptic ulcer occurs in 4−5% of the population. The period of exacerbation is characterized by the presence of damage to the gastric mucosa or duodenum due to exposure to hydrochloric acid and pepsin.

Chronic active gastritis precedes the onset of recurrent gastric ulcer (GU). Chronic active duodenitis precedes recurrent duodenal ulcer (DUD). It is known that *H.pylori* persists in humans for years, and often for a lifetime. Due to their activity, they cause specific inflammation in the gastroduodenal mucosa, and against this background, an erosive-ulcerative process in the stomach and duodenum can develop and recur. *H.pylori* stimulates the production of gastrin, hydrochloric acid; reduces the production of prostaglandins, underestimating the protective properties of the mucosa; inhibits dew factors and slows down the regeneration of the mucosa.

GU develops more often in people over 40 years of age. DU is more common in people under 40 years of age. Pathogenetic factors in the development of ulcerative disease of any localization are neuro-emotional overstrain, dietary disturbances, smoking, alcohol and medication abuse, hereditary predisposition − violation of mucosal protection factors, mucosal susceptibility to *H. pylori* infection, parietal cell hyperplasia, hypervagotonia, hypergastrinemia, etc.

In the pathogenesis of DU, accelerated evacuation from the stomach, acid-peptic factor play a role. With an ulcer of the antrum of the stomach, the role of duodeno-gastric reflux, antral stasis, and a high concentration of hydrochloric acid is great. With a mediogastric ulcer, an insufficient mucous barrier, microcirculation disorders, and insufficient prostaglandin production are significant.

The most common complaint of PU is pain. Features of the pain syndrome in peptic ulcer were discussed above. It is important to understand that with an ulcer of the pyloric stomach and duodenal bulb, pain occurs 1.5−2 hours after eating, almost on an empty stomach, the pain is often nocturnal, it is stopped by antacids and food. With an ulcer of the body and cardia of the stomach, the pain is early, occurs 30−60 minutes after eating, stops after emptying the stomach. With a post-bulbar ulcer, pain is intense, throbbing, occurs 3−4 hours after eating, and often that is stopped only by drugs.

The course of PU is associated with the development of **ulcerative dyspepsia syndrome**, which includes the following manifestations:

- bad taste in the mouth;
- empty belching of air;
- feeling of pain and/or discomfort in the epigastric region, heaviness, overflow;
- heartburn, sometimes very unpleasant;

- at the height of pain, nausea and vomiting may occur;

- possible development of intestinal dyspepsia with flatulence, constipation, «sheep» feces.

Astheno-vegetative syndrome in PU is also pronounced and manifests itself in the form of anxiety, hypochondria, irritability, weakness, egocentrism and demonstrative behavior appear less often. Also, the structure of the syndrome includes signs of autonomic dystonia − arterial hypotension, pulse lability, acrocyanosis, cold hands, hyperhidrosis. In diagnosis, the leading method is FEGDS with targeted biopsy and further histological examination of the biopsy. X-ray diagnosis of PU is of auxiliary importance. The course of PU is cyclic, periods of exacerbation are replaced by periods of remission. It is known that relapse of GU is preceded by exacerbation of chronic active gastritis, and exacerbation of DU is preceded by exacerbation of chronic active duodenitis associated with *H. pylori*.

Complications of peptic ulcer in the clinic of internal diseases acquire independent significance, as they involve many organs and systems in the pathological process. The following most typical complications are distinguished: ulcerative bleeding; ulcer perforation; ulcer penetration; peritonitis; perivisceritis (perigastritis, periduodenitis); pyloric stenosis, malignancy. All of these complications are treated with the use of surgical methods of treatment. Often, against the background of PU, reactive hepatitis and reactive pancreatitis occur.

CHAPTER 5.
THE MAIN CLINICAL SIGNS OF DISEASES OF THE SMALL AND LARGE INTESTINES, DIAGNOSTIC VOLUME, FEATURES OF THE CLINICAL PICTURE OF SOME DISEASES

Considering the clinical symptoms in diseases of the intestine, it is necessary to indicate the commonality and difference in the structure and functions of the small and large intestines. The small intestine consists of three sections − the duodenum (diseases of the duodenum discussed above), the jejunum and the ileum. From the inside, throughout the entire length of the small intestine is covered with villous epithelium, the maximum number of villi is located in the jejunum. Chyme enters the small intestine at a rate that provides intracavitary and parietal digestion and absorption. After the assimilation of nutrients, unabsorbed food residues are evacuated into the large intestine. Motility of the small intestine prevents the entry of colonic microflora into it.

The pathology of the small intestine is manifested by violations of the motor function of the intestine, abdominal pain, flatulence, intestinal bleeding, malabsorption, maldigestion. Separately, **the syndrome of intestinal dyspepsia** is distinguished, which includes such concepts as flatulence, rumbling, diarrhea, constipation and unstable stools − alternating diarrhea and constipation. Violation of the motor function of the small intestine is manifested in a decrease in its propulsive activity, for example, with intestinal paresis with peritonitis, or with intestinal obstruction.

One of the most common complaints in bowel diseases is abdominal pain. When the patient complains of abdominal pain, their localization, irradiation, intensity, nature, duration and

conditions that lead to pain relief should be established. Intestinal pain differs from gastric pain in the absence of a strict connection with food intake; the exception is the inflammatory process in the transverse colon (transversitis), in which abdominal pain occurs immediately after eating; the pathogenesis of pain in this case is associated with reflex peristaltic contractions of the transverse colon when food enters the stomach. It is possible to associate pain with the act of defecation, so pain can occur before, during and rarely after a bowel movement. a close connection of pain with the act of defecation (may occur before, during and rarely after a bowel movement; Often the pain is relieved after a bowel movement or gas passage.

Colicky pains are characterized by short repeated attacks that begin and end abruptly. The pains can very quickly change their place, and yet they are mainly localized around the navel.

Aching pains are sometimes persistent, aggravated by exertion and coughing. When the peritoneum is involved in the inflammatory process, pain is accompanied by pronounced muscular protection.

It is important to accurately determine the localization of pain. Pain in the right iliac region occurs with appendicitis, and pathology (tuberculosis, cancer, inflammation) of the caecum (typhlitis). Acute pain in the left lower abdomen is observed with obstruction and inflammation of the sigmoid colon (sigmoiditis). Pain in the navel can occur with inflammation of the small intestine (enteritis), inflammation and cancer of the colon. Pain in the perineum, especially at the time of defecation, combined with the presence of blood in the stool, is a characteristic feature of rectal disease (proctitis, cancer).

Pain can radiate to the left half of the chest with damage to the splenic angle of the colon and its descending section (taken for angina attacks), with appendicular colic − to the right leg, with acute damage to the lower left sections of the colon (dysentery)

pain radiates to the sacral region. Contribute to the cessation of pain, the use of heat, antispasmodics, the passage of gases, the release of the intestines.

Abdominal pain in diseases of the small intestine often occurs in the navel (mesogastrium), but may not be limited to this area. With intestinal obstruction, they are colicky or intermittent in nature, due to stretching of the intestinal lumen, tension of its wall, inflammation of the surrounding peritoneum. As the bowel stretches and muscle tone decreases, the intensity of pain decreases. In acute inflammation of the small intestine with involvement of the visceral and parietal layers of the peritoneum, the pain becomes constant, aching, with the appearance of rigidity, peritoneal symptoms, and sharp pain in the abdominal wall.

At the base of intestinal pain are: a violation of the patency of the intestines and a disorder of their motor function. More often, intestinal pain appears against the background of intestinal spasm (spastic pain), or distension of the intestine with gases (distension pain). Often both pain mechanisms are combined.

Distension pains associated with tension and irritation of the mesentery differ from spastic pains in two main features: 1) lack of periodicity (long-term and gradually dull with prolonged swelling); 2) fairly accurate localization.

With intestinal obstruction (complete or partial), colicky pains are combined with almost constant pain in the abdomen. They are characterized by localization in the same place (the area of the navel and colon) and an increase in connection with intestinal peristalsis.

With appendicular colic, the pains are first localized around the navel and in the epigastric region, and then after a few hours descend to the right iliac region, gradually increasing here. Sometimes pain immediately occurs in the right iliac region.

Rectal colic, or the so-called tenesmus, is manifested by frequent and painful urge to the bottom with a feeling of convulsive contraction of the intestine and sphincter. Defecation does not occur; sometimes there are lumps of mucus. Tenesmus occurs in dysentery, ulcerative colitis (UC), Crohn's disease, and rectal cancer.

The appearance of pain before a bowel movement is associated with a disease of the descending or sigmoid colon, during a bowel movement − with hemorrhoids, anal fissures, cancer.

Small intestinal bleeding − may be hidden or appear as chalky or red-brown blood in the stool (with intense blood loss).

Diarrhea, as a symptom of intestinal inflammation, has been discussed previously.

Flatulence (from the Greek. μετεωρισμός − lifting up, swelling) − excessive formation / accumulation of gases in the lumen of the digestive tract. The gastrointestinal tract (GIT) of a healthy person contains no more than 200 ml of gases. Normally, the removal of gases from the lumen occurs due to belching, emission of gases through the rectum, diffusion into the blood and excretion with exhaled air. It should be recognized that these processes have not been sufficiently studied, and the norm indicators are not quite clearly defined. Belching in a healthy person can be observed several times a day, mainly after eating and has no smell. The frequency of gas release through the rectum is normally estimated as 13–21 times per day**.** The rate may vary depending on the diet. The causes of flatulence may be increased gas formation; impaired motor function due to a drop in the tone of the intestinal wall or intestinal obstruction; a decrease in the absorption of gases by the intestinal wall during their normal formation; with aerophagia − excessive swallowing of air, followed by its passage into the stomach and intestines. Separately, hysterical flatulence is distinguished − a rapidly

swelling belly to the size of a woman's belly in the last weeks of pregnancy, which occurs under the action of complex nervous mechanisms. In connection with the above, according to the main mechanisms of development, flatulence can be conditionally divided into alimentary (associated with dietary habits), digestive (associated with impaired digestion), dysbiotic (associated with the predominance of gas-forming bacteria), dynamic (associated with peristalsis disorders), high-rise, psychogenic .

There are three main groups of complaints characteristic of flatulence:

1) excessive belching;

2) bloating (in English publications referred to as «bloating»), an increase in the volume of the abdomen («distention»), described in the literature as a «symptom of tight clothing», sometimes noticeable to other people;

3) excessive discharge of gases, or flatulence («flatus»).

When questioning, it is imperative to find out the nature of the patient's diet, to establish the localization of swelling − the entire abdomen or a limited area, to find out whether swelling is always observed in the same area.

In case of the intestinal obstruction, the patient, in addition to limited bloating, feels rumbling, transfusion, increased peristaltic movements above the site of obstruction.

Intestinal absorption deficiency syndrome (malabsorption) is a disorder of absorption processes in the small intestine, often combined with maldigestion. There is primary and secondary malabsorption. Primary malabsorption develops with hereditary disorders of the structure of the mucous membrane of the intestinal wall and is genetically determined by intestinal fermentopathy. Secondary malabsorption occurs in the case of acquired changes in the mucous membrane of the small intestine − enteritis, resection of the small intestine, accelerated passage of food chyme. In acute and subacute malabsorption, the

time of contact of the chyme with the intestinal wall is reduced. In chronic malabsorption, dystrophic, sclerotic changes in the epithelium and the proper layer of the mucous membrane of the intestinal wall develop, microvilli and crypts shorten, flatten and disappear. The intestinal mucosa becomes «bald». Metaplasia of the prismatic intestinal epithelium, which has a suction capacity, into the epithelium that secretes mucus. Fibroplastic processes develop in the mucous membrane of the intestinal wall, blood circulation and lymph flow are disturbed, as well as the processes of parietal digestion. All of these changes lead to insufficient intake of the breakdown products of proteins, fats, carbohydrates, mineral compounds, and vitamins into the body. Alimentary dystrophy develops. Conditionally pathogenic and pathogenic microflora is abundantly settled in the intestine. The development of intestinal dysbiosis further exacerbates the violation of intestinal digestion and absorption, contributing to accelerated peristalsis.

Thus, the basis of malabsorption is a decrease in the total absorptive surface, a decrease in the absorptive capacity, an acceleration of the motor activity of the intestine, and a violation of the mesenteric blood flow. Malabsorption occurs in many diseases. Among them are celiac disease, chronic pancreatitis, liver cirrhosis, Crohn's disease, biliary obstruction (intrahepatic and extrahepatic cholestasis); after acute interstitial bacterial or parasitic infection, resection of the stomach and terminal small intestine, bacterial overgrowth (contamination), pancreatic cancer, small bowel lymphoma, Whipple's disease, thyrotoxicosis, Zollinger-Ellison syndrome, mesenteric ischemia in metabolic defects and AIDS, starvation and some other states.

The clinical manifestations of malabsorption are varied and include the following symptoms:

1) weakness, fatigue, anorexia, bloating, rumbling in the abdomen. Symptoms are nonspecific. For example, they can also

occur in irritable bowel syndrome. The occurrence of weakness and fatigue in malabsorption syndrome is associated with an imbalance of electrolytes, anemia, hyperphosphatemia;

2) diarrhea due to the most likely causes − bacterial overgrowth and celiac disease;

3) weight loss associated with impaired digestion and absorption of proteins, fats and carbohydrates, but with impaired absorption of only individual ingredients, for example, vitamin B_{12}, however, weight loss is not observed with microbial contamination of the small intestine;

4) steatorrhea − a sign of exocrine pancreatic insufficiency, the stool becomes light, shiny, voluminous, odorless, with drops of fat;

5) dry skin, hair loss, increased fragility of nails, dermatitis, ecchymosis, glossitis and other conditions associated with deficiency of iron and most vitamins, especially B_{12}, folic acid, ascorbic acid.

Specific signs of malabsorption include:

1) peripheral edema, less often − ascites associated with protein malabsorption, loss of endogenous protein, hypoalbuminemia. With hypoproteinemia below 4−5 g, hypoproteinemic edema begins to appear;

2) paresthesia, tetany, symptoms of Chvostek, Trousseau − associated with hypocalcemia, hypomagnesemia, malabsorption of vitamins D and B_1. Osteoporosis, anemia develops (hypochromic in case of impaired absorption of iron and hyperchromic in case of impaired absorption of vitamin B_{12}, etc.). (Chvostek's symptom − light tapping with a percussion hammer along the zygomatic arch is manifested by involuntary spasm of facial muscles. Trousseau's symptom − with a strong compression of the shoulder by the tonometer cuff, a hand cramp occurs, giving it the shape of an «obstetrician's hand»);

3) vitamin deficiency that occurs with malabsorption syndrome may be accompanied by the corresponding symptoms: night blindness or xerophthalmia (deficiency of vitamin A), cheilitis (deficiency of riboflavin), subcutaneous hemorrhages (deficiency of vitamin K), skin rashes, anemia, leukopenia (deficiency of zinc and copper), but these disorders are not common and are usually accompanied by symptoms of hypovolemia. In addition, the content of cholesterol, calcium, and sugar in the blood serum decreases.

The syndrome of insufficiency of digestion (maldigestion) is a violation of digestion in the stomach and intestines. There are 6 groups of digestive disorders:

1) deficiency of digestive enzymes − lactase deficiency, celiac disease and others;

2) alimentary maldigestion − fermentative dyspepsia, putrefactive, fatty;

3) intoxication − endogenous (poisoning with mushrooms, industrial poisons, medicines − digoxin, laxatives, etc.); and exogenous (renal, liver failure);

4) neuro-vegetative disturbances in the regulation of bowel function: neurogenic dyspepsia in stressful situations, conditioned reflex neurogenic dyspepsia;

5) acute and chronic infections, parasitic infestations;

6) violation of the secretion of digestive juices or a decrease in digestive enzymes in them − resection of the stomach or intestines, achlorhydria, pancreatogenic dyspepsia, dyspepsia associated with impaired secretion and intestinal motility in chronic diseases.

Digestive disorders can be caused by malnutrition, insufficient secretion of digestive juices, low levels of enzymes in them, and accelerated passage of contents through the intestines. With incomplete breakdown of dietary fiber, dysbiosis often

develops, while the intestinal flora actively participates in the enzymatic breakdown of nutrients in the intestine, contributes to the formation of toxic products (ammonia, indole, skatole, etc.), which cause irritation of the intestinal mucosa, increased peristalsis and intoxication of the body. With long-term dyspepsia, chronic inflammatory bowel diseases develop, and then fibrosing processes in the intestinal wall.

Syndrome of intestinal dyspepsia. Symptoms of intestinal dyspepsia include a feeling of pressure, heaviness in the epigastrium and throughout the abdomen, belching with an unpleasant odor, nausea, regurgitation, loss of appetite, rumbling, transfusion in the abdomen, bloating, profuse gas formation, stool disorders up to diarrhea. So with fermentative dyspepsia, stools with a sour smell, plentiful, frequent. With putrid dyspepsia, stools with an unpleasant putrefactive odor are increased up to 5 times a day. With fatty dyspepsia, the stool is plentiful, semi-liquid, with a greasy sheen, with the smell of rancid oil, it is poorly washed off the walls of the toilet bowl (the so-called acholic stool). It is more often observed in violation of the flow of bile into the intestine, with a stone or narrowing, compression of the common bile duct. In a scatological examination of feces, micro-remains of food are revealed − amylorrhea, creatorrhea, steatorrhea. An X-ray examination studies the passage of barium suspension through the intestines. Against the background of maldigestion, the passage of barium will be accelerated.

The large intestine, like the small intestine, consists of several sections − the cecum, the ascending colon, the transverse colon, the descending colon, the sigmoid colon, and the rectum. The large intestine differs from the small intestine in the form of haustra, ribbons, and omental processes. In the large intestine, fluid and salts are absorbed, the formation and accumulation of dense fecal masses, which are dehydrated intestinal contents, and their removal from the body until evacuation occurs. Up to 75 %

of the mass of raw feces are microorganisms, which are very abundant in the large intestine.

The main complaints of patients with diseases of the large intestine are few − pain, rectal bleeding, changes in the rhythm of defecation and the consistency of feces.

Pain in diseases of the colon is usually localized in the lower abdomen. When the rectum is involved in the process, pain is felt deep in the pelvic cavity, with diseases of the anal canal − in the perineum. Overflow of the colon with gases or liquid contents causes colicky or cramping pains, which can sometimes be accompanied by contraction and spasm of the muscular layer of the intestine. After flatulence and / or defecation, these pains stop. Prolonged pain in the lower abdomen, which does not facilitate bowel movements, may accompany intestinal tumors with tumor inflammation or infiltration of the intestinal wall. Acute inflammation of the colon involving the parietal and visceral layers of the peritoneum is accompanied by severe persistent pain.

Constipation in diseases of the colon. Constipation (lat. *obstipatio*) − prolonged, more than 48 hours, stool retention. The duration of stool retention is a relative indicator and depends on the nature of food and drinking regimen. In the case of the predominance of a meat diet, the stool becomes rarer. Constipation is usually divided into organic and functional.

Organic constipation is associated with mechanical obstructions in the large intestine − narrowing of the intestinal lumen due to a tumor, scar, adhesions, as well as in connection with anomalies in the development of the intestine (megacolon, dolichosigma, diverticulosis, etc.).

Functional constipation is divided into alimentary; neurogenic − associated with inflammatory lesions of the colon, for example − with dysentery of the colon; toxic; endocrine; caused by hypodynamia; caused by weakness of the abdominal

muscles. Alimentary constipation appears due to an insufficient amount of coarse dietary fiber in food. Neurogenic constipation − caused by a reflex effect on intestinal motility from another diseased organ, for example − with inflammation of the gallbladder, prostatitis, adnexitis. In addition, neurogenic constipation may occur when the function of the intramural innervation of the large intestine or the pathology of the vagus nerve is impaired. Toxic constipation is observed with intoxication with lead, morphine, cocaine. Endocrine constipation − with hypothyroidism, pathology of the pituitary gland. The character of stool in constipation is about 1st or 2nd types in Bristol stool classification.

Examination of patients with diseases of the guts

In severe prolonged malabsorption, the patient is depleted − cachexia. With the loss of protein and simultaneous fluid retention, edema is observed. The skin may be dry, pale, mucous membranes are pale. Vitamin deficiency associated with insufficient absorption is manifested by rough skin, cracked lips − seizures. The tongue acquires a crimson-red color, its papillae are smoothed – «cardinal tongue». The gums may be loose and bleed. When the patient is asked to «breathe with his stomach», he cannot take a deep breath in the presence of pain.

It is necessary to ask the patient about the postoperative scars on the anterior abdominal wall, their age, since previous operations on the abdominal organs can cause the formation of adhesive disease, accompanied by pain in the abdomen and constipation. It is possible that in urgent situations, anti-peristaltic movements in the epigastric region or along the intestine will be visible, indicating an obstacle to the movement of food masses. In terminal patients, you can see the «face of Hippocrates» (facies Hyppocratica) − a mask-like face with sunken eyes, sunken cheeks, a pointed nose, cyanosis of the lips and sometimes ears,

drops of cold sticky sweat on the forehead, a general suffering expression, observed in patients with diffuse peritonitis, perforation of the intestine, stomach, with intestinal obstruction, is an unfavorable prognostic sign (fig. 27).

Figure 27. The face of Hippocrates (or the «mask of Hippocrates»)

With peritonitis, a forced position of the patient is possible − in the «embryo position» − in the position on the side, with bent knees pulled up to the chest, a pillow can be clamped between the knees − this position somewhat alleviates the pain syndrome in the abdomen. With intestinal obstruction and in the presence of a fistula between the stomach and the transverse colon, a fecal odor may come from the patient's mouth.

Palpation of the abdomen in diseases of the intestine

For the diagnosis of intestinal diseases, the method of deep palpation according to Obraztsov−Strazhesko (V.P. Obraztsov and N.D. Strazhesko − Russian therapists) is used as the most informative manual method of research. This method is also called sliding, methodical, topographic palpation. All results of palpation in the presence of pathological changes must be monitored radiographically and even laparoscopically.

Starting palpation, it is necessary that the muscles of the anterior abdominal wall of the patient be relaxed. The patient lies on his back on the couch, under his head − a low pillow. His hands are folded on his chest, his breathing is shallow, diaphragmatic. The doctor sits on the right side on a hard chair, facing the patient. The height of the doctor's chair should be equal to the height of the patient's couch. The doctor's hands are warm, clean and dry.

The goals of palpation are to establish whether the organs are in a normal physical condition and topographic relationships; detection of the pathological process, its size, shape, consistency. For this, both superficial and deep palpation are used. (The technique of superficial palpation was described in the section «Palpation of the abdomen» in Chapter 4).

Deep methodical sliding palpation according to Obraztsov−Strazhesko in case of bowel diseases

The position of the patient is the same as with superficial palpation. Palpation is carried out with the right hand. The doctor lays his right hand flat on the anterior abdominal wall of the patient, perpendicular to the axis of the intestine under study or on the edge of the organ under study. An important point is the shifting of the skin and the formation of a skin fold so that further movements are not limited to skin tension. The hand plunges deep into the abdomen. Gliding with the fingertips occurs in the

direction transverse to the axis of the organ under study, while pressing the organ against the back wall and, continuing to slide, roll over the palpable intestine or curvature of the stomach. The movements of the palpated hand are made only together with the skin. Diving into the abdominal cavity occurs gradually, using the period of relaxation of the abdominal wall on exhalation. Sliding movements of deep palpation are performed in the direction from the inside to the outside (sigmoid colon, caecum), or from top to bottom (stomach, transverse colon). Having palpated the intestine, determine its localization, mobility, consistency, diameter, surface condition (smooth, bumpy), the presence or absence of rumbling during palpation, pain. All of these signs make it possible to judge the nature and intensity of the pathological process. The following sequence of deep palpation is most acceptable: sigmoid colon, cecum with appendix, terminal ileum, stomach with its departments, transverse colon, liver, spleen, duodenum, pancreas and kidneys.

Having palpated the abdominal organ, determine its localization, mobility, consistency, diameter, surface condition (smooth, bumpy), the presence or absence of rumbling during palpation, pain; All these signs make it possible to judge the presence or absence of a pathological process.

Palpation of the sigmoid colon. The sigmoid colon is palpated from the top right, perpendicular to the axis of the intestine, which is usually located obliquely in the left iliac cavity at the border of the middle and outer third of the line connecting the umbilicus with the anterior superior iliac spine (linea umbilico-iliaceae). Palpation is carried out with four fingers folded together and slightly bent. Having immersed the fingers inwards from the intended position of the intestine, having reached the posterior wall of the abdominal cavity, they slide along it in the outward and downward direction. In this case, the intestine, pressed against the back wall, slides along it, and then

slips out from under the fingers. At this moment, the palpable surfaces of the fingers bypass the intestine almost along the entire circumference. Normally, the sigmoid colon is palpable for 20−25 cm in the form of a smooth dense cylinder with a thickness of the thumb or forefinger, painless on palpation, not rumbling, very sluggish and rarely peristaltic. It can be shifted to the sides within 3−4 cm. The technique allows you to palpate the intestine in 95 % of people. Only with excessive bloating and in excessively obese people, the sigmoid colon is not palpable. If the sigmoid colon is not palpable in its usual location, it may be closer to the umbilicus due to the long mesentery.

Palpation of the caecum. Palpation of the intestine is carried out along the line connecting the navel and the upper anterior axis of the ilium. The intestine is normally located on the border of the middle and outer third of the linea umbilico-iliaceae, 5 cm from the iliac spine (fig. 28).

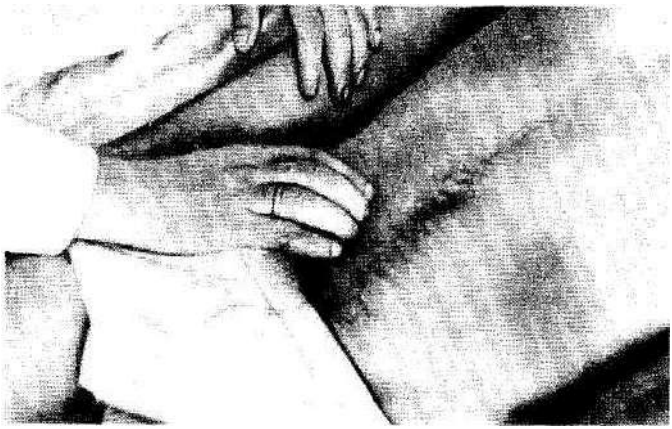


Figure 28. Palpation of the caecum

The caecum is palpable in 80−85 % of cases in the form of a moderately tense, slightly expanding downward cylinder, 2−3 cm

in diameter, rumbling when pressed. Palpation of the intestine does not cause pain and allows you to determine its mobility within 2−3 cm.

Palpation of the ileum and appendix. The ileum is palpable in 80% of cases in the right iliac region for 15−20 cm immediately behind the caecum. It rises in the direction from the small pelvis, connects with the large intestine. Palpation is carried out parallel to the linea umbilico iliaceae, but below it. The final part of the ilium is palpated in the depth of the right iliac cavity in the form of a soft, peristaltic, movable cylinder as thick as a little finger or a pencil. The cylinder, when slipping out from under the fingers, intensively rumbles. Having found the terminal process of the ileum, you can look above or below its appendix. It is easier to find it if you first palpate the m.psoas (it is easier to find it if the subject slightly raises the straightened right leg) and palpate the process on the contracted belly of the muscle. The process is palpable in 20−25 % of cases in the form of a thin, goose-feather-thick, painless cylinder that does not rumble and does not change its consistency under the hands. However, the appendix can imitate mesenteric duplication and lymphatic bundle. In many people, the appendix is located retrocaecally, and palpation is impossible. With inflammation of the process, its deformation, thickening, fixation, compaction will take place, which increases the likelihood of its palpation. Palpation of the cecum, ileum and appendix is carried out with the right hand, four fingers folded together and slightly bent at the joints. When the abdominal muscles are tense, in order to cause relaxation in the palpation zone, it is useful to press the navel area with the radial edge of the left hand.

Colon palpation. For palpation of the ascending and descending colon, bimanual palpation is used: the left hand is placed under the left and right halves of the lower back, and the right hand is immersed in the abdominal cavity. At the same time,

on exhalation, the doctor's hands «meet» in the abdominal cavity (V.Kh. Vasilenko's method). Palpation of the transverse colon is carried out with the right hand with folded and slightly bent fingers or with both hands (bilateral palpation). Since the position of the transverse colon is variable, to search for it, it is recommended to find the lower border of the stomach percussion (method – «Obraztsov's percussion palpation»), and then begin palpation of the intestine 2−3 cm below the lower border of the stomach. The right hand or both hands are placed on both sides of the white line of the abdomen, pushing the skin slightly upwards, the hands begin to sink into the abdominal cavity on exhalation. Having reached the back wall, they slide down along it and in case of palpation of the intestine they find it in the form of an arcuately and transversely located cylinder of moderate density, 2−2.5 cm thick, easily moving up and down, painless, non-rumbling (fig. 29).

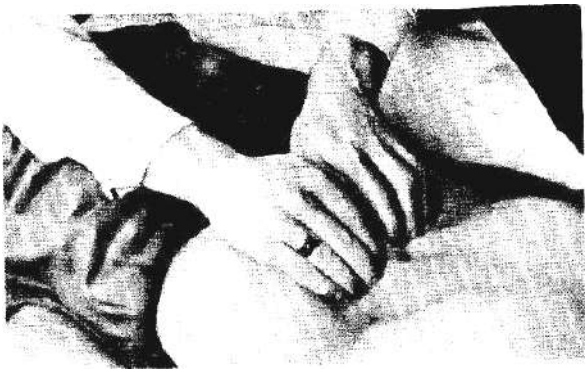


Figure 29. Deep palpation of the transverse colon

If the intestine is not found by this method, then it is palpated both lower and laterally, changing the position of the palpating

hands accordingly. The transverse colon is palpable in 60−70% of cases.

Finger examination of the rectum. It is carried out after a cleansing enema. The patient is in the knee-elbow position. Dressed in a disposable latex glove and lubricated with medical vaseline, the index finger is inserted into the rectum and gently advanced to the possible depth with slow movements. Having passed the sphincter, the finger meets the prostate in front of men, and the vaginal part of the uterus in women. On it, the finger moves up, bypassing the sacrococcygeal fold, and, if possible, reaches the final fold (plica terminalis recti), which closes the entrance to the sigmoid colon. After examining the anterior wall with a finger, turn the finger backwards and palpate the posterior sacral, and then the side walls, making an interpretation of the mucous membrane by palpation (the presence of ulcers, papillomas, polyps, varicose veins, swelling and swelling of the mucosa, cicatricial narrowing, neoplasms, etc.) as well as the fiber surrounding the rectum, Douglas space, prostate, uterus with its appendages, pelvic bones (fig. 30).

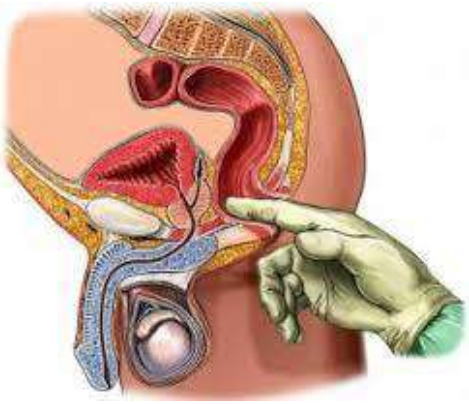
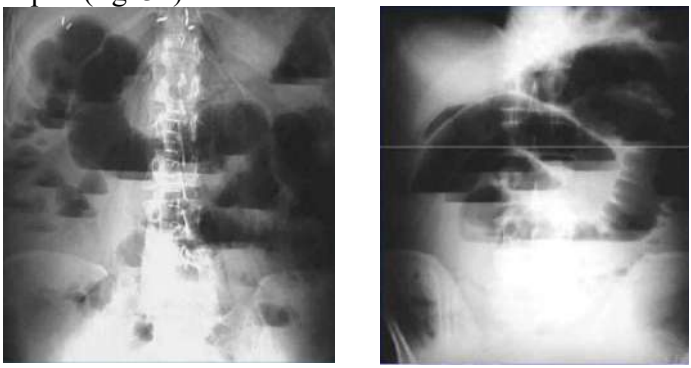


Figure 30. Digital examination of the rectum

Auscultation of the bowel. Auscultation allows to examine the motor function of the intestine. In the phase of gastric digestion and the movement of chymus in the small intestine, a long periodic rumbling is heard. Rhythmic intestinal noises are heard in the caecum 5−7 minutes after eating. With mechanical intestinal obstruction, peristalsis is very loud, with large sound waves. With paralytic obstruction of the intestines, peristalsis disappears, and with perforation of an ulcer with secondary paralysis of the intestines, the so-called «deathly silence» occurs in the abdomen. In patients with fibrinous peritonitis, during respiratory movements, a friction rub of the peritoneum may be heard. Instrumental and laboratory diagnostics of intestinal diseases.

The FEGDS method is actively used for the study of the duodenum, it is allows you to see all parts of the intestine, take tissues for a biopsy if necessary. Visualization of other parts of the small intestine was difficult until a certain time.

X-ray examination of the small intestine has been used for a long time, but has many errors. Criteria for diagnosing intestinal obstruction using the X-ray method of research have been developed (fig. 31).



A *B*

Figure 31. X-ray with *A* − small bowel obstruction, *B* − with large bowel obstruction

With small bowel obstruction, pathological levels are located mainly in the central sections of the abdominal cavity; the diameter of the levels exceeds the height, since the small intestine is capable of stretching; in the swollen loops of the intestine above the levels, transverse folds of the mucous membrane are visible; bowel loops, swollen with air, can give the symptom of «arches» above the levels. With colonic obstruction, Kloiber's bowls are usually located along the periphery; the diameter of the levels is less than their height, since the large intestine is not able to expand like the small intestine, due to the haustra; in the swollen loops above the levels, gaustral retractions can be seen along the contours.

For examination of the rectum, in addition to the mandatory digital examination, which must be performed by doctors of any specialty, the method of sigmoidoscopy is routinely used.

Rectoscopy (sigmoidoscopy) is a method for examining the mucous membrane of the rectum and the distal sigmoid colon using an endoscopic apparatus − a rectoroscope. The procedure allows not only to examine the entire surface of the intestine, but also to take a biopsy, as well as to perform minor operations, for example, to remove a polyp. The device is a flexible or rigid tube (depending on the model) with built-in lighting and an air blower (fig. 32). Indications for rectoscopy are pain in the anus; bloody, purulent or mucous discharge; chronic constipation or diarrhea, suspicion of a malignant neoplasm of the intestine.

Figure 32. Rectoscopy procedure

There are practically no absolute contraindications to rectoscopy. Relative contraindications are cardiac decompensation, severe general condition, narrowing of the lumen of the anal valve and rectum, acute inflammatory processes in the anus (acute paraproctitis, thrombosis of hemorrhoids), in these cases, the examination is best done after subsiding acute phenomena; stenosing tumors of the anal canal; chemical and thermal burns in the acute stage

The rectoscopy procedure requires special preparation − a thorough cleansing of the colon from the contents. On the eve of rectoscopy during the day, a diet low in dietary fiber is prescribed, and in the evening − only tea. The study is carried out on an empty stomach. In the evening and 2 hours before the study, the intestines are cleansed with an enema. A preliminary examination of the anus and its digital rectal examination is mandatory. At the same time, anal eczema, dermatitis, condylomas, external fistula openings, perianal thrombosis, skin ulcerations, prolapsing hemorrhoids or polyps, pararectal abscesses, prolapse of the

rectum and tumors are easily detected. If you offer the patient to push, then the external hemorrhoids become visible and filled. The study is best done immediately after a bowel movement, because at this time it is possible to clearly diagnose the prolapse of internal hemorrhoids.

Colonoscopy. Colonoscopy is the gold standard for colonoscopy today. During the procedure, it is possible to detect ulcers, polyps, tumors. The procedure allows you to examine up to 120−152 cm of the intestine − the entire length of the large intestine, while rectoscopy − no more than 60 cm. During colonoscopy, as well as with FEGDS, it is possible to perform a biopsy and some therapeutic measures (fig. 33).

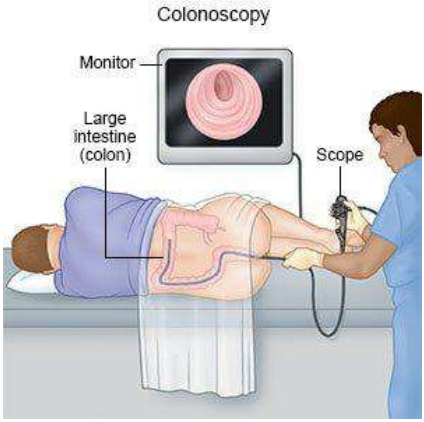
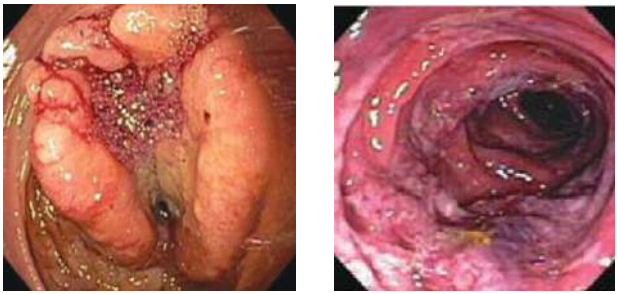


Figure 33. Conducting a colonoscopy study



A *B*

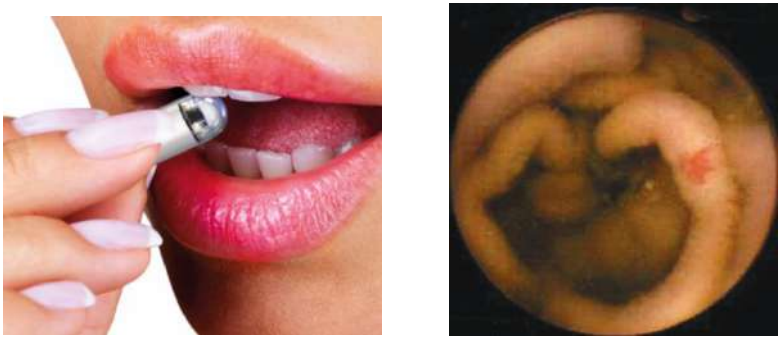
Figure 34. Colonoscopy pictures. *A* – adenocarcinoma of the large intestine, growing in its lumen; *B* – ischemic colitis with partial mucosal edema, cyanosis and small submucosal hemorrhages

Indications for a planned colonoscopy are the repeated presence of pus, mucus and blood in the stool; chronic constipation and diarrhea; bloating and frequent pain along the colon; subfebrile temperature of unclear etiology for a long period; unmotivated weight loss; anemia of unknown etiology; benign and malignant neoplasms diagnosed using other research methods (irrigoscopy, CT, MRI); search for a primary tumor in the presence of metastases; screening for colon cancer; evaluation of the effectiveness of conservative or surgical treatment (fig. 34).

Contraindications for colonoscopy are acute myocardial infarction and decompensated heart failure; acute violation of cerebral circulation; fulminant form of colitis; acute infectious process; aortic aneurysm; acute diverticulitis; tense ascites. According to WHO recommendations, every healthy person aged 55 years and older should have a colonoscopy every 10 years.

In recent years, a new method for examining the gastrointestinal tract, capsule endoscopy, has been actively introduced in the world. (fig. 35). The patient swallows a miniature tablet-sized capsule that passes freely through the

gastrointestinal tract and captures a video image, transferring it to a recording device previously attached to the patient's belt. After a set period of time, the patient returns the recording device to the clinic. The doctor uses special software to process the received data. The technique is non-invasive, but does not allow taking tissue for a biopsy.



A *B*

Figure 35. *A* – the patient swallows the video camera capsule; *B* – ectasia of the vessels of the small intestine, registered in the process of capsule endoscopy. This ectasia needs additional examination, as it may be the earliest manifestation of a malignant disease

In the nowadays, robotic capsules are already appearing for endoscopy with biopsy, as well as intra-intestinal surgical interventions. A team of researchers from Vanderbilt University and the University of Leeds have developed a magnetized endoscope capsule. The direction of movement of the capsule is set by an outside robotic arm with a magnet. Currently, the robotic capsule is successfully used to study the large intestine. The robotic platform for colonoscopy consists of two main subsystems:

1) endoscopic device – a soft-tie capsule with all the functionality of a traditional endoscope, including an endoscopic

camera, white light LEDs, a therapy channel for instruments, and air/water channels for blowing and cleaning. In addition, the capsule has a magnet that provides a non-rigid connection with an external magnet that sets the movement of the capsule through the colon;

2) a robotic arm (Robot Unit) and a control unit (Control Unit) allow you to control the capsule. The proposed approach reduces the physical load during the procedure. In addition, the management of the procedure is separated from the site of the procedure. If necessary, the study can be carried out by a doctor from another clinic or even from another country. The control box supports multiple interfaces, including the Phantom Omni tactile interface that provides force feedback; in addition, the device is equipped with touch screens, Kinect and classic interface devices − a mouse and keyboard. The development of robotic capsules for the study of the small intestine, which, most likely, is the future of gastroenterology, is actively underway.

Features of the diagnosis of certain diseases of the intestine

Celiac disease − celiac disease (non-tropical sprue), is characterized by atrophy of the villi of the small intestine, which are restored only after the exclusion of gluten from food. Abnormal gluten sensitivity causes damage to the intestinal mucosa. Gluten is a group of proteins found in wheat, rye and barley. Gluten contains L-gliadin, which is a toxic substance, but its mechanism of action is not fully understood. The antibodies formed as a result of the interaction damage the enterocytes of the intestinal villi. Complaints of patients with celiac disease − diarrhea, steatorrhea, weight loss, bloating. Symptoms may appear in childhood, then decrease and reappear in the 30s to 60s.

Excessive bacterial growth (microbial contamination syndrome of the small intestine) is a multifactorial disease characterized by an excess of the number of gram-positive bacteria in the small intestine of more than 104 per 1 ml of jejunal juice. The causes are infectious diseases, diverticulitis, Crohn's disease, radiation strictures, scleroderma and others. Bacteria deconjugate bile acids, absorb vitamin B_{12}, but do not lead to malabsorption of folic acid and fat-soluble vitamins. Complaints and symptoms: diarrhea, malabsorption, B_{12} deficiency anemia.

Disaccharide deficiency − chronic diarrhea due to congenital or acquired deficiency of disaccharide hydrolysis enzymes − lactase, sucrase, maltase, treclase.

Adverse reactions to food − various types of food allergies, intolerances, sensitivities. Characterized by the appearance of abdominal pain, diarrhea, steatorrhea, as well as extraintestinal

manifestations. There may be nausea, vomiting, skin reactions, reactions from the upper and lower respiratory tract.

Diverticulosis of the small intestine is often asymptomatic. Rarely, diverticula can cause small bowel obstruction, bacterial overgrowth, bowel perforation, bleeding, and inflammation. Diverticula can be located in the duodenum, on the mesenteric edge of the entire small intestine. Separately, Merkel's diverticulum is distinguished − a sac-like protrusion in the terminal ileum, which, when damaged and inflamed, is most often complicated by intestinal bleeding. The clinic resembles acute appendicitis, surgical treatment.

Appendicitis is an inflammation of the appendix of the caecum that develops as a result of the introduction of bacterial microflora into its mucous membrane with further obstruction of the appendix as a result of inflammation, fecal stone, foreign body or neoplasm.

Acute appendicitis is a common cause of an «acute abdomen». After obstruction of the process, its necrosis and perforation occur. Clinical symptoms: occurs at any age, but more often in young men. The attack of pain begins suddenly. The pain usually starts in the center of the abdomen and shifts to the right iliac region, but there may be other options, especially with atypical locations of the process. Pain is accompanied by nausea, vomiting due to irritation of the peritoneum. There may be stool retention. Palpation pain in the right iliac region at the McBurney point (fig. 36), a positive Shchetkin-Blumberg symptom (fig. 37) and a number of other symptoms of acute appendicitis and peritonism are characteristic.

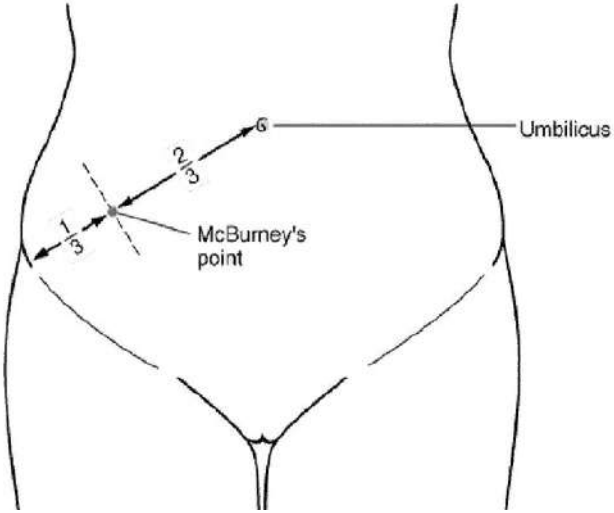


Figure 36. The position of the Mac-Burney point in the case of the diagnosis of acute appendicitis. The point is located on the border between the lower and middle third of the line connecting the navel and the upper anterior iliac spine

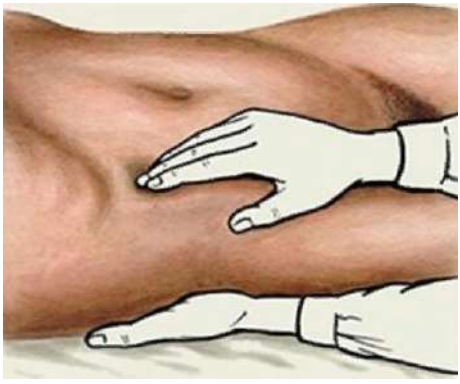


Figure 37. Definition of the Shchetkin-Blumberg symptom

A sharp increase in pain in the abdomen with the rapid removal of the palpating hand from the anterior abdominal wall after pressure. Indicates irritation of the peritoneum. Symptom Shchetkin−Blumberg refers to the symptoms of peritonism.

Polyps. More often than others in clinical practice, there are multiple polyps of the small intestine: nodular lymphoid metaplasia, Peutz−George syndrome (polyposis of the small intestine in combination with hyperpigmentation of the skin and mucous membranes), Cronkite−Canada syndrome (polyposis in combination with early baldness and atrophy of the nail plates), lymphomatous polyps, endometriosis of the small intestine. Clinical manifestations of polyposis are nonspecific, the symptoms are common − abdominal pain, fever, diarrhea, malabsorption. Polyposis can lead to intestinal obstruction, perforation, bleeding.

Alpha heavy chain disease is a lymphoproliferative disorder resulting in diffuse infiltration of the small intestinal mucosa and mesenteric lymph nodes by plasma cells secreting an abnormal immunoglobulin devoid of light chains and containing only defective heavy chains. Its manifestation is often hypogammaglobulinemia. The disease is considered as precancerous.

Small bowel carcinoma is a rare disease with an undetermined etiology. It is more common in patients with celiac disease, a heavy chain disease. The tumor grows rapidly and leads to narrowing of the intestinal lumen. Accompanied by hypochromic anemia, bleeding.

Chronic nonspecific inflammatory diseases of the large intestine. The most common diseases in this group are Crohn's disease and ulcerative colitis (NUC). The cause of these diseases is not fully understood. The leading hypothesis is the pathological immune mechanism. In patients, the use of hormones and cytostatics gives a positive effect in the treatment, and humoral antibodies to intestinal cells, bacterial and viral antigens, *E. coli*, foreign protein and various polysaccharides are found in the

blood. Perinuclear antibodies (pANCA) can be identified in 60−70 % of patients with UC and in 40 % of patients with Crohn's disease.

Ulcerative colitis (UC) is a necrotizing inflammatory disease of the colon mucosa characterized by periods of exacerbation and remission. UC occurs in 50−230 cases per 100,000 people (twice as common as Crohn's disease), more often between the ages of 20 and 40 years. With UC, the rectum is almost always affected. The entire colon (pancolitis) or its descending part, the sigmoid colon, may be involved in the pathological process. The main clinical variants of UC are shown in fig. 38. UC never spreads to the small intestine. The characteristic pathoanatomical sign of UC is crypt microabscesses observed in 70 % of cases. The inflammatory process is limited to the mucous membrane. The main complaint of patients is diarrhea with blood, mucus, sometimes pus. The onset of the disease is spontaneous, gradually progressive, the symptoms may subside and recur.

The frequency of stools varies throughout the day, with more stools occurring at night or early in the morning. Often there are non-intense pains throughout the abdomen, discomfort in the abdomen. Anemia, fever, weight loss, weakness may appear. Signs of systemic inflammation in UC (endotoxemia syndrome) include fever, leukocytosis, elevated ESR, and elevated acute phase proteins (CRP, fibrinogen, seromucoid).

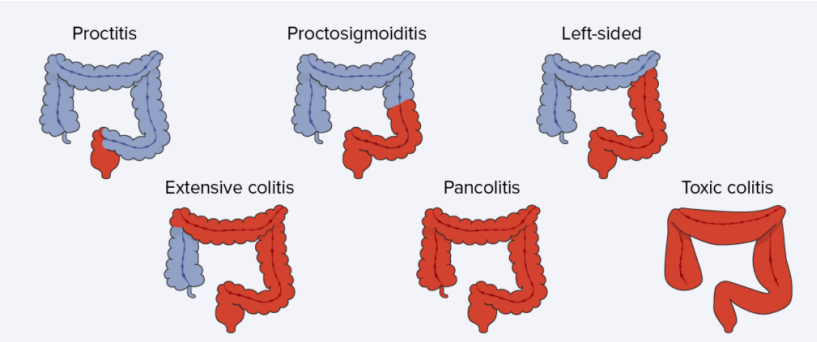


Figure 38. Clinical variants of ulcerative colitis

The frequency of stools varies throughout the day, with more stools occurring at night or early in the morning. Often there are non-intense pains throughout the abdomen, discomfort in the abdomen. Anemia, fever, weight loss, weakness may appear. Signs of systemic inflammation in UC (endotoxemia syndrome) include fever, leukocytosis, elevated ESR, and elevated acute phase proteins (CRP, fibrinogen, seromucoid).

The syndrome of metabolic disorders associated with UC includes weight loss, general weakness, anemia, hypoproteinemia, and electrolyte imbalance.

Intestinal complications of UC include toxic dilatation and perforation of the colon, intestinal bleeding, and colorectal cancer.

Extraintestinal manifestations of UC are described, which can be divided into the following groups: autoimmune, associated with the activity of the disease; autoimmune, not associated with the activity of the disease; as well as extraintestinal complications due to a long-term inflammatory process and metabolic disorders.

Autoimmune complications associated with the activity of the disease include various arthropathies (arthralgia, arthritis; skin lesions in the form of erythema nodosum, pyoderma gangrenosum; mucosal lesions in the form of aphthous stomatitis;

eye damage by the type of uveitis, iritis, iridocyclitis, episcleritis. Autoimmune manifestations, not associated with disease activity include primary sclerosing cholangitis and pericholangitis, ankylosing spondylitis and sacroileitis, seronegative rheumatoid arthritis, psoriasis Extraintestinal manifestations due to prolonged inflammation and metabolic disorders include cholelithiasis, hepatic steatosis, steatohepatitis, peripheral vein thrombosis, pulmonary embolism, amyloidosis.

In the diagnosis of the disease, a contrast x-ray examination of the colon with barium is used − irrigography. Signs in the form of a decrease in intestinal haustration and others are characteristic. The main diagnostic method is colonoscopy with a biopsy of the altered part of the intestine. On fig. 39 shows a picture of a colonoscopy of the large intestine affected by UC.

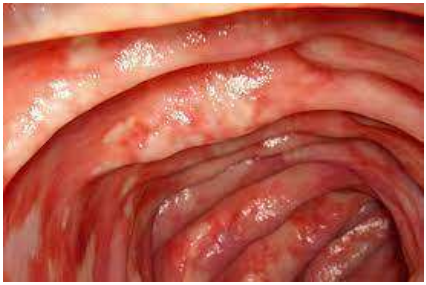


Figure 39. Colonoscopy of the colonic mucosa in nonspecific ulcerative colitis

The colonoscopic signs of UC include continuous inflammation, limited to the mucous membrane, starting in the rectum and continuing proximal to the borders of inflammation is always clear. The activity of UC is determined by the presence of contact vulnerability of the mucosa − the release of blood when touching the endoscope; the absence of a vascular pattern and the presence of erosions and ulcerations. The detection of persistent

narrowing of the bowel in UC requires the exclusion of colorectal cancer.

The scope of examinations for UC includes a thorough physical examination, examination of the perianal region, digital examination of the rectum, sigmoidoscopy, plain radiography of the abdominal cavity − in pain syndrome to exclude toxic dilatation of the intestine and its perforation. To establish the diagnosis, a mandatory total colonoscopy with ileoscopy is necessary. If a colonoscopy is not possible, a double-contrast irrigoscopy should be performed to assess the extent of the colon lesion. At the initial diagnosis, in case of doubts about the diagnosis, a biopsy is recommended, and with a long duration of the disease for more than 7 years, a stepped biopsy is recommended to exclude dysplasia of the epithelium of the intestinal mucosa. In addition, all patients undergo an ultrasound examination of the abdominal cavity. The coprogram is performed by everyone to exclude acute infection and parasitic invasion in the primary diagnosis of UC; and also in the feces, a study of toxins A and B of Clostridium difficile is carried out after a course of antibiotic therapy or hospital stay, with a severe exacerbation of the disease, with resistance to treatment. For the primary differential diagnosis of UC with functional bowel diseases and to assess the activity of the inflammatory process in the intestine, a study of the level of fecal calprotectin is carried out.

Crohn's disease (CD) is a chronic relapsing disease of the gastrointestinal tract of unclear etiology, characterized by transmural segmental granulomatous inflammation with the development of local and systemic complications.

An exacerbation of CD should be understood as the appearance of typical symptoms of the disease in patients with CD who were previously in clinical remission, supported by medication or spontaneous. Remission of CD refers to the

disappearance of typical symptoms of the disease. Remission is distinguished as clinical and endoscopic. Clinical remission is understood as the absence of symptoms and compliance with a CD activity index of less than 150. Endoscopic remission is understood as the absence of microscopic signs of inflammation during endoscopic examination.

The disease occurs at any age, but more often in young people. The prevalence of CD is higher in the northern latitudes and in the west and is 322 cases per 100,000 people. Characteristically, in contrast to UC, there is an inflammatory lesion of all layers of the intestine according to the «cobblestone pavement» type, where areas of preserved mucosa alternate with deep slit-like ulcers penetrating into the submucosal and muscular layers, where fistulas, abscesses, and strictures of the intestine can form.

The etiology of the disease is associated with many factors such as genetic predisposition, defects in innate and acquired immunity, intestinal microflora and various environmental factors.

The classification of CD is based on the Montreal classification, which includes 3 main forms of the disease − terminal ileitis, colitis and ileocolitis. In addition, lesions of the upper gastrointestinal tract and / or anorectal zone can join each individual localization.

According to the prevalence of the lesion, a localized form and a widespread form are distinguished. The localized form includes a lesion less than 30 cm long in each part of the intestine, more often localized in the ileocecal zone. A common lesion includes a total length of inflammation of more than 100 cm.

According to the nature of the course of CD, an acute course is distinguished − less than 6 months from the onset of the disease; chronic continuous course − the absence of more than 6-

month periods of remission during treatment; as well as a chronic relapsing course − in the case of more than 6-month periods of remission. The severity of CD exacerbation is determined by the intensity of the next inflammation, the presence of extraintestinal manifestations and complications, the extent of the lesion, resistance to therapy, and the development of resistance.

For a clinical assessment of the severity of an exacerbation of CD, the Crohn's Disease Severity Scale is used in accordance with the criteria of the Society for the Study of Inflammatory Bowel Diseases at the Association of Coloproctologists of Russia (tab. 2), the Harvey−Bradshaw index (tab. 3), as well as the CD activity index (tab. 4).

Table 2

Scale for assessing the severity of Crohn's disease in accordance with the criteria of the Society for the Study of Inflammatory Bowel Disease at the Russian Association of Coloproctologists

Index	The severity of the exacerbation		
	mild	moderate	severe
Average stool frequency per day for the last 3 days	<4	4–6	≥7
Abdominal pain	absent or minimal	moderate	severe
Fever, ºC	absent	<38	≥38
Tachycardia, beats per minute	absent	less then 90	≥90
ESR, mm/h	norm	less then 30	≥ 30
Leukocytosis	norm	moderate	High with formulaes change
C-reactive ptotein, g/l	norm	<10	>10
Hypoproteinemia	absent	mild	severe
Extraintestinal manifestations	absent	present	present
Intestinal complications (any)	absent	present	present

Table 3

The severity of the CD attack according to the CDAI activity index (Best index)

Criterion	Counting system	Coefficient	Sum points
The frequency of the liquid or mushy stool	The amount of bowel movements for the last 7 days	x2	=
Abdominal pain: 0 – no; 1 – weak; 2 – moderate; 3 – strong	The total points for 7 days are taken into account	x5	=
General well-being: 0 – good; 1 – satisfactory; 2 – bad; 3 – very bad; 4 – terrible	The total points for 7 days are taken into account	x7	=
Other symptoms (extraintestinal or intestinal complications): - arthritis or arthralgia; - iritis or uveitis; - nodular erythema; - pyoderma gangrenosum; - aphthous stomatitis; - anal lesions (cracks, fistulas, abscesses); - other fistulas	The presence of each of the above complications adds 1 point	x20	=

Ending of table 3

Criterion	Counting system	Coefficient	Sum points
Fever ≥ 37,5	The sum of episodes of fever for 7 days	x20	=
The use of loperamide (other opiates) for relief of diarrhea: 0 – no; 1 – yes		x30	=
Tension of the abdominal muscles (or palpable infiltrate): 0 – missing; 2 – doubtful; 5 – clearly	The assessment is made once at a time. inspection	x10	=
Hematocrit 47 minus the score sick (M); 42 minus the indicator sick (F)	The difference between normal level and indicator of the patient (taking into account sign «+» or «-»)	x6	=
Body weight in kg	1 − (actual weight: ideal weight)	x100	=
Total	Total points		

Note: < 150 points − inactive CD (clinical remission), 150−300 points − mild attack, 301−450 points − moderate attack, > 450 − severe attack.

Table 4

Harvey-Bradshaw index

Symptom	Severity	Grade
General well-being	good slightly below average bad very bad terrible	0 1 2 3 4
Abdominal pain	not weak moderate strong	0 1 2 3
Diarrhea		1 point for each bowel movement liquid stool per day
Abdominal infiltrate	not availability doubtful availability presence with muscle tension abdominal wall	0 1 2 3
Complications	arthralgia, uveitis, nodular erythema, gangrenous pyoderma, aphthous stomatitis, anal fissure, new fistula, or abscess	1 point for each complication

Note: sum ≤4 – remission, 5–6 – mild attack, 7–8 – moderate attack, ≥9 – severe attack.

In the diagnosis of CD, the Leonard−Jones criteria are generally accepted, including the definition of six main signs of the disease:

1) a possible lesion from the oral cavity to the anal canal. Chronic granulomatous inflammation of the mucous membrane of the lips and cheeks, pyloroduodenal lesions, lesions of the small intestine, perianal lesions;

2) The nature of the lesion is intermittent;

3) Transmural lesion - deep ulcers, cracks, abscesses, fistulas;

4) Formation of fibrosis and intestinal strictures;

5) Aphthous ulcers and transmural lymphoid accumulations on histology;

6) Normal content of mucin in the area of active inflammation of the colon mucosa;

7) Presence of sarcoid granuloma.

The diagnosis of CD is considered reliable in the presence of 3 or more signs out of 7, as well as in the presence of a granuloma in combination with any sign.

Typical pathological changes in CD are described thickening and transmural lesions of the intestine; enlarged dim mesenteric lymph nodes; focal granulomas; deep tortuous and linear ulcerations in the form of a «cobblestone pavement», sometimes with fistulas (fig. 40); secondary strictures in scarring; alternation of areas of normal and affected mucous membranes. The clinical manifestations of Crohn's disease depend on the site of the lesion.

Common symptoms of the CD are fever, diarrhea, abdominal pain and weight loss. The pain syndrome can be in the nature of an «acute abdomen». There may be the formation of fistulas, arthritis, skin manifestations, nonspecific hepatitis. In the diagnosis of the disease, the leading place is occupied by endoscopic studies − FEGDS (in case of damage to the upper

gastrointestinal tract) and colonoscopy with biopsy − when the process is localized in the large intestine (fig. 40).

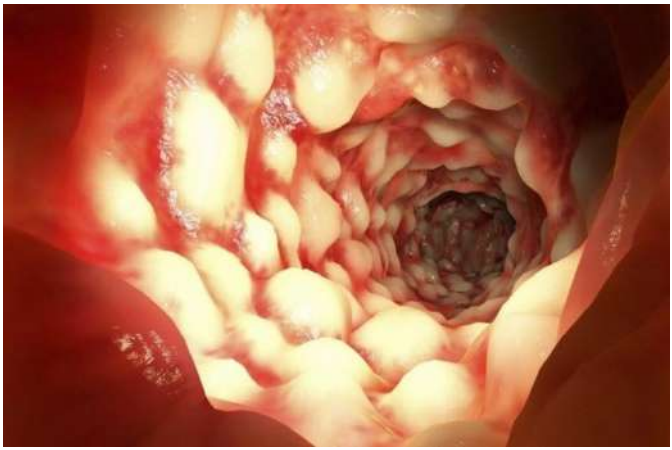


Figure 40. View of the colon affected by Crohn's disease during colonoscopy − the wall is thickened, the mucosa is in the form of a «cobblestone pavement»

Chronic diarrhea for more than 6 weeks is the main symptom of the disease. More often, diarrhea does not have blood impurities, is accompanied by abdominal pain, fever or anemia of unknown origin, symptoms of intestinal obstruction, as well as chronic anal fissures, paraproctitis, rectal fistulas. Many patients may also have extraintestinal manifestations of CD. There are three groups of extra-viscous manifestations of CD − autoimmune, associated with disease activity; autoimmune, not related to the activity of the disease and due to prolonged inflammation and metabolic disorders. Autoimmune complications associated with the activity of the disease include arthropathy, skin lesions (erythema nodosum, pyoderma gangrenosum), mucosal lesions of the type of aphthous stomatitis,

eye damage − uveitis, iritis, iridocyclitis, episcleritis. to autoimmune manifestations. Not associated with the activity of the disease include ankylosing spondylitis (sacroiliitis), primary sclerosing cholangitis, osteoporosis, psoriasis. Extraintestinal manifestations of CD caused by long-term inflammation and metabolic disorders include cholelithiasis, hepatic steatosis, steatohepatitis, peripheral vein thrombosis, pulmonary embolism, and amyloidosis. Physical examination includes a mandatory examination of the perianal region, digital examination of the rectum and sigmoidoscopy. Physical examination may reveal malnutrition fever, abdominal infiltration, external intestinal fistulas, perianal fissures and fistulas, and various extraintestinal manifestations.

Laboratory diagnostics includes a mandatory complete blood count, determination of CRP, coagulograms, determination of the concentration of protein, albumin, electrolytes in the blood serum, determination of the activity of liver enzymes. In the acute onset of diarrhea, a fecal analysis is performed to exclude intestinal infection, the study of toxins A and B to Clostridium difficile, a parasitic infestation. 4 different stool samples are needed to detect infection in 90% of cases. Fecal calprotectin is a sensitive marker of inflammatory bowel disease, as in UC. If it is necessary to identify a variant of anemia, it is necessary to determine the level of ferritin, the total iron-binding capacity of serum, serum gland, vitamin B_{12} and folic acid.

CHAPTER 6.
LIVER AND BILIARY TRACT − HIGHLIGHTS OF ANATOMY AND PHYSIOLOGY

Patients with diseases of the liver, gallbladder and pancreas meet daily both at outpatient appointments and in the emergency department of a multidisciplinary hospital. The clinical manifestations of this group of diseases are very diverse. Diffuse liver lesions are more common − chronic hepatitis, cirrhosis, cholecystitis (stone, stoneless), cholangitis, and chronic pancreatitis. Somewhat less common are focal lesions of the liver − abscesses, echinococcosis, primary liver tumors, liver metastases of tumors of other localizations (for example, ovarian tumors in women). Genetic diseases of the liver with the gradual development of cirrhosis are described − hepatocerebral dystrophy (Wilson−Konovalov's disease); hereditary hemochromatosis; liver damage in alpha-1 antitrypsin deficiency. Every year, the list of diseases is updated with new ones, including orphan diseases.

The liver is the largest gland in the digestive system. Its normal weight is about 1.5 kilograms, and when the liver vessels are filled with blood, it reaches 2 kg. Figure 40 shows the topography of the liver, gallbladder, pancreas and duodenum. The liver, biliary tract and pancreas are closely related to each other anatomically and functionally, so the pathology of one of them is always accompanied by damage to neighboring organs. The liver consists of two lobes − right and left, covered with a visceral peritoneum, under which there is a thin, dense fibrous membrane − Glisson's capsule (fig. 41). The gates of the liver are located on its lower surface. The gates of the liver include the portal vein, own hepatic artery and nerves, and the common hepatic duct and lymphatic vessels exit the gates. The common hepatic duct joins with the cystic duct of the gallbladder to form the common bile

duct, or common bile duct. Choledoch flows into the descending part of the duodenum, connecting with the pancreatic duct (Wirsung duct), forming a common hepatic-pancreatic ampulla for them, which, ending with the Futter nipple, carries the contents of the ducts into the duodenum (fig. 42).

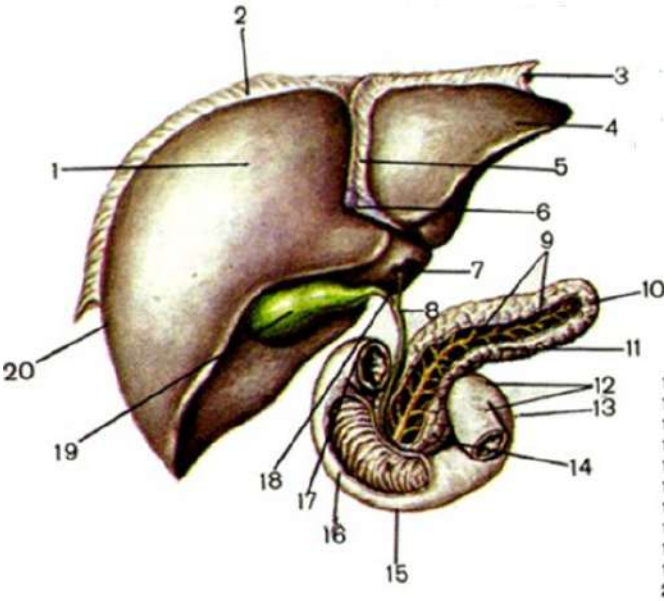
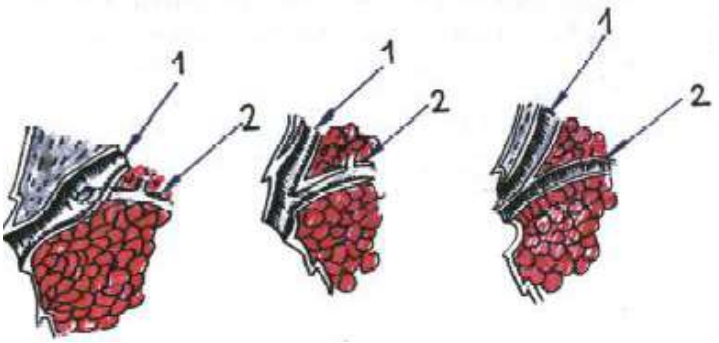


Figure 41. Topographic features of the liver, gallbladder, pancreas and duodenum. 1 – right lobe of the liver; 2 – coronary ligament; 3 – left triangular ligament; 4 – left lobe of the liver; 5 – falciform ligament of the liver; 6 – round ligament of the liver; 7 – common hepatic duct; 8 – common bile duct; 9 – pancreatic duct (Wirsung duct); 10 – tail of the pancreas; 11 – the body of the pancreas; 12 – duodenal-jejunal flexure; 13 – ascending part of the duodenum; 14 – head of the pancreas; 15 – horizontal part of the duodenum; 16 – descending duodenum; 17 – upper part of the duodenum (initial section); 18 – cystic duct; 19 – gallbladder; 20 – right triangular ligament

The functional unit of the liver is the hepatic lobule − a honeycomb-like formation built from hepatic beams. At the center of each lobule is the central lobular vein. Between the hepatic beams are sinusoids, into which blood is collected from the portal vein system and the hepatic artery. Sinusoids flowing into the hepatic vein come into contact with each hepatocyte, carrying out the entry into and out of the blood of many biologically important substances (fig. 43). Inside each hepatic beam, between rows of hepatocytes, there are bile canaliculi. From the lobules through the bile ducts, bile exits into the interlobular bile ducts, which join into larger bile ducts, then into two large bile ducts − the right and left, and then into the common hepatic duct, which, connecting with the cystic duct, forms the common bile duct. The blood supply to the liver comes from the portal vein and the hepatic artery.



a *b* *c*

Figure 42. The most common options for the confluence of the choledochus and the Wirsung duct into the duodenum: *a, b* − with the formation of a common hepatic-pancreatic ampulla; *c* − at some distance from each other; 1 − choledochus, 2 − Wirsung duct

The main functions of the liver can be very briefly summarized as follows:

1. Carbohydrate metabolism: gluconeogenesis, synthesis and breakdown of glycogen.

2. Fat metabolism: synthesis of fatty acids; synthesis and excretion of cholesterol; synthesis of lipoproteins; ketogenesis; synthesis of bile acids; 25-hydroxylation of vitamin D.

3. Protein metabolism: synthesis of plasma proteins, including some coagulation factors, but not immunoglobulins; urea synthesis.

4. Hormone metabolism: metabolism and excretion of steroid hormones; metabolism of polypeptide hormones.

5. Metabolism and excretion of drugs and foreign substances.

6. Storage of glycogen, vitamins A, B_{12}, iron.

7. Metabolism and excretion of bilirubin.

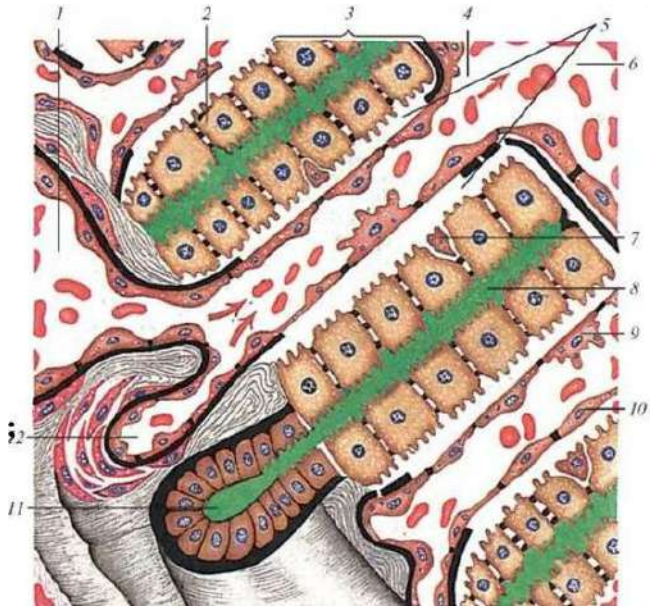


Figure 43. The structure of the hepatic lobule, where 1 is the perilobular vein, 2 is the hepatocyte, 3 is the hepatic beam, 4 is the sinusoidal hemocapillary, 5 is the presinusoidal space, 6 is the central lobular vein, 7 is the sinusoidal lipocyte, 8 is the bile capillary, 9 is stellate macrophagocyte, 10 – endothelial cell, 11 – perilobular bile duct, 12 – perilobular artery

Most liver diseases are accompanied by hepatocellular insufficiency in varying degrees of severity, as well as hepatocyte necrosis. Symptoms of liver diseases can be directly related to liver damage (jaundice during acute hepatitis), as well as those associated with damage to other organs as a result of liver failure (bleeding, portal hypertension, and others). Necrotically altered hepatocytes can regenerate, however, with significant damage to a huge number of liver cells, regeneration is impossible. In this case, fibrosis begins to form.

The gallbladder is a fibromuscular sac located in the fossa of the gallbladder on the visceral surface of the liver and covered by the peritoneum. The shape of the bladder is unstable, depending on the position of the body, the size of the liver, and age. In the vertical position of the body, the shape of the gallbladder can be in the form of an elongated oval (normotonia), rounded (hypertension) or pear-shaped (hypotension). The gallbladder concentrates bile, evacuates it into the duodenum, regulates the level of pressure in the bile ducts for the most favorable secretion of bile. The synthesis of bile in the liver occurs around the clock, but at night it decreases slightly. Bile acids are produced in the hepatocyte and then transported to the bile ducts. Following the acids, water enters the tubules and bile becomes more isotonic. Further formation of bile occurs in the bile ducts and it is constantly excreted with the liver at an average rate of 0.4 ml per minute.

Bile acids are the end product of cholesterol metabolism. During the day, the liver secretes 20−30 g of bile acids, while from 3 to 5 g are involved in the enterohepatic circulation (fig. 44).

About 80 % of bile acids are absorbed in the intestine and returned to the liver. A small amount of bile acids enters the blood and lymph. A small daily loss of bile acids in the feces (200−600 mg) is replenished by the formation of new acids by the liver.

CHAPTER 7.
COMPLAINTS AND ANAMNESIS OF PATIENTS WITH DISEASES OF THE LIVER AND GALLBLADDER

The most common complaints in patients with diseases of the liver and biliary tract are:

- **pain syndrome**;
- **intense itching** of the skin, aggravated at night and depriving the patient of normal sleep;
- **jaundice** at the time of examination or in history;
- an increase in the size of the abdomen and the associated weight gain (**ascites**);
- an unpleasant, usually **bitter taste in the mouth**;
- **belching, nausea and vomiting**, often triggered by fatty or fried foods;
- **unstable stool** (constipation is replaced by diarrhea), bloating;
- **asthenic complaints** − weakness, malaise, poor appetite, fatigue, irritability, headaches, decreased potency and libido, menstrual irregularities.

Initially, many liver diseases begin to manifest non-specific symptoms, such as fever, anorexia, nausea, vomiting, loss of appetite, fatty stools that do not flush off the toilet walls (steatorrhea). Fever often accompanies viral or alcoholic hepatitis. Steatorrhea appears with cholestasis. Jaundice can appear both with hepatocellular dysfunction and with cholestatic disorders. Jaundice is the most specific symptom of liver damage. It is often accompanied by dark urine and light-colored stools.

Pain syndrome in liver's diseases. Pain is not necessarily a sign of liver disease. Many very severe liver diseases can occur without pain − liver cancer, liver syphilis, liver echinococcus, gallbladder cancer, blockage of the common bile duct.

Pain in diseases of the liver and biliary tract can occur in the following circumstances:

1) inflammation of the peritoneum covering the liver (perihepatitis) or gallbladder (pericholecystitis);

2) with a rapid and significant increase in the liver, leading to stretching of its peritoneal cover − Glisson's capsule;

3) with spastic contractions of the muscles of the gallbladder and large bile ducts;

4) with significant stretching of the gallbladder.

If perihepatitis or pericholecystitis appears, it means that the inflammatory process from the liver or other organs has passed to the peritoneum surrounding the liver and gallbladder. In acute perihepatitis and pericholecystitis, pain appears in the right hypochondrium, is intense, sometimes «shooting», and in case of pericholecystitis it often radiates to the right shoulder, right shoulder blade, interscapular region. The pain is aggravated by breathing and by pressure on the right hypochondrium, by tapping on the right costal arch. In chronic perihepatitis, pericholecystitis, the pain is less intense, it becomes aching, dull. When the glisson capsule of the liver and gallbladder is fused with neighboring organs and tissues, the pain begins to increase when the patient is turned from one side to the other, when the body is bent backward, lifting weights, straining.

A rapid increase in the liver with stretching of the Glisson capsule with the occurrence of pain as a result of this is most often observed with stagnation of venous blood in the liver due to heart failure. The so-called «stagnant liver» is formed. Due to overfilling of the right ventricle and right atrium with blood, there is stagnation of venous blood in the system of the inferior vena cava, dilatation of the inferior vena cava. Then there is a plethora of hepatic veins and the liver rapidly increases in volume. Due to the anatomical features of the liver, its left lobe increases faster,

which gives pain in the epigastric region. The pain is aggravated by palpation, exercise, after eating.

It is necessary to define the main clinical criteria for «congestive liver»:

1) the lower edge of the liver protrudes from under the edge of the right costal arch;

2) the lower edge of the liver is rounded;

3) the lower edge of the liver is dense, but not of stony density;

4) soreness of both the lowest edge of the liver and the palpated anterior surface, this pain is constant, and aggravates on palpation;

5) with strong pressure on the anterior surface of the enlarged congestive liver, the jugular veins in the neck swell. This swelling instantly disappears when the pressure stops;

6) with a strong venous congestion in the liver, the flow of bile in the intrahepatic bile ducts becomes difficult, therefore, subicteric skin and mucous membranes may appear, especially noticeable on the conjunctiva.

If stagnation in the liver lasts for a long time, then the transformation of the liver begins, the germination of connective tissue in all hepatic structures, cirrhosis of the liver is formed. The cirrhotic liver differs from the stagnant one in greater density, reaching the stony one. The lower edge of the liver becomes sharp and painless. With cardiac cirrhosis of the liver, blood circulation through the intrahepatic branches of the portal vein becomes difficult, which can cause secondary ascites. Ascites can exist without edema in other parts of the body. If ascites becomes very intense, then anasarca develops secondarily.

Pain during spastic contraction of the muscles of the gallbladder and large bile ducts is most often observed when the ducts are blocked by stones (biliary colic), as well as in acute cholecystitis, when normal coordination between the contractions

of the muscles of the gallbladder and the biliary tract and their sphincters is disturbed when bile moves to the duodenum. This phenomenon is called «biliary dyskinesia». Less often, the cause of obturation may be a tumor or helminthic invasion.

Normally, under the influence of impulses from n.vagus, there is a contraction of the muscles of the gallbladder and relaxation of the sphincter of Oddi (located at the confluence of the bile ducts into the duodenum, this process contributes to the normal promotion of bile. With excessive excitation of the vagus nerve, coordination between contraction of the gallbladder and relaxation of the sphincter of Oddi disturbed and often there can be simultaneous contraction of both the gallbladder and the sphincter of Oddi.This causes stagnation of bile in the biliary tract, impaired emptying of the gallbladder, reflex intense contraction of the muscles of the gallbladder, intense pain syndrome − dyskinetic pain.The causes of excessive excitation of the vagus nerve can be its reflex irritation from foci in the abdominal cavity (appendicitis, inflammation of the pelvic organs), less often − intense emotional disturbances.

The main differences between dyskinetic pain and pain in biliary colic and cholecystitis:

1) pain is not associated with food intake and its quantity;

2) lasts for several minutes, while with biliary colic and cholecystitis, the duration of pain is long − for hours and even days;

3) dyskinetic pains are never accompanied by chills, fever and other signs of inflammation (leukocytosis, accelerated ESR, increased concentration of C-reactive protein, etc.);

4) at the end of an attack of dyskinetic pain, the patient does not feel any spontaneous or palpatory pain in the liver and gallbladder.

Gallbladder (or hepatic) colic − an attack of very intense pain in the right hypochondrium, due to spasmodic contraction of the smooth muscles of the distended gallbladder or bile ducts due to obstruction of the bile ducts.

An attack of biliary colic can be triggered by abundant fatty or spicy foods, alcohol, physical activity, bumpy driving, emotional experiences.

Pain during biliary colic is very intense, sharp, excruciating, often unbearable. The pain makes the patient literally kneel down, not finding a comfortable position for himself, rush around the room, scream or moan loudly. It hurts the right hypochondrium, epigastric region, the pain can radiate to the right shoulder, right shoulder blade. At the height of the pain, nausea and vomiting with an admixture of bile are characteristic, which does not bring relief. There is a taste of bitterness in the mouth. The duration of the pain can vary from several hours to several days. Attacks often occur at night. In women, attacks of biliary colic often occur on the eve of or during menstruation, as well as during pregnancy (more often in the second trimester) or in the postpartum period. Frequent recurrence of attacks, accompanied by chills and fever, as well as aching pain in the gallbladder between attacks indicates the presence of cholecystitis.

On palpation, the abdomen is swollen, sharply painful in the right hypochondrium, at the point of the gallbladder there is a muscular defense. The patient's breathing is quickened, the skin is covered with profuse cold sweat, the tongue is lined with a dirty coating. After a few hours, jaundice may appear. The pancreas may be involved in the pathological process.

An attack of biliary colic is often accompanied by fever in a patient with choledocholithiasis; at the end of the attack, the fever disappears. Pain from stretching of the gallbladder occurs only when this stretching develops very quickly, for example, when the bile ducts are bent. These pains are less intense than with biliary

colic, they are aching in nature. Sometimes the pain can be aggravated by the attachment of spastic contractions of the muscles of the bladder. Irradiation of pain is less pronounced than with biliary colic, but may also be present.

Pain syndrome in diseases of the liver and gallbladder is more often described as heaviness and pressure in the right hypochondrium. Pain may be associated with irritation of the peritoneum covering the liver, with perihepatitis (for example, with a tumor or abscess of the liver) and pericholecystitis. They are usually intense and radiate up to the right shoulder, and are aggravated by palpation of the right hypochondrium.

Stretching of the liver capsule associated with an increase in its size (hepatomegaly) also causes pain. Such pain syndrome may accompany liver congestion against the background of decompensated heart failure.

Paroxysmal intense pain can be caused by biliary dyskinesia, as well as spastic contraction of smooth muscle cells of the gallbladder, biliary tract in biliary colic. Gallstone colic is usually caused by the movement of a stone through the bile ducts. The pains begin suddenly, quickly become unbearable, often radiate upwards, are accompanied by nausea and vomiting, which does not bring relief.

With chronic inflammation of the gallbladder, patients may complain of aching, dull pain in the right hypochondrium, aggravated by palpation of the gallbladder point (the angle between the right costal arch and the outer edge of the right rectus abdominis muscle), as well as by pressing on the point in the neck on the right between the legs sternocleidomastoid muscle (phrenicus syndrome).

Liver diseases are often accompanied by chronic pancreatitis, peptic ulcer of the stomach or duodenum, which can also be causes of abdominal pain.

Nausea and vomiting in diseases of the liver and biliary tract. Nausea and vomiting are often manifested at the height of biliary colic, dyskinetic pain, pain in cholecystitis. Vomiting does not bring relief from pain, unlike vomiting in diseases of the stomach.

Asthenic syndrome. The appearance of asthenic syndrome (fatigue, irritability, headaches) is associated with intoxication due to violations of the synthetic and detoxifying (disinfecting) functions of the liver, which is clinically manifested by disorders of the central nervous system − hepatic encephalopathy. With an increase in intoxication, these signs intensify, sleep disorders (dyssomnia, insomnia, sleep inversion) join, and then loss of consciousness (hepatic coma). The detoxification function of the liver is to neutralize toxic substances formed during the decay of proteins in the large intestine and entering the liver through the portal vein system. A number of mild intoxication phenomena, such as irritability, fatigue, headaches, are detected in many liver diseases − hepatitis, cirrhosis, etc.

A sharp degree of intoxication is called «hepatargia». Hepatargia occurs in the terminal stages of severe liver diseases, such as liver cancer, acute liver failure. Hepatargia is manifested by intense general weakness, irritability, headaches, persistent insomnia, or vice versa − drowsiness, anxiety, delirium, convulsions, and may result in hepatic coma (coma hepatica), during which the patient dies in an unconscious state.

The presence of blood in the vomit often indicates bleeding from esophageal varices, or the presence of erosive gastritis, erosive esophagitis, as manifestations of portal hypertension with blood stasis in the portal vein system.

Anamnesis. There are many risk factors for developing liver disease. First of all, alcohol. Alcoholic liver disease is characterized by erectile dysfunction and the formation of gynecomastia. When questioning any patient, it is necessary to clarify the presence of chronic viral hepatitis B, C, D, G. It is important that there is a history of blood transfusions, a homosexual history, injection drug addiction, which theoretically can cause infection with viral hepatitis. The profession of medical workers is also a risk factor for the development of viral hepatitis. In epidemiological terms, it is necessary to clarify the fact of the patient's stay in endemic foci of opisthorchiasis, leptospirosis, yellow fever. The fact of taking hepatotoxic drugs − tetracycline, furadonin, methyldopa, anti-tuberculosis drugs (isoniazid, ethambutol), some psychotropic drugs, estrogens (including oral contraceptives), blood transfusions according to history is important. A risk factor is the use of certain herbs and plant products. A family history of advanced liver disease is also important. So it is possible to identify the hereditary nature of Wilson−Konovalov's disease, hemochromatosis, alpha-1-antitrypsin deficiency, familial benign hyperbilirubinemia.

Jaundice. Jaundice is of particular diagnostic value in liver pathology and is a sign of hyperbilirubinemia (fig. 44). Pathogenetically, jaundice is a consequence of the deposition in the skin and mucous membranes of the bile pigment bilirubin due to its increased concentration in the blood.

The terms «jaundice» and «icterus» are used to describe the yellow coloration of the skin and sclera. Jaundice is best seen on the face, sclera, torso. To understand the cause of jaundice, it is necessary to remember the mechanism of hyperbilirubinemia, the mechanism of formation of bile pigment and its release into bile.

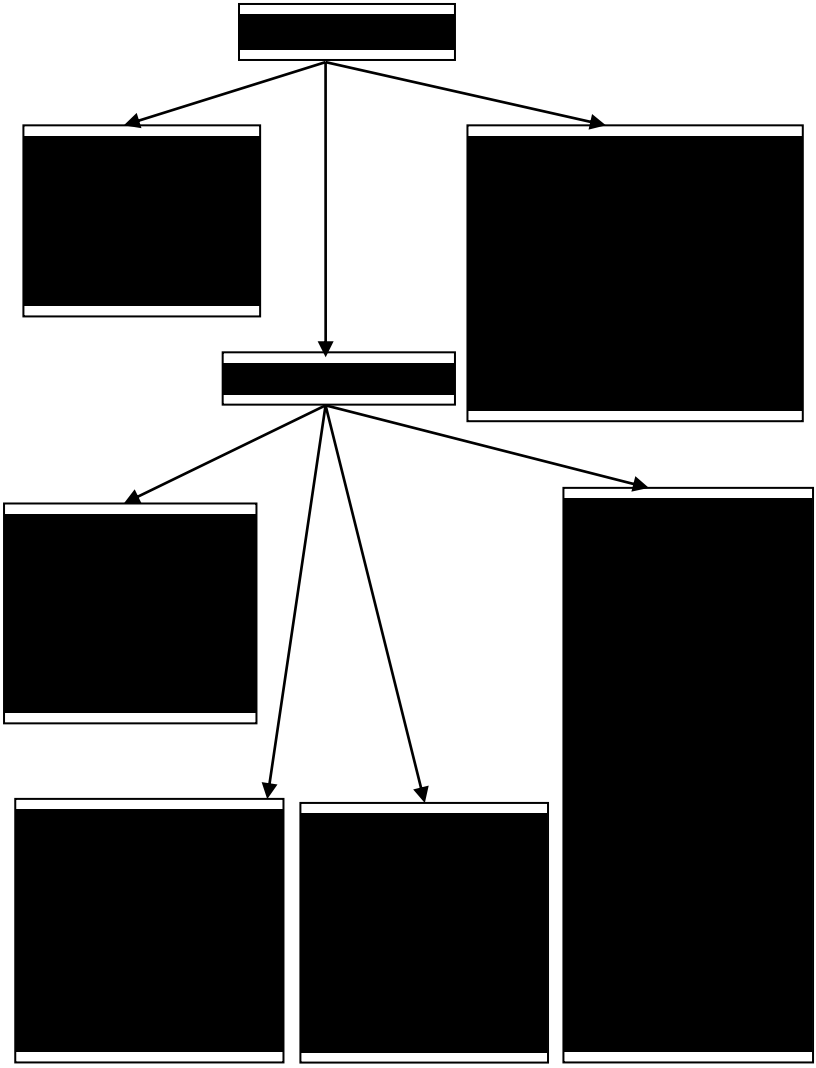


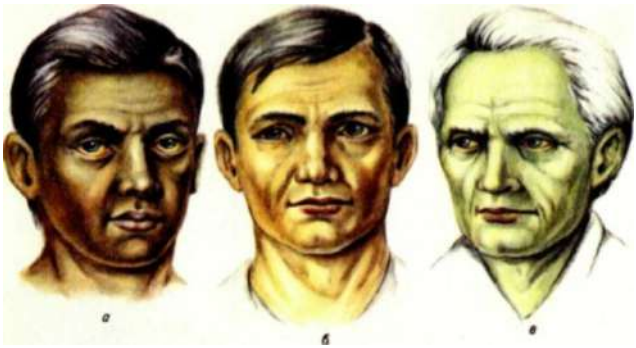
Figure 44. All possible pathogenic variants of Jaundice

In adults with new onset jaundice, **8 diseases explain 98 % of all probable diagnoses:** viral hepatitis, alcoholic liver disease,

chronic hepatitis, drug-induced liver injury, gallstone disease and its complications, pancreatic cancer, primary biliary cirrhosis, and primary sclerosing cholangitis

The formation of bilirubin occurs in the cells of the reticuloendothelial system. Elements of the reticuloendothelial system are scattered throughout the body (bone marrow, spleen, skin, etc.), but are also present in the liver in the form of Kupffer cells. The formation of bilirubin occurs in these Kupffer cells of the liver. The raw material for the formation of bilirubin is the hemoglobin of erythrocytes, which is released during their physiological decay. From the hemoglobin of erythrocytes, a protein body is formed − globin and iron-containing hematin. Hematin is captured by the cellular elements of the reticuloendothelial system (mainly the spleen), iron is split off from it, water is added, and eventually free bilirubin is formed.

In accordance with these three options for the development of hyperbilirubinemia, there are three main mechanisms of jaundice − prehepatic (hemolytic), posthepatic (mechanical) and hepatic (parenchymal) (fig. 45). The most common jaundice is due to parenchymal liver damage.



а *б* *в*

Figure 45. Skin integuments with various types of jaundice: a − obstructive jaundice, b − parenchymal jaundice; c − hemolytic (prehepatic) jaundice

Direct bilirubin, or bilirubin-diglucuronide, or bound bilirubin is formed in hepatocytes from free bilirubin in three stages.

The first stage is the capture of bilirubin by the liver cell after the cleavage of albumin. The second stage is the formation of a water-soluble bilirubin-diglucuronide complex. The third stage is the release of conjugated bilirubin into the bile ducts.

An increase in the content of bilirubin in the blood, preceding the development of jaundice, can have three development options:

1) due to the reabsorption of bile bilirubin into the blood when its entry into the duodenum is obstructed;

2) with insufficient release of bilirubin from the blood by pathologically altered Kupffer cells of the liver;

3) due to increased production of bilirubin by the cells of the reticuloendothelial system and its increased entry into the blood, as a result of which even normally functioning liver cells do not have time to isolate it from the blood. This occurs with increased hemolysis of erythrocytes.

Mechanical (subhepatic) jaundice occurs when the reabsorption of bilirubin into the blood with obstacles to the flow of bile from the gallbladder and ducts into the duodenal ampulla. The patient's skin color becomes olive-yellow.

Obstructive jaundice is observed:

1) when a gallstone obstructs the hepatic or common bile ducts; blockage of only the cystic duct will not cause jaundice, since this does not stop the flow of bile into the duodenum;

2) with blockage of the hepatic and common bile ducts by a cancerous tumor;

3) with compression of the hepatic or common bile ducts by a primary or metastatic cancerous tumor in the region of the liver gate;

4) in case of compression of the final process of the common bile duct, which passes through the thickness of the head of the pancreas, by a cancerous tumor;

5) in case of chronic inflammation of the pancreas, with the localization of the main inflammatory process in the head, due to compression of the final segment of the common bile duct;

6) obstructive jaundice is possible with cancer or duodenal ulcer, especially if they are located at the confluence of the common bile duct into the intestine;

7) with some liver diseases that cause compression of the hepatic or common bile ducts − a large echinococcal cyst, liver cancer, liver syphilis;

8) with cicatricial narrowing of the hepatic or common bile ducts as a result of inflammation;

9) very rarely − due to spasm of the sphincter of Oddi («emotional» jaundice) caused by neuropsychic moments.

The intensity of skin coloration in obstructive jaundice is more pronounced than in other forms, and increases with the duration of the disease. With a prolonged state of obstructive jaundice (within months), the yellow-green color of the skin begins to acquire a blackish tint, such jaundice is called melas icterus and is more often observed in cancer of the pancreatic head. With obstructive jaundice, more often than with other forms, skin itching is felt.

With obstructive jaundice, bradycardia is often observed due to irritation of the cardiac branch of the vagus nerve by bile acids circulating in the blood.

With true obstructive jaundice, there will never be an enlargement of the spleen, in contrast to parenchymal and hemolytic jaundice.

The most important sign of obstructive jaundice is acholic stool (light, gray-white, reminiscent of the color of old window putty). There is no stercobelin pigment in the acholic stool; stool

analysis for the presence of stercobelin can be performed in the laboratory.

Parenchymal (hepatic) jaundice is observed:

1) with infectious diseases that cause damage to the liver parenchyma − chronic viral hepatitis, Botkin's disease, malaria, syphilis, relapsing fever;

2) with some intoxications − poisoning with phosphorus, chloroform, mushrooms, male fern;

3) with sepsis;

4) rarely − with stagnation of venous blood in the liver due to heart failure. The patient's skin color becomes saffron-yellow.

Parenchymal jaundice can also be differentiated into three variants, depending on the level of microsomal damage (scheme) − pre-microsomal, microsomal, post-microsomal.

In 50−60 % of cases of parenchymal jaundice, an enlarged spleen is observed. In the urine with parenchymal jaundice, an increased content of urobilin and bilirubin is found. The appearance of urobilin is a violation of the function of Kupffer cells of the liver. Feces with parenchymal jaundice are not acholic, it is colored, although weaker than normal.

Hemolytic (prehepatic) jaundice occurs when hyperbilirubinemia occurs due to increased production of bilirubin in the cells of the reticuloendothelial system due to pathological enhanced hemolysis of red blood cells. The patient's skin color acquires a lemon-yellow hue.

Hemolytic jaundice occurs when there is an increased breakdown of red blood cells:

1) some cases of croupous pneumonia (increased breakdown of red blood cells in the alveolar exudate);

2) Biermer's anemia;

3) lung infarction (destruction of erythrocytes of blood that has entered the area of infarction);

4) rupture of the tube during ectopic pregnancy;

5) internal hemorrhages;

6) some septic processes caused by pathogens that have hemolytic properties;

7) malaria − the breakdown of red blood cells under the influence of parasites that have invaded them;

8) poisoning with hemolytic poisons − arsenic hydrogen, phenylhydrazine;

9) hemolytic jaundice, as a special chronic condition accompanying chronic hemolytic anemia, when the resistance of erythrocytes is reduced, and they disintegrate due to imperfect erythropoiesis.

With hemolytic jaundice, there will never be bilirubin in the urine, in contrast to the mechanical and parenchymal forms of jaundice. However, there will be urobilin in the urine. The absence of bilirubin in the urine is due to the fact that an excess amount of free bilirubin circulates in the blood, and the liver is not able to completely convert it into direct, bound. Indirect bilirubin does not dissolve in water, so it does not pass into the urine. The increase in the concentration of urobilin in the urine is due to its increased intake from the intestine, which contains an increased concentration of bilirubin. Normally, all urobilin from the intestine is absorbed into the blood of the portal vein and enters the liver, where it is processed into bilirubin or destroyed, only a very small amount of it can enter the blood. With hemolytic jaundice, the amount of urobilin coming from the intestine is so large that the liver does not physically have time to process it, and a significant part of the urobilin enters the bloodstream, and from there it is excreted in the urine.

Fecal masses in hemolytic jaundice are more intensely colored than normal (stool pleiochromia), the color is up to brick red. This is the result of the release of a significant amount of bilirubin from the blood into bile by normally functioning liver cells and an increased content of stercobilin.

An icteric shade of the skin is not always a manifestation of jaundice. It is necessary to carefully collect an anamnesis. So eating a large number of carrots can cause a saffron yellow or orange-yellow skin tone. In this case, the color of the mucous membranes and sclera will be normal. The use of quinacrine, even in therapeutic doses, can cause jaundice, but here, too, the sclera will remain white.

Upon admission of a patient with jaundice, it is necessary to clarify the following points from the anamnesis:

1) with regard to the development of viral hepatitis:

− whether the patient had blood transfusions (especially before 1990);

− whether there has been intravenous community administration of medicinal substances or drugs in the last 3 years;

− features of sexual life − anal sex, sex with a prostitute, a history of sexually transmitted diseases, many sexual partners, sexual contact with patients with hepatitis B, C, sexual contact with intravenous drug users;

− contacts with patients with jaundice;

− changes in taste and smell;

− an injection with an injection needle threatening infection with infectious diseases;

− whether the patient worked in the hospital − a nurse in the treatment room, hemodialysis department; operating surgeons and traumatologists;

− use of shared toothbrushes, shaving accessories;

− piercing, ear piercing, recent tattoos;

− inhalation of cocaine.

2) with regard to the development of viral hepatitis A (Botkin's disease):

− travel to endemic areas, settlements;

− eating raw mollusks (probably grown in contaminated water);

− stay in places of possible accumulation of patients with hepatitis − restaurants, canteens, preschool institutions.

3) in relation to drug-induced liver injury:

− analysis of all medications taken by the patient;

− taking drugs without a doctor's prescription (self-medication);

− long-term use of multivitamins (especially vitamin A);

Separately clarify the intake of medicinal herbs, home remedies, dietary products, alcohol-containing tinctures.

4) with regard to alcoholic liver disease:

− Clarify with the patient and his family members the amount of alcohol consumed both now and in the past.

− Ask if you have ever experienced withdrawal symptoms.

− CAGE-criteria (cessation of alcohol intake, irritability, guilt, the need to get drunk).

− Identification of diseases comorbid with alcoholism − chronic pancreatitis, peripheral polyneuropathy.

5) other liver diseases:

− itching (possible due to intra- and extrahepatic cholestasis);

− dark urine, light stools;

− recent changes in the menstrual cycle − amenorrhea often accompanies chronic liver disease, cirrhosis;

− symptoms of biliary colic, chronic cholecystitis;

− chronic inflammatory bowel disease in history (may accompany primary sclerosing cholangitis, history of blood transfusions).

− occupational history, especially exposure to hepatotoxic substances.

Skin itching. Skin itching is a symptom of liver disease, indicating cholestasis. The extreme degree of pruritus (excruciating, aggravated at night, with a large number of often infected skin scratches) is observed in the presence of intrahepatic or extrahepatic obstruction of the biliary tract. Itching is often associated with jaundice, but may occur without it. Sometimes itching reaches a significant degree, depriving the patient of sleep. Scratches can get infected, which causes various skin diseases. The cause of itching, is the retention of metabolic products in the blood, which being deposited in the skin, irritate its nerve endings. These products are thought to be bile acids. It is possible that itching is also caused by entering the bloodstream with subsequent deposition in the skin of some poisonous products of intestinal decay that are not subject to decontamination in a diseased liver. Escoriations (skin scratches) indicating itching may indicate intra- and extrahepatic cholestasis accompanying primary biliary cirrhosis, primary sclerosing cholangitis, cholangiolithiasis, cholangitis.

Stool changes. Changes in color, consistency, amount of feces are specific for diseases of the hepatobiliary system. In adults, the color of feces is brown-brown due to the presence of the stercobelin pigment. With the termination or a sharp decrease in the flow of stercobelin into the intestine, the stool becomes gray or off-white. This is typical for situations where bile for some pathological reasons does not enter the intestine (tumor of the head of the pancreas with compression of the bile ducts, gallstone or common bile duct with obstruction of the bile ducts, atresia of the bile ducts, compression of the bile ducts by a tumor of neighboring organs and tissues, and other reasons). Discolored feces are called acholic.

A lighter than normal color of feces can be observed in diseases of the liver parenchyma, against which there is

insufficient synthesis of bile and its entry into the intestine (chronic hepatitis, cirrhosis).

The main «complexes» of hepatic symptoms and signs are as follows:

1) The presence of jaundice, careful history taking, physical examination and analysis of basic laboratory data allows the attending physician to establish the correct diagnosis with a certainty of 85 %.

2) The triad of ascites, splenomegaly, and dilated venous collaterals on the anterior abdominal wall indicates obvious portal hypertension.

3) The presence of two symptoms (ascites and asterixes) and two laboratory signs (an increase in prothrombin time of more than 3 s and hypoalbuminemia of less than 2.8 g/dl) makes it possible to establish the diagnosis of liver cirrhosis.

4) Three symptoms − enlargement of the parotid glands, gynecomastia and Dupuytren's contracture − point to the likely chronic alcohol abuse.

5) The appearance of fresh blood in the feces is due to bleeding from varicose hemorrhoidal veins. Melena is caused by bleeding from the upper gastrointestinal tract. The causes of bleeding can also be gastritis of alcoholic origin and Mallory-Weiss syndrome.

Anorexia is one of the main symptoms of viral heptitis and tumors that affect the liver, biliary tract, pancreas, and large intestine. With a loss of body weight of more than 4.5 kg, it is necessary to think about the tumor.

Chills, fever, myalgia, headaches suggest viral hepatitis, especially hepatitis A. Chills, fever and pain or heaviness in the right hypochondrium − biliary tract disease − cholelithiasis, especially choledocholithiasis, ascending cholangitis.

Arthritis, «flying» joint syndrome can be symptoms of viral hepatitis, autoimmune hepatitis, primary sclerosing cholangitis, granulomatous liver diseases (for example, sarcoidosis).

Alcohol use is often and long-term associated with the development of alcoholic liver disease. It is important to ask the patient and his relatives about the amount of alcohol consumed previously and currently. To evaluate the data, it is convenient to use the following information: 30 ml of whiskey or 360 ml of beer or 120 ml of red wine contain 10−11 g of alcohol. 10−11 g of alcohol corresponds to 1 unit of alcohol. Drinking more than 3 units per day or more than 21 units per week is dangerous, especially for women. The threshold for the development of alcoholic liver damage is the daily consumption of 30 g of alcohol for women and 60 g for men for 5−10 days. In the presence of other liver diseases for example = chronic viral hepatitis C − the damaging doses of alcohol will be lower. It is necessary to clarify whether the patient has ever experienced hangover symptoms recently. It is convenient to use the CAGE criteria for this, including answers to 4 questions:

1. Has the patient tried to stop drinking alcohol?

2. Did the patient become angry when asked about the use of alcohol?

3. Does the patient feel guilty about drinking alcohol?

4. Does the patient have a need to get drunk in the morning? Positive answers to these questions can suggest alcoholism.

CHAPTER 8.
PHYSICAL METHODS FOR EXAMINING THE LIVER AND BILE DUCTS

The main physical method of examining the liver is palpation, but it should be preceded by a general examination and percussion.

Examination of a patient with liver diseases. Sclera icterus is an obvious symptom of liver pathology, better visible in daylight (fig. 46).

Figure 46. Icteric sclera

This symptom is usually detected when the bilirubin level is more than 3 mg/dl. Yellowness of the skin appears at higher values of bilirubin. Paleness of the skin may indicate advanced liver disease or a malignant tumor. Marks from intravenous and subcutaneous injections, especially on the shins, thighs and other atypical places suggest drug addiction. Excoriations indicate pruritus, which is most intense in patients with primary biliary cirrhosis and sclerosing cholangitis. Petechiae and ecchymosis indicate coagulation disorders, especially thrombocytopenia.

In a patient with jaundice, it is necessary during a general examination to assess the presence of icterus of the sclera (maybe false jaundice), pallor of the skin, emaciation, traces of injections, excoriations, ecchymosis and petechiae, soreness and weakness of the muscles, damage to peripheral lymph nodes, signs of pneumonia, congestive heart failure.

In a healthy person, the liver, gallbladder and pancreas are not visible during examination. With a significant increase and omission of the liver, emaciation of the patient, the liver can be determined visually below the costal arch.

The liver may be visible in the following situations:

1) liver cancer − the liver can be significantly enlarged, sometimes deformed, the patient is very emaciated;

2) with syphilis of the liver − the liver is very enlarged and deformed;

3) echinococcosis of the liver − the size of the liver can reach gigantic;

4) liver abscess;

5) sometimes − congestive liver;

6) hypertrophic variant of liver cirrhosis.

In all these cases, the abdomen loses its symmetry, a visible protrusion appears in the right hypochondrium, the lower edge of the protrusion corresponds to the lower edge of the liver. From above, the protrusion goes under the right costal arch, while there is no normal slight retraction of the costal arch. With echinococcosis and liver cancer, an increase in the liver is possible not only downwards, but also upwards. Then the protrusion of the right half of the chest, similar to exudative pleurisy, can be determined, but unlike the latter, the intercostal spaces will not be smoothed out. It is possible that the respiratory excursion of the lower edge of the liver will be visually determined. In this case, general exhaustion is accompanied by an increase in the abdomen due to the accumulation of fluid in the

abdominal cavity (ascites) (fig. 47). The presence of dilated veins of the anterior abdominal wall («head of a jellyfish») makes it possible to state with a high probability the presence of portal hypertension.

Figure 47. Examination of the abdomen of a patient with ascites in a vertical position. The abdomen is sagging, the navel is protruding, the vascular venous network on the anterior abdominal wall

In the presence of ascites in the vertical position of the patient, the abdomen looks saggy, because the fluid flows down, you can see a protruding navel due to an increase in intra-abdominal pressure, this sign distinguishes an increase in the abdomen with ascites (figure), while with significant obesity and the presence of large intra-abdominal tumors, because with them the navel sinks. In a horizontal position, the abdomen is flattened, and its lateral sections swell («frog belly»).

With long-term cirrhosis, changes in the terminal phalanges of the hands are often found in the type of drumsticks. With tricuspid valve insufficiency, it is possible to visually determine the pulsation of the liver.

Some progressive liver diseases are accompanied by a number of non-specific symptoms − non-infectious fever, arthritis, vascular changes with Raynaud's syndrome (dead fingers syndrome), dry syndrome (Sjögren's syndrome − insufficient production of saliva (xerostomia), lacrimal fluid (dry conjunctivitis), widespread caries teeth). Sometimes liver disease is accompanied by only similar symptoms, and only a study of the anamnesis and the results of the study of the liver, including biopsy, allow the diagnosis of primary liver disease with extrahepatic manifestations. Tremor, drowsiness, uncritical behavior indicate the development of hepatic encephalopathy.

The tongue in patients with liver disease often has a smooth surface and a bright red color – «crimson tongue» (fig. 48).

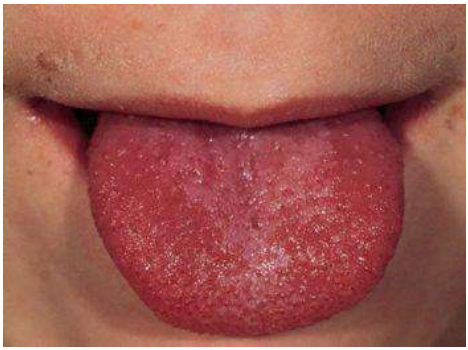


Figure 48. «Raspberry» tongue in cirrhosis of the liver

In men, unilateral or bilateral enlargement of the mammary glands (gynecomastia) and impaired hair growth on the

chin, chest, and abdomen are common. In women, hair growth in the armpits and pubis decreases. An increase in the size of the abdomen (sometimes very rapid) may be due to the accumulation of ascitic fluid in the abdominal cavity as a result of obstruction of the outflow of blood from the intestine, through the portal vein, significant flatulence (due to a violation of the digestive processes in the intestine in violation of bile secretion) or a sharp hepatosplenomegaly.

There are a number of signs that allow not only to suspect liver pathology, but also to suggest its etiology. In relation to these signs, there is a term «hepatic signs», which are divided into small and large.

Minor «hepatic signs» include:

1) Telangiectasia or «spider veins» − local excessive expansion of capillaries and small vessels (fig. 49). Asterisks are pulsating angiomas slightly rising above the surface of the skin, from which small vascular branches branch, ranging in size from a pinhead to 0.5−1 cm. They are located on the neck, face, shoulders, hands and back, less often on the mucous membranes of the nose, mouth, body. Their number can vary from single to scattered in large numbers – «fields of spider veins».

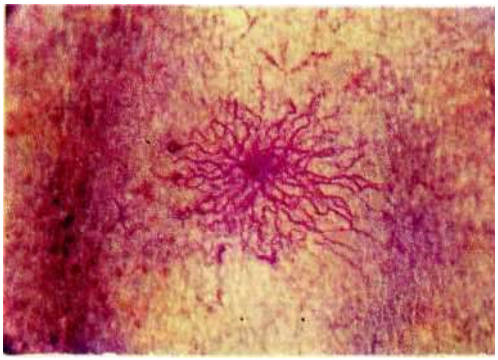


Figure 49. Large spider vein (telangiectasia)

The arterial asterisk consists of a central arteriole and numerous small vessels diverging from it, resembling the legs of a spider. With point pressure on the central part of the asterisk, it turns pale. Telangiectasias are characterized by a peculiar increase in the skin vascular pattern, which, when viewed close, resembles a pattern of a banknote interspersed with colored threads (a symptom of a «banknote»). Spider angiomas are usually found in the basin of the superior vena cava, more often on the anterior surface of the chest. If the number of these vascular «spiders» exceeds 12 pieces, then you need to think about portal hypertension.

2) Palmar erythema or «liver palms» − bright red erythema in the area of \u200b\u200b the elevation of the thumb and little finger. Often similar changes occur on the feet (fig. 50). The clinic is due to hyperestrogenemia − an increased content of estrogens in the blood due to their reduced destruction in the liver, and possibly − the opening of arteriovenous anastomoses. The symptom is more common in liver cirrhosis of alcoholic or viral etiology; symmetrical redness of the palms and soles is characteristic, especially in the thenar and hypothenar regions. When pressed, the reddened places turn pale, and when the pressure stops, they quickly turn red again.

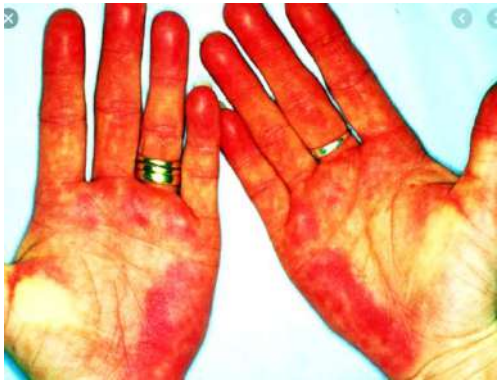
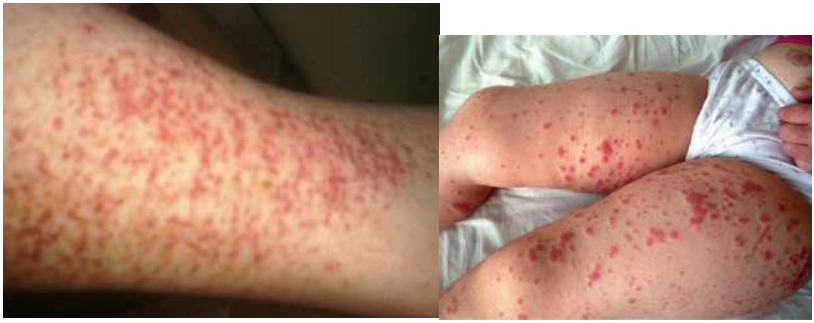


Figure 50. Palmar erythema

3) Hemorrhagic syndrome with the appearance of petechiae and ecchymosis on the skin, bleeding of the mucous membranes of the nose and mouth − a frequent syndrome, sometimes ulcers form at the site of vascular changes. Under petechiae, it is necessary to understand small-point skin hemorrhages, under ecchymosis − small-spotted hemorrhages exceeding 3 mm in diameter. These manifestations are associated with a violation of the synthesis of coagulation factors in the liver − primarily prothrombin or thrombocytopenia (fig. 51, *a, b*).



a *b*

Figure 51. Petechiae − punctate hemorrhagic rash −*a*; Ecchymosis is a larger hemorrhagic rash, more than 3 mm in diameter − *b*

4) Xanthomas and xanthelasmas with typical localization in the eyelids may indicate manifestations of primary biliary cirrhosis. Xanthomas or xanthomatosis - deposition in the skin, tendons and other tissues, sometimes in the internal organs of cholesterol and (or) triglycerides in the form of focal accumulations (fig. 52). Xanthomatosis develops with an increased concentration of lipoproteins in the blood, which includes all cholesterol and triglycerides in blood plasma. Distinguish between tuberculate and flat xanthomas of the skin and tendon xanthomatosis, which are characterized by intracellular deposition of mainly cholesterol, giving the cells a

foamy appearance (foamy, or xanthoma, cells). also eruptive xanthomas with extracellular deposition of predominantly triglycerides.

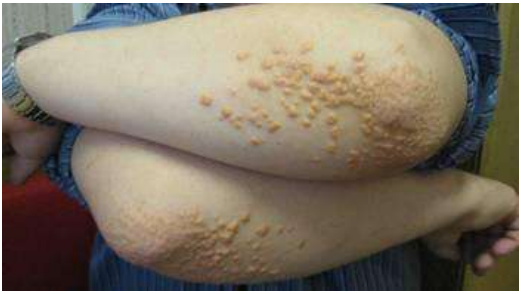


Figure 52. Multiple xanthomas – xanthomatosis

There are primary xanthomatosis caused by a hereditary disorder of lipoprotein metabolism, and secondary − due to hyperlipoproteinemia, which develops in various diseases, including diabetes mellitus, hypothyroidism, nephrotic syndrome, biliary cirrhosis of the liver.

Xanthelasmas are neoplasms localized on the upper eyelid at the inner corner of the eye and resembling a small plaque in shape. It can be single or multiple (fig. 53).

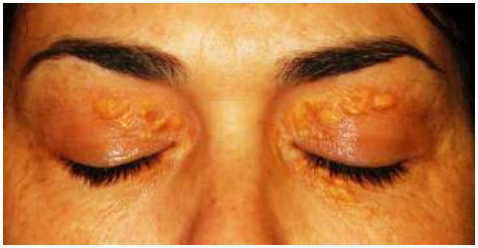


Figure 53. Multiple xanthelasmas − neoplasms on the upper eyelids

5) Gynecomastia − an increase in the mammary glands in men, due to hyperestrogenemia as a result of insufficient metabolism of excess estrogens by the affected liver (fig. 54).

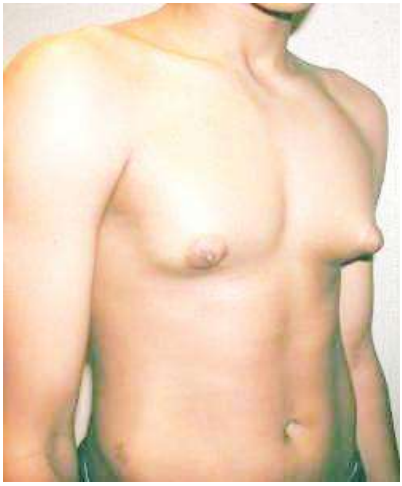


Figure 54. Gynecomastia in a young man with liver disease

6) Giant parotitis − an increase in the parotid salivary glands (fig. 55) is often found in patients with cirrhosis of the liver or alcoholic hepatitis.

Figure 55. Giant mumps in an adult - visible asymmetry of the face

7) Dupuytren's contracture − fibrous cicatricial seals of the palmar aponeurosis and tendons of the flexor muscles of the fingers (fig. 56).

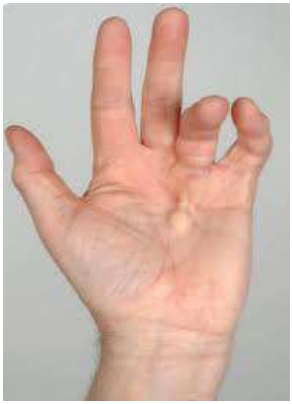


Figure 56. Dupuytren's contracture

8) Injection of scleral vessels (fig. 57).

9) The Kaiser–Fleischer ring, characteristic of Wilson–Konovalov's disease − a change in the cornea in the form of yellowish-green or greenish-brown pigmentation along its periphery, due to copper deposition (fig. 57).

Figure 57. Kaiser-Fleischer ring in a patient with Wilson–Konovalov disease and injection of scleral vessels

A sign of Kaiser−Fleischer ring with a high degree of reliability allows you to identify a long-term and genetically determined violation of copper metabolism, leading to cirrhosis of the liver in Wilson−Konovalov disease.

10) Testicular atrophy in alcoholic liver damage, defined as a decrease in the diameter of the testicles less than 3 cm (fig. 58).

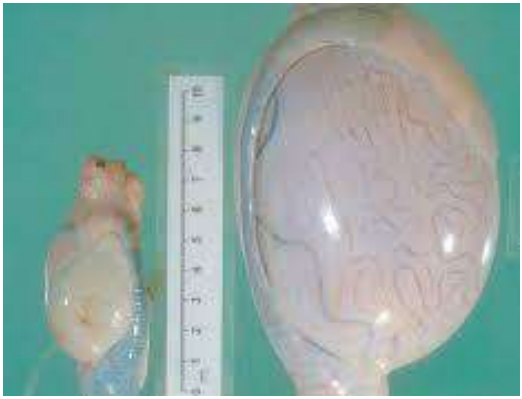


Figure 58. Normal testis and a testicle with atrophy

11) Weak hair growth of the armpits and pubis.

12) Ocular symptoms resembling hyperthyroidism are possible.

In the diagnosis of liver diseases, great importance is given to the definition of the underlying hepatic syndrome. It is the syndromes that are the «great hepatic signs» of diseases of the hepatopancreatobiliary system.

Major «hepatic signs» include:

1) jaundice;
2) hepatomegaly;
3) portal hypertension syndrome;
4) syndrome of hepatocellular insufficiency;

5) hepatorenal syndrome;

6) hepatic encephalopathy syndrome;

7) cholestasis syndrome.

There are a number of specific signs of liver diseases, which makes it possible to make a correct diagnosis to the patient with a certain accuracy (tab. 5).

Table 5

Signs of liver disease and probable diagnosis

Signs	Disease
Fingers in the form of «drumsticks»	Chronic hepatitis and cirrhosis of any etiology
«Fields» of spider veins on the skin of the collar zone, intense palmar erythema, Dupuytren's contracture, alcoholic's face (facies alcoholica), gynecomastia, giant mumps	Icoholic hepatitis and cirrhosis of the liver
Xanthelasma, xanthomas, skin hyperpigmentation	Primary biliary cirrhosis
Scratching, skin hyperpigmentation	Bile duct obstruction, primary sclerosing cholangitis, primary biliary cirrhosis
Expansion of the veins of the anterior abdominal wall, ascites, bulging of the umbilicus	Cirrhosis of the liver with portal hypertension
Intense jaundice, edematous-ascitic syndrome, hemorrhagic syndrome – bruises, purpura, «liver» breath, tremor of the hands, tremor of the tongue	Cirrhosis of the liver with hepatocellular insufficiency
Kaiser–Fleischer ring (when examining the cornea with a slit lamp)	Wilson–Konovalov disease

Examination of the abdomen in patients with liver disease

The abdomen is examined in a vertical and horizontal position, using both direct and side lighting. Normally, the left and right halves of the abdomen are symmetrical, the navel is slightly retracted. In normosthenics, the stomach is slightly protruding. In hypersthenics, the stomach is usually voluminous. Asthenics have a small belly, flattened or slightly retracted. A uniform increase in the size of the abdomen is observed with obesity, excessive accumulation of gases in the intestines (flatulence), the appearance of free fluid in the abdominal cavity (ascites), as well as in the third trimester of pregnancy.

In patients with moderately severe ascites, the abdomen in the supine position flattens out due to flattening in the umbilical region and protrusion in the lateral sections – «frog belly». In a standing position, the abdomen with ascites is enlarged in volume, sags in the lower half due to the movement of fluid. With severe ascites, the abdomen has a capsule shape, regardless of the position of the body, its skin becomes smooth, shiny, the navel is smoothed or protrudes. One of the causes of ascites may be portal hypertension. A symptom characteristic of ascites is noted – «proud posture», since the upper half of the body is thrown back, and the stomach protrudes forward.

When examining the area of the liver, one can notice its increase (hepatomegaly) only with a significant size of the liver and a small thickness of the anterior abdominal wall, which is also true for volumetric formations − nodes, tumors, echinococcal cysts, large abscesses. An asymmetric enlargement of the abdomen on the right due to bulging of the liver, as well as lagging movements of the abdominal wall in the right hypochondrium and epigastrium during respiratory movements may attract attention.

«Decollete symptom» − spider veins (telangiemias), similar to a spider or asterisks. The most typical localization is the neck, shoulder girdle, back. They are a marker of liver diseases (fig. 59).

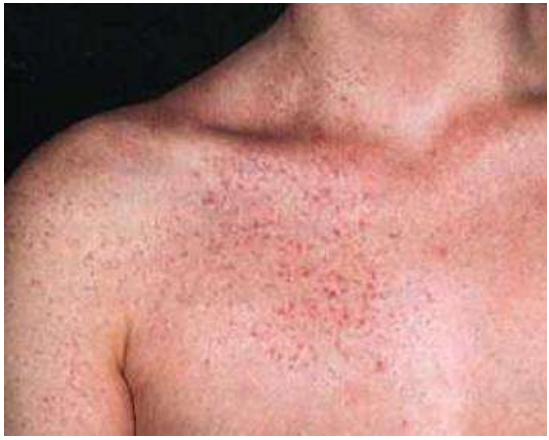


Figure 59. «Decollete symptom» − a scattering of spider veins in the decollete area

With tricuspid valve insufficiency, you can see the pulsation of an enlarged liver due to a wave of blood regurgitation in the right heart.

An enlarged gallbladder (empyema, dropsy) can also be the cause of a protrusion in the right hypochondrium. It is seen in emaciated patients with gallbladder cancer. The visually defined gallbladder is characterized as a pear-shaped body protruding above the abdominal wall, making respiratory excursions, unless the gallbladder is fused with surrounding organs and with the abdominal wall. An enlarged gallbladder can also be seen with tumors that compress the common bile duct (cancer of the head of the pancreas, duodenal cancer in the area of the Ampula Vateri. (Courvoisier−Terrier symptom), while bile does not enter the

duodenum and jaundice develops in patients. When observing a patient with ascites, it is important to measure the circumference of the abdomen daily and control body weight.

There may be dilated thrombosed saphenous veins on the anterior surface of the abdomen, which is characteristic of the Budd–Chiari syndrome (fig. 60).

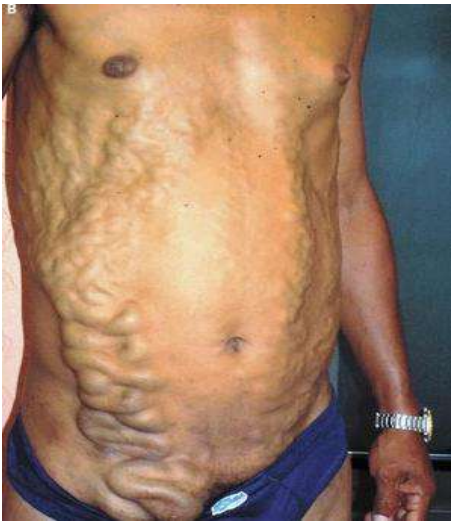


Figure 60. Changes in the saphenous veins – thrombosis, cyanosis in Budd–Chiari syndrome

Percussion of the liver

The purpose of the technique is to determine the upper and lower boundaries of the liver. Relative dullness of the liver – the borders of the liver, covered with tissue of the right lung. The borders of the liver that are not covered by the lung tissue are called the borders of the absolute dullness of the liver. The upper limit of the absolute dullness of the liver coincides with the lower edge of the right lung. The lower limit of absolute dullness corresponds to the lower border of the liver. To determine the

absolute dullness of the liver, the method of quiet percussion is used. The dimensions of the absolute dullness of the liver are somewhat smaller than the true dimensions of the organ.

For percussion determination of the upper border of the liver, percussion is carried out from top to bottom. The border is found by the contrast between a clear pulmonary sound and a dull hepatic percussion sound. The found border is marked with dots on the skin, along the upper edge of the finger − a plessimeter along each vertical line. Normally, the upper limit of the absolute dullness of the liver in front is located along the linea parasternalis dextra at the upper edge of the VI rib, along the linea medioclavicularis dextra on the VI rib and along the linea axillaris anterior dextra on the VII rib (fig. 61).

For percussion determination of the lower limit of absolute hepatic dullness, it is necessary to use quiet percussion. Otherwise, the air contained in the stomach and intestines enters the percussion zone and a tympanic shade is added, which interferes with the correct determination of the lower limit of the absolute dullness of the liver. There are two methods of quiet percussion of the lower edge of the liver − according to F.G. Yanovsky and according to V.P. Obraztsov. In both cases, percussion is performed with one finger.

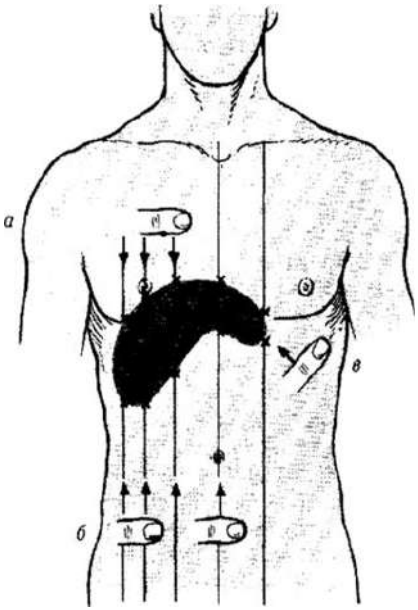


Figure 61. Scheme of percussion of the upper and lower borders of the liver with the direction of movement of the finger-plessimeter

Percussion of the lower edge of the liver according to the method of F.G. Yanovsky

Percussion should begin from the right flank of the abdomen at a level obviously below the lower edge of the liver, presumably from the level of the navel. If there is already a dull sound there, then it is necessary to go down even lower, to the place where the tympanic sound is determined.

Gradually, moving the plessimeter finger up, they reach a level where the tympanic percussion sound changes to dull. This percussion can be carried out along topographic lines − anterior axillary, mid-clavicular, right parasternal, median, left parasternal. By connecting the obtained points, you can get the exact border of the lower edge of the liver (fig. 62).

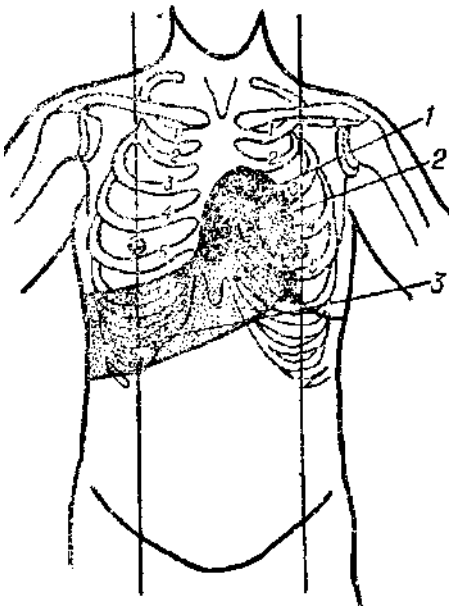


Figure 62. Determination of the border of absolute dullness of the lower edge of the liver: 3 – absolute hepatic dullness and the border of the lower edge; 2 – absolute dullness of the heart; 1 – relative dullness of the heart

Percussion of the borders of the liver according to the method of V.P. Obraztsov

Method V.P. Obraztsov received more fame than the method of Yanovsky F.G. The boundaries of the liver are determined along five lines − the right parasternal, mid-clavicular, anterior axillary, anterior median and along the left costal arch. The upper limit according to this method is determined by three lines − the right parasternal, midclavicular and anterior axillary. The lower limit is determined by all five lines (fig. 63).

Normally, in an adult with a normal body mass index, a normosthenic chest, in a horizontal position, with an average

depth of breathing, the lower limit of absolute hepatic dullness along the right anterior axillary line is located at the level of the X rib, along the mid-clavicular line − at the level of the lower edges of the right costal arch, along the right parasternal line − 2 cm below the edge of the right costal arch, along the midline − on the border between the upper and middle thirds of the line connecting the lower edge of the xiphoid process with the navel, along the left parasternal line − along the lower edge of the left costal arcs.

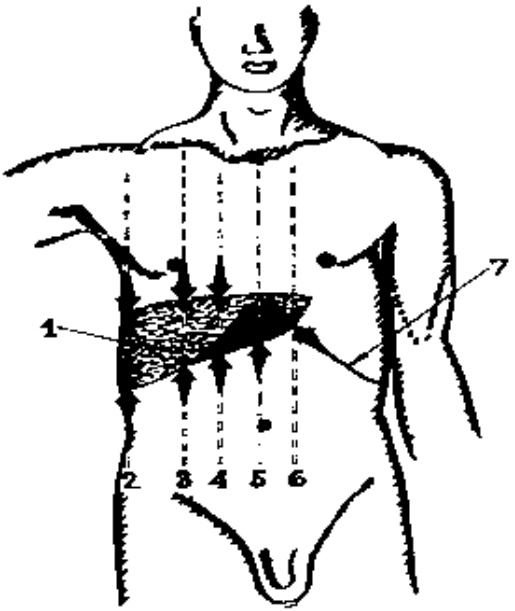


Figure 63. Scheme for determining the boundaries of absolute hepatic dullness according to the method of V.P. Obraztsova, where 1 − absolute dullness of the liver, 2 − anterior axillary line, 3 − right midclavicular line, 4 − right parasternal line, 5 − anterior midline, 6 − left parasternal line

In hypersthenics, the lower edge of the liver is located higher than in normosthenics, approaching the lower edge of the xiphoid process (1 cm higher than in normosthenics). In asthenics, the lower edge of the liver is located lower than in normosthenics, on average, on the border between the middle and lower thirds of the indicated line (fig. 64). In women, especially multiparous ones, the lower edge of the liver is located below the indicated norms by 1−2 cm. In the standing position, the lower edge of the liver shifts down by 1−1.5 cm also can fall below the edge of the right costal arch.

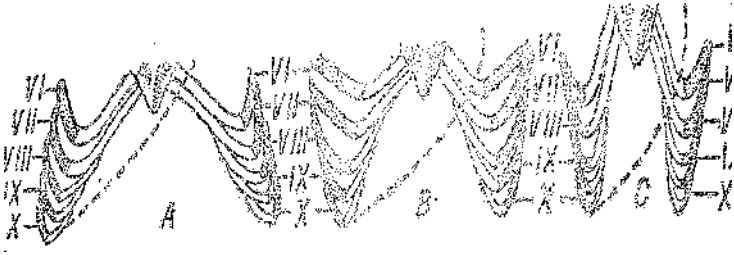


Figure 64. location of the lower edge of the absolute dullness of the liver, depending on the shape of the chest: a − hypersthenic; b − normosthenic; c − asthenic

The vertical line connecting the points of the lower and upper boundaries of the absolute hepatic dullness of the liver is called the height of hepatic dullness and is equal to 8−10 cm along the right parasternal line, 9−11 cm along the mid-clavicular line on the right, and 10.5−12 cm along the anterior axillary line. On the left, the height of hepatic dullness is difficult to determine, as hepatic dullness and cardiac dullness merge.

Diagnostic value also has a shift in the position of the upper border of the liver upwards. This is observed under the following circumstances:

1) wrinkling of the right lung, especially in the region of its lower lobe;

2) compaction of the anterior-lower part of the right lung (cancer, pneumonia), when its dullness merges with the dullness of the liver;

3) accumulation of inflammatory fluid in the right pleural cavity, when the dullness of the exudate merges with hepatic dullness;

4) high standing of the diaphragm with a change in the standing of the liver to the edge. At the same time, the height of the absolute dullness of the liver decreases, it should be remembered that the stagnant liver is only displaced downwards.

If suddenly the upper border of the congestive liver has shifted upward, it is necessary to exclude the presence of encysted cardiac pleurisy in the right pleural cavity. The liver occupies, as it were, a marginal position, turning around the frontal axis, so that its lower edge moves upward, and its upper edge moves backwards and downwards. If the rotation around the frontal axis is very significant, then the anterior-superior surface of the liver will be located horizontally, and hepatic dullness may completely disappear (fig. 65);

5) changes are also possible in the liver itself, increasing it not only downwards, but also upwards. These include liver echinococcosis, liver tumors, abscesses of the anterior-upper surface of the liver;

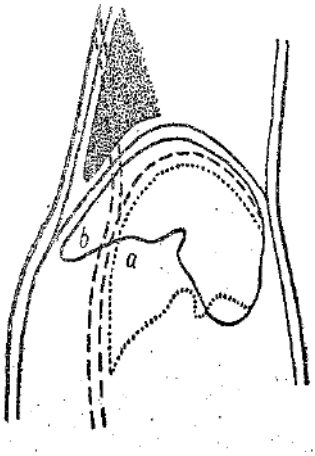


Figure 65. The position of the liver depending on the level of the dome of the diaphragm: A – normal, B – marginal standing of the liver with a high dome of the diaphragm

6) with subphrenic abscesses, when pus accumulates between the anterior-upper surface of the liver and the lower surface of the diaphragm covered with peritoneum, there is an apparent displacement of the upper edge of absolute hepatic dullness upwards.

The shift of the upper limit of the absolute dullness of the liver downward is observed when the diaphragm is low, for example, with emphysema, right-sided pneumothorax, enteroptosis. In the clinic, another method is also widely used to determine the boundaries of the liver – the definition of three ordinates M.G. Kurlov.

Method for determining the ordinates M.G. Kurlova – the size of the liver

This method is fast, convenient, and widely used in medical practice all over the world. Medical researcher M.G. Kurlov developed a method for determining the boundaries of the liver along three lines − the right mid-clavicular, anterior median and the line of the left costal arch, respectively, these measurements are called the first, second and third Kurlov's ordinates. Normally, the upper border of the liver is located at the level of the VI rib. Lower border of liver is definite only with method of quiet percussion (fig. 66).

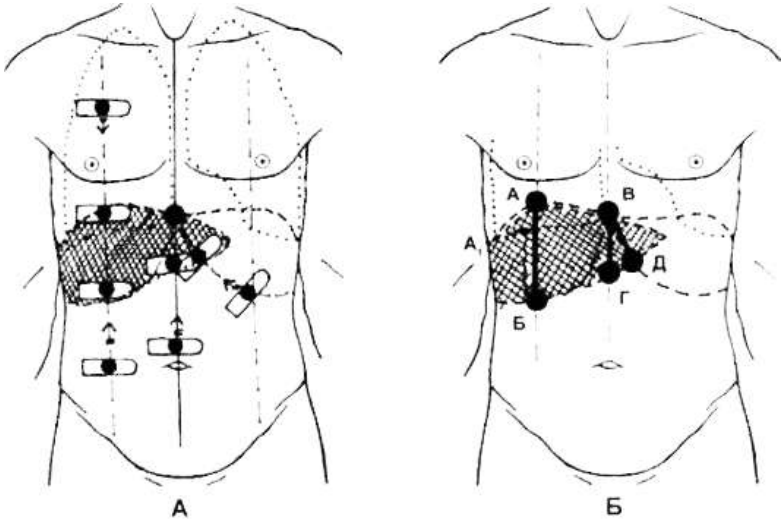


Figure 66. Method for determining Kurlov's ordinates

To determine the first Kurlov ordinate along the right midclavicular line, percussion is performed from the navel to the lower border of the liver and from a clear pulmonary sound down

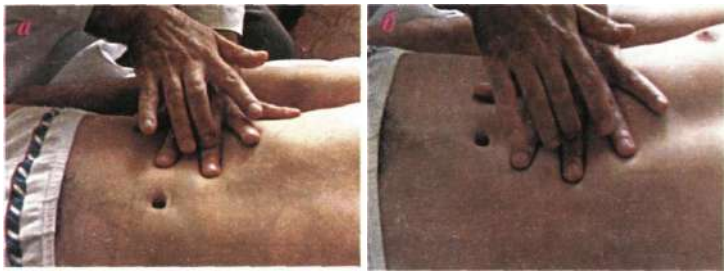
the intercostal space until hepatic dullness appears. By connecting two points, measure the first ordinate. Normally, it is 9 cm.

To determine the second Kurlov's ordinate along the midline of the abdomen, percuss up until hepatic dullness appears. It is difficult to determine the upper border of hepatic dullness along the midline due to the location of a dense sternum under the skin, damping percussion sounds. Therefore, it is customary to draw a perpendicular from the upper border of hepatic dullness of the first Kurlov ordinate to the midline and take this point as the upper border of the liver along the second ordinate. By connecting the upper and lower points, the value of the second Kurlov ordinate is obtained. Normally, it is 8 cm.

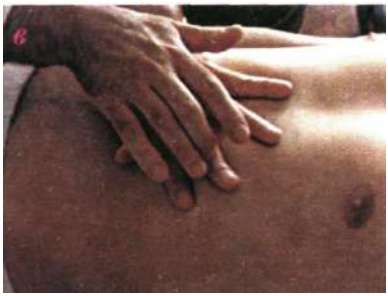
To determine the third Kurlov ordinate, percussion is performed directly under the left costal arch, parallel to it, starting from the anterior axillary line. The dull hepatic sound point is the lower limit of the third ordinate. The upper boundary of the third Kurlov ordinate will be the point of the upper boundary of the second ordinate. The distance between the upper and lower boundaries is measured, normally the value of the third Kurlov ordinate is 7 cm. **It is very easy to remember − 9 − 8 − 7!**

If the liver is enlarged in size, then the first Kurlov ordinate is denoted by a fraction, in which the numerator is the total size of the liver along the right midclavicular line, and the denominator is its part corresponding to the size extending beyond the costal arch down, for example − 14/5 cm.

The lower border of the liver is determined along the midclavicular line in the norm − along the edge of the right costal arch, along the anterior midline − at the border of the upper and middle third of the distance from the navel to the xiphoid process, and along the left costal arch − at the level of the left parasternal line (fig. 67).



a *b*



c

Figure 67. Determination of the Kurlov ordinates: *a* – determination of the lower border of the liver along the right mid-clavicular line; *b* – along the anterior midline; *c* – on the left costal arch

Palpation of the liver

Before describing the technique of palpation of the liver, it is necessary to dwell on the main points of palpation of the abdomen. Until recently, until ultrasound diagnostics became a publicly available research method, palpation of the abdomen was the main diagnostic method. But even now it has not lost its relevance. Depending on the goals set by the doctor, two types of palpation are distinguished − superficial and deep. Both types of palpation should be used in every patient, and superficial should precede deep. The doctor sits on a chair to the right of the patient.

The seat of the chair should be at the same level as the bed of the patient. The patient lies horizontally, his head is on a low pillow or without a pillow, which will allow more relaxation of the abdominal muscles. The patient's legs should not rest against the back of the bed for the same reason. The hands of the doctor should be warm, the nails should be short. The touch of a cold hand reflexively causes tension in the patient's abdomen.

Palpation (palpation) is the main method of physical examination of the liver and gallbladder. Palpation allows you to judge the state of the liver, based on the state of its lower edge. In addition, palpation allows you to study the properties of the anterior surface of the liver. Normally, only the anterior surface of the left lobe of the liver is accessible to palpation, located between the left and right parasternal lines and not covered by the ribs. With an increase or a strong omission, it is possible to palpate the right lobe of the liver.

The technique is carried out according to the rules of deep sliding palpation according to Obraztsov. The patient lies on his back, on a flat surface, his arms lie along the body, his legs are slightly bent at the knees, the muscles of the anterior abdominal wall are relaxed. This position of the patient somewhat limits the respiratory movements of the ribs and contributes to a greater participation of the diaphragm in breathing, providing a greater respiratory excursion of the liver. The doctor is positioned to the right of the patient.

The lower edge of the liver is preliminarily determined by percussion along the midclavicular line. You can also determine the border of the lower edge of the liver by sliding. To do this, the fingers of the right hand slowly slide along the anterior surface of the abdominal wall along the anterior axillary or mid-clavicular line from the edge of the right costal arch down. In the place where the anterior surface of the liver ends, and accordingly, the lower edge of the liver is located, there is a change in the body of

a dense consistency to a softer one. If both methods do not lead to the goal, then it is necessary to repeat the attempt, palpating the lower edge of the liver at various levels below the costal arch and the xiphoid process. The doctor puts his left hand with his palm on the anterior surface of the right half of the chest, on the costal arch and overlying ribs, exerts some pressure on the ribs. The doctor's palpable right hand lies flat on the right half of the patient's abdomen, the line formed by the ends of 2−5 fingers should be parallel to the supposed lower edge of the liver and be slightly below it. In this case, the doctor's fingertips are immersed deep into the abdomen in sync with the patient's breathing. When the patient exhales, the doctor's fingers sink into the abdominal cavity, while inhaling, the fingers slip out of the abdominal cavity, touching the descending lower edge of the liver from under which they slip (fig. 68).

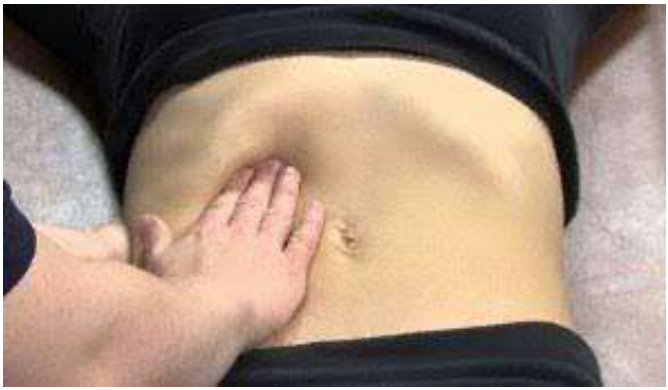


Figure 68. Palpation of the lower edge of the liver

Since the lower edge of the liver lies superficially and is normally thinned, the fingers should be immersed to a depth of no more than 1−2 cm. The patient is asked to breathe deeply and slowly. The lower edge of the liver, starting during inhalation

towards the palpating fingers, falls into the duplication bag of the peritoneal wall, formed due to the fact that the fingers, plunging into the abdominal wall, press it inward (fig. 69). The posterior surface of the thin edge of the liver is in front of the nail surfaces of the palpating fingers, separated from the fingers by the posterior leaf of the duplication. The liver begins to move downward, its elastic flexible edge first bends forward, and then bypasses the tops of the immersed fingers, slipping out of the duplication, straightening again, it is already behind the fleshy surface of the fingers, separated from them by the straightened abdominal wall. At the moment when the edge of the liver bypasses the tops of the fingers, a palpation sensation appears, giving an idea of the properties of the edge.

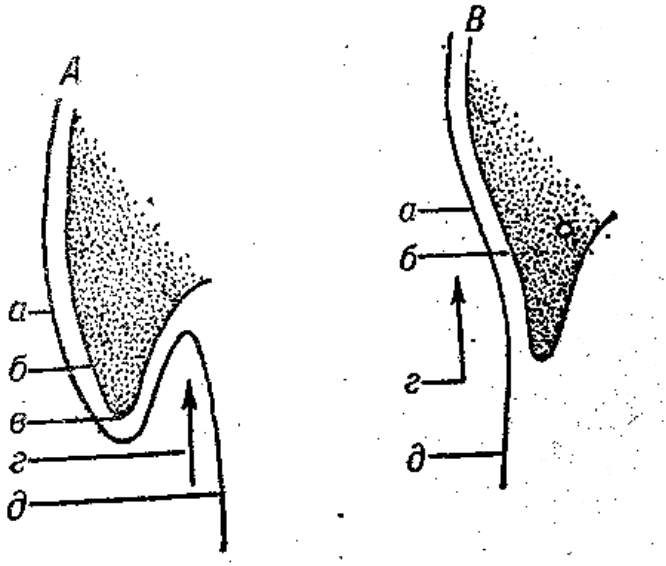


Figure 69. Palpation of the lower edge of the liver. A − the first moment, B − the second moment: а − skin, б − peritoneum, в - the lower edge of the liver, г − depression in the abdominal wall and the direction of the fingers during palpation, д - the surface of the abdomen

When lowered, the liver sometimes makes a turn around its sagittal (anterior-posterior) axis in such a way that its right lobe descends, and the left lobe rises, and the lower edge of the liver begins to occupy an almost vertical position (fig. 70). To palpate such an edge of the liver, the line of palpating fingers should be directed parallel to the hepatic edge.

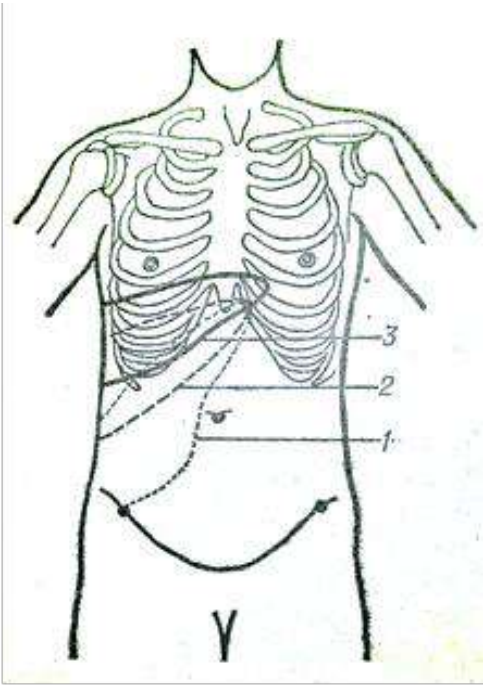


Figure 70. Scheme of changes in the position of the lower edge of the liver during its omission: 1 – strong omission, 2 – moderate omission, 3 – normal position

With severe ascites, ordinary percussion and palpation of the liver are difficult, therefore, the method of balloting palpation

is used, while revealing the symptom of a «floating floe». To do this, the right hand is placed in the mesogastric region to the right below the navel and with jerky movements of the fingers, the hands move up until a dense displaced organ is felt under the fingers. Using this technique, you can get an idea about the features of the edge of the liver and its surface.

With the help of palpation of the liver, it is important to correctly assess its lower edge. Characteristics of the lower edge of the liver: shape (sharp, rounded), density, smoothness or irregularities, pressure sensitivity, consistency.

Normally, the edge of the liver is soft on palpation, smooth, pointed, painless.

The downward displacement of the lower edge of the liver may be due to either a downward displacement of the entire non-enlarged liver, or an increase in the liver. To clarify the true cause of the displacement, it is necessary to determine the upper limit of absolute hepatic dullness with the help of percussion with the determination of the height of hepatic dullness along topographic lines, or by determining the Kurlov ordinates.

The downward displacement of the lower edge of the liver may not be due to its pathology, but to general splanchnoptosis, in that case there will be a downward displacement of the upper border of the liver.

Hepatomegaly is a formidable symptom. Most often, this symptom occurs in congestive heart failure, acute and chronic hepatitis, hypertrophic cirrhosis of the liver, in the initial stages of atrophic cirrhosis of the liver, tumor or metastatic liver damage, fatty lipodystrophy of the liver, including in tuberculosis patients. Hepatomegaly can accompany fatty liver disease, alcoholic liver disease, diseases associated with biliary tract obstruction. Hepatomegaly is also found in many poisonings, for example, in poisoning with chloroform, phosphorus. Hepatomegaly is possible with the development of severe anemia, diabetes mellitus, with

amyloid degeneration of the liver. Hepatomegaly has been described in syphilis, including the development of syphilitic gums in it, chronic purulent processes − osteomyelitis, severe periodontitis, and also in septic processes. With leukemia, the liver increases due to the formation of foci of extramedullary hematopoiesis in it.

The liver reaches the most gigantic sizes in cancer, amyloidosis and hypertrophic cirrhosis. The most common causes of liver enlargement are congestive liver and cirrhosis (fig. 71).

The edge of the congestive liver is usually more rounded, painful on palpation. Pressure on the enlarged liver in congestive heart failure causes swelling of the right jugular vein − hepatojugular reflux. This symptom is a sure symptom of stagnation of blood in the systemic circulation. The edge of the cirrhotic liver is usually dense, uneven, painless. Hepatocellular carcinoma results in significant hardening of the liver tissue, described as «stony-density liver». Palpable regional lymph nodes confirm the diagnosis of a malignant neoplasm of the liver. The edge of the liver in the gummous form of syphilis will also be bumpy, dense, uneven, having an unequal consistency in different places.

The edge of the congestive liver is usually more rounded, painful on palpation. Pressure on the enlarged liver in congestive heart failure causes swelling of the right jugular vein − hepatojugular reflux. This symptom is a sure symptom of stagnation of blood in the systemic circulation. The edge of the cirrhotic liver is usually dense, uneven, painless. Hepatocellular carcinoma results in significant hardening of the liver tissue, described as «stony-density liver». Palpable regional lymph nodes confirm the diagnosis of a malignant neoplasm of the liver. The edge of the liver in the gummous form of syphilis will also be bumpy, dense, uneven, having an unequal consistency in different places.

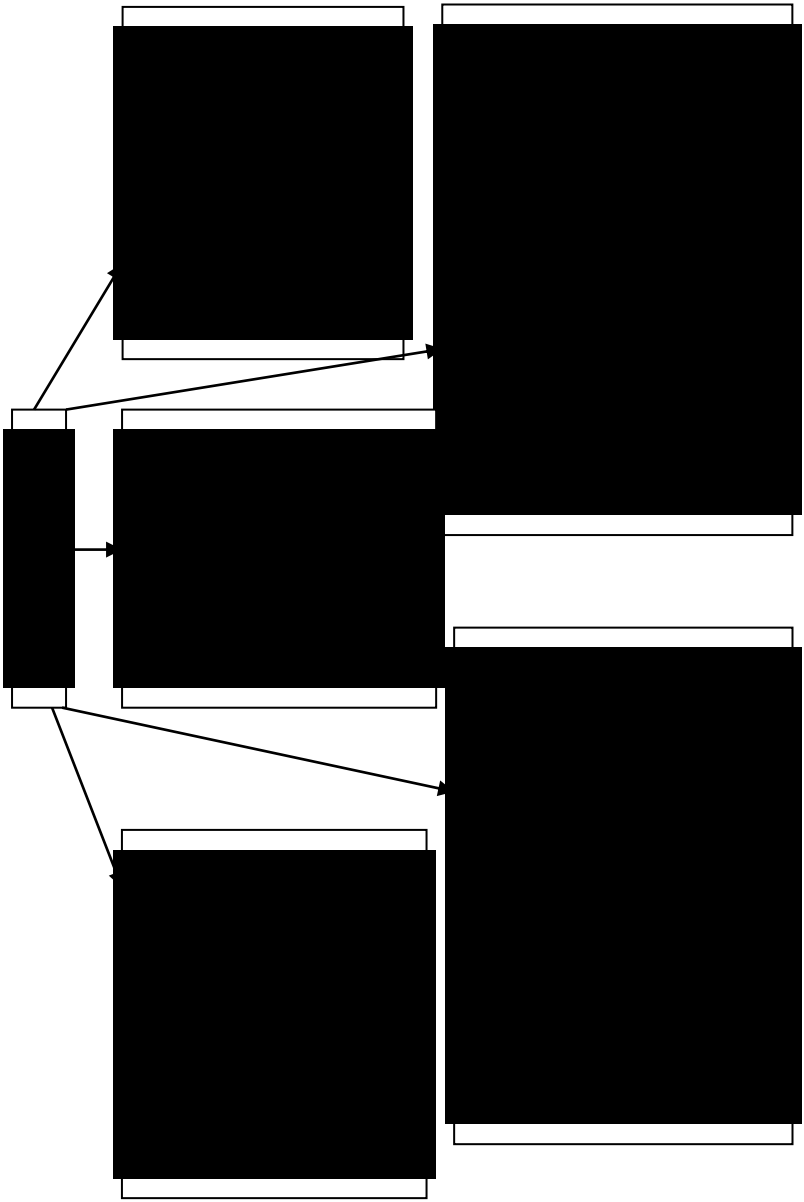


Figure 71. The most common causes of hepatomegaly

Of great importance is the dynamics of changes in the size of the liver. A rapid increase is usually seen in liver cancer. A rapid decrease in size − with cirrhosis and acute hepatitis of the fulminant course. With successful treatment of congestive heart failure, the size of the liver will also decrease rapidly.

Simultaneously with hepatomegaly, there may be an enlargement of the spleen (splenomegaly), in this case it is appropriate to use the term «hepatolienal syndrome».

A decrease in the size of the liver is noted in the late stages of atrophic cirrhosis, senile atrophy of the liver, and acute dystrophic processes in the liver tissue.

Palpation of the lower edge of the liver will be painful with congestive liver, perihepatitis, inflammatory bulging of the liver with angiocholitis, with liver abscess. With liver lipodystrophy and amyloid degeneration, the liver is insensitive to palpation.

Determination of ascites by physical methods

Ascites in modern realities should be considered as the presence of various origins and composition of fluids in the abdominal cavity, caused by certain diseases, injuries or therapeutic effects. The definition of ascites as an accumulation of transudate in the abdominal cavity (from the Greek askos − a bag for water, wine) only partially reflects the essence of this pathological process. Transudate in the abdominal cavity appears with cirrhosis of the liver and its appearance is associated with protein imbalance, increased pressure in the portal vein, sodium and water retention, peripheral vasodilation, increased levels of renin, aldosterone, vasopressin and norepinephrine in plasma, changes in membrane permeability of the peritoneum.

Determination of ascites by physical methods

Ascites in modern realities should be considered as the presence of various origin and composition of fluids in the

abdominal cavity, caused by certain diseases, injuries or therapeutic effects. The definition of ascites as an accumulation of transudate in the abdominal cavity (from the Greek ascos − a bag for water, wine) only partially reflects the essence of this pathological process. Transudate in the abdominal cavity appears with cirrhosis of the liver and its appearance is associated with protein imbalance, increased pressure in the portal vein, sodium and water retention, peripheral vasodilation, increased levels of renin, aldosterone, vasopressin and norepinephrine in plasma, changes in membrane permeability of the peritoneum.

In the presence of ascites, it is important to determine the amount of fluid, its infection and the degree of susceptibility to drug treatment. From these positions, ascites are small, moderate and significant (tense, massive). According to the infection of the contents − sterile, infected, bacterial peritonitis. According to the drug response − amenable to drug therapy or refractory.

Ascites in a patient with decompensated liver cirrhosis is a sign of impending death – 50 % of such patients die within a year and only 20 % live for more than 2 years.

Clinical manifestations of ascites are very bright. Many of the author's well-established symptoms of ascites are described.

«Frog belly» − in a patient with ascites in a horizontal position, the stomach is flattened, and its lateral sections swell.

The symptom of «floating ice» − in the presence of a large amount of fluid in the abdominal cavity, the liver can be palpated using jerky ballot palpation. Hands on both sides apply light jerky blows to the abdominal wall, upon impact, the liver first deviates deep into the abdominal cavity, and then hits the fingers, «swings», becomes palpable, «floats like an ice floe».

«Head of a jellyfish» − an expanded venous network on the anterior abdominal wall, a sign of portal hypertension, is often detected against the background of ascites.

«Wind before rain» − flatulence, against which ascites develops in a patient with cirrhosis of the liver.

Ascites develops when the collateral pathways described above are unable to drain all venous blood into the vena cava systems. Ascites usually develops slowly, gradually. But in the case of development of thrombosis of the portal vein or overlapping of its lumen by a tumor, ascites will form quickly, in two to three days.

According to one of the pathogenetic theories of the development of ascites, fluid seeps into the abdominal cavity from crowded capillaries through diapedesis. An important point for the formation of ascites is sodium retention in the blood due to hyperfunction of the adrenal cortex and increased production of aldosterone. This contributes to an increase in sodium reabsorption in the loop of Henle, the release of antidiuretic hormone into the blood, and the accumulation of large volumes of fluid in the abdominal cavity.

There are three methods for the physical detection of ascites − the fluctuation method; percussion method № 1 and percussion method № 2.

The fluctuation method is used to detect large volumes of fluid in the abdominal cavity. The palm of the left hand is firmly applied to the lateral surface of the abdomen, and light jerky blows are applied with the right hand from the opposite side. If there is free fluid in the abdominal cavity, then these blows are felt with the left hand in the form of a wave. You can use the help of an assistant to eliminate additional fluctuation waves that occur in obese people. In this case, the assistant places his hand with the edge of the palm along the midline of the patient's abdomen. The assistant's hand extinguishes the waves that come from the vibration of the adipose tissue of the anterior abdominal wall, but does not affect the fluctuation waves of the free fluid in the abdominal cavity (fig. 72, 73).

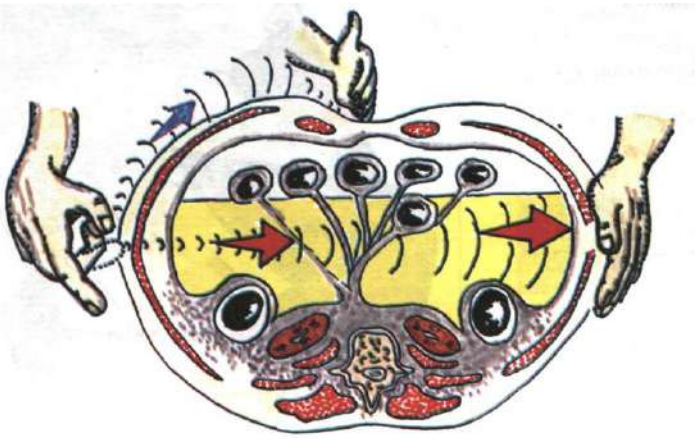


Figure 72. Scheme for determining ascites by the fluctuation method

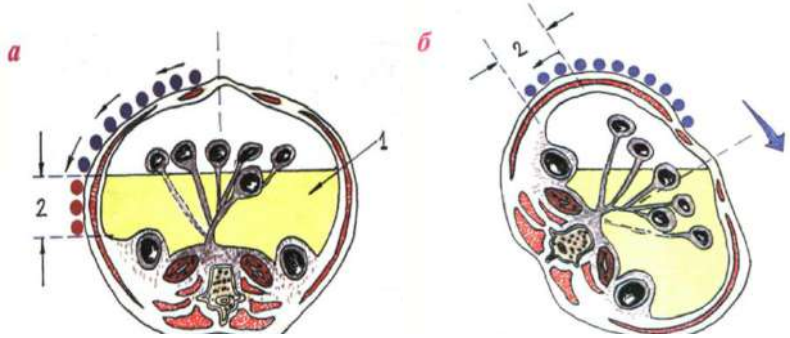


Figure 73. Scheme for determining ascites by percussion method № 1 in a horizontal position lying on the back and turning on its side:
***а*: 1 – free fluid in the abdominal cavity, 2 – zone of dull percussion sound,**
***б*: 2 – zone of tympanic sound in the same place with a change in body position**

Percussion Method № 1 − used to detect large volumes of fluid in the abdominal cavity. The position of the patient is horizontal, lying on his back. The finger-plessimeter is placed parallel to the midline of the abdomen and percussed from the navel towards the flanks of the abdomen, from tympanic sound to a dull percussion sound formed by free fluid accumulated along the flanks in the abdominal cavity. Then, without removing the finger from the border of the beginning of a dull percussion sound, the patient is asked to turn on the opposite side and continue percussion in the direction of the flank. If a tympanic sound appears in place of a dull percussion sound after turning to the opposite side, then we are really dealing with free fluid in the abdominal cavity. When turned on its side, the liquid flows to another place (fig. 73). If a dull sound persists when the patient is turned on its side, then the cause of its appearance is not a liquid, but, possibly, a tumor of the intestines, kidneys, ovaries, feces, and more.

Percussion Method № 2. A small amount of fluid in the abdominal cavity can be detected by percussion of the abdomen in the upright position of the patient. To do this, the patient stands still, the doctor, standing from the patient to the right, begins percussion along the midline of the abdomen from top to bottom, from the level of the navel to the pubis. If a dull sound is detected in the lower abdomen (above the pubis), then the patient is asked to lie horizontally on his back, having previously marked on the patient's skin the point above which a percussion blunt sound was received in a vertical position (fig. 74).

If there is indeed a small amount of fluid in the abdominal cavity, then when the patient takes a horizontal position, it will flow along the flanks of the abdomen and in the place where there is a mark, there will no longer be a dull percussion sound. The remaining dullness in the suprapubic region will indicate other

causes − an enlarged bladder, uterus, intestinal tumor and other causes.

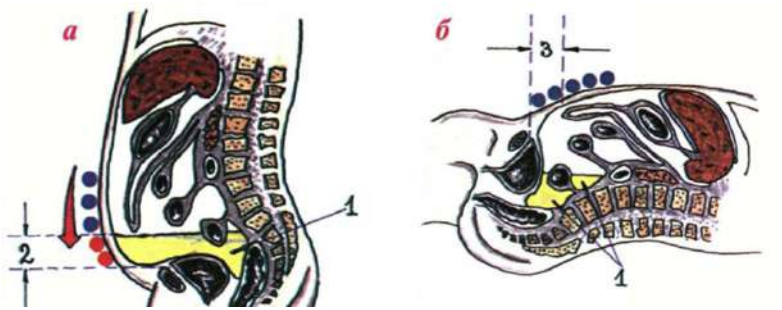


Figure 74. Scheme for determining ascites by percussion method № 2: 1 − fluid level, 2 − zone of dull percussion sound, 3 − zone of tympanic sound in the same place with a change in body position

Palpation of the gallbladder

Normally, the gallbladder is not palpable, but with a significant increase it can be palpated. An increase in the gallbladder can be caused by its dropsy, empyema, chronic cholecystitis, malignant neoplasm.

The technique of palpation of the gallbladder was developed by V.P. exemplary. Palpation is carried out in the region of the notch of the edge of the liver lateral to the edge of the rectus abdominis muscle. However, with a significant increase in the bladder and stretching of the cystic duct, it can shift to the sides. An enlarged gallbladder is defined as a sac-shaped, ovoid or pear-shaped formation of a dense or elastic consistency (depending on the nature of the contents) in the area between the lower edge of the liver and the outer edge of the right rectus abdominis muscle. In chronic cholecystitis, the bladder wall is dense, and in dropsy, empyema, the wall will be elastic, tense. In the case of pericholecystitis, the bladder wall is uneven when

palpated, bumpy due to inflammatory layers. With cancer of the gallbladder, its wall can also be uneven, bumpy. The gallbladder, full of large stones, also feels like a dense, bumpy formation. In the absence of inflammatory adhesions between the gallbladder and the surrounding organs or the anterior abdominal wall, its excursion during breathing will be noticeable during palpation, in addition, it can be shifted by a palpating hand to the right and left in a small amplitude.

With all the inflammatory processes in the gallbladder and around it, its palpation is very painful. Sometimes, during inflammatory processes in the gallbladder, it remains inaccessible to palpation, but it is possible to palpate a small part of the liver, located directly above the bladder in the form of a tongue − this is the so-called «Riedel lobule», which can be inflamed, swollen, dense, painful.

Comparative characteristics of the palpation features of the gallbladder are presented in table 6.

Auscultation of the liver and adjacent areas of the abdomen

Abdominal noises play an important diagnostic value in the pathology of the hepatobiliary system and not only. Noises by origin are systolic sounds of turbulent blood flow passing through pathologically altered, compressed blood vessels. The appearance of abdominal noise may be associated with hepatocellular carcinoma, renal artery stenosis, fibromuscular hyperplasia of the renal arteries, abdominal ischemia, aortic aneurysm, pancreatic cancer.

So, noises in the liver area can appear with alcoholic hepatitis, hepatocellular carcinoma, portosystemic shunt, hepatic artery aneurysm, hepatic arteriovenous fistula, as well as after surgical interventions on the liver (fig. 75).

Table 6

Palpation characteristics of the gallbladder in some pathological conditions

Diseases	Morphological changes	Features of the gallbladder
Cholecystitis	Inflammatory infiltration of the wall, the presence of stones (an optional sign), possibly pericholecystitis – the transition of inflammation to the visceral peritoneum covering the bladder	The gallbladder is sharply painful, somewhat compacted, enlarged, with the development of pericholecystitis it is poorly displaced
Gallbladder cancer	Germination of the tumor wall of the gallbladder, adhesions around the gallbladder, there are signs of inflammation	The gallbladder is enlarged, painful, dense, may be bumpy, poorly displaced
Hydrocele of the gallbladder	Obstruction of the bile duct, overflow of the bladder with bile and mucus – «white bile»	The bubble is significantly enlarged, moderately painful, slightly compacted or of an elastic consistency, the wall is tense, displaced
Cancer of the head of the pancreas	Compression by a tumor of the common bile duct, obstructive jaundice, distended, full of bile bladder	The bubble is significantly enlarged, painless, elastic, the wall is tense, there are clinical signs of obstructive jaundice

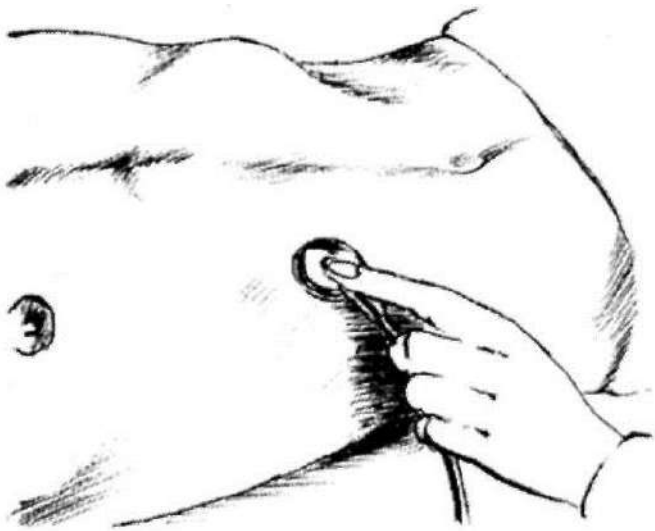


Figure 75. Auscultation of the liver

A peritoneal rub over the liver may be seen in liver metastases or primary hepatocellular carcinoma, liver infarction (eg, sickle cell anemia, periarteritis nodosa), and liver abscess.

Sometimes after a liver biopsy, you can hear a short friction noise caused by the formation of a hematoma around the puncture site, but usually after 4−6 hours it is no longer audible.

There are no noises over a healthy liver. However, in case of pathology over the liver area, it is possible to listen to the peritoneal friction rub with the development of perihepatitis or pericholecystitis, this noise is similar to a weak pleural friction rub. Noise occurs due to friction of the inflamed peritoneum covering the liver and gallbladder against the parietal sheet of the peritoneum. A rough rubbing sound of the peritoneum can sometimes be identified by hand.

In rare cases, with an echinococcal cyst of the liver, located on its anterior surface, a quiet trembling noise of hydatids

is heard in the cyst area. Even better, this noise is felt percussion, for which the fingers of the left hand are placed on the cyst and percussed for each of them in turn, finding the locus with the most pronounced trembling, resembling the trembling of springs. This symptom is rare and its absence cannot rule out the diagnosis of echinococcal cyst of the liver.

It is possible to auscultate the noise of moving blood with superficially located nodes of vascularized tumors, as well as over the area of significantly dilated venous trunks of the anterior abdominal wall, forming the «head of a jellyfish», due to recanalization of the umbilical vein.

Noises in the left hypochondrium may appear due to cancer of the body or tail of the pancreas. The murmur in this case may be due to envelopment of the splenic artery or splenic vein, or both. Noise is present in 25 % of pancreatic cancers.

An umbilical murmur has been described as the Cruvelier-Baumgarten syndrome and may be caused by increased umbilical venous blood flow due to portal hypertension. However, noise in the umbilical region can also be a manifestation of renal artery stenosis, fibromuscular hyperplasia of the renal artery, renal artery aneurysm.

The importance of examining the spleen in liver disease

Examination of the spleen is of particular importance in liver diseases, since changes in the size and function of this organ give an idea of the development of many irreversible complications of liver diseases. The most common causes of splenomegaly (enlargement of the spleen) are:

1) portal hypertension caused by cirrhosis of the liver;

2) viral, bacterial, fungal infections, as well as those caused by some protozoa;

3) leukemia, lymphoma and Hodgkin's disease;

4) connective tissue diseases (systemic lupus erythematosus, rheumatoid arthritis);

5) infiltrative diseases (amyloidosis, sarcoidosis);

6) hemolytic syndrome;

7) myelofibrosis.

Most pathological processes in the spleen are painless. Pain can appear only in case of damage to the peritoneum covering the spleen. This lesion can be in the form of inflammation of the peritoneum covering the spleen (perisplenitis), or in the form of stretching of this peritoneum.

Pain in the spleen with the reaction of the peritoneum surrounding it can occur with malaria, relapsing fever, spleen infarction (in this disease, the pain in the spleen is the most intense).

Perisplenitis occurs when the inflammatory process passes to the peritoneum covering the spleen, both from the spleen itself and from the surrounding organs and tissues. Pain during perisplenitis can be excruciating, aggravated by breathing, coughing, sneezing, changing body position.

More rare causes of pain in the spleen include rupture of the spleen during trauma, with an attack of malaria, relapsing fever. Pain can occur when the movable splenic pedicle is twisted. Pain in the spleen is felt in the left hypochondrium, sometimes radiating to the left half of the lower back. With a long-term enlargement of the spleen (splenomegaly), if it reaches a significant degree, patients complain of a feeling of heaviness and fullness in the left hypochondrium. This discomfort is due to a significant increase in the weight of the organ and stretching of its ligamentous apparatus. The appearance of acute pain in chronic splenomegaly is usually due to the development of perisplenitis or infarction of the spleen.

Examination of the spleen

Normally and with moderate enlargement, the spleen is not available for examination. Only with significant splenomegaly (more often with myeloid leukemia) does the spleen become visible through the anterior abdominal wall in the form of a protrusion in the left half of the abdomen, violating its symmetry. This protrusion passes to the left hypochondrium and causes smoothing of a small depression, which is normal under the costal arch. With significant splenomegaly, there may be a protrusion under the left costal arch and the left half of the chest, causing a slight asymmetry of the chest (fig. 76).

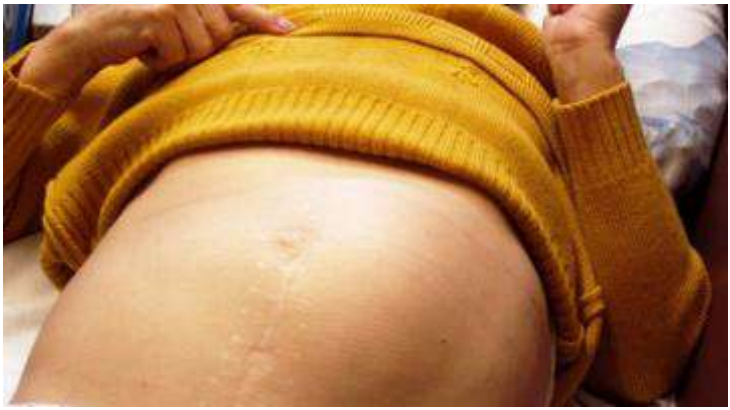


Figure 76. Visible splenomegaly.If there is no fusion of the peritoneum covering the spleen with the surrounding organs, then a respiratory excursion of the lower edge of the enlarged spleen will be visible

Percussion of the spleen

The spleen is located in the abdominal cavity in the region of the left hypochondrium, at the level of the IX−XI ribs. Percussion of the spleen is performed to determine the size of the

organ. The technique was developed by a domestic Russian therapist, professor of the Imperial Siberian Tomsk University M.G. Kurlov at the beginning of the 20th century. For this, a variant of quiet percussion is used (so that there is no influence of intestinal tympanitis on the overall percussion picture). The patient lies on the couch on the right side (fig. 77).

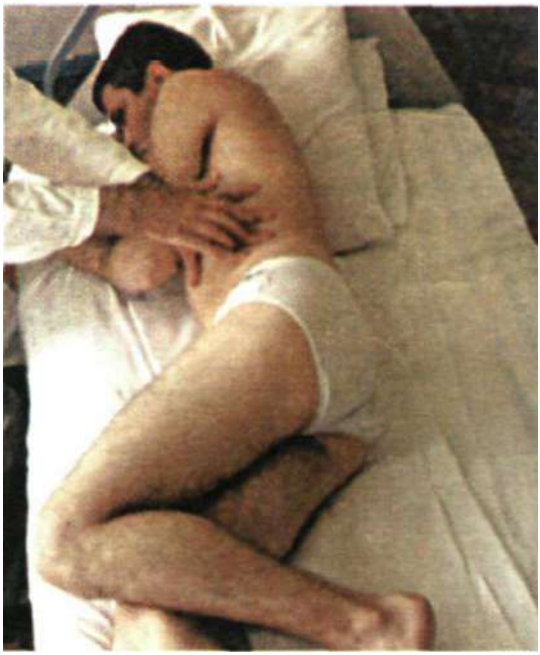


Figure 77. The position of the patient during percussion of the spleen

The finger-plessimeter is installed at the edge of the left costal arch perpendicular to the X rib. Percussion is carried out directly along the left costal arch in the direction from the sternum to the spine, marking the zones of dull percussion sound. And then in the opposite direction along the costal arch from the

posterior axillary line to the sternum − we mark the second point of the beginning of a dull percussion sound.

Two marked points connected to each other form the length of the spleen, normally equal to 6−8 cm. To determine the diameter of the spleen from the middle of the length of the spleen perpendicular to the X rib, a quiet percussion is performed up and down, marking 2 points of the end of splenic dullness. Normally, the diameter of the spleen is 4−6 cm (fig. 78).

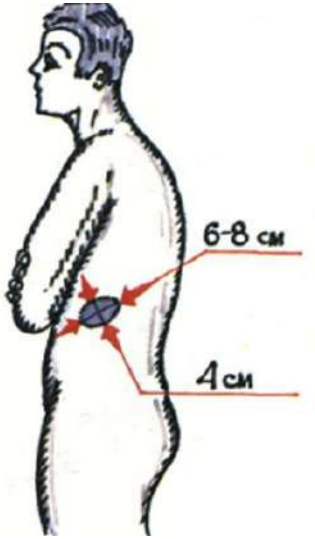


Figure 78. The patient lies on the right side. The dimensions and topography of the spleen are normal, the length is 6−8 cm, the diameter is 4 cm

Reasoning about the change in the size of the spleen according to the results of percussion may be erroneous due to the changes that may be imposed by the organs surrounding it. So, when the lower lobe of the left lung is compacted or with left-sided hydrothorax, the definition of the upper limit of splenic

dullness becomes incorrect, since the dullness of the spleen turns into dullness of the altered lung tissue or pleural fluid. At the same time, with emphysema, the upper limit of splenic dullness shifts downward and the area of the spleen in the upper part of the organ decreases. With severe hepatomegaly, especially its left lobe, the border of hepatic dullness can merge with the border of spleen dullness. With a strong filling of the intestinal loops adjacent to the spleen, liquid or solid feces, splenic dullness seems to be enlarged downwards. Severely gassed intestines can penetrate between the spleen and chest and displace the spleen deep under the diaphragm, significantly reducing the size of splenic dullness utill its disappearance. Therefore, conclusions about the change in the size of the spleen must be obtained from two diagnostic methods − percussion and palpation.

Palpation of the spleen

Palpation is the main method for examining the spleen. It is more convenient to carry out palpation according to the Sali method - the patient lies horizontally on his right side. His head is slightly tilted forward to the chest, the left arm is bent at the elbow joint and lies on the chest, the left leg is bent at the knee and hip joint, the right leg is extended. Normally, the spleen is never palpated!

Palpation involves both hands of the doctor. The doctor's left hand is on the left half of the patient's chest, presses on the chest. The half-bent fingers of the right bow are set on the left hypochondrium, so that the middle finger of the hand is the «continuation» of the X rib of the patient. The patient is asked to take a breath. With the right hand of the doctor, the skin is shifted down. As the patient exhales, the doctor's right hand plunges into the abdominal cavity. The patient is again asked to take a deep

breath. On inspiration, the spleen, if enlarged, descends under the pressure of the diaphragm and, with its lower pole, collides with the doctor's fingertips, and then slips under them (fig. 79).

If necessary, the spleen can also be palpated with the patient lying on his back with arms extended along the body, legs extended and a low headboard. The palpation technique is exactly the same as in the position on the right side.

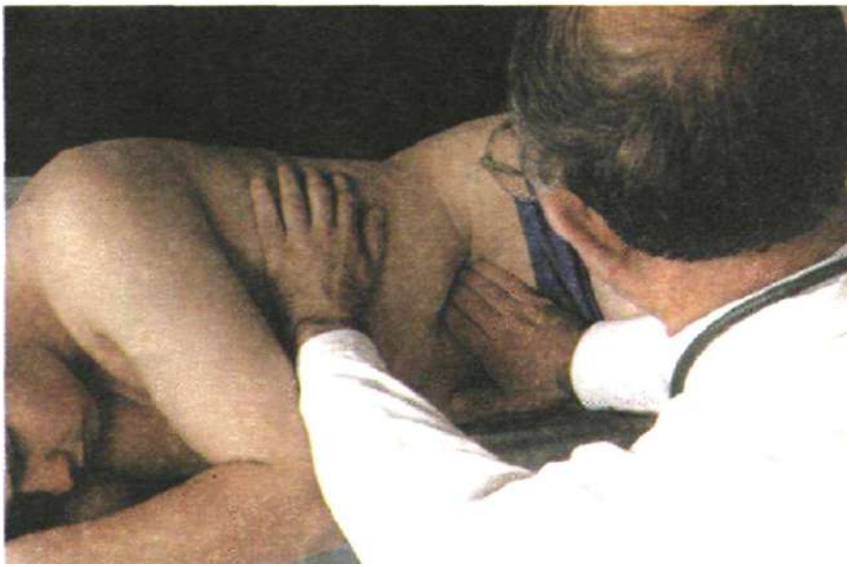


Figure 79. Palpation of the spleen on the right side in the Saly position

While palpating the spleen, it is necessary to pay attention to the following characteristics of its edge: localization; form; density; surface relief (smooth or bumpy); the presence of clippings on the front edge; mobility; soreness. It is important to follow a certain sequence when palpating the spleen (fig. 80).

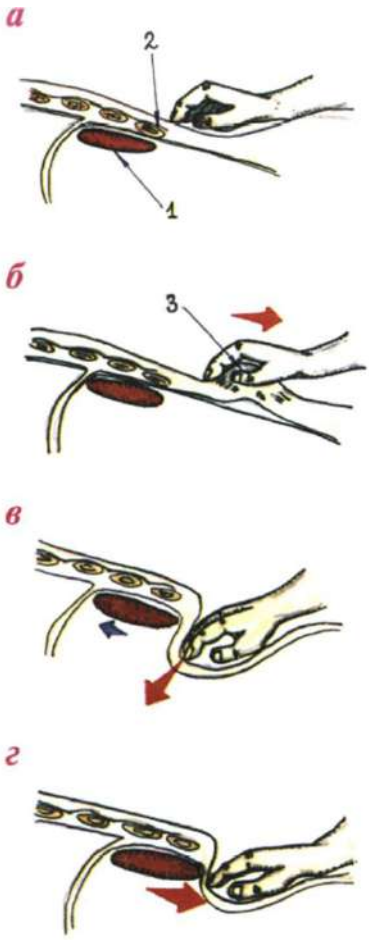


Figure 80. Step-by-step scheme of palpation of the spleen: *а* – installation of the doctor's hands; *б* – creation of a skin fold; *в* – immersion of the hand deep into the abdominal cavity; *г* – actual palpation of the lower pole of the spleen. 1 – spleen, 2 – costal arch, 3 – skin fold

In the left upper quadrant of the abdomen, organs other than the spleen are often detected – the kidney, the left lobe of the liver, the enlarged pancreas, and the colon. Sometimes it is difficult to distinguish these organs from the spleen on palpation, then it is advisable to use ultrasound or other imaging diagnostic methods to identify the palpable formation.

Conditionally allocate a slight (moderate, small) and a significant increase in the spleen. In the first case, the lower edge of the spleen comes out from under the left edge of the costal arch from 2 to 8 cm. In the case of a significant increase, the lower edge of the spleen comes out more than 8 cm from under the edge of the left costal arch.

Moderate enlargement of the spleen is observed:

1) in acute infectious diseases − typhoid, typhus, relapsing fever, paratyphoid, malaria, sepsis − due to hyperplasia of the spleen pulp;

2) with some chronic infectious diseases − syphilis, chronic malaria, hepatolienal syndrome with hepatitis and cirrhosis of the liver;

3) with atrophic cirrhosis of the liver of the Laennec type;

4) with spleen infarction, which is observed more often with infective endocarditis;

5) with Biermer's anemia, with polycythaemia rubra, with Werlhof's disease, tuberculosis of the spleen, lymphogranulomatosis, hemolytic anemia;

6) with systemic diseases of the connective tissue;

A significant increase in the spleen is observed with: leukemia; amyloidosis; Bunty's disease; Gaucher disease, leishmaniasis, in some cases chronic malaria, with hypertrophic cirrhosis of the liver; thrombosis of the splenic vein, thrombosis of the portal vein, echinococcal and other cysts of the spleen; spleen cancer; in case of large abscesses of the spleen.

A giant spleen that occupies the entire left half of the abdomen, partially the right half of the abdomen and sometimes extends into the small pelvis is described in chronic myeloid leukemia.

Soreness on palpation of the spleen is detected with its infarction, perisplenitis, thrombosis of the splenic vein. The soft

consistency of the enlarged spleen is more often observed in acute infectious diseases, more often in sepsis.

Higher the density of the spleen, longer the splenomegaly exists. In acute diseases, the density is less than in chronic diseases. A dense spleen occurs in chronic infectious diseases, cirrhosis of the liver, amyloidosis, and spleen cancer. With sepsis − on the contrary, the spleen is soft, doughy.

The tuberosity of the surface of the enlarged spleen is observed with perisplenitis due to the deposition of fibrin in it, with syphilitic gums, spleen cancer, sometimes with chronic leukemia, as a result of heart attacks or perisplenitis.

With echinococcosis, small abscesses and cysts of the spleen, limited protrusions and even fluctuations can be found on its anterior surface.

With a significant increase in the spleen size, when a distinct palpation of its lower edge is possible, it is possible to feel from one to four different depths of horizontal grooves on it.

The mobility of the spleen is quite significant, while its increase is moderate. With severe splenomegaly, the respiratory mobility of the organ decreases, but passive mobility (the ability to move the organ with a palpating hand) remains.

Soreness of the spleen on palpation is significant in the case of perisplenitis. In acute infectious diseases, an enlarged spleen is usually painless, with the exception of malaria and relapsing fever.

In the differential diagnosis of an enlarged spleen and an enlarged or wandering left kidney, it is important to understand that the upper pole of the left kidney can be bypassed with a palpating hand, which cannot be done with the lower edge of the spleen.

CHAPTER 9.
FEATURES OF SOME DISEASES OF THE LIVER AND BILIARY TRACT

Cholecystitis is an inflammation of the gallbladder, the process can be either acute or chronic. The cause is often bacterial inflammation, the infectious agent enters hematogenously. The cause of the development of the disease can be stones of the neck or cystic duct, a gall plug, an inflection of the cystic duct due to a congenital anomaly − a long cystic duct, and more. An inflammatory process develops in the wall of the gallbladder − catarrhal, purulent, gangrenous or phlegmonous.

Acute cholecystitis − the disease begins acutely, often it is preceded by gross violations of the diet. There are sharp, sometimes unbearable pains in the right hypochondrium, radiating to the right shoulder, collarbone. Dyspeptic joins the pain syndrome in the form of nausea, vomiting. The temperature rises to subfebrile and febrile numbers, dryness of the tongue appears. The abdomen is swollen on palpation, participates in the act of breathing to a limited extent, there is a muscular defense in the upper floor. Positive symptoms of acute cholecystitis will be observed − Ortner−Grekov's symptom, Frenikus-symptom (Mussi−Georgievsky), Murphy's symptom, Kera's symptom, Lepine's symptom, Boass's symptom. With intense inflammation, positive symptoms of peritoneal irritation may appear. In the general blood test, neutrophilic leukocytosis will appear, an increase in the erythrocyte sedimentation rate.

There are a number of specific symptoms that allow you to assess the condition of the gallbladder.

Symptom Terrier − Courvoisier − palpation of a significantly enlarged gallbladder with normal elastic walls, filled, tense, painless, in combination with jaundice. This may be due to

blockage of the common bile duct or blockage of the Futter's nipple by a tumor in the head of the pancreas or a stone.

Murphy's symptom − careful, gentle insertion of the hand into the gallbladder area and with a deep breath, the palpating hand causes sharp pain (a sign of cholecystitis)

Kehr's symptom − pain on palpation in the right hypochondrium on inspiration.

Symptom Mussi-Georgievsky (phrenicus-symptom) − pain when pressed between the legs of the right sternocleidomastoid muscle.

Ortner's symptom − soreness with slight tapping along the edge of the right costal arch. The symptom is checked by comparing the discomfort of the patient on both sides.

Riedel's symptom − in the case of a slight increase in the gallbladder, a lobule of the liver is palpated, located above the bladder, which can be mistaken for the gallbladder (a complex sign of acute cholecystitis).

Mussy's symptom is a pain when pressing on the phrenic nerve between the legs of the sternocleidomastoid muscle on the right (a sign of gallbladder damage, often acute cholecystitis).

Mackenzie's symptom is hyperesthesia of the abdominal skin, characteristic of peritonitis. In destructive forms of acute cholecystitis, skin hyperesthesia can be observed in the right hypochondrium with a maximum projection zone in the gallbladder area.

Yonash's symptom − pain on pressure at the site of attachment of the right trapezius muscle in the occipital region in the area of the projection of the occipital nerve (a sign of acute cholecytitis).

Symptom of Lyakhovitsky (or the phenomenon of the xiphoid process) − pain occurs with slight pressure on the right half of the xiphoid process or when it is taken upward. The origin of pain is associated with the transition of the inflammatory

process to the lymph nodes located behind the xiphoid process (a sign of acute cholecystitis).

These symptoms, with the exception of Courvoisier's symptom, are characteristic of cholecystitis (acute or chronic exacerbation).

Chronic cholecystitis. The development of chronic cholecystitis is facilitated by factors such as malnutrition, physical inactivity, various toxic effects, reflux in the bile and pancreatic ducts, the presence of a chronic focus of infection in the body with the spread of infectious agents by hematogenous, lymphogenous and ascending routes. These reasons contribute to the difficulty of the outflow of bile (duct dyskinesia, stones, elongation of the cystic duct, increased pressure in the duodenum, compression of the choledochus by enlarged lymph nodes or adhesions. Bile can change its biochemical composition due to chronic liver diseases − the lithogenic and antimicrobial properties of bile are violated. Chronic stagnation of bile in the gallbladder contributes to the development of chronic cholecystitis Violation of the integrity of the endothelium of the ducts (stones, Giardia, etc.) contributes to the intensification of inflammation.

Complaints of patients with chronic cholecystitis will be less specific than with acute cholecystitis. There will be dull, not very intense pain in the right hypochondrium, often radiating to the right shoulder blade, collarbone, right shoulder. Pain often occurs after eating, especially after eating fried, fatty foods, eggs, alcohol. Sometimes the pain is provoked by a jolting ride, physical exertion. Very rarely, pain can be identical to that of acute cholecystitis. Patients are accompanied by chronic dyspepsia − belching, heartburn, nausea, unpleasant taste in the mouth, stool instability, flatulence. Characterized by moderate asthenic syndrome and subfebrile condition.

On physical examination, there is tenderness on palpation at the point of the gallbladder (the point of intersection of the rectus abdominis muscle with the right costal arch). All symptoms of acute cholecystitis may be positive. However, in addition to this, other symptoms may appear − Kharitonov's symptom, Konash's symptom, Pekarsky's symptom, Shofar−Gubergrits' symptom (fig. 81).

Chronic cholecystitis can be complicated by sclerosis of the walls of the bladder and ducts with the formation of shrinkage of the gallbladder; the development of a periprocess, including acute peritonitis; stone formation; chronic cholangiohepatitis, pancreatitis.

Pekarskiy's symptom − pain when pressing on the xiphoid process (due to irritation of the solar plexus) − a symptom of chronic cholecystitis.

Symptom Ortner−Grekov − pain when tapping the ulnar side of the palm along the costal arch − a symptom of acute cholecystitis.

Lepine symptom − soreness with slight tapping with the fingertips in the right hypochondrium − a symptom of acute cholecystitis.

Boass symptom − soreness with pressure in the interscapular region at the level of IX-XI thoracic vertebrae, 3 cm laterally to the right − a symptom of acute cholecystitis.

Kharitonov's symptom − pain on palpation lateral to the right of the IV thoracic vertebra − a symptom of chronic cholecystitis.

Konash symptom -increased sensitivity in the occipital region at the site of attachment of the trapezius muscle − a symptom of chronic cholecystitis.

The phenomenon of the «xiphoid process» is pain that occurs with slight pressure on the xiphoid process of the sternum.

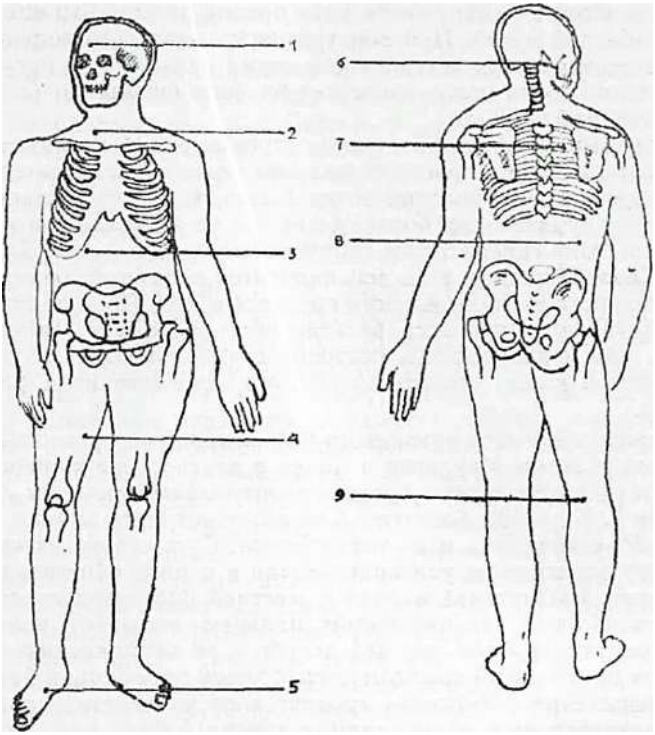


Figure 81. Pain points detected by palpation in patients with chronic cholecystitis: 1 – Bergman's point; 2 – Mussi point; 3 – Mackenzie point; 4 – Lapinsky point; 5 – plantar point; 6 – Ionash point; 7 – Kharitonov point; 8 – Boas point; 9 – point of the popliteal fossa

Maybe a symptom of acute cholecystitis or exacerbation of chronic cholecystitis.

«Principle of five F» – a set of signs inherent in patients with cholelithiasis – a woman, a blonde, over 40 years old, overweight, who had a pregnancy.

Frenikus symptom (Mussi–Georgievsky) – pain when pressing on the point of the phrenic nerve – between the legs of

the sternocleidomastoid muscle on the right − a symptom of acute cholecystitis.

Murphy symptom − a sudden interruption of inspiration with increased pain when touching the gallbladder − a symptom of acute cholecystitis.

Kera symptom − increased pain on palpation of the gallbladder on inspiration − a symptom of acute cholecystitis.

In chronic diseases of the biliary system, general neurotic disorders are also observed − irritability, tearfulness, insomnia, paresthesia, numbness of the hands, palpitations.

Chronic diseases of the biliary system can be complicated by food allergies, which have corresponding symptoms.

«Pseudo-ulcerative» syndrome occurs in chronic cholecystitis and cholelithiasis, often accompanied by ulcer-like symptoms. At the same time, there is a seasonality of exacerbations, hungry pains. The atypical course of chronic diseases of the biliary system is usually due to severe duodenal dyskinesia, and can also be caused by a periprocess extending from the choledochus to the duodenum.

Cholecystocardial syndrome (cardialgic or cholecystocoronary) − can occur with cholelithiasis. The possibility of reflex pains in the heart in cholelithiasis was first noted in his works in 1883. S.P. Botkin. In these cases, an attack of biliary colic does not occur with characteristic irradiation along the right phrenic nerve, but in the form of severe pain in the heart and behind the sternum. In this case, the duration of pain in the heart will not be coronary (up to 30 minutes), but can be very long − from 8−10 hours to several days. Cardiac symptoms such as tachycardia, increased pulse and venous pressure, impaired coronary circulation, and heart rhythm are observed. Occasionally, against the background of biliary colic,

hypertension in the pulmonary artery system can be detected. On the ECG at the time of biliary colic, the following changes can be detected: a shift in the ST interval below the isoline, a decrease in the voltage of the teeth and negative E waves mainly in the chest leads, high P waves in II, III, avf leads. Patients with cholecystocardiac syndrome, especially those with concomitant arterial hypertension, are more at risk of developing myocardial infarction.

Laryngeal syndrome is a very rare manifestation in patients with various diseases of the biliary tract. Acute pain in the larynx and phenomena of laryngospasm occur in the evening or at night after a heavy meal with a significant amount of fatty or fried foods. The attack can last for a long time, up to 6 hours or more. It is believed that this syndrome is based on overexcitation of the central nervous system, a violation of the transmission of impulses along the vagus nerve, and an increase in its tone.

Saint's syndrome is a rather rare triad of symptoms: cholelithiasis, diaphragmatic hernia and diverticulosis of the large intestine. According to the literature data, patients with diaphragmatic hernia suffer from cholelithiasis twice as often as those without diaphragmatic hernia. It is assumed that with a diaphragmatic hernia, the vagus nerve is involved in the pathological process, as a result of which the emptying of the gallbladder slows down, which leads to a violation of the circulation of bile acids, an increase in the lithogenicity of bile, and stone formation.

Werbraik's syndrome − described in 1940 to refer to a symptom complex in patients with adhesions between the right flexure of the colon and the gallbladder. It is characterized by a feeling of tension, dull pain in the epigastric region, nausea, and sometimes moderate muscle tension in the right hypochondrium. These changes occur with the tension of the gallbladder caused by the filling of the colon.

Mirizzi syndrome is an inflammatory stenosis of the common hepatic duct, most often occurs when the cystic duct is blocked (calculus, inflammation, tumor) with the transition of the inflammatory process to the common hepatic duct. Characterized by pain in the right hypochondrium, nausea, hyperbilirubinemia.

Cholelithiasis

Gallstone disease (cholelithiasis) is a polyetiological disease characterized by the formation of stones in the bile ducts − the gallbladder (cholecystolithiasis) and ducts (choledocholithiasis).

The first mention of gallstone disease dates back to ancient times, when gallstones were used as ritual ornaments, in religious ceremonies. The disease is mentioned in the writings of Galen, Vesalius. In 1824, cholesterol was isolated from bile for the first time.

Gallstones are formed as a result of the precipitation of insoluble components of bile − cholesterol, bile pigments, calcium salts, and certain types of proteins. Sometimes there are also chemically pure stones. Traditionally, stones are divided into cholesterol, pigment, calcium carbonate and mixed options. About 10% of the population suffers from cholelithiasis worldwide. People of any age, including children, are ill, however, more cases are recorded in the elderly and senile age, women get sick more often than men, since the lithogenicity of bile in women is higher. During pregnancy, the lithogenicity of bile increases significantly, the emptying of the gallbladder worsens, and favorable conditions for stone formation are created. The effect of estrogen therapy on the development of cholelithiasis has been noted. There is evidence of obesity, diabetes mellitus as predisposing factors for the development of cholelithiasis.

Pigmented gallstones often appear in patients with cirrhosis of the liver, with hemolytic anemia, and in diseases and conditions with an increased release of bilirubin into bile.

Clinically, during gallstone disease, 4 forms are distinguished − latent, dyspeptic, painful torpid form, painful paroxysmal form (biliary colic), and each of these forms can go into other options.

Cholangitis

Cholangitis is a catarrhal or purulent inflammation of both extra- and intrahepatic bile ducts. Cholangitis can occur as an acute process (catarrhal, purulent, purulent-septic), and as a chronic disease (recurrent, protracted septic, latent).

The clinical picture of acute cholangitis is close to the picture of acute cholecystitis, however, it is aggravated by damage to the intrahepatic bile ducts (fig. 82).

Characterized by an acute onset with febrile fever, chills, and heavy sweat. Pain in the right hypochondrium is intense, radiating to the right shoulder, right arm, right shoulder blade, interscapular region. The clinic is accompanied by nausea, vomiting, severe intoxication. On physical examination, the liver is often enlarged and tender to palpation. Jaundice is characteristic in varying degrees of severity. In the general blood test, high neutrophilic leukocytosis, accelerated ESR, increased activity of ALT and AST, alkaline phosphatase will be observed.

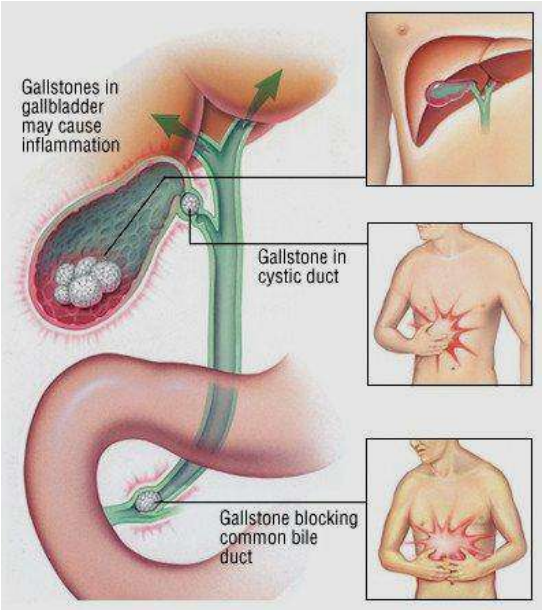


Figure 82. A stone in the common bile duct causes inflammation of its walls (cholangitis) with a characteristic intense pain syndrome

The clinical picture of chronic cholangitis does not differ significantly from that of chronic cholecystitis. However, the symptoms of intoxication, fever, chills will be more pronounced. Hepatomegaly appears with violations of the basic functions of the liver, including pigment metabolism (jaundice).

Complications of acute and chronic cholangitis − liver abscess, peritonitis, subdiaphragmatic abscess, liver fibrosis, sclerosis and deformation of the bile ducts, pleural empyema, lung abscesses.

Gallbladder cholesterolosis

Gallbladder cholesterolosis is the accumulation of lipids in the gallbladder mucosa, consisting of free and esterified cholesterol. As the disease progresses, lipids are deposited in the submucosal and muscular layers (figure). The disease was first described by R. Virkhov in 1857, when he saw a gallbladder, the mucosa of which contained many small yellowish inclusions, which made the bladder itself look like a strawberry («strawberry» gallbladder).

Cholesterolosis of the gallbladder is often combined with cholesterol calculi, so sometimes the disease is described as a prestage of gallstone disease. Cholesterolosis can be complicated by inflammation, stone formation, malignant degeneration of the bladder mucosa (fig. 83).

The classification of gallbladder cholesterolosis is based on morphological changes. So the following forms of the disease are distinguished:

1) «strawberry» or speckled gallbladder;

2) cholesterol polyps;

3) erased forms;

4) deep intramural cholesterol granulomas;

5) deposits of cholesterol in dropsy of the gallbladder.

In Russia, a classification based on the macroscopic lesion of the gallbladder is used. There are the following forms of cholesterolosis of the gallbladder:

1) focal-mesh form;

2) diffuse mesh form;

3) mixed mesh-polyposis form;

4) polyposis form.

5) extravesicular form of cholesterosis with lipid deposition in the mucous membrane of the cystic, common bile, pancreatic ducts. Each of the forms can be stone or stoneless.

Some scientists tend to suggest that cholesterolosis is a metabolic disorder of fat metabolism in the human body.

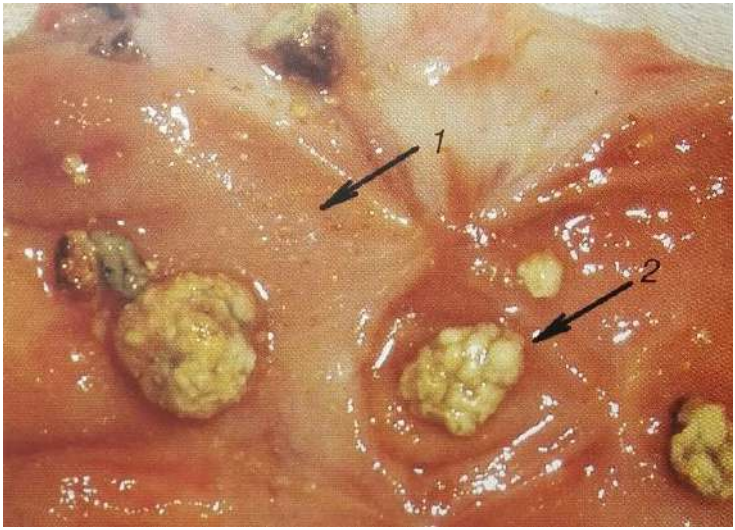


Figure 83. Cholesterolosis of the gallbladder (1) with the formation of stones (2)

Chronic hepatitis

Chronic hepatitis is a polyetiological inflammatory liver disease caused by hepatotropic factors with multiple mechanisms of transmission, with a very different course and outcome, proceeds cyclically, and liver damage occupies a central place in the clinical picture. In some patients, recovery is possible, in others − a sluggish, non-progressive course for years, in others − a rapidly progressive course with an outcome in cirrhosis.

Morphological changes in chronic hepatitis are characterized by histiolimphoplasmic cell infiltration of the portal tracts, periportal or portoseptal fibrosis in combination with

degeneration of liver cells while maintaining the lobular structure of the liver.

Comprehensive data on the epidemiology of chronic hepatitis are not yet available, but men are more likely to get sick. Only autoimmune hepatitis occurs predominantly in women. More than 70% of chronic hepatitis are clinically asymptomatic and are often detected incidentally. This is partly because in the event of a fatal outcome in the final of chronic hepatitis, as a rule, cirrhosis of the liver develops and records are kept for this final disease.

Currently, 5 nosological forms of viral hepatitis have been identified: A, B, C, D and E. In addition, there is a group of undifferentiated viruses G, F, TTV, etc. It is known that there are 350 million carriers of HBsAg in the world and at least 115 million patients chronic viral hepatitis B (HBV). Patients with chronic viral hepatitis C (HCV) in the world, there are at least 60 million, very often chronic hepatitis C is presented together with HIV infections.

Viral hepatitis B and C are more common in developed countries. In some patients with chronic hepatitis B (HBV), viral hepatitis D can be detected at the same time. Such combined liver lesions (HBV + HDV) are more common in the Mediterranean and South America and in Russia- in Tuva and Yakutia. In HBV and especially HCV hepatitis, HGV and TTV are often found. TTV is often detected in alcoholic hepatopathy. The role of HGV, TTV and SEN in the development of progressive forms of chronic viral hepatitis is small. Detection of these three viruses generally does not require antiviral therapy, and they are not included in tests for blood donors. In recent years, alcoholic-viral chronic hepatitis has become increasingly important.

Hepatitis A and E have a fecal-oral transmission mechanism.

Hepatitis B, C, D, G, F and TTV viruses are transmitted parenterally (from patients with hemophilia, drug addicts, homosexuals) during blood transfusion and blood products, operations, and other parenteral interventions; in violation of the integrity of the skin and mucous membranes; in case of casual sexual intercourse during the last 6 months or close contact with carriers of HBsAg, as well as patients with various CVH; vertical and perinatal transmission of infection from mother to child is possible.

Chronic alcoholic hepatitis is registered in 10−15 % of persons suffering from alcoholism. The total number of people affected by this form of hepatitis is comparable to the number of patients suffering from viral hepatitis.

There are significantly fewer patients with chronic drug-induced hepatitis than those with viral and alcoholic hepatitis.

Autoimmune hepatitis (AIH) is a rare disease. It is detected in 170 persons per 1 million population, women are ill 4 times more often than men.

The pathogenesis of chronic hepatitis is determined by the etiological features, as well as the mechanism of self-progression of cirrhosis common to all forms of this disease. In the pathogenesis of viral liver damage, the persistence of a viral infection and the immunoinflammatory process caused by it, the cytopathic (hepatotoxic) effect of viruses B and C, and the development of autoimmune reactions are important.

In the development of autoimmune hepatitis, the main role is played by autoimmune reactions that cause a pronounced immuno-inflammatory process with necrosis of the liver tissue.

In the pathogenesis of chronic drug-induced and chronic toxic hepatitis, damage to hepatocytes by direct, side effects of a drug or toxic substance, as well as their metabolites, manifested by metabolic disorders or an immunoallergic response resembling autoimmune hepatitis, is of primary importance.

In the development of viral hepatitis, an important role is played by the phase of the infectious process, and the disease proceeds most actively and progresses during the replication (multiplication) of the virus. Prolonged replication of the virus is more often observed in various immunity defects and can be maintained by chronic alcohol intoxication (with daily doses of ethanol more than 30 g in men and 20 g in women).

In the classification of the morphological features of chronic hepatitis, the components of the histological activity index according to the Knodell system are used, which include three histological criteria for hepatitis activity and the severity of fibrosis indicators in points (tab. 7).

Table 7

Determination of the Histological Knodell Activity Index

Components	Range of digital evaluation
Periportal necrosis with or without bridging necrosis	10 points
Interlobular degeneration and focal necrosis	0–4 points
Portal inflammation	0–4 points
Fibrosis	0–4 points

Histological activity indices (the first three components in points) are summed up and make it possible to roughly assess the severity of chronic hepatitis, and the fourth – the severity of fibrosis.

1–3 – chronic hepatitis with minimal activity of the pathological process; 4–8 – mild chronic hepatitis; 9–12 – moderate chronic hepatitis; 13–18 – severe chronic hepatitis.

The formulation of the diagnosis should be as follows:

1. Chronic viral hepatitis B, in the acute stage (HBsAg+, HBV+ DNA), inactive.

2. Chronic viral hepatitis C (anti HCV +, HCV RNA +), pronounced activity (persistent cytolytic syndrome).

3. Chronic drug-induced hepatitis (inactive form) as a result of intoxication with dopegyt.

4. Autoimmune hepatitis, highly active form.

In 1994, a new classification of chronic hepatitis was proposed at a convention of gastroenterologists in Los Angeles. Etiological factors were brought to the fore, which determine not only the clinic of the disease, but also its prognosis. In clinical and etiological terms, among chronic hepatitis are:

- viral B, C, D;
- undifferentiated viral G, TTV and SEN;
- autoimmune;
- medicinal;
- genetically determined.

In addition, it is proposed to refer to viral hepatitis cases of the disease, according to clinical signs, close to the forms that occur with the presence of virus markers, but without their detection. Among the autoimmune forms of chronic hepatitis, three types of the disease are distinguished, which are characterized by the presence of various types of antibodies. More than 70 % of chronic hepatitis are clinically asymptomatic or with minimal symptoms, especially during remission. Therefore, most often the disease is first detected relatively by accident during medical examinations, examinations for other diseases, as well as during examinations of people who have had acute HCV in the past, drug addicts or those suffering from alcoholism. The doctor's attention is usually attracted by the presence of liver signs, hepatosplenomegaly, the detection of HbsAg, anti-HCV, etc., as well as elevated levels of bilirubin and serum aminotransferases.

Depending on the characteristics of the pathological process, cholestatic, cytolytic or autoimmune syndromes may predominate.

Indirectly, the degree of activity and severity of the inflammatory process can be judged by the level of increase in aminotransferases (AlAT, AsAT):

− the minimum degree of activity − with a slight increase in the level of aminotransferases;

− weakly expressed activity − with their increase by no more than 2 times;

− moderately pronounced activity - with an increase of 2−5 times;

− a pronounced degree of activity − with a more than five-fold increase in the level of aminotransferase activity.

The degree of activity of liver pathology is also judged by the severity of dysproteinemia, an increase in the thymol test, and low numbers of the prothrombin index.

With the immunocomplex variant of the course of chronic hepatitis of the pathological process, the level of gamma globulin in the blood serum increases by 2 or more times;

electrophoresis appears polyclonal, rarely monoclonal gammopathy.

Albumin levels remain within the normal range until late stages of liver failure.

By the nature of the course, the pathological process in chronic hepatitis and cirrhosis of the liver can proceed latently, without obvious clinical and laboratory manifestations. The diagnosis in these cases is made only with a morphological study of liver biopsy specimens taken during examination for another disease.

Clinical manifestations of chronic viral hepatitis B

Typically, an oligosymptomatic course in the integrative phase and with «virus carriage» is more common; the diagnosis is confirmed by elevated ALT, HBV markers, and liver biopsy. Replication is evidenced by: general symptoms of intoxication of

varying severity; dyspeptic disorders; sometimes dull pain in the epigastrium and right hypochondrium; subfebrile condition; dark urine; not very much enlarged, but denser, in contrast to acute hepatitis, the liver.

Sometimes hemorrhagic syndrome and enlargement of the spleen are detected; possible jaundice with pruritus, sometimes with xanthelasma; vascular «asterisks», palmar erythema; vasculitis, periarteritis nodosa, myalgia, glomerulonephritis, fibrosing alveolitis, myocarditis, cryoglobulinemia, damage to the lymph nodes, sex glands, adrenal glands, thyroid gland, pancreas; violation of the immunological function of lymphocytes and monocytes infected with HBV. Urobilinogen, sometimes bile pigments, are found in the urine. Anemia and thrombocytopenia are possible. Moderately elevated ALT activity, reduced prothrombin index, dysproteinemia.

Clinical manifestations of chronic viral hepatitis C

Viral hepatitis C is caused by the RNA-containing hepatitis C virus (HCV). It is characterized by: incubation period from 2 to 26 weeks; proceeds more easily than viral hepatitis B and A, but more often acquires a chronic course; anicteric forms are more common, which are tolerated without inpatient treatment; often turns into chronic hepatitis and cirrhosis of the liver; dyspeptic syndrome, weakness, malaise are noted; may be complicated by aplastic anemia. Often the only manifestation of hepatitis C is a «wave-like» increase in the activity of ALT in the blood serum; fulminant course of hepatitis C is possible in carriers of HBs Ag. Chronic hepatitis C slowly progresses with further development of liver cirrhosis. Histological examination often reveals minimal or mild activity of the pathological process. A specific triad includes the presence of lymphoid follicles in the periportal connective tissue, alteration of the bile ducts, and fatty degeneration of hepatocytes. In the area of stepped necrosis, T-

helpers predominate, and in hepatitis B, T-suppressors. The heterogeneity of hepatocytes in chronic hepatitis C is more common and more pronounced than in chronic hepatitis B. Among the periportal necrosis, stepped ones predominate.

The reactivation (activity) of HCV is evidenced by:

- strengthening of astenization, decrease in working capacity;

- enlargement and hardening of the liver;

repeated subfebrile condition, weight loss, but there is no jaundice;

the development of numerous extrahepatic manifestations;

- increase in ALT;

- HCV RNA and/or anti-HCV IgM markers, the presence of the entire spectrum of antibodies to structural and non-structural proteins in the immunoblotting reaction.

Viral hepatitis D

It is caused by the RNA-containing hepatitis D virus (HDV). It is typical for him:

- incubation period 20−40 days;

- occurs only in persons infected with hepatitis B;

- proceeds in the form of acute co-infection or superinfection.

Mixed hepatitis (co-infection) is the combined development of two or more viral hepatitis with simultaneous infection with several pathogens. Clinical, laboratory and epidemiological data more often indicate co-infection with hepatitis C and B. In some patients, markers of HDV infection may be detected. A combination of acute hepatitis A and hepatitis B, C, D is possible.

Autoimmune hepatitis

Autoimmune hepatitis is a chronic inflammatory disease of the liver of unknown etiology, characterized by predominantly periportal or more extensive inflammation with lymphomacrophage and plasma cell infiltration, usually accompanied by hypergammaglobulinemia, the presence of a wide spectrum of tissue autoantibodies, and usually amenable to immunosuppressive therapy.

Autoimmune hepatitis is characterized by a violation of the immunological tolerance of the liver tissue; frequent combination with other autoimmune diseases (thyroiditis, Sjögren's syndrome, ulcerative colitis, etc.); connection with HLA antigens involved in immunoregulatory processes (B8 DR3 and DR4 in patients with AIH-1, and AIH-2 it is associated with DR3 and DQ2); often there is a defect in the autoimmune regulator type 1 (AIRE-1); frequent onset of AIH after infection with viruses A, B, C, herpes, Epstein-Barr disease. In the debut of the disease, a decrease in working capacity is characteristic; arthralgia; yellowness of the skin and sclera; weakness, anorexia; sometimes fever; extrahepatic manifestations are possible, occurring under the guise of systemic lupus erythematosus, rheumatoid arthritis, systemic vasculitis, etc. The stage of advanced clinical manifestations of autoimmune hepatitis is characterized by the following symptoms: increased asthenic syndrome, and jaundice; skin itching is not typical; myalgia; abdominal manifestations; various skin rashes: «spider veins»; bright pink striae on the abdomen and thighs; hemorrhagic and acne skin rashes; hepato- and splenomegaly; systemic manifestations (vasculitis, polymyositis, lymphadenopathy, pneumonia, pleurisy, myocarditis, pericarditis, glomerulonephritis, Hashimoto's thyroiditis, ulcerative colitis,

diabetes mellitus, hemolytic anemia, idiopathic thrombocytopenia, hypereosinophilic syndrome, etc.).

The following laboratory changes for autoimmune hepatitis are characteristic: increased ESR, moderate leukopenia, thrombocytopenia, hemolytic anemia; positive direct Coombs test; violation of iron content; conjugated hyperbilirubinemia, a sharp increase in transaminase activity, AST / AlAT <1, a mild increase in alkaline phosphatase activity, hypergammaglobulinemia; transient liver failure (hypoalbuminemia, decreased prothrombin index, increased prothrombin time); predominant increase in IgG; positive immunoserological reactions to some bacteria and viruses (*E. coli*, *salmonella, bacteroids, measles, rubella, cytomegalovirus*, etc.).

The first type of autoimmune hepatitis is the most common; in the blood serum, antinuclear (anti-ANA) and / or anti-smooth muscle (anti-SMA) autoantibodies are determined, including those to actin (anti-F-actin), often in combination with p-type antineutrophil cytoplasmic antibodies (p-ANCA); more often women are ill, at the age of 10−20 years, or in the postmenopausal period; cirrhosis of the liver is often formed within 3 years; most patients have a good response to corticosteroid therapy with stable remission after discontinuation of drugs.

The second type of autoimmune hepatitis is rare (less than 4%), mainly in children aged 2−14 years. It is characterized by a higher biochemical and histological activity, and cirrhosis of the liver for 3 years is formed 2 times more often than with AIH-1. This hepatitis is more difficult to respond to immunosuppressive therapy, drug withdrawal leads to relapse.

Chronic drug-induced hepatitis can be caused by a side effect of a drug due to its direct toxic effect or the toxic effects of metabolites of this drug, and can also be caused by an idiosyncratic reaction to the drug or its metabolites, manifested by

metabolic disorders or an immunoallergic response; similar to autoimmune hepatitis. Chronic drug-induced hepatitis is characterized by direct damaging effect on the liver or idiosyncratic stimulation of hyperimmune reactions, regardless of the dose of the drug; hypersensitivity, manifested by skin rash, fever, eosinophilia; can develop latently, without an episode of acute hepatitis with prolonged use of the drug; discontinuation of the drug usually prevents further progression of the lesion.

In liver punctures of patients with chronic drug-induced hepatitis, morphological changes are similar to those in acute viral hepatitis: more often centrolubular and less often diffuse necrosis, more pronounced in the center of the lobules; cholestasis, infiltration of portal zones with monocytes, lymphocytes, eosinophils, the presence of granulomas and fatty changes, connective tissue septa, fibrosis.

Depending on the clinic and the degree of increase in the levels of biochemical blood tests (AlT, AST, APH, GGTP, etc.), three types of drug-induced liver damage are distinguished: hepatocellular type; cholestatic type; mixed type.

Alcoholic liver disease

Alcoholic liver disease is a variety of violations of the structure and functional ability of the liver caused by prolonged systematic use of alcoholic beverages. Alcoholic liver disease is characterized by: dependence on the dose and duration of alcohol consumption; the reversibility of changes in the initial, and often in the advanced stages, provided that the patient completely stops drinking alcohol and the ineffectiveness of any method of treatment against the background of its use. The disease usually develops 10−15 years after the onset of alcohol abuse in men with daily consumption of 60 g of alcohol, in women − 20 g;

When using toxic doses of alcohol, 5 phases of alcoholic liver damage develop sequentially or simultaneously:

1st – initial adaptive alcoholic hypertrophy of the liver;

2nd – alcoholic fatty degeneration of the liver with and without fibrosis;

3rd – alcoholic fibrosis of the liver;

4th – chronic alcoholic hepatitis;

5th – chronic alcoholic cirrhosis of the liver (fig. 84);

Episodes of acute alcoholic hepatitis may occur at any stage after heavy drinking; biochemical parameters of blood change little, but GGTP, Ig A are often elevated; impaired protein metabolism in the liver; histologically, in acute poisoning, centrolobular widespread necrosis of the liver with an inflammatory reaction occurs.

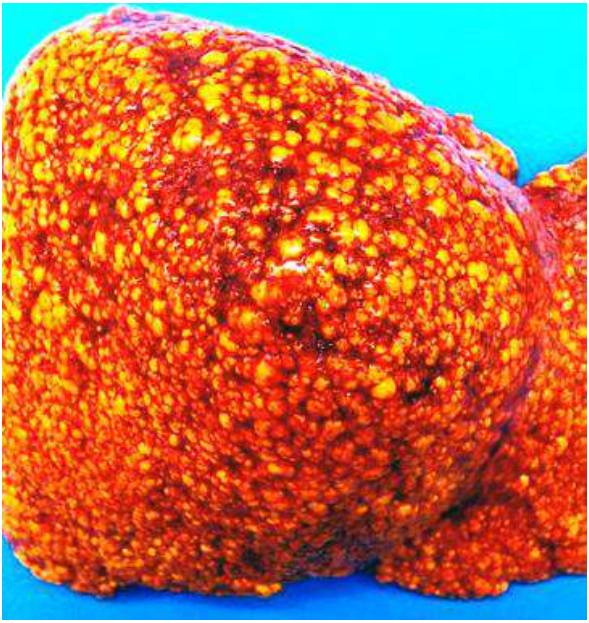


Figure 84. Chronic alcoholic cirrhosis of the liver. autopsy material

Morphological picture of alcoholic liver disease: signs of nonspecific reactive hepatitis and moderate fatty degeneration; enlargement and rounding of hepatic cells with a dull glassy transformation of their cytoplasm, resembling the cytoplasm of cells producing HBs Ag, but not giving orcein-specific staining; compression of the hepatic sinusoids due to hypertrophy of hepatocytes; the formation of inflammatory, predominantly neutrophilic infiltrates; central hyaline necrosis (alcoholic hyaline, Mallory bodies), perivascular fibrosis.

Portal hypertension syndrome and liver cirrhosis

Portal hypertension is an increase in pressure in the portal vein system (vena porta), and secondarily − the veins of the stomach, pancreas, spleen, mesenteric veins due to stagnation of blood in it. Stagnation of blood in the portal vein system is more often caused by mechanical factors that impede the flow of blood from the portal vein through the liver into the inferior vena cava. The cause is liver disease. The most common cause is an overgrowth of connective tissue in the liver, followed by scarring of the liver (liver cirrhosis). Wrinkled fibers of the connective tissue narrow, compress the intrahepatic ramifications of the portal vein, which impedes the flow of blood from the portal vein system to the system of the hepatic veins and the inferior vena cava.

The most common cause of portal hypertension syndrome is atrophic cirrhosis of the liver. In second place − malignant tumors, enlarged lymph nodes, scars located in the gates of the liver, a little less often − thrombosis of the portal vein with inflammation of its wall.

The development of portal hypertension is accompanied by a number of compensatory pathological mechanisms:

1) development of the collateral venous network;

2) the formation of ascites;

3) enlargement of the spleen.

The formation of a collateral venous network occurs due to the direction of blood flow from the portal vein system through additional pathways connecting the portal vein system with the superior and inferior vena cava, bypassing the liver. Normally, these collaterals are thin, inconspicuous veins, since the amount of blood flowing through them is small. With stagnation of blood in the portal vein system, a very large amount of blood rushes along these collaterals, the veins dilate significantly. The collateral veins located on the abdominal wall take the form of thick writhing strands and become accessible to inspection, palpation, and auscultation**.**

In case of portal hypertension the blood goes to the venous net of esophagus and from there through the system of little anastomoses its goes to the lower venacava (fig. 85). There are inter systemic anastomoses between the veins of the portal system and the veins of the vena cava system. These anastomoses play an extremely important role in the distribution of blood in areas of the body and are of particular importance in pathology in case of violations of blood flow in the main venous highways or their tributaries, providing collateral circulation.

Portocaval anastomoses are a system of fistulas between the tributaries of the portal vein and tributaries of the superior and inferior vena cava, and cavacaval anastomoses are a system of fistulas between the tributaries of the superior and inferior vena cava. In the case of the development of this collateral path, the dilated veins of the esophagus acquire a florid, knotty appearance, the so-called «esophageal varicose veins» are formed. These nodes are detected on fluoroscopy of the esophagus, fibroesophagogastroscopy. The probability of both spontaneous rupture of the esophageal venous nodes and their traumatization with further rupture during the intake of solid food, strong drinks,

chest blows, and intense coughing becomes very high. There may be cases of rupture of nodes during diagnostic procedures.

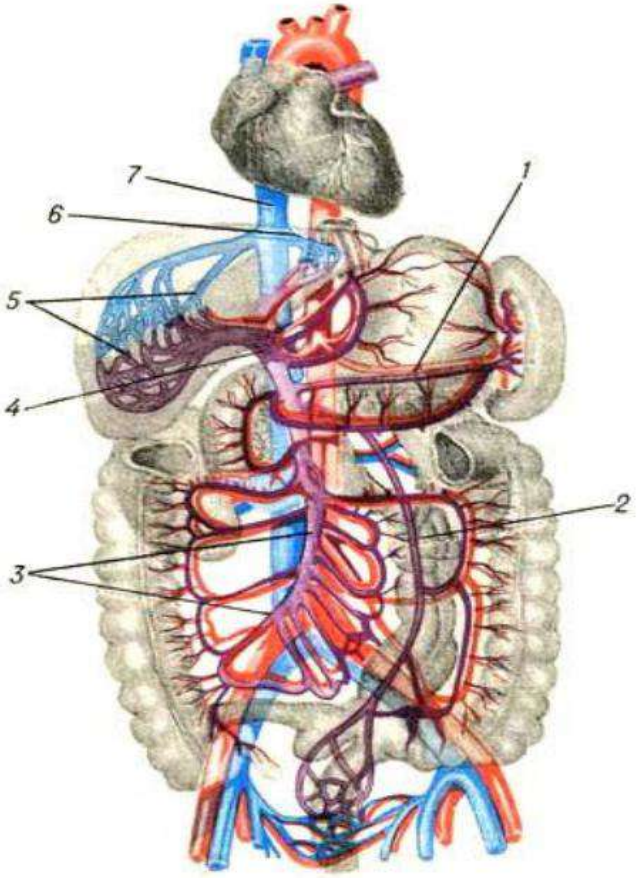


Figure 85. Scheme of portal circulation: 1 – splenic vein; 2 – inferior mesenteric vein; 3 – superior mesenteric vein; 4 – portal vein; 5 – branching of blood vessels in the liver; 6 – hepatic vein; 7 – inferior vena cava

Rupture of esophageal varices leads to intense esophageal bleeding, often leading to the death of the patient. Clinically,

bleeding will be manifested by profuse bloody vomiting, and blood entering the intestines will be chalky (liquid black, tarry stools).

From the inferior mesenteric veins with portal hypertension, blood rushes into the hemorrhoidal veins, and from there, bypassing the liver, into the system of the inferior vena cava. With the development of this collateral pathway, by analogy with the esophagus, varicose expansion of the hemorrhoidal veins is formed, their nodular deformation, which threatens the development of hemorrhoidal bleeding.

From the veins of the round ligament of the liver, leading to the navel, blood rushes through the remaining non-obliterated umbilical vein and paraumbilical veins, and from there through the veins of the abdominal wall into the systems of the superior and inferior vena cava. A corresponding venous pattern is formed on the anterior abdominal wall, resembling the head of the Gorgon Medusa. This symptom is called the head of a jellyfish.

Main portocaval anastomoses:

1. Portocaval anastomoses. In the wall of the abdominal part of the esophagus there are venous plexuses, from which blood flows through: the left gastric vein (*v. gastrica sinistra*) into the portal vein; esophageal veins (*vv. esophageae*) into the unpaired *(v. azygos*) and semi-unpaired veins (*v. hemizygos*) of the system of the superior vena cava.

2. Portocaval anastomoses of the rectum. In the wall of the rectum there are rectal venous plexuses (*plexus venosus rectalis*), the venous outflow paths of which are: veins. The middle rectal veins (*vv. rectales mediae*) drain blood from the middle section of the rectum through the internal iliac vein (*v. iliaca interna*) into the common iliac vein (*v. iliaca communis*) of the inferior vena cava system. The lower rectal veins (*vv. rectales inferior*) create an outflow of blood from the lower rectum (*anus*) through the

internal pudendal vein (*v. pudenda interna*) and the internal iliac vein (*v. iliaca interna*) into the common iliac vein (*v. iliaca communis*) system of the inferior vena cava (fig. 86).

3. Portocaval anastomoses of the anterior abdominal wall. In the anterior abdominal wall in the navel, tributaries are anastomosed:

- paraumbilical veins (*vv. paraumbilicalis*), passing in the round ligament of the liver, the portal vein system;

- superior epigastric vein (*v. epigastrica superior*), which flows through the internal thoracic vein (*v. thoracica interna*) and subclavian vein (*v. subclavia*) into the brachiocephalic vein (*v. brahiocephalica*) of the superior vena cava system;

- thoracic vein (*v. thoracoepigastrica*), which flows into the axillary vein (*v. axillaris*) of the superior vena cava system;

- superficial epigastric vein (*v. epigastrica superficialis*), which through the femoral vein (*v. femoralis*), and then the external iliac vein (*v. iliaca externa*) flows into the common iliac vein (*v. iliaca communis*) systems of the inferior vena cava;

- the inferior epigastric vein (*v. epigastrica inferior*), which, through the external iliac vein (*v. iliaca externa*), flows into the common iliac vein (*v. iliaca communis*) of the inferior vena cava system.

4. Portocaval anastomoses of the posterior wall of the abdominal cavity in the lumbar region, in the parts of the ascending line not covered by the peritoneum and the descending colon of the duodenum, the pancreas anastomose with each other:

- tributaries of the splenic vein (*v. lienalis*) of the portal vein system;

- tributaries of the superior and inferior mesenteric veins (vv. mesenterica superior et inferior portal vein system;

- tributaries of the lumbar veins (*vv. lumbales*) system of the inferior vena cava. The formation of ascites is also natural in the formation of portal hypertension syndrome, ascites will be discussed later.

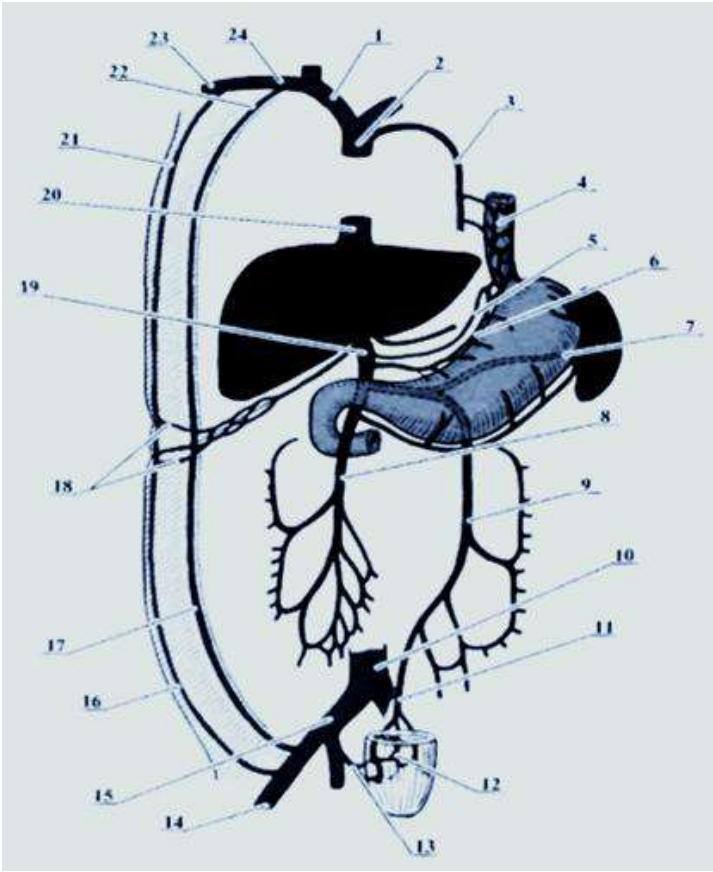


Figure 86. Scheme of intersystem venous anastomoses:
1 – *v. brachiocephalica*; 2 – *v. cava superior*; 3 – *v. azygos*;
4 – *vv. oesophageales*; 5 – *v. gastrica sinistra*; 6 – *v. gastrica dextra*;
7 – *v. lienalis*; 8 – *v. mesenterica superior*; 9 – *v. mesenterica inferior*;
10 – *v. cava inferio* r; 11 – *v. rectalis superior*; 12 – *plexus venosus rectalis*;
13 – *vv. rectales media et inferior*; 14 – *v. femoralis*; 15 – *v. iliaca communis*;
16 – *v. epigastrica superficialis*; 17 – *v. epigastrica inferior*;
18 – *vv. paraumbilicales*; 19 – *v. porta hepatis*; 20 – *v. cava inferior*;
21 – *v. thoracoepigastrica*; 22 – *vv. Epigastricae superiores*;
23 – *v. axillaris*; 24 – *v. subclavia*

Enlargement of the spleen in the syndrome of portal hypertension occurs necessarily. The cause of splenomegaly is the stagnation of blood in the splenic vein. It is important to understand the difference between splenomegaly and hypersplenism. Splenomegaly is an anatomical increase in the size of an organ. Hypersplenism − increased destruction of blood cells in the spleen, which is also possible in the case of severe portal hypertension syndrome.

Clinical criteria for hypersplenism (W. Dameshek):

1) a decrease in the number of one or more cellular elements of peripheral blood (pancytopenia; thrombocytopenia, leukopenia, anemia);

2) hyperplasia of the corresponding sprouts of the bone marrow;

3) splenomegaly;

4) improvement of clinical and hematological parameters after splenectomy.

It has now been proven that in liver cirrhosis, hypersplenism is often combined with autoimmune cytopenias, therefore it is recommended to use the term «cytopenic syndrome». To detect splenomegaly, it is necessary to master the technique of percussion and palpation of the spleen. This block of information is also located in this tutorial. In addition to these changes, with portal hypertension syndrome, pronounced dyspeptic disorders appear − loss of appetite, belching, heartburn. Their cause is a violation of the blood circulation of the stomach, intestines due to venous stasis. There is a decrease in the absorption capacity of the intestine and a decrease in the secretory function of the pancreas. As a result of recent facts and dyspeptic phenomena, patients with portal hypertension syndrome begin to lose weight dramatically. Ultimately, with long-term portal

hypertension, a sharp contrast is striking − a large belly with ascites and a sharply emaciated patient's body (fig. 87).

There is increased dryness of the skin, its peeling is observed. The patient is often thirsty due to slight absorption of fluid, fluid retention in the abdominal cavity with increasing ascites. Daily diuresis decreases, oliguria occurs, as ascites progresses.

In violation of cholesterol metabolism in patients with cirrhosis of the liver, intradermal cholesterol deposition occurs − xanthomatosis in the form of yellow plaques, which are located especially often on the eyelids (xanthelasma), less often on the hands, elbows and feet (xanthomas); xanthomatosis is also observed in other diseases accompanied by impaired cholesterol metabolism (with atherosclerosis, diabetes mellitus, essential hyperlipidemia, etc.).

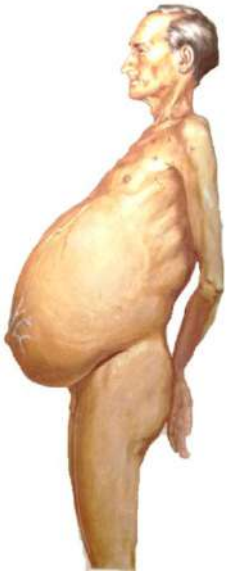


Figure 87. Appearance of a patient with a long-term portal hypertension syndrome

Cirrhosis of the liver is the last, irreversible stage of the pathological process. It is characterized by the formation of parenchymal nodules surrounded by fibrous septa, a violation of the architectonics of the liver, its vascular system, the formation of false lobules, intrahepatic anastomoses, and the development of portal hypertension. Cirrhosis of the liver ranks first among the causes of death from diseases of the digestive system (excluding tumors).

In accordance with the classification (Los Angeles. 1994), cirrhosis is distinguished by etiology, the degree of activity determined by biochemical tests (ALT activity), morphological changes in the liver, and the severity of hepatocellular insufficiency.

Among the etiological variants of liver cirrhosis, there are viral, alcoholic, autoimmune, toxic, genetic (hemochromatosis, Wilson−Konovalov's disease, alpha1-antitrypsin deficiency), cardiac, due to intra- and extrahepatic cholestasis (primary and secondary biliary cirrhosis, primary sclerosing cholangitis, venous congestion). − Budd−Chiari disease), cryptogenic.

According to the activity and phase of the cirrhosis process, there are: exacerbation (active phase) − activity is minimal, moderate, pronounced; remission (inactive phase). According to the course of cirrhosis, there are: slowly progressive, rapidly progressive and stable course.

Most often, cirrhosis of the liver develops as a result of chronic viral (B, C, D), alcoholic, drug, autoimmune hepatitis. Less commonly, cirrhosis occurs as a result of hemochromatosis, Wilson−Konovalov disease, alpha-1 antitrypsin deficiency, primary biliary cirrhosis, primary sclerosing cholangitis, venous stasis (Budd−Chiari disease). Often, two or more etiological factors are involved in the development of liver cirrhosis. In about 20% of cases of liver cirrhosis, the etiology remains unknown.

The development of irreversible changes in cirrhosis is due to diffuse damage to the liver parenchyma with prolonged exposure to a damaging factor, as well as the predominance of collagen synthesis over its breakdown. In all cases of the development of liver cirrhosis, the mechanism of self-progression of the pathological process and stimulation of the formation of connective tissue is central in the pathogenesis:

- an organic scar is formed in the foci of liver necrosis at the site of dead hepatocytes;
- the vessels of the portal tract approach the central vein;
- conditions are created for the passage of blood from the hepatic artery and portal vein into the central vein;
- blood flow bypassing the sinusoids of intact areas of the liver leads to their ischemia, and then necrosis;
- with necrosis, substances stimulating liver regeneration are released;
- nodes of regeneration develop, which compress the vessels and contribute to further disruption of blood flow in the liver;
- decay products of hepatocytes (biologically active substances) stimulate the inflammatory response;
- infiltrates are formed that spread from the portal fields to the central departments, contributing to the development of the post sinusoidal block.

In addition to the above, the inflammatory process in liver cirrhosis is characterized by intense fibrosis, connective tissue septa are formed that contain vascular anastomoses (intrahepatic porto-portal shunts); they connect the central veins and portal tracts, forming pseudolobules. Blood enters immediately into the hepatic vein system bypassing the pseudolobular parenchyma,

which causes ischemia and necrosis of the following hepatic cells (a vicious circle closes).

The clinical picture of liver cirrhosis depends on the degree of activity of the process and the degree of compensation of hepatocellular function.

For cirrhosis of the liver with a minimum degree of activity, the following changes are characteristic: subjective manifestations of the disease are unstable. There may be a temporary decrease in performance, bleeding gums and darkening of the urine that appear after an intercurrent infection. Patients are worse than before, endure physical and emotional stress. An objective study reveals «spider veins», telangiectasias in 40−50 % of patients. Hepatomegaly occurs in 80−90 % of patients, splenomegaly in 30 %.

For cirrhosis of the liver of a moderate degree of activity, it is characteristic: complaints of increased fatigue, poor appetite, weight loss, there may be skin itching, jaundice, nosebleeds. Objectively, icterus of the sclera, mucous membranes, skin, «spider veins» (telangiectasias), palmar erythema, angiomas at the edge of the nose, in the corner of the eyes are often detected; varnished, edematous, not coated tongue, gynecomastia in men; atrophy of the genital organs;

For cirrhosis of the liver of a pronounced degree of activity is characteristic: The liver is enlarged, (sometimes small in size), dense, often bumpy, with a sharp edge. Many patients with cirrhosis of the liver have an enlarged spleen and pancytopenia syndrome (anemia, leukopenia, thrombocytopenia). With a pronounced activity of cirrhosis of the liver, there may be subfebrile body temperature. Enlargement of the spleen is characteristic, which is explained by the development of portal hypertension, venous congestion in the organ, pulp fibrosis

Three stages of liver cirrhosis are described − initial, advanced and terminal.

The initial stage of cirrhosis is characterized by bloating, pain and a feeling of heaviness in the upper abdomen; weight loss, weakness, increased fatigue, decreased performance; enlargement and hardening of the liver; splenomegaly is moderately expressed or not yet obvious; sometimes this stage proceeds latently and is detected during a routine examination or morphological examination of a liver biopsy for another disease.

The advanced stage of liver cirrhosis is characterized by the appearance of the following symptoms: fingers and nails − in the form of «drum sticks» and «watch glasses»; often the appearance of patients resembles a «spider figure»; there are jaundice, skin hemorrhagic syndrome, gynecomastia, genital hypoplasia; pastosity and swelling of the legs, ascites; symptoms of portal hypertension (dilation of the veins of the anterior wall of the abdomen, esophagus, stomach, intestines).

The end stage of liver cirrhosis is characterized by: increased hepatocellular insufficiency up to the development of coma; portal hypertension with refractoriness of ascites to drug therapy; development of hepatorenal syndrome; accession of a bacterial infection; frequent profuse bleeding from varicose veins of the esophagus and stomach; transformation into liver cancer.

With a slight violation of liver function, periodically appear heaviness in the right hypochondrium, asthenia; there is no hemorrhagic syndrome, laboratory parameters have changed slightly, including the prothrombin index is reduced to 60 %, and the albumin-gamma-globulin coefficient is down to 2.5. With moderate liver dysfunction, astheno-vegetative syndrome, heaviness in the right hypochondrium, bleeding gums, easy bruising, repeated nosebleeds, moderate changes in laboratory

parameters in blood serum are detected, including a decrease in the prothrombin index up to 50 %, and the albumin-gamma-globulin coefficient up to 2. With a significant violation of liver function, asthenic-vegetative and hemorrhagic syndromes are expressed. There are symptoms of portal hypertension and hepatic encephalopathy, disorders of the genital organs. The change in blood serum parameters is significantly expressed, including the prothrombin index decreases below 50 %, and the albumin-gammaglobulin coefficient is below 2.

Some known symptoms of cirrhosis of the liver are:

«Biliary rheumatism» − joint pain in a patient with chronic hepatitis, cirrhosis of the liver − an expression of French doctors.

«Night blindness» − a violation of twilight vision with vitamin A deficiency in a patient with cirrhosis of the liver.

«Hands of beer lovers», a synonym – «palmar erythema» − symmetrical reddening of the thenar and hypothenar areas, accompanies the clinic of liver cirrhosis.

«White nails» is a symptom of long-standing cirrhosis of the liver.

Hepatocellular function in cirrhosis of the liver is assessed by Child−Pugh (tab. 8). Group A indicators indicate compensated cirrhosis. Group B and C indicators correspond to decompensated cirrhosis.

In the phase of remission of cirrhosis of the liver, the pathological process, as a rule, has a benign course. It is asymptomatic, with normal or slightly altered blood biochemical parameters.

In the phase of exacerbation of liver cirrhosis, manifestations of the pathological process appear or intensify (clinical and laboratory-instrumental).

Table 8

Child−Pugh scoring for liver cirrhosis

Index	Points		
	1	2	3
Ascites	no	small	moderate or large
Encephalopathy	no	small	moderate or large
Bilirubin (mg/dl)	<2	2−3	>3
Albumin (mg/l)	>3,5	2,8−3,5	<2,8
Prothrombin time (s)	1−3	4−6	>6

The total number of points in this table is summarized. With a total score of 5−6, liver cirrhosis is classified as category A; 7−9 − category B, 10−15 − category C.

Biochemical indicators in liver cirrhosis: in cirrhosis of viral etiology, markers of hepatitis viruses (B and C) can be detected. Hepatitis D virus markers are detected in the most active viral cirrhosis of the liver. In patients with alcoholic cirrhosis, the activity of gamma-glutamyl transferase increases to a greater extent.

Secondary biliary cirrhosis − prevalence unknown. The disease is based on extrahepatic cholestasis as a result of obstruction of the biliary tract (calculous cholecystitis, choledocholithiasis, operations on the biliary tract with subsequent formation of strictures, congenital defects of the biliary system). The disease occurs as a result of a prolonged violation of the outflow of bile at the level of large extrahepatic bile ducts. Patients are concerned about pain in the right hypochondrium, different in nature and intensity. An increase in

the liver is objectively determined, an increase in the gallbladder is also possible. Liver signs are less common than in primary biliary cirrhosis.

Atresia of the bile ducts

Biliary atresia occupies a leading position in the structure of diseases of the hepatobiliary system in young children and is the most common indication for liver transplantation. The main morphological features of this disease are cholestasis (intracellular, intrasinusoidal), bile duct proliferation, fibrosis, portal and periportal inflammation. The severity of cholestasis, proliferation of bile ducts and the prevalence of fibrosis is directly proportional to the age of the child. The intensity of inflammatory changes does not reflect the true severity of the disease.

The gold standard for diagnosis is a liver biopsy. There is a high diagnostic value of determining the activity of the enzyme gamma-glutamyl transpeptidase (GGTP) in the blood serum. An increase in GGTP activity up to 10 times indicates a significant severity of fibroplastic changes in the liver tissue. An exceptionally early diagnosis of the disease is important, which allows timely correction of pathological changes and prevention of the development of biliary cirrhosis**.**

Hepatorenal syndrome

Hepatorenal syndrome (HRS) is a common pathology in patients with liver cirrhosis and ascites. The first descriptions of the syndrome were presented in 1863. A.Flint in patients with liver cirrhosis and ascites.

The term «hepatorenal syndrome» was introduced in 1916 by P. Merklen as "a combination of an anatomically defined liver disease with a significant limitation of kidney function with little or no morphological changes in them.

Currently, hepatorenal syndrome is understood as a functional, oliguric, progressive, but at the same time reversible pathology of the kidneys that occurs in severe liver diseases with liver failure, when other causes that contribute to kidney damage are excluded.

The functional nature of renal failure in patients with ascites on the background of liver cirrhosis was confirmed by the complete restoration of kidney function after liver transplantation.

Liver diseases most commonly associated with HRS include:

1) cirrhosis, especially alcoholic, in the presence of ascites and diuretic therapy, hepatic encephalopathy, esophageal-gastric bleeding;

2) fulminant liver failure;

3) acute viral hepatitis;

4) hepatocellular carcinoma;

5) acute fatty liver of pregnant women.

With HRS, a multiorgan pathology develops, characterized by an acute dysfunction of the cardiovascular system, kidneys, liver, adrenal glands, brain and other organs and tissues. For the diagnosis of HRS at the first stage, it is important to identify the glomerular filtration rate (GFR). However, in patients with cirrhosis of the liver, the mass of muscle tissue is reduced, and, accordingly, the synthesis of creatinine. Similarly, urea produced by the liver can be reduced. These features often lead to false negative diagnoses. With this in mind, it was decided to diagnose HRS when the serum creatinine level rose above 1.5 mg/dL.

At the second stage, differential diagnosis of HRS and other kidney diseases is carried out.

In 1996 The International Ascites Society was the first to formulate diagnostic criteria for HRS.

Big GDS criteria:

1. The presence of chronic liver disease with liver failure, portal hypertension and fulminant liver failure.

2. Low GFR (increased serum creatinine more than 1,5 mg/dl and decreased creatinine clearance less than 40 ml/min).

3. Absence of shock, infections and data on the use of nephrotoxic drugs; no indication of hypovolemia due to pathology of the gastrointestinal tract (uncontrollable vomiting, diarrhea) or kidneys (fluid loss of more than 500 ml per day during the day in a patient with ascites without peripheral edema or more than 1000 ml per day in a patient with peripheral edema).

4. Lack of improvement in kidney function (decrease in serum creatinine to 1.5 mg/dl or less or increase in creatinine clearance to 40 ml/min or more) after discontinuation of diuretics and administration of 1.5 liters of isotonic solution.

5. Proteinuria less than 500 mg/day and no ultrasound evidence of obstructive or parenchymal kidney disease.

Minor criteria for hepatorenal syndrome:

1. Decreased daily diuresis less than 500ml/day.

2. The concentration of sodium in the urine is less than 10 meq/l.

3. The content of sodium in the blood serum is less than 130 meq/l.

4. The osmolarity of urine is greater than the osmolarity of blood serum (coefficient above 1.3).

5. Absence of hematuria (less than 50 erythrocytes in the field of view).

The diagnosis of HRS required the presence of all major criteria and preferably the presence of minor ones. However, in 2005, these criteria were revised in San Francisco.

New criteria for hepato-renal syndrome (2005):

1. Cirrhosis of the liver with ascites;

2 Serum creatinine more than 133 mmol/l (1,5 mg/dl);

3. Absence of normalization of serum creatinine (reaching a level less than or equal to 133 mmol/l) after 2 days of discontinuation of diuretics and administration of albumin − the recommended dose is 1 g per 1 kg of body weight per day (up to a maximum dose of 100 g/day).

4. No shock.

5. Lack of data on the use of nephrotoxic drugs.

6. The absence of any parenchymal kidney disease, manifested by proteinuria, hematuria, microhematuria and / or the corresponding ultrasound picture.

Acute liver failure

Acute liver failure is a clinical and laboratory syndrome that develops as a complication of many diseases, accompanied by necrobiotic changes in hepatocytes. There is a blockade of all the main functions of the liver, severe endogenous intoxication and, as a result, multiple organ failure. The term «Acute liver failure» refers to both an acute condition, accompanied by the development of encephalopathy over the next 8 hours, and the culmination of chronic liver disease. Fulminant («lightning») liver failure is very difficult. The main sign of fulminant liver failure is the development of symptoms within 8 weeks of the onset of the first signs of liver disease, more often jaundice. The development of massive necrosis of the liver is confirmed by a rapid decrease in its size (symptom of «melting liver», «melting ice»); it becomes flabby, doughy, its lower edge ceases to be palpated, the zone of hepatic dullness decreases and disappears. A distinct hepatic odor from the mouth and the same smell of sweat is revealed.

The main syndromes of acute liver failure are as follows:

1. Malnutrition syndrome − loss of appetite, development of nausea, intolerance to fatty and protein foods, abdominal pain, bloating, unstable stools, weakness, emaciation, skin changes

(dryness, thinning, wrinkling), development of neuritis, anemia. The basis of this syndrome is a violation of metabolic processes.

2. Hepatic encephalopathy is a reversible neuropsychiatric disorder that complicates the course of liver diseases, manifested by a complex set of disorders. Clinical manifestations of hepatic encephalopathy can vary from minimal drowsiness, impaired attention to deep coma.

The development of hepatic encephalopathy goes through the following stages:

1) the stage of precursors: sleep disturbance (insomnia at night and drowsiness during the day), conflict, deterioration of orientation in space;

2) stupor stage: drowsiness while maintaining the ability to follow simple commands, the patient reacts to painful stimuli or a loud cry, but, following simple instructions, quickly gets tired;

3) stage of hepatic coma: lack of consciousness: a) with a reaction to pain stimuli; b) with a generalized reaction (convulsions) to strong pain stimuli; c) without reactions to painful stimuli;

4) stage of irreversible coma: clinical picture of intravital brain death.

It should be noted that the drowsiness of a liver patient may very much resemble natural sleep, which may not alert relatives or medical personnel: there are no characteristic changes in the rhythm of breathing, the patient seems to be sleeping and breathing calmly. The body temperature begins to rise, but it is also possible to change from hyperthermia to hypothermia.

Coma in acute liver failure is often accompanied by psychomotor agitation and cerebral edema. For chronic encephalopathies associated with liver failure, lethargy and drowsiness are characteristic.

3. Feverish syndrome − with terminal liver diseases, fever may develop up to 38°C, even up to 40°C. Hyperthermia is resistant to antibiotics and is associated only with liver damage. The pathogenetic aspect of fever is necrosis, the entry of toxic products into the blood, bacteremia, and possibly the entry of microorganisms from the intestine with the development of sepsis in the terminal stage. The intestinal microflora enters the systemic circulation either through a damaged hepatic filter or through portosystemic collaterals, systemic toxemia occurs, the function of the reticuloendothelial system (Kupffer cells and polymorphonuclear leukocytes) is disturbed, spontaneous bacterial peritonitis develops in the terminal stage (in patients with liver cirrhosis at 75 % of cases), urinary tract infections, pneumonia, lymphadenitis, endocarditis.

4. Jaundice syndrome − massive necrosis is almost always accompanied by an increase in jaundice due to an increase in the concentration of free bilirubin.

5 Arterial hypotension with high cardiac output and low peripheral vascular resistance. The state of increased vasodilation in liver failure contributes to tissue hypoxia. In liver cirrhosis, increased permeability of the intestinal mucosa and portosystemic shunting lead to the entry of endotoxins and cytokines into the vascular bed, which may explain the changes in hemodynamics. It is assumed that nitric oxide and prostaglandins (E_1, E_2) play a certain role in the formation of the hyperdynamic type of blood circulation with the subsequent development of portal hypertension, ascites, and hepatorenal syndrome. Under the influence of vasoactive metabolites, normally inactive arteriovenous anastomoses develop, arterial and vascular volume increases, as a result of which the effective arterial blood volume decreases, and hypoxia develops. The blood circulation of the kidneys, liver, brain suffers. Clinical changes in hemodynamics are manifested, in addition to arterial hypotensionzia, hyperemia

of the extremities, jumping pulse and capillary pulsation. Characterized by tachycardia, hypotension, decreased sonority of heart tones, flatulence, decreased diuresis.

6. Hepatopulmonary syndrome − changes occurring in the small vessels of the lungs, in severe cases, lead to the development of hepatopulmonary syndrome in the form of cyanosis, a decrease in oxygen saturation. In 2 % of cases, patients with portal hypertension may develop pulmonary hypertension. The probable reason for this is the entry into the bloodstream of vasoconstrictor substances of intestinal origin (endothelin-1) through portosystemic and portopulmonary bypass.

7. Edema-ascitic syndrome is associated with a decrease in albumin synthesis in the liver and a drop in oncotic pressure; aldosterone inactivation is also disturbed, which leads to secondary hyperaldosteronism with the development of hypernatremia and hypokalemia.

8. A specific hepatic sweet smell of the skin, which is associated with the accumulation of methyl mercaptan, which is formed from methionine, which accumulates in excess due to a violation of demethylation processes in the liver.

9. Hemorrhagic diathesis syndrome − synthesis of blood coagulation factors and a sharp increase in the consumption of existing coagulation factors, which leads to widespread thrombosis and even to disseminated intravascular coagulation (DIC) and bleeding. In the liver, all blood coagulation factors, with the exception of VIII, coagulation inhibitors and proteins of the fibrinolytic system are formed. The liver is also involved in the elimination of activated clotting factors. Coagulopathy in acute liver failure has a complex genesis and is caused not only by a deficiency of coagulation factors, but also by an increase in fibrinolytic activity, the most likely cause of which is

intravascular coagulation. The resulting coagulopathy predisposes to spontaneous bleeding in the form of gastrointestinal, nasal, uterine bleeding, gums bleed, petechial rashes appear, hematomas at the injection sites, and a tourniquet is applied. With gastrointestinal bleeding, melena is observed, in the washings of the stomach – «coffee grounds». Typical complaints are muscle and headaches, weakness. Brain hemorrhages are possible.

10. The syndrome of endocrine disorders is extremely diverse (testicular atrophy, infertility, gynecomastia, hair loss, atrophy of the mammary glands, uterus, menstrual irregularities, the formation and rapid increase in spider veins, palmar erythema, the phenomenon of white nails). The mechanism is the accumulation of estrogens or a decrease in their inactivation.

Criteria for acute liver failure in the clinic are:

- the presence of jaundice of the skin and mucous membranes;

- a decrease (more often) or an increase (less often in leptospirosis) in the size of the liver;

- hemorrhagic syndrome in the form of punctate rashes, bruises, hematomas at injection sites, bleeding gums, nasal,

- uterine, gastrointestinal bleeding;

- laboratory criteria: the content of total bilirubin is more than 100 mmol/l and above; hypoproteinemia and dysproteinemia; increased activity of cytolytic enzymes; decrease in prothrombin index up to 60 % and below; decrease in the level of pseudocholinesterase to 5000 mmol / l and below;

- a cerebral deficit of 14 points or less on the Glasgow scale; lack of effect or deterioration of the patient's condition in dynamics; lack of effect or deterioration of the patient's condition in dynamics with conventional conservative therapy; violation of coordination of movements according to functional tests

(structural, exit from the maze, writing on unlined paper, determining the reaction rate, etc.).

Acute liver failure in the terminal stage can have formidable complications:

1. Edema-swelling of the brain, which is manifested by signs of irritation of the meninges, hyperemia, convulsive muscle twitching, symptoms of damage to the cranial nerves, progressive respiratory disorders, increased blood pressure, a tendency to bradycardia.

2. Gastrointestinal bleeding (including stress erosion and ulcers of the gastrointestinal tract, gastroenteritis with the addition of renal failure).

3. Secondary infection: pneumonia, including aspiration, angiogenic and urethral sepsis.

4. Fulminant liver failure occurs with multiple organ damage: the kidneys, cardiovascular system, lungs, pancreas, and brain are involved in the process. In 55% of patients, acute tubular necrosis is noted. For the diagnosis of disorders of the nitrogen excretion function of the kidneys, it is advisable to use the indicators of plasma creatinine. Due to the violation of the urea-forming function of the liver, the level of urea cannot be an adequate criterion for assessing the degree of azotemia.

Diseases that cause the development of liver failure can be represented by 5 groups.

1. Liver diseases: acute and chronic hepatitis, liver cirrhosis (portal, postnecrotic, biliary), malignant neoplasms, alveolococcosis, etc. In these cases, liver failure is the main manifestation of the disease, which determines the clinical picture and the nature of changes in biochemical parameters. Among the diseases of this group, the most common development of liver

failure occurs in viral hepatitis (due to the development of acute massive liver necrosis).

2. Obstruction of the bile ducts.

Mechanisms for the formation of liver failure: an increase in pressure in the bile ducts leads to the development of bile hypertension and a violation of the secretion of hepatocytes, in order to secrete the secretion into the lumen of the bile capillaries (in which there is stagnation of bile), the liver cell has to overcome great resistance. Biliary hypertension leads to impaired blood and lymph circulation in the liver, changes in organ microcirculation, which leads to the development of degenerative changes in hepatocytes and biliary cirrhosis of the liver. Changes in organ hemodynamics and secretion of hepatocytes are also observed with a sharp decompression of the bile ducts after their prolonged obstruction during surgery.

3. Diseases of other organs and systems − blood vessels, heart, systemic connective tissue diseases, endocrine and infectious diseases. As a rule, liver failure occurs in the chronic course of these diseases.

4. Poisoning by hepatotropic toxic substances.

5. Diseases caused by hereditary pathology of bilirubin metabolism

Substances with a hepatotoxic effect include:

1) natural substances (phalloidin, falloin in lines, beta-amanitin in pale grebe);

2) chemicals used in everyday life, agriculture, industrial poisons;

3) organic compounds (solvents).

Depending on the chemical nature and dose of the poison, the mechanism of its action is different, the main mechanisms are as follows:

1) violation of the enzyme systems of the endoplasmic reticulum of the hepatocyte, resulting in necrosis of the liver cell (poisoning with carbon tetrachloride);

2) blockade of sulfhydryl groups of hepatocyte enzymes (poisoning with salts of heavy metals);

3) damage to mitochondrial membranes (resulting in inhibition of oxidative phosphorylation), endoplasmic reticulum, lysosomes in case of damage by phallotoxins;

4) suppression of actin polymerization with the development of cholestasis (phalloidin);

5) inhibition of DNA, RNA with inhibition of protein synthesis, resulting in cell autolysis (amanitotoxins).

A variety of hepatotoxic poisons are medicinal substances. According to the mechanism of damage, they are divided into 2 groups.

True hepatotoxins. Means drugs that cause damage to hepatocytes through indirect action through metabolites leading to disruption of any metabolic process. In this group, cytotoxic (tetracycline) and cholestatic − causing liver damage by selective violation of secretion into the bile ducts (anabolic steroids) are distinguished.

Hepatotoxins cause idiosyncrasy with facultative reactions. It is assumed that their metabolites act as haptens that bind to cell macromolecules and form antigens. This induces immunopathological reactions leading to damage to the hepatocyte.

In some cases, drug metabolism disorders are genetically determined and can be caused by any drug. Each drug has a

specific morphological variant of changes in the liver. However, a number of drugs can cause, depending on individual sensitivity, various morphological changes. For example, halothane (halothane) and isoniazid cause massive liver necrosis; alpha-methyldopa − acute or chronic hepatitis, and in some cases − massive liver necrosis (tab. 9).

Table 9

Some drugs that cause liver damage

Dose-dependent hepatotoxicity	Paracetamol (overdose), salicylates (high doses), tetracyclines (high doses), Azathioprine, Methotrexate
Idiosyncratic hepatotoxicity	isoniazid, Halothane, Methyl-Dopa, Rifampicin, Dantrolene, Nitrofurantoin
Dose dependent cholestasis	Methyltestosterone
Idiosyncratic cholestatic hepatitis	Chlorpromazine, Erythromycin, Chlorpropamide, Tolbutamide

Extreme exposure to the body can also affect the liver. As a rule, such effects are a combination of factors (trauma, burns, crushing of tissues, severe surgery, accompanied by the formation of an extensive wound surface, massive blood loss, blood transfusions, severe purulent complications). Allergization of the body can have a certain significance in the development of liver failure, against which even small irritants lead to the development of a symptom complex of liver failure. With regard to fulminant liver failure, its development is often determined by a geographical factor. Thus, acute viral hepatitis is the most common cause in the United States and in some European countries. At the same time, in England, the overwhelming number of cases of fulminant liver failure is due to an overdose of paracetamol. If viral hepatitis A, B, D and E cause predominantly

fulminant liver failure, then hepatitis C causes delayed liver failure. Rare causes of fulminant liver failure include Wilson's disease, autoimmune chronic active hepatitis, Budd-Chiari syndrome, acute fatty liver of pregnancy, diffuse malignant liver tumors, hepatitis B reactivation, and hyperthermia. However, in many patients, the causative factor for fulminant liver failure remains unidentified. Table 10 presents the main liver diseases caused by hereditary metabolic pathology, which have vivid clinical manifestations.

Table 10

Diseases caused by hereditary pathology of bilirubin metabolism

Disease	Defect		Inheritance type	Clinical manifestations
Gilbert's syndrome	Decreased conjugation, in some cases, reduced absorption of bilirubin		autosomal dominant	Slight hyperbilirubinemia due to free bilirubin, increases with starvation, does not affect life expectancy
Crigler-Najjar Syndrome	Type 1	Lack of bilirubin-conjugating enzyme	autosomal recessive	Severe hyperbilirubinemia due to free bilirubin, patients die early, death is preceded by bilirubin encephalopathy
	Type 2	Insufficient activity of the conjugating enzyme	autosomal dominant	Severe hyperbilirubinemia due to free bilirubin is treatable with phototherapy, phenobarbital, and patients often survive to adulthood

Ending of table 10

Disease	Defect	Inheritance type	Clinical manifestations
Dubin-Johnson Syndrome	Decreased hepatic excretion of bilirubin	autosomal recessive	Mild hyperbilirubinemia due to conjugated bilirubin. Deposition of melanin pigment in the liver. Increased ratio of coproporphyrins I and III in the urine, bilirubinuria. For duration has no effect on life
Rotor syndrome	Defect unknown	autosomal recessive	Mild hyperbilirubinemia due to conjugated bilirubin. Deposition of melanin pigment in the liver. Increased ratio of coproporphyrins I and III in the urine, bilirubinuria. For duration has no effect on life

Hepatic encephalopathy

Hepatic encephalopathy is a reversible neuropsychiatric disorder that accompanies many liver diseases accompanied by liver failure and / or portosystemic shunting. The most common hepatic encephalopathy occurs with cirrhosis of the liver. The diagnosis of hepatic encephalopathy can be made during a physical examination with the following signs:

− hypothermia with a temperature below 36.0 degrees Celsius;

− liver odor from the mouth - an unpleasant odor caused by the excretion of sulfur-containing by-products of amino

acid metabolism, such as dimethyl sulfide, methyl mercaptan, ethyl mercaptan;

− asterixes and impaired cognitive functions.

Asterixes is a «flapping» tremor of the hands, which occurs more often in acute encephalopathy, poisoning with barbiturates, lead, mercury, manganese, ammonia, anticonvulsants (fig. 88). It is not strictly pathognomonic for liver diseases, as it is also detected in renal, respiratory and congestive heart failure.

Hepatic encephaloratia is characterized by a combination of asterix with cognitive impairment. There are many tests to detect cognitive impairment, but the Reiten test and the A-deletion test are most commonly used (the patient is asked to cross out all the letters A from one or two paragraphs of the newspaper, then they count how many letters were not crossed out.

The classification of hepatic encephalopathy based on changes in mental status is the most widely used in clinical practice. According to this classification, 5 degrees of hepatic encephalopathy are distinguished in individuals with liver diseases.

0 degree − there is no violation of consciousness, mental activity, personality and behavior;

Grade 1 − minor disorientation, euphoria or anxiety, decreased attention;

Grade 2 – sickness, malaise, personal changes, disorientation of a person, time and place;

Grade 3 – since somnolence till the stupor, disturbance of consciousness, inadequate answer in a pain stimuli.

Grade 4 – coma, absent any reactions in a pain stimuli.

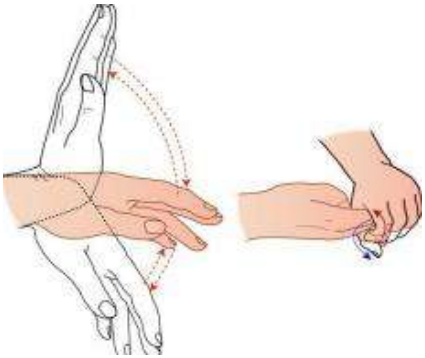


Figure 88. Asterixes and the method of its detection - the doctor asks to grab his fingers with a brush, in this case, the doctor's brush is trembling

Hepatic encephalopathy can be either acute or chronic. In patients with cirrhosis with any neurological disorders, hepatic encephalopathy should be suspected. Neurological manifestations of hepatic encephalopathy are nonspecific. However, patients with alcoholic liver cirrhosis have Wernicke–Korsakoff encephalopathy, seizures, alcohol intoxication, or alcohol withdrawal syndrome. A common diagnostic error is the lack of definition of vitamin B_1 deficiency.

The course of chronic hepatic encephalopathy is accompanied by spontaneous relapses and remissions. Most often, chronic hepatic encephalopathy accompanies conditions with a large porto-systemic shunt.

Separately, it is necessary to highlight the syndrome of fulminant liver failure. Fulminant liver failure occurs in patients with severe liver damage without previous liver disease and is associated with high mortality. Fulminant liver failure includes cerebral edema, hemodynamic instability, renal failure,

coagulopathy, profound metabolic disturbances, and increased susceptibility to bacterial and fungal infections. The etiological factor in the development of fulminant liver failure plays a leading role, as it determines the histological picture of liver damage. Particularly acute are states with acetaminophen poisoning, acute liver damage by hepatitis A and B viruses. Among other equally significant causes of fulminant hepatic disease, it is necessary to note the herpes simplex virus, cytomegalovirus, as well as autoimmune hepatitis, pregnancy, Budd−Chiari syndrome, obliterating endophlebitis of the hepatic veins, heart failure, septic shock, Wilson−Konovalov disease, toadstool venom (Amanita phalloides), and lymphoma.

There are 2 classifications of fulminant liver failure based on the duration of the interval between the onset of jaundice and the onset of encephalopathy − the Bernois classification (France) and the O'Grady classification (UK).

The Bernois classification identifies fulminant liver failure when the jaundice-encephalopathy interval is no more than 2 weeks and subfulminant liver failure with an jaundice-encephalopathy interval of 2 to 12 weeks.

According to the O'Grady classification, hyperacute liver failure (within 1 week), acute liver failure (1−4 weeks) and subacute liver failure (from 5 to 12 weeks) are distinguished.

In most cases of fulminant liver failure, liver regeneration is noted to varying degrees, more active in hyperacute and subacute degrees.

CHAPTER 10.
LABORATORY DIAGNOSTICS OF DISEASES OF THE HEPATOBILIARY SYSTEM

The liver contains a huge amount of enzymes. It is believed that an increase in the level of these enzymes in the blood reflects an increase in the entry into the blood of the contents of damaged hepatocytes.

There are several laboratory hepatic syndromes.

Cytolysis syndrome is a group of laboratory tests reflecting hepatocyte necrosis.

The main laboratory markers of cytolysis are the activity of aminotransferase enzymes − alanine aminotransferase (ALT) and aspartate aminotransferase (AST). The activity of both aminotransferases is necessarily present in the blood serum, but does not exceed 30−40 IU/l. AST is present in the liver, myocardium, skeletal muscle, kidney, brain, pancreas, lungs, leukocytes, and erythrocytes. ALT is present in the highest concentration in the liver. An increase in the activity of transaminases in the blood serum is associated with damage to tissues rich in aminotransferases or a change in the permeability of cell membranes, which leads to the penetration of enzymes into the blood serum. A significant increase in serum AST activity occurs after extensive tissue necrosis, so this analysis is informative even in myocardial infarction. Also, mitochondrial AST is elevated in chronic alcoholic liver disease.

AST activity depends on age and gender. Table 11 shows normal values for the activity of this enzyme.

Table 11

Normal values of aspartate aminotransferase activity

Age	Units/l
newborns	25−75
10 days − 23 month	15−60
2−3 years	10−56
4−6 years	20−39
7−19 years	12−32
20−49 years, males	20−40
20−49 years, females	15−30
Males aand females older then 50 years old	10−35

Allocate a significant, moderate and slight increase in AST activity. A significant increase in AST activity is equivalent to an increase of more than 5 times from normal values, moderate − an increase of 3−5 times, mild − an increase in AST activity up to 3 times. The main reasons for the change in enzyme activity are presented in Table 12.

Of great importance in the differential diagnosis of diseases of the liver and biliary tract is the ratio of AST/ALT. So the ratio of AST / ALT more than 2 allows suspecting alcoholic liver disease, and the ratio of more than 3 confirms this diagnosis if there is an appropriate history. An AST/ALT ratio greater than 1 may indicate cirrhosis of the liver. An increase in the activity of serum transferases is not typical for the pathology of the biliary tract. An increase in AST activity with normal or slightly increased values of ALT activity is characteristic of the pathology of the heart muscle (in particular, myocardial infarction), as well as skeletal muscles.

In addition to ALT and AST transaminases, there are other laboratory tests that reflect hepatocyte necrosis. These include

determining the activity of glutamate dehydrogenase, isocitrate dehydrogenase, lactate dehydrogenase, and sorbitol dehydrogenase.

Table 12

The main causes of changes in the activity of aspartate aminotransferases

Increase in AST activity	Increase from normal values	The main reasons
Severe degree	more than 5 times	Acute viral hepatitis, acute hepatocellular insufficiency toxic, alcoholic, drug (eg acetaminophen), shock, acute pancreatitis
Moderate	3–5 times	Alcoholic cirrhosis of the liver, obstruction of the biliary tract, condition after operations and interventions on the heart, heart failure, myocardial infarction, HELLP syndrome, infectious mononucleosis, liver tumors, muscle diseases (dermatomyositis, polymyositis, gangrene, rhabdomyolysis, trichinosis), Ray's syndrome, trauma
Mild degree	Up to 3 times	Cerebrovascular stroke, fatty liver with slowly progressive cirrhosis, hemolytic anemia, pericarditis, pulmonary infarction, alcoholic psychosis
Decrease in AST activity		Hemodialysis, uremia, deficiency of vitamin B_6 or cofactors

Cholestasis syndrome

Cholestasis − stagnation of bile, difficulty in excreting bile, but this condition is not an analogue of jaundice. Cholestasis is understood as insufficient secretion of all or the main components of bile. Cholestasis syndrome is observed in all hepatobiliary diseases. The morphological manifestation of cholestasis is the accumulation of bile pigments in hepatocytes and bile ducts. Biochemical manifestations of cholestasis are associated with an increase in the content of bile components in the blood serum − cholesterol, phospholipids, beta-lipoproteins, bilirubin, bile acids and the activity of enzymes − alkaline phosphatase, 5-nucleotidase, leucine aminopeptidase, gamma glutamyl transpeptidase.

Cholestasis is a symptom complex that accompanies complete or partial obstruction of the biliary tract. To detect cholestasis in clinical practice, markers are used − the content of free and conjugated bilirubin in the blood, the activity of alkaline phosphatase enzymes, gamma-glutamyl transpeptidase, 5'-nucleotidase, leucine aminopeptidase.

Determining the concentration of bilirubin in the blood is the long-term criterion for cholestasis. The determination of total bilirubin, direct (conjugated, bound) bilirubin and indirect (unconjugated, free) bilirubin is important. Normal bilirubin values vary in the human population with age and sex. Table 13 shows the normal value of bilirubin to serum.

With an increase in the concentration of bilirubin in the blood, icterus of the sclera and yellowness of the skin appear.

The concentration of bilirubin increases with suprahepatic, hepatic and subhepatic jaundice.

An increase in the concentration of bilirubin in suprahepatic jaundice is associated with an intense breakdown of erythrocyte heme, which may be associated with neonatal erythroblastosis, extensive hematomas, hemolytic anemia,

pernicious anemia, post-hemotransfusion period, as well as the pathology of erythrocytic enzymes − glucose-6-phosphate dehydrogenase, pyruvate kinase), spherocytosis erythrocytes.

Table 13

Dynamics of normal values of bilirubin in blood serum during a person's life

Index	Mg/dl	Mmol/l
Total bilirubin newborns at 1 day 1 − 2 days 3 − 5 days 6 − 7 days 8 − 9 days 10 − 11 days 12 − 13 days 14 − 30 days Over 1 month, all children and adults	 Less then 5,8 Less then 8,5 Less then 11,7 Less then 8,4 Less then 6,5 Less then 4,6 Less then 2,7 Less then 0,8 Less then 1,2	 Less then 99 Less then 140 Less then 200 Less then 144 Less then 111 Less then 79 Less then 46 Less then 14 Less then 21
Unconjugated bilirubin (unbound, free)	Less then 1,1	Less then 19
Conjugated bilirubin (bound) newborns from day 29 − all children and adults	 Less then 0,6 Less then 0,3	 Less then 10 Less then 5

An increase in bilirubin in hepatic jaundice is associated with a deficiency of conjugated bilirubin in Crigler−Najjar syndrome, and may also be associated with a violation of bilirubin transport, as in Dubin−Johnson and Gilbert syndromes. Hepatic jaundice can also be caused by cholangitis, alcoholic acute and

chronic hepatitis, cholecystitis, cirrhosis, infectious mononucleosis.

An increase in the concentration of bilirubin in subhepatic jaundice may be associated with a tumor of the liver, pancreatic head, and other causes of biliary obstruction.

Alkaline phosphatase is a group of enzymes − catalysts of organic phosphate esters. In the body, alkaline phosphatase (ALP) is localized in bone osteoblasts, hepatocyte membranes, brush border cells of the small intestine mucosa, proximal convoluted renal tubules, leukocytes, and placenta. The maximum enzymes are localized in the liver and bone tissue. The range of normal values of ALP activity depends on the method of its determination, of which at least seven are used (tab. 14).

Table 14

Normal values of serum alkaline phosphatase activity

Method	Units	Normal values
Bessey–Lowry–Brock	1 μmol/L p-nitrophenol for 60 min	0,8 – 3,0
Bodansky	1mg/100ml inorganic phosphate for 60 min	1,5 – 4,0
international	1μmol/l p-nitrophenol per minute	21,0 – 85,0
international	1mg/100ml phenol for 30 min	3,0 – 13,0
King–Armstrong	1mg/100ml phenol for 30 min	3,0 – 13,0
Klein–Reed–Babson	1mg/100ml phenolphthalein for 30 min	1,0−4,0
Shinowar–Jones–Reinhart	1mg/100ml phenol for 60 min	2,2 – 8,6

In cholestasis, ALP is synthesized de novo in the liver and released into the blood. In 75 % of patients with prolonged cholestasis, the activity of alkaline phosphatase increases by 4 or more times, both with intra- and extrahepatic biliary obstruction. A significant increase is also observed in hepatobiliary disorders in patients with AIDS (for example, in tuberculosis with liver damage or in sclerosing cholangitis caused by cytomegalovirus infection).

A smaller increase in ALP activity, less than 4 times, is nonspecific and can occur with all liver diseases and congestive heart failure.

5'-Nucleotidase is a nucleotide catalytic enzyme found in the liver, intestines, brain, heart, blood vessels, and pancreas. The activity of 5'-nucleotidase increases primarily in diseases of the hepato-biliary system, as does the activity of alkaline phosphatase. Most studies state that ALP and 5'-nucleotidase have similar sensitivity and specificity in bile duct obstruction and infiltrative and voluminous liver lesions.

Gamma-glutamyl transpeptidase (GGTP) is an important enzyme in the metabolism of glutathione that regulates its intracellular level. Glutathione is involved in the transport of amino acids across cell membranes. The process is catalyzed by the GGTP enzyme. Normally, GGTP is always present in the blood serum, in slightly higher concentrations in men than in women. An increase in serum GGTP activity is noted in diseases of the liver, biliary tract, and pancreas. An increase in enzyme activity was also noted in myocardial infarction. GGTP is more specific than ALP, since its concentration does not change during bone diseases.

Leucine aminopeptidase (LAP) is a proteolytic enzyme that hydrolyses tissue amino acids from N-terminal proteins and peptides. LAP is maximally present in the bile epithelium of the liver, therefore, the highest activity of the enzyme is observed in

cholestasis. PLA is present in much lower concentrations in the tissues of the small intestine and pancreas. In diseases of the liver and biliary tract, several peaks of the enzyme were recorded. It is believed that the determination of LAP activity is a more sensitive marker in infiltrative liver diseases than ALP and 5'-nucleotidase. In addition to diseases of the hepatobiliary system, pregnancy leads to an increase in the activity of the LAP.

Syndrome of hepatocellular insufficiency

It is customary to discuss about the syndrome of hepatocellular insufficiency in the case when there is a deficiency in the synthesizing function of the liver, which is most noticeable with a deficiency in protein synthesis.

The blood serum contains a large amount of proteins, the main source of which is the liver. Liver cells synthesize albumin, fibrinogen and other clotting factors, as well as alpha and beta globulins. Only gamma globulins are synthesized by B-lymphocytes.

The total protein of blood serum is usually determined using a type of biuret reaction, and then it can be divided into fractions with the release of individual components - albumin and globulins.

Albumin is the main protein in blood serum, the normal content of which is 35−45 g/l. In a day in an adult, the liver should produce about 200 mg/kg.

In patients with acute viral hepatitis, hepatotoxic effects of drugs, obstruction of the biliary tract, the level of albumin in the blood serum does not change. A decrease in the level of albumin less than 30 g/l may indicate chronic hepatitis, cirrhosis of the liver.

Alcoholic beverages, protein starvation, and chronic inflammation can inhibit albumin synthesis. However, hypoalbuminemia does not necessarily mean liver disease. It can

occur in protein-losing enteropathy, nephrotic syndrome, chronic and severe infections.

The liver is the main organ for the synthesis of 11 blood coagulation proteins.

factor I − fibrinogen;

factor II − prothrombin;

factor V − proaccelerin, labile factor;

factor VII – proconvertin, stable factor, serum prothrombin conversion catalyst;

factor IX, a component of plasma thromboplastin, Christmas factor;

factor X − Stuart−Prawer factor;

factors XII − prekallikrein;

factor XIII is a high molar kininogen.

In liver diseases, coagulation disorders are often manifested, which can be assessed using special tests. For example, the Quick prothrombin time is used to determine the rate at which prothrombin is converted to thrombin. Results are expressed either in seconds or as a ratio of patient plasma prothrombin time to control plasma time. The normal value of prothrombin time according to Quick is 9−11 s. An increase of up to 13 s is a deviation from the norm, up to 15 s is a risk group for uncontrolled bleeding.

The international normalized ratio (MHO) is standardized and represents the prothrombin ratio (the ratio of the patient's prothrombin time to the prothrombin time of normal plasma), raised to the power of the international sensitivity index (MIC). With the help of INR, the degree of hypocoagulation is assessed in the treatment of indirect anticoagulants (warfarin and others), regardless of the thromboplastin used, and the results obtained by different laboratories are compared.

Vitamin K is required for the synthesis of coagulation factors II, VII, IX and X of the liver. Vitamin K deficiency due to

inhibition or reduction of its synthesis (for example, in hepatocellular carcinoma), taking vitamin K antagonists can cause severe coagulation disorders. In the case of vitamin K deficiency, des-γ-carboxyprothrombin (abnormal prothrombin) appears in the blood serum. In healthy individuals, there is no abnormal prothrombin in the blood serum. Deviation of prothrombin time from the norm by more than 5−6 s is a laboratory test confirming the possible development of fulminant liver necrosis in acute viral hepatitis. An increase in prothrombin time is a prognostic factor for patients with alcoholic steatonecrosis. Determination of prothrombin time is a very important test for all patients with liver diseases, which allows assessing preoperative risks, the quality of treatment, and adjusting therapy.

Laboratory syndrome of hepatic fibrosis

Diagnosis of hepatic fibrosis is one of the global issues in hepatology. Liver biopsy has been the «gold standard» for many years, but in modern realities it is not performed very often. To date, there are laboratory markers of hepatic fibrosis − hyaluronan (the most sensitive and specific), as well as type IV collagen, procollagen III, laminin. Also of interest are some multiparametric tests − fibrotest (multiparametric marker, including haptoglobin, bilirubin, GGTP, apolipoprotein A1, α2-macroglobulin), the ratio of AST activity to platelets, as well as glycomics.

Hyaluronan is a glycosaminoglycan synthesized in mesenchymal cells and widely present in the intercellular space. Hyaluronan is destroyed by the sinusoidal cells of the liver, therefore, with hepatocellular insufficiency, its concentration in the blood increases dramatically. Fasting serum hyaluronan levels greater than 100 mg/dl have been shown to have 78 % specificity and 83 % sensitivity in the diagnosis of liver cirrhosis. It has been proven that the serum level of hyaluronan correlates with the rate

of liver regeneration in patients after hepatectomy with the development of liver failure after surgery, and also normalizes in patients after liver transplantation.

Fibrotest showed adequate results in the group of patients with chronic hepatitis B, C and alcoholic liver disease.

Glycomics is the analysis of the DNA sequence encoding the total protein in blood serum. Only N-glycans are subject to analysis. According to this method, with a sensitivity of 79 % and a specificity of 86 %, it is possible to distinguish compensated liver cirrhosis from non-cirrhotic disease.

Immune inflammatory syndrome

Serum immunoglobulins are synthesized by beta-lymphocytes, so their determination is not a significant liver test. Most of the increased amount of immunoglobulins in patients with liver cirrhosis are antibodies against antigens of the normal intestinal microflora. In liver cirrhosis, these antigens are not destroyed by reticuloendothelial cells; antigens reach the lymphoid tissue outside the liver, where they cause an intense inflammatory reaction. In acute hepatitis, immunoglobulin levels remain within the normal range or are minimally elevated. A persistent moderate increase in γ-globulins is possible with chronic active hepatitis, but a significant increase in γ-globulins indicates autoimmune chronic hepatitis.

In most types of cirrhosis, a nonspecific diffuse polyclonal increase in IgG and IgM levels is found. An increase in Ig M is characteristic of primary biliary cirrhosis of the liver. An increase in IgA is characteristic of patients with alcoholic cirrhosis of the liver. In obstructive jaundice, IgA levels are normal.

In conclusion, it is advisable to present a comparative table of changes in laboratory markers in liver diseases (tab. 15).

Table 15

Comparative table of changes in laboratory markers in various liver diseases

Condition	Bilirubin	AST and ALT	ALP
hemolysis	norm	norm	norm
Gilbert's syndrome	5 mg / dl, more than 85% − indirect bilirubin. Bilirubin in the urine is not determined	norm	norm
acute necrosis of hepatocytes	increased direct and indirect	elevated more than 500 IU, with ALT higher than AST	norm or increase up to 3 times
chronic hepatitis	upgraded both factions	both elevated, but less than 300 IU	from normal to 3-fold increase
alcoholic hepatitis	bilirubinuria	AST/ALT>2	Norm
intrahepatic cholestasis	normal or elevated both fractions	normal or moderate increase, rarely >500 IU	increased 4 times and more
obstructive jaundice	bilirubinuria	norm	norm
infiltrative liver diseases (tumor, granulomas)	norm	normal or slight increase	raised 4 times or more

Ending of table 15

Albumen	Globulins	Prothrombin time
norm	norm	norm
norm	norm	norm
norm	norm	norm, or increased, which indicates an unfavorable prognosis
decreased or normal	increase in γ-globulins	norm or insignificant elongation
norm	norm	norm or lengthening in cirrhosis
norm in the absence of chronization	γ-globulins are normal	norm
norm	may increase beta globulins	norm
norm	normal or elevated γ-globulins	norm

CHAPTER 11.
THE MAIN FEATURES OF THE ANATOMY, COMPLAINTS AND ANAMNESIS IN DISEASES OF THE PANCREAS

The pancreas is located retroperitoneally, behind the stomach. This is a lobed organ of an oblong shape, rather soft, but elastic. The pancreas is projected onto the anterior abdominal wall in the epigastric region, occupying its middle part and the left hypochondrium. The organ is conditionally divided into the head, body and tail (fig. 89).

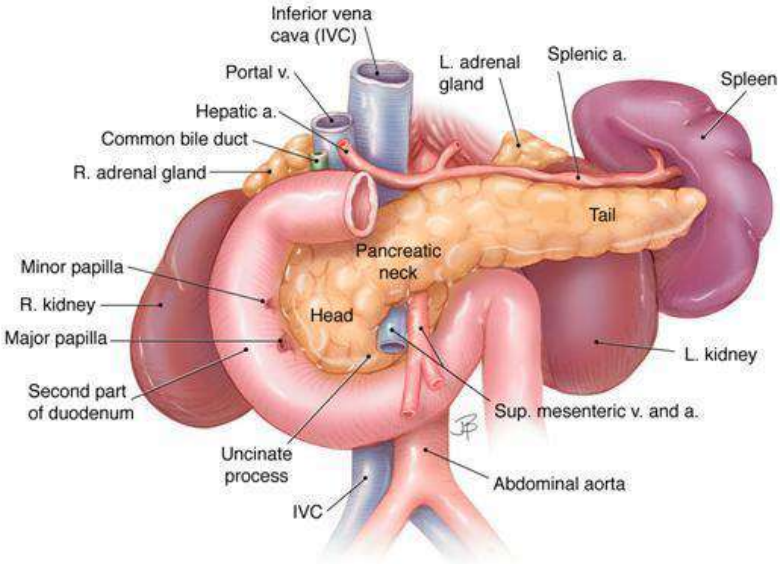


Figure 89. Topography of the pancreas

The lobules of the pancreas consist of a group of cells called acini that secrete enzymes. One acinus unites from 5 to 8 pancreatic cells, lobular pancreatic ducts gather in the center of the acinus, later flowing into second- and first-order flows, and

the latter open into the main pancreatic duct (Virsungov) or accessory duct (Santorini).

Allocate endocrine and exocrine tissue of the pancreas. Endocrine tissue is represented by a cluster of cells (islets of Langerhans) of three types − alpha, beta and delta cells. Alpha cells of the islets of Langerhans produce glucagon, beta cells produce insulin, and delta cells produce somatostatin. The endocrine tissue of the pancreas accounts for no more than 5 % of its mass.

The exocrine tissue of the pancreas is responsible for the production of pancreatic juice (1.5−3 liters per day), which plays an important role in the digestion process. Pancreatic juice is rich in over 20 enzymes, is alkaline and essential for digestion and maintaining optimal intestinal pH. Pancreatic juice enzymes provide the breakdown of proteins, fats and carbohydrates. So proteins are cleaved by endopeptidases − trypsin, chymotrypsin, which break the internal peptide bonds of proteins and polypeptides; exopeptidases − carboxypeptidase, amino-peptidase − which react with free carboxyl and amino-terminal ends of peptides, as well as elastase. Proteolytic enzymes are initially secreted in an inactive state and then become active through complex chemical reactions. Enterokinase, an enzyme found in the mucosa of the duodenum, breaks the lysine-isoleucine bond, which transforms trypsinogen into trypsin. Trypsin further activates other proteolytic enzymes, entering into a cascade of reactions with phospholipase A.

Carbohydrates are cleaved by alpha-amylase, maltose, and nucleic acids by ribonuclease and deoxyribonuclease. Fats are broken down by pancreatic lipase, phospholipase A and cholesterol esterase. In addition to enzymes, pancreatic juice contains bicarbonates, the concentration of which depends on the hormonal effects of secretin. Vagus nerve stimulation affects the release of vasoactive intestinal peptide, which is a secretin

agonist. Exocrine pancreatic function is influenced by neuropeptides such as somatostatin, pancreatic polypeptide, peptide YY, neuropeptide Y, enkephalin, pancreastatin, as well as calcitonin-related gene peptides, glucagon and galanin. Nitric oxide is also an important neurotransmitter for the pancreas. Bicarbonate is an ion essential for pancreatic secretion.

The secretion of the main enzymes in an inactive form, intracellular calcium homeostasis, acid-base balance, as well as the secretion of a whole group of protease inhibitor enzymes - pancreatic trypsin secretion inhibitor (PSTI), which can inactivate up to 20 % of intracellular trypsin activity, saves the pancreas from self-cleavage. These proteases are found in acinus cells, in pancreatic secretions, and in plasma alpha-1 and alpha-2 globulins. Loss of any of these four mechanisms leads to the primary activation of enzymes, self-digestion of the pancreas and the development of acute pancreatitis.

Complaints and anamnesis of patients with diseases of the pancreas

The etiology of pancreatic diseases is very diverse. The most common disease is chronic pancreatitis. Pathogenetically allocate primary and secondary pancreatitis.

The main complaints of patients with diseases of the pancreas:

1. Pain in the upper abdomen associated with a gross violation of the diet (a lot of fatty, fried foods), alcohol consumption. Sometimes the pain is preceded by an attack of biliary colic or abdominal trauma. «Pancreatic pain syndrome» is characterized by an episodic onset, localization in the epigastrium, irradiation to the back, spine, the pain spreads to the left hypochondrium, the left costovertebral angle in the form of a half-belt, less often to the region of the heart and

left collarbone, girdle pain is also possible (fig. 90). The nature of pain in the pathology of the pancreas is often pressing, boring, burning. Pain is not stopped by eating, antacids, thermal procedures. Abundant food, alcohol provoke the appearance or intensification of pain. The cold brings relief. Pain is rare in the morning, more often in the afternoon. The pain is aggravated in the prone position and decreases in the knee-elbow position or sitting with an inclination forward. Sometimes the pains are of a crisis nature, last up to 3−5 hours, accompanied by repeated vomiting that does not bring relief, sometimes − loose stools and collapse (Zimmerman Y.S., 1992).

2. Vomiting that does not bring relief, discomfort in the abdomen, heaviness in the epigastrium, constipation and diarrhea − all these manifestations together are called pancreatic dyspepsia syndrome.

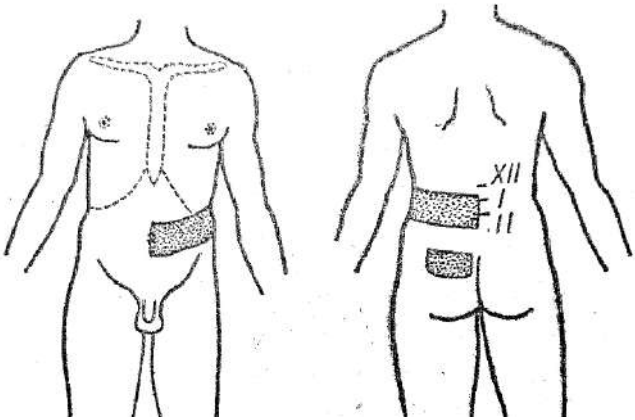


Figure 90. Areas of typical distribution of pain in chronic pancreatitis

3. Poor tolerance of a large assortment of dishes − meat, fish, dairy, fried − in the presence of appetite.

4. Sitophobia syndrome − fear of food due to the fear of developing abdominal pain and dyspepsia.

5. Loss of body weight during illness.

6. «Pancreatic stool» − a violation of absorption in the small intestine leads to the development of malabsorption and maldigestion syndromes, which is manifested by the appearance of «pancreatic stool» − mushy, sticky, brilliantly opalescent, abundant, having a sharp unpleasant odor, grayish color, poorly washed off with water from toilet walls.

The main features of the anamnesis of patients with diseases of the pancreas, which have a pathogenetic basis:

- Alcohol consumption. When questioning patients, it is necessary to ask a question about the use of alcohol, both single and systemic. Alcohol consumption is a leading factor in primary chronic pancreatitis.

- Overeating and frequent consumption of fatty foods.

- There was a high percentage of chronic pancreatitis in people with obesity and dyslipidemia types I, IV, V.

- Hereditary predisposition to the development of chronic pancreatitis. Separately, there is a variant of familial pancreatitis with an autosomal dominant path of development.

- Congenital malformations of the pancreas, especially the ductal system. Various congenital variants of gland aplasia are possible, for example, Shwachman's syndrome, which has an autosomal recessive inheritance path. What matters is the violation of the metabolism of individual amino acids − lysine, cysteine.

- Presence of various vascular diseases − periarteritis nodosa and other systemic vasculitis, arterial hypertension, atherosclerosis of the abdominal aorta and vessels extending from it.

- The use of certain drugs contributes to the development of chronic pancreatitis. For example − steroid pancreatitis, pancreatitis against the background of long-term estrogen intake.

- The use of the following drugs is definitely associated with the development of chronic pancreatitis: azathioprine, hypothiazide, 6-mercaptopurine, methyldopa, estrogens, sulfonamides (sulfasalazine, etc.), tetracycline, furosemide.

- The presence of the use of the following drugs is probably associated with the development of chronic pancreatitis: corticosteroids, cimetidine, ethacrynic acid, metronidazole, piroxicam.

- The possible causes of chronic pancreatitis are the use of beta-blockers, cholestyramine, colchicine, indomethacin, ibuprofen, paracetamol, rifampicin, phenolphthalein, salicylates.

- The presence of the first (I) blood group is more common in patients with chronic pancreatitis.

- For the development of secondary pancreatitis, diseases of the liver and biliary tract are of the greatest importance − calculous and acalculous cholecystitis, anomalies in the development of the bile ducts, postcholecystectomy syndrome, cirrhosis of the liver.

- The presence of parasites in the body, primarily affecting the hepatobiliary system, and secondarily − the pancreas − opisthorchiasis, echinococcosis. Cases of obstruction of the pancreatic ducts by roundworms with subsequent deposition of calcium salts are described.

- The presence of diseases of the duodenum − peptic ulcer, duodenitis, etc.

- Hemochromatosis is a hereditary disease in which chronic hepatitis, cirrhosis of the liver develop first, then chronic pancreatitis with diabetes mellitus, called «bronze diabetes» due to the specific color of the skin (accumulation of pigments containing iron).

- Cystic fibrosis is a chronic hereditary disease that leads to cystic fibrosis and obstruction of the pancreatic ducts, intestinal glands and lungs.

- Pathology of the parathyroid glands − hyperparathyroidism can be complicated by the development of chronic pancreatitis with the formation of calcifications in the pancreas, and sometimes stones in the pancreatic ducts.

- A history of mumps is very often associated with the development of chronic pancreatitis.

CHAPTER 12.
PHYSICAL EXAMINATION OF PATIENTS WITH PANCREATIC PATHOLOGY

Visual inspection

When examining the skin on the abdomen, on the back, chest, and less often on the skin of the upper and lower extremities, clear, bright red small convex spots are found – «blood dew spots» or «red droplets» (symptom of Tuzhilin S.A.), the origin of which is associated with the presence of chronic pancreatitis. In the projection area of the pancreas on the anterior abdominal wall, atrophy of the subcutaneous fat (Grott's symptom) can be determined.

With compression of the head of the pancreatic and bile ducts, the head of the pancreas by a tumor, or sclerosis of the ducts, an obstructive inflammatory process, icteric coloration of the skin and mucous membranes of the soft palate and sclera occurs. In such cases, it is first necessary to exclude cancer of the head of the pancreas.

With a long course of chronic pancreatitis, patients noticeably lose weight, the thickness of the subcutaneous fat decreases, the skin becomes dry and loses elasticity. It is possible that the skin will acquire a dirty gray tint, various pigmentation will occur. When examining the oral cavity, it is likely to detect smoothness, atrophy of the papillae of the tongue, ulceration in the corners of the mouth (cheilitis), aphthous stomatitis, and bad breath.

In diseases of the pancreas, the abdomen is often swollen due to flatulence. Flatulence is caused by exocrine pancreatic insufficiency, the development of secondary intestinal dysbacteriosis, less often by intestinal paresis.

Of great diagnostic importance is the determination of pain points and zones specific for diseases of the pancreas.

Pain in the Chauffard zone − pain on superficial palpation in the epigastrium and right hypochondrium, medially from the bisector dividing the right angle between the midline of the abdomen and the line drawn perpendicular to the midline of the abdomen through the navel (fig. 91).

Soreness at the Desjardins point − is determined on the initial segment of the line drawn from the navel to the right armpit at the border of the middle and upper third of the distance between the navel and the right costal arch. Corresponds to the head of the pancreas.

Soreness in the Gubergrits-Skulsky zone − is determined in the epigastrium to the left of the midline symmetrically to the Chauffard zone − corresponds to the body of the pancreas.

Soreness at the Gubergrits point − is determined on the initial segment of the line drawn from the navel to the left armpit, 5−6 cm above the navel − corresponds to the body of the pancreas.

Soreness in the Mayo−Robson zone − is determined in the left costovertebral angle, corresponds to the lesion of the tail of the pancreas.

Determination of zones of skin hyperesthesia-hyperanalgesia of Zakharyin−Ged on the anterior abdominal wall is carried out with superficial palpation of the area of the thoracic calving of the spine at the level of VIII−X vertebrae (fig. 92). It is possible to detect pulsation in the region of the first vertebrae of the lumbar spine due to the transmission pulsation of the abdominal aorta in chronic pancreatitis

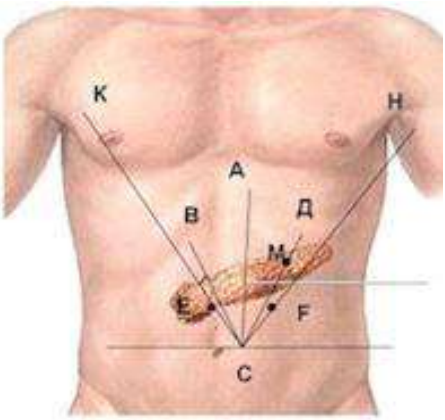


**Figure 91. Triangle ABC − Chauffard zone;
triangle ADC – Gubergritsa−Skulsky zone; point E − Desjardin's point,
located 6 cm above the navel along the SC line; point F − point Gubergritsa,
located 6 cm above the navel along the CH line;
point M − Mayo−Robson point, located on the border of the outer and middle
third of the line connecting the navel with the middle of the left costal arch**

Palpation of the pancreas

A healthy pancreas is very rarely palpated. Fibrosed, calcified pancreas is palpated in 50 % of cases, mainly in lean people − in the form of a transversely located dense painful cord 2−3 cm wide. For palpation of the pancreas, the Grott method, the Shelagurov method, the Malle−Guy method are used.

Method of deep palpation of the pancreas according to I.W. Grott

Palpation is carried out in three positions of the patient's body − lying on his back, standing and lying on his right side. In the first position − the patient lies on his back, placing both hands under the lumbar spine, alternately the right and left hands must

be clenched into a fist. The doctor stands to the right of the patient.

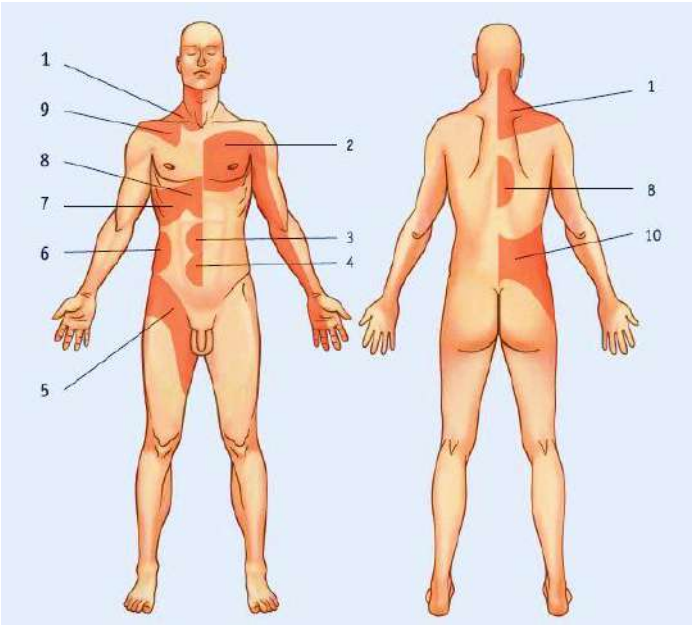


Figure 92. Zakharyin–Ged zones on the surface of the body, where 1 – lungs and bronchi, 2 – heart, 3 – intestines, 4 – bladder, 5 – ureters, 6 – kidneys, 7 and 9 – liver, 8 – stomach and pancreas gland, 10 – genitourinary system

With half-bent fingers of both hands, on exhalation, it plunges into the abdominal cavity between the navel and the left hypochondrium. In the second position, the patient is standing, slightly leaning forward and to the left. The doctor stands to the right of the patient. With the right hand, while the patient is exhaling, the doctor penetrates deep into the patient's abdominal cavity between the navel and the left hypochondrium. In the third position, the patient lies on his right side, his legs are slightly bent and brought to the body. The doctor with his right hand during

exhalation conducts an immersion in the abdominal cavity of the patient at a level between the navel and the left hypochondrium.

The method of deep palpation of the pancreas according to A.A. Shelagurov

The patient lies on his back. The doctor's left hand is located under the patient's back, under the left half of the lumbar region below the edge of the ribs. The doctor's right hand is placed in the projection of the pancreas between the navel and the left hypochondrium. The patient is asked to breathe with the involvement of the abdominal muscles.

On inhalation, a superficial skin fold is made with the right hand from the bottom up (according to the method of V.P. Obraztsov), and on exhalation, with half-bent fingers of the right hand, the doctor plunges deep into the abdominal cavity towards the posterior abdominal wall, and at the beginning of inhalation makes a sliding movement from the bottom up, trying feel the pancreas with your fingertips. Then, using this method, palpation of the pancreas is performed in the position of the patient lying on his right side with legs half-bent at the knees. Then, similarly, deep palpation of the pancreas is performed in a standing position.

The method of deep palpation of the pancreas according to Mallet-Guy (P. Mallet-Guy)

The patient lies on the right side, tilting the body at 45º, the left leg lies on the right leg, slightly bent at the knee. The doctor puts his right hand on the edge of the left costal arch, fixes it, and palpates the pancreas with his left hand along the line between the navel and the left costal arch during the exhalation of the patient.

The methods of Grott, Shelagurov and Malle-Guy make it possible to palpate the fibrous pancreas in the region of its body and tail. For palpation of the head of the pancreas, all these methods are also acceptable, only it is necessary to carry them out in the mirror direction − in the right hypochondrium, in the

Chauffard zone, and the patient should be examined, including on the left side.

In order to the enlarged and compacted pancreas to become more accessible for palpation, it is better to carry out the procedure on an empty stomach, according to the recommendations of earlier sources − on the eve of the evening, the patient was washed out the stomach and did a cleansing enema.

CHAPTER 13.
FEATURES OF THE DIAGNOSIS OF CERTAIN DISEASES OF THE PANCREAS

Acute and chronic pancreatitis are the two most common diseases of the pancreas. It is important to note that chronic pancreatitis very often develops in patients who have had acute pancreatitis.

Acute pancreatitis is an aseptic inflammation, which is based on necrosis of pancreatic acinar cells and enzyme aggression, followed by expanding necrosis and dystrophy of the gland, in which it is possible to damage surrounding tissues and distant organs, as well as systems and the addition of a secondary purulent infection. Acute pancreatitis is the most common diagnosis in patients with abdominal pain in the emergency department of a multidisciplinary hospital. There are many known causes of acute pancreatitis. In the US and Europe, 80−90 % of cases of acute pancreatitis are caused by gallstones and alcohol use. It has been proven that the risk of developing acute pancreatitis in individuals with gallstones smaller than 5 mm is significantly higher than in individuals with large stones. Hypertriglyceridemia with triglyceride levels above 1000 mg/dl is the cause of acute pancreatitis in 1.3−3.8 % of cases. Patients with diabetes mellitus complicated by ketoacidosis and patients taking oral contraceptives account for 0.1−2 % of the cohort of all patients with acute pancreatitis. The main reasons for the development of acute pancreatitis are presented in tab. 16. At the same time, rare and hidden diseases of the biliary tract, especially microlithiasis, biliary sludge, can be the causes of frequent recurrences of acute pancreatitis; side effects and drug interactions; alcohol addiction; metabolic causes: hypercalcemia, hypertriglyceridemia; anatomical causes − pancreas divisum, pancreatic cancer; intraductal papillary mucinous neoplasm; hereditary pancreatitis; cystic fibrosis; autoimmune pancreatitis; idiopathic pancreatitis.

The leading role in the pathogenesis of acute pancreatitis belongs to toxemia, which develops as a result of exposure to pancreatic enzymes − trypsin, lipase, phospholipase-A2, lysosomal enzymes.

Table 16

The main causes of acute pancreatitis

Cause group	Varieties
Most Common Causes	Gallstones, including microlithiasis; Alcohol (both acute and chronic alcoholism); Hypertriglyceridemia; Endoscopic retrograde cholangiopancreatigraphy (ERCP), especially after biliary manometry; Drugs: azathioprine, 6-mercaptopurine, sulfonamides, etrogens, tetracycline, valproic acid, ART drugs for the treatment of HIV infection, 5-ainosalicylic acid; Trauma (especially blunt abdominal trauma); Condition after any abdominal surgery
Infrequent causes	Vascular causes and vasculitis (ischemia, condition after cardiac surgery); Connective tissue diseases and thrombocytopenic purpura; Pancreas cancer; hypercalcemia; Periampullary diverticulum; Doubling of the pancreas (pancreas divisum); Hereditary pancreatitis; cystic fibrosis; kidney failure; Infectious diseases (mumps, Coxsackie virus, cytomegalovirus, echovirus, parasites); Autoimmune causes (type 1 and type 2)

The main symptom of acute pancreatitis is abdominal pain. Pain can vary in intensity from mild discomfort to intolerable acute conditions. It is usually localized in the epigastrium and umbilical region and can radiate to the back, chest, both sides, and also to the lower abdomen. Nausea, vomiting, and bloating due to increased peristalsis of the stomach and intestines and the development of chemical peritonitis often accompany acute pancreatitis. On physical examination, the patient is often agitated and stressed. Perhaps the appearance of subfebrile fever, tachycardia, hypotension. In some cases, the patient may be in shock due to hypovolemia and the development of retroperitoneal exudation of whole blood or blood plasma; increasing the formation of renal peptides that cause vasodilation and vascular permeability; and also due to the systemic effects of proteolytic and lipolytic enzymes, which enter the bloodstream in large quantities.

Occasionally, jaundice may appear, which indicates swelling of the pancreatic head, which compresses the bile duct, interfering with the passage of bile into the duodenum. Still, jaundice is more often manifested during the passage of gallstones or sludge through the bile ducts, which is less associated with the pancreas. Rarely, erythematous skin nodules may appear due to necrosis of subcutaneous fat. In 10−20 % of patients, moist rales are found in the lower parts of the lungs, atelectasis and hydrothorax, mainly on the left side. Heaviness in the abdomen and rigidity of the muscles of the anterior abdominal wall are present in varying degrees of severity, but are always associated with the intensity of the pain syndrome. Intestinal peristalsis is usually reduced or not identified. The pancreas significantly increases in size due to the acute accumulation of fluid in it, necrosis of the walls, and possibly the formation of pseudocysts, which can be palpable from 4−6 weeks of the disease in the projection of the pancreas.

Acute pancreatitis is characterized by Cullen's symptom − cyanosis around the navel, which indicates hemoperitoneum, as well as Turner's symptom − blue-red tuberosity or green-brown spots on both flanks of the abdomen, indicating the breakdown of hemoglobin in necrotizing pancreatitis with bleeding. It is necessary to confirm the diagnosis of acute pancreatitis on computed tomography (CT) with clarification of its morphological variant. The main morphological variants of acute pancreatitis are presented in tab. 17.

Table 17

Main morphological variants of acute pancreatitis

Morphological variant	Specification
Acute edematous pancreatitis	Acute inflammation of the parenchyma of the gland and peripancreatic tissue, but without the formation of necrosis
Acute necrotizing pancreatitis	Diffuse or focal areas of non-viable pancreatic parenchyma, which, as a rule, are combined with necrosis of retroperitoneal fatty tissue
Sterile pancreatic necrosis	The appearance of pancreatic necrosis, which does not contain pathogenic microflora and is not accompanied by the development of purulent complications
Acute peripancreatic fluid collection	Acute peripancreatic fluid accumulation without signs of necrosis of the pancreatic parenchyma and retroperitoneal fatty tissue in the first 4 weeks of the disease, without a clear delineation

Ending of table 17

Morphological variant	Specification
Acute pancreatitis with pseudocyst formation	Fluid accumulation (with or without sequesters) delimited by fibrous or granulation tissue following an attack of acute pancreatitis. Occurs after 4 weeks from the onset of the disease, in the phase of aseptic sequestration of necrotizing pancreatitis. As a rule, it is the outcome of an infiltrate. The contents of the cyst may be aseptic and infected. Bacterial contamination of the contents of the cyst often does not have a clinical manifestation, but the likelihood of infection is always higher in the presence of sequesters
Acute necrotic accumulation	An accumulation containing varying amounts of both fluid and necrotic tissue in the pancreatic parenchyma and/or retroperitoneal adipose tissue in the first 4 weeks of the disease, without a clear demarcation
Acute pancreatitis with demarcated foci of necrosis	Foci of necrosis are encapsulated, formed against the background of intense inflammation of the pancreatic wall, appear after the fourth week of acute pancreatitis
Infected pancreatic necrosis	Necrosis of pancreatic tissue and retroperitoneal tissue seeded with bacteria with their purulent fusion and sequestration

On examination: the patient's abdomen is involved in breathing, swollen; the skin and mucous membranes are often pale, there may be jaundice and icterus of the sclera, cyanosis of the face and extremities (purple spots on the face − a symptom of Mondor, spots of cyanosis on the side walls of the abdomen − a

symptom of Gray−Turner, cyanosis of the umbilical region − a symptom of Grunwald). In the later stages of the disease, cyanosis can be replaced by a bright hyperemia of the skin, especially the upper half of the body. Tongue dry, lined. On palpation, pain in the epigastric region is determined, rarely in combination with tension in the muscles of the anterior abdominal wall and a positive Shchetkin−Blumberg symptom; symptoms of Voskresensky (lack of pulsation of the abdominal aorta due to an increase in the size of the pancreas and swelling of the retroperitoneal tissue), Mayo-Robson (severe pain on palpation of the lumbar region, especially the left costovertebral angle), Kerte (transverse painful resistance of the anterior abdominal wall in projection of the pancreas). When percussion is observed: severe tympanitis due to paresis of the transverse colon; dullness in sloping places of the abdomen in the presence of effusion in the abdominal cavity. During auscultation, there is a weakening or absence of intestinal noises as a manifestation of dynamic intestinal obstruction.

Laboratory examination necessarily includes a general blood test and a general urinalysis, a biochemical blood test with the determination of total protein, bilirubin, aminotransferases, urea, creatinine, glucose, amylase. In case of moderate and severe disease, the necessary studies are a coagulogram, as well as the determination of C-reactive protein and lipase.

The diagnosis of acute pancreatitis is established in the presence of clinical signs of the disease obtained during the collection of anamnesis and physical examination, as well as laboratory tests and instrumental confirmation using modern imaging methods − ultrasound, computed tomography, magnetic resonance imaging, positron emission tomography.

According to the severity, acute pancreatitis of mild, moderate and severe degree is distinguished. A mild degree is established in the absence of pancreatic necrosis and organ

failure. Moderate severity is characterized by the manifestation of one of the signs: acute peripancreatic fluid accumulation, acute necrotic accumulation, peripancreatic infiltrate, pseudocyst, limited necrosis and / or the development of general manifestations in the form of transient organ failure for no more than 48 hours. A severe degree is established in the presence of infected pancreatic necrosis and / or the development of persistent organ failure for more than 48 hours from the onset of the disease.

All patients are recommended to undergo an ultrasound examination (fig. 93) of the abdominal organs, and if the diagnosis is unclear and differential diagnosis with other diseases, spiral computed tomography (SCT) or magnetic resonance imaging (MRI) is necessary. The morphological variant of acute pancreatitis can be clarified only with the help of SCT and MRI. The presence of peritoneal syndrome, including the presence of ultrasound signs of fluid in the abdominal cavity (fig. 94), as well as the need for differential diagnosis with other diseases, it is necessary to perform laparoscopy and percutaneous drainage of the abdominal cavity under ultrasound control or laparocentesis. Therefore, patients with acute pancreatitis should be observed and treated in a surgical department.

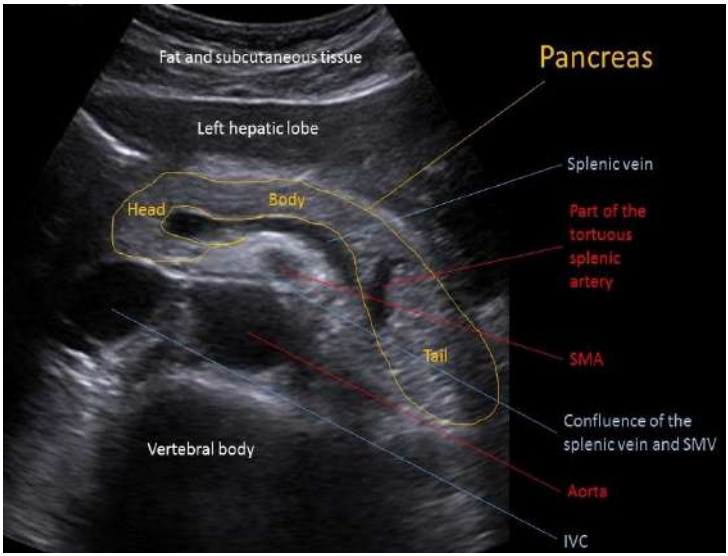


Figure 93. Ultrasound topography of normal pancrease

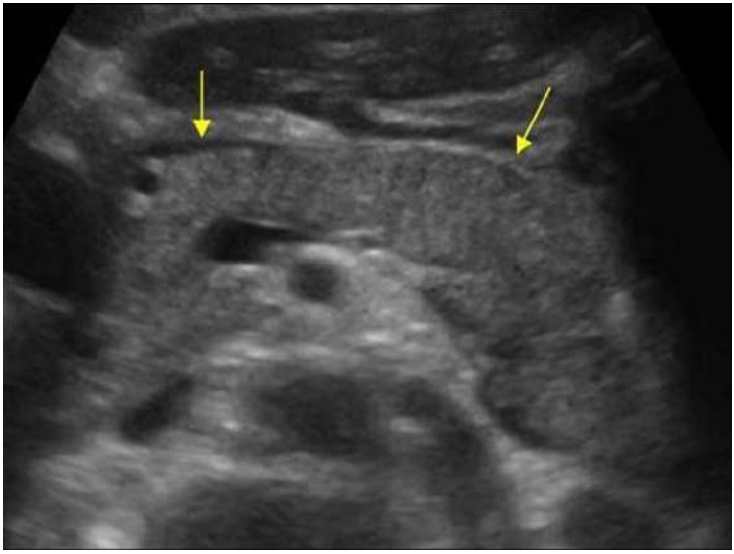


Figure 94. Acute pancreatitis with peripancreatic fluid (yellow arrows)

Chronic pancreatitis is a long-term inflammatory disease of the pancreas, manifested by irreversible morphological changes that cause pain and/or a permanent decrease in function. Mortality after the initial diagnosis is up to 20 % during the first 10 years and more than 50 % after 20 years, with an average of 11.9 %.

Depending on the etiological factor, the following variants of chronic pancreatitis are distinguished:

- toxic − associated with alcoholism, smoking, hypercalcemia, hyperparathyroidism, chronic renal failure, exposure to drugs and toxins;

- idiopathic − variants with early pain syndrome, late pain syndrome, rapid development of pancreatic calcification, exo- and endocrine insufficiency, as well as tropical pancreatitis (fibrocalculous pancreatic diabetes, tropical calcific pancreatitis);

- hereditary forms of chronic pancreatitis (autosomal dominant, cationic trypsinogen, autosomal recessive, CTFR gene mutations; SPINK1 gene mutations, cationic trypsinogen mutations, alpha-1-antitrypsin variant;

- autoimmune chronic pancreatitis − an isolated variant, as well as a variant associated with other autoimmune diseases;

- chronic pancreatitis, as a result of recurrent and severe acute pancreatitis − postnecrotic, severe acute pancreatitis, recurrent acute pancreatitis, pancreatitis due to vascular diseases and / or pancreatic ischemia, as well as radiation pancreatitis;

- obstructive chronic pancreatitis − stenosis of the sphincter of Oddi; duct obstruction, post-traumatic scars of the pancreatic ducts and pancreas divisum.

Autoimmune chronic pancreatitis can occur alone or in association with Sjögren's syndrome, inflammatory bowel disease, and other autoimmune diseases. There are two types − I and II. Type I is characterized by manifesting IgG4-associated damage to the pancreas involving other target organs, being a systemic autoimmune disease with a specifically altered immune response.

Type II is a distinct disease of the pancreas characterized by granulocytic epithelial damage and occasional coexistence with ulcerative colitis. Clinically relevant serum parameters for diagnosing autoimmune pancreatitis include hypergammaglobulinemia, increased serum IgG and IgG4 levels, and the presence of autoantibodies (antinuclear, lactoferrin, carbonic anhydrase II, and smooth muscle).

The main classification of chronic pancreatitis today includes the following items:

1. By etiology: biliary-dependent; alcoholic; dysmetabolic; infectious; drug; autoimmune; idiopathic.

2. According to clinical manifestations: pain, dyspeptic; combined; latent.

3. According to morphological features: interstitial-edematous, parenchymal, fibrous-sclerotic, hyperplastic, cystic.

4. By the nature of the clinical course: rarely recurrent, often recurrent, with constantly present symptoms.

5. Complications: violations of the outflow of bile, portal hypertension (subhepatic), endocrine disorders (pancreatogenic diabetes mellitus, hypoglycemic conditions, etc.), inflammatory changes (abscess, cyst, parapancreatitis, «enzymatic» cholecystitis, pneumonia, exudative pleurisy, paranephritis, etc.).

The clinical picture of chronic pancreatitis is most often characterized by the development of recurrent abdominal pain, but it can also manifest as a clinical picture of complications of the disease. Abdominal pain in chronic pancreatitis is often localized in the epigastrium with irradiation to the back, increases after eating, decreases in a sitting position or leaning forward. Pain can appear and disappear (type A) or continue continuously for up to several days (type B) − more often with alcoholic chronic pancreatitis.

Insufficiency of exocrine secretory function can manifest itself clinically with a decrease in the function of the gland by

more than 90 % − in the form of steatorrhea, flatulence, weight loss, malabsorption of fat-soluble vitamins and vitamin B_{12}. In 70 % of patients with chronic pancreatitis, impaired glucose tolerance develops on average 10 years after the onset of the disease. Pancreatogenic diabetes mellitus (Type 3C diabetes mellitus) develops rapidly, often complicated by ketoacidosis,

Depending on the clinical picture, the stages of chronic pancreatitis are determined.

Stage I − preclinical, established according to ultrasound diagnostics, while there are no clinical manifestations;

Stage II − initial manifestations − is characterized by the development of pain type «A» − repeated attacks that reduce the quality of life. The duration of this stage is 4−7 years, there is a recurrence of pancreatic necrosis of complications of pancreatitis.

Stage III − persistent symptoms, characterized by the development of type «B» pain, endo- and exocrine insufficiency, and even trophological paedosufficiency.

Stage IV − atrophy of the pancreas − is characterized by severe insufficiency of the gland, a decrease in the intensity of the pain syndrome, and sometimes the pain syndrome stops altogether. Diabetes mellitus, trophological insufficiency often appear. There is a high risk of pancreatic cancer.

Complications of chronic pancreatitis − the formation of pseudocysts at the site of tissue necrosis due to destruction of the ducts by the gland. Edema and fibrosis of the pancreatic head can cause compression of the common bile duct with the formation of jaundice. Inflammation and fibrosis of the parapancreatic tissue can lead to compression and thrombosis of the splenic, superior mesenteric and portal veins, however, a detailed picture of subhepatic portal hypertension is extremely rare.

Perhaps the development of erosive esophagitis, Mallory-Weiss syndrome, gastric and duodenal ulcers, which is due to a long and significant decrease in bicarbonate production in the

gland. Less common is chronic duodenal obstruction and abdominal ischemic syndrome. Exacerbation of chronic pancreatitis can be complicated by pancreatic necrosis with the development of bacterial complications − inflammatory infiltrates, abscesses, purulent cholangitis, sepsis. Often, in persons with chronic pancreatitis, during a significant progression of the disease, ductal adenocardcinoma of the gland develops. A common complication due to progressive fibrosis and maldigestion is the formation of severe malabsorption with the formation of micronutrient deficiency, primarily fat-soluble vitamins A, D, E, K, and vitamin B_{12}. Due to severe malabsorption, anemia of chronic disease, osteoporosis develop.

In chronic pancreatitis, unlike acute pancreatitis, there is rarely an increase in the level of enzymes in the blood or urine, so if this occurs, the formation of a pseudocyst or pancreatic ascites can be suspected. A persistently elevated level of amylase in the blood suggests macroamylasemia (in this case, amylase forms large complexes with plasma proteins that are not filtered by the kidneys, and normal amylase activity is observed in the urine) or the presence of extrapancreatic sources of hyperamylasemia (chronic renal failure; diseases of the salivary glands − epidemic mumps, stones, radiation sialadenitis); complications of maxillofacial surgery; «cancerous» hyperamylasemia (lung cancer, oac of the esophagus, ovarian cancer); true macroamylase; burns; diabetic ketoacidosis; pregnancy; kidney transplant; brain injury; morphine use; cholecystitis, choledocholithiasis, perforation or penetration of ulcers; bowel obstruction or infarction; ectopic pregnancy; peritonitis, aortic aneurysm; postoperative hyperamylasemia). With high exocrine pancreatic insufficiency, the determination of elastase-1 in feces has a diagnostic value for clarifying the diagnosis of chronic pancreatitis. Decreased fecal elastase-1 levels are indicative of primary exocrine insufficiency (0−100 ccrg severe,

101−200 moderate to mild), which is an indication for lifelong high-dose pancreatic enzyme replacement therapy. The diagnosis of endocrine insufficiency is confirmed by determining the level of glycated hemoglobin.

The diagnosis must be confirmed by an imaging diagnostic method − ultrasound of the pancreas (the diagnosis is confirmed by calcification of the gland, intraductal calcium calculi, pseudocysts, dilatation of the duct and its side branches, parenchyma atrophy) (fig. 95). However, transabdominal ultrasound is not effective for detecting the disease in the early stages and is significantly inferior to MSCT and endoscopic ultrasound. MSCT is the method of choice for the primary diagnosis of chronic pancreatitis and its complications. MRI is the most effective method for detecting calculi and tumors of the gland.

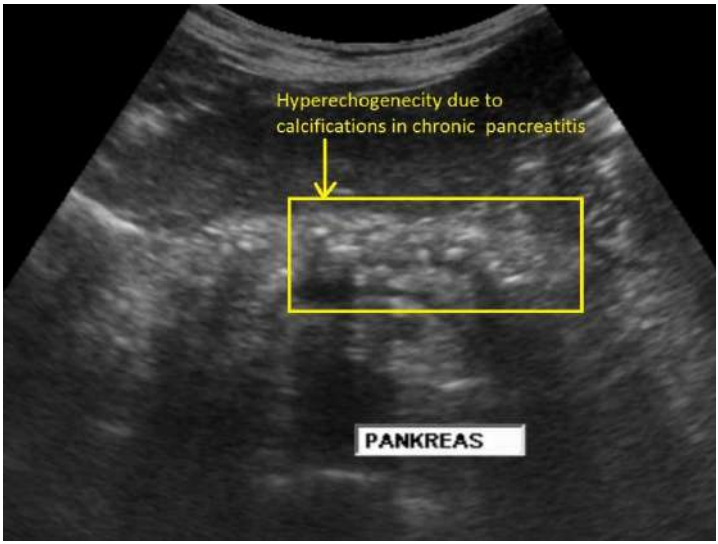


Figure 95. One of the samples of chronic pancreatitis in ultrasound examination

Doubling of the pancreas (pancreas divisum) − manifests itself in 7−10 % of the population and is formed in embryogenesis. This is the most common congenital anomaly of the pancreas, when initially two pancreas are determined in a person, often having one common duct, or separate ducts in the duodenum. Often one gland is much larger than the other (fig. 96). The presence of an anomaly does not imply the unambiguous development of pancreatitis, however, the combination of pancreas divisum and a narrow opening of the pancreatic sphincter or narrow Wirsung duct significantly increases the likelihood of developing chronic pancreatitis.

Patients with pancreas divisum need dynamic monitoring of pancreatic function throughout their lives. Their risk of developing chronic pancreatitis is much higher than that of other people. Every patient with pancreas divisum needs to undergo ultrasound of abdomen atleast once a year. (fig. 97).

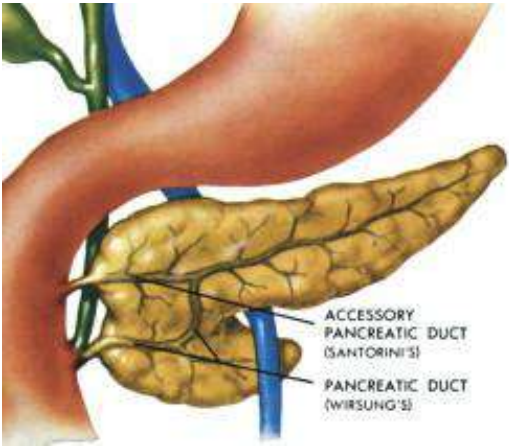


Figure 96. Variant of the formed pancreas divisum

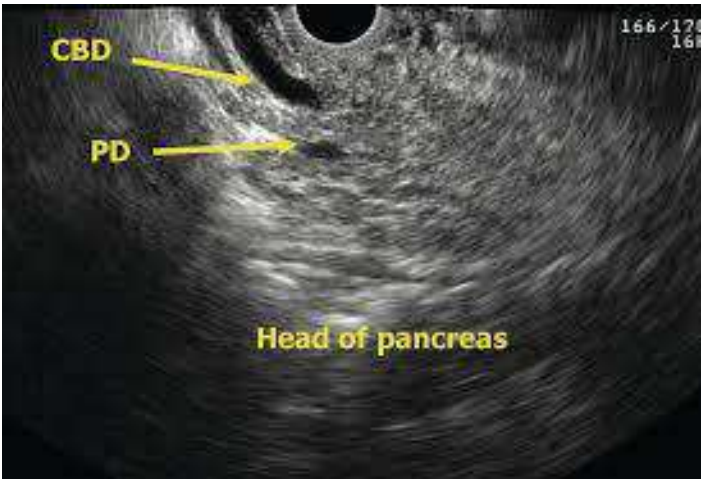


Figure 97. Pancreas Divisum (PD) in ultrasound picture

MCQ TESTS

(single correct answer for each question)

1. THE FUNCTIONAL UNIT OF THE LIVER IS

1) hepatic lobule
2) hepatocyte
3) alveolocyte
4) bile ducts

2. FOR LIVER DISEASES, ALL THE FOLLOWING COMPLAINTS ARE CHARACTERISTIC, EXCEPT

1) asthenic complaints − weakness, malaise, poor appetite, fatigue, irritability, headaches, decreased potency and libido, menstrual irregularities
2) intense itching of the skin, aggravated at night and depriving the patient of normal sleep
3) the appearance of protein in the urine
4) belching, nausea and vomiting, often provoked by the intake of fatty or fried foods

3. PAIN IN DISEASES OF THE LIVER AND BILIARY TRACT MAY OCCUR IN ALL OF THE FOLLOWING CIRCUMSTANCES EXCEPT

1) inflammation of the peritoneum covering the liver (perihepatitis) or gallbladder (pericholecystitis)
2) with a rapid and significant increase in the liver, leading to stretching of its peritoneal cover − the Glisson capsule
3) with spastic contractions of the muscles

of the gallbladder and large bile ducts
4) when cysts or tumors appear in the liver

4. THE MAIN DIFFERENCES BETWEEN DYSKINETIC PAIN AND PAIN IN BILIARY COLIC AND CHOLECYSTITIS

1) pain associated with food intake and its quantity
2) last for hours and even days
3) dyskinetic pains are accompanied by chills, fever and other signs of inflammation (leukocytosis, accelerated ESR, increased concentration of C-reactive protein, etc.)
4) at the end of an attack of dyskinetic pain, the patient does not feel any spontaneous or palpatory pain in the liver and gallbladder

5. BILIARY (OR HEPATIC) COLIC IS

1) an attack of sharp pain behind the sternum, provoked by stress or physical exertion, stopped by taking nitrates
2) an attack of very intense pain in the right hypochondrium, which can be triggered by the itake of abundant fatty or spicy foods, alcohol, shaky driving, emotional experiences
3) an attack of very intense, sharp pains in the lumbar region, radiating to the groin, provoked by a bumpy ride
4) an attack of intense pain in the abdomen, accompanied by positive symptoms of peritoneal irritation

6. THE CAUSE OF POSTHEPATIC JAUNDICE CAN BE

1) hemolytic anemia

2) active phase of chronic viral hepatitis C
3) tumor of the head of the pancreas
4) alcoholic liver disease

7. THE NATURE OF PAIN IN THE PATHOLOGY OF THE PANCREAS IS MORE OFTEN

1) pressing, boring, burning, pains are not stopped by eating, antacids, thermal procedures
2) abundant food and alcohol do not provoke the appearance or intensification of pain
3) more often pains appear in the morning
4) pain is relieved by lying down

8. CHOLECYSTOCARDIAL SYNDROME CAN OCCUR WHEN

1) alcoholic liver disease
2) cholelithiasis
3) acute coronary syndrome
4) renal colic

9. ON THE ECG AT THE TIME OF BILIARY COLIC, THE FOLLOWING CHANGES CAN BE DETECTED

1) displacement of the ST interval below the isoline, a decrease in the voltage of the teeth and negative T waves mainly in the chest leads, high P waves in II, III, avf leads
2) formation of S-type ECG
3) the appearance of a monophasic curve – ST elevation of more than 2 mm in the chest leads
4) the appearance of atrioventricular blockade of the 1st degree, often in combination

with the blockade of the right leg
of the bundle of His

10. MANIFESTATIONS OF EDEMATOUS-ASCITIC SYNDROME ARE ASSOCIATED WITH

1) with the synthesis of blood coagulation factors and a sharp increase in the consumption of existing coagulation factors, which leads to widespread thrombosis and even to disseminated intravascular coagulation (DIC) and bleeding
2) with a decrease in the synthesis of albumin in the liver and a drop in oncotic pressure; in the future, aldosterone inactivation is disrupted, which leads to secondary hyperaldosteronism with the development of hypernatremia and hypokalemia
3) with a deterioration in appetite, the development of nausea, intolerance to fatty and protein foods, the appearance of abdominal pain, bloating, unstable stools, weakness, emaciation, skin changes (its dryness, thinning, wrinkling), the development of neuritis, anemia
4) with the occurrence of obstruction of the bile ducts

11. LIVER DISEASES, IN WHICH HEPATO-RENAL SYNDROME MOST OFTEN DEVELOPS, INCLUDE ALL, EXCEPT

1) liver cirrhosis, especially alcoholic, in the presence of ascites and diuretic therapy, hepatic encephalopathy,

esophageal-gastric bleeding
2) acute viral hepatitis
3) cholelithiasis with obstruction of the bile ducts
4) hepatocellular carcinoma

12. TELANGIECTASIA IS
1) local excessive expansion of capillaries and small vessels
2) the appearance on the anterior abdominal wall of an excess of dilated, nodular-changed saphenous veins
3) scarlet heel, appearing on the entire surface of the skin
4) irreversible changes in the nail plates

13. «FIELDS» OF SPIDER VEINS ON THE SKIN OF THE COLLAR ZONE, INTENSE PALMAR ERYTHEMA, DUPUYTREN'S CONTRACTURE, THE FACE OF AN ALCOHOLIC (FACIES ALCOHOLICA), GYNECOMASTIA, GIANT PAROTITIS − ALL OF THESE SIGNS TOGETHER ARE OBSERVED WITH
1) chronic hepatitis and cirrhosis of any etiology
2) alcoholic hepatitis and liver cirrhosis
3) obstruction of the bile ducts
4) Wilson−Konovalov disease

14. INTENSE JAUNDICE, EDEMATOUS-ASCITIC SYNDROME, HEMORRHAGIC SYNDROME − BRUISES, PURPURA, «LIVER» BREATH, TREMOR OF THE HANDS, TREMOR OF THE TONGUE − ALL OF

THE ABOVE SIGNS TOGETHER ARE OBSERVED WHEN

1) cirrhosis of the liver with hepatocellular insufficiency
2) primary biliary cirrhosis
3) Wilson−Konovalov disease
4) chronic hepatitis and cirrhosis of any etiology

15. WHEN EXAMINING THE CORNEA WITH A SLIT LAMP, THE KAISER−FLEISCHER RING IS OBSERVED WITH

1) cirrhosis of the liver with hepatocellular insufficiency
2) primary biliary cirrhosis
3) Wilson−Konovalov disease
4) chronic hepatitis and cirrhosis of any etiology

16. WITH PERCUSSION OF THE SPLEEN ACCORDING TO THE METHOD OF M.G. KURLOV'S FINGER-PLESSIMETER IS INSTALLED

1) at the edge of the left costal arch perpendicular to the X rib
2) at the navel diagonally to the right costal arch
3) in the right hypochondrium along the mid-clavicular line
4) under the left shoulder blade

17. THE LENGTH AND DIAMETER OF THE SPLEEN ARE NORMALLY

1) 9−10 cm and 7−9 cm, respectively

2) 6−8 cm and 4−6 cm, respectively
3) 4−5 cm and 2−3 cm, respectively
4) 17−20 cm and 16−18 cm, respectively

18. THE FLUCTUATION METHOD, WHICH IS USED TO DETECT LARGE VOLUMES OF FLUID IN THE ABDOMINAL CAVITY, IS CARRIED OUT AS FOLLOWS
1) the finger-plessimeter is installed at the edge of the left costal arch perpendicular to the X rib. Percussion is carried out directly along the left costal arch in the direction from the sternum to the spine, marking the zones of dull percussion sound. And then in the opposite direction along the costal arch from the posterior axillary line to the sternum − we mark the second point of the beginning of a dull percussion sound
2) the doctor's palpating right hand lies flat on the right half of the patient's abdomen, the line formed by the ends of 2−5 fingers should be parallel to the supposed lower edge of the liver and be slightly below it. In this case, the doctor's fingertips are immersed deep into the abdomen in sync with the patient's breathing. When the patient exhales, the doctor's fingers sink into the abdominal cavity, while inhaling, the fingers slip out of the abdominal cavity, touching the descending lower edge of the liver from under which they slip
3) the palm of the left hand is firmly

applied to the lateral surface of the abdomen, and light jerky blows are applied with the right hand from the opposite side. If there is free fluid in the abdominal cavity, then these blows are felt with the left hand in the form of a wave

4) Percussion should begin from the right flank of the abdomen at a level obviously below the lower edge of the liver, presumably from the level of the navel. If there is already a dull sound there, then it is necessary to go down even lower, to the place where the tympanic sound is determined

19. ACUTE LIVER FAILURE IN THE TERMINAL STAGE MAY HAVE ALL OF THE LISTED COMPLICATIONS. BESIDES

1) edema-swelling of the brain, manifested by signs of irritation of the meninges, hyperemia, convulsive muscle twitches, symptoms of cranial nerve damage, progressive respiratory disorders, increased blood pressure, a tendency to bradycardia
2) gastrointestinal bleeding (including stress erosion and ulcers of the gastrointestinal tract, gastroenteritis with the addition of renal failure)
3) secondary infection: pneumonia, including aspiration, angiogenic and urethral sepsis
4) retinal detachment

20. ORTNER−GREKOV SYMPTOM IS

1) pain when tapping the ulnar side of the palm along the costal arch − a symptom of acute cholecystitis
2) soreness with light tapping with the fingertips in the right hypochondrium − a symptom of acute cholecystitis
3) pain on pressure in the interscapular region at the level of the IX−XI thoracic vertebrae, 3 cm lateral to the right – a symptom of acute cholecystitis
4) pain on palpation lateral to the right of the fourth thoracic vertebra is a symptom of chronic cholecystitis

21. HEPATARGIA MANIFESTS ITSELF

1) the presence of blood in the vomit, which often indicates bleeding from varicose veins of the esophagus, or the presence of erosive gastritis, erosive esophagitis
2) intense general weakness, irritability, headaches, persistent insomnia, or vice versa − drowsiness, anxiety, delirium, convulsions, and may result in hepatic coma (coma hepatica), during which the patient dies in an unconscious state
3) the appearance of fresh blood in the stool
4) the appearance of undigested dietary fiber in the feces

22. TRIGGER FACTORS FOR THE DEVELOPMENT OF CHRONIC PANCREATITIS INCLUDE
    1) history of acute pancreatitis
    2) chronic pyelonephritis
    3) chronic heart failure
    4) tumor of the parenchyma of the kidney

23. WHAT IS «CHOLESTEROSIS OF THE GALLBLADDER»
    1) this is the accumulation of lipids in the mucous membrane, and later in the submucosal and muscular layers of the gallbladder, these lipids consist of free and esterified cholesterol
    2) this is the germination of the hepatic lobules by connective tissue
    3) the appearance of atherosclerotic plaques in the hepatic arteries
    4) development of fibrosing processes in the wall of the gallbladder

24. THE DEVELOPMENT OF PORTAL HYPERTENSION IS ACCOMPANIED BY A NUMBER OF COMPENSATORY PATHOLOGICAL MECHANISMS, EXCEPT FOR
    1) development of the collateral venous network
    2) the formation of ascites
    3) enlargement of the spleen
    4) the formation of hypersplenism

25. THE DEVELOPMENT OF HEPATIC ENCEPHALOPATHY GOES THROUGH THE FOLLOWING STAGES, EXCEPT
    1) the stage of precursors: sleep disturbance (insomnia at night and drowsiness during the day), conflict, deterioration of orientation in space
    2) stupor stage: drowsiness while maintaining the ability to follow simple commands, the patient reacts to painful stimuli or a loud cry, but, following simple instructions, quickly gets tired
    3) stage of hepatic coma: lack of consciousness
    4) recovery stage

26. GILBERT'S SYNDROME IS CLINICALLY MANIFESTED BY THE FOLLOWING SYMPTOMS
    1) severe hyperbilirubinemia due to free bilirubin, patients die early, death is preceded by bilirubin encephalopathy
    2) mild hyperbilirubinemia due to conjugated bilirubin, characterized by deposition of melanin pigment in the liver, an increase in the ratio of coproporphyrins I and III in the urine, bilirubinuria, but does not affect life expectancy
    3) slight hyperbilirubinemia due to free bilirubin, increases during starvation, does not affect life expectancy
    4) severe hyperbilirubinemia due to free bilirubin is treatable with phototherapy, phenobarbital, patients often survive to adulthood

27. THE FOLLOWING SYMPTOMS ARE TYPICAL FOR THE ADVANCED STAGE OF LIVER CIRRHOSIS

1) bloating, pain and a feeling of heaviness in the upper abdomen; weight loss, weakness, increased fatigue, decreased performance; enlargement and hardening of the liver; splenomegaly is moderately expressed or not yet obvious; sometimes this stage proceeds latently and is detected during a routine examination or morphological examination of a liver biopsy for another disease
2) the appearance of the following symptoms: fingers and nails − in the form of «drum sticks» and «watch glass»; often the appearance of patients resembles a «spider figure»; there are jaundice, skin hemorrhagic syndrome, gynecomastia, genital hypoplasia; pastosity and swelling of the legs, ascites; symptoms of portal hypertension (dilation of the veins of the anterior wall of the abdomen, esophagus, stomach, intestines)
3) increased hepatocellular insufficiency up to the development of coma; portal hypertension with refractoriness of ascites to drug therapy; development of hepatorenal syndrome; accession of a bacterial infection; frequent profuse bleeding from varicose veins of the esophagus and stomach; transformation into liver cancer
4) decrease in the number of one or more cellular elements of peripheral blood (pancytopenia; thrombocytopenia, leukopenia, anemia), hyperplasia of the corresponding bone mar-

row sprouts, splenomegaly, improvement of clinical and hematological parameters after splenectomy

28. THE INITIAL STAGE OF LIVER CIRRHOSIS IS CHARACTERIZED BY

1) bloating, pain and a feeling of heaviness in the upper abdomen; weight loss, weakness, increased fatigue, decreased performance; enlargement and hardening of the liver; splenomegaly is moderately expressed or not yet obvious; sometimes this stage proceeds latently and is detected during a routine examination or morphological examination of a liver biopsy for another disease
2) the appearance of the following symptoms: fingers and nails − in the form of «drum sticks» and «watch glass»; often the appearance of patients resembles a «spider figure»; there are jaundice, skin hemorrhagic syndrome, gynecomastia, genital hypoplasia; pastosity and swelling of the legs, ascites; symptoms of portal hypertension (dilation of the veins of the anterior wall of the abdomen, esophagus, stomach, intestines)
3) increased hepatocellular insufficiency up to the development of coma; portal hypertension with refractoriness of ascites to drug therapy; development of hepatorenal syndrome; accession of a bacterial infection; frequent profuse bleeding from varicose veins of the esophagus and stomach; transformation into liver cancer

4) decrease in the number of one or more cellular elements of peripheral blood (pancytopenia; thrombocytopenia, leukopenia, anemia), hyperplasia of the corresponding bone marrow sprouts, splenomegaly, improvement of clinical and hematological parameters after splenectomy

29. PALPATION OF THE SPLEEN IS PAINFUL IN ALL CONDITIONS, EXCEPT FOR ONE

1) perisplenitis
2) hypersplenism
3) malaria
4) relapsing fever

30. PALPATION OF THE LOWER EDGE OF THE LIVER WILL BE PAINLESS WITH

1) congestive liver
2) perihepatitis
3) liver amyloidosis
4) inflammatory bulging of the liver with angiocholitis

31. IDIOSYNCRATIC CHOLESTATIC HEPATITIS CAN OCCUR WITH THE USE OF THE FOLLOWING DRUGS

1) isoniazid, halothane, methyldopa, rifampicin, dantrolene, nitrofurantoin
2) chlorpromazine, erythromycin estolate, chlorpropamide, tolbutamide
3) methyltestosterone
4) paracetamol, salicylates, tetracyclines, azathioprine, methotrexate

32. CLINICAL CRITERIA FOR HYPERSPLENISM (W.DAMESHEK) INCLUDE ALL OF THE FOLLOWING EXCEPT

1) a decrease in the number of one or more cellular elements of peripheral blood (pancytopenia; thrombocytopenia, leukopenia, anemia)
2) hyperplasia of the corresponding sprouts of the bone marrow
3) the formation of portal hypertension
4) improvement of clinical and hematological parameters after splenectomy

33. MAJOR CRITERIA FOR HEPATO-RENAL SYNDROME − ALL ITEMS EXCEPT

1) the presence of chronic liver disease with liver failure, portal hypertension and fulminant liver failure
2) low glomerular filtration rate
3) proteinuria less than 500 mg/day and the absence of an ultrasound picture of obstructive or parenchymal kidney disease
4) Availability of data on the use of nephrotoxic drugs

34. THE STAGE OF ADVANCED CLINICAL MANIFESTATIONS OF AUTOIMMUNE HEPATITIS IS CHARACTERIZED BY THE FOLLOWING SYMPTOMS, EXCEPT

1) increased asthenic syndrome, and jaundice
2) skin itching is characteristic
3) abdominal manifestations; various skin rash-es: «spider veins»; bright pink striae on the

abdomen and thighs; hemorrhagic and acne skin rashes; hepato- and splenomegaly
4) systemic manifestations (vasculitis, polymyositis, lymphadenopathy, pneumonia, pleurisy, myocarditis, pericarditis, glomerulonephritis, Hashimoto's thyroiditis, ulcerative colitis, diabetes mellitus, hemolytic anemia, idiopathic thrombocytopenia, hypereosinophilic syndrome

35. THE LIVER REACHES THE MOST GIGANTIC SIZES
1) with edematous-ascitic syndrome
2) with infectious diseases of the liver
3) in cancer, amyloidosis and hypertrophic cirrhosis
4) with autoimmune hepatitis

36. NORMALLY, IN AN ADULT WITH A NORMAL BODY MASS INDEX, NORMOSTHENIC CHEST, IN A HORIZONTAL POSITION, WITH AN AVERAGE DEPTH OF BREATHING, THE LOWER LIMIT OF ABSOLUTE HEPATIC DULLNESS ALONG THE RIGHT ANTERIOR AXILLARY LINE IS LOCATED AT THE LEVEL OF
1) VII ribs
2) VIII ribs
3) IX ribs
4) X ribs

37. FEVERISH SYNDROME IN TERMINAL LIVER DISEASES OCCURS DUE TO
1) the formation of necrosis of hepatocytes, the entry of toxic products into the blood, bacter-emia, the entry of microorganisms

from the intestine with the development of sepsis in the terminal stage is possible
2) arterial hypotension with high cardiac output and low peripheral vascular resistance
3) the development of normally inactive arteriovenous anastomoses, an increase in vascular volume, resulting in a decrease in the effective volume of arterial blood, hypoxia develops
4) a decrease in the synthesis of albumin in the liver and a drop in oncotic pressure; aldosterone inactivation is also disturbed, which leads to secondary hyperaldosteronism with the development of hypernatremia and hypokalemia

38. THE SYNDROME OF ENDOCRINE DISORDERS IN LIVER FAILURE IS MANIFESTED BY ALL OF THE FOLLOWING SYMPTOMS, EXCEPT

1) testicular atrophy, infertility
2) gynecomastia
3) hypertrichosis
4) the formation and rapid increase in «spider veins»

39. FOR CIRRHOSIS OF THE LIVER, CLASS A ACCORDING TO CHILD−PUGH, IT IS CHARACTERISTIC

1) severe encephalopathy, PTT 1−3 s; mild ascites, bilirubin 3 mg/dl, albumin 3.0 mg/dl; prothrombin time 6 s
2) absence of ascites, slight encephalopathy, bilirubin less than 2 mg/dl, albumin 5 mg/l, prothrombin time 2 s

3) severe ascites, slight encephalopathy, albumin 1.5 mg/dl, bilirubin 4 mg/dl, prothrombin time 10 s
4) mild ascites, mild encephalopathy, albumin 5 mg/l, bilirubin 4 mg/dl, prothrombin time 11 s

40. DOSE-DEPENDENT HEPATOTOXICITY OCCURS WITH THE USE OF THE FOLLOWING DRUGS

1) isoniazid, halothane, methyldopa, rifampicin, dantrolene, nitrofurantoin
2) chlorpromazine, erythromycin estolate, chlorpropamide, tolbutamide
3) methyltestosterone
4) paracetamol, salicylates, tetracyclines, azathioprine, methotrexate

41. MALABSORPTION SYNDROME IS

1) violation of digestion in the intestine
2) malabsorption in the stomach
3) malabsorption in the intestine
4) colonization of the intestines by pathogenic microflora

42. SUDDEN ACUTE «DAGGER-LIKE» ABDOMINAL PAINS ARE CHARACTERISTIC OF

1) for perforation of the stomach
2) for chronic gastritis
3) for duodenal ulcer
4) all of the above

43. WHAT DOES RUMBLING DURING PALPATION OF THE CAECUM TESTIFY TO

1) the symptom is normal

2) there is free fluid in the abdominal cavity
3) there is a pyloric stenosis
4) there is a large amount of gas in the large intestine (flatulence in a patient with colitis)

44. WHAT DOES THE RUMBLING DURING PALPATION OF THE ASCENDING AND TRANSVERSE COLON TESTIFY TO

1) the symptom is normal
2) there is free fluid in the abdominal cavity
3) there is a pyloric stenosis
4) there is liquid content in the large intestine and gases accumulate (for example, in a patient with acute enteritis)

45. ON PALPATION OF THE STOMACH, THE FOLLOWING ARE BEST STUDIED

1) greater curvature of the stomach
2) gatekeeper
3) greater and lesser curvature of the stomach
4) the cardial part of the stomach

46. DIFFERENCE BETWEEN ESOPHAGEAL VOMITING AND VOMITING OF GASTRIC ORIGIN

1) occurs without nausea
2) vomiting is preceded by nausea
3) vomit consists of undigested food
4) vomit does not contain hydrochloric acid and pepsin

47. CROHN'S DISEASE IS CHARACTERIZED BY EVERYTHING EXCEPT

1) fever, diarrhea, abdominal pain and weight loss

2) the pain syndrome can be in the nature of an «acute abdomen»
3) the leading diagnostic methods are endoscopic − FEGDS and colonoscopy
4) only the intestinal mucosa is affected

48. IN NONSPECIFIC ULCERATIVE COLITIS, EVERYTHING HAPPENS, EXCEPT

1) with NUC, the rectum is almost always affected
2) typical pancolitis
3) the formation of fistulas, fistulas, abscesses, strictures is possible
4) characterized by diarrhea with blood, mucus, sometimes with pus

49. A DIGITAL EXAMINATION OF THE RECTUM IS PERFORMED (REMOVE THE WRONG ANSWER)

1) after a cleansing enema
2) is carried out by a doctor in the presence of complaints of pain during defecation
3) is carried out with the release of blood from the rectum
4) is a method for diagnosing NUC

50. CONSTIPATION IS (CHOOSE THE INCORRECT OPTION)

1) stool retention for more than 36 hours
2) stool retention for more than 48 hours
3) prolonged delay in bowel movements
4) their presence depends on the nature of food and drinking regimen

CLINICAL SITUATIONAL TASKS

Task № 1

Patient V., 40 years old, noticed yellowing of the sclera and skin in a saffron-yellow tone, urine the color of dark beer, weakness, aversion to food, mild nausea. Three months ago, there was a single use of an intravenous narcotic substance − heroin. Considers himself sick for 15 days. During this time, the body temperature remained within 37.2–37.5 °C, large joints ached, appetite disappeared, in the last 2 days − dark urine, yellowness of the sclera and skin. General condition of moderate severity. Temperature 36.8 °C. Sclera and skin moderately icteric. Peripheral lymph nodes are not changed. Pulse − 50 beats / min, blood pressure − 110/55 mm Hg. Art. Respiration is vesicular. The abdomen is soft, slightly painful in the epigastrium. The liver protrudes from under the right edge of the costal arch by 3 cm, the lower pole of the spleen is palpated. Urine dark brown, feces gray, acholic, mushy.

Questions:

1. Establish a preliminary diagnosis based on the data obtained, taking into account the syndromic approach, and justify it.
2. Give an etiological description of the pathogen that allegedly caused the disease, name the sources of infection and ways of infection.
3. Name the diseases with which it is necessary to carry out differential diagnostics.

Task № 2

A 60-year-old patient (who had just arrived by train) turned to the station paramedic with complaints of sudden onset of sharp, sharp pains in the right hypochondrium after a diet

violation (she ate smoked fish and fried pies). Pain radiates to the right side of the chest. The patient is agitated, groans, tries to find a comfortable position. The pain has been disturbing for 5 hours already, the intensity persists while taking non-steroidal anti-inflammatory drugs. The skin is olive-yellow, covered with sweat. Pulse 70 bpm. BP 130/80 mmHg Temperature 38.0 degrees Celsius. On palpation, pain in the right hypochondrium. The gallbladder is not palpable. The phrenicus symptom is revealed. History of gallstone disease.

Questions:

1. Establish a preliminary diagnosis based on the data obtained, taking into account the main syndromes, and justify it.
2. Give the etiological characteristics of the disease.
3. What will be the changes in feces and urine in this case?

Task № 3

Patient A., 46 years old, a worker, was admitted to the clinic with complaints of general weakness, fatigue, sleep disturbance, loss of appetite, dry mouth, itching of the skin, dark urine, yellowness of the skin and sclera, an increase in the abdomen. Viral hepatitis markers are negative. Drinks 1−2 bottles of 1.5 liters daily for many years. He was sent to a therapeutic hospital for examination and treatment.

Objectively: the state of moderate severity, yellowness of the skin and sclera. On the chest and back – «spider veins», spots of «blood dew». Vesicular breathing in the lungs, no wheezing. Heart sounds are muffled, rhythmic. Heart rate 89 per minute, BP 155/90 mm Hg. The abdomen evenly participates in the act of breathing, increased in volume due to ascites. A dense painless edge of the liver is palpable. Kurlov's ordinates are 19−16−9 cm. The spleen is not palpable. There are no peripheral edema.

FGDS: varicose veins in the lower third of the esophagus.

Ultrasound: the liver is enlarged, echogenicity is increased, the structure is heterogeneous, the vascular pattern is depleted. The spleen is enlarged. Ascites.

Questions:

1. Formulate a preliminary diagnosis.
2. List the anamnestic and clinical signs confirming the etiology of the disease.
3. What organs and systems are involved in the pathological process?

Task № 4

Patient R., 44 years old. Delivered to the hospital in an unconscious state; there is no reaction to painful stimuli, stiffness of the neck muscles and muscles of the extremities, clonus of the muscles of the foot, reflexes of Babinsky, Gordon are determined. «Liver smell» from the mouth. The skin is pale gray with an icteric tint, subicteric sclera; exhausted, on the skin − telangiectasia, petchial rashes.

Pulse 120 beats / min, weak filling and tension. The heart is moderately enlarged in diameter, the tones are quickened, deaf. The abdomen is significantly enlarged in volume; on the anterior abdominal wall, dilated tortuous saphenous veins form a picture of the «head of a jellyfish». On palpation and percussion of the abdomen, there are signs of free fluid. Due to ascites, the abdominal organs are inaccessible to palpation. The method of «balloting» palpation is determined by an enlarged dense liver.

Question:

1. What is your probable diagnosis?

ANSWER KEY TO MCQ TESTS

1 – 1	11 – 3	21 – 2	31 – 2	41 – 4
2 – 3	12 – 1	22 – 1	32 – 3	42 – 4
3 – 4	13 – 2	23 – 1	33 – 4	43 – 3
4 – 4	14 – 1	24 – 4	34 – 2	44 – 4
5 – 2	15 – 3	25 – 4	35 – 3	45 – 4
6 – 3	16 – 1	26 – 3	36 – 4	46 – 3
7 – 1	17 – 2	27 – 2	37 – 1	47 – 4
8 – 2	18 – 3	28 – 1	38 – 3	48 – 3
9 – 1	19 – 4	29 – 2	39 – 2	49 – 1
10 – 2	20 – 1	30 – 3	40 – 4	50 – 2

ANSWERS TO CLINICAL SITUATIONAL TASKS

Task № 1

1. Preliminary diagnosis: viral hepatitis, it is necessary to specify the causative virus. There is a cholestasis syndrome, an inflammatory syndrome, a syndrome of hepatocellular insufficiency. According to the laboratory analysis, cytolysis syndrome will definitely be detected.

2. The diagnosis was established on the basis of an epidemiological history: three months ago, parenteral manipulations, based on the presence of symptoms of damage to the liver parenchyma: jaundice, dark urine, clarified feces.

3. It is necessary to carry out differential diagnostics with various viral liver lesions: viral hepatitis A, E, C, as well as with alcoholic and drug-induced hepatitis. Additionally, check for HIV infection.

Task № 2

1. The most likely diagnosis is an attack of biliary colic on the background of cholelithiasis. Most likely there was an obstruction of the bile ducts by a stone.

2. After a jolting train ride and a diet violation, the gallbladder stones moved, there was an obstruction of the biliary tract, which grew within 5 hours and provoked the development of obstructive (subhepatic) jaundice.

3. The feces will be acholic, the urine will be dark.

Task № 3

1. Alcoholic cirrhosis of the liver, portal hypertension syndrome, ascites.

2. Drinking a large amount of beer for a long time is an etiological factor in the development of alcoholic liver disease, and later − cirrhosis, portal hypertension syndrome.

3. Involved in the process: liver, spleen, kidneys, heart, lungs, and also the brain.

Task № 4

Hepatic coma. Cirrhosis of the liver is terminal. portal hypertension syndrome. Ascites. Edema of the brain. Jaundice is parenchymal. Cancer of the liver is excluded. Secondary toxic cardiomyopathy.

BIBLIOGRAPHY

1. *Бейтс Б., Байкли Л.* Энциклопедия клинического обследования больного: пер. с англ./ под ред. *И.Н. Денисова, В.Т. Ивашкина, В.А. Княжева*. – М.: ГЭОТАР-Медиа, 2001.

2. *Бобылев Ю.М., Зубарев М.А.* Симптомы и синдромы – из прошлого в будущее. – Пермь: ГОУ ВПО «ПГМА Минздрава России», 2002. – 24 с.

3. *Голованова Е.С.* Дифференциальная диагностика гепатомегалий. − Пермь: Пресстайм, 2008. − 252 с.

4. *Гребенев А.Л., Шептулин А.А.* Непосредственное исследование больного. – М.: МЕДпрес-информ, 2005.

5. *Григорьев П.Я., Яковенко А.В.* Клиническая гастроэнтерология. − М.: Мед. информ. агентство, 2004 – 768 с.

6. *Губергриц А.Я.* Непосредственное исследование больного. – Ижевск: Удмуртия, 1996.

7. *Губергриц А.Я.* Непосредственное исследование больного. Курс физикальной диагностики. – 3-е изд. − Ижевск: Удмуртия, 1996. – 332 с.

8. *Затурофф М.* Симптомы внутренних болезней: пер. с англ. − М.: Mosby-Wolf – Практика (совместное издание), 1997. – 493 с.

9. *Иванченкова Р.А., Свиридов А.В., Грачев С.В.* Холестероз желчного пузыря: современный взгляд на патогенез, клинику, диагностику и лечение. - М.: Мед. информ. агентство», 2005. – 200 с.

10. *Ивашкин В.Т., Султанов В.К.* Пропедевтика внутренних болезней. − М.: Литтерра, 2007. – 560 с.

11. Клинические рекомендации по диагностике и лечению болезни Крона у взрослых (проект) / *В.Т. Ивашкин, Ю.А. Шелыгин, Д.И. Абдулганиева* [и др.]. // Колопроктология. − 2020. – Т 19, №2 (72). − С. 8−38.

12. Клинические рекомендации Российской гастроэнтерологической ассоциации и Ассоциации колопроктологов России по диагностике и лечению язвенного колита / *В.Т. Ивашкин, Ю.А. Шелыгин, Д.И. Абдулганиева* [и др.] // Колопроктология. – 2017. − №1(59). − С. 6−30.

13. Морфологическая диагностика атрезии желчевыводящих путей/ *А.В. Дегтярева, Е.Л. Туманова, А.Ю. Разумовский* [и др.]// Российский журнал гастроэнтерологии, гепатологии, колопроктологии. – 2008. − Т.18, №5. − С. 54−62.

14. *Мухин Н.А., Моисеев В.С.* Пропедевтика внутренних болезней. – 2-е изд., доп. и перераб. – М.: ГЭОТАР-Медиа, 2009. – 849 с.

15. *Никонов В.В.* Медицина неотложных состояний, избранные клинические лекции. – Т. 2. – 2007.

16. Основы семиотики заболеваний внутренних органов/ *А.В. Струтынский, А.П. Баранов, Г.Е. Ройтберг, Ю.П. Гапоненков*. – М.: РГМУ, 1997. – 224 с.

17. Практикум по пропедевтике внутренних болезней/ под ред. *Ж.Д. Кабалава, В.С. Моисеева*. – М.: ГЭОТАР-Медиа, 2008.

18. Пропедевтика внутренних болезней/ *В.Х. Василенко, А.Л. Гребенев, В.С. Голочевская* [и др.]. – М.: Медицина, 1989. – 512 с.

19. Пропедевтика внутренних болезней: ключевые моменты/ под ред. *Ж.Д. Кабалава, В.С. Моисеева*. – М.: ГЭОТАР-Медиа, 2008.

20. *Ратников В.А*. Диагностические возможности и методика магнитно-резонансной томографии печени, билиарного тракта, поджелудочной железы и селезенки. // Российский журнал гастроэнтерологии, гепатологии, колопроктологии. – 2001. − Т. XI, №6. − С. 28−32.

21. Синдром портальной гипертензии / *А. Г. Скуратов* [и др.]. – Гомель: ГомГМУ, 2014. – 48 с.

22. *Циммерман Я.С.* Очерки клинической гастроэнтерологии. − Пермь: ФГБОУ ВО ПГМУ им. академика Е.А. Вагнера Минздрава России, 1992. – 336 с.

23. Чучалин А.Г., Бобков Е.В. Основы клинической диагностики. – 2-е изд. − М.: ГЭОТАР-Медиа, 2008 – 584 с.

24. *Шапошников А.В.* Асцит: современные подходы к классификации и лечению // Российский журнал гастроэнтерологии, гепатологии. – 2001. – Т. XI, №3. − С. 19−23.

25. *Шифф Ю.Р.,Сорелл М.Ф., Уилисс С.М.* Введение в гепатологию / пер. с англ. под ред. *В.Т. Ивашкина, А.О. Буеверова, М.В. Маевской.* – М.: ГЭОТАР-Медиа, 2011. – 704 с.

26. *Шульпекова Ю.О.* Метеоризм: круг знания или круг незнания? // Медицинский совет. − 2013. − № 10. − С. 52−60.

27. *Avundik C.* Mannual of gastroenterology. Diagnosis and therapy. – Philadelphia, USA, 2008. – 516 p.

28. Harrison's principles of internal medicine / *J.L. Jameson, D.L. Kasper, D.L. Longo* [et al.]. – 20th ed. – New-York, USA. – 2018. – 3790 p.

29. Harrison's Principles of Internal Medicine/ eds. *J. Jameson, A.S. Fauci, D.L. Kasper* [et al.]. − 20 th. – McGraw Hill, 2018.

30. *Innes J.A., Dover A.R., Fairhurst K.* Macleod's Clinical Examination. – 14th ed. – Hariana, India: ELSEVIER, 2015. – 402 p.

31. *Sainani G.S., Joshi V.R., Sainani R.G.* Mannual of clinical and practical medicine. – Hariana, India: ELSEVIER, 2010. – 512 p.

LIST OF LITERATURE RECOMMENDED FOR SELF STUDY

1. *Innes J.A., Dover A.R., Fairhurst K.* Macleod's Clinical Examination. – 14th ed. – Edinburgh, London, New-York, Oxford, Philadelphia, St. Louid, Sidney: ELSEVIER, 2018. − 402 p.

2. *Duda R.W.* Systems Gastrointestinal Tract. – Philadelphia: Lippincott Willams &Wilkins a Wolter Kluwer business, 2010. – 193 p.

3. *Ralston S.H., Penman I.D., Strachan M.W.J., Hobson R.P.* Davidson's Principles and Practice of Medicine. – Edinburgh, London, New-York, Oxford, Philadelphia, St. Louid, Sidney: ELSEVIER, 2018. – 1440 p.

4. *Glynn M., Drae W.M.* Hutchison's Clinical Methods. Ah integrated approach to clinical practice. – Edinburgh, London, New-York, Oxford, Philadelphia, St. Louid, Sidney: ELSEVIER, 2012 – 485 p.

5. *Block B.* Abdominal ultrasound: Step by Step. – 2nd ed. – Thieme, Stuttgart: New-York, 2012. – 305 p.

6. *Laposata M.* Clinical Diagnostic Tests. How to Avoid Er-rors in Ordering Tests and Interpreting Results. – New-York: DemosMEDICAL, 2016. – 311 p.

7. *Pal K.P., Pal P., Nanda N.* Comprehensive Textbook of Medical Physiology, Vol.1. – JAYPEE The Health Sciences Publisher, New Delhi, 2017 – 1395 p.

Educational edition

SYMPTOMS AND SIGNS OF GASTROINTESTINAL DISEASES

Study guide for students of medical universities studying in English

Compiled by:
Goryacheva Olga Georgievna

Corrector A.A. Efimova

Signed in the press 14.11.2023.
The format is 60×90/16. Conditional printing sheets 19,75.
Circulation 50 copies. Order № 23.

Editorial and Publishing Department
FGBOU V RGMU named after Academician E.A. Vagner
Ministry of Health of Russia
27 Petropavlovskaya str., Perm, 614990

Printed in the printing house of IP Seregina O.N.
Address: 21, Metallistov St., Perm, 614107, apt. 174.

Учебное издание

СИМПТОМЫ И ПРИЗНАКИ ЗАБОЛЕВАНИЙ ЖЕЛУДОЧНО-КИШЕЧНОГО ТРАКТА

Учебное пособие для студентов, обучающихся на английском языке

Автор:

Горячева Ольга Георгиевна

Корректор А.А. Ефимова

Подписано в печать 14.11.2023 г.
Формат 60×90/16. Усл. печ. л. 19,75.
Тираж 50 экз. Заказ № 23

Редакционно-издательский отдел
ФГБОУ ВО ПГМУ им. академика Е.А. Вагнера Минздрава России
614990, г. Пермь, ул. Петропавловская, 27

Отпечатано в типографии ИП Серегина О.Н.
Адрес: 614107, г. Пермь, ул. Металлистов, д. 21, кв. 174