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# Pathophysiology, Pathogenesis, Prognosis, and Treatment of Pathological Changes in the Endocrine System

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**Abstract:** The endocrine system coordinates the functions of various organs through hormones, which are chemicals secreted into the bloodstream from specialized cells located within endocrine glands. Once in the bloodstream, hormones affect the function of target tissues, which may be other endocrine glands or receptors. Some hormones affect the cells of the same organ as their source (paracrine action), and sometimes the cells that produce them (autocrine action).

**Key words:** Endocrine system, pathology, pathogenesis, prognosis and treatment.

**Introduction:** Hormones bind selectively to receptors located within or on the surface of target cells. Receptors located within cells interact with hormones that regulate gene function (e.g., corticosteroids, vitamin D, thyroid hormone). Receptors located on the cell surface bind to hormones that regulate enzyme activity or affect ion channels (e.g., growth hormone, thyrotropin-releasing hormone).

Endocrine diseases result from dysfunction of the endocrine glands and/or their target tissues.

The functions of the peripheral endocrine organs are controlled to varying degrees by pituitary hormones. Some functions (e.g., insulin secretion by the pancreas, which is primarily regulated by blood glucose levels) are minimally controlled or independent of pituitary control (e.g., parathyroid hormone secretion by the parathyroid glands, primarily in response to blood calcium levels), while many (e.g., thyroid or gonadal secretion) are dependent on the secretion of hormones. The secretion of pituitary hormones is controlled by the hypothalamus.

The relationship between the hypothalamus and the pituitary gland (called the hypothalamic-pituitary axis) is a feedback system. The hypothalamus receives signals from almost all areas of the central nervous system and uses them to produce signals that are sent to the pituitary gland. In response, the pituitary gland secretes a variety of hormones that stimulate many endocrine glands in the body. Changes in the blood levels of hormones produced by these endocrine glands are sensed by the hypothalamus, which increases or decreases stimulation of the pituitary gland accordingly, thus maintaining homeostasis.

**Research methods and materials:** The hypothalamus modulates the activity of the anterior and posterior pituitary glands in various ways. Neurohormones synthesized in the hypothalamus reach the anterior pituitary gland (adenohypophysis) through a special portal vascular system and regulate the synthesis and secretion of 6 major peptide hormones of the anterior lobe (see figure: Pituitary gland and its target organs ). The latter regulate the functions of peripheral endocrine glands (thyroid gland, adrenal glands and gonads), as well as growth and lactation. There are no direct neural connections between the hypothalamus and the anterior pituitary gland.

In contrast, the posterior pituitary (neurohypophysis) consists of neuron cell bodies located in the hypothalamus. These axons serve as a depot for two peptide hormones: vasopressin (antidiuretic

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hormone) and oxytocin, which are synthesized in the hypothalamus; in the periphery, these hormones regulate water balance, milk production, and uterine contractions.

Almost all hormones produced by the hypothalamus and pituitary gland are released into the blood in pulses; periods of secretion alternate with periods of rest. The secretion of some hormones [e.g., adrenocorticotropic hormone (ACTH), growth hormone, prolactin] has a clear circadian rhythm; the secretion of others (e.g., luteinizing and follicle-stimulating hormones during menstruation) obeys a monthly rhythm, to which the circadian rhythm is superimposed.

**Results:** To date, 7 physiologically significant hypothalamic neurohormones have been identified. Except for the biogenic amine dopamine, all of them are small peptides. Some of them are produced not only in the hypothalamus, but also in the environment, especially in the gastrointestinal tract, and act as local paracrine factors. One of them is vasoactive intestinal peptide, which also stimulates prolactin secretion.

Neurohormones control the secretion of many pituitary hormones. The secretion of most of them depends on stimulating signals from the hypothalamus. The exception is prolactin, the secretion of which is controlled by inhibitory stimuli. After transection of the pituitary stalk (which connects the pituitary gland to the hypothalamus), prolactin secretion increases, while the secretion of other hormones of the anterior pituitary gland decreases.

Most pathological processes in the hypothalamus (including tumors and inflammatory lesions) are accompanied by changes in the secretion of hypothalamic neurohormones. Since neurohormones are synthesized in different centers of the hypothalamus, some pathological processes alter the production of only one neuropeptide, while others affect the production of several of them. In this case, both hypo- and hypersecretion of neurohormones can occur, which, accordingly, weaken or enhance the secretion of pituitary hormones. Clinical syndromes resulting from dysfunction of pituitary hormones (for example, diabetes insipidus, acromegaly, and hypopituitarism) are discussed elsewhere.

The cells of the anterior pituitary gland (which account for 80% of the weight of the entire pituitary gland) synthesize and secrete a number of hormones necessary for normal growth and development, as well as stimulate the activity of several target glands.

ACTH is also called corticotropin. The main stimulator of ACTH secretion is corticotropin-releasing hormone (CRH), but vasopressin is also involved in stress. Under the influence of ACTH, the adrenal cortex secretes weak androgens such as cortisol and dehydroepiandrosterone (DHEA). Once in the blood, cortisol and other corticosteroids (including exogenous corticosteroids) inhibit the secretion of CRH and ACTH. Changes in the CRH-ACTH-cortisol system are a key component of the stress response. In the absence of ACTH, the adrenal cortex atrophies and virtually ceases to produce cortisol.

TSH regulates the structure and function of the thyroid gland, stimulating the synthesis and secretion of thyroid hormones. The synthesis and secretion of TSH itself is stimulated by the hypothalamic thyrotropin-releasing hormone (TRH) and inhibited by thyroid hormones present in the blood (according to the principle of negative feedback).

**Discussion:** LH and FSH control the production of sex hormones. The synthesis and secretion of LH and FSH are primarily stimulated by gonadotropin-releasing hormone (GnRH) and are inhibited by estrogens and testosterone. One factor that controls the release of GnRH is kisspeptin, a hypothalamic peptide that is activated by increased leptin levels during puberty. Two sex hormones, activin and inhibin, only affect FSH; activin is stimulating, while inhibin is inhibiting.

In women, LH and FSH stimulate the development of ovarian follicles and ovulation.

In men, FSH acts on Sertoli cells and is necessary for spermatogenesis, and LH acts on Leydig cells in the testes, stimulating testosterone biosynthesis.

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GH stimulates somatic growth and regulates metabolism. The main stimulator of GH synthesis and secretion is growth hormone-releasing hormone (GHRH), and the inhibitor of these processes is somatostatin. GH regulates the synthesis of insulin-like growth factor 1 (IGF-1), also called somatomedin-C, which is responsible for tissue growth. IGF-1 is produced by many tissues, but its main source is the liver. One form of IGF-1 is found in muscles, where it plays a role in increasing their strength. This variant of the protein is less regulated by GH than its hepatic variant.

The metabolic effects of GH occur in two phases. Initially, GH acts like insulin, increasing glucose uptake by muscle and adipose tissue, stimulating amino acid uptake and protein synthesis in the liver and muscle, and inhibiting lipolysis in adipose tissue. After a few hours, a more pronounced anti-insulin metabolic effect develops. These include inhibition of glucose uptake and utilization, which is accompanied by increased lipolysis with increased blood glucose levels and increased plasma free fatty acids. Increased GH levels during fasting maintain blood glucose levels and mobilize fat as an alternative energy source. GH production decreases with age. Ghrelin (a hormone produced by cells in the fundus of the stomach) promotes GH release from the pituitary gland, increases food intake, and improves memory in animal models.

Prolactin is produced by cells called lactotrophs; they make up about 30% of all cells in the adenohypophysis. During pregnancy, the size of the pituitary gland doubles, mainly due to hyperplasia and hypertrophy of the lactotrophs. The main function of prolactin in humans is to stimulate milk production. Prolactin is also released during sexual intercourse and during stress. The level of prolactin is a sensitive indicator of pituitary dysfunction; it is this hormone that is most often produced by pituitary tumors, and in the presence of infiltrative processes or tumors that compress the pituitary gland, its content, like the level of other hormones, decreases.

The anterior pituitary gland also produces a number of other hormones. These include proopiomelanocortin (POMC, which helps produce ACTH), alpha and beta-melanocyte-stimulating hormones (MSH), beta-lipotropin (β-lipotropin), enkephalins, and endorphins. POMC and MSH can cause skin hyperpigmentation, which is clinically significant only in diseases characterized by a sharp increase in ACTH levels (such as Addison's disease or Nelson's syndrome). The function of β-LPG is unknown. Enkephalins and endorphins are endogenous opioids that bind to and activate opiate receptors in the central nervous system.

The posterior pituitary gland secretes vasopressin (also called arginine vasopressin or antidiuretic hormone [ADH]) and oxytocin. Both of these hormones are released in response to nerve impulses and have a half-life in the blood of about 10 minutes.

The main function of vasopressin is to conserve water in the body by the kidneys by increasing the permeability of the distal tubular epithelium. High concentrations of vasopressin also cause vasoconstriction. Like aldosterone, vasopressin plays an important role in maintaining fluid homeostasis and the hydration of blood vessels and cells. The main stimulus for vasopressin secretion is an increase in the osmotic pressure of water in the body, which is "sensed" by osmoreceptors in the hypothalamus.

Another important stimulus is a decrease in fluid volume, which is "sensed" by baroreceptors in the left atrium, pulmonary veins, carotid sinus, and aortic arch; signals from the baroreceptors are transmitted to the central nervous system via the vagus and glossopharyngeal nerves. Other stimuli for vasopressin secretion include pain, stress, vomiting, hypoxia, exercise, hypoglycemia, cholinergic agonists, beta-blockers, angiotensin, and prostaglandins. Inhibitors of vasopressin secretion include alcohol, alpha-blockers, and glucocorticoids.

Vasopressin deficiency causes central diabetes insipidus. Failure of the kidneys to respond normally to vasopressin causes nephrogenic diabetes insipidus. Removal of the pituitary gland does not usually result in permanent diabetes insipidus because some surviving hypothalamic neurons continue to produce small amounts of vasopressin.

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Copeptin is produced in the posterior pituitary gland at the same time as vasopressin. Measuring its levels can help determine the cause of hyponatremia.

Oxytocin production is stimulated by breastfeeding, which causes contraction of myoepithelial cells. This contraction helps milk move from the alveoli into the large ducts, from where it is expelled (called the let-down reflex in nursing mothers). Oxytocin causes contraction of uterine smooth muscle cells, and the sensitivity of the uterus to oxytocin increases during pregnancy. However, there is no significant increase in plasma oxytocin levels during labor, and the role of oxytocin in labor induction remains unclear.

The stimuli for oxytocin secretion in men are unknown; its concentration in male plasma is very low.

Insufficient stimulation of the pituitary gland due to pituitary dysfunction or insufficient stimulation of the pituitary gland by the hypothalamus (secondary disorders)

Hyperstimulation due to pituitary dysfunction or hyperstimulation of the pituitary gland by the hypothalamus (secondary disorders)

In rare cases, abnormal tissue response to hormones (usually hypofunction).

In this case, there may be an increase (hyperfunction) or decrease (hypofunction) in hormone production by peripheral glands.

Hyperfunction of the endocrine glands can result from either intrinsic dysfunction of the pituitary gland or from overstimulation of the pituitary gland by the hypothalamus. However, hyperfunction of the endocrine glands is most often caused by hyperplasia or neoplasia of the gland itself. In some cases, hormones are produced by malignant tumors of other tissues (ectopic hormone production).

Excess hormones can also be the result of their exogenous effects. In some cases, patients take over-the-counter medications containing hormones and do not know or tell their doctors about it.

Hypersensitivity of tissues to hormones is also possible. Peripheral endocrine glands can be stimulated by antibodies, as occurs in hyperthyroidism, typical of Graves' disease. Destruction of a peripheral endocrine gland may be accompanied by a rapid release of stored hormones into the blood (for example, the release of thyroid hormones in subacute thyroiditis).

Defects in enzymes involved in the long steps of hormone synthesis can lead to hyperproduction of hormonally active compounds formed proximal to the enzyme block. Finally, overproduction of a hormone may occur as an appropriate response to a disease state.

Hypofunction of the endocrine gland can be the result of insufficient stimulation from the pituitary gland due to intrinsic dysfunction of the pituitary gland or insufficient stimulation of the pituitary gland by the hypothalamus.

Peripheral hypofunction of the gland itself can result from congenital or acquired diseases (including autoimmune diseases, tumors, infections, vascular diseases, and toxins).

**Conclusion:** Genetic disorders that cause hypofunction may involve deletion of the gene encoding the hormone or abnormal hormone synthesis. Decreased hormone production by the peripheral endocrine gland, coupled with increased secretion of the regulatory pituitary hormone, can lead to peripheral endocrine gland hyperplasia. For example, if there are abnormalities in thyroid hormone synthesis, thyroid-stimulating hormone (TSH) is produced excessively, leading to the development of a goiter.

After secretion in peripheral endocrine glands, some hormones require conversion to the active form. In a number of disorders, this step in hormone activation is blocked (for example, in renal pathology, the formation of the active form of vitamin D is inhibited). Antibodies to a circulating hormone or its receptor can block the binding of the hormone to the receptor.

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Diseases or pharmacological agents can accelerate the clearance of hormones. Substances present in the blood are also capable of blocking the action of hormones. Hypofunction can also be the result of defects in the receptors or in the pathways of transmission of activating signals from receptors to peripheral endocrine gland cells.

Because the symptoms of endocrine disorders are insidious and nonspecific, their clinical recognition often takes months or years. Therefore, biochemical diagnosis is usually of the greatest value; this usually requires measuring blood levels of peripheral endocrine hormones, pituitary hormones, or both.

Since the secretion of many hormones has a circadian rhythm, measurements should be made at a specific time of day. Hormone levels (e.g., luteinizing hormone) can vary over shorter time periods, necessitating the need to measure them 3-4 times within 1-2 hours or to use pooled blood samples. Hormones that show weekly variability (e.g., estrogen) should be measured separately each week.

**List of used literature:**

1. Sarkisova V., Xegay R., Numonova A. ENDOCRINE CONTROL OF THE DIGESTION PROCESS. GASTROINTESTINAL ENDOCRINE CELLS //Science and innovation. – 2022. – T. 1. – №. D8. – C. 582-586.
2. Sarkisova V. ASPECTS OF THE STATE OF THE AUTONOMIC NERVOUS SYSTEM IN HYPOXIA //Science and innovation. – 2022. – T. 1. – №. D8. – C. 977-982.
3. Vladimirovna S. V. et al. Analysis of Women's Reproductive and Somatic Health, Hospitalized for Endometrial Hyperplasia and Uterine Bleeding //Eurasian Medical Research Periodical. – 2023. – T. 17. – C. 91-96.
4. Vladimirovna S. V. Epidemiology, Theories Of The Development, Conservative And Operative Treatment Of The Endometriosis //The Peerian Journal. – 2023. – T. 15. – C. 84-93.
5. Vladimirovna S. V. et al. Adenomyosis as an Independent Unit of Dysfunction of the Endometrium and Uterine Myometrium //Scholastic: Journal of Natural and Medical Education. – 2023. – T. 2. – №. 3. – C. 85-91.
6. Sarkisova V. et al. ESSENTIAL ROLE OF BRADIKININ IN THE COURSE OF BASIC LIFE PROCESSES //Science and innovation. – 2022. – T. 1. – №. D8. – C. 576-581.
7. Sarkisova V., Xegay R. CAUSES, DIAGNOSIS, CONSERVATIVE AND OPERATIVE TREATMENT OF UTERINE MYOMA //Science and innovation. – 2022. – T. 1. – №. D8. – C. 198-203.
8. Vladimirovna S. V. About the Causes of Endometrial Hyperplasia and Forms of Endometrial Hyperplasia //Global Scientific Review. – 2023. – T. 12. – C. 25-32.
9. Vladimirovna S. V. et al. Hyperplastic Processes of the Endometrium: Issues of Ethiopathogenesis, Clinic, Diagnosis, Treatment //Scholastic: Journal of Natural and Medical Education. – 2023. – T. 2. – №. 3. – C. 72-77.
10. Саркисова В. В. Патогенетические отношения артериальной гипертензии и сопротивления инсулина //IQRO JURNALI. – 2023. – T. 2. – №. 1. – C. 727-731.
11. Vladimirovna S. V. PATHOGENETIC RELATIONSHIPS OF ARTERIAL HYPERTENSION AND INSULIN RESISTANCE //IQRO JURNALI. – 2023. – T. 2. – №. 1. – C. 685-691.
12. Vladimirovna S. V. ABOUT THE CAUSES OF ENDOMETRIAL HYPERPLASIA AND FORMS OF ENDOMETRIAL HYPERPLASIA //ResearchJet Journal of Analysis and Inventions. – 2022. – T. 3. – №. 11. – C. 66-72.
13. Sarkisova V. et al. UTERINE ARTERY EMBOLIZATION AS A METHOD OF TREATMENT OF UTERINE FIBROIDS //Science and innovation. – 2023. – T. 2. – №. D3. – C. 115-121.

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14. Vladimirovna S. V. et al. Ovarian Apoplexy and its Impact on Reproductive Health //Central Asian Journal of Medical and Natural Science. – 2023. – Т. 4. – №. 2. – С. 381-388.
15. Vladimirovna S. V. et al. Menstrual Cycle Disturbances in the Reproductive Period //Central Asian Journal of Medical and Natural Science. – 2023. – Т. 4. – №. 2. – С. 389-397.