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REGULATION OF THE SECRETION OF THYROID HORMONES

Abstract: *This article discusses specific aspects of the regulation of thyroid function.*

Key words: *thyroid gland, histology, physiology, follicle, follicular cell*

РЕГУЛИРОВАНИЕ СЕКРЕТАЦИИ ГОРМОНОВ ЩИТОВИДНОЙ ЖЕЛЕЗЫ

Резюме: *В данной статье обсуждаются конкретные аспекты регуляции функции щитовидной железы.*

Ключевые слова: *щитовидная железа, гистология, физиология, фолликул, фолликулярная клетка.*

Thyroid stimulating hormone of the pituitary gland is considered a specific stimulant of the thyroid gland. The thyrotropic function of the anterior pituitary gland, in turn, is activated by thyroliberin secreted by the hypothalamus (see. Hypothalamic neurohormones). Therefore, damage to the hypothalamus leads to the same weakening of the thyroid gland, as well as hypophysectomy (see. Pituitary gland). This method of regulation can be referred to as transadenohypophyseal.

In turn, thyroid hormones (especially triiodothyronine) inhibit the thyroid-stimulating function of the pituitary gland (and, presumably, the secretion of thyrotropic hormone by the hypothalamus), that is, the relationship between the functional activity of the thyroid gland and the intensity of the thyroid-stimulating function of the pituitary gland is a system of negative feedback (see), ensuring the preservation of oscillations functional activity of the thyroid gland within the physiological norm.

Thyroid stimulating hormone, which enters the thyroid gland with the blood stream, is perceived by specific receptors localized in the plasma membrane of thyrocytes. These receptors, when combined with thyroid-stimulating hormone, activate the adenylate cyclase system of thyrocytes, which, through cyclic adenosine monophosphate (cAMP), activates the enzyme systems of thyrocytes, as a result of which their functional activity increases.

It has been established that the secretion of thyroid hormones is activated directly by sympathetic impulses, although not as intensely as thyroid-stimulating hormone. Parasympathetic impulses suppress these processes. Thus, the regulating effects of the hypothalamus (see) on the thyroid gland can occur both through the pituitary gland and bypassing it (parahypophyseal).

At the same time, afferent signals from the thyroid gland, arriving along the centripetal nerve pathways, reaching the hypothalamus, weaken the thyrotropic function of the pituitary gland; therefore, negative feedback between the thyroid gland and the pituitary gland is also manifested in the direct action of nerve impulses. The state and activity of the parafollicular cells of the thyroid gland do not depend on the pituitary gland and are not disturbed after hypophysectomy; their function is stimulated by sympathetic impulses, while parasympathetic impulses inhibit. At the same time, the secretory activity of parafollicular cells is directly dependent on the concentration of calcium in the blood: an increase or decrease in it entails, respectively, an increase or decrease in the secretion of calcitonin by parafollicular cells. Antagonistically interacting with the parathyroid hormone (see Parathyroid hormone) of the parathyroid glands (see Parathyroid glands), calcitonin ensures that the level of calcium in the body remains constant.

Almost all thyroxine entering the blood reversibly binds to serum proteins, mainly to L-globulin - the so-called thyroxine-binding globulin, and partly to thyroxine-binding prealbumin and albumin. Therefore, the concentration of protein-bound iodine (see) in the blood is often considered as

an indicator of the secretory activity of the thyroid gland. The binding of thyroxine to serum proteins prevents its destruction, but prevents its active action on cells. A dynamic equilibrium is established between bound and free thyroxine in the blood, and only free thyroxine has an effect on the reacting cells and tissues. Triiodothyronine binds to serum proteins weaker than thyroxine. The half-life of thyroxine in the blood lasts 6-7 days, triiodothyronine decomposes faster (half-life is 2 days).

The reception of thyroxine takes place inside the cells. Penetrating into the cell, thyroxine immediately loses one iodine atom, passing into triiodothyronine. The point of application of triiodothyronine (both received from the blood and formed from thyroxine) is DNA, where triiodothyronine stimulates transcription (see) and the formation of RNA.

In the cells, further deiodination of thyroxine and triiodothyronine, deamination, rupture of the diphenyl ether bond and decarboxylation take place (see Iodine metabolism).

In the metabolism of thyroid hormones, the liver plays the main role, in which the decay products of deiodinated iodothyronines bind to glucuronic and sulfur conjugates and then enter the intestines with bile, from where the released iodine is absorbed back into the blood, transferred to the thyroid gland and reutilized.

The role of thyroid hormones in morphogenesis and regulation of physiological processes

The effects caused by thyroid hormones are based on their influence on oxygen uptake and oxidative processes in the body. It has been established that thyroxine in toxic doses acts on the mitochondria of cells, uncoupling the synthesis of ATP with the transfer of electrons along the respiratory chain and thus blocking oxidative phosphorylation (see).

Thyroid hormones increase heat production, and if they are insufficient (hypothyroidism), the body temperature decreases. Simultaneously

hypothyroidism (see) is accompanied by water retention in the body and a decrease in the excretion of calcium and phosphorus in the urine.

Thyroid hormones increase the breakdown of glycogen (see) and reduce its formation in the liver. The lack of these hormones is accompanied by a disorder in the regulation of carbohydrate metabolism (see) and an increase in the body's tolerance to glucose. With hyperthyroidism (see Thyrotoxicosis), the excretion of nitrogen in the urine increases and is disturbed (phosphorylation of creatine (see). Under hypothyroidism, the content of cholesterol increases (see) in the blood, and with an excess of thyroid hormones, its level decreases. At the same time, with hyperthyroidism the excitability of the higher nervous system (especially its sympathetic division) increases, which is manifested by tachycardia (see), arrhythmias (see Arrhythmias of the heart), an increase in blood flow rate, an increase in systolic blood pressure.At the same time, the motility of the gastrointestinal tract and the secretion of digestive juices increase.

Thyroid hormones are essential for the normal functioning of the central nervous system. Lack of thyroid hormones in the embryonic period and at the beginning of the postnatal period can lead to a delay in the differentiation of the cerebral cortex and the mental development of the child up to cretinism (see).

Thyroid hormones together with growth hormone (see) are involved in the regulation of body growth (especially stimulate ossification).

Features of the function of the thyroid gland in the antenatal and postnatal periods

During pregnancy, the functional activity of the mother's thyroid gland increases; an increase in the content of total thyroxine in the blood is associated with an increase in the synthesis of thyroid-stimulating hormone under the influence of placental estrogens.

The ability of the thyroid gland to concentrate and accumulate iodine appears in the fetus at 10-12 weeks of intrauterine development. At the same

time, the synthesis of monoiodothyronine, diiodothyronine, triiodothyronine, thyroxine, thyroxine-binding globulin begins. In fetal blood serum (see) thyroliberin (thyrotropin-releasing hormone) and thyroid-stimulating hormone of pituitary origin appear. The regulatory relationship between thyroid-stimulating hormone and thyroid hormones is established from the 30th week of intrauterine development.

There was no parallelism between the content of thyroid-stimulating and thyroid hormones in the blood of the mother and the fetus, since the transplacental transport of these hormones is less than 1%. The highest concentration of thyroid hormones in the prenatal period is detected in the fetus before its birth.

Immediately after birth, there is a period of increased functional activity of the thyroid gland. The level of thyroid-stimulating hormone increases in the 30th minute after birth, and after 24-48 hours it decreases to the same level as in adults. The content of triiodothyronine increases maximally by the end of the first day. The maximum increase in thyroxine content is noted 24-48 hours after birth, then a gradual decrease in its level occurs.

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