INTRODUCTION

The necessity of investigation of hypoxia-related changes in living organisms has been mostly stimulated by the practical needs of mankind in the course of mastering the hydrosphere, higher atmospheric layers, and mountain landscapes, as well as by the existence of a number of related medical problems. As early as in the 19th century, studies of the preventive and therapeutic effects of hypoxia were initiated [1, 2], but an overwhelming part of the investigations for the past two centuries were directed toward the pathophysiological aspects of this phenomenon. Only in the last decades of the 20th century has special attention been focused on hypoxia from the preventive medicine standpoint. An important contribution to the studies of hypoxia along the mentioned lines of investigations has been made by Ukrainian [3-11] and Kirghizian [12-18] scientists.

Due to urbanization, ecological accidents, and excessive and unwarranted use of pharmaceuticals in clinics and everyday life, problems of hypoxia and application of a graduated decrease in the O$_2$ partial pressure for stimulation of hemopoiesis, immunogenesis, and other adaptive functions of the organism have assumed even higher importance at the turn of the 21st century.

It was always assumed in the specialized literature that those cellular systems that show the highest functional activity are the most sensitive to oxygen deficiency. Among such elements are O$_2$-receptor cells of the sinocarotid glomus (glomus caroticus), tissues of the eye retina, and brain cortex neurons. In general, experimental data on the consequence of manifestations of the reactions of different systems of the organism to gradual reduction of pO$_2$ in the air and arterial blood give support to this standpoint.

The first reflex systemic reactions of the organism to hypoxia are revealed in the shifts of ventilation and circulation parameters, namely in an increase in lung ventilation and cardiac output [19]. It should be noted that adaptation to new conditions...
can occur with full compensation of the \( \text{pO}_2 \) level in the blood. Subsequent hypoxia-related reactions can be manifested as limitations in peripheral vision, development of euphoria, and disorders of self-assessment. In this case, a great variability of the individual’s sensitivity to \( \text{O}_2 \) deficiency in the inhaled air is observed [3, 12, 13].

**SYSTEMIC STABILITY AND RESPONSIVENESS OF THE CNS TO HYPOXIA**

Stability of the structures of the brain and spinal cord to hypoxia is also rather variable. However, according to the data of most researchers, there is a definite \( \text{pO}_2 \) level necessary for the maintenance of a minimum adequate functional state of the entire CNS and, therefore, of the entire organism. In this case, the oxygen tension in brain cells should be not less than 3-5 mm Hg. A complex of homeostatic mechanisms counteracts drops in the \( \text{pO}_2 \) in the organism’s tissues. Therefore, when the \( \text{pO}_2 \) level in lung alveoli drops at 2,500 m altitude to 70% of that observed at sea level, the oxygen tension in the blood can, for some time, be kept (depending on the adaptability of the organism) at the level of 90% of the corresponding initial value.

It should be taken into consideration that within the initial period of adaptation to hypoxia, which is observed at moderate altitudes above sea level, the reactions of some systems and organs are realized through “centralization” of circulation with the prevailing blood supply of the CNS, heart, and lungs. In ascending to higher altitudes, a gradual “superposition” of changes in the systems, which have already responded to hypoxia of a lower level, occurs [16]. This process is of an individual nature; not all people who have successfully adapted themselves to one altitude can re-adapt to a higher locality [15].

It is natural that the CNS supply with oxygen depends not only on the \( \text{pO}_2 \) in the air but also on the state of the blood supply. The critical level of blood pressure at normal \( \text{pO}_2 \), which provides satisfactory parameters of energy metabolism in all brain structures, is equal to 30-40 mm Hg. With further drop in blood pressure, the concentration of macroergs is reduced to dangerous values [30, 32, 33]. In acute hemic hypoxia, the first link for preservation of the necessary level of energy metabolism is redistribution of the blood flow and intensification of \( \text{O}_2 \) extraction by tissues [31, 34].

One-fifth of the general blood flow is utilized for the cerebral blood supply (the mass of the human brain constitutes only about 1/20 of the body mass). This confirms the existence of extremely high requirements of the CNS for oxygen and energy carriers and the corresponding sensitivity to their deficiencies [20, 21]. It should be noted that about 20% of oxygen is consumed in the electric transport system of the endoplasmic reticulum. The bulk of oxygen is utilized in the processes of mitochondrial oxidation. In this case, up to 50% of the energy released in the brain is consumed for the maintenance of transmembrane ion gradients [22]. Oxygen homeostasis in CNS tissues is provided by a complex system of regulation of brain circulation [23]. Disorders of such a system (irrespectively of their trigger mechanisms) shift the parameters of neuronal oxygenation from physiological limits. Brain ischemia is one of the basic reasons for hospitalization of neurological patients [24, 25].

The hypothesis of a higher resistance of brainstem structures to hypoxia compared with that of the brain cortex cannot now be interpreted unambiguously. For example, Martz et al. [26] demonstrated that various brainstem structures adapt to hypoxia in a different manner. Earlier, Ukrainian researchers showed significant changes in the reactivity of the olivary structures, pons, and brainstem reticular formation in \( \text{O}_2 \) deficiency that considerably exceeded analogous reactive modifications in spinal neurons [27].

A mosaic, in the functional respect, pattern of different brain structures is also reflected in their sensitivity to hypoxia. The central part of the cortical area of the motor analyzer and cerebellar Purkinje (pear-like) cells are most vulnerable to oxygen deficiency. Klatzo [28] reported that Purkinje cells and brain cortex neurons are very close with respect to their sensitivity to hypoxia. Less sensitive are cells of deep cortical layers and the hypothalamus. We should note that it would be reasonable to differentiate more definitely various meanings of the term “sensitivity.” Under this term, a threshold, on the one hand, for any functional disorder (sometimes even positive) and, on the other hand, for only negative, pathological disorder is meant. Experimental findings show that moderate hypoxia even enhances the homeostatic activity of hypothalamic neuronal systems [29]. Researchers believe that the hippocampus and nuclei of pairs X and XII of the cranio-cerebral nerves (CCN) [30, 31] are the brain structures with the lowest susceptibility to hypoxia. The physiological mode of