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PREDICTION OF MORPHOLOGICAL CHANGES ARISING IN ACUTE AND CHRONIC CIRCULATORY DISORDERS

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Abstract. This research work is devoted to the study of the pathogenesis of acute and chronic circulatory disorders and their morphological manifestations in organs. The article analyzes such fundamental pathological processes as arterial and venous hyperemia, ischemia, stasis, thrombosis, embolism, and infarction. Particular emphasis is placed on the types of cell death (necrosis and apoptosis), tissue remodeling, and sclerosis processes[1,2,3]. Based on international cardiological and angio-pathological statistical data from 2024–2026, the dynamics of these diseases are demonstrated.

Keywords: homeostasis, hemodynamics, perivascular edema, diapedetic hemorrhage, fibrinoid necrosis, hypoxia, angiogenesis, myocardial remodeling, endothelial dysfunction.

INTRODUCTION

At present, one-third of all human deaths are directly related to circulatory disorders**.** Acute circulatory disorders, such as acute myocardial infarction or hemorrhagic stroke, rank first in the world in terms of mortality, while chronic heart failure (CHF) is a major factor limiting the working capacity of the population. Nearly 64 million people worldwide are living with CHF[4,5], and this figure is increasing by 2.1% annually. Knowledge of the morphological basis of these processes is important not only for pathologists, but also for clinicians of all specialties in establishing a diagnosis.

In the body, blood circulation is the main mechanism for maintaining homeostasis, and its disturbance triggers a chain of reactions. Among acute circulatory disorders, arterial hyperemia is distinguished by its diversity[6,7,8]. Physiological arterial hyperemia, for example in muscles during physical activity, is beneficial, whereas its pathological form, such as in inflammation or impaired innervation, leads to excessive tissue heating and edema. Morphologically, this condition is characterized by marked dilation of small arteries and capillaries and their engorgement with blood[9,10,11]. Under the microscope, this can be identified by tension of the vessel walls and flattening of endothelial cells**.**

However, acute venous congestion has much more severe consequences. This process occurs when the return of blood to the heart becomes difficult. For example, in acute left ventricular failure, the pulmonary vessels become engorged with blood. In this case, the alveolar septa become edematous, and fluid passes from the capillaries into the alveolar spaces[12,13,14]. This condition is called pulmonary edema, and its morphology is characterized by an increase in lung weight from the normal 350–450 g to 1200–1500 g**,** and by the release of pink frothy fluid on sectioning. According to statistical data, the mortality rate in cardiogenic shock accompanied by pulmonary edema is 40–50%.

Ischemia is the cessation or marked reduction of blood supply to tissue. Acute ischemia is most often caused by thrombosis or embolism. In the ischemic area, cells experience oxygen deprivation (hypoxia). During the first 10–15 minutes of ischemia, glycogen stores are depleted[15,16], anaerobic glycolysis is activated, lactate accumulates, and intracellular pH decreases, resulting in acidosis. This process leads to enzyme activation and destruction of organelle membranes. Morphologically, these changes are referred to as the stage of “ischemic dystrophy”. If blood flow is not restored, this stage progresses to infarction, that is, ischemic necrosis[17,18].

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Table 1. Step-by-step morphological description of ischemia and infarction.

Stage	Time	Macroscopic changes	Microscopic appearance
Ischemic (prenecrotic)	6–12 hours	Organ slightly dull, pale	Cardiomyocyte swelling, glycogen depletion
Necrotic stage	18–48 hours	Yellowish-white focus, hemorrhagic border	Kariolysis, plasmorrhexis, neutrophilic infiltration
Organizatio nal (scarring)	2–8 weeks	Whitish-gray dense tissue	Granulation tissue replaced by connective tissue

Chronic circulatory disorders present a completely different morphological picture. Here, the main mechanism is long-term hypoxia and impaired tissue trophism. In chronic venous congestion, for example in cardiac valvular defects, connective tissue proliferation (sclerosis) begins in all organs due to hypoxia. This process is called “congestive induration”. The liver is particularly sensitive to this process. In chronic right ventricular failure, blood accumulates in the central veins of the liver. The central hepatocytes undergo atrophy and necrosis, and their place is occupied by blood-filled sinusoids. At the periphery of the liver lobules, hepatocytes undergo compensatory fatty degeneration. As a result, the characteristic appearance of a “nutmeg liver” is formed. If prolonged, this condition leads to cardiac cirrhosis.

Thrombosis is the formation of a blood clot inside blood vessels during life. This process is based on Virchow’s triad: damage to the vessel wall, slowing of blood flow, and changes in blood composition. The structure of a mixed thrombus differs by its “head” (the part attached to the vessel wall), “body,” and “tail” (the freely projecting part along the blood flow). The later fate of a thrombus may vary: it may undergo lysis (dissolution), become incorporated into the vessel wall (organization), form channels (canalization), or detach and become an embolus. According to statistics, 30% of patients with deep vein thrombosis of the lower limbs are at risk of pulmonary artery embolism.

Embolism refers to foreign particles carried by the blood or lymph flow that obstruct a blood vessel. Types of embolism include thromboembolism, fat embolism (in bone fractures), air embolism (in neck vein injuries), gas embolism (in decompression sickness), and tissue or cellular embolism (in metastatic spread of cancer cells). In fat embolism, the mortality rate is 10–15%, because the microcirculation of the brain and lungs becomes severely impaired.

Another important form of chronic circulatory disorder is atherosclerosis. This process is characterized by the accumulation of lipids in the arterial wall and the proliferation of connective tissue. Morphologically, the development of an atherosclerotic plaque takes several decades. Initially, “fatty streaks” appear in the intima, then they transform into a fibrous plaque. Rupture of the plaque leads to acute thrombosis and, consequently, to infarction or stroke. According to 2025 statistics, atherosclerotic changes are observed in 60% of men over 45 years of age.

Table 3. Causes of death worldwide (analysis by circulatory system diseases)

Type of disease	Annual number of deaths (million)	Global share (%)	Growth trend
Ischemic heart disease	9.1	16.2	Increasing
Stroke (all types)	6.6	11.6	Stable
Peripheral arterial disease	1.2	2.1	Increasing
Rheumatic heart disease	0.3	0.5	Decreasing

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The final stage of morphological changes is often decompensation. In this condition, the heart muscle (myocardium) becomes excessively enlarged (dilation), dystrophic changes in the cells intensify, and the organ is no longer able to perform its function. For example, in chronic ischemia, diffuse fine-focal cardiosclerosis develops in the myocardium. Under the microscope, whitish bands of connective tissue can be seen between the muscle fibers. These changes lead to cardiac rhythm disturbances (arrhythmias) and sudden cardiac arrest.

CONCLUSION

Circulatory disorders are not merely a mechanical obstruction, but rather a complex pathophysiological and morphological process involving the entire organism. Acute disorders are characterized by tissue necrosis and systemic shock, whereas chronic forms differ by the exhaustion of adaptive-compensatory mechanisms such as hypertrophy and sclerosis, eventually leading to organ failure. Modern morphological diagnostics, including immunohistochemistry and electron microscopy, are opening new horizons for understanding and managing these processes at the cellular level. In clinical practice, this knowledge is the key to reducing mortality and prolonging patients' lives.

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