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CHANGES IN THE MORPHOFUNCTIONAL PROPERTIES OF THE THYROID GLAND AND BLOOD INDICATORS DURING THE HYPOXIC PROCESS.

Khojiyev Dilmurod Yakhshiyevich., Karimova MuattarSharipovna
Bukhara State Medical Institute named after Abu Ali ibn Sino

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Abstract:

Throughout a person's life, the thyroid gland (TG) is exposed to various adverse factors (environmental pollution, drug abuse, bad habits, malnutrition, etc.), which often lead to the development of its pathology. This necessitates the search for new effective means and methods for the prevention and treatment of thyroid dysfunction. During the hypoxic process, changes are observed not only in the morphological structures of the thyroid gland, but also in blood parameters. This process can also include periods of hypoxic state and oncogenesis in the perinatal period, on the basis of which this article analyzes studies of the effect of hypoxic conditions on the morphogenesis of the thyroid gland.

Keywords: thyroid gland, hypoxic state, oncogenesis, hormone.

Thyroid hormones take an active part in the formation of the body's adaptive response to the action of various environmental factors. Studies [1] have shown that the pituitary-thyroid system is involved in the stress response already in the early stages and, according to this author, plays an important role in the development of the general adaptation syndrome described by G. Selye. At the same time, the hormonal activity of the thyroid gland (TG) is largely determined by the nature, nature and intensity of exposure. However, the question of the patterns of changes in the functional reserves of the thyroid gland, depending on the specifics of the current stress factor, remains unresolved. It has been shown that short-term immobilization causes a significant increase in the secretion of thyroid hormones of the thyroid gland [12], but already a day after the onset of immobilization stress, according to morphometry in animals, a decrease in the synthetic activity of thyrocytes is revealed [3]. Under conditions of long-term waiting stress, in rats of various lines, selected for the excitability of the nervous system, the hormone-synthetic activity of the thyroid gland decreases and signs of its hypofunction appear with the restoration of the morphofunctional state 2 weeks after the end of neurotic exposure [4, 5].

An imbalance between oxygen demand and delivery leads to the development of hypoxia and thus poses a significant threat to bioenergetic homeostasis and cell survival. The term "hypoxia" (hypoxia) comes from the Greek hypo - "below" and the Latin oxygenium - "oxygen" and means an insufficient supply of O2 to the tissues of the body or a violation of its utilization in the process of biological oxidation. The problem of oxygen deficiency is one of the fundamental problems in modern biology and medicine. Interest in hypoxia as a typical pathological process that occurs and accompanies animals and humans throughout life has existed for hundreds of years. A characteristic feature of various hypoxic conditions is that they are an important pathogenetic mechanism of almost any disease. As is known, hypoxia determines the development of ischemic damage to the heart and brain, the formation of multiple organ failure in the syndrome of disseminated intravascular coagulation (DIC), is an invariable companion of shock and collaptoid conditions, diseases of an infectious and non-infectious nature, as well as stressful effects (5).

A morphological study of the thyroid gland of rabbits with parallel measurement of the level of hormones in the blood under conditions of prolonged emotional and pain stress with increasing intensity of exposure showed that with stress depletion of the body's adaptive reactions, a parallel decrease in the secretory activity of the thyroid gland is observed [6]. The specific role of thyroid hormones in the reactions of thermogenesis and in the processes of adaptation of the body to prolonged exposure to cold is well known 114]. In this work, we evaluated the functional activity of the thyroid gland during extreme cooling of laboratory animals.

Systemic hypoxia can cause inflammatory changes in organs and tissues. The concept that hypoxia itself can induce inflammation was adopted following a series of studies showing that hypoxia-induced signaling pathways are associated with activation of the pro-inflammatory factor NF-κB (5).

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The development of inflammation in response to tissue hypoxia has been shown in organ transplant patients: ischemia of donor organs increases the risk of inflammation and their rejection in the recipient (3). The relationship of hypoxia and inflammation has also been found in acute respiratory distress syndrome, in which, as a rule, tissue hypoxia and inflammatory reactions potentiate each other (4). In obesity, an imbalance between oxygen delivery and consumption causes hypoxia and increases the content of pro-inflammatory adipokines in adipose tissue (7). In healthy volunteers exposed to 2-hour normobaric hypoxia (12% O2) in vivo, neutrophil chemotaxis, phagocytosis, and ROS production increased (9). In addition, when exposed to hypoxia equivalent to an altitude of 5500 m, the activity of HIF-1α and NF-κB factors increased in the peripheral blood cells of healthy people (8). Exposure to hypoxia during mountain climbing in some people is associated with the development of altitude sickness and is characterized by hypoxic pulmonary and cerebral edema (4). Similar effects are observed in short-term and chronic hypoxic exposure in mice, which leads to an increase in the concentration of pro-inflammatory cytokines and chemokines, as well as pulmonary edema (7).

The study of the gas composition of the blood is important for determining the state of the function of external respiration and the cardiovascular system. For the clinic, continuous dynamic monitoring of arterial oxygen saturation is especially valuable when the patient performs various functional tests. These requirements are best met by the oximetry method, which in this respect, despite its inherent shortcomings, has enormous advantages over the vanSlyk method. There is quite a lot of information in the literature about the gas composition of the blood both in practically healthy people and in people with various diseases of the respiratory organs and the cardiovascular system [1-7]. All these data indicate that in case of insufficiency of the function of external respiration and cardiac activity, there is a decrease in the saturation of arterial blood with oxygen of varying severity. As for such studies in patients with plutonium pneumosclerosis, we found only two works in the available literature [8, 9]. Meanwhile, the study of blood in patients with plutonium pneumosclerosis, in our opinion, is of significant theoretical and practical interest, since the degree of blood oxygen saturation, as well as changes in its gas composition, depend primarily on the state of the function of external respiration and, therefore, can to a certain extent reflect the degree violations of this function. Carrying out oxiometric studies in dynamics using various functional tests makes it possible to detect the presence of latent pulmonary and heart failure, and also makes it possible to judge, to a certain extent, the body's compensatory ability to maintain blood oxygen saturation at a normal level. For this purpose, we examined a group of people who worked in conditions of air pollution with plutonium aerosols in quantities significantly exceeding the maximum permissible levels, some of whom developed plutonium pneumosclerosis of varying severity.

Thyroid diseases occupy an important place among endocrine pathologies. They are accompanied by a violation of the production of thyroid hormones with the development of hypothyroidism syndrome or thyrotoxicosis. Thyrotoxicosis is a syndrome caused by a persistent increase in the level of thyroid hormones in the blood [1, 3]. Thyrotoxicosis develops in various diseases: diffuse toxic goiter, thyrotoxic adenoma, nodular toxic goiter, chronic autoimmune Hashimoto thyroiditis, subacute thyroiditis, ectopic goiter, pituitary adenoma [6]. In these diseases, the high content of thyroid hormones is due to their increased secretion by the thyroid gland itself (hyperthyroidism). However, thyrotoxicosis can develop with excessive intake of thyroid hormones from outside: during treatment with thyroid hormones, iodine-based syndrome, etc. [5, 7]. In modern cardiology, the drug "amiodarone" is widely used, which has unique antiarrhythmic properties. However, the drug has a toxic effect on the thyroid gland due to the high content of iodine. Iodine-containing preparations are used as radiopaque agents (RCS). During coronary angiography as part of the RCS, the patient is injected with iodine from 15 g to 100 g, which exceeds the total iodine content in the human body by 1500–10,000 times. Prolonged use of iodine-containing antiarrhythmic drugs or the introduction of radiopaque agents can lead to the development of iodine-induced disorders with the development of thyrotoxicosis. However, iodine-induced pathological processes have not been sufficiently studied in our country [2]. In the domestic literature, there is only fragmentary information on this issue; the nature and dynamics of morphological changes in the gland in exogenous thyrotoxicosis have not been studied. This issue needs further in-depth study.

The role of respiratory, circulatory, tissue, and hemic hypoxia in the pathogenesis of severe thyroid dysfunction has been studied for a long time. Most of these studies were performed under conditions of acute and chronic exposure to hypobaric hypoxia, of varying severity and duration [1, 2]. And, as you

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know, low atmospheric pressure exacerbates the negative effects of hypoxia on the human body. Berezovsky V.A. et al. [3] developed the concept of sanogenic hypoxia. In accordance with this concept, hypoxia, corresponding to an altitude of 1500-3000 meters above sea level (17-12% oxygen), has a healing and healing effect on the human body. Exposure to hypoxic gas mixtures corresponding to a given altitude in intermittent mode under normal atmospheric pressure, on the one hand, eliminates the adverse effects of hypobaria and hypoxia, and on the other hand, potentiates the sanogenic effects of the latter.

The problem of the occurrence of stress and its impact on human life and health is given great attention by specialists in various fields of science. One of the most relevant areas of modern experimental medicine is the study of the response of a growing organism to chronic stress [1, 3, 4]. Stress can trigger autoimmune thyroiditis by damaging the thyroid gland [2, 6]. Therefore, it is very important to know the features of adaptation of the hypothalamic-pituitary-thyroid system to stress. The identification of regularities in the morphological structure of the thyroid gland in stressful situations is relevant, since it meets the requirements of modern fundamental science [5]. The regulation of stress and related diseases is of great practical and theoretical importance.

Interest in studying the structure and function of the thyroid gland is steadily growing due to the importance and diversity of the influence of thyroid hormones on vital processes. The thyroid gland is involved in the regulation of oxygen metabolism in the body. Hypoxia is a strong effect on the body, endangering its very existence. However, interval hypoxic stimulation (IGS) is used in clinical practice for the prevention and treatment of a certain number of diseases [1], and in aviation and occupational medicine to increase the stress resistance of people in dangerous professions to extreme impacts. In connection with the foregoing, the purpose of this work was to study the morphofunctional state of the thyroid gland during experimental hypoxia.

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