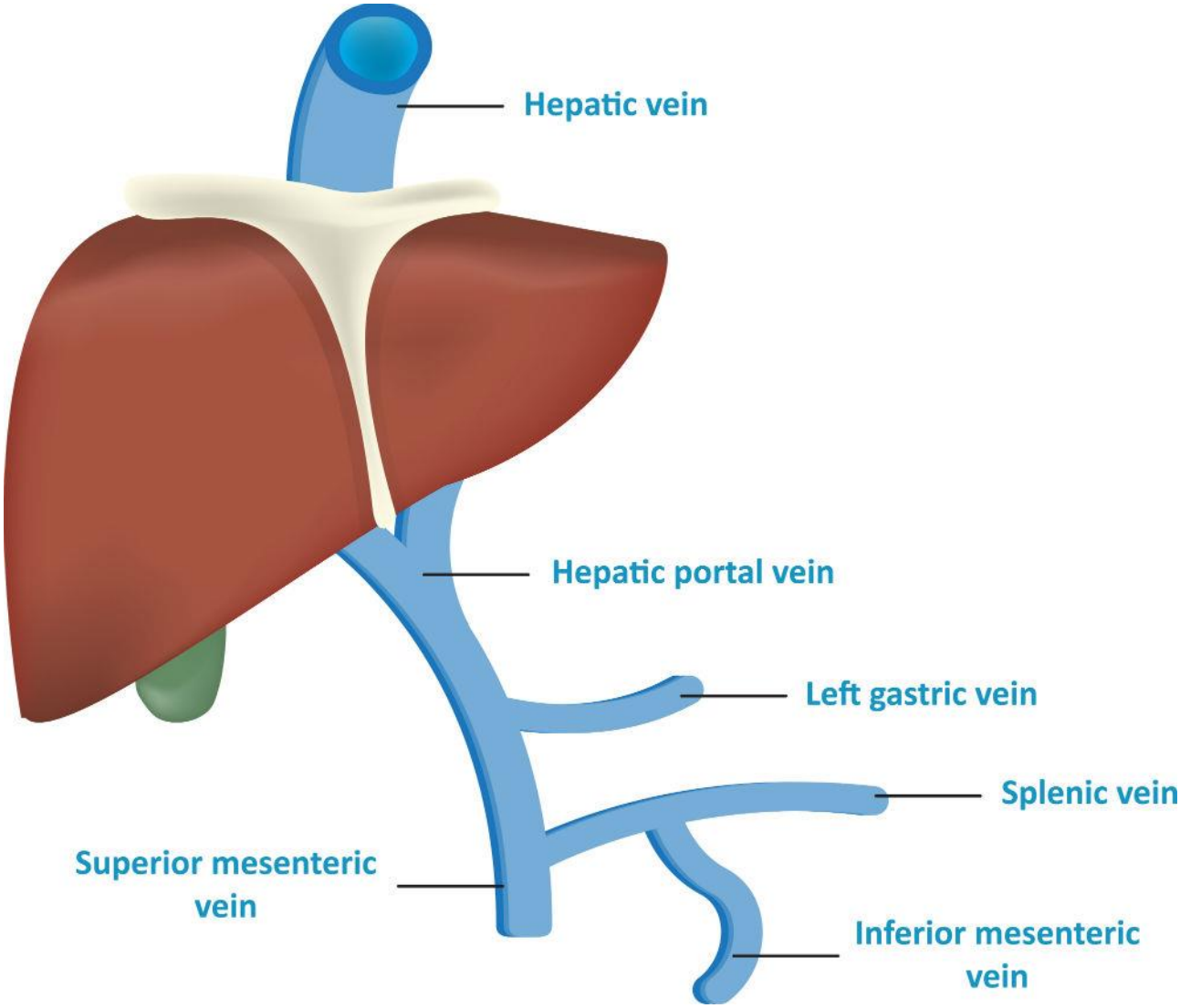
ROYAL METROPOLITAN UNIVERSITY

Department of Clinical Disciplines

PORTAL HYPERTENSION

Educational and methodical manual



BISHKEK 2022

UDC616.149.66(575.2)

P85

Printed on the recommendation of the RMU Academic Council, Protocol № 1 dated 09.09.2022.

Compiled by:

Toktosunov U.T.,

Nazir Muhammad Imran,

Jaxymbaev N. B.

Saliev A.Zn.

The educational and methodical manual is devoted to portal hypertension. Objectives and tasks are spelled out in a form understandable to students. The presentation of the material is intended to provide students with a unified picture, since this material is based on a universally recognized academic structure in the world, starting with definition, etiology / pathogenesis and ending with diagnosis, treatment. In order to check the level of students' knowledge, control questions, tests and tasks, and situational problems are given.

The educational and methodical manual is written in English at the highest level and is intended for international students of the 6th semester of the Faculty of "Medical Science" of the RMU.

Reviewers:

Doctor of Medical Sciences, Professor of the Department of Clinical Disciplines RMU Kenzhaev M.G.

Doctor of Medical Sciences, acting Professor of the Department of Faculty Surgery named after Kakish Ryskulova of I.K. Akhunbaev KSMA Aitbaev S.A.

Table of contents

1. Introduction

Relevance: The prevalence of portal hypertension syndrome is judged by the incidence of cirrhosis of the liver, which in recent years has a distinct upward trend in many countries around the world. According to autopsies, the incidence of cirrhosis of the liver varies between 1 and 11%, averaging 2-3%, including in Europe about 1%. Most often, this disease is diagnosed in men over 40 years of age. The mortality rate from cirrhosis of the liver is 14-47 cases per 100,000 population **[1].**

The purpose of the lesson: To teach students the methods of clinical, laboratory, additional methods of research of patients with portal hypertension and its complications, paying attention to the congenital and acquired causes of the disease, the choice of therapeutic tactics depending on the cause of the disease and complications.

Training tasks:

1. On the basis of data on anatomy, physiology, pathological physiology and pathological anatomy, to form students' knowledge about the etiopathogenesis of portal hypertension and its complications.
2. To familiarize students with various forms of the disease and their complications.
3. Teach students to identify the clinical symptoms of portal hypertension.
4. Teach students to interpret the results of radiation (ultrasound, X-ray, CT, etc.) research methods.
5. To show the importance of traditional and modern methods of studying portal hypertension complications.
6. To form students ' knowledge about existing methods of treatment of portal hypertension and its complications.
7. To give an idea of conservative and surgical tactics in patients with various forms of portal hypertension.

What a student should know, be able to represent and represent:

1. Know the anatomy of the portal system.
2. Know the classification of portal hypertension.

3. Know the clinical manifestations of the suprahepatic, intrahepatic, and subhepatic forms of portal hypertension.

4. Have an understanding of the mechanism of portal hypertension development and its possible complications.

5. Know the types of portal hypertension complications and their clinical manifestations.

6. Know the methods of portal system research.

7. Know conservative and surgical methods of treatment for bleeding from varicose veins of the esophagus.

8 Learn the indications and techniques for using the Blackmore probe

Requirements for the initial level of knowledge. For a complete assimilation of the topic, it is necessary to repeat:

- from normal and topographic anatomy: the anatomy of the liver and portal system, the patterns of blood circulation in the liver and portal vein;
- histology: features of the histological structure of the wall of the hepatic arteries and veins of the portal system;
- normal physiology: the function of the portal system, features of blood flow and outflow in the liver.

2. Definition

Portal hypertension (PH) is a syndrome that occurs due to obstruction of the blood flow in the portal system, leading to high portal pressure (over 140-160 mm of water), splenomegaly, varicose veins of the esophagus, stomach, rectum, bleeding from them, the development of ascites and liver failure. The isolation of intra -, extrahepatic and mixed forms of PH was proposed by A. Whipple (1945), R. Linton (1949) and has found wide distribution.

3. Etiology

The etiology of PH is as follows:

1. Hepatitis, fatty hepatosis, cirrhosis of the liver of various etiologies (alcoholic, viral, biliary), which make up 70-80 % of all causes. In the United States, between 26,000 and 35,000 people die each year from chronic liver disease and cirrhosis, with cirrhosis being the 9th most common cause of death and accounting for about 1.2 % of all deaths in the country.

2. Portal liver fibrosis of inflammatory and post-traumatic etiology, Karolyi's disease, myelofibrosis, tumor and parasitic diseases (schistosomiasis, echinococcosis, alveococcosis in endemic areas) of the pancreatobiliary organs.

3. Cardiac (cardiac) cirrhosis of Peak.

4. Congenital and acquired liver vascular abnormalities (aneurysm, thrombosis, hepatic artery embolism, aplasia, hypoplasia, cavernous transformation and portal vein atresia, Kruweye– Baumgarten disease associated with portal vein hypoplasia, liver atrophy, and preserved umbilical vein).

5. Pileflebitis, phlebosclerosis, obliteration and thrombosis of the portal vein, congenital stenosis and atresia of the portal vein and its branches, caused by the spread of the physiological process of postnatal obliteration of the umbilical vein and the venous (arantium) duct to the portal vein, compression of the portal vein and its branches by scars, aneurysm of the splenic and hepatic arteries, benign and malignant tumors, cysts, infiltrates.

6. Congenital and acquired pathology of the hepatic veins and inferior vena cava: Chiari disease (obliterating endophlebitis of the hepatic veins was first described by the Prague pathologist Chiari in 1899), Budd-Chiari syndrome (congenital membranous overgrowth of the inferior vena cava, compression of its adhesions, tumors, increased pressure in the inferior vena cava-constrictive pericarditis, heart defects, heart failure).

7. Omphalitis phenomena experienced in childhood or the consequences of umbilical vein catheterization.

4. Pathogenesis

The key to understanding the pathogenesis of PH is knowledge:

-anatomy of the human venous system, primarily the portal vein system, which receives venous blood from the spleen, stomach, cardiac esophagus, small and large intestines;

-the structure of the walls of the veins of the above organs, adapted to the level of venous pressure in the portal system, not exceeding 140-160 mm of water. art., an increase in which leads to varicose veins, primarily the lower third, the cardiac esophagus and stomach;

- factors that initiate erosion, rupture of varicose veins, which leads to intense esophageal-gastric bleeding, the volume and intensity of which directly affects the progression of liver failure (hepatocyte necrosis), the results of treatment and prospects for the patient's life;

- the degree of compensation for hepatic insufficiency according to Child–Pugh, primarily in cirrhosis of the liver, which is a fundamental pathogenetic and prognostic factor in the treatment of patients with PH;

- the degree of adaptive mechanisms of organs and systems of a person suffering from PG, with timely medical and surgical correction of etiological and pathogenetic factors.

Blood enters the liver through the portal vein system and the hepatic artery. In the portal vein (the speed of portal blood flow is about 1 l/min), blood comes from the upper and lower mesenteric veins, the splenic vein, which collect it from the gastrointestinal organs, the spleen and the pancreas.

Hepatic blood flow combines arterial and venous flows in the central vein of the hepatic lobule. Despite the difference in pressure in the arterioles (110-112 mm Hg) and venules (5-10 mm Hg), arterial blood flow does not prevail. In addition, 75% of the hepatic blood flow passes through the portal vein, and only 25% passes through the artery. In the portal vein of the liver, the portal vein is divided into the right and left, and later-into segmental branches that accompany the hepatic arteries.

The terminal branches of the portal interlobular veins form sinusoids, into which the arterioles of the hepatic arteries also flow. In the sinusoids, venous and arterial blood is mixed, with the help of presinusoidal and postsinusoidal sphincters, arterial and venous pressure is leveled, blood flow and outflow are regulated, and its contact with liver cells occurs. This feature of microcirculation in the liver is provided by a rather complex system of special sphincters that regulate the amount of blood entering the hepatic vein.

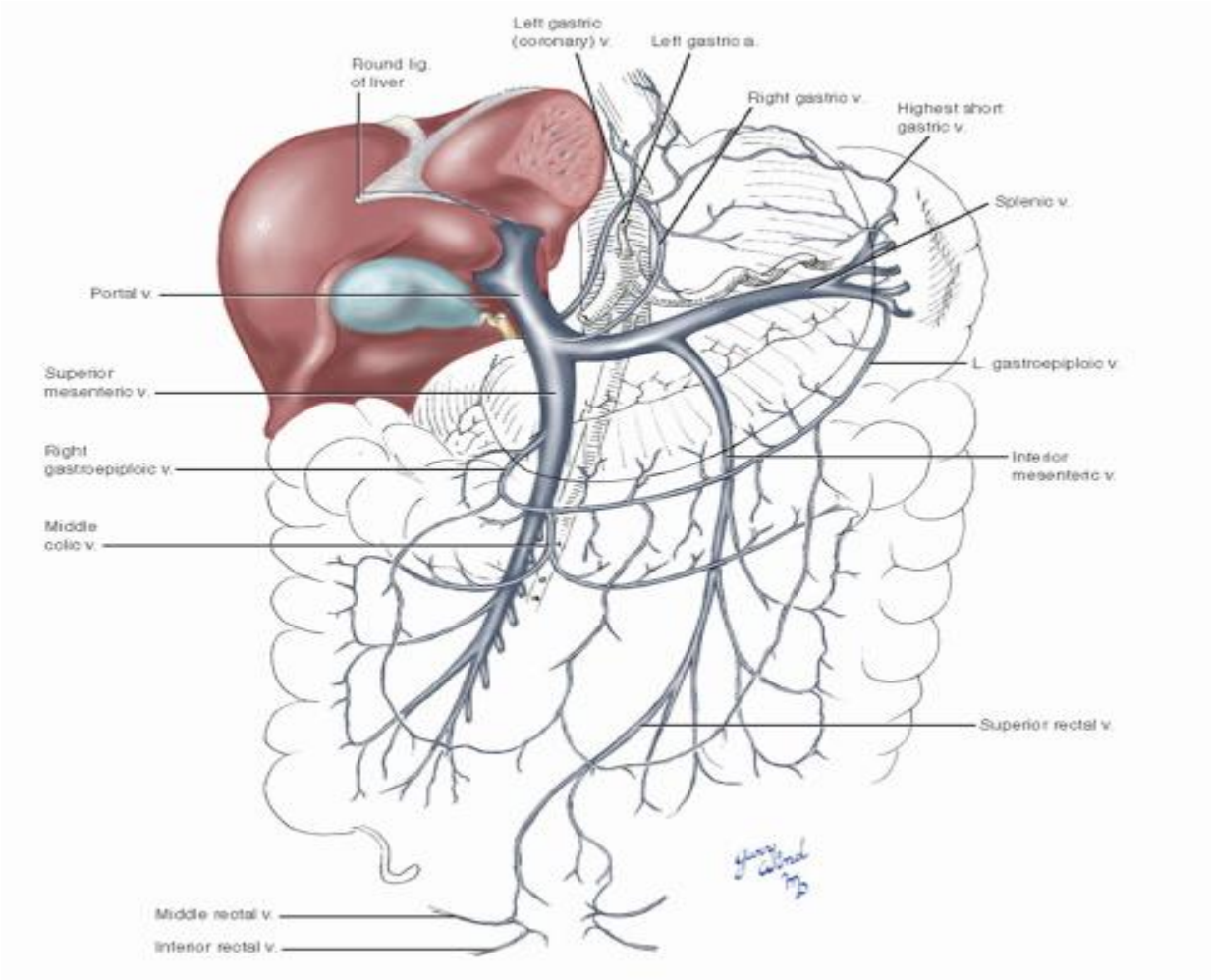


Fig. 4.1 Portal venous system

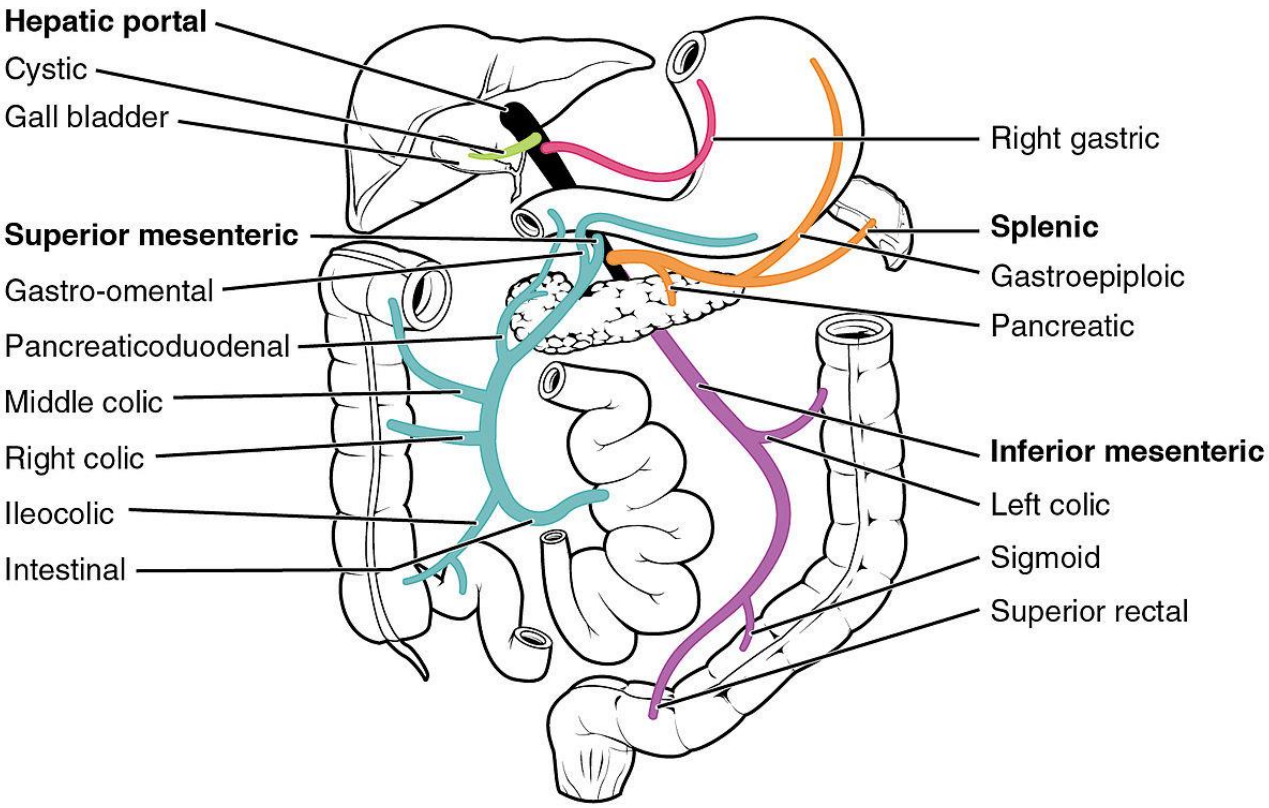


Fig. 4.2 Hepatic portal vein system

In the sinusoids, mixed portal and arterial blood contacts the microvilli of hepatocytes in the Disse spaces, which ensures that the liver performs metabolic functions. Normally, the pressure in the portal vein system is 5-10 mm Hg. Its increase indicates the development of PH. This syndrome is a consequence of increasing pressure in the portal vein or in one of its branches (physiological portal pressure reaches a maximum of 7-12 mm Hg.), which is due to both an increase in venous resistance in the prehepatic, hepatic and posthepatic parts of the portal system, and an increase in abdominal blood flow. This occurs against the background of a decrease in arterial vascular resistance.

The collaterals divert part of the portal blood flow from the liver, which helps to reduce PH, but never completely eliminates it. Most often, varicose veins occur in the esophagus and stomach.

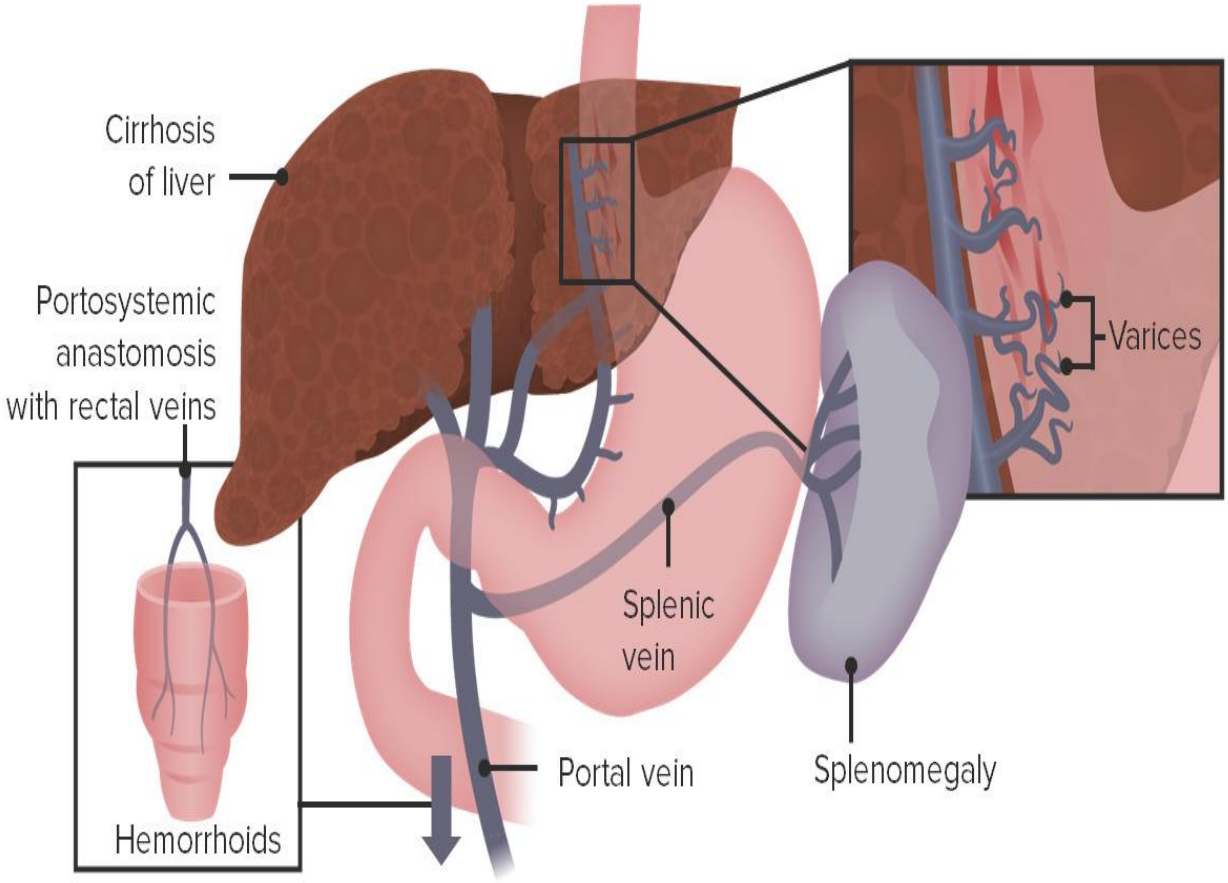


Fig. 4.3 Blood circulation in the liver and the mechanism of development of PH and esophageal varicose veins.

The most common cause of the intrahepatic form of PH is cirrhosis of the liver. Morphological features of cirrhosis determine the restructuring of the cellular and vascular architectonics of the liver. A single sinusoidal network for the entire liver in cirrhosis is divided into many isolated fragments. The false lobules formed as a result of regeneration and fibrosis have their own sinusoidal network, which differs significantly from that in normal hepatic lobules. The sinusoidal network of false lobes is many times larger than normal lobes: it lacks the sphincter mechanisms that regulate blood flow. In addition, the vascular plexuses of the neighboring lobules anastomose with each other. Through them, the branches of the portal vein, the hepatic artery connect directly with the branches of the hepatic veins, i.e., direct portopehepatic shunts develop.

Collateral circulation is an important feature of portopehepatic circulation in cirrhosis, it is carried out not only by intrahepatic shunts, but also by extrahepatic

portocaval anastomoses. A significant part of the blood passes through the liver through the septum vessels, bypassing the active parenchyma. "Useless" blood flow through the liver can account for more than 50 % of the blood flowing through the hepatic artery and portal vein. Blood circulation bypassing the active parenchyma significantly impairs the metabolism of liver cells, leads to periodic bacteremia and endogenous intoxication with febrile episodes. Clinically, this is manifested by encephalopathy of varying severity.

Due to spasm and significant resistance to blood flow in the hepatic artery, its redistribution occurs in the system of the celiac trunk, which leads to an increase in the volume of blood flow in the splenic artery. In combination with the difficulty of blood outflow through the splenic vein, this leads to the development of splenomegaly, and in combination with hyperplasia of the reticuloendothelial tissue — the occurrence of hypersplenism (pancytopenia in the general blood test). A reduction in the flow of portal blood to the liver is accompanied by a slowdown in its metabolic processes due to a decrease in the volume of blood flow and a corresponding decrease in the number of hepatocytes.

The level of pressure in the portal vein is determined by three main factors: the amount of portal blood flow, the vascular tone of the branches of the portal vessels, and the total intrahepatic vascular resistance. PH in cirrhosis of the liver entails dilation of the vessels of the abdominal cavity. This in turn leads to an increase in portal blood flow. It follows from the above that the pathogenesis of PHcan not be reduced only to the obstruction of intrahepatic venous blood flow on the basis of mechanical obstacles to it, the restructuring of the angioarchitectonics of the liver and other local factors. The above functional deviations are also important, which opens up the possibility of pharmacological effects on them. Arteriovenous anastomoses between the branches of the hepatic artery and the portal vein in the fibrous septa (septa) also contribute to an increase in pressure in the portal vein system, leading to additional blood flow to the portal system.

Portal vein hypertension is accompanied by an increase in lymph formation and hyperdynamic hypertension in the lymphatic vessels, which in turn leads to a

variety of structural and functional disorders of the abdominal organs. There is no doubt that the organic substrate of PH and excessive lymph production due to the difficulty of venous outflow from the liver occupy almost a central place in the genesis of one of the main manifestations of intrahepatic portal block — ascites.

Nevertheless, it would be a mistake to reduce everything to just this. One of the prerequisites for the development of ascites is shifts in the renin-aldosterone-angiotensin system. It should be remembered about the role of increased activation of the renin-angiotensin mechanism, which leads to hypersecretion of aldosterone. This is due to a violation of renal perfusion on the basis of the general hemodynamic shifts inherent in cirrhosis of the liver. Another factor in the development of ascites is hypoalbuminemia with a drop in oncotic pressure of blood plasma, which, as is known, contributes to the release of intravascular water beyond the vascular bed

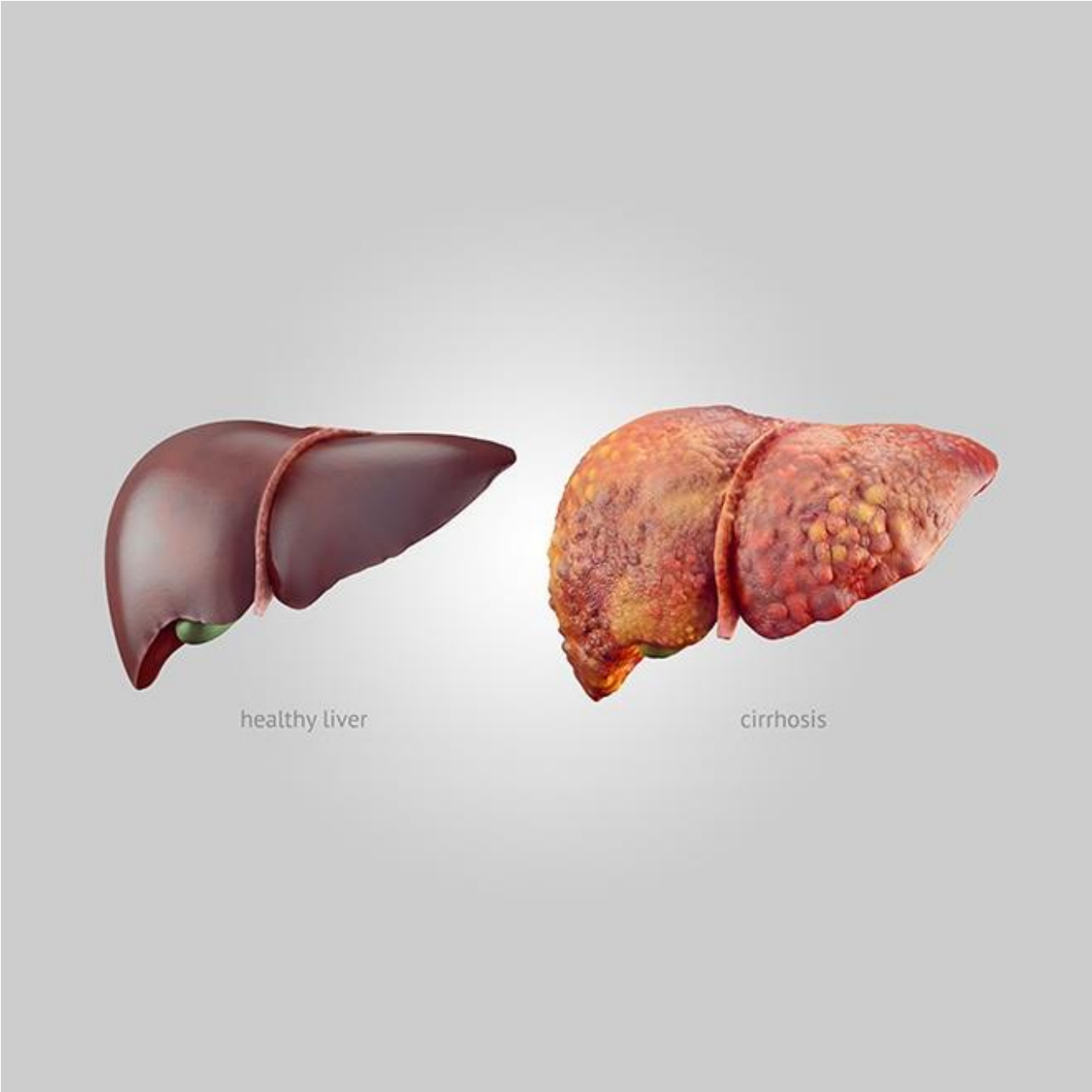


Fig. 4.4 Cirrhosis of the liver

The vasodilation of the arterioles of the abdominal organs that develops in cirrhosis of the liver leads to a consistent activation of sympathetic impulses, which stimulates the release of renin in the kidneys and the secretion of antidiuretic hormone by the pituitary gland. Another consequence of sympathetic hypertension is a violation of kidney perfusion, and in some cases-and a drop in their production of prostaglandins. This leads to a decrease in glomerular filtration with sodium and water retention, which in turn contributes to the formation of ascites. This is expressed in the deterioration of blood circulation conditions in the inferior vena cava

and in the abdominal organs. At the same time, the respiratory excursion of the lungs is limited and cardiac activity is hindered. The resulting increase in intra-abdominal pressure contributes to gastroesophageal reflux, which in turn can provoke bleeding from the varicose veins of the esophagus. The greatest significance for the prognosis of PH is the degree of activity and progression of the cirrhotic process in the liver, which affects the functional capabilities of the liver (liver failure). In patients with PH, the degree is evaluated on the Child-Pugh scale.

Gradually, extrahepatic portosystemic shunts are formed in patients with PH. Knowledge of the anastomoses connecting the portal vein and its tributaries to the hollow vein system is of great importance in understanding the processes that develop during the formation of the block in the portal system. There are 4 main groups of portocaval anastomoses

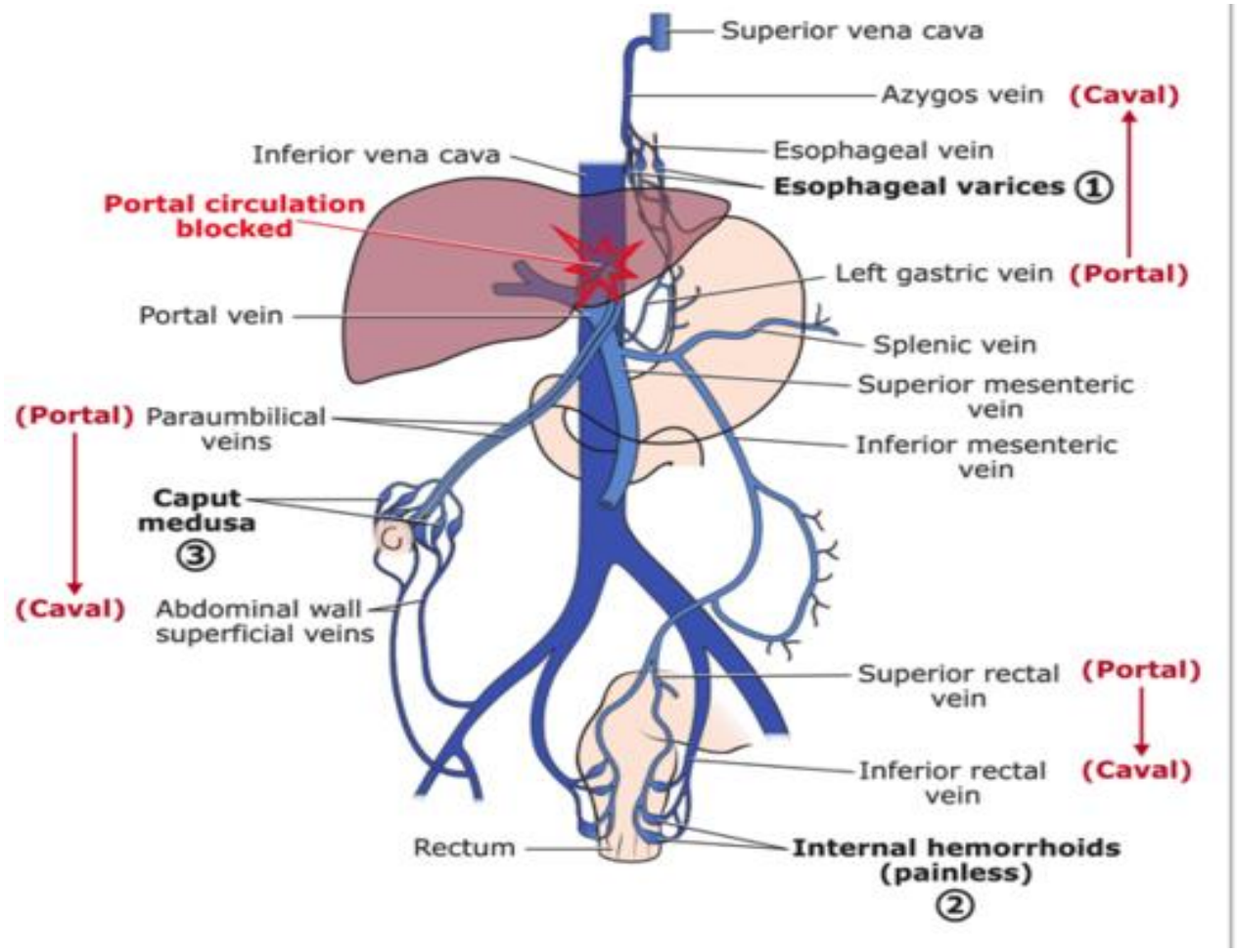


Fig. 4.5 Main groups of portocaval anastomoses

– gastroesophageal (in the cardiac part of the stomach and the abdominal part of the esophagus), connecting the portal vein with the superior vena cava through the vena cava of the stomach, unpaired and semi-paired veins;

– between the branches or main trunk of the portal vein and the veins of the anterior abdominal wall and diaphragm, formed by the parotid veins (between the upper epigastric and umbilical veins);

- between the venous plexuses of the rectum and the inferior vena cava, through the upper (portal vein basin) and lower hemorrhoidal veins (inferior vena cava basin);

- between the veins of the gastrointestinal tract and the veins that divert blood to the retroperitoneal and mediastinal veins (Guillot anastomoses between the veins of the stomach, diaphragm and kidney capsules, the veins of Sappey, Retzius-anastomoses between the splenic vein or one of its branches and the left renal veins, ovarian veins or kidney capsules).

However, in some cases, despite the development of significant portosystem collaterals, the pressure does not decrease either in the portal system or in the collaterals themselves. Increased vascular resistance is the most common cause of PH. There are no valves in the portal venous system, and any structural abnormalities in it lead to an increase in pressure.

Along with increased intra-abdominal pressure (due to ascites, constipation, heavy physical labor), which increases the likelihood of rupture of the varicose veins of the esophagus, the provoking factors for the development of esophageal-gastric bleeding are:

- peptic factor (reflux-esophagitis);
- injury of the esophageal veins with coarse food (bones, etc.);
- hemostatic disorders due to liver failure and hypersplenism.

The main sources of bleeding from the upper gastrointestinal tract include:

1. Esophageal varicose veins (EVV). It is assumed that the onset of bleeding, as a rule, is associated with two factors: tear of the thinned wall of varicose veins due to constant high intraluminal pressure and the damaging effect of hydrochloric acid reflux on the thinned wall of the EVV.

The detection rate of EVV in patients with cirrhosis of the liver ranges from 25 to 80 %, averaging 60 %. Every year, varicose veins develop in 10-15 % of patients with cirrhosis. An increase in the size of varices occurs in 10-20 % of patients within a year after the first detection. The process of formation is carried out faster in the alcoholic etiology of the disease.

2. Varicose veins of the stomach (VVS). Bleeding from the VVS accounts for 20-30 % of all varicose veins. The incidence of gastric varices in patients with PH ranges from 6 to 78 %. This variability is explained by the different etiology of PH and the limitations of diagnostic techniques. VVS is more often observed in the subhepatic form of PH than in cirrhosis.

Bleeding from gastric varices develops less often than from esophageal varices. However, the severity of bleeding, especially from fundal varices, and mortality are higher. These bleeds are almost impossible to stop by endoscopic methods.

3. Portal hypertension gastropathy, which is another manifestation of hyperdynamic circulation in PH. Morphologically, it is characterized by the expansion of submucosal and subserous veins. In patients with cirrhosis, the incidence of portal hypertension gastropathy is 50-60 %. Gastropathy is the second most common cause of bleeding in patients with cirrhosis of the liver. They can manifest as acute bleeding or as chronic iron deficiency anemia. Recurrent bleeding occurs in 62-75 % of patients.

Portal hypertension gastropathy is classified from moderate to severe. Changes can occur in any part of the stomach, but usually the most severe are found in its body. Moderate gastropathy is characterized by the so-called snakeskin: a mosaic pattern of the gastric mucosa with multiple fields of hyperemia, surrounded by a thin white reticulin network. This is the most frequent and quite specific endoscopic picture. Other signs of moderate gastropathy include small pink spots and superficial erythema on the gastric folds, leading to the outline of the folds in the form of ribbons. In severe gastropathy, individual cherry-red spots, erosions, or diffuse hemorrhagic gastritis appear.

Varicose veins of the rectum and ectopic varices are rare sources of bleeding in PH, but present significant difficulties for controlling bleeding.

In the subhepatic form of PH, the liver remains intact, blood circulation in it is not disturbed, and its parenchyma is not affected. There are several options for obstructing venous blood flow: block at the level of the portal vein trunk; thrombosis, compression from the outside of the splenic vein; block of the superior mesenteric vein, as well as their combinations (block of the splenic and superior mesenteric veins, trunk of the portal vein and splenic or superior mesenteric).

Starting as a type of subhepatic portal block, the pathological process leads to the development of blood stagnation in the entire portal system, the formation of portoportal and portocaval pathways of venous blood outflow, the occurrence of ascites, splenomegaly. The end result of this form of PH is: bleeding from the EVV and VVS, splenomegaly with hypersplenism, and increasing ascites.

The suprahepatic form of PH is characterized by an initial difficulty in the outflow of blood from the unaffected liver. The pressure increases in the entire portal vein system: inside the liver and under it. In the future, this is accompanied by swelling of the liver, an increase in its size and the subsequent occurrence of secondary cirrhosis. Stagnation of blood in the entire portal system causes the development of collateral venous circulation, ascites, splenomegaly. As a result, complications such as bleeding from varicose veins, ascites and liver failure develop.

In the case of a mixed form of PH, the pathogenesis includes those factors that are typical for each of the above forms of PH. There is a situation in which several intra - and extrahepatic causes act simultaneously: cirrhosis of the liver in combination with portal vein thrombosis as a complication, or portal vein thrombosis with subsequent cirrhosis of the liver and the occurrence of a number of complications that pose a real threat to the patient's life, which include bleeding from EVV, ascites and hepatic encephalopathy.

Thus, the PH syndrome as a complication of diseases of the liver and vessels of the portal system in the formation of esophageal varicose veins and the threat of fatal bleeding acquires a primary role in the prognosis for the patient's life and puts in the

first place therapeutic measures aimed at preventing and stopping esophageal-gastric bleeding.

5. Classification

According to the level of the portal system block, there are:

- subhepatic block (portal vein thrombosis, congenital portal vein abnormality, compression of the portal vein by a tumor, parasitic (alveococcosis) and inflammatory (pancreatitis) formations of the pancreatobiliary region). Splenic vein thrombosis with the development of varicose veins of the cardiac department and the bottom of the stomach is classified as segmental PH, being a type of subhepatic block;

-intrahepatic block (cirrhosis, liver fibrosis, neoplastic, parasitic liver disease, trauma, cystic masses, hemangiomas of the liver);

– suprarenal unit (violation of the outflow of venous blood from the liver into the system of the inferior Vena cava due to lesions of the hepatic veins, inferior vena cava (suprarenal segment) — the Budd–Chiari and endoplamic hepatic veins — a disease Budd–Chiari);

mixed unit (liver cirrhosis complicated by portal vein thrombosis, etc.).

Based on the level of increase in pressure in the portal system the following is distinguished:

– PH of the first degree (pressure of 250-400 mm of water);

- PH of the second degree (pressure of 400-600 mm of water)

– PH of the third degree (pressure of more than 600 mm of water).

Classification of the degree of varicose veins of the esophagus and stomach. To date, several classifications have been proposed. The division of varicose veins into degrees is not only of theoretical importance, but also allows you to compare the results of treatment and carry out subsequent dynamic control.

In the classification of K.-J. Paquet (1983), 4 degrees of EVV are distinguished:

- Grade I-single venous ectasia (visible in FGDS, but not radiologically determined);
- Grade II — single well-defined venous trunks, mainly in the lower third of the esophagus, which remain clearly pronounced during insuflation. At the same time, there is no narrowing of the esophageal lumen and thinning of the epithelium on the veins;
- III degree-trunks of varicose veins in the lower and middle third of the esophagus, narrowing its lumen and partially decreasing only with strong air insufflation. Single red markers or angiectasias are detected on the tops of varicose veins;
- IV degree-varicose veins, which completely make the lumen of the esophagus, even with maximum air insufflation. Endoscopically, thinning of the epithelium above the veins is detected. Multiple erosions and/or angiectasias are detected at the apices of varicose veins.

N. Soehendra, K. Binmoeller in 1997 proposed a three-step classification, in which the authors differentially consider PH in the esophagus and in the stomach:

1. Esophageal varicose veins:
- grade I: the diameter of the veins does not exceed 5 mm, they are elongated, located only in the lower third of the esophagus;
- Grade II: the diameter of the veins is from 5 to 10 mm, they are convoluted, spread in the middle third of the esophagus;
- III degree: the size of the veins is more than 10 mm, they are tense, with a thin wall, located close to each other, red markers on the surface of the veins.

2. Varicose veins of the stomach:
- grade I: the diameter of the veins is not more than 5 mm, they are barely visible above the gastric mucosa;
- II degree: the size of the veins is from 5 to 10 mm, they have a solitarnopolipoid character;
- III degree: the diameter of the veins is more than 10 mm, they are thin-walled, polypoid in nature, and represent an extensive conglomerate of nodes.

The Japanese Scientific Society for the Study of PH in 1991 published the basic rules for the description and registration of endoscopic signs of varicose veins of the esophagus and stomach. The rules contain 6 main criteria according to which the state of varicose veins is evaluated both before and after treatment. In general terms, this classification is currently used by most specialized clinics dealing with this problem. It is presented as follows:

1. Localization (prevalence) EVV:

- lower third of the esophagus-Li;

– middle third — Lm; - upper third-Ls.

Localization (location relative to the cardia) VVS — Lg:

in the cardia — Lg-c;

– remotely from the cardia — Lg-f.

2. Shape (type and size of varicose veins):

– no veins — FO;

– short, small caliber-F1; - moderately expanded, convoluted-F2; — significantly expanded, nodular-F3.

3. Color (this category reflects the wall thickness of varicose veins):

- white-Cw;

- blue (indicates a significant thinning of the wall) - Cb.

4. Red wall markers:

– red cherry spots — CRS; - hematocyst spots-HCS; - telangiectasia-TE.

5. Signs of bleeding (in acute bleeding, it is necessary to determine its intensity, and in the case of spontaneous hemostasis — to assess the nature of the blood clot) –

- during the bleeding period: jet, in the form of seepage; – after reaching hemostasis: red blood clot, white blood clot.

6. Changes in the esophageal mucosa (can be both a manifestation of reflux disease, often combined with diseases occurring with PH syndrome, and a consequence of therapeutic endoscopic exposure):

- erosion-E; - ulcer-U;

- rumen-S.

Many classifications have been proposed to determine the severity of liver cirrhosis, but at present the Child–Turcott classification in the Pag modification (1964-1972) is more often used.Determination of the Child–Turcott cirrhosis severity class in the Pag modification

Child-Turcotte-Pugh Classification for Severity of Cirrhosis			
	Points*		
	1	2	3
Encephalopathy	None	Grade 1-2 (or precipitant induced)	Grade 3-4 (or chronic)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
INR	<1.7	1.7-2.3	>2.3
*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)			
Class A = 5 to 6 points (least severe liver disease)			
Class B = 7 to 9 points (moderately severe liver disease)			
Class C = 10 to 15 points (most severe liver disease)			

Table 5.1 Child–Turcott classification in the Pag modification

6. Clinical picture

The manifestations of PH depend on the level of portal block, the main disease that caused the violation of blood flow in the portal vein, the severity of liver damage and the nature of the complications that occurred.

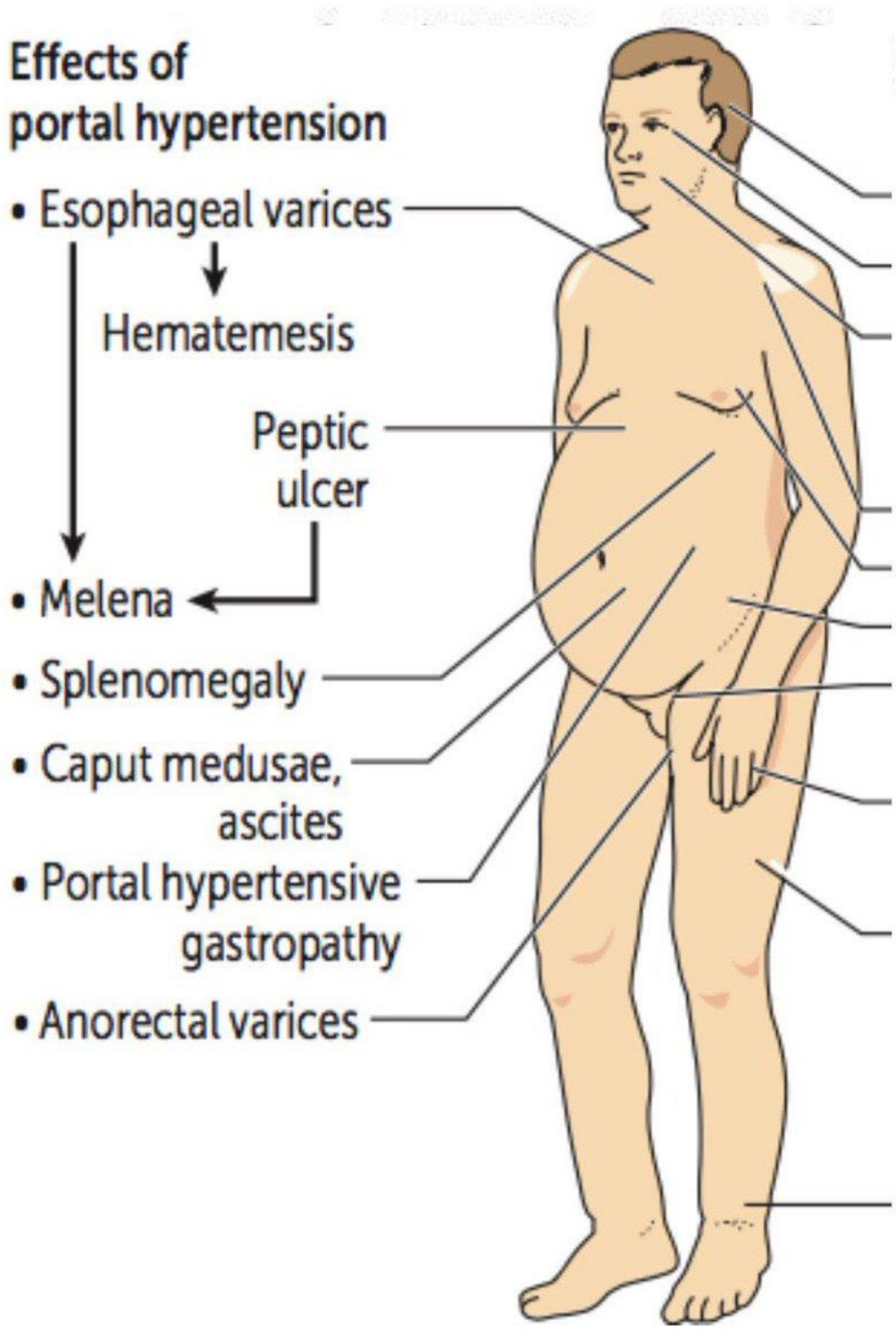


Fig. 6.1 The clinical picture with portal hypertension

The main symptoms of PH are:

- symptoms of the underlying disease that caused PH;

- varicose veins of the esophagus, cardiac stomach, rectum, complicated by esophageal-gastric (rectal) bleeding;
- size change and impaired liver function;
– hepatic insufficiency with the development of encephalopathy;
- progressive accumulation of ascitic fluid; - splenomegaly with hypersplenism.

The intrahepatic form of PH develops with liver damage. In the anamnesis, patients have a history of hepatitis, cirrhosis of the liver, parasitic (echinococcosis, alveococcosis) and tumor damage (more often metastatic) of the liver, etc. The symptoms of the underlying disease are joined and progressed by signs of liver failure: weight loss, jaundice staining and dryness of the skin with a decrease in their turgor, expansion of the subcutaneous veins of the anterior abdominal wall around the navel — "medusa's head", "liver signs" (vascular asterisks, "liver palms", etc.). The patient is concerned about general weakness, decreased memory and intelligence, recurrent headaches, dyspeptic disorders. On examination, the abdomen is enlarged due to the accumulation of ascitic fluid (percussion isdetermined by bluntness in the sloping areas of the abdominal cavity and in the small pelvis). With palpation and percussion, a moderately painful, enlarged or reduced (with cirrhosis) liver is revealed in size, its edge is rounded, uneven or lumpy. As a complication, bleeding from EVV and VVS often occurs with a typical gastrointestinal bleeding clinic.

The subhepatic form of PH develops with a block of the portal vein or its branches — the splenic or superior mesenteric (segmental form of PH). This disease most often develops in young people due to trauma, pileflebitis, pancreatitis, phlebosclerosis, obliteration and thrombosis of the portal vein and its branches, compression of the veins of the portal system by scars, tumors, and infiltrates of the abdominal cavity. This blockade is characterized by the absence of symptoms of liver damage, since it is not involved in the pathological process. When examining the patient, the size of the liver is not changed, there is an accumulation of ascitic fluid, splenomegaly, which is not always manifested by hypersplenism. The disease progresses slowly with occasional episodes of esophageal-gastric bleeding and the development of anemia.

The suprahepatic form of PH is caused by the occurrence of a block for the outflow of venous blood from the liver, which is observed in constrictive pericarditis, cardiac cirrhosis, Budd– Chiari disease or syndrome. Patients complain of general weakness, pain and a feeling of heaviness in the right hypochondrium, jaundice, weight loss, abdominal enlargement due to ascites, edema of the lower extremities. At the beginning of the disease, with palpation and percussion, the liver is enlarged, the edge is rounded, and later its size decreases, the contours become lumpy. An enlarged spleen is determined. It is characterized by the rapid development and progression of ascites that do not respond to diuretic therapy. In the later period, there are bleeding from the EVV and VVS.

The mixed form of PH is not common and is a combination of two or more types of the course of the pathological process (cirrhosis of the liver with portal vein thrombosis as a complication or portal vein thrombosis with the subsequent development of cirrhosis and the occurrence of a number of complications). The clinical manifestations of this pathology are very diverse. In patients, there are signs of liver damage with the development of hepatic insufficiency and encephalopathy, an enlarged spleen with hypersplenism, as well as the occurrence of gastrointestinal bleeding that poses a real threat to the patient's life.

7. Diagnosis

Diagnosis of PH syndrome is based on the patient's complaints, anamnesis of the disease, life, objective examination data, as well as laboratory and instrumental methods of examination.

Complaints

The examination of the patient reveals:

- pain syndrome
- asthenic syndrome;
- dyspeptic syndrome;
- cholestatic syndrome;

– esophageal-gastric (rectal) bleeding;

- venous pattern on the anterior abdominal wall;

- signs of liver failure;

- ascites (an increase in the size of the abdomen).

Anamnesis

To clarify the etiology of PH, it is necessary to find out:

- whether the patient had hepatitis, cirrhosis of the liver (intrahepatic block);

– have you been treated for alveococcosis or echinococcosis of the liver (intrahepatic or subhepatic block);

– are there any signs of a tumor lesion of the abdominal organs;

– did you suffer from purulent diseases of the navel in childhood (omphalitis);

– if he was operated on for appendicitis — whether there were purulent postoperative complications (development of pileflebitis);

- whether there were blunt abdominal injuries;

- at what age the symptoms of PH began to appear.

Objective research data

Objective examination reveals the following signs of PH: clinical signs of chronic hepatitis (cirrhosis), dilation of the veins of the anterior abdominal wall ("medusa's head"), hepatomegaly, splenomegaly, cutaneous hemorrhagic syndrome (hypersplenism — pancytopenia), edema (dysproteinemia). Every third patient with cirrhosis of the liver has pronounced clinical symptoms of encephalopathy-a syndrome that combines neurological and psychoemotional disorders caused by metabolic disorders in the central nervous system.

The latter are associated with liver failure and, in particular, with the influence of ammonia, phenols, and enterobacteria toxins.

Encephalopathy is manifested first by euphoria, then by depressive syndrome, slow speech, tremor of the fingers, and changes in handwriting. After that, there are drowsiness, inappropriate behavior, sleep disorders, changes in the electroencephalogram. Often, hepatic encephalopathy is provoked by electrolyte

disturbances (a consequence of the use of large doses of diuretics, diarrhea, vomiting), bleeding, infection, and alcohol abuse.

Instrumental diagnosis

Ultrasound examination allows to detect an increase in the size of the liver and spleen, the appearance of portal collaterals, the presence of free fluid in the abdominal cavity (ascites), an increase in the size of the veins of the portal system; to visualize varicose veins in the retroperitoneal, subhepatic space, small pelvis. Under the control of ultrasound examination, a puncture biopsy is performed to determine the type of liver damage (hepatitis, cirrhosis, metastases).

Doppler imaging evaluates both hepatic venous systems by visualizing the lobar and segmental branches of the portal vein, as well as the three hepatic veins that flow into the inferior vena cava. Normally, the blood flow in the portal vein is directed to the liver, it changes depending on the phase of respiration. The amount of blood flowing in the portal vein in 1 minute is called the volumetric blood flow of the portal vein. Normally, the maximum blood flow rate in the shallow exhalation phase is 0.15–0.20 m/s. With the development of the disease, the maximum speed of blood flow in the portal vein decreases to 0.09–0.12 m/s. This indicator is significantly lower in severe fibrosis than in moderate fibrosis. With the development of fibrosis, the spleen also undergoes significant changes. Moderate enlargement of the spleen, the presence of an additional lobe that reflects organ hyperplasia, and visualization of an expanded venous network in the spleen gate are indirect signs of increased pressure in the portal vein system. Normally, the volume of blood flow in the splenic vein is 28 ± 10 % of the portal blood volume per minute. The destruction of the normal structure of the liver lobule in cirrhosis in the bulk of the liver parenchyma leads to the formation of an insurmountable obstacle to the blood flow in the vessels of the portal vein basin. In most cases, the maximum rate of blood flow in the portal vein in cirrhosis of the liver in the initial period is significantly reduced (0.11 ± 0.02 m / s) compared to that in healthy people.

To exclude cirrhosis of the liver in the compensation stage, the presence of a triad of symptoms is determined:

1) reducing the maximum rate of blood flow in the portal vein;

2) the length of the spleen increases more than the age norm;

3) a decrease in the number of platelets, which is typical in 98 % of cases for cirrhosis of the liver.

If all 3 of the proposed symptoms do not go beyond normal, then in this case, cirrhosis of the liver with a high degree of probability can be excluded.

An absolute sign of PH syndrome is the detection of spontaneously formed collaterals — anastomoses during ultrasound examination. A sharp increase in the flow rate in the main trunk of the portal vein and a slowdown in the lobar branches is a sign of intrahepatic portosystemic bypass surgery.

A decrease in the maximum flow rate in the portal vein of less than 0.10 m / s in cirrhosis of the liver is a prognostically unfavorable sign. With a sharp slowdown in the blood flow in the portal vein, spontaneous cancellation of the portal blood flow can occur, as well as complete or partial thrombosis of the portal, splenic and other veins of the portal system. The absence of portal vein pulsation during respiration is a late, but in 94% of cases sensitive and in 90% specific sign of PH syndrome.

X-ray examination of the esophagus and stomach using a contrast agent can reveal the presence of varicose veins (in the form of multiple defects in filling the corresponding sections of the esophagus and stomach with contrast), signs of reflux esophagitis.

Magnetic resonance imaging allows you to get an image of the parenchymal organs of the abdominal cavity, retroperitoneal space, large vessels in which the flowing blood acts as a natural contrast; it makes it possible to diagnose diffuse and focal diseases of the liver and other organs.

Computed tomography confirms (excludes) diffuse (focal) liver damage.

Angiography with selective celiac or mesenterica in the venous phase of the study allows for clear visualization of the portal vein and its tributaries. When a contrast agent is injected into the splenic artery, the splenic vein and its main tributaries are clearly identified. When contrast is introduced into the superior mesenteric artery, the superior mesenteric and portal veins are visualized. This study

also makes it possible to clearly identify the size of the vessels, the nature and direction of blood flow in the main venous collaterals, such as the left gastric and inferior mesenteric veins.

The bottom (top) cavography performed for suspected suprarenal block.

Scintigraphy of the liver and spleen allows you to set reduced and uneven accumulation of radioisotope in the liver, a marked decrease in the intensity of isotope separation from the blood, increase or decrease the size of the liver, enlargement of the spleen, actively accumulating isotope.

Endoscopic examination.

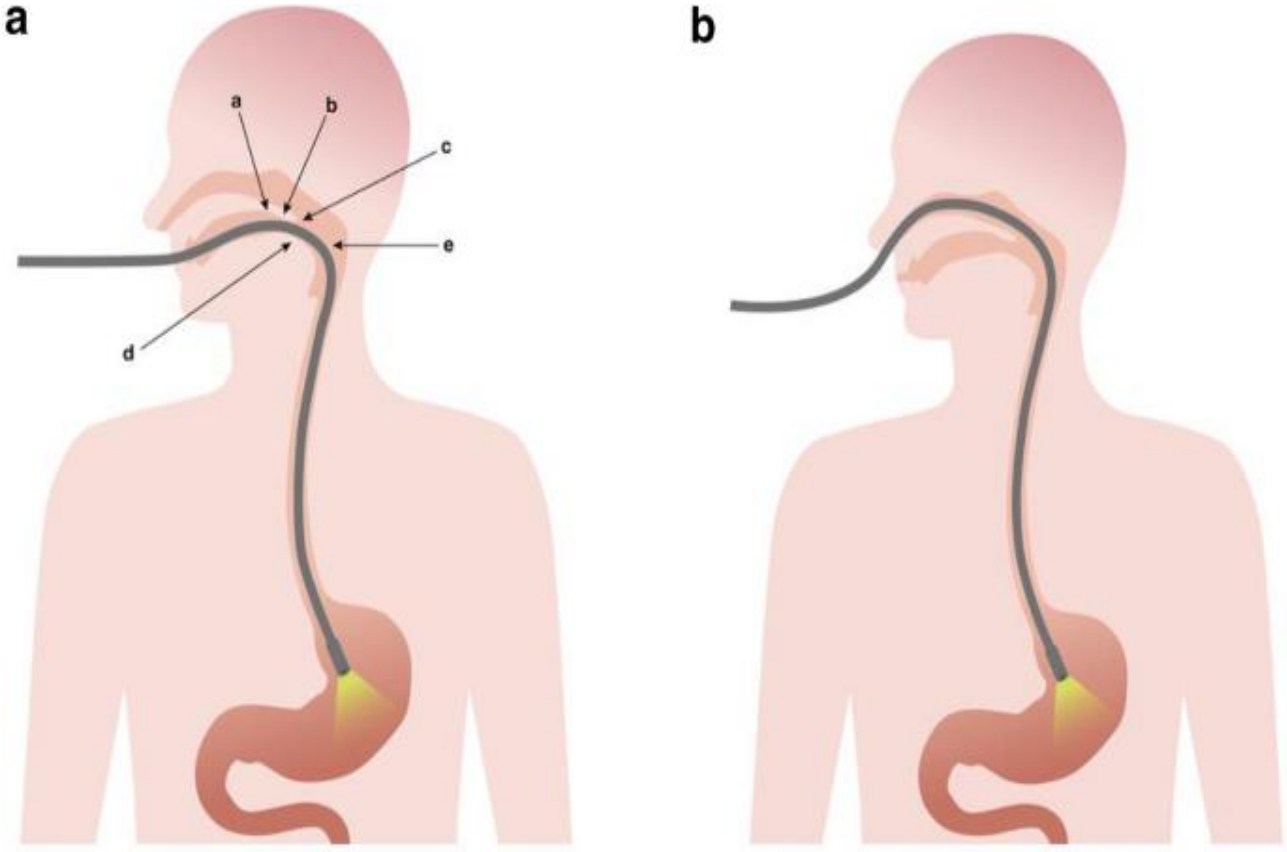


Fig. 7.1 Esophagoscopy

Splenoportography (invasive technique)

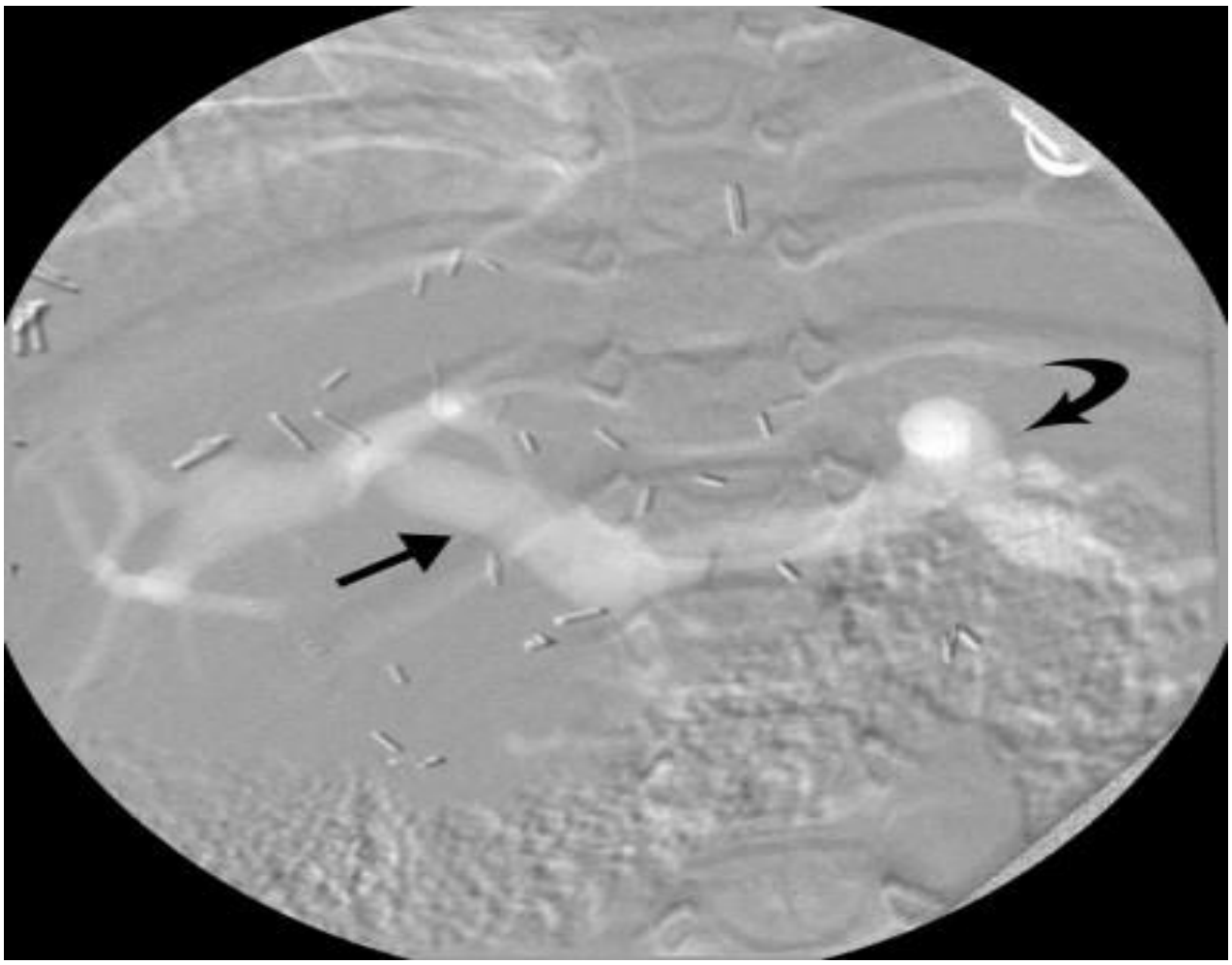


Fig. 7.2 Splenoportography

Laboratory diagnosis

When conducting laboratory studies, the phenomena of hypersplenism (anemia, thrombocytopenia, leukopenia in different combinations or pancytopenia), signs of cholestasis (hyperbilirubinemia, bilirubinuria, elevated levels of alkaline phosphatase, cholesterol, reducing blood iron), signs of liver cell failure (increase in AST, ALT, aldolase, a decrease of cholinesterase activity), dysproteinemia, protein-generating violation of liver function and, above all, a violation of blood coagulation (coagulation hemostasis) with a tendency to hypocoagulation.

8. Treatment

8.1. Tactics of management and treatment of patients with bleeding from varicose veins of the esophagus

Bleeding from EVV is the most frequent and life-threatening complication of PH, developing in 80 % of cases. Significant risk factors for bleeding include:

- grade III esophageal varicose veins;
- esophageal dilatation;
– erosive or ulcerative esophagitis;
– portal pressure above 330-350 mm of water;
–severe liver function disorders (Child-Pugh group C).

A single treatment program for this complication of PH syndrome has not yet been developed. The poor tolerance of patients with cirrhosis of the liver to extensive, traumatic operations has now led to the development and search for a rational combination of minimally invasive interventions, the priority of which are methods of endoscopic and X-ray endovascular hemostasis.

If there is bleeding from the EVV in PH, the following measures are taken:

1. Exposure to the source of bleeding. The use of an esophageal probe with pneumoballons is quite effective and allows you to stop bleeding in 70-80 % of cases. The most widely probe Sengstaken of Blackmore. The lack of effect from the use of the probe suggests bleeding from the veins of the cardiac part of the stomach, which are not compressed by the gastric balloon.

The technique of setting up the Sengstaken-Blackmore probe is as follows:

1) the probe is placed through the nose, previously liberally lubricated with glycerin;
2) the patient is psychologically prepared and informed that the placement of the probe is the salvation of his life;
3) check the tightness of the cylinders by inflating them with a syringe;
4) after the probe is carried out (the flow of gastric contents, blood through the inner lumen of the probe), first inflate the small gastric balloon (the one that will stand in

the cardiac part of the stomach) to 30-50 ml of air, pull it up to fixation in the cardia (at this level, the probe is fixed to the nose) and inflate the large esophageal balloon (80-100 ml of air) until the patient has unpleasant sensations in the chest;

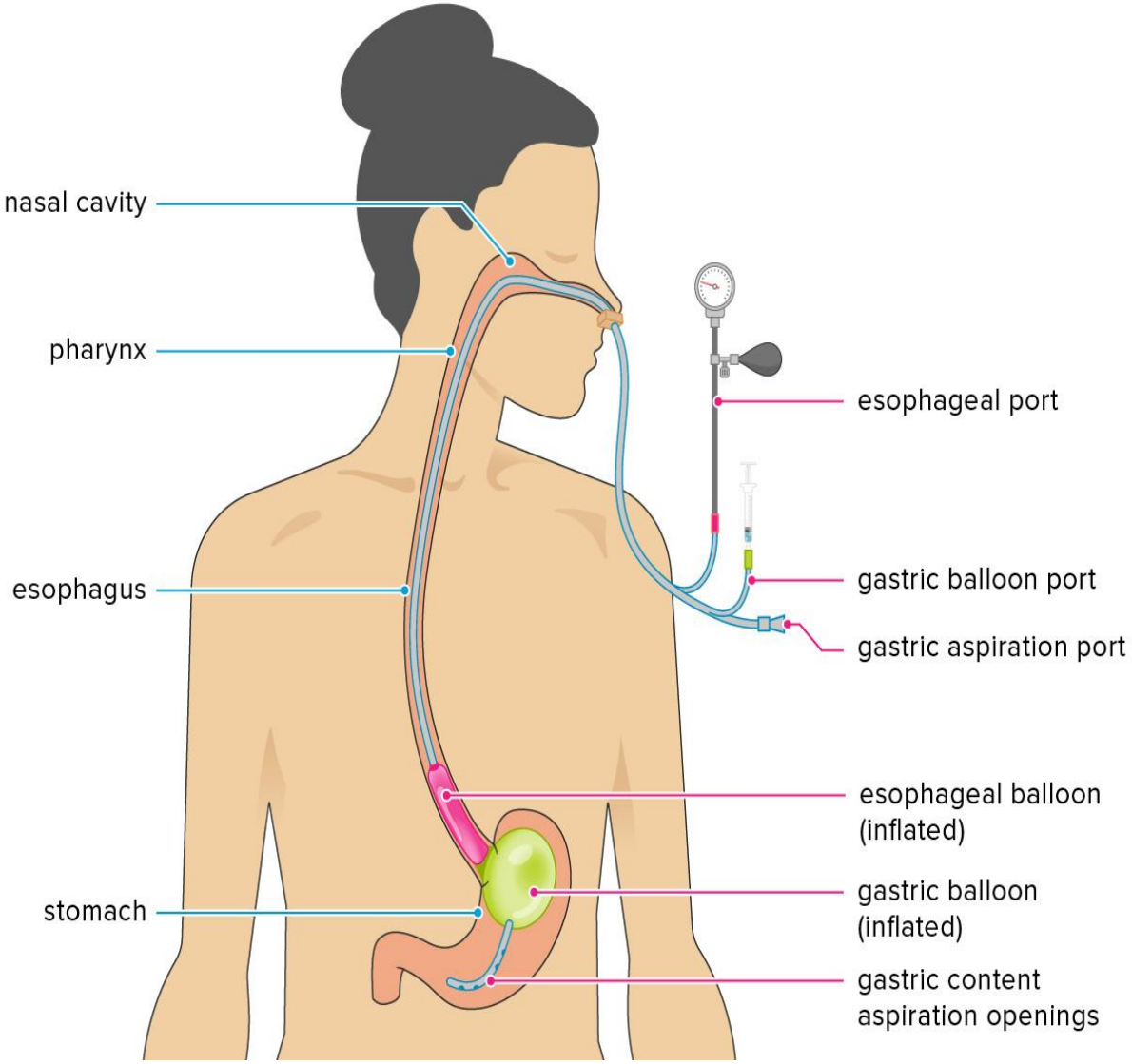


Fig. 8.1.1Sengstaken-Blackmore probe

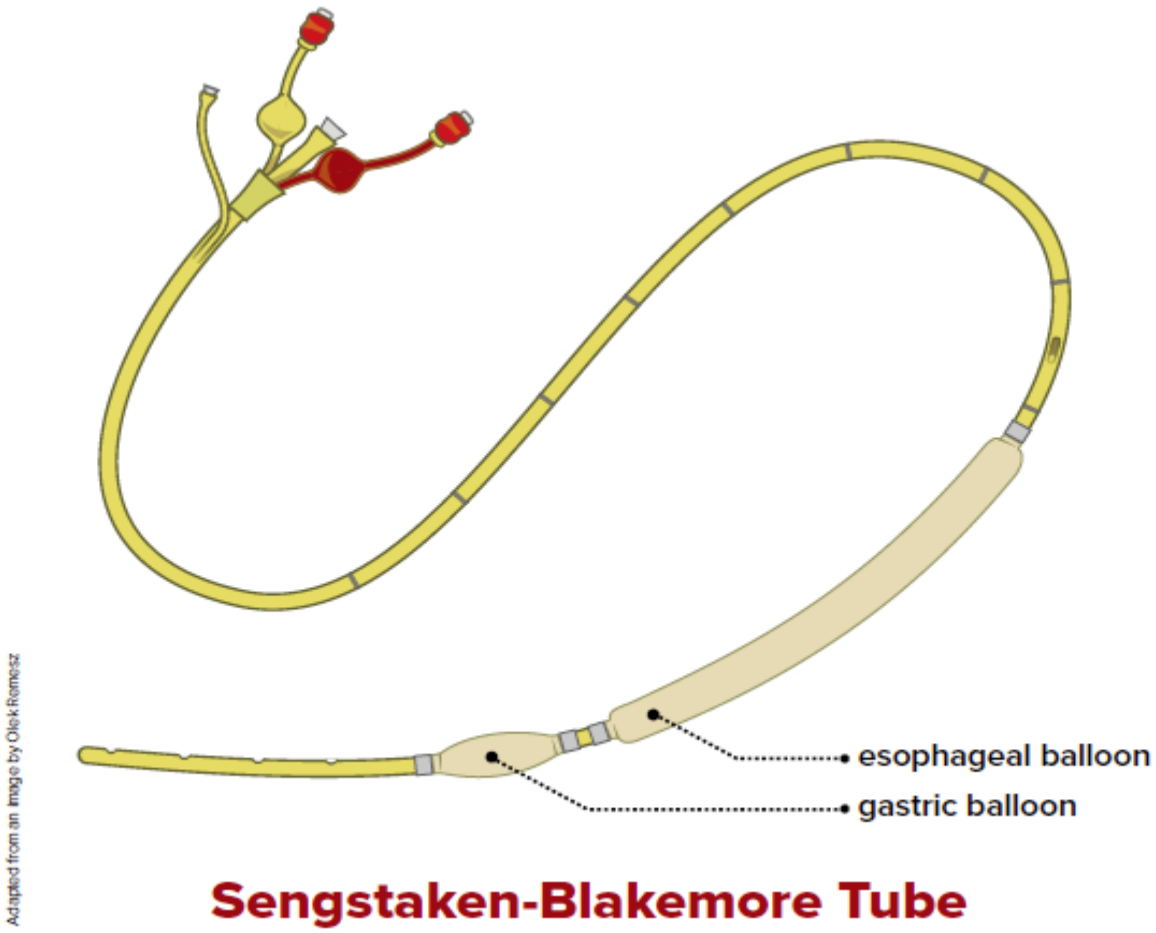


Fig. 8.1.2Sengstaken-Blackmore probe

5) in the filled state, the probe can be held for up to 4-6 hours, then the large balloon is lowered for 20-30 minutes, observe whether the bleeding continues or has stopped, then the balloon is inflated again. It is advisable to put the probe on for 24-72 hours (with breaks after 4-6 hours).

Complications in the case of using the probe are as follows: esophagitis, ulcers, bedsores of the esophagus, respiratory and heart failure, acute splenomegaly. Early relapses of bleeding after the removal of the probe are observed in 20-50 % of patients.

8.2. Conservative treatment

The decrease in portal pressure.

To reduce the pressure in the portal vein system and reduce the risk of bleeding, non-selective beta-blockers are used: propranolol, anaprilin, obsidan 20 mg 3 times a day. The dose should be such that the pulse rate is reduced by 25%

compared to the initial one. Non-selective beta-blockers with prolonged administration can reduce portal pressure by 30-38 %.

Nitrates (nitroglycerin, nitroprusside) reduce portal pressure by 28-30 %. The drugs are used both independently and in combination with others, in particular with pituitrin, since the latter can cause ischemia of the myocardium and internal organs.

The first drug used to reduce portal pressure was pituitrin. Due to its pronounced effect on the central hemodynamics (increased blood pressure, decreased pulse), it is contraindicated in patients with general atherosclerosis,angina, hypertension. Vasopressin and somatostatin have a similar effect to pituitrin. The advantage of the latter is the possibility of a significant reduction in portal pressure and blood flow with minimal side effects.

3. Effects on the blood clotting system. Assign aminocaproic acid, menadione, etamsilat sodium, produce transfusion of fresh-frozen plasma, cryoprecipitate, platelets (2-3 doses), the introduction of a 10% solution of calcium chloride.

4. Antiulcer therapy, including H2-histamine receptors (FAMO, famotidine, quamatel), proton pump inhibitors (omez, omeprazole, rabeprazole) to maximum therapeutic doses. It is advisable to use almagel, rosehip oil, sea buckthorn, antioxidants (vitamin E).

5. Compensation of blood loss — transfusion of a single group of red blood cells (better washed red blood cells) and the fight against hypoxia (oxygen therapy, intravenous administration of actovegin, solcoseril).

6. Prevention of liver failure — transfusion of glucose solutions, vitamin therapy, the appointment of hepatoprotectors (essentiale forte, hepatitis, heptral), antibacterial agents inside to suppress intestinal microflora (neomycin-4-6 g per day, metronidazole-0.25 g 3 times a day for 10-15 days), 10-30 ml of lactulose 3 times a day to a laxative effect. Ornicetil is administered intravenously by drip of 50 mg per 150 ml of saline solution (binds ammonia) up to 6 times a day.

7. Correction of water-electrolyte disorders and maintenance of cardiovascular activity.

Conservative therapy is applied persistently, within a few days after the patient's admission with bleeding, and in the future, if the treatment is ineffective, it should be supplemented with endoscopic sclerotherapy (ES) of EVV and only after that — an emergency operation (only for patients in stage A-B according to Child–Pugh).

There are 2 main methods of administration of the sclerosing agent (thrombovar, varicocide, ethoxysclerol) — intra - and perivasal. In the first case, the sclerosant is injected directly into the EVV, followed by their thrombosis. In the perivasal method, the sclerosant is injected into the submucosal layer around the vein. In this case, the effect is achieved due to edema of the submucosal layer, followed by perivasal fibrosis.

ES allows you to stop bleeding in 70 % of cases after the first and in 90-95 % - after repeated sessions. Relapses of bleeding after ES are observed in 30-60 % of patients. The high frequency of relapses is explained by the fact that ES does not eliminate the cause of the formation of EVV — portal hypertension.

As an independent method of treatment of EVV ES is indicated:

1) patients with an extremely high risk of surgery (decompensated cirrhosis of the liver, jaundice, ascites), in whom conservative therapy is ineffective;

2) persons over 60 years of age with cirrhosis of the liver and severe concomitant diseases;

3) patients who have been repeatedly operated on for PH.

Contraindications to ES are hepatic coma, profuse bleeding in agonizing patients, and severe disorders of the blood clotting system.

An alternative to ES in patients with intrahepatic PH can be the embolization of bleeding veins, which has similar indications. Embolization may involve the left gastric, splenic, and short veins of the stomach, as well as the left gastric and splenic arteries. Embolizing materials are thrombin, ethyl alcohol, hemostatic sponge, spirals.

Complications of this procedure can be bleeding from the liver, bile peritonitis, portal vein thrombosis. The recurrence rate after percutaneous transhepatic embolization of bleeding veins is 43 %.

Contraindications to embolization are considered to be the interposition of the intestine between the liver and the abdominal wall, volume formations in the right lobe of the liver.

In the presence of a special set of instruments, endoscopic ligation of the EVV is performed.

Treatment of ascites

Long-term PH in combination with changes in oncotic pressure in the blood and an increase in the content of aldosterone lead to the development of ascites. This is usually preceded by persistent flatulence. Therapeutic measures for ascites include the restriction of table salt, the appointment of diuretics (furosemide 40-120 mg per day in combination with spironolactone 100-400 mg per day). Treatment of ascites with furosemide and spironolactone should be started with low doses (40 mg of furosemide and 100 mg of spironolactone per day). It is necessary to monitor the amount of fluid taken and the amount of urine released per day, as well as daily weighing of the patient. The dose of furosemide and spironolactone is increased every 3-4 days until the patient's weight loss reaches 400 g per day, and the amount of liquid consumed corresponds to the amount of urine excreted. If this effect cannot be achieved at a dose of furosemide 120 mg, and spironolactone 400 mg per day, then ascites is considered refractory and it is necessary to resort to laparocentesis with the removal of fluid from the abdominal cavity and intravenous administration of albumin solution (6-8 g per 1 liter of removed fluid). Progesterone has a good antialdosterone effect, which is administered in 3 ml of a 0.5% solution intramuscularly 2 times a day for 7-8 days with a torpid edematous ascitic syndrome. With repeated accumulation of fluid, it is necessary to resort to the imposition of peritoneal-venous shunts.

Treatment of hypersplenism

Hypersplenism is a syndrome in which an increase in the spleen is combined with the development of fibroadenia and, as a result, one of the functions of the spleen increases — the capture of red blood cells, granulocytes, platelets from the blood. This leads to a decrease in their peripheral blood (thrombocytopenia,

granulocytopenia, anemia). Treatment of hypersplenism syndrome, which often accompanies cirrhosis of the liver, is carried out with prednisone (20-25 mg per day for 15-20 days, followed by a reduction in the dose to 5 mg every 10 days) under the control of the blood pattern. At the same time, you can prescribe pentoxil 0.2 mg 3 times a day, especially if hypersplenism is manifested by severe granulocytopenia.

The lack of effect of conservative treatment raises the question of surgical intervention (splenectomy).

8.3. Surgical treatment

The main indication for surgery for PH is the treatment and prevention of bleeding from the EVV and VVS, which are the main cause of death. This symptom "owes" almost the entire arsenal of surgical aids used in this disease. It is this complication of PG that explains the increased interest of surgeons in this problem.

Russian scientists play a major role in the development of diagnostic methods and new types of operations for PH (F. G. Uglov,T. O. Koryakina, L. M. Grozdov, M. D. Paciora, B. V. Petrovsky,K. N. Tsatsanidi, A. K. Eramishantsev).

By 1980, the total number of developed operations and their modifications for PH exceeded 100 variants. In the following years, their number increased. However, due to the development and introduction of liver transplantation into clinical practice, the nature of operations has changed due to the emergence of "minimally invasive" endovascular and endoscopic technologies that allow for a short-term hemostatic effect.

The beginning of surgical treatment of PH should be considered the 80-90s of the XIX century, when the idea of decompression of the portal system, i.e. the creation of new ways of blood outflow from the portal vein system, was taken as the basis for surgical intervention. To solve this problem, 2 types of operations were proposed almost simultaneously:

1) omentoparietopexy (suturing the large omentum to the anterior abdominal wall), proposed by the Dutch physician A. S. Talma (1898);

2) the formation of a vascular portocaval anastomosis, developed by the Russian surgeon N. V. Ekk (1877).

The idea of Talma gave rise to a new direction in PH surgery, called organoanastomoses. His supporters used for this purpose not only the omentum, fixing it to the liver, kidney and vena cava, but also such organs as the small intestine, stomach, liver, spleen. They sewed them, depending on the level of the block, to each other, to the lung, diaphragm, muscles, or abdominal wall.

The first operations of partial esophagogastrectomy in the clinic were performed by Professor M. D. Pacior in 1960-1965. However, out of 9 patients, 4 died from the failure of the esophageal-gastric anastomosis sutures. And only since 1968, after the introduction of the invagination method of esophageal anastomosis into the practice of K. N. Tsatsanidi, the immediate results of this operation have significantly improved. According to the world literature of the 1960s and 1970s, great hopes were placed on this operation as the most radical and last attempt to stop bleeding from esophageal and gastric phlebectases, when bypass operations proved impossible.

Since 1980, the attitude to splenectomy as an independent operation in PH has been revised: the indications for its implementation are sharply narrowed and limited to the presence of three situations:

1) segmental PH, when the main manifestation of the disease was profuse bleeding from the VVS as a result of obstruction of the splenic vein;

2) arteriovenous fistula between the splenic artery and the splenic vein, i.e. when the cause of PH is a load of blood volume;

3) infantilism in adolescents with cirrhosis of the liver, when the removal of a large spleen leads to the normalization of the physical parameters of the body.

Operations for PH should be performed in the "cold period", in the interval between bleeding, during the period of the greatest compensation of all body functions. The task of the operation is to reduce the pressure in the portal vein system-hemodynamic correction.

Classification of operations in PH. Currently, 6 groups of operations are used:

1. Operations that promote the removal of ascitic fluid:

1) laparocentesis;

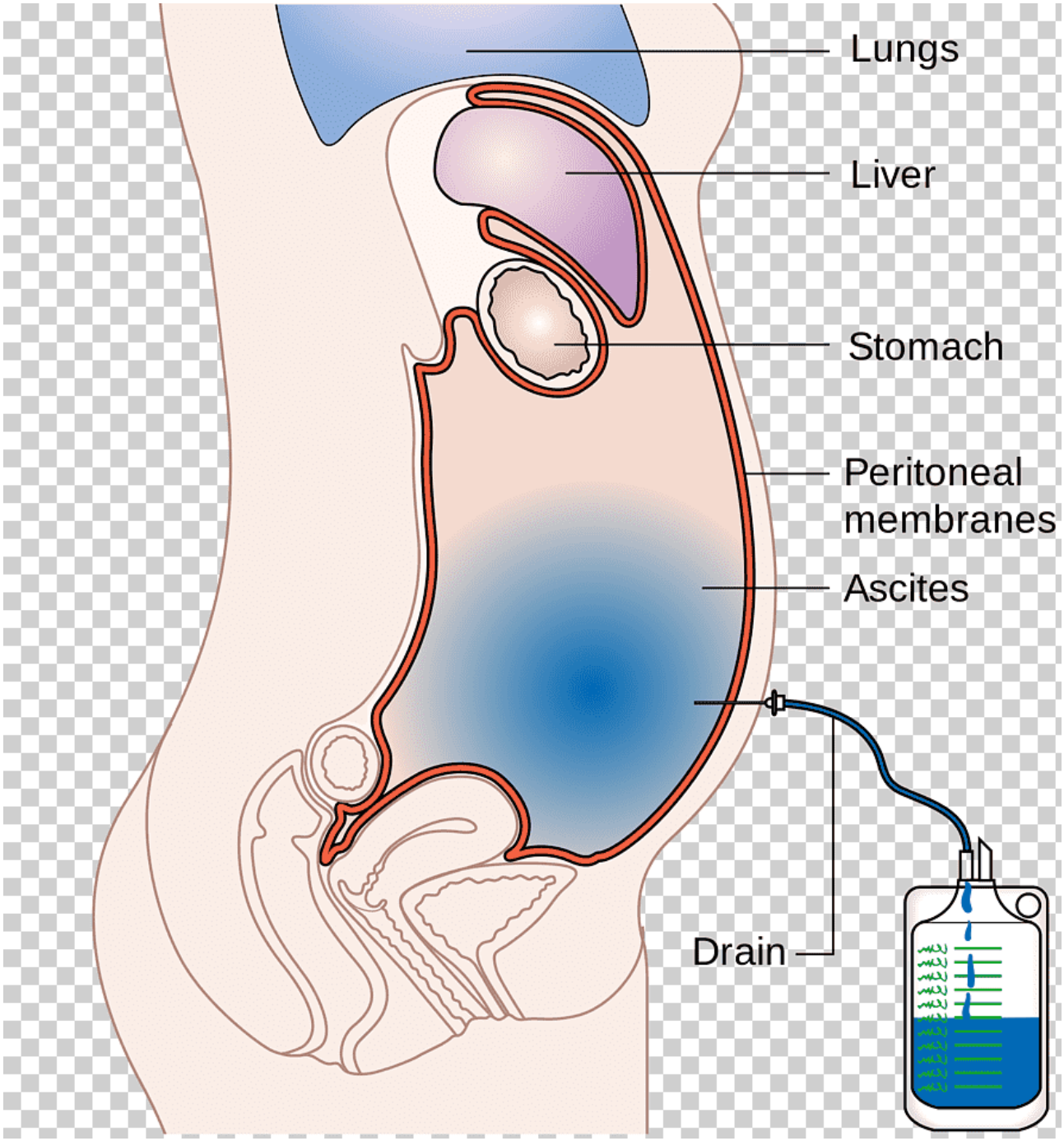


Fig. 8.3.1.Laparocentesis

2) Operation Kalba

- excision of the parietal peritoneum and abdominal muscles in the lumbar triangle for a constant outflow of ascitic fluid;

3) Ruott's operation-isolation of the large saphenous vein of the femur and suturing into the hole in the peritoneum above the inguinal ligament;

4) abdominal-jugular bypass surgery with Levin valve-conducting a tube with a valve under the skin between the jugular vein and the abdominal cavity;

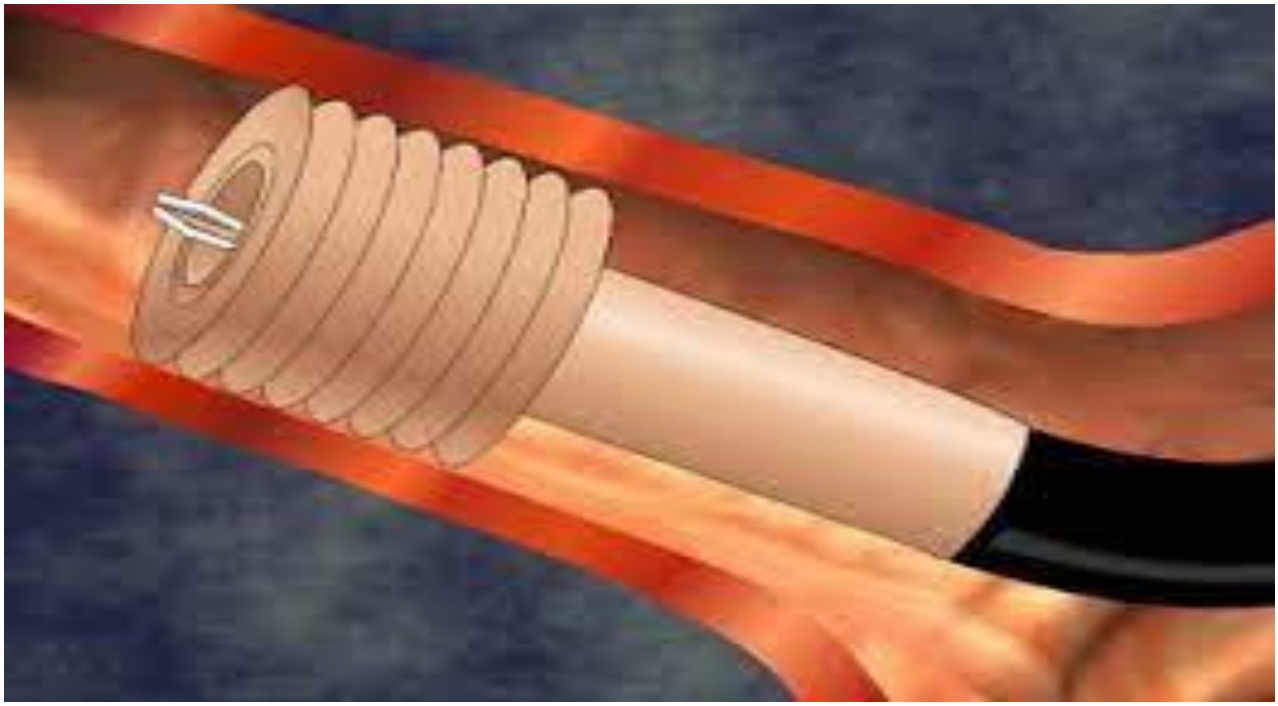


Fig. 8.3.2.Levin valve

5) thoracolization of the decapsulated liver — resection of the left dome of the diaphragm and movement of the liver into the pleural cavity.

2. Operations that disconnect the connection of the veins of the esophagus and stomach with the veins of the portal system:

1) sclerosis of varicose veins;

2) stapler transection of the esophagus-the imposition of a circular suture in the lower third of the esophagus with the help of stitching devices;

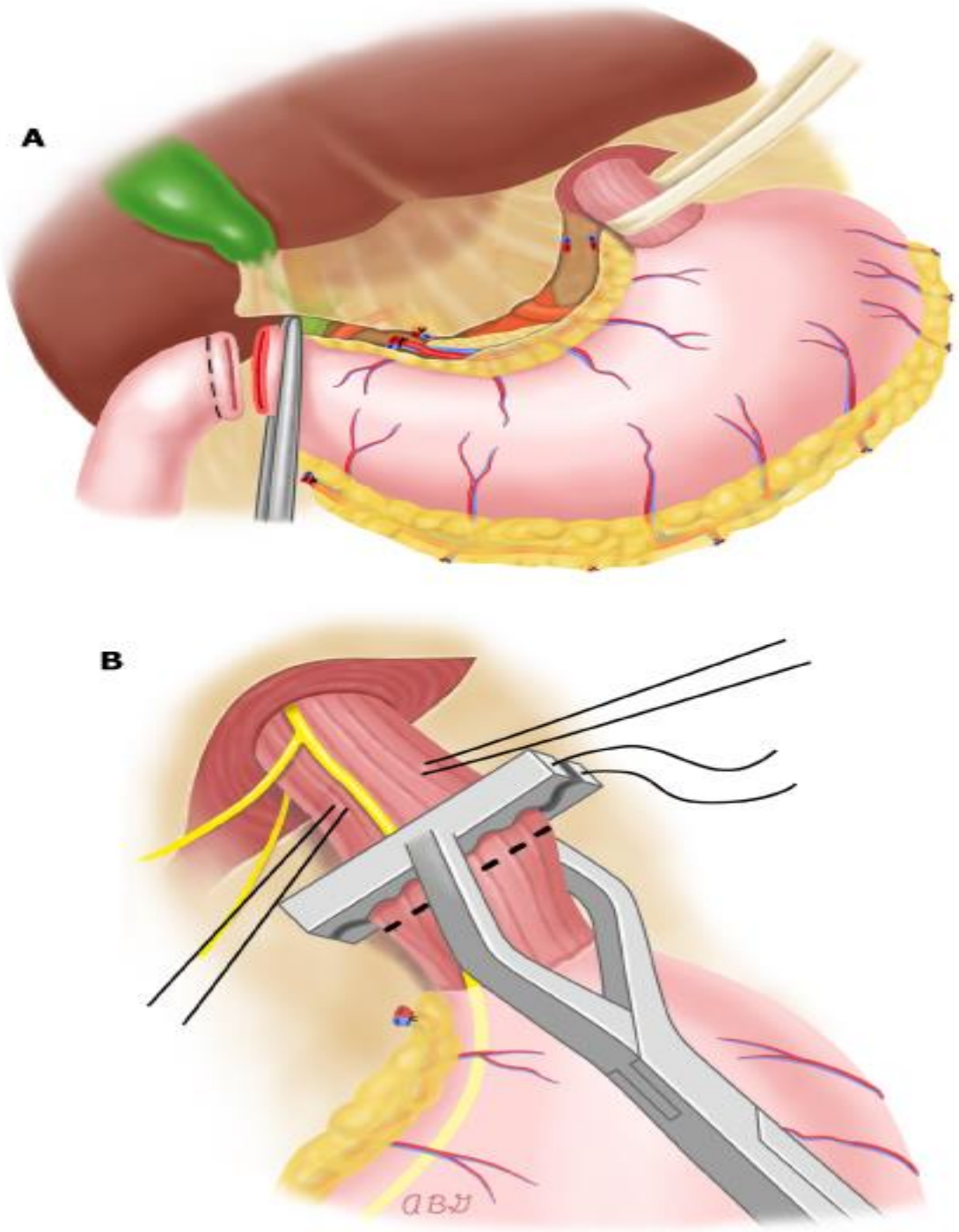


Fig. 8.3.3.Stapler transection of the esophagus

3) operation of Patsiora-gastrotomy and stitching on the part of the mucous membrane of the EVV and VVS. The operation can be combined with ligation and crossing of the splenic artery;

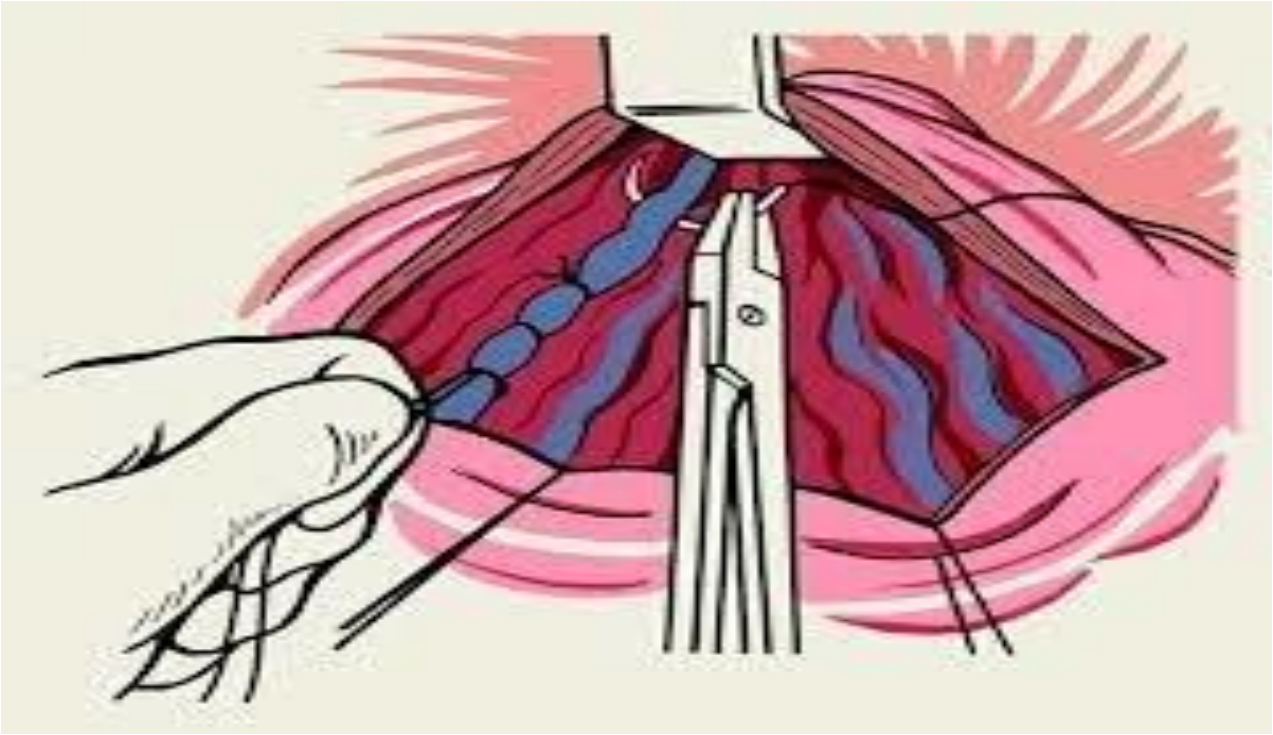


Fig. 8.3.4. Patsiora operation

4) The operation of Tanner, who proposed to cross the stomach in the cardiac department with the subsequent restoration of its integrity.

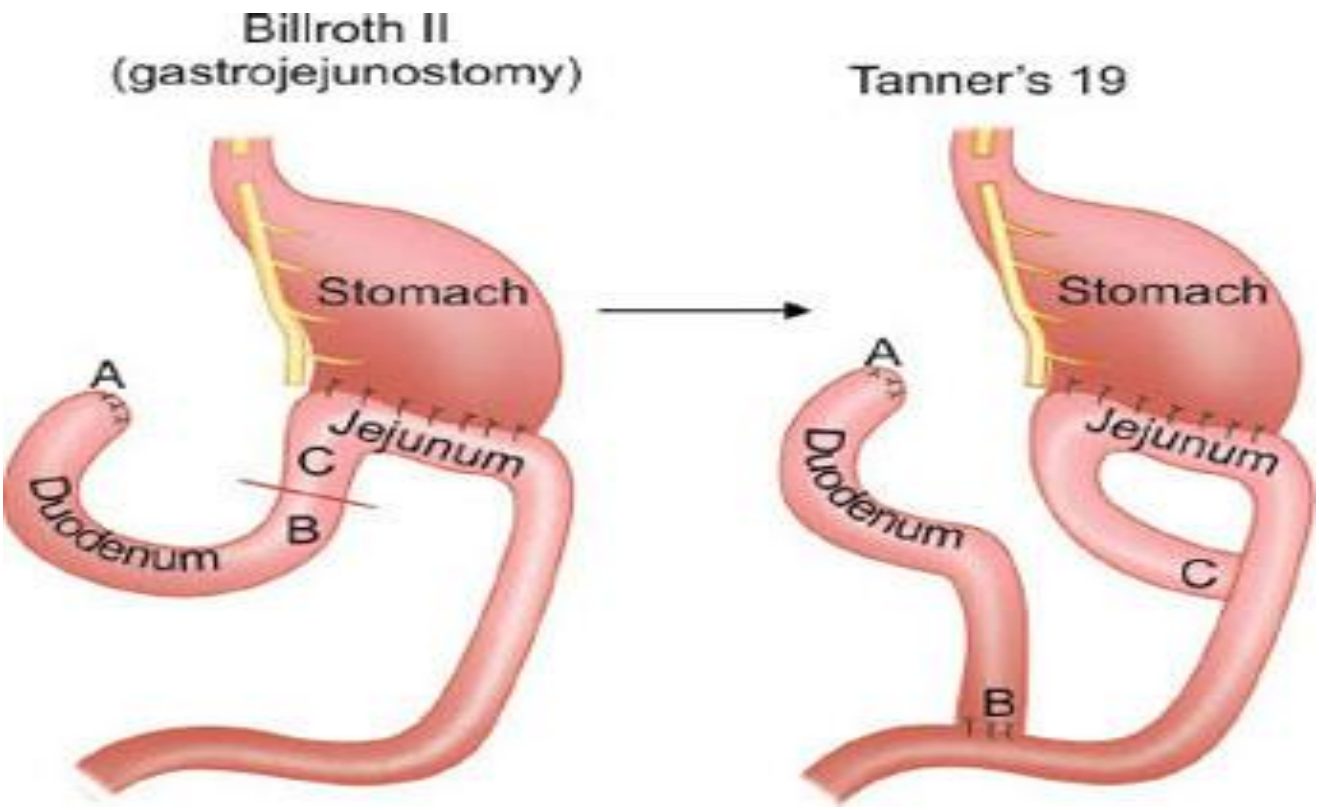


Fig. 8.3.5. Tanner operation

After devascularization of the abdominal esophagus and the cardiac part of the stomach, the latter is crossed in the upper third, after which the integrity of the stomach is restored using crosslinking devices. Surgical intervention is completed with fundoplication and pyloroplasty;

5) Berem-Krail operation — left-sided thoracoabdominal incision with a longitudinal section of the esophagus. Sew and bind the veins from the side of the lumen of the esophagus.

Sclerosing solutions are injected into the venous nodes between the ligatures;

6) Sugiura operation,

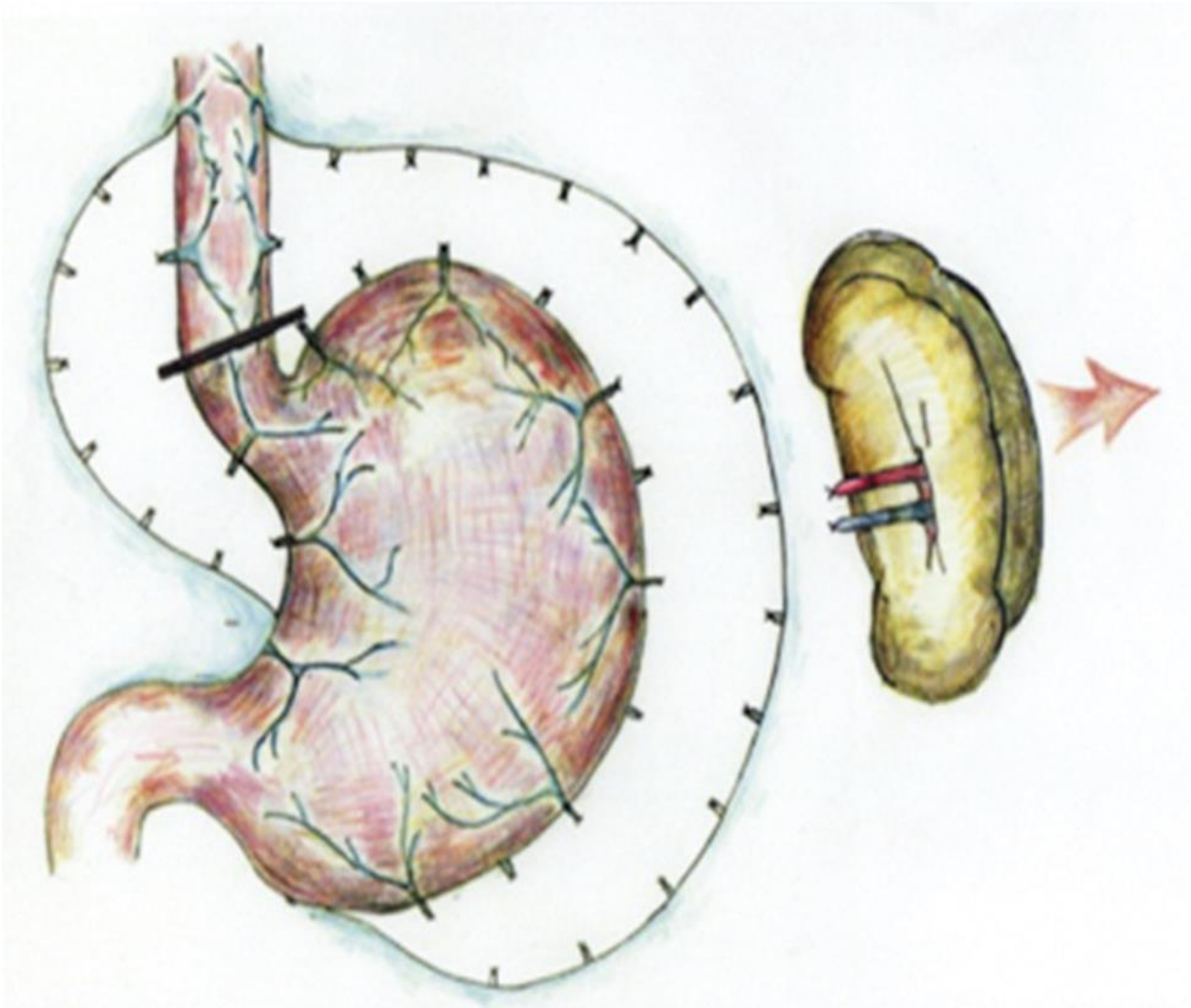


Fig. 8.3.6 Sugiura operation

which is performed in two ways:

- from thoracic and abdominal access. From the thoracic access, the esophagus is devascularized from the cardia to the inferior pulmonary vein and a partial intersection of the esophagus is performed, followed by suturing at the level of the diaphragm. Splenectomy, devascularization of the abdominal esophagus and stomach to the middle of the small curvature, selective vagotomy, and pyloroplasty are performed from the abdominal access;

- from the thoracic access. Abdominal stage performing transdiaphragmatic.

3. Operations that limit blood flow to the portal system:

1) splenectomy (strictly according to the indications);

2) ligation of arteries and veins: left gastric and right gastric omental arteries, inferior mesenteric vein, hepatic artery, splenic artery;

3) x-ray endovascular embolization of the arteries.

4. Operations that create new ways of blood outflow from the portal system-portocaval anastomoses and organoanastomoses (omento-and organopexy):

1) Bogoraz operation-creation of direct portocaval andmesentericocaval anastomoses

2) Whipple–Blackmore operation - formation of distal splenorenal anastomosis;

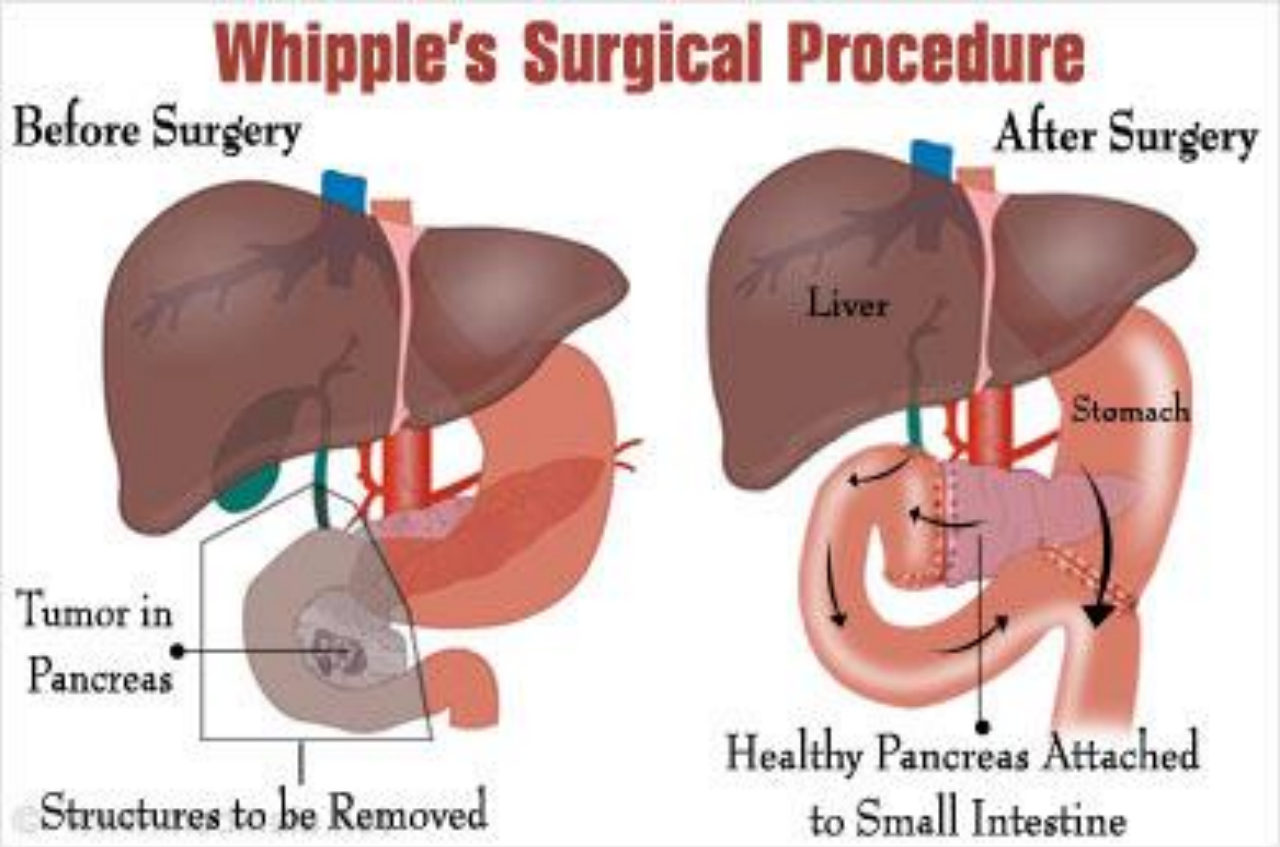


Fig. 8.3.7. Whipple–Blackmore operation

3) omentopexy to the liver, kidney, diaphragm, pleura;

4) organoponico — hepatopancrease, gastropexy, colorautomatic.

During the operations of Bogoraz and Whipple-Blackmore, the liver does not perform its detoxification function, blood from the gastrointestinal tract together with toxic substances flows into the general bloodstream, bypassing the liver, thereby leading to encephalopathy.

5. Operations that improve liver regeneration and thereby improve intrahepatic arterial blood circulation:

1) liver resection;

2) Mallee-Gi operation-periarterialneurectomy of the common hepatic artery;

3) creation of arterioportal anastomoses.

6. Radical operations (removal of tumors, cysts, blood clots, opening of abscesses, extensive resections of the liver together with the tumor, liver transplantation).

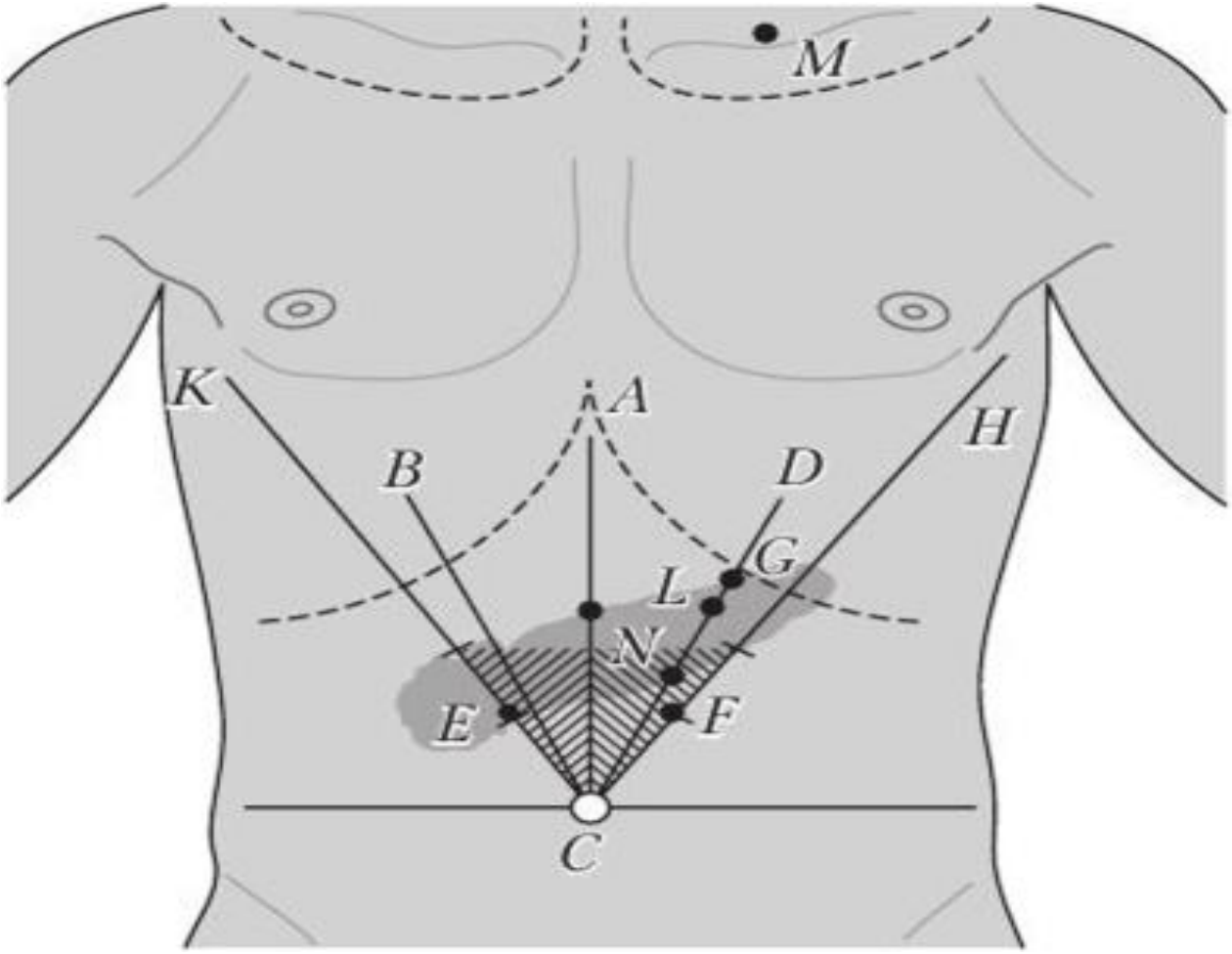


Fig.8.3.8. G=a Malle-Gi point

Currently, of the dozens of proposed options for portocaval anastomoses, the most commonly used are non-selective splenorenal, mesentericocaval, and selective (selectively, dosed "unload" the esophageal-cardiac pool with minimal theft of the hepatic portal blood flow) — distal splenorenal shunt.

The advantage of direct portocaval anastomosis is its effectiveness in preventing recurrent bleeding from the EVV due to a significant reduction in portal pressure. However, the outcomes of the operation are significantly affected by encephalopathy and liver failure. Frequent (30-50 %) deaths after direct portocaval bypass surgery occur not due to repeated bleeding, but due to progressive decompensation of liver function, since blood perfusion through the liver is significantly reduced. The frequency of postoperative encephalopathy reaches 30-40 %, and the 5-year survival rate rarely exceeds 50 %. In this regard, this type of bypass surgery is not used and is of more historical interest.

Distal splenorenal anastomosis meets the conditions of rational surgical management in patients with PH to the greatest extent: portal perfusion through the liver is preserved, selective decompression of the gastroesophageal veins is achieved, the pressure in which is reduced through the short veins of the stomach and the splenic vein.

The frequency of postoperative encephalopathy after splenorenal anastomosis does not exceed 15 %, recurrent bleeding is observed in 12-14% of patients, they are associated with anastomotic thrombosis.

If there are symptoms of hypersplenism or pronounced splenomegaly, a central splenorenal anastomosis with the removal of the spleen is preferred. With a short splenic vein, a splenocaval shunt is used. Both traditional methods of shunt operations and modifications are used: H-prosthesis with an insert made of teflon, dacron or autovene, which makes it possible to significantly simplify the operation, reduce the time required for its execution, and reduce the frequency of shunt thrombosis.

Transjugular portosystemic shunt

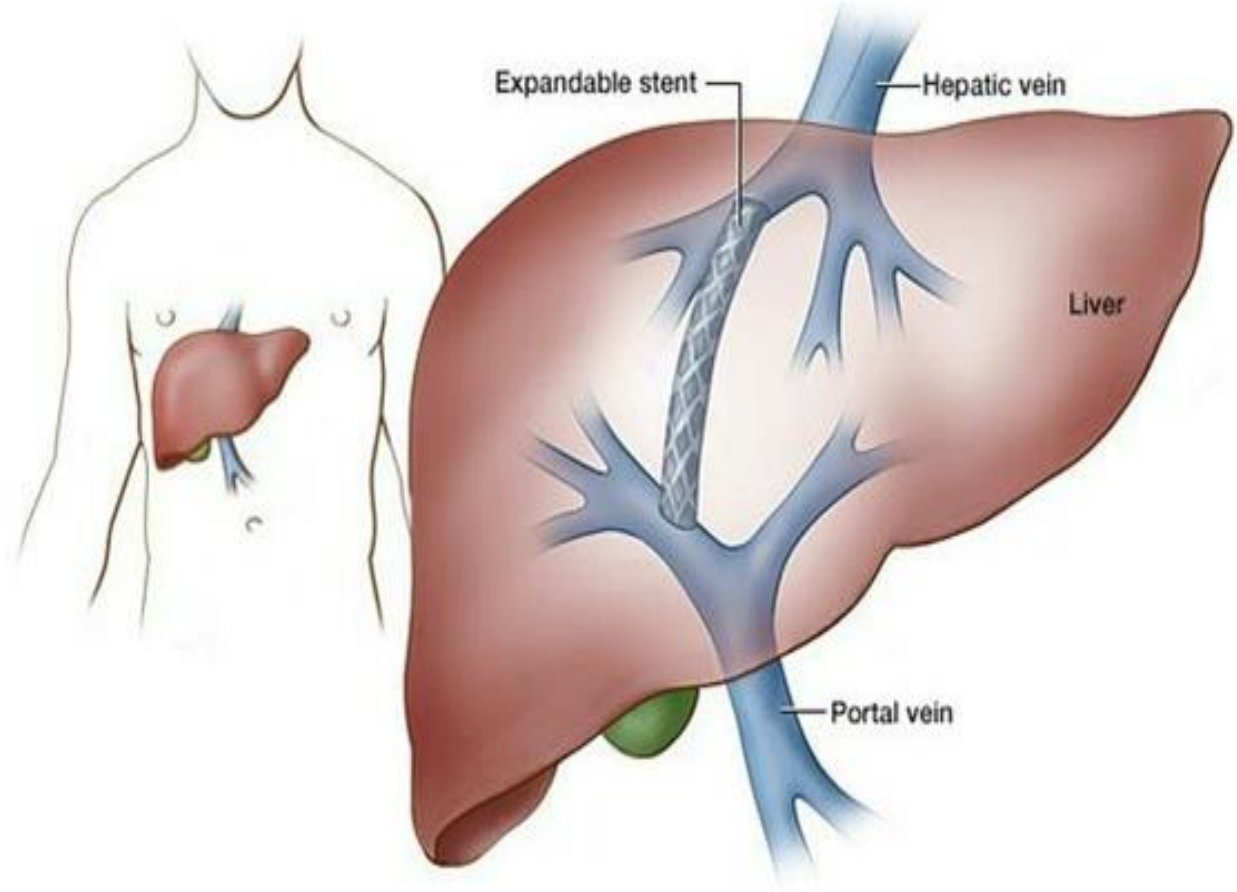


Fig. 8.3.9. Transjugular intrahepatic portosystemic shunt (TIPS)

Fig. 8.3.10. Portosystemic shunts (TIPS)

This intervention is an endovascular method of creating a junction between the hepatic vein and the portal vein branch by installing a stent through the liver parenchyma. Under local anesthesia, the right hepatic vein is catheterized through the right jugular vein in the angiography room. Performed selective cavography.

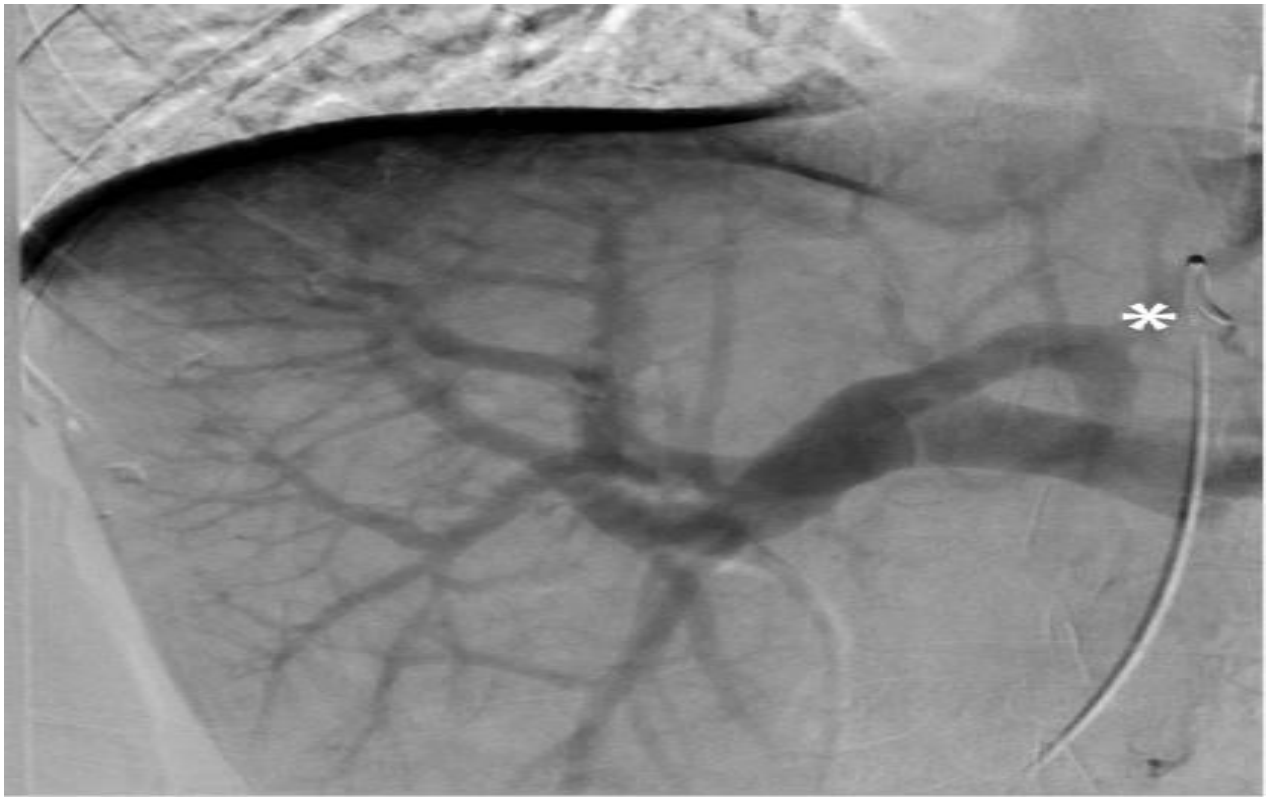


Fig. 8.3.11 Selective cavography

A simulated stiletto-catheter is inserted, a needle is passed through it, with the help of which the liver tissue is punctured until the portal vein system is cannulated. Then, a balloon-catheter is installed along the conductor, with the help of which the channel in the liver tissue is inflated (exposure of 30-40 s). After that, a metal stent is installed in the formed channel. This operation reduces the portal pressure by approximately 50 %. The frequency of recurrent bleeding during the year is from 5 to 19 %.

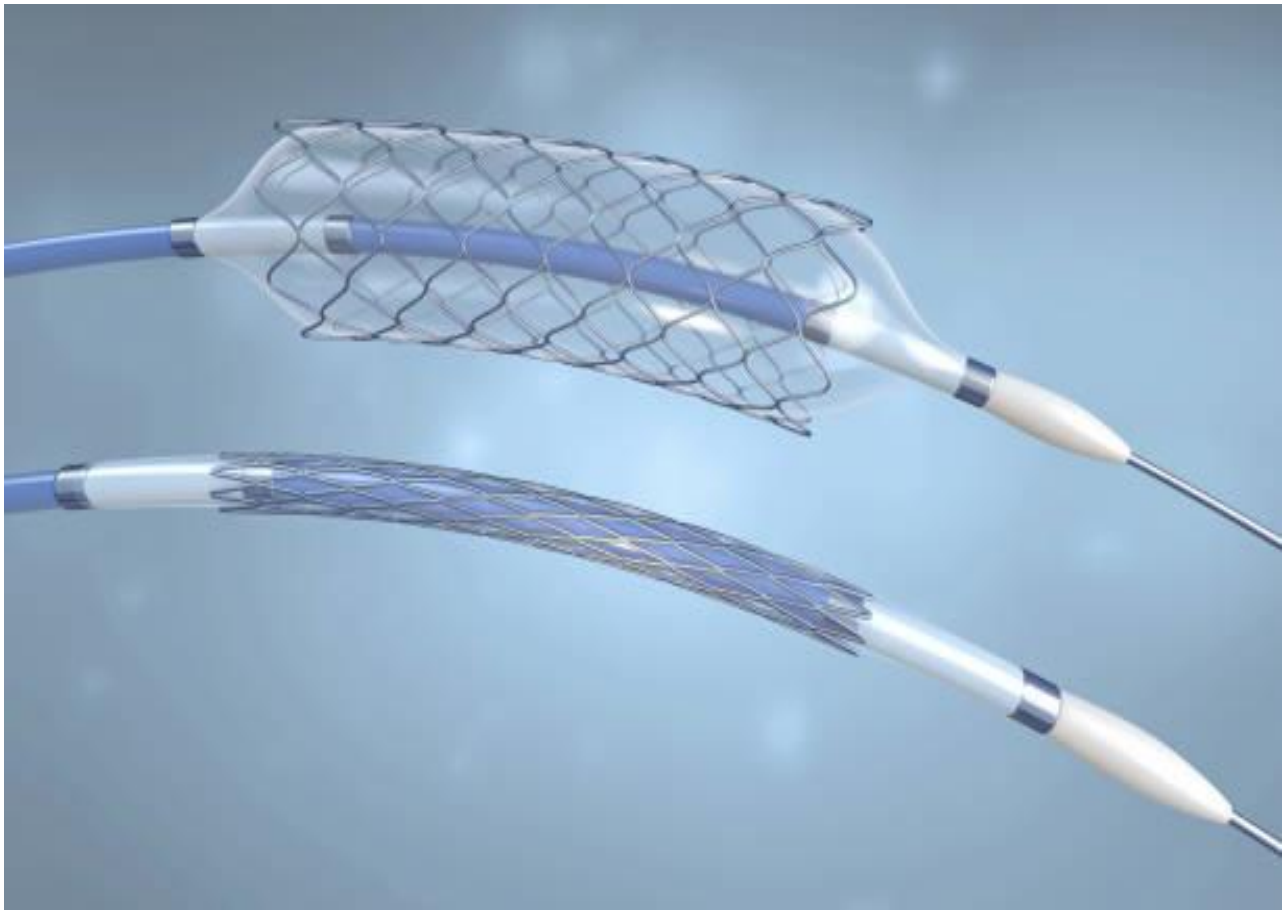


Fig.8.3.12. Balloon-catheter

Despite the wide variety of proposed methods for the treatment of EVV bleeding in patients with intrahepatic PH, the results of treatment, especially in the group of patients with decompensated cirrhosis, remain unsatisfactory. Currently, it is believed that the only effective treatment for these patients is liver transplantation. In terms of preoperative preparation for liver transplantation of patients with bleeding or high risk of its occurrence, both drug therapy and bypass surgery are widely used. Mortality after liver transplantation is 5-7 %, 5-year survival in group B (according to Child–Pugh) — 92 %, in group C-71-73 %.

9. Control questions

1. Anatomy of the portal system.
2. The definition of the syndrome of portal hypertension.
3. Classification of portal hypertension.

4. Clinical manifestations of portal hypertension.

5. Pathogenesis and pathological anatomy of portal hypertension complications.

6. Pathogenesis of bleeding from gastroesophageal phlebectasias.

7. Diagnosis of portal hypertension:

- x-ray examinations
- endoscopic examinations
- Ultrasound studies
- computer, spiral, magnetic resonance, tomographic

methods of research .

8. Clinic of bleeding from varicose veins of the esophagus and stomach.

9. Diagnosis of bleeding from varicose veins of the stomach and esophagus.

10. Treatment of bleeding from varicose veins of the esophagus and stomach.

11. Methods of conservative therapy:

- method of application of the obturator probe.
- medical therapy and nutrition of patients.

12. Surgical methods of treatment of bleeding:

- radical operations
- combined operations
- minimal operations

13. Therapeutic and preventive measures in the postoperative period.

14. The complication of the postoperative period.

15. Causes of ascites.

16. Methods of treatment of ascites.

17. Methods of surgical treatment of portal hypertension.

- splenorenal
- portocaval
- mesenterical anastomoses.

18. Prevention of postoperative complications.

10. Tests

1. What is the normal pressure in the portal system:

a) up to 140-160 mm of water;

b) up to 220-240 mm of water;

c) up to 280-300 mm of water?

2. What is not an etiological factor of PH:

a) cirrhosis of the liver;

b) Budd-Chiari syndrome;

c) thrombosis of the splenic artery?

3. From the confluence of which veins is the portal vein formed:

a) splenic, inferior mesenteric, inferior vena cava;

b) splenic, inferior mesenteric, superior mesenteric;

c) splenic, inferior mesenteric, right common iliac?

4. What is the most frequent source of bleeding in PH:

a) hyoid veins;

b) the veins of the esophagus and the cardiac part of the stomach;

c) the hemorrhoidal veins?

5. What is splenomegaly:

a) hyperfunction of the spleen;

b) spontaneous rupture of the spleen;

c) an increase in the size of the spleen?

6. What is hypersplenism:

a) hyperfunction of the spleen;

b) spontaneous rupture of the spleen;

c) an increase in the size of the spleen?

7. Tanner's operation is:

a) gastrotomy, stitching of the bleeding veins of the esophagus and the cardiac part of the stomach;

b) complete cross-section of the cardiac part of the stomach with its subsequent cross-linking;

c) formation of splenorenal anastomosis.

8. What is the name of the probe to stop bleeding from the VVS:

a) the Kocher probe;

b) the Babcock probe;

c) probe Sengstaken of Blackmore?

9. Operation Pacioli is:

a) gastrotomy, stitching of the bleeding veins of the esophagus and the cardiac part of the stomach;

b) complete cross-section of the cardiac part of the stomach with its subsequent cross-linking;

c) formation of splenorenal anastomosis.

10. During what time is it advisable to use the Sengstaken–Blackmore probe:

a) up to 3 days;

b) up to 4 days;

c) up to 5 days?

Answers: 1 — a; 2-d; 3-b; 4-b; 5-c; 6-a; 7-b; 8-c; 9 — a; 10-a.

11. Situational tasks

1. During esophagogastroscopy, a 47-year-old patient was found to have bleeding from the EVV, which could not be stopped endoscopically. Determine the next steps.

2. A 56-year-old patient with prolonged bleeding hemorrhoids has an enlarged liver and ascites. Make the intended diagnosis and determine further tactics.

3. A 49-year-old patient with gastric bleeding has an enlarged liver, ascites, and a "jellyfish head" on the anterior abdominal wall. Make a preliminary diagnosis and determine the tactics.

4. In a 37-year-old patient with anemia, leukopenia, and thrombocytopenia enlarged liver and spleen, developed ascites. Make a diagnosis and determine what to do next.

5. In a 41-year-old patient, splenoportography revealed a sharp expansion and "amputation" of the splenic vein, splenomegaly. Make a diagnosis and determine the next tactics.

6. A 44-year-old patient was admitted to the hospital as an emergency medical aid. He fell ill suddenly, when after sneezing there was a profuse bloody vomiting. The skin is pale, pronounced weakness. From the anamnesis, it was possible to find out that for many years he has been abusing alcohol. When examining the patient on the anterior abdominal wall, the head of a jellyfish, the liver is not enlarged, the spleen is sharply enlarged, dense. What was the source of the bleeding? What diseases should bleeding be differentiated from?

7. A 45-year-old patient was admitted to the hospital for bloody vomiting. The patient has vomited twice in the last three years. The patient was examined -bleeding from varicose veins of the esophagus was revealed.

8. During a preventive examination, you found that the patient has a pronounced subcutaneous venous network on the anterior abdominal wall. What diseases can cause such changes?

9. A 45-year-old patient was taken to the surgical department with a clinic with gastric bleeding. From the anamnesis, it is known that 4 years ago he was operated on for a gangrenously altered vermiform process. In the postoperative period, hyperthermia up to 38-39 degrees C was observed for a long time, with unspecified abdominal pain. Gastrofibroscopy revealed bleeding from the dilated veins of the esophagus and the cardiac part of the stomach. In the anamnesis, he did not suffer from infectious diseases, he denies bad habits. Determine the possible cause of the developed pathology and research methods to clarify the diagnosis.

10. A 57-year-old patient was taken to the emergency room for emergency medical care. Pronounced shortness of breath, blue lips. On examination: the skin is icteric, earthy, the abdomen is sharply enlarged in size. A positive symptom of the "wave". The liver is not palpable. The spleen is enlarged in size, dense. From the anamnesis, it

was found out that the patient has been abusing alcohol for a long time. What led the patient to this condition? What is the volume of emergency assistance.

11. A 48-year-old patient was operated on for intrahepatic portal hypertension. The operation was performed - the imposition of a direct portocaval anastomosis. In the early postoperative period, there was a change in the mental status of the patient, he became not critical, aggressive, euphoric, excited. In biochemical analyses, a sharp increase in residual nitrogen and serum urea was noted. What led the patient to this condition?

12. During a preventive examination, a 56-year-old patient revealed varicose veins of the esophagus. The general condition of the patient is satisfactory. The patient was examined and diagnosed with intrahepatic portal hypertension-cirrhosis of the liver. What is the minimum amount of care that should be provided to prevent bleeding from dilated esophageal veins?

13. In a 48-year-old patient, endoscopy revealed dilated esophageal veins. Further examination revealed that there is a splenic vein thrombosis (segmental thrombosis), splenomegaly with hypersplenism. The general condition of the patient is compensated. How much surgical treatment should be performed?

14. A 49-year-old patient was admitted to the emergency room with pronounced encephalopathy - agitated, not critical, not oriented in space.He fell ill, according to the relatives of the patient after taking an abundant amount of smoked sausage.Ahistory of liver disease that was accompanied by jaundice. What is the cause of this condition of the patient?

15. A 45-year-old patient suffering from cirrhosis of the liver, portal hypertension,ascites, after performing abdomenocetesis (the amount of fluid is 10 liters), acute intestinal obstruction(clinically) developed and the patient died from the progression of multiple organ failure. What is the cause of acute intestinal obstruction?

Answers:

1. You should immediately put the probe Sengstaken of Blackmore.

2. The patient has the syndrome PH. It is necessary to conduct a full range of diagnostic studies to establish the etiology and form of PH and determine the tactics of adequate treatment.

3. The patient has the syndrome PH. Esophagogastroduodenoscopy should be performed urgently, urgent measures should be taken to stop the bleeding; then additional examination and adequate, pathogenetically justified treatment should be carried out.

4. The patient has PH syndrome, splenomegaly, and hypersplenism. Careful follow-up and subsequent treatment is necessary, depending on the form and etiology of PH.

5. The patient has a splenic vein thrombosis. Splenectomy is indicated after a short follow-up examination and appropriate preoperative preparation.

6. It is most likely that bleeding from varicose veins of the esophagus is due to alcoholic cirrhosis of the liver. It is necessary to exclude a stomach ulcer, 12 p. k., a stomach tumor.

7. Probe obturator for 1-3 days. Replenishment of blood loss. Drugs that reduce portal pressure (nitroglycerin, pituitrin, sandostatin).

8. 1)Cirrhosis of the liver with portal hypertension; 2)A tumor process with a violation of the outflow from the portal system.

9. It is possible that acute appendicitis, suffered 4 years ago, was complicated by acute pileflebitis, followed by a violation of the venous outflow through the portal system, followed by the development of portal hypertension, dilation of the esophagus and stomach veins, and bleeding. To clarify the diagnosis, it is necessary to perform: The safest way to obtain information about the state of the vessels of the portal system is a computer spiral tomogram with intravenous bolus amplification. Ultrasound scanning in combination with Doppler examination provides information about the diameter of the spleen and portal veins, the patency of blood flow in these vessels. Invasive methods of contrasting the vessels of the portal system (direct splenoportography, direct percutaneous transhepaticportography) are not currently used.

10. Alcoholic cirrhosis of the liver, intrahepatic portal hypertension. Ascites. It is necessary to perform an abdominocentesis.

11. The operation of direct portocaval bypass surgery is flawed due to the inability to detoxify from nitrogenous slags in the blood flowing from the small intestine.

12. Endoscopic coagulation of varicose veins of the esophagus.

13. Splenectomy.

14. High protein load led to liver failure.

15. In response to a decrease in intra-abdominal pressure, thrombosis of the intestinal tube vessels occurred, and as a result, dynamic intestinal obstruction

12. References

[1] Sauerbruch T, Schierwagen R and Trebicka J. Managing portal hypertension in patients with liver cirrhosis [version 1; peer review: 2 approved] F1000Research 2018, 7(F1000 Faculty Rev):533 (https://doi.org/10.12688/f1000research.13943.1

[2] Iwakiri Y. Pathophysiology of portal hypertension. Clin Liver Dis. 2014;18(2):281-91

[3] Hirsch S, Guo J, Reiter R, Schott E, Büning C, Somasundaram R, et al. Towards compression-sensitive magnetic resonance elastography of the liver: sensitivity of harmonic volumetric strain to portal hypertension. J MagnReson Imaging. 2014;39(2):298-306

[4] Stefanescu H, Procopet B. Noninvasive assessment of portal hypertension in cirrhosis: liver stiffness and beyond. World J Gastroenterol. 2014;20(45):16811-9

Gerstenmaier JF, Gibson RN. Ultrasound in chronic liver disease. Insights Imaging. 2014;5(4):441-55

[5] Park JK, Saab S, Kee ST, et al. Balloon-occluded retrograde transvenous obliteration (BRTO) for treatment of gastric varices: review and meta-analysis. Dig Dis Sci. 2015;60(6): 1543-1553

[6] Reinglas J, Amjadi K, Petrcich B, Momoli F, Shaw-Stiffel T. The palliative management of refractory cirrhotic ascites using the PleurX ((c)) catheter. Can J GastroenterolHepatol. 2016;2016: 4680543.

[7] de Franchis R, Baveno VI Faculty: Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. J Hepatol. 2015; 63(3): 743–52

[8] Orloff MJ: Fifty-three years' experience with randomized clinical trials of emergency portacaval shunt for bleeding esophageal varices in Cirrhosis: 1958-2011. JAMA Surg. 2014; 149(2): 155–69

[9] Luo X, Wang Z, Tsauo J, et al.: Advanced Cirrhosis Combined with Portal Vein Thrombosis: A Randomized Trial of TIPS versus Endoscopic Band Ligation Plus Propranolol for the Prevention of Recurrent Esophageal Variceal Bleeding. Radiology. 2015; 276(1): 286–93

[10] Qi X, Tian Y, Zhang W, et al.: Covered TIPS for secondary prophylaxis of variceal bleeding in liver cirrhosis: A systematic review and meta-analysis of randomized controlled trials. Medicine (Baltimore). 2016; 95(50): e5680.

[11] Khanna R, Sarin SK. Non-cirrhotic portal hypertension—diagnosis and management. J Hepatol 2014; 60:421–441

[12] Dhiman RK, Saraswat VA, Valla DC, et al. Portal cavernomacholangiopathy: consensus statement of a working party of the Indian National Association for Study of the Liver. J Clin Exp Hepatol 2014;4(Suppl 1):S2–S14.

[13] Luo X, Nie L, Zhou B, et al. Transjugular intrahepatic portosystemic shunt for the treatment of portal hypertension in noncirrhotic patients with portal cavernoma. Gastroenterol Res Pract 2014;2014:659726.

[14] Qi X, He C, Guo W, et al. Transjugular intrahepatic portosystemic shunt for portal vein thrombosis with variceal bleeding in liver cirrhosis: outcomes and predictors in a prospective cohort study. Liver Int 2016; 36:667–676.

[15] Fares N, Robic MA, Péron JM, et al. Transjugular intrahepatic portosystemic shunt placement before abdominal intervention in cirrhotic patients with portal hypertension: lessons from a pilot study. Eur J GastroenterolHepatol. 2018;30:21–6