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PATHOPHYSIOLOGICAL BASIS OF ANEMIA TYPES AND HEMODYNAMIC COMPENSATORY MECHANISMS: AN IN-DEPTH ANALYSIS

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Abstract: This comprehensive study is devoted to one of the most pressing problems of modern medicine—the molecular pathogenesis of anemias of various etiologies and the analysis of the body's multistage adaptive responses to hypoxia. The article thoroughly examines the cellular-level mechanisms of the main forms of anemia—iron deficiency anemia (IDA), megaloblastic anemia (vitamin B_{12} and folate deficiency), hemolytic anemia, and aplastic anemia—as well as the hormonal and genetic regulation of erythropoiesis.
The central part of the study scientifically substantiates hemodynamic alterations, including changes in blood rheological properties, increased cardiac output, decreased peripheral vascular resistance, and redistribution processes within the microcirculatory bed. The obtained results make it possible to identify the functional reserves and "fatigue points" of compensatory mechanisms depending on the severity of anemia, as well as to prevent cardiohemodynamic complications.

Keywords: Anemia, pathophysiology, hematogenic hypoxia, hemodynamics, compensatory mechanisms, erythropoiesis, cardiomyopathy, iron deficiency, 2,3-diphosphoglycerate, oxyhemoglobin dissociation

Introduction: Relevance of the Problem and Pathophysiological Essence: Anemia is a complex pathological condition characterized by a decrease in hemoglobin concentration per unit volume of blood (<120 g/L in women, <130 g/L in men) and/or a reduction in the number of erythrocytes, leading to impaired oxygen delivery to tissues. According to World Health Organization (WHO) data, approximately 25–30% of the global population suffers from anemia, making it one of the most significant public health challenges worldwide [1]. In Uzbekistan, the prevalence of anemia has regional, ecological, and socio-biological characteristics. In particular, the unfavorable environmental conditions of the Aral Sea region, high salinity of drinking water, and insufficient dietary intake of iron and vitamins pose a serious threat to national public health [3]. Regardless of its form, the main pathophysiological link of anemia is hematogenic hypoxia, i.e., a reduction in the oxygen-carrying capacity of the blood. Under hypoxic conditions, mitochondrial respiration is disrupted, resulting in a sharp decrease in ATP synthesis, intracellular acidosis, and increased oxidative stress due to free radical accumulation [2]. In response to this life-threatening condition, the body rapidly activates hemodynamic (increased cardiac activity), hormonal (erythropoietin synthesis), and metabolic (increased 2,3-DPG) compensatory mechanisms. Hemodynamic compensation represents an "emergency mode" of cardiovascular system function aimed at maintaining stable oxygen delivery to tissues.

Literature Review: Analysis of National and International Studies: Uzbek scientists have systematically studied various aspects of anemia for decades. Academician H.A. Hakimov, in his fundamental works, interpreted anemia not merely as a change in hematological parameters but

as a disruption of the body's overall adaptive capacity—an anemic syndrome [2]. His studies demonstrated that iron deficiency impairs not only hemoglobin synthesis but also the activity of iron-containing enzymes (cytochromes, myoglobin, peroxidases), thereby exacerbating tissue-level hypoxia. Professor S.N. Bobojonov analyzed cardiovascular changes in anemia from the perspective of "hyperdynamic circulation" [4]. He associated functional systolic murmurs, tachycardia, and left ventricular hypertrophy observed in anemia with decreased blood viscosity and increased blood flow velocity. Cardiologist R.D. Qurbonov demonstrated that anemia is an independent factor that worsens the course of chronic heart failure and increases mortality risk [5]. International studies widely discuss erythropoietin synthesis in the kidneys in response to hypoxia via the HIF-1α pathway, highlighting its role not only in erythropoiesis but also in endothelial protection [8]. Recent clinical guidelines emphasize the importance of not only iron supplementation but also cardiometabolic therapy and antihypoxic agents in anemia management [3].

Materials and Methods: The study employed the following comprehensive methods. Systematic bibliographic analysis: More than 100 scientific sources from the last 15 years, including national clinical protocols and authoritative monographs indexed in the National Library of Uzbekistan, Google Scholar, and PubMed [1,2,6]. Pathophysiological modeling: Analysis of changes in cardiogeodynamic parameters (heart rate, stroke volume, cardiac output) across three severity levels of anemia (mild, moderate, severe) [4,5]. Molecular-biochemical analysis: Evaluation of iron metabolism (ferritin, transferrin), erythrocyte 2,3-diphosphoglycerate concentration, and its effect on hemoglobin oxygen affinity (P50 index) [2,7]. Biophysical modeling: Simulation of increased venous return resulting from reduced blood viscosity and peripheral resistance based on the Frank–Starling mechanism and Poiseuille's law [7,8]. Retrospective statistical analysis: Assessment of cardiac complications of anemia using data from specialized hematology and cardiology centers of the Republic. Results. Pathophysiological Analysis and Hemodynamics. Differential Pathogenesis of Anemia Types. Iron Deficiency Anemia (IDA): The most prevalent form, caused by impaired heme synthesis due to iron deficiency, resulting in microcytosis and hypochromia. Megaloblastic Anemia (Vitamin B_{12} and Folate Deficiency): DNA synthesis impairment halts cell division while cytoplasmic growth continues, leading to the formation of large, immature, and fragile megaloblasts (ineffective erythropoiesis) [7]. Hemolytic Anemia: Characterized by increased erythrocyte destruction. Elevated bilirubin levels contribute to endogenous intoxication and aggravate hypoxia [6].

Stages of Hemodynamic Compensation: Changes in Blood Rheology: Reduced erythrocyte mass lowers blood viscosity, accelerates blood flow, decreases total peripheral vascular resistance, and enhances venous return [4]. Cardiac Compensation (Hyperdynamia): Cardiac output increases by up to 200–300% to stabilize oxygen delivery. Tachycardia: Due to sympathetic-adrenal system activation. Increased stroke volume: According to the Frank–Starling mechanism. Tissue-Level Adaptation: Increased erythrocyte 2,3-diphosphoglycerate shifts the oxyhemoglobin dissociation curve to the right, facilitating oxygen release to tissues [2].

Discussion: The findings indicate that hemodynamic compensation is a highly energy-consuming process that eventually exhausts myocardial reserves. Observations by Uzbek researchers confirm that patients with chronic anemia develop stable "anemic cardiomyopathy" within 3–5 years [5]. Persistent hyperdynamic cardiac activity leads to mitochondrial dysfunction in cardiomyocytes and eccentric left ventricular hypertrophy. In iron deficiency

anemia, reductions occur not only in hemoglobin but also in myocardial myoglobin and enzymatic iron, directly impairing myocardial contractility. Under Uzbekistan's hot climate conditions, anemia-associated hypoxia combined with electrolyte imbalance (potassium, magnesium) predisposes patients to severe cardiac arrhythmias [3].

Conclusion and Recommendations: The pathophysiological basis of anemia is hematogenic hypoxia, which disrupts bioenergetic processes in all organs and systems [2]. Hemodynamic adaptation occurs through hyperdynamic circulation, increasing cardiac output but ultimately leading to heart failure with prolonged persistence [4,5]. In Uzbekistan, anemia prevention and treatment strategies should focus not only on restoring hemoglobin levels but also on cardiometabolic protection through antihypoxic and metabolic therapies [3].

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