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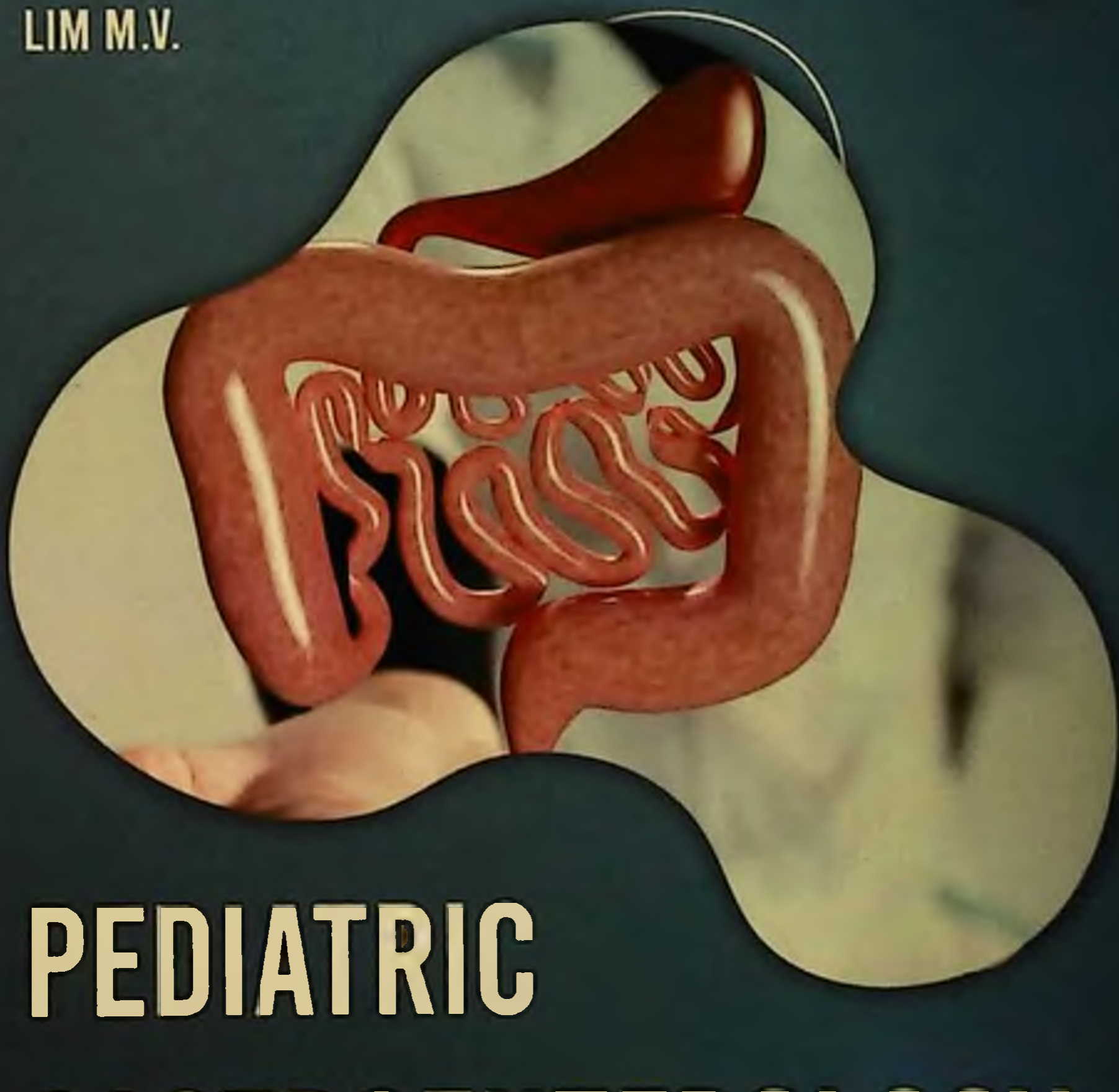

PEDIATRIC GASTROENTEROLOGY

MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION OF THE REPUBLIC OF UZBEKISTAN
MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN

Lim M.V.

PEDIATRIC GASTROENTEROLOGY

The textbook is intended for students of medical universities

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This textbook has been developed in compliance with the State Educational Standard for Higher Professional Education in Pediatrics - 5510200. It encompasses sections addressing prevalent pediatric gastroenterological diseases, covering their etiology, pathogenesis, classification, clinical manifestations, examination methodologies, differential diagnoses, principles of treatment, complications, and preventive measures. It is designed for students enrolled in the pediatric faculty.

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CONTENT

List of abbreviations

Here's the translation of the list of abbreviations into English:

AB - antibiotics
BP - blood pressure
AST - aspartate aminotransferase
ALT - alanine aminotransferase
AT - antibody
GC - glucocorticoids
NSAIDs - nonsteroidal anti-inflammatory drugs
CG - chronic gastritis
CP - chronic pancreatitis
CC - chronic cholecystitis
CGD - chronic gastroduodenitis
US - ultrasonography
AC - acute cholecystitis
EGD - esophagogastroduodenoscopy
ESR - erythrocyte sedimentation rate
UD - peptic ulcer disease
UC - ulcerative colitis

INTRODUCTION

The analysis of contemporary literature demonstrates an escalating trend in the incidence of gastrointestinal disorders, with no indication of a declining pattern. Gastro-duodenal pathology plays a pivotal role in the structure of gastroenterological conditions. In Uzbekistan, there is also an observable rise in gastrointestinal diseases, which, if left untreated, may lead to serious consequences. The etiology, pathogenesis, diagnostic methods, and treatment principles of gastroenterological disorders are enriched by novel scientific research foundations. This manual emphasizes the significance of risk factors, considering the traditions of our region's peoples in the development of gastro-duodenal pathology in Uzbekistan and their diagnostic coefficients.

Given that the majority of gastrointestinal disorders are associated with abdominal syndrome, this topic receives extensive coverage. Considering the high frequency of diseases in the small and large intestines among children, we deemed it necessary to include this topic in the manual. Additionally, topics such as chronic hepatitis, pancreatitis, exocrine pancreatic insufficiency, and cholecystitis in children are encompassed in this manual.

The purpose of this manual is to assist students, postgraduates, and clinical residents in mastering the etiopathogenesis, clinical manifestations, and treatment principles of gastroenterological disorders. This educational manual is designed for students in the 5th and 6th years of the pediatric faculty, master's students, and clinical residents to enhance their knowledge.

ABDOMINAL SYNDROME

Abdominal Syndrome

According to the definition by the International Association for the Study of Pain, pain is a symptom of many conditions that cause unpleasant, intolerable sensory (emotional) and emotional burdens due to tissue damage or changes within them.

By the course of pain, there are:

— Acute pain

— Chronic pain

Abdominal Syndrome encompasses:

— Functional disorders of the digestive system

— Thoracic organ diseases accompanied by abdominal pain
— Abdominal cavity and wall diseases that do not require emergency surgery
— Abdominal pain unrelated to the gastrointestinal tract

Types of abdominal syndrome include:

— Visceral pain - constant transient pain often dispersed in the middle of the abdomen
— Somatic pain - acute pain with clear localization, developing during acute abdominal organ diseases
— Radiating pain

Characteristics of pain:

— Constant (aching) pain arises due to inflammation from constant stimulation of the nerve cells in the mucous and submucous membranes
— Periodic (intermittent - sporadic, nocturnal) pain caused by hypersecretion, specific spasms
— Colicky pain induced by spasmodic contractions of smooth muscles
— Seasonal pain (autumn or spring)
— Pain associated with food intake, antacids, and antispasmodics, excitement, and physical exertion.

Causes of Pain in Children Up to 6 Months

— Intestinal colic (ingestion of air into the stomach or intestines)
— Inflammation of the gastrointestinal tract in infants

Causes of abdominal pain syndrome in children older than 6 months:

— Gastroenteritis
— Abdominal syndrome during influenza
— Inguinal hernia
— Dysbacteriosis
— Dismetabolic nephropathy

Causes of abdominal pain syndrome in preschool-aged children:

— Constipation
— Urinary tract infection
— Pneumonia, pleurisy
— Food poisoning
— Dysbacteriosis
— Appendicitis

Causes of abdominal pain syndrome in school-aged children:

- Gastroenteritis
- Stomach and duodenal ulcers
- Pancreatitis
- Dyskinesia of the biliary tract
- Cholecystitis and cholangitis
- Gallstone disease
- Kidney stone disease
- Abdominal trauma
- Pneumonia
- Urinary tract infection
- Malabsorption syndrome
- Constipation
- Dysmenorrhea

Common symptoms of pain associated with bowel disease

— Lack of a clear relationship with food intake (except for inflammatory bowel disease - pain associated with reflex contraction of the intestine as a result of eating);

— Associated with the act of defecation;

— Reduction of pain after the act of defecation or gas discharge.

Pain localization

Under the right hypochondrium:

— Inflammation of the gallbladder - cholecystitis.

— Dyskinesia of the biliary tract.

— biliary colic - gallstones

— ascending cholangitis

Navel area

— Stomach and small intestine: small bowel obstruction, appendicitis

— Aorta and its branches: abdominal aortic aneurysm, mesenteric circulation disorders.

— -Right region of the transverse colon:

— Appendix

— Cecum and colon - Crohn's disease

Groin area:

— Testicular torsion

— Inguinal hernia

Epigastric region:

— Esophagus: esophagitis
— Stomach: duodenal ulcer
— Gallbladder: cholecystitis
— Pancreas: pancreatitis
— Lungs: lower lobe pneumonia
— Myocardial infarction in adults

Left hypochondrium and lumbar region:

— Renal colic, urinary tract infections
— Splenic infarction

Hypogastric zone:

— Colon obstruction
— Left iliac fossa-descending colon and sigmoid colon: diverticulitis

Suprapubic region:

Bladder cystitis
— Uterus and its appendages: endometritis, fallopian tube rupture, salpingo-oophoritis
— Functional diseases of the digestive system
— Indigestion Syndrome
— Aerophagia
— Constipation
— Colon irritation
— Biliary dyskinesia
— Nervous system disorders manifest as neuroses.

Abdominal pain syndrome not associated with diseases of the gastrointestinal tract

— Dry pleurisy
— Pleuropneumonia
— Abdominal epilepsy
— Congenital heart defects
— Rheumatism
— Nodular periarteritis

Diseases of the abdominal cavity and retroperitoneal space that do not require emergency surgery

Pain associated with peptic ulcer

— Cholecystitis and gallstones
— Chronic pancreatitis
— Enteritis
— Chronic colitis: Crohn's disease, ulcerative colitis.
— Renal colic
— Abdominal purpura (Schonlein-Henoch disease)
— Dysmenorrhea

Abdominal pain is the most common complaint among children. In young children, it manifests as constant crying, screaming, restlessness, refusal to eat, drawing legs towards the stomach, and continual leg movement. These symptoms can arise not only from abdominal organ diseases but also from other conditions such as cerebral palsy, brain tumors, ear or bone diseases, among others. Hence, a doctor's experience and intuition play a crucial role in carefully observing the child's condition and movements to identify the cause of distress and locate the source of pain.

Abdominal pain is not solely linked to abdominal organ diseases but also to other bodily systems and organs. Gastrointestinal tract diseases predominantly cause abdominal pain, with intestinal colic being the most prevalent among children below 1 or 2 years of age. It commonly occurs in infants within the first two weeks, characterized by continuous crying, flushed skin, heightened anxiety, and rhythmic leg movements toward the stomach. The infant's eyes are shut, and perspiration covers the forehead. Tense abdominal muscles, bloating, and vomiting are common symptoms. Typically occurring in 2-3-month-old infants, these episodes are more frequent during the evening and night, lasting from a few minutes to 6-8 hours.

Food allergies, stomach and intestinal cramps, inadequate or excessive feeding, aerophagia, improper feeding techniques, or inappropriate formula feeding are common causes of colic. Sudden onset of abdominal pain in young children may indicate intussusception, marked by vomiting, weakness, and potential shock. Blood in stools appears within the first 12 hours, followed by jelly-like, mucous, and bloody stools. Abdominal swelling and tension, sometimes with a palpable, tender, sausage-shaped mass, are evident. Intussusception usually occurs on the right side above the navel, with increased swelling during an episode. Some cases may release blood and mucus without

feces during rectal examination. X-ray confirmation is necessary for diagnosis.

Infectious diseases of the small and large intestine frequently cause abdominal pain in children and often present with fever. Symptoms are prominent in gastroenteritis, enteritis, and enterocolitis, including abdominal pain, vomiting, diarrhea, reduced appetite, and weakness. Enteritis results in liquid, occasionally watery stools, whereas enterocolitis yields frequent stools with mucus, pus, and blood. Nonspecific ulcerative enterocolitis brings stabbing pain, mainly in the lower left abdomen, accompanied by stools containing blood clots. Children may appear undernourished and anemic, with an ESR of 16-30 mm/h and Hv around 50 g/L.

Abdominal pain in children often indicates appendicitis, presenting symptoms such as nausea, vomiting, and a gradual rise in body temperature. Children over 2 years of age typically indicate the location of abdominal pain around the navel and lower right quadrant using their hand. They may also experience restlessness, discomfort, irritability, crying, and loss of appetite. The pain can be persistent, progressive, and intense. Stool consistency may vary, ranging from liquid to constipation. An increase in stool indicates the onset of local peritonitis. Objective examination often reveals tense abdominal muscles, increased pain upon palpation, and positive Shchetkin-Blumberg and Rovsing signs. Preference for lying on the stomach with the right leg bent, limping on the right leg while walking, and increased pain during coughing are common. Blood tests often show increased neutrophilic leukocytosis.

Abdominal pain in children may also be associated with the intestinal form of cystic fibrosis, particularly observed in infants with meconium ileus. Symptoms include discomfort, diarrhea, vomiting, and vasodilation of the anterior abdominal wall. The baby's general condition deteriorates, manifesting as pallor, dry skin, decreased turgor, shortness of breath, tachycardia, and toxicosis. Severe complications include intestinal perforation and peritonitis.

Mechanical intestinal obstruction is another cause of abdominal pain in children, where symptoms, such as nausea, vomiting, bloating, cessation of gas formation, and constipation, appear immediately. The pain gradually intensifies, hernia size increases, and the condition becomes irreversible. Intussusception, appendicitis, mechanical intestinal

obstruction, and strangulated hernia can lead to purulent peritonitis, causing widespread pain that intensifies with deep breathing and coughing. Patients with purulent peritonitis often exhibit severe hyperesthesia of the abdominal skin, reluctance to touch the abdomen, reduced intestinal motility, stopped stool passage, vomiting, weakness, pale skin, sunken eyes, and disinterest in the surroundings. Accelerated neutrophilic leukocytosis and ESR are observed in blood tests.

Abdominal pain syndrome is more common in children, particularly those over 5 years old, with gastric and duodenal ulcers. Initially, discomfort occurs in the upper abdomen, later localizing around the navel, often presenting before breakfast or at night. Pain intensity may vary, sometimes subsiding after meals or completely disappearing following vomiting, a primary symptom of gastric and duodenal ulcers. Abdominal pain in the umbilical region is also observed in preschool and primary school-aged children with helminthic infestations, alongside complaints of weakness, headaches, and dizziness. These children often exhibit pale skin with white facial spots, intermittent abdominal pain that occasionally intensifies. Reactive arthralgia may also be detected, with stool analysis for worm eggs being a crucial diagnostic criterion in this disease.

Biliary dyskinesia typically presents with abdominal pain in the right hypochondrium, extending to the upper abdomen and right shoulder. Pain often occurs after consuming fatty broths, soups, smoked, fried, or fatty foods. Palpation exacerbates pain in the right hypochondrium. Cholecystograms often reveal gallbladder kinetics abnormalities.

In cases of cholecystitis and cholangitis, abdominal pain worsens during physical activities like walking, jumping, or running. Patients experience elevated body temperature, pain upon palpation in the right hypochondrium and gallbladder, and enlargement of the liver. Blockage of bile ducts by mucus can result in jaundiced skin and sclera. Blood tests often show increased lymphocytosis and ESR.

Pancreatitis is another cause of abdominal pain in children. During the acute phase, severe pain primarily occurs in the upper abdomen, radiating to the left shoulder. Patients often experience vomiting. Fermentation processes lead to gas accumulation in the intestines. Fecal volume increases, containing traces of fat and blood. Palpation typically reveals bloating, a key symptom of pancreatitis. Additionally, the amylase content in urine rises.

Gallstone disease (cholelithiasis)

Definition. Gallstone disease is a condition affecting the hepatobiliary system, marked by the development of stones within the gallbladder and/or bile ducts due to disruptions in bilirubin and/or cholesterol metabolism. These stones are categorized as either cholesterol or pigment stones.

Cholelithiasis.

Symptoms, syndromes, physical condition:

Complaints: Biliary colic manifests as recurrent pain in the right upper quadrant of the abdomen, sometimes extending to the epigastric region and often radiating towards the right shoulder. Cholecystitis is marked by fever, nausea, and occasional vomiting, frequently triggered by the consumption of fatty foods. The distinction between chronic and acute cholecystitis is typically identified by an increase in body temperature and the presence of pain in the right hypochondrium after consuming meals, particularly with calculous cholecystitis, where pain can escalate after physical exertion.

Physical Findings: Cholecystitis often presents with bloating, flatulence, muscular tension over the gallbladder area, and tenderness upon palpation. Liver enlargement upon palpation is more commonly observed in cases of cholangitis.

Diagnostic Protocol: Initial Assessment: Laboratory tests: Blood and urine analysis, cholesterol, amylase, glucose levels, coprogram, blood group typing, bacteriological examination of duodenal juice, total bilirubin and its fractions, AST, ALT, total protein and its fractions, C-reactive protein. Diagnostic imaging: Abdominal cavity radiography, ultrasound of the liver, gallbladder, pancreas, and spleen; endoscopic retrograde cholangiopancreatography (if indicated), electrocardiography. Treatment approach: Determined based on the diagnosis.

Chronic cholecystitis.

Chronic cholecystitis (without cholelithiasis) is a persistent inflammation of the gallbladder wall resulting from motor abnormalities in the biliary system.

Chronic cholecystitis can be categorized by etiology into bacterial infections and parasitic infestations.

Symptoms, syndromes, physical data:

Complaints: Patients typically report enduring pain in the right hypochondrium, occasionally extending to the epigastric region, lasting

several hours. Pain is provoked and exacerbated by the consumption of fatty, fried foods, eggs, cold sodas, and spicy dishes. In some instances, pain is coupled with nausea, belching, bloating, fever, and persistent upper abdominal discomfort.

Physical data: enderness in the region of the gallbladder projection, especially during breathing, along with a positive Murphy's sign.

Recommended Evaluations:

Initial Assessment:

Laboratory Studies: Comprehensive blood and urine analysis, cholesterol levels, amylase levels, blood type, and Rh factor determination, coprogram, bacteriological, cytological, and biochemical examination of duodenal juice, bilirubin and its fractions, AST, ALT, total protein and its fractions, serum C-reactive protein.

Instrumental Studies: Ultrasound examination of the liver, gallbladder, pancreas, duodenal sounding, esophagogastroduodenoscopy, chest x-ray.

Additional assessments are performed based on the diagnosis and its complications. Consultation with a surgeon is essential.

Consultation of the surgeon is necessary.

Therapeutic Interventions: Dietary Guidelines: Advisable to avoid fatty and fried foods, carbonated and chilled beverages, as well as spicy cuisine. Recommended dietary habits include consuming frequent small portions (4-5 times a day) of steamed or boiled warm food.

Chronic pancreatitis (CP).

Definition: Chronic pancreatitis is a progressive pancreatic disease characterized by acute inflammation signs during an attack and the gradual replacement of parenchyma with connective tissue, leading to exocrine and endocrine pancreatic insufficiency.

Complaints: Patients experience pain in the epigastric region and left hypochondrium, sometimes accompanied by bloating and diarrhea. Pain often correlates with overeating and is alleviated by antacids (including histamine H2 receptor blockers and proton pump inhibitors).

Physical data: Dry skin, glossitis, stomatitis, and weight loss are observed. Palpation of the abdomen reveals tenderness in the epigastric region, under the left rib, and in the intestines. Positive Mayo-Robson's symptoms (pain in the left hypochondrium and left side) might be present. Occasionally, a compacted and enlarged pancreas may be palpated.

Diagnostic Methods:

Laboratory tests: Comprehensive blood and urine analysis, coprogram, total bilirubin and its fractions, AST, ALT, blood amylase and lipase, blood sugar, calcium, total protein and its fractions, blood sugar curve.

Imaging studies: Plain radiography of the abdominal cavity, abdominal ultrasound, endoscopic retrograde cholangiopancreatography, pancreatic ultrasound.

Specialist consultation required: Surgeon, endocrinologist.

Treatment:

1. On the 1st day of hunger, give Borjomi, Essentuki-4 water, feed parenterally.

Day 2 - unsweetened tea, crackers, porridge on the water.

Day 3 - porridge cooked on a mucous broth, tea, crackers.

Day 4 - butter, porridge.

From the 5th day - vegetable puree from potatoes and carrots.

For 7-8 days - you can give a bun, cutlets.

It is necessary to give rest to the pancreas and reduce the production of the enzyme.

2. Detoxification therapy, elimination of metabolic disorders. Removal from the body of products with toxic properties.

3. Elimination of pain and narrowing of the pancreatic ducts, improvement of secretion.

4. Desensitizing therapeutic measures.

5. Antienzymatic therapy.

6. Measures to prevent secondary damage, prevention of infectious lesions.

7. Restoration of the secretory function of the pancreas.

8. General supportive therapeutic measures that increase the body's immunity.

9. Phytotherapy.

Among the most prevalent causes of abdominal pain in children are liver-related conditions (acute and chronic hepatitis, liver cirrhosis, hepatic vein thrombosis, and hepatocellular carcinoma). Pain typically manifests on the right side of the abdomen. Left-sided abdominal pain may occur with splenomegaly and spleen rupture.

Another significant contributor to abdominal pain is kidney and urinary tract diseases. In cases like urolithiasis and obstructive

pyelonephritis, pain occurs in the lower abdomen and back, particularly in the lumbar region. Urolithiasis often presents as paroxysmal pain, and urinalysis indicates leukocyturia, erythrocyturia, and proteinuria.

Abdominal pain syndrome can also manifest in children due to various conditions like mesenteric lymphadenitis, acute pneumonia in young children, rheumatism, pericarditis, pleurisy, leukemia, hemolytic anemia, hemorrhagic vasculitis, hemophilia, paranephritis, precoma in diabetes mellitus, typhoid fever, tuberculosis, and reflex pains. Furthermore, certain metabolic disorders such as diabetic acidosis, hypoglycemia, acetonemic vomiting, and Addison's disease may accompany abdominal pain. Notably, lead poisoning in children has the potential to harm the nervous system and trigger stomach pain.

CHRONIC GASTRITIS AND GASTRODUODENITIS IN CHILDREN

The formation of the stomach initiates during the 3rd week of gestation, progressing with the establishment of circular muscles by the 6th week, and later, the outer longitudinal and inner oblique muscular layers around the 13-14th week. By the 2nd month of fetal development, all sections and glands of the stomach are established.

Throughout childhood and adolescence, the stomach undergoes rapid growth, with its volume tripling by the end of the first year, increasing sixfold by ages 4-5, expanding tenfold by age 10, and reaching up to 24 times its initial volume by age 20. Interestingly, the stomach's growth rate surpasses the child's overall growth. The gastric mucosa's surface area increases substantially with age: by 3 months, it triples; by 6 months, quadruples; at 2 years, it grows to fivefold; and at 15 years, it expands up to tenfold. Epithelial regeneration in the gastric mucosa takes place within 12-24 hours.

At birth, some parts of the stomach are incompletely formed, particularly the bottom and cardial sections, leading to a specific structure in the cardiac sphincter causing frequent vomiting in newborns. The development of the cardial part of the stomach finalizes by age 8.

In newborns, the stomach lining (mucosa) is relatively thick. Folds at the stomach's entrance develop by 8-9 months. As the child grows, the number of stomach cavities increases, into which pancreatic ducts open. Newborns have around 200,000 folds, increasing to 700,000 by 3 months,

2.3 million by 5 months, reaching 1.7 million from 6 to 14 years, and expanding to 4 million by age 15. Although integumentary and stem cells emerge prenatally, the pancreas at birth is morphologically and functionally immature.

The pancreas grows in tandem with the child's switch to enteral feeding. At 2 months, gland numbers increase 3.5-4 times. By 2 years, a child possesses 8 million glands, 10 million by age 6, 18 million by age 15, and an adult typically has 25 million glands

The duodenum in newborns measures 7.5-10 cm in length, increasing gradually with age (in adults, it ranges from 24-30 cm). The duodenal sphincter apparatus comprises the bulbo-duodenal (Kapanji) and medioduodenal (Oxner) sphincters. The medioduodenal sphincter creates a distinct low-pressure area in the lower section of the ascending duodenum, differentiating it from the upper and lower sections.

This unique configuration leads to lower pressure in the ascending part compared to other intestinal segments, attributed to the presence of the bulboduodenal and Oxner sphincters. The duodenal mucosal relief differs from that of the stomach, featuring transverse, yellowish annular folds due to bile. Newborns exhibit fewer and lower folds compared to older children, with the bulb's folds situated beneath the folds of the bulboduodenal and ascending intestines.

Chronic gastritis (CG) is a persistent inflammatory condition affecting the gastric mucosa and submucosa, characterized by cell infiltration and compromised physiological regeneration. Inadequate treatment of chronic hepatitis can lead to gradual pancreatic atrophy within the stomach, resulting in disruptions in secretory, motor, and endocrine functions. CG is observed as an independent disease in only 10-15% of children. Antral gastritis often coexists with duodenitis-gastroduodenitis. The prevalence of chronic hepatitis among the elderly population ranges from 30 to 50%.

Stomach and duodenal ailments are among the most common digestive system issues in children, accounting for 58-65% of gastroenterological conditions, totaling 100-150 per 1000 children. Although Helicobacter pylori (HP) is a known culprit in chronic gastritis and peptic ulcers in children, the influence of various risk factors on the onset and progression of these pathologies should also be acknowledged.

Exogenous risk factors for chronic hepatitis:

— Diet: Consisting of "dry food," excessive consumption of spicy and fried foods, protein and vitamin deficiencies, an overuse of spices, and eating disorders.

— Psycho-emotional factor - stress, depression

— Environmental factors: Atmospheric conditions, nitrates in food, and poor-quality drinking water

— Medications - non-steroidal anti-inflammatory drugs (indomethacin, acetylsalicylic acid, corticosteroids, etc.)

— Bad habits - smoking, alcohol consumption

— parasitic infections (mainly giardiasis)

— food allergies and intolerances to specific foods

— Frequent maxillofacial system diseases

Endogenous factors of hormonal dysfunction of chronic hepatitis:

— Helicobacter pylori (HP) infection.

— Reflux

— Disorders within the endocrine system

The development of highly effective therapeutic technologies, endoscopy, morphological examination of the gastric mucosa, some biochemical and bacteriological methods made it possible to divide gastritis into the following independent types (Sydney Classification (2000)).

Type A gastritis (endogenous autoimmune gastritis)

Endogenous gastritis arises due to the generation of autoantibodies targeting the cells of the gastric mucosa. This form of gastritis is infrequent in children, constituting only 1-3% of all cases. It is distinguished by primary atrophic alterations in the body and fundus of the stomach, leading to reduced gastric secretions and elevated levels of gastrin in the bloodstream.

Type B gastritis (bacterial). The pathogenesis of Type B chronic gastritis involves a persistent HP infection, often confirmed by the presence of this microorganism in the pyloric region of the stomach in the majority of patients. Infection can arise through oral ingestion or as a result of endoscopic procedures or probing.

Type C gastritis (reactive, chemical gastritis, reflux gastritis). The main contributor to gastritis development is duodenogastric reflux, resulting in reduced bile acid levels and subsequent damage to the stomach's mucous membrane and epithelium. Additional factors for this

condition involve non-steroidal anti-inflammatory drugs like acetylsalicylic acid. These medications, through their anti-prostaglandin action, disrupt the production of bicarbonates and mucus, leading to erosion and microcirculation issues. In children, chronic gastritis often occurs due to hereditary influences and the mentioned risk factors. The clinical presentation of chronic gastritis varies and is contingent upon the stomach's secretory and evacuation functions, the child's age, and individual characteristics.

During flare-ups, clinical features of chronic gastritis related to hydrochloric acid secretion are observed, including increased or normal levels of hydrochloric acid secretion, which commonly align with type B gastritis.

Pain syndrome: intense and persistent pain related to meals characterizes gastritis. Morning pain typically aligns with fundic gastritis, while night pain is a hallmark of antral gastritis. Nighttime discomfort doesn't appear linked to seasonal eating habits. In older children, palpation often reveals moderate pain in both the epigastric region and the pyloroduodenal zone.

Dyspeptic syndrome: "sour" belching, flatulence, heartburn, nausea, a tendency to constipation. Syndromes of nonspecific intoxication and asthenia are varied. There are vegetative disorders, irritability, rapid fatigue during mental and physical stress. With reduced secretion of hydrochloric acid (more often type A gastritis), the pain syndrome is mild, characterized by diffuse imitation of pain in the epigastric region. Feeling of heaviness and fullness in the upper abdomen after eating; pain occurs and increases depending on the quality and volume of food. On palpation, there is a slight "diffuse" pain in the epigastric region.

Dyspeptic syndrome, prominent over the pain syndrome, presents symptoms such as bloating, nausea, mouth bitterness, reduced appetite, flatulence, and occasional diarrhea. Gastritis with reduced secretory activity often leads to anorexia for specific foods like cereals or sour-milk products.

Nonspecific intoxication syndrome tends to be more pronounced, with predominant asthenia, resulting in patients appearing very pale, emaciated, with anemia and hypovitaminosis due to gastrointestinal and secondary pancreatic disorders. In pediatric gastroenterology, significant attention is given to stomach acid-forming function, crucial not only in

determining gastroenterological manifestations but also in guiding appropriate antisecretory therapy and reparations.

Intramedial Ph-metry is a contemporary method to assess stomach pH, with normal values in children older than 5 years ranging between 1.7-2.5 on an empty stomach and 1.5-2.5 after histamine introduction. A significant discrepancy between the pH of the body and the antrum of the stomach (more than 2) indicates a compensated state, whereas a reduction in this difference signifies a decrease in the antral region's neutralizing properties and an increase in duodenal acidity (decompensated state). For institutions where intramedial Ph-metry isn't feasible, fractional methods using stimulants are employed to study gastric pH.

Chronic gastritis diagnosis involves clinical and morphological assessment, often determined through comprehensive biopsy examinations of the antrum, fundus, and lateral sections of the stomach. Notably, leading gastroenterologists stress the impossibility of diagnosing gastritis without a morphological study. It is advisable to preliminarily term it 'ulcerative dyspepsia' pending a morphological examination.

Accurate examination schemes for chronic gastritis hinge on its type, stomach secretory function, the child's age, and the status of their autonomic nervous system and mental health. Given that children's abdominal pain is often psychogenic, the diagnosis of CG should be verified through endoscopic and histological assessments before commencing therapy, considering a comprehensive blend of gastroenterological, somatic, and psycho-somatic approaches.

Principles of treatment of type A gastritis. Therapy aims to normalize gastric juice acidity and compensate for atrophic processes in the gastric mucosa. The primary treatment direction is clinical nutrition. During the acute stage, the prescribed diet (No. 1a) includes 5-6 meals, offering functional, mechanical, thermal, and chemical protection for the gastric mucosa. This diet excludes foods that irritate the gastric mucosa, such as salty, smoked, fatty soups, marinades, hot spices, and fried meats or fish. Additionally, items like whole milk, grape juice, and sour cream, which patients often cannot tolerate, should be excluded. Salt, strong tea, and coffee varieties are also limited. As inflammation subsides, there's a gradual increase in functional stimulation of the fundic glands, transitioning to dietary tables No. 2 or even No. 15. However, consumption of fatty meats and fish, insoluble animal fats, fried potatoes, pancakes, canned foods, smoked meats, and sweets is limited. Fresh

fermented milk products like sweet yogurt, curdled milk, cottage cheese, and non-acidic cottage cheese are preferred over milk. Consumption of fresh and black bread, muffins with butter, sour cream, heavy cream, cabbage, and grapes is restricted.

For type A gastritis, anticholinergic and antacid drugs are not usually prescribed. To manage pain and dyspeptic syndrome, oral administration of metoclopramide, sulpiride, no-shpa, and butylscopolamine bromide (buscopan) proves effective. Enveloping and strengthening herbal remedies such as tinctures, plantaglucid granules, and decoctions of chamomile, mint, rosehip, and valerian root are widely used. Herbal tinctures are typically prescribed at 1/3-1/2 cup 4-5 times daily before meals for 2-4 weeks. Stimulating the stomach's secretory function might involve a combination of herbal stimulants like herbogastrin, herbion honey drops, yarrow, and its preparations (pantaglucid). Substitution therapy might include hydrochloric acid, pepsin, and other agents. Drugs enhancing microcirculation, protein synthesis, and reparative processes—such as nicotinic acid preparations, vitamins B and C, methyluracil, and solcoseryl—are used to improve the trophism of the gastric mucosa. For combined megaloblastic anemia, additional injections of vitamin B12 might be necessary. During remission, physiotherapy and mineral water treatments could be utilized, while sanatorium-and-spa treatments are recommended.

Although treatment principles for type B gastritis often consider its association with Helicobacter pylori infection, the primary treatment focuses on eradicating this infection. Initial treatment involves prescribing Diet No. 1, which reduces mechanical and chemical effects on the gastric mucosa, along with increasing food intake to 4-6 times a day. For severe pain, antispasmodics like drotaverine (drotaverine-KMP, no-shpa), halidor, and papaverine may be prescribed. In certain cases, anticholinergics like atropine or buscopan prove effective. For elevated gastric juice acidity, selective M-anticholinergics and antisecretory drugs like pirenzepine (gastrocepin) are prescribed for up to 4 weeks.

For preschool children, the tablet form dosage of the drug is 12.5 g twice a day, and in school-age children, it's increased to 25 g twice daily. Histamine H2 receptor blockers like famotidine and ranitidine are typically prescribed for a duration of up to 2 weeks. Children over 10 years old might be given famotidine at a dosage of 0.02-0.04 g at bedtime. After the antisecretory therapy course, complex antacids such as

phospholugel or preparations containing preservatives like almagel, almol, or maalox are used. Diosmectin (smecta) can be prescribed in an aqueous solution at 6-9 g per day for children over 2 years old. To maintain treatment effectiveness and alleviate residual effects like dyspepsia and pain syndrome post-eradication therapy, cytoprotectors like sucralfate (Antruxal, Venter) are administered. The sucralfate dosage for children is typically 0.5-1.0 g four times daily, preferably with one dose at night, for a month. Sea buckthorn oil and multivitamins can be used for up to 3-4 weeks to enhance the trophism of the gastric mucosa. Tranquilizers such as diazepam (seduxen, sibazon), tazepam, and others are used for up to 2-3 weeks in complex therapy. Soothing agents like valerian extract and persen are also effective.

For type C gastritis focused on motor disorders, duodenogastric, and gastroesophageal reflux (reflux gastritis), medications like metoclopramide (raglan, cerucal) might be prescribed to normalize cardiac sphincter closure and reduce gastroesophageal reflux. Metoclopramide also aids gastric emptying and enhances gastric mucosa resistance to mechanical stress, although rare side effects like hyperkinetic phenomena, drowsiness, tinnitus, and dry oral mucosa can occur. Domperidone (motilium) is another option to normalize gastric motility, known for its milder effects compared to cerucal with minimal side effects. Phosphalugel can be prescribed to neutralize bile's aggressive effect on the gastric mucosa by not only providing antacid action but also adsorbing bile acids and covering the mucosa. Sucralfate (Incruzal, Venter, Ulgastran, Sucrase) offers cytoprotective effects by forming complex compounds with tissue proteins near the damaged mucosa, providing resistance to the acid-pepsin factor, and adsorbing pepsin and bile acids. Diosmectite (smecta) also exhibits cytoprotective effects. However, synthetic prostaglandin cytoprotectors like cytotex and arboprostil, while effective in reducing gastric secretion and stimulating regenerative processes, are limited due to potential dyspepsia, side effects on the reproductive system, and allergic reactions. Hence, they are used cautiously, primarily in adolescents with erosive gastritis. Isolated hCG or duodenitis in children is rare, whereas gastroduodenitis is more commonly encountered according to leading pediatricians.

Chronic gastroduodenitis (CGD) - a chronic, recurring inflammatory condition characterized by non-specific structural changes in the mucous membranes of the stomach and duodenum, involving

dystrophic, inflammatory, and regenerative alterations in the glandular apparatus. This condition represents the most prevalent form of chronic gastroduodenal disorders, accounting for 58-74% of stomach and duodenal diseases. When diagnosing CGD, the presence of risk factors and hereditary tendencies toward its development should be considered.

The clinical course of CGD varies based on the stage and manifestation of the inflammatory process, the stomach's secretory function, and motor-evacuation issues in the stomach and duodenum. Clinical syndromes like pain, dyspepsia, and signs of intoxication typically arise during exacerbations, akin to peptic ulcer disease. While the clinical symptoms resemble those of peptic ulcers, the pain pattern is non-seasonal, with night pains being infrequent.

Pain syndrome. The most characteristic pains in the abdomen, they are dull, prolonged, begin in the morning on an empty stomach and 1.5-2 hours after eating. Often acute, spastic, short-term pain localized in the epigastric region, under the right costal arch, around the navel. Pain worsens after eating and physical activity. With erosive CHD with high acidity, late night pains in the lower abdomen are observed. Palpation reveals diffuse pain in the epigastric region, a positive Mendel's symptom in the pyloroduodenal zone, with erosion - local muscle tension.

Dyspeptic syndrome: frequent belching, heartburn, nausea, feeling of heaviness after eating, bitterness in the mouth, flatulence, constipation, and in rare cases, intermittent diarrhea.

Intoxication syndromes are characterized by emotional instability, frequent headaches, irritability, general weakness, and asthenia.

In the treatment of CG and CGD in modern conditions, it is necessary to perform adequate paraclinical examination methods for adequate diagnosis and treatment:

Laboratory research:

a) mandatory (one-time):

— clinical blood test;

— clinical analysis of urine;

— total protein and protein fractions of blood;

— Tests for Helicobacter pylori (rapid urease, bacteriological, respiratory urease test, serological (ELISA), fecal analysis to determine the concentration of HP antigen, PCR);

b) if necessary:

— Examination of feces for occult blood (Gregersen's reaction);

— Histological (cytological) examination of biopsy specimens by the method of histological diagnostics - the "gold standard";

— immunogram; instrumental studies and diagnostic criteria;

Required:

— fibroesophagogastroduodenoscopy, targeted biopsy and express diagnostics of HP (for erosive CHD - twice);

— intragastric pH-metry (or fractional study of the contents of the stomach) - once;

— Ultrasound of the abdominal organs - once to determine concomitant pathology,

If necessary:

— X-ray examination of the stomach and duodenum (motor and evacuation disorders, developmental anomalies);

— rheography;

— Additional examinations in accordance with the nature of concomitant pathology.

The fundamental principles of treatment typically align with those applied in chronic hepatitis therapy. These principles hinge on the disease stage, the specifics of clinical and endoscopic alterations, as well as the condition of stomach secretory functions and motor-evacuation patterns in the stomach and duodenum. It's crucial to decide on the treatment setting (inpatient or outpatient) and regulate physical activity during exacerbations. Dietary prescriptions consider the patient's condition, often involving Table No. 1 or No. 5.

Complex therapy includes:

— If HP is present: anti-PH eradication therapy (usually within 7 days);

— Antisecretory medications, such as histamine H2 receptor blockers, are typically prescribed for 2-3 weeks, alongside selective M1 anticholinergics like pirenzepine for 4 weeks. Supplementary use of cytoprotective agents and effective antacids with sorption properties might be necessary for 10-14 days. Smecta, administered as 1 sachet 3-4 times daily, could also be included when needed.

— Prokinetics (domperidone) are prescribed for reflux and duodenostasis - for 10 days.

— antispasmodics (drotaverine, papaverine, metacin) - 7-10 days;

— Sedatives, tranquilizers, and herbal remedies are commonly prescribed as part of the treatment regimen.

— Following the cessation of antisecretory drugs, reparative agents like smecta, sucralfate, liquitron, and sea buckthorn oil are administered for a duration of 4-6 weeks. In cases involving pancreatic damage, enzyme preparations are utilized. For adolescents, intestinal antispasmodics (such as dicetel, pinaverium bromide), laxatives (like macrogol), and other appropriate medications are prescribed if constipation occurs. During exacerbations, physiotherapy methods such as electrotherapy and heat therapy are applied. Laser and magneto-laser therapies aid in normalizing stomach and duodenal motor function while enhancing gastric mucosa trophism. Reflexology is also utilized as a non-pharmacological treatment method. In clinical remission periods, herbal medicine, balneotherapy, physiotherapy, exercise therapy, and non-traditional therapies yield positive outcomes. Typically, the average hospital stay lasts for 21 days (extending to 28 days for erosive CHD cases). Continuation of treatment at a local gastroenterological sanatorium is recommended.

Dispensary observation for CG and CGD should occur at least twice a year for 5 years post-treatment initiation. Patients undergo evaluation by a pediatrician every 6 months and by a pediatric gastroenterologist annually. Fibrogastroduodenoscopy is conducted once a year, while in cases of erosive CHD, the frequency of examinations increases to 3 times a year, with biannual endoscopic assessments. Discharge from dispensary care for individuals with CGD or CG is considered following 5 years of complete clinical and radiological remission.

DUODENAL ULCER

When analyzing the rise in digestive organ pathologies, two essential factors merit consideration: firstly, the escalation of diseases due to genuinely adverse influences, and secondly, the advancements and widespread adoption of new diagnostic methodologies (such as gastroduodenofibroscopy, biochemical and histological analyses of mucosal biopsies, intragastric pH-metry, ultrasound, etc.) leading to increased disease registration. Considering the significance of risk factors in the disease's pathogenesis, a table containing information on risk group indicators (DC - Diagnostic Coefficients) for gastroduodenal pathologies, including peptic ulcers, has been devised.

TABLE OF INFORMATION OF SYMPTOMS OF GASTRODUODENAL PATHOLOGY

signs	DK
1. Toxicosis of pregnancy.	+0.75
2. Mothers over 30 years old.	+1.93
3. The work of the mother in the state work	+0.12
4. Type of baby feeding in the first year: Natural Artificial	 -1.11 +0.46
1. Violation of the diet	+0.17
2. Dry food	+0.55
3. late dinner	+1.43
4. The presence of diseases of the digestive system in relatives	+3.71
5. stressful situations	+2.43
6. Emotional lability	+2.93
7. Attitude of parents towards children: Hard treatment Weakness	 +2.58 -2.12
8. Frequent illnesses of the child	+2.94
9. Dysentery	+1.76
Helminthiasis	+1.76
Dental caries	+3.20
Chronic tonsillitis	+0.61

The table indicates that the most revealing symptoms (DC) include late dinner (DC + 1.43), family history of digestive system ailments (DC + 3.71), stress (+ 2.43 DC), among others.

Discussing infant nutrition requires consideration of the specificities of Uzbekistan's dietary habits. Notably, the improper calorie distribution throughout the day, with lower-calorie meals consumed at noon and a concentration of 40-50% of daily calories in the evening instead of the recommended 15-20% (in dishes like pilaf, manti, kebab, etc.). Overeating in the evening with a full stomach poses a significant burden on the digestive system, contributing to gastroduodenal issues.

Among the serious conditions within gastroduodenal pathology is duodenal ulcer (12-13 cases per 1000 children). Without timely

prevention and treatment, this ailment can lead to significant adversities and disabilities in adulthood.

There are many factors that lead to stomach and duodenal ulcers.

1. Alimentary factors: Eating disorders significantly impact the onset of peptic ulcer disease. Prolonged deviations from a healthy diet, overeating, consumption of unhealthy foods, or a monotonous diet can contribute. Extended fasting without food can trigger excessive gastric juice secretion, potentially damaging the gastric mucosa.

2. Stressful situations: Emotional stress, illness, and conflicts at home or school lead to brain excitation, creating a cycle of impulses between the brain and stomach. This continuous loop is evident when students returning to school after summer break show increased vulnerability to illness in the fall, highlighting stress's role in disease onset.

3. Hormonal factors: Clinical and experimental studies emphasize the importance of pituitary-adrenal system changes in peptic ulcer development. Glucocorticoids, for instance, can elevate gastric juice production and, in some cases, contribute to stomach bleeding.

4. Role of hypoxia: There's substantial evidence in literature demonstrating the significance of hypoxia in duodenal ulcer development. Studies indicate that local blood vessel damage during wound formation might result in mucosal hypoxia, leading to ulceration.

5. Significance of dysbiosis: Research, including that by Zakirova B.I. and colleagues, supports dysbiosis as a significant contributor to gastroduodenal pathology, including duodenal ulcers. The intestinal microflora's activity correlates closely with the physiological function of digestive organs. The onset of the disease has been linked to the development of dysbiosis, prompting the use of eubiotics in pathogenetic therapy.

6. Hereditary factors: A comprehensive medical history often reveals digestive system diseases among parents and relatives of affected children. Observations by department staff indicate that 73% of patients have digestive system ailments among their relatives.

7. Role of Helicobacter pylori: Recent scientific evidence strongly supports the significant role of Helicobacter pylori in peptic ulcer development.

Classification of peptic ulcer of the stomach and duodenum in children
(A.V. Mazurin, 1984)
Supplemented in 2005 by V.F. Privorotsky and N. Luppoval

Stages:	**Flow:**	**Localization:**	**Form:**	**H. pylori infection**
1. exacerbations 2. Incomplete clinical remission. 3. Clinical remission.	1.discovered for the first time, 2. rare remission for more than 3 years, 3. continuously relapsing (remission less than 1 year)	stomach, duodenum - Bulb - Double localization in the postbulbar region	1. uncomplicated 2. complicated 1) bleeding 2) penetration 3) perforation 4) pyloric stenosis 5) perivisceritis)	1.HP-positive 2.HP-negative
Functional characteristics: Acidity and gastric motility: may increase, decrease and remain normal.				
Clinical and endoscopic stages of peptic ulcer: Stage 1 - fresh wound Stage 2 - the beginning of the epithelialization of the wound Stage 3 - wound healing in severe gastroduodenitis. Stage 4 - clinical and endoscopic remission				
Accompanying illnesses: - pancreatitis - esophagitis - cholecystocholangitis				

Clinical picture.

The clinical presentation of peptic ulcers is highly characteristic. Children primarily report experiencing pain, initially localized and enduring. This pain is mainly felt in the epigastric and pyloroduodenal regions. Another notable symptom is heartburn, often challenging for younger children to articulate accurately. Nausea, often accompanied by pain and vomiting, is also a prevalent symptom.

Vomiting might occur after meals, with subsequent improvement in the child's condition. Despite maintaining a reasonable appetite, affected children often eat less due to fear of vomiting. Constipation, characterized by rectal pain, is common among children with peptic ulcers.

During examination, children might assume a forced half-bent position. Abdominal palpation reveals pain in the epigastric region. Positive signs of Mendel-Boas-Oppel are observed. Children might exhibit irritability, nervousness, fatigue, disrupted sleep patterns, red dermographism, decreased blood pressure, and lowered heart rate—all indicative of autonomic nervous system disturbances.

Characterizing the disease in stages:

In stage 1, pain emerges 2-4 hours post-meals, often sudden and paroxysmal in 50% of patients, sometimes radiating to the lumbar region. Profuse sweating and slowed pulse may accompany the pain. Abdominal palpation reveals tenderness and tense abdominal muscles. Nausea, heartburn, and constipation are prevalent. Endoscopic examination typically reveals a 6-8 mm round or oval ulcer in the mucous membrane.

Stage 2 differs from the first stage as pain mainly occurs during the day. Pain patterns following food intake or hunger (Moinigan's rhythm) are characteristic. Lower back radiation is less than in the first stage. Pain in the epigastric and pyloroduodenal regions is felt upon deep abdominal palpation. Abdominal muscle tension persists. Endoscopy may show signs of ulcer surface epithelialization, reduced redness around the ulcer, and clearing of the ulcer base.

In stage 3, pain occurs on an empty stomach, sometimes in the epigastric and pyloroduodenal regions. Moinigan's rhythm persists, but pain subsides after eating. Abdominal muscle tension diminishes, allowing for deeper abdominal palpation. Endoscopic examination reveals no defects or scarring.

Stage 4 patients are asymptomatic, exhibiting a satisfactory condition, with no observable endoscopic changes.

Complications of peptic ulcer.

According to numerous scientific studies, the most common complication arising from peptic ulcers is stomach bleeding, followed by stenosis of the pyloric part of the duodenum, penetration, and perforation. Bleeding episodes from stomach and duodenal ulcers might occur due to physical or emotional stress, dietary imbalances, or specific medications (such as aspirin or hormones). Often, abdominal pain precedes bleeding,

accompanied by vomiting resembling coffee grounds and dark stools. Affected children typically report malaise, weakness, and dizziness. Blood pressure (BP) and hemoglobin levels in patients decrease significantly, sometimes dropping to 20-40 g/L. Examining feces for occult blood is crucial in diagnosing peptic ulcers. Even minor bleeding can be identified using the Gregersen reaction.

Apart from bleeding, another noteworthy complication is pyloric stenosis. In this scenario, an ulcer in the narrow pyloric region forms a scar, leading to difficulty in food passage. Consequently, the stomach expands, causing food stagnation, constipation, and flatulence. The abdomen's upper part appears significantly enlarged upon examination. Patients often experience vomiting, with visible remnants of previously consumed food.

Insufficient digestion and improper assimilation of food lead to general weakness, weight loss in children, rendering them debilitated. Dry skin becomes noticeable, indicative of dehydration symptoms.

Penetration of ulcers is less frequent in children than in adults, occurring more often in severe cases. Symptoms include unrelieved vomiting, severe pain often radiating to the lumbar region. Penetration commonly occurs into the pancreas.

Ulcer perforation, though rare in children, exhibits symptoms like increased weakness, intensified abdominal pain, and dyspepsia before onset. Key signs of perforation include sudden severe epigastric pain, often causing shock. Symptoms such as weakened pulse, abdominal tenderness, loss of liver dullness, nausea, and irregular bowel movements ensue. Subsequently, peritonitis symptoms manifest with a board-like abdomen and a positive Shchetkin-Bleimberg sign.

Diagnosis of peptic ulcer is based on:

1. A comprehensive patient history.
2. Analysis of gastric juice (often hyperacid).
3. X-ray of the stomach and duodenum (identifying the symptoms of a "niche").
4. Gastroduodenofibroscopy (detection of a mucosal defect).
5. Detection in the biopsy of Helicobacter pylori.
6. Gregersen's reaction (positive for occult bleeding).

Differential diagnostics.

Chronic gastritis: Pain in the epigastric region intensifies after consuming coarse food. Abdominal swelling is present along with belching that smells rotten. Fluoroscopy of the stomach reveals mucosal hypertrophy or atrophy. Endoscopy shows mucosal hyperemia.

Cholecystocholangitis: Symptoms include elevated body temperature, bitter taste in the mouth, pain in the right hypochondrium, and a positive Ortner's symptom. Upon duodenal sounding, a significant amount of mucus, leukocytes, and epithelial cells are detected.

Pancreatitis: Constant and intense pain is observed in the left hypochondrium. Additional characteristic symptoms include bloating, heartburn, nausea, and unrelieved vomiting. Stools are oily, foul-smelling, and contain undigested food particles. Diastasis levels in the blood and urine increase.

Treatment.

The diet should aim to reduce gastric acidity, enhance digestion, and expedite ulcer healing. For this purpose, the diet should be chemically, mechanically, and thermally appropriate. Gentle nutrition involves excluding or limiting foods containing coarse plant fibers that stimulate gastrointestinal secretion and peristalsis. It also involves special culinary processing, adhering to appropriate temperatures, and adopting a fractional eating pattern.

When planning dietary guidelines for children with gastroduodenal issues, it is essential to:

- provide for the compliance of the nutritional and energy value of nutrition with the age needs of the child;
- provide mechanically, chemically and thermally sparing treatment for the diseased organ;
- prevent unreasonably long use of sparing diets and restriction of nutrients;
- observe the principle of gradualness when expanding nutrition;
- strictly monitor the observance of the diet, ensure the variety and high taste of ready meals.

Diet No. 1a, the most gentle, is prescribed during the acute phase of severe gastric or duodenal ulcers, typically limited to 1 to 7 days. This diet minimizes dietary fiber content and emphasizes milk and unleavened cottage cheese. All meals are steamed, boiled, mashed to a puree

consistency, served warm (30-40°C), and administered in a liquid or semi-liquid form, provided in 6-7 small portions throughout the day.

Avoid: Meat, fish, and mushroom broths; fried, spicy, and fatty foods; hard animal fats; smoked items; spices; pickled or marinated foods; dairy and carbonated beverages; bread; raw vegetables; untreated fruits and berries; nuts; mushrooms; coffee; cocoa; chocolate.

Recommended: Slimy soups based on vegetables and grains; mashed milk-based porridge (excluding millet and barley); mashed cottage cheese; steamed omelets; steamed soufflé-style meat and fish dishes; milk-based fruit and berry jellies and mousses; rosehip broth. Use butter and vegetable oil only in prepared dishes.

Diet No. 1b, less restrictive, is intended for the healing phase post-inflammation in the stomach, duodenum, reflux esophagitis, or after abdominal surgeries for 10-14 days. Diet No. 1b slightly relaxes limitations on chemical and mechanical stimuli while maintaining a controlled dietary regime. The diet's energy and nutritional value are enhanced by expanding the range of allowed foods, yet it remains modest compared to normal dietary requirements.

Include: Crushed wheat crackers; homogenized vegetable and fruit purees suitable for infant food; ripe fruits and berries after heat treatment; sweet fruit and berry juices diluted with boiled water (1:1 ratio); steamed meat and fish in the form of cutlets or meatballs; replacing slimy soups with pureed versions. All meals are still prepared through steaming, boiling until soft, and then crushed, kneaded, or rubbed to a puree consistency, served warm. Maintain a fractional meal pattern.

During convalescence and periods of disease remission, a moderately gentle Diet No. 1 is recommended, aligning with the physiological norm in energy value and chemical composition. The diet expands to include individually boiled vegetables (excluding white cabbage, turnips, radishes, radishes, and legumes), sweet ripe fruits, dried wheat bread, and select confectionery. However, broths, fried, fatty, spicy foods, smoked meats, soft bread, and pastries remain excluded.

Diet No. 1 offers two variants: mashed and non-mashed dishes.

The initial version of Diet No. 1 is prescribed for patients in the convalescence phase after an acute period of peptic ulcer, typically lasting around 1 month. As patients' conditions improve, there's a gradual transition from mashed to non-mashed foods. The diet plan aims to augment the variety of dishes from Diet No. 1b with mashed soups

prepared from a variety of vegetables (excluding cabbage soup and borscht) and cereals or noodles. Boiled vegetables with tender fiber (such as zucchini, cauliflower, Brussels sprouts, tomatoes, carrots, and beets) are included as side dishes. Consumption of dried wheat bread is permissible. All meals are prepared by steaming, boiling until soft, crushing, and mashing to a puree consistency, served warm.

Regarding medication, the treatment regimen should align with the etiopathogenesis of the disease, possibly including sedatives such as Novopassit, three tablespoons, Tenoten, Persen, Adaptol, among others.

For the eradication of Helicobacter pylori, the following course of treatment is recommended:

1. First line (omeprozole, omegast) 1 mg/kg
+ antibiotic clarithromycin (Cluber 15 mg/kg of milk - 2 times)
+ amoxicillin 50 mg/kg 2 times
2. second line:
Omez 1 mg/kg
+ antibiotic-tetracycline
+ metronidazole
+ bismuth preparations - Denol
The course of treatment is 7-14 days.

Antispasmodics: No-shpa 2% -1 ml intramuscularly, papaverine 2% -1 ml., intramuscularly, platifillin 0.2% -1 ml. intramuscularly.

Antacids - almagel, phospholugel, maaolox 1 tablespoon 3 times a day and 1 time at bedtime. Denol is taken 1 tablet 3 times a day.

Given the importance of hypoxia in ulcers, a violation of the redox process, the following drugs are prescribed. Solcoseryl 1ml. intramuscularly, 5% ascorbic acid, 4-5 ml. in / in. At the same time, it is advisable to prescribe the following metabolite complex:

Riboflabin mononucleotide - 1% -1.0
Lipoic acid - 0.5% -1.0
Cocarboxylase - 50-100 mg.
Calcium pantothenate-20%- 1.0

All this is mixed in 50 ml. with boiled and chilled water, and taken 1-2 hours before meals 1 time per day for 15 days, also prescribed nicotinamide 0.01-0.05 x 2 times a day, vitamin B6-5% 1-2 ml peros in the morning on an empty stomach or in / m.

To enhance the intestinal microflora, Enterogermin, Linex, and Lacto-Ji are prescribed. For sorbents, Carbolene is recommended at a dosage of 1 tablet taken three times a day, along with Filtrum-STI.

In gastroduodenal pathology, including duodenal ulcer, to improve the disturbed fat metabolism, zigir oil is prescribed. Zigir oil contains a lot of unsaturated fatty acids up to 300 mg%, copper and cobalt, so it is used as an antioxidant and immunomodulator. Zigir oil also accelerates the epithelization of the ulcer. Sigir oil is prescribed in the amount of 30 ml x 3 times a day.

In the uncomplicated course of peptic ulcer, physiotherapeutic procedures are widely used: electropheresis and inductothermia in the abdomen, novocaine, papaverine or platifillin relieve pain, reduce inflammation.

Clinical examination.

Children with peptic ulcer should be registered with the "D". Dispensary observation is carried out for at least 5 years from the onset of the disease.

FUNCTIONAL DISEASES OF THE STOMACH AND INTESTINE

The most common functional diseases of the gastroduodenal zone in children include:

- Functional dyspepsia
- Aerophagy
- Functional regurgitation

Functional dyspepsia.

Functional dyspepsia in children is an irritation of the gastric mucosa, which manifests itself in conjunction with a violation of the ability of the stomach to ferment (the breakdown of organic substances under the influence of enzymes). The symptom complex of stomach dysfunction is manifested by pain and discomfort in the epigastric region. If it hurts under the right and left ribs, this is not a sign of dysfunction. When discomfort occurs, there may be severe pain. The patient may complain of heaviness in the epigastric region, heartburn, sometimes nausea, morning discomfort that develops after eating.

Epidemiology.

Recurrent pain is more common in school-age children, 20% of cases predominantly in older children, in girls 5% and in boys 3% of cases.

Etiology and pathogenesis.

Recurrent pain in many children arises from functional dysfunction caused by dietary issues like irregular meals, consumption of dry and high-fat foods, smoked meats, and excessive carbohydrates. Dysfunctional disorders can also stem from psychosocial factors such as the loss of loved ones, family stress, conflicts, and poor social conditions.

These dysfunctions may lead to disruptions in gastric secretion, affecting the islet apparatus and causing excessive HCl secretion. Additional contributors include the overuse of non-steroidal anti-inflammatory drugs, smoking, and Helicobacter pylori-induced gastritis.

Motor-evacuation dysfunction of the stomach can lead to gastroparesis. Reports indicate that 75% of patients with gastrointestinal tract dysfunction experience functional dyspeptic complaints.

Gastroduodenal motility disorders.

1. Gastroparesis (violation of the motor-evacuation function of the stomach).

2. Violation of antraduodenal coordination.

3. Violations of the motor function of the antrum.

4. Violations of the distribution of food in the gastrointestinal tract (disturbances in the relaxation of the stomach, disturbances in the accommodation of food in the stomach).

5. Violations of the stage of gastric cyclic activity.

There are 3 options:

1. Ulcerative

2. Dyspeptic

3.Non-specific

Clinical signs: pain in the epigastric region typically arises on an empty stomach, occasionally occurring at night. The dyspeptic variant is characterized by discomfort in the upper abdomen; children may complain of pain after eating or during meals, along with symptoms like nausea, heartburn, and anorexia. If the observed symptoms in the patient do not align with the first two options, the third, i.e., non-specific option, can be considered.

Diagnostics. The diagnosis of FD is established on the basis of the study and analysis of symptoms, anamnesis of the disease, the results of a physical examination of patients, as well as data from a laboratory and instrumental examination, in essence, by excluding organic diseases in which symptoms of dyspepsia occurred, i.e., excluding organic dyspepsia. Diagnosis is carried out with the help of FGDS, ultrasound, radiography with barium, the nature of nutrition, the study of feces.

Differential Diagnosis: carried out with chronic gastritis, dystrophic changes, peptic ulcer.

Treatment: the primary objective in treating FD patients is to enhance their objective and subjective well-being, alleviating pain and resolving dyspeptic disorders. Treatment involves adopting general measures to normalize lifestyle and diet, using medications, and in some cases, employing psychotherapeutic approaches.

The approach to treating FD should be comprehensive and tailored. As motor disorders are frequently at the core of FD pathogenesis, the preferred medications include prokinetics and gastrointestinal motility regulators. Additionally, evidence-based medicine advocates the use of antisecretory drugs (proton pump inhibitors), antacids, anti-Helicobacter therapy, neuromodulating agents, enzymes, and biological products.

When abdominal pain prevails in the clinical presentation, priority should be given to antacids, proton pump inhibitors, and their combinations. Antacids operate via an integrative mechanism, not solely based on their acid-neutralizing effect but also due to their coating effect, reducing mucous membrane hypersensitivity, and their ability to absorb bile acids and lysolecithin.

Aerophagia

Aerophagia, characterized by the excessive swallowing of air, either voluntarily or involuntarily, leading to subsequent eructation, causes functional indigestion. The build-up of excess air exerts pressure on the stomach walls, resulting in discomfort and pain.

Clinic: difficulty in eating, anxiety, box sound in the epigastrium, vomiting of unchanged milk 5-10 minutes after feeding. With the help of X-ray examination, you can detect the amount of gases in the digestive organs, as well as follow the external changes in the internal organs.

Swallowing air in older children is associated with eating or chewing gum.

Treatment: teach mothers how to breastfeed and not talk to adults while eating.

Functional regurgitation includes:

- Regurgitation is the backward flow of a small amount of food (uncurdled or partially curdled milk) from the stomach up the digestive tract, moving into the esophagus and subsequently into the oral cavity.

- Vomiting is the abrupt, involuntary emptying of the stomach. Nausea and vomiting serve as symptoms for various diseases and conditions. In the majority of cases, they do not present a significant threat to the body.

- Rumination is the generally involuntary regurgitation of small amounts of food from the stomach into the mouth, typically occurring 15 to 30 minutes after a meal. Often, it involves repeated chewing and swallowing without accompanying symptoms like nausea and abdominal pain. Rumination can be observed in children as young as 1 month old and is attributed to upper gastrointestinal tract issues, including weakness in the cardial part and the stomach's horizontal positioning.

Etiopathogenesis. the mechanism behind functional vomiting and regurgitation in one-month-old children involves several factors:

— Impaired coordination of swallowing and esophageal peristalsis

— Gastrointestinal tract peristalsis disorders

— Challenges in stomach evacuation

— Pylorospasm

Vomiting often accompanies neurotic reactions and intense emotional arousal triggered by various unpleasant experiences, alongside anorexia.

Causes and clinical signs: neurological pathologies, metabolic disorders (such as acetone vomiting, diabetes mellitus), hereditary diseases, parasitic infestations in the gastrointestinal tract, and infections contribute to these symptoms.

Diagnostics: diagnosis involves general analysis of blood and urine, coprological, parasitological, and bacteriological studies, along with biochemical blood tests to determine electrolytes, glucose, creatinine, uric acid enzymes, liver function, and hereditary diseases.

Treatment: antireflex, sedative, antispasmodic drugs, hinolitic riabon 0.4 ml 2 mg x 3 times a day after meals.

Esophageal Diseases.

Esophageal diseases in children, though not among the most prevalent digestive organ pathologies, exhibit considerable diversity. These encompass conditions associated with movement disorders (such as achalasia, esophageal spasm), those linked to reflux (cardia insufficiency, gastroesophageal reflux, chronic esophagitis), acute inflammatory and destructive diseases (acute esophagitis, esophageal burns, Mallory-Weiss syndrome), and anomalies and developmental defects (esophageal atresia, fistulas, stenosis, esophageal diverticula, diaphragmatic-esophageal hernia).

Dysfunctional Esophageal Disorders.

Impaired motility in the esophagus leads to two types of food passage issues. One occurs due to retrograde movement where food moves back up. These disorders arise from compromised esophageal peristalsis, typically hypermotor, clinically presenting as esophageal spasms and lower esophageal sphincter dysfunction—namely, sphincter insufficiency, seen in achalasia and cardia achalasia. Cardia insufficiency naturally predisposes to reflux diseases, predominantly gastroesophageal reflux and reflux esophagitis. These diseases are more common in older children and adults, while esophageal malformations and abnormalities are frequently observed in infants.

Achalasia of the esophagus

Achalasia is a rare idiopathic esophageal disease stemming from neuromuscular dysfunction, causing the absence of esophageal peristalsis and hypertonicity in the lower esophageal sphincter. The term 'achalasia' denotes a lack of movement. This condition arises due to an impairment in the movement of the cardiac sphincter, wherein the sphincter fails to relax promptly during food swallowing, disrupting food passage from the esophagus to the stomach. This occurs due to the blockage of motor impulses from the vagus nerve in the cardial esophagus. Regular sphincter spasms lead to disrupted esophageal peristalsis, reduced muscle tone, and persistent dilation in this area.

The most common symptoms of esophageal achalasia include dysphagia (difficulty swallowing), regurgitation, and retrosternal pain. Dysphagia is often the initial symptom, with breastfed infants showing discomfort during breastfeeding, kindergarten children experiencing slow feeding, and older children complaining of swallowing difficulty.

Swallowing, especially solids, may cause pressure in the cardiac region and discomfort around the xiphoid process.

Dysphagia is frequently accompanied by regurgitation (vomiting food) without nausea, occurring suddenly during or post meals. Vomit typically contains undigested food and may occasionally happen during sleep. Patients often exhibit a vagotonic type of neurocirculatory dystonia, characterized by bradycardia, arterial hypertension, sweaty extremities, and duodenal spasms.

From an anatomical standpoint, two achalasia variants are observed: subcompensated and decompensated. In the former, esophageal movement is hypermotile, with moderate or absent dilation. Symptoms like dysphagia, regurgitation, and pain are intermittent. In the latter variant, X-ray examinations reveal substantial esophageal dilation amidst a transition from hyperkinesis to hypokinesis.

The diagnosis primarily relies on X-ray and endoscopic studies. X-ray imaging depicts prolonged barium retention in the cardia, an enlarged and narrowed esophageal segment, forming a characteristic 'carrot tip.' Endoscopic examination shows esophageal dilation, sluggish peristalsis, and signs of mucosal inflammation.

Treatment: proper attention to nutrition is crucial, ensuring mechanically, chemically, and thermally processed foods rich in proteins and vitamins, easily digestible, and served in liquid or semi-liquid form. It's recommended to maintain a gap of at least 3-3.5 hours between dinner and bedtime.

Drug Treatment: local anesthetics like 0.25%-0.5% novocaine or anesthesin are administered before meals. Nitroglycerin under the tongue is prescribed for older children to open the cardial esophagus. Considering the pathogenetic origin, buscopan is recommended during periods of primary and esophageal hyperkinesis. However, prokinetic drugs such as cerucal, motilium, and sisapride are not advised as they tend to increase rather than reduce the tone of the cardiac sphincter.

Psychotherapy, incorporating autogenic training, hypnosis, etc., can yield favorable results when combined with physiotherapy techniques like baths, Shcherbak reflexology, and acupuncture.

Cardiodilation, a common symptomatic treatment involving balloon pressure on the esophageal sphincters, proves effective. If cardiodilation or bougienage proves ineffective, surgical methods such as Geller's

cardiomyotomy are considered. Favorable outcomes have been achieved in 70%-80% of patients

Esophagospasm.

Esophagospasm is an idiopathic diffuse spasm affecting the esophagus, characterized by intermittent yet robust and prolonged contractions. It involves segmental and diffuse spasms occurring in significant areas. In children, esophagospasm often exhibits a segmental nature, predominantly occurring in the distal sections or the lower 1/3 of the esophagus.

Mental factors, especially negative emotions, predominantly contribute to the etiology of this condition. Additionally, some patients experience spasms while consuming solid foods, drinking excessively cold water, or during forced feeding.

The primary symptoms of esophagospasm include chest pain and dysphagia. Intense pain is typically felt in the sternum or epigastric region, extending (in older children) along the chest's anterior surface, reaching the neck, lower jaw, and shoulder. Pain might initiate during eating or occur spontaneously, lasting for several hours or subsiding suddenly upon changes in body position or gargling with warm liquids like water or tea.

DISEASES OF THE SMALL AND LARGE INTESTINE

Chronic diseases affecting the small and large intestines are frequently observed, particularly in preschool children. These conditions pose significant medical and social challenges due to their prevalence, intricate diagnostic procedures, and severe implications for a child's development and growth. Intestinal diseases induce both functional and morphological changes, yet early-stage differential diagnosis of the disease remains infrequent.

Considering the anatomical and physiological peculiarities of the digestive system in young children, the pathological process often manifests concurrently in both the small and large intestines (enterocolitis). Conversely, in school-age children, distinct lesions of the intestines are more commonly observed.

CHRONIC ENTERITIS.

Chronic enteritis is a recurrent inflammatory-dystrophic condition affecting the small intestine, resulting in disrupted intestinal functions such as digestion and absorption, subsequently impacting various

metabolic processes. It ranks among the primary digestive system diseases in about 4-5% of cases.

Etiology.

The origins of chronic enteritis are diverse, presenting both primary and secondary causes.

— Bacterial infections (like dysentery, salmonellosis, yersiniosis, campylobacteriosis), parasitic infestations (giardiasis, cryptosporidiosis, trichuriasis, hymenolepiasis), or fungal diseases often instigate small intestine damage.

— Nutritional factors, such as a dry diet, excessive food intake, high carbohydrate and fat content, and inadequate protein, vitamin, and micronutrient intake, can significantly contribute.

— Recent identification of etiological factors includes exposure to poisons like heavy metals (lead, phosphorus, cadmium), certain medications (salicylates, glucocorticoids, NSAIDs, immunosuppressants, cytostatics, long-term use of specific antibiotics), and ionizing radiation (e.g., x-ray therapy).

— Congenital enzymopathies, intestinal malformations, immune disorders (local and systemic), food allergies, previous intestinal surgeries, and diseases affecting other digestive organs (primarily the duodenum, pancreas, and biliary tract) are also associated with chronic enteritis. Pinpointing a specific etiological factor in a child's chronic enteritis is often challenging, as multiple endogenous and exogenous factors may contribute.

Pathogenesis.

Irrespective of the cause, inflammation in the small intestine's mucous membrane ensues, evolving into a chronic condition due to compromised immunity and inadequate compensatory responses. This leads to disrupted enzymatic activity in intestinal glands, altered food movement pace, microbial proliferation, and impaired digestion and nutrient absorption.

Clinical picture.

The clinical presentation of chronic enteritis is varied and contingent upon the disease's duration, developmental stage, small intestine function changes, and concurrent illnesses. Two primary clinical syndromes emerge—local and general.

Local intestinal (enteric) syndromes manifest due to prewall (membrane) and cavity digestion irregularities, often causing flatulence, abdominal pain, rumbling, and diarrhea. Stools typically contain undigested food debris and mucus, with occurrences of either copious stool or constipation. Abdominal palpation reveals tenderness around the navel, positive Obraztsov's and Porges' symptoms, and in severe cases, the appearance of "pseudoascites." Bowel dysfunction symptoms frequently follow the intake of milk, raw vegetables and fruits, and confectionery.

The generalized intestinal (enteric) syndrome involves water-electrolyte imbalance, macro- and microelement malabsorption, and multi-organ damage due to the pathological process (malabsorption syndrome). This syndrome presents as fatigue, nervousness, headaches, varying degrees of weight loss, and impotence. Other noticeable signs include skin dryness, nail changes, glossitis, gingivitis, cracked lips, hair loss, blurred vision, vein fragility, and hemorrhages.

These symptoms primarily stem from polyhypovitaminosis and trophic disorders. In children under three, anemia and metabolic disturbances often lead to osteoporosis, bone fragility, and seizures. The degree of both local and general enteral symptom manifestation determines the disease's severity.

Diagnosis and differential diagnosis.

The diagnosis of this disease relies on patient history, clinical symptoms, and outcomes of instrumental studies. Differential carbohydrate loads with mono- and disaccharides, as well as tests with d-xylose, are conducted. Endoscopy is performed to obtain biopsies for a histological examination, aiding in precise diagnosis. Coprogram results reveal creatorrhea, steatorrhea, and amylorrhea.

Differential diagnosis involves distinguishing this condition from hereditary and acquired diseases that present with a malabsorption syndrome. These conditions include acute enteritis, the intestinal manifestation of cystic fibrosis, gastrointestinal food allergies, celiac disease, and disaccharidase deficiencies.

CHRONIC ENTEROCOLITIS.

Chronic enterocolitis is an inflammatory-dystrophic disease with a polyetiological nature, characterized by a combined lesion affecting both the small and large intestine.

Etiology.

The disease often develops after acute intestinal infections (such as salmonellosis, dysentery, escherichiosis, typhoid fever, viral diarrhea), helminthiases, protozoal diseases, severe eating disorders (prolonged irregular, insufficient, or excessive nutrition), and allergic reactions to food. Additionally, congenital and acquired enzymopathies, compromised immunity, stomach, liver, biliary tract, and pancreas disorders, intestinal malformations, dysbiosis, vitamin deficiencies, neurological and hormonal disorders, radiation exposure, and irregular use of medications, primarily antibiotics, can predispose individuals to the development of this disease.

Pathogenesis.

The complete pathogenesis of this condition remains incompletely understood. One assumption suggests that infectious agents disrupt the integrity of tissues within the digestive tract, leading to destruction or morphological changes in the cells. Consequently, genetically foreign antigens (Ag) form, inciting autoimmune reactions within the body. This triggers the accumulation of cytotoxic lymphocyte clones and the production of antibodies (AT) against these antigens, which are recognized as part of the body's own tissue in the digestive tract. Additionally, there is a deficiency in secretory IgA, which normally prevents the invasion of bacteria and allergens. Changes in the usual intestinal microflora can increase the permeability of the intestinal mucosa to microbial allergens, contributing to the development of chronic enteritis. Furthermore, dysbiosis, or the imbalance in the intestinal microbial community, is often associated with the progression of this disease.

Clinical picture.

The course of chronic enterocolitis typically follows a fluctuating pattern, alternating between periods of relapse and remission. During the relapse phase, the primary clinical symptoms revolve around abdominal pain and gastrointestinal disturbances.

Pain characteristics vary in nature and intensity. Children frequently report discomfort on the left or right side near the navel or in the lower abdomen. Pain can occur at any time, often in the afternoon or about two hours after meals. It intensifies before bowel movements, during physical activities like running or jumping, and while traveling. Dull, prolonged pain is commonly associated with small intestine involvement, whereas

severe pain is linked to large intestine affliction. Equivalent symptoms include constipation post-meals, food withdrawal (especially in young children), and selective eating habits.

Chronic enterocolitis exhibits alternating symptoms of diarrhea (in small intestine involvement) and constipation (in large intestine involvement). Diarrhea manifests as frequent bowel movements (5-7 times a day) with small, varied consistency stools—liquid, undigested food lumps mixed with mucus, often gray, shiny, frothy, and foul-smelling due to decomposition. Sometimes, the stool appears as 'sheep' or ribbon-like formations, potentially causing anal fissures and minor bleeding. Bloating, rumbling, and gas difficulty are common symptoms. In some cases, the disease may manifest a psychovegetative syndrome, encompassing fatigue, disrupted sleep, nervousness, and headaches. Despite these symptoms, complaints related to bowel dysfunction remain secondary. Prolonged disease duration can lead to weight loss, stunted growth, anemia, signs of vitamin deficiency, and metabolic irregularities (protein, minerals).

Diagnosis and differential diagnosis.

The diagnosis of chronic enterocolitis relies on a thorough medical history, the clinical presentation (prolonged intestinal dysfunction leading to dystrophy), laboratory findings (anemia, hypo- and dysproteinemia, hypoalbuminemia, reduced serum cholesterol concentration, total lipids, β-lipoproteins, calcium, potassium, sodium levels, presence of mucus, leukocytes, steatorrhea, creatorrhea, amylorrhea in stool samples), and results from various instrumental examinations (sigmoidoscopy, colonofibroscopy, radiological and morphological studies).

Differential diagnosis is essential to distinguish chronic enterocolitis from prolonged dysentery, congenital enzymopathies (such as cystic fibrosis, celiac disease, disaccharidase deficiency), and exudative enteropathy syndrome.

Treatment.

The treatment approach for chronic enteritis and chronic enterocolitis focuses on restoring bowel function and preventing disease recurrence. Therapeutic nutrition, typically following Pevzner's Table No. 4, is recommended as a foundational measure. Additionally, treatment involves the use of multivitamins, enzymatic preparations (such as Pancreatin), pre- and probiotics (like bifidobacteria bifidum + activated

carbon (Probifor), Linex, Lactobacillus acidophilus+Kefir grains (Acipol), Khilak-forte), enterosorbents (such as dioctahedral smectite), and prokinetics (like trimebutine, loperamide, mebeverine, etc.). When necessary, antibacterial drugs such as Intetrix, nitrofurans, nalidixic acid, metronidazole, etc., might be recommended. Additionally, phytotherapy, symptomatic therapy, physiotherapy, and exercise therapy are included in the treatment plan. It's advised to avoid spa treatments until 3-6 months after a relapse.

Consequences.

With timely and adequate therapeutic measures, the outcome at any stage of rehabilitation will be positive.

INFLAMMATORY DISEASES OF THE INTESTINE

Inflammatory bowel disease comprises a group of conditions characterized by superficial or transmural nonspecific immune inflammation of the intestinal wall.

Presently, diseases within this group include:

- Ulcerative colitis (UC);
- CD (Crohn's disease);
- Undifferentiated colitis.

UC is a chronic condition where the inflammatory process affects the mucous membrane (occasionally extending to the submucosa), predominantly involving various parts of the large intestine. CD, also known as intestinal granulomatosis or terminal ileitis, is a chronic relapsing disease marked by segmental transmural granulomatous inflammation affecting different sections of the digestive system. Due to similarities in epidemiology, etiopathogenesis, and clinical presentation among these diseases, early-stage diagnosis can be challenging. In such instances, the accurate diagnosis might be termed 'undifferentiated colitis,' signifying a chronic bowel disease characteristic of both Crohn's disease and ulcerative colitis.

ICD-10 code.

In the XI class 'diseases of the digestive system,' the K50-K52 block categorizes 'non-infectious enteritis and colitis,' encompassing types of inflammatory bowel diseases:

- K50. Crohn's disease (regional enteritis).
- K51. Ulcerative colitis.
- K52.9. Noninfectious gastroenteritis and nonspecific colitis.

Epidemiology.

Prevalence: UC accounts for 30 to 240 cases per 100,000 inhabitants, while Crohn's disease affects 10 to 150 cases per 100,000 individuals. These conditions demonstrate a consistent trend of affecting younger populations.

In Germany, approximately 200,000 people suffer from inflammatory bowel disease, with 60,000 being children and adolescents. Pediatric records indicate about 800 new cases of inflammatory bowel disease annually [Berens R., Buderus S., 2005].

The indicators registered in the Russian Federation do not differ from other countries in negative trends: higher mortality as a result of severe forms of the disease (3 times higher than in other countries), late detection of the disease (ulcerative colitis is detected only in 25% of cases in the first year of the disease), a large number of complicated forms of inflammatory bowel disease. As a result of late detection of the disease, life-threatening complications are observed in 29% of cases. Crohn's disease, detected within 3 years after its onset, gives a complication in 55% of cases, and late diagnosis in 100% of cases.

Classification.

At the World Congress of Gastroenterology in Montreal in 2005, the International Classification was adopted, replacing both the Vienna classification of Crohn's disease and the international classification of ulcerative colitis.

International classification of Crohn's disease

(Montreal International Congress of Gastroenterology, 2005)

Criteria	Index	Concept
Manifestation Age (ageat diagnosis)	A1 A2 A3	Up to 16 years old From 17 to 40 years old Over 40 years old
Localization (location)	L1 L2 L3 L4	Ileith Colitis Ileocolitis Individual lesions of the upper GI tract*
Flow (behavior)	B1 B2 B3 P	Non-stenosing No penetration Stenosing with penetration Perianal lesion**

* L4 can be added alone or with L1-L3 (upper and lower GI injury).

** Index "p", additional B1-B3, is introduced in case of damage to the perianal sphere.

International classification of ulcerative colitis

(Montreal International Congress of Gastroenterology, 2005)

Criteria	Index	concept	Concept
Prevalence	E1	Ulcerative proctitis	lesion of the distal region in the rectosigmoid region
	E2	left hand (distal) ulcerative colitis	lesion distal to the angle of the spleen
	E3	scattered ulcerative colitis (pancolitis)	colon completely damaged (proximal inflammation from the angle of the spleen)
severity (severity)	S0	Clinical remission	Symptoms are not observed
	S1	Easy	Diarrhea 4 times a day or less (presence of blood in the stool or not); there will be no system-specific symptoms; in the acute phase, the protein concentration is normal
	S2	Medium	Stool more than 4 times a day, and symptoms of chronic intoxication are minimal
	S3	Heavy	The number of stools 6 or more times a day and with an admixture of blood; The number of pulses is 90 or more per minute; Body temperature - 37.5 ° C and above; Hb - 105 g / l or less; ESR - 30 mm/h and above

Prevention.

Since the causes of inflammatory bowel disease development have yet to be established, specific preventive measures are not available. However, preventive efforts focus on maintaining a healthy lifestyle, combating detrimental habits, stress prevention, and adopting a balanced diet rich in nutritional fibers and essential substances. Screening for inflammatory bowel disease involves regular health monitoring of individuals with a hereditary predisposition to the condition, often including genetic tests among this group.

Etiology and pathogenesis.

The etiology of inflammatory bowel disease remains incompletely understood. According to modern concepts, it is considered a multifactorial disease, involving genetic predisposition, impaired immunoregulation, and an autoimmune component in its pathogenesis

The pathological process is rooted in immune system dysfunction, although the specific antigen responsible for these changes remains unidentified. These antigens might include bacterial antigens along with self-antigens. Subsequent effector mechanisms drive alterations in the body's immune response to antigenic stimulation, resulting in non-specific immune inflammation of the intestinal wall and mucous membrane.

A pivotal factor is the genetic predisposition to heightened intestinal wall permeability, causing a reduction in the intestine's barrier function against bacteria and toxins.

The compromised immune response is marked by the selective activation of various T-lymphocyte subpopulations and changes in macrophage activity. This cascade leads to the release of inflammatory mediators such as eicosanoids, activation of platelet factors, histamine, kinins, cytokines, and reactive oxygen species, ultimately contributing to tissue destruction.

Clinical picture.

The clinical symptoms of intestinal obstruction are grouped into several main clinical syndromes:

- irritable bowel syndrome;
- syndrome of extraintestinal changes;
- endotoxemia syndrome;

IRRITABLE BOWEL SYNDROME

The global prevalence of irritable bowel syndrome (IBS) ranges from 18% to 30%. However, a significant number of individuals with this condition do not seek medical attention, with only about one-third of cases being diagnosed through specialized examinations. While the typical age of onset for IBS is between 30 to 40 years, it can also affect children. Despite extensive research, a universally accepted terminology for IBS remains elusive. Current terms in use include 'irritable bowel syndrome' or 'irritable colon syndrome,' along with 'chronic spastic colitis.'

The term 'irritable bowel syndrome' was introduced by DeLore in 1967, but the historical roots of this condition trace back to the 19th century. W. Gumming detailed its clinical features in 1849, and later, in 1892, W. Osler described a similar condition as congenital colitis. Subsequently, the condition was referred to as 'spastic colitis' or 'intestinal neurosis.' However, the diagnostic term 'chronic spastic colitis' used at that time did not entirely capture the underlying pathological process, as it implied inflammation of the colon mucosa. Contrarily, histological findings in IBS indicate more dystrophic changes rather than classic signs of inflammation.

IBS significantly impacts the health of affected individuals, including children, necessitating continual improvements in treatment and rehabilitation procedures.

Etiology.

Irritable bowel syndrome (IBS) is a multifactorial condition characterized by various functional disruptions in motility, absorption, and secretion in the gastrointestinal tract. Several etiological factors contribute to its development, including adverse events, infections, medication exposure, and other triggers. Alterations in both the central and peripheral nervous systems play a pivotal role in the onset of IBS. Many patients experience somatoform disorders, presenting symptoms such as a sense of danger, fear, depression, hysterical reactions, and other psychiatric conditions.

Numerous factors can provoke the onset of IBS. Dysfunctions in the central and autonomic nervous systems, disturbances in the intestinal lymphatic system, changes in gut microflora (dysbacteriosis), disrupted nutrient absorption, psychological changes, inadequate consumption of dietary fiber, and various social and emotional stressors contribute to the

development of the disease. IBS involves both the small and large intestines in its pathological process. Certain infectious agents like shigella, hepatitis B virus, among others, have been identified as potential contributors to the etiology of IBS. After acute intestinal illnesses, functional issues affecting the intestines and stomach often arise, sometimes leading to colitis.

Presently, one of the primary etiological factors identified is a disruption in the microflora of the colon (intestinal dysbacteriosis). Long-term use of laxatives or regular enema usage may result in IBS by desensitizing hemo- and baroreceptors in the intestinal wall, causing reduced sensitivity. These conditions can lead to disturbances in the intestinal motor-evacuation function, causing transit time changes, manifesting as hyperkinetic (diarrhea) or hypokinetic (constipation) patterns. These alternating disruptions in bowel movements contribute to the disorder. Emotional stress plays a significant role not only in IBS but also in functional gastrointestinal disorders. Patients with IBS often exhibit signs of neurosis or psychopathy. Other contributing factors include the inability to absorb specific nutrients, irregular eating habits, nutritional deficiencies, high consumption of carbohydrates, and physical inactivity.

Pathogenesis.

In irritable bowel syndrome (IBS), psychoemotional factors influence changes in intestinal motility and sensitivity to neurohumoral and mechanical stimuli, forming a cyclic pattern. Notably, alterations in the transverse intestinal motor function play a pivotal role. Patients with IBS often exhibit an increased content of serotonin in the intestinal mucosa. Disturbances in serotonin distribution can alter nerve impulse conduction, leading to reduced intestinal contractions.

Studies employing biomechanical and electrophysiological methods have identified various types of IBS: hypersegmental hyperkinesis (present in 52% of patients), characterized by increased segmental contraction; high activity of dystonic hypo- or akinesis (observed in 36% of patients) with low-amplitude wave action; and antiperistaltic hyperkinesis (occurring in 12% of children), signifying elevated motor activity in the large intestine.

Enterocolosynthography studies of colon function in IBS reveal accelerated passage of intestinal contents in the distal colon but slowed transit in the proximal part. Accelerated passage is observed in the

sigmoid colon in 49% of pediatric cases and in the entire colon in 17% of cases. Pain impulses in IBS stem from the excitation of afferent nerve roots of the vagus nerve. Factors affecting these nerve endings influence intestinal secretory and motor function, leading to bloating or diarrhea. These pain signals are then transmitted to the brain. According to modern hypotheses, abdominal pain in IBS is linked to impaired perception and regulation of pain impulses within the central nervous system.

Classification.

According to the classification proposed by F. Weber and McCallum in 1992, irritable bowel syndrome (IBS) manifests in three primary variants:

1. Diarrhea-predominant variant (characterized by diarrhea as the predominant symptom)
2. Constipation-predominant variant
3. Flatulence and abdominal pain-predominant variant.

In IBS, stool characteristics can vary, ranging from whitish, hard, ribbon-like, and pellet-like (polyfecal) to very liquid consistency. Patients often experience urgent bowel movements due to dysfunction of the anal sphincter. In this condition, patients typically maintain stable body weight, although in rare cases, weight loss or gain may occur.

Clinic.

At an international working meeting in Rome in 1988, irritable bowel syndrome (IBS) was described as a complex of functional disorders lasting more than 3 months, encompassing major clinical signs such as reduction of abdominal pain post-defecation, flatulence, bloating, false urge to defecate, constipation, diarrhea, and their alternation. In milder forms of IBS, a latent course is observed in 1/3 of patients seeking specialist consultation, often spanning over 5 years since the onset of complaints. Carcinophobia is frequently reported among most patients, and the disease's history, variable complaints, and worsening patient condition are linked to psycho-emotional factors. Psychological changes in IBS patients vary, with risks of developing depression. Patients may experience migraine-like headaches, weakness, bad mood, drowsiness, and appetite disorders.

Abdominal pain is the primary symptom of IBS, varying in intensity from mild discomfort to colic, lasting from minutes to hours. Typically, pain is experienced in the lower left abdomen, occasionally spreading from one side to the other. Notably, abdominal pain and diarrhea are

absent during sleep. For 1/3 of patients, pain exacerbates after meals, irrespective of the nature of the pain, heightened by emotions, stress, or physical exertion.

Stool changes are common in IBS. Constipation may persist or alternate with diarrhea, influencing the colon's neuro-impulsive motility. Diagnosis of constipation involves observing constant straining during bowel movements, hard stools, and a sensation of incomplete bowel movement. Bowel movements occurring 2 times or less per week may indicate constipation associated with various diseases. Four types of constipation are identified:

1. Idiopathic constipation (impaired motor and evacuation function of the large intestine)
2. Constipation due to mechanical rectal obstruction
3. Medication-induced constipation
4. Colostasis (common in mental illness, craniocerebral injuries, and strokes)

Constipation can also result from various pharmacological drugs such as amitriptyline, antacids, antidepressants, non-steroidal anti-inflammatory drugs, diuretics, iron preparations, and cytostatics. Causes of constipation include mechanical, gastroduodenal, neurogenic, and metabolic-endocrine factors.

Diarrhea, persisting for more than 2 weeks and recurring, is a characteristic of IBS. Its manifestations vary, including diarrhea in a latent clinical form. Constipation accompanied by pain can progress to diarrhea.

Abdominal pain (80-90%) and diarrhea are the primary symptoms, while constipation occurs in 75% of IBS cases. These symptoms relate to impaired intestinal motility and heightened visceral sensitivity. IBS is classified into three variants based on the prevalence of abdominal pain, defecation disorders, and bloating:

— Abdominal pain and flatulence
— Constipation
— Diarrhea

In 1978, the clinical signs of irritable bowel syndrome (IBS) were initially described alongside the proposal of six clinical criteria. The Mann criteria, which include various symptoms, serve as primary diagnostic criteria distinguishing IBS from other diseases:

— Reduction of pain after defecation

— Experiencing pain during defecation
— Bloating
— Presence of mucus impurities in stool and false urge to defecate
— Sensation of incomplete bowel movement
— Frequent intestinal contractions at the disease's onset

The classic criteria for diagnosing IBS encompass symptoms such as abdominal rumbling, flatulence, decreased abdominal pain after defecation, altered defecation patterns, and significant intestinal motility disturbances. Patients often seek hospital admission due to severe abdominal pain, initially diagnosed as "intestinal colic." Pain localization varies and may extend to the chest, back, or lumbar region. These pains often correlate with defecation, decreasing or disappearing post-bowel movement.

Flatulence, often accompanied by strong gas emissions, may not be persistent and can occur temporarily, particularly after meals. Intestinal dysfunction is a key symptom, but children may not perceive it as significant. A doctor considers the shape, nature, and amount of a patient's stool and examines for the presence of mucus. Currently, the Bristol stool scale categorizes stools into seven variants based on visual characteristics, indicating transit speed. Types 1 and 2 represent slow transit, types 6 and 7 indicate accelerated transit, while types 3 and 4 are considered normal.

Clinical features of IBS include:

— A syndrome characterized by prolonged, subtle progression
— A variety of patient complaints
— Variable nature of complaints
— Deterioration of patient condition associated with psycho-emotional factors
— Absence of nighttime pain and intestinal disorders
— Lack of identifiable "risk signs".

Diagnostics.

In 1999, an international working group defined the clinical criteria for irritable bowel syndrome. It should be noted that in irritable bowel syndrome, in addition to clinical criteria, the main attention in the diagnosis should be given to "signs of risk". These signs will help to exclude organic bowel diseases:

1. history of bleeding from the rectum

2. weight loss
3. fever
4. nocturnal symptoms
5. the presence of inflammatory diseases or tumors of the colon in the intestines in the offspring.

When examining patients with irritable bowel syndrome, the general condition of patients is not disturbed, despite the abundance of symptoms of the disease. Be sure to pay attention to the presence or absence of "signs of risk" (presence of blood in the stool, fever, causeless emaciation, anemia and increased ESR) of this disease. Patients with irritable bowel syndrome undergo general and biochemical blood tests, sigmoidoscopy, colonoscopy and irigography. indications, a simultaneous biopsy with gastroduodenojunoscopy is carried out.In case of extraintestinal and retroperitoneal changes, ultrasound, computed tomography, angiography should be performed.In November 1999, an international consensus was adopted in Rome on the diagnostic criteria for irritable bowel syndrome.Diagnostic criteria, currently used in irritable bowel syndrome (Rome III criteria). Main signs: a state of discomfort or pain in the abdomen that occurs at least 1-3 days after a bowel movement, a decrease in pain after a bowel movement, accompanied by a change in the shape of the stool, tension during a bowel movement over the past 3 months indicates irritable bowel syndrome.

Signs confirming the diagnosis of irritable bowel syndrome:

— no stool for 1 week
— stool more than 3 times within 1 and
— hard stool
— mushy or watery stools
— Fake calls
— presence of mucus in the stool
— flatulence

Differential diagnosis.

The differential diagnosis of irritable bowel syndrome (IBS) depends on whether the patient experiences constipation or diarrhea. When observing diarrhea in IBS, it's crucial to differentiate it from various conditions such as infectious diarrhea, rectal mucosa diseases (ulcerative colitis, Crohn's disease), pancreatic insufficiency, short bowel syndrome, use of laxatives, rectal tumors, carcinoid syndrome, Zollinger-Elisson

syndrome, hyperthyroidism, lactase deficiency, celiac disease, food allergies, intestinal lymphoma, amyloidosis, and diabetic enteropathy.

During patient observation, differential diagnosis involves considering appendicitis, cholecystitis, cholelithiasis, peptic ulcers in the stomach and duodenum, and stomach tumors.

Crohn's disease and nonspecific ulcerative colitis share several clinical features (diarrhea 80%, Melena 50%, weight loss 85%, stunting 35%). However, their disease courses differ significantly. Crohn's disease tends to progress sluggishly with latent, concealed bleeding, and may present with anal and perianal issues like paraproctitis, fistula, or chronic deep fissures.

Dysentery manifests abruptly with rapid onset of intoxication symptoms, showing positive response to antibiotic treatment. Endoscopic examinations in dysentery reveal ulcers and erosions without scarring. On the other hand, nonspecific ulcerative colitis usually develops gradually, often marked by blood in stool initially, occasionally followed by grayish, liquid stool. Mucus and blood might appear in stool after 1-2 months. The endoscopic picture in nonspecific ulcerative colitis varies based on disease degree and activity. During flare-ups, the intestinal mucosa swells, reddens, and loses its vascular network, often demonstrating contact bleeding.

Treatment.

The treatment of irritable bowel syndrome (IBS) emphasizes improving the psycho-emotional state, adopting a healthy diet, increasing physical activity, and utilizing physiotherapeutic measures to normalize intestinal functioning and motility. Dietary modifications involve reducing carbohydrate intake, limiting milk consumption, fiber-rich foods, pickled or smoked items, and carbonated beverages.

For constipation, replacing sugar with xylitol or sorbitol is recommended, alongside consuming carrots, radishes, figs, and honey. If lifestyle changes aren't effective, drug therapy is considered.

To alleviate IBS pain, the following medications are suggested:

— Enkephalin receptor stimulants like trimebutine (debridate) in varying dosages based on age.

— Calcium channel blockers such as dicetel or spasmomen taken 1 tablet 2-3 times daily.

— Antispasmodics for smooth muscles like spasmomen, no-shpa, papaverine at similar intervals.

— Anticholinergics like platifillin, buscopan, or belladonna preparations. For flatulence, simethicone (espumizan, Sub-simplex in tablet or drop form) 2-3 times a day is recommended to break up gas bubbles in the intestine.

Constipation in IBS may require:

— Fiber supplements like Mucofalk, psyllium.

— Osmotic laxatives such as forlax or lactulose.

— Prokinetics like cisapride.

For predominant diarrhea in IBS, consider:

— Adsorbents like activated carbon, smectite, cholestyramine.

— Astringents such as tannins, birch, oak bark, blueberries, or bird cherry fruits.

— Antidiarrheal medications like loperamide or imodium.

Alterations in intestinal biocenosis are common, so normalization of microflora with probiotics (bifiform, linex, enterol) and prebiotics (lactulose 5-10 ml) for a 3-4 week course is recommended. Hemicellulose-containing enzyme preparations (Festal, Digestal) at 1 tablet 3 times a day for 1-3 months can yield good clinical results.

If IBS is accompanied by depression symptoms, antidepressants (e.g., fevarin 50 mg nightly) for up to 1-6 months may be prescribed. For pronounced vegetative dysfunctions, consider herbal remedies.

There's no definitive 'gold standard' for IBS treatment. Bifidobacteria deficiency and opportunistic microorganism growth alter intestinal enzymatic properties. To enhance intestinal microbiota, incorporating bifidus and lactobacilli-containing supplements is advised. Anticholinergic and antispasmodic drugs are also part of the treatment program. Cholestyramine is used if bile acid absorption is disrupted with diarrhea. Remedy responses for constipation vary; calcium ion antagonists are successful in normalizing colon peristalsis. Serotonergic drugs, targeting specific receptors, are widely used in treatment.

Dispensary observation.

Dispensary observation spans three years. After a comprehensive clinical and instrumental examination, if no pathological changes are detected, the patient is deregistered. The patient undergoes evaluation by a pediatric gastroenterologist twice yearly and by a pediatrician once

every three months. Assessments by other specialists are conducted based on specific indications. Anti-relapse treatment is administered biannually.

Prognosis.

Timely diagnosis and treatment are crucial for children with irritable bowel syndrome. Managing these patients demands significant attention from the doctor, often necessitating collaborative consultations with a psychoneurologist. This condition warrants physiotherapy, intestinal hydrotherapy, and spa treatments for effective patient care.

PANCREATITIS

The timely diagnosis and treatment of pancreatic diseases in children represent one of the most challenging issues in clinical gastroenterology. A critical aspect involves differentiating between primary and secondary lesions affecting the pancreas.

Secondary pancreatitis is currently described using various terms such as 'secondary pancreatitis,' 'reactive pancreatitis,' or 'dyspancreatism.'

Functional changes in the pancreas related to gastroenterological disorders range from 10% to 80%. Dyspancreatism is frequently associated with gastroduodenal pathologies:

— 25% in cases of gastritis

— 57% in instances of gastroduodenitis

— 82% in cases of peptic ulcers in the stomach and duodenum.

Clinical picture.

The disease exhibits an atypical progression. Intense pain radiates from under the right rib (associated with biliary pathology) and extends from the pyloroduodenal region (related to duodenal disease) to the left hypochondrium.

Dyspeptic symptoms include nausea, vomiting, belching, bloating, and alternating episodes of loose stools and constipation. Additionally, an asthenovegetative syndrome is observed.

In chronic pancreatitis, specific pain points are identified:

— Desjardins Point: Located 6 cm above the navel along the line connecting the right armpit.

— Kacha Point: Positioned 4-7 cm above the navel at the outer edge of the left rectus abdominis muscle.

— Mayo-Robson Point: Positioned at the juncture of the upper and middle thirds of the line connecting the navel to the middle of the left costal arch.

— Chauffard Zone: Originating from the Desjardins Point, a perpendicular line extends to the midline, defining a conditional triangle that corresponds to this area. Tenderness in this zone typically indicates inflammation around the pancreatic head.

— Palpation often elicits pain in the Chauffard, Mayo-Robson, Kach, and Desjardins areas.

The disease course may vary in the severity of symptoms and is characterized by pancreatic lesions associated with gastritis, duodenal disease, gallbladder, and its ducts.

Chronic pancreatitis.

Chronic pancreatitis is a prevalent condition characterized by persistent inflammation of the pancreas.

Etiological factors include:

— Acute gastrointestinal infections

— Inflammatory conditions affecting the gallbladder and biliary tract

— Dyskinesia of the biliary tract

— Chronic gastrointestinal diseases

— Persistent infection foci in the nasopharynx

— Infestation by worms

— Giardiasis affecting the biliary tract and intestines

— Allergic diseases and related conditions

— Non-compliance with dietary guidelines

— Trauma

— Familial history of the condition

— Metabolic disorders (such as obesity)

— Developmental delays in mental and physical growth during infancy

— Excessive weight.

Clinical picture.

The disease manifests with paroxysmal pain localized in the upper abdomen, above the navel, occasionally extending under the left rib. The pain is girdling and cramp-like in nature, frequently radiating towards the

lumbar region, left chest, left arm, and left lower limb. Tender areas include the projection of the pancreas and biliary tract, the Choffard-Rive zone in the anterior abdominal wall, and the possibility of palpating muscle resistance in the epigastric region (Kerte's symptom). Additionally, Mayo-Robson's symptom presents as pain upon palpation in the left costovertebral corner. Positive Bergman and Kalka symptoms involve experiencing pain upon percussion along the left costal margin.

During an episode, the child is restless, finding no comfortable position. Pain slightly subsides when lying on the stomach or with bent legs towards the left side. Nausea, vomiting, flatulence, and either constipation or loose stools occurring once or twice a day are common. The tongue appears dry and covered with a white coating. Patient temperatures usually range from subfebrile to normal, occasionally reaching 38-39.50 C.

Laboratory data.

The stool analyses reveal numerous muscle fibers (creatorrhea), starch, fatty acids, and mucus. Other observations include polyfecal matter, steatorrhea, and malodorous stools. Additionally, this pathology is associated with an elastase level exceeding 150 μg/kg.

Biochemical studies indicate hyperamylasemia (16-30 g/l), hyperamylasuria (28-160 g/l), lipasemia (13-60 g/l), and increased trypsin activity ranging from 25-30 g/l. Peripheral blood tests reveal leukocytosis, eosinophilia, thrombocytopenia, and elevated ESR.

Instrumental Research.

Fluoroscopy conducted during an attack indicates the presence of gases in the intestinal cavities. Ultrasound examination revealed swelling of the head of the pancreas.

Substantiation of the diagnosis.

The diagnosis is established based on patients presenting with pain above the navel and radiation towards the left side, along with pancreatic insufficiency syndrome symptoms such as creatorrhea, steatorrhea, amylorrhea, elevated levels of amylase in serum and urine, increased trypsin activity, and the results obtained from the pancreamisin test.

Treatment:

1. Provide functional rest or reduce the secretory function of the pancreas.
2. Implement detoxification therapy and address metabolic disorders.

3. Eliminate toxic products from the body.
4. Alleviate pain, relax the pancreas, and improve secretion outflow.
5. Apply desensitizing therapy.
6. Administer anti-enzymatic treatments.
7. Implement measures to prevent secondary damage or infection.
8. Restore the external secretion function of the pancreas.
9. Employ general strengthening therapeutic measures to enhance the body's immune system.
10. Utilize phytotherapy.

Diet.

- Day 1: Fasting with water intake (Borjomi, Essentuki-4); parenteral feeding for the patient.
- Day 2: Unsweetened tea, dry bread, porridge cooked in water.
- Day 3: Porridge cooked in skim milk, tea, dried fruits.
- Day 4: Porridge with butter.
- From Day 5: Mashed potatoes and carrots.
- On Day 7-8: Introduction of berries and cutlets.

Treatment plan:

1. Decreased pancreas secretion:
- Anticholinergic drugs: atropine, platfillin, methacin.
- Carbonic anhydrase inhibitors: hypothiazide, furosemide.

2. Detoxification therapy:
- Days 1-2: 10% glucose solution (200-400 ml) with insulin. Also recommended: Reopoliglyukin, Ringer's lactate, polyglucin, albumin, infuzol, and vitamins.
- Glucocorticoids used: methylprednisolone, hydrocortisone, dexamethasone.
- Antispasmodics and analgesics: no-shpu, papaverine, halidor, analgin, baralgin, spazmalgon, duspatalin.

3. Desensitization:
- Drugs include diphenhydramine, pipolfen, suprastin, tavegil, diazolin, and loratadine.

4. Biologics:
- Prescribed: duphalac, enterozhirmin, lactobacterin, linex.

5. Anti-enzymatic therapy:

- Inhibitors of pancreatic proteolytic enzymes: trizolol, contrical, Gordox diluted in isotonic sodium chloride solution.
- Chemical inhibitors of trypsin: pentoxyl, amino acid pronic acid, methyluracil.

6. Antibiotic therapy:

- Utilization of synthetic and semi-synthetic penicillins, cephalosporins.

7. Enzyme substitutes:

- Recommended enzyme replacements: festal, degestal, pancreatin, panzinorm, mezim-forte. Administration of Creon preparations (1 capsule 3-4 times daily).

Phytotherapy.

Patients with pancreatitis are recommended to consume a mixture of clove tincture, calendula root, walnut leaves, sage, eucalyptus, and rosehip. The suggested dosage is ½ cup three times a day for 1-2 months.

CHRONIC HEPATITIS

Anatomical and physiological features of the digestive system in infant children.

The foundation of the digestive system originates very early in embryonic development. Around 7-8 weeks, the primary intestine takes shape as a tube emerging from the endoderm. By the 12th day, this primary intestine splits into two components: the intrauterine, evolving into the future digestive tract, and the extrauterine, developing as the icteric sac. The primary intestine concludes with a blunt end, owing to the presence of the oral ring and the cloacal membrane.

During fetal development, the oral ring dissolves around the 3rd week, followed by the cloacal membrane by the 3rd month. Any disruption in these processes can lead to malformations. Around the 4th week of embryonic development, distinct segments of the digestive tract begin to take shape:

— The anterior intestine contributes to the development of the larynx, esophagus, stomach, part of the duodenum, pancreas, and liver nucleus.

— The middle intestine forms part of the duodenum, small intestine, and ileum.

— The hindgut is responsible for the formation of all segments of the large intestine.

Liver and bile ducts. Formation of the hepatic ducts and gall sac commences from the liver diverticulum during fetal development around the 4th week, originating from the ventral part of the primary endoderm of the midgut. The future gall sac and bile ducts arise from the proximal portion of the diverticulum, while the hepatic ducts emerge from the distal section.

At birth, the liver is among the largest organs, occupying 1/3 to 1/2 of the abdominal cavity and constituting approximately 4.38% of a newborn's body weight. Initially, the left side of the liver grows larger due to a robust blood supply. However, by 18 months postnatal development, the left portion diminishes in size. Hepatic lobules (lobuli hepatis) in newborns lack distinct demarcation and consist of a fibrous capsule comprising thin collagen and elastic fibers.

As a child grows, the liver also expands, although its growth rate lags behind body mass increments. For instance, by 10-11 months, it doubles (while body weight triples), reaching a threefold increase by 2-3 years, fivefold by 7-8 years, tenfold by 16-17 years, and thirteenfold by 20-30 years (with a twentyfold increase in body weight). This growth discrepancy causes the lower liver edge to protrude under the right rib in children under 5-7 years, usually 2-3 cm below the ribs, gradually decreasing thereafter.

The liver size, as observed via ultrasound, varies with age. For instance, the right liver section measures approximately 4 ± 1 cm by age 5, doubles by age 12, and reaches around 9.7 + 1 cm by age 15. Notably, newborn livers contain more water (75-80% by the 8th week) than adult livers (65-70%) and fewer proteins, fats, and glycogen. The content of dense matter increases with age.

Microscopically, liver cells in children exhibit age-related structural alterations. At birth, approximately 1.5% of hepatocytes are two-nucleated, increasing to 8.3% in adults. Palpation remains the most reliable method for liver examination due to its relatively larger size in children. Palpation methods involve various techniques to ascertain the liver's edge, consistency, shape, and sensitivity.

In various diseases, the lower liver edge becomes denser and painful. Hepatitis, cirrhosis, parasitic diseases, amyloidosis, and hepatosis

often cause liver enlargement. Conversely, acute liver dystrophy, especially in viral hepatitis B, can cause a sudden decrease in liver size. Conditions such as cirrhosis, fibrocholangiocystosis, or neoplastic damage lead to liver compaction or alterations in surface texture.

The Kurlov method for assessing liver size is applicable only in older children (5-7 years). The most effective technique involves simultaneously determining the upper and lower boundaries of the liver along the anterior axillary line, mid-clavicular line, and midline. The upper edge is identified using the slow percussion method, while the lower edge is located by palpation along these designated lines. If the lower border isn't palpable, percussion is used to locate it. This percussion technique enables dynamic monitoring of liver size in various conditions.

In conditions such as right-sided exudative pleurisy, the lower liver border shifts downward, while in cases of flatulence or ascites, it shifts upward. However, the overall size of the liver remains unchanged.

Ultrasonography.

Ultrasonography of the liver is widely utilized in pediatric practice due to its harmless nature. This method helps assess liver size and its parenchymal state, revealing conditions such as increased connective tissue (cirrhosis), cysts, or tumors. Changes in liver size can indicate pathological conditions affecting the organ.

In cases of portal hypertension, this imaging technique assesses the diameter of the hepatic portal vein and splenic vein, as well as the thickness of the ventricle wall. A wall thickness exceeding 4 mm suggests the presence of inflammatory processes (like gastritis or gastroduodenitis) or damage due to tumors.

Biochemical research methods.

The method of biochemical study of blood serum serves as a crucial tool for evaluating the functional status of the gastrointestinal tract, particularly the liver. Regarded as the body's central chemical laboratory, the liver undergoes intricate chemical processes, often reflected in various inflammatory diseases, notably acute and viral hepatitis, evidenced by increased levels of bilirubin, primarily due to direct bilirubins and monoglucuronides. In cases of hemolytic anemia, hyperbilirubinemia results from unbound bilirubin.

The liver plays a pivotal role in carbohydrate metabolism, synthesizing glycogen from glucose while converting galactose and fructose into glucose. To gauge the liver's carbohydrate activity, tests

involving glucose, galactose, or levulose loads determine the glycemic curve. In acute parenchymal hepatitis (viral or toxic), these curves exhibit significant alterations, notably with a galactose load.

Protein metabolism is another essential function of the liver, involved in synthesizing albumins, fibrinogens, various protein fractions, and most blood coagulation factors. Additionally, the liver facilitates deamination of amino acids and urea formation. Assessing the liver's protein activity involves determining total protein, protein fractions, and blood clotting factors such as prothrombin (factor II), proconvertin (factor VII), proaccelerin (factor V), and fibrinogen. Notably, in liver cirrhosis, there's a continuous decline in albumin levels, aiding in distinguishing it from chronic hepatitis.

Fat metabolism is also crucial, encompassing cholesterol esterification, bile acid conversion, and phospholipid formation. In parenchymal hepatitis (acute and chronic), decreased cholesterol esterification correlates with the severity of liver damage, while cholestasis typically leads to increased total cholesterol levels. Investigations also cover total lipid content and fractions.

Moreover, the liver actively participates in metabolizing fat-soluble vitamins (A, D, E), serving both as a storage site and converting these vitamins into active forms through processes like hydroxylation of vitamin D or the conversion of thiamine to thiamine phosphate.

The liver's vital function lies in neutralizing external poisons or internally formed toxic substances by transforming them into harmless compounds and eliminating them with bile. However, the liver's disinfecting function gradually matures after birth. Early processes include sulfitation within the first 10 days of a child's life, followed by acetylation from the third week, glucuronization from the second month, and conjugation from the third month. Elimination of toxic substances through urine develops later. Notably, the liver's disinfecting function is weaker in children than in adults, leading to rapid intoxication (toxicosis) during various diseases or poisonings, influencing drug pharmacokinetics. Multiple tests, including the Quick-Pitel test or occasionally the santonin test with sodium benzoate, are used to determine liver disinfecting activity, often reduced in parenchymal lesions.

There are numerous other research methodologies available for assessing the liver's functional state.

At present, damage to the hepatobiliary system is categorized into four groups:

1. Cytolysis syndrome, involving the direct impairment or decay of hepatocytes.
2. Cholestasis syndrome, characterized by disruptions in both intracellular and extracellular bile transport. The first type leads to an interruption in bile flow into the blood, while the second type affects the movement of bile along the bile ducts.
3. Syndrome of hepatocyte insufficiency, marked by a reduction in various substances synthesized by hepatocytes, including those vital to blood composition.
4. Inflammatory syndrome, resulting from damage to the reticulohistiocytic elements, which are abundant in the liver.

Chronic hepatitis (CHG, ICD-10 code for chronic hepatitis B: B18, Chronic hepatitis of unknown etiology: C73.9) is an inflammatory liver condition persisting for six months or longer.

Frequency of occurrence. Approximately 2 billion individuals globally are presently affected by hepatitis B, which accounts for roughly a third of the world's population. Among them, 400 million have received a diagnosis of HBsAg, indicating chronic hepatitis. Additionally, approximately 400 million people worldwide are carriers of HCV.

Etiology. In children with chronic hepatitis, the predominant virus detected is hepatitis C (HCV) in 30-50% of cases, occasionally accompanied by hepatitis B virus (HBV) at 15-20%, usually found alongside the D-virus (HDV), cytomegalovirus, herpes, rubella virus, enteroviruses, and Epstein-Barr virus. HBV is a DNA virus, while S, D, F, G viruses are RNA viruses. HDV is a hybrid particle enclosed by the surface of HBV - HBsAg.

Transmission of HCV, HDV, HFV, HGV occurs solely through parenteral means, such as blood transfusions, use of contaminated needles, and syringes. HBV can be transmitted through parenteral, household (e.g., saliva), and sexual contact (including perinatal infection during birth). Chronic hepatitis typically arises due to exposure to parenterally transmitted viruses.

According to a clinical study led by V.F. Uchaikin, parenteral transmission accounts for the majority (63.7%) of chronic hepatitis B cases in children, occasionally through contact-household routes (24.5%), and rarely (9.3%) via blood transfusions. HAV and HEV are transmitted

through the fecal-oral and water-food route but do not lead to chronic hepatitis.

HBV and HCV are identified as risk factors for liver cirrhosis and hepatocarcinoma. Hepatocarcinomas, even in adults, are believed to develop due to perinatal infection. The likelihood of a child contracting hepatitis B from an HBsAg carrier mother is 5-10%, while for an HBeAg carrier, it ranges from 75-100%. If a mother acquires hepatitis B in the last trimester of pregnancy, the risk of transmission can be 50-90%, depending on the severity of the infection.

According to foreign data, the risk of chronic hepatitis B is 5% in adults and older children, while among breastfed children, it is as high as 90%. V.F. Uchaikin and B.A. Svyatsky (1998) suggest that acute hepatitis B might progress into a chronic form. According to their clinic, all chronic hepatitis cases caused by HBV in children manifest as an anicteric atypical form of the disease, primarily resulting from a latent manifestation of the disease. Simultaneously, according to their data, the transition from an atypical (anicteric) form of hepatitis C to jaundice occurs in 20% of cases, rising to 44% in typical mild cases and 75% in moderate cases. V. F. Uchaikin and B. A. Svyatsky (1998) note that while there is no doubt about the liver's susceptibility to drug-induced damage, their clinic's findings indicate that chronic hepatitis developing under the influence of a drug is not encountered in children. Even when suspecting drug-induced chronic hepatitis, deeper investigation reveals persistent viral infections (B, C, D, among others).

Pathogenesis. The main stages of chronic hepatitis:

— Infection with hepatitis C, B or B + D viruses;

— high viral load - the main risk factor;

— genetically determined weakness of T-cell immunity and an imbalance of immunoregulatory subpopulations, a decrease in interferonogenesis, a deficiency in macrophage activity due to a certain decrease in the activity of T-suppressors;

— the influence of effector cells on the membrane of hepatocytes with the expression of viral antigens and necrosis of hepatocytes;

— observation of immunopathological processes in the liver, development of fibrosis;

— In addition, disorders of intrahepatic hemodynamics, as well as microcirculation disorders, leading to the development of intrahepatic

hypoxia, are of great importance. According to the American Academy of Pediatrics (Red Book, 2015), a diagnosis of chronic hepatitis B is made if the virus DNA titer is above 20,000 IU/ml for more than 6 months.

Autoimmune CHG - this is an exacerbated inflammatory disease affecting liver tissue, characterized by an unknown origin, with the presence of various autoantibodies in the blood serum and hypergammaglobulinemia. In autoimmune hepatitis type 1, there's an association with HLA-A1-V8-DR3 or 4, while type II is linked to -V14, DR3, C4ADQ.

Classification According to the recommendation of the World Congress of Hepatologists (October 1994, Los Angeles), there are:

Forms of chronic hepatitis:

— chronic viral hepatitis or a virus with a pathogen (B, C, D) was not detected;

— autoimmune hepatitis,

— Chronic toxic or drug-induced hepatitis.

Chronic hepatitis activity level:

— minimal (alt content more than 3 times);

— medium (alt content up to 10 times);

— explicit (alt content more than 10 times);

— inactive hepatitis.

The basis for assessing the level of activity is, first of all, determining the severity of morphological changes:

— periportal necrosis with bridging necrosis;

— intralobular degeneration;

— inflammation of the portal tract.

At a minimum level of activity, periportal necrosis is observed only in the periportal zone, only a part of the portal tract is affected, at a moderate level of activity, all portal tracts are affected, at a pronounced level of activity, necrosis passes into lobules, additional bridging necrosis develops.

Periods of chronic hepatitis:

— Mild periportal fibrosis.

— Moderate fibrosis with portoportal partitions (barriers).

— Significant fibrosis with portocentral partitions (barriers).

— Disruption of the lobular structure.

— Development of liver cirrhosis.

Phases of virus development (for chronic viral hepatitis):

— replication,

— integration.

Clinical manifestations. In children with minimal to moderate activity chronic hepatitis, symptoms often arise after school hours. Evenings become characterized by increased fatigue, malaise, weakness, irritability, disrupted sleep, headaches, nausea, and loss of appetite. They may struggle with ingesting fatty foods and experience frequent constipation or diarrhea. Abdominal pain, typically under the right rib, manifests post-meals or after physical activity.

During examination, there is often a slight liver enlargement and induration observed. Some children may exhibit spleen enlargement, and in rare cases, transient jaundice, skin hemorrhages, skin pallor, subfebrile condition, weight loss, telangiectasia, and liver enlargement upon palpation (presenting with thenar and hypotensive hyperemia). Most patients tend to experience tenderness when the liver edges are palpated, as well as in the pancreas projection (Mayo-Robson point).

Hyperfermentemia, predominantly higher levels of ALT and AST, along with moderate hyperbilirubinemia, are typical indications of liver dysfunction. Recurrences of this disease can persist for months or years, often manifesting with varied symptoms. Some children may present with no or fewer complaints, showing symptoms limited to hepatomegaly, moderate splenomegaly, and hepatic hyperenzymemia.

Symptoms can include asthenovegetative syndrome (fatigue, malaise, sleep disturbances, headache, sweating, low-grade fever, anorexia, arterial hypotension), dyspeptic symptoms (nausea, vomiting, heartburn), and abdominal pain, often under the right rib. Other signs might include minor indications of liver failure, such as drowsiness or insomnia, transient jaundice, hemorrhagic syndrome, hand tremors, vascular spots and angiomas, skin redness, hepatic palms, anemia, and myocardial infarction. On palpation, an enlarged liver with tender edges and sometimes moderate splenomegaly are observed.

Laboratory tests reveal signs of dysproteinemia, hypergammaglobulinemia, increased enzyme activities including transaminases and glutamate dehydrogenase, liver pigmentation, and dysfunction in carbohydrate metabolism and other aspects. Clinical and laboratory signs of disease activity persist even during remission.

Cholestasis is rare in children with chronic hepatitis, where the main clinical symptoms are jaundice and itching due to elevated bile acids in the blood. Cholestatic hepatitis in children might progress to biliary cirrhosis.

The main clinical symptoms of cholestasis-related chronic hepatitis are jaundice and itching, which arise due to elevated bile acids in the blood. Persistent jaundice might not always be present. In children, cholestatic hepatitis can advance to biliary cirrhosis. Moderate splenomegaly might be occasionally observed. Blood tests often reveal dysproteinemia, hypergammaglobulinemia, increased enzyme activities (like transaminases and glutamate dehydrogenase), liver pigmentation, and dysfunction in carbohydrate metabolism and other areas. Clinical and laboratory signs of disease activity typically persist even during remission. Cholestasis is rare in children with chronic hepatitis.

Comparison of the three main types of chronic hepatitis [Sherlock Sh., Duli Dj., 1999]

Signs	Autoimmune hepatitis type 1	Hepatitis B	Hepatitis C
gender dominance	Woman	Man	Same for both sexes
Age	15-25 years old. Menopause	newborns, over 25 years old	All age groups
HBS Ag in serum	Not tracked	Discovered	Not tracked
Antibodies to HCV in serum	Not tracked	Not tracked	Discovered
Autoimmune diseases	Mostly	In rare cases	Sometimes
Increase in the amount of y-globulin in serum	Expressed	Average	Average
Antibodies to smooth muscle and antinuclear factors	High titer (70%)	The titer is low or not observed	The titer is low or not observed
Risk of primary liver cancer	Small	High	High
Outcome of corticosteroid treatment	Positive	Not efficient	Not efficient

Autoimmune chronic hepatitis (ICD-10, code K75.4) Hypergammaglobulinemia with severe symptoms similar to chronic active hepatitis, hyperglycemia and other non-hepatic immunopathological (autoimmune) disorders: autoimmune thyroiditis, ulcerative colitis, synovitis, arthritis or diffuse-toxic, pulmonary infiltrates, erythema, hemolytic anemia, thrombocytopenic purpura, heart damage, etc. Type I autoimmune hepatitis is diagnosed in patients with HLA-AI-B8-DR3 or 4, and type II - V14, DR3, C4A-DQ. The main variants of chronic hepatitis are shown in the table below.

Drug-induced liver injury in children less common than in adults. However, with polypharmacotherapy, the risk is even higher. There are two groups of drug-induced hepatopathy:

- Predictable (drugs with hepatotoxic effects in high doses);
- idiopathic (optional).

The first group comprises dose-dependent hepatic drugs like acetaminophen (paracetamol), methotrexate, 6-mercaptopurine, valproic acid, etc. These drugs, when taken at a dose of 140 mg/kg of body weight, directly damage the liver by affecting the hepatocyte membrane and forming reactive metabolites. The second group of drugs is not dose-dependent; they induce liver damage in individuals who produce abnormal metabolites affecting tissue macromolecules, subsequently triggering immunopathological reactions. Sometimes, this leads to hypersensitivity and extrahepatic symptoms such as fever, skin rash, and arthralgia. Toxic hepatopathy is conditionally divided into three groups:

- Functional disorders of the liver (like transient hyperbilirubinemia, increased transaminase activity, etc.),
- Changes resembling viral hepatitis (such as hepatocyte cytolysis and signs of cholestasis),
- Granulomatous hepatitis and intrahepatic circulation disorders.

Hepatitis-like reactions encompass symptoms such as abdominal pain, fever, liver enlargement, jaundice, dark urine, and elevated transaminase activity (2-5 times the normal level). Certain drugs exhibit a cytolytic effect on the liver, including antibiotics (tetracycline, rifampicin, meropenem, oxacillin, ampicillin, carbenicillin), diuretics (thiazides, furosemide, ethacrynic acid), general anesthetics (fluorotanate, citamate, chloroform), cytostatics (azathioprine, leukeran, methotrexate,

6-mercaptopurine), anti-tuberculosis drugs (PASK, isoniazid), and others. Drugs with predominant hepatotoxic effects and cholestasis encompass antibiotics (erythromycin, nitrofurans), psychotropic drugs (chlorpromazine, diazepam, meprotan), and hormonal drugs (nerobol, dianabol, methyltestosterone). Children experiencing such reactions may report itching, malaise, dyspeptic disorders, hepatomegaly, and jaundice.

In children, hepatitis-like lesions (toxic hepatopathy) can result from poisoning (often due to toadstool or insecticides), hereditary metabolic abnormalities (fructosemia, tyrosinosis, galactosemia, etc.). Occasionally, children may develop toxic-allergic reactions to drugs, ranging from mild to severe toxicoderma (Stevens-Johnson, Lyell syndromes). Liver damage in the form of granulomatous hepatitis is rarely seen in children treated with sulfonamides, penicillins, or fluorine-containing anesthetics, characterized by intrahepatic cholestasis, fever, and hepatosplenomegaly. Granulomatous hepatitis may also indicate mycosis and tuberculosis. Diagnosis typically involves hepatobiliary scintigraphy and liver biopsy, although ultrasound (US) can assist in the diagnostic process.

Chronic hepatitis is diagnosed during a comprehensive examination of patients:

- biochemical;
- virological;
- immunological;
- morphological;
- The state of portal hemodynamics is assessed.

An elevation in blood serum enzyme activity (AST, ALT, fructose mono- and diphosphate aldolase, etc.) results from hepatocyte cytoplasmic membrane disruption and mitochondrial integrity compromise (increased glutamate dehydrogenase, urokinase activity), or lysosomal damage (elevated ribonuclease, leucine aminopeptidase, cathepsins). This indicates compromised secretory (reduced cholinesterase activity) and excretory (raised alkaline phosphatase activity) liver functions. The heightened activity of mitochondrial and lysosomal enzymes corresponds to the severity of liver damage.

Pigment metabolism disorders in chronic hepatitis may result from:

- Impaired conjugation and secretion of bilirubin (parenchymal component of jaundice).
- Bile duct obstruction (mechanical component of jaundice).

- Heightened breakdown of red blood cells (hemolytic component of jaundice).

Elevated Direct Hyperbilirubinemia (DHB) characterizes recurrent chronic hepatitis. However, severe autoimmune disorders might exhibit direct hyperbilirubinemia alongside diminished retention of hepatocyte bilirubin in the blood. The manifestation of parenchymal jaundice involves bilirubinuria, characterized by dark urine and a positive reaction to bile pigments in the urine. Additionally, during jaundice exacerbations, an increase in urobilinuria might be observed. With the hemolytic component of jaundice, besides direct hyperbilirubinemia, reticulocytosis, increased carboxyhemoglobin, and reduced serum haptoglobin are observed. A decrease in the liver's protein synthesis capability is associated with hypoalbuminemia, reduced concentrations of other vitamin K-dependent blood coagulation factors (VII, IX, X), as well as factors I, V, transferrin, ceruloplasmin, and haptoglobin.

Disturbances in lipid metabolism are evident in chronic hepatitis, signified by elevated phospholipid content in the blood serum and reduced cholesterol and its ether-bound fraction. Cholestatic jaundice is concurrent with hyperlipidemia and hypercholesterolemia.

The Bauer test, which involves a galactose load (0.5 g per 1 kg of body weight), is a reliable method for detecting liver regulatory function insufficiency in carbohydrate metabolism. Patients with chronic hepatitis often display relatively high galactose excretion (exceeding 8% within 12 hours), indicating disrupted galactose excretion. The evaluation of the liver's cleansing and excretory function involves a radioactive gold load.

Safe methods for morphological liver assessment include ultrasound, radioisotope scanning, computed tomography, and laparoscopy. Liver puncture biopsies conducted using these methods help identify diffuse liver damage. In moderate chronic hepatitis, a histological examination often does not show significant changes in liver architecture. Instead, it typically reveals moderate dystrophic changes in liver cells such as hydropic degeneration and ballooning hepatocytes, limited lymphohistiocytic infiltration, and moderate focal fibrosis in the portal zone without parenchymal transformation into nodes.

Histological manifestations of hepatocellular carcinoma in lupoid hepatitis involve pronounced plasma cell and portal lymphocyte infiltration, diffuse liver fibrosis, and impaired architectonics.

Liver elastography stands as the most effective method for studying chronic hepatitis, replacing the need for painful biopsies. It has become the global diagnostic standard in hepatology, effectively assessing tissue condition and playing a significant role in determining fibrosis stage in hepatitis patients. Utilizing the fibroscan apparatus, this innovative method relies on the relationship between tissue elasticity and the degree of fibrosis.

It is an essential procedure for individuals exhibiting evident signs of impaired liver function in various conditions such as:

- Different types of viral hepatitis.
- Alcoholic fatty disease or fatty hepatosis.
- Alcoholic steatohepatitis.
- Autoimmune liver diseases.
- Hereditary conditions affecting organ function.

Additionally, it is crucial for individuals at risk or with a medical history of conditions leading to tissue damage, including:

- Diabetes.
- Endocrine disorders.
- Atherosclerosis.
- Hematopoietic disorders.
- Obesity.
- Elevated bilirubin levels in the blood.

Fibroelastometry offers several advantages over standard biopsy methods:

- Conducted without anesthesia.
- No surgical intervention required.
- Outpatient procedure.
- Carried out without sedation.
- Typically completed within 15 minutes.
- More cost-effective than traditional biopsy procedures.
- Minimal risk of complications.
- No need for a recovery period.
- Provides assessment of various body tissues.
- Results are independent of human influence or error.

The virological examination of chronic hepatitis is designed to detect specific antigens and antibodies in the patient's blood. HBsAg, HBcAg, and HBeAg are identified using enzyme-linked immunosorbent assay (ELISA). Additionally, DNA of HBV and RNA of HCV and HDV

are detected through polymerase chain reaction (PCR) analysis conducted on both blood samples and liver biopsies.

HBsAg represents the surface antigen of hepatitis B, while HBcAg constitutes the core nucleoprotein of the virus. HBeAg, derived from HBcAg, is a marker indicating active viral replication present in the blood.

The immunological assessment involves evaluating the patient's immune status and searching for antibodies against various antigens, including those found in liver cells, skin basement membranes, DNA, and smooth muscles. In cases of chronic hepatitis, there is often a mild elevation in immunoglobulins A and G levels in the blood. However, highly active chronic hepatitis typically displays significantly increased IgG levels.

In chronic hepatitis caused by HBsAg, the count of T-lymphocyte suppressors tends to remain within the normal range. Conversely, in cases of autoimmune-type hepatitis, this count is often diminished.

Differential diagnosis. We need to initially differentiate chronic hepatitis from residual symptoms of acute hepatitis A. Recovery from acute hepatitis A typically takes 12–16 weeks. Hence, it's erroneous to consider a child discharged from the hospital after hepatitis A treatment as entirely healthy. This is because there might be alterations in laboratory parameters or a decline in the general condition. Children often report fatigue, headaches, joint pain, loss of appetite, nausea, vomiting, right hypochondrial pain, and diarrhea. On objective examination, an enlarged liver, yellow skin, and sclera are observed, along with persistent impaired liver function. Asthenodyspeptic syndrome may manifest in some children during the disease, while others display jaundice. In some cases, patients solely exhibit liver enlargement without any complaints, yet liver function tests yield satisfactory results.

Focal fibrosis development in the liver following acute hepatitis A treatment can lead to an anatomical defect. Functional hyperbilirubinemia might persist for months, and sometimes even years, post-treatment without affecting the child's general condition or liver function parameters. Acute hepatitis A does not progress to chronic hepatitis or liver cirrhosis.

If, despite clinical and epidemiological data confirming epidemic hepatitis, antibodies against HBsAg, HBcAg, HBeAg are absent even after a year following the acute phase, symptoms of acute hepatitis are still evident. The differentiation between chronic hepatitis and liver

cirrhosis relies on conducting morphological studies (ultrasound, computed tomography, radioisotope scanning, biopsy) alongside analyzing clinical and laboratory data over time.

Chronic hepatitis is distinguished from hereditary pigmentary hepatoses and metabolic liver diseases through differential diagnosis. Clinical indicators suggesting metabolic liver diseases include periodic vomiting, physical and/or mental retardation, shortness of breath, dysmorphia, cataracts, hypoglycemia, organic acidemia, lactic acidosis, and hemorrhages.

Treatment of patients with chronic hepatitis.

In the event of a recurrence of chronic hepatitis, patients are advised to observe bed rest, reducing hepatic load and enhancing hepatic blood flow. As liver function and the patient's overall condition improve, the regimen is gradually expanded. For those without disease recurrence, engaging in physical activities, sports, and daytime rest is recommended. However, excessive movement should be avoided, and patients can benefit from exercise therapy and walks in fresh air. It's crucial to shield patients from all medications, particularly those detoxified by the liver.

Dietary considerations vary based on the disease stage and degree of liver failure. Typically, patients adhere to diet table number 5, moderately hypochlorite (3-4g of salt per day), with a total energy value slightly exceeding the age norm. There's a slight reduction in fat and a proportional increase in carbohydrates. In periods without decompensation (1 and 2), protein content should align with age norms. However, in cases of liver failure and high protein levels due to hepatic-portal vein anastomoses, blood nitrogenous products, often ammonia, increase, posing a risk of liver failure and coma. Consequently, protein intake is halved compared to physiological requirements.

Dietary restrictions involve limiting mechanical and chemical agents. Vegetables are finely grated, and meat is served as steam cutlets or meatballs. Coarse vegetable fibers, extracts, purines, and salt are excluded in cases of liver failure. Including vegetable oils high in unsaturated fatty acids is beneficial. The diet should be rich in vitamins and lipotropic substances. Dairy products like yogurt, cottage cheese, cream, salmon, eggs, lean meat, fish, oatmeal, and fresh fruit juices (mainly grape juice) are recommended.

Prohibited foods include fatty foods, soups, mushroom soups, sausages, canned items, spices, spicy or salty foods, smoked meats,

coffee, ice cream, and carbonated drinks. This prescribed diet is typically lifelong, with meal frequency increased to 4-6 times a day.Primary therapy involves hepatoprotectors (e.g., heptral, silibor), litholytics (e.g., ursofalk, lithofalk), enterosorbents, pre- and probiotics, herbal remedies, exercise therapy, psychotherapy, and managing concurrent diseases. Antiviral therapy for chronic viral hepatitis is specifically conducted in specialized departments and clinics and is recommended during the virus replication phase. The Red Book (2015) outlines indications: for hepatitis B, DNA levels above 20,000 IU/ml and the presence of HBeAg; for hepatitis C, virus RNA levels above 2,000 IU/ml. The drug of choice typically involves standard, recombinant, or pegylated interferon. α-2-interferons such as Roferon A and others, and α-2-interferons like Intron A at a standard dose of 3 million IU/ml are administered three times a week for 6-12 months. These are given via subcutaneous, intramuscular, or intravenous routes for children over 1 year old. Interferons enhance the patient's immune response to the viral infection, reduce viral replication by activating endoribonuclease, increasing protein kinase activity, and inducing 2'3-oligoadenylate synthetase.

Antivirals are not recommended during inactive phases. The use of interferons may result in flu-like reactions such as fever, headache, severe joint and muscle pain, and in some cases, hematological disorders, ischemic retinopathy, or thyroid dysfunction.

Virological control is typically performed at the 12th week of treatment. In cases where interferon therapy is ineffective, combined dual therapy is adopted (as recommended from the onset of chronic hepatitis C) involving pegylated α-interferons and ribavirin. Additionally, triple therapy comprising pegylated interferon, ribavirin, and protease inhibitors like telaprevir or boceprevir has shown promising results.

In hepatitis C, the cure rate for dual therapy is 1a - 40%, for triple therapy - 75-100%. The American pharmaceutical company Gilead Sciences has developed a new method of treating hepatitis C with a combination of drugs sofosbuvir (sofosbuvir) and ledipasvir (ledipasvir), which eliminates 97-100% of the hepatitis C virus. Sofosbuvir (lefasbuvir) and ledipasvir (ledipasvir) stop the replication of the hepatitis C virus by inhibiting the phosphoprotein of the ledipasvir virus, the NS5A protein, while sofosbuvir is an analogue of uridine (triphosphate), binding to viral RNA through NSSB polymerase. The combination drug is called Harvoni and contains 90 mg of ledipasvir and 400 mg of sofosbuvir. Take

once a day for 12-24 weeks until the virus disappears from the blood. Side effects are minimal (headache, malaise, fatigue) in 1% of patients. A 12-week course of Harvoni costs $94,500 and a 24-week course costs $189,000, which is half the price of a liver transplant. According to Red Book (2015), there is no direct treatment for hepatitis C in children. In chronic hepatitis with systemic symptoms (autoimmune hepatitis), glucocorticoids with or without immunosuppressants and cytostatics are recommended. The dose of prednisolone is 2 mg/kg at the beginning of treatment, which is administered until transaminase activity normalizes. Then it is constantly reduced to 10-15 mg / day. This dose is prescribed for several months (at least 6 months) against the background of potassium-enriched foods (raisins, sorrel, plums, etc.) and potassium supplements. The daily dose of the hormone is always administered in 2 doses (2/3 morning dose, 1/3 daily dose) to prevent adrenal insufficiency. In this case, it is necessary to monitor the possible side effects of the hormone and eliminate them in a timely manner. Autoimmune hepatitis is the basis for the joint recommendation of corticosteroids and immunosuppressants - imuran (azathioprine) at a daily dose of 2-2, 5 mg/kg. After 1 month, the dose of imuran is reduced to 1 mg / kg and prescribed for a long time - at least 1 year (usually up to 3 years) in combination with prednisolone (0.25–0.5 mg / kg / day). The effectiveness of cyclosporine Ani is currently being studied. Contraindications to the use of glucocorticoids are ulcerative colitis, severe osteoporosis, and contraindications to the use of cytostatics are precoma and coma of the patient, severe infections, severe leukopenia and hypoproteinemia. For skin itching, cholestyramine or cholestipol, ursodeoxycholic acid, heptral, rifampicin, ondansetron, nakolson, phenobarbital (2.5 mg / kg / day) in minimal doses, N1-histamine receptor antagonists are recommended. Ondansetron (serotonin receptor antagonists) is more effective. Adults are given 8 mg intravenously as a bolus. Ammonia is reduced as a result of: Ondansetron (serotonin receptor antagonists) is more effective. Adults are given 8 mg intravenously as a bolus. Ammonia is reduced as a result of: Ondansetron (serotonin receptor antagonists) is more effective. Adults are given 8 mg intravenously as a bolus. Ammonia reduction can occur through several methods:

- Decreasing ammoniagenic substances involves reducing protein intake initially with a protein-free diet, purging the gastrointestinal

tract using laxatives, improving kidney function, or reducing urea levels through hemodialysis or peritoneal dialysis.

- Lowering ammonia production entails using non-absorbable antibiotics (like neomycin), lactulose, or lactobacterin, which displaces pathological intestinal microflora.
- Eliminating ammonia involves various methods such as dialysis, hemosorption, gastrointestinal tract clearance, and medications causing acidification (like lactulose, sorbitol, cation exchangers).
- Employing medications to convert ammonia into less toxic forms such as glutamine-asparagine (glutamine and aspartic acid), urea (arginine, protamine), or the introduction of non-amino acid amines.

Empirical therapy, including L-DOPA and monoamine oxidase inhibitors, is being administered. Anticonvulsant therapy is used for symptomatic treatment, addressing infections, anemia, hypoxemia, acid-base and electrolyte imbalances, fever, and other related issues. The initial volume of parenteral infusion therapy is set at 1 liter per square meter per day. Effective results in portosystemic encephalopathy were obtained using Hepamerz, as indicated by SD Podymova's study in 1996.

Children with chronic hepatitis undergo regular monitoring until they transition to an adult polyclinic. The daily routine for these patients includes 10–11 hours of sleep per day for children aged 5–9 and 9–10 hours for those aged 10–14 (with an additional 1–2 hours of daytime sleep). Morning hygienic exercises are recommended for the child. Following a 2-3 month recovery period post the last recurrence, the child can engage in sports within a physiotherapy exercises group at school. Daily activities should involve outdoor walks (preferably 2-3 times a day), skiing, and outdoor games, ensuring the level of physical activity is appropriate. The daily diet should be regular, consisting of at least 4 meals a day and including cottage cheese or other dairy products, oatmeal, fruits, juices, fresh fish, salads with vegetable oil, vegetables, buckwheat porridge, and honey.

During the initial 2-year period post-hospitalization, the patient undergoes a biochemical blood test (bilirubin, transaminase activity, protein and protein fractions) every 3 months, reducing to twice a year thereafter. Evaluations include a thorough assessment of the patient's well-being, liver size, presence of cholecystitis, and other infections. The child undergoes clinical blood tests and fecal examinations to detect

helminth eggs biannually. Additionally, referrals to an otolaryngologist and a dentist are made.

For the first 2 years post-hospitalization, monthly treatment courses are scheduled as follows: twice a week intake of tincture of medicinal plants (immortelle, corn poppy, shamrock, dandelion root, mint leaves, chamomile tea) and lactobacterin or bificol. Vitamin courses (C, A, B15, B5, B6, etc.) are alternated every two weeks, followed by stimulating therapy involving pentaoxide, dibazol, pantocrine, ginseng, etc. Courses lasting 1 month are recommended twice a year, involving heated mineral water (Slavyanovskaya, Essentuki No. 4, Dzhermajur, Arzni, Jermuk, Arshan, Sairme, Vytautas, Izhevskaya, Mirgorodskaya) at a rate of 3-5 ml/kg and ozokerite applications to the liver area.

Spa treatment: Children with chronic liver diseases are treated in local sanatoriums, as well as in Zheleznovodsk, Essentuki, Truskovets, Pyatigorsk, Dzhermajur, Mirgorod, Morshin, etc. A contraindication to sending children to sanatorium treatment, in addition to general resistance, is the presence of an active destructive-necrotic process in the liver or signs of severe liver failure.

Consequences of the disease. The outcome of chronic hepatitis depends on its form, level of activity, and systemic treatment using interferon preparations. In moderate stages of chronic hepatitis, the process stabilizes, showing reparative dynamics and subsequent recovery. Periodic relapses of the disease might occur without morphological signs of exacerbation. Side effects of chronic hepatitis encompass cirrhosis and liver failure, predominantly observed in cases with high activity (approximately 50% of cases).

Prevention. Per WHO guidelines, active immunization against hepatitis B targets individuals at high risk of infection:

- Newborns delivered by mothers with acute hepatitis B or carriers of HBsAg (administered with specific hepatitis B immunoglobulin within 12 hours of birth).
- Medical personnel in hemodialysis and hematology departments, alongside students in medical institutions and secondary medical schools who have constant contact with patients' blood before commencing internships.
- Family members of chronic hepatitis patients or HBsAg carriers.

- Residents in areas with a high prevalence of HBsAg carriers (8-15% or higher).
- Travelers visiting epidemiologically vulnerable regions.
- Teenagers leading promiscuous sex life.
- Patients undergoing hemodialysis.
- Individuals with blood disorders receiving blood transfusions or blood products.
- Intravenous drug users.
- Children born in regions where HBsAg carriers exceed 2% of the population.
- Individuals in contact with acute hepatitis B patients who haven't been vaccinated or diagnosed (vaccine administration includes specific hepatitis B immunoglobulin).

ACUTE CHOLESYSTITIS

ANATOMICAL AND PHYSIOLOGICAL FEATURES OF THE DIGESTIVE ORGANS IN YOUNG CHILDREN

In infants, the gallbladder is typically obscured by the liver, making palpation challenging or unclear on x-rays. Its shape can vary, from cylindrical or pear-shaped to rarer forms like fusiform or S-shaped, often associated with the hepatic artery's different position. As children age, the gallbladder size increases. Ultrasound findings suggest an average gallbladder size of 2.5-4 cm between 2-7 years, 5 cm between 8-12 years, and 7 cm between 13-15 years, with a maximum size of 3 cm. In children over 7 years old, the gallbladder's projection is typically identified at the intersection point of the external rectus abdominis muscle and the chest, laterally while in the supine position.

In newborns, the lower part of the gallbladder is enveloped on all sides by the abdominal cavity, while the body and neck are covered on three sides (laterally and inferiorly). The lower part of the gallbladder intersects with the small intestine, the body with the transverse small intestine, and the neck with the upper horizontal surface of the duodenum. Typically, newborns have a larger gallbladder compared to the common bile duct, which runs along the free edge of the liver-duodenum and may show a bend in its initial section, aiding in distinguishing between the ascending and descending parts of the intestine. The bile duct merges with the bile duct at the bile duct to form the common bile duct (ductus

choledochus). The total length of the bile ducts varies even among newborns (5-18 mm), increasing with age.

In children, the gallbladder is typically not palpable. However, in gallbladder-related conditions such as cholecystitis, pain is often localized in the same area, extending from the lower edge of the liver to the outer right muscle of the lateral part of the abdomen.

Cholecystography serves as a diagnostic method to examine the hepatobiliary system. Oral cholecystography involves administering a contrast agent (cholevid, bilitrast) orally, typically biligrafin a day prior to the study (12-14 hours) or intravenously before the examination (15-45 minutes). X-rays are taken to assess the gallbladder's shape, size, location, and sometimes its condition. Following this, a choleretic breakfast (e.g., egg yolk) is given, and another image is captured. This approach helps determine the gallbladder's contractility, often reduced to half of the normal function. Cholecystography is instrumental in detecting abnormalities within the biliary system and identifying dyskinetic disorders such as hypo- and hypermotor dyskinesia.

Duodenal sounding, particularly fractional sounding, has gained popularity in recent decades. It involves the use of a specialized thin probe with an olive tip. This procedure is aimed at analyzing the volume of duodenal fluid and determining the duration of its formation.

The first period, marking the formation of initial bile before the introduction of the stimulant (25% magnesium sulfate solution, xylate, etc.), indicates the amount of bile secretion from the common bile duct, averaging at 1.2 ± 0.3 ml/min.

The second period denotes the closure time of the hepatic-pancreatic sphincter, determined by the duration between the stimulant's delivery and the appearance of a new portion of bile, averaging at 4 ± 2 minutes.

The third period (portion time A) encompasses the opening duration of the pancreas and liver sphincters, which typically lasts 2.5 ± 0.5 minutes.

The fourth period signifies the release time of the cystic portion (average 25 ± 5 min). Additionally, it identifies the number of bladder portions (norm 33 ± 11 ml) and the speed (norm 1.3 ± 0.2 ml/min).

After retrieving all portions, an irritant is administered to assess the gallbladder's contraction completion. Deviations from the normal bile duct functions indicate dystonia of the hepatic-pancreatic ampullae (hypo-

or hypertension) and disrupted motility of the gallbladder (hypo- or hyperkinesia).

The collected duodenal fluid undergoes microscopic and biochemical examination, revealing mucus, leukocytes, epithelial cells, and concentrations of bilirubin, cholesterol, lipids, and bile acids in each portion. These analyses aid in diagnosing biliary system diseases.

Acute cholecystitis is an uncommon inflammatory condition of the gallbladder in children, with boys being twice as likely to be affected compared to girls.

Etiology: The causative agents primarily include Escherichia coli, staphylococci, streptococci, anaerobic flora, and, in very rare instances, typhoid bacilli.

Pathogenesis: Infection reaches the gallbladder via hematogenous, lymphogenous, or enterogenous routes. The primary cause of acute cholecystitis is the stagnation of bile in the gallbladder, often associated with various anomalies or cholelithiasis.

Classification: In children, acute cholecystitis manifests in catarrhal, phlegmonous, and gangrenous (destructive) forms. Acute catarrhal inflammation involves swelling of the gallbladder, while the phlegmonous form may lead to gangrene or empyema.

Clinic. The typical onset of the disease is sudden, often in the evening, accompanied by a febrile rise in body temperature and sharp abdominal pain localized on the right side or throughout the abdomen. These pain episodes may last from minutes to hours, rarely radiating to the lower back, right shoulder, sternum, back, or limbs. Around half of affected children experience nausea and vomiting. Pain usually intensifies on the right side.

Common signs of intoxication are evident, including oily or sweaty skin, dry lips, a coated tongue, headache, loss of appetite, constipation, tachycardia, and in some cases, convulsions, fainting, or meningeal symptoms. About half of the patients with acute cholecystitis exhibit jaundice. In one-third of cases, body temperature might remain normal despite signs of gallbladder inflammation.

During examination, abdominal swelling and a delay in the upper abdomen during breathing might be observed. Palpation on the right side of the anterior abdominal wall often reveals rigidity in the epigastric and hypochondral muscles. Sometimes, the precise localization of pain might not be obvious. Positive signs such as Mendel's, Ortner's, Murphy's, and

Kera's can be observed. Additionally, in some instances, Shetkin-Blumberg symptoms may also be positive.

Blood test changes like leukocytosis, neutrophilia, and increased ESR are common. Diagnosis of acute cholecystitis relies on transabdominal ultrasound findings, particularly the thickening of the gallbladder wall (2–3 mm) or ill-defined contours. The appearance of a hypoechogenic area in the gallbladder wall might indicate mucosal tumors

Differential diagnosis. In its early stages, the disease is differentially diagnosed from conditions such as appendicitis, epidemic hepatitis, right-sided croupous pneumonia, acute gastritis, pyelonephritis, abdominal Shenlein-Genoch disease, and relapse of chronic cholecystitis. Generally, acute cholecystitis in children progresses without complications. Body temperature elevation and pain attacks can persist for several days, gradually diminishing. Conservative treatment suffices in most cases, although surgery might be necessary for purulent, phlegmonous, or gangrenous cholecystitis with gallbladder rupture. Only 30% of children experience complete recovery from acute cholecystitis. Often, acute cholecystitis is indicative of underlying chronic cholecystitis.

Treatment. In the early stages of the disease, patients are advised to adhere to bed rest, increase fluid intake (mineral water, saccharin tea, followed by a diet consistent with Table No. 5), use sedatives, antibiotics (such as cefuroxime, ceftriaxone with metronidazole), antispasmodics (like drotaverine, papaverine, metacin), and analgesics (such as baralgin, atropine). Infusion therapy and anti-enzymatic drugs (such as kontrykal) are also prescribed. The patient's treatment is a collaborative effort between a pediatrician and a pediatric surgeon.

CHRONIC CHOLECYSTITIS

This is a chronic inflammatory process of the gallbladder or biliary tract (MKV-10 K81.1), which develops against the background of dyscholia and cholelithiasis, congenital and acquired anomalies of the biliary tract.

Etiology. Inflammation affecting the gallbladder and biliary tract can stem from infectious and non-infectious origins. Infectious processes within the gallbladder predominantly involve bacteria, occasionally extending to rare viral occurrences. Bacterial pathogens encompass auto-

flora such as E. coli, staphylococcus, enterococcus, proteus, and sporadically typhoid fever, paratyphoid fever, and dysentery pathogens. In some cases, infants receiving breastfeeding may develop sepsis leading to gallbladder inflammation. Viruses like epidemic hepatitis viruses (occasionally adenoviruses and enteroviruses) can cause inflammation of the bile duct wall, even in the absence of bacterial presence. Additionally, certain regions may experience chronic cholecystitis due to parasitic causes (feline and liver flukes). Nonspecific processes affecting the biliary tract may arise due to hypotonic dyskinesia resulting from duodenobiliary reflux, gastric and pancreatic secretions, and allergic reactions in individuals with atopic diathesis.

Pathogenesis. Chronic cholecystitis is devided into calculous and non-calculous forms, with young children being more susceptible to non-calculous types. In children, cholecystitis seldom impacts the bile ducts, typically initiating around the funnel-shaped neck area, progressing to inflammation in the bile duct walls (cholecystocholangitis), and occasionally involving the intrahepatic ducts. Various anomalies in the biliary tract, such as dyscholia and dysbacteriosis, contribute to the onset of cholecystitis.

Individuals with cholecystitis, as well as healthy individuals, have microbes and Giardia eradicated by bile and experience leukocyte breakdown, albeit this process is delayed or absent in patients. Factors like liver infections (e.g., epidemic hepatitis, infectious mononucleosis), nutritional, and metabolic disorders (such as obesity, diabetes mellitus), can lead to dyscholia. The role of dyskinesia in cholecystitis' pathogenesis has been previously discussed. Cholecystitis tends to rapidly develop in patients with hypotonic dyskinesia, dysbacteriosis, constipation, and chronic infection foci. In most cases, the pathological process initially affects the stomach, pancreas, liver, and occasionally later involves the cardiovascular system. This results in purulent infections and functional disturbances in the biliary tract, leading to duodenitis, gastritis, dysbacteriosis, and dyskinesias. Thus, determining whether a process is primary or secondary in a specific patient can be challenging.

Gallbladder infections can be transmitted through the ductus choledochus or through lymphatic, hematogenous routes (via the oral cavity, lungs, kidneys, and other organs). Microbes enter the intestines through the gallbladder, then reach the liver via the portal vein and subsequently invade the bile ducts. Through the lymphogenous route,

microbes travel from the gallbladder to the pancreas, then through the portal vein to the liver.

If morphological changes occur solely in the mucous membranes, the process tends to be catarrhal in nature, allowing the gallbladder function to persist for an extended period. However, if the process encompasses the entire gallbladder wall, it can lead to thickening, sclerosis, pericholecystitis, dysfunction, and the formation of mucoepithelial stones. Functional inflammatory processes in the gallbladder have been shown to cause chronic, recurrent allergic dermatoses and bronchial asthma. In the elderly, chronic cholecystitis might result in leukopenia.

Clinic. The primary symptoms of cholecystitis include pain, which may not be directly linked to food intake but can be triggered by physical activities or consumption of fatty, cold, spicy foods, or carbonated drinks. The pain can be severe, stabbing, or cutting, lasting anywhere from half an hour to several hours. Pain localization varies: approximately 50% of patients experience it in the right hypochondrium, 30% in the epigastrium, and some individuals have indistinct localization. After 2-3 hours, the pain might subside.

Other reported complaints consist of fatigue, headaches, irritability, a subfebrile condition (present in about a quarter of patients), sweating, nausea, bitter taste in the mouth, loss of appetite, occasional vomiting, constipation, and dermatitis. Upon objective examination, patients with cholecystitis exhibit muscle resistance in the right hypochondrium and show positive signs such as Kera's symptom (gallbladder pain worsened by breathing), Ortner's symptom (gallbladder pain with light tapping on the chest), Lepin's symptom (pain with gentle tapping on the gallbladder with bent fingers), and Mendel's symptom. In rare cases, children may display positive phrenicus symptoms on the right, Georgiev-Mussi symptoms on the right, pain in the right shoulder blade (Kharitonov's symptom), and pain in the xiphoid process (Voskresensky's symptom). The clinical presentation of cholecystitis and giardiasis cannot be distinguished without these symptoms.

When inflammation primarily affects the biliary tract, pain and dyspepsia develop, accompanied by liver enlargement and thickening. Abdominal pain is typically around the navel and radiates to the right shoulder. Intermittent jaundice, hepatic duct obstruction (Mirizzi syndrome), pruritus, and obstructive cholangitis (Ano-Ressle syndrome)

may also occur. In chronic cholecystitis in children, the stomach is generally affected to some degree. The initial cause of cholecystitis, if it involves hyperacid gastritis, can transition to hypoacid in subsequent years. Around 40-50% of patients with chronic cholecystitis experience impaired pancreatic secretory function in the form of dyspepsia. Dermatitis, whether atopic or non-allergic, is more prevalent in patients with biliary tract pathology.

While hereditary predisposition and atopic diathesis before the onset of biliary tract pathology play a significant role, the pathogenetic relationship is intricate. Clinical experience has demonstrated that treatment aimed at normalizing hepatobiliary function can yield positive outcomes in some patients, even when treatment for atopic diseases has proven ineffective.

Diagnostics. The diagnosis of gallbladder diseases includes anamnesis (patients having a history of biliary tract diseases in the family), patient complaints, clinical symptoms, as well as clinical and biochemical changes in the blood.**Instrumental and hardware research methods:** Ultrasound stands as a primary diagnostic method for assessing diseases of the biliary tract and liver, and it is also utilized in screening procedures.

* Diagnostic tests with sorbitol, magnesium sulfate or choleretic breakfast.

* * Diagnostic tests with Ursofalk.

Cholecystography may be oral or intravenous. In both cases, iodine-containing contrast agents (bilignost, biligrafin) are used, X-ray 12-14 hours, oral cholecystography after 15 minutes. Subsequently, it is carried out after 20-30 minutes. When the shadow of the gallbladder is clearly visible, you need to give a choleretic breakfast (2 raw egg yolks or 100 g of sour cream) and re-radiography is performed after 45-60 minutes. Normally, the gallbladder is 1/3 smaller than its original size. Cholecystography shows that if the ultrasound results do not match the anamnestic and clinical signs, various anomalies of the gallbladder are suspected.

CT scan is important in the differential diagnosis of various pathologies of the abdominal organs - the liver and pancreas, tumors, diverticula, gallbladder dyskinesia, liver abscess.

Radionuclide cholecystinography. Hepatobilistin-tygraphic dynamics allows not only to assess the obstruction of bile outflow, the

concentration and contractility of the gallbladder, sphincter dysfunction, but also to determine the size and deformation of the liver and gallbladder. Children with hepatomegaly undergo laparoscopy to determine the location, size, surface area, and color of the gallbladder.

General blood analysis: usually unchanged in biliary tract disease, leukocytosis in acute cholecystitis is accompanied by neutrophilia, increased ESR, leukopenia in chronic cholecystitis and increased ESR.

Biochemical blood test: An increase in the level of bilirubin, alkaline phosphatase, cholesterol, lipoproteins, triglycerides, glutamyl transpeptidases, transaminases associated with impaired conduction of the biliary tract in cholestasis and cholangitis is of great diagnostic value. Exocrine pancreatic insufficiency is determined by the results of the coprogram (primarily by the presence of neutral fats, fatty acids) and is clearly confirmed by a low concentration of pancreatic elastase in the feces. Diseases of the biliary tract are compared with gastroduodenitis, ulcers (hence esophagogastroduodenoscopy), chronic pancreatitis, nonspecific mesadenitis and pseudotuberculosis, nonspecific ulcerative colitis, parasites, pyelonephritis. check for grammar and stylistic mistakes.

DYSKENESIA OF THE BILIC TRACKS

Biliary dyskinesia refers to the narrowing of the gallbladder and bile ducts, impeding the proper flow of bile.

According to statistics, the incidence of this disease in Uzbekistan stands at 70%, accounting for a ratio of 1000:700. This high prevalence is attributed to factors such as helminthic invasions among the child population, inadequate nutrition, psycho-emotional stress, post-viral hepatitis status, among others. Consequently, this disease remains a pressing issue. Given these factors, a deeper understanding of the etiopathogenesis, clinical manifestations, early diagnosis, differential diagnosis, effective treatments, and modern approaches to reducing or eliminating its prevalence is essential.

Etiology.

The factors contributing to biliary dyskinesia include:

- Neurocirculatory dysfunction from various origins
- Acute viral hepatitis
- Neuroses

- Food allergies
- Chronic gastrointestinal tract diseases
- Gastrointestinal tract parasitosis (such as giardiasis)
- Hereditary predisposition
- Chronic infection foci within the body
- Poisoning
- Endocrine diseases

Pathogenesis

Since the gallbladder and bile ducts form a single system, their inflammatory process often occurs together. The infection is transmitted by hematogenous, lymphogenous and ascending routes, and stagnation in the gallbladder also plays an important role in the development of the inflammatory process. Dyskinesia of the gallbladder causes an increase in pressure in the biliary tract system in violation of the function of the sphincters. This, in turn, leads to a violation of the physicochemical properties of bile, a violation of the ability to kill (kill) bacteria in bile. As soon as the colloidal composition of bile is destroyed, bilirubin, cholesterol and calcium precipitate in the form of a precipitate in the gallbladder and bile ducts. This condition leads to disruption of local blood circulation and excessive production of histamine, serotonin, heparins from biologically active substances. If the patient has duodenobiliary reflux, the fluid from the duodenum enters the bile ducts, which, under the influence of proteolytic enzymes, violates the anatomical integrity of the mucous membranes of the bile ducts, causing an inflammatory process in the neck of the gallbladder. The immunobiological state of the body is also important in the mechanism of development of inflammatory diseases of the biliary tract and gallbladder.

PATHOMORPHOLOGY

In acute cholecystitis, the inflammatory process can induce significant changes, ranging from mucous membrane damage to extensive alterations in the gallbladder walls. Various manifestations such as catarrhal, phlegmonous, and gangrenous changes are observed. Meanwhile, chronic cholecystitis often presents with gallbladder and duct deformation, along with scarring, coupled with signs of ongoing inflammation in the gallbladder and bile ducts. Additionally, biliary stenosis and impaired gallbladder motility can be detected.

CLASSIFICATION

For the first time M.Ya. Studenkin proposed the following classification.

1. Congenital defects of the gallbladder and biliary tract.
2. Tumors of the biliary tract.
3. Gallstone disease (atypical) typical.
4. Inflammation of the bile ducts.

Depending on location

A) cholecystitis
B) cholangitis
V) cholecystocholangitis

Flow:

1. Sharp
2. Chronic

A) hidden
B) Recurrent

The nature of the inflammation.

A) catarrhal.
B) phlegmatic.
B) gangrenous.

5. Biliary dyskinesia

a) giardiasis
b) opisthorchiasis
c) ascariasis
d) fascioliosis

Classification of biliary dyskinesia according to A.A. Mazurin, A.M. Zaprudnov, V.L. Paykov 1998.

1. For the sphincter apparatus of the biliary tract: (sphincter spasm), hypotonic (sphincter insufficiency), atonic, mixed.
2. From the side of the gallbladder and biliary tract: hyperkinetic, hypokinetic, mixed.

The clinical presentation of inflammation often manifests in both latent and acute courses. Cholecystocholangitis can remain latent, sometimes escaping the attention of both the patient and their parents. Initial complaints related to the digestive system include loss of appetite, nausea, belching, abdominal pain post-meals, and a sense of heaviness beneath the right ribs. According to M.Ya. Studenkin and E.F. Chomakov, primary complaints usually revolve around dyspeptic symptoms and pain

syndromes. Pain is commonly felt in the right hypochondrium, epigastrium, around the navel, occasionally varying in intensity. In severe cases, excruciating pain might radiate to the infraclavicular and subscapular regions, accompanied by elevated body temperature, flatulence, nausea, vomiting, and potentially leading to hospitalization under a misdiagnosis of appendicitis.

Intense pain often induces muscle tension in the right hypochondrium. Dyspeptic syndrome presents with heartburn, nausea, vomiting, and diarrhea. Children with chronic hepatocholecystitis exhibit signs of intoxication, such as pale skin and mucous membranes, general malaise, weakness, mood swings, dry skin, morning bad breath, and a white-coated tongue.

Objectively, examination of patients reveals various symptoms indicative of biliary tract and gallbladder diseases. These symptoms commonly include signs of intoxication, such as pallor of the skin and mucous membranes, general malaise, weakness, mood swings, dry skin, morning bad breath, and a white-coated tongue.

During an objective examination of the patient, several symptoms indicative of biliary tract and gallbladder diseases become apparent:

1. Zacharin's symptom: Pain in the gallbladder area upon palpation and tapping with a hand.
2. Obraztsov's symptom: Sharp pain in the right hypochondrium during deep breathing while palpating.
3. Ortner's symptom: Pain in the right chest upon tapping the right costal arch with a hand.
4. Glinchikov's symptom: Tension in the anterior abdominal wall around the right hypochondrium during comparative jerky palpation.
5. Lepin's symptom: Pain in the gallbladder area upon tapping with bent fingers.
6. Kharitonov's symptom: Pain experienced in the right shoulder blade region.
7. Mendel's symptom: Pain upon hitting the abdominal wall during breathing.
8. Mirizzi's syndrome: Jaundice and pruritus due to blockage of the hepatic bile ducts.
9. Apo-Ressle syndrome: Jaundice and pruritus resulting from obstructive cholangitis.

10. Werbraik's syndrome: Pain and tension sensation in the epigastric region, combined with dyspeptic symptoms, occurring when the gallbladder adheres to the hepatic angle of the colon. Pain intensity often decreases in the evening and while the patient is in a horizontal position.

In the hypertensive form, sharp, cutting, stabbing pains occur around 30-40 minutes after eating, lasting between 5-15 minutes. The maximum basal pressure of this sphincter should not exceed 40 mm Hg.

Differential Diagnosis.

Differential diagnosis primarily involves distinguishing between chronic cholecystocholangitis, cholelithiasis, gastroduodenitis, peptic ulcer, chronic hepatitis, and pancreatitis.

Treatment.

Diet number 5. Food should be prepared in a manner that protects it chemically, mechanically, and thermally. Meals, comprising 5-6 servings a day, aim to ensure a rhythmic release of bile. Evening choices should lean towards fermented milk products like kefir, yogurt, or fermented baked milk. Children should have dinner 2-3 hours before bedtime, focusing on lean foods. The diet excludes extractive foods such as pepper, onion, garlic, black pepper, radish, turnip, smoked meats, salty foods, mushrooms, meat, fatty foods, lamb meat, and beef fat. This exclusion is due to the challenges posed by fat digestion, constant bile penetration into the intestines, and reduced pancreatic lipase enzyme activity. Digesting vegetable fats like sunflower or olive oil requires minimal bile and enzymes, making them beneficial for this condition.

Additionally, chocolate, cocoa, and coffee confectionery should be removed from the diet. For children with hypertensive and hyperkinetic dyskinesia types, it's advisable to limit gas-producing foods like peas and legumes. Extremely cold food and liquids can trigger sphincter spasms, leading to discomfort. For individuals with hypotonic and hypokinetic forms, foods with choleretic properties such as butter, vegetable oils, sour cream, and eggs are recommended. Increased intake of fruits and vegetables is also encouraged. This dietary regimen is suitable for children over 1 year old.

Treating biliary tract diseases in children places significant emphasis on diet. During the acute phase of cholecystitis, a diet similar to that for acute liver diseases is prescribed, with limited salt intake. During

fever peaks, easily digestible diet days are suggested, including options like milk-curd, apple, compote, watermelon, and grapes.

Patients with biliary tract diseases are advised to increase their meal frequency to 4-6 times a day, which aids in improving bile secretion. The daily caloric intake should match that of a healthy child. In a hospital setting, patients are placed on the Pevzner's diet number 5. Protein and carbohydrate intake should match the child's age or slightly exceed it. Restricting proteins is impractical as they stimulate bile acid formation, influence cholesterol ratio, prevent stone formation, and enhance the body's immune reactivity. However, limiting nitrogen-containing extractives formed during high-temperature cooking processes (e.g., frying) is advisable. It's recommended to enrich the diet with foods rich in dietary fiber, lipotropic substances, and methionine (such as wholemeal bread, cottage cheese, egg whites, oatmeal, fish, and yeast drinks).

While many authors suggest limiting fat intake, excessive restriction can be detrimental as fats strongly stimulate bile secretion and aid in fat-soluble vitamin absorption. Patients with cholecystitis should avoid foods like lard, fatty meats (poultry, fish, lamb), egg yolks, fresh bread, chocolate, legumes, salty and sour foods, and cream. Insoluble fats should be minimized, with only animal fat butter being permissible. Vegetable oils (such as corn, sunflower, olive) are highly beneficial due to their unsaturated fatty acids (like arachidonic, linoleic, and linolenic acids), which increase bile secretion and improve liver function. These oils can be added to salads, snacks, sauerkraut, or consumed in teas and desserts, approximately 2-3 tablespoons per day before meals. However, vegetable oils should not be used for frying.

Carbohydrates are only restricted in foods containing indigestible fiber (like cabbage, turnip, etc.). Increased fluid intake is recommended as it enhances bile secretion. Patients are provided with dairy, vegetarian and fruit-based soups, dense foods such as boiled vegetables, cereals, puddings, boiled fish, and meat (avoiding pork, lamb, fatty poultry, brain, and kidneys due to their high extractive substance content). Liquids such as weak tea, compotes, fruit juices (not canned), milk, yogurt, kefir, fermented baked milk, stale white or black bread, processed cheese, and mild cheeses are permissible. Foods rich in magnesium salts like wholemeal bread and pastries, buckwheat, oatmeal, raw fruits, and vegetables are beneficial.

The alleviation of pain should be a priority. Atropine solution (0.1%, 5 drops per year of age), belladonna extract (1 mg per year of age), papaverine, no-shpu, theophylline, theobromine, aprofen, or tramal may be prescribed. If oral medications are ineffective, intramuscular administration of baralgin, 0.2% platyphylline solution, 0.1% atropine sulfate, 1-2% papaverine hydrochloride solution, moderate ganglion blocker doses, and 0.5% novocaine solution (3-5 ml) with 10-15 ml of 5% glucose IV may be considered. In persistent painful attacks, a combination of 1% promedol or pantopon with atropine can be administered. Mild heat in the form of warm heating pads over the right hypochondrium or warm compresses is recommended during painful episodes. However, if complications indicating surgical treatment are suspected (peritoneal reaction, perforation, pus), it's advisable to use ice on the abdomen to limit the inflammatory process.

Treatment with antibiotics. Indications for antibiotic treatment in biliary tract diseases include pain, fever, leukocytosis, accelerated ESR, and biliary tract inflammation. The course of antibiotic treatment typically lasts 7-10 days. Prolonged or non-exacerbated use of antibiotics can be ineffective and even harmful, leading to dysbiosis and fungal growth. With cholecystitis, antibiotics should be used in combination with bactisubtil and, of course, vitamins (C, B group, A). When choosing an antibiotic, it is advisable to consider the sensitivity of the flora from bile culture, especially portion B. If the clinical signs do not allow waiting, broad-spectrum antibiotics such as ampiox, gentamicin, and cephalosporins are necessary. Additionally, Nicodin (a product consisting of nicotinic acid amide and formaldehyde) exhibits antibacterial effects similar to oxaphenamid, cyclovalone, and furazolidone.

In cholecystitis treatment, herbal preparations play a crucial role in reducing bile stagnation in the affected gallbladder and alleviating inflammation. The herbal mixture by N.G. Kovaleva is widely used, containing dome spruce (fruits) - 10 g, medicinal chamomile (flowers) - 20 g, fenugreek (branches) - 30 g, white birch (leaves) - 10 g, medicinal cloves (aerial parts), dill (seeds) - 10 g, forest gnafalium (grass) - 10 g, uterine column of cornflower - 30 g, wild rose hips (visible fruits) - 40 g, wild strawberries (fruits) - 20 g, white roses (flower petals) - 20 g. Infusing 5-6 g of this mixture in 500 ml of boiling water, then taking 50-150 ml of this tincture 3 times a day 10-15 minutes before meals. The taste is slightly bitter, but the smell is pleasant. In cases with hyperkinetic

symptoms, peppermint can be added or the following collection used: yarrow and valerian root, field tea (herb) - 5 g, marsh gnafalium (herb) - 5 g, peppermint - 3 g, wild rose (fruits) - 10 g, medicinal chamomile (flowers) - 5 g. Pouring this mixture with 200 ml of boiling water, infusing for 5-6 hours, cooling, and straining. This decoction is consumed in a tablespoon between meals. The course of treatment is 3 weeks.

Blind probing, proposed by G.S. Demyanov in 1948, has been widely recognized for treating cholecystitis. The patient consumes a warm 33% magnesium sulfate solution on an empty stomach in the morning and lies on their right side with a heating pad on the gallbladder area. For children, a 50-75 ml 20% xylitol solution is administered in the morning on an empty stomach, followed by lying on the right side with a heating pad. An hour later, the child takes 1 tablespoon of a 30% solution of magnesium sulfate or half a glass of hot borax. After two hours, the child performs 8-10 sitting motions, resulting in a bitter taste in the mouth, indicating successful probing. This process is carried out 1-2 times weekly for a course of 10-16 probes. Tubage is not possible in cases of cholecystitis with erosive and ulcerative processes in the stomach and duodenum.

For chronic cholecystitis, consuming water rich in fine and medium mineralized hydrocarbons, sulfates, chlorine, magnesium, sodium, and calcium is recommended. Thermal (35-420 C°) or hyperthermal (42-500 C°) mineral waters stimulate bile formation, liquefy it, and reduce viscosity. Water intake should be in small portions at a rate of 3 ml per 1 kg of body weight. Commonly used mineral waters include Essentuki No. 4, 17, 20, Smirnovskaya, Borjomi, Slavyanskaya, Naftusia, Izhevskaya, Old Russian, Arzni, thermal, and Jermuk. For cholecystitis complicated by hyperacid gastritis, mineral water (Essentuki No. 4, Slavic, Smirnovskaya, Borzhom) is taken 1-1.5 hours before meals, and for hypoacid or normoacid gastritis, 40 minutes before meals. The treatment course with mineral water is 1-1.5 months, followed by subsequent courses every 3-6 months.

Physiotherapy treatment. During the onset of cholecystitis, several high-frequency sessions are administered in the solar plexus region, followed by subsequent procedures like electrophoresis, diathermy, paraffin and ozokerite applications, typically comprising 10-15 sessions involving magnesium sulfate (or alternatives like novocaine,

papaverine, dionine). The specific physiotherapeutic properties are tailored to address any gastritis complicating the course of cholecystitis.

Physiotherapy plays a crucial role in enhancing gallbladder motility and forms a vital component of treatment for patients dealing with biliary dyskinesia and chronic cholecystitis. However, patients should avoid excessive physical exertion, sudden movements, pushing, or lifting heavy weights as these activities are prohibited during treatment.

TEST:

1. The most commonly observed source of bleeding from the upper parts of the digestive system:
   a. stomach ulcer;
   b. stomach cancer;
   c. Mallory-Weiss syndrome;
   d. duodenal ulcer;

2. What method is the most informative for suspected duodenal ulcer:
   a. fibrogastroscopy
   b. pH-metry
   c. probing of the stomach
   d. biopsy

3. Diagnosis of Helicobacter pylori infection includes:
   a. Urease breath test
   b. X-ray of the stomach
   c. electrogastrography;
   d. caprological studies

4. A common complication of peptic ulcer in children is:
   a. bleeding
   b. penetration
   c. Perforation
   d. Perivisserite

5. The following indications for Helicobacter pylori therapy in children include:

a. erosive lesions of the gastric and duodenal mucosa
b. focal superficial changes in the gastric mucosa;
c. the presence in the family of patients with cholelithiasis;
d. hemolytic anemia;

6. What antacids should not be used for peptic ulcer?
a. Baking soda
b. Almagel
c. Phosphalugel
d. De-nol

7. The most common etiological factors of peptic ulcer are:
a. gastroduodenal reflux
b. Helicobacter pylori
c. Neuro-psychic factor
d. That's right

8. A sign of endoscopic healing of duodenal ulcer in children is:
a. Severe local hyperemia
b. Deformation of the duodenum 12
c. Wound size reduction
d. Epithelialization of the wound defect

9. What group does Omeprazole belong to?
a. M-anticholinergics
b. proton pump inhibitor
c. H2-blocker of histamine receptors
d. Adrenoblockers

10. Protective factor of the gastric mucosa:
a. Mucus formation
b. Pepsin
c. Hydrochloric acid
d. Gastrin

11. The main syndromes of peptic ulcer:
a. Painful, dyspeptic, intoxication
b. Painful, dyspeptic, hemorrhagic

c. Dysuric, dyspeptic intoxication
d. Pain, hemorrhagic, dysuric

12. Cushing's ulcers are symptomatic ulcers, on what background do they develop?
a. When taking glucocorticoids
b. Traumatic brain injury
c. Neurosurgical operations
d. General burns

13. What indicators of the gallbladder cannot be determined by ultrasound?
a. gallbladder functions
b. tone of the sphincter apparatus of the gallbladder
c. abnormal forms of the gallbladder
d. concentration function of the gallbladder

14. Specify the main clinical signs of cholestasis syndrome:
a. jaundice, hepatosplenomegaly
b. jaundice, pruritus
c. abdominal pain, jaundice
d. pale, itchy skin

15. Changes characteristic of the hypotonic type of dyskinesia in the gallbladder with ultrasound:
a. gallbladder reduction
b. gallbladder enlargement
c. liver shrinkage
d. the shape of the gallbladder does not change

16. Changes characteristic of the hypertonic type of dyskinesia in the gallbladder with ultrasound:
a. liver shrinkage
b. gallbladder enlargement
c. gallbladder reduction
d. the size of the gallbladder did not change

17. What drugs are used in the treatment of hypotonic form of biliary dyskinesia?

a. Analgesics
b. Antispasmodics
c. Choleretics and cholekinetics
d. Hepatoprotectors

18. What drugs are used in the treatment of hypotonic form of biliary dyskinesia?

a. Analgesics
b. Hepatoprotectors
c. cholekinetics
d. Antispasmodics and sedatives

19. Specify the main syndromes characteristic of chronic cholecystocholangitis:

a. Pain, dyspepsia
b. Pain, dysuria
c. Intoxication, hemorrhagic
d. Dyspeptic, hemorrhagic

20. What determines the nature of pain in cholecystocholangitis?

a. By patient's age
b. State of the nervous system
c. Type of dyskinesia
d. For the duration of the disease

21. The main etiological factor of chronic hepatitis is:

a. bacteria
b. Viruses
c. parasites
d. Mushrooms

22. What is included in the required biochemical studies in hepatobiliary pathology?

a. Total protein fractions, transaminase, bilirubin, cholesterol
b. Total protein, C-reactive protein, seromucoid, bilirubin
c. Total protein fractions, urea, creatinine, cholesterol

d. Total protein fractions, C-reactive protein, seromucoid, urea

23. Based on what signs is chronic cholecystitis diagnosed?
a. If there are signs of intoxication
b. If it hurts under the right rib
c. If the walls of the gallbladder are thickened on ultrasound
d. If a complete blood count shows signs of inflammation

24. In the chronic course of what pathological process does viral hepatitis end with cirrhosis of the liver?
a. autoimmune hepatitis
b. Viral hepatitis A
c. Viral hepatitis E
d. Viral hepatitis C

25. What drug is most effective in the treatment of autoimmune hepatitis?
a. Prednisolone
b. Azathioprine
c. Ribavirin
d. Essentuki

26. What is the most informative method for diagnosing chronic hepatitis?
a. Morphological
b. Clinical and biochemical
c. Immunological
d. Biological

27. Specify the drugs most effective for the treatment of patients with autoimmune hepatitis:
a. cytostatics + interferon;
b. corticosteroids + interferon;
c. Ursodeoxycholeic acid + corticosteroids
d. corticosteroids + cytostatics;

28. Specify the types of chronic hepatitis in children:
a. Viral, autoimmune, drug-induced, toxic

b. Persistent, active, autoimmune
c. drug, autoimmune, alcohol
d. cryptogenic, viral, toxic

29. Stages of activity of chronic hepatitis:
a. active (light, medium, heavy), inactive
b. active (minimal, pronounced, explicit), inactive
c. active, incomplete clinical and laboratory remission, complete clinical and laboratory remission
d. incomplete clinical and laboratory remission, complete clinical and laboratory remission

30. Is it typical for minimal activity of chronic hepatitis?
a. excess of ALT above the norm by 3 times
b. Normal amount of ALT
c. Excess ALT above the norm by 5-10 times
d. Excess ALT above the norm by 10 times

Answers:

No.	Answer	No.	Answer	No.	Answer
1	d	11	a	21	b
2	a	12	d	22	a
3	a	13	b	23	c
4	a	14	b	24	d
5	a	15	b	25	a
6	a	16	c	26	a
7	b	17	c	27	d
8	d	18	d	28	a
9	a	19	a	29	b
10	a	20	c	30	a

SITUATIONAL TASKS

Task #1

The patient is 10 years old. Complaints at admission: pain in the epigastric and pyloroduodenal region, decreased pain after eating, constipation. From the anamnesis, the child has been ill for a year. The mother has been suffering from duodenal ulcer for 5 years. She is regularly treated. Upon arrival at the hospital, the general condition is moderate, the skin is pale, the tongue is covered with a white coating. Palpation of the abdomen is determined by pain in the epigastric and piloduodenal region. The liver and spleen are not enlarged. Prone to constipation. On palpation, pain in the projection of the duodenum is determined. With gastroduodenofibroscopy, edema, hyperemia and a defect in the form of an oval on the mucous membrane of the duodenum are observed.

Question:

1. Your clinical diagnosis.
2. Principles of treatment.

Task #2

A 10-year-old patient was brought to the children's department with complaints of weakness, nausea, vomiting, and abdominal pain. Sick since 2 years. From the anamnesis, he complains of constant nausea, burning in the throat, stuttering. Due to the fact that the parents did not pay special attention to this, the child was not checked anywhere. The mother does not associate the onset of the disease with anything. But, according to her, the child always violates the diet (does not have breakfast at home in the morning. He has lunch after the arrival of his mother at 15.00, he eats dinner very late). Eats in a hurry, after eating and physical exertion, pain in the epigastric region increases. The pain lasts 10-20 minutes. Constant nocturnal and hungry pains are observed. The child is pale, appetite is weakened, sometimes an unpleasant smell comes from the mouth. Palpation of the abdomen reveals severe pain in the pyloroduodenal zone, X-ray examination of the stomach and duodenum changes the mucous membrane of the stomach and duodenum 12, the folds are thickened and expanded, the contour from the abundance of mucus is clear and heterogeneous. In endoscopic examination, the

mucosal folds are swollen and thickened, on the duodenal mucosa there is a defect 0.3-0.4 mm in size.

Question:

1. Your clinical diagnosis.
2. Dispensary control.

Task #3

A 13-year-old patient was admitted to the children's department with complaints of aching pain in the epigastric region, weakness. From the anamnesis, there is constant pain in the epigastric region, more than 2 hours, increases before eating, subsides when eating. Hunger pains are often observed. The child's diet is irregular, spicy, salty foods predominate. The child has been sick since the age of 2. My paternal grandmother, maternal aunt and sister had stomach problems. Fractional study of gastric juice acidity and secretion increased.

Question:

1. What additional methods of examination should be carried out to establish the diagnosis.
2. Your preliminary diagnosis.
3. Treatment plan.

Task #4

A 13-year-old girl complains of abdominal pain, the pain is aggravated by eating solid food, heartburn, constipation, headache. The patient has been ill for 12 months. The child violates the diet. Tongue wet, covered with white coating. The abdomen is soft, palpation is determined by pain in the epigastric region.

Question: what methods of examination should be carried out for the diagnosis of the patient.

Task number 5.

A 12-year-old patient complains of diarrhea, copious stools more than 3 times a day, abdominal pain and a feeling of incomplete bowel movement. According to the mother, the child has been sick for several years. On objective examination, the condition is moderate, the skin is pale. Slightly decreased turgor and elasticity of the skin. On palpation, abdominal pain and flatulence are observed. In scatology: stools are liquid, mushy, with an admixture of mucus.

Your preliminary diagnosis.

Task number 6.

A 10-year-old patient complains of constipation, stools no more than 2 times a week, abdominal pain and straining during defecation, bloating. According to the mother, these signs have been observed for several years, he has not been treated anywhere. On palpation of the abdomen, pain and flatulence are observed. In scatology: feces are hard, formed.

Your preliminary diagnosis.

Task number 7.

A 9-year-old patient, according to her mother, complains of bloating, frequent stools more than 3 times a day. These signs are observed in a child for several years. The mother did not go anywhere and did not treat, pain and flatulence are observed during palpation of the abdomen.

In scatology: stool-liquid (watery) with mucus.

Question: What diagnostic methods do you use?

Task number 8.

According to the mother, a 5-year-old child complains of pain and bloating in the abdomen, loss of appetite. The stool is mushy and slimy. The patient was not treated. On palpation of the abdomen, pain and flatulence are observed. In coprology: stools are liquid (watery) with an admixture of mucus.

Your treatment strategy

Task number 9

Patient, 4 years old. Complaints of paroxysmal pain in the right iliac region, heartburn, nausea, bitter taste in the mouth in the morning. From the anamnesis: two years ago he was treated for helminthic invasion (giardiasis). The general condition of the patient is moderate. The skin and visible mucous membranes are pale. The eye sclera is yellowish, the abdomen is soft on palpation, the liver is +2 cm. The spleen is not enlarged. Urination and stool are normal. In a laboratory study: A blood test showed anemia, elevated AST, ALT.

1. What is your diagnosis?

2. What instrumental methods of examination should be carried out?
3. What is your treatment strategy?

Task number 10

The patient is 3 years old. Complaints of pain in the epigastric and right iliac regions, nausea, constipation. From the anamnesis: the child is often sick and suffers from chronic tonsillitis. The general condition of the patient is moderate. The skin and visible mucous membranes are pale pink. The eye sclera is yellowish. The abdomen is soft on palpation, the liver is +2 cm. On palpation of the abdomen, pain is observed in the epigastric region. Ortner's sign is positive. Urination and stool are normal. Laboratory: a blood test showed anemia and an increase in ESR, ALT and AST are elevated, thymol test is positive.

1. What is your diagnosis?
2. What additional methods of laboratory research are carried out?
3. Diet therapy.

Situational questions sample answers:

Task#1

Answer: 1. Main: duodenal ulcer, acute course.

Violation of the functional state of the stomach. Uncomplicated type.

1. Table No. 1A, 1B.

sedative therapy.

Antispasmodic drugs.

Triple or quadruple therapy.

Physiotherapy.

Task#2

Answer: 1. duodenal ulcer, acute course, increased functional state of the stomach, uncomplicated type.

2. Children suffering from peptic ulcer must remain in department "D" on the basis of Form No. 30. Dispensary observation must be at least 5 ails from the onset of the disease.

TaskNo. 3

Answer: 1. Contrast fluoroscopy of the duodenum and stomach, gastrofibroscopy, detection of helicobacter pylori, examination of feces for the Grigersen reaction.

2. Chronic gastroduodenitis with an increase in the secretory function of the stomach, a period of exacerbation.

3. Diet therapy. Table number 1, sedatives, antioxidants, Helicobacter pylori eradication, antispasmodics, antacids.

Task#4

Answer: Ph-metry, fractional study of gastric juice, contrast fluoroscopy, esophagogastrofibroscopy, Grigersen reticulation, determination of helicobacter pylori in a biopsy.

TaskNo. 5. Irritable bowel syndrome with diarrhoea.

TaskNo. 6. Irritable bowel syndrome with a predominance of constipation.

TaskNo. 7. General analysis of blood, urine and stool, bacteriological examination of stool, 3-fold examination of stool for vomiting, biochemical blood test, sigmoidoscopy or colonoscopy, irrigoscopy.

TaskNo. 8. Diet. Restricted products: milk, marinades, carbonated drinks, smoked meats. Imodium, Loperamide, adsorbents (Smecta, Kaopektate, Polyphepan, Enterodez, Multisorb), probiotics (Enterol).

Task#9

1. Biliary dyskinesia

2. Ultrasound, X-ray examination of the gallbladder with a contrast agent.

3. Table No. 5, antispasmodics (No-shpa, papaverine, spasmalgon), choleretic agents (cholasas, allahol, Cholenzim), sedatives (valerian, motherwort, sedovitis), physiotherapy.

Task No. 10

1. Biliary dyskinesia.

2. Coprogram, general analysis of feces for worm infestation. biochemical study of bile.

3. Table number 5 (fatty, spicy, smoked, fried foods are limited)

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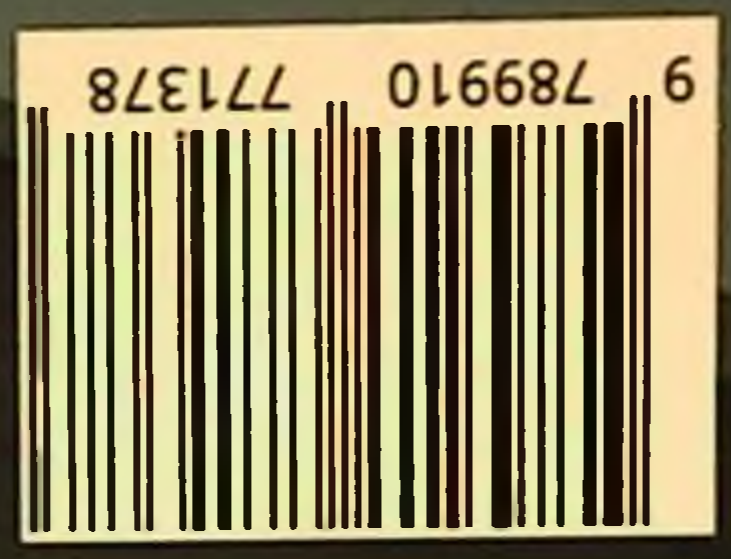
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