

(50–100  $\mu\text{m}$ ) cuts, then half-thin (1–2  $\mu\text{m}$ ) 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 RSCSPC 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 reticular fibres. Their thickness increases in broaden and compacted outer layer where first collagen fibres appear. The first smooth myocytes appear, muscular coat forms on boundary between layers with different 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 fibrogenesis. 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. Communications 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 proto-capillary 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 magistral. In conditions of intensive organogenesis and increasing blood pressure the basal membrane forms under thickening 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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