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Навчальний посібник призначений для підготовки до практичних занять студентів закладів вищої медичної освіти, клінічних ординаторів-іноземців за спеціальністю «Хірургія», які навчаються англійською мовою.

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INTRODUCTION

The most widespread in structure of urgent abdominal surgery are acute diseases of abdominal organs. In most cases, with acute pathology of the abdominal cavity organs, timely recognition of the acute pathological process, adequate and qualified medical care saves both the patient's health and life. All urgent situations in surgery require urgent decision-making regarding diagnosis and treatment. Therefore, for better studying and increase the level of self-training, the manual systematized modern views on the etiology and pathogenesis, treatment and diagnostic tactics and rehabilitation of patients with urgent surgical pathology of the abdominal cavity organs. The authors focus not only on the "classical" course of diseases, but also on their atypical, modern features.

The contents of the manual correspond to the program "Surgery" of the 4th year students of high medical educational institutions of Ukraine and allows you to use it for self-study during training for practical seminars of the content module "Urgent Abdominal Surgery". In addition, it will be useful for students of the 6th year and interns in the specialty "Surgery", students of the thematic improvement study "Actual issues of emergency abdominal surgery".

ACUTE APPENDICITIS

Definition. Acute appendicitis is a non-specific inflammatory process of the appendix, which occurs as a result of a number of factors: non-specific primary infection, changes in general and local reactivity, blood supply disorder due to the dysfunction of the neurohumoral apparatus of local or general origin. The term "appendicitis" refers to an inflammation of the appendix. It was proposed by the American doctor Fitz in 1886, although this disease was first described by Lauer in 1824.

Acute appendicitis is one of the most common acute surgical diseases of the abdominal cavity. The incidence is higher among women and mostly depends on the age. For children under the age of one the incidence rates 3.48 per 10 thousand inhabitants, for children aged between 1 and 14 it is 11.4, for people between the ages of 15 and 59 it is 114, 9, in the 60 to 69 age group it is 29.7, for 70 and more it is 15.8. Thus, with the average life expectancy of 60 years, every 12^{th} -15^{th} person will have a vermiform appendix removed till the end of their life.

Anatomical and physiological features. The existence of the vermiform appendix is known since the times of ancient Egypt. The first descriptions and sketches of a human appendage belong to Leonardo da Vinci (1472). The appendix (Processus vermiformis s. appendix) as an independent organ is formed when the embryo is 60-75mm long. The newborn's appendix is funnel-shaped with a broad base passing into the head of blind colon. It is finally formed in the tubular organ by 7-8 years of life.

The vermiform appendix branches from the back of the inner segment of the head of blind colon 2-3cm down from the ileocecal angle at the point of ascension of three teniae. However, it is possible that the appendix discharges not from the blind, but from the ascending colon and arcuate appendix, which at both ends opens into the lumen of the intestine.

The appendix's wall has five layers: 1) mucous, 2) submucosal with a large number of lymphoid follicles, 3) muscular, 4) subserous, 5) serous (peritoneum). Most often, the appendix is covered with peritoneum from all sides and has a mesentery. The muscular membrane at the base of the appendix forms a circular thickening – Robinson's sphincter. The mucous membrane at the mouth of the appendix forms 1-2 folds - the valve, or the Gerlach's valve. The cecum ends with the apex. There is a large number of lymphoid follicles in the submucous membrane of the appendix - up to 70-80 percm^2 for an adult.

Blood supply of the appendix is carried out by the appendicular artery (a. Apendicularis), which branches from a. ileocolica, and the last one goes from a. mesenterica superior. The appendicular artery is considered to be an artery of the finite type. Blood supply of the appendix has a clear segmental character, that is, prolonged spasm or blockage of one branch of the appendicular artery provokes an adequate zone of ischemia of the appendix's wall, which may also be the cause of the inflammatory process. There are four types of structure of the appendicular

artery: 1) one trunk, which supplies blood only to the appendix; 2) one or two trunks that supply 4/5 of the appendix; proximal 1/5 is supplied by the ileocecal artery; 3) one or two trunks that supply the bloodstream to the appendix and part of the wall of the cecum at the point of its inflow; 4) multiple anastomoses between one or two trunks that supply blood to the appendix and the adjacent part of the intestine. Excluding these types, ligation of the main trunk a.appendicularis can provoke ischemic necrosis of the cecum. Flood from the appendix is carried out on the self-titled vein v. appendicularis to system v. portae. This may cause the spread of the inflammatory process (pylephlebitis) on the latter reaching the liver (solitary or multiple abscesses) in case of complicated forms of acute appendicitis.

Lymphatic system. The main lymphatic collectors of the appendix (appendicular and ileocecal lymph nodes) are connected with the lymphatic systems of many other organs (the blind gut, the right kidney and around the colon cells, the stomach and duodenum, gall bladder, internal genital organs). This connection can lead to the spread of the pathological process in both directions.

Invasion of the appendix is carried out from the solar, upper limb and sacral plexus, which have both sympathetic and parasympathetic nerve fibers in their composition. As in other parts of the intestine, they form two major plexuses: muscular (Auerbach's) and submucosal (Meisner's). Unlike other parts of the intestine, the number of nerve cells per unit appendage is approximately three times greater. This may lead to the primary localization of pain in the epigastric region, the peculiarity of the spread of pain, the severity and display of dyspeptic syndrome, etc.

The physiological function of the appendix is not fully clarified. Most researchers think that this is a rudimentary part of body. It is considered, that the appendix secretes mucus that is stimulating the motor function of the intestine. Also, owing to the accumulation of lymphoid tissue, the appendix can serve as a barrier to infection. It is assumed that the appendix can be a source of B-lymphocytes.

Possible positions for the appendicular appendix and the colon. The appendix branches from the posterior-medial wall of the cecum, occupying a different position in relation to the cecum.

Most often (40-50%) the appendix branches from the intestine downward medially, sometimes it can fall into a small pelvis and reach the bladder, rectum, ovaries, fallopian tubes. An appendix can branch from the cecum in the medial and lateral directions, and also can be located on the anterior surface of the blind and ascending gut, reaching the lower surface of the liver and the bottom of the gall bladder. Often (in 9-15% of observations), the appendix is located behind the cecum (retrocecal) and even retroperitoneally, adjacent to the right ureter or kidney. Observation of the location of the appendix in the wall of the cecum (intramural), in the peritoneum (intraperitoneally) is described. It has its own mesentery, which

contains adipose tissue, vessels and nerves. Very rarely there are two worm-like appendages.

Due to the features of embryonic development, the appendix sometimes has a ligament (lig. apppendiculo-ovarien) that connects it with the appendages of the uterus. In this connection, lymphatic and blood vessels pass, which makes transition of pathological processes from one organ to another possible.

The cecum may be placed intraperitoneally (and sometimes it even has its own mesentery) and mesoperitoneally (with the back wall of the cecum not covered by the peritoneum).

The most frequent and most important in practical terms deviations from the normal position of the cecum are the following:

1. High, (hepatic) position.
2. Low, (pelvic) position.
3. The rarest positions: left-sided, in the middle of the abdomen, in the left hypochondrium, in the hernia sac etc.

Projection of the base of the appendix on the anterior abdominal wall:

McBurney's point is over the right side of the abdomen that is one-third of the distance from the anterior superior iliac spine to the umbilicus (navel).

Lance's point is situated on a line connecting the two anterior superior iliac spines one third of the distance from the right spine.

Kummel's point is located 2cm down and 2cm to the right of the navel.

Etiology and pathogenesis of acute appendicitis. Factors contributing to the inflammation of the appendix:

- peculiarities of the anatomical structure of the appendix ("blind" empty organ, overwhelming amount of lymphoid tissue, significant development of the neuro-receptor apparatus);
- presence of pathogenic and opportunistic microflora;
- damaged wall of the appendix (mechanical, chemical factors);
- neuro-humoral regulation disorder of the ileocecal region (poor diet, diseases of stomach and bile duct);
- diseases that reduce immunological resistance of the organism;
- sensitization (food allergy, helminth infestation, intoxication of different genesis).

The leading factor in the occurrence of acute appendicitis is the combination of pathogenic and opportunistic microflora. There are three ways of microorganisms' entry into the mucous membrane of the appendix: enterogenic, hematogenous and lymphogenous.

Inflammatory process in the appendix results from an increase in the pathogenicity of microorganisms in its lumen against the disorder of the protective barrier function of the appendix epithelium and reduction of the microorganism's resistance.

Approximately 1/3 of the cases of acute appendicitis is due to obstruction of the lumen of the appendix with fecal stones (coprolites), foreign bodies, helminthes

and others. Coprolites are found in the bodies of almost 40% of patients with simple appendicitis, and 65% of those with destructive appendicitis and 99% of those with perforated appendicitis. With obstruction of the proximal part of the appendix in its distal part, the secretion of mucus continues, which leads to a significant increase in intravitational pressure and circulatory disturbances in the wall of the appendix. As a result of circulatory disorders in the appendix, swelling of the wall occurs, resulting the closure of the orifice of the appendix. The contents that build up in it, stretch it, push on the wall of the appendix, disordering its trophism. As a result, the mucus loses its resistance to microbes that are always present in its lumen (coli, staphylococci, streptococci, enterococci, and other microbes). They penetrate into the wall of the appendix and inflammation occurs.

The topographic proximity of the appendix to the small intestine and pelvic organs often determines the acute appendicitis simulating their inflammation (adnexitis, cystitis, enteritis). It leads to the formation of delimited "side" infiltrates (abscesses) while having destructive appendicitis. Acute appendicitis in this position simulates enteritis, and while having destructive forms - creates favourable conditions for the occurrence of general peritonitis and intervertebral infiltrates (abscesses).

Thus, the inflammatory process in the appendix can develop in the following directions:

1. The inflammatory process either passes into a chronic appendicitis or does not shift to the destructive stage and regress without consequences.
2. The inflammatory process progresses with the formation of a destructive stage with the development of complications: a) appendicular infiltrate of different localization, which can regress with the transition to secondary chronic appendicitis or progress with the transition to appendicular abscess; b) appendicular abscess, which can be revealed in the free abdominal cavity (general purulent peritonitis) or into the intestine (self-cure); c) perforation of the appendix and the appearance of diffuse purulent peritonitis (or a delimited abscess); d) pylephlebitis (purulent thrombophlebitis of the portal vein), single or multiple abscesses of the liver or sepsis.

Pathomorphology of acute appendicitis. There are simple (superficial) and destructive (phlegmonous, primary gangrenous and secondary gangrenous) appendicitis, which are the morphological expression of the phases of acute inflammation, which end with necrosis.

In ***catarrhal*** (simple) appendicitis, changes occur primarily in the distal wart. There is stasis in capillaries and venules, edema and hemorrhage. After 1-2 hours, a suppurative inflammation of the mucous membrane with a defect of the epithelial lining (the primary Aschoff's affect) is formed.

In ***phlegmonous*** (destructive) appendicitis, the phlegmon of the appendix develops by the end of the day. The organ is enlarged, the wall is thickened, the serum is dull, swollen, reddish, full-blooded, with ulceration. On the surface there are layers of fibrin, and in the lumen - purulent, sometimes hemorrhagic fluid with

fecal odor. Peritoneum is swollen, hypersensitive and thickened. Transition of the process to the wall of the cecum is possible.

Gangrenous (destructive) appendicitis is divided into primary, when the process begins with thrombosis of the mesentery, and secondary, when necrosis is a consequence of the phlegmonous process progression. It may be focal, with a perforation, segmental or total. The appendix is flabby, thinned, dirty-grey, easily broken. In the lumen of the appendix there is a dirty-grey liquid with a sharp unpleasant odor. The mucous membrane is not differentiated.

Perforated appendicitis (complication of destructive forms) - the integrity of the wall is disordered as a result of purulent necrotic process. The perforation aperture is almost not visible (microperforation) but can be large sometimes. Fecal stones can fall into the free abdominal cavity through it.

Classification of acute appendicitis. According to ICD-10 appendicitis is classified into:

K35 - Acute appendicitis

K35.2 - Acute appendicitis with generalized peritonitis

K35.3 - Acute appendicitis with localized peritonitis

K35.8 - Acute appendicitis, other and unspecified

K38 - Other diseases of appendix

K38.8 - Other specified diseases of appendix

K38.9 - Disease of appendix, unspecified

Classification of acute appendicitis by V. S. Savelyev (1976):

A. Acute appendicitis:

1) Simple (catarrhal) appendicitis.
2) Destructive appendicitis:
   - phlegmonous
   - gangrenous;
   - perforated.
3) Complicated appendicitis:
   - appendicular infiltration;
   - appendicular abscess;
   - general purulent peritonitis;
   - pylephlebitis;
   - sepsis.

B. Chronic appendicitis:

1) Primary.
2) Residual
3) Recurrent

Classification of acute appendicitis by V. I. Kolesov (1972):

1) Appendicular colic.
2) Simple (superficial).

3) Destructive:
   - phlegmonous
   - gangrenous;
   - perforated.
4) Complicated:
   - appendicular infiltrate;
   - appendicular abscess;
   - general peritonitis;
   - others (pylephlebitis, sepsis).

Clinical picture and diagnostics. The clinical picture of acute appendicitis responds to the nature of morphological changes in the process, its location and the development of complications of the underlying disease.

The main symptoms of acute appendicitis. In the clinical course of acute appendicitis, four phases are distinguished: 1) epigastric; 2) local symptoms; 3) subsidence; 4) complications.

The disease begins with sudden abdominal pain. It is localized in the right club area, in most cases it has a constant character, it has a moderate intensity without irradiation, but may increase with progression or decrease with the remitting of the inflammatory process and its delineation. It's important to remember that in case of gangrenous appendicitis the intensity of the pain may decrease. The characteristic feature is the pain in the epigastric area, which then moves down, mainly to the right iliac region – a Koher-Volkovich's symptom. Localization of pain in the around-umbilical region (at the medial location of the appendix) is possible, as well as at the bottom of the abdomen (with pelvic placement of the appendix), in the lumbar region (with the retrocecal localization of the appendix). Nausea (more often) and vomiting (less commonly) are frequent, but not constant symptoms, which appear because of irritation of the peritoneum that covers the appendix. In case of destructive appendicitis, vomiting is more common. Fecal disorder is a volatile and ambiguous symptom. Delayed bowel movements are more often observed in case of destructive appendicitis (as a result of irritation of the peritoneum), and there is an accelerated liquid defecation, if the appendix is placed in pelvic station. Dysuric phenomena are noted if the wall of the bladder or ureter is involved in the inflammation process. The body temperature is heightened in most cases: in case of a simple appendicitis it does not go higher than 37.5 ° C, while having destructive forms it doesn't exceed 38 ° C. In case of a simple appendicitis, the general condition at the beginning of the disease is satisfactory, and of a destructive it gets worse rapidly, especially in case of gangrenous and perforative appendicitis. Cardiac acceleration indicates distinct intoxication which is a signature of a destructive appendicitis.

During the physical examination attention is paid to the following features:

- the tongue is predominantly wet, covered with fur, but in case of destructive forms it is dry;

- palpation of the right illiac area is painful, the intensity of pain depends on the degree of pathological-anatomical changes in the process;
- muscle tension - the symptom is impermanent, but weighty. The degree of muscle tension of the anterior abdominal wall depends on the degree of parietal peritoneum involvement in the inflammatory process;
- Shyotkin-Blumberg sign is a symptom of irritation of the peritoneum: after soft pressure on the abdominal wall the fingers are removed of the abdomen abruptly. In case of inflammation of the peritoneum, the pain is greater at the moment of hand removal rather than at the moment of pressure. When the appendix is in the retrocecal position, this symptom is unclear or absent at all.

The pain movement from the epigastric to the iliac region (Volkovich-Kocher sign), the muscle tension in the iliac region and the symptom of peritoneal irritation are the three most common and important symptoms of acute destructive appendicitis:

1) Rovsing’s sign - if palpation of the left lower quadrant of a person's abdomen increases the pain felt in the right lower quadrant, the patient is said to have a positive Rovsing's sign and may have appendicitis. The symptom is positive in 60-75% of cases.
2) Sitkovskiy’s sign - a patient, that lies on left, feels the pain which arises or increases in a right iliac area. The symptom is positive at almost 50%.
3) Voskresensky’s sign (slipping sign, a symptom of a "shirt") - the doctor is on the patient’s right side. They pull patient's undershirt downward with the left hand. The doctor puts second and forth fingertips of the right hand on the abdomen and during the inhalation makes rapid sliding movements. At the moment of these movements, the patient experiences severe pain in case of the inflammation of the peritoneum. Symptom is positive in 97% of cases.
4) Krymov’s sign ("inguinoscrotal") - if pain occurs when the index finger is inserted into the external buccal ring and while palpating posterior wall of the inguinal canal (transverse fascia with the peritoneum adjacent to it). This symptom is characteristic of the pelvic location of the appendix.
5) Rozdolsky's sign – pain occurs while doing percussion in the right iliac region.
6) Bartomier-Michelson's sign. During palpation, patient being examined lies on his/her left side, and the pain in the right iliac region increases as the appendix becomes more accessible for palpation.
7) Dumbadze’s sign – pain occurs while inserting the fingertip in the umbilical ring, the occurence or intensification of pain while palpating the right and the posterior vaginal vault during vaginal examination or the anterior wall of the rectum while examining men.
8) Cheremsky-Kushnirenko’s sign ("cough") - the appearance or strengthening of pain while coughing.
9) Obraztsov’s sign – palpation causes the increasing of pain in the right iliac region while raising the straightened right lower limb (indicates that the sacrum muscle (m. ileopsoas), to which the appendix adjoins, is involved in the inflammation process).

The presence, expression, character and localization of the presented subjective and objective clinical signs and symptoms depend on the clinical form of acute appendicitis, variants of the particular patient's position of the appendix and the individual characteristics of each patient. The diagnosis is established on the basis of a set of characteristic clinical signs.

Features of the clinical picture of atypical forms of acute appendicitis, depending on the location of the appendix.

***Retrocale location*.** In case of free placement of the appendix in the pockets of the peritoneum behind the intestine, the clinical picture is normal. If the appendix is "immured" by the commissures behind the intestine, the pain occurs acutely, it is moderate, localized in the usual place, often in the lumbar region, irradiated in the pelvis, in the right hip. Sometimes the pain is localized over the combs of the iliac bone closer to the spine (it is identified more clearly and depends on the degree of involvement in the inflammatory process of retroperitoneal fiber). In case of inflamed appendix, which is located retroperitoneally, there is significant hyperthermia, fever, severe intoxication.

Local symptoms: muscle tension, pain in the right iliac region, Schotkin-Blumberg's symptom is expressed weakly. With this localization, the following symptoms are determined:

- Obraztsov's;
- Yaure-Rozanov's - pain occurs when a finger is pressed in the area of the lumbar triangle (observed in case of retrocecular destructive appendicitis);
- Gabai's symptom - in the projection of the lumbar triangle, the finger is pressed on the right side, and then the hand is removed quickly (as with the symptom of Shchotkin-Blumberg). At the moment of the hand removal there is a sharp pain. If the pain in the lumbar triangle region intensifies when finger is removed after previous pressing, it indicates the involvement of the peritoneum in the inflammation of the posterior leaf of peritoneum.

The pelvic position is most common for women. The pain is localized over the puortal bundle, on the anterior wall of the rectum during its examination, and an accelerated painful urination is observed (if the inflammatory process passes over to the peritoneum that covers the organs of the small pelvis); diarrhoea (in case of the proliferation of the process on the rectum).

Medial position (between the intestinal loops) is a rare form of acute appendicitis. The clinical picture is explained by the early inclusion of the loops and erythema of the small intestine in the inflammatory process and is characterized by: rapid flow, the spread of pain throughout the abdomen, abdominal distension, muscle tension and a positive symptom of Shchotkin-Bljumberg.

Left-side appendicitis is due to either excessively moving and prolonged blind intestine or full or partial inverse position of organs and is characterized by the same clinical features as in the normal localization of the appendix, only on the left.

Peculiarities of the clinical picture of acute appendicitis for pregnant women. Acute appendicitis of pregnant women occurs in 0,7-1,2% of cases, that is

much more often than among other population. This is due to the presence of factors contributing to the inflammation process in the appendix: a shift of the intestine and the appendix upwards and to the front caused by the uterus, which gradually increases, resulting in overgrowth and stretching of the appendix, its abnormal emptying, also the blood supply of the appendix is disordered in the conditions of changes of anatomical relations between the organs. Significant role is played by the susceptibility to constipation during pregnancy, which leads to stagnation of the contents and increased virulence of the intestinal flora. Also the hormonal shifts which lead to a decrease in immunity are important. These factors often lead to a severe course of appendicitis, which ends with a destructive process, especially during the second half of pregnancy. Destructive appendicitis can lead to abortion and fetal death. Such complications occur in 4-6% of cases of the appendicitis during pregnancy.

Particular attention to the occurrence of appendicitis during pregnancy is paid due to the fact that a number of features that are specific to this disease (abdominal pain, vomiting, leukocytosis), is observed in the normal course of pregnancy, making it difficult to diagnose.

The clinical course of acute appendicitis in the first half of pregnancy is almost the same as its course in the nonpregnant state. Significant differences are common only for the appendicitis which complicates the second half of pregnancy. First of all, attention is drawn to the intensity of the pain syndrome which is usually identified by patients as the pain which often occurs in the second half of pregnancy. However, careful questioning will allow to state the beginning of the pain in the epigastric area and its gradual movement to the localization site of the appendix (a Koher-Volkovich sign). Vomiting is not critical because it is commonly observed during pregnancy. While examining the abdomen, it is necessary to take into account the fact that the localization of appendix changes, moving upward with growth of pregnancy period. Examination by touch should be performed while the pregnant woman is lying on her left side.

Thus, local pain during acute appendicitis in the second half of pregnancy is determined not in the iliac region, but much higher. Due to the stretching of the abdominal wall by the uterus, the localized muscle tension is expressed weakly. During late terms of pregnancy, when the cecum and its appendix remain behind the enlarged uterus, there may be negative symptoms of subactivity of the peritoneum: Shchotkin-Blumberg's, Voskresensky's and others. In this period, as a rule, Obraztsov's sign is well expressed and Sitkovskii's sign is expressed as well in some cases. And there will also be positive symptoms of:

- Brindeau - pain occurs in the right area of the club while pushing the left border of gravid uterus;
- Taranenko-Bogdanova – while the pregnant woman is lying on the left side, the pain decreases because pressure on the inflamed appendix diminishes, and when the patient lies on the right side - the pain intensifies;
- Michelson's - when the patient is on the right side the pain increases in the right part of abdomen, as the gravid uterus pushes the inflamed appendix.

The fever response is less expressed than in the nonpregnant state. In the blood analysis, leukocytes increase moderately, but it must be taken into account that leukocytosis up to $12x10^9$/L is a physiological phenomenon for pregnant women.

The differential diagnosis between acute appendicitis and right-side pyelitis in the second half of pregnancy has some difficulties, because the disease data has many similar features: uncertainty of the pain syndrome, high localization of the pain, vomiting. As for the dysuric phenomena, they can be observed in the case of acute appendicitis as well because of the appendix and the right kidney being close in the later stages of pregnancy.

Objective examination of abdomen shows the presence of high localized pain, but the symptoms of peritoneum inflammation may not appear because the hieroclical angle remains behind the enlarged uterus.

Only the beginning of the disease is significantly different: it is known that appendicitis always begins with pain, and then there is a marked increase in body temperature and vomiting, whereas during the pyelitis the disease begins most often with a sharp ague, vomiting, fever, and only then pain occurs. With careful objective research it is possible to notice that the maximum pain in the case of pyelitis during pregnancy is localized closer to the lumbar region, whereas during appendicitis it appears in the area of the lateral and anterior wall of the abdomen. Palpation of the abdomen while the patient is on the left side can help to orientate correctly - in this case, due to some uterine shift to the left, it is possible to palpate the area of the appendix and the right kidney more accurately. The analysis of urine is important (taken with a catheter), with which the presence of a pyuria (the main symptom of purulent pyelonephritis) can be detected. During an ultrasound examination, changes in the cystic-loch system of the right kidney are detected. However, if doubts remain, it’s better to operate the patient rather than provide a conservative treatment with the risk of appendicular peritonitis. For pregnant women, surgical tactics should be more active than for other categories of patients.

Features of the clinical picture of acute appendicitis for elderly and old people. Acute appendicitis occurs in the old and declining age somewhat less frequently than in youth. The number of elderly and aged patients is about 10% of the total number of patients with acute appendicitis.

In the old and declining age destructive forms of appendicitis dominate. A preconditions of destructive forms are not only the irradiation but also morphological changes of the appendix at this age: atrophy of the entire appendix, as well as its individual layers, obliteration of its lumen, reduction of the number of follicles and vessels in it, as well as the presence of various concomitant diseases that can significantly affect the symptoms and development of acute appendicitis. In this age so-called primary gangrenous appendicitis occurs, which develops immediately, passing the stage of catarrhal and phlegmonous inflammation, which is the main reason of postoperative complications and the cause of lethality.

Symptom complex of acute appendicitis for patients within this group is rarely erased. In most cases, it is not possible to indicate the beginning of the disease, often there is a "silence" period of symptoms on the background of general adynamia. One

of the most common symptoms of acute appendicitis is acute pain which is less pronounced and may be localized not in the right iliac region.

As a result of the physiological increase in the threshold of pain sensitivity, patients often do not pay attention to the epigastric phase of abdominal pain that occurs at the onset of the disease. Flabery of the abdominal wall and obesity mask the tension of its muscles. During aging, the tactile, temperature and pain sensation is dimmed. It reduces the diagnostic value of many symptoms during acute appendicitis.

Nausea and vomiting occur more often than in mature age, which is associated with the rapid development of destructive process. The acute symptoms of appendicitis are not clear in the old age, but the delay of emptying, dry tongue, general malaise can be observed almost always. The older the patient is, the more expressed these phenomena are. Although it should be noted that the delay in the act of defecation is not crucial, since in the old age there is a physiological tendency of intestinal emptying slowdown.

During the examination of the abdomen, only moderate pain in the right iliac region is revealed, even in the case of destructive forms of appendicitis. Due to age-related relaxation of the abdominal muscles, their tension is not expressed, but the Schotkin-Blumberg's sign is usually positive. Often, the positive symptoms of Voskresensky and Sitkovsky are determined.

In some cases, especially in the case of destructive forms of the disease, pronounced flatulence is observed as a result of paresis of the intestine. For 41% of the elderly patients body temperature is normal and even with destructive appendicitis it increases moderately. In the blood test, normal blood levels of white blood cells are observed, or insignificant increase in the range of 10 - $12x10^9$/L occurs, which usually appears late (after 2-3 days or more from the beginning of the disease), neutrophilic changes are not expressed.

In the old and declining age appendicular infiltration occurs more often than in mature age. Patients often pay attention to tumour-like formation in the right iliac region, which occurs a few days after the onset of non-intense pain, which makes particular attention paid to the differential diagnosis between appendicular infiltration and tumour of the intestine. The peculiarity of the course of acute appendicitis in the old age depends on the fact that the exact recognition of one or another clinical form of acute appendicitis before doing the operation is difficult. This suggests the need for active surgical tactics, especially since the risk of appendectomy in the old age is often exaggerated. When choosing the method of anesthesia local anesthesia is preferred, especially for patients with concomitant diseases of the respiratory and cardiovascular system.

Diagnosis of acute appendicitis. To clarify the diagnosis of "acute appendicitis" in clinical practice most often are used:

- general analysis of blood - the most characteristic change is: neutrophilic leukocytosis (in case of simple appendicitis, the number of leukocytes increases to $12x10^9$/L, with destructive - up to $15\text{-}18x10^9$/L); shift of leukocyte formula

to the left (more expressed in case of destructive forms); acceleration of the ESR;

- general urine analysis - urine in the early period of the disease is unchanged; With an increase in intoxication, proteinuria, erythrocyturia, cylinduria appear (granular).

In addition, in some cases you can use: an overview of the X-ray of the abdominal cavity, ultrasound of the abdominal cavity, laparosecenesis, laparoscopy, CT of the abdominal cavity.

During ***ultrasound examination***, the pathology of the appendix is determined by its thickening of up to 6mm and more, with the presence of a "gland" thickened echogenically and, often, by the presence of local accumulation of fluid in the right ileum fossa, in the retrovesical or Douglas space.

During perforation of the appendix free fluid in the abdominal cavity appears, sometimes it is possible to see a concrement in the neck of the appendix. However, it is often possible to detect the intestine-associated formation in the right ileum with accumulation of fluid. In such a situation it is impossible to differentiate acute appendicitis with other diseases of the intestine and the appendix: tumorous formations, tuberculosis, and others. A healthy appendix is not visualized in most cases. Informativity of ultrasonography is 85-98%, but its specificity is 92-100%. Advantage of the method is low-invasiveness, short-term examination, slight radiation load, wide possibilities of diagnosing other causes of abdominal pain, especially for women of childbearing age and for children. The main disadvantage of ultrasonographic research is that it completely depends on how it is interpreted by the performer.

The method of ***laparoscopic examination*** is important in the diagnosis of acute appendicitis. However, in some cases, laparoscopy cannot visualize the appendix, for example, with retrocecal and retroperitoneal placement. It is not always possible to correctly interpret the intraoperative picture. Carrying out laparoscopy may be difficult for patients who have previously undergone surgical intervention on the abdominal organs. In this regard, diagnostic laparoscopy is indicated only in cases where after the use of all available non-invasive methods of study the diagnosis of acute appendicitis remains questionable. However, laparoscopy is increasingly used for surgical treatment with clearly established diagnosis using other methods.

To assess patients with an atypical clinical picture of acute appendicitis, ***computer tomography (CT)*** of the abdominal cavity is currently the most important method of examination. This method helps to reduce the number of unreasonable appendectomies. The advantage of computer tomography is the greater informativity and accuracy compared with other methods of research, the possibility of rapid preparation of the equipment and the patient, non-invasiveness and the possibility of diagnosis of another pathology. The disadvantages of the method are significant radiation load, the possibility of anaphylactoid reaction with intravenous use of contrast medium.

A high-precision and cost-effective way of diagnosing acute appendicitis for adults is a limited ***spiral computer tomography with rectal contrast***. Further improvement of the technology of spiral computer tomography and its interpretation makes it a method of choice in the future. The use of intravenous and oral contrast increases the sensitivity of the computer tomography to 98%.

The main task of the surgeon is, first of all, to establish the correct diagnosis for patients with suspected acute appendicitis, to minimize cases of removal of the appendix without acute inflammation, while preventing an increase in the frequency of its perforation.

Differential diagnosis of acute appendicitis. In most cases, the diagnosis of acute appendicitis is not particularly difficult. Most often, acute appendicitis has to be differentiated with diseases of the:

- chest organs (basal pleuropneumonia, myocardial infarction, intercostal neuralgia);
- abdominal cavity (acute gastritis, stomach phlegmon, exacerbation of stomach ulcer and duodenal ulcer, perforated ulcer, acute cholecystitis, acute pancreatitis, acute intestinal obstruction, mesenteric blood vessel thrombosis, acute diverticulitis, acute diseases of the internal genital organs);
- retroperitoneal space (urolithiasis, pyelonephritis, sciatica, aortic dissection);
- infectious diseases (food poisoning, acute enterocolitis, epidemic hepatitis, shingles)
- and other diseases (diabetes, Henoch's disease, lead colic etc.).

Right-sided basal pleuropneumonia and basal pleurisy – during this pathology the reflex tension of the muscles in the upper parts of the anterior abdominal wall can be determined. Often, the need to differentiate occurs when the position of the appendix is high (hepatic). The diagnosis is based on the discovery of objective data of the pleuro-pulmonary pathology, radiological signs in the examination of the plan radiography of the chest organs.

Myocardial infarction, epigastric form - it is necessary to differentiate at the expressed epigastric phase of the course of acute appendicitis and its hepatic disposal. The presence of hemodynamic disorders, changes in auscultation of the heart, ECG, and echocardioscopy allow verifying the diagnosis.

Intercostal neuralgia on the right side sometimes can provoke abdominal pain, tension of the abdominal muscles, but dyspeptic syndrome is not characterized for it. Performing a paravertebral block, which reduces pain, allows to clarify the diagnosis.

Acute gastritis, food poisoning is necessary to differentiate at the expressed clinical picture in the epigastric phase of acute appendicitis, rarely – in case of the ascending position of the appendix. It is different from acute appendicitis in the colicky abdominal pains, the permanent localization of the process in the epigastric region, the dyspeptic syndrome (nausea, multiple vomiting with a temperature above 38^0C at the beginning of the disease, fever, diarrhoea) is dominant. Usually, patients indicate the poor quality food intake. During palpation of the abdomen there is no

ability to determine the place of the greatest pain accurately, there is no tension of the abdominal muscles and symptoms of peritoneal irritation. The normal amount of leukocytes is determined in the blood analysis. Clarification of the diagnosis is possible using epidemiological data, bacteriological examination, gastroscopy.

Phlegmon of the stomach is a rare disease. Differential diagnosis is based on the presence of the triad of signs characteristic of phlegmon of the stomach: intensive lancinating pain in the epigastric region and upper abdomen, repeated exhausting vomiting, high body temperature with fever, in combination with the rapid development of severe intoxication and septicemia. Clarification of the diagnosis is possible through gastroscopy and laparoscopy.

Exacerbation of gastric ulcer and duodenal ulcer, covered perforation - difficulties in differential diagnosis occur when the clinical picture is expressed in the epigastric phase of acute appendicitis, in case of ascending position of the appendix, with a of the primary epigastric localization of the pain similar to the Volkovich-Koher's sign with its subsequent disposition in the right iliac region in case of covered perforation of the ulcer (due to the movement of aggressive duodenal or gastric contents on the right lateral canal of the iliac region). Differentiation is based on the presence of a peptic anamnesis. Clarification of the diagnosis is possible on the basis of the results of gastroduodenoscopy, determination of the presence of free gas and fluid in the abdominal cavity, the disappearance of liver dullness during the percussion, dullness of percussion sound over the abdominal cavity, during rectal examination – overhang of the anterior wall of the rectum. Plain radiograph of the abdominal cavity in a direct projection shows a serous-like strip of free gas under the right dome of the diaphragm, ultrasound investigation and laparocentesis indicate the presence of gastric or duodenal contents in the abdominal cavity.

Acute cholecystitis - a similar clinical picture is possible both in the hepatic upward position of the appendix and in the low placement of the gall bladder. Differential diagnostics is based on the possibility of patient having cholelithiasis in the anamnesis, irradiation of pain in the right shoulder blade and the right shoulder and jaundice. Clarification of the diagnosis is possible based on the results of ultrasound (the size of the gall bladder, the thickness of its wall, the nature of the contents, changes in the liver and bile ducts).

Acute pancreatitis - a similar clinical picture is possible in case of the medial placement of the appendix and the expressed clinical picture in the epigastric phase of acute appendicitis. The refinement of the diagnosis is based on the results of urine and blood tests (hyperamylasuria, hyperamylasaemia), ultrasound examination and laparocentesis.

Acute intestinal obstruction (strangulative, obstructive, invagination) - reminds the clinical picture of a dyspeptic syndrome during acute appendicitis, appendicular infiltration. Clarification of the diagnosis is possible by conducting a survey radiograph (to indicate the presence of the Kloiber's bowls), ultrasound investigation, irrigoscopy, irrigography, and laparoscopy of the abdominal cavity organs.

Acute mesenteric thrombosis - refinement of the diagnosis is possible on the basis of the presence of micro-(rarely macro-) haematochezia, ultrasound investigation, laparoscopy and mesentericography results.

Acute diverticulitis and Crohn's disease - the clinic is almost identical to the acute appendicitis clinic. Non-specific inflammation of the terminal ileum and inflammation of the Meckel's diverticulum may give a clinical picture that is akin to acute appendicitis. Diagnosis is verified during surgery. If during a surgery changes in the appendix do not correspond to the severity of the clinical picture of the disease, the area of the ileum within a meter should be inspected, so as not to miss the Crohn's disease or the inflammation of the Meckel's diverticulum.

Acute diseases of the internal female genital organs (ovary apoplexy, abdominal pregnancy, ovarian cyst torsion, acute adnexitis, endometritis, pelvoperitonitis) - a clinic similar to a pelvic placement of the appendix. Differentiation is based on the analysis of obstetric and gynaecological history, the results of bimanual vaginal examination, puncture of the posterior arch of the vagina (blood, pus), ultrasound investigation of the internal genital organs.

Diseases of the urinary tract (renal colic, pyelonephritis) - a clinic is similar as if in case of retrocecal, especially retroperitonial (parurethermal) placement of the appendix. Differentiation is based on the presence of urolithiasis, micro- or macrohaematuria, pyuria in the anamnesis, ultrasound scan of the urinary tract and excretory urography.

Mesenteric lymphadenitis (inflammation of the lymph nodes of the mesentery) sometimes gives a clinical picture, similar to acute appendicitis. Observed among young children and young people. High body temperature is characteristic, signs of acute respiratory disease. Unlike acute appendicitis, palpation of the abdomen reveals pain in the course of attachment of the mesentery of the small intestine.

Treatment of acute appendicitis. Therapeutic tactic in the case of acute appendicitis is common. Emergency surgery is justified when the diagnosis of acute appendicitis is established, regardless of the form of acute appendicitis, the patient's age, the time elapsed from the onset or duration of pregnancy. Only in case of the presence of appendicular infiltration conservative-anticipatory tactics should be observed.

Preparing the patient for surgery:

- the patient empties the bladder (or urine is released by the catheter);
- the operating field is shaved (all stomach and pubis);
- premedication is performed (narcotic analgesics, antihistamines and atropine).

Anesthesia: general anesthesia or local anesthetic are needed. The volume of the operation is appendectomy (removal of the appendix). Access is typically oblique in the right iliac area for McBurney's incision through McBurney's point (the point on the boundary between the outer and middle third of imaginary line connecting right front-top outgrowth of the ilium and the navel) perpendicular to the

mentioned imaginary line so that one-third of a cut was higher, and two were below the line (mostly 8-10cm in length).

Vertical Lenander's incision and transverse Sprengel's incision can also be used.

There are two typical appendectomy methods:

- antegrade - is used in most cases with a mobile appendix and dome of the cecum. Initially, the appendix is mobilized by one-stage or step-by-step ligation of mesentery, then it is removed.
- retrograde - is applied when the appendix is fixed with joints and is not removed from the abdominal cavity. First, the appendix is cut at the base, its stump is treated, then the mesentery is bundled gradually.

There are also different ways of appendix stump treating:

A. peritonitization of the stump with the help of cystic and Z-shaped sutures (used with the unchanged wall of the cecum);
B. peritonitization of the stump using the separate nodal grey-serous sutures (in case of moderate inflammation of the wall of the cecum at the base of the appendix);
C. ligature (in case of the inflammatory infiltration of the wall of the cecum, mostly among children under 3 years of age) – appendix is tied with nylon ligature, stump is not ligated, and in some cases even extraperitonitization of the dome of the cecum is done. The operation is mostly ended with a layered sewing of an operational wound.

In the case of having local peritonitis with the large amount of serous or purulent fluid with colibacillar smell in the abdominal cavity, right iliac area and pelvic cavity is drained with vinyl chloride or silicone tubes, which are usually displayed through additional incisions.

Postoperative period. Bed regime for twenty-four hours (Fowler's position in bed in case of effusion in the abdominal cavity) is required. In the future (the next day after the operation), active getting up from the bed and motor regime, therapeutic physical training are needed.

1. Therapeutic gymnastics is prescribed in 6 hours after the surgery.
2. Ice bag is put on the wound for 2 hours.
3. Patient is fed in 10-12 hours after the operation: the first 1-2 days patient should drink water, tea, kefir. After restoration of the motor function of the intestine - rubbed vegetable soups, porridges, lean broth, mashed potatoes, cheese can be eaten with a gradual transition to a general diet till the end of the week;
4. To reduce pain during the first day, 1ml of 2% promedol solution is appointed in 6 hours, on the second day non-narcotic analgesics are prescribed.
5. If necessary, adequate parenteral antibacterial therapy is performed within 3-5 days;

6. In the presence of intoxication within 1-2 days, detoxification therapy is carried out;
7. In the absence of a self-sustained intestinal defecation micro-enema is appointed on the 3rd day.
8. Seams are removed on the 6th-8th day after surgery (uncomplicated cases).

Complications during surgery: bleeding from the vessels of mesentery and damage (mainly deserosation) of the wall of the small intestine.

Postoperative complications:

A. early: haemorrhage into the abdominal cavity (from the stump of the mesentery of the appendix); bleeding into the lumen of the large intestine (from the the mesentery of the appendix); insufficiency of the mesentery of the appendix with the development of unlimited (dispersed) peritonitis; post-appendicular inflammatory infiltration in the right iliac region; suppuration of postoperative wound; early conjunctival obstruction; embolism of the pulmonary artery.
B. late: conjunctival obstruction; ligaturic, intestinal fistulae; post-appendicular inflammatory infiltrates; postoperative ventral keel.

Complications of acute appendicitis. appendicular infiltration; general purulent peritonitis; pylephlebitis (septic thrombophlebitis of the portal system).

Appendicular infiltration. This is a conglomerate of interconnected organs and tissues around a destructively-altered appendix. The formation of infiltrate is a consequence of a protective response from the peritoneum formations that delineate the inflammatory process in the abdominal cavity. The basis of infiltration is often the modified appendix. The formation of infiltrate is the result of ill-timed treatment of a patient and / or diagnostic error at the prehospital stage.

A typical clinical picture of infiltration develops on the 3rd-5th day starting from the onset of the disease. By this term pain syndrome almost disappears, the patient's well-being improves, but the body temperature remains subfebrile. There are no muscle tension or peritoneal irritation symptoms. In the right iliac region dense nebolic formation of various sizes with limited mobility is palpated. There may be positive signs of Rovsing and Sitkovsky. Blood tests usually show moderate leukocytosis.

The diagnosis is based on anamnestic data and the presence of dense formation. The surgeon's tactics are conservative and expectant. With a smooth flow of infiltrate the surgery is not justified. Limited motor status is assigned, the right ilium should stay in the cold, a high-calorie diet is prescribed, with the exception of the food rich in fiber. Complex antibiotic therapy directed to the colon cartilage is assigned (preferably parenteral injection), as well as detoxification infusion therapy, stimulation of the body's protective forces.

After the disappearance of the pain syndrome, reduction of leucocytosis and normalization of temperature for accelerated resorption of infiltrate, physiotherapeutic treatment is appointed (UHF, thermal procedures). With the positive result of this treatment, appendicular infiltrate is gradually resolved (on

average in 1-2 weeks), during this period, the volume of conservative treatment is adequately reduced. After resorption of the infiltrate, the patient leaves the hospital with a recommendation to perform appendectomy in a planned manner in 2-3 months.

Abscess formation of infiltration. It manifests itself by the appearance and intensification of pain in the right iliac region, intoxication, an increase in leukocytosis, hyperthermia, which is hectic in nature, signs of irritation of the peritoneum over volumetric formation. When diagnosis of periapendicular abscess is stated, surgical intervention is justified. Under general anaesthesia, the incision is performed in the right iliac region, most often by the method of M.I. Pyrohov and the cavity of the abscess is opened with the extra-peritoneal access. At the same time pus is evacuated, the cavity is washed with antiseptic solution and 1-2 rubber drainages are set. Pus should be necessarily seeded to the microflora and its sensitivity to antibiotics should be determined. Through the additional puncture a drainage is put into the cavity for the introduction of antibiotics (the antibioticogram is taken into account).

Interloop abscesses. The abscesses are located between the loops of the small intestine, colon and omentum. There are primary and residual abscesses. Primary absesses are formed in the area of the primary centre of infection, and residual are between the loops of the intestine after previous diffuse peritonitis. Interloop abscesses are single and multiple; complicated and uncomplicated. Their frequency ranges from 1.8 to 5.8% of all cases of acute appendicitis.

Causes of interloop abscesses: suppuration of appendicular infiltrate, imperfect treatment of appendix stump (ligature method), insufficient rehabilitation of the abdominal cavity and its infection in the time of the appendix searching.

Complications of interloop abscesses: opening of the abscess to the free abdominal cavity, to the hollow organ, formation of intestinal fistulas, intestinal obstruction, pneumonia, exudative pleurisy, liver abscess and others.

Clinical picture is characterized by the above-mentioned features. Often, moderate paresis of the intestine is observed.

Surgical tactics: operative. If possible, abscesses are opened using the extra-peritoneal access (the treatment is the same as with an appendicular abscess).

Pyleflebitis (purulent thrombophlebitis of the portal system). Complications are noted occasionally. The inflammatory process begins in the veins of the appendix, extends to the mesentery veins, the outside and inside veins of the portal system with the formation of multiple abscesses in the liver.

The main symptoms of the clinical course of pylephlebitis: body temperature increases (septic fever), expressed adynamia, signs of intoxication, icteriosity of sclera and skin, pain in the right hypochondria with irradiation to the back, right collarbone and shoulder, hepatomegaly, right-sided reactive pleurisy, tachycardia up to 100-120 beats per minute. The blood analysis shows: neutrophilic leucocytosis with a shift of the leukocyte formula to the left, liver function disorder (hyperbilirubinemia, hyperfibrinogenaemia, etc.).

Treatment: antibiotic therapy (direct introduction of antibiotics in the portal system), anti-inflammatory, anticoagulant therapy, elimination of the infection centre, detection of liver pusoids.

Chronic appendicitis. Changes in the appendix during chronic appendicitis do not display the chronic inflammatory process. The disease in the vast majority of cases is the result of the changes that occurred during acute inflammation of the appendix. In chronic appendicitis during morphological examination, more or less common sclerotic (fibrous) changes in the various layers of the wall of the appendix are observed, as well as its merging with the surrounding organs, which leads to deformation and obliteration of the lumen of the appendix, violations of its motor-evacuation function.

ICD-10 classification:

K36 - Chronic appendicitis

Chronic appendicitis are:

1) Primary - pathological changes in the process develop gradually without signs of acute attack.
2) Secondary:
   A. residual (remanent) - pathological changes appear after an attack of acute appendicitis, appendicular infiltration, appendicular abscess;
   B. recurrent - repeated acute attacks occur.

Clinical picture of chronic appendicitis. Subjective signs are past acute appendicitis (not operated), appendicular infiltrate:

- pain in the right iliac region connected with food intake, physical activity of the patient; presence of unpleasant sensations in the right iliac region;
- unstable, moderate (or insignificant) signs of disorder of the passage of intestinal contents, intestinal motoric;
- absence of signs of inflammation.

Objective features:

- pain during deep palpation in the right iliac region (in the area of the appendix region);
- absence of local signs of inflammation and signs of irritation of the peritoneum;
- possible positive appendicular symptoms.

Characteristic symptoms for chronic appendicitis:

1) Volkovich's - atrophy and relaxation of muscles in the hypogastria on the right;
2) Ivanov's - an enlargement in the distance between the navel and the anterior-top awn of the iliac bone on the right;
3) Przhevalsky's – the right leg gets tired faster while raising both legs at an angle of 45 in lying a position.

Differential diagnosis of chronic appendicitis. Because of the absence of clinical signs of chronic appendicitis, differential diagnosis is carried out by the method of exclusion: It is necessary to exclude the presence of ulcer, gallstone desiases, urolithiasis, chronic pancreatitis, enterocolitis, diseases of female genital organs, organs of the retroperitoneum for patients with pain in the right ilium region with the help of additional instrumental methods of investigation. Clinical diagnosis of chronic appendicitis is carried out radiologically: irrigography, appendicography. Signs: deformation, narrowing of the lumen, disturbance of motor-evacuation function.

Treatment is a planned appendectomy**.**

Tumours of the appendix are rarely observed. During the histological examination of the removed appendixes, they are detected among 0,2-0,3% of patients. Benign neoplasms of the appendix are neuromas, myomas, lipomas, angios, fibromas and polyps (adenomatous and villous), malignant neoplasms are cancer, carcinoid and reticuloblastoma.

Benign and malignant tumours of the appendix are detected only during the histological examination of the removed appendix, since they do not provide any specific displays, but may contribute to the development of acute or chronic appendicitis.

The cancer of the appendix has the appearance of a polypoid tumour, sometimes with an ulcer. During histological studies adenocarcinoma is detected. Tumour metastases (in the liver, large omentum) are rarely observed. In case of the cancer of the appendix, the obturation of its lumen comes rapidly enough, which leads to stagnation of the content and development of acute appendicitis, which lead to the operation. In case of detecting a malignant tumour in the appendix removed due to the acute appendicitis, a reoperation is required - right-sided hemicolectomy. The prognosis is the same as in the case of caecal cancer.

Carcinoid of the appendix is the most carcinoid tumour of the gastrointestinal tract, which occurs in almost 55% of cases. The tumour derives from enterochromaffin cells which produce serotonin. The tumour is small in size (1-2cm), most often in the region of the apex of the appendix, it has a yellowish-gray colour in the sectional view. Carcinoid metastases are rare (in 3% of cases).

The main displays of the disease are connected with high levels of serotonin in the blood - cyanosis or reddening of the face, hot flushes, diarrhoea, asthmatic attacks (the so-called "carcinoid syndrome"). Fibrosis of the endocardium with the damaging of valves is often observed in case of carcinoid, which gives the appropriate clinical displays. In the diagnosis, the determination of blood serotonin level and urine hydroxyindoleacetic acid (a product of serotonin metabolism) level are of a great importance. Radical surgery is appendectomy.

The cysts of the appendix occur as a result of obturation of its lumen or obliteration in a limited area. This leads to a cluster of the secretion of the mucous membrane in the appendix lumen. In this case, a closed cavity is formed, which is filled with gelatinous content (mucocoele). A rupture of a cyst with the penetration of its contents in the abdominal cavity can lead to the development of

pseudomyxoma of the peritoneum. This disease is displayed by a cluster of a large number of gelatinous or mucous membranes in the abdominal cavity, which are formed in cells of the mucous membrane of the appendix, implanted on the surface of the peritoneum after rupture of mucocoele. There is a chronic granulomatous-cystic inflammation in the peritoneum. The disease proceeds hard and leads to a fatal outcome, that is why pseudomyxoma is referred to malignant processes.

Self-control questions

1) Anatomical and functional information about the appendix.
2) Atypical position of the appendix.
3) Etiology and pathogenesis of acute appendicitis.
4) Classification of acute appendicitis ..
5) Methods of examination of patients with acute appendicitis.
6) A typical clinical picture of acute appendicitis.
7) Atypical clinical picture of acute appendicitis.
8) Features of the clinical picture of acute appendicitis among the elderly.
9) Features of the clinical course of acute appendicitis among pregnant women.
10) Differential diagnosis of acute appendicitis.
11) Diagnostic program of examining the patients with suspected acute appendicitis.
12) Therapeutic program for patients with acute appendicitis.
13) Features of the clinical picture of acute appendicitis in the presence of comorbidity.
14) The nature of operant treatment in case of acute appendicitis.
15) Preoperative preparation of patients with acute appendicitis.
16) Postoperative management of patients with acute appendicitis.
17) Complications of acute appendicitis, their diagnosis and treatment.

Tests for self-control

1. In case of suspected acute appendicitis in the case of ambiguous results of instrumental study methods, it is necessary:
   a. to perform laparotomy in the right ilium region;
   b. continue to observe the patient;
   c. conduct an ultrasound study in dynamics;
   d. to perform laparoscopy;
   e. to perform mid-laparotomy.

2. With appendicular infiltration it is shown:
   a. laparoscopic surgery;
   b. conservative therapy;
   c. typical appendectomy;
   d. laparotomy;
   e. endoscopic intervention.

3. A 45 year old patient appealed to a doctor with complaints of pain in the right illiterate area. The pain arose in the epigastric area, three hours later, then moved to the right ilium, was a single vomiting. At inspection: body temperature 37,6 ° C, wet tongue, marked tension of the muscles of the anterior abdominal wall in the right iliac region. Positive Symptoms of Shchotkina-Blumberg, Roving, Sitkovskii. In the blood test, leukocytes are 11,0 x 1012 / l. General urine analysis without peculiarities. Formulate a preliminary diagnosis.
   a. right-side renal colic;
   b. acute appendicitis;
   c. acute cholecystitis;
   d. Crohn's disease;
   e. regional mesenteric lymphadenitis.

4. A 52-year-old patient is operated for acute appendicitis. The next day, she had flatulence, abdominal pain, and fever to 39 ° C. Symptoms of peritoneal irritation are not defined. Despite massive antibiotic therapy, the patient's condition progressively deteriorates, joints become attached. About the development of which complication need to think?
   a. subdiaphragmatic abscess;
   b. acute pancreatitis;
   c. pylephlebitis;
   d. gangrenous cholecystitis;
   e. poured peritonitis.

5. The patient, 43 years old, came to the reception office with complaints of pain in the right ilium, nausea, and one-time vomiting. About seven hours ago, she had a pain in the epigastric area, nausea. After a few hours, the pain moved to the right ilium and became less intense. At examination, the patient has a diagnosis: acute appendicitis. What symptom corresponds to such a course of the disease?
   a. Koher-Volkovich;
   b. Roving;
   c. Sitkowski;
   d. Barthomey-Mikhelson;
   e. Obraztsova;

6. A patient 55 years old, complains about pain in the right ilium region. I got sick about 7 hours ago, when there were pains in the epigastrium, and then they shifted to the right iliac region. The act of defecation was, feces of ordinary color, decorated. Urination is not affected. Body temperature 37.2 C. Make a preliminary diagnosis.
   a. acute intestinal obstruction;
   b. perforation of the stomach;
   c. acute pancreatitis;

d. acute cholecystitis;
e. acute appendicitis.

7. A patient 43 years old, complained about the pain that suddenly appeared in the right half of the abdomen, which gives to the inguinal and right lumbar dyelans. Frightened 3 hours ago. Previously, such a pain never existed. The pain was accompanied by one-time vomiting. Body temperature during inspection 37.5 ° C. Pulse 102 oz. for a minute, the tongue is wet, covered with white layers. The abdomen in the right half is painful, the Schotkine-Blumberg symptom is negative. The symptom of tapping on the right lumbar region is positive. Leukocytosis 14,0x109 / l. In the general urine analysis, protein traces, relative density 1018, fresh erythrocytes 8-10 in sight, and white blood cells 8-10 in sight. He was hospitalized in the urological department with a diagnosis of urolithiasis, right-sided renal colic. Three days after the onset of the disease there were positive symptoms of Obraztsov and Shchetkin-Blumberg. Make a preliminary diagnosis.
   a. acute appendicitis, with retroperitoneal arrangement of the appendix;
   b. Urolithiasis, right-sided renal colic;
   c. acute cholecystitis;
   d. thrombosis of mesenteric vessels;
   e. acute pancreatitis.

8. A patient of 63 years old entered the surgical department 4 days after the onset of the disease with complaints of moderate pain in the right iliac region, an increase in body temperature to 37.6 ° C. From anamnesis: 5 days ago there was an attack of pain in the right iliac region. At inspection: the tongue is moist, the stomach is involved in the act of breathing, soft. When palpation in the right iliac region is determined by a rounded form of formation. Make a preliminary diagnosis.
   a. omission of the right kidney;
   b. tumor of the cecum;
   c. appendicular infiltration;
   d. Right-sided inguinal hernia;
   e. swelling of the small intestine.

9. A patient 27 years old, fell ill 8 hours ago when there were pains in the lower abdomen, in connection with which he turned to the doctor and after inspection was hospitalized with the diagnosis: "Acute appendicitis. Ectopic pregnancy? "What is the most informative research for differential diagnosis?
   a. fibrogastroduodenoscopy;
   b. laparocentesis;
   c. laparotomy;
   d. puncture of the posterior arch of the vagina;
   e. irrigoscopy;

10. A patient 47 years old, was hospitalized with acute appendicitis, peritonitis. He is ill for 6 days, body temperature 38.5 ° C. Complains with pain in the lower abdomen. At examination: the tongue is dry, the pulse is 98 beats per minute, the abdomen is palpated sharply in the right iliac region and in the lower parts, where the positive symptom of Shchotkin is determined. What is the therapeutic tactic?:
    a. laparotomy;
    b. infusion detoxification therapy;
    c. laparocentesis;
    d. ultrasound of the abdominal cavity;
    e. appointment of anti-inflammatory therapy;

11. A 38 year old patient appealed to the hospital with complaints of pain in the lower abdomen. When examining a gynecologist and a surgeon in the patient can not exclude acute appendicitis. Established indicators for surgical intervention. During the operation, right side pyosalping and secondary appendicitis were found. What is the amount of surgical intervention?
    a. resection of the right appendages, appendectomy, drainage of the abdominal cavity;
    b. resection of the right uterine tube and the ovary, drainage of the abdominal cavity;
    c. appendectomy, drainage of the abdominal cavity;
    d. sanitation and drainage of the fallopian tube;
    e. resection of the uterine tube, appendectomy, drainage of the abdominal cavity.

ACUTE CHOLECYSTITIS

Definition. Acute cholecystitis is a non-specific inflammation of the gall bladder, which is characterized by varying degrees of inflammation of its wall.

By frequency, acute cholecystitis ranks third among all acute surgical diseases of the abdominal cavity. Often acute cholecystitis affects women over the age of 40 years. The male to female ratio is 1:3.

The incidence of acute cholecystitis in Ukraine and throughout the world is gradually increasing and reaches 6.3 per 10,000 population. In the structure of emergency surgical operations this pathology takes second place after appendicitis. Postoperative lethality with acute cholecystitis is 6-8%, and in elderly patients with destructive forms of the disease reaches 50%.

Anatomical and physiological features. The bile is produced in the lobes of the liver with subsequent gradual passage through the formed intra-liver ducts (segmental), the left and right hepatic duct, the general hepatic, and after connecting with the bubble duct - the common bile duct, which flows into the lower part of the duodenum.

The total bile duct is 7-8cm length, has diameter of 7-10mm and has 4 parts: supra- and gastroduodenal (are in the hepato-duodenal ligament), pancreatic and interstitial, which passes through the wall of the duodenum and forms in it. In the submucosal layer located a sphincter of Oddi that plays an important role in ensuring the rhythmic flow of bile to the duodenum.

The gall bladder is placed in the longitudinal fissure of the lower surface of the liver, 2/3 of it is covered by peritoneum, 1/3 - by the liver. The length of the gall bladder is 6-8cm, diameter 3-4cm. It consist of the bottom, body, neck with Hartmann's pocket and bladder duct. The volume of a bubble 40-70ml. The wall of the gall bladder consists of mucous, fibromuscular, subserous and serous membranes. In the wall of the bubble are the branches of the bladder veins and arteries, as well as lymph vessels and nerves. Near the cervix of the bubble are lymph nodes. The mucous membrane of the bubble has a mesh velvet appearance and is covered by a cylindrical epithelium, performing a suction function.

Cavities, which is formed through deep bursts of the mucous membrane of the bubble between weak and diffuse muscle fibers, sometimes reaches almost to the serous cover of the bladder (Lush walk). Together with the bile, they may get an infection that can stay there and support the inflammatory process.

The bladder duct is 3-7cm long, connected to the general liver, which goes into the general bile duct. In the bladder duct from the mucous membrane, spiral folds-valves of Geyster or Lyuksen sphincter are formed.

The supra-duodenal part of the common bile duct is surrounded with the portal vein and its hepatic artery in the hepatic-duodenal ligament.

From the right branch of the hepatic artery, the bubble artery most often departing from the bloodstream of the gall bladder. The space defined by the bladder artery, the bladder duct and the lateral edge of the common liver duct is called the triangle Kalo.

The size of the gall bladder, its shape, degree of peritoneum coverage, the relationship between the bladder duct and the bladder artery are very variable.

Lymphatic drainage occurs in the lymph nodes of the gates of the liver, paraaortic nodes and the chest duct.

The innervation of the gall bladder and extrahepatic bile duct is due to the branches of sympathetic and vagus nerves, as well as diaphragmatic nerve.

The function of the gall bladder. The bile consists of water (97%), bile salts (up to 2%), cholesterol, pigments, fatty acids, lecithin and other substances (1%). The most important function of the bile is activation of lipase and intestinal digestion, absorption from the intestines of vitamin K. During the day, the liver produces up to 800-1000ml of bile.

As a result of the action of food, gastric juice, fats on the mucous membrane of the duodenum, hormones - serotonin and cholecystokinin - are released into the blood, which in turn causes a reduction of the gall bladder and relaxation of the sphincter of sphincter of Oddi. After taking food, the alkaline environment of the duodenum promotes closure of the sphincter of Oddi (reflex Sklyarova).

Etiology and pathogenesis of acute cholecystitis. The appearance of acute cholecystitis is associated with the action of some etiological factors: mechanical, infectious, chemical, functional, endocrine and vascular.

Mechanical factors are causes that disturb the outflow of bile from the gall bladder. The level of blockage can be on any segment of the biliary tract distal to the neck of the gall bladder. The most common cause of blockage are concretions which located in the neck of the gall bladder, bladder duct, common bile duct, duodenal papilla. Among other reasons, the importance of blockage of biliary tract by parasites, inflammatory processes in the large duodenal papilla, duodenostasis, dyskinesia of the gall bladder.

The most common (85-90%) acute cholecystitis occurs as a consequence of cholelithiasis. Injury of the mucous membranes with concretions on the background of biliary hypertension leads to a decrease in the barrier function of the mucous membrane of the gall bladder.

In the majority of patients with acute inflammation of the gall bladder in the bile, virulent microflora appears. Most inflammation pathogens are enterococci, streptococci, staphylococci, coliform, which can enter the gall bladder in various ways: hematogenous, lymphogenous, ascending.

To acute cholecystitis, in the background of chemical factors include the so-called enzyme cholecystitis, which is due to the reflux of pancreatic juice in the gall bladder. Functional disorders of the gall bladder (dyskinesia) and hormonal disorders also lead to its atony, stasis of bile and the vulnerability of the mucous membrane to the pathogenic microflora.

Vascular factors are especially important in the development of acute non-stone cholecystitis in the elderly, whose occurrence is due to bladder artery thrombosis. Among other factors of acute cholecystitis, allergic, stressful reactions and heredity should be noted.

Often, the appearance of acute cholecystitis leads to a combination of factors, such as bile drainage, the presence of infection (in the background of bile hypertension), mechanical or chemical damage to the gall bladder mucosa, sensitization of the body.

Intravenous bile hypertension, which occurs as a consequence of the aforementioned etiological factors, leads to an overlapping wall of the gall bladder accompanied by pain syndrome, deterioration of blood supply to the mucous membrane of the gall bladder.

Decrease of hemoperfusion of the wall promotes the violation of the mucous barrier, penetration, and growth of microorganisms, inflammatory exudation in the lumen of the gall bladder. Under these conditions, the endogenous saprophytic infection becomes pathogenic and the serous inflammation becomes purulent. Through the crypt of Luchka and sinuses of Rokitinsky of the process extends to the muscular and serous layers. Purulent-inflammatory exudate accumulates in the gall bladder, which increases in volume and becomes tense. The gall bladder gradually acquires a dark-purple color. May form an empyema of the gall bladder, and with a slightly virulent infection, it becomes mucocele. With further development of the inflammatory process, phlegmonous changes can go into gangrenous form. The rate and severity of the development of the inflammatory process in the gall bladder depend on the vascular changes in its wall.

The proliferation of bile and purulent contents of the gall bladder through the wall leads to the development of biliary abscesses, biliary peritonitis, and necrotic changes in the wall - perforation.

Classification of acute cholecystitis. There are many classifications of acute cholecystitis. A rational classification makes it possible to correctly determine the optimal therapeutic tactics, the nature of the surgical intervention, postoperative period. The clinical and statistical classification of acute cholecystitis is presented in accordance with the standards and clinical protocols of medical care provision.

Clinical and Statistical Classification of Disease (ICD-10):

K80.0 - Acute calculous cholecystitis

K81.0 - Acute cholecystitis

Classification of acute cholecystitis (by Kondratenko P.O., 2005)

1. Depending on the presence or absence of gallstones concretions:
   - acute calculous cholecystitis;
   - acute non-stone (calculous) cholecystitis.
2. Form of ignition:
   - catarrhal;
   - destructive: phlegmonous, gangrenous.
3. Clinical course:
   - uncomplicated;
   - complications:
   - Biliary or purulent peritonitis;
   - Obturation of the neck of the gall bladder or the bladder duct;

- perivesical infiltrate;
- perivesical abscess;
- perforation of the wall of the gall bladder;
- septic cholangitis;
- abscess of the liver;
- acute pancreatitis;
- liver-renal insufficiency;
- internal bile duct;
- obstructive jaundice.

Clinic and diagnostics. The clinical picture of acute cholecystitis is varied, which depends on the pathomorphological form of gallstone inflammation, the prevalence of inflamed processes and associated changes in the bile duct, and the reactivity of the patient's body.

Depending on the severity of acute calculous cholecystitis, they distinguish between mild, moderate and severe disease.

Clinical manifestations of acute cholecystitis are characterized by pain, the presence of local symptoms and general disorders that are associated with the development of intoxication. Clinical picture, which is caused by inflammation in acute calculous and non calculous cholecystitis, has no fundamental differences.

The disease begins, in most cases, suddenly, with severe pain of cutting or bruising in the right hypochondrium and epigastric area with irradiation in the right shoulder and shoulder blade. The pain is long-lasting. At the beginning of the disease, it may resemble the hepatic colic but continues to be permanent. At the moment of increasing, pain patients become restless, moan.

At the height of the pain, nausea, and vomiting often occur, which does not bring relief. The emesis is represented by gastric contents, mucus, and bile. Patients experience bitterness and dryness in the mouth, biting with a bitter taste. Subsequently, bloating, delay in the discharge of gases and feces, which is more often in the destructive process and in the development of peritonitis, joins. There is an elevated body temperature from the subfebrile to 38-39 ° C, sometimes there are chills. The pulse is noticeably accelerated, the tongue is dry. In many cases you can notice the yellowing of sclera, skin, which may be the result of a blockade of choledochal, the development of hepatitis or pancreatitis.

During the examination, the lag in the right half of the chest and abdomen is noted. When palpation of the abdomen a significant pain in the right hypochondrium, it is often determined painful, tense and enlarged in the size gall bladder. With destructive forms of inflammation - the tension of the anterior abdominal wall.

The clinical picture of acute cholecystitis is characterized by the following symptoms:

- Ortner-Grekov's symptom - pain when the palm is tapped by the right edge arch;

- Murphy's symptom - increased pain and breakage of inhalation with deep palpation in the projection of the gall bladder;
- Mussy-Georgievsky's symptom - when you squeeze between the legs of the right sternoclavicular muscle, pain occurs (frenicus-symptom);
- Boas's symptom - pain when a finger is pressed to the right of the VIII-X vertebra on the back;
- Zakharin's symptom - pain when tapped or pushed on the gall bladder projection area;
- Kehr's symptom () - pain that occurs during inhalation with palpation of the right hypochondrium;
- Kehr's point - located at the intersection of the outer edge of the direct muscle of the abdomen and the edge arch - when pushed in it, the patient feels pain;
- Lyakhovitsky's symptom - the appearance of pain when it is easily pushed into a bovine sprout due to lymphangitis and an inflammatory reaction of the lymph nodes located behind the bovine appendix.
- Shelestyuk's test: on the inner surface of the forearm, 2ml of a 0.9% sodium chloride solution are injected intradermally and the papule resorption period is detected. The degree of dehydration, the time of resorption of the papule and the daily need for fluid are shown in the table.

The degree of manifestation of clinical manifestations of acute cholecystitis depends on its form and course of the disease. With uncomplicated forms, a mild virulent infection, the disappearance of inflammation factors (for example – a stone, a lump of mucus, etc. moved into the duodenum), the clinic of cholecystitis gradually disappears. This can happen spontaneously or under the influence of conservative treatment. In destructive forms of acute cholecystitis, the general condition of the patient is severe, expressed signs of intoxication, abruptly positive symptoms as described above. When pouring infected bile into the abdominal cavity or perforation of the gall bladder, peritoneal symptoms are determined.

In patients of the elderly and aged, inflammation in the wall of the gall bladder is often accompanied by non-fibrotic changes, which lead to the melting and perforation of the wall due to thrombosis of the bladder artery. In such persons, the disease may be accompanied by toxic encephalopathy and respiratory failure, as well as reflex angina (cholecystitis-cardiac syndrome) characterized by remitting pain that occurs after the pain in the right hypochondrium. The peculiarity of the course of acute cholecystitis among patients of the elderly is a large frequency of development of destructive forms of the disease and the discrepancy of the clinical picture with pathomorphological changes in the gall bladder.

Pregnant women have often aggressive course of acute cholecystitis with the development of destructive forms, which leads to complications of pregnancy, like abortions, premature births, and the like.

Complications of acute cholecystitis.

Dropsy of the gall bladder is a consequence of the obstruction of the bubble duct with calculus or scar of a fibro-sclerotic altered wall. When regressing the

inflammatory process, pigments of uninfected bile are absorbed through the wall of the bladder, and the bile that is in it becomes transparent and diluted with the exsudate. The patient is concerned about a minor pain in the right hypochondrium, palpated tense, moderately painful enlarged gall bladder.

With the infection of the gall bladder in the background of the blocked cystic duct, empyema of the gall bladder develops. With this pathology, the lumen of the gall bladder is filled with pus. Clinical picture becomes exacerbated, the temperature rises to 38-39 ºC, there is a fever. The gall bladder is enlarged in size, tight, painful. Positive symptoms of Ortner, Ker, Murphy.

Fissure abscess. As a consequence of phlegmonous inflammation in the wall of the gall bladder and the spread of infection in the circulatory space, an inflammatory infiltrates may be formed, in which organs and structures adjacent to the gall bladder are drawn. Most often it is a large cap, abdominal wall, duodenal or rumen gut, stomach. The patient experiences dull pain in the right hypochondrium, moderately positive symptoms characteristic of acute cholecystitis. Body temperature is 37-38 ºC.

In the case of the progression of the inflammatory process, infiltrate may become an abscess that is formed in patients with destructive forms of the disease. At a clinical examination a sharp deterioration of the general condition of the patient, an increase in body temperature to 38-39 ° C, pain and positive symptoms of peritoneal irritation in the region of the right hypochondrium is noted. Signs of intoxication are intensifying, there is a fever of a hectic nature. Often, jaundice is increased by intoxication genesis or by compression by the inflection (abscess) of choledochal. In the blood, high leukocytosis, the shift of the leukocyte formula to the left.

Untimely diagnosis of the adductor abscess can lead to extremely negative consequences - sepsis, purulent cholangitis, acute liver-kidney failure.

Cholangitis is a severe non-specific disease of the common bile duct, which is usually combined with cholecystitis or hepatitis. Isolated cholangitis is rare. Necessary conditions for the emergence of purulent cholangitis is the presence of infected bile and violation of its passage. The classic clinical picture is characterized by the triad Charcot: pain in the right hypochondrium, body temperature up to 39-40 ° C with fever, mechanical jaundice. In the severe course of purulent cholangitis, the following clinical symptoms are accompanied by arterial hypotension and abnormal consciousness (Reynolds Pentate), as well as the occurrence of cholangiogenic abscesses in the liver, which requires adequate timely treatment with the restoration of the bile passage through the biliary tract. With purulent cholangitis, sepsis may develop, toxic liver dystrophy, multiple organ failure syndromes.

Biliary peritonitis is a threatening complication, which occurs as a result of destructive changes in the wall of the gall bladder with phlegmonous or gangrenous cholecystitis and is divided into needle, perforative, limited and poured.

Perforation of the gall bladder occurs against the background of pronounced clinical signs of cholecystitis, often with gangrenous form. Often perforation occurs under the influence of the pressure of the concrete. The clinical picture depends on the zone of destruction of the wall, the rate of formation of the joint barrier and

infiltration with adjacent organs and the reactivity of the organism. In limited forms of peritonitis, the clinic corresponds to the one given with the biliary abscess.

The most threatening situation occurs when perforating in the free vein of the cavity - the spread of peritonitis develops, which is the greatest danger to the patient. With perforation of the wall of the gall bladder against the background of the clinic of acute cholecystitis suddenly there is a sharp abdominal pain, vomiting. The patient is pale, moans, has frequent pulse, hypotonia. Abdominal palpation is extremely painful and intense, mostly in the right half, and later in all areas. There are positive symptoms of peritoneal irritation.

The course of the propitious peritonitis is slower, the frequency of flutter peritonitis is much smaller, which is associated with a greater probability of formation of the joint barrier.

Biliary-intestinal fistula. Fistulae are pathological combinations of bile ducts with adjacent organs of the abdominal cavity. Through the perforation opening of the inflamed wall of the gall bladder, the contents and concrements of the bubble enter into their lumen, forming an internal biliodigestive bile duct: cholecyst-gastric, cholecyst-duodenal, cholecystitis-rim, and others. Migration of large concrement in the intestine can cause obstructive intestinal obstruction.

Perforation of the wall of the gall bladder in the choledochal, often with the localization of the stone in the Hartmann's pocket, leads to the appearance of a biliary-biliary fistula with the migration of the concrement into the supraduodenal part of the choledochal (Miritstsi syndrome), which may cause obstructive jaundice. Possible formation of biliary fistulas and other organs of the abdominal cavity and, even - the thoracic cavity.

Mechanical jaundice. The clinical picture in patients with acute cholecystitis complicated by mechanical (obstructive) jaundice consists of symptoms of the corresponding form of acute cholecystitis and signs of obstruction of the biliary tract.

Migration of small stones in the choledochal occurs through the expanded bladder duct, and their accumulation in the distal parts of the choledochal - before its obturation. Stones of large size may enter the choledochal in the formation of cholecyst-choledochal fistulae - Miritstsi's syndrome. The probability of the development of obstructive jaundice increases with the accompanying complication of acute cholecystitis with purulent cholangitis, in which the thickening of the wall of the choledochal, its swelling, accumulation in the lumen of manure, mucus, congestive bile is observed.

When choledochal is obturated, the symptoms are characterized by a stone, the sequence of which is characteristic and has diagnostic value: pain (liver colic), fever, jaundice, sclera, skin, enlargement of the liver, discolored (acholic) feces.

Laboratory and instrumental diagnostic methods. Laboratory specific tests do not exist to clarify the diagnosis of acute cholecystitis, but a comprehensive assessment of laboratory general-clinical and biochemical data provides important information for the establishment of this pathology.

In order to clarify the diagnosis of acute cholecystitis in clinical practice, general tests of blood, urine, biochemical blood tests (bilirubin, amylase, urea,

creatinine, AlT, AST), x-ray (examination of the radiograph of the abdominal cavity organs), ultrasound examination of the liver, biliary bubble, and bile duct, pancreas, laparoscopy.

General blood test. Observed neutrophilic leukocytosis, the degree of severity of which depends on the stage of the inflammatory process in the gall bladder, displacement of the leukocyte blood formula left to the appearance of even immature forms of granulocytes and toxic grains of neutrophils, lymphopenia, eosinopenia, monocytopenia, possible increase in the rate of erythrocyte sedimentation;

Urinalysis - moderate proteinuria, hematuria (more commonly eroded red blood cells), pyuria, cylinduria, as manifestations of intoxication and gipostenuria, as a manifestation of disturbance of the concentration of renal function, is characteristic of amylase in the process of absorption into the pancreas;

Biochemical blood tests - possible hyperbilirubinemia, mainly due to direct bilirubin, increased urea, creatinine, hyperamylasemia, a slight increase in enzymes AlT, AST.

The most common methods of instrumental diagnosis of acute cholecystitis are ultrasound and laparoscopy. Ultrasound is a non-invasive and at the same time the most informative method of diagnosis of acute cholecystitis, its course, the presence of complications and the effectiveness of conservative treatment. Ultrasound gives the most complete information to clarify the diagnosis with an accurate description of the size of the bladder, its wall, ducts, calculi, common bile duct and qualitative characteristic of the expression of inflammatory changes in the surrounding space.

The catarrhal cholecystitis has thickened of a wall of the bladder up to 6mm (normally 3-4mm), the phlegmonous - to 7-9mm with the presence of a hypoechoic rim with rounded cells, microabscesses, the gangrene cholecystitis - up to 10mm or more, a wall of multilayer, contours of the fuzzy. In the spread of inflammatory process beyond the gall bladder, the contours of it are not differentiated and merge with surrounding tissues, which indicates the formation of the infiltration of infiltration, and the definition in this area of hollow formations - the presence of the adductor abscess. In acute calculous cholecystitis, concrements are determined in the gall bladder, and with complications of obstructive jaundice, enlargement of the haematopoiesis and intrahepatic bile ducts.

Laparoscopy is use with the uncertainty of diagnosis in patients with high operational risk.

To *tomography* (CT, MRI) and laparoscopy are resorted to with difficulties in diagnosis and patients with high operational risk.

X-ray diffraction (radiography) is used for differential diagnosis of intestinal obstruction, perforated stomach ulcer or duodenal ulcer, acute inflammation of the lungs and pleura.

Differential diagnostics. Differential diagnostics of acute cholecystitis is most often performed with acute surgical diseases of the abdominal cavity (breakthrough ulcer, exacerbation of peptic ulcer, acute appendicitis, acute intestinal obstruction), right-sided basal pneumonia, myocardial infarction (cholecystitis-cardiac syndrome), complicated by urolithiasis. Decisive for clarification of the diagnosis is

an ultrasound of the liver and biliary tract, an overview of the abdominal cavity organs, chest x-ray, ECG.

The algorithm for differential diagnosis of acute cholecystitis with diseases that have the most common manifestations are given in the table.

Differential diagnosis of acute cholecystitis

№ п/п	Diseases with which to be differentiated	Definitive differential factors: data of anamnesis, physical examination, laboratory and instrumental research
1	Acute appendicitis	Anamnesis (the nature of the pain, the Coherera symptom), physical data (characteristic symptoms), changes in the blood test.
2	Perforated ulcer of the stomach and duodenum	Ulcer anamnesis, the nature of pain, overview R-copy (-graphic) of the abdominal cavity.
3	Acute pancreatitis	The nature of the pain is irradiation in the lumbar region and frequent vomiting, elevated diastase levels> 256 OD, blood lipase, ultrasound data.
4	Acute mechanical intestinal obstruction	The nature of the pain, abdominal examination data, overview R-copy (-graphic) of the abdominal cavity, ultrasound examination of the abdominal cavity.
5	Right-sided renal colic	Character, localization and irradiation of pain, changes in urine (leukocytes, red blood cells, protein), data of urography, ultrasound.
6	Right-sided pleurisy	Anamnesis, data of physical examination, review R-copy (R-graphy) of the chest.
7	Myocardial infarction	Anamnesis, ECG, troponin test.

Acute pancreatitis. Acute pancreatitis has pain that localized predominantly in the epigastric region of the abdomen, rarely in the left hypochondrium, becoming narcissistic, accompanied by multiple vomiting. The course is characterized by fast-growing symptoms of intoxication. Positive symptoms of Mayo-Robson, Resurrection, and Kerta. In urine - high level of diastase. When ultrasound - increased, increased echogenicity with blurred contours of the pancreas.

Aggravated peptic ulcer disease. Differential diagnostics is based on the nature and features of pain (moderate, inactive, "hungry"), the presence of a history of gastritis, absence of symptoms of intoxication. Decisive in the differential diagnosis is ultrasound data and fibro gastro duodenoscopy (FGDS).

Perforated ulcer of the stomach or duodenum. This pathology is characterized by the triad of Mondorus: a sudden "dagger" pain in the epigastric region, the tension in the anterior abdominal wall muscles and ulcerative anamnesis. Pain, as a rule, is not accompanied by nausea or vomiting. Unlike acute cholecystitis, the tension of the anterior abdominal wall from the very beginning acquires a sharp degree and is of a generalized nature. With percussion, there is no hepatic dullness - a positive symptom of Sklyarova. From the auxiliary methods of diagnostics, overview radiography of the abdominal cavity and FGDS is used.

Acute appendicitis. The difficulty in differential diagnosis often occurs in cases where the appendix is located under the top of the liver, or when the bottom of the enlarged gall bladder reaches the right iliac region and resembles appendicular infiltration. The differential diagnosis will help analyze the development of the disease, data ultrasound, in an uncertain situation - laparoscopy.

Acute intestinal obstruction. This pathology is characterized by cramping abdominal pain without clear localization, abdominal distension, enhanced, with a metallic tinge, peristalsis. Small-billed obstruction is followed by vomiting initially with bile admixture, and then thin-tissue contents. In case of obstruction of the distal intestinal tract, there may be asymmetry of the abdomen. A true diagnosis helps timely delivery of radiography of the abdomen and ultrasound examination.

Right lower left ventricular pneumonia. Patients with lower limb pneumonia or right-sided pleurisy often have complaints of pain in the right hypochondrium similar to acute cholecystitis. Analysis of the disease - complaints of a recent acute respiratory infection, cough, shortness of breath, sputum discharge, percussion data and auscultation of the lungs, will allow suspecting their inflammatory process. A true diagnosis is established in the lung radiograph.

Right-sided renal colic. Unbearable, cramping pain in the right half of the abdomen, restless and fussy behaviour of patients with the renal prickle reminds of the onset of acute calculous cholecystitis. But in this case, the pain begins in the lumbar region to the right, accompanied by pronounced dysuric disorders - frequent and painful urination. In differential diagnostics, ultrasound determines. Often, stone in the ureter can also be seen on the review radiography.

Myocardial infarction. Abdominal pain in the abdominal form of myocardial infarction is difficult to distinguish from pain in severe forms of acute cholecystitis in elderly patients with cardiac pathology - pain is given to the heart and the left half of the chest (cholecystocardial syndrome). A mistake in the diagnosis can have severe consequences. The main means of differentiation is electrocardiography.

Dyskinesia of the biliary tract. The clinic of dyskinesia of the biliary tract is marked by a variety of manifestations and the absence of individual pathognomy. In the hypertensive form of dyskinesia, the nature of the pain resembles biliary colic, but often the colic is not prolonged. The abdomen on palpation is soft, there is a variable pain in the right hypochondrium. The pain may disappear spontaneously, or after the introduction of antispasmodics. In the clinical analysis of blood, with the ultrasound of the gall bladder, there are no signs of inflammation.

Treatment of acute cholecystitis. Patients with acute cholecystitis should be hospitalized to the surgical department because the dynamics of the development of the inflammatory process in the gall bladder is unpredictable.

Therapeutic tactics for acute cholecystitis should take into account the form of the disease, the presence and nature of the complications, the age of the patient and the degree of violation of homeostasis.

When determining the indications for the operation, it must be remembered that the threat of the development of destructive and complicated forms of acute cholecystitis increases significantly if more than 3 days have elapsed since the

disease. In some cases, the process becomes threatening in the first day, in others - the clinical picture, quite often, does not correspond to the morphological changes in the gall bladder. This is especially true for the elderly.

A complication of cholecystitis with peritonitis is an indication for emergency surgery after a short-term, 2-3 hours preoperative preparation. In other cases, treatment of acute cholecystitis begins with conservative measures, while simultaneously monitoring data from clinical, laboratory and instrumental examinations in dynamics to determine the presence and nature of complications, concomitant pathology and treatment effectiveness.

Conservative therapy includes:

1. Functional rest for the liver and bile ducts - bed rest, local hypothermia (cold on the right of hypochondrium), hunger.
2. Anesthesia is the administration of non-narcotic analgesics, with acute pain - intramuscularly 2% solution promedol.
3. Removing spasm of the sphincter Oddi - the introduction of antispasmodics and blockade of the circular ligament of the liver.
4. Nonsteroidal anti-inflammatory drugs.
5. Antibiotic therapy: cefazolin 1-2 g / m 2 times a day, cefuroxime 750 mg / m 2 times per day;
   - in destructive forms: cefotoxim intramuscularly combined with 0.5% solution of metronidazole 100ml 2 times per day, pefloxacin 0.4 g and metronidazole or medetsef 1.0 intravenously + 0.5% solution of metronidazole -100ml intravenously. Other combinations of anti-inflammatory drugs are possible.
   - in destructive forms: cefotaxime (Loraxim) in combination with a 0.5% solution of metronidazole 100ml 2 times a day iv or pefloxacin 0.4 g and metronidazole or cefoperazone 1.0 + 0.5% solution of metronidazole - 100ml iv. Other combinations of anti-inflammatory drugs are possible.)
6. Correction of dehydration of electrolyte balance and detoxification. Correction of dehydration is carried out by solutions of electrolytes and solutions of glucose, taking into account the Shelestyuk test and under the control of central venous pressure (CVP).
7. Antihistamines.
8. Cardiovascular agents, vitamin therapy, in the presence of severe jaundice - vikasol 10 mg / m 2 times a day.

Degrees of dehydration	**Time for papule resorptioning**	**Amount needed liquid per day**
I	40-30 min.	50-80ml / kg per weight
II	30-15 min.	80-120ml / kg per weight
III	15-5 min.	120- 160ml / kg per weight

According to calculations, with the III degree of dehydration, an average of 70 kg of the patient's body weight is administered 8400-11200ml, but not more than 10-12% of the body weight.

In the absence of the effect of conservative treatment, surgical intervention is indicated. To solve further treatment tactics, it is necessary to evaluate the clinical picture in dynamics, changes in the general condition of the patient, indicators of laboratory research methods and data from ultrasound of the gal lbladder Indications for the operation and terms of performing surgical intervention.

Indications for surgery and the timing of surgery:

Emergency (urgent) operations are performed in patients with acute cholecystitis with complicated common peritonitis 3-4 hours after hospitalization and intensive short-term pre-operative preparation.

Early surgery - performed in patients within 10 days of the onset of the disease.

Postponed surgery - performed 10 days later and after the calm of the acute process in the gall bladder and improvement the general condition of the patient. During this period ,operations are the least dangerous.

The purpose of the surgery for acute cholecystitis is the elimination of the inflammation center in the abdominal cavity and hypertension in the bile ducts during their obstruction.

Methods of surgical treatment. In acute cholecystitis the surgery of choice is cholecystectomy. In patients with severe forms of concomitant pathology from the side of the heart, lungs, liver, kidneys and the presence the severe forms of diabetes - surgical interventions are performed only on vital signs (peritonitis).

In some cases, in extremely serious patients of the elderly with a clinic of acute cholecystitis, but without the peritonitis phenomena, it is expedient to perform decompression cholecystitis, as the first stage of surgical treatment. The essence of the surgery is the creation of an external fist. At the same time, the bottom of the gall bladder is applied to the edges of the wound so that it is isolated from the abdominal cavity and into the lumen of it is introduced a drainage tube.

Excessive punctural cholecystostomy could be considered as an alternative to cholecystectomy in the case of ineffectiveness of conservative therapy in patients with contraindications to emergency surgery-severe concomitant diseases.

Cholecystectomy is the removal of the gall bladder, that is, the removal of the inflammation cell. The surgery is performed under general anaesthesia by open laparoscopic or laparoscopic method taking into account the degree of operational risk. Under the existing capabilities (equipment, respectively, an operating team is prepared), it is expedient to perform laparoscopic cholecystectomy.

Laparoscopic cholecystectomy was first performed by French surgeons Muret and Dubois in 1987, using a multi trocar technique of surgery using laparoscopic equipment and CO2 insufflation to create pneumoperitoneum.

Laparoscopic cholecystectomy has a small percentage of complications and reduces the patient's hospital stay.

Early laparoscopic cholecystectomy is better than delayed in patients with acute calculous cholecystitis in the case when it is performed within 10 days from the onset of symptoms.

Laparoscopic cholecystectomy should not be offered to patients after 10 days from the onset of symptoms unless there is a deterioration in the patient's condition in the form of development of peritonitis or sepsis which requires emergency surgery. In patients with symptoms over 10 days, delayed cholecystectomy (after 45 days from the time of illness) is more appropriate.

A complex of special equipment is used to perform video laparoscopic cholecystectomy: an operational laparoscope with a video camera and a colour video monitor. After creating a pneumoperitoneum and introducing a laparoscope into the abdominal cavity, manipulator tools are introduced through separate punctures of the abdominal wall. With their help, the gall bladder is removed under visual control following the image on the monitor. To mobilize the gallbladder, two metal clips are placed on the cystic duct and artery at a distance of 5-6mm from each other and these structures intersect between them. Subsequently, the gallbladder is removed from its bed and by the manipulator through contraperture - from the abdominal cavity.

Compared with the open laparotomic method, the laparoscopic method has several advantages: minimal trauma and insignificant number of postoperative complications, good cosmetic effect, shortening the length of hospital stay and the period of complete rehabilitation.

Contraindications to Videopalaroscopic Cholecystectomy:

- acute myocardial infarction;
- acute cerebrovascular accident;
- uncorrected coagulopathy;
- widespread purulent peritonitis;
- dense infiltration in the zone of the neck of the gall bladder;

The need for the transition to open laparotomy (conversion) with acute cholecystitis occurs in 5-30% of cases.

Indications for conversion are:

1) impossibility to identify the elements of the triangle Kahlo and elements of the hepatic-duodenal ligament (infiltrate, sclerotic tissue changes);

2) Detection during the operation of previously unidentified tumors of the intestine, stomach, gall bladder;

3) the emergence of an operation complications that can not be eliminated - damage or threat of damage to the elements of the hepatic-duodenal ligaments (choledochal, portal vein, hepatic arteries);

Among the large number of surgical accesses for laparotomy cholecystectomy, the greatest advantage is given to upper-middle laparotomy, skew sections in the right hypochondrium (according to Kohher, according to Fedorov), or transrectal (behind the Mason).

Distinguish cholecystectomy from the neck (retrograde), from the bottom (anterograde) and combined (atypical).

There is cholecystectomy from the neck (retrograde), from the bottom (antegrade) and the combined atypical.

The retrograde method of cholecystectomy is the most rational and common - first, the cystic duct and cystic artery are ligated (by manipulating in the Kahlo triangle), intersected between ligatures, and then the gall bladder is removed from its bed.

Advantages of retrograde cholecystectomy:

1. the ability to remove the gall bladder to conduct cholangiography, manometry or sounding choledochal using the cousin of the cystic duct;
2. small calculi of the gallbladder during manipulations do not migrate to the common bile duct;
3. minor bleeding during surgery.

In the presence of infiltration or scar macroscopic joints in the area of the cervix of the gall bladder and hepatic-duodenal ligaments, an integrand cholecystectomy is performed, which is initially in the removal of the gall bladder from the bottom to the neck of the bed and the subsequent separation of the ligation and the intersection between the two ligatures of the bladder artery and the bladder straits Bleeding when applying such a technique is much greater, migration of small stones to choledochal is possible.

In some cases, with expressed tissue infiltration, edema and adhesions in the neck of the gall bladder, Kahlo triangle and the hepatoduodenal ligament, it is impossible to visualize the cystic artery and cystic duct, and it is dangerous to manipulate in this area - the threat of damage to the common bile duct, portal vein. In such a situation, the lumen of the gall bladder is opened longitudinally and all further manipulations are performed under the control of the index finger of the left hand introduced into the cavity of the gall bladder (atypical cholecystectomy).

With significant infiltrate in the area of the distal gallbladder, when the anatomical structure in the area of the Kahlo triangle is impossible to understand and damage to the common bile duct is possible, laparoscopic or open subtotal cholecystectomy can be considered an operation of choice.

During surgery (before the removal of the gall bladder it is necessary to conduct an intraoperative revision of the abdominal organs).

When conducting intraoperative visual and manual, and if necessary instrumental diagnosis, the final diagnosis and the nature of the surgical intervention are determined. Attention is drawn to the type, shape and degree of inflammation of the gallbladder, the presence of calculi in it, the ability to empty, adhesion to adjacent organs, the presence of infiltrate in the region of the Kahlo triangle, the state of the common bile duct (diameter, tension and infiltration of the wall, the presence of palpable calculi).

If the patient has signs of obstructive jaundice and calculi in the choledochal according to preoperative ultrasound, and during intraoperative revision, an expanded and intense bile duct is determined, it is necessary to use instrumental

methods for studying the biliary tract. An audit can be carried out using the stump of the cystic duct or through a choledochal incision in the supraduodenal section (choledochotomy).

The most common instrumental methods of intraoperative diagnosis of bile duct obstruction in acute cholecystitis are:

- intraoperative cholangiography - contrasting of the bile ducts by introducing water-soluble iodine-containing drugs into them through the stump of the cystic duct. Cholangiography makes it possible to determine the choledochal diameter, the presence or absence of calculi in it, - the phenomenon of “claws” (flowing around with the contrast of the bile calculus), as well as the cone-shaped narrowing of the terminal part of the choledochal (with a pancreatic head tumour) characteristic for the stenosis.
- cholangiomamometry – is the method, that allows using a water manometer (Waldman apparatus) to detect in the ducts the degree of biliary hypertension. Normal pressure is in the range of 80-120mm of water. Art. (0.78-1.17kPa), and higher - indicates biliary hypertension.

Bile duct sensing. Normally, a 4mm diameter probe freely passes into the duodenum through the large duodenal nipple. The probe is performed through the cough of the bladder duct (with sufficient diameter), or through the choledochotomy hole.

An indication for supra-duodenal choledochotomy is the presence of signs of obstructive jaundice, purulent cholangitis, and stones palpated in the lumen of the supra-duodenal part of the choledochal.

The incision of the front wall of the choledochal in the length of 1-1.5cm is done at a distance of about 5-6mm from the wall of the 12th-digestive gut. With the help of clamps, the Fogarty probe, stones (choledocholithotomy) and ointment bile are removed. Be convinced of the passage of bile ducts by sensing them in the distal and proximal directions and performing control cholangiography. Through the choledochotomy hole, bile duct washing is performed for the final removal of ointment bile and manure. With confidence in the passage of biliary tract, the operation ends with external drainage choledochal, which is temporary and is used for the rehabilitation of the duct system in the presence of cholangitis and for the purpose of decompression in bile duct hypertension to prevent insolvency sutures on the handsome duct.

External drainage of choledochal is carried out by drainage of various structures, most often - by Ker, Holsted-Pikowsky or Vyshnevsky.

According to the method proposed by Ker, the choledoch is drained by the T-shaped latex tube of the corresponding one, according to Vishnevsky - the vinyl chloride tube is installed in the choledoch in the direction of the portal of the liver, according to Holsted-Pikovsky - the drainage is introduced into the common bile duct through the stump of the cystic duct or through the choledochotomy opening in the direction of the duodenum guts.

Drainages are sealed in a choledochal or bubble coke with a catgut or synthetic self-extruding thread. Removal of drainages usually takes 12-14 days after surgery and conducting of control cholangiography. The external drainage of choledocha completes most of the operations performed in an urgent manner on the destructive forms of acute cholecystitis.

If it is impossible to eliminate the obstruction of the distal common bile duct during choledochotomy (wedged calculus in the large duodenal nipple, multiple calculi that cannot be completely removed, tumors of the large duodenal (Faterova) papilla, its strictures, tumors of the pancreatic head and other obstacles that cause a violation of the pancreatic head passage of bile of a constant nature), as an addition to choledocholithotomy, they perform internal drainage of the bile ducts - the application of choledochoduode noanastomosis, choledochojunoanastomosis, or transduodenal papillosphincteroplasty.

In the formation of choledochoduodenoanastomosis, the most famous methods are Finsterer, Flerkin, Juras. Their difference lies in the ratio of the direction of the section of the common bile duct and duodenum. It should be noted that with destructive forms of acute cholecystitis, the implementation of internal drainage of the biliary tract is most often delayed until the inflammatory process subsides in the postoperative period, and is performed as planned against the background of biliary tract debridement. The best method to eliminate obstruction of a benign nature (stones, strictures) in the distal choledochal during this period is endoscopic.

The operation for acute cholecystitis ends with drainage of the abdominal cavity with vinyl chloride drains (Blake drainage) and rubber semi-gloves, which is brought to the subhepatic space, and in the presence of peritonitis, to the subphrenic space and to the pelvis.

Complications of cholecystectomy are divided into suboperative and postoperative (early and late).

Suboperative complications are most often associated with damage to the elements of the hepatoduodenal ligament: ligation of the cystic duct with part of the common bile duct, iatrogenic injuries of the hepatic ducts (dissection, perforation), damage and bleeding from the portal vein or hepatic artery (most often the right branch). With a pronounced adhesive process in the area of the subhepatic space, with the isolation of the neck of the gallbladder, damage to the wall of the hollow organs - the duodenum or colon, stomach, is possible. With destructive forms of cholecystitis after removal of the gallbladder from its bed, especially with severe jaundice of mechanical origin, significant bleeding of a parenchymal nature is possible, which requires careful electrocoagulation or suturing of the bed for hemostasis.

In the early postoperative period, early postoperative complications are possible: bleeding (from the bed of the removed gallbladder, sliding off the ligature from the cystic artery), bile secretion through the drainage to the outside or into the free abdominal cavity (sliding of the ligature from the cystic duct, bile secretion through the additional duct of the non-diagnosed gallbladder) damage to the hepatic ducts during surgery). With minor bleeding and prolonged bile secretion through the

drainage, observation, conservative treatment, laboratory and ultrasound monitoring are indicated. With severe bleeding, bile secretion, especially in the free abdominal cavity, with the development of signs of peritonitis - emergency relaparotomy and the elimination of identified defects.

Management of the postoperative period in the absence of early postoperative complications coincides with the nature of conservative treatment and preoperative preparation.

Self-control questions

1. Anatomical and functional information about the gall bladder and extrahepatic bile ducts.
2. Etiology and pathogenesis of acute cholecystitis.
3. Classification of acute cholecystitis.
4. Methods of examination of patients with acute cholecystitis.
5. Typical clinical picture of acute cholecystitis, its features in persons of young and old age.
6. Differential diagnosis of acute cholecystitis.
7. Diagnostic program of examination of patients with suspected acute cholecystitis.
8. The treatment program for patients with acute cholecystitis.
9. Features of the clinical picture of acute cholecystitis in the presence of concomitant pathology.
10. Indications for emergency operations with acute cholecystitis.
11. The nature of surgical interventions with acute cholecystitis.
12. Indications for minimally invasive surgical interventions (endoscopic, laparoscopic, CT and ultrasound).
13. Preoperative preparation of patients with acute cholecystitis.
14. Postoperative management of patients with acute cholecystitis.

Tests for self-control

1. For acute destructive cholecystitis is characteristic:
   a. basophilia;
   b. leukopenia;
   c. eosinophilia;
   d. leukocytosis with a shift of the leukocyte formula to the left;
   e. positive response to the Australian antigen.

2. Duodenal sounding with acute cholecystitis is performed at the patient's position on:
   a. back
   b. left side;
   c. sitting;
   d. right side;
   e. not conducted.

3. For the clinical picture of acute cholangitis is characterized by:
   a. nausea, vomiting;
   b. high fever, pain in the right hypochondrium, jaundice;
   c. ground;
   d. unstable liquid feces;
   e. girth pain in the epigastrium.

4. Participation of the anterior abdominal wall in the act of respiration with acute cholecystitis:
   a. limited in the region of the right hypochondrium;
   b. limited in the navel area;
   c. limited in mesogastric region;
   d. limited in the hypogastric region;
   e. limited in the area of the left hypochondrium.

5. In acute cholecystitis, the most commonly occurring operation is:
   a. Choledochotomy;
   b. cholecystectomy and choledochotomy;
   c. cholecystectomy;
   d. cholecystostomy;
   e. cholecystodiuodenostomy.

6. The patient, 38 years old, complains about pain in the right hypochondrium, nausea, vomiting. The skin is clean. Pulse – 88 beats per 1 minute, AT - 120/70mm Hg. Art. Abdominal limb takes part in the act of breathing, painful and tense in the right hypochondrium. When you touch the edge of the palm on the edge of the arch, pain is noted. Positive symptom of Shchetkina-Blumberg. What disease do these clinical symptoms correspond to?
   a. perforated ulcer of the duodenum;
   b. acute cholecystitis;
   c. acute intestinal obstruction;
   d. acute appendicitis;
   e. acute pancreatitis.

7. The patient 62 years old, complains about pain in the right half of the abdomen. She became ill two days ago, after a diet violation. On examination: skin of normal color, sclera icteric. The abdomen is moderately swollen, with palpation pain and muscle tension in the right hypochondrium and iliac areas are noted. There are also identified positive symptoms of peritoneal irritation. Positive symptoms of Ortner, Mussi. What research should be performed first of all for diagnosis?
   a. retrograde cholangiopancreatography;
   b. fibrogastroscopy;
   c. X-ray examination of the abdominal cavity;
   d. ultrasonography of the gall bladder and biliary tract;

e. instrumental research methods not shown.

8. The patient is 50 years old, complains about pain in the right hypochondrium, nausea, vomiting, which arose after taking fatty food. From the anamnesis it is known that such an attack is not the first time. At inspection: the stomach is tense, sharply painful in the right hypochondrium. There is a suspiction of acute cholecystitis. What treatmment tactics should be choosen?
   a. home treatment;
   b. send a sick person to the surgeon in the clinic;
   c. urgent hospitalization to a therapeutic hospital;
   d. outpatient observation until the next attack of pain;
   e. urgent hospitalization to a surgical hospital.

9. Choose the most informative survey to identify the cause of obstructive jaundice in acute cholecystitis:
   a. X-ray examination of the abdominal cavity;
   b. biochemical blood test;
   c. computer tomography;
   d. ultrasound;
   e. intravenous cholangiography.

10. The patient, 68 years old, suffers from gallstone disease for a long time, suddenly fell ill 7 days ago. The pain appeared in the right hypochondrium, there was nausea, vomiting. It was treated independently, conservatively. She noted some improvement, but the day before hospitalization, her condition suddenly worsened. The tongue is dry. The pain spreads down the right side of the abdomen, bloating appeared, and the gases stopped flowing. With percussion, blunting in the sloping places of the abdomen, a positive symptom of Shchetkin-Blumberg, with auscultation, there is no peristalsis. White blood cells 16.7 × 109 / L, shift of the white blood cell to the left. What is the most likely diagnosis?
   a. Perforated duodenal ucler;
   b. hepatic colic;
   c. peritonitis of biliary origin;
   d. pancreatic necrosis;
   e. thrombosis of mesenteric vessels.

11. The patient, 40 years ,old complains of acute pain in the right hypochondrium, vomiting, fever up to 38.5 0 C. He became ill 3 days ago. He has been suffering from gallstone disease for 2 years. Objectively: in the right hypochondrium a tumor-shaped formation of a thickly-lactic consistency is determined, moderately painful, symptoms of irritation of the peritoneum are not detected, percussion - dullness. AT 130/90mm Hg. Art. Pulse - 86 az. in 1 minute. Formulate a preliminary diagnosis.
   a. peri-bubble inflammatory infiltrate;

b. echinococcus liver;
c. liver hemangioma;
d. tumour of the transverse colon;
e. hydronephrosis of the right kidney.

ACUTE PANCREATITIS

Definition. Acute pancreatitis is a disease in which the exocrine part of the pancreas is most often damaged due to activation of pancreatic enzymes with parenchyma autolysis and development of specific local changes and the general reaction of the body to endotoxins of vasoactive substances.

Among all acute diseases of the abdominal cavity, acute pancreatitis ranks third after acute appendicitis and acute cholecystitis and is 9-11%. The morbidity rate of acute pancreatitis in different countries varies from 4.9 to 73.4 cases per 100 thousand people. The incidence of this pathology in Ukraine is 67-69.5 cases per 100,000 population (from 28 to 124 in different regions). In recent years there has been an upward trend in the incidence of this disease. This disease occurs at the age of 30-50 years. Women fall ill twice as often as men. The total mortality rate for acute pancreatitis ranges from 4% to 15%, while for the necrotic form it is 24-60%, postoperative mortality reaches 70%. Therefore, the most important problem of surgical gastroenterology is diagnostics and treatment of acute pancreatitis.

Anatomy-physiological features. Pancreas (rapeseed) - an unpaired glandular organ located in retroperitoneal space at the level of I-II lumbar vertebrae. The gland is elongated as a draft 15-25cm long, 3-9cm wide and 2-3cm thick, with an average weight of 80-100g. It has four anatomical sections: head, isthmus, body and tail. In the head of the organ, which is the largest glandular section, there is a distinction between the upper part, the "upper pole", which borders on the upper-horizontal branch of the duodenum, the middle part, which is mainly behind the intestine, and the lower part, which is the largest part. The body of the pancreas adjoins the anterior surface of the stomach. These organs are separated from each other by a narrow slot, the glandular bag. The tail reaches the spleen gate. The spleen corner of the colon adjoins it from the front. The tail of the gland is sometimes covered with peritoneum from all sides, which is sometimes associated with a well-defined lig. pancreaticolienale.

Of particular importance is the study of the anatomical features of the left half of the pancreas, as cystic formations and fistulas are most often located here. And this very half is the object of surgical intervention at the resection of the gland and hemipancreatectomy.

Blood supply to the pancreas comes from three sources:

1. gastroduodenalis, which comes from a. hepatica
2. pancreatoduodenalis inferior
3. lienalis, which runs along the upper edge of the gland in its own checked case and supplies blood mainly to the body and tail of the pancreas

The veins pass along with the arteries. Blood from the body and tail flows through small pancreatic veins through the splenic vein into the gate. Blood flows from the head through the upper pancreatoduodenal vein, which flows into the final part of the splenic vein, and the lower pancreatoduodenal vein, which flows into the

upper mesenteric vein. Blood flows from the splenic vein and the upper mesenteric vein into the portal vein.

On its way, lymphatic vessels of the pancreas accompany the blood vessels and exit ducts, forming a thick mesh, widely anastomizing with lymphatic vessels of the gallbladder and common bile duct. Lymphatic vessels of the pancreas flow into pancreatic, pancreatoduodenal, pyloric and lumbar lymph nodes. Regional nodes of the first stage are the mainly anterior and posterior pancreas and nodes lying in the region of the tail of the gland along the splenic artery. Regional nodes of the second stage are abdominal nodes. Besides, lymph flow to the adrenal glands, liver, stomach and spleen.

Pancreas innervation is performed by branches of vagus nerves and five plexuses (solar, hepatic, splenic, upper mesenteric and left renal). Pancreas innervation is performed mainly by the left vagus nerve and post-ganglion fibres of the left abdominal nerves. It is believed that all the nerves of the pancreas are mixed - cute and parasympathetic.

Duct system. Inside the gland passes the main pancreatic duct (ductus pancreaticus Wirsungi), which is formed by the fusion of lobular ducts and passes in the thickness of the organ from the tail to the head, closer to the rear surface of the organ. The strait length is 18-20cm and its diameter in the head area is 3-4mm and 1-2mm in the tail and body area. The main duct in the pancreas head connects with the additional duct (ductus Santorini) and then flows into the common bile duct, forming 2cm above the large duodenal papilla of the duodenal papilla. The femoral papilla ampulla as well as the intramural sections of the common bile duct and pancreas duct, surrounded by smooth muscle fibres that form the Oddi sphincter, regulate the portion of bile and pancreatic juice into the duodenum.

Pressure in the Virsung duct reaches 300- 500mm of water (i.e., 100-200mm higher than in glandular slices and 40-60mm higher than in the common bile duct). The resistance of the Oddi sphincter is 250mm of water column (i.e. 100-200mm higher than in glandular slices, and 40-60mm higher than in the general bile duct).

The pancreas is one of the most important glands of the digestive tract. It has external and internal secretion and has an impact not only on digestion but also on other aspects of metabolism.

According to the histology structure of the pancreas is classified as highly differentiated alveolar glands with a lobular structure.

The exocrine part is a complex alveolar-tubular gland, which is divided into slices by very thin connective tissue septum, departing from the capsule. The slices contain acinuses of 100-150 microns in size, formed by one layer of pyramidal-shaped acinocytes in the amount of 10-12. In the centre of the acinus, there are central acynosis epithelial cells, which form the wall of the insertion strait with which an acinus is a structural-functional unit of the exocrine part of the pancreas. The secret enters the lumen of the acinus through the apical surface of the cell (merocrine secretion).

With the help of two pancreatic ducts: the main one - ductus pancreaticus (Wirsungi), running along with the pancreas, along its axis from the tail to the head I additional - d. pancreas accessorius (Santorini) carries out its external secretion.

The external secretion is the secretion of pancreatic juice into the duodenum, which plays an important role in the digestive process. Pancreatic secretion is secreted in slices of parenchyma in the amount of 30-50ml / h, which is 1.5-2 litres per day. Pancreatic juice is a colourless transparent liquid with a specific weight of 1015, contains 1.3% solids of organic and inorganic origin, has an alkaline character (pH 8.3-8.9) and a clear ratio of anions and cations.

Its excretion is stimulated and regulated by the neurohumoral way:

1. by the vagus and sympathetic nerves
2. humoral:
    a. gastric acid
    b. chemical mediators - secretin and pancreosymine, which are produced in the intestines. The mechanism of pancreatic double secretion - nervous and humoral, operate simultaneously and synergistically.

There are three phases of pancreatic secretion: **cerebral, gastric and intestinal.**

The cerebral (first) phase has a complex reflex mechanism, which is realized through the central nervous system using conditional and unconditional reflexes. The phase is stimulated by the sight, taste or smell of food through the cholinergic innervation of the vagus nerve. The secretion in this phase is small (up to 15% of the stimulated secretion). Pancreatic juice is secreted and contains a large number of enzymes. The introduction of atropine reduces the secretion of pancreatic juice.

The gastric (second) secretion phase has more complex mechanisms and is accompanied by the secretion of about 10% of total stimulated secretion. It occurs in response to dilation of the stomach and also through the cholinergic reflex. Pancreatic juice has a liquid consistency and contains a small amount of enzymes.

Intestinal (third) phase. The main period of pancreatic secretion after a meal occurs in the intestinal phase. This phase is responsible for 70-80% of pancreatic secretion and is regulated by ejection of secretin or cholecystokinin. Secretin, which goes into circulation with the duodenum, is responsible for the secretion of bicarbonates and water by ductal cells of the pancreas. Cholecystokenin is mainly responsible for the secretion of enzymes (proteases) in acinaric cells.

Pancreatic juice contains some enzymes:

1. amylase (diastase) - released in the active state, break down carbohydrates, cause hydrolysis of starch and its transition to maltose.
2. lipase (steapsin) - released in the semi-active state, the activator are bile acids and calcium salts - break down fats into fatty acids and glycerine.
3. proteases - proteolytic enzymes that break down proteins into amino acids:
    a. trypsinogen;
    b. carbopeptidase;

c. collagenase;
d. desnuclease;
e. chymotrypsin;
f. rypsin;
g. nuclease, which enters the duodenum as enzymes that are activated under the influence of enterokinase.

However, if these enzymes collide with cytokinesis, which is excreted from pancreatic cells at their death, activation may also occur in the middle of the gland.

Regulation of the excretor function of the pancreas is carried out in a neurohumoral way:

1. conditional and unconditional irritants through sympathetic and parasympathetic fibers stimulate secretion of the pancreas;
2. Gastric Syme, which contains hydrochloric acid, getting into the duodenum, irritates its mucous membrane and the formation of secretin and pancreosymine, which humorally stimulates the secretory part of the pancreas.

Normally, the secret of the pancreas does not affect its undamaged tissue and does not itself destroy its cellular elements. Self-digesting of the pancreas occurs only under the influence of various etiological factors and their combination.

Incremental function of the pancreas provides regulation of water exchange, participates in fat exchange and regulation of blood circulation.

The endocrine part is formed by groups of insulae pancreatici (Langerhans islands).

Intra-secretory activity of the pancreas is due to the secretion of hormones produced in the Langerhans islets, which are located among the cells of the gland parenchyma, mainly in the tail and body and have no ducts, secrete the hormone directly into the blood. The main ones are insulin, somatostatin and glucagon.

Langerhans Islands contain 10-30% A-cells, which are the site of glucagon formation. The other 60-80% are B cells, which also synthesize deposited insulin, D cells (about 10%) produce somatostatin, and C cells produce gastrin.

The main role of insulin is to regulate carbohydrate metabolism, it reduces blood sugar levels, contributes to the deposition of glycogen in the liver, its absorption by tissues and reduces lipemia. Violation of insulin production causes an increase in blood sugar, thus - the development of diabetes.

Etiology and pathogenesis of acute pancreatitis. At the heart of the pathogenesis of acute pancreatitis is the activation of own pancreatic enzymes with subsequent autolysis. The activation of proteases, which first occurs in the ducts of the gland, and then they penetrate the parenchyma, destroying it, becomes essential.

Factors contributing to the development of the disease:

1. Neurohumoral - disorders of fat metabolism, systemic diseases of blood vessels, functional diseases of the stomach, secondary circulatory disorders in the pancreas, liver, heart, pregnancy, childbirth.
2. Toxic-allergenic - food and drug allergies, alcoholism, various poisonings, hotbeds of acute and chronic infections.

Factors that cause disease:

1. Ductogenic (48%):
   a. Duodenobiliary (gallstone disease, duodenostasis, primary and secondary duodenopapilitis, postcholecystectomic syndrome) - 25%;
   b. b) genovine (chronic pancreatitis, inflammatory infiltrates, pancreatic tumors) - 23%.
2. Acute cholecystitis - 10%.
3. Contact (gastric and duodenal ulcer with penetration into pancreas) - 1.5%.
4. Vascular (occlusion of sacreavian arteries, veins, portal hypertension) - 4%.
5. Posttraumatic (gland injuries, open and closed, surgical) - 5%.
6. Kryptogenic (diseases that reduce the body's defenses) - 29%.
7. Complication of other diseases and associated pathologies (poisoning, terminal states of acute intestinal obstruction, etc.) - 2.5%.

The combined effect of three active factors - autosensitization with tissue antigens, violation of the intraorganic capillary blood circulation and changes in the blood coagulation system - is the pathogenetic basis of acute pancreatitis.

The influence of any damaging factor leads to the isolation of the active substance from cells - cytokinase, which activates trypsinogen, turning it into trypsin. Trypsin, affecting the gland parenchyma, damages blood vessels, leads to stasis and massive edema of the gland, and later - to hemorrhage. It's what's called the trypsin phase. Microcirculation disruption causes necrosis.

As a result of hemorrhagic necrosis, bile acid salts are excreted from the blood and lipase - the "lipase phase" - is activated. Both processes occur simultaneously and are interconnected. As a result of glandular damage, some active enzymes enter the bloodstream. The release of proteolytic and lipolytic enzymes of the pancreas into the blood causes the activation of the kalikreinkininin system, accompanied by a decrease in tone and vascular permeability, the release of blood plasma outside the vascular channel and, as a consequence, cardiovascular and renal failure, pulmonary and brain oedema, that is, polyorgan failure. And then there are purulent-septic complications.

Pathomorphology of acute pancreatitis. Changes in the gland depend on the phases of the disease, change rapidly and are extremely polymorphic simultaneously in different parts of the gland. Interstitial edema (pale, oedematous, dense, stressed gland) is observed at the initial stage. Infiltration of lymphoidnema by cells is noted histologically. In the phase of hemorrhagic necrosis there are observed overflowing blood vessels, centers of hemorrhage and necrosis (red, dense, oedema gland). In

histological examination, edema, lymphoid infiltration and diapaedesis of red blood cells are observed.

Hemorrhagic fluid appears in the abdominal cavity. At the same time it develops and fat gland necrosis, which makes the gland look like a sebum candle (flabby, cloudy, foci of fat necrosis).

Accompanying the infection forms a focal or diffuse purulence. Gypsologically, tissue necrosis and continuous leukocytic infiltration are noted.

Classification. Emphasizes the clinical and statistical classification of the disease (ICD-10):

K85 - acute pancreatitis.

Indications for inpatient treatment: clinical signs of acute pancreatitis.

International classification of acute pancreatitis (Atlanta, USA), 1992.

I. Acute pancreatitis:
   a. soft;
   b. heavy.

II. Acute fluid accumulation (parapancreatic tissue in the pancreas) - acute interstitial pancreatitis.

III. Pancreonecrosis:
   a. sterile;
   b. infected.

IV. Pancreatic false cyst.

V. Pancreatic abscess.

1. Clinical and anatomical forms:
   a. edema pancreatitis (abortive pancreatic necrosis).
   b. fat pancreonecrosis;
   c. hemorrhagic pancreonecrosis.

2. Propagation of necrosis:
   a. local (focal) glandular lesion;
   b. subtotal lesion of the gland;
   c. total glandular lesion.

3. Course:
   a. abdominal glandular disease;
   b. progressive.

4. Periods of disease:
   a. period of hemodynamic disorders and pancreatogenic shock;
   b. period of functional failure of parenchymatous organs;
   c. period of dystrophic and purulent complications.

5. Interstitial pancreatitis

It is a mild (moderately heavy) form, which has a stroke with minimal organ dysfunction and is well amenable to conservative treatment (acute interstitial pancreatitis). Its main morphological criteria are macroscopic-interstitial edema,

microscopic-leukocytic, mainly neutrophilic imbibition of the pancreas with small foci of acinaryngeal necrosis and parapancreatic fatty tissue.

It is a severe form in which 20-25% of cases of the disease is accompanied by the development of complications (pancreatic necrosis proper): in early terms - polyorgan failure, in late - pancreatic infection, which is accompanied by necrosis of more than 30% of the volume of pancreatic tissue. In macroscopy, widespread necrosis of parapancreatic fibers with cloudy hemorrhagic perspiration into the abdominal cavity is noted. Microscopically - drainage zones of organ parenchyma necrosis, vascular wall necrosis, centers of hemorrhage and rupture of pancreatic ducts. An important feature is also the presence of fat necrosis inside the pancreas and pronounced inflammation phenomena.

Pancreonecrosis

a. Non-infected pancreatic necrosis is a diffuse or restricted area of the devitalized pancreatic parenchyma, most commonly with associated necrosis and a tendency to become infected. In microscopy, damage to the parenchymatous mesh of the pancreas capillaries, acinar and islet cells, and the flow system from the perilobular necrosis
b. infected pancreatic necrosis - a widespread purulent-necrotic affection of pancreatic tissue and/or peripancreatic tissue with the presence of pathogens (fungi, bacteria) in them in the amount of 1x105-109 per 1 g of tissue, which occurs as a result of infection of non-viable tissues, with no accumulation of manure. Purulent pancreatonecrosis most often occurs in the early stages of the disease and is characterized by the development of severe complications: respiratory distress syndrome, acute renal failure, coagulopathy, which are an immediate threat to the life of the patient

Pancreas abscess - located, more than 5mm in diameter, accumulation of manure in or around the pancreas, which is formed by melting necrotic tissues or secondary infection of the false cyst. It occurs more often in the late stages of acute pancreatitis. It is relatively well predicted.

Acute false cyst of the pancreas is a limited pseudocapsular accumulation of pancreatic juice in the pancreas or parapancreatic tissue, which occurs not earlier than 4 weeks after the disease. (The cyst is considered acute within 2-3 months from the disease).

Two clinical phases of the course of severe acute pancreatitis can be distinguished conditionally:

The first, associated with sterile necrosis of the pancreas and cellular formation. It is caused by local and systemic disorders, mainly by activated pancreatic enzymes, cytokines and factors of endothelial origin (1-7 days).

The second is associated with infection of necroses of any localization, the development of secondary SIRS (syndrome of systemic inflammatory response) and MODS (syndrome of multiple organ dysfunction - 8-14 days).

Acute Pancreatitis Classification (Based on input from the Atlanta Classification Revision Working Group, 2012)

<table>
<tr><td rowspan="5">1. Ethiology:</td><td colspan="3">A) alcoholic</td></tr>
<tr><td colspan="3">B) biliary</td></tr>
<tr><td colspan="3">B) alimentary</td></tr>
<tr><td colspan="3">D) post-traumatic</td></tr>
<tr><td colspan="3">D) idiopathic</td></tr>
<tr><td>2. For severity:</td><td>A) mild form</td><td>Б) moderate severity</td><td>B) severe form</td></tr>
<tr><td>3. Form:</td><td>A) edematous (interstitial)</td><td colspan="2">Б) necrotizing pancreatitis (pancreatic necrosis):</td></tr>
<tr><td rowspan="2">4. Prevalence of necrosis:</td><td rowspan="2"></td><td rowspan="2">a) limited necrosis (up to 30% necrosis of the pancreatic parenchyma)</td><td>a) common pancreatic necrosis (from 30 to 50% necrosis of the pancreatic parenchyma)</td></tr>
<tr><td>b) subtotal-total pancreatic necrosis (> 50% necrosis of the pancreatic parenchyma)</td></tr>
<tr><td rowspan="2">5. Systemic complications of acute pancreatitis:</td><td rowspan="2"></td><td rowspan="2">a) systemic inflammatory response syndrome (up to 48 hours from the onset of the disease)</td><td>a) systemic inflammatory response syndrome (> 48 hours from the onset of the disease)</td></tr>
<tr><td>b) multiple organ failure (respiratory, renal, liver failure, DIC)</td></tr>
<tr><td colspan="4">6. Complications of acute pancreatitis</td></tr>
<tr><td rowspan="3">6a.Phase of aseptic inflammation (up to 2 weeks from the onset of the disease)</td><td rowspan="3"></td><td colspan="2">a) enzymatic peritonitis, pleurisy</td></tr>
<tr><td colspan="2">b) acute parapancreatic fluid accumulation (in the omental bag, in the retroperitoneal tissue)
c) acute pancreatic and / or parapancreatic necrosis</td></tr>
<tr><td colspan="2">d) obstructive jaundice</td></tr>
<tr><td>6b. Phase of aseptic inflammation (> 2 weeks from the onset</td><td></td><td colspan="2">a) pancreatic pseudocyst (formed from 4 weeks from the onset of the disease)</td></tr>
</table>

diseases)			
6c. Phase of purulent complications			- infected pancreatic and parapancreatic fluid accumulations (in the stuffing box, retroperitoneal phlegmon) - infected pancreatic and parapancrotic necrosis (up to the 4th week from the onset of the disease) - limited infected necrosis (> 4 weeks from the onset of the disease)
			- purulent peritonitis, abscesses of the abdominal cavity, pleural empyema
6d. Secondary complications of necrotic pancreatitis			- acute bleeding in the gastrointestinal tract
			- internal and external fistulas of the digestive tract
			- arosive bleeding into the cavity of the pseudocyst or out

The clinic of acute pancreatitis. The clinical picture of acute pancreatitis is very diverse and depends on the form of lesion, localization and scope of pathological changes, as well as on the degree of activation of enzymes, localization of the process, the reactivity of the body and the emergence of complications.

The disease begins suddenly with attacks of severe pain, mainly in the upper abdomen, which more often occurs after diet disturbance (excessive consumption of fat and spicy foods, especially when combined with alcohol, diseases of the biliary tract and after physical and mental exhaustion). Painless forms are very rare. The pain is very severe, unbearable, and in some cases so intense that the patients even lose consciousness.

The onset of pain with acute pancreatitis is often preceded by precursors: dyspeptic phenomena, short-term, uncertain nature of abdominal pain, a sense of gravity in the epigastric region. To detect such prodromal phenomena is difficult, because the subjective feelings of patients may be due to concomitant diseases of other abdominal organs (gall bladder, stomach, duodenum, etc.), or their

simultaneous disease with involvement in the process of the pancreas itself. The pain is usually constant, but in 10-15% of patients, it may be of a contractile nature due to the phenomena of dynamic intestinal obstruction. The intensity of pain in acute pancreatitis in most cases corresponds to the severity of the process and reaches its greatest strength in acute hemorrhagic pancreatic necrosis, but in some cases, it can be very sharp, even in acute pancreatic oedema.

Pain localization may vary depending on which part of the organ is involved in the inflammatory process. In a lesion of the head of the pancreas, pain is localized in the epigastric region or to the right of the median line, when involved in the process of the body - in the epigastric region, the tail - in the upper left half of the abdomen, in a diffuse lesion of the gland - throughout the upper half of the abdomen, becoming shingles.

Irradiation of pain is varied: to the lumbar region, right scapula and shoulder, sometimes to the left half of the chest, often acquiring the character of pain in the form of a feeling of tightness "belt", "hoop", etc.

The duration of the pain also varies. In some cases it lasts several hours, in others, it lasts several days.

Vomiting is the second most common symptom of acute pancreatitis. Vomiting usually occurs after an attack of pain, is very frequent (sometimes endless), first with food and then with bile and sometimes blood. Blood impurities in the vomit masses indicate severe damage to the pancreas. The volume of vomiting masses can be up to 2-6 litres per day, which further leads to pronounced dehydration of the body. Some patients suffer more from vomiting than pain.

Body temperature may be different - normal, sometimes even low, and later on with the development of collapse and the progression of subfebrilic or elevated disease. High body temperature indicates that an infection has joined (more often on 5-6 days). Prolonged and significant increase in body temperature within 8-10 days may be the result of joining the main disease of secondary infection or the development of the purulent process in the iron.

At the external examination of the patient in most cases skin pallor is marked, jaundice of sclera and skin is often marked (it can be caused by oedema of pancreas head, blockage of the common bile duct, exacerbation of inflammatory process of gallbladder and bile ducts as a result of liver damage and erythrocyte disintegration). In severe cases, skin cyanosis is observed (the result of enzymes and hemodynamic disorders), which may be of different intensity and prevalence. In addition, the lesion of surface vessels, with acute pancreatitis may occur the same changes in the internal vessels, resulting in bloody vomiting and intestinal bleeding.

The general condition of patients is severe due to pain syndrome, intoxication and dehydration. The patient's position is often forced - half-bent. The tongue is wet at the beginning of the disease, covered with white or grey plaque, but later with increasing intoxication becomes dry. Heart rate is first slow, then accelerates, becomes weak, persistent tachycardia develops before the body temperature. Transistor compensatory hypertension is changed by persistent hypotension due to hypovolemia, blood thickening, increased vascular permeability,

increased portal pressure and decreased central venous pressure, reduced volume of circulating blood, oliguria (less than 500ml of urine per day) appears. Attention is drawn to anxiety, as well as disorders of the mental state of the patient (phenomena of acute psychosis), which sometimes requires psychiatric care (especially in alcoholic pancreatitis).

Development of shock and collapse is typical for severe forms of acute pancreatitis and may have a different duration (sometimes several days). Often, patients with acute pancreatitis experience respiratory distress, which becomes more frequent (30-40 or more respiratory movements per minute) and superficial, which is due to a pronounced intoxication and direct involvement in the inflammatory process of the diaphragm and pleura, as well as nerve-reflective effects of the solar plexus and pancreas.

When examined in the first hours of the disease, asymmetry of the abdomen in the upper parts of the body may be observed as a result of moderate bloating of the abdomen in the epigastric (due to the paresis of the stomach and mainly cross-sided intestine of a reflex character and secondary affection of its mesentery with enzymes of the pancreas). Later the asymmetry of the abdomen is evened out due to uniform bloating of the entire abdomen. The paresis of the intestine is accompanied by a delay in the discharge of gases and stools.

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When percussion is detected blunting of percussion sound in epigastria and the course of infiltration. Shortening of percussion sound in adjunctive places and displacement of the dullness boundary when changing body position indicates the presence of exudate in the abdominal cavity. High thympanitis throughout the abdomen at percussion is determined at pronounced intestinal paresis.

On palpation of the abdomen during the first period of acute pancreatitis, despite severe pain in the epigastric region and left subcostal, patients do not show any abdominal muscle tension or symptoms of peritoneal irritation ... Therefore, often at examination the "soft" abdomen is defined against the background of a pronounced pain syndrome, typical for acute pancreatitis. The area of pain and rigidity corresponds to the projection of the pancreas. Painful resistance of the abdominal wall in the epigastric area in the form of a transverse strip, which is located 6-7cm above the navel and corresponds to the topographic position of the pancreas (**Körte's symptom**), painfulness in the epigastric odiolancy when the abdominal wall is pushed from bottom to top and front to back by the doctor's hand, located across the abdomen below the navel (**Chukhrienko's symptom**) is determined. Besides, pain in the **Chauffard area** can be determined - the angle between the middle line of the abdomen and the bisector of the angle formed by the middle line of the abdomen and the horizontal line drawn at the navel level, and

Dejardin's point - 6cm above the navel line, with the navel joining the right axillary depression (if the head of the pancreas is affected), and **Hubergritz** - similar only to the left (if the tail of the pancreas is affected).

In diagnostics of acute pancreatitis the **symptom of Mayo - Robson** - revealing at palpation of a painful point in the left rib-vertebral angle (at pancreas tail affection) is given importance. The symptom is not constant, so its absence does not exclude the presence of acute pancreatitis.

Patients with acute pancreatitis often lack the abdominal aortic pulsation in the epigastric region due to glandular edema (Voskresensky symptom). If the muscle tension does not interfere with the study, then on palpation there is dough, "execution" of the epigastric region behind the pancreas, and sometimes - stretched in the transverse direction of painful infiltration. When a cyst or abscess is formed, which happens a few days after the onset of the disease, it is possible to determine the rounded formation of different sizes, takes up the entire epigastric area.

For the detection of acute pancreatitis is also determined by some characteristic symptoms, the diagnostic value of which increases when detected together.

- *Lagerlof symptom* - cyanosis of the face, extremities;
- *Mondor's symptom* - purple spots on the face, torso;
- *symptom of Halsted* - abdominal skin cyanosis,
- *Grey-Turner's symptom* is cyanosis of the side walls of the abdomen,
- *Cullen's symptom* is a yellowish-cyanotic color in the navel area;
- *Grunvald is a symptom* of cyanotic spots around the navel;
- *symptom of Davis* - abdominal skin cyanosis, petechial hemorrhages in the lumbar and buttock area;
- *Makov's symptom* is hyperesthesia of the skin above the navel;
- *Katsch symptom* - hyperesthesia of the skin on the left paravertebrally at the thoracic vertebrae level (Th6-Th8).
- *Razdolski's symptom* is pain at percussion above the pancreas.

If the inflammatory process involves the entire peritoneum, palpation reveals spilled pain and tension of abdominal muscles, even increased due to edema of the pancreas can not be felt because of its deep placement and a pronounced flatulence. Therefore, the data on palpation in the study of the abdomen to some extent dependent on the form of acute pancreatitis, which suffers from the patient.

Optionally, in the first hours of the disease, the peristalsis is normal or even intensified. When involved in the process of peritoneum there is a flatulence, there is a sharp weakening of intestinal noises or their complete disappearance, dynamic intestinal obstruction develops. Symptoms of irritation of the abdomen and moderate tension of the anterior abdominal wall muscles appear, mainly in the epigastric region, less often along the abdominal wall if the entire abdomen is involved in the inflammatory process. At the same time, peritonitis phenomena are much weaker than in gastrointestinal perforations.

Depending on the stage of the course of the destructive form of acute pancreatitis, its clinical picture has significant differences. Therefore, based on

pathophysiological changes in the body in the clinical course of pancreatitis three distinct periods can be distinguished:

- hemodynamic disorders and pancreatogenic shock (1-3 days).
- functional failure of parenchymatous organs (3-7 days).
- organic (local abdominal) dystrophic and purulent complications (over 7 days).

The first period is characterized by sharply pronounced pain syndrome and changes in hemodynamics (bradycardia, fluctuations in blood pressure). As the destructive process progresses, tachycardia develops. Blood pressure changes in different ways: it rises or falls. Pancreatogenic shock develops in 20% of patients, in which the condition of patients is heavy, the skin is cold, moist, pale, often spotted, marble. There is acrocyanosis, frequent breathing, thready pulse, hypotension with a sharp decrease in pulse pressure, changes in ECG of ischemic type. Many patients may have mental disorders: psychomotor excitation, hallucinations, delirium.

The second period is manifested mainly in the dysfunction of vital organs and systems, there are signs of cardiovascular, hepatic, renal and respiratory failure. During this period, there may be lesions of the central nervous system, manifested by mental disorders of patients, the appearance of delirium and coma, and later leads to death of patients.

The third period is characterized by the development of local changes after necrotic processes, which may occur aseptically with the formation of parapancreatic infiltration or the formation of a false pancreatic cyst or with the addition of infection, when the phlegmons of the retroperitoneal space, intra-abdominal abscesses, fistulas, erosive bleeding is possible.

The so-called lightning (fulminant) form of acute pancreatitis must be remembered. In this case, the severity of symptoms is so great that the patients die a few hours after the beginning of the disease. Sometimes they do not even have time to seek medical attention. This course of acute pancreatitis is more often observed in young people and almost all cases is due to the emergence of total pancreatitis.

Diagnosis of acute pancreatitis. To diagnose acute pancreatitis and determine the state of severity of the patient, it is necessary to rely on:

a. clinical manifestations of the disease and its severity (the Mondor triad - presence of pain that is localized above the navel, invincible vomiting, bloating of the abdomen that occurs after eating error, alcohol consumption)
b. data of physical diagnostic methods
c. data of the main laboratory and instrumental methods of diagnostics

Labolatoric diagnistics:

- clinical blood analysis
- general urine test;
- blood sugar
- Amylase content in serum (sign: level 3 times the upper limit of the norm);

- serum lipase content (sign: level 3 times higher than the upper limit of the norm);
- Urinary diastase content (indication: level 3 times higher than normal upper limit).
- blood type, rhesus factor;
- Blood biochemical analysis (bilirubin, ALT, ACT, SCP, GHT, thymol sample, electrolytes, urea, protein, creatinine, urea nitrogen, uric acid)
- protein and protein fractions;
- coagulogram;
- medium molecules;
- serum calcium
- C - reactive protein (in quantitative units).

A general blood test. Changes in the red blood count are relatively rare. In the first days of the disease there may be a relative increase in hemoglobin levels as a result of fluid sequestration. Hypochromic anemia may occur later on. Changes in white blood parameters are of diagnostic value. High leukocytosis with shift of leukocyte formula to the left, lymphopenia (Herfort symptom), eosinopenia and neosinophilia, increase of SER, hematocrit (due to blood thickening) is determined in general blood analysis.

Biochemical blood test. Rapidly developing metabolic disorders: hypoproteinemia, hyperglobulinemia, hypoprothrombinemia, hypofibrinogenemia, hyperbilirubinemia, which are associated not only with a mechanical factor, but also with damage to the liver parenchyma due to its hypoxic or toxic damage. Besides, in acute pancreatitis, especially its severe course, there is an increase in aminotransferase, lactate dehydrogenase and alkaline phosphatase activity, not always correlated with bilirubinemia.

As a result of leakage of enzymes there is an increase in blood amylase to 300 units. Amylase in urine (diastase) appears after 12-24 hours, its values range from 512 to 32786 IU and above Volgemutom (normal 4-64 IU, moderate increase - 128-512 IU, sharp increase - more than 512 IU). From 2-3 days of the disease hypocalcaemia is observed due to binding of calcium in necrosis foci of fatty acid salts. Expressed hypocalcaemia is a negative prognostic sign. Violation of carbohydrate metabolism is manifested by hyperglycemia (associated not only with damage to the islet apparatus of the pancreas, but also with a violation of oxidation of major energy substrates in the liver parenchyma) and, as a consequence, glucose (in 10-15% of patients). Progressive decrease of protein and especially albumin concentration has an important diagnostic value.

Other laboratory indicators are markers of complications and are used to predict the course of acute pancreatitis. An increase in serum urea and creatinine levels indicates impaired renal function, while changes in blood gas composition indicate respiratory failure.

Additional instrumental diagnostic methods. Review X-ray and chest and abdominal cavity radiography allows to reveal high standing of the left diaphragm dome and fluid level in subdiaphragmal space, pleural sinuses, restriction of mobility

of diaphragm domes, intestinal pneumatosis, fluid accumulation in abdominal cavity. In contrast study of the digestive tract with barium sulfate can be found characteristic signs of oedema pancreatitis and pancreatic cysts. The increase of retrogastric space with the formation of the so-called "cascade stomach" is observed in the lesion of the body of the pancreas, and in the lesion of the head - "unfolded horseshoe" and flattened clearance of the duodenum. There are some radiological symptoms typical for acute pancreatitis:

- *The symptom of the "guard loop"* - local bloating of the first loop of the hungry intestine;
- *The symptom of the "severed intestine"* is an inflatation of the ascending colon and its hepatic angle with a sharp break directly to the left of the right bend of the colon;
- *Stewart's symptom* is inflating the ascending and descending colon with no gas in the cross-rimmed intestine;
- *Gobi's symptom* is a local paresis of the transverse colon;
- *Poppel's triad* - presence of air in the stomach, duodenum and small intestine;
- *Toby's symptom* is the blurred contour of the left lumbar muscle.

Gastroduodenofibroscopy - allows to reveal the signs of papillite, sometimes - the presence of a wedged stone in the large duodenal papilla, signs of gastroduodenite of the back wall.

Ultrasonic (sonographic) monitoring (the main method of screening instrumental diagnosis). Currently, it is the most common method of diagnosing diseases of the pancreas, which can determine the degree of increase in the size of the pancreas and the disappearance of the characteristic length of its structure, the presence of infiltration in the projection of the gland and the presence of perspiration in the abdominal cavity, as well as the state of the gallbladder and extrahepatic bile ducts. Characteristic ultrasonographic picture for the oedema form of acute pancreatitis is an increase in the size of the pancreas, increasing its hydrophilicity, the presence of signs of moderate edema of parapancreatic fibers while maintaining a clear contour of the organ itself. In pancreatic necrosis the contours of the pancreas are fuzzy and blurred, and the structure of its tissue is heterogeneous with hypoechogenic areas in the necrosis zones. Often the accumulation of fluid in the glandular bag or free abdominal cavity is determined. In the severe course of disease ultrasound is performed at least once a day (pay attention to the condition of the pancreas and abdominal cavity).

Computed tomography (CT) of abdominal organs with bolus injection of a contrast substance (method of instrumental diagnostics, clarifies the method that determines the severity of the pancreas affection) - the most informative of noninvasive diagnostic methods at present. This method has some advantages over ultrasound, which may have difficulty interpreting the data in cases of flatulence and obesity of the patient. Computed tomography is currently the only method of radiation diagnostics, which allows to obtain a clear detailed image of the pancreas, assess its shape, size, structure, relationships with other organs and structures. Conducted with the contrast of the digestive canal and with angiography, it becomes

even more possible to identify some characteristic symptoms: increased swollen pancreas, foci of necrosis in the adipose tissue behind the omentum, in paranephritis, in the root of the mesentery of the small and transverse colon, the lack of visualization of the pancreas in angiography in cases of necrosis. Indications for CT in case of acute pancreatitis are:

- specification of the acute pancreatitis diagnosis in case of insufficient information from other research methods;
- in case of established pancreatic necrosis diagnosis to determine the prevalence of pancreatic lesion;
- in the development of complications of acute pancreatitis;
- in case of worsening of the patient's condition against the background of therapy
- to perform percutaneous puncture and aspiration of pancreatic tissue to confirm or exclude infection of necrosis foci;
- to evaluate the postoperative flow.

Five topographic anatomical CT variants for acute pancreatitis are distinguished (E. S. Valthazag and co-authors, 1990):

a. normal pancreas;
b. local or diffuse expansion of the pancreas borders, without a couple of pancreatic changes;
c. expansion of pancreatic boundaries or changes in pancreatic parenchyma with signs of parapancreatic inflammatory process;
d. enlargement of size of pancreas with presence of parapancreatic fluid in anterior superior around renal area;
e. increase in the size of the pancreas and the presence of more than 2 liters of pancreatic fluid.

Laparoscopy - has greatly enhanced the diagnosis of acute pancreatitis. It allows to receive direct objective signs of acute pancreatitis:

- plaques of fat necrosis on the peritoneum and omentum ("steatonecrosis").
- hyperemia and hemorrhagic imbination of the peritoneum and the large omentum, perspiration with a hemorrhagic hue (at joining the hemorrhagic component in the abdominal cavity, the study of which find a high content of enzymes).

Indirect signs include:

- gastric and transverse colorectal paresis;
- swelling of the small stuffing box;
- hepatic duodenal ligament oedema;
- swelling of the stomach forward, dilation of its veins.
- an enlarged and stretched gallbladder without signs of inflammation.

Angiographic research is a rather complicated modern method and its use is justified in cases when it is impossible to make a diagnosis of diffrentiation using other methods.

Other methods of acute pancreatitis diagnostics, such as selective aorthography, thermography and radioisotope examination, have not found wide application and are used as auxiliary. Retrograde cholangiopancreatikography is more commonly used to diagnose acute pancreatic pseudocysts.

Clinical manifestations of the disease depending on the severity of the acute pancreatitis patient

Initial assessment	Clinical manifestations of the disease, depending on the severity of the patient Body mass index> 30 kg / m2 Pleural effusion on radiographs, RDS Score for APACHE II> 8
24 hours after hospitalization	Score for APACHE II> 8 Stance organ failure (special multiple)
48 hours after hospitalization	48 hours after hospitalization Glasgow score reduced by 3 points or more. C-reactive protein> 150 mg / l Stance organ failure (special multiple)

After 24 hours of inpatient hospitalization, the degree of severity of the patient's condition can also be determined using the Marshall Polyorgan Dysfunction Scale or the SOFA (Sequential Organ Failure Assessment) Scale.

Marshall Score

	The mark				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (pO2 / FiO2)	>400	301-400	201-300	101-200	≤101
Renal **Whey creatinine (mmol / L)** **Serum creatinine (mg / dL)**	≤134 <1,4	134-169 1,4-1,8	170-310 1,9-3,6	311-439 3,6-4,9	>439 >4,9
Cardiovascular (systolic blood pressure,mm. Hg.)	>90	<90 with response to infusion therapy	<90 without response to infusion therapy	<90, pH<7,3	<90, pH<7,2
Hepatic (bilirubin,	≤20	21-60	61-120	121-	>240

mol / l)				240	
Hematologic (platelets, x109 / l)	>120	81-120	51-80	21-50	≤20
Neurological (number of points for the Glasgow scale)	15	13-14	10-12	7-9	≤6

SOFA Scale Rating Interpretation

	Score of SOFA	Mortality
0	0-2	до 9%
1	3-4	до 22%
2	6-8	до 38%
3	9-12	до 69%
4 and more	13 and more	83% and more

Patient severity assessment on APACHE II scale

Indicators	**Score**								
		+3	+2	+1	0	+1	+2	+3	+4
Temperature, ° C	1	39-40,9		38,5-38,9	36-38,4	34-35,9	32-33,9	30-31,9	≤29,9
Mean arterial pressure,mm Hg	60	130-159	110-129		70-109		50-69		≤49
HR for 1 min.	180	140-179	110-139		70-109		55-69	40-54	≤39
Respiratory rate for 1 min.	50	35-49		25-34	12-24	10-11	6-9		≤5
GradientAa and pO2,mm Hg 2 PaO2,mm Hg.	00	350-499	200-349		<200 >70	61-70		55-60	<55
arterial	,7	7,6-		7,5-	7,33-		7,2	7,1	<7,15

pH 3 Content of HCO3 in serum (mmol / l)	2	7,6 9 41-51, 9		7,59 32-40,	7,49 23-31,9		5-7,3 2 18-21, 9	5-7,2 4 15-17, 9	<15
The content of Na + in serum, mmol / l	80	160 - 179	155 - 159	150-154	130-149		120 - 129	111 - 119	≤110
K content in blood serum, mmol / l		6-6,9		5,5-5,9	3,5-5,4	3-3,4	2,5-2,9		<2,5
Serum creatinine content, mg%	3,5	2-3,4	1,5-1,9		0,6-1,4		<0, 6		
Hematocrit (%)	60		50-59, 9	46-49,9	30-45,9		20-29, 9		<20
The total number of cells, x109 / l	0		20-39, 9	15-19,9	3-14,9		1-2,9		<1

Differential diagnosis. Differential diagnosis should be made with the following diseases:

A. Most commonly:
   - perforative or Penetrating gastroduodenal ulcer
   - with acute cholecystitis
   - with acute intestinal obstruction
   - spleen infarction
   - kidney colic
   - with mesenteriotrombosis

B. less frequently:

- with pneumonia;
- with myocardial infarction
- with acute appendicitis;
- with food toxicosis;
- purulent paranephritis

Differential diagnosis of acute pancreatitis

Symptoms of the disease	***Nosological forms***		
	Acute pancreatitis (first period)	***Acute pancreatitis (first period)***	***Acute pancreatitis (first period)***
Complaints			
Pain			
Localization	In the upper abdomen	Behind the sternum	Whole belly
Intensity	Very intense	Intense pressing	"Dagger"
Character	Dumb constant	Sharp constant	Sharp constant
Irradiation	In the back, shingles	In the epigastric region, left arm and shoulder girdle	In the shoulder girdle
Vomiting	Unquenchable - first taken food, then bile or intestinal contents	Reflex, accepted food	No or single meal
Defecation act	Delay	Delay	Delay
Objective data			
Integuments	Pale, dry, have color symptoms	Cold, covered with cold sweat, acrocyanosis	Pale, covered with cold sweat
Pulse	Tachycardia	Tachycardia, arrhythmia	Bradycardia that changes tachycardia
Arterial pressure	Declining	Declining	Declining
Tongue	Dry	Wet	Wet first, then dry.
Examination of the abdomen			
Inspection	Takes part in the act of breathing	The upper part lags behind in the act of breathing	Does not participate in breathing
Palpation	The abdomen is soft, painful in the epigastrium.	Moderate muscle tension in the epigastrium	Dose-like muscle tension and soreness over the entire surface of the abdomen

Percussion	Dullness in sloping places, tympanitis above the transverse colon	Without features	Lack of liver dullness
Auscultation	Weakened or missing peristalsis	Without features	Weakened peristalsis
Pathognomonic symptoms	Kurt, Chukhriyenok, Resurrection, Mayo-Robson, "color symptoms"	-	Spizharny, the triad of Mondor
Additional methods			
Clinical blood test	Moderate leukocytosis with a left shift	Moderate leukocytosis with a left shift	Moderate leukocytosis with a left shift
Urinalysis	Oligoanuria, later toxic changes	Without changes	Without changes
ECG	Possible toxic ischemic changes in myocardium	Signs of acute myocardial ischemia	Without changes
Survey X-ray	Hyperpneumatosis, Gobier symptom	Not informative	Free gas under the diaphragm domes
Ultrasound scanner	Gland and fiber edema around	Not informative	Free fluid

Continuation

Symptoms of the disease	***Nosological forms***	
	Mesenteric thrombosis (phase of hemodynamic disorders)	***Renal colic***
Жалобы		
Pain		
Localization	Without clear localization	In mesogaster and epigastrium
Intensity	Very intense	Very intense
Character	Acute paroxysmal	Acute paroxysmal
Irradiation	Without irradiation	to the genitals, thigh
Vomiting	Uninterrupted, sometimes mixed with "coffee grounds"	Reflex, food taken

Defecation act	Feces with blood	Not changed
Objective data		
Integuments	Pale	Pale
Pulse	Tachycardia, often arrhythmia	Tachycardia
Arterial pressure	Declining	Does not change
Tongue	Dry	Wet
Examination of the abdomen		
Inspection	Takes part in the act of breathing	Takes part in the act of breathing
Palpation	Muscle tension and soreness without clear localization	Muscle tension and soreness in the lower sections, disappears when the patient is distracted
Percussion	Without features	Without features
Auscultation	Peristalsis enhancement	Without features
Pathognomonic symptoms	-	Pasternatskiy
Additional methods		
Clinical blood test	At the begun unchanged, later leukocytosis	Without changes
Urinalysis	Oligoanuria, later toxic changes	Protein, fresh red blood cells
ECG	Atrial fibrillation is possible	Without features
Survey X-ray	Not informative	Shadow of stones
Ultrasound scanner	Not informative	The presence of stones, hydronephrosis

Treatment of acute pancreatitis. The basics of rational surgical tactics for acute pancreatitis are the correct and timely diagnosis, a reasonable combination of conservative measures and surgical methods of treatment, clear compliance with the phased provision of specialized care to patients. In case of severe condition or development of pancreatogenic shock, patients need treatment in the intensive care unit.

Conservative treatment of acute pancreatitis should be comprehensive and include:

1. Diet - hunger, followed by oral feeding of patients immediately after reducing the pain syndrome and the level of pro-inflammatory markers.
2. Removal of pain syndrome and neurovegetative disorders. It is used for this purpose:
    a. analgesics and non-steroidal anti-inflammatory;
    b. antispasmodics;
    c. combined preparations: Pitophenone in combination with analgesics; d) epidural analgesia (performed to relieve pain syndrome, improve

hepatosplastic microcirculation and stimulation of intestinal motor skills), is performed under the condition of hemodynamic stability against the background of dehydration correction; the catheter is set at ThVIII - ThIX. Drugs should not be injected, as they have a vagomimetric effect, which leads to spasm of the Oddi sphincter and, as a result, an increase in pressure in the bile ducts and ducts of the pancreas. To correct neurovegetative disorders, a novocaine blockade (paranefral) and epidural anaesthesia are applied.Blockade of secretory activity of the pancreas. To reduce the acidity of gastric juice and suppression of stomach secretion is used:

3. Nasogastric aspiration;
4. Proton pump inhibitors;
5. H2-blockers of histamine;
6. to interrupt the synthesis of the enzymes: dalargin, 5-fluorouracil (fluorofur) for 3-4 days. The best means to suppress the secretion of the pancreas is Sandostatin, which is administered 0.1 mg 2-3 times a day or stylamin;
7. Correction of circulatory disorders (occur due to the loss of fluid and electrolytes due to their transudation into the abdominal cavity, vomiting, the emergence of a stable intestinal paresis, etc.). Infusion therapy to make up for a fluid deficiency in the amount of 2.5-4 litres consists of balanced saline solutions and solutions with metabolic effect, for correction use colloidal solutions, hemosubstitutes, saline solutions, glucose, protein preparations which infusion rate should not exceed 200ml/hour. In this case, the proteins not only restore the amino acid state but also inhibit the activity of proteolytic enzymes and lipase. The daily volume of infusion therapy for a patient weighing 70 kg should be 3.5-6 litres.
8. To reduce the permeability of the vessel wall, cortisone or hydrocortisone shall be prescribed. To improve microcirculation, it is advisable to use solutions of 6-10% hydroxyethylcrachmal (HEC) - Stabilizol, Reformed, Hecodes, Rheosorbylact. For correction of anaemia and hypoxia, small doses of fresh single-group erythrocyte mass of washed erythrocytes are used transfusions.
9. Fighting enzymes. Protease inhibitors are used for this purpose: counterrikal 80-120 thousand units per day for 3-5 days (Gordox, Trazilol - in equivalent doses), blockade of cytokines (pentoxifylline - 20ml/day for 5-7 days). Pancreatic enzymes are also inactivated under the influence of serum albumin, aminocapronic acid, para-aminobenzoic acid.
10. Immunomodulatory and immunosupplementation therapy: recombinant human interleukin-2, immunoglobulins.
11. Adequate protein and energy supply. Stop feeding the patient by mouth for 3 days, correction of metabolic disorders: protein at the rate of 1-2.5 g / kg per day, amino acids - up to 2 litres, vitamins, ATP, glucose-electrolyte mixtures of 1.2-2 litres, fat emulsions - up to 2 litres.
12. Detoxication therapy is aimed at the removal of endotoxicosis signs and prevention of poly organic insufficiency. This therapy includes:

a. Accelerated diuresis. It is performed in cases of stable hemodynamics and the absence of renal failure. This method is based on the introduction of solutions of crystals (up to 4.5-6 litres) followed by the administration of diuretics: torasemide 5% 4ml or furosemide 1% ofmlvs per day under diuresis control.
b. extracorporeal detoxification (plasmapheresis, chemisorption). These methods are based on the removal of toxic substances from blood and plasma using sorbents. In severe cases, hyperbaric oxygenation, antioxidant therapy is used.

13. Prevention of purulent infection (translocation of intestinal microflora):
- enterosorption, colon lavage
- correction of metabolic disorders with subsequent restoration of intestinal barrier function (glutamine, arginine, antioxidants)
- selective intestinal decontamination (polymixin, norfloxacin, amphotericin B)
- early via probe feeding

14. antibacterial therapy: routine preventive use of antibiotics in the phase of sterile pancreatic necrosis is not recommended. Antibiotic therapy is prescribed only in the presence of extrapancreatic infection (cholangitis). It is used for this purpose:
- carbapenems (thienam 500-1000 mg 3-4 times a day intravenously drip or meronem 500-1000 mg 3 times a day intravenously drip).
- fluoroquinolones of II-IV generation (ciprofloxacin - 400 mg 2 times a day, offloxacin (tarivide) 400-800 mg 2 times a day intravenous drip, pefloxacin (abactal) 400 mg 2 times daily intravenous drip, levofloxacin (Lefloxacin) 500- 1000 mg 1-2 times daily intravenous drip, gatifloxacin 400 mg 1-2 times daily.
- cephalosporins of III-IV generation; ceftriaxone (LENDATIONINA, Oframax) - 1-4 g 1-2 times a day, cephoperazone (cephobid) - 1-4 g 2 times a day, cefotaxim (klaforan) 1-3 g 4 times a day, cephepim (Maxipim) 1 -2 g 4 times a day.
- semi-synthetic penicillins, except protected (in combination with clavam) and aminoglycosides (except for Netromycin and amicacin) in this situation, is not appropriate to apply due to their insufficient accumulation in pancreatic tissues and poor tropicity to the corresponding microflora. All antibiotics, except for carbapenemes, are combined with antianaerobic imidazole preparations (metronidazole or tinidazole) in a dose of 500 mg (100ml) 3 times a day. The course of antibiotic therapy lasts at least 14 days and is supplemented by antifungal therapy (ketoconazole (nizoral) 200 mg per day, fluconazole 50-400 mg per day. It should be noted that Candida has an important role in the development of infectious complications of pancreatic necrosis, with the main route of infection being translocation of microorganisms from the colon. In pancreatonecrosis, intraabdominal candidiasis leads to an irreversible pathological process.

15. Prevention of necrosis and micro thrombosis proliferation. To normalize microcirculation in the pancreas preventive use of low-molecular-weight heparins or unfractionated heparin according to generally accepted methods, prescription of the group of pentoxifillin (pentoxifillin on Ringer's lactate solution or combined preparation Latren) is performed.
16. Prevention of stressful acute ulcers and gastric and duodenal erosions
    - H2 histamine blockers for 10-12 days
    - proton pump inhibitors, antacids
    - Enveloping agents: almagel, secretin
    - aminocapron acid with thrombin (in the presence of erosions)
    - endoscopic methods to stop the bleeding
17. Hepatoprotectors: essential, heptral, glutargyn.

Surgical treatment of acute pancreatitis. The volume and nature of surgical interventions in acute pancreatitis is determined by the degree of spread of destruction of pancreatic tissue and involvement in the inflammatory process of the surrounding organs and tissues.

Indications for surgical minimally invasive treatment of acute necrotic pancreatitis in the first period of the disease is limited:

a. enzymatic peritonitis (according to ultrasound data, if there is fluid in the abdominal cavity from 500 to 1000ml).
b. biliary pancreatitis (choledocholithiasis, acute cholecystitis).

The following surgical interventions are mainly used:

1. Endoscopic:
- endoscopic retrograde cholangiopancreatography (ERCPG) is indicated in patients with biliary pancreatitis with signs of cholangitis
- urgent ERCPG (<24 h) has been indicated in patients with acute cholangitis
- magnetoresonance cholangiopancreatography (MRCPG) and endoultarsonography may reduce indications for ERCPG in patients with no cholangitis. Endoultarsonography has the highest diagnostic accuracy compared with MRCPG in detecting concrements of common bile duct less than 5mm in diameter
- single or prolonged nasopancreatic drainage or nasopancreatic drainage with papiloviersungotomy in the presence of pinched stone, large papilla stenosis of the duodenum or pancreatic duct, polype, around the papilla diverticulum
- papillosphincterotomy with decompression of pancreatic and bile ducts;

2. Point methods under ultrasound and/or CT control:
- puncture of liquid accumulations in the abdominal cavity, glandular bag and abdominal space
- thin-needle puncture of the pancreas to determine infection with pancreatic necrosis

- puncture and catheterisation of pancreatic cysts and abscesses, glandular fluid accumulation

3. Laparoscopic interventions

Laparoscopic treatment is currently one of the main methods of treatment of acute pancreatitis patients and includes:

- laparoscopic drainage of the abdominal cavity at enzymatic peritonitis to clarify the diagnosis and evacuate the exudate (drainage is set for 4-6 days, to limit its infection)
- in the presence of isolated accumulations of liquids perform percutaneous ultrasound-controlled punctures and drainage
- laparoscopic drainage of limited areas of pancreatic necrosis, abscesses, pseudocysts, retroperitoneal tissue
- The advantage of this method of surgical treatment is as follows:
- Reduction in the frequency of local postoperative complications (fistulas, hernia, etc.)
- reduction in the severity of postoperative trauma, and as a result - a minimal stress response to the patient.

4. Laparotomy.

Laparotomy without intervention on the pancreas is used for edematous acute pancreatitis due to the progression of peritonitis due to:

- the assumption of acute surgical disease of other organs;
- presence of pinched stones in the large papilla of the duodenum at unsuccessful attempt to remove them using endoscopic methods;
- the inefficiency of conservative treatment within 1 dob in calculous cholecystitis;

If HP is detected during diagnostic laparotomy, the volume of the operation should include drainage of the abdominal cavity in the iliac and subdental areas. The liquid obtained during the operation should be examined for amylase activity and sent for bacterioscopic and bacteriological examination.

Patients with proven necrosis infection, any intervention should be postponed until as much as possible until the 4th week from the beginning of the disease, when the necrosis is separated from surrounding tissue. Routine fine needle aspiration of fluid clusters in acute pancreatitis to verify infection is not indicated, as there are accurate prognostic markers for infection (persistent fever, increased levels of proinflammatory cytokines and signs on the CT scan - the presence of gas bubbles in fluid and necrotic clusters).

The best time for surgical intervention is after the 4th week of the disease. The primary type of intervention for verified necrotically infected pancreatitis is percutaneous drainage of fluid and necrotic clusters or endoscopic transluminal drainage, the next step, if indicated, is conventional surgery, video-systemic retroperitoneal necrectomy or endoscopic transluminal necrectomy.

5. Drain the pustule, sewing up the perforation of the cavity organ and aromatic vessels. Perform open intervention with external drainage. The

perforated organ is sewn, at the perforation of the duodenum - with its exception, the large intestine - by ileostomy, pierced vessels, as a rule, simultaneously perform a partial distal resection of the pancreas with the removal of the spleen.

In the postoperative period, it is necessary to continue intensive therapy and to take measures to prevent purulent-septic complications.

Complications of acute pancreatitis. The time of acute pancreatitis complications is divided into early and late:

Early complications are due to the generalized effect of pancreatic enzymes, biologically active amines and other vasoactive substances. They include:

- shock
- the enzymatic spilled peritonitis;
- acute hepatic renal insufficiency
- acute cardiac insufficiency
- early acute sores and gastrointestinal bleeding
- jaundice
- pneumonia
- pleurisy;
- atelectases;
- vascular thrombosis;
- intoxicating psychoses.

Late complications occur on the 10th-12th day of the disease and are caused by the addition of infection. Among them stand out:

- purulent pancreatitis and para pancreatitis;
- pancreatic abscesses,
- subdiaphragmatic abscessiflegmons of the retroperitoneal space;
- abdominal abscesses;
- pancreatic fistulas;
- haemorrhage erosions in the rejection of necrotized tissues.
- pylephlebitis;
- sepsis.
- pancreatic cysts (real, false).

Systemic complications of acute pancreatitis
(By M.Buchler and co-author, 2004)

Organ / System	**Патологические состояния**
Lungs	Pleurisy Atelectasis Pneumonia
A heart	Pericarditis Arrhythmia

Circulatory system	Hypovolemia Shock
Central nervous system	Encephalopathy Patcher syndrome (angiospathic retinopathy, which is characterized by loss of visual fields to complete blindness)
Homeostasis	Hypocalcemia Hyperglycemia Hypokalemia Hypoalbuminemia Hyperlipidemia
Coagulation	Disseminated intravascular coagulation
Kidney	Oliguria Anuria
Other organs and systems (rare complications)	Subcutaneous fat necrosis Arthritis Osteolysis Rhabdomyolysis

Methods that predict the development of complications with acute pancreatitis include the clinical course of the disease, increased body weight and an APACHE II>8 scores in the first 24 hours after hospitalization, C-reactive protein level> 120 mg / L, a decrease in Glasgow score of 3 or more, or organ failure, which is stored after 48 hours after admission to hospital.

Rehabilitation and evaluation of work capacity. The duration of inpatient treatment is determined individually depending on the nature of the disease, the presence of complications. In uncomplicated cases, the average length of stay in hospital is 7-15 days, and the average period of restriction of hard physical labour is up to 2-3 months.

In complicated forms of the disease (presence of postoperative complications) these terms are increased. Permanent disability is possible with the definition of the disability group. Rehabilitation is carried out through outpatient treatment at the place of residence, treatment in rehabilitation hospitals, hospitals, preventive clinics, resorts (Truskavets, Morshin). Dispensary observation is organized at the place of residence or work.

Prognosis. In the oedema form of acute pancreatitis and sterile pancreonecrosis is favourable. In these conditions, conservative therapy is effective in 98-100% of cases. In infected pancreatonecrosis and complications, the lethality rate reaches 50-80%, and in severe complications and their combination 80-100%. Among patients in this group, 72 per cent become disabled.

Prophylaxis. Prevention of acute pancreatitis consists of timely elimination of all etiological factors. To prevent alcohol intake, especially in patients with acute pancreatitis, in the presence of gallstone disease, timely remove the gallbladder with concrements, prevent excessive intake of fat and protein foods.

Self-control questions

1. Anatomical topographic features of the pancreas.
2. Functional features of the pancreas.
3. Aetiology and pathogenesis of acute pancreatitis.
4. Classification of acute pancreatitis.
5. The main clinical symptoms of acute pancreatitis.
6. Laboratory diagnostics of acute pancreatitis.
7. Instrumental methods of acute pancreatitis diagnostics.
8. Prediction of the severity of acute pancreatitis flow.
9. Non-invasive methods of acute pancreatitis diagnostics.
10. Invasive methods of diagnosis and treatment of acute pancreatitis.
11. Differential diagnostics of acute pancreatitis.
12. Conservative treatment of acute pancreatitis.
13. Indications for minimally invasive surgical interventions (endoscopic, laparoscopic, CT-controlled and ultrasound) in acute pancreatitis.
14. Indications and methods of surgical treatment of acute pancreatitis.
15. Preoperative preparation of patients with acute pancreatitis.
16. Postoperative management of patients with acute pancreatitis.

Tests for self-control

1. What's the most common cause of acute pancreatitis?
    a. Pancreatic trauma;
    b. choledocholithiasis;
    c. alcohol abuse;
    d. hyperlipidemia;
    e. infectious mumps.

2. Which method of instrumental diagnosis of acute pancreatitis is most informative?
    a. X-ray examination;
    b. fibrogastroduodenoscopy;
    c. computer tomography;
    d. angiography;
    e. laparoscopy.

3. The first stage of treatment of patients with severe acute pancreatitis is:
    a. Laparotomy and abdominal cavity drainage;
    b. intensive conservative therapy;
    c. Pancreatic resection;
    d. oentopancreatopexy;
    e. laparotomy.

4. The pancreas is located relative to the peritoneum:
    a. retroperitoneal;
    b. mesoperitoneal;
    c. intraperitoneal;
    d. behind lumbar fascia;
    e. Extraperitoneal.

5. Pains most typical of initial acute pancreatitis:
    a. Constant severe banding pain in the upper abdomen,
    b. constant whining in epigastria;
    c. dagger pains in the upper abdomen,
    d. Stomach pains in the stomach
    e. Seizure abdominal pains.

6. The patient had a scheduled resection of the stomach for a chronic stomach ulcer three days ago. The general condition is severe. The tongue is dry. Stomach swelled, soft on palpation, no peristalsis. Symptoms of peritoneal irritation are negative. Urine released in the last 24 hours, 500ml, pink. Diastase of urine 512 IU, amylase of blood 55 mkg / L, total blood bilirubin - 45mmol / L. Formulate a preliminary diagnosis:
    a. Acute pancreatitis
    b. acute intestinal obstruction;
    c. Acute renal failure
    d. intra-abdominal bleeding
    e. choledocholithiasis, mechanical jaundice.

7. The patient complains of severe acute pain in the epigastric region of the girdle, nausea, bloating of the abdomen, general weakness. The patient's condition is severe, marked pale skin and acrocyanosis. Pulse rate 100 beats per minute, tongue dry, covered. The stomach is swollen, painful in the epigastrium and peritoneal region, palpation marks the tension of the abdominal wall muscles. Blood amylase - 63mlg / l. Formulate a preliminary diagnosis:
    a. acute appendicitis;
    b. acute cholecystitis
    c. perforated stomach ulcer;
    d. acute mesenterial vessel thrombosis;
    e. Acute pancreatitis.

8. The patient entered the surgical department with complaints of bandage pain, nausea, vomiting, general weakness, bloating. The disease is associated with taking alcohol surrogates. At examination: mucous membranes are pale, marble colour of skin, cyanosis of lips is marked. Heart rate 126 in 1 minute, weak filling. Positive peritoneal symptoms in the epigastric region. Urinary diastase 1024 IU. Formulate a preliminary diagnosis:

a. acute mesenterial vessel thrombosis;
b. acute pancreatitis
c. acute poisoning by alcohol surrogates;
d. perforated stomach ulcer;
e. acute appendicitis.

9. The patient complains of severe pain in the epigastric region, which is bandaging, nausea, repeated vomiting, which does not bring relief, dry mouth, general weakness. The onset of the disease is associated with eating fatty foods and alcohol. Skin and mucous membranes pale. A / T 100/60mm Hg. Stomach swelled, on palpation soft, painful in epigastria. Liver stupidity preserved. On the review X-ray of the abdominal organs in the left half of the abdominal cavity, a single cup of Cloiber is observed. White blood cells 22.0x109 / l, urine diastase 256 units. Formulate a preliminary diagnosis:
a. perforated stomach ulcer;
b. mechanical intestinal obstruction;
c. food toxicosis;
d. acute pancreatitis
e. acute cholecystitis.

10. The patient became acute after a fatty meal. After 2:00 there were pains in epigastria, repeated vomiting, chills, dry mouth. On the review X-ray of the abdominal organs in the direct projection of the loop of the bowel pneumatically, no free gas under the diaphragm dome. Serum amylase 88 µg/l, leukocytes 18.0x109/l. Urinary diastase 256 units. Formulate a preliminary diagnosis:
a. peritonitis,
b. perforated stomach ulcer;
c. perforation of the hollow organ is covered;
d. acute cholecystitis
e. Acute pancreatitis.

ACUTE COMPLICATIONS OF PEPTIC ULCER AND DUODENAL ULCER
ULCEROUS HEMORRHAGE

Definition. Ulcerous bleeding of the stomach and duodenum is a severe complication, which is associated with the progression of necrosis in the ulcer and the blood intrusion into the gastrointestinal tract (GIT).

Ulcerous hemorrhage occurs more often in the stomach than in the duodenum. The most frequently ulcers of lesser curvature of the stomach bleed and the posterior wall of the duodenum. Ulcerous hemorrhage occurs among 20-22% of patients, mainly after their 35-40. The emergence of such bleeding is a critical situation that threatens the life of the patient and requires the surgeon to determine the causes of bleeding and the choice of tactics for further treatment. Ulcerous bleeding reaches into more than 60% of gastrointestinal bleeding and occupies the first place as the immediate cause of death among patients with ulcer disease.

Anatomical and physiological features of the stomach and duodenum. The stomach is located in the epigastrium, mainly in the left hypochondriac region. It includes the cardiac part, the fundus, the corpus and the janitor.

The cardiac part of the stomach is located at a distance of 5cm from the gastro-esophageal junction, the fundus - above the level of the cardiac opening. The body of the stomach is located between the cardiac part, the fundus and the janitor. At the edge of the stomach and duodenum, the pyloric muscle is located. There are greater and lesser stomach curvature. The stomach wall consists of serous, muscular, mucous and submucosal membranes. The serous membrane forms a ligamentous apparatus of the stomach at the transition to other organs. Between the cardiac part, the fundus of the stomach and the diaphragm there is a gastrophrenic ligament, between the stomach and the liver there is hepatogastric ligament, between the fundus of the stomach and the spleen there is gastrolienal ligament. The stomach is connected with the segmented intestine by gastrocolic ligament. The posterior surface of the fundus, the cardiac part, the corpus of the stomach and the anterolateral edge of the pancreas are connected by the gastro-pancreatic bundle.

The blood supply to the stomach is carried out by the branches of the coeliac trunk (truncus coeliacus), in particular, the left gastric (a. gastrica sinistra), splenic (a. lienalis) and the common hepatic (a. hepatitis communis) arteries. The vessels of the stomach are connected by anastomoses between themselves and with the branches of the superior mesentric artery.

Venous blood flows from the stomach into the portal vein system. In the submucosal membrane of the the cardiac part the veins are anastomosed with the lower veins of the esophagus, forming portacaval anastomoses.

The lymphatic system of the stomach begins with a mucous membrane, there is submucosal, intramural, subserocal-serous lymphatic plexus. There are lymph glands of the first order along the branches of the left gastric, hepatic and splenic arteries; there are lymph glands of the second order along the trunks of these vessels; there are lymph glands of the third order along the abdominal trunk.

The innervation of the stomach is driven by the aid of sympathetic and parasympathetic nerve fibers, which form posterior-gastral nerves and intramural nerve plexus. Synpatic nerve fibers reach the stomach from the abdominal plexus, and the parasympathetic innervation of the stomach is carried out by the branches of the vagus nerves. From the anterior vagus nerve, the hepatic branch extends to the gates of the liver under the diaphragm. Gastric branches grow away from it to the stomach, they form anterior gastric plexus on the anterior surface of the stomach. The fibers of the posterior vagus nerve form the posterior gastric plexus, from which the branches to the abdominal plexus go.

The "criminal nerve of Grassi" branch goes away from the back of the vagus trunk behind the esophagus, recurrence of ulcers often occurs as a result of the preservation of this nerve during vagotomy. Each trunk of the vagus nerve ends with the anterior and posterior Latarjet branches, which form the so-called “goose foot” in the stomach area.

On the basis of the secretory and incretory function the stomach is divided into three glandular zones. Glands of the cardiac part secrete mucus, which provides the nutrition of the masses. Fundal, or major glands, consist of four types of cells. The main cells secrete pepsine proferment - pepsinogen, parietal cells secrete chloride acid and the gastric intrinsic factor, required for the absorption of vitamin B12, additional cells secrete soluble mucus, which has buffer properties. Non-differentiated cells are the source for other mucosal cells.

The janitor's glands secrete soluble mucus and gastrin hormone. D-cells synthesize somatostatin, which is an inhibitor of the hydrochloric acid production and gastrin secretion. There is no clear border between the areas of the janitor and the fundus of the stomach. However, this site is extremely vulnerable to the effects of damaging factors - this is where the ulcer is most often formed.

The mucous membrane of the stomach and duodenum is able to recover from damage quickly enough. This process is mainly performed with the help of their movement from the crypt of the glands along the basement membrane. Secretion of prostaglandins by the gastric mucosa is provided by the main, additional or parietal cells. Prostaglandins contribute to the protection of the mucous membrane of the stomach by inhibiting the activity of parietal cells, stimulating the formation of mucus and hydrocarbonates, increasing the blood supply to the mucous membrane, accelerating cellular regeneration. The use of non-steroidal anti-inflammatory agents predetermines damage to the mucous membrane of the stomach and duodenum, as these agents inhibit the synthesis of prostaglandins.

The main functions of the stomach are motor and secretory, which provide deposit, physical and chemical processing of food. The stomach also performs endocrine, suctorial and exocrine functions. Under normal conditions, almost two liters of gastric juice, containing hydrochloride acid, enzymes (pepsin, cathepsin), biologically active substances and electrolytes are released daily.

The duodenum has a form of horseshoe, bordering the head and partly the body of the pancreas, and is located on the front of the right kidney, crosses the lower cavity of the cava, the spine, the aorta and passes into the intestine. The length of the duodenum is 25-30cm, most of it is not covered by the peritoneum. It is divided

into: the upper part of the level T_{XII} - L_I; descendant part of the level of L_I - L_{III}; the horizontal part at the level L_{III} of the vertebrae.

The length of the upper part of the duodenum is 5-6cm, it is covered by the peritoneum from the three sides and it passes to the descendant part which is 7-12cm long, and is placed extraperitoneally. On the right side of the spine, it passes into the horizontal side which is 6-8cm long, which lays over the spine in a transverse direction and extends to the ascendant part. The ascendant part of a duodenum with the length of 4-5cm forms a curve and passes into the intestine. A common bile duct falls into lower or middle third of the descendant part of the duodenum. The superior mesenteric artery and vein cross the anterior surface of the horizontal duodenum.

Blood supply of the duodenum is carried out by four pancreatic and duodenal arteries, which are branches of the gastroduodenal and superior mesenteric artery.

Lymphatic vessels are located on the anterior and the posterior surface of the duodenum. They widely anastomose with centric medial and mesenteric, hepatic and pre-aortic lymph glands.

The duodenum is innervated by the branches of the abdominal, posterior-mesenteric, hepatic and renal nerve plexuses.

The duodenum is called the hypophysis of the abdominal cavity. It digests all food ingredients with proteo-, lipo- and amylolytic enzymes. It participates in the regulation of motor and secret function of the stomach and intestine, as well as hepatobiliary system and pancreas.

Etiology and pathogenesis. The main cause of superior mesenteric artery ulcerous hemorrhage is an aggravation of peptic ulcer disease with progression of necrosis in the ulcer area. Exacerbation of the pathological process is associated with various factors (stress, poor diet etc.), which cause a disfunction of tissue trophy, increased inflammation, as well as peptic force of gastric juice. The source of bleeding may be an artery, vein or capillaries.

Pathogenic disorders in case of acute gastrointestinal bleeding.

1. Acute blood loss leads to a rapid decrease in venous return and cardiac output.
2. Reflex activation of the sympathoadrenal system leads to generalized vasospasm and centralization of blood circulation.
3. The mechanisms of compensation are supplemented by interstitial fluid inflow to the vascular bed and increased production of aldosterone, which leads to an increase in reabsorption of water and sodium in the renal tubules.
4. Loss of the circulating blood volume(CBV) up to 20% is compensated by the above-mentioned mechanisms without additional correction.
5. If the volume of blood loss exceeds 20% of CBV, there is no treatment, compensatory possibilities of the organism are insufficient or bleeding continues, then hypovolemia, tissue peripheral perfusion decrease, electrolyte disturbances and progressive decrease of immunoresistance of the organism develop.
6. Insufficient blood supply to the periphery leads to oxygen deprivation of tissues, stimulates anaerobic glycolysis and development of metabolic acidosis.

7. There is a hemoconcenration, an increase in blood viscosity, aggregation of formed elements in vessels with subsequent development of microtromboses.
8. Arteriolovenular shunts open, which leads to the capillary bed exclusion from the bloodstream, develops multiple organ failure: lungs, liver and kidneys suffer, and then brain and heart get included in this reaction.

In case of bleeding can be observed:

- hypovolemic shock as a result of CBV reduction;
- renal failure due to decreased filtration and kidney hypoxia;
- liver failure;
- ischemia and myocardial infarction;
- hypoxia and brain edema;
- intoxication with decay products of blood, which effused to the intestine.

The speed and nature of pathological changes in the body during the acute bleeding depend not only on volume but also on the rate of blood loss. In cases of damaging of a large arterial vessel with a high rate of bleeding, the loss of more than 30% of the CBV can be fatal.

Classification. According to the clinical-statistical classification of diseases (ICD-10) hemorrhagic ulcers are divided into:

K25.0 - Acute gastric ulcer with hemorrhage.
K25.2 - Acute with both hemorrhage and perforation.
K25.4 - Chronic or unspecified with hemorrhage.
K25.6 - Chronic or unspecified with both hemorrhage and perforation.
K26.0 – Acute duodenal ulcer with hemorrhage.
K26.2 - Acute with both hemorrhage and perforation.
K26.4 - Chronic or unspecified with hemorrhage.
K26.6 - Chronic or unspecified with both hemorrhage and perforation.
K27.0 – Acute peptic ulcer with hemorrhage, site unspecified.
K27.2 - Acute with both hemorrhage and perforation.
K27.4 - Chronic or unspecified with hemorrhage.
K27.6 - Chronic or unspecified with both hemorrhage and perforation.
K28.0 – Acute gastrojejunal ulcer with hemorrhage.
K28.2 - Acute with both hemorrhage and perforation.
K28.4 - Chronic or unspecified with hemorrhage.
K28.6 - Chronic or unspecified with both hemorrhage and perforation.

<u>According to the reasons</u> of origin (V.D. Bratus, 1980) hemorrhagic ulcers are divided into:

I. Bleeding that is associated with diseases of the stomach and duodenum:
   1. during peptic ulcer;

2. non-ulcer genesis - in case of tumors of the stomach; hemorrhagic erosive gastritis; hernia of the esophageal hiatus; diverticulum of the esophagus and duodenum; Mallory-Weiss syndrome.

II. Bleedings associated with non-gastric diseases – in case of portal hypertension against the background of liver cirrhosis; hemorrhagic diathesis; atherosclerosis and hypertension; hemophilia, aneurysm or mechanical damage to the thoracic part of aorta.

According to the duration of bleeding: acute and chronic.

According to the localization – corresponding to the location of ulcers.

According to the clinical course: profuse, torpid, continuing, stopped bleeding.

According to the classification of O.O. Shalimov hemorrhages are divided into 3 severity levels of blood loss. The first level - easy, with a loss of CBV up to 20% (up to 1000ml for a patient with a weight of 70 kg). The second is moderate, with a loss of 20% to 30% of CBV (up to 1500ml). The third level - heavy, with blood loss of more than 30% of CBV.

In terms of bleeding intensity (according to V.I. Struchkov and E.V. Lutsevich), they are divided into: The ***first level*** - bleeding, which causes minor changes in hemodynamics. The overall condition is satisfactory. The pulse is slightly accelerated; the blood pressure is normal. The CBV deficit is not more than 5% of the proper. Hemoglobin is above 100 g/l. Capillaroscopy: pink background, 3-4 capillary loops with fast homogeneous blood flow. ***The second*** is severe bleeding. General condition is of moderate severity, there is atony, dizziness, unconscious condition. Pale skin. Significant increase in pulse rate. Reduced blood pressure up to 90mm Hg. A patient has a single-shot vomit or melena. Deficiency of CBV is 15% of proper, hemoglobin is 80 g/l. Capillaroscopy: pale background, reduced number of capillaries, fast blood flow, homogeneity is disordered. The ***third*** level - the general condition is serious. Skin is pale, covered with cold sweat. Mucous membranes are pale. The patient yawns and is thirsty. Pulse is quick, filiform. Blood pressure is lowered to 60mm Hg. Deficiency of CBV is 30% of proper, hemoglobin is 50 g/l. Capillaroscopy: pale background, there are 1-2 loops with arterial and venous parts which are difficult to see. Frequent vomiting, melena. Bleeding is accompanied by an unconscious condition. The **fourth** is a profuse bleeding with prolonged loss of consciousness. The general condition is critical, bordering with agonal. The pulse and arterial pressure disappear. The CBV deficit is more than 30% of the proper. Capillaroscopy: gray background, open loops of capillaries are not noticeable.

Clinics. The clinical picture of bleeding depends on the patient's age and concomitant pathology, the duration of bleeding, the level of blood loss. The patient has general progressive weakness, dizziness, maybe loss of consciousness, blood vomiting (haematemesis). With hemin formed in the stomach, vomit may be a "coffee grounds". "Coffee grounds" is the most characteristic symptom of bleeding, it can be single-shot and occasional, indicating the continuation of bleeding. In case of stomach ulcer, "coffee grounds vomiting occurs more often than during a

duodenal ulcer. Before the bleeding, sometimes there is an increase in the pain that disappears after the onset of bleeding (the Bergman symptom). In case of a profuse bleeding, low-level blood is released, not "coffee grounds". A possible sign of bleeding is a black tar-like dejection with bad odor (melena). Melena often accompanies vomiting with blood, is more characteristic of bleeding from the duodenum, but often occurs also with highly located ulcers. In case of profuse duodenal bleeding and rapid blood flow through the intestine, fecal masses can be of a liquid consistency, dark cherry color. During moderate bleeding, fecal masses are formed, of a normal consistency and black color. Feces of black color may be observed while taking drugs with iron, bismuth, absorbent charcoal, while eating blood sausage etc.

On examination, the patient is pale, scared, lying mostly on the back, afraid of moving, so as not to provoke bleeding, frequent breathing is noted. The face is covered with cold sweat, pallor of the face and visible mucous membranes is seen. The patient complains of dryness in the mouth. Pulse is frequent, sometimes it is filiform, sometimes arrhythmic, arterial pressure is lowered. During examination, the stomach is hollow, and during severe bleeding - it is swollen as a result of the blood decay in the intestine and intoxication. During auscultation of the stomach – diastaltis is increased, which is connected with irritation of the intestines with blood. During palpation the moderate pain in the projection of ulcers is determined. Deep palpation cannot be performed, it can provoke bleeding. During percussion the symptom of Mendel is determined by the pain in the projection of ulcers on the anterior abdominal wall. A compulsory survey method for such patients is a digital rectal examination. In this case, there are stomach-shaped dejecta (melena), and also an anal fissure, tumors, hemorrhoidal piles can be found. In order to diagnose bleeding and perform a gastric lavage nasogastric tube is administrated.

Diagnosis

Laboratory examination methods:

1. General blood test
2. General urine test
3. Determination of blood group and Rh-factor
4. Coagulation test
5. Platelet count
6. Coagulation time
7. Biochemical blood test
8. ECG

Laboratory studies show a decrease in hemoglobin, the number of red blood cells, hematocrit. At the beginning of the bleeding, these indices do not reflect the true extent of it, the decrease is seen only in a few hours. First of all, there are changes in blood hematocrit in 20-30 minutes. There is anisocytosis, poikilocytosis, there may be neutrophilic leukocytosis as well. A decrease in the level of total protein is also observed, an electrolyte metabolism disorder, an increase in the amount of urea in the blood because of the reduction of the function of the kidneys.

Approximately, the volume of blood loss can be defined by determining the Allgower shock index, which is calculated as the ratio of the pulse rate to the level of systolic blood pressure. Normally, the Allgower index is equal to 0.5, but during the internal hemorrhage it increases.

Classification attributes of the blood loss volume severity:

Estimation of the blood loss volume severity. The degree of blood loss severity is a complex concept that, based on laboratory and clinical parameters, characterizes the volume of blood loss, the intensity of bleeding and the patient's body's response to blood loss.

Endoscopic methods are the most important out of the instrumental methods of examination, more specifically, the fibrogastroduodenoscopy (FGDS), which makes it possible to determine the source of the bleeding, the cause of its occurrence, determine whether the bleeding has stopped, perform endoscopic hemostasis, and assess the risk of bleeding recurrence.

Indication for conduction of FGDS are signs or a suspicion of acute gastrointestinal bleeding (AGIB). Contraindications: absolute - agonal condition; circumstantial – acute cerebrovascular disease, unstable hemodynamics (risk of asystole). Timely performance: primary FGDS - in the first 2 hours in case of the severe blood loss; in the first 24 hours, if the bleeding already occurred; secondary FGDS – in case of recurrence of the AGIB and the source is unidentified, monitoring of the hemostasis degree (2-3 days).

In 1987, J. Forrest offered an endoscopic classification of ulcer bleeding:

Class I - active hemorrhage:

a. Active spurter;
b. Active oozing.

Class II - signs of recent hemorrhage:

a. Non-bleeding visible vessel;
b. Adherent clot;
c. Flat pigmented hematin on ulcer base.

Class III - Lesions without active bleeding, clean-based ulcer.

During the massive bleeding, endoscopy is performed to investigate the bleeding source localization to select surgical access. After establishing the source of the hemorrhage and its intensity, it is necessary to evaluate the possibility of stopping of blood with the help of an endoscope.

A stable hemostasis is characterized by the absence of blood in the stomach and duodenum, ulcer base is covered the fibrin, the lumen of the vessel of the ulcer emerging above the hollow is closed by the clot of a white color. Signs of unstable hemostasis are the presence of light blood, blood clots in the stomach, quaggy blood clot of a dark color over the ulcer. Absolute sign of unstable hemostasis is the extravasation from the vessel.

Due to the wide adoption of endoscopy, the role of X-ray methods of examination reduced significantly. In some cases, selective angiography of the

branches of the abdominal aorta, celiacography, is used for the gastrointestinal bleeding diagnosis. If the bleeding continues, the contrast is determined in the lumen of the gastrointestinal tract.

According to clinical signs, blood loss can be mild, moderate, severe and extremely severe.

Clinical and laboratory signs of blood loss of different degrees of severity

Criteria	**The degree of hemorrhage involves**			
	mild	**moderate**	**severe**	**extremely severe**
1	2	3	4	5
CBV deficit (% of proper)	10 – 20 Up to 1000*ml	21 – 30 1000-1500*ml	31 – 40 1500-2000*ml	41 – 70 2000-3500*ml
Pulse rate (beats per min.)	Up to 90	90 - 110	110 - 120	>120
Blood pressure (mmHg)	>120	120 - 80	80 - 70	<70
CTV (mm w.g.)	120 - 80	80 - 60	<60	0
Shock index (P/BP)	054 - 078	0,78 – 1,38	1,38 – 1,5	>1,5
Erythrocytes ($x10^{12}$/l)	5,0 – 3,5	3,5 – 2,5	2,5 – 2,0	<2,0
Hemoglobin (g/l)	120 – 100	100 – 80	80 – 60	<60
Hematocrit (%)	44 - 38	38 - 32	32 - 22	<22
Diuresis (ml/h)	50 - 60	40 - 50	30 - 40	<30

Notes:

- a collaptoid state during the bleeding indicates a blood loss of more than 30% of the CBV;
- surgical approach in case of severe and extremely severe blood loss is the same;
- laboratory parameters are correct in case of bleeding that lasts more than 12 hours;

*values are given in ml. for a patient weighing 70-80 kg.

The determination of the high- and low-risk groups is an objective criteria of bleeding recurrence emersion and death:

- high-risk group - quick methods of diagnosis and treatment,
- low-risk group - examination, follow-up examination, early discharge.

The risk of a bleeding recurrence

Criteria	0 points	1 point	2 points	3 points
Age	< 60	**60-79**	**>80**	-
Signs of shock	None	Orthostatic tachycardia. Orthostatic hypotension.	Pulse> 100 / min Blood pressure <100mm Hg	-
Co-morbidity	None	None	Cardiovascular insufficiency. Other organ dysfunction	Renal / hepatic insufficiency. Metastatic tumor
The cause of bleeding	Mallory-Weiss syndrome. There are no signs of the gastrointestinal bleeding.	Other causes	Malignant neoplasms	-
Signs of "fresh" bleeding	Missing.	-	There is blood in the lumen. Ajar vessel is bleeding	-

Note:

The number of points:

0-2 – 3-5% of hemorrhage recurrence, mortality rate of 0-0,2%
3-4 – 11-14% of hemorrhage recurrence, mortality rate of 3-5%
5-6 – 24-33% of hemorrhage recurrence, mortality rate of 11-17%
7-8 – 44% and more of hemorrhage recurrence, mortality rate of 27-40%

Differential diagnosis. Differential diagnostics of bleeding during a stomach ulcer and a duodenal ulcer is carried out with:

1. stomach diseases, complicated bleeding accompanying gastric and esophageal cancers, diverticula, Mallory-Weiss, Zollinger-Ellison syndromes, stomach polyposis, erosive gastritis
2. bleeding of non-ulcer nature with diaphragmatic hernia, bulge of the esophagus veins, atherosclerosis of the vessels of the abdominal aorta, telangiectasia, accompanying blood diseases and coagulation system disease

Treatment. The tactic of treating such patients depends on the cause, the degree of blood loss, the duration of bleeding, its dynamics, the continuation and stopping of bleeding.

Forrest I-II class: endoscopic and surgical hemostasis. Medicinal therapy: intensive treatment of blood loss - restoration of CBV, procoagulants (etamsylate, dicinone, ACA, tranexamic acid), antisecretory therapy - proton pump inhibitors (PPIs).

Forrest III: *endoscopic and surgical hemostasis are not justified.* Drug therapy: restoration of CBV, PPI.

Principles of conservative treatment hemorrhages from the upper gastrointestinal tract are emergency hospitalization, restoration of CBV, hemostatic therapy, hemotransfusion.

All patients with a suspicion of gastrointestinal bleeding should be hospitalized to a surgical department, and in case of their severe condition, to the resuscitation department.

Conservative treatment of hemorrhage is justified in case of:

- doubtful diagnosis;
- bleeding of the first level, which is stopped;
- stopped bleeding, when there is no data about its recurrence;
- the absence of conditions for operative treatment;
- the presence of severe accompanying pathology.

Depending on the level of bleeding, the following things are justified:

1. Strict bed confinement, bowel rest.
2. Constant gastric intubation.
3. Cardiotherapy, Oxygen Therapy.
4. Autotransfusion (a bandage of the legs), low head position.
5. Heat waste prevention.
6. Catheterization of two veins, one of which is central.
7. The monitoring of central venous pressure (CVP) allows to adjust the amount of fluid injected, detect recurrence of bleeding and it is important in case of massive bleeding, especially among elderly patients.
8. Transfusion of erythrocytic mass and plasma. Introduction of hemostatic drugs. Hemostatic (procoagulant) therapy does not stop the bleeding but promotes hemostasis! Ethamsylate (dycinone) (5-10ml 12.5%), tranexamic acid (10-15 mg / kg), α -aminocaproic acid (100-200ml 5%), cryoprecipitate, frozen plasma, calcium chloride, vicasol, platelet concentrate.
9. Intravenous administration of protease inhibitors (contrykal, trasylol).
10. Gastric secretion reduction (H_2 receptor blockers, proton pump inhibitors).
11. Inhibition of fibrinolytic activity (aminocaproic acid per os).
12. Guided hypotension.
13. Recovery of CBV: stream infusion of crystalloid solutions up to three volumes of blood loss. Early introduction of colloids - only during reanimation measures: they fill the intravascular sector, reinforce the venous return to the heart. Rheological substances (rheopolyglucin, 5% albumin solution), improving central hemodynamics, create conditions for the normalization of

microcirculation. Tachycardia and arterial hypotension indicate a low CBV, in this case it is necessary to enter up to 1 liter of the colloidal solution inwards, then slowly continue infusion to obtain blood products.

14. Local hypothermia, including gastric lavage with cold water.
15. If the bleeding stops - a Meulengracht's diet is administrated (sour cream, raw chilled eggs).
16. Local bleeding stop. Using a nasogastric tube, a mixture of 4.0ml of norepinephrine, 50.0ml of physiological saline sodium chloride solution, 50.0ml of aminocaproic acid, 200.0 g of thrombin are injected.
17. In the time of FGDS conduction, the bleeding can be stopped locally.

Medical endoscopy in case of bleeding into the lumen of the gastrointestinal tract is used to: temporarily or permanently stop bleeding; influence on the pathological process to prevent its recurrence in the coming hours. In the presence of bleeding from a chronic or acute ulcer of the stomach, the duodenum it is used for: treating the source of bleeding with Adrenergic agonists, diathermocoagulation, electrocoagulation, cryoagulation, laser coagulation; clips appliance on the vessel, glue application. Also combined methods are used: a combination of injection hemostatic with coagulation or clipping.

Recurrence of acute gastrointestinal bleeding after treatment endoscopy: frequency - 15-20%, in the first 24 hours - 80%. Evaluation of the necessity of surgical hemostasis: repeated endoscopic hemostasis and monitoring using a nasogastric probe, repeated recurrence demands a surgical treatment.

Cryocoagulation – the place of bleeding is treated with cold, due to the introduction of chlorethyl to the probe, led to the bleeding area. During the cryoelectrocoagulation, the bleeding area has to be washed with chladone. During conducting diathermocoagulation, the electrode is led to the blood vessel and at the expense of high-frequency current, tissue coagulation is performed. Laser coagulation is the most effective, because it is possible to regulate the depth of coagulation. These methods are effective in 80-90% of cases, however, 20% of patients have the recurrence of bleeding.

Assessment of hemostasis stability. Signs of unstable hemostasis are:

- clinical and laboratory parameters: the collaptoid state of the patient in case of vomiting and the presence of melena;
- the presence of laboratory signs of increased blood loss;
- recurrence of bleeding based on data from clinical and dynamic laboratory observation;
- Forrest's endoscopic signs of hemostasis stability.

The presence of a clinical or endoscopic sign of instability of hemostasis, as well as their comparison, is the basis for a general conclusion about the hemostatic instability.

Indications for hemotransfusion and its volume determination depends on the degree of blood loss and amount of time lasted after the onset of bleeding. Hemotransfusion should be performed among patients with a clinical picture of hemorrhagic shock, ongoing bleeding, tachycardia and systolic pressure of less than 80mm Hg, the rate of hemoglobin falling below 80 g/l during the continued bleeding (unstable hemostasis) and below 60 g / l in case of stable hemostasis. It gives a positive effect during continued bleeding, it is a prevention of its recurrence.

Rules for replenishment of blood loss and correction of the CBV deficit

The aim of blood loss compensating is to replenish the deficiency of the liquid part of the blood to improve microcirculation, perfusion of organs and tissues, and oxygen supply and to restore the red blood cell deficit.

Delay in the initiation of infusion-transfusion therapy after the diagnosis of bleeding has been stated is unacceptable!

For adequate therapy among patients with acute gastrointestinal bleeding it is mandatory to provide reliable venous access - central venous catheterization in case of severe and extremely severe hemorrhage, regular monitoring of CVP to correct the rate of infusion. After transfusion, it is necessary to monitor the red blood cells and hemoglobin. Infusion-transfusion therapy is performed depending on the amount of blood loss.

Principles of infusion-transfusion therapy of acute blood loss

Level*	Blood loss volume			Total volume of transfusion (in% of	Infusion-transfusion therapy						
	Deficit of CBV (%)	% of body weight	Blood loss (ml) (for a patient with a body		Crystalloids (physiological solution, Ringer	Colloids			Erythrocyte mass	Cryoprecipitate **	Thromboconcentrate ***
						synthetic	natural				
						HES III - II, Gelofusine	plasma	albumin			
I	10 - 20	1-1,5	500,0-1000,0	200-300 (up to 2,5 л)	10-15 ml/kg	10ml/kg	-	-	-	-	-
II	21 - 30	1,5 - 2,0	1000,0 - 1500,0	200 (up to 3 liters)	10ml/kg	10ml/kg	5-10 ml/kg	-	5-10 ml/kg	-	-
III	31 - 40	2,0 - 2,5	1500,0 - 2000,0	180 (up to 4	7ml/kg	10ml/kg	10-15 ml/	200 ml	15-20ml/kg	-	-

				liters)			kg				
IV	41 - 70	2,5 - 3,6	2000,0 - 3500,0	170 (up to 5 liters)	7-10ml/ kg	10-20ml/kg	15-20 ml/ kg or >	200 ml or >	30 ml/kg or >	5-10 doses	Selectively 4-10 units

Note:

* in the blood loss of III-IV levels, it is desirable to add an artificial carrier of oxygen perftoranum of a dose of 2.5-5ml/kg of body weight

** the volume of 1 dose of cryoprecipitate is 15ml

*** 1 unit of thromboconcentrate contains not less than $0{,}5\times10^9$ of platelets, one therapeutic dose contains from 4 to 10 units

Algorithm for infusion-transfusion therapy calculation

1. Calculate the proper CBV, which is: for men - 70ml/kg; for women - 60ml/kg; for pregnant women - 75ml/kg of body weight.
2. Calculate the volume of blood loss or CBV deficiency by the Moore formula:

$$BL = CBVproper \text{ x} \frac{\text{Ht proper} - \text{Ht actual}}{Ht\ proper}$$

**Where BL is a blood loss, Ht_{proper} is a normal hematocrit (45% for men, 42% for women), Ht_{actual} is a factual hematocrit, determined after the bleeding stopping and the stabilization of hemodynamics deficiency.*

3. Determine the percentage of CBV deficit from the proper CBV:
4. Depending on the percentage of CBV deficiency, determine the level of blood substitution and the total volume of infusion.
5. Using the table, make an infusion-transfusion therapy program.

Carrying out the infusion-transfusion therapy under the conditions of acute gastrointestinal bleeding:

- infusion therapy is begun with solutions of electrolytes, through the second venous access the preparations of gelatin, hydroxyethyl starch is entered;
- simultaneously it is necessary to order and freeze one-group plasma and prepare the erythrocytic mass, determine its suitability for transfusion, check the blood group and make tests for group and individual compatibility;

Transfusion therapy should be performed taking into account the changes in CBV during the different periods after the bleeding. In the first 2 days, there is hypovolemia due to the deficiency of CBV and the volume of circulating plasma, therefore transfusion of whole blood and blood substitutes is justified. On the 5th day the oligocythemic hypovolemia is observed, therefore it is expedient to transfuse

the red blood cell mass. After 5 days, transfusion of red blood cells and whole blood is indicated. Infusion therapy should be controlled by measuring central venous pressure. Treatment of a seriously ill patient should be carried out in conditions of emergency departmen. After the transfusion of citrate blood, serum calcium levels may decrease, so after the transfusion of every 400ml of blood, 10ml of calcium gluconate solution administration is required.

Compensation of blood loss of IV level begins with intravenous administration of solutions of electrolytes by stream infusion, up to 250-300ml / min, after the stabilization of hemodynamic parameters - up to 30ml / min. In the first venous access, solutions of electrolytes are introduced at a rate of 7ml/kg of the patient's body weight: sodium chloride solution 0.9% or Ringer's solution. Frozen plasma is introduced at the rate of 10-15ml/kg, up to 1000ml; solution of 10% albumin. In the second venous access colloidal plasma substitutes infusion at the rate of 7ml/kg of the patient's body weight is introduced: preparations of gelatin 500ml, or preparations of hydroxyethyl starch 500ml; erythrocyte concentrate at the rate of 15-20ml/kg, up to 1000ml. It is necessary to prevent volume overload, which leads to an increase in blood pressure, pulmonary or cerebral edema.

Control of conservative treatment is performed with the definition of pulse, blood pressure every 30 minutes, and CVP - hourly. The erythrocytes, hemoglobin, hematocrit hourly blood test is performed. To control the bleeding gastric intubation and diuresis control are performed due to permanent catheterization of the bladder.

Evacuant enemas are used to remove blood from the intestine.

Surgical treatment. Surgical approach for gastroduodenal bleeding includes: determining the indications for surgery, the duration of the operation, the choice of the method of surgery.

Absolute indications for surgery: profuse bleeding that continues; impossibility of conducting / ineffectiveness of endoscopic hemostasis. Relapse of bleeding after it stops. The combination of bleeding and perforation.

The ***nature*** of surgical interventions for gastrointestinal bleeding:

a. emergency operation - performed within 2 hours after delivering the patient to the hospital with the presence of intense bleeding and with the state of the patient at the stage of decompensation of vital functions. Such patients come to the operating room from the hospital admissions unit after the primary diagnosis, and all diagnostic and therapeutic measures connected with the stabilization of vital functions of the body are performed during a brief pre-operative preparation and the operation itself;
b. urgent operation - performed within 2-24 hours after patient's admission to hospital, with the state of the patient at the stage of subcompensation or relative compensation, with continued bleeding, or with unstable hemostasis or recurrence of bleeding;
c. delayed operation - is performed at unstable hemostasis after replenishment of blood loss and stabilization of vital functions within 24-72 hours after the patient enters the hospital.

The ***character and volume*** of operative interventions is determined by the following circumstances: the nature of the pathological process, which was a cause of bleeding; the patient's condition; qualification of the surgical brigade; maintenance of operational intervention.

1. Gastroduodenotomy with underruning of ulcers and erosions, which are the source of bleeding, and with conduction of stem or selective vagotomy (except for the extremely severe condition of the patient) is performed in case of bleeding from acute ulcers of the stomach, duodenum, as well as during acute hemorrhagic gastritis.
2. In case of ulcers of the pyloric part of the stomach and duodenum, the ulcer excision or its exteritorization with underruning of the bleeding vessel, are performed. Together with pyloroduodenoplasty and under the stable state of the patient, one of the types of vagotomy. Stem vagotomy - the intersection of the trunks of the vagus nerves above the discharge of the hepatic and abdominal branches. Selective vagotomy - vagus nerves intersect below the discharge of these branches. Selective proximal vagotomy (SPV) - proximal branches of the body and the bottom of the stomach intersect.
3. In case of bleeding from chronic ulcer of the stomach an adequate volume of intervention would be the resection of the stomach, and in case of severe condition of the patient it is possible to perform the incision of the ulcer;
4. When there are doubts about the benign nature of ulcers, the operation of choice should be resection of the stomach on cancer principles;
5. If it is impossible to provide all conditions for a typical resection of the stomach, a segmental resection with excision of the ulcer or gastrectomy with underruning of a bleeding ulcer and a biopsy of the ulcer is performed.

Premedication of the patient for the operation lasts up to 2 hours, in some cases, profuse bleeding is practically absent, and the volume of preoperative preparation depends on the degree of blood loss.

Operative treatment of gastroduodenal bleeding is performed under endotracheal anesthesia. Surgical access – superior-medial laparotomy. A review of the stomach and intestines is carried out to detect blood in them. In case of the absence of data indicating viruses, direct gastrostomy is performed, which begins at a distance of several centimeters from the pylorus, a revision of the stomach and duodenum is carried out.

After establishing a source of bleeding, before choosing the optimal method of surgery, urgent measures are needed to supply local hemostasis. To do this, during gastrostomy, it is necessary to underrun, ligate, coagulate blood vessels or the corresponding area of the mucous membrane, apply an 8-shaped stitch to the ulcer.

In case of a duodenal ulcer underruning of a vessel that bleeds (or excision of an anterior wall ulcer) is performed in combination with pyloroplasty and vagotomy. In case of combined ulcers of the duodenum and stomach, vagotomy with pyloroantrumectomy are carried out. For extremely severe patients, palliative operations are justified: underruning a bleeding vessel from the mucosa, wedge-

shaped excision of an ulcer, embolization of bleeding vessels, underruning the stomach wall along the perimeter of the ulcer with the following imposition of serous-muscular sutures, suturing of a bleeding stomach and duodenal ulcer.

A patient with a short history of an ulcer disease or without it, with a bleeding of the I-II levels, after its stop conservative therapy is shown.

In the **postoperative period**, the basic principles of treatment are:

1. Restoration of CBV, substitution therapy.
2. Fighting post-hemorrhagic anemia.
3. Improvement of rheological properties of blood.
4. Prevention or treatment of hepatorenal insufficiency.
5. Deintoxication therapy.
6. Antiulcerous therapy, which includes blockers of gastric secretion, proton pump drugs, antacids and cytoprotectors.
7. Decompression of the stomach to restore peristalsis and passage of intestinal contents.
8. Fighting paresis of the intestine, a cleansing enema.
9. Vitamin therapy.
10. Symptomatic therapy.
11. Antibacterial therapy according to indications.
12. Prevention of cardiovascular complications, complicated by the lungs, thromboembolic complications, especially among old and elderly patients.

The patient's diet starts from the 2^{nd}-3^{rd} day, and from the 7^{th}-8^{th} day there should be 5-6 meals a day. In the absence of complications, seams are removed on the 7^{th}-8^{th} day, and in 10 days in case of weakness of the patient. In the uncomplicated course of the postoperative period, ambulatory treatment with the continuation of antiulcerous therapy are prescribed.

Patients with bleeding from the superior gastrointestinal tract with blood loss of a severe or moderate degree sign out of the hospital on the 12^{th}-14^{th} day after the operation and the control endoscopic examination, to be under the supervision of a surgeon and gastroenterologist of the clinic later, to solve the issues of the subsequent medical treatment. Patients, who went through the acute gastrointestinal bleeding of any origin with a blood loss of medium or mild severity require a control endoscopy on the 12^{th}-14^{th} day after hospitalization to decide an issue of further tactics.

Complication. Early postoperative complications include: bleeding in the gastrointestinal tract and abdominal cavity, disorder of the anastomosis patency, the anastomosis seam failure, peritonitis, pancreatitis, complications of the cardiovascular and respiratory systems.

Working capacity examination includes staying in the hospital for 14-18 days after the operation to cure the ulcer disease. The term of temporary disability - 45-60 days with the transition to work, which is not connected with physical activity.

In the postoperative period, patients have the regular medical check-up. They must find a proper employment, adhere to the diet. The transition to disability is established by the DDS, which depends on the operation volume and complications.

Prognosis. The prognosis of acute gastrointestinal bleeding depends on the causes, the degree of blood loss and patient's state: their age, concomitant disease. The risk of an unfavorable result is always extremely high.

Self-control questions

1. Etiology and pathogenesis of ulcerous bleeding.
2. Methods of examination of patients with the ulcerous bleeding.
3. Classification of the ulcerous hemorrhage.
4. Forest classification of the ulcerous bleeding.
5. Clinical picture of the ulcerous hemorrhage.
6. Differential diagnosis of the ulcerous bleeding.
7. Features of the flow depending on the degree of its activity.
8. Features of the clinical course of the hemorrhage.
9. Features of the clinical course of stomach ulcer bleeding that has stopped.
10. Choosing a method of a bleeding stopping.
11. Indications for conservative treatment of the ulcerous bleeding.
12. Conservative treatment of the ulcerous bleeding.
13. Methods of surgical interventions for the ulcerous hemorrhage.
14. The choice of the method of surgical intervention, depending on the patient's condition, the intensity of the bleeding, the degree of blood loss, the localization of the ulcer.
15. Complications in the postoperative period and their treatment, the features of management of patients with ulcerative bleeding of the early and late postoperative period.

Tests for self-control

1. What are the signs for the determination of the blood loss amount in case of the acute gastrointestinal bleeding?
   a. the globular volume;
   b. the CBV indicators;
   c. the number of red blood cells, hemoglobin, hematocrit;
   d. the central venous pressure;
   e. the arterial pressure, pulse, state of the patient.

2. A 47-year-old patient was hospitalized with complaints of general weakness, dizziness, temporary loss of consciousness. From the anamnesis it is known that periodically, for four years, patient has complained about the pain in the epigastrium, especially at night, the heartburn, to reduce which he used soda. Has not been examined. Two weeks ago, he was disturbed by the pain that disappeared on its own two days ago. On the day of admission there was a severe weakness, nausea, dizziness, the bowel movement was of a black color

twice, tarry feces and the patient fainted twice. General blood analysis: hemoglobin - 96 g/l, leukocytes – $16*10^9$/l. What is the previous diagnosis?
a. acute pancreatitis;
b. abdominal form of myocardial infarction;
c. bleeding from the esophageal phlebectasia;
d. stomach cancer with bleeding;
e. bleeding from duodenal ulcer.

3. Identify the most informative method of diagnosis of bleeding from ulcers of the stomac:
a. ultrasound examination;
b. computed tomography;
c. laparoscopy;
d. gastroduodenoscopy;
e. X-ray of the stomach.

4. A 43-year-old patient, came to the department with complaints of weakness, dizziness, nausea, vomiting with a "coffee grounds", melena. In the anamnesis, there are periodic nocturnal, "hunger" pains in the superior abdomen, nausea, heartburn. Heart rate - 110 per 1 min., blood pressure - 90/60mm Hg., hemoglobin - 78 g/l, red blood cells - $2,6*10^{12}$/l, leukocytes - $9,8*10^9$/l. Formulate a preliminary diagnosis.
a. Peptic ulcer disease, acute gastrointestinal bleeding;
b. stomach cancer, acute gastrointestinal bleeding;
c. Mallory-Weiss syndrome;
d. hemorrhagic gastritis;
e. duodenal ulcer, acute gastrointestinal bleeding.

5. The patient complains about the general weakness, dizziness, pain in the epigastrium, nausea, and feces of black color. Got sick two weeks ago, when the abdominal pain appeared. Three days passed as a general weakness, ailment and terry feces occurred. After that, the pain slightly decreased. In the past, he felt abdominal pain fasted and at night. The abdomen is not swollen, symmetrical, takes part in the act of breathing. During the palpation it is soft, somewhat painful in the epigastrium. The liver is near the edge of the arch of the ribs, the spleen is not palpated. Formulate a preliminary diagnosis.
a. tumor of the stomach, hemorrhage;
b. bleeding of the ulcerous etiology;
c. Mallory-Weiss syndrome;
d. hemorrhagic gastritis;
e. bleeding from the varicose veins of the esophagus.

6. Indicate the most common cause of acute gastrointestinal bleeding:
a. colon pathology;
b. cirrhosis of the liver;

c. esophageal disease;
d. portal hypertension;
e. gastroduodenal ulcers.

7. A 43-year-old patient has been suffering from ulcer duodenum for many years. Two days ago pain in the abdomen subsided, but there was weakness, dizziness. On that day, after getting up from the bed, there was a dizziness. During the examination: pale skin, palpation is painful in the epigastric area. What complication did the patient have?
   a. pyloric stenosis;
   b. Malignancy of the ulcer;
   c. bleeding from the ulcer;
   d. Penetration of the ulcer;
   e. perforation of the ulcer.

8. A 50-year-old patient complains of severe general weakness, dizziness, and rare dejection of the black color. Suffers from illness during a day after eating spicy food and\or drinking alcohol. Objectively: the condition is severe, the skin is pale, moist. Pulse is 120 beats per minute. BP is 90/60mm Hg. Tongue is covered, dry. Abdomen is mild, moderately painful in the epigastrium. Peristalsis is enhanced. Which diagnosis is most probable?
   a. gastrointestinal bleeding;
   b. poisoning by alcohol surrogates;
   c. acute pancreatitis;
   d. food toxic infection;
   e. nonspecific ulcerative colitis.

9. A patient has peptic ulcer disease, during the regular examination, pain in the abdominal region disappeared. At the same time, the patient noted the appearance of liquid black feces. What do you think the reduction in pain is typical for?
   a. perforation of the ulcer;
   b. pyloroduodenal stenosis;
   c. penetration into the pancreas;
   d. malignancy of the ulcer;
   e. bleeding from the ulcer.

10. The patient was hospitalized to the clinic with the fifth ulcerous bleeding over the past two years. Bleeding was stopped endoscopically. There is anemia of a moderate severity. There is an ulcer in the duodenal bulb up to 2.0cm in diameter. Identify treatment policy:
    a. operative treatment;
    b. antiulcerous ambulatory treatment;
    c. coagulation of ulcer with argon plasma;
    d. antiulcer stationary treatment;

e. anti-ulcer and physiotherapeutic treatment.

11. According to the Forrest classification of ulcerous hemorrhage, an ulcer with a blood clot, when the clot is fixed is:
    a. Forrest Ib;
    b. Forrest IIc;
    c. Forrest IIa;
    d. Forrest Ia;
    e. Forrest IIb.

12. Indicate the average degree of blood loss on the upper limit of the CBV deficit:
    a. up to 30%;
    b. up to 25%;
    c. up to 20%;
    d. up to 5%;
    e. up to 15%.

13. The patient was hospitalized being in an extremely difficult condition in 14 hours after the onset of bleeding, which was signed by blood and melena vomiting. The state is confusional. BP is 60/40mm Hg., the pulse is 128 beats per minute. The blood analysis shows red blood cells amount of $2,6*10^{12}$/l. Determine the rational management for the patient:
    a. complex anti-ulcer therapy;
    b. antishock therapy, cryotherapy;
    c. Emergency laparotomy;
    d. antishock therapy, endoscopy in 2-3 hours;
    e. endoscopic hemostasis, anti-ulcer treatment.

14. To measure the patient's condition and volume of blood loss, the shock index of Algover is used. Specify its definition:
    a. the ratio of systolic pressure to heart rate;
    b. the ratio of heart rate to systolic pressure;
    c. the ratio of pulse pressure to central venous pressure;
    d. the ratio of blood pressure to central venous pressure;
    e. the ratio of the magnitude of central venous pressure to arterial pressure.

15. A 62-year-old patient entered the hospital with gastrointestinal bleeding. Using the endoscopy, bleeding from ulcers of the stomach was found. Electrocoagulation and arteriography with embolization were ineffective. What is the next step in the treatment?
    a. a planned operation;
    b. an urgent operation;
    c. a massive transfusion;
    d. high doses of antibiotics;
    e. repeated endoscopic hemostasis using diathermocoagulation.

ACUTE COMPLICATIONS OF PEPTIC ULCER AND DUODENAL ULCER
PERFORATED ULCER OF STOMACH AND DUODENUM

Definition. Perforation of the ulcer is a life-threatening complication of the peptic ulcer which is a transverse defect in the wall of the stomach or duodenum with the contents getting to the abdominal cavity and the developing of peritonitis.

Perforation may occur on the background of exacerbation of the chronic course of peptic ulcer and acute ulcer, even observed among patients with no clinical signs (cases of asymptomatic course). Perforated ulcers are thought to occur in the youth and adulthood, and it's more common among men.

Etiology and pathogenesis. Among the factors that contribute to perforation of the ulcer of the stomach and duodenum, the most likely are physical stress with an increase in intra-abdominal pressure, overflow of the stomach with food due to the poor diet affecting by the use of alcohol.

During the perforation of the ulcer, the contents of the stomach or duodenum enter the abdominal cavity, which primarily carries out a chemical irritation of the peritoneum. Depending on the location of the perforation, the higher the concentration of hydrochloric acid in this content, the degree of burn wound of peritoneum is more pronounced. Therefore, the perforation of the ulcer of the stomach leads to the development of general serofibrinous peritonitis in a shorter period of time than in the case of duodenal ulcer. In addition, pain in the beginning of the disease is similar to the pathogenesis of shock and is more intense in case of the perforation of the stomach than in case of the perforation of duodenal ulcer.

Classification.

According to the international classification of diseases (ICD-10) there are:

K25 - Gastric ulcer.

K25.1 – Acute with perforation.

K25.2 – Acute with both hemorrhage and perforation.

K25.3 - Acute without hemorrhage or perforation.

K26 – Duodenal ulcer.

K26.1 – Acute with perforation.

K26.2 – Acute with both hemorrhage and perforation.

K26.3 - Acute without hemorrhage or perforation.

Classification of perforated ulcers

1. By etiology:
   1.1. perforation of chronic ulcers.
   1.2. perforation of acute ulcer (hormonal, stressful)
2. By localization:
   2.1. perforation of the ulcer of the stomach: small curvature, front wall, back wall;
   2.2. Perforation of the ulcer of the duodenum: the anterior wall, the posterior wall.

3. By progress:
    3.1. perforation to the free abdominal cavity.
    3.2. perforation is hidden.
    3.3. perforation is atypical.

The perforations of the anterior wall of the duodenum and the prepyloric part of the stomach are most common. And ulcers of the anterior wall of the stomach are rarely complicated by perforation.

In cases of perforation of the ulcer in the free abdominal cavity (open perforation), the flow of the contents of the stomach or duodenum into the free abdominal cavity with the development of peritonitis occurs. When after perforation of ulcers in the near future there was a cover of a perforation aperture with a pellicle of the fibrin, a fold of the mucous membrane, a neighboring organ (a liver, a large cap, a pancreas), or even a piece of solid food from the inside of the stomach, it would be a covered perforation. The ulcers of the posterior wall of the stomach can perforate in the omental sac, as they are similar to a covered perforation in clinical flow. Also perforation of several ulcers of different localization simultaneously is possible.

Perforation of the ulcer with the contents going not in the abdominal cavity, but in the retroperitoneal space, large or small omentum, hepatoduodenal ligament etc. is atypical.

Clinical picture of the perforated ulcer. Perforation of gastroduodenal ulcers is accompanied by classic signs: knife-like pain, wooden belly, ulcerous anamnesis.

Clinical signs of perforation in a free abdominal cavity correspond to three periods. **The first period** - (primary shock) - lasts 3-6 hours. Its duration depends on the size of the ruptured opening and the amount of content that has fallen into the free abdominal cavity. The first signs of perforation are sudden, distinct, permanent, knife-like pains in the epigastric region. The intensity of the sensations is so severe that the patient may have a severe abdominal shock with a collapse and loss of consciousness. Because of the great pain, patient cannot make active movement; they take up a forced position, lying on the right side with the legs lifted to the belly, which reduces the severity of pain. Gradually the intensity of the pain decreases and it extends to the right half of the abdomen. Also, irradiation of pain in the main site is connected with irritation of the branches of the diaphragmatic nerve (frenicus-symptom). The pain intensifies more with deep breathing and minimal movement. The skin of the patient's face becomes pale, covered with cold sticky sweat.

In the first hours of the illness, so called vagus pulse is determined - bradycardia (50-60 beats per minute) or vice versa, there may be a moderate tachycardia (80-90 beats per minute). Blood pressure is decreasing progressively. The frequency of respiratory movements is increased, respiration is superficial.

At examination of a stomach the absence of movability of the anterior abdominal wall during breathing attracts attention. During the palpation a sharp muscular straining in the anterior abdominal cavity is determined – wooden belly, positive symptoms of of Shchotkin-Blumberg, Voskresensky (the symptom of

"shirt"). During percussion, hepatic dullness disappears, almost the half of patients have the tympanitis in the hepatic projection. It should be noted that with a small amount of free air in the abdominal cavity, hepatic dullness can only decrease or not change at all. In the right ileal fossa and the right lateral canal dullness (Kerven's symptom) appears.

The second period ("imaginary" well-being) lasts 6-12 hours. Due to the dilution of hydrochloric acid with exudate in the abdominal cavity, a certain adaptation of the patient's body to aggression, the intensity of the symptoms that prevailed in the first period decreases, the patient feels subjectively better, abdominal pain decreases, euphoria or concern appears. Facial skin can be of normal color, tongue and mucous membranes are dry. The muscle tension of the abdominal wall decreases, the positive symptoms of peritoneal irritation persist, hepatic dullness is absent, bloating appears, and weakened peristalsis is heard over the intestine auscultatory. During the rectal examination, it is possible to diagnose the overhang of the anterior wall of the rectum and its soreness. Thus, in the clinical course of the second period, symptoms of diffuse or delimited peritonitis and intoxication increase.

The third period – of peritonitis, begins 12 hours after the perforation. A progressive deterioration of the patient's condition occurs, symptoms of intoxication increase. Facial features are sharpened, the skin and mucous membranes of the oral cavity are dry. Body temperature rises to 38-40 ° C (sometimes it decreases), blood pressure decreases (hypotension), there are severe tachycardia (pulse to 110-120 beats per minute), frequent and shallow breathing. Repeated vomiting is observed. On examination, the abdomen is swollen due to the development of paralytic intestinal obstruction. Symptoms of peritoneal irritation become sharply positive, signs of the presence of free fluid in the abdominal cavity are determined. Because of the dehydration due to vomiting, the deposition of fluid in the lumen of the intestine and abdominal cavity, connected with intestinal obstruction and a disorder of tissue microcirculation, oliguria or even anuria develops. Without surgery, patients inevitably face death.

The clinical course of covered perforation has its own characteristics. Similar symptoms are observed in case of perforation of an ulcer of the posterior wall of the stomach to an omental bursa. The onset of the disease is typical: acute knife-like pain occurs, but its duration is much shorter, the intensity is lower, and signs of abdominal shock, as a rule, do not develop or are not severe. After a short period of time, the pain subsides, becomes insignificant and local, mainly in the epigastric region or right hypochondrium.

If there is no adhesion process in the area of the Vinsel hole, then perforation of the ulcer in the omental bursa is clinically signed by a gradual increase of moderate intensity of pain in the right lateral canal and iliac region because of the contents of the omental bursa. Such a clinical picture without a detailed analysis of the medical history of the disease may be the reason for deciding whether to conduct surgery for acute appendicitis. Consequences of covered perforations can be abscesses of the abdominal cavity or omental bursa or diffuse peritonitis.

Diagnosis of perforated ulcers. The primary mandatory diagnostic method for perforated ulcers is an X-ray examination of the abdominal cavity. So, with a survey radiography (fluoroscopy) of the abdominal cavity in the vertical position of the patient, in most cases, a strip of free gas is determined in the subphrenic space or under the liver (pneumoperitonium). In cases of the adhesion process between the liver and the diaphragm or with small sizes of the perforated hole, there may be no free gas in the abdominal cavity. Then pneumogastrography is performed, when after the introduction of a small amount of air (1.0-2.0 L) through the tube into the stomach, an X-ray examination is repeated. It is also possible to use a contrast fluid instead of air. If, after this, either air or contrast appears in the abdominal cavity, then the presence of perforation is considered confirmed.

Fibrogastroduodenoscopy, which allows you to determine the exact location of the ulcer, its size, to determine the presence of other complications at the same time, is of certain importance for clarifying the diagnosis.

Laparoscopy, which helps to clarify the diagnosis can be important. So, in case of the localization of an ulcer on the anterior wall of the stomach or duodenum, a perforated hole or the presence of a specific effusion in the abdominal cavity can be detected. Also, laparoscopy allows you to choose an adequate treatment plan for a dubious clinical picture, and in some cases to convince one or another method of surgical treatment.

Using ultrasound diagnostic methods, it is possible to diagnose free fluid in the abdominal cavity during the development of diffuse peritonitis.

Laboratory blood tests allow you to determine leukocytosis with a shift in the leukocyte blood count to the left, signs of blood thickening. A biochemical examination determines hyperkalemia, metabolic acidosis, and an increase in urea and creatinine. In the analysis of urine, the appearance of blood cells, protein and cylinders is noted.

Differential diagnostics. In spite of the bright clinical picture of perforated ulcer, in some cases it is necessary to conduct differential diagnostics with other acute diseases of the abdominal cavity, with one of the main similar symptoms is pain in the epigastric region. These diseases include acute cholecystitis, acute pancreatitis, acute appendicitis. Sometimes it is also difficult to differentiate it with acute cardiovascular disease.

An attack of acute cholecystitis usually begins after eating spicy, fatty, fried foods. The pain is localized in the right hypochondrium, is permanent and is accompanied by repeated vomiting, which does not bring relief. Muscle tension in the right hypochondrium is palpated, but not so severe as with a perforated ulcer, since it is sometimes possible to feel the bottom of the gallbladder or the entire enlarged tense gallbladder. In addition, the positive signs of Ortner, Kehr, Murphy are determined. In addition to irradiating pain in the right shoulder, a positive sign of Musi-Georgievsky is determined ("phrenicus symptom"). The chronic course of cholecystitis is characterized by a recurrence of pain attacks, the similarity of the causes of their occurrence. An ultrasound reveals signs of inflammation of the gallbladder and perivesical region.

During acute pancreatitis, the onset of the disease is connected with vomiting and with the appearance of constant sharp pain in the epigastric region, a characteristic feature of which is belt-like character. The causes of the attack are usually the consumption of excessive amounts of fatty foods and alcohol. During the palpation, the abdomen is slightly tense, painful in the projection of the pancreas, where inflammatory infiltrate can be determined. Another characteristic of acute pancreatitis is bloatedness due to intestinal paresis. Additional diagnostic methods provide assistance in conducting differential diagnostics, primarily ultrasound, radiography, fibrogastroduodenography, and determination of the level of pancreatic enzymes in the blood serum. Similarities in clinical signs between perforated ulcers and acute appendicitis occur at early stages of the disease. Difficulties arise due to the localization of pain in the epigastrium. So, in case of acute appendicitis, Kocher's sign is not accompanied by such severe muscle tension in the epigastrium as during a perforated ulcer. Movement of pain sensations to the right iliac region with palpation signs of peritonitis during a perforated ulcer differs from Kocher's symptom during appendicitis because it occurs while maintaining tension in the muscles in the epigastrium. In cases when the patient is operated due to the misdiagnosis of acute appendicitis, during the operation, the presence of a blurred effusion in the abdominal cavity with the admixture of mucus, bilis, food residues on the background of an unaltered or catarrhally changed appendix may remedy the situation. In such cases, it is necessary to perform medial laparotomy and to eliminate the cause of peritonitis. Performing an X-ray examination of the abdominal cavity in cases of diagnosis of pneumoperitonium facilitates differentiation with acute appendicitis.

Mesenteric vascular thrombosis, in contrast to a perforated ulcer, is characterized by an acute onset with the appearance of severe abdominal pain without clear localization. The general condition of patients is severe due to hemodynamic disturbances that leads to arterial hypotension and tachycardia. The abdomen is evenly swollen, soft on palpation, signs of paralytic intestinal obstruction are determined. With a digital examination of the rectum, bloody flux may be detected. As a rule, thrombosis of mesenteric vessels occurs against the background of chronic diseases of the cardiovascular system. In addition to the features of the medical history, additional diagnostic methods allow to perform differential diagnosis: panoramic radiography and ultrasound investigation of the abdominal organs.

The gastralgic form of acute myocardial infarction, with similar localization of pain in the epigastric region with moderate muscle tension and peritonitis, in contrast to perforated ulcers, is characterized by ECG changes in the form of fresh focal myocardial abnormalities.

Treatment of perforated ulcer. Surgical treatment is indicated for all patients with perforated ulcers, the main purpose of which is to save the patient's life. Perforation of an ulcer in the free abdominal cavity is an absolute indication for emergency surgery, with the exception of patient agony, which is in some cases considered a contraindication for surgery.

In addition, as an exception, with the patient's categorical refusal of the operation or the absence of conditions for its implementation, a conservative Taylor treatment method can be used. The essence of the method is the constant aspiration of gastric contents through a nasogastric tube, drug-induced reduction in gastric secretion, intensive antibiotic therapy, infusion therapy, and laboratory and radiological monitoring of the process. The creation of negative pressure in the stomach during aspiration should help cover the perforated hole with a neighboring organ, therefore, the criterion for the effectiveness of the method is the absence of contrast in the abdominal cavity during a contrast X-ray examination. In cases where the Taylor method did not lead to perforation covering, it is regarded as a preoperative preparation.

In general, preoperative preparation should be carried out in all cases and should be the more intense the more pronounced the phenomena of peritonitis is. Antishock measures and detoxification are usually carried out, metabolic disturbances and disorders in the activity of organs and systems of the body, and stomach decompression are corrected.

Depending on the type and location of the ulcer, the severity of the patient’s condition, the stage of development and prevalence of peritonitis, the duration of peptic ulcer disease and the conditions for surgery, various methods can be used:

1. Palliative surgery (suturing an ulcer; excision of an ulcer followed by drug treatment; tamponade of the perforated opening with a part of the greater omentum according to the Graham or Oppel-Polikarpov method.
2. Radical operations (excision of an ulcer with pyloroplasty and vagotomy, types of stomach resection).

So, in case of widespread purulent peritonitis, a high degree of operational risk due to concomitant diseases, the patient's age over 60 years, the duration of the disease, during the perforation of acute (stress, medicamentous) ulcers or in the absence of conditions for performing more extensive interventions, it is advisable to perform ulcer closure. The essence of the method is to suture an ulcerative defect in the transverse direction in relation to the axial axis of the stomach or duodenum using separate serous-muscular sutures. Before suturing, it is advisable to economically carve the edges of the ulcer.

If it is impossible to suture a perforated ulcer (large size, callous ulcer, if there are contraindications to gastric resection and when the sutures are cut), the ulcer tamponade is performed with a lock of a large omentum on a crus using the Oppel-Polikarpov method or the Graham method.

Indications for laparoscopic suturing of a perforated ulcer are the absence of peritonitis, the localization of the perforated hole on the anterior wall of the stomach and duodenum, the size of the perforated hole up to 5mm and the stability of hemodynamic parameters of the patient. It should be noted that suturing a perforated ulcer does not cure the patient with peptic ulcer and does not guarantee them from relapse, therefore, it is considered to be a palliative method.

The optimal amount of surgical intervention for perforated ulcers is the excision of a gastric ulcer (sphenic or from the side of the lumen of the stomach

according to Jadu-Tanaki) or duodenal ulcer (according to Jadu-Horsley) with pyloroplasty and vagotomy. It should be noted that due to the success in the medical treatment of peptic ulcer disease, the performance of any type of vagotomy during surgery has reduced its importance due to the use of highly effective drugs to reduce acidity (the so-called "medicamentous vagotomy"). Therefore, the implementation of vagotomy in an urgent surgery, for today, is not recommended.

In cases of covered ulcer perforation, emergency surgery is indicated, since covering the ulcer and temporarily stopping the contents of the stomach or duodenum from entering the free abdominal cavity cannot be considered reliable and sufficient for complete cure.

In any case, surgery for perforated ulcers should end with a thorough rehabilitation of the abdominal cavity. It is necessary to completely evacuate the inflammatory exudate and gastro-duodenal contents, especially from the subphrenic space, the lateral canals and the pelvic cavity. After rehabilitation, drainage of the abdominal cavity is mandatory, sometimes with intubation of the intestine with a nasogastric tube. In severe forms of peritonitis, a programmed laparostomy is performed.

After surgery, drug treatment of peritonitis, peptic ulcer continues, prevention of purulent-inflammatory complications is performed.

Complications. In the postoperative period, possible complications are pneumonia, abdominal abscesses, disordered functional or mechanical genesis evacuation from the stomach, and postoperative intestinal paresis.

For the prevention of pulmonary complications, the whole spectrum of measures should be applied: early activation of patients, adequate pain relief in the early postoperative period, percussion massage of the chest, camphor preparations and physiotherapeutic procedures.

Diffused peritonitis due to the failure of anastomotic sutures or delimited forms of it (abscesses) are treated exclusively operatively.

In case of disorder of the evacuation from the stomach due to functional reasons, the patient is given anti-inflammatory, infusion, desensitizing therapy, measures to eliminate intestinal paresis. If the symptoms of stagnation in the stomach do not disappear within 5-7 days of treatment, mechanical factors are the most likely cause of stenosis, among which the narrowing of the lumen of the pyloric section due to errors in suturing the ulcer, and narrowing of the anastomosis due to inflammation and cicatricial deformity are most common. In such cases, relaparotomy is indicated.

Prevention.

Primary preventive measures include:

- early diagnosis of peptic ulcer of the stomach and duodenum;
- giving up bad habits (smoking, drinking)
- compliance with diet and dietary regime;
- eradication of HP infection with control;

- the appointment of gastroprotectors while taking non-steroidal anti-inflammatory drugs and anticoagulants;
- sanatorium-resort treatment in specialized sanatoriums.

Prevention of secondary complications includes:

- prevention of the progression of peritonitis, intraperitoneal and wound suppurative complications (an adequate choice of the volume of the operation and method of eliminating of the perforated hole, thorough sanitation and drainage of the abdominal cavity, well-timed determination of indications for programmed relaparotomy, antibiotic prophylaxis and adequate initial antibiotic therapy).
- detoxification therapy;
- prevention of paralytic ileus, thrombohemorrhagic complications, pulmonary complications and stress ulcers.

Self-control questions

1. What are the clinical signs of perforation of the ulcer in the free abdominal cavity?
2. What are the clinical signs of the closed perforation of the ulcer?
3. What are the clinical signs, specific nature of the diagnosis of atypical perforation of the ulcer?
4. List the classification of perforated ulcers.
5. What are the methods for diagnosing perforation of the ulcer in the free abdominal cavity?
6. What are the methods of diagnosis of the closed perforation of the ulcer?
7. Perform a differential diagnosis of perforated ulcer.
8. What are the methods of surgical treatment of perforated ulcers?
9. What are the features of treatment of perforation of the ulcer using the Taylor method?
10. What are the postoperative complications of perforated ulcers?

Tests for self-control

1. A 53-year-old patient was hospitalized to the surgical department 28 hours after perforation of a gastric ulcer with severe peritonitis. The patient's condition is serious. After preparation, he was immediately operated. The operation revealed that the ulcer with a perforated hole is located in the pyloric stomach. During suturing, the exit from the stomach narrowed significantly, which objectively could lead to pyloric stenosis. What kind of surgery does the patient need?
   a. resection of the stomach according to Billroth-I;
   b. pyloroplasty;
   c. gastroenterostomy;
   d. a resection of the stomach according to Billroth-II;
   e. gastric resection according to Ru.

2. A 25-year-old patient was hospitalized in the surgical department with complaints of severe pain in the epigastric region. Objectively: the tongue is dry, the abdomen is swollen, painful in all departments upon palpation, mostly in epigastrium. The symptom of Shchotkin-Blumberg is sharply positive. On a survey radiograph of the abdominal organs, free gas was found in the abdominal cavity. Identify further diagnostic tactics.
   a. fibrogastroduodenoscopy;
   b. laparotomy with a decision on the volume of surgical intervention
   c. radioscopy of the stomach with barium;
   d. dynamic observation;
   e. radioscopy of the stomach with urographin.

3. The patient went to the doctor with complaints of bloating, the absence of an act of defecation and gas discharge within 3 days. Nine days ago, there was pain in the right hypochondrium, which itself stopped. Pulse was 68 beats per min., rhythmic, with satisfactory properties. The abdomen is swollen, peristalsis is satisfactory. During an x-ray examination revealed free gas under the right dome of the diaphragm and fluid level. An endoscopic examination revealed two duodenal ulcers with no signs of perforation. What is the diagnosis?
   a. duodenal ulcer, perforation of the small intestine;
   b. duodenal ulcer, complicated by bleeding;
   c. an interloop abscess, duodenal ulcer;
   d. covered perforation of a duodenal ulcer with the formation of a subphrenic abscess;
   e. acute intestinal obstruction.

4. The patient complains of general weakness, vomiting, sharp pain in the epigastric region. Suffers from peptic ulcer of the duodenum for 10 years, such attacks of pain have been observed repeatedly. Objectively: the skin is pale, covered with sticky sweat. The tongue is dry; the stomach is not involved in the act of breathing. During palpation, the anterior abdominal wall is tense. Symptoms of peritoneal irritation are sharply positive in the epigastrium, the right half of the abdomen. Make a preliminary diagnosis:
   a. perforated duodenal ulcer;
   b. right-sided renal colic;
   c. acute perforated cholecystitis;
   d. acute perforated appendicitis;
   e. penetration of a duodenal ulcer into the head of the pancreas.

5. The patient complains of abdominal pain, delayed discharge of gases and feces, general weakness. For 8 years, he was repeatedly treated in a hospital for gastric ulcer. A few hours ago there was a sharp pain in the epigastric region (after a defect in the diet). Objectively: the tongue is dry. The abdomen is distended, during palpation it is painful in all departments, mostly in the epigastrium. The

symptom of Shchotkin-Blumberg is sharply positive. Pulse - 120 beats per minute., rhythmic, of a weak filling. The activity of the heart is rhythmic. BP - 100/60mm Hg. Formulate a preliminary diagnosis.

a. penetration of a stomach ulcer into the pancreas;
b. acute cholecystitis;
c. thrombosis of mesenteric vessels;
d. acute pancreatitis;
e. perforated gastric ulcer.

6. The patient complains of sharp pain in the epigastrium. Suffers from peptic ulcer of the duodenum for 10 years. The patient's position is forced on the right side with the hips brought to the stomach. The abdomen during the palpation is sharply painful in the epigastrium, there is a severe protective tension of the muscles of the anterior abdominal wall. Formulate a preliminary diagnosis.
   a. exacerbation of peptic ulcer;
   b. acute pancreatitis
   c. perforated duodenal ulcer;
   d. penetration of the ulcer into the pancreas;
   e. thrombosis of mesenteric vessels.

7. The patient complains of a sharp constant pain in the epigastrium and in the right half of the abdomen. Ulcerative history lasts 5 years. The abdomen during the palpation is sharply painful in the epigastrium, a positive symptom of Shchotkin-Blumberg is determined. Hepatic dullness is absent. What is the diagnosis?
   a. acute myocardial infarction
   b. acute perforated appendicitis;
   c. perforated ulcer;
   d. exacerbation of peptic ulcer;
   e. acute cholecystitis.

8. The patient is concerned about intense pain in the epigastrium, nausea, vomiting. The moment the pain appeared was like a burn with boiling water. The abdomen is not swollen, does not participate in the act of breathing, sharply painful in the epigastric region. Also, the protective tension of the muscles of the anterior abdominal wall is determined. Peritoneal symptoms are positive. The symptom of Spizharny is determined. What is the diagnosis?
   a. thrombosis of mesenteric vessels;
   b. acute appendicitis;
   c. aortic aneurysm
   d. perforated ulcer;
   e. acute pancreatitis.

9. The patient complains of sharp pain in the epigastrium and in the right half of the abdomen. The pain radiates to the right scapula and subclavian region on

the right. The abdomen is not swollen, does not participate in the act of breathing, it is sharply painful during the palpation in the epigastrium, less in the mesogastrium and in the right iliac region. Positive symptoms of Shchotkin-Blumberg, Spizharny. What is the diagnosis?

a. penetration of the ulcer into the pancreas;
b. perforated ulcer;
c. acute appendicitis;
d. acute pancreatitis
e. exacerbation of peptic ulcer.

10. A patient with a anamnesis of peptic ulcer experienced a sharp pain in the epigastrium, which disappeared in 30 minutes. A day later, the patient went to the doctor. Objectively: palpation of the abdomen is soft, moderately painful in the right hypochondrium. The symptom of Shchotkin-Blumberg is positive, hepatic dullness is saved. Body temperature is 37.8 C. What is the diagnosis?
    a. acute intestinal obstruction;
    b. acute stomach ulcer;
    c. subphrenic abscess;
    d. perforation of the small intestine;
    e. covered ulcer perforation.

11. What is the name of the radiological symptom characteristic of perforation of a duodenal ulcer?
    a. a lace;
    b. a hammer;
    c. a nail;
    d. a sickle;
    e. a pot.

ACUTE INTESTINAL OBSTRUCTION

Definition. Acute intestinal obstruction is a pathological condition caused on a complete or partial defect of the natural passage of the contents of the intestine by the origin of mechanical obstruction or disorders of the motoric-evacuation function. This is one of the most actual problems of abdominal surgery, which is caused by a high prevalence (up to 10% of the total number of patients with surgical pathology) and high (from 15 to 25%) mortality and a lot of a number of complications. Somewhat more often acute intestinal obstruction occurs in men (55%). The exception is adhesive intestinal obstruction, which is more common in women.

Anatomical and physiological information. Intestine is divided into small and large intestine. In the small intestine, there are three sections: the duodenum, the jejunum and the ileum. The large intestine consists of the cecum, ascending colon, transverse colon, descending colon, sigmoid and rectum. The site of transition the small intestine in the large bowel is called the ileocecal angle.

The beginning and the end of the small intestine is fixed by the root of the mesentery to the back wall of the abdominal cavity. Another mesentery provides its mobility and position in the form of loops. About the three sides they are surrounded by the colon: from above - the transverse colon, to the right - the ascending colon, to the left - the descending colon. Intestinal loops in the abdominal cavity are located in several layers, the surface layer faced with a large septum and anterior abdominal wall; the deep layer is adjacent to the back wall.

The **large intestine** begins from the ileocecal angle, ends with a rectum and a back hole (anus). Ileocecal angle is located in the right iliac recess and represents the transition of the small intestine to the first part of the colon - cecum. The transition point of the ascending colon in the lumbar is the hepatic angle, and the transitional point of the transverse loop in the descending is the spleen angle.

The **cecum** is located below the upper side of the ileum and is covered by a peritoneum from all sides (intraperitoneally). Where the cecum does not have a complete peritoneal cover, its back wall is strongly fixed to the retroperitoneum and iliac fascia. In the area of the appendicular process, all three muscle strips of the intestine come together. The appendix is also covered from the peritoneum on all sides.

The ascending part is covered by a peritoneum from three sides (mesoperitoneally). Its right bend is in contact with the lower surface of the right lobe of the liver, the bottom of the gall bladder and located mesoperitoneally.

Transverse colon is located intraperitoneally. It begins in the right ribs' angle, passes into the epigastric area, and then reaches the left hypochondrium, where it passes into the left loop, which is located intraperitoneally. The transverse colon on the top is limited by the liver, gallbladder, large curvature of the stomach, spleen, from the bottom with the loops of the small intestine, the front with the anterior abdominal wall, and from the back with the duodenum, pancreas and the left kidney, which are separated from it by the erythematous and mobility peritoneum The lower abdominal wall is located mesoperitoneally. It is separated from the anterior

abdominal wall by the loops of the small intestine and a greater omentum, behind it the muscles of the back abdominal wall.

The **sigmoid colon** is located intraperitoneally and has significant mobility.

Blood supply of the duodenum is carried by the branches of the gastro-duodenal and upper mesenteric arteries, the jejunum and ilium intestine - the upper mesenteric artery. Initially, the upper mesenteric artery passes like crescent behind the neck of the pancreas and splenic vein, and then goes out of the lower edge of the gland and enters the mesentery of the small intestine. Entering the mesentery of the small intestine, the upper mesenteric artery passes from the top-down from left to right, creating an arcuate bend that is directed bulge to the left. In this place, the branches of the small intestine (aa jejunal et ileac) leave from the upper mesenteric artery to the left, and branches of the ascending and transverse colon departing upward a. colica media et. a. colica dextra. In the thickness of the mesentery of the small intestine, branches form arterial arches I and II parts, which provide reliable blood supply. From them to the wall of the intestine goes straight short arteries. The upper mesenteric artery ends in the right iliac recess with its ultimate branch - a. Ileocolica, which supplies blood to the end of the ilium and the beginning of the large intestine.

Blood supply to the colon provides by upper and lower mesenteric arteries that depart from the abdominal aorta.

The branches of the upper mesenteric artery, to the ileocecal angle, are the anterior and posterior ciliary arteries (aa. caecalis anterior et posterior), leading the branches to the apex part of the ileum, the caecum and the appendix. The artery of the appendix (appendicularis) passes in through own mesentery. The right colon artery (a. colica dextra) supplies the ascending colon. It is divided into a descending branch, which connects to the ascending branch of the caecum artery, and ascending, which connects to the right branch of the middle colon artery. The rim of the intestinal artery is divided into two branches and connects to the left and right colon arteries, respectively.

The lower mesenteric artery supplies the colon from the left bend to the upper third of the rectum. It gives the left iliac artery (a. colica sinistra) to the intestine, which is divided into ascending and descending branches. The ascending level of the left bend is connected with the left branch of the middle mesenteric artery, and the descending - with sigmoid arteries. The anastomosis between the left branch of the median rim and the ascending branch of the left rim artery connects the basins of the upper and lower mesenteric arteries and is called the Riolan's arch.

To the sigmoid colon go 2-4 branches of sigmoid arteries, which are interconnected.

The upper rectal artery (a. rectalis superior) is the ultimate branch of the lower mesenteric artery and goes to the rectum ampulla. It is associated with anastomosis of the lower sigmoid and medial rectal arteries.

Venous blood from the small intestine flows out into the upper mesenteric, and then into the portal vein system. From the large intestine, the blood flows out with the same name arteries and the veins, which form the upper and lower mesenteric veins. From there the blood enters the portal vein. From the lower part of the sigmoid

colon, the blood may flow out to the portal vein or the branches of rectum veins to the lower vena cava. Thus, interstitial portocaval anastomosis is formed.

Lymph outflow occurs by many drainage vessels to the mesenteric and abdominal lymph nodes. Intraintestine lymphatic channel in the small intestine is developed relatively faintly. The lymph from the large intestine flows into the lymph nodes located along the vessels: the nodes of the appendix, the anterior and posterior nodes of the caecum, iliac and transversal; right, middle, left transversal, pre-transversal, sigmoid, upper rectal, and upper and lower mesenteric nodes.

The innervation of the small intestine provides by branches of the abdominal cavity, liver (duodenum) and upper mesenteric plexus. The innervation of the large intestine is provided by the branches of the upper and lower mesenteric plexus (sympathetic innervation) and the branches of the vagus nerves and pelvic internal nerves (parasympathetic innervation).

Etiology and pathogenesis of acute intestinal obstruction. To the etiological factors of the development of acute intestinal obstruction are:

- anatomical and morphological features of the intestine, mesentery, peritoneum: congenital disorders (dolichosigm, mobile cecum, pockets in the abdominal cavity, intraperitoneal adhesions), various formations in the lumen of the intestine (tumours, gallstones, parasites), the effects of injuries or operations on the organs of the abdominal cavity (adhesion disease), various types of external abdominal hernias, inflammatory diseases of the abdominal cavity.
- a high increase in intraabdominal pressure, changes in the motor-evacuation function of the intestines in case of disorders of diet or against the background of their inflammatory diseases.

Anatomic-morphological changes in the abdominal cavity that contribute to the development of intestinal obstruction:

Causes of <u>obstructive</u> intestinal obstruction:

1. Congenital disorders of the progress of the intestinal tube (doubling, atresia, Meckel diverticulum).
2. Acquired: benign and malignant tumours of the intestine, foreign bodies, inflammatory infiltrates.

Causes of <u>strangulation</u> in forming of intestinal obstruction:

1. Congenital: anomalies of the progress of the peritoneum, mesentery and intestine.
2. Acquired: adhesions, hernia, cicatricial changes and traumatic defects in mesentery.

Causes of <u>invagination</u> in intestinal obstruction:

1. Congenital: disorders of innervation and motility of the intestine.
2. Acquired: acute and chronic intestinal diseases, intoxication.

Causes of <u>connective type</u> intestinal obstruction:

- Congenital: Lenn's formations, Jackson's membranes.
- Acquired: inflammatory, post-traumatic and postoperative adhesions.

Causes of dynamic intestinal obstruction:
- Spastic: helminthic invasion, lead poisoning, hysteria.
- Paralytic: alimentary disorders, postoperative paresis, inflammatory processes in the abdominal cavity.

Among the ways for the pathogenesis of acute intestinal obstruction are ischemic and obstructive. The mechanism of ischemic disorders is associated with general disorders of the microcirculation and with the effect of intestinal hypertension to the circulatory system. Hemodynamic and volume changes arise as a result of the decreasing of arterial supply and venous outflow or compression of the vesicles of the loops (with strangulation obstruction) or of the intratumoral vessels by increasing the critical pressure in the lumen of the bowel in functional or obstructive acute intestinal obstruction. In the influence of biologically active substances, there is a paralysis of precapillary muscles-locks with the subsequent forming of stasis and aggregation of the blood cells. Free tissue kinins and histamine break the permeability of the vascular wall, which help the appearance of interstitial oedema. Together, with the action of endotoxins and ischemia, it leads to the destruction of the intestinal wall with necrosis of all its structures. The bowel gets dark-purple and later black-brown colour.

Along with it, the primal role in the emergence of endogenous intoxication have a microbial factor. Already from the first hours of acute intestinal obstruction, the normal microbiological ecosystem of the bowel is disturbed by the stasis of contents, which support to the quick growth of flora, as well as the migration of the microflora from the distal parts of the digestive canal into the proximal parts (colonization of the small intestine by colon flora). The isolation of exotoxins and endotoxins with the breach of the barrier function of the intestinal wall results in translocation into the portal blood flow, lymphatic and peritoneal exudates of bacteria whose toxins damage the function of many organs and systems. These processes are the basis of systemic inflammatory reaction and abdominal sepsis. The progress of intestinal necrosis and purulent peritonitis is a source of internal toxicosis, which leads to the forming of multiple organ dysfunction and failure characterized by severe sepsis, the mortality of which rates 80% or more.

Classification of acute intestinal obstruction. According to the clinical-statistical classification of diseases (ICD-10), acute intestinal obstruction divided:

K56.0 Paralytic ileus. Paralysis of: bowel, colon, intestine.

K56.1 Intussusception. Intussusception or invagination of: bowel, colon, intestine, rectum.

K56.2 Volvulus. Strangulation. Torsion. Twist of colon or intestine

K56.3 Gallstone ileus. Obstruction of intestine by gallstone.

K56.4 Other impaction of intestine. Enterolith. Impaction (of): colon, faecal.

K56.5 Intestinal adhesions [bands] with obstruction. Peritoneal adhesions [bands] with intestinal obstruction.

K56.6 Other and unspecified intestinal obstruction. Enterostenosis. Obstructive ileus NOS. Occlusion. Stenosis. Stricture of colon or intestine.

K56.7 Ileus, unspecified.

By **cause**:

- Congenital
  - malformations of the intestinal tube;
  - malformations of the intestinal wall;
  - disruption of bowel movement;
  - defects of development of other organs of the abdominal cavity.
- Acquired

By mechanism of occurrence:

1. Dynamic obstruction:
   - spastic;
   - paralytic
2. Mechanical obstruction:
   - obturation (disturbance of the lumen of the intestine) - compression from the outside, tumor obstruction, fecalith and gallstones, ascaridic globe, coprostasis;
   - strangulation (compression of the bowel and its loops with simultaneous violation of patency and blood circulation) - pinching, twisting, knot formation;
   - mixed (conjunctival acute intestinal obstruction, invagination).

By **level of obstruction**:

1. Small bowel obstruction:
   - high;
   - low
2. Colon obstruction:
   - acute
   - chronic
   - recurrent

By **stages**:

– the stage of "ileus cry" (stage of acute offense of the intestinal passage), occurs after 12-14h.

– the stage of intoxication (the stage of acute violations of intrathecal intestinal hemocirculation) occurs after 12-36h.

– terminal stage (peritonitis) / occurs more than after 36 hours

By the **rate of closure** of the lumen of the intestine:

– full

– partial.

Clinic of acute intestinal obstruction. The clinical manifestations of intestinal obstruction is quite various and depends by many factors: level, type and rate of obstruction, time of origin. Most often, the disease begins suddenly and quickly progresses. The main symptoms of intestinal obstruction are pain, vomiting, gases delay and stomach cramps.

Abdominal pain appear from the beginning of the disease and has an attack-like character with irradiation in the epigastric region, in the waist, crotch, groin, back, and coccyx. The intensity of the pain relies on the size and rate of the occlusion of the intestines and the involvement in the process of the mesentery. Quite often, it is so intense that the patient cries, moans, constantly changes the position in the bed. In obstruction, the pain gradually increases, insignificant. When the motor function of the intestines weakens (after 4-6 h.), paresis of the intestine arise and the pain subsides partly.

Vomiting appears almost at once with pain and has a reflexive character. With narrow-necked obstruction, vomiting occurs after 10-15 minutes. Subsequently, it becomes more constant and does not bring relief. First, vomiting is the contents of the stomach, duodenum and gall bladder. Then, from 2-3 days of the disease, it becomes of faecal odour by decay and decomposition of proteins in the small intestine and the formation of indole and scatole. Faecal vomiting is a poor prognostic sign and indicates an increase in the severity of the process in the abdominal cavity. About colorectal obstruction, vomiting occurs 1-2 times at the beginning of the disease and arise again at the end of the first day. At high-level invaginations and in case of thrombosis of mesenteric vessels, an admixture of blood appears in vomiting.

Delay of faeces and gases is a pathognomonic sign of intestinal obstruction. This is an early symptom of lower obstruction. In the case of high intestinal obstruction at the beginning of the disease, especially by the influence of therapeutic measures, the act of defecation may be caused by emptying of the intestines below the obstruction.

In the clinical following of acute intestinal obstruction, distinguish three **stages**:

- Initial or "ileus cry" - from 2 to 12 hours. It develops as a result of disorders of the passage in the intestine and is characterized by cramping pain, a quick passage, and vomiting. Bowel necrosis comes quickly.
- The stage of intoxication - from 12 to 24 hours. The pain becomes permanent, the stomach is bloated, asymmetric, there is a complete delay in the excretion of faeces and gases. Peristalsis of the intestine is seldom, there are hemodynamic changes that are associated with hypovolemia and centralization of blood circulation. There is thirst, dryness of the tongue, decreased blood pressure, tachycardia, increased hematocrit, decrease diuresis.
- Terminal stage - more than 36 hours. The condition of the patient is extremely severe, the "face of Hippocrates", dry tongue, faecal vomiting, the abdomen is strongly swollen, peristalsis is not heard, the temperature of the body is hectic,

tachycardia, blood pressure 60-70mmHg. Hypovolemic shock, hypokalemia, blood acidosis evolves. There is general weakness, muscle hypotonia, decreased reflexes, apathy, heart rhythm disorders, systolic noise at the apex of the heart, paresis of the intestine. In severe cases - paralysis of breathing, asystole.

Diagnostics of acute intestinal obstruction. For the diagnosis of acute intestinal obstruction are the importance of complaints, anamnesis of disease and the life, physical examination methods (palpation, percussion, and auscultation).

During an objective examination, attention is drawn to the patient's behaviour. In the case of an attack-like pain, he becomes restless, often changes the position of the body. In the intervals between pain attacks, the patient is calm.

The general condition of the patient is moderate or severe and rely on the form, level and time from the beginning of the disease. The temperature at the beginning of the disease does not increase. In the future, with the occurrence of peritonitis, there is an increase in temperature, tachycardia. The tongue is dry, covered with grey layers. It should be noted that the clinical manifestation of high intestinal obstruction is significantly pronounced, with early manifestations of dehydration, disorders of acid-alkaline and water-electrolyte balances.

When looking at the stomach in patients with acute intestinal obstruction, it is important to examine all possible places of the output of hernias wall, to exclude their strangulation. Special attention is for femoral hernia in elderly women. Postoperative scars may cause connective intestinal obstruction. During the examination of the abdomen, flatulence is might help to diagnose the localization of the barrier, the level of obstruction and the duration of the disease. Uneven bloated abdomen (Bayer's symptom) is, in some cases, characteristic of a sigmoid torsion. At high obstruction, is insignificant and often asymmetric, for paralytic and obstructive intestine obstruction characterized by diffuse flatulence. Abnormal configuration of the abdomen and its asymmetry is inherent in the case of strangulation intestinal obstruction. The longer the intestinal obstruction proceeds, the more pronounced swelling of the abdomen.

Peristalsis is observed in the early stages of the disease, in the first 4-6 hours, until the progress of paresis of the intestine. In case of invagination, adhesion disease and chronic obstruction, the peristalsis can be saved much longer. Sometimes, especially exhausted patients, it is possible to see bloated and periodically peristaltic loops of the intestines under the abdominal wall.

Palpation of the stomach in the period between attacks is usually not painful, the tension of abdominal muscles and symptoms of peritoneal irritation are absent. When strangulation of the intestine caused by a torsion of the small intestine, is a positive symptom Tenevara - a severe pain during pressing the abdominal wall on two fingers below the navel in the median line (the location of the projection of the mesenteric root). By palpation, it is possible to detect a limited elastic sausage-shaped formation with a site of high tympanitis - Valya's symptom, which use to diagnosing the tumour, the body of the invaginate or inflammatory infiltrates, which

became the cause of obstruction. Peristaltic may be called or intensified by shaking the abdominal wall with hand or by its palpation - the symptom of the Shlange.

By percussion and auscultation of the abdomen, you can hear the "noise of the splash" - the symptom of Sklyarov and the "noise of a falling drop" - a symptom of Spasokukotsky in combination with high tympanitis with a metallic tint over overflowing gases and a bloated bowel loop - a symptom of Kivul's. With the progression of intestinal obstruction and the beginning of paresis, intestinal noises appear less often, they are short and of high tone. In the late period, all the peristaltic noises completely disappear and paralysis of the intestines arises - a symptom of "grave silence". In this period, the impossible to hear the peristalsis over the bloated abdomen, but hearing of respiratory noises and heart tones are the symptom of Loteisen.

An examing of a patient with acute intestinal obstruction necessarily ends with the rectal examination by finger. In this case, an empty bowel and an enlarged rectal ampule are diagnosed - the symptom of Grekov ("Obukhivska's Hospital symptom"). Also, it is possible to find out "faeces", rectal tumour, invaginate, to estimate the tone of the external anal sphincter, the contents of the ampulla of the rectum and the impurities in the defecation. In case of invagination on the glove, the blood is often found. Small capacity of the rectum in the setting of a siphone clyster - a symptom of Ceghe-Manteyfel are signs of low obstructive intestine obstruction.

In the general blood analysis, erythrocytosis, leukocytosis, the shift of the leukocyte formula to the left, haemoglobin, hematocrit and ESR are determined. In the general analysis of urine - oliguria (in severe cases, anuria), presence of protein, leukocyturia, cylinders). In the biochemical analysis of blood - dysproteinemia, decrease in total protein, decrease in the content of K +, Na +, SI, an increase of creatinine, urea, and nitrogen.

Instrumental diagnostics in suspicion of intestinal obstruction is used, both for confirmation of the diagnosis, and for clarification of the level and causes of forming of acute intestinal obstruction. X-ray examination - the main special method of diagnosis, which carried out with the slightest suspicion of intestinal obstruction. Initially perform the radiographic review examination of the abdominal cavity.

Here are some of the following symptoms:

1. Intestinal arcs - appears when the small intestine is swollen by gases, while in the lower parts of the arcs there are horizontal levels of the liquid. They characterize the predominance of gas over the fluid content of the bowel and occurs in the early stages of disease.
2. Kloiber's bowl is the horizontal level of liquid with a dome-like clarification (gas) above it, which looks like an inverted bowl. When the width of the fluid level is greater than the height of the gas bubble, the folds of the mucous membrane in the form of a spring - the bowls are localized in the small intestine. In the large intestine, the vertical dimensions predominate over the horizontal, there are infrequent half-mucous folds of the mucosa (gaustration). With strangulation obstruction of the bowl of Cloyber may appear in an hour, with an obstructive - 3-5 hours from the moment of the disease. With narrow-neck obstruction, the number of bowls varies, sometimes they can be laid one by one

in the form of stairs. When the bowls are localized only in the left hypochondrium, this indicates a high obstruction. For low obstruction in late terms, both small and large-bowel bowls of Kloiber are characteristic. The placement of a fluid in one intestinal loop on one level indicates a deep paresis of intestines, which is characteristic of the late stages of acute mechanical or paralytic intestinal obstruction.

3. a symptom of a feather (transverse stretching of the intestine in the form of a stretched spring) occurs at high intestinal obstruction and is associated with stretching and oedema of the intestine, which has high circular folds of the mucous.

Contrast X-ray examination of the digestive canal is important in questionable cases and with difficulty in the diagnosis of intestinal obstruction. The patient gets 50ml of barium suspension and have a dynamic study of the passage. Delay of contrast for more than 6 hours in the stomach and 12 hours - in the small intestine indicates a violation of patency or intestinal motility. In the case of mechanical obstruction, the contrast mass does not occur lower the barrier.

Endoscopic diagnostic methods. Urgent irrigoscopy is performed on suspicion of low obstruction and can detect the obstruction of the colon of the tumour, as well as to reveal the symptom of the trident - a sign of ileocecal invagination. Fibrocolonoscopy, rectoromanoscopy are performed according to indications and have an important role in the timely diagnosis and treatment of tumours, which causes the colon obstruction. Their implementation makes it possible not only to accurately localize the pathological process, to take a biopsy for the morphological verification of the process but also by the intubation of the narrowed part of the intestine to eliminate the acute obstruction and to perform a surgical intervention about the tumour in more favourable conditions.

In recent years, for the diagnosis of intestinal obstruction are increasingly using ultrasonography as a safe and highly informative method. Sonographic signs of mechanical intestinal obstruction are:

- enlargement of the gut lumen more than 2cm with the phenomenon of "sequestration of fluid";
- thickening of the wall of the small intestine more than 4mm;
- the presence of reciprocal movement of the chyme in the intestine;
- an increase in the height of the Kerckring folds over 5mm and an increase in the distance between them more than 5mm;
- hyperpneumatization of intestines in the previous part.

For the dynamic intestinal obstruction are characterised by other sonographic features:

- the phenomenon of "sequestration of fluid" in the bowel space;
- absence of reciprocal movement of chyme in the intestine;
- no relief of the bowel folds;
- hyperpneumatization of intestines in all departments.

Diagnostic laparoscopy - conducted only in case less informative results of previous methods of instrumental diagnostics.

Computer tomography is used in case insufficient informative instrumental diagnostics, as well as for the detection of various diseases of organs of the abdominal cavity, which may be the cause of acute intestinal obstruction.

Variants of the clinical course of acute intestinal obstruction.

Strangulated intestinal obstruction. This form of intestinal obstruction characterized by an ischemic component that occurs as a result of compression of the vessels of the mesentery. The disease begins suddenly by severe pain, paroxysmal character. The acuity of the pain syndrome, the suddenness of the disease and ischemic disorders lead to necrotic changes involving the intestinal tissue, which leads to deterioration of the patient's condition and body intoxication.

Obstructive intestinal obstruction. The disease begins with a strong attack pain, which disappears in the period between, and then increases. In the future, the pain of the attack becomes permanent. In opposite to the strangulation, it does not progress so fast. In the clinical picture of this type of intestinal obstruction, the symptoms of disorders of passage intestines that are manifested by prolonged intermittent pain and flatulence. More rather than symptoms of bowel destruction and peritonitis, which prevail.

Mixed mechanical intestinal obstruction. Includes the invagination and obstruction caused adhesions.

Invagination is the occurrence of one or more segments of the bowel in a segment below or above. It occurs more often in children under 4 years of age. In adults, it is caused by the presence of diverticular disease, polyps, tumours of the intestines. Are different types of the disease: thin-mesenteric invagination, iliac-loop (blind-loop) and thick-intestine itself. The disease begins with a sudden paroxysmal pain, then there is often vomiting, bloody overflow from the rectum. In the abdominal cavity, a tumour-like formation of a tightly-lustrous consistency is founded, which, during palpation, is spastically reduced, leading to increased pain.

Adhesive intestinal obstruction takes the first place among all types of intestinal obstruction and is a result of previous surgical interventions, inflammatory processes in the abdominal cavity and traumas. The occurrence of acute obstruction, as a rule, is preceded by chronic manifestations of adhesive disease: periodic paroxysmal pain in the abdomen, bloating, nausea, sometimes vomiting, periodical stool and gas retention after eating excessive amounts of food, physical activity. When acute adhesive obstruction occurs, these symptoms are stable, do not change under the influence of conservative therapy, but may progress. General condition worsens significantly due to severe metabolic disorders.

Paralytic intestinal obstruction. For this form of intestinal obstruction are characterized by pain, vomiting, delayed passage of gases and faeces. In the examination, a flattened stomach is visible, by palpation - the tension of the muscles of the anterior abdominal wall. Peristalsis abruptly weakened, determined the symptom of "grave silence". On the X-ray review - uniform swelling of all sections of the digestive canal.

Spastic obstruction. It is characterized by paroxysmal pain without a plain localization, there is no delay in gas and faeces, the general condition is satisfactory, the stomach is normal or retracted, sometimes muscle tension, small Kloiber cups on X-ray review.

Differential diagnosis. Acute intestinal obstruction has several signs that are diagnosed in other diseases and cause the need for differential diagnosis with diseases that have similar clinical signs.

Acute appendicitis. Common symptoms are abdominal pain, delayed stool, vomiting. However, pain with appendicitis begins gradually and does not reach the same strength as with obstruction. In the case of appendicitis, the pains are localized, and with obstruction, they are cramping in nature, more intense. Stronger peristalsis and sound of it that are heard in the abdominal cavity are characteristic of intestinal obstruction, and not appendicitis. In acute appendicitis, there are no radiological signs characteristic of obstruction.

Perforated ulcer of the stomach and duodenum. Common symptoms are sudden onset, severe abdominal pain, delayed bowel passage. However, with a perforated ulcer, the patient has a forced pose, and with intestinal obstruction, the patient is restless, often changes position. Vomiting is not characteristic of perforated ulcers, but is often observed with intestinal obstruction. In the case of a perforated ulcer, the abdominal wall is tense, painful, does not assist in the act of breathing, while with intestinal obstruction, the abdomen is distended, soft, not painful. With a perforated ulcer, peristalsis is absent from the very beginning of the disease, and the “splashing noise” is not heard. X-ray with a perforated ulcer determines free gas in the abdominal cavity, and with intestinal obstruction - Kloiber’s bowls, arcades.

Acute cholecystitis. Pain in case of acute cholecystitis is permanent, localized in the right hypochondrium, radiating to the right scapula. In case of acute intestinal obstruction, the pain is cramping, not local. Acute cholecystitis is characterized by hyperthermia, which does not happen with intestinal obstruction. Enhanced peristalsis, peristaltic sounds, radiological signs of the obstruction are absent in case of the acute cholecystitis.

Acute pancreatitis. Common signs are the sudden onset of severe pain, a severe general condition, frequent vomiting, bloating and a delay in defecation. But with pancreatitis, the pain is localized in the upper abdomen, a girdle, and not cramping in character. A positive Mayo-Robson symptom is determined. There are no signs of increased peristalsis, characteristic of mechanical intestinal obstruction, in case of the acute pancreatitis. Acute pancreatitis is characterized by an increased level of diastase in the urine and amylase in the blood. Radiologically with pancreatitis, a high standing of the left dome of the diaphragm is define, and with obstruction - Kloiber’s cups (bowls), arcades.

In the case of ***intestinal infarction***, as with acute intestinal obstruction, severe sudden abdominal pain, vomiting, severe general condition, and a soft stomach are diagnosed. However, pains with intestinal infarction are constant, peristalsis is absent, bloating is slight, and there is no asymmetry of the abdomen. During

auscultation, "grave silence" is determined. Intestinal infarction is characterized by the presence of embolic diseases, atrial fibrillation, pathognomonic high leukocytosis (20-30x10^9/l) is characteristic.

Renal colic and acute intestinal obstruction have similar symptoms - pronounced abdominal pain, bloating, delayed discharge of gases and stool, restless behaviour of the patient. However, the pain with renal colic radiates to the lumbar region, genitals, dysuric appearance with characteristic changes in the urine analysis, a positive symptom of Pasternatsky. On the survey radiograph in the kidney or ureter, shadows of calculus may be visible.

Pneumonia. In the case of pneumonia, abdominal pain and bloating may appear which gives reason to think about intestinal obstruction. However, high fever, rapid breathing, a blush on the cheeks characterize pneumonia, and a physical examination reveals crepitus wheezing, pleural friction noise, severe bronchial breathing, and dull percussion lung sound. An X-ray examination can detect a focus of pneumonia.

Myocardial infarction. In the case of myocardial infarction, severe pain in the upper abdomen, bloating, sometimes vomiting, weakness, decreased blood pressure, tachycardia, that is, signs like in case of strangulated intestinal obstruction can occur. However, with myocardial infarction, there is no asymmetry of the abdomen, increased peristalsis, symptoms of Valya, Sklyarov, Spasokukotsky and there are no radiological signs of intestinal obstruction. An electrocardiographic study helps to clarify the diagnosis of myocardial infarction.

Treatment of acute intestinal obstruction. Therapeutic measures for acute intestinal obstruction should be complex, taking into account the stage of the clinical course and the nature of possible complications. All patients with suspected acute intestinal obstruction should be urgently hospitalized in a surgical hospital. Depending on the type of obstruction, treatment may be conservative or surgical.

In cases of strangulated and decompensated bowel obstruction with peritonitis are the absolute indications for emergency surgery, which is carried out after short-term (within 2-4 hours) preoperative preparation. In other cases, treatment begins with conservative measures, which will usually be effective in case of dynamic intestinal obstruction, and can also eliminate some forms of mechanical obstruction - coprostasis, intussusception, sigmoid colon torsion, adhesive obstruction, tumour obstruction of the intestine.

The complex of conservative therapy measures includes:

- decompression of the upper digestive tract by aspiration of the contents through the nasogastric tube and the release of the distal parts by setting cleansing and siphonic clysters;
- elimination of pain, hyperperistalsis with antispasmodic therapy, bilateral perirenal novocaine blockade;
- infusion therapy, which is carried out with the aim of replenishing the volume of intravascular and intercellular fluid, correcting water-salt disorders, disturbances in acid-base balance, providing parenteral nutrition, regulating the

rheological properties of blood, replenishing the formed elements and individual blood components, and administering drugs. Among the infusion solutions, hemodynamic preparations based on hydroxyethyl starch (reftan, stabizol, gecodesis), correctors of water-electrolyte metabolism and acid-base conditions (Ringer's solution, sorbilact, lactasol) are widely used.

To improve microcirculation, reosorbilact, reopoliglukin, pentoxifylline are prescribed. Correction of protein metabolism disorders is carried out according to the principle of component therapy using drugs such as albumin, freshly frozen plasma. However, protein deficiency cannot be eliminated only by transfusion of albumin, plasma or other protein preparations, since a certain amount of time is required for their absorption. While the introduction of a mixture of amino acids (infezol, aminosol) allows you to quickly achieve a positive protein balance. However, it is advisable to combine them with solutions of sorbitol, xylitol or glucose.

An indication for transfusion of red blood cells or washed red blood cells is an increasing anaemia of the body due to exit from the active circulation of red blood cells, in which the haemoglobin content in the blood of patients and the level of hematocrit are respectively below 80 g/l and 0.3. The criterion for the sufficiency of infusion therapy is the normalization of the volume of circulating blood, hematocrit, central venous pressure, increased urine volume (at least 40 ml/h).

The outflow of a large amount of gases and faeces, the disappearance of pain and the improvement of the patient's condition after a series of conservative measures indicates the elimination of the fact of acute intestinal obstruction. With unresolved intestinal obstruction, it is contraindicated to use drugs that stimulate peristalsis. After effective conservative treatment, the patient requires further examinations to determine the cause of obstruction. If conservative treatment does not have a positive effect within 2-4 hours, then the patient must be operated.

Stages of surgery:

I. Access. To ensure adequate revision and rehabilitation of the abdominal cavity, a median laparotomy is usually performed. In some cases, in the presence of anatomical and technical conditions, it is possible to use laparoscopic and minilaparotomic accesses.

II. After laparotomy, the abdominal cavity is revised. The localization of the site of obstruction is founded by the state of the intestinal loops, which are inflated above the obstacle, and inactive below. At the same time, it is necessary to examine the intestines throughout, since the passage obstacles can be in several places and at different levels. With a pronounced intestinal overstrain of gas and liquid contents after laparotomy, intestinal intubation and evacuation of stagnant contents are primarily performed in order to improve the conditions for a full examination of the abdominal cavity and further surgical intervention

III. The scope of interventions is specific to the cause of the obstruction.

Radical operation:

- strangulated hernia - hernia removal;

- torsion - detorsion;
- nod formation - elimination of nodule formation;
- tumour formations - bowel resection;
- foreign bodies - entero- or colectomy;
- invagination - disinvagination;
- adhesive obstruction - dissection of adhesions.

Palliative surgery. Includes the entero- or colostomy over the level of obstruction. These interventions provide the elimination of acute intestinal obstruction without treatment of the main pathology that caused the obstruction (colostomy with intestinal obstruction of the tumour genesis) and, as a rule, are forced.

IV. Assessment of intestinal activity. It is carried out after the elimination of obstruction and restoration of blood supply. The main signs of intestinal viability are:
- pink;
- presence of peristalsis;
- pulsation of mesenteric vessels.

When in doubt about viability, using the following methods: introduction of a 0.25% solution of novocaine into the mesentery; warming the intestines with warm napkins; test with 10% sodium chloride solution (if the intestine is not viable, it wrinkles in response to the application of a hypertonic solution).

Assessing the viability of the intestine by the state of the serous membrane, it is important to take into account that the extent of own mucosal necrosis is much greater. It must be remembered that the drive segment of the intestine feels excessive intraluminal pressure. In this regard, the resection is carried with the obligatory removal of at least 40 cm of the drive and 20 cm of the outlet segment from the visible borders of necrosis. Then form the intestinal anastomosis. In extreme cases, the intestinal stoma can be removed.

V. Decompression of the intestine. The aim of intestinal decompression is to prevent the development of both intra-abdominal and systemic complications associated with ischemic damage to the intestinal wall, which persist for a long time after the elimination of obstruction. There are variants for conducting a probe for the decompression of the intestine - nasointestinal intubation, transanal intubation, intubation of the intestine through a gastro-, entero-or appendicstomy. Preference should be given to non-invasive bowel decompression methods. The most gentle and effective method of decompression of the small intestine is the nasogastrointestinal drainage with a Miller-Ebbott probe. Intestinal decompression involves the introduction into the small intestine of an elastic probe 8–9mm thick and about 3–3.5m long with multiple openings 2–2.5mm in diameter along the entire probe, except for the part that will be in the oesophagus, pharynx, and outside. Trying to pass the

probe into the small intestine as far as possible, and remove it the next day after the appearance of peristalsis and gas discharge, but no later than on the 7th day, since prolonged exposure of the probe threatens the formation of pressure sores in the intestinal wall. The colon is drained through the anus.

VI. Prevention of relapse of obstruction. This stage involves the elimination of factors that cause the intestinal obstruction. An example is Hagen-Thorn mesosigmoplication during inversion of the sigmoid colon. The method consists in fixing the elongated mesentery of the sigmoid colon to the parietal peritoneum with individual sutures. Also, a preventive measure is the performance of hernioplasty by removing the hernia.

A feature of the postoperative period in case of acute intestinal obstruction is the presence of severe paresis or paralysis of the intestine, water-electrolyte disorders, severe intoxication, and acid-base condition. Therefore, therapeutic measures aimed at eliminating these pathogenetic mechanisms started in the preoperative period and during surgery should be fully continued in the postoperative period. Moreover, specific therapeutic measures aimed at restoring the motor-evacuation function of the intestine should not be considered as an alternative, but as simultaneous and coordinated actions. Intensive stimulation of intestinal motility without adequate correction of cardiovascular, respiratory and hemodynamic disorders, energy, water-electrolyte and other metabolic disorders is considered erroneous and pathogenetically unreasonable.

Conducting the fulfil infusion therapy is the necessary background on which special therapeutic measures can be carried out aimed at solving postoperative paralytic intestinal obstruction. The daily volume of infusion solutions reaches 3-4 litres. Mandatory is anticoagulant and antibiotic therapy. The main component of complex therapy aimed at eliminating postoperative intestinal paresis, especially in severe forms, is a long decompression of the gastrointestinal tract, which eliminates the source of pathological nerve effects that arise when the intestinal wall is stretched due to the accumulation of fluid and gases, creates conditions for improving microcirculation and restoration of lost functions (motor, secretory, suction). Thus, intoxication is reduced, intestinal motility is enhanced.

Decompression prevents the failure of anastomotic sutures, eventration, provides further patency of the digestive tract after surgery about adhesive obstruction (carcass function). The decompression efficiency is significantly increased when, despite the regular removal of stagnant contents, the intestinal lumen is periodically washed with small portions (300-500ml) of saline solutions, which are identical in the consist with the chyme. It is also possible to prescribe targeted antibiotics through the probe (kanamycin, neomycin, polymyxin M) for selective intestinal decontamination. During this time, full parenteral nutrition should be provided. If, as a result of the therapy, the digestive and absorption functions of the small intestine are partially or completely restored, then enteric infusion of electrolyte solutions or polysubstrate mixtures through the probe becomes an effective means of stimulating motility and correcting metabolic disorders.

Mandatory components of therapy in the postoperative period are the use of cleansing, siphon and hypertonic enemas. An effective means of stimulating intestinal motility is the infusion of hypertonic solutions of sodium chloride, 20% sorbitol solution (sorbylact, lactosorbal). The restoration of intestinal motility is facilitated by perirenal novocaine blockade, prolonged epidural anesthesia, the positive effect of which is associated with the limitation of the sympathetic effect, reduction of pain, restoration of microcirculation and improvement of oxygenation in the abdominal organs. For the prevention and treatment of paresis and paralysis of the digestive canal, neurotropic drugs of a targeted action are used - ganglion blockers (dicoline, dimecoline, benzohexonium, pentamine, etc.), sympatholytics, which remove the "hypertonus" of the sympathetic nervous system, and do not block stimulating parasympathetic influences (isobarin, ornide, pyrroxan, chlorpromazine, nibufin). Sympatholytics are recommended to be introduced already 16-18 hours after surgery, however, stable hemodynamics and normovolemia are a prerequisite for their using.

Widely used in the clinic were: anticholinesterase drugs (proserin, prostigmine, uretide), to enhance the parasympathetic effect on intestinal motility, as well as antagonists of dopamine receptors (metoclopramide, cerucal, domperidone). A pronounced therapeutic effect on the restoration of the motor activity of the digestive tract was obtained using electrostimulation.

Complications of acute intestinal obstruction. Untimely treatment started is fraught with the development of serious complications. Obstruction in the intestine causes necrosis in its walls. This contributes to the out of intestinal contents into the abdominal cavity, and as a result, there is inflammation of the peritoneum - peritonitis. This condition usually goes into abdominal sepsis and, as a result, the patient’s death.

Forecast. The prognosis of acute intestinal obstruction depends entirely on the timely and complete volume of treatment. The poor prognosis is due to the late diagnosis of obstruction in the elderly with inoperable neoplasms and in people with low immunity.

Prevention. Preventive measures should include timely examination and treatment of tumours in the intestine, prevention of adhesion disease, preferring laparoscopy during abdominal surgery, after which the formation of adhesions is minimal. Timely elimination of helminthic infestations, proper nutrition, and, if possible, avoid injury.

Self-control questions

1. Anatomical and functional information about parietal and visceral peritoneum.
2. Etiology and peculiarities of the pathogenesis of acute intestinal obstruction.
3. Classification of acute intestinal obstruction.
4. The main clinical symptoms of acute intestinal obstruction.

5. Clinical picture of acute intestinal obstruction, depending on the cause and stage of development.
6. Characteristic clinical symptoms of acute intestinal obstruction.
7. Additional research methods for acute intestinal obstruction.
8. Dynamics of changes in laboratory parameters at different stages of acute intestinal obstruction.
9. Differential diagnosis of mechanical acute intestinal obstruction.
10. Preoperative preparation with acute intestinal obstruction.
11. Volume of surgical intervention and methods of surgery
12. How is elimination of causes of obstruction?
13. How is choosing the volume of resection of the intestine?
14. Define impressions for one-, two-, and three-step operations.
15. Conducting postoperative period, postoperative complications and their prevention.

Tests for self-control

1. What method of intestinal decompression is used during surgery?
   a. intestinal intubation;
   b. gastrostomy;
   c. introduction of novocaine into the udder;
   d. drainage of the abdominal cavity;
   e. anastomosis overlay.

2. The most common cause of fine-motor mechanical obstruction are:
   a. alien bodies;
   b. gallstones;
   c. tumor;
   d. connective tissue of the abdominal cavity;
   e. helminths.

3. A patient 45 years old, entered the surgical department with complaints of nausea, abdominal pain, multiple vomiting congestive intestinal contents, abdominal distension, delayed release of gases. Ill ill for 8 hours. Pulse - 108 beats per minute. The abdomen is symmetrically blown, soft, painful. When percussion marks a tympanitis, with auscultation - a splashing noise. Which diagnosis is most probable?
   a. acute pancreatitis;
   b. perforated ulcer of the stomach;
   c. acute intestinal obstruction;
   d. acute nonspecific colitis;
   e. thrombosis of mesenteric vessels.

4. For acute intestinal obstruction characterized by symptoms:
   a. Valya, Kivul, Grekov, Sklyarova;
   b. Obraztsova, Krymova, Shchotkina-Bljumberga;

c. Kerah, Ortner, Murphy;
d. Davis, Cullen, Gerbiah;
e. Voskresensky, Chuhriyenko, Meio-Robson.

5. A child aged 9 months during the day has become restless, crying, there were seizures. In the intervals between attacks - lies calmly. Several times it was vomiting. Feces in the form of "raspberry jelly" are observed. Belly palpation is soft, in the right half palpated painful formation of elastic consistency. What is the most likely diagnosis?
   a. dysentery;
   b. paralytic obstruction;
   c. spastic obstruction;
   d. acute appendicitis;
   e. invagination.

6. Decisive methods of research in the diagnosis of acute intestinal obstruction will be:
   a. X-ray examination of the abdominal cavity;
   b. angiography;
   c. esophagogastroduodenoscopy;
   d. laparoscopy;
   e. colonoscopy.

7. In what kind of intestinal obstruction can be observed the bloody discharge from the rectum:
   a. paralytic;
   b. spastic;
   c. invaginations;
   d. inversion of the small intestine;
   e. infarction of the intestines.

8. To what type of intestinal obstruction is the swelling of the small intestine:
   a. obstructive;
   b. strangulation;
   c. mixed;
   d. spastic;
   e. dynamic.

9. The cause of obstructive intestinal obstruction can be anything except:
   a. inflammatory conjunctions;
   b. swelling of the intestinal mucus;
   c. gallstones;
   d. invagination;
   e. tumor contraction.

10. The patient 69 years old complained about chronic constipation, periodic discharge of blood and mucus from the rectum. During the past week, the patient's condition gradually deteriorated: nausea appeared, periodic vomiting, emptying was not three days, during the last day gases did not escape. In the history: surgery and injuries of the abdominal cavity was not. The patient is diagnosed with acute intestinal obstruction. What is the most likely cause of obstruction?
    a. conjunctive disease;
    b. colon cancer;
    c. bowel movement;
    d. Stomach cancer;
    e. cancer of the small intestine.

11. The patient is 73 years old, complains about pain and bloating, nausea. Sufferers an ischemic heart disease, in a patient - postinfarction and atherosclerotic coronary heart disease. At inspection: the condition is heavy, the stomach is blown, the abdominal wall is weakly involved in breathing. At laparoscopy it was discovered that in the abdominal cavity there is a small amount of cloudy effusion, one of the loops of a thin, dark-colored colon cartilage. Formulate a preliminary diagnosis.
    a. Bashiha;
    b. spinal cord;
    c. acute intestinal obstruction;
    d. ischemic abdominal syndrome;
    e. thrombosis of mesenteric vessels.

ACUTE PERITONITIS

Definition. Acute peritonitis is an acute non-specific inflammation of the peritoneum, caused by different etiologic factors. Mortality in this disease, despite some progress achieved in its diagnosis and treatment, still rather high. Improvement of the results of peritonitis treatment is possible only with a comprehensive approach to its diagnosis, operative and postoperative treatment.

Anatomical and physiological features of the peritoneum. Peritoneum is a thin connective tissue, which covering all the walls of the abdominal cavity (parietal layer of peritoneum) and covers the surface of the organs of the abdominal cavity (visceral layer of peritoneum). The abdominal cavity is the space between the parietal and vissceral peritoneum. In men, it is closed. In women by the fallopian tubes, the uterus and the vagina is is interconnected with outside. The parietal peritoneum lining the anterior, posterior and lateral abdominal walls. The visceral is covers the organs of the abdominal cavity: stomach, part of duodenum, intestine, liver, gallbladder, spleen, uterus and Fallopian tubes. The area of the peritoneum is approximately equal to the area of the skin and equals 2-3m.

It is formed by six layers that have a different histological structure:

1. Mesothelium is present by cells of mesodermal origin - mesotheliocytes. They are characterized by fibrinolytic activity. When the cells are producing, a tape of fibrin is forming that protects the layers of the peritoneum under it.
2. Boundary, or basal, membrane is the superficial part of which is homogeneous, and deep is contains by delicate plexus of reticular fibers.
3. The superficial collagen layer is present by thin collagen fibers, located longitudinally.
4. The superficial diffusely elastic net, fibres of which are located closely.
5. The deep longitudinal elastic net, the fibers of which are thick and connected by thin communacations.
6. Deep latticed collagen-elastic layer.

In the different parts of the abdomen, the peritoneum has a different number of layers. Thus, the peritoneum, which covers the small intestine and abdominal wall, consists of six layers, the greater omentum it is for four, and on the diaphragmatic part is for three. The number of layers of the peritoneum depends on the intensity of absorption and secretion of the fluid. Absorption is more intense if fewer layers are in the area. The blood and lymphatic vessels of the peritoneum, which are located only within the sixth layer, take an active role in these processes.

The peritoneum covers the internal organs in different types. On all sides (intraperitoneal location), it covers the ileum, transverse colon and sigmoid colon, the cecum with the appendix, spleen, stomach, and fallopian tubes. On three sides (mesoperitoneal location) - the liver, gall bladder, ascending and descending colon, the middle part of the rectum, uterus, bladder. On the one side or it does not cover at all (extraperitoneal location) - the descending and horizontal parts of the duodenum, pancreas, kidneys, adrenal glands, prostate, lower part of the rectum.

The peritoneum passes from the wall of the abdominal cavity to the organs, and then, again to the wall, forms folds (plicae), ligaments (ligamenta), omentum and mesentery (mesenteria). From the lower surface of the diaphragm, it passes to the upper surface of the liver by the two ligaments: sickle-shaped (lig. falciforme) and coronary (lig. coronarium). After covering the upper surface of the liver in the gate area, both leaves converge and descend in by the hepatoduodenal (lig. hepatoduodenal) and hepatic-gastric ligaments (lig. hepatogastricum), which together with a small gastro-diaphragmatic ligament forming a lesser omentum (omentum minus). Its layers are fixing to the small curvature of the stomach and to the upper edge of the upper part of the duodenum. At small curvature, these layers diverging and cover the front and rear walls of the stomach. On the great curvature of the stomach, the peritoneum layers come together and go down in the shape of a wide “apron” - the anterior duplicate of the greater omentum, which returns below and goes up - the ascending duplicate of the omentum and grows together with the transverse colon and its mesentery. At the posterior wall of the abdominal cavity, the layers of the ascending duplicate diverged again. One lay on the wall rises up and merges with the posterior lay of the coronary ligament of the liver, and the second is goes down and goes into the posterior parietal peritoneum. Between the lays of the peritoneum of the greater omentum contains adipose tissue.

One part of the greater omentum, which stretches from the greater curvature of the stomach to the transverse colon, consists of two sheets of the peritoneum and it has called the gastrointestinal ligament (lig. gastrocolicum), and two anterior (descending) and two posterior (ascending) lays below the transverse colon more often fused together. Two posterior (ascending) lays of the peritoneum, which extend above the transverse colon, are mostly fuse with the mesentery of this intestine. At the top left, a large omentum passes into the gastro-splenic and diaphragmatic-splenic ligaments of the peritoneum. Therefore, the upper part of the greater omentum, which represented by the gastro-colon, gastro-splenic and splenic-renal ligaments, consists of two sheets of the peritoneum, and the lower part, which is located below the transverse colon, from four sheets.

The lower sheet of the mesentery of the transverse colon descends down the back wall of the abdominal cavity and wraps around the loops of the jejunum and ileum, forming the right and left sheets and the root of the mesentery of the small intestine, from which the right sheet of the peritoneum passes to the ascending colon and then to the right lateral and the anterior abdominal wall. The left sheet going to the left and has the same path as to the right side, differing in that it covers the mesoperitoneal lower and intraperitoneally with the formation of a mesentery and sigmoid colon. On the posterior wall of the abdominal cavity, the mesentery, on both sides, goes down in ferst to the cavity of the large, and then to the small pelvis.

In the pelvic cavity, the peritoneum, which is called the urogenital, covers the mesoperitoneally rectum, and then passes to the urinary bladder in men, and in women, first to the posterior wall of the vagina, uterus, and then to the bladder. Here, in men, a recto-cystic deepening (excavatio rectovesical) forms, and in women, a rectal-uterine (excavatio rectouterina) deep and vesicoureteral (excavatio vesicouterina) shallow deepening. From the right and left edges of the uterus to the

walls of the pelvis, two wide ligaments of the uterus stretch, along the upper edge of which between the sheets of the peritoneum the fallopian tubes are located.

After covering the bladder, the peritoneum goes up the front wall of the abdominal cavity, in the lower third of which it forms five umbilical folds (odd median, paired medial and paired lateral).

The median umbilical fold (plica umbilicalis mediana) covers the rudiment of the urinary duct. The medial umbilical folds (plicae umbilicales mediates), located on both sides of the median fold, contain desolate umbilical arteries. On the sides are lateral folds (plicae umbilicales laterales), which are formed by the relief of the lower epigastric arteries. Between these folds above the inguinal ligaments and pubic symphysis, paired fossae form: supravesical (fossa supravesical), which is located between the median and medial umbilical folds; the middle inguinal fossa (fossa inguinalis medialis), which is limited in the middle and side by the umbilical folds and corresponds to the projection of the superficial inguinal ring (this fossa is the exit site of a direct inguinal hernia); lateral inguinal fossa (fossa inguinalis lateralis), which is located on the side of the lateral umbilical fold. It is a projection of a deep inguinal ring - a place for an oblique inguinal hernia to exit from the abdominal cavity.

From the navel to the liver, a round ligament of the liver stretches in the midline, which lies in the duplication of the peritoneum and is a desolated embryonic umbilical vein. On the front wall of the abdominal cavity, the peritoneum rises up and passes to the lower surface of the diaphragm.

Blood supply the peritoneum receives from the organ that it covers. In the small intestine, the straight mesenteric arteries give two arterial branches lenght to 5mm long into the serous membrane of the colon. The straight artery in the submucosal layer of the intestine forms powerful vascular plexuses, the branches of which penetrate into the deep ethmoid collagen layer of the peritoneum.

Venous outflow from the peritoneum is via the portal and vena cava inferior systems.

The lymphatic system is closely connecting to the peritoneal lymph vessels of muscular body shell, which it covers.

The innervation of the visceral and parietal peritoneum is significantly different, which is the great clinical importance in diagnostics of diseases of the abdominal organs. So, the parietal peritoneum is innervated by the spinal (therefore, has a high pain sensitivity) and sympathic nerves. The innervation of the visceral part of the peritoneum is mainly presents by sympathetic nerves. In addition, the diaphragmatic and vagus nerves are participate in the innervation of the peritoneum. The deep nerve plexus of the peritoneum is located in its deep ethmoid collagen layer. It contains myelin and non-myelin nerve fibers, as well as ganglion cells, which are many in the parietal part of the peritoneum. Nerve fibers accompanied by the vessels of the peritoneum and partially woven into collagen fibers. The non-

myelin nerve fibers partially enter the surface layer of the peritoneum. A large number of sensitive nerve endings in the peritoneum is a combination of interoreceptors.

Pain is a protective function of the body arising from the effects of various agents (physical, biological and chemical) to pain receptors or sensitive nerve fibers. Pain as the protective function due to the fact that it occurs only when the pathological changes in tissues or as a result of exposure to and threatens their damage (inflammation, ischemia, mechanical traumatization, changes in osmotic pressure, the acid-base status and others). Pain is conducting along two nervous systems: peripheral (PNS) and central (CNS).

The PNS cunducts pain by the fast nerve fibers from the body surface or slowly, from deeply located tissues and organs. The central nervous system carries a painful irritation to the cerebral cortex (cortical pain) by thin myelinous rapid fiebers up to the thalamus (optical hill). For this kind, the exact localization of pain is characteristic.

Mobility of the hollow organs of the abdominal cavity relative to each other and the abdominal wall is possible due serosa that is wetted by the serous fluid released due to the active secretory function of the peritoneum. Secretion of the fluid is carried out by the blood vessels of the peritoneum, and its resorption is provided by lymphatic gaps and blood vessels. In this case, some sections of the peritoneum carry a secret, others - resorption of produced serous fluid. In the upper floor of the abdominal cavity, the intestine carries mainly secretion (peritoneum of the duodenum, small intestine), which is determined by the structure of the peritoneal intestinal system in these parts, where the blood vessels are more superficial than lymphatic. Absorption of fluid from the abdominal cavity is carried out by a peritoneum that covers the pelvic bed, diaphragm, and cecum. In these areas, on the contrary, the lymphatic vessels are more superficial than blood vessels. In the diaphragmatic and pelvic parts, the peritoneum, the lymphatic vessels are applied directly to the mesothelium lay, forming particular sucking hatches. In addition, in these parts, the peritoneum is devoid of frontier membrane.

Normally, in the abdominal cavity contains no more than 20ml of serum fluid containing cellular elements: mesothelium cells, lymphocytes, histiocytes, etc. It also has bactericidal properties, which greatly increases its sensitivity to infection. The main part of the roaming cells (histiocytes and macrophages) thats provide phagocytosis, is located in the abdominal cavity with a greater omentum, which, in addition, has a high plasticity and mobility, by limiting the focal points of infection in the abdominal cavity.

In case of abnormal processes in the abdominal cavity, the exudate initially has a serous component, and with the progression of inflammation there is an increase in the number of fluid and the transfer of the exudate in the pus, which leads to degenerative changes of the mesothelium, its damage and, as a result, to the formation of adhesive joints.

Consequently, the basic **physiological functions of the intestine** are:

1. Absorbtive, or resorptive. The peritoneum sucks in 1 hour 3-6 liters, per day - up to 70 liters of tissue fluid.

2. Excretion, or transudative. For 1 hour, the same amount of liquid is released, how much is absorbed. The most excretioric function has the peritoneum, which covers the duodenum and the small intestine.
3. Protective, or barrier. Exudate of the abdominal cavity has bactericidal and bacteriostatic properties.
4. Plastic. The uniiriated peritoneum, the peritoneum reacts with the release of fibrin and the formation of joints, thereby limiting the locus from the inflammatory process. Due to this property after performing surgical interventions on the cavity organs, their sutures are pressurized with fibrin.

Consequently, the peritoneum makes a lot of functions that provide vital functions of the organism.

Etiology and pathogenesis of acute peritonitis. The most common cause of acute peritonitis is an infection that enters the abdominal cavity with a violation of the entire cavity organs of the digestive system or their necrosis (gangrenous cholecystitis and appendicitis, peptic ulcer of stomach and duodenum, nonspecific ulcerative colitis and Crohn's disease, acute obstruction of the intestine) or permeability violation of the organ walls on acute surgical diseases (acute appendicitis, acute cholecystitis, acute obstruction of the intestine, etc.). Acute peritonitis may occur with pancreatic necrosis (acute pancreatitis). Less commonly, it occurs when an exogenous infection enters in abdominal cavity with traumatic damage of the organ wall, or after surgical interventions.

The factors of the occurrence of acute peritonitis are:

- complication of gross inflammation of the organs of the abdominal cavity, small pelvis and retroperitoneum, perforation of hollow organs;
- closed and penetrating injuries of the abdominal cavity, small pelvis;
- postoperative peritonitis;
- primary hematogenous and cryptogenic peritonitis (occurring very rarely).

In addition, spontaneous bacterial peritonitis, which is one of the most severe complications of ascites, is also distinguished. In this case, there are no other sources of intraabdominal infection in the body, and microorganisms or neutrophilic granulocytes in an ascitic fluid in excess of 250 cells perml detected. Most often spontaneous bacterial peritonitis complicates liver cirrhosis (in 6-30% of patients), rare - nephrotic syndrome or systemic lupus erythematosus, sometimes - cardiovascular failure.

The main cause of the occurrence of acute peritonitis is the entry into the abdominal cavity of microorganisms from different parts of the digestive system. In addition, the disease or damage to one or another body depends not only the composition and number of microflora, and its virulence and the rate of spread of inflammation in the peritoneum, the severity of the disease.

So, normally 1ml of gastric secret contains up to 1000 microorganisms, or 0-10^3 colony-forming units. These are mainly anaerobic streptococci and lactobacilli,

while anaerobes founded very rarely. With low acidity of gastric secret, the number of microorganisms increases highly. In the initial parts of the small intestine, the number of microorganisms is $0\text{-}10^5$ colony forming units in 1ml, and the ratio of aerobic/anaerobic is 10:1. In the terminal part of the small intestine, it is $10^5\text{-}10^9$ colony-forming units in 1ml, with an aerobic/anaerobic ratio of 1:100, and in the colon, more than 10^9 colony-forming units. The microflora of the colon characterized by a significant predominance of anaerobic bacteria over aerobic, with an aerobic/anaerobic ratio of 1:1000. Thus, these data suggest that the heaviest clinical case is acute peritonitis, which caused the microorganisms of the colon.

Pathogenesis of acute diffuse peritonitis. In response to the profress of the inflammatory process and intoxication syndrome, caused by the formation of many toxins that occur during the death of microorganisms, cells and tissues of the human organism, activation of biologically active substances, which take a main role in the subsequent course of peritonitis and syndrome of endogenous intoxication, occurs. Caused by their action, interoreceptors are damaged; capillary permeability is increases, blood stasis forming. Systemic disorders appear by changes in the water-electrolyte balance, the acid-base state, the metabolism of proteins, carbohydrates and fats. This causes tissue hypoxia, the development of metabolic acidosis, increased blood coagulability, capillary toxicosis, blood stasis, which raise to the arising of disseminated intravascular coagulation syndrome (DIC).

In the damage of interoreceptors, the tone decreases, and subsequently, intestinal paresis occurs with sequestration of a large amount of liquid, salts, microelements, proteins, carbohydrates, fats and blood cells in the intestinal clearance and in the abdominal cavity. As a result, ischemic changes progress in the intestinal wall, it becomes permeable to microorganisms, which cause to their entry into the abdominal cavity and into the bloodstream, as a result of which cytokines appear, which induce to heavy disorders of organism vital functions. It should be noted that the intestines constantly contain various microorganisms that coexist peacefully in a human's body. They participate in many essential processes, particularly, in providing trophic processes of the intestine wall, microbial fermentation at the phases of digestion, synthesis of vitamins, etc.

At the background of acute peritonitis, the pathological changes arise in all organs and tissues of the body. Stasis progresses, capillary permeability increases significantly, which cause tissue oedema with the forming of hypovolemia and a decrease in the volume of circulating blood. The body accumulates toxic metabolic products, which increase toxicosis and worsen the patient's condition. A further increase in endotoxemia causes a high offence of the functions of organs that provide detoxification processes (liver, kidneys). In the progression of these conditions, signs of multiple organ failure syndrome appears, the first manifestation of which is damage to the respiratory system, followed by cardiovascular, liver and renal failure. The main symptom of multiple organ failure in peritonitis is toxic encephalopathy.

In the pathogenesis of peritonitis, inflammatory mediators - cytokines, which are low-molecular protein mediators produced by different cells (endothelial cells, leukocytes, fibroblasts, etc.) have an important role. Their biological activity is

appeared by action on high-specific receptors, which are located in the cells. However, interleukins, and the tumour necrosis factor acting on all the cells, present a systemic effect.

The main ***functions of the cytokines*** are:
- participation in the inflammatory response;
- regulation of growth and differentiation of individual cells;
- influence on growth of tumors ;
- participation in regeneration of damaged cells;
- immunity protection.

In a healthy body, self-regulation processes constantly occurs, including self-regulating immune responses, and the part of cytokines in supporting these processes is very important. They can regulate the course of metabolic processes, increase of energy generation, help to the appearance of a hyperthermic reaction, stimulate the regeneration of damaged tissues and wound healing.

At critical conditions, particularly in acute peritonitis, the production of cytokines is stimulated or the pathways of their formation are activated, as a result of which the autoregulation reactions modify and become uncontrollable. The result of these processes is the beginning of interstitial oedema, which causes is not regeneration but the tissue destruction. Since endothelium, blood cells and tissue macrophages, the main sources of cytokines synthesis, are which are available in the body, therefore that an increase in the level of cytokines cause to impaired functions of not only those organs that reduce to the emergence of a critical condition, but also of all organs and systems of the body (multiple organ failure syndrome). About diseases of the abdominal organs, a pronounced aggression of inflammatory mediators is as a result of the appearance of gram-negative microorganisms in the blood of endotoxin.

Bacterial infection and the intoxication syndrome associated with it in acute peritonitis cause the immunodeficiency progression, which causes the progression peritonitis progression and significant deterioration in the patient's health. Temporary immunodeficiency state of the patient during the first days of the occurrence of acute peritonitis plays a positive role, limiting the appearance of secondary, autoimmune in nature, necrobiosis. This is the physiological essence of transitional immunodeficiency.

Thus, to prevent a disruption of the activity of internal organs and body systems or to rise their condition in acute peritonitis and to achieve success in its treatment, given the pathogenesis of the disease, it is possible only if the peritonitis source is tin-time removed, abdominal cavity sanitation, organs function normalizing, and rational antibiotical therapy, eliminating homeostasis disorders.

Pathomorphology of acute peritonitis. The pathomorphology of acute peritonitis is based on a combination of local and general changes. Local changes include the condition of not only the peritoneum, but also the entire abdominal cavity.

The main macroscopic signs of peritonitis during autopsy is the full-blood peritoneum, often with the presence of pinpoint hemorrhages, including in the form of stripes on the adjoining loops of the intestines or organs, and also the dull appearance of the surface of the peritoneum as a result of the layering of fibrin. If there is described signs of inflammation of the peritoneum and the absence of free liquid in the abdominal cavity conclude so-called "dry peritonitis". Most often, same peritonitis develops after surgery or a large diagnostic revision of the abdominal organs. Typically, such postoperative "traumatic" peritonitis for 2-3 days or a reverse development proceeds in exudative forms upon infections.

A very rare form of "dry peritonitis" is a tuberculous damage of the peritoneum. In these cases, severe hyperaemia and a dull appearance of the peritoneum, the presence of multiple petechiae and small tubercles are appearing. In a microscopic examination, they have the structure of typical tuberculous granulomas.

In most cases of peritonitis, is presence the one or another type of exudate. However, it may be general or localized. As a rule, the first is formed if it is delimited by joitns and organs. In the early stages the joints are friable, sometimes even almost imperceptible, with a long course with a long course of localized peritonitis, dense adhesions form, among which there are accumulations of thick purulent exudate - abscesses. And with common peritonitis, exudate can accumulate in the subdiaphragmatic and subhepatic spaces, forming the typical abscesses, as well as in the bag of the lesser omentum and in the pelvic cavity.

Another critical moment is the characteristic of the exudate, which reflects a certain extent of the cause and progress timing of peritonitis. Exudate can be serous, fibrinous, purulent, hemorrhagic, but more often it is unifying character: seroplastic, purulent-fibrinous, purulent-fibrinous with a pronounced necrotic component. The character of the inflammatory reaction of the peritoneum depends on the cause and time of the progess of peritonitis.

The inflammation of the peritoneum in the first three days have a serotic or fibrous character. In this case, there is a hyperemia of the visceral and parietal layers of the peritoneum and omentum, and also are little areas of hemorrhages. Microscopic examination: the serous membrane thickened and diffused due to edema. Small arteries and arterioles of subserous membrane are bloodlessed, their walls are thickened due to plasmatic seeping. Venous vessels are dilated and bloodlessed. There is a low infiltration by lymphocytes and neutrophilic granulocytes, there are slight layering of fibrin. Electronic microscopy: seeping and partial desquamation of mesotheliocytes, expansion of intercellular gaps.

If peritonitis is long over, it has fibropurulent or purulent course. With fibrinous peritonitis, the surface of the peritoneum covered with a thin “tape” of fibrin, which looks rough and dull (not smooth). Later, this tape becomes green and pus appears. In this case, the volume of exudate can be from several mililiters at local type to several liters with diffuse peritonitis.

The main places of pus cumulation at peritonitis are:

- a site at the source of inflammation (appendix, gall bladder, stomach);
- the lowest departments of the abdominal cavity (lateral canals, small pelvis).

Therefore, with peritonitis of appendicular origin, pus accumulates in the right ileum, around the cecum; against cholecystitis - under the liver; against perforated ulcer of the stomach or duodenum - in the area of their location, as well as in the small pelvis, where it flows through the lateral channels.

With common peritonitis, the arteries and veins of the mesentery can be involved in the inflammatory process, with the development, in such cases, of purulent thrombophlebitis, which (when spread through the portal vein system) can lead to the formation of multiple abscesses in the liver. The spread of infection through the lymphatic vessels accompanied by the development of mesenteric and retroperitoneal lymphangitis and lymphadenitis. In histological studies, the peritoneum is much thickened, fibrinous or fibrinous-purulent exudate with a large number of neutrophils and lymphocytes observed; there are small abscesses and points of necrosis. Roughness of the lay of the peritoneum caused the presence of inflammatory infiltrates. Vascular stasis, fibrin, mixed blood clots and signs of infiltrative proliferative vasculitis are visualised in the vessels.

In fibrinous-purulent or purulent-necrotic peritonitis, the peritoneum is much thicked due to fibrinous-necrotic overlays, severe sclerosis and inflammatory infiltration (lymphocytes, macrophages, neutrophils and single eosinophils). The subserous base of the peritoneum has signs of severe fibrosis. In a month, the exudate organized with the formation of joints between the bowel loops with the development of ulcers encysted.

Characteristic morphological signs that caused by the cause of the development of peritonitis are not detected. Although there are features that are associated with the predominant causative agent of peritonitis. Therefore, purulent exudate with a clear and mixed coli-bacillary infection has a strong fecal smell, and with anaerobic non-clostridial peritonitis there is pungent odor of contents of the abdominal cavity, which mainly called “ichorous”. In this case, the exudate has a dirty green or brown color with accumulations in the form of jelly-like masses or massive overlays of fibrin on the peritoneum. Exudate in peritonitis, which is caused by hemolytic streptococcus, liquid, watery, odorless. In moist TB-peritonitis, the exudate is liquid with a small amount of fibrin, and with attached to another flora, it is a cream-like pale-green color with small tubercles present on the peritoneum.

In case of destructive pancreatitis, infiltrated peritonitis may be aseptic. Hemorrhagic exudate contains a large number of proteolytic enzymes and almost no pathogenic microorganisms. Among patients with secondary pancreatogenic peritonitis, which occurs during purulent-necrotic processes in the pancreas and parapancreatic tissue, the exudate is mainly purulent with a large number of microorganisms and does not contain pancreatic enzymes. In such cases, steatonecrosis observed in the retroperitoneal tissue and omentum.

Permanent signs of peritonitis (including postoperative) include intestinal paresis expressed to one degree or another. The walls of the stomach and intestines are thickened, swollen, with dilated vessels and hemorrhages in the mucous and serous membranes. Histological: inflammatory infiltrates that spread from serous to muscle and submucous membranes.

A characteristic sign of the common changes observed in peritonitis are disorders of circulations. Macroscopic: uneven blood supply to organs and tissues, peritoneum (in particular), intestines, liver and kidneys; revealed uneven blood vessel enlargement, erythrocyte aggregation, leukocyte stasis and areas of hemorrhage. In the small vessels of most internal organs, fibrin clots are often founded which evidence of the development of DIC.

The main morphological substrate of organ dysfunction and multiple organ failure is cell damage to parenchymal organs. A macroscopic sign of such disorders is a dull appearance of organs. In the section, the liver has a typical «cut of a nutmeg». Histological examination shows varying degrees of damage and necrosis of hepatocytes (from focal to massive) usually there is damage and a decrease in the number of endotheliocytes and stellate macrophagocytes (Kupffer cells). Such damage to the liver cells is at the core of the disorders of its barrier and detoxification functions.

Kidneys damage occurs in the prerenal form of acute renal failure, which characterized by hypoperfusion and ischemia of the cortex with tubular necrosis, as well as the renal form of acute renal failure, the clinical and morphological manifestation of which is acute glomerulo- or interstitial nephritis.

The occurrence of pulmonary complications by peritonitis with the development of respiratory failure significantly aggravate the condition of patients and worsen the prognosis of the disease. Inhibition of breathing generally correlates with stage of peritonitis. Thus, the spread of peritonitis increases blood levels of toxic compounds, including endotoxin, which leads to disorders intraorganic hemodynamics, tissue hypoxia, acidosis, pulmonary surfactant system damage. Reduced surface activity of surfactant is due to a decrease in its synthesis with type II alveolocytes and loss of functional activity. In patients who die with peritonitis, pathomorphological changes that occurs in the lungs caused by the development of pneumonia and (or) acute respiratory distress syndrome. In most cases of peritonitis, inflammation localized in general in the VII-IX and X segments of the lungs.

Pathological anatomy of the heart under peritonitis characterized by an increase microcirculation disorders and severe damage to cardiomyocytes. In autopsy, the presence of blood bundles in the cavities of the heart is finded. In this case, the heart muscle has a dull outlook with yellowish spots. On histological preparations of the heart, the vessels of the microvasculature are full-blooded, sometimes with signs of blood stasis in the capillaries and small venules. With general purulent peritonitis, there is a parietal location of leukocytes and signs of diapedetic hemorrhages. In various parts of the myocardium, plasma seeked of artery walls and damaged cardiomyocytes are visualizated.

Morphological changes in the adrenal glands characterized by circulatory disorders and a decrease in lipid content and the presence of micronecrosis in adenocorticocytes. There is a much full-blood sinus in the cortex and medulla, aggregation of blood cells and little hemorrhages.

Morphologic study of the immune system organs shows significfat inhibition of lymphoid tissue of both central and peripheral organs immunogenesis. Histological examination of the lymph nodes and spleen shows the absence of light

centers in the lymphatic follicles and even their disappearance and a decrease in the content of blasts and mitotic activity.

In pathomorphological studies of deaths caused general peritonitis, swelling of the membranes and brain matter usually finded, and in severe cases, small hemorrhages in them.

Thus, the main microscopic signs of general changes in organs and tissues by peritonitis are pronounced circulatory disorders, recurrent and irreversible damage, as well as cell necrosis. These processes make the material substrate of organ dysfunction and multiple organ failure. Clinical expression organ failure depends on the etiology of peritonitis, the incidence and duration of the inflammatory process, and tolerance of individual organs cells to damage factors and their compensatory capacity. The degree of organ dysfunction may range from slight to severe decompensation latent shifts.

Classification of peritonitis:

There are several classifications of acute peritonitis. Per ICD-10, peritonitis classified as:

K65.0 Acute peritonitis

Abscess (of):

- abdominopelvic
- mesenteric
- omentum
- peritoneum
- retrocaecal
- retroperitoneal
- subdiaphragmatic
- subhepatic
- subphrenic

Peritonitis (acute):

- generalized
- pelvic, male
- subphrenic
- suppurative

Use additional code (B95-B98), if desired, to identify infectious agent.

K65.8 Other peritonitis

Chronic proliferative peritonitis

Mesenteric:

- fat necrosis
- saponification

Peritonitis due to:

- bile
- urine

K65.9 Peritonitis, unspecified

Excl.: peritonitis:

- aseptic (T81.6)
- benign paroxysmal (E85.0)
- chemical (T81.6)
- due to talc or other foreign substance (T81.6)
- neonatal (P78.0-P78.1)
- pelvic, female (N73.3-N73.5)
- periodic familial (E85.0)
- puerperal (O85)
- with or following:
  - abortion or ectopic or molar pregnancy (O00-O07, O08.0)
  - appendicitis (K35.-)
  - diverticular disease of intestine (K57.-)

The most common in clinical practice is the **classification**:

***Etiology*:** primary, secondary. Primary - occurs due to the ingestion of microorganisms into the abdominal cavity by the hematogenous or lymphogenous, as well as during their translocation from other organs. Secondary - occurs due to the ingress of microorganisms into the abdominal cavity with acute surgical diseases of its organs or its damage.

The causes of secondary peritonitis: traumatic; postoperative; perforated; inflammatory - with the spread of the inflammatory process from the affected organ of the abdominal cavity without its perforation.

According to the stages of the course: reactive (first 24 hours from the onset of the disease) toxic (24 to 72 hours after the onset of the disease) terminal, which according to actual data is not instance stage, but only a manifestation of the maximum expression of endotoxicosis.

By the nature of the exudate: serous, serous-fibrinous, fibrinous, fibrinous-purulent, purulent, putrid, hemorrhagic, biliary, fecal, chemical, mixed.

By the microflora: aerobic (staphylococcus, streptococcus, intestinal wand, etc.) anaerobic (bacteroids, peptococci, etc.) mixed - aerobic-anaerobic; specific (syphilitic, tuberculous, chlamydial, gonococcal, etc.).

By the clinical course: acute, chronic, and slowly progressing.

By prevalence: delimited, not delimited, which divided into local, diffuse, spilled and common. *The delimited peritonitis* if inflammation dissociates itself from the rest of the abdominal cavity by anatomical structures - a large omentum, intestinal loops. This occurs when there is a high reactivity of the organism and low microflora virulence. Example: infiltrates and abscesses of appendicitis, cholecystitis, pancreatitis or abscesses of other localization. *Not delimited peritonitis* if the inflammatory process from the place of occurrence freely spreads through the abdominal cavity. Depending on which parts of the abdominal cavity it spreads, it is define:

a. *local* - extends to one of the 9 anatomical zones of the anterior abdominal wall, that is, adjacent to the site of peritonitis. Example: for appendicitis - to the right iliac region, for cholecystitis - to the right hypochondrium.

b. *diffuse* if the inflammatory process extends not only to the peritoneum at the place of origin, but also to the adjacent areas, but not more than three. Thus, in acute appendicitis, the pelvic peritoneum, the right iliac and suprapubic regions may be involved in the inflammatory process.
c. *spilled* if the inflammatory process extends to more than three, but not more than six anatomical sites. Example: the phenomena of peritonitis appear in the lower floor of the abdominal cavity during appendicitis or perforation of the sigmoid colon or intestinal obstruction.
d. *general* if the faetures of peritonitis spreads to the entire abdominal cavity.

By severity:

I. *grade (mild)* - signs of endotoxicosis are not pronounced;
II. *grade (moderate)* - to eliminate the signs of endotoxicosis, it is enough to eliminate the cause of the development of acute peritonitis by surgery and conduct intensive therapy
III. *grade (severe)* - signs of endotoxemia after the elimination of the causes of acute peritonitis may be eliminate only by using extracorporeal detoxification methods (hemosorption, lympho-sorption, plasma exchange, etc.)
IV. *grade (very heavy)* - requires re-sanation of the abdominal cavity;
V. *grade (terminal),* when there is a syndrome of multiple organ failure, life-incompatible.

The clinical picture of acute peritonitis. The course of acute peritonitis depends on the cause of the occurrence, nature and virulence of microorganisms, the spread of the pathological process, the stage of the course, status of the body's immune system.

In clinical practice, define subjective and objective signs of acute peritonitis. Subjective signs include abdominal pain, nausea, vomiting, dysfunction of the intestines (***delayed stool and discharge of gas, bloating).***

Usually acute peritonitis has a sudden begiening with abdominal pain, which localized in the area of peritonitis, then spreads throughout the abdominal cavity, and becomes more intense. In the later stages, the intensity of pain decreases due to damage to the peritoneal sensory nervous endings.

Nausea and vomiting are permanent signs of acute peritonitis that appear at the begiening of the disease in the reactive stage and usually are reflexive. The nature of the vomit masses depends on the stage of peritonitis, at the beginning of the disease is the content of the stomach is prevalent, and when the paresis of the intestine - there are impurities of the contents of the small intestine. Usually vomiting does not bring ease.

In case of disorders of intestinal function, patients complains of delay of the gases and stool, bloating. These signs are the result of acute paralytic intestinal obstruction. At the same time, the patient's position in bed is forced and he is passive, trying to protect the painful, inflamed part of the abdomen affected by additional irritation.

The appearance of the patient is characteristic of acute common peritonitis. At the beginning, patient's face is hyperemic, eyes are gloss, and tongue is dry, covered with white layers. With the progression of the inflammatory process, the facial features become sharper; bruises appear under the eyes, lips bluish, face covered with cold sweat. The body temperature rises to 38 ° C or more.

There is a disorder of cardiovascular activity, which caused by endotoxicosis and manifested by pain in the heart zone. The pulse is frequent, mild, more than 100 beats per minute, the blood pressure is lowered; with the more frequent pulse, the lower the blood pressure. The ratio of these indicators used to estimate the severity of the course of peritonitis and the prognosis of disease.

As result of the diaphragm high standing, which caused by flatulence, as well as the tension of the anterior abdominal wall muscles, the respiratory disfunctions. The rate of respiratory movements reaches 24-30 in 1 minute; respiration is superficial thoracic type. Moreover, more pronounced superficial breathing and what it is more rate, the prognosis of the disease is worse.

At the starts of the disease, the abdomen is tense, does not take stand in the act of breathing, and after the beginning of intestinal paresis, flatulence appears and it is swollen.

Palpation of the abdominal wall causes pain and tension, which much pronounced in the site of the source of peritonitis. It is necessary to start palpation from a site far from the locus of peritonitis, slowly approaching to it. In this case, there are positive symptoms of peritoneal irritation: Shchotkina-Bljumberga and Rosdolsky (the emergense of pain by tapping the fingers on the front abdominal wall), which is most expeessed in the projection of process.

Percussion of the front abdominal wall characterized by a high percussion sound (tympanitis). If the abdominal cavity accumulate the fluid in gentle places and over the pubis appears dulling percussion sound.

In the early stages of acute peritonitis, in the auscultation of the abdomen, intestine peristalsis is preserved, but weakened. With spread of inflammation and the progression of intoxication syndrome, paresis of the intestine is occurs, peristaltic noises are not audible at all (the symptom of "trembling silence").

Rectal examination may present overhanging and pain in the front wall of the rectum, and in the case of vaginal pain is the incontour pain, which stronger with uterine displacement.

However, recent clinical cases of acute peritonitis were changed somewhat. There is an atypical course of the clinical course, the fuzziness of classical peritoneal symptoms among weak patients, especially with secondary immunodeficiency, and patients with haevy concomitant pathology and old. Complicated is the diagnostics of specific peritonitis, which appears as result of intestinal syphilitic ulcers perforation, tuberculosis, typhoid fever, and mortality about such diseases can reach 90%.

Recently, however, the clinical manifestations of acute peritonitis have changed somewhat. There is an atypical course of the clinical picture, lack of clarity of classical peritoneal symptoms in debilitated patients, especially with secondary immunodeficiency, in patients with severe comorbidities, and in elderly and senile

age. Difficult is the diagnosis of specific peritonitis, which occurs due to the perforation of intestinal ulcers in syphilis, tuberculosis, typhoid fever, and mortality that diseases can reach 90%.

In addition, recently the following trends been noticed in the clinical course of acute peritonitis: 1) fast progressive - among patients with saved body immune protection; 2) slowly progressive or torpid - patients with immunodeficiency that must be consider for diagnostis.

In case of delayed treatment of the cause of peritonitis (untimely treatment of the patient by medics, inadequate treatment tactics), the reactive stage becomes toxic, when symptoms cause intoxication syndrome resulting from ingress of toxic substances as a result of the microorganisms death and tissue damage, as well as contents stasis of the intestines. The intensity of the pain decreases, intestinal paresis emergence and vomiting becomes much frequent. The tongue is dry, the features are pointed, bruises under the eyes, the gases outcome stops. Pulse up to 120 rates per 1 minute, the blood pressure decreases. The breathing is rapid, superficial. The abdomen is swollen, does not involvement in breathing, peristalsis is absent, positive symptom of Shchetkin-Blumberg throughout the abdomen. In addition, the symptoms of intestinal obstruction - Kivul's (with percussion of the anterior abdominal wall tympanitis with a metallic tinge is auscultated), Sklyarov (with jerky vibrations of the anterior abdominal wall with a phonendoscope, the "sound of a splash"), etc.

The severity of acute peritonitis caused by the action of toxins on the nervous, cardiovascular and respiratory systems, liver, kidneys, as result of which they have degenerative changes. An important role in the course of this stage belongs to the paralytic ileus of the intestine, which leads to disruption of homeostasis and all types of metabolism.

With the fast progression of the disease comes the terminal stage, in which there is a worse in the patient's condition with the leading of adynamia, a significant expression of intoxication syndrome, inhibiting and disorientation of the patient. The pulse rate increases (more than 120 per 1 minute), thus the blood pressure significantly decreases, diuresis decreases - less than 500ml per day, significant metabolic disorders are occours, and prognostically unfavorable manifestations are forming. The tongue is dry, the features are pointed, the eyes are lowed, the skin is covered with cold sweat. The abdomen is swollen, does not involvement in breathing, painless about palpation. Peristalsis of the intestine disappearing (a symptom of "dead silence"). The cause of peritonitis in this stage is usually difficult to set.

Different methods give the possibility to evaluate the severity of the patient’s condition to the prognosis of the disease course. The most common in surgical practice definitions of the Mannheim Peritonitis Index (MIP), the APACHE II system (Acute Physiology and Chronic Health Evaluation - the evaluation of physiological and chronic adult health status), the peritoneal index ALTONA, etc. Laboratory and clinical indicators, gender, and age of the patient, the presence of chronic and concomitant diseases.

Diagnosis of acute peritonitis. The diagnosis of acute common peritonitis is based on the patient's complaints (abdominal pain, nausea, vomiting, delayed gas, and stool vomiting, bloating); disease anamnesis (patient's presence of diseases that can cause peritonitis - gastric or duodenal ulcers, acute cholecystitis, acute pancreatitis, open or closed abdominal damage); objective status of the patient (skin is pale, bluish; blueness under the eyes, pointed features; dry, coated tongue, low mobility, stiffness, the forced body position) and the objective status of the abdomen (strained, does not involve in the act of breathing, positive symptoms Schotkin-Blumberg, Rozdolskogo, bowel disfunction - flaccid or absent peristalsis); cardiovascular and respiratory systems (fast pulse rate, reduced blood pressure, shallow breathing, chest-breathing); indicators of laboratory analyses (high leukocytosis, white blood cell count shift to the left, the emersion of young forms of leukocytes, an increase in ESR, impaired ion exchange, etc).

A diagnostic program for suspected acute peritonitis: clinical tests of blood and urine, determination of the blood group using the ABO system and rhesus factor, urea, potassium, sodium, chlorides, glucose; total protein and its fractions, creatinine, residual nitrogen in the blood serum, indicators of the blood coagulation system, blood tests for sterility and blood culture, electrocardiography in rest.

To confirm the diagnosis, especially in dubious clinical cases, additional methods of examining the organs of the abdominal and thoracic cavities are used: fluoroscopic and radiographic examination, CT scan of the abdominal and retroperitoneal space, US; laparacentesis and laparoscopy.

With the X-ray examination of the abdominal organs, as a rule, gas is detected in the intestine, on the horizontal fluid levels (Kloyber bowls); and by perforated or traumatic damage to the cavity organ - free-gas under the dome of the diaphragm (pneumoperitoneum). Using CT scan and magnetic resonance imaging (MRI) diagnose delimited peritonitis and determine the presence of fluid in the abdominal cavity. The diagnostic value of these methods is very high - up to 96%.

Recently, using US in the case of the assumption of the presence of acute peritonitis has received a high propagation. Using this method it is possible to define the contours of the abdominal organs, the thickness of their walls, their content, the presence of gas and fluid in the intestine, abscesses in the abdominal cavity. About acute peritonitis, it is possible to detect the fluid in the abdominal cavity, and an acute inflammatory process in organ, which is the origin of peritonitis.

Laparocentesis is indicate for a dubious diagnosis and unconsciousness of the patient. It confirms the diagnosis of acute peritonitis the presence of fluid in the abdominal cavity, and the color and character provide it possible to determine the cause of peritonitis (an admixture of bile, blood, food pieces, etc.).

The use of laparoscopic methods, like laparocentesis, helps to determinate the diagnosis. However, this informative method for the diagnosis of acute peritonitis should be used very carefully. According to their results, not only may confirm the diagnosis of acute peritonitis, but also determine the nature of the effusion, detect diseases or organ damage that cause the progress of peritonitis. In difficult diagnostic cases, if they suggest the presence of acute peritonitis, a microlaparotomy or diagnostic laparotomy is carried to confirm the diagnosis.

Differential diagnosis of acute peritonitis. Differential diagnosis of acute diffuse general peritonitis should be performed with diseases that are similar in clinical course but do not require surgical treatment. These are diseases which the occurrence of so-called false acute peritonitis is possible. These include diseases of the lungs and pleura (lower segments pneumonia, basal pleurisy), cardiovascular system (abdominal form of acute myocardial infarction), gynaecological (adnexitis) and urological (renal colic), as well as infection intoxication, diabetes, etc.

Diseases of the lungs and pleura (lower segment pneumonia and basal pleurisy), as a result of irritation of the rib nerves and inflammation of the diaphragmatic surface of the peritoneum, abdominal pain and abdominal reflex increase. Palpation of the anterior abdominal wall reveals the tension of its muscles, diffuse pain in the epigastric region. The body temperature rises, leukocytosis occurs. The complex of symptoms depends on the kind of peritonitis. However, the key signs of diseases of the lungs and pleura is a disorder of the function of external respiration, which is appeared by an increase in its frequency, shortness of breath, and others. During auscultation of the lungs, wheezes are heard, with percussion - the suppression of percussion sound. In the diagnosis, data from an x-ray examination of the chest cavity organs (darkening in the lungs, restriction of the respiratory excursion of the diaphragm, the presence of fluid in the pleural cavity — with pleurisy, etc).

On background cardiovascular diseases (abdominal form of acute myocardial infarction; rheumatismal abdominal syndrome) sometimes there is pain in the abdominal cavity, tension of the muscles of anterior abdominal wall, paresis of the intestine. Unlike peritonitis, pain in the abdominal cavity is unstable. Patients, for the most part, complain of pain in the heart region. During examination, tachycardia is present. By an electrocardiography: changes are founding which are the typical for heart disease. Rarely, there are essential changes in the leukocyte's formula and in general clinical urine test and do not observe the disorders of metabolic processes.

On background gynaecological diseases, in particular adnexitis, the origin and following of the first hours are the same as an acute peritonitis. The disease begins with acute pain in the abdomen, muscle tension in the lower abdominal part, and intestinal dysfunction. However, the pain, for the most part, is localized at the lower levels of the abdomen, irradiates in the region of the perineum and the genitals. The tension of the muscles of the anterior abdominal wall is less pronounced, it is detected only in the lower abdomen. Despite the fact that at the adnexitis is a strong pain syndrome, the condition of the patient remains passable. Vaginal examination detects painful and thickened uterine tubes, enlarged painful ovaries and vaginal arch, pain begin by displacement of the cervix. According to the ultrasound examination, the uterus is enlarged and the fallopian tubes are thickened. Confirms the diagnosis of the gynaecological disease the presence of mucous-purulent, purulent or bloody secretions from the vagina.

The similar symptoms of renal colic and acute peritonitis are hard pain in the abdominal cavity, nausea, vomiting, paresis of the intestine, protective tension of muscles of the anterior abdominal wall during palpation, fever, leukocytosis.

However, the pain in the renal zone occurs suddenly, with the background of satisfactory condition of health, usually in the lumbar region, and subsequently extends to the same half of the abdomen, or even the all abdomen. Most patients with renal colic the pain is irradiated along the ureter, in the bladder, ilium and inguinal areas. Unlike the case of acute peritonitis, the patient is restless, constantly changing the body position. Typical for renal colic are dysuric conditions, a positive symptom of Pasternatsky, the presence of erythrocytes in the urine. Information by cystoscopy, reviewal urographic and ultrasound studies confirm the diagnosis of kidney and urinary tract diseases.

Toxicoinfection is characterized by dyspeptic signs (vomiting, nausea, diarrhoea), which come before the appearance of pain in the abdominal cavity, and the pain has spastic character. The stomach is mild, a little painful by palpation. Bloating, symptoms of peritoneal irritation are absent. After the detoxification therapy, the condition of the patient is quickly refining.

Treatment of acute peritonitis. Modern pathogenetic-proved treatment of acute general peritonitis is based on three basic principles:

1. An urgent surgery with adequate and complete sanitation of the abdominal cavity.
2. Management of full antibacterial therapy course taking into estimation the sensitivity of emitted microorganisms to antibiotics.
3. The course of complex intensive therapy aimed at correction of disorders of the functional state of organs and systems of the body and metabolic disorders.

Preoperative preparation is carried out in the short-term mode, its features are determined by the common condition of the patient and the stage of peritonitis.

Amount of preoperative preparation in case of acute peritonitis.

Stage of peritonitis: reactive

Place and scope of preparation: on the operating table

Infusion Therapy: Depending on the patient's condition. Ringer's solution - 800ml, glucose solution 20% or 10% 400ml with potassium chloride solution (1,5-3 g for preserved diuresis), polyglycan, albumin, 5% glucose solution 1200ml, vitamins of group B, ascorbic acid, antibiotics , eufillin.

Stage of peritonitis: toxic

Place and scope of preparation: Resuscitation and intensive care department. Duration 2-3 hours. Catheterization of the central vein, the introduction of the probe into the stomach, catheterization of the bladder. Checking the central venous pressure, acid-alkaline state, potassium, sodium, and urea in the blood.

Infusion therapy: Polyglyucine or gelatinol, 5% glucose solution 1200ml, Ringer solution, 10% glucose solution 400ml with potassium chloride solution, albumin, cardiac glycosides, antibiotics, vitamins, eufilin, etc.

Important positions in preoperative preparing are the carrying of detoxification therapy, correction of homeostasis disorders (hypovolemia, disorders of water-electrolyte balance, acid-alkaline state, etc.) and actions to improve the functional

state of the cardiovascular and respiratory systems, as well as antibacterial and symptomatic therapy. In 2 hours prior to surgery, you need to infuse 1/3 of the calculated volume, but not more than 10-12% of the body weight. Correction of dehydration is carried out by solutions of electrolytes and solutions of glucose, taking the Shelestyuk test at the same time, which is used to determine the degree of dehydration and the need for fluid infusion) and under the control of CVP. The essence of the test is as follows:

- the skin of the forearm surface is treated with antiseptic solution;
- 0.25ml of 0.9% solution of sodium chloride is injected intradermally;
- marked the time of complete resorption of \ "lemon peel \";
- by the time of resorption on nomogram determined approximate degree of dehydration.
    - 1 stage of dehydration (the papule is absorbed within 40-30 minutes) - 50-80ml / kg (on average 70 kg of body weight of the patient 3500-5600ml);
    - 2 stage of dehydration (the papule is resolved within 30-15 minutes) - 80-120ml / kg (on average 70 kg of body weight of the patient 5600-8400ml);
    - 3 stage of dehydration (papule is absorbed in 15-5 minutes) - 120-160ml / kg (on average 70 kg of body weight patient 8400-11200ml).

Preoperative preparation of patients with acute peritonitis in toxic and terminal stages is carried out in intensive care units. The intensity of infusion preoperative preparation depends on the stage of acute peritonitis.

The way of choosing anaesthetics during surgery for acute general peritonitis is combined endotracheal anaesthesia.

Treatment of acute general peritonitis is carried out in several stages:

1. Early surgery, which involves eliminating the cause of the peritonitis, effective sanitation of the abdominal cavity. Surgical access - median-middle laparotomy. If necessary, depending on the location of the peritonitis, this access can be extend up and down. This gives a possibility to carry a complete revision of the organs of the abdominal cavity and its sanitation.

After the laparotomy, by exhaustion, remove the exudate, carry the revision of its organs to detect the cause of peritonitis. Begin the revision from the stomach, then examine the small and large intestine, gall bladder, pelvic organs, pancreas. Eliminate the cause of peritonitis. Carefully attention is taken to a revision of the intestine. In it is necrosis, after the removal of a non-viable area of the small intestine, surgical intervention, in most cases, is completed by forming the thin-intestinal anastomosis. When necrosis of the large intestine, the overlay of the primary anastomosis is very dangerous, so they form a colostomy.

According to the critical condition of the patient and the presence of questionable pathological changes in the intestine, the solution to the issue of its viability and the need for resection of the affected segment is postponed for 6-12 hours, it is carried out during programmable laparotomy with simultaneous

rehabilitation of the abdominal cavity. After eliminating the cause of peritonitis, the final rehabilitation of the abdominal cavity is performed on clean rinsing waters. Effective is the use of tissue sorbents, which are injected into the affected organ, abdominal side flaps, small pelvic cavity.

2. Postoperative rehealing of the abdominal cavity is carried by its drainage using polychlorinated tubes with additional holes. The tubes are injected into the lateral parts of the abdomen, the pelvic cavity, into the subhepatic or under the diaphragmatic space (depending on the location of the peritonitis and pus cluster). Through those drainages, not only remove the exudate, but also scour the abdominal cavity.

Previously, for the sanitation of the abdominal cavity, a routine used method of peritoneal dialysis - a constant cleaning of the abdominal cavity using the dialysis solution. However, in recent years the method is rarely used. An indication for its use is a general peritonitis with accumulation of purulent exudates in the abdominal cavity. Two types of dialysis are used: flow and fractional. During the process of dialysis in the abdominal cavity, 4 tubes with a lot of holes (two - in lateral parts, two - in the small pelvis cavity) are introduced. The abdominal cavity is cleaning for several days, until the solution released through the tubes, will not become pure, without purulent impurities. The flaw of flow dialysis is that after 3-4 days around the tubes fibrin is lying, and they fail to function properly, the peritoneal exudate begins to flow only through the channels formed around the tubes.

In fractional peritoneal dialysis in the abdominal cavity 2 liters of dialysis solution are injected with lysis agents that dissolve fibrin. The solution is left in the abdominal cavity for 2 hours, and then removed. Together with the liquid, fibrin with impurities of manure is removed. In this way a thorough rehabilitation of the abdominal cavity is carried out. The procedure is carried out every 8 hours. However, the best results are obtained by combining these two methods: during 3-4 days, conduct flow dialysis, and then - fractional. Effective is the alternate application of methods.

The study for rehealing of abdominal cavity and rehabilitation methods and the evaluation of treatment results led to the introduction to the medical practice the programmed laparotomy - reopening and rehabilitation of the abdominal cavity. For this during the first laparotomy, after the elimination of the causes of peritonitis, rehabilitation of the abdominal cavity, the need for its reopening is determined. After 24-48 hours, re-opening the abdominal cavity and carry out its sanitation, while controlling the course of inflammation. After the eliminating the purulent process, when re-scouring the abdominal cavity is not required, the surgical wound is seamed layer-by-layer tightly. The programmed laparotomy can also be used as a diagnostic method for monitoring the viability of organs and tissues, the capacity of intestinal sutures, etc. During the last decade, in the complex of treatment of acute general purulent peritonitis, the laparoscopic surgery is widely used, the advantage of which are small injuries.

3. Detoxification of the digestive system and the rehabilitation of the intestine by evacuating the contents of the stomach through the probe introduced into it, intestinal intubation, and the physiotherapeutic and physiotherapeutic stimulation of its functions. The intubation of the intestines is carried by antegrade methods (nasogastric, gastric, jejunal) and retrograde (through a stoma, stump of the appendix, blind or rectum). For intubation of the intestines, thin, usually twin-path probes with a lot of lateral holes, through which evacuate its contents, are used. The probes are put into the gut lumen after performing the main stage of surgical intervention before suturing the laparotomy wound. With nasogastric intubation, the probe is carried through the nose, oesophagus and stomach, followed by intubation of the small intestine. The probe is used not only for the passive removal of the contents of the stomach and intestines, but also for the management of enteral nutrition and medical stimulation of the intestine.

Intubation of the stomach and small intestine provides not only a detoxication effect caused the removal of toxic content, but also support the early recovery of its peristalsis. An important disadvantage of antegrade intubation of the intestine is the development of respiratory failure (tracheitis, bronchitis, inflammation of the lungs), which has a particularly negative effect on the elderly patients. For the aim of detoxification is successfully used through probe introduction of sorbents (enterosgel, enterodose, enterosorb, bilosorb, carboline, etc).

To quickly restoring the function of the intestine, may be carrying out its early stimulation with the help of cleansing enema, drugs (prozerin, tserukal, etc.), the implementation of a paranephral blockade. For resistance paresis using long-term epidural anaesthesia in combination with the cleansing enema.

4. Antibacterial therapy should begin in the period of preoperative preparing, to continue during surgery and in the postoperative period, is one of the most important components of the complex of acute peritonitis treatment. The presence of a polymicrobial infection and its toxins in the abdominal cavity, fast absorption of exudate, trigger the reaction of the systemic inflammatory response and cause the beginning of multiple organ failure syndrome. Therefore, the main task of antibiotic therapy - to blockade the inflammatory response at the level of microbial mediators of damage.

Antibiotics and other antimicrobial agents should be prescribed taking into account the sensitivity of each microorganism to those. However, the results of determining the sensitivity of microorganisms to antibiotics might be only about 2-3 days after the start of the disease. Therefore, in the first days after surgery, antibiotics of a wide range of effects (tienam, netromicin, meropenem, etc.) are prescribed, that is, those active about gram-negative and gram-positive, aerobic and anaerobic bacteria. Antianaerobic action is also made by metronidazole and metrogil.

Antibacterial therapy guess the using of adequate doses of drugs and their timely replacement, if necessary. The optimal duration of use of one antibiotic is

7-8 days, which prevents the emergence of antibiotic-resistant strains of microorganisms.

5. General detoxification of the body is carried out not only by the sanitation of the intestine, aspiration of its contents, peritoneal dialysis, enterosorption, and the implementation of hemosorption, lymphosorption, forced diuresis, infusion therapy. The efficiency of detoxification therapy is controlled about the dynamics of the leukocyte formula, the presence of toxic changes of neutrophil granulocytes, the level of creatinine, urea, the activity of aspartate aminotransferase in the blood, and also on the indicator of leukocytic index of intoxication (LII), which is calculated by the formula A.Ya. Calf-Caliph.

6. The adequate correction of disorders of proteins, carbohydrates, fats, acid-alkaline state, water-electrolyte balance and microcirculation is beginning before the intervention, are continue during its implementation, and especially intensively - in the postoperative period. The volume of infusion therapy is calculated taking into account the average physiological daily need for the body and pathological losses caused an increase of body temperature, hyperventilation, and the like. Up to 25% of this volume is compensated by proteins. Half is by crystalline solutions (isotonic sodium chloride solution, Ringer-Locke solution, Lactasol), 10% glucose solution, 10% - other meds. Parenteral nutrition is performed using amino acids, fat emulsions, glucose solutions.

7. For prophylactic objectives, all the patients are prescribed the direct anticoagulants (heparin, Fraxiparin, Clexan, Fragmin), and also the elastic compression of the lower limbs to early activation, conducting the medical physical Culture, etc.

8. An important factor in the favourable course of the postoperative period is the stimulation of the body's protective resources and immunocorrection, because in the patients with acute general peritonitis, as a rule, diagnose an immunodeficiency. Active immunization is carried by using antistaphylococcal toxoid, passive anti-staphylococcal gamma-globulin, antistaphylococcal plasma; donors-reconvalescents plasma, whom in the past have suffered acute peritonitis; T-activin, laferon, and the like.

9. As symptomatic therapy, aimed at improving and normalizing the activity of the cardiovascular system, microcirculation, hepatic and renal blood flow, central venous pressure, stabilizing blood pressure, increasing diuresis (up to 50-60ml of urine per hour), use cardiac glycosides, nitrates, antiplatelets and the like.

For prophylactics and treatment of disorders of the functions of the respiratory organs begin the dynamic activity, oxygen therapy, respiratory gymnastics,

inhalations, massage of the chest, etc. If necessary, patients who are in a difficult condition, the forced ventilation of the lungs in hyperventilation mode during 7 days.

Patients who were operated about acute widespread peritonitis, there is a risk of some complications associated with the presence of an infectious component in the abdominal cavity. The cause for that may be the inadequate treatment of the abdominal cavity during the intervention, and especially during the postoperative period, the failure of seams on the abdominal organs, infection of the surgical wound, sepsis, infiltration and abdominal abscess formation and internal organs, postoperative bowel obstruction.

In the postoperative period, the treatment of patients with acute general peritonitis incurs in a reanimation and intensive care department. Only complex treatment, aimed for normalizing the functions of all organs and systems of the body, can provide recovery of the patient.

The prognosis for acute widespread peritonitis depends on the cause of peritonitis, the virulence of the flora, the timeliness of diagnosis and the surgery aimed at removing the source of peritonitis, and subsequent intensive care.

Postoperative complications. After surgical interventions on the abdominal organs, early signs of postoperative peritonitis are a deterioration in the patient's condition, which can not be explained by evident reasons (recurrence of pain in the abdominal cavity, tachycardia, increased heart rate, pain about the palpation of the abdomen). The common symptoms of this complication are nausea and vomiting. There are pronounced changes in general-clinical and biochemical blood tests (leukocytosis, hypoalbuminemia, etc.).

In order to clarify the diagnosis, additional instrumental methods of examination are widely used: ultrasound, X-ray and CT. If the diagnosis of postoperative peritonitis is confirmed, performs relaparotomy, sanation of the abdominal cavity, continue the intensive therapy. The earlier performed relaparotomy - the more chances of success.

The most common postoperative complications are:

1. Insolvency of intestinal sutures - is accompanied by the return of pain syndrome, deterioration of the patient's condition, progressing tachycardia, dryness of the tongue, bloating, progression of symptoms of peritoneal irritation. In the presence of drainage in the abdominal cavity, the diagnosis of insolvency of the seams is not difficult to confirm, revealing the exit of contents of the intestine through it. If the patient's condition does not deteriorate, and signs of peritonitis are absent, this indicates the delimitation of the inflammatory process of the peritoneum, therefore the urgent relaparotomy is not indicated. The results of ultrasound and X-ray examination confirm the diagnosis of seams failure. In that case (the progression of peritonitis) immediate relaparotomy, sanation of the abdominal cavity with the formation of the intestinal stoma is indicated.

2. Abscessing of the abdominal cavity. About its appearance, the local pain intensifies, chills arise, and body temperature becomes hectic. The assumption of abscess is confirmed by the results of ultrasound and X-ray examination. The treatment is operative.

The intraorganic abscess is more often formed in the liver and pancreas in reactive widespread peritonitis with traumatic origin. For diagnostics of these abscesses, most informative methods are ultrasound and X-ray examination. During an ultrasound examination, rounded forms of one or more echo-negative inclusions with edema of nearby tissues and fluid inside are diagnosed. During the X-ray examination, an enlargement of the organ that contains the abscess is observed, in some cases, the high standing of the dome of the diaphragm is present.

3. Early postoperative bowel obstruction may be mechanical or paralytic genesis, delayed gas, intestinal passage, abdominal distension. The X-ray examination reveals the characteristic symptoms: horizontal levels of fluid in the intestine, Kloyber's bowl, positive symptoms of Kivul, Sklyarova and others. When the paralytic intestinal obstruction is diagnosed, the conservative treatment performed, and in mechanical obstruction - surgical intervention.

4. Suppuration of the wound occurs quite often, as during the surgical intervention wound is often infected. Even carrying antibiotic therapy does not always prevent the development of such complication. If signs of suppurating appears, the wound is exposed, rehab, and set the drainage. Sometimes, when the postoperative wound becomes inflamed, there are changes, which requires urgent intervention.

5. Thromboembolic complications. Venous thrombosis and pulmonary embolism are dangerous complications of the postoperative period. Non-specific and specific prophylaxis is carried out to prevent thromboembolic complications. Unspecific precautions include early conducting of breathing exercises, massage, active mode, early getting out of bed. By specific - the appointment of direct anticoagulants (heparin, Fragmin, Fraxiparin, Kleksan, etc.). Particular attention is needed for patients with a high risk of thromboembolic complications.

The main principle of the prevention of acute general peritonitis is timely diagnosis and treatment of acute surgical diseases and injuries of the abdominal cavity, which cause the occurs and progression of acute inflammation of the peritoneum.

Local forms of peritonitis. Local (delimited) peritonitis is an inflammation of the parietal and visceral peritoneum of one anatomical region of the abdominal cavity, which is accompanied by both local and general manifestations and disorders of functions of different organs and systems. Local peritonitis is often a complication

or a primal outcome or stage of development of different acute surgical diseases and injuries of the abdominal cavity. The origin of local peritonitis occurs at the condition of low virulence of the microflora on the background of normal activity and well resistance of the organism. Characteristic of this form of peritonitis is that the inflammation zone is detached from other parts of the abdominal cavity by anatomical structures: large and small omentum, mesentery and walls of the intestine, and others.

Differ the primary local forms of peritonitis (appendicular and paranephral infiltrate) - when the delimitation is in parallel with the run of the inflammatory process, and secondarily formed with the infection of the abdominal cavity (abscesses of the abdominal cavity). In this case, microorganisms that have come into the sac or pit of the peritoneum, are limited by forming around the inflammation zone by the connective tissue capsule. By localization, the abscesses are divided into subdiaphragmatic, pelvic and between-loops.

Subdiaphragmatic abscesses are an accumulation of purulent substance in the subdiaphragmatic space, which is delimited from other parts of the abdominal cavity. Subdiafragmal space is the part of the abdominal cavity, which is limited: on the top by the diaphragm, on the bottom by a transverse colon and its undulation, on the sides - lateral abdominal wall. It is delimited by the liver and its ligaments on the supra- and subhepatic areas.

The causes of localized peritonitis are diseases of the stomach and duodenum, inflammatory diseases of the liver and bile ducts, acute appendicitis, operations performed on these diseases and purulent diseases of the lungs and pleura.

By localization, the following types of subdiaphragmatic abscess are defined:
- right-side (diaphragm-hepatic);
- left-sided (diaphragm-splenic);
- medial (diaphragm-gastric).

By the nature of the disease:
- gas (in the abscess content contains not only pus but also the gas);
- non-gas

Often, sub-diaphragmatic abscesses formed under the right diaphragm dome. There is a congestion of pus between the diaphragm and the upper surface of the right lobe of the liver. In this dome, diaphragm pushed up, and the liver going down. In the left-sided localization of abscess, it is located to the left of the suspending ligament of the liver between the surface of its left lobe and the diaphragm. In this case, abscesses lift up the left diaphragm dome, and the liver is pushed down and can move to the front surface of the stomach.

In more than 25% of cases in the subdiaphragmatic abscess, along with the pus its contains gas, as by perforation into the abdominal cavity enters the contents of hollow organs and air. In other cases, gas is produced by microbial flora. The clinical running of subdiaphragmatic abscesses is often accompanied by the formation on the simile side the pleural exudate.

The clinical course may have several variants: 1) a sudden acute start of manifestations of suppuration; 2) distorted character; 3) a gradual increase in the manifestation of inflammation following laparotomy and surgical intervention.

Patients complain of a feeling of compression pain in the upper abdomen, which increases with a deep breath, pain in the subribs and lumbar area appears. The body temperature is hectic.

The forming of the sub-diaphragmatic abscess is accompanied by the origin of the following symptoms: pain in the right or left subrib, pain in the chest area, nausea, the appearance of hiccups, chill, increased sweating, edema of the abscess area tissues, increased heart rate, shortness of breath. Positive symptoms are: Duchenne (paradoxical breathing - retraction of the epigastric region during inhalation and exhalation), Myus-Georgievsky (increased pain in the right subrib when the finger is pressed between the sternum and clavicular processes of the sternocleidomastoid muscle), Senator (immobilized spine during walking caused the rigidity of the muscles), Litten's (retraction of intercostal spaces in the places of attachment of the diaphragm with a deep breath).

In the laboratory tests, leukocytosis with a shift of the formula to the left and endotoxicosis is detected: hypodysproteinemia, progressive anaemia of hypochromic type, toxic grainy of neutrophils, eosinophilia and an increase in ESR, increased urea concentration, creatinine, and others.

During the X-ray view, there is a displacement of the dome of the diaphragm and its real estate at the breath, the presence under the diaphragm of gas with a horizontal fluid level, displacement of organs of the gastrointestinal tract and urinary system. With ultrasound - the display of hollow formation with the presence of fluid.

Treatment of the sub-diaphragmatic abscess. The established diagnosis of the sub-diaphragmatic abscess is an absolute indication for the surgery - the disclosure and drainage of the abscess. It is important to avoid infection of other parts of the abdominal cavity or pleura, which will lead to the generalization of the inflammatory process. Interventions about sub-diaphragmatic abscesses are divided into transthoracic and trans-abdominal, which in turn are divided into circular and non-transverse.

When performing a transthoracic pleural access to prevent infection of the pleura, joint up the subdiaphragmatic lay of the pleura with its rib part and puncture and drain the abscess through the diaphragm.

Transthoracic non-circular access to the abscess is made by cutting below the transition site of the pleura or by mobilizing the edge-diaphragm angle to the apex.

Taking into account that the lower bound of the rib-diaphragm angle is at the level of the XII rib, AV Melnyk proposed to perform a periosteal resection of the XII rib. Create a transverse incision at right angles to the spine through the bed of the removed rib on the level of the lumbar vertebra. Then blunt move apart tissues to the abscess cavity, wash it and drain by tubing drainage. Hilerault suggested resection of the X rib, carefully separating the upper pleura and thus abscess. Access Clairmont (at the anterior abscesses localization) is carried by parallel to the edge immediately below it, crossing the abdominal muscles and transverse fascia to the

parietal peritoneum. It moved with finger and separated from the lower surface of the diaphragm until it reaches the cavity of the abscess, with the helping of a large needle, it removes its contents, after by irrigation the cavity with solutions of antiseptics and antibiotics. Additionally, the antibiotics of a wide spectrum of is appointed.

The prevention of sub-diaphragmatic abscesses is by the following: timely treatment of inflammatory diseases of the stomach, duodenum, liver and other organs of the abdominal cavity; instant call to the doctor in the case of pain feeling in the abdomen in order to timely detection of inflammation of the internal organs, strict implementation of medical recommendations after intervention on organs of the abdominal cavity in order to prevent the complications of inflammatory type.

Pelvic abscesses appear as a result of infection of the lower level of the abdominal cavity where the exudate is accumulating. The ways for the dissemination of ulcers are caused by numerous cellular spaces and fascias. Most often abscess is localized in women between the rectum and uterus ("Douglas space"), and men - between the bladder and rectum. Also, abscesses can be forming in the pararectal space and ovarian space.

The causes for their forming are inflammatory diseases of the abdominal organs, primarily appendicitis, gynaecological pathology, perforation of the large intestine, traumas of the pelvic organs, and after performing operative interventions by these causes.

Clinic. for a constant, rather intense pain in the lower part of the abdomen, tenesmus (the urges to defecation). When the rectal wall is involved, there is pain during the defecation, the hiatus of the anus, the absence of excrement and gases outlet, frequent painful urination. The body temperature is hectic. By palpation of the abdomen, as a rule, there are no symptoms of irritation of the peritoneum. By the rectal examination, there is impended anterior wall of the rectum, increasing its pain by palpation, and the presence of the soft consistency compaction.

The vaginal examination shows hanging up the back wall of the vagina, abrupt pain in the displacement of the cervix. By bimanual examination, it is sometimes possible to detect an abscess and determine its size.

By the laboratory tests, the same changes are observed as about subdiaphragmatic abscesses.

The treating of pelvic abscesses is operative. Accesses for surgical intervention are divided into extra-large (for drainage of the anterior pelvic zone), perineal (for drainage the anterior and posterior zones) and through the rectum or vagina (for drainage the back pelvic zone).

By conducting a suprapubic incision, abscesses of the space of the prostate gland are opened, while it is important to peel the peritoneum. The perianal rectal recess is drained by perineal access. To do this, one deep incision is made in the region of each buttock-rectal recess, the tissues of the pelvic diaphragm are separated in a blunt way, the abscess is opened, it is pared and drained. Through the rectum - after catheterization the bladder and a cleansing enema, the external sphincter of the rectum is expanded, a rectal mirror is inserted into the rectum. With the big-size

needle punctures the anterior wall of the rectum at the point of bulging. After evacuating pus with a lancet in the transverse direction, open the anterior wall of the rectum, wide the aperture, irrigate and drain the abscess.

Through the vagina - the cervix is gripped by the clamps and pushed to the top and to the front. Through the back arches with a thick needle, an abscess is punctured. When receiving pus from the needle, a scalpel incision is performed and the abscess is drained. With help of rectum and vagina are possible to reveal only 30% abscesses of a small pelvis.

Intestinal abscesses are abscesses that are placed between the small intestine loops, between the small and large intestines, between the intestines and the cape or abdominal wall. Allocate the interstitial abscesses primary and residual.

Primitives arise directly near the primary source of infection (near the appendix, gall bladder, diverticulum of the intestine, in the place of the closed perforation, etc.), residual (or residual abscesses) are more often formed after the common forms of peritonitis with insufficiently effective rehabilitation of the abdominal cavity.

Allocate: single and multiple interstitial abscesses. According to the clinical picture they are: uncomplicated and complicated. Complications: 1) breakthrough abscess in the free peritoneal cavity and the development of pervious peritonitis; 2) breakthrough of the abscess into the lumen of the hollow organ with the formation of fistula; 3) bowel obstruction; 4) pylephlebitis.

Clinic. Patients complain of stupid pain in the abdomen of moderate intensity, periodic bloating, elevated body temperature to 38-39 ° C, chills.

With objective examination, the symptoms of peritoneal irritation are not detected. Rarely, you can palpate moderately painful and dense infiltration. Laboratory manifestations - the same as with abscesses of another pathology.

At X-ray examination (including computed tomography), it is possible to detect a darkening area with a level of fluid. More informative is the ultrasound investigation, which allows you to determine the localization of the abscess, its size.

Treatment is operative. Surgical access depends on the localization of the abscess. With para-adenocarcinoma abscess, use is given by extra-occipital access. The incision of the layers of the abdominal wall to the parietal peritoneum is the same as with appendectomy. The ovaries are dull medially. Through infiltrated retroperitoneal cellulose, open the abscess and drain its cavity. In other localizations, the incision is carried out over the cavity of the abscess. Cut the layers of the abdominal wall into the peritoneum, it is separated off and on the ground fluctuations open and drain abscess.

If the abscess is placed on the back wall, after laparotomy the tissue is dissected over the infiltrate, revealing the abscess. Around the abscess place tampons, into the cavity of the abscess set drainage tubes.

Prevention of abscesses forming: timely effective removing of the source of infection and effective rehealing of the abdominal cavity.

Self-control questions

1. Anatomical and functional information about parietal and visceral peritoneum.
2. Etiology and pathogenesis of acute peritonitis.
3. Classification of acute peritonitis.
4. Methods of examination of patients with acute common and local peritonitis.
5. Clinical picture of common acute peritonitis.
6. Clinical picture of local acute peritonitis.
7. Differential diagnosis of acute peritonitis.
8. Diagnostic algorithm for a patient with acute peritonitis.
9. Therapeutic tactics in a patient with acute peritonitis.
10. Therapeutic tactics of the patient for various forms of local peritonitis.
11. Complications of acute peritonitis.
12. Features of preoperative preparation in acute peritonitis.
13. Methods of surgical interventions with acute local and widespread peritonitis.
14. Programmable laparocentesis in the treatment of acute peritonitis.
15. Postoperative complications in acute peritonitis, their diagnosis, prevention and treatment.

Tests for self-control

1. Local peritonitis is the spread of the inflammatory process to:
   a. three anatomical sites;
   b. two anatomical sites;
   c. one anatomical site;
   d. four anatomical sites;
   e. more than six anatomical sites;

2. Indications for surgical intervention in diffuse peritonitis are:
   a. the operation is shown to all patients with a diagnosis;
   b. exceptionally reactive stage of peritonitis;
   c. exclusively toxic stage of peritonitis;
   d. no effect of conservative treatment within 24 hours;
   e. lack of hypertension.

3. Clinically distinguish the following stages of peritonitis:
   a. acute, chronic;
   b. latent, intoxication, reconvalescence;
   c. acute, subacute, chronic;
   d. prodromal, acute, terminal;
   e. reactive, toxic, terminal.

4. What is the treatment for acute peritonitis?
   a. delayed operation;
   b. urgent operation;
   c. conservative;
   d. dynamic observation;

e. Expectant tactics.

5. Poured peritonitis is the spread of the inflammatory process:
   a. per anatomical site;
   b. up to four anatomical sites;
   c. up to three anatomical sites;
   d. up to eight anatomical sites;
   e. up to six anatomical sites.

6. A patient 53 years old, 4 hours ago, suddenly arose a lot of pain in the epigastrium, giving right shoulder. Objectively: the skin is pale, covered with sticky sweat, lying motionless on the back. Breathing superficial to 20 in 1 minute. Pulse 96 in 1 minute. Tongue dry The abdomen is tense and dragged. On the review X-ray of the abdominal cavity - free air under the diaphragm. Make a preliminary diagnosis.
   a. acute pancreatitis;
   b. acute intestinal obstruction;
   c. acute appendicitis;
   d. perforated ulcer of the duodenum;
   e. appendicular infiltration.

7. The patient complains of pain throughout the abdominal cavity, which arose a half hour ago. Ill pale, facial expression suffering, lying motionless on the back, pulse - 90 per minute, rhythmic. Blood Pressure - 90/60mm Hg. Art. The tongue is dry, clean. The abdomen in the act of breathing does not take part, it is sharply tense and painful in all departments. The symptom of Shchotkin-Blumberg is positive. What is the treatment of a patient?
   a. conservative therapy in day care;
   b. conservative therapy in outpatient settings;
   c. conservative therapy at home;
   d. urgent hospitalization in a surgical hospital;
   e. hospitalization in hospital after examination.

8. The patient is 50 years old, got into a hospital with complaints of severe abdominal pain, which arose suddenly 6 hours ago. At inspection: the condition is heavy, the skin is pale, body temperature 37,6 ° C. Pulse 110 per 1 minute, AT - 100/60mm Hg. Art. Breathing is superficial. Abdominal palpation is intense, marked positive symptoms of peritoneal irritation, with percussion - bluntness in the abdominal areas of the abdominal cavity. In blood tests: leukocytes - 14,7x109 / l. Established diagnosis: poured peritonitis. Which of the revealed symptoms is most informative for this diagnosis?
   a. temperature rise;
   b. symptom of irritation of the peritoneum;
   c. tachycardia;
   d. leukocytosis;

e. superficial breathing

9. During an operation that was performed on the patient for 36 years, with regard to acute appendicitis, a large amount of clear serous fluid appeared on the abdominal cavity. The crustal appendage is unchanged, the Meckel diverticula has not been found during rectal examination. At the same time, on the ripples of the large and small intestines, white rashes and lymph nodes were found. He suffers from pulmonary tuberculosis. Suspected specific peritonitis. What do you need to do during an operation to clarify the diagnosis?
   a. crops liquid;
   b. macroscopic evaluation of the exudate;
   c. Mantou's reaction;
   d. peritoneal biopsy with tubercles and cytological examination;
   e. puncture of the pleural cavity with a microscopic punctate.

10. The patient 42 years old complains of pain in the right iliac region, which occurred 2 days ago, which began in the epigastrium, and after 3 hours moved to the right ilium, it was a single vomiting. At inspection: body temperature is 37.9 ° C, pulse - 90 for 1 minute, tongue is moist. In the right iliac region there is pain, muscle tension, positive symptoms of Shchotkina-Blumberg, Sitkovsky, Roving. In the blood analysis, white blood cells - 10.7x109 / l, the leukocyte formula shift to the left. Formulate a preliminary diagnosis.
   a. acute pancreatitis;
   b. acute cholecystitis;
   c. acute appendicitis, local peritonitis;
   d. acute intestinal obstruction;
   e. perforated ulcer of the stomach.

11. In a 32-year-old patient, who had been operated 9 days ago with a pelvic disruptive appendicitis, despite the treatment being performed, complications developed in the form of an abscess of the Douglas's space. At palpation of the abdomen a moderate pain over the womb is observed, the abdomen is mild. At the finger study of the rectum, its front wall hangs, and then painful infiltrate with softening is determined. What should be the further tactics of a surgeon?
   a. relaparotomy for drainage of abscess;
   b. warm microclysters and candles with antibiotics;
   c. presacral blockade;
   d. puncture of the abscess through the rectum;
   e. electrophoresis with antibiotics on the bottom of the abdomen.

RIGHT RESPONSE TO TESTS

Acute appendicitis

1 – D, 2 – B, 3 – B, 4 – C, 5 – A, 6 – E, 7 – A, 8 – C, 9 – D, 10 – A, 11 – A.

Acute cholecystitis

1 – D, 2 – E, 3 – B, 4 – A, 5 – C, 6 – B, 7 – D, 8 – E, 9 – C, 10 – C, 11 – A.

Acute pancreatitis

1 – B, 2 – C, 3 – B, 4 – A, 5 – A, 6 – B; 7 – E; 8 – B; 9 – D; 10 – E.

The stomach ulcer and duodenal ulcer are complicated by hemorrhage

1 – C, 2 – E, 3 – D, 4 – E, 5 – B, 6 – E, 7 – C, 8 – A, 9 – E, 10 – A, 11 – E, 12 – A, 13 – C, 14 – B, 15 – B.

Perforated ulcer of the stomach and duodenum

1 – B, 2 – B, 3 – D, 4 – A, 5 – E, 6 – C, 7 – C, 8 – D, 9 – B, 10 – E, 11 – D.

Acute intestinal obstruction

1 – A, 2 – D, 3 – C, 4 – A, 5 – E, 6 – A, 7 – C, 8 – C, 9 – B, 10 – B, 11 – E.

Acute peritonitis

1 – C, 2 – A, 3 – E, 4 – B, 5 – E, 6 – D, 7 – D, 8 – B, 9 – D, 10 – C, 11 – D.

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