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ZAPORIZHZHYA STATE MEDICAL UNIVERSITY
DEPARTMENT OF INTERNAL DISEASES -1

EVALUATION DATA OF LABORATORY

AND INSTRUMENTAL STUDIES IN GASTROENTEROLOGY

TRAINING MANUAL

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The training manual contents the methodological approaches to evaluation data of laboratory and instrumental studies in patients under the program of discipline "Internal Medicine" Content Module 2" Principals of diagnostics, treatment and prophylaxis digestive system diseases" specialties: 7.12010001 "Medicine" and 7.12010002 "Pediatrics". These educational materials will help students better mastering of skills and abilities to analyze the results of clinical-laboratory and instrumental studies in the most widespread diseases in gastroenterology. The need for the proposed publication of the manual due to the changes in the discipline program, approved by Ministry of Health of Ukraine in 2014.

CONTENT

INTRODUCTION

In gastroenterology clinic play of great importance laboratory and instrumental studies, permitting the identification of pathological changes in the organism, to make an early diagnostics of diseases of the gastrointestinal tract and monitor the effectiveness of treatment. Knowledge of research methods, their diagnostic value in gastroenterology will allow students to select the most informative ones at every nosology and interpret the results. Training manual includes information on the most informative research methods that are appropriate to use in the diagnostics of diseases of the digestive system. The manual contains information material for self-training students for practical lessons, a description of the main clinical syndromes, modern diagnostic methods in gastroenterology with the assessment of research results, tests, tasks with state exams "STEP 2" required for mastering the subject matter "Internal medicine" contents module "Fundamentals of diagnosis, prevention and treatment of major diseases of the digestive system." The need for the proposed publication of the manual due to the changes in the discipline program, approved by Ministry of Health of Ukraine in 2014.

1. THE MAIN SYMPTOMS GASTROINTESTINAL SYMPTOMS AND SYNDROMES

Violation of appetite (decrease or increase) occurs in many diseases. Decreased appetite is often seen in patients with chronic gastritis with reduced gastric function, at various poisoning, infectious diseases. Complete loss of appetite (anorexia) is characteristic for gastric cancer. Increased appetite occurs in diabetes mellitus, in convalescent patients, at duodenal ulcer.

Pain is one of the main symptoms of the lesions of the gastrointestinal tract. Often the pain associated with food intake. Accordingly, the time elapsed since eating before the appearance of pain, decided to devote early and late pain. Early pain occurs in 30-60 minutes after meals and more characteristic for the stomach lesions with its localization in the epigastric region**.**

Late pain occurs in 1.5 - 3 hours after a meal and characteristic for the patients with duodenitis and duodenal ulcer. Such patients often observed hungry, night and seasonal (spring, autumn) pain with localization in the epigastric region to the right of the median line. Pain during bowel lesions is often spastic character associated with the act of defecation and its localization depends on what his department affected. In patients with liver and biliary diseases pain most often localized in the right upper quadrant. By the nature of pain can be long or attacks, dull or sharp, strong or moderately expressed. Paroxysmal pain (biliary colic or hepatic) occurs suddenly and rapidly becomes extremely intense, often observed in cholelithiasis.

Vomiting - complex reflex act of spontaneous ejection of stomach contents through the esophagus, throat, mouth and nasal passages. Customary to distinguish central, reflex and toxic vomiting. Vomiting of central origin appears at elevated intracranial pressure (with brain tumors, hypertensive crisis), the sea and air disease. Toxic vomiting occurs when exogenous (alcohol, poisons) and endogenous (chronic renal failure, toxemia of pregnancy) intoxication. Vomiting reflex - any severe pain, such as renal or biliary colic can trigger vomiting reflex. A characteristic feature of vomiting in diseases of the stomach (eg, ulcers) is that it brings relief; therefore, patients can call it artificially to reduce pain. The great diagnostic importance are following features the amount of vomit, their smell, color, texture, nature of food

residues, presence of pathological impurities. Thus, a large amount of vomit food eaten before, with the smell of hydrogen sulfide, or "rotten eggs," is observed for pyloric stenosis. Bloody vomiting or vomiting "coffee ground" is a symptom of gastrointestinal bleeding.

Nausea is reflected in an unpleasant pressure in the epigastric region, accompanied by dizziness, sweating and skin pale. Nausea often preceded by vomiting due to the same reasons as vomiting. Nausea is often observed in patients with chronic gastritis, peptic ulcer and gastric cancer; can also occur after a breach of of the diet.

Heartburn - a feeling of heat and burning behind the sternum and in the xiphoid process, resulting in irritation of the lower esophagus often acidic content that is thrown from the stomach. Violations tonus of the lower esophagus and cardiac part of the stomach leads to backflow (reflux) of gastric contents into the esophagus.

Belching is the expulsion of gas from the gut through the mouth (eructations). Belching may be acidic (at elevated gastric secretion) and bitter (when hit in stomach bile from the duodenum). Belching putrid smell of hydrogen sulphide ("rotten eggs") indicates the long delay food in the stomach in patients with uncompensated pyloric stenosis. Belching air swallowing (aerophagia) may be voluntary or involuntary. Air usually enters the esophagus and is expelled as a belch. It usually observes in healthy individuals with overeating, use of carbonated drinks.

Gastrointestinal bleeding - one of the most serious complications of various diseases of the stomach and duodenum. Most often bleeding occurs at gastric ulcers, stomach cancer, erosive gastritis, gastric varicose veins. The main signs of gastric bleeding is vomiting with blood and black stool (as tar, melena). In patients with preserved stomach acidity the vomit has type coffee ground, which is caused by the formation of hematin hydrochloric. Melena is often accompanied by bloody vomiting, although it can occur without it, and appears usually 8-12 hours after bleeding. In cases where the source of bleeding is localized in the colon, the stool is marked admixture of unchanged blood. In dysentery, colon cancer blood usually mixed with feces, in hemorrhoids fresh drops of blood at the end of the act of defecation selection is observed.

Flatulence (bloating) is result from enhanced gas production in the intestine. The reason may be eating foods that contain large amounts of fiber and starch (beans, cabbage, and potatoes), excessive swallowing air, violating the emission of gas from the intestine (bowel obstruction), digestive disorders due to enzyme deficiency etc.

Diarrhea - loose, watery stools occurring more than three times in one day. Diarrhea occurs in different intestinal infections (dysentery, cholera), inflammatory bowel disease (enteritis, colitis), chronic gastritis with decreased secretion, chronic pancreatitis with exocrine insufficiency, functional disorders (irritable bowel syndrome).

Constipation (obstipation) - bowel emptying is delayed more than two days. If constipation stool water content decreases, it becomes hard, released from tension. Constipation may be organic (colon tumors, adhesions) and functional. Among functional reasons distinguished: alimentary (due to sedentary lifestyles, inadequate intake of fiber and fluid), neurogenic (spastic or atonic constipation), proctogenic constipation (hemorrhoids, anal fissures) etc.

2. INTERPRETATION OF LIVER FUNCTION TESTS

State of pigment metabolism determine by the content of general, conjugated (direct) and unconjugated (indirect) bilirubin in blood serum, bilirubin and urobilinogen in urine, stercobilin in feces.

Liver enzyme tests. Intracellular enzymes - indicators of hepatocyte cytolysis (ALT, AST, GLDH, GGT, LDH); membrane-bound enzymes are indicators of cholestasis (alkaline phosphatase); secretory enzymes are indicators of hepatic insufficiency (cholinesterase).

Used for the assessment of severity of the inflammation and necrosis in the liver and cholestasis.

On protein metabolism is judged by the content of serum total protein, protein fractions, the level of prothrombin index (prothrombin time), clotting factors, α-fetoprotein level (in patients with hepatocellular tumors). Hypoalbuminaemia and violation of the synthesis of coagulation factors (hypocoagulation) is indicator of hepatic insufficiency. Increased ß- and α2-globulins levels confirm in cholestasis; elevated γ-globulin level is one of the inflammatory process criteria in the hepatocytes (mesenchymal-inflammatory syndrome).

Lipid metabolism measured by serum total cholesterol, triglycerides, high density, low density and very low density lipoproteins level. In liver diseases (cholestasis, hepatitis, cirrhosis) can cause significant changes in lipid metabolism.

For differential diagnosis and assessment of severity of parenchymal liver disease and bile duct lesions is used to determine the activity of various liver enzyme tests. The sensitivity and specificity of these tests are limited and none of them reliably determine the location of the lesion**.** It should be remembered that the increase in enzyme activity can occur in extrahepatic diseases. At first determine the activity of aminotransferase, alkaline phosphatase, 5'-nucleotidase and amma-glutamyltransferase. Determination of the total activity of LDH and its isoenzyme for diagnosis of liver diseases shallow - this enzyme found in all tissues. A moderate increase of LDH activity observed in viral hepatitis, cirrhosis, malignant tumors of the liver and biliary tract lesions. The marked increase in activity of LDH,

accompanied by changes and other biochemical parameters of liver function may be observed at hemoblastoses (lymphoma).

Ornitinkarbamoiltransferasse – an enzyme involved in the urea cycle - found only in the liver and small intestine. The activity of this enzyme increases with various liver diseases and also has limited diagnostic value.

Biochemical parameters of liver injury: aminotransferase (transaminase).

About hepatocyte damage can be judged by to change the activity of some serum enzymes, the most important of which - alanine aminotransferase (ALT) and aspartate aminotransferase (AST). They catalyze the transfer of alpha-amino group of alanine and aspartate to alpha-keto group alpha ketoglutarate, resulting from the alanine and aspartate are formed according pyruvic and oxalic acid, and alpha-ketoglutarate - glutamic acid**.** ALT is mainly in the liver. AST is less specific and contained in many tissues, including the heart, muscles, kidneys and brain. Normally, AST and ALT activity in serum is less than 0.58 mckat / l (35-40 IU/l).

In hepatocytes ALT contains exclusively in the cytosol, various isoenzymes of AST are in the cytosol and mitochondria. Increased activity of these enzymes may occurred especially myocardial infarction and skeletal muscle lesions. But they can be distinguished from liver disease based on the clinical picture. In uremia aminotransferase activity may be reduced. AST and ALT activity increased in almost all diseases of the liver, but - especially in cases accompanied by widespread necrosis of hepatocytes (in severe acute viral hepatitis, toxic liver disease or severe shock). To a lesser extent their activity is increased in mild viral hepatitis, as well as chronic diffuse and local damage of the liver (chronic hepatitis, liver cirrhosis, liver metastases). A single definition of aminotransferase level does not indicate severity and prognosis of disease. Because enzymes values determine several times. Thus, in patients with severe liver necrosis aminotransferase activity may significantly rise in the early stages (the first 24-48 hours), but in 3-5 days it can be 3,34-5,8 mckat/L (200-350 IU/l). In severe alcoholic hepatitis there is only a moderate rise aminotransferase activity (usually less than 5 mckat/l). Mild increase of ALT and AST (less than 1.67 mckat/l) is possible with bile duct obstruction; more pronounced increase indicates the development of cholangitis and necrosis of hepatocytes. The

activity of ALT and AST changing the same, except for two cases. In alcoholic hepatitis ratio AST/ALT may be more than 2. This is due to decrease ALT activity caused by deficiency its coenzyme - pyridoxalphosphate. Ratio AST/ALT greater than 1 can occur in acute fatty liver of pregnancy. In fatty liver of other etiologies this ratio is less than 1.

Alkaline phosphatase (ALP) - an enzyme is detached from the cell membranes and is able to hydrolyze synthetic phosphoric esters at pH equal to 9. Its physiological role is unknown. In human serum contains a number of isoenzymes of alkaline phosphatase. The source of ALP are bones, intestines, liver and placenta. There are various methods of determining this enzyme. In the absence of pregnancy or bone damage elevated alkaline phosphatase indicates biliary diseases (cholestasis). Increased activity of alkaline phosphatase reflects its increased synthesis by hepatocytes and biliary tract epithelial cells and to a lesser extent, the reverse flow of the blood enzyme caused by obstruction of the biliary tract. Bile acids, on the one hand, induce the synthesis of alkaline phosphatase, and the other - contribute to its cleavage from cell membranes. Moderate (no more than in 2 times) increase of alkaline phosphatase activity occurs in many parenchymal liver diseases - such as hepatitis and cirrhosis. Generally, this transient increase in activity may at any liver disease. In malignant tumors of the liver or infiltrative liver diseases (leukemia, Hodgkin's disease, lymphoma, and sarcoidosis) alkaline phosphatase activity moderately elevates but sometimes may increase sharply (for example, mycobacterial infections). However, the most significant increase in alkaline phosphatase activity (10 or more times) is observed in extrahepatic bile duct obstruction or intrahepatic cholestasis (e.g., drug-induced liver disease or primary biliary cirrhosis). Conversely, normal alkaline phosphatase activity under these conditions is rare. Incomplete obstruction of bile duct or hepatic duct obstruction serum bilirubin level normal or slightly increased, but the ALP activity increases. The activity of this enzyme increases with extrahepatic diseases - disease of bones (Paget's disease, osteomalacia and bone metastases) and, sometimes, malignant tumors of bones. Some tumors synthesize isoenzyme, similar to placental alkaline phosphatase (isoenzyme Regan). Typically, you can always decide whether caused by increased activity of alkaline

phosphatase liver disease or other causes. In difficult cases, resorting to determine the activity of certain isoenzymes. So, liver isoenzyme of alkaline phosphatase unlike bone resistant to heat (56 °C for 15 minutes) and the action of urea. Isoenzymes of alkaline phosphatase can be divided electrophoretically, but in practice this does not apply. More informative is the simultaneous determination of ALP activity and 5'-nucleotidase: increased activity of both enzymes confirms liver damage.

It is necessary to take into account the age and sex of the patient (alkaline phosphatase activity is higher in children and older women). In adults with no signs of disease occurs isolated increase in alkaline phosphatase.

Biochemical parameters of liver injury: 5'-nucleotidase

5'-nucleotidase catalyze hydrolytic cleavage of phosphate in position 5 pentose nucleotides. Although this enzyme is distributed in all tissues, increasing its activity is usually observed in diseases of the liver and biliary tract.

5'-nucleotidase level determined mainly in order to confirm or rule out liver causes elevated alkaline phosphatase in children, pregnant women and patients with lesions of bones. However, the activity of 5'-nucleotidase and alkaline phosphatase is not always increased at the same time as the first normal values do not rule out liver damage.

Biochemical parameters of liver disease: amma-glutamyltransferase

Gamma-glutamyltransferase (GGT) catalyzes the cleavage residue gamma-glutamic acid from peptide as glutathione or transfer the balance to another peptide or amino acid. This enzyme may also participate in the transport of amino acids. It is found in all structures of the liver and biliary tract and other tissues. In liver diseases activity of GGT and alkaline phosphatase increased simultaneously**.** The increased activity of GGT is the most sensitive indicator of biliary tract lesions, but it nonspecific. This activity also increases in diseases of the pancreas, heart, kidneys, lungs, diabetes mellitus and alcoholism.

Biochemical syndromes of liver damage

Cytolysis syndrome. Accumulation of intracellular enzymes serum indicates necrotic processes in the body. Indicator enzymes (indicate necrosis of hepatocytes) are transaminases (AST, ALT), glutamate dehydrogenase (GLDH), LDH), serum iron

and B-12 vitamin level. Liberate enzymes from hepatocytes occurs either because of increased permeability of cell membranes, or due to destruction of cells. Necrotic hepatocytes can cause not hyperaminotransferasemia. Activity cytosolic enzymes (ALT, LDH) increases in a relatively mild process. Activity of mitochondrial enzymes (AST, GLDH) increases only in severe necrotic processes.

Cholestasis syndrome. Excretory enzymes (cholestasis indicators) located on the plasma membrane of hepatocytes and biliary cells in the tubules. Increasing their activity in the blood is a result of enzyme induction (increased synthesis) and increased permeability of membranes hepatocytes. Obstruction of bile flow intrahepatic cholangiols or in extrahepatic bile ducts is accompanied by selective stimulation of synthesis excretory enzymes. Cholestasis indicators are membrane-bound enzymes - alkaline phosphatase, gamma-glutamyltranspeptidase, leucinaminopeptidase, 5'-nucleotidase. The characteristic violation of pigment metabolism - increase of conjugated (direct) bilirubin and total bilirubin level. Cholestasis accompanied by the accumulation of cholesterol in the blood serum.

Immune inflammation syndrome. Elevated total serum protein level due to the accumulation globulins, particularly beta- and gamma-globulins. Positive sedimentary sample – thymol test. Elevated immunoglobulins A, M, G values.

Hepatic insufficiency syndrome. On the reduction of synthetic liver function shows low serum albumin level formed only in the liver, low protrombin index (prolong protrombin time), low values of other clotting factors. The sharp decrease of cholinesterase activity may lead to reduction of esterified cholesterol in serum of blood.

Hepatic hyperazotemia syndrome. There is increase total serum nitrogen level and aromatic aminoacids (tyrosine, fenilalanin, tryptophan) levels, high phenol and indicant serum values.

3. ESTIMATION OF ELISA BLOOD AND FECES TEST

The basis of the enzyme immunoassay (ELISA method) is the immune reaction of antigen antibody. Immuno-enzyme assay (ELISA) used in the diagnostics of urogenital infections to identify the presence of serum specific immunoglobulin (Ig or antibodies) - proteins produced by cells of the immune system to combat the body taken root infectious agent. Contacting the microbial cells or products of its life, immunoglobulins neutralize them and facilitate removal. Thus, immunoglobulins are the essence of humoral immunity, that immunity caused by substances contained in body fluids, unlike cellular immunity associated with the presence of specific immune cells. For every type (and even subspecies) pathogen specific antibodies produced that are usually not associated with any other infectious agent. This definition of antivirus antibodies provides a sufficiently high specificity analysis. Of the five classes of Ig in the diagnosis of infectious diseases are most commonly used three types of Ig A, M and G. For research by ELISA using blood from cubital vein obtained fasting. The advantages of ELISA include high sensitivity, specificity, reproducibility, uniformity and suitability for mass screening. Ability to evaluate the results of instrumental factor eliminates subjectivity.

ELISA serologic tests for diagnostics Helicobacter pylori. Normally, antibodies IgG to Helicobacterpylori at their qualitative determination in serum absent; the quantitative research of antibodies` titer IgG - at least 8 E/ml, 8-12 F/ml - "border zone". The most common method for serological diagnostics Helicobacter pylori is ELISA test. The method is non-invasive and indirect, in the patient's blood to determine antibodies to Helicobacter pylori, belonging to the IgA, IgM and (mostly) IgG. When using this method in general titer BP consider most valuable titer determination of IgG class of antibodies Helicobacter pylori. Its sensitivity ranges from 87% to 98%, specificity - 75-100%. A simple qualitative determination of antibodies to Helicobacter pylori by ELISA are mainly used for the diagnosis of infection. In recent years, received diagnostic test systems based on ELISA with high sensitivity and allow quantitative determination of antibodies to Helicobacter pylori different classes. Such test systems can be used for evaluation of eradication. It was

shown compared to invasive methods (histologic, urea) that if 30-40 days after treatment blood pressure IgG titer values fell by 20% or more can be assumed that as a result of treatment occurred eradication of Helicobacter pylori, if the value titer increased, unchanged or decrease of less than 20%, it should be regarded as a lack of eradication. Determination of titer of antibodies to Helicobacter pylori is necessary for the diagnosis of diseases caused by Helicobacter pylori, in including gastric and duodenal ulcer, gastric cancer and esophageal ulcer.

ELISA serologic tests for diagnostics of hepatitis. Today hepatitis A, B, C, D, E, G, TT are distinguished. Hepatitis A and E are transmitted through enteral not cause the chronic process, but may activate the disease in the presence of other hepatitis virus carriers. Hepatitis B, C, D, G, TT transmitted parenterally and cause usually chronic hepatitis. In practice, the laboratory research hepatitis A, B, C, D, G are subject.

Hepatitis A

- **Anti-HAV IgM**- antibodies to virus hepatitis A arise at the end of the incubation period, detect acute hepatitis or recent infection; stored in blood from 3 to 6 months.
- **Anti-HAV IgG** - antibodies to virus hepatitis A indicate the recent infection and the formation of stable immunity; can be used to evaluating the effectiveness of vaccination.
- **Hepatitis B**

- **HB surface antigen.** A protein on the surface of HBV; it can be detected in high levels in serum during acute or chronic HBV infection. The presence of HBsAg indicates that the person is infectious. The body normally produces antibodies to HBsAg as part of the normal immune response to infection. HBsAg is the antigen used to make hepatitis B vaccine.
- **HBe** antigen reveals in patients with acute hepatitis or in patients with chronic hepatitis from the end of the incubation period to the end of replication phase; indicates high levels of HBV. It don’t reveal at pre-core HBV mutants and HDV superinfection.
- **HBcor** antigen - don’t reveal in the blood serum;

- **Anti-HBs IgM and IgG** - antibodies to superficial antigen HBV. They appear in the end of the acute phase or in 3-8 months; are stored 10 and more years. The presence of anti-HBs is generally interpreted as indicating recovery and immunity from HBV infection. Anti-HBs also develops in a person who has been successfully vaccinated against hepatitis B.
- **Anti-HBe IgM and IgG** - antibodies to HBe-antigen. They appear on 2-3 weak of jaundice period; save to 2-5 years. They show the beginning of non-replication phase and help to detect chronic hepatitis due to pre-core HBV mutants in the absence HBe-antigen. Spontaneous conversion from E antigen to E antibody (a change known as seroconversion) is a predictor of long-term clearance of HBV in patients undergoing antiviral therapy and indicates lower levels of HBV.
- **Anti-HBcor IgM** - antibodies to nuclear antigen class M. Positivity indicates recent infection with HBV (≤6 months). Its presence indicates acute infection.
- **Anti-HBcor IgG** - antibodies to nuclear antigen class G. Appear with the beginning of the acute phase of hepatitis are stored during the entire future life. The presence of anti-HBc IgG in the absence of HBsAg is usually indicative of a past self-limiting HBV infection. But it is frequently associated with co-infection with HCV which can worsen the existing status of chronic liver disease. Isolated anti-HBc IgG is also frequently observed in intravenous drug abusers, HIV infected individuals, HBV and HCV co-infected patients and pregnant females.
- **HBV DNA** is the first marker to be detected by polymerase chain reaction (PCR) before HBsAg reaches detectable levels**.** DNA virus appears in blood at the end of the incubation period and appears during the whole period of replication in chronic hepatitis.

Hepatitis D

The assays are conducted only in patients with hepatitis B.

- **Anti-HDV** (total) at acute hepatitis reveal in low titer (may be only in the phase of reconvalescence), are stored up to 1-2 years after the acute HDV-infection. The development of chronic HDV-infection is usually associated with the presence of high titer antibodies persist permanently. In addition, the observed so-called

transient expression replication markers HBV, which is due to hepatitis D virus inhibitory effect on replication of hepatitis B virus.

- If the patient is anti-HDV positive, the ongoing replication is evaluated by the presence of **serum HDV RNA**, since only a part of the seropositive patients are viremic and viremia indicates potential for liver damage. Reverse transcriptase polymerase chain reaction is the most frequently selected method for viral RNA detection due to its high sensitivity.

Hepatitis C

- **Anti-HCV**. An HCV antibody test is used to screen for infection. It detects the presence of antibodies to the virus, indicating exposure to HCV. HCV antibodies usually do not appear until several months after exposure but will always be present in the later stages of the disease.
- **HCV** genetic material **(RNA)** testing uses polymerase chain reaction to identify an active hepatitis C infection. The RNA can be found in a person's blood within 2 weeks after exposure to the virus. HCV RNA testing may be done to double-check a positive result on an HCV antibody test, measure the level of virus in the blood (called viral load), or show how well a person with HCV is responding to treatment.
- **HCV quantitative test** (also called viral load) is often used before and during treatment to find out how long treatment needs to be given and to check how well treatment is working.
- **HCV viral genotyping** is used to find out which genotype of the HCV virus is present. HCV has six genotypes, and some are easier to treat than others..

Hepatitis G

GBV-C RNA has been detected in patients with acute non-A to non-E viral hepatitis, in patients with chronic hepatitis of presumed viral etiology, in patients with cryptogenic cirrhosis, and in some patients with primary hepatocellular carcinoma. However, it is often difficult to tease out the direct role of GBV-C in these settings since coinfection with HCV is so common. Studies in patients with apparently isolated GBV-C infection suggest that the acute liver injury is similar to and may be less severe than that with HCV.

Hepatitis E

Hepatitis E infection is found worldwide. Two different patterns are observed, where hepatitis E is found in: resource-poor areas with frequent water contamination; and areas with safe drinking water supplies. The disease is common in resource-limited countries with limited access to essential water, sanitation, hygiene and health services. Sporadic cases are also believed to be related to contamination of water or food, albeit at a smaller scale. The hepatitis E virus is transmitted mainly through the faecal-oral route due to faecal contamination of drinking water. This route accounts for a very large proportion of clinical cases with this disease. The risk factors for hepatitis E are related to poor sanitation, allowing virus excreted in the faeces of infected people to reach drinking water supplies.

In rare cases, acute hepatitis E can be severe, and results in fulminant hepatitis (acute liver failure); these patients are at risk of death. Fulminant hepatitis occurs more frequently when hepatitis E occurs during pregnancy. Pregnant women with hepatitis E, particularly those in the second or third trimester, are at an increased risk of acute liver failure, fetal loss and mortality. Case fatality rates as high as 20–25% have been reported among pregnant women in their third trimester.

- **Antibodies HEV IgM** appear lately - on 12-13 day after infection, are stored up to 2-3 months.
- **Antibodies HEV Ig G** indicate immunity, which is not life. In some cases, possible reinfection.

ELISA tests for diagnostics parasite infections

Using ELISA for the diagnostics of various parasitic diseases

Helmintoze:

Nematodoze

Ascariasis - for diagnosis of parasitic infestations commonly used ELISA method, the method has high specificity in the presence of roundworm.

Trichinosis - ELISA must be repeated several times to show the process dynamics. The maximum level of antibodies detected 4 - 12 week disease.

In enterobiosis, Trichuriasis ELISA method is not informative.

Ancylostomiasis. The IgM-ELISA was neither specific nor sensitive.

Cestodoze

Teniasis and cysticercosis - the only representatives of this kind, for which IFA has diagnostic value.

When echinococcosis and alveococcosis ELISA is used rarely, since even during the expanded clinical picture of past results may falsely positive.

In diphyllobothriasis, hymenolepiasis, beef tapeworm infection serological method ELISA is not specific.

Trematodoze

Fasciolosis - already in the acute fase informative serological survey methods, which are leading with ELISA.

Opisthorchiasis - serological methods, including ELISA ranked first, while informative chronic process. The method allows for the differential diagnosis between acute and chronic opisthorchiasis.

Protozoan Diseases

Giardiasis - the most sensitive ELISA method deployed during clinical manifestations. At this time, are in more immunoglobulin M, they quickly disappear and within weeks of release as the body of Giardia there are immunoglobulin G, which may still remain 12 - 15 months.

Malaria - a serological ELISA widespread use in clinical practice has not found despite the complexity and duration of the study. The antibodies appear in the blood after 2 - 3 attacks, peaking at 4 - 6 weeks, after decreasing, determined to 6 - 24 months. The inhabitants of endemic malaria regions are antibodies in the blood continuously.

Cutaneous **leishmaniasis** and visceral - ELISA method is quite informative and widely used in the diagnosis of these diseases.

Amebiasis - ELISA method acquires some value in the presence of amebic abscess, especially in cases where no intestinal manifestations and parasitological methods are uninformative.

Toxoplasmosis - serological methods in including ELISA are the main methods the simplest definition of a human body. In the acute phase defined in more immunoglobulin M, chronic - immunoglobulin G.

ELISA for the diagnosis of Helicobacter pylori in feces

Stool antigen detection. This method detects H pylori antigen in stool specimens and can be used for diagnosis, therapeutic monitoring, and proof of eradication post treatment. Determination of Helicobacter in stool by ELISA performed in acute diseases**.** A sample of stool after defecation self-collected in a single, tightly closed container overnight and sent to the laboratory for enzyme-linked immunosorbent study to determine specific antigens.

ELISA for evaluating the condition pancreatic elastase 1 in stool (E1)

Pancreatic elastase 1 - proteolytic enzyme digestion, exclusively produced by the pancreas. Elastase cleaves internal communications protein (neutral amino acid). It is present in human pancreatic juices and feces. The enzyme is not affected during the passage through the intestinal tract. At exocrine pancreatic insufficiency level of pancreatic elastase 1 in feces reduce. Reduced activity in E1 feces also found in patients with chronic pancreatitis, pancreatic cancer, children with cystic fibrosis. In cystic fibrosis encouraged to detect the level of elastase 1 in stool at least once every 6 months. Content E1 in stool is not changed by a number of other diseases, celiac disease, inflammatory bowel disease, infectious diarrhea. High stability pancreatic elastase 1 allows not to limit the delivery of diagnostic analysis in the laboratory. The material for the determination of E1 can be kept for a week in the refrigerator. One stool sample is sufficient for diagnosis (no need to collect feces daily). Definition E1 is used to evaluate exocrine pancreatic function. Unlike chymotrypsin fecal E1 determination results are not dependent on patients taking pancreatic enzymes.

4. INTERPRETATION OF RADIATION (X-RAY AND ULTRASOUND) STUDIES OF THE GASTROINTESTINAL TRACT AND ABDOMINAL ORGANS

X-ray study enables determine the form, size, position, stomach mobility, localization of tumors, ulcers, relief gastric mucosa, the presence or absence of folds, symptom of "niche" at peptic ulcer, "filling defect" in tumors. An hour later, the stomach is 1/3 or less barium. Important X-ray study is artificial contrast, especially used for examinations of the esophagus, stomach, intestines. For this purpose fasting patient takes barium sulphate aqueous suspension, insoluble in water, which slows down x-rays. In polypozition X-ray (roentgenoscopy, series sighting images) study the phases of swallowing, functional and morphological changes of the esophagus, stomach, duodenum. For 1.5-3 hours stomach completely liberated from 200 ml suspension of barium sulfate. Within 3-4 hours after its adoption loop filled with fine, then - colon. After 6-9 hours conrast substance enters the caecum, ascending, and for 18-24 hours fills the entire colon. The method used to diagnose the diseases of the esophagus, stomach, small intestine and determining the functional disorders of colon. To identify morphological changes of the last hold irrigoscopy or retrograde filling of barium sulfate suspension for using the Bobrov`s apparatus.

The second principle of the digestive tract study is phased character. The study of each department should be conducted in "tight" filling the contrast agent to determine the position, shape, size, contours, mobility and function of the organ. Relief mucosa studied at small filling. The sequence of these two phases varies for different departments. At researching stomach examine the relief of gastric mucosa at first. Then create a "tight" content, is at research of of the colon, these steps are reversed. Mandatory condition of success is research palpation and compression body using special tubes. All parts of digestive tract, except for esophageal and rectal study using measured compression with varying degrees of contrast filling body substance. Another step in the research may be study the thickness and elasticity of the walls of certain gastrointestinal channel with the introduction of X-raynegative

contrast agents, particularly the gas inside the body with a small filling barium sulfate suspension (double-contrast or pariyetography and triple staining).

The third principle is multipozition study of the digestive tract in which to determine the state of the walls of the body being studied, its relationship with the surrounding tissue, changing the patient's position. Patient is returned to the vertical and horizontal position, lying on his back and stomach in Trendelenburg position with a raised to 10-15° pelvic and lying on its side. It is very important correctly prepare the contrast agent.

Indications for this test: 1) dysphagia; 2) complaints of heartburn, spitting up, loss of appetite, bloating, abdominal pain, nausea, vomiting; 3) enhanced intestinal noises; 4) evidence for the diagnosis of chronic gastritis, ulcers, tumors stomach, etc; 5) unwarranted weight loss; 6) the presence of seals abdominal palpation; 7) enlarged liver or spleen; 8) ascites; 9) anemia of unknown etiology; 10) presence the stool occult blood.

In recent years, science discusses the appropriateness and consistency of the use of radiological and endoscopic examinations of the digestive tract. Most scientists believe that it should begin with endoscopy, in which a radiologist for a specified area of increased attention (changes in mucosal ulceration small size, exophytic lesions). Radiographic studies help assess the state neighboring authorities to clarify the scope and nature of complications. This sequence of procedures significantly reduces radiation exposure to the patient.

Radioisotope researching of the liver. Hepatobiliary scintigraphy (intravenous administration of Tc-99m) enables evaluate the function of parenchymal and reticuloendothelial cells of liver. Hepatobiliary scintigraphy showed the excretory function of the liver quantitatively and was helpful in detecting hepatic excretory dysfunction early in chronic liver disease.

Computer tomography allows to estimate the external contours of the liver, gets detailed picture of its internal structure. Normally images liver celebrated mostly clear, smooth contours, homogeneous structure. Parenchymal density corresponds to 50-79 units. Possible variations within 5- 10 units. For the diagnosis of digestive tract tumors are increasingly using ultrasound imaging. However, the information obtained

should be compared with the findings of clinical, endoscopic and radiological examinations.

Ultrasound (Sonography)

Ultrasonography is currently one of the essential components of the clinical examination of the patient.

Ultrasound examination of the stomach. Carry on the device, which operates in "real time." Explore an empty stomach in the morning. First, find a pylorus (conducting longitudinal glow right epigastric midline). Over or under the lower edge of the liver it can be seen in cross-section (diameter - 2.0-2.5 cm wall thickness - 0.5 cm). By moving echozond left, you can see the body and antral part of stomach in the form of an oval or a triangle. In 70-80% of visible folds at intervals of 0.5 cm. During the study, you can change the patient's position (horizontal, vertical, on his side, sitting). Determine the projection stomach to the anterior abdominal wall. Then identify where the greatest pain under control image. To monitor the stomach tone and peristalsis give the patient drink 200-300 ml of warm water. Evacuation capacity assessed by rhythmic contractions of the pylorus and change the volume of fluid-filled stomach.

Ultrasonography enables identify pathological processes in the liver, to trace them in dynamics. In many cases, the ultrasound can not establish the nature of jaundice, identify signs of portal hypertension, etc. In unclear cases resort to fine needle biopsy sighting controlled by sonography or CT scan. Fine needle biopsies under ultrasound guidance is carried out in order to obtain material for morphological studies; medicinal purposes - for biliary decompression with hypertension, irrigation and external drainage of pus and other cavities, injection of medications (antibiotics, chemotherapeutic agents, sclerosing solutions, etc.). Puncture conducted sighting, significantly reduces the risk of complications encountered in the blind manipulations, increases diagnostic accuracy and expands the range of possible diagnostic and therapeutic measures.

Ultrasound scan of the liver. Ultrasound scan the liver can estimate the position, size, form, shape and structure of the liver, its respiratory mobility, condition of the vascular system and intrahepatic bile duct, liver relationship with

surrounding organs and tissues. The liver has an irregular dome form with convex upper surface and facing down and back bottom surface of the depression of internal organs - the gallbladder, right kidney, aorta, inferior vena cava, the hepatic flexure of the colon. The upper surface of the liver is confronted with the diaphragm. The angle formed surfaces liver on mid-clavicular line not exceeding 70 °, the median - 45 °. The right and left lobe of the liver and separated coronary round ties. The latest to cross tomograms sometimes mistaken for focal lesions of the liver. Suspicion of focal lesions removed in obtaining long-axis images. Square and caudate lobe of the liver are more visible on longitudinal (relative to the axis of the body) scans. Measuring liver spend on standard lines. It is necessary to take into account the human constitution: the sagittal hyperstenics increased size and reduced verhnenizhniy in asthenics - on the contrary. The contours of the liver, limited by glisson capsule (fibrous membrane of the liver) are clear, smooth. Any changes of the contour, protrusion, retraction, discontinuity, etc. trace at ultrasound, spending intersection in perpendicular planes. Particular attention is paid to the structure of tissues in the uneven path. On vessels of the liver is most clearly manifested the portal vein and hepatic veins. The branches of the portal vein most noticeable at oblique section below the right costal arch with a slope echozond down (feet patient) at an angle of 45-90 °. The direction right and left branches of the portal vein is longitudinal and parallel section plane; structure vascular network mainly branched; notably the gradual narrowing of blood vessels to the liver surface to which they normally do not reach; the largest diameter at the gate of the liver - 14 mm. The walls of the portal vein branches give a large number of reflected signals (echogenicity). Hepatic veins as seen at an oblique section below the right costal arch, but echozond location closer to the line and tilt it down at an angle of 0-45 °. Hepatic veins have oblique direction relative to the plane of the section, vessels mostly straight, extending toward the aperture, which have the largest diameter (4.5 mm); they fall into the inferior vena cava. The walls of the hepatic veins at this intersection almost did not make the reflected signals (like embedded in the structure of the liver), hepatic artery and its branch are not always visible within a small gate in the liver. They run parallel to the portal vein as a tubular structure with a diameter of 1-2 mm. Intrahepatic bile ducts

with no pathology are not seen. The exception is area of fusion of left and right hepatic duct diameter of 3 mm, which is located above the branch portal vein into right and left branches. Echostructure liver tissue as a whole seems netted and uniform. By increasing or decreasing the echogenicity (degree mappings) and violation of the homogeneity of the structure and contours liver conduct review sighting of suspicious areas, changing signal intensity and brightness. Topography location of structures is carried out according to the segmental structure of liver.

Liver cysts - a round or other form formation that does not allow of reflected signals (echonegative), followed by strengthening the image of underlying tissues (distal strengthening effect) with a lateral weakening. Their contours are equal, clear; with thin wall (posterior wall is visible clearly than anterior). Typically, cysts are in unchanged tissue in the liver and near the surface. Larger cysts can deform the contour of the liver; the localization in the right lobe, near the gate of liver cyst can be mistaken for gallbladder. To avoid errors research are conducting after cholagogue use (this changes the gall bladder volume). Sometimes it is difficult to differentiate liver cyst from the cyst of right kidney; in such cases, conduct additional examination of the kidney. Liver cysts are single and multiple, often combined with kidney cysts. A large number of cysts, deforming and increase the organ indicates a polycystic liver. Inequalities of internal or external contour of cysts, various overlays on the walls of partitions, small daughter cysts, calcinates in the cavity or calcination of capsules may indicate liver echinococcosis. However, even when detecting typical benign liver cysts researcher can not always determine its nature. Diagnosis is specified using serological tests.

Echinococcos node has the form a small, irregularly shaped hypo-echogenic formation, which is then converted to cyst formation.

Hemangiomas – are the most often benign liver tumors. Typically, they are located near the vascular structures of the liver, sometimes possible to trace their connection.

There are two types of hemangiomas: the so-called capillary hemangiomas, localized predominantly in the right lobe of the liver in the VI-VII segment. They are small (13 to 40 mm), have clear, equal, sometimes wavy contours, uniform

hyperechogenic structure. Lacunar hemanhyomas localize in the right lobe more often. Such hemangiomas typically are large (sometimes gigantic), taking the whole lobe of the liver. They are clear, but wavelike contours, uneven structure with areas that do not allow the reflected signals that corresponds cavities filled with blood. In this form, as well as multiple hemangiomas in some cases there is a need for differential diagnosis of metastasis and with primary liver cancer. Outside hemangiomas occasionally observed increased image of underlying tissues (distal strengthening effect). Color Doppler record low speed flows. Hemangiomas are single and multiple. At the last possible triad of symptoms: hepatomegaly, skin lesions and heart failure.

Liver adenomas - benign tumors originating from liver cells or bile duct epithelium. They are oval formation, less round shape with equal contours clear. They have uniform hypoechogenic structure that follows the pattern of the surrounding liver tissue (sometimes containing uniform inclusion); enhance the image of tissue to be observed. They can change the course of surrounding blood vessels (gradual flow). If you suspect a liver adenoma needed morphological verification. Differential diagnosis is made primarily of hypoechoic metastases.

Liver amyloidosis is usually characterized by diffuse changes. The differential diagnosis is conducted with liver cirrhosis. In several cases with the uneven deposition of amyloid areas of reflections of low echogenic various shapes (from round to irregular star) with unclear contours are in the background of strengthening diffuse echogenic. Liver amyloidosis is usually combined with amyloidosis of other organs, renal amyloidosis. For the latter absence of differentiation of parenchyma and pyelocaliceal system is characteristic.

Liver hematoma - rounded or other form formation with slightly pronounced effect of distal strengthening without lateral weakening. The contours are unclear, content is nonhomogenic. Severe tenderness under control sonography, and at the location close to the surface of the liver - limited mobility of the diaphragm. In history - often trauma.

Organized hematoma looks like a cyst with nonhomogenic content in the form of small flaky inclusions. They shift when patient change position. Ther is mild

strengthening of underlying tissues. Especially difficult differente the organize hematoma and cysts if hemorrhage is in the cyst cavity.

Liver abscess. During abscess formation marked change in ultrasound picture. Initial signs are abscess area of low echogenicity without clear boundaries, often localized in the right lobe of the liver. In the development of it heteroehcogenic zone appears in the center or around the formation due to melting of tissues; but hypoehcogenic area around the formation is usually expressed marked due to strengthening the image of underlying tissues (distal strengthening effect). Differential diagnosis is made with a cyst of the liver. At organization of abscess capsule and pus separation appears. When identifying dense content and unclear borders of abscess differential diagnosis of the tumor is required. When spontaneous resolution of abscess is observed ther is calcification of the capsule, followed by ultrasound shadow.

Puncture controlled ultrasound performed for external drainage, antibiotic therapy and collection of material for morphological studies, sowing. From anamnesis pay attention to the presence of purulent process in the abdominal cavity and surgical interventions.

Subhepatic abscess is usually located under the lower surface of the liver, anterior to the right kidney.

Suprahepatic abscess is frequently on the right, adjoining is directly to the diaphragm (as in liver abscess); usually pleural effusion is in the right sinus. The mobility of the liver and the diaphragm is limited.

Fatty (degeneration, steatosis) is characterized by moderate liver (rarely more pronounced) increase in liver uniform increased echogenicity depending on the degree of fatty infiltration caused by strong reflection of ultrasonic vibrations tiny inclusions of fat in the liver cells (scans as if covered with small grains). Vascular picture differentiated uncertain; structure under the diaphragm can not be seen, due to the pronounced absorption and reflection of ultrasonic energy (in modern devices, this feature is losing its significance as a result of a more powerful signal amplification depth). Sometimes the background of diffuse changes defined areas of intact liver tissue, preserving its structure (necessary to carry out differential

diagnosis of focal lesions of the liver). For fatty liver disease is unusual enlargement of the spleen; splenomegaly usually indicates the presence of concomitant inflammatory diffuse process in the liver as expanding the diameter of splenic and portal veins.

Chronic hepatitis is not always accompanied by any change ultrasonography of the liver, so put this diagnosis only according sonohramy difficult. When ultrasound can sometimes detect moderate hepatomegaly without deformation of contours, edges rounded with a moderate increase of liver angles. Deep-lying structures and diaphragm are viewed clearly. The structure of the liver is frequently homogeneous, echogenicity may be increased moderately and evenly. There is a moderate enlargement of the spleen. No signs of portal hypertension.

Liver cirrhosis. In the early stages of cirrhosis increased body mainly by the left lobe is marked, increase (bulging) tailed share. As the growth process and the appearance of dystrophy decreased liver size is observed. In the expanded stage of the disease line between right and left lobes is well defined as well as the fibrous membrane of the liver**.** The structure of the liver has heterogeneous mosaic picture, mixed echogenicity, sometimes the type of focal changes (due to the presence of necrosis, nodes, inflammatory reaction and the development of connective tissue, abnormal vascularization, that is in violation of the architectonics of the liver). Increased echogenicity of periportal fields can be traced almost to the periphery of the liver. Liver edges are rounded, angles are extended (more than 45° the median and 75° on media clavicular lines). The contours of liver are deformed: serrature is determined at micronodular cirrhosis and big bulging or retraction at macronodular cirrhosis; particularly clear terrain contours on the background of ascites. The spleen is enlarged, it increased echogenicity (spleen structure resembles the structure of the liver). Identify signs of portal hypertension. The diagnosis is more likely in identifying extensions splenic and portal veins, ascites and other signs of portal hypertension.

Alveococcosis. Ultrasound picture is characterized by diffuse echogenicity enhancement, often absence of clear borders between affected and intact tissue of the

liver, underlined reticulate liver structure. In history there are indications for surgery of alveococcosis.

Ultrasound signs of portal hypertension: 1) expanding of the splenic vein (at the gate of the spleen> 5 mm for the vein> 10 mm); tortuosity, detect veins inside spleen, enlargement of the spleen; 2) expansion of the portal vein> 14 mm; 3) enlargement of the superior mesenteric vein> 9 mm; 4) ascites; 5) varicose vein cardiac part of stomach (thickening of its walls); 6) spleno-renal anastomoses; 7) recanalization of the umbilical vein; 8) slowing of blood flow in the portal vein on Doppler results; 9) reducing the volume of blood flow in the portal vein and its branches on the results duplex angiography (color Doppler). The diagnosis is significant in the presence of at least three of the above symptoms.

The main features of the liver at congestive heart failure is expanding of the hepatic veins (>8.9 mm when measuring 1 cm below their confluence tothe vena cava inferior); throbbing veins; enlargement of the vena cava inferior (>15-16 mm); no change of vena cava diameter inferior to inhale and exhale. Enlargement of the liver, as well as the presence of other signs of the heart failure depends on its duration and stages. At the early stage of heart failure observe varying degrees of liver enlargement, equal, clear contours, rounded edges, increasing angles; it structure is clearly visible, echogenicity is normal or reduced; portal vein is not changed; can be detected ascites and hydrothorax (usually right sided). In severe heart failure and its prolonged duration due to the formation of cardiac cirrhosis border between lobes of the liver, severe liver fibrous membrane becomes noticeable. The liver edge is acute, diffusely increased echogenicity of structure, periportal fields. The liver is deformed, it becomes jagged contour. There are signs of portal hypertension, splenomegaly, ascites, and hydrothorax. In the final stages of disease liver size decreases.

Cholangitis. Chronic cholangitis. For this process characterized by uneven expansion of the bile ducts that appears unstable, uneven hyperechogenic walls.

Acute cholangitis is characterized by enlargement of the liver, local tenderness to palpation liver under ultrasound control, limiting the mobility of the diaphragm to the right, sometimes enlarged spleen.

Bile ducts are usually unevenly dilated; their contours are not clearly and merge with the surrounding tissue. Can be seen small abscesses, combined with bile ducts. When unfavorable course possible to form larger abscesses that clearly stand out against liver parenchyma. For external drainage and antibiotic cyst puncture spend under control sonography.

Anaerobic cholangitis accompanied by the appearance of small gas bubbles, followed by ultrasonic shade (like ultrasound data observed in the presence of stones in the bile ducts, but without clinic cholangitis). They may be complicated by thrombosis of the portal vein.

Malignant tumors of the liver is divided into primary and metastatic. In clinical practice often are metastases of malignant tumors, which are focus of various sizes and shapes, often with unequal and unclear contours, varying degrees of echogenicity; from hyper - to hypo- and anechogenic formations.

Formations which give more than the normal liver tissue, the amount of reflected signals (hyperechogenic varying degrees of severity and mixed structure) is the most visible with ultrasound and are found more often. The differential diagnosis is carried out primarily with hemangiomas of the liver. If metastatic lesions suggests the presence of hypoechogenic rim around the formation, which can be caused by a layer of actively multiplying tumor cells that have the same type of structure as well as compression and liver tissue pathological vascularization around the formation.

The most difficulty is the detection of metastases did not differ in structure from the surrounding tissue of the liver. This so-called izoehohennye metastases. The main diagnostic reference of these cases is round identifying such areas hypoechoginic rim or displacement of the vessels. In the absence of these features identify such metastasis is virtually impossible, these metastases occur in 1-3% of cases.

Mixed structure metastases with irregular distribution of reflected signals often caused by uneven growth of the tumor, the presence of foci of hemorrhage, necrosis, purulent fusion, inflammatory response and the development of abnormal blood vessels and connective tissue, and so on. D. Among them should be allocated so-called bullish eyes (in severe hyperechogenic periphery - hypoechogenic center) is usually caused by a massive collapse of tissue in the center of the formation.

Anechogenic metastases are rare; in appearance they resemble cysts of the liver. For metastases may occur enhance the image underlying tissue (less pronounced than in the kitty, volumes). Some of them are caused by tumors that produce mucin, as part consists of fairly dense tissue (eg metastatic soft tissue sarcoma). In analyzing the images must pay attention to the unevenness of internal and external peripheral circuits of the formation. The differential diagnosis is made with abscesses and liver.

Hipoehochenic metastases have a structure that allows a limited number of reflected signals. They sometimes resemble cysts, but for them has no strengthening the image of underlying tissures. Such a view may have metastatic seminoma, lymphoma, sarcoma, melanoma, and other young metastases that have not undergone further development: over time you can watch their transformation into hyperechogenic or mixed form. Detection of compression, displacement, breakage of blood vessels and bile ducts expansion helps diagnose metastases, but these signs are not always reliable. Enlargement of the liver and changing its contours are visible only when a significant location and spread of metastases in the periphery of the body under the fibrous sheath liver. Single metastasis can not be distinguished from primary liver cancer.

Primary malignant liver tumors. Primary liver cancer out of the liver cells (hepatocellular carcinoma) or bile duct (holangiogenic cancer). Diagnosis of liver cancer is difficult, especially in cases where the patient has changes characteristic of liver cirrhosis, against which the tumor develops. Primary liver cancer can be conditionally divided into two forms, nodular and diffuse infiltrative. Revealing the last form represents the greatest difficulties. At ultrasound is necessary to pay attention to areas of parenchymal unusual structure, with fuzzy boundaries, heterogeneous structures, mixed echogenicity. These areas are often not seen or deformed vascular network. In the liver vessels clots can be detected. The decay in the center of the tumor appears hypoechogenic cavity. Enlargement of the liver and changing its contours are marked with large tumor size or its location close to the surface of the liver.

Detection of enlarged lymph nodes (retroperitoneal or at the gate of the liver) confirms the assumption malignant nature of the disease, but the rather late stage, as well as the shift of the gallbladder, limited mobility hollow organs (stomach, colon), the fluid in the right pleural sinus and others.

Nodal form of primary liver cancer is revealed worse than metastases, as often occurs against the background of existing diffuse liver changes. Like metastasis, tumor can have a different look and hypoechogenic rim around it. Mixed, hyperechogenic and isoechogenic forms are dominated. Hypoechoic form can occur in the early stages of primary liver tumors. If multiple foci in the liver one of them is large display provides the most intense signals can be assumed liver cancer with liver metastases. For holangiogenic cancer tumor presence directly in the bile duct and bile duct expansion above the location of the tumor is characteristic. With this type of tumors at earlier stages appear jaundice and markedly expanded bile ducts.

Nodular form primarily differentiated from metastases that are not always possible, as well as other liver tumors (hemangioma, cysts, etc.). With benign tumors most difficult differential diagnosis of teratoma, which may have several varieties. Typically, teratomas have irregular heterogeneous structure due to the presence of germs of skin, hair and secretions of sweat and sebaceous glands, bones, teeth and so on. As a result, teratomas may be represented by three components: superdense, medium and liquid. For dense inclusions (bones, teeth) should ultrasonic shadow. In some cases there is malignant teratoma.

Diagnostics of obstructive jaundice

The main diagnostic feature to distinguish the obstructive jaundice from other types of jaundice is the expansion of the bile ducts. On sonograms intrahepatic bile ducts are visualized. The site of confluence right and left hepatic duct is more 3 mm, diameter of the common hepatic duct - > 4 mm, the common bile duct - > 5 mm, enlarged gall bladder (length - > 11 cm, width > 3.5-4 cm).

Through US examination can determine the level of obturation biliary system. Extension of intrahepatic duct in one lobe indicates the block of lobular bile duct; diffuse expansion of intrahepatic duct without enlarged gallbladder – obstruction of common hepatic duct, expansion of biliary tract and increased gallbladder shows the

obstruction in the major duodenal papilla (Vater papilla). Ultrasonography in some cases to set the cause of obstruction (gallstone disease, metastases in the liver gate, pancreas cancer, gallbladder cancer, liver cancer, cancer and stenosis major duodenal papilla, liver echinococcosis, alveococcosis liver, liver abscess, cyst in the liver gate, exacerbation of chronic pancreatitis, tumors of adjacent organs, etc.).

Gallbladder ultrasonography allows studying its content, form, volume, thickness and structure, the presence of additional inclusions in its cavity and contractile ability. Longitudinal section of the gallbladder is pear-shaped. Normally its length - 10.8 cm, width - 3 cm. The contours of the gallbladder clear. The thickness of wall is less than 3 mm. Cavity is homogeneous. After 40 minutes of bile breakfast volume of gallbladder is reduced by 30-60%.

5. ESTIMATION OF ENDOSCOPIC PROCEDURES OF THE DIGESTIVE TRACT

Fibroesophagogastroduodenoscopy (FEGDS) - a review of the mucosa of the esophagus, stomach and duodenum using an endoscope, which is administered to the patient from anesthesia throat. It allows you to determine the color of the mucous, vascular status, presence of erosions, ulcers, hemorrhage, tumor, to a piece of mucous for morphological study (biopsy).

To assess the state of the mucous small bowel mucosa morphological study carried postbulbar duodenal part obtained during the execution FEGDS.

All the organs of the gastrointestinal tract are interconnected and any pathological processes in one of them necessarily affect the state of the gastrointestinal tract. So often you need to study all these organs - esophagus, stomach and intestines.

Endoscopy of the esophagus called esophagoscopy and conducted by introducing gastroscope through the mouth. It rarely performed independently, often studied all upper gastrointestinal tract - esophagus, stomach and duodenum. FEGDS can be performed for diagnostic and therapeutic purposes. Diagnostic endoscopy of the esophagus is made in inflammatory diseases of the esophagus caused by burns, diseases of the stomach from throwing the contents into the esophagus, suspected tumor of the esophagus. With endoscopy can be good to examine all the walls of the esophagus and detect the initial stages of any process that can not be seen, such as X-ray. Endoscopy is also used to control the spread of the pathological process in the esophagus, including during treatment.

Therapeutic endoscopy of the esophagus often performed using rigid endoscope due to the fact that the required narrow esophagus endoscopic instruments. Planned medical esophagoscopy performed to remove polips esophageal sclerotherapy (injection of medicines that cause desolation of esophageal varices, that is a warning possible bleeding, enlargement of the lumen of the esophagus during its contraction and so on. Sometimes endoscopy of the esophagus spends on an emergency basis, mainly required if it enters the esophagus foreign body or bleeding from the blood vessels of the esophagus.

Endoscopy stomach - gastroscopy is one of the main methods.

Studies performed using fibrogastroscopy (flexible gastroscopy). It consists of the heads of the appliance where the eyepiece levers distal end of the appliance, buttons feeding the stomach and absorption the air, liquid; entrances to the channels with valves through which the stomach can enter various instruments (biopsy forceps, catheters, miniature scissors, and polypectomy loop for removal of foreign bodies, electrodes for coagulation, etc.); working part gastroscope, which is injected into the stomach (a flexible tube with a diameter of 8-12 mm, length 860-1200 mm) and the connecting fiber.

Indications for FEGDS are: 1) the need to establish or clarify the diagnosis of any primary disease of the stomach (gastritis, peptic ulcer disease, tumors, etc.); 2) determine the nature of the changes in the stomach due to changes in diseases of adjacent organs (liver, gall bladder, pancreas); 3) detection foreign objects and so on.

Contraindications for FEGDS are: 1) esophageal disease (scarring and narrowing of the tumor, diverticulitis) and surrounding organs (aortic aneurysm, a tumor of the esophagus, large curvature of the spine); 2) expressed cardiovascular and pulmonary insufficiency; 3) veins of the esophagus. The general grave condition of the patient, if gastroscopy performed for health reasons, is not a contraindication to its implementation.

Planned gastroscopy spends the morning on an empty stomach, emergency - at any time. For 15-20 minutes before the test is administered subcutaneously 0.5-1 ml of 0.1% solution of atropine. Oral cavity is anesthetized by lidocain solution. After examination of the patient for 1-2.5 hours should not drink, eat, smoke, and if biopsy was performed, you can not use this day a hot meal.

Normally folds of gastric mucosa are more pronounced at lesser and larger curvature. Mucosa has from pale pink to red color. When air injection in the stomach wrinkles smoothed. Pylorus takes the form of the socket and at considerable fanning the stomach reaches 1.5 cm in diameter.

Complications - perforation of the esophagus and stomach bleeding after the biopsy, disorders of the cardiovascular and respiratory systems and more.

Compliance with the rules of gastroscopy with regard to indications and contraindications and proper preparation of patients allows for this survey is quite safe and prevent complications.

Gastroscopy can detect inflammation in the stomach, erosive and ulcerative processes (gastric ulcer), polyps, benign and malignant tumors, as well as a means of monitoring the effectiveness of treatment. Therapeutic gastroscopy conducted to injection of medications, removing polyps and small benign tumors as well as to expand the holes between the stomach and duodenum.

Endoscopy of the duodenum usually performed with endoscopy of the esophagus and stomach to detect the process of ulcer in the duodenum, and congenital disorders of digestion process that hinder the process of absorption of food. Disturbance of absorption of food is often associated with congenital lack of synthesis of certain digestive enzymes. Food is digested with the wrong wall and annoying duodenum, causing it to poor circulation and metabolism. All this is clearly visible during duodenoskopy.

Rectoscopy - a review of the mucous rectum and sigmoid intestines using rectoscope (35 cm); normal mucosa is smooth, moist and moderately red. Acute inflammation is swelling, hyperemic, muddy, covered with slime. You can detect bleeding, erosions, ulcers, hemorrhoids, anal fissures, tumors, biopsy done.

Colonoscopy allows you to see the colon mucosa almost throughout, to biopsy and photography. Colonoscopy performed in difficult diagnostic cases after irrigoskopy (contrast medium filling the large intestine using an enema followed by X-ray). Colonoscopy requires careful preparation of the patient by means of cleansing enemas and laxatives. It provides valuable information, especially with suspected tumor, bleeding.

Endoscopy of the colon using a colonoscope is carried out, which is introduced through rectum. We investigate the whole colon, are virtually all diseases of the body in the initial stages. This is very important when detecting polyps of the intestine, which are considered precancerous diseases and immediate removal and the detection of early signs of bowel cancer. Widely used and therapeutic colonoscopy - local treatment of various diseases, removal of polyps and benign tumors.

From the study of the small intestine it is much more difficult. Today is the ability to study using special videocapsule endoscopy. Unfortunately, the widespread this technique has not yet, so the problem of detecting tumors of the small intestine in the early stages and is still relevant.

Endoscopic retrograde cholangiopancreatography (ERCPG). The method is important in the diagnosis of diseases of pancreatic and biliary zone. ERCPG is filling the bile and pancreatic ducts with X-ray contrast substance. ERCP is used as a diagnostic and therapeutic purpose. In addition, it allows you to explore the state of the esophagus, stomach, duodenum, large duodenal papilla, pancreatic duct. There is a possibility of biopsy in the studied organs, eliminate stenosis at sphincters, remove the stones from ducts.

Indications: a suspicion of diseases of the pancreas, jaundice of unknown etiology, pain in the upper abdomen, especially arising after operations on the biliary tract, suspected choledocholithiasis and stenosis of the bile and pancreatic ducts. Complications - acute pancreatitis, sepsis, anaphylactic reactions to contrast.

6. INTERPRETATION OF MICROBIOLOGICAL AND BIOCHEMICAL ANALYSES OF BILE

Research bile. To assess the functional status of biliary tract used method duodenal fractional tubage (general clinical research duodenal contents) which can diagnose pathology in different parts of the biliary tract. Laboratory studies bile helps to clarify the nature of the disease process. In bahatomomentnomu fractional sensing bile collected in separate tubes every 5 or 10 minutes, a record outflow of each portion of bile, it volume. The results reflect the charts. For portions of bile from the gall bladder (portion B) as a stimulant commonly used 33% solution of magnesium sulfate (50 ml). Magnesium sulfate as cholecystokinin cause contraction of the gallbladder.

Phases bile: I phase - bile A - the contents of the duodenum before introduction of the stimulus. Within 20-40 minutes normally allocated 15-45 ml of bile. Reducing the amount of bile that evolved for this phase indicates its hiposecretion which is often observed with cholecystitis. Hypersecretion is possible after cholecystectomy, in the phase of incomplete remission aggravation cholecystitis, the non-functioning gall bladder, hemolytic jaundice. Bold lighter bile observed in lesions of liver parenchyma, violation of the common bile duct patency. Irregular secretion shows to the Oddi`s sphincter hypertension (duodenitis, cholangitis, gallstone disease, cancer). Portion A may be non-existent in patients with acute hepatitis.

**Phase II (Oddi`s sphincter close) -** since the introduction of a stimulus to the appearance of bile A - 3-6 minutes. Reducing II phase can be caused by Oddi`s sphincter hypotonia or increased pressure in the common bile duct. Prolongation of II phase may be associated with Oddi`s sphincter hypertrophy, papillary stenosis. The slower passage through the cystic duct, particularly in gallstone disease, also leads to prolongation of this phase.

Phase III - bile A1 - the content of the common bile duct; for 3-4 minutes secretes 3-5 ml of bile. Prolongation of the third phase to 5 minutes may occur in the atonic gallbladder or it block caused by spastic or organic origin (gallstones). Volume

of bile (fraction A1) decrease at severe liver failure and increase at dilatation of the common bile duct.

Phase IV - bile B - the contents of the gallbladder. Within 20-30 minutes secreted 20-50 ml of bile. Acceleration in the secretion of bile indicates hyperkinetic dyskinesia while maintaining normal volume of gallbladder. Continuous or intermittent secretion of bile of large volume is observed at hypomotoric gallbladder dyskinesia. Reducing the volume of bile indicates a decreased volume of the gallbladder, in particular at its sclerotic changes and gallstone diseases.

No B fraction at: cystic duct occlusion by stone or tumor; violation of the contractility of the gall bladder due to inflammatory changes; loss of ability to concentrate gallbladder bile due to inflammatory changes; absence of so-called cystic reflex, that is emptying the gall bladder in response to the conventional stimulants. There is 5% of healthy people, but can be caused by even dyskinesia biliary tract.

Phase V - "liver" - bile portion C; flows continuously until the probe is. Slower outflow point in lesions of liver parenchyma. The complete absence of all portions of bile in probing the oil in the correct position of the probe in the duodenum may be the result of: compression of common bile duct stone or tumor; termination of biliary function in severe lesions of the liver parenchyma.

Physical and chemical properties of bile. The color of bile is normal: A portion - golden yellow, amber; portions - saturated yellow, dark olive brown; piece C - bright yellow. Change the color portions A: dark yellow - when throwing portion in bile and hemolytic jaundice; bright yellow - with liver parenchyma lesions, viral hepatitis, cirrhosis, occlusion the Oddi`s sphincter with stone, compression by increased pancreatic head, spasm of the sphincter; color of blood - at duodenal ulcer, major duodenal papilla tumors, hemorrhagic diathesis; greenish color (clear bile) - with its stagnation or infection.

Change the color in portions, poor color (white bile) - chronic inflammation with mucosa atrophy of gallbladder; very dark color - pathological thickening of bile in the bladder (stagnation) and hemolytic states.

Change the color portions C: pale color - in viral hepatitis, cirrhosis of the liver; dark color (pleohromia) - hemolytic jaundice; green color - with cholangitis; red - at duodenal ulcer, malignant tumors of the pancreas or pyloric ulcer.

Normally, all portions of bile transparent. Even small opacities that appears quite often due to impurities hydrochloric acid and does not indicate any other changes. A portion of turbidity is possible with high acidity of gastric juice, pylorus or failure in conditions of presence of duodenal reflux. Flakes out at duodenitis.

Turbidity portion is observed in inflammatory processes in the gallbladder. Flakes of mucus falls portions C in inflammatory processes intrahepatic moves cholecystocholangitis. A portion normally a neutral or basic reaction. Portions B and C - major. A portion of the acidic reaction happens when the inflammatory process in the duodenum. Acid reaction portion in characterizing bladder inflammation, and other portions - the relevant parts of biliary tract.

Normally, the relative density portions A - 1,003-1,016 g/l; B - 1,016-1,032 g/l; C - 1,007-1,011 g/l. Relative density increases at portions A throwing portions B hemolytic jaundice decreases with abnormal liver function, its parenchyma lesions (viral hepatitis, cirrhosis), violation of the flow of bile into the duodenum. Relative density portions in of condensation increases in bile (stasis), cholelithiasis, with biliary dyskinesia; reduced - at lower concentration ability of the gallbladder. Relative density increases at portions C hemolytic jaundice and decreases with decreasing secretion of bilirubin (hepatitis, cirrhosis).

In a healthy person the content of bile acids in the A portion of 17,4-52,0 mmol/l, in portions B - 57,2-184,6 mmol/l, in portions C - 13,0-57,2 mmol/l. The increase in the portion of C occurs in increased secretion of cholic acid by liver cells, decrease - at secretory deficiency liver cells. In a healthy person cholesterol in bile portions A - 1,3-2,8 mmol/l, in portions B- 5,2-15,6 mmol/l, in portions C - 1,1-3,1 mmol/l . The increase in the A and B portions marked with cholelithiasis, cholecystitis, and decrease - in violation of the concentration ability of the gallbladder. The content of bilirubin in the bile normally given in the table.

Table 6.1 The content of bilirubin in the bile of various portions of the norm

Bile portion	**Van den Berg method, g/l**	**Yendrashek`s method, mmol/l**
A	До 0,25	0,17–0,34
B	>2–4	6–8
C	>0,25	0,17–0,34

The content of bilirubin in the bile decreases with jaundice, Botkin's disease, cirrhosis, and calculous cholecystitis increases with hemolytic jaundice, B-12-deficiency anemia, malaria.

Microscopic examination of the bile. Microscopy is performed under low and high magnification after receiving bile. Bile acids can quickly destroy the leukocytes. In white blood cells in the bile can identify other cellular elements (epithelium), crystalline formations (cholesterol, bilirubin, "gall sand" microliths, crystals of fatty acids), parasites (Giardia, eggs, liver, roundworm). Mucus in the form of small flakes evidence of biliary tract catarrh, there is also at duodenitis. Red blood cells have no diagnostic value, because often there due to injury in probing. Diagnostic value are white blood cells that are found in small flakes of mucus in conjunction with the epithelium of bile ducts or gallbladder. The presence of white blood cells only portions A characteristic of duodenitis and inflammation in the large bile ducts. Detection of leukocytes mainly in portions at smaller portions of their presence in the A and C indicates the localization process in the gallbladder. The advantage of leukocytes in portions C observed in cholangitis. A large number of white blood cells in the bile fractions observed in frail elderly patients with septic cholangitis or liver abscess. Eosinophilic leukocytes detected in allergic cholecystitis, cholangitis and worm infestation. High prismatic villous epithelium characteristic in samples for cholecystitis; small prismatic cells or liver moves high prismatic epithelium of the common bile duct - for cholangitis. Large cylindrical cells from the cuticle and fibers indicate pathology in the duodenum. The cells of malignant tumors found in the contents of the duodenum with tumors. Crystals of cholesterol present in large

numbers by changing the colloidal stability of bile (cholelithiasis). They tend to accumulate with other crystalline elements of bile - microlite, calcium salts (calcium bilirubinat), fat and bile acids. Normally, all the crystalline elements are missing. Their presence indicates colloidal disruption of normal properties of bile, ie the pathological process of cholelithiasis.

Bacteriological study of bile has a relative value, as set highlighted flora origin difficult. Normal sterile bile. However, identifying microorganisms in repeated studies bile, we can conclude that they are isolated from the biliary tract. When parasitic diseases in the gall exhibit vegetative forms lamblia, helminth eggs (opisthorchiasis, fasciolosis, clonorchosis, strongyloidiasis, human trichostrongyloidosis). Detection of Fasciola hepatica and Strongyloides stercoralis in bile is very difficult. For fasciolosis and strongyloidiasis diagnostics multiple studies are needed.

7. INTERPRETATION OF GASTRIC FUNCTION TESTS (PH-METRY)

Types of intragastric pH-metry

There are main types of intragastric pH-metry:

1. Daily esophageal pH-metry (24 hours or more);

2. Daily gastric pH-metry (24 hours or more);

3. The short-term intragastric pH-metry (within 2-3 hours);

4. Express pH-metry (for 15-20 minutes);

5. Endoscopic pH-metry (during gastroscopy).

Daily esophageal pH metry to determine the presence or absence of gastroesophageal reflux, especially in clinically unclear cases.

Daily esophageal pH meter is required:

1) Absence of severe endoscopic changes in patients with typical clinical signs of gastroesophageal reflux disease (GERD);

2) For suspected extraesophageal manifestations of GERD:

cardiac - chest pain not associated with diseases of the cardiovascular system. In patients with normal coronary angiography data in 40-50% chest pain attacks are associated with GERD;

bronchopulmonary - connection asthma attacks with episodes of reflux in 34-89% of cases and in 20% of healthy individuals over a lifetime marked by bouts of bronchospasm associated with acid reflux into the esophagus;

laringofaringeal - acid reflux in 10-50% of cases the cause of pathological voice hoarseness, chronic cough, chronic laryngitis, granulomas of the vocal cords, throat or trachea stenosis, and sometimes even neoplastic processes;

Dental - erosion of tooth enamel, tooth decay, erosion of the mucous membrane of the mouth. More than 80% of patients with gingivitis and gum disorders, GERD appears;

3) before and after surgery on the reflux esophagitis;

4) to evaluate the effectiveness of the treatment (especially in patients with mild clinical symptoms of GERD).

Information obtained during 24-hour pH meters, accurately determine how long mucosa of the esophagus is exposed to acid vozdeystviyusolyanoy and evaluate the effectiveness of esophageal clearance. Under normal conditions in the lower third of the esophagus meets pH 6.0. At pH meter in gastroesophageal reflux taken to mean the episodes in which esophageal pH drops below pH 4. Level 4 was set as the threshold, because this level allows the most reliable statistical divide patients with GERD and healthy.

In analyzing the pH in the esophagus grams made use of the following indicators:

1. The percentage of time the pH <4. This is the most significant difference between pathological and physiological reflux. This figure does not depend on whether the episodes are rare, but long or, short but frequent.

2. The percentage of time the pH <4 in the upright position of the patient.

3. The percentage of time the pH <4 in the horizontal position of the patient.

4. The total number of reflux pH <4 for the day.

5. The amount of reflux pH <4 lasting more than 5 minutes per day.

6. The duration of the longest reflux pH <4.

The last two parameters characterize the ability of the esophagus to cleanse itself and therefore may indicate the severity of the violations. Increasing the number of reflux lasting more than 5 minutes and increase the duration of the longest reflux suggests the presence hypomotoric dyskinesia of the esophagus. The severity of gastroesophageal reflux disease (GERD) estimate the indexes in the table. 7.1

Table 7.1 The severity of reflux in terms of 24-hour pH metry.

Index	**Norma**	**Mild GERD**	**Moderate GERD**	**Severe GERD**
Time, pH <4, total, %	4,5	From 4,5 to 6,0	From 4,5 to 7,5	More 7,5
Time, pH <4, standing, %	8,4	From 8,4 to 9,3	From 9,3 to 10,2	More 10,2
Time, pH <4, lying, %	3,5	From 3,5 to 4,0	From 4,0 to 4,5	More 4,5

Duration of reflux with pH <4	47	From 47 to 56	From 56 to 67	More 67
Amount of reflux lasting more than 5 minutes	3,5	From 3,5 to 4,0	From 4,0 to 6,5	More 6,5
The longest reflux, min	20	From 20 to 46	From 46 to 66	More 66

In addition to these settings often use index de Meystera which integrally combines all of these parameters. If the value more 14.72 is a finding of GERD.

Daily gastric pH metry.

Daily gastric pH metry allows you to:

1) Judge the process of acid throughout the day in the wild with the assessment of various factors (eating, smoking);

2) Evaluate the effect of various drugs on intragastric acidity (H2-receptor antagonists, blockers of H +/K + -ATPase, antacids, etc.);

3) identify resistance to accept different antisecretory drugs;

4) detect nocturnal acid breakthrough, when patients receiving proton pump inhibitors, a decrease in pH below lasting more than 4 hours;

5) evaluate the functional state of the stomach before and after surgery;

6) to choose effective scheme taking antisecretory drugs, especially in patients with bleeding ulcers.

The main indicator of gastric pH monitoring is the total time with pH 3-4. For quick repair of gastric ulcers and duodenal ulcers should provide pH 3-4 for 18-20 hours a day (Bell`s rule).

Electrogastroenteroghraphy (or electrogastrogram) - a research method of motor-evacuation function of gastrointestinal tract using simultaneous registration of biopotentials its various departments.

Short-term intragastric pH meter is used to study acid-and gastric acid-neutralizing function in basal conditions and after stimulation (histamine or pentagastrin). Measured average pH level in different parts of the stomach, and concludes on them. The evaluation criteria are given in Table 7.2.

Criteria for assessment of body stomach pH. Table 7. 2

Status of acid in the stomach	pH level	
	Basal	**Stimulated**
Hyperacidyty, continuous acid	0,9-1,5	0,9-1,2
Normal acidyty, continuous acid	1,6-2,0	1,2-2,0
Hypoacidyty	2,1-6,0	2,1-3,0
Subanacidyty	3,1-5,0	
Anacidyty	more 6,0	more 5,0

The evaluation functions neutralizing antrum spend the difference minimum pH in the stomach and maximum - in the antrum (Table. 7.3).

Evaluation of neutralizing function of antrum. Table 7. 3

pH max (antrum)-pH (body)	**Conclusion**
4,0 and more	Compensation
1,5-3,9	Subcompensation
Less 1,5	Decompensation

The main functional test in short-term intragastric pH metry is alkaline test by Noller. It is that the patient is administered through the mouth into the stomach of 0.5 g of sodium bicarbonate (soda) dissolved in 30 ml of water, and using the instrument for intragastric pH measuring pH dynamics registered in the stomach. The result is the introduction of alkali neutralization reaction of hydrochloric acid HCl + NaHCO3 = NaCl + CO2 + H2O, the pH increases, and a so-called base time back to the original level after the allocation of hydrochloric acid in the stomach.

Criteria for evaluation of acid-forming function of the stomach are given in Table. 7.4.

Criteria for evaluation of acid-forming function of the stomach with alkaline test. Table 7.4

Evaluation alkalify function of the stomach	**Alkaline time, min.**	
	Fasting	**Stimulated**
Sharp increases production of hydrochloric acid	less 10	less 5
Increases production of hydrochloric acid	10-20	5-10

Normal production of hydrochloric acid	20-25	10-15
Reducing production of hydrochloric acid	more 25	more 15

At express- pH metry is determined only basal level of acidity, that is the question of the presence or absence of hydrochloric acid and determined the pH in the stomach.

For short-term and rapid pH measuring instrument commonly used computer "Gastroscan-5M" allows you to explore the 5 patients simultaneously.

Indications for intragastric pH metry:

1. Gastroesophageal reflux disease (GERD);
2. Gastric ulcer and duodenal ulcer;
3. Various forms of chronic gastritis, duodenitis, dyspepsia;
4. Zollinger – Ellison`s syndrome;
5. Barrett's esophagus;
6. Evaluation of action of drugs that reduce the secretion of individual selection for the patient;
7. State after gastrectomy.
8. Contraindications for intragastric pH metry
9. Contraindications to study consisting of contraindications to gastric probe input and contraindications to the use of certain stimulants or inhibitors of gastric secretion.

Contraindications for intragastric pH metry

1. Gastric bleeding (bleeding during and for 10 days after its completion);
2. Aortic aneurysm;
3. Burns, diverticula, esophageal stricture;
4. Severe hypertension and coronary insufficiency;
5. Nasal obstruction;
6. Severe maxillo-facial trauma;
7. Severe coagulopathy.

Relative contraindications for intragastric pH metry

1. Recent surgery of the upper gastrointestinal tract;
2. Tumors and ulcers of the esophagus;
3. The presence of esophageal varices;

4. Bleeding from the upper gastrointestinal tract (after stopping the bleeding may be prolonged pH meter to monitor the effectiveness of the antisecretory drugs that prevent the development of recurrent bleeding).

Endoscopic pH metry is made using a special endoscopic pH-probe introduced through the instrumental channel of the endoscope. Research conducted during the gastroscopy, extending the normal procedure for about 5 minutes. During this time, measure the acidity in 9 standard points of the stomach and duodenum under visual control.

When interpreting the results of endoscopic pH meter, be aware that endoscopy is itself a factor that stimulates acid production. Therefore, the measured pH value should be compared with the criteria for stimulated secretion in the table. 2. Endoscopic pH measuring conducted using acydogastrometr "AGM-03".

The study of the functional state of the pancreas

Exogenous function: determination of enzyme activity (lipolytic, proteolytic, amylolytic enzymes) in duodenal contents (Lund`s test), blood (ELISA, breath test), urine (amilase test - indirect PABA test) in the stool (ELISA of elastase-1).

Endocrine (incretory) function: determination of insulin, glucagon (ELISA), glucose in serum and urine as a baseline, and after glucose load (glucose).

Coprogram - macroskopy, microscopy (steatorrhea, creatorrhea, amylorrhea).

Ultrasound examination of pancreas - to evaluate it macrostructure, provisions concerning vessel-orientation, to get an idea of the shape, size, state of the pancreatic duct. Pancreatic tissue is homogeneous echostructure and compared with liver echostructure. The diameter of the pancreatic duct is less than 2 mm. The average thickness of the head of the pancreas - no more than 32 mm, body and tail -not more than 25 mm.

CT scan of pancreas. Normally, the pancreas reveal on computer tomograms in a relatively homogeneous body of variable form with clear, smooth contours. The density of the pancreas normally is 20-40 units. On the computer tomograms clearly distinguish the head, body, tail of the pancreas, which is a measure of the thickness of all parts of the body. The transverse size of the pancreatic head is 25 mm body - 20 mm tail - 15 mm.

TESTS OF THE MAIN SYMPTOMS AND RESEARCH METHODS IN GASTROENTEROLOGY

1. The main causes of obstructive jaundice:

a) Closing the lumen of the common bile duct;

b) Closing the lumen of the gallbladder duct;

c) Poisoning mushrooms;

d) cancer of the caudal part of pancreas;

d) cancer of the pyloric part of stomach.

2. The main reason of biliary diskinesia:

a) acute viral hepatitis;

b) toxic effects;

c) the presence of foci of chronic infection in the body;

d) change of neuro-reflex regulation of the gallbladder;

e) fasting.

3. What is the most appropriate of stimulants at research of gastric secretion?

a) Alcohol;

b) Caffeine;

c) Histamine;

d) **Pentagastrin.**

4. Obstructive jaundice characterized by:

a) hyperbilirubinemia;

b) hyperurobilirubinemia;

c) increase of serum aminotransferase;

d) Increased γ-globulin;

d) Increased alkaline phosphatase.

5. Gilbert's disease is accompanied by:

a) hyperbilirubinemia due to conjugated bilirubin;

b) hyperbilirubinemia due to unconjugated bilirubin;

c) hyperurobilirubinemia;

d) increased serum aminotransferase;

d) Increased alkaline phosphatase.

6. The least typical of portal hypertension:

a) the development of collaterals;

b) bleeding from varices;

c) scites;

d) fever;

e) splenomegaly.

7. If you find change ultrasonography of focal liver should appoint research:

a) A-fetoprotein;

b) ALT;

c) AST;

d) HBs Ag;

d) anti HCV.

8. Obstructive jaundice at Gallstone disease occurs when:

a) bocking the gallbladder to the neck;

b) the cystic duct blockage;

c) a blockage in the common bile duct;

d) a blockage of pancreatic ducts;

d) all listed.

9. Hyperkinetic dyskinesia the gallbladder unlike the Gallstone disease is not characterized by:

a) vomiting by bile;

b) nausea;

c) constipation;

d) pains in right hypochondrium after fatty foods;

e) leukocytosis.

10. What are pH changes considered normal after stimulation of gastric secretion pentagastrin?

a) 2,1-3,5;

b) 3,6-5,9;

c) 1.2-2.0;

d) 6.0 and>.

11. How many portions of bile obtained in the phase 5 duodenal tubage:

a) 3;

b) 5;

c) 4;

d) 6;

d) 2.

12. At ultrasound enlargement choledochus and intrahepatic bile ducts is revealed. What research is necessary to clarify the diagnosis?

a) Liver biopsy;

b) Endoscopic Retrograde holangiopancreatography;

c) Viral hepatitis markers;

d) Proteinogram;

e) US of portal system.

13. What are the most informative investigations in diagnostics Gallstone disease?

a) X-ray;

b) Thermography;

c) Laparoscopy;

d) Ultrasound;

e) Radionuclide studies.

14. The most significant sign of bleeding from esophageal varices:

a) abdominal pain;

b) heartburn;

c) light blood in vomit;

d) the dark blood in vomit;

e) all listed.

15. Bilirubinuria takes place at:

a) hemolytic jaundice;

b) **obstructive jaundice;**

c) Gilbert's syndrome;

d) all answers are correct.

16. In the pathogenesis of anemia after a stomach resection primary are:

a) malabsorption of Fe;

b) malabsorption of B12;

c) the sharp decline HCl production;

d) development of secondary colitis;

d) limitations in the diet.

17. Gilbert's disease is accompanied by:

a) hyperbilirubinemia due to conjugated bilirubin;

b) **hyperbilirubinemia due to unconjugated bilirubin;**

c) hyperurobilirubinemia;

d) increased serum aminotransferase;

d) Increased alkaline phosphatase.

18. Ortner`s symptom is characteristic for:

a) steatosis;

b) exacerbation of calculous cholecystitis;

c) hypotonia of Oddi`s sphincter;

d) Violation of secretory liver function.

19. What cholekinetik administered through a tube in the fractional duodenal tubage?

a) 25% solution of magnesium sulfate;

b) 33% solution of magnesium sulfate;

c) 10% solution of Carlsbad salt;

d) 20% solution of sorbitol;

d) 40% solution of sorbitol.

20. Specify the contraindication for washing of a stomach:

a) gastric bleeding;

b) epilepsy;

c) stenosis of the pharynx and esophagus;

d) all listed.

21. How many servings of bile obtained in the duodenal tubage:

a) 3;

b) 1;

a) 2;

d) 5;

22. For hyperkinetic dyskinesia gallbladder unlike the cholecystytis is not characteristic:

a) vomiting by bile;

b) nausea;

c) constipation;

d) pain in right hypochondrium after fatty foods;

e) leukocytosis.

25. CT-scan is the necessary image to diagnose:

a) Chronic hepatitis;

b) fatty liver;

c) cirrhosis;

d) liver cancer;

e) amyloidosis liver.

GASTROESOPHAGEAL REFLUX DISEASE

1. Choose dose of omeprazole for the treatment of non-erosive form GERD/

a) 20 mg / day;
b) 10 mg / day;
c) 40 mg / day
d) 80 mg / day.

2. What middle age of patients with GERD?

a) 18-20
b) 20-30 years
c) 30-40 years
d) 40-60 years

3. The dysphagia is manifested:

a) GERD, in tumors of the esophagus;
b) Crohn's disease;
c) Gallstone disease;
d) Liver cirrhosis.

4. On the tone of the lower esophageal sphincter act:

a) glucagon, somatostatin, secretin
b) beta-blockers, sedatives, hypnotics
c) nitrates, theophylline
d) all listed

5. What are the main "esophageal" GERD symptoms:

a) nausea
b) odynofagia (pain when swallowing and passage of food through the esophagus)
c) sense of chest "heaviness", vomiting, hiccups
d) all listed

6. What are the main "extraesophageal " GERD symptoms:

a) heart pain (night), "like coronary" heartbeat
b) reflex bronchospasm (cough, scratchy throat)
c) . burning tongue, oral mucosa
a) all listed

7. List the orofaryngeal symptoms of GERD:

a) inflammation of the nasal reed tonsil
b) throat
c) feeling of "lump" in the throat
d) all listed

8. List the otolaryngial symptoms of GERD:

a) laryngitis, croup
b) granulomas, polyps of the vocal cords
c) medial otitis, rhinitis
d) all listed

9. What drugs are prescribed at duodeno-gastric reflux:

a) NSAD
b) prokinetic
c) antispasmodics
d) ursodesoxycolic acid (ursofalc)

10. Barrett`s Esophagus is:

a) intestinal metaplasia of incomplete type in the distal part of esophagus;
b) infiltration in the distal part of esophagus;
c) infiltration of the stomach;
d) intestinal metaplasia of the stomach

11. List the performance criteria for the treatment of GERD:

a) elimination of clinical symptoms
b) achieving endoscopic remission (healing of defects, eliminating inflammation)
c) Barrett`s esophagus prevention
d) all listed

12. Specify the position of the Maastricht III Consensus for use in the treatment of GERD Helicobacter therapy:

a) prolonged use of antihistamines
b) prolonged use of NSAD
c) prolonged use of PPIs in young people
d) all listed

13. Secondary prevention of GERD is:

a) mandatory components of primary prevention performance
b) meals on the presence of symptoms of GERD avoid torso, lifting heavy objects, dressing, tight corsets, do not go immediately after a meal
c) limit the drugs and foods that relax the esophageal sphincter
d) all listed

14. All patients with signs of GERD erosive reflux require follow-up with endoscopic control at least:

a) 1 every 2-3 years
b) 1 per month
c) 1 per year
d) 2 times a year

15. Key provisions in GERD diet:

a) The need to avoid eating acidic fruit juices, foods that enhance gas production, limit intake of fats, chocolate, coffee, garlic, onion, pepper
b) should exclude alcohol, spicy, hot and cold foods, carbonated beverages
c) avoid overeating and not eating for several hours before bedtime
d) all listed

16. Recurrences of GERD occur in:

a) . 80%
b) . 20% of cases
c) . 10%
d) 2%

17. What drugs are the drugs of choice in the treatment of GERD:

a) antihistamines;

b) NSAD
c) PPI
d) antibiotics

18. What preparations relief heartburn in patients with GERD:

a) antibiotics;
b) PPI;
c) probiotics;
d) NSAD.

19. Add a dose of pantoprazole:

a) 10 mg;
b) 25 mg;
c) 30 mg;
d) 40 mg.

20. The duration proton pump inhibitors use in patients with erosive form GERD is:

a) 1-2 weeks;
b) 1-3 weeks;
c) **8-12 weeks**;
d) 4-8 weeks**.**

21. The duration proton pump inhibitors use in patients with non-erosive form GERD is:

a) **1 time per day for 4-8 weeks**
b) 1 time per day for 1-2 weeks
c) 1 time per day for 10 days
d) 1 time per day for 1 year

22. To diagnose Barrett's esophagus is the method of choice:

a) Barium meal test;
b) intraesophageal daily pH-monitoring;
c) **FGDS, biopsy or chromoendoscopy with methylene blue;**
d) bilimetry.

23. Barrett's Esophagus is:

a) **complication of GERD**;
b) complication of peptic ulcer disease;
c) complication of pancreatitis;
d) all listed

24. What diseases can lead to reflux:

a) asthma, hiatal hernia;
b) ascites, abdominal tumor;
c) duodenal ulcer;
d) all listed

CHRONIC GASTRITIS

1. The most informative method of diagnosis of chronic gastritis are:

a) upper endoscopy with biopsy of the mucous membrane;
b) barium meal test;
c) urea breath test;
d) antibodies to parietal cells.

2. The basic morphological changes in chronic gastritis are:
a) The predominance of proliferation of the epithelium;
b) The predominance of the processes of differentiation of the epithelium;
c) The predominance of processes atrophy of the mucous membrane;
d) All listed.
3. The obligatory condition for the existence Helicobacter pylory in stomach is:
a) The level of pH (3 - 6) around the colonies of bacteria;
b) The presence of urea in gastric juice;
c) The presence of gastric epithelium;
d) All listed.
4. Violation of regeneration in chronic gastritis is characterized by:
a) rapid movement are not fully differentiated cells from generation area;
b) the emergence of mixed-cell structural features several specialized epithelial cells;
c) flattening pits and change the gastric epithelium;
d) All listed.
5. The syndrome of gastric dyspepsia includes:
a) loss of appetite;
b) unpleasant taste in the mouth;
c) belching, nausea, sometimes vomiting;
d) All listed.
6. Which option is most likely chronic gastritis in patients with rheumatoid arthritis, which lasted (5 months) take diclofenac?
a) Chronic gastritis, type C;
b) Chronic gastritis, type A;
c) Chronic gastritis type B;
d) Chronic eosinophilic gastritis.
7. In a normal physiological process of updating the mucous membrane of the stomach lasts for:
a) 2 - 4 days
b) 3 - 6 days
c) 10 - 14 days;
e) 20 - 25 days.
8. What is a prerequisite for settling Helicobacter pylory mucosal duodenum?
a) atrophy of the mucous membrane of the stomach;
b) Intestinal metaplasia of duodenal epithelium;
c) duodenal gastric metaplasia epithelium;
d) Increased regeneration of epithelial cells.
9. For final diagnosis of chronic gastritis is need:
a) Serological studies;
b) Endoscopy;
c) Bacteriological research;
f) Histological examination of biopsy.
10. For atrophic chronic gastritis with secretory insufficiency is characterized by:
a) Bad taste in the mouth;
b) Belching food, rotten or rancid;
c) loss of appetite, nausea;

d) All listed.

11. Which of the following antibiotics not used in the treatment of Hp (+) chronic gastritis in primary resistance to disease pathogen him?

a) Amoxycyllin;

b) Clarithromycin;

c) Benzylpenicyllin;

d) Metronidazole.

12. The development of chronic gastritis type A major role is:

a) Infection with Helicobacter pylory;

b) hereditary immunological reactivity;

c) Infection St. aureus;

e) Alimentary factor.

13. By reducing the gastric pH Helicobacter pylory migrate to:

a) The body of the stomach;

b) the fundus of the stomach;

c) the duodenum;

d) All of the above is true.

14. Clinical symptoms of chronic gastritis include:

a) pain;

b) the syndrome of gastric dyspepsia;

c) pain syndrome and gastric dyspepsia;

d) malabsorption syndrome.

15. The method to evaluate the gastric secretion is:

a) FGDS;

b) urease test;

c) intragastric pH metry;

d) All listed.

16. Etiological factors of chronic gastritis are:

a) Nutritional factors;

b) Occupational hazard;

c) Effect of medicines;

d) All listed.

17. Chronic gastritis type A mostly localized:

a) In the fundus of the stomach;

b) In the small curvature;

c) In the body of the stomach;

d) In the antral part.

18. The main cause of duodenal epithelium metaplasia:

a) gastric hyperacydity;

b) gastric hypoacydity;

c) reduction motor function of the stomach;

d) increased production of mucus.

19. Pain in chronic gastritis are located in:

a) epigastric area;

b) mesogastrium;

c) Around the navel;

d) All of the above is true.

20. The value of pH in the stomach area of the body in conditions of normal basal secretion is:
a) pH 2.9 - 2.0;
b) pH 4.9 - 3.0;
c) pH 6.9 - 5.0;
e) pH 1.9 - 0.9.
21. Chronic gastritis Helicobacter pylory appears mainly:
a) In the cardia;
b) In the body of the stomach;
c) At the fundus of the stomach;
e) In antral part.
22. Chronic gastritis type B immunological activity of the body:
a) higher;
b) reduced;
c) normal;
d) moderately reduced.
23. In patients with chronic gastritis in the gastric mucosa develops:
a) inflammation;
b) atrophy;
c) violation of cell regeneration;
d) all listed.
24. Pain in chronic gastritis often are:
a) cutting nature;
b) dull, aching character;
c) cramping in nature;
e) barbed character.
25. Stimulating with pentagastrin leads to pH level of gastric secretion:
a) pH 1.5 - 1.1;
b) pH 1.8 - 2.5;
c) pH 2.0 - 2.9;
e) pH 1.0 - 0.9.

PEPTIC ULCER

1. The main etiological factor in peptic ulcer present is:
a) Helicobacter pylori;
б) St. aureus;
c) E. coli;
д) Enterococcus.
2. Duodenal ulcer often accompanied by:
a) metaplasia of intestinal epithelium in the duodenum;
b) intestinal epithelial dysplasia;
c) decrease of parietal cells;
f) Erosions of the mucosa.
3. Infection Helicobacterpylori accompanied by:
a) reduction in serum gastrin;
b) increasing the concentration of somatostatin;
c) increasing the concentration of gastrin-releasing factor;

d) reducing production of HCl.

4. The main stimulant of gastric secretion is:

a) Histamine;

b) Gastrin;

c) Acetylcholine;

e) Insulin.

5. What factor inhibits gastric secretion?

a) Thyroid hormones;

b) Cholecystokinin;

c) Glucagon;

e) Insulin.

6. Bicarbonate secretion is stimulated:

a) Adrenaline;

b) Cholecystokinin;

c) Lipase;

e) Bile acids.

7. In gastric ulcer leading role in the pathogenesis performs:

a) Increasing the activity of acid-peptic factor;

b) Rapid gastric emptying;

c) Increased activity of the parasympathetic division of the autonomic nervous system;

d) Impressions gastric mucosa by reducing its regenerative ability.

8. Typical complaints ulcers include:

a) Pain shaking nature;

b) cutting pain, sometimes cramping nature;

c) Pain aching character;

d) Pain dull character.

9. Pain ulcers differ:

a) rhythm associated with eating;

b) consistency;

c) chaos;

e) episodic.

10. For ulcers duodenum characterized by:

a) Early pain;

b) Night, hunger pains;

c) cramping;

e) The sharp, stabbing pain.

11. Gastric hypersecretion features include:

a) A sense of bitterness in the morning;

b) Heartburn, belching sour, vomiting acidic content;

c) Belching rotten;

e) Belching air.

12. When duodenum ulcer meal:

a) Increases pain;

b) Brings temporary relief;

c) Does not affect the pain;

e) It is irradiation of pain.

13. In gastric ulcer vomiting at the height of pain brings:

a) Relief;

b) Increased pain;

c) Strengthening regurgitation;

d) The changing nature of pain.

14. When ulcer pain is localized:

a) In the epigastric region;

b) In umbilical area;

c) In the area of right upper quadrant;

d) In the area of left upper quadrant.

15. Pain at peptic ulcer:

a) has a local character;

b) has spilled character;

c) There are no clear localization;

e) Has typical localization.

16. In uncomplicated ulcer:

a) no irradiation of pain;

b) pain irradiation available;

c) there is no circadian rhythm of pain;

e) the pain is diffuse in nature.

17. The appearance of irradiation of pain ulcers indicates:

a) aggravation of ulcerative process;

b) penetration into adjacent organs;

c) bleeding;

e) malignancy ulcers.

18. On examination the patient tongue ulcers observed:

a) marginal papillae hypertrophy;

b) atrophy papillae;

c) Brown patches;

e) "Crimson" color language.

19. Symptom Mendel determined by:

a) The percussion epigastric area;

b) palpation of the epigastric area;

c) The percussion in costal arc edge of his hand;

e) The percussion on the spinous processes XI-XII thoracic vertebrae.

20. An examination of a patient with suspected ulcer must necessarily hold:

a) Endoscopy;

b) Biochemical blood;

c) scatological research;

e) Immunological research.

21. The reaction Gregersen made:

a) once;

b) twice;

c) three times;

e) four times.

22. The most informative method of diagnosis of peptic ulcer are:

a) Endoscopy;

b) X-ray study;
c) Fractional study of gastric contents;
f) biochemical blood.

23. Which of the following listed research methods used to diagnose H.pylori:
a) X-ray study;
b) FGDS;
c) intragastric pH-metry;
e) urea breath test.

24. Direct signs of a stomach ulcer at Ro-study include:
a) Symptom "index finger";
b) Symptom "niche";
c) The convergence of folds;
e) Inflammatory shaft.

25. Ro-study ulcers when necessary to:
a) suspected bleeding;
b) suspected penetration;
c) suspected malignancy;
d) suspected peryvistseryt.

26. Shock Algover`s index use for:
a) assessing the severity of coma;
b) assess the degree of blood loss;
c) Assessment of the internal organs;
f) evaluation.

27. Shock Algover`s index - is:
a) The ratio of systolic blood pressure to heart rate;
b) The ratio of diastolic blood pressure to heart rate;
c) The ratio of the pulse rate to the value of systolic blood pressure;
e) ratio of the pulse rate to the value of diastolic blood pressure.

28. Ulcer perforation is characterized by:
a) The disappearance of liver dullness on percussion;
b) Local resistance anterior abdominal wall palpation:
c) Increased splenic dullness;
d) Pain in umbilical area.

29. For the diagnosis of ulcer perforation is necessary to:
a) Endoscopy;
b) Plain abdominal X-ray;
c) Complete blood;
d) Serological studies.

30. Perforation in retroperitoneal space is characterized by:
a) Developing sharply, characterized by a sharp "stabbing" pain;
b) Acute pain that is accompanied by signs of peritoneal irritation;
c) gradually start radiating pain in the back;
e) Dizziness, drop in blood pressure.

31. The most informative method of research at penetrations ulcers are:
a) Ultrasound;
b) FGDS;
c) X-ray;

d) Laparoscopy

32. At ulcer localization in a bulb of duodenum or pyloric part of stomach often develop complications such as:

a) perforation;
b) Bleeding;
c) deformation and stenosis;
e) Penetration.

33. The best combination for eradication is:

a) Clarithromycin + amikacin;
b) Clarithromycin + amoxicillin;
c) Clarithromycin + vancomycin;
d) amoxicillin + metronidazole.

34. The dose of clarithromycin for eradication Helicobacterpylori is:

a) 500 mg x 2 times/day;
b) 250 mg x 2 times/day;
c) 250 mg 3 times/day;
e) 1000 mg x 2 times/day.

35. The dose of amoxicillin for eradication Helicobacterpylori is:

a) 500 mg x 2 times/day;
b) 1000 mg x 2 times/day;
c) 250 mg x 4 times/day;
d) 500 mg 3 times/day.

36. In the treatment of peptic ulcer disease first line of metronidazole assigned a dose:

a) 500 mg x 2 times/day;
b) 500 mg 3 times/day;
c) 500 mg x 4 times/day;
d) 250 mg 3 times/day.

37. Nitrofurans use in the treatment of H. pylori infection in:

a) triple scheme;
b) quadriple scheme;
c) As preparations reserve;
d) Not used.

38. Preparations of bismuth in the treatment of peptic ulcer disease are in:

a) first-line therapy;
b) second-line therapy;
c) as monotherapy;
d) not used.

39. De Nola dose in the treatment of peptic ulcer are:

a) 120 mg x 4/day;
b) 120 mg 3 times/day;
c) 120 mg x 2 times/day;
d) 240 mg 3 times/day.

40. Preparations colloidal bismuth:

a) affect the motor function of the stomach;
b) affect the secretion of hydrochloric acid;
c) combining with proteins to form insoluble film on the surface;

e) Connect hydrochloric acid.

41. Proton pump inhibitor in addition to the blockade of proton pump:

a) promotes mucous discharge;

b) improves the motility of the stomach;

c) Has secondary Helicobacter activity;

e) It forms a protective film.

42. By selective m cholinoblockers include:

a) Atropine;

b) Metacin;

c) Gastrotsepin;

e) Platifillin.

43. In accordance with the Maastricht 4-2010 consensus Hp-positive ulcer is an absolute indication for:

a) eradication H.pylori;

b) carrying out preventive treatment;

c) surgery method;

d) immunomodulators use.

44. The successful eradication of Hp infection can:

a) Apply physiotherapy;

b) Do not diet;

c) To reduce the risk of recurrence and complications;

e) Leave endoscopic research.

45. Triple therapy includes:

a) clarithromycin, Omeprazole Ranitidine;

b) amoxicillin Ranitidine, De-nol;

c) clarithromycin, amoxicillin, metronidazole;

e) pantoprazole, clarithromycin, amoxicillin.

46. When proton pump inhibitors you can`t use administers:

a) Antacids;

b) H2 –histamin blokers;

c) M-anticholinergics;

e) M-cholinomimetics.

47. When triple therapy ineffective is necessary:

a) Repeat the course after 1 week;

b) Start quadriple therapy;

c) Add antacids;

d) increase the dose of drugs used.

48. The second-line treatment of peptic ulcer includes:

a) omeprazole, ofloxacin, ranitidine, metronidazole;

b) tetracycline, clarithromycin, de-nol, metronidazole;

c) pantoprazole, tetracycline, de-nol, metronidazole;

e) omeprazole, amoxicillin, metronidazole, almagel.

49. What are the numbers necessary to maintain pH in the body of the stomach to better scarring ulcers?

a)> 2.0;

b)> 2.5;

c)> 3.0;

d)> 4.0.

50. When therapy is performed on demand?

a) If there is no effect of the eradication;

b) Within one year after scarring ulcers;

c**) When the first symptoms of dyspepsia gastric ulcer after effective treatment of duodenal;**

d) If there is a concomitant reflux gastritis and reflux esophagitis.

51. The duration of maintenance therapy duodenal ulcer:

a) 1 year;

b) 2 years;

c) 3 years;

e) 4 years.

52. The duration of maintenance therapy gastric ulcer:

a) 1 year;

b) 2 years;

c) 3 years;

e) 4 years.

53. Weekends therapy - is:

a) Option prolonged maintenance therapy;

b) Option intermittent therapy;

c) Alternative therapy on demand;

d) Option second-line therapy.

54. In the treatment of ulcers Hp-negative drugs of choice are:

a) proton pump inhibitors;

b) macrolides;

c) H2 -histaminoblokatory;

f) preparation of bismuth.

55. Which of the following conditions are absolute indications for surgical treatment?

a) Penetration ulcer;

b) repeated bleeding history;

c) Lack of effect of adequate treatment of benign gastric ulcers within 3 months;

e) Kallosus ulcer that long rumen.

CELIAC DISEASE AND OTHER ENTEROPATHY, CROHN'S DISEASE, NONSPECYFIC ULCERATIVE COLITIS

1. Crohn's disease is characterized by:

A. chronic or night diarrhea

B. abdominal pain, weight loss, fever, rectal bleeding

C. infiltrates and abscesses that palpable in the abdomen (intestinal and perianal)

D. **All listed**

2. Extraintestinal symptoms of Crohn's disease:

A. Inflammatory diseases of the eye (iridocyclitis, episcleritis, etc.).

B. arthritis (affects large joints)

C. impression kidneys (the appearance of oxalate, obstructive hydronephrosis)

D. Impressions liver (steatosis, hepatitis)

E. **All listed**

3. Best`s index (Crohn's disease activity) is:

A. Changes in general condition, availability resistance areas palpation of the abdomen

B. Degree abdominal pain and the number of episodes of diarrhea

C. extraintestinal symptoms (arthritis, erythema nodosa)

D. All listed

4. Localization distinguish Crohn's disease:

A. Terminal ileitis

B. Ileocolitis

C. Preferential impression colon

D. All listed

5. Severe Crohn's disease is not typical:

A. Diarrhea> 6 times a day with the release of microscopic blood;

B. Diarrhea 2-4 times a day, ESR <20 mm/h;

C. Fever> 37.5 ° C, tachycardia, ESR> 50 mm / h.

D. The presence of gastrointestinal complications (abscesses, intestinal obstruction, bleeding, high intestinal fistulas)

6. Complications of Crohn's disease:

A. Intestinal obstruction

B. The emergence of fistulas, abscesses, fractures, bowel stricture

C. Toxic colon dilatation

D. All listed

7. To verify the diagnosis of Crohn's disease compulsory studies are:

A. Endoscopic examination of morphological examination of biopsy samples

B. Morphological study biopsy (reveals specific transmural granulomatous inflammation)

C. Irrigoscopy and barium passage through the small intestine

D. All listed

8. Endoscopic examination for Crohn's disease is characterized by:

A. asymmetric cell transmural granulomatous impression of any part of the intestinal tube type "Bridge"

B. Diffuse congestion, lack of vascular picture, erosion, inflammation of the rectum

S. "Grainy mucus", petechiae, contact bleeding, ulcerated areas of irregular shape

D. The intense inflammatory exudate triple, total impression colon

9. Crohn's disease with characteristic morphological study:

A. Segmentary impression intestine, transmural bowel inflammation, the presence of longitudinal ulcers, fractures, changes in the mucous type "Bridge"

B. inflammatory infiltration predominantly mucosa, submucosa sometimes, swelling of the mucosa, bleeding in her order, abscesses, superficial ulcers

C. Infiltration mainly lamina propria mucosa big foamy PAS-positive macrophages

D. All listed

10. For the treatment of Crohn's disease use:

A. Budesonide + mesalasine

B. Prednisolone

C. Metronidazole and ciprofloxacin or their combination

D. All of the above listed in certain combinations depending on the current and severity of Crohn's disease

11. For the treatment of Crohn's disease use:

A. Salofalc (mesalasine)

B. Meloxicam

C. Azathioprine

D. All listed

12. Ulcerative colitis is characterized by extraintestinal complications:

A. Skin and mucous membranes: erythema nodosa, uveitis, keratitis, iridocyclitis

B. Joints: arthritis, sacroileitis

C. Internal organs - liver (hepatitis), bronchopulmonary, glomerulonephritis, vasculitis

D. All listed

13. Surgical treatment of ulcerative colitis is shown:

A. When failure of conservative therapy

B. When lightning current in very severe cases

C. Fistula, light epithelial dysplasia

D. All listed

14. For the treatment of ulcerative colitis apply:

A. Budesonide + mesalasine

B. Nimesulide

C. Metronidazole and ciprofloxacin or their combination

D. All of the above listed in certain combinations depending on the current and severity of Crohn's disease

16. For endoscopic picture of ulcerative colitis is characterized by:

A. Diffuse hyperemia. The absence of vascular pattern. Erosion. Single ulcer surface area. Inflammation limited to the rectum.

B. "Grainy" slimy. Petechiae. Contact bleeding. Not ulcer surface area, merging, irregular shape, covered with slime. Preferably Left hand experience.

C. Intensive necrotizing inflammation. The purulent exudate. Spontaneous hemorrhage, microabscesses.

D. All listed

17. For ulcerative colitis are not typical symptoms:

A. toxic dilatation of the bowel, Colon Cancer

B. perforation, bleeding

C**. hepatic coma**

D. stenosis, pseudopoliposis

18. As the prevalence identify the following forms of ulcerative colitis:

A. Forensic colitis

B. Proctosigmoiditis, proctitis

C. Left-hand colitis

D. All listed

19. Adrift distinguish ulcerative colitis:

A. Acute ulcerative colitis (lightning)

B. Chronic

C. Chronic recurrent course

D. All listed

20. For ulcerative colitis is not typical:

A. diarrhea mixed with blood and mucus;

B. abdominal pain, low-grade fever body;

C. tenesmus, weight loss;

D. excessive sweating

21. When endoscopic examination for ulcerative colitis is characterized by:

A. asymmetric cell transmural granulomatous impression of any part of the intestinal tube type "Bridge";

B. Diffuse congestion, lack of vascular picture, erosion;

C. "Grainy mucus", petechiae, contact bleeding, ulcerated areas of irregular shape;

D. The intense inflammatory exudate triple, total impression of the colon.

22. What violation of protein metabolism observed in celiac disease:

A. hypoproteinemia, hypoalbuminemia;

B. proteinuria;

C. hypovitaminosis;

D. All listed.

23. Sodium deficiency in celiac disease leads to:

A. lack of exercise;

B. fatigue, muscle pain, decreased muscle tone;

C. nausea and vomiting;

D. **All listed**.

24. For the diagnosis of colon pathologies most commonly used:

A. Ultrasound;

B. Jejunoscopy;

C. Colonoscopy;

D. Retropnevmoperytoneum.

25. Amylorrhea - a detection by microscopic examination of stool:

A. muscle fibers;

B. free extracellular starch;

C. neutral fat;

D. remnants of undigested food.

26. What are the clinical variants characteristic for irritable bowel syndrome?

A. With the advantage of diarrhea

B. With the advantage of constipation

C. With the advantage of abdominal pain and bloating

D. All listed.

27. For patients with irritable bowel syndrome is characterized by:

A. The absence of organic changes in the colon

B. The presence of ulcers of the colon mucosa

C. Swelling submucosal layer of the colon

D. Lack of mucosal vascular pattern colon

28. What is the prevalence of IBS?

A. 14-48%
B. 10-30%
C. 50%
D. 60%
E. 20-40%

29. Persons whose age and sex often suffer from IBS?

A. young women
B. middle-aged men
C. men and women regardless of age
D. middle-aged women
E. young men

30. What features does not fulfill the small intestine?

A. absorption of vitamins
B. production of intestinal hormones
C. stool formation and release him
D. protective function
E. cavity, membrane and intracellular digestion

31. The cause of IBS can be:

A. acute intestinal infection
V. stress
C. psycho-emotional stress
D. genetic predisposition
E. all of the right

32. The main cause disease of IBS is:

A. dynamic obstruction
B. increased mucus
C. thrombosis mesenteric arteries and their branches
D. impression intramural nerve plexus
E. heightened perception of pain impulses

33. Specify the mechanism of diarrhea in IBS:

A. violation of the motor (motor) functions intestine
B. increasing the synthesis of cholecystokinin-pancreozymine
C. increased pancreatic secretion
D. increased osmotic pressure, irritation of receptors organic acids
E. abuse enterohepatic circulation of fatty acids

34. Discomfort in patients with IBS following:

A. during the menstrual cycle
B. after drinking alcohol
S. after eating legumes
D. after stress
E. all of the right

35. Which of the following symptoms contradicts the diagnosis "IBS"?

A. abdominal pain which decreases after a bowel movement
B. lactose intolerance
C. constipation alternating with diarrhea
D. mucus in feces

E. bloating

36. What feces are not characteristic for IBS?

A. "sheep" feces mixed with mucus
B. unformed stool
C. watery stool
D. ribbon-shaped stool
E. mushy stools of bloody

37. At what time of day is not characterized by the appearance of a chair at the IBS?

A. predominantly in the daytime
B. predominantly during sleep
C. regardless of the time of day
D. regardless of the meal

38. Which of the following symptoms does not apply to clinical A.Manninh criteria?

A. The change in stool frequency of appearance of pain
B. changing stool consistency with the appearance of pain
C. discoloration of feces with the appearance of pain
D. reduce pain after defecation
E. apparent to the eye bloating
F. feeling of incomplete emptying of intestine mucous discharge from the rectum

39. Which of the following symptoms can not be in the IBS?

A. painless diarrhea
B. progression of symptoms
C. steatoreja
D. hypochondria
E. gluten intolerance

40. Add a symptom, non extraintestinal manifestations of IBS:

A. vasospastic reactions
B. feeling of "lump" in swallowing
C. urination disorder
D. lack of appetite and weight loss
E. sexual dysfunction

41. IBS characterized by the following symptoms:

A. The presence of blood in stool
B. Rush
C. weight loss
D. flatulence

42. Endoscopic signs of IBS:

A. Painting "Bridge"
C. in the presence of erosions sq.m.
C light congestion, shiny surface of the intestinal wall
D. mucosa in contact with the colonoscope bleeding

43. Prohibited foods during diarrhea of IBS include all listed, except for:

A. milk
B. dietary fiber
C. tannin foods (blueberries, strong tea)

D. cabbage, beans
E. coffee

44. What preparations are the means of choice for IBS with pain?

A. Gepabene
B. Espumizan
C. Creon
D. Duspatalin
E. Talcid

22. What prokinetics prescribed for IBS with constipation?

A. motilium
B. cerucal
C. cyzaprid
D. mosid
E. metaclopramid

25. With increased anxiety symptoms in patients with IBS should appoint:

A. tricyclic antidepressants
B. analgesics
S. sleeping
D. magnesium preparations

GALLSTONE DESEASE, CHRONIC CHOLECYSTITIS AND BILIARY DYSKINESIA

1. Leading etiopathogenetic factors of chronic cholecystitis:

A) violation of the outflow of bile;
B) violation of colloidal stability;
C) infection of bile;
D) all answers are correct.

2. Ways of penetration infection to the gall bladder:

A) hematogenous, lymphogenous, enterogenic
B) hematogenous, airborne, sexual;
C) lymphogenous, aspiration, transplacental;
D) enterogenous, nutritional, hematogenous.

3. Classification of biliary dyskynesia:

A) hyperkinetic or hypokinetic type;
C hypermotoric or hypomotoric type;
C) hypertrophic or hypotrophic type;
D) hyperergic or hypoergic type.

4. Syndrome "pentalogy F» includes:

A) blonde woman over 40, which tends to be overweight and have children;
B) blonde woman of 40 years, which tends to be overweight and have no children;
C) brunette woman over 40, which tends to be overweight and have children;
D) brown hair, thin man of 40 years who has no children.

5. What is the characteristic symptom exacerbation of chronic cholecystitis?

A) Ker`s symptom;
B) Voskresensky`s symptom;
C) Schetkin-Blumberg`s symptom-;
D) all answers are correct.

6. What is Murphy`s symptom?

A) pain increase at palpation in the gallbladder point during deep inhalation;

B) pain at palpation in the projection of the gallbladder;

C) pain in the area of the gall bladder when tapped on the right costal arch;

D) pain at palpation between the legs of the sternum-clavicular-mastoid muscle.

7. The method to take diagnosis of biliary dyskinesia:

A) fractional duodenal tubage;

B) blood test;

C) X-ray;

D) rectoromanoscopy.

8. Name the leading syndrome in exacerbation of chronic cholecystitis:

A) pain;

B) hemodynamic disturbances;

C) hepatic insufficiency;

D) biliary hypertension.

9. The method of choice in the treatment of gallstones is:

A) surgery;

B) antibiotic therapy;

C) detoxication therapy;

D) diet.

10. The leading method of diagnosis of chronic cholecystitis:

A) ultrasound;

B) duodenal tubage;

C) FGDS;

D) CT-scan.

11. What preparations are indicated for pain in the right upper quadrant, resulting hypotonic biliary dyskinesia?

a) analgesics

b) cholekinetics

c) sedatives

d) antispasmodics

e) the enzyme

12. Exacerbation of chronic non-calculous cholecystitis characterized by:

a) alternating diarrhea with constipation

b) heartburn

c) pain in the left upper quadrant

d) poor tolerance of fatty foods

d) none of the above

13. What drugs has lithogenic effect?

а) pancreatin

б) metoclopramide,

в) essenciale

г) ursodesoxicholic acid

14. Use ursodesoxicholic acid to dissolve gallstones is contraindicated:

a) cholesterol stones

b) smaller than 2 cm

c) stones in the gallbladder

d) stones in the bile ducts

15. What drugs you administer the patient with biliary hypotonic dyskinesia?

a) analgesics

b) cholekinetics

c) sedatives

d) antispasmodics

e) enzymes

16. Choose the most typical clinical symptoms of cholangitis:

a) pain in the right upper quadrant after taking fatty foods, nausea, vomiting

b) an attack of pain in the right upper quadrant of the development of jaundice, the appearance of discolored feces, dark urine

c) short-term pain in the epigastric region, vomiting, diarrhea

d) febrile temperature with a fever, enlarged liver, jaundice, leukocytosis

e) dull pain in the right upper quadrant, belching bitter

17. What is symptom of hypertonic biliary dyskinesia?

a) heaviness in the right upper quadrant

b) dyspeptic symptoms

c) pain in the right upper quadrant

d) spherical gallbladder (ultrasound)

d) positive effect of cholekinetiks

18. Cholestatic syndrome is characterized by:

a) increase of enzymes activity

b) increased bilirubin, cholesterol and alkaline phosphatase

c) reduction of albumin and clotting factors

d) dysproteinemia and sediment samples positive

19. Cholestasis leads to these violations, except for:

a) retention of bile acids in the blood

b) increased indirect bilirubin

c) increased alkaline phosphatase

d) osteomalacia

e) fatty

20. At hyperkinetic biliary dyskinesia observed:

a) sharp pain in the right hypochondrium

b) sharp pain in the left hypochondrium

c) aching pain in the right hypochondrium

d) nagging pain in the left hypochondrium

21. At hypokinetic type biliary dyskinesia observed:

a) sharp pain in the right hypochondrium

b) sharp pain in the right iliac region

c) aching pain in the right hypochondrium

d) aching pain in the right iliac region

22. What drugs relief pain at hyperkinetic biliary dyskinesia?

a) antibiotics

b) nitrofurans

c) antispasmodics

d) sulfonamides

23. Bile action has:

a) everlasting
b) calendula
c) nettle
d) plantain

24. At duodenal tubage magnesium sulfate is used to receive
a) stomach contents
b) A portion
c) B portions
d) C portions

25. In preparation for duodenal tubage cleansing enema:
a) does not apply
b) refers night
c) refers morning
d) concerns morning and evening

CHRONIC HEPATITIS

1. In what time can be diagnosed chronic hepatitis?
1. 3 months
2. 1 year
3. 6 months
4. 1 month

2. What virus of acute hepatitis is not etiological factor of chronic?
1. C
2. A
3. B
4. D

3. Which of chronic hepatitis does not exist (etiological classification)?
1. Cryptogenic chronic hepatitis
2. Toxic chronic hepatitis
3**. Persistent chronic hepatitis**
4. Chronic viral hepatitis

4. What was observed during the inspection of the skin of patients with chronic hepatitis?
1. Xanthoma
2. Annulare erythema
3. Yellowness of the skin
4. Xanthelasma

5. Which symptom is not typical for chronic hepatitis B?
1. Fatigue
2. Allopetcia
3. Nausea
4. Jaundice

6. What is normal size of the liver (by Kurlov?)
1. 14-14-15 sm
2. 7-6-6 sm
3. 9-12-14 sm
4. 9-8-7 sm

7. What is the upper limit of normal total bilirubin content in the serum?

1. 50 g/
2. 20.5 mmol/l
3. 4%
4. 8 mmol/l

8. What index don't increase in cytolytic syndrome?

1. Alanine aminotransferase
2. Lactate dehydrogenase
3. C-reactive protein
4. Glutamatdehidrohenase

9. Increased index in cytolytic syndrome is

1. γ-globulins
2. Cholesterol
3. Total protein
4. Total bilirubin

10. Increased enzyme at cholestasis syndrome is

1. Alanine aminotransferase
2. Aspartate aminotransferase
3. Alkaline phosphatase
4. Lactate dehydrogenase

11. What index reflects the level of activity of chronic hepatitis B?

1. Alanine aminotransferase
2. Creatinin
3. Glucose-6-phosphate dehydrogenase
4. Alkaline phosphatase

12. What is the characteristic feature of chronic autoimmune hepatitis?

1. Hyper-γ-globulinemia
2. leykocituria
3. Hypercholesterolemia
4. Hypokaliemia

13. What is the normal level of total protein?

1. 65-85 g/l
2. 20-45 g/l
3. 80-105 g/l
4. 100-125 g/l

14. What distinguishes morphological characters of alcoholic liver disease?

1. Lymphocyte infiltration of the portal fields
2. The presence of eosinophilic hyaline alcohol particles (Malory`s cells)
3. Mild fibrosis combined degeneration of liver cells
4. Hyperplasia stellate retyculoendoteliocytes

15. What morphological change is not typical for chronic hepatitis B?

1. Lymphocyte infiltration of the portal fields
2. Hyperplasia stellate retyculoendoteliocytes
3. Mild fibrosis combined degeneration of liver cells
4**. Violation of lobular structure of the liver**

16. What diagnostic criteria for chronic autoimmune hepatitis?

1. The positive effect of antihypertensive drugs

2. The positive effect of antibiotics
3. The positive effects of glucocorticosteroids
4. The positive effect of hepatoprotective drugs

17. What maintenance dose of prednisolone in chronic autoimmune hepatitis?
1. 100 mg
2. 50 mg
3. 5 mg
4. 10 mg

18. Which drug is the drug of choice for treatment of alcoholic hepatitis?
1. Amoxicillin
2. Ursodesoxicholic acid
3. Metionin
4. Prednisolone

19. What is the most hepatotoxic antibiotic?
1. Penicillins
2. Macrolydes
3. Cephalosporins
4. Tetracyclines

20. What drug administer the patient with chronic viral hepatitis in the phase of viral replication?
1. α-interferon
2. NASD
3. Azatioprin
4. Prednisolone

21. Which drug you choose for treatment of autoimmune hepatitis?
1. α-interferon
2. Prednisolone
3. Papaverine
4. Cephalosporins

22. What complication occurs during corticosteroid therapy?
1. Hyperglycemia
2. Hypotension
3. Nasal bleeding
4. Glaucoma

23. What complication occurs during corticosteroid therapy?
1. Bronchospasm
2. Hyper-γ-globulinemiya
3. Arterial hypertension
4. Hypoglycemia

24. What drug is hepatoprotector?
1. Azithromycin
2. Papaverine
3. Ribavirine
4. Metionin

25. What medicines and procedures is undesirable to appoint patients with chronic hepatitis
1. Psychotropic means

2. Physical therapy procedures
3. Large infusion
4**. All listed**

LIVER CIRRHOSIS

1. Which hepatitis is not the cause cirrhosis of the liver?
1. Hepatitis B
2. Hepatitis A
3. Hepatitis C
4. Hepatitis D

2. In the development of hepatic encephalopathy leading role plays:
1. Ammonia
2. Creatinine
3. Hypoalbuminemia
4. Urea

3. Increased enzyme at cholestasis is:
1. GGT
2. Aspartate aminotransferase
3. Alkaline phosphatase
4. Lactate dehydrogenase

4. Jaundice in liver cirrhosis is:
1. Obstructive
2**. Cell-parenchymal**
3. Hemolytic
4. Nuclear

5. At cirrhosis in the blood there is increased concentration of:
1. Lactate dehydrogenase
2. Creatinekinase
3. Aspartate aminotransferase
3. Alkaline phosphatase
4. Amylase

6. Sign of cirrhosis is everything, except for:
1. Hepatomegaly
2. Severe pain in the right upper quadrant
3. Enlarged spleen
4. Ascites
5. Hepatic signs

7. The signs of portal hypertension are all listed, except for:
1. Jaundice
2. Ascites
3. Bloating
4. Varicose veins on the anterior abdominal wall.

8. The main complication of liver micronodular cirrhosis is:
1. Hepatic coma
2. Stroke
3. Bleeding from veins of the esophagus and stomach

4. Liver cancer
5. Acute heart failure

9. Teleangioectasia is:

1. The accumulation of cholesterol
2. Erythema
3. Subcutaneous arterio-venous shunts
4. Subcutaneous hemorrhage

10. Greenish skin tone liver cirrhosis depends on the accumulation in the skin:

1. Biliverdin
2. Bilirubin
3. Bile acidS
4. Melanin

11. Hormonal disorders in cirrhosis are all listed, except for:

1. Jaundice
2.Teleangioectasia
3. Palmar erythema
4. Gynecomastia

12. Xantoma is:

1. The accumulation of bilirubin in the skin
2. Intradermal deposits of cholesterol
3. Vascular formation
4. The accumulation under the skin triglycerides

13. Hypersplenism is not characterized by:

1. Thrombocytopenia
2. Anemia
3. Neutrophilic leukocytosis
4. Leukopenia

14. Early portal hypertension is characterized for:

1. Macronodulare cirrhosis
2. Biliary cirrhosis
3. Micronodulare cirrhosis
4. Wilson`s disease

15. Portal hypertension leads to dilatation all veins, except for:

1. Esophageal-gastric
2. Intercostal
3. Hemorrhoidal
4. Umbilical

16. The cause of hemorrhage syndrome in cirrhosis all listed, except for:

1. Thrombocytopenia
2. Varicose veins of the esophagus and stomach
3. Violation of synthesis of prothrombin
4. Violation of albumin synthesis

17. In total blood count in liver cirrhosis observed everything, except for:

1**. Eosinophilia**
2. Leukopenia
3. Anemia
4. Thrombocytopenia

5. Increased ESR

18. The highest level of conjugated bilirubin levels observed at:

1. Micronodulare cirrhosis
2. **Biliary cirrhosis**
3. Macronodulare cirrhosis
4. Cardiac cirrhosis

19. Sedimentation liver samples include all, except for:

1. Reberg`s
2. Timol
3. Sulema
4. Cadmium
5. Gold

20. Morphological signs of cirrhosis can detect at:

1. CT- scan
2. Ultrasound
3. X-ray
4. Percutaneous liver biopsy

21. For the detection of esophageal varices is the most effective method:

1. X-rays of the esophagus
2. Ultrasound
3. Radioisotope scans of liver
4. **FGDS**

22. The manifestations of decompensated cirrhosis are all, except for:

1. Severe jaundice
2. Insomnia
3. **Increased pain in the right upper quadrant**
4. Weight loss
5. Hepatic smell

23. Hepatoprotectors are all drugs, except for:

1. Kreon
2. Silibor
3. Hepabene
4. Essenciale

24. In the development of ascites play role all factors, except for:

1. Hypergammohlobulinemia
2. Hypoalbuminemia
3. Hyperaldosteronism
4. Portal hypertension

25. Which drug lowers blood pressure in the portal vein?

1. Prednisolone
2. Lactulose
3. Essentiale
4. **Propranolol**

CHRONIC PANCREATITIS

1. Which cells do not belong to the exocrine pancreatic tissue?

1. Parietal
2. Acinar
3. Mucin
4. Interstitial

2. Etiological factors biliary pancreatitis include all, except for:
1. Choledocholithiasis
2. The dysfunction of the lower esophageal sphincter
3. Oddi`s sphincter dysfunction
4. Organic pathology of major duodenal papilla

3. For development exocrine pancreatic insufficiency enzyme level must be:
1. 50% of normal
2. 30% - "-
3. 60% - "-
4. 90% - "-

4. What factor absent is a history of chronic pancreatitis?
1. Alcohol abuse
2. Acute pancreatitis in the past
3. Acute hepatitis A
4. Organic pathology of biliary tract

5. The most specific pancreatic enzyme is:
1. Amylase
2. Elastase-1
3. Lipase
4. Phosphatase

6. What are ultrasonographic signs of early stages of chronic pancreatitis?
1. Reduced mobility pancreas during movement of the diaphragm
2. Concretions in pancreatic strait
3. Inhomogeneous distribution echoes with alternating solid and cystic areas
4. The painting "Bridge" which echoes give medium intensity

8. What research you use to diagnose chronic pancreatitis at first?
1. Ultrasound
2. Endoscopic retrograde cholangiopancreatography
3. CT - scan
4. Magnetic resonance imaging

9. What drugs relief pain in chronic pancreatitis?
1. Atropine
2. Analgesics
3. Proton pump inhibitors
4. H2-blokers

10. Polyenzyme microspherical drugs do not provide:
1. Uniform mixing with food
2**. The presence of bile acids in preparations**
3. Simultaneous passage of food enzymes in duodenum
4. The presence of acid resistant membrane in preparations

11. Specify normal pH level of pancreatic juice
1. 1,0-1,2
2. 3,0-3,5

3. 7,5-9,0
4. 5,0-6,5

12. Etiological factors biliary pancreatitis don't include:
1. The chronic non-calculous cholecystitis
2. Chronic calculous cholecystitis
3. Biliary dyskinesia
4. Liver cysts

13. The syndrome of biliary hypertension does not include:
1. Portal hypertension
2. Jaundice
3. Cholangitis
4. Pathology large duodenal papilla

14. Clinical symptom are absent if significant swelling of the pancreas is:
1. Jaundice
2. Forced position on the back
3. Forced genucubital position
4. Voskresensky`s ymptom

15. Choose the drug for treatment of Oddi`s sphincter dysfunction in pancreatitis.
1. Atropine
2. Analgin
3. Mebeverine
4. Domperodone

16. Polyenzyme microspheric drugs provide:
1. The immediate release of enzymes in the upper small intestine
2. Slow release of enzymes in the upper small intestine
3. The rapid release of enzymes in the lower small intestine
4. Slow release of enzymes in the lower small intestine

17. Specify the normal daily volume of pancreatic juice
1. 0,2-0,4 l
2. 0,8-1,2 l
3. 1,5-2,0 l
4. 0,6-0,8 l

18. The syndrome of endocrine pancreas insufficiency in the early stages of chronic pancreatitis appears:
1. Hyperinsulinemia
2. Hypercorticism
3. Hypokorticizm
4. Hypoyinsulinemia

19. The symptom is not characteristic for chronic pancreatitis.
1. Voskresensky`s
2. Left phrenicus
3. Mendel`s
4. Kacha`s

20. The main functions of the pancreas does not include:
1. Neutralization of acid
2. The synthesis and secretion of digestive enzymes

3. The synthesis and secretion of hydrochloric acid

4. Synthesis of hormones that regulate carbohydrate metabolism

21. Physiological hypersecretion pancreas provides:

1. Select 2 liters per day secretions containing enzymes 10 times more than is necessary for adequate digestion

2. Isolation 1 liter per day secretions containing enzymes 10 times more than is necessary for adequate digestion

3. Bold 0.5 liters per day secretions containing enzymes 5 times more than necessary for adequate digestion

4. Bold 3 liters per day secretions containing enzymes 5 times more than necessary for adequate digestion

22. Choose drugs for replacement therapy of exocrine pancreatic insufficiency.

1. Polyenzyme drugs in tablets
2. Polyenzyme drugs content bile

3. Polyenzyme drugs with enteric shell

4. Polyenzyme drugs without enteric shell

23. What group of enzymes producing acinar tissue of the pancreas?

1. Proteolytic
2. Amylase
3. Lipase
4. Nuclease

5. All listed

24. What research is prescribed for suspected parapancreatitis?

1. FGDS
2. Endoscopic retrograde cholangiopancreatography
3. Ultrasound
4. CT scan
5. Laparoscopy

25. expectant management in chronic pancreatitis follow at:

1. asymptomatic pseudocyst diameter < 5 sm

2. symptomatic pseudocyst diameter> 5 sm
3. asymptomatic pseudocyst diameter> 10 sm
4. asymptomatic pseudocyst diameter> 15 sm
5. symptomatic pseudocyst diameter <10 sm