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PERFUSION COMPUTER TOMOGRAPHY IN PORTAL HYPERTENSION OF VARIOUS GENESIS: FEATURES OF INTRAHEPATIC HEMODYNAMICS

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Abstract
The study of intrahepatic hemodynamic features remains an urgent problem. This is due to the influence of the degree of change of this factor on all the main criteria of severity of the course and prognosis of the pathologic process, the frequency of functional and hemodynamic complications, as well as on the choice of optimal treatment tactics and, accordingly, the duration of its effectiveness. One of the newest and promising options for studying the features of intrahepatic hemodynamics is perfusion computed tomography. Three groups of patients were included in the study taking into account the level of portal blood circulation block: intrahepatic postsinusoidal block in liver cirrhosis - 63 patients, intrahepatic presinusoidal block in liver fibrosis - 10 patients and subhepatic block in extrahepatic portal hypertension - 13 patients. Also, to analyze the nature of changes in perfusion indices relative to normal values, the fourth group consisting of healthy individuals - 24 people - was included in the study. In total, the study was conducted in 110 people aged 18 to 67 years. The analysis of the results obtained in liver PCI showed statistically significant differences of indices in any variant of portal blood flow block from those in healthy individuals, except only arterial fraction in liver fibrosis. The obtained values of liver perfusion fractions showed a high degree of linear dependence of the nature of changes in intrahepatic blood flow in relation to normal parameters depending on the etiology of portal hypertension. Thus, the etiological factor of portal hypertension development affects the peculiarities of changes in hepatic perfusion in relation to the normal values, with a paradoxical increase in the portal fraction with insignificant changes in arterial blood flow and perfusion index being characteristic of liver fibrosis; in liver cirrhosis, reliable changes with a decrease in portal blood flow with a compensatory increase in arterial inflow and perfusion index were noted; in extrahepatic portal hypertension, a mini-infarction of intrahepatic blood flow was verified.

Keywords: CT liver perfusion, liver cirrhosis, liver fibrosis, portal hypertension, arterial perfusion, portal perfusion, hepatic perfusion index.

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Introduction

The study of intrahepatic hemodynamic features remains an urgent problem. This is due to the influence of the degree of change of this factor on all the main criteria of severity of the course and prognosis of the pathological process, the frequency of functional and hemodynamic complications, as well as on the choice of optimal treatment tactics and, accordingly, the duration of its effectiveness. In particular, the following points should be noted. Firstly, the degree of intrahepatic circulation disorder affects the functional status of hepatocytes, respectively, the state of liver perfusion will affect the effectiveness and long-term effectiveness of hepatoprotective therapy. Secondly, decrease in liver perfusion together with the progression of liver dysfunction determines the development of decompensation of cirrhosis and the decision of the issue of radical treatment - liver transplantation. Thirdly, the features of hemodynamic restructuring affect the severity of the course of portal hypertension and, accordingly, the frequency of complications such as bleeding from varicose veins of the esophagus and stomach, splenomegaly and hypersplenism, vascular component of edema-ascitic syndrome. Fourth, compensated indices of hepatic perfusion expand the choice of surgical methods of esophageal-gastric bleeding prophylaxis. Thus, if in decompensated cirrhosis these techniques are strictly limited to endoscopic or endovascular interventions, the compensated or subcompensated course of the pathological process allows us to consider variants of traditional surgeries characterized by more effective and long-term prognosis in terms of bleeding prevention (portosystemic shunt, dissociation surgeries).

Another aspect of maintaining the relevance of studying the peculiarities of liver hemodynamics is the continued development and introduction of new methods of radiology diagnostics. Among them, one of the newest and promising options for studying the features of intrahepatic hemodynamics is perfusion computed tomography.

Materials and Methods

Three groups of patients were included in the study taking into account the level of portal circulation block: intrahepatic postsinusoidal block in liver cirrhosis (LC) - 63 patients, intrahepatic presinusoidal block in hepatic fibrosis (HF) - 10 patients and subhepatic block in extrahepatic HF (EHF) - 13 patients. Also, to analyze the nature of changes in perfusion indices relative to normal values, the fourth group consisting of healthy individuals - 24 people - was included in the study. In total, the study was conducted in 110 people aged from 18 to 67 years. PCT study included determination of liver perfusion parameters: arterial fraction (AF), portal fraction (PF) and hepatic perfusion index (HPI). In each patient the mentioned parameters were studied in all anatomic segments of the liver - in 8 points. That is, 880 parameters of AF (ml/100ml/min), PF (ml/100ml/min) and PI (%) were obtained for 110 patients studied.

All patients underwent perfusion computed tomography (PCT) on a wide-detector computed tomography scanner “Aquilion One - 640” GENESIS version (Canon Medical Systems, Japan). The following scanning protocol was used to perform CCT: native examination - 120 kV, 200 mAs; dynamic examination -100 kV, 100-450 mAs (depending on the patient's weight). The thickness of the liver perfusion scan was 19.2 mm. Data processing of the study was performed using Liver Perfusion program, with determination of the following quantitative indices: arterial hepatic perfusion (AF); portal hepatic perfusion (BF); hepatic perfusion index (HPI). Total liver perfusion

(TLP) was calculated by summing arterial and portal perfusion indices. After obtaining a series of TCT images, data processing was performed on the Vitrea workstation in off-line mode. The quality of images by respiration and noise of heterogeneity heterogeneity of tissue density parameters was necessarily corrected. For quantitative analysis, the basic anatomical areas of perfusion parameters calculation were fixed - aorta, portal vein, spleen and several areas of interest in the liver parenchyma. The peak of contrast (the highest values of unit H) in the aorta was reached at the 20th second (±2 s), the peak of portal vein contrast at the 32nd second (±3 s), the peak values of spleen contrast were detected slightly earlier - at the 28th-30th second (regardless of the patient's age and physique).

Result and Discussion

The analysis of the results obtained at the liver SCT showed statistically reliable differences of the indices at any variant of portal blood flow block from those in healthy subjects, except only arterial fraction at FP (Table 1).

Table 1. CT perfusion of the liver in portal hypertension of different genesis

Pathology	AF			PF			PI		
	Significance	σ	m	Significance	σ	m	Significance	σ	m
Norm	34,4	11,9	0,9	154,9	26,2	1,9	18,2	4,2	0,3
Liver cirrhosis	41,4	15,7	0,7	146,3	68,4	3,1	23,8	9,2	0,4
t-criterion to normal	6,32 (p<0,001)			2,40 (p<0,05)			10,94 (p<0,001)		
Liver fibrosis	35,1	15,1	1,8	169,4	42,2	5,0	21,1	6,1	0,7
t-criterion to normal	0,35 (p>0,05)			2,73 (p<0,01)			3,72 (p<0,001)		
t-criterion to LC	3,29 (p<0,01)			3,97 (p<0,001)			3,25 (p<0,01)		
EHH	49,5	17,0	1,7	107,0	56,3	5,5	35,6	15,5	1,5
t-criterion to normal	8,04 (p<0,001)			8,20 (p<0,001)			11,23 (p<0,001)		
t-criterion to LC	4,46 (p<0,001)			6,22 (p<0,001)			7,49 (p<0,001)		
t-criterion to LF	5,89 (p<0,001)			8,40 (p<0,001)			8,62 (p<0,001)		

The obtained values of liver perfusion fractions showed a high degree of linear dependence of the nature of changes in intrahepatic blood flow in relation to the normal parameters depending on the etiology of PG (Fig. 1).

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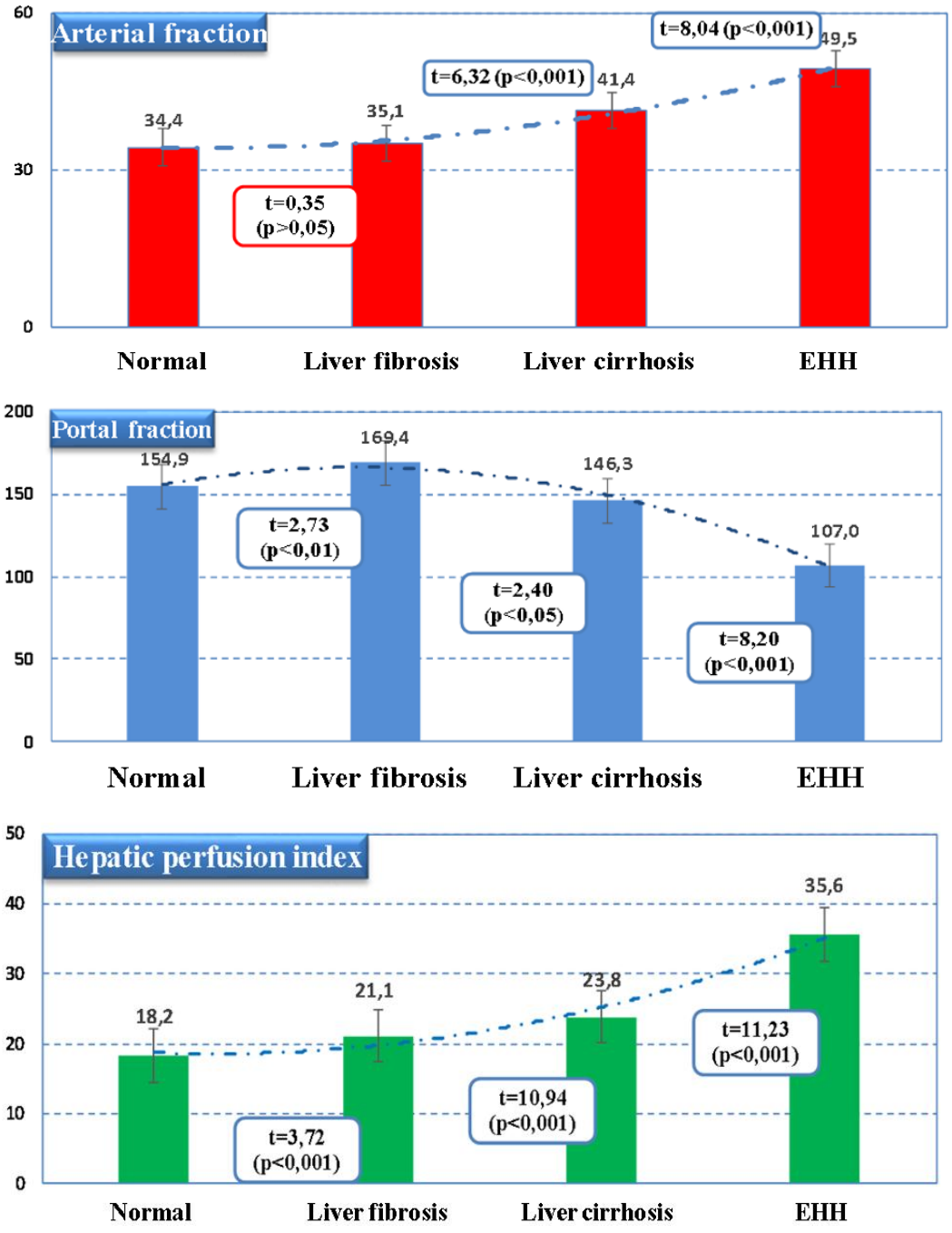

Figure 1: CT perfusion indices of the liver.

Thus, in CKD there was determined a decrease in portal blood flow (PF) up to 146.3±3.1 ml/100ml/min with the norm - 154.9±1.9 ml/100ml/min ($p<0.05$). Corresponding to the decrease in portal fraction, there was a compensatory increase in arterial blood flow (AF) up to 41.4±0.7 with the norm of 34.4±0.9 ml/100ml/min ($p<0.001$). These changes caused the increase of perfusion index (PI) with its growth in relation to the norm due to the increase of arterial and decrease of portal fraction up to 23.8±0.4% (normal 18.2±0.3; $p<0.001$) (Fig. 2-3).

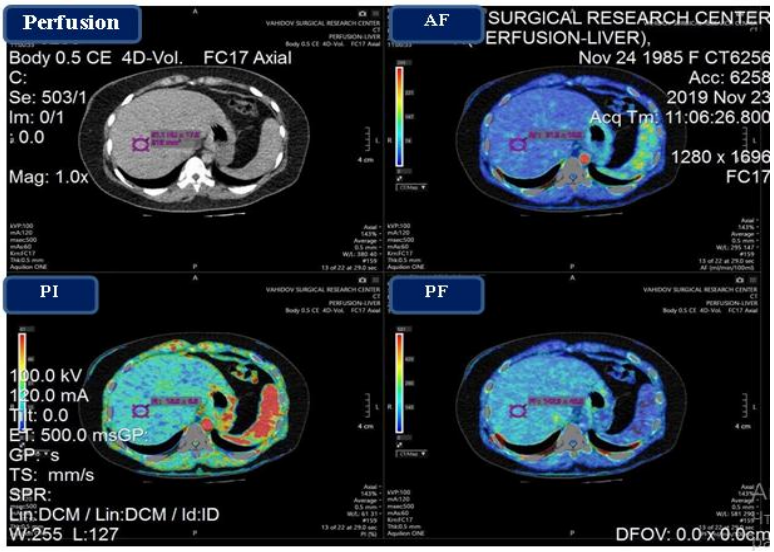

Figure 2: CT perfusion of normal liver

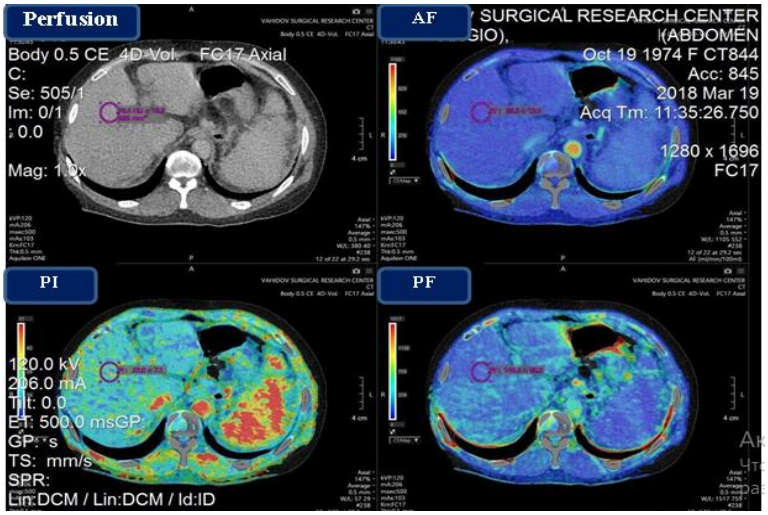

Figure 3: CT perfusion in liver cirrhosis.

In contrast to the indices in norm, cirrhosis or fibrosis, the peculiarity of liver perfusion in HLH was the maximum deviation of fractions with the lowest portal blood flow (107.0±5.5 ml/100ml/min; $p<0.001$) and the highest compensatory amplification of the arterial fraction (up to 49.5±1.7 ml/100ml/min; $p<0.001$). This indicates that in the formation of subhepatic block with partial preservation or complete absence of portal blood flow, significant arterialization of the liver is the result of the development and progression of PH in the perinatal period. Thus, in 8 out of 13 cases of HLH was the result of intrauterine malformation of portal system - cavernous transformation of portal vein, in other 5 cases thrombosis of portal vein or the whole splenoportal channel was the result of early pathology of newborns - umbilical sepsis. In other words, the peculiarity of PG formation in the perinatal period is a significant decrease in the fraction of portal perfusion due to partial or complete block of portal blood flow (the indices of which were maximally low in our study). In this case, the formation of compensatory venous inflow to the liver due to the development of neoportal vessels occurs within several years. Accordingly, in conditions of pronounced decrease in portal perfusion, in parallel with the progression of PG and compensatory development of non-portal venous collaterals,

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there is a significant redistribution of arterial blood supply to the liver. Reserve possibilities of this fraction of hepatic blood flow taking into account the perinatal period are characterized by the maximum possibility of arterial channel transformation (Fig.4).

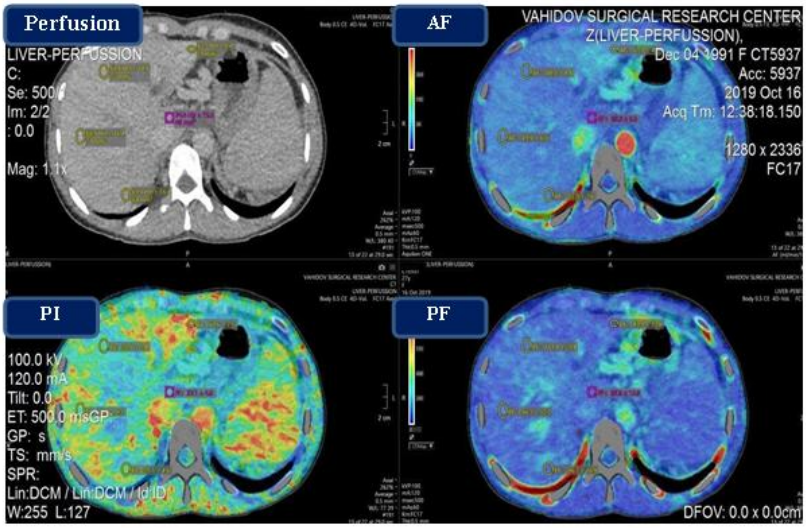

Figure 4: CT perfusion of the liver in extrahepatic portal hypertension

That is why, in our study, the level of arterial perfusion in IDH reached maximum values - 49.5±1.7 ml/100ml/min, whereas, for example, in CKD this fraction compensatory increased on average only to 41.4±0.7 ml/100ml/min ($p<0.001$). Corresponding to the maximum redistribution of intrahepatic blood flow fractions, the highest perfusion index was obtained in the same group - 35.6±1.5% ($p<0.001$) (Fig. 5).

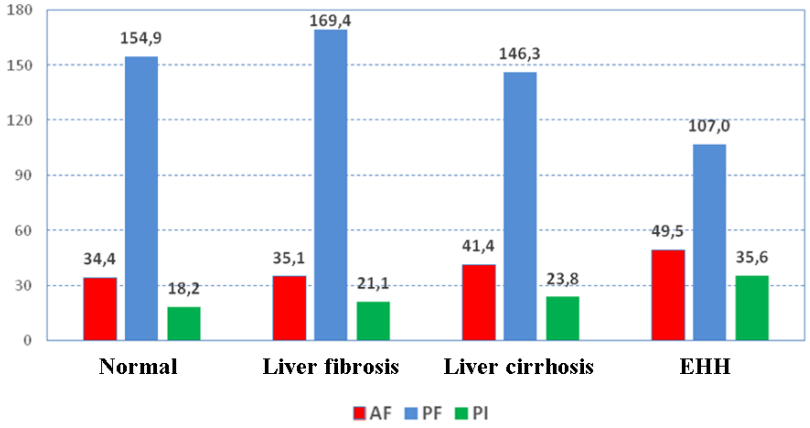

Figure 5. Summary data on CT-perfusion indices of the liver depending on the cause of portal hypertension.

Paradoxical changes of portal perfusion fraction in FP should be emphasized. In contrast to CKD and CHF, the characteristic feature of presinusoidal block was not a decrease, but an increase in portal blood flow relative to normal values up to 169.4±5.0 ml/100ml/min ($p<0.01$). That is, hyperkinetic type of portal circulation was determined in these cases. Scientific interest to the research of this type of blood flow peculiarities is conditioned by pathogenetic interrelations of PG formation on the background of increased portal perfusion. Literature analysis indicated that

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excessive blood flow in the portal vein system was the primary mechanism of PG occurrence, and moderate sclerotic changes in the liver were secondary. Further studies showed that in the formation of hyperkinetic PG, in addition to arterio-venous fistulas, the development of portohepatic venous collaterals is also important. This suggests that against the background of FP with presinusoidal block the obtained high indices of portal perfusion are a consequence of functional activity of portohepatic venous collaterals. Expressed sclerotic process with nodular transformation in CKD due to necrosis of hepatocytes leads to a decrease in portal perfusion, which aggravates the course of the pathological process forming a vicious circle. In this situation, the functional status of hepatocytes is crucial in terms of disease prognosis. In turn, at FP the function of hepatocytes is not disturbed, and the aggression factor is the progressive PG and its complications (esophageal-gastric bleeding). This proves the fact that bypass surgery in FP does not lead to deterioration of hepatocyte function, but allows to radically solve the problem of PG. This is due to the fact that the initial hyperkinetic type of portal blood flow against the background of portal system decompression at portosystemic shunting provides preservation of hepatopetal blood flow.

Another peculiarity of liver perfusion in fibrosis is that against the background of increased values of portal fraction there was no significant increase in arterial blood flow (35.1±1.8 ml/100ml/min; p>0.05). In turn, the mean value of perfusion index (PI) despite the increased portal fraction was significantly higher in relation to the norm (21,1±0,7%; p<0,001). This indicates that despite the absence of difference in the volume of arterial inflow at FP and in norm, still at presinusoidal block there is a change in the ratio of blood flow fractions in the direction of arterial fraction increase. This also proves that hyperkinetic type of portal blood flow in FP is caused by the development of portohepatic collaterals and despite the inflow enhancement this effect in qualitative value does not allow to maintain adequate blood supply to the liver, due to which the perfusion index changes with the increase of arterial fraction (Fig.6).

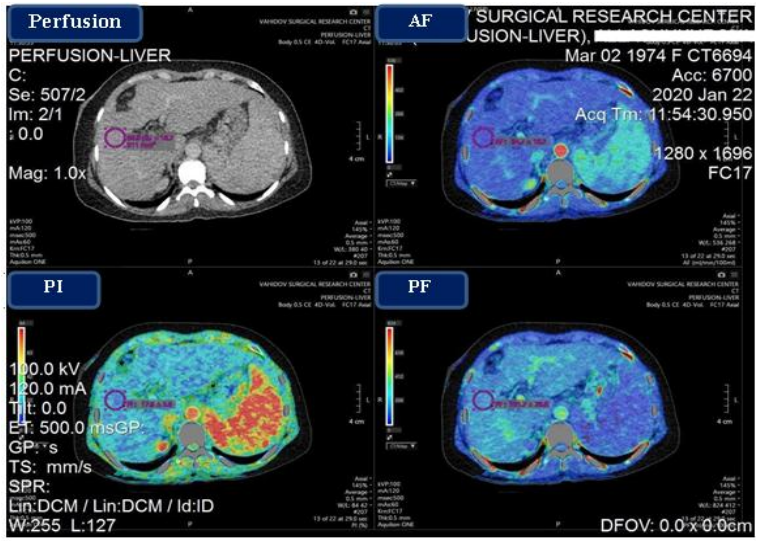

Figure 6. CT perfusion in hepatic fibrosis.

Conclusions

Thus, the etiological factor of PG development affects the peculiarities of changes in hepatic perfusion in relation to the normal values, with a paradoxical increase in the portal fraction with an insignificant change in arterial blood flow and perfusion index being characteristic of presynusoidal block against the background of FP. In CP with postsinusoidal block, reliable changes with a decrease in portal blood flow with compensatory increase in arterial inflow and perfusion index were noted. Progression of cirrhotic process with increasing block for portal blood flow causes the development of compensatory increase of arterial fraction, the reserve possibilities of which are strictly limited by the peculiarities of angioarchitectonics and hemodynamics of the arterial basin of the liver, accordingly, one of the conditional signs of the beginning of decompensation of hepatoperfusion is stabilization of AF index growth (on average at the level of 44,8±1,3 ml/100ml/min) against the background of significant increase of PI (up to 25-30%; $p<0.001$) due to a decrease in the portal fraction of hepatic blood flow (from 150.9±4.0 to 119.6±3.3 ml/100ml/min; $p<0.001$).

In subhepatic block (SHB) the minimal level of portal blood flow was verified against the background of maximal reserve stimulation of arterial inflow with a significant increase in perfusion index.

References

1. Accia M., Ainora M.E., Ponziani F.R. et al. Portal vein thrombosis in cirrhosis: Why a well-known complication is still matter of debate. Wld J. Gastroenterol. 2019; 25 (31): 4437-4451. https://doi.org/10.3748/wjg.v25.i31.4437
2. Babadjanov A. Kh, Djuraeva N.M. Amirhamzaev A.T. The features of liver perfusion in various hepatic diseases and portal hypertension according to data of multidetector computed tomography. American Journal of Medicine and Medical Sciences 2020, 11(14): 509-514
3. Jiang H., Zheng T., Duan T. et al. Noninvasive in vivo imaging grading of liver fibrosis // J. Clin. Transl. Hepatol. 2018. V. 6. № 2. P. 198-207.
4. Margini C., Berzigotti A. Portal vein thrombosis: The role of imaging in the clinical setting. Dig. Liver Dis. 2017; 49 (2): 113-120. https://doi.org/10.1016/j.dld.2016.11.013
5. Mezikova E.A., Zavadovskaya V.D., Beloborodova E.V., Dolgalev I.V., Tonkikh O.S. Perfusion multislice computed tomography of the hepatolienal zone in patients with diffuse liver diseases. Pharmateka. 2019;26(2):42–47. DOI: https://dx.doi.org/10.18565/pharmateca.2019.2.42-47
6. Moller S., Bendtsen F. The pathophysiology of arterial vasodilatation and hyperdynamic circulation in cirrhosis. Liver Int. 2018; 38: 570-580. https://doi.org/10.1111/liv.13589
7. Nazirov F.G., Djuraeva N.M., Vakhidova N.T, Omonov O.A., Salimov U.R., Amirhamzaev A.T. Diagnostic informativity of volume MDCT-angiography and MR- cholangiography in pre- and intraoperative periods for the examination of donors of a liver fragment. Journal of Life Science and Biomedicine. J Life Sci Biomed, 9 (6):, 2019
8. Stashuk G.A., Moisyuk Ya.G., Smirnova D.Ya., Sumtsova O.V. The role of CT liver perfusion in detecting predictors of portal vein thrombosis in patients with compensated and

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subcompensated liver cirrhosis. Medical Visualization. 2022;26(1):119-129. (In Russ.) https://doi.org/10.24835/10.24835/1607-0763-1092

9. Stashuk G.A., Moisyuk Ya.G., Smirnova D.Ya., Sumtsova O.V. The Role of Perfusion Computed Tomography of the Liver in the Assessment of Hemodynamics in Patients with Compensated Cirrhosis of the Liver and the Presence of Type 2 Diabetes Mellitus. Pilot Study. Radiology - Practice. 2021;(2):62-71. (In Russ.) https://doi.org/10.52560/2713-0118-2021-2-62-71
10. Wang Q., Koniaris L. G., Milgrom D. P. et al. CT and MRI imaging and interpretation of hepatic arterioportal shunts // Transl. Gastroenterol. Hepatol. 2019. V. 4. № 34. P. 36-58.
11. Zhan Y., Wu Y., Chen J. et al. Value of liver perfusion maging of 256-slice CT in evaluation of the cirrhosis // Zhong Nan Da Xue Xue Bao Yi Xue Ban. 2016. V. 41. № 1. P. 44-50.

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