

ULTRASOUND DIAGNOSTICS OF EXTRAHEPATIC PORTAL HYPERTENSION IN PATIENTS WITH CHRONIC PANCREATITIS

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Expressed fibro-infiltrative changes of the pancreas, parapancreatic and paravasal fiber in chronic pancreatitis (CP) determine the development of extravasal compression (EVC) or portal vein thrombosis system that is the cause of the development of extrahepatic portal hypertension (EPH).

The frequency of EPH upon lesions of distal parts of the pancreas is from 7-20% of cases [12], up to 45% of cases by S. M. Weber et al. [18] and 80% — by P. A. Banks et al. In CP with a primary lesion of pancreatic head, involving veins mesentericoportal axis, frequency ranges from 13.2% (according to P. Bernades et al.) to 40% of cases [7, 8]. E. L. Avdyei notes the development of EPH in 47.8% of cases of proximal CP, and according to S. P. Chikoteeva and I. K. Boyko, EVC and portal vein thrombosis system were identified in 72.7% of patients [1, 4].

Unlike patients with primary EPH whose clinical manifestations of the syndrome are associated with sudden bleeding of esophageal varices and/or stomach [2], in patients with CP clinic syndrome of EPH is not so expressed. Bleeding episodes in patients with portal EPH origin due to CP, ranging from 5% to 18% of the cases [1, 8, 9, 10, 11, 12, 15, 16, 17]. According to J. R. Izbicki et al., recurrent bleeding episodes were not fixed [8], and T. R. Heider et al. marked them in no more than 4% of patients [12]. A number of authors do not describe a single death from acute blood loss [1, 6, 8, 9, 12, 15].

In spite of rare bleeding episodes, the importance of preoperative diagnosis of EPH is not in doubt. As shown by J. R. Izbicki et al., among patients with EPH, when performing pancreatectomy, had significantly bigger intraoperative blood loss,

respectively, these patients often needed blood transfusions, increased operation time and recovery period [8].

Currently, the main methods of EPH diagnosis in pathology of pancreatoduodenal zones are non-invasive, having high sensitivity and specificity. Ultrasound study using color duplex scanning helps assess the condition of the pancreas and anatomically interconnected with it organs, detect compression or portal vein thrombosis, visualize extended venous collaterals.

The aim of our work is the definition of the diagnostic capabilities of color duplex scanning in the detection of EPH in patients with CP.

M. D. Patsiora proposed classification of EPH according to clinical manifestations and state of portohepatic and central circulation in patients with liver cirrhosis. Author allocated 1 step (compensated), which was characterized by compensated portohepatic circulation, development of splenomegaly with or without hypersplenism. In stage 2 (subcompensated) she revealed varicose veins of the esophagus and stomach bleeding or without bleeding, splenomegaly, and violations portohepatic circulation. Upon EPH decompensation (stage 3), except for violations of portohepatic circulation, changes occur in central hemodynamics (hyperdynamic circulation type) [3].

We examined patients with CP, complications of EPH, identified two stages of EPH: EPH in stage 1 (EPH-1) hemodynamic changes in the portal system (thrombosis, compression) led to the development of portoportal venous collaterals designed to bypass the obstruction area and restore circulation of the liver; in stage 2 (EPH-2), which, in fact, was subcompensated, increased portal pressure contributed to the development of portosystemic and intraorgan collaterals with clinical manifestation of the syndrome.

Materials and methods. The work is based on the analysis of complex examination of 94 CP patients, in 61 (65%) pancreatitis was complicated by EPH, and 33 (35%) CP patients had no signs of increased portal pressure. Among the patients studied the men (78%) of working age prevailed — average age $47 \pm 9,9$ years (from 22 to 72).

In 82 (87%) patients the cause of CP was the nutritional factor, in particular, alcohol and fatty foods, in 9 (10%) patients had onset of the disease associated with an attack of acute pancreatitis on the background of gallstone disease and in 3 (3%) patients with CP it developed after pancreatic injury.

Surgery on the pancreas upon CP was performed in 78 patients. Distribution of patients by type of intervention and the presence of signs of EPH is shown in Table 1.

Table 1

Distribution of patients with CP depending on the type of surgery and the presence of EPH

Type of surgery	CP patients with EPH	CP patients without EPH
	Number of patients abs., %	
Operation of internal drainage (formation pancreatojejunal anastomosis, pancreatocystojejunal anastomosis, cystopancreatic anastomosis)	27 (52%)	15 (56%)
Resection of the pancreas with a transversal intersection	14 (27%)	7 (26%)
pancreatoduodenal resection	3 (6%)	3 (11%)
Beger operation	6 (12%)	2 (7%)
distal resection	5 (10%)	2 (7%)
Resection of the pancreas with a transversal intersection (Frey operation)	10 (19%)	5 (18%)
Overall	51 (66%)	27 (34%)

Results. In 64% of cases (60 patients) CP affecting mainly the pancreatic head dominated. CP with fibro-inflammatory changes in the body-tail or the tail of the pancreas was diagnosed in 28 patients (30%), and lesion of all the parts was noted in 6 (6%) patients.

Ultrasound examination in B-mode showed that among patients with EPH calculary CP was significantly more frequently diagnosed — in 48% of cases, 75% noted the development of pancreatic hypertension, in 58% of patients an increase in the anterior-posterior part of the head of pancreas was more than 40 mm ($p < 0.05$).

We found no significant difference between patients with and without EPH in presence of pancreatic pseudocysts, enlargement of intra- and extrahepatic bile ducts, fiber parapancreatic infiltrative changes ($p>0.05$). Postnecrotic pancreatic cysts occurred among 44 (72%) CP patients with EPH and in 25 (76%) patients without signs of increased portal pressure. In comparing the linear sizes of the pseudocysts, it was observed that among patients with EPH half (48%) had cyst size from 40 mm to 80 mm, and 19% — pseudocysts size from 80 mm to 140 mm. In patients with EPH in substantially equal proportions — in 39% of cases we diagnosed cyst size from 20 mm to 40 mm, and in 32% — from 80 mm to 140 mm. Among CP patients with EPH pancreatic head lesion was seen in 69% of cases and in 55% of patients without EPH, lesion of the distal part, including a combination of body and tail, was found in 28% of patients with EPH and in 33% without EPH. Fibro-inflammatory changes in all the parts of the pancreas were diagnosed among 3% of patients with EPH and in 12% without EPH.

One of the diagnostic criteria for hepatic portal hypertension is a form of enlargement of the spleen. In our work, this trend was not identified. Increasing area of the spleen for more than 40 cm² we noted in 63% of patients with EPH and in 48% non-EPH ones ($p>0.05$). Increasing area of spleen was comparable to the increase in regional lymph nodes, which suggested greater proliferative inflammation. Increasing area of the spleen for 10% or more than 100% of normal was significantly more frequent among patients with portal vein thrombosis system ($p<0.05$).

Among 61 patients with EPH there were 40 patients with EPH-1, in which the results of preoperative examination and intraoperative data diagnosed portoportal venous collaterals. In 21 patients with EPH-2 ultrasonography and computed tomography diagnosed portosystemic collaterals, except portoportal, while esophagogastroscopy identified extended intraorganic collaterals.

To study the assessment of central hemodynamics in patients with EPH-2, we conducted echocardiography. The results showed that in patients with CP complicated by the development of EPH hyperdynamic circulation type was not

formed, there were no changes in the central hemodynamics (heart volume indicators, contractility of left ventricular were consistent with normal values).

In this paper, the development of EPH in 24 (60%) patients with EPH-1 was due to portal vein EVC system, a combination of EVC with thrombosis was diagnosed in 4 (10%) patients, thrombosis of the main tributaries of the portal vein system was detected in 5 (13%) patients, and in 7 (17%) portal hemodynamics was not changed.

Among patients with EPH-2 patients with thrombosis of the main tributaries of the portal vein dominated in 14 (66%) cases, one half was represented by persons with combination of thrombosis and EVC. In 7 (34%) cases, the cause of EPH-2 was the EVC portal venous system.

Among the 23 patients with diagnosed thrombosis, occlusive thrombosis was detected in 21 observations, of which 6 (26%) had all the portal vein main tributaries thrombosed, thrombosis of the portal vein confluence area developed in 2 (9%) patients, thrombosis of the portal vein trunk — 5 (22%) cases, superior mesenteric vein — 1 (4%) observation, splenic vein (segmental) — 7 (30%) cases. In 2 (9%) mural thrombosis localized: in one case — in the trunk of the superior mesenteric vein, in the second case — in the portal vein with the transition to the superior mesenteric vein.

In patients without signs of increased portal pressure the portal system veins were completely passable in 58%. Among the 42% of patients with compression of the portal veins 86% recorded moderate increase in linear velocity of blood flow without significant turbulence in blood flow on average 0.5 m/s.

Visualization of venous collaterals is the most important criterion of diagnosis of EPH. Our results showed that the incidence of venous collaterals visualization depends on the stage of EPH. In the stage of EPH-1 frequency of visualization of collaterals according to the results of duplex ultrasonography was 45%, while in patients with EPH-2 collaterals were determined in 76% of patients. In 100% of cases during duplex ultrasonography venous collaterals were diagnosed among patients with thrombosis in the portal system. Development of venous (portoportal) collaterals

occurs within a single "anastomotic field" (L. L. Gugushvili). We identified the following trend: the level of obstruction in the portal system depends on the location of the lesion of pancreas in CP. Thus, we conducted the diagnosis of venous collaterals considering the pancreatic lesions. Upon CP with proximal lesion of mesentericoportal axis, we visualized collaterals in thicker hepatoduodenal ligament (pericholedochal plexus) in the gallbladder wall, around the head of the pancreas, in the anatomical course of the portal and superior mesenteric vein (right-sided). Upon splenic vein thrombosis due to lesions of the tail of pancreas, portoportal collaterals were determined in projection of anatomical stroke of splenic vein in gastrolial area at the gate and at the poles of the spleen (left-sided localization).

In CP patients with a primary lesion of pancreatic head in 24% of cases we detected collaterals of right-sided localization and in 21% — a combination of right- and left-sided localizations. Patients with changes in the distal parts of the pancreas in equal proportion (26%) had left-sided localization of collaterals and a combination of right- and left-sided.

To determine the diagnostic significance of ultrasound, we performed a comparison of examination results with intraoperative data. In the initial stage of EPH-1 accuracy of ultrasound in detecting venous collaterals is low and equal to 53%, a sensitivity is only 47% and specificity — 83%. With increasing changes in the portal system, the role of ultrasound in the diagnosis of EPH rises too. Thus, upon EPH-2 diagnostic accuracy of the method in detecting venous collaterals is 79%, sensitivity — 77%, specificity — 100%.

We conducted a quantitative analysis of hemodynamic parameters in the portal system in patients with thrombosis in the portal system and EVC. We analyzed the following parameters: veins diameter, linear velocity of blood flow on passable plot with laminar blood flow and the volume of blood flow velocity, which is presented in the Table 2.

Table 2

**Comparative analysis of venous hemodynamics in patients with EPH and CP
without increasing portal pressure**

Vessel/blood flow parameter	CP patients with thrombosis in the portal system (n=23) M±m	CP patients with EPH due to EVC (n=30) M±m	CP patients without EPH (n=25) M±m
Diameter of portal vein on extrahepatic segment (mm)	5,9±1,4*	11,9±1,4	12,2±0,3
Linear velocity of blood flow through the portal vein on the extrahepatic segment (cm/sec)	9,9±2,4*	20,5±2,4	19,7±1,5
Volume of velocity of blood flow through the portal vein on extrahepatic segment (L/min)	0,272±0,08*	0,498±0,08	0,480±0,02
Superior mesenteric vein diameter (mm)	6,1±0,8	9,1±0,5	8,9±0,2
Linear velocity of blood flow through the superior mesenteric vein (cm/sec)	12,9±2,0*	15,8±2,0*	20,1±1,7
Volume of velocity of blood flow through the superior mesenteric vein (L/min)	0,160±0,03*	0,237±0,03	0,277±0,03
Splenic vein diameter (mm)	3,8±0,8	7,8±0,8	7,3±0,2
Linear velocity of blood flow through the splenic vein (cm/sec)	8,7±2,1*	17,6±2,1*	21,2±1,8
Volume of velocity of blood flow through the splenic vein (L/min)	0,087±0,02*	0,225±0,02	0,228±0,05

Note: * — statistically significant difference ($p < 0.05$) in indices of the portal hemodynamics upon comparison of groups of patients with EPH and without increasing portal pressure.

A statistically significant change in the hemodynamic parameters of the portal vein in patients with venous thrombosis reveals reducing linear and space velocities indices ranging from 37% to 58% when compared to patients without EPH. Among patients whose EPH is not associated with EVC of portal venous system, significant differences from the group of patients without EPH are defined in terms of the linear velocity of blood flow — a tendency towards a decrease in speed indices through the superior mesenteric and splenic veins on average by 15%. Blood flow velocity indices correspond to the values in patients with no evidence of an increased portal pressure.

Thus, in patients with EVC of portal vein system venous collaterals compensates for violations of portal blood flow. In patients with thrombosis, whose majority in our study had EPH subcompensated stage, development of collateral blood flow does not provide full compensation of portal blood supply.

The second part of our study aimed to evaluate the parameters of portal blood flow after the operation on the pancreas about CP. We believed that the improvement of portal hemodynamics was associated with an increase of the linear velocity of blood flow by eliminating EVC of portal venous system. Among the complications in the early postoperative period, which may have affected the portal hemodynamics, we examined the development of acute postoperative pancreatitis, failure of biliodigestive and pancreatodigestive anastomoses. Formed infiltration or limited presence of fluid accumulation in the area of intervention may lead to the development of EVC of portal vein system. The frequency of postoperative complications depending on the type of operation is shown in Table 3.

Table 3

The complication rate of early postoperative period in patients with CP

	PDR		Beger operation		Distal resection		Frey operation		Internal drainage		Overall n=78
	I n=3	II n=3	I n=6	II n=2	I n=5	II n=2	I n=10	II n=5	I n=27	II n=15	
BDA failure	1 33%	1 33%	2 33%						2 7%		6 7,7%
PEA failure						1 50%	1 10%		2 7%	1 7%	5 6%
Acute p/o pancreatitis	1 33%	1 33%	2 33%	—	—	—	4 40%		—	1 20%	9 11,5%
Infiltration	1 33%	1 33%	2 33%	—	1 20%	—	2 20%	2 40%	2 7%	1 7%	12 15%
Fluid accumulation	1 33%	1 33%	1 17%	—		1 20%	1 10%	—	2 7%	2 13%	9 11,5%

Note: I — patients with EPH; II — patients without EPH.

Ultrasonographic evidence of acute postoperative pancreatitis were: increasing size of the pancreas, blurred front circuit along with a decrease in echogenicity of the parenchyma, which was found in 9 patients. Among patients with acute postoperative pancreatitis, in 78% of cases (7 patients) we noted the presence of effusion in the packing bag, ascites. According to our data, infiltrative changes and fluid

accumulation in the area of intervention were significantly more ($p<0,05$) among patients diagnosed with complicated postoperative period.

To estimate blood flow in the portal vein system, on the 3rd and 7th day after surgery, all patients underwent ultrasound duplex scanning.

When analyzing the data in Tables 3 and 4, it was noted that on day 3 after the operation frequency of EVC registration depended on the presence of complications of early postoperative period. On day 7 after surgery EVC of portal vein system was significantly more common among patients who had undergone surgery of internal drainage ($p<0.05$), which indicated the failure of venous decompression.

Table 4

Comparative evaluation of the frequency of EVC portal venous system in patients with CP complicated by EPH, depending on the type of surgery

Ultrasono- graphy data	PDR (n=3)	Beger operation (n=6)	Distal resection (n=5)	Frey operation (n=10)	Internal drainage (n=27)
Day 3	1 (33%)	2 (33%)	1 (20%)	4 (40%)	15 (56%)
Overall	8 (33%)				15 (56%)
Day 7	—	1 (17%)	—	1 (10%)	8 (30%)
Overall	2 (8%)				8 (30%)

As shown in Table 5, EVC of portal venous system in the early postoperative period is also recorded among patients with no evidence of an increased portal pressure. In most patients, we noted normalization of portal blood flow on day 7 and only in one case EVC of portal vein by infiltration that developed on the background of postoperative acute pancreatitis was preserved.

Table 5

Comparative evaluation of the frequency of EVC of portal venous system in patients with CP without signs of EPH

Ultrasono- graphy data	PDR (n=3)	Beger operation (n=2)	Distal resection (n=2)	Frey operation (n=5)	Internal drainage (n=15)
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Day 3	1 (33%)	—	1 (50%)	1 (20%)	4 (27%)
Overall	3 (25%)				4 (27%)
Day 7	—	—	—	—	1 (7%)
Overall	—				1 (7%)

Quantitative indicators of portal blood flow (linear and volumetric flow velocity) we counted on day 7 after surgery on the pancreas, which we compared with preoperative indices and variant of surgery (Fig. 1a, b, c).

When comparing with preoperative indices, increase and normalization of indices of linear velocity of blood flow in the portal vein system were observed in patients who underwent resection of the pancreas with transversal intersection (pancreatoduodenal resection, Beger operation, distal resection). After Frey surgery increase of blood flow velocity was recorded only in the splenic vein, in other vessels indices corresponded to the preoperative data. Among patients who had undergone surgery of internal drainage, speed indices through the portal and superior mesenteric veins have not been changed, and in the splenic vein there was a significant decrease of blood flow velocity.

Among the 23 patients with thrombosis in the portal system we operated on 14 patients (61%). In half of the cases resection of the pancreas in different amounts was performed, and 7 patients (50%) had surgery of internal drainage of duct system and/or cysts of the pancreas. It should be noted that in two patients the indications for distal pancreatectomy with splenectomy, along with fibro-inflammatory changes in the tail of pancreas, included splenic vein thrombosis with veins fundus of the stomach.

Among all the patients with thrombosis in the portal system after surgery we revealed no progression of thrombosis with extension to the veins traversed before the operation, and no recanalization of previously thrombosed veins occurred. They had dilated venous collaterals, which localization corresponded to the level of occlusion of mesentericoportal trunk. In the analysis of quantitative indicators of portal hemodynamics, significant differences with indices before surgery were

identified. Reducing volume of the portal blood flow by reducing the linear flow velocity was maintained after surgery in the same range.

Discussion. EVC or portal vein thrombosis leads to the development of EPH in patients with CP. In our study and in the literature, EVC has etiological factor — compression of the portal venous system by the enlarged head of pancreas or pseudocysts [8, 18]. Violation of portal venous drainage system leads to increased pressure in the veins of prestenotic segment and development of collateral venous network. We have identified two stages of EPH in patients with CP, based on the degree of development of collateral circulation. In the initial stage (EPH-1) upon violation of the outflow in the portal system, portoportal collateral network designed to bypass the narrowed area is expanding. Most of this group consisted of patients with EVC of portal venous system (60%), and 17% according to the color duplex scanning did not have registered hemodynamic changes in the portal system. With further increase in portal pressure there is the formation of portosystemic and intraorganic collateral circulation. Under subcompensation (EPH-2), we observed clinical manifestations of EPH in the form of varicose veins of esophagus and stomach, and increased risk of bleedings of portal genesis. In our paper we show that among patients with CP we did not observe decompensated EPH.

Thus, color duplex scanning is the exact method to identify not only the degree and extent of changes in pancreatoduodenal zone in patients with CP, but the hemodynamic changes in the portal system caused by the pathological process. Moreover, this method is prescribed for the dynamic control of portal hemodynamics in the postoperative period.

References

1. Авдей Е. Л. Клиника, диагностика и лечение синдрома вторичной портальной гипертензии : автореф. дис.... канд. мед наук : 14.00.27 / Е. Л. Авдей ; Бел. гос. ин-т усовершенствования врачей. — Минск, 1993. — 16 с.
2. Ерамишанцев А. К. Первичная внепеченочная портальная гипертензия и ее хирургическое лечение : автореф. дис. ... докт. мед. наук / А. К. Ерамишанцев. — М., 1983. — 41 с.
3. Пациора М. Д. Хирургия портальной гипертензии / М. Д. Пациора. — Ташкент : Медицина, 1984. — 319 с.
4. Чикотеев С. П. Синдром внепеченочной портальной гипертензии при проксимальном хроническом панкреатите / С. П. Чикотеев, Е. А. Ильичева, И. К. Бойко // Сиб. мед. журн. — 1998. — № 13 (2). — С. 14–15.
5. Ahluwalia A. S. Extrahepatic portal hypertension following abdominal surgery / A. S. Ahluwalia, J. J. Mazza, S. H. Yale // WMJ. — 2007. — Vol. 106, No 5. — P. 266–269.
6. Alcoholic chronic pancreatitis with simultaneous multiple severe complications — extrahepatic portal obliteration, obstructive jaundice and duodenal stricture / S. Kakizaki, T. Hamada, T. Yoshinaga [et al.] // Hepatogastroenterology. — 2005. — Vol. 52, No 64. — P. 1274–1275.
7. Chronic pancreatitis / H. G. Beger, M. Buechler, H. Ditschuneit, P. Malfertheimer. — Berlin ; Heidelberg ; New York : Springer, 1990. — 574 p.
8. Extrahepatic portal hypertension in chronic pancreatitis: an old problem revisited / J. R. Izbicki, E. F. Yekebas, T. Strate [et al.] // Ann. Surg. — 2002. — Vol. 236, No 1. — P. 82–89.
9. Isolated gastric varices resulting from iatrogenic splenic vein occlusion : report of a case / S. Tsuchida, Y. Ku, T. Fukumoto [et al.] // Surg. Today. — 2003. — Vol. 33, No 7. — P. 542–544.
10. Marn C. S. CT diagnosis of splenic vein occlusion: imaging features, etiology and clinical manifestation / C. S. Marn, K. A. Edgar, I. R. Francis // Abdom. Imaging. — 1995. — Vol. 20, No 1. — P. 78–81.

11. Moossa A. R. Isolated splenic vein thrombosis / A. R. Moossa, M. A. Gadd // World J. Surg. — 1985. — Vol. 9, No 3. — P. 384–390.
12. The natural history of pancreatitis-induced splenic vein thrombosis / T. R. Heider, S. Azeem, J. A. Galanko, K. E. Behrns // Ann. Surg. — 2004. — Vol. 239, No 6. — P. 876–879.
13. Septic thrombosis of the portal vein due to peripancreatic ligamental abscess / M. Wakisaka, H. Mori, H. Kiyosue [et al.] // Eur. Radiol. — 1999. — Vol. 9, No 1. — P. 90–92.
14. The significance of sinistral portal hypertension complicating chronic pancreatitis / G. H. Sakorafas, M. G. Sarr, D. R. Farley, M. B. Farnell // Am. J. Surg. — 2000. — Vol. 179, No 2. — P. 129–133.
15. Significance of splenic vein thrombosis in chronic pancreatitis / A. K. Agarwal, R. Kumar, S. Agarwal [et al.] // Am. J. Surg. — 2008. — Vol. 196, No 2. — P. 150–153.
16. Splenic and portal venous obstruction in chronic pancreatitis. A prospective longitudinal study of a medical-surgical series of 266 patients / P. Bernades, A. Baetz, P. Lévy [et al.] // Dig. Dis. Sci. — 1992. — Vol. 37, No 3. — P. 340–346.
17. Thavanathan J. Splenic vein thrombosis as a cause of variceal bleeding / J. Thavanathan, C. Heughan, T. M. Cummings // Can. J. Surg. — 1992. — Vol. 35, No 6. — P. 649–652.
18. Weber S. M. Splenic vein thrombosis and gastrointestinal bleeding in chronic pancreatitis / S. M. Weber, L. F. Rikkerts // World J. Surg. — 2003. — Vol. 27, No 11. — P. 1271–1274.
19. Yamashita Y. Clinical study of transient portal vein stenosis induced after pancreatic head resection / Y. Yamashita, H. Ryo, K. Takasaki // Surg. Today. — 2004. — Vol. 34, No 11. — P. 925–931.

Ultrasound diagnostics of extrahepatic portal hypertension in patients with chronic pancreatitis

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Key words: extrahepatic portal hypertension, chronic pancreatitis, sonography, internal drainage of duct or pseudo-cyst, resection of pancreas

Aim is to evaluate the potential of the color duplex scanning in revealing of the portal hypertension in patients with chronic pancreatitis (CP).

Materials and methods. 94 patients with chronic CP were examined. Pancreatitis was complicated by extrahepatic portal hypertension (ECPH) in 61 patients (65%), and 31 patients (35%) had no signs of the portal pressure rising.

Results. Investigations in B-regime shown that chronic calculous pancreatitis was revealed in patients with ECPH in 48%, development of pancreatic hypertension — in 75%, increase of the pancreatic head up to more than 40 mm — in 58% of patients. We didn't reveal statistically significant differences in pseudo-cysts in the pancreas, extension of extra- and intrahepatic ducts and infiltrative changes in parapancreatic cellular tissue in patients with and without ECPH.

ECPH development in 31 (51%) patients was preconditioned by an extravasal compression of veins of portal system, combination of extravasal compression with thrombosis was found in 11 (18%) patients, thrombosis of the magistral veins of portal system was revealed in 12 (20%) patients and hemodynamics wasn't changed in 7 (11%) patients.

Resections turned out to be preferable operations for recovery of portal circulation. Increase and normalization of portal circulation were found after transversal section of the pancreas (the Beger operation, pancreaticoduodenal resection, distal resection of the pancreas). A tendency to normalization of the blood flow was observed after the Frey operation.

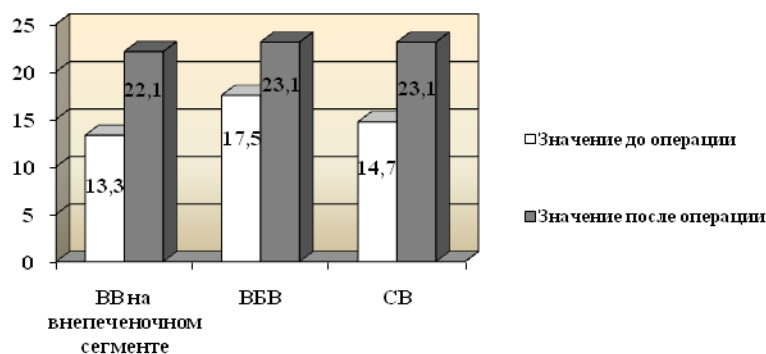


Fig. 1a. The magnitude of BFV (cm/sec) in the veins of the portal system in patients undergoing resection of the pancreas with a transversal intersection. BB — portal vein; BBV — superior mesenteric vein; CB — splenic vein.

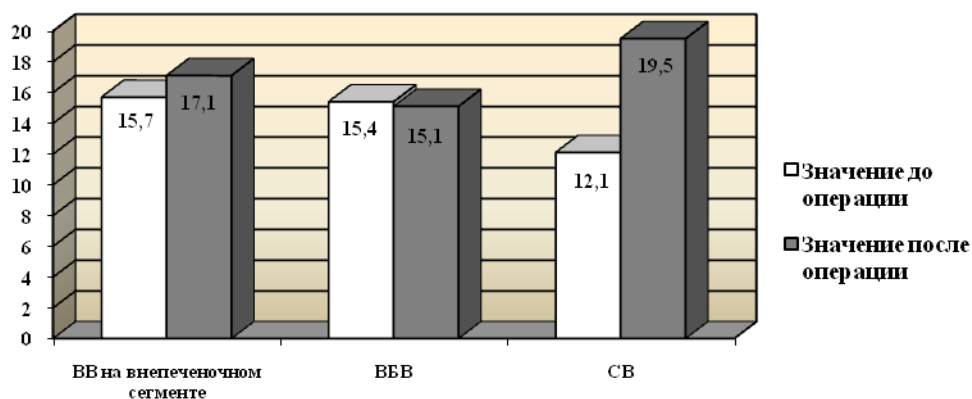


Fig. 1b. The magnitude of BFV (cm/sec) in the veins of the portal system in patients undergoing Frey operation.

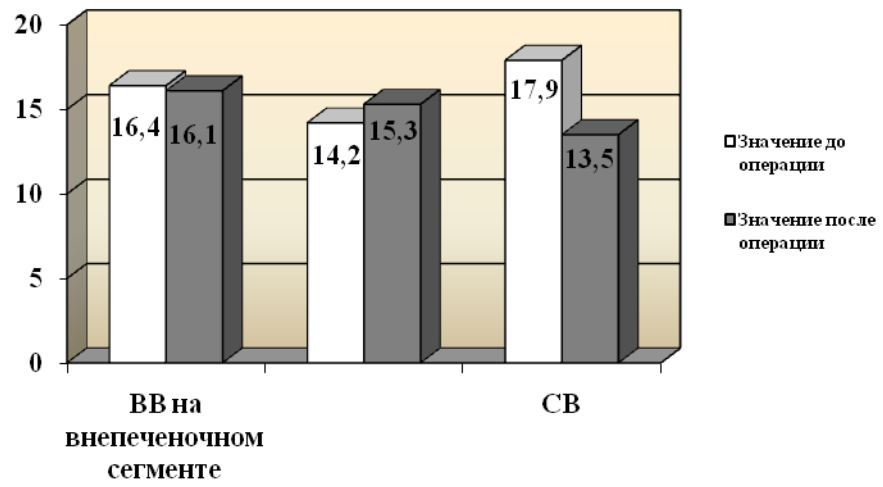


Fig. 1c. The magnitude of BFV (cm/sec) in the veins of the portal system in patients undergoing surgery of the internal drainage.