Researches showed that while the rise of the level of portal pressure there lowered the speed of blood flow in the portal vein. Thus patients with CH of moderate activity while the level of portal activity 148,0 mm of water column had the speed of blood flow of 23,0 sm/sec, while the rise of portal pressure till 181,0 mm of water column there was observed the decrease of the blood flow's speed till 16,0 sm/sec. While the rise of the level of pressure in the portal vein from 188,0 till 204,0 mm of water column patients with CH of high activity had an observed lowering of the blood flow's speed in the portal vein from 20,0 sm/sec till 15,0 sm/sec, correspondingly. Received facts testify to that by the rising of portal pressure there is registered the lowering of the blood flow's speed in the portal vein. In the clinical practice about the degree of portal hypertension on judge by the extent of diameter of portal and splenic veins. In connection with this there were compared extents of portal pressure and diameter of portal vein. Researches were carried out of all observed patients with CH of different activity. It was found that while the rise of portal pressure there was observed the increase of the diameter of portal vein. Thus sick people with CH of moderate activity while the portal pressure 148,0 mm of water column the diameter of portal vein was 11,0 ± 0,2 mm, and while the rise of portal pressure till 181,0 mm of water column the diameter of portal vein increased till 14,0 ± 0,3 mm. While the rise of portal pressure from 188,0 till 204,0 mm of water column of the patients with CH of high activity there was observed the dilatation of portal vein from 12,0 ± 0,2 mm till 16,0 ± 0,1 mm, correspondingly. Received facts testify that in the majority of cases there was registered right parallelism between the level of portal pressure and extent of the diameter of portal vein. At the same time not infrequent were cases when this parallelism wasn't observed. From the received results it follows that parameters of liver haemodynamics are connected between each other while the main role of activity of inflammatory process. In connection with this we can't exclude the situation when with the degree of intensity of morphological changes between the level of portal pressure and extent of the diameter of portal vein there not always observed the interconnection.

Conclusion

Practically all healthy people by the facts of ultrasonic Doppler sonography with the use of empiric formula of calculation the average extent of portal pressure was 113,0 ± 4,4 mm of water column with rippling at the limits from 103 till 143 mm of water column. Portal hypertension, while the viral diseases of liver, develops at the stage of chronic hepatitis, the degree of its intensity depends on the activity of inflammatory process in the liver. Widening of the diameter of portal and splenic veins, and also increase of the square of their cross section not always correlate with the level of portal pressure.

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SOME PROBLEMS IN COMPUTER RECONSTRUCTION OF ORGANS, TISSUES AND CELLS IN HUMAN AND ANIMAL MORPHOLOGY

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Modern computer equipment allows us to create 3-D models of methodical – biological objects that find a wide implementation in typographic anatomy and surgery, as well as in computer tomography of diagnosis purposes (Maykaya, 2000; Blinov, 2005). Along with that another area of medical science exists, in which a work with visual images goes on that could make a breakthrough in our knowledge of human and animals organ, tissue, and cells structure. It is the morphology that includes anatomy, histology, and cytology. It could allow us to carry out a multi-level reconstruction of organs and their components. This knowledge was especially important for defining anatomic, histological, and cytological peculiarities of various knots and tracts distribution in heart, where significant blanks in topographic determination of all the organ's leading systems and its nervous and vascular components exist. It leads to the fermentation of our knowledge on the organ's function morphological substance in its normal and pathological condition. Classical heart leading systems and knot operative myocardium metastructure reconstruction methods are very rare to find in literature (Thaemert, 1978) because of their laboriousness and significant technical difficulties (a cutting of serial ultrathin cuts on numerous nets of one block takes years of work). We can try to make the researcher's problems easier if we use little animal organs enclosed in one block of epoxy gums and further its cutting into thick

(50–100 mcm) cuts, then half-thin (1–2 mcm) cuts, and, if necessary, from them – into ultrathin cuts (50–70 nm). Further, as we obtain analog pictures, we can try, using a perspective, to form a single image (like it is done in animation). Or, as we digitize a cut series, we can obtain the same result if we use a 3D graphic editor. It should be outlined that special program products for metastructure and histological pictures analysis are extremely rare and expensive, and their development requires work of competent programmers along with the labor of morphologists. Structure modeling, first of all, of inner muscular organs (heart, uterus) with the usage of serial half-thin and thick cuts will allow us to define the layer organs structure and then will reveal functionally-important part of these organs (for example, sphincters, nerve ganglions, leading ways). An involvement of cybernetics students of RCSPC medical-biological faculty will aim them for the development of program applications that can be used in future to reconstruct all the various mammal types organs, including those in normal ontogeny and under various pathologies. Besides, it will, if provided with the digital organ model, to rotate it in different angles and reveal the peculiarities of its tissue, cellular, and subcellular structure.

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UNEVEN GROWTH AND GISTOGENESIS OF THORACIC DUCT

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Thoracic duct (TD) has endothelial wall in embryo of 7 week with first endothelial valves in embryo of 8 week. It transformates into multi-layered TD wall in ending of human uterine development when thickness of the wall increases in a 20 times. Uneven growth of TD wall connects with its gistogenesis intimately. Correlation of thickness of TD wall and width of its lumen is less 0,05 before 12 weeks of uterine human life what permit to regard TD as thin-walled vessel. In fetuses of 4–5 months TD become thick-walled vessel. Inner layers of TD wall try the largest tension under inner pressure and stretch more than outer layers. Moreover subendothelial layer of connective tissue remains thin and crumbly with net of thin retcular fibres. Their thickness increases in broaden and compacted outer layer where first collagen fibres appear. The first smooth myocyties appear, muscular coat forms on boundary between layers with diferrent hard. The coat brakes moving distortion (relative displacement of layers) and prevents destruction of TD wall. Lymph from TD lumen penetrates through endothelium into connective tissue and erodes it, brakes weak intermolecular connections, slows down fibregenesis. Speed of diffusion diminishs rapidly in thickness of the wall. And thus subendothelial layer of connective tissue remains thin and plastic. Outer coat of TD promotes new formation and growth of valves because it slows down outer dilatation of TD and stabilizes structures of residual deformation. Muscular coat limits dilatation of TD and stretching of inner layers of TD wall, its folden deformation. Thus uneven growth is in the base of TD morfogenesis and gistogenesis.

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DIFFERENTIAL FACTOR IN ANLAGE OF LYMPHATIC BED

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Anlage of primary lymphatic system comes by means of formation of lateral pockets in large primary veins with thin endothelial wall under the pressure of adjacent arteries with external coat. Comunications of central canal and lateral pockets of increasing veins narrow. The pockets separate from secondary veins as lymphatic chinks with endothelial covering. Some of primary veins turn off blood flow together with lymphatic chinks as tributaries preceding venous pockets including part of protocapillary net. Lymphatic capillaries preserve very thin endothelial wall without basal membrane and lose vascular connection with blood bed. Therefore usually lymph pressure is lower than the venous and prone to the greater fluctuations right to zero. Lymphovenous connections preserve usually in the neck of human and mammals where negative venous pressure originates periodically. Thus lymphatic bed including roots in microcirculatory bed develops from venous collaterals by means of reducing their connections with venous magistrals. In conditions of intensive organogenesis and increasing blood pressure the basal membrane forms under thickenning endothelium of blood capillaries. The membrane cuts off lymphatic collaterals with thin endothelium without basal membrane. The pressure of differentiated advential coat of secondary veins is conducive to separation of venous pockets. Thus transverse gradient of blood and mechanical pressure originates in primary drainage system of parallel vessels [collateral – magistral – collateral] and diferentiates the system on secondary or true veins and primary lymphatic vessels as modificate or persistent primary veins.

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