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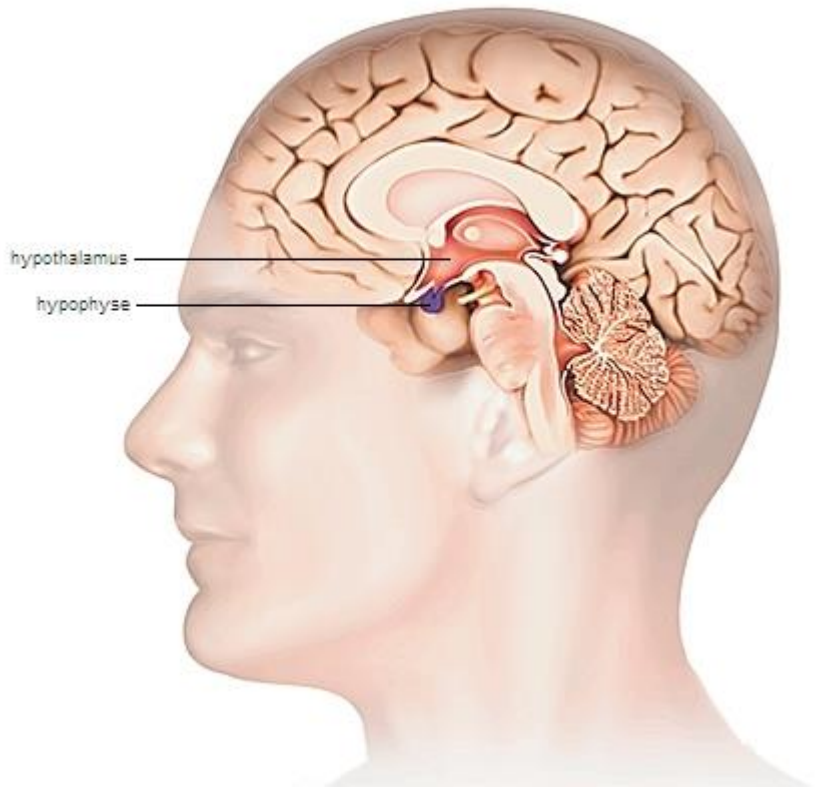
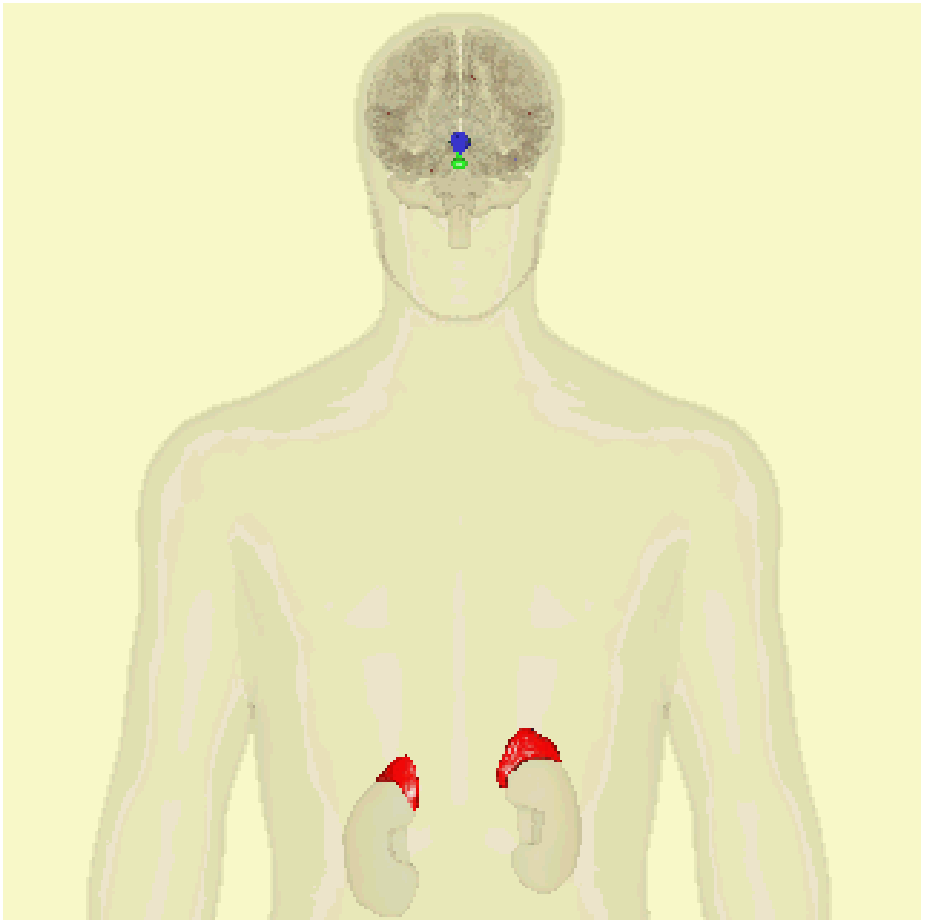
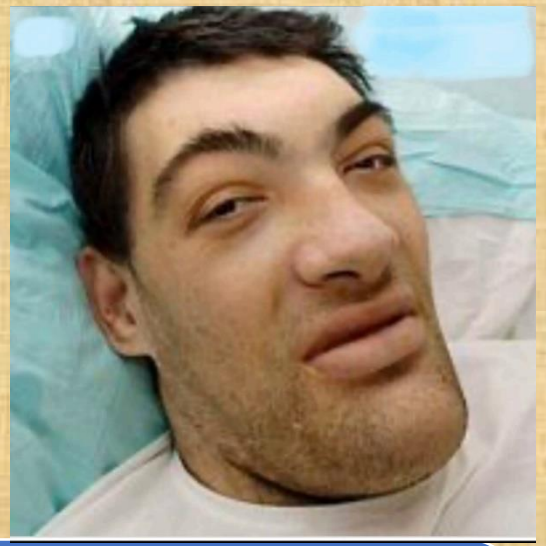




- **The level of somatotropic hormone in the blood**
- **STH blood level is normally 0.4-10.0 ng/ml in male and 1-14 ng/ml in female.** The secretion of this hormone increases during physical load and deep sleep.
- STH stimulates cell growth directly, but also through insulin-like growth factor I and II (somatomedin).
- STH is secreted unevenly throughout the day. During most of the day, the amount of this hormone in the blood is recorded at a low level. Therefore, special provocation tests are used. STH secretion is mainly regulated by hypothalamus-synthesized STRF (somatotropin-releasing factor), somatostatin, and insulin-like growth factor.
- An increase in the concentration of insulin-like growth factor I in the blood slows down the transcription of STH genes in the hypophysis gland through feedback.
- The main changes in the somatotropic function of the hypophysis gland are related to the increase and decrease of the hormone. Gigantism and acromegaly develop as a result of chronic hyperproduction of STH from the somatotroph cells of the anterior part of the hypophysis gland. Hyperproduction of STH in the period of osteogenesis before the closure of

STH hipersekresiyası

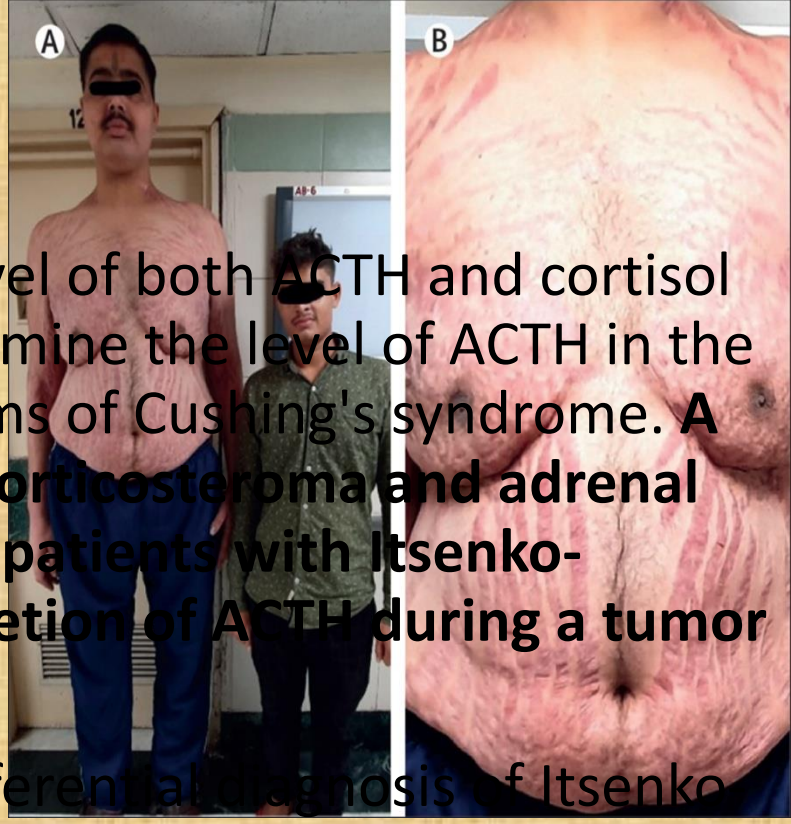


Somatotropic hormone (STH) is a peptide of 191 amino acids secreted by the anterior lobe of the hypophysis gland. The daily dose of this hormone is 500 mcg. STH stimulates protein synthesis, mitotic division, accelerates lipolysis. Inactivation of the hormone in the blood is carried out by hydrolysis. Compared to other hormones, STH is secreted from the hypophysis gland in a small amount (5-15 µg of tissue). The main function of STH is to stimulate neck elongation in the body. STH stimulates the transport of amino acids into the cell in an insulin

The concentration of insulin-like growth factor I in the blood

- The concentration of insulin-like growth factor I in the blood varies depending on STH and T4 hormone. A low concentration of insulin-like growth factor I is found in severe forms of T4 deficiency. Replacement therapy with sodium-levothyroxine leads to an increase in the concentration of insulin-like growth factor I in the blood to normal. Another factor that ensures the concentration of insulin-like growth factor I is nutrition. In children and adults, meeting the protein-energy supply of the body in accordance with the demand is an important condition for ensuring the normal concentration of insulin-like growth factor I. Although the concentration of insulin-like growth factor I in the blood of children with acute protein deficiency decreases, it normalizes after proper nutrition. Liver failure, inflammatory diseases of the intestines, and kidney failure also cause a decrease in the concentration of insulin-like growth factor I in the blood.

Itsenko-Cushing’s disease



- Itsenko-Cushing’s disease is characterized by an increase in the level of both ACTH and cortisol and 17-OKS (oxyketosteroids) in the blood. It is important to determine the level of ACTH in the differential diagnosis of Itsenko-Cushing’s disease and various forms of Cushing's syndrome. **A decrease in ACTH secretion is noted in patients diagnosed with corticosteroma and adrenal gland cancer. The level of this hormone in the blood increases in patients with Itsenko-Cushing’s disease and ectopic ACTH syndrome (pathological secretion of ACTH during a tumor of the bronchi or thymus gland).**
- Corticotropin-releasing hormone (CRH) test is used in the differential diagnosis of Itsenko-Cushing’s and ectopic ACTH syndrome. Thus, in the case of Itsenko-Cushing’s disease, the injection of CRH leads to a significant increase in ACTH secretion, while in non-hypophysis localized tumors that secrete ACTH, the level of ACTH does not change because there are not receptors against CRH.
- An increase in the level of ACTH in the blood occurs during cancer of the thyroid gland and thymus, ovaries and mammary gland and cancer of the stomach and large intestine. Diagnostically, in ACTH-ectopic syndrome, the level of ACTH in the blood is more than 44 pmmol/l and it is clinically important to study the level of the hormone in different veins.

Adrenocorticotropic hormone (ACTH) in blood plasma

The level of ACTH in blood plasma consists normal as < 22 pmmol/l at 8:00 am and <6 pmmol/l at 10:00 pm.

ACTH - adrenocorticotropic hormone consists of 39 amino acids, and mainly controls the synthesis and secretion of hormones of the adrenal cortex - cortisol, cortisone, corticosterone. In addition, the secretion of progesterone, estrogen and androgens also increases. Also, the effect of ACTH and its components on memory motivation and acquisition processes has been proven.

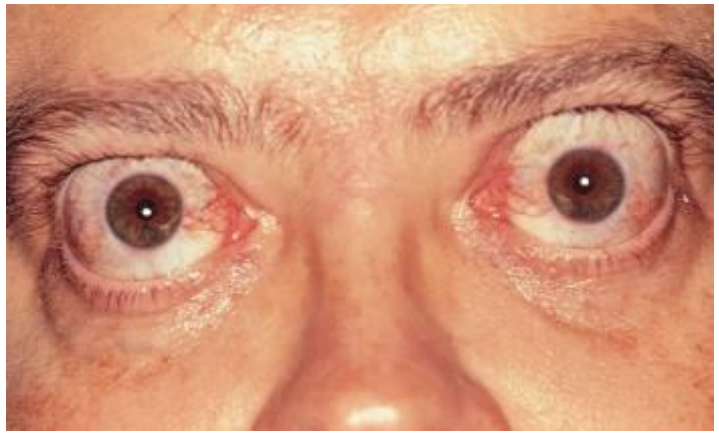
Excretion of ACTH into the blood occurs with a daily rhythm, the maximum concentration is recorded at 6 am, and the minimum concentration is recorded at 10 pm. Stress is considered a strong stimulator of this hormone.

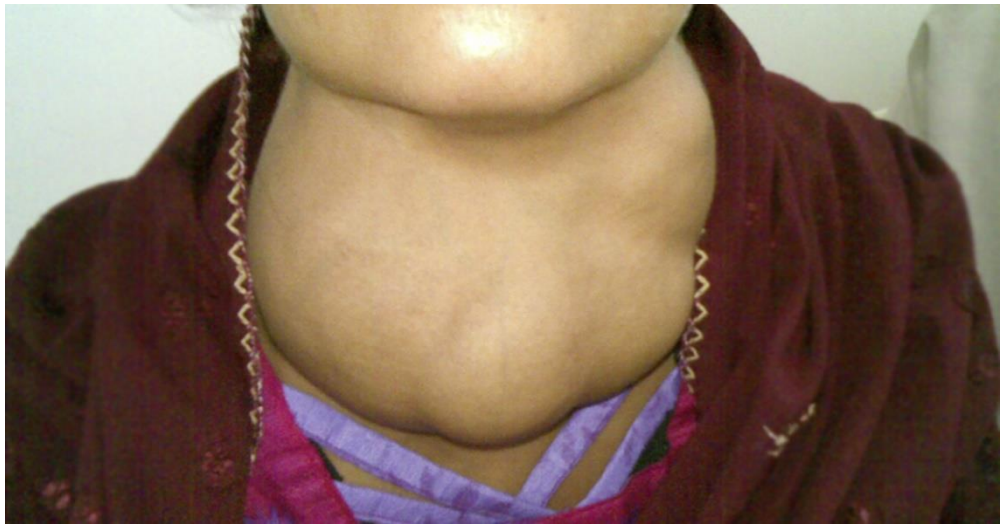
An increase in the level of ACTH in the blood occurs during cancer of the thyroid gland and thymus, ovaries and mammary gland and cancer of the stomach and large intestine. Diagnostically, in ACTH-ectopic syndrome, the level of ACTH in the blood is more than 44 pmmol/l and it is clinically important to study the level of the hormone in different veins.

tropin hormone (TTH) in the blood

borns is normally 3-20 mME\l, in adults 0.2-3.2

one (TTH) is a glycoprotein secreted by the which mainly stimulates the synthesis of hyronine and secretion from the thyroid gland. , the TTH level increases. At this time, the ncentration of free T4 and T3 in the blood, and range in subclinical mild hypothyroidism, osis. A low level of TTH in hypothyroidism sis or hypothalamic dysfunction and rules out n





Indicators characterizing reproductive function

The reproductive system consists of certain structures of the hypothalamus and hypophysis and target cells /codocyte/ (fallopian tubes, uterus, etc.). The elements of the reproductive system interact with information signals that allow them to function in a complete way.

Studying the level of reproductive system hormones plays an important role in discovering the causes of male and female infertility based on hormonal regulation disorders.

Classification of hormones that regulate reproductive function:

- Hypothalamus: Gonadotropin releasing hormone, Prolactin RH, GRIQ, PRIQ
- Hypophysis: Luteinizing (LH), Follicle stimulating (FSH), prolactin

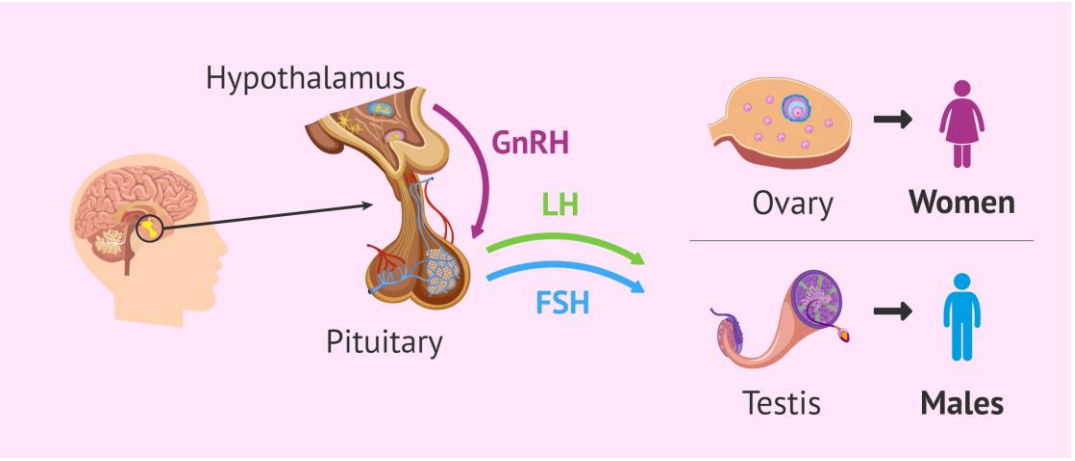
Ovaries: estrogen, gestagen, androgen, inhibin

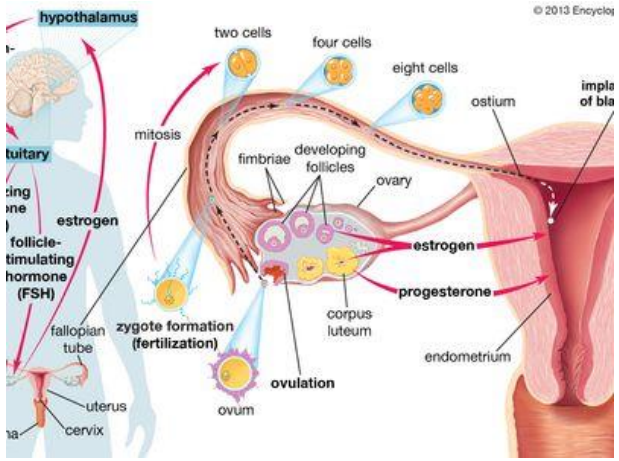
Placenta: estrogen, gestagen, chorionic hormone, prolactin

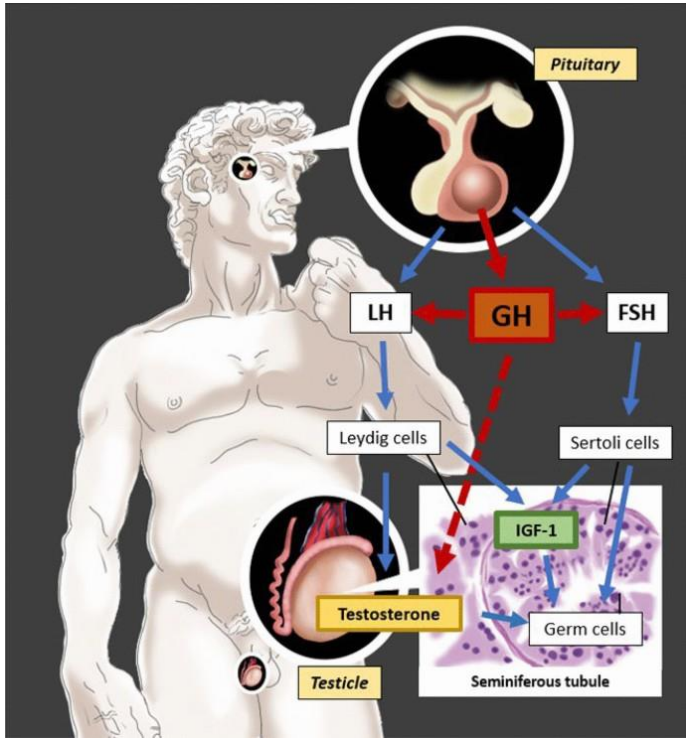
Seminiferous tubules: androgen, inhibin

Adrenal gland substance: androgens, estrogens

Qonadotrop hormonun hipo və hiperfunksiyası







Follicle stimulating hormone (FSH)

- **Follicle stimulating hormone (FSH)** Follicle-stimulating hormone (FSH) is secreted from the anterior lobe of the hypophysis gland and regulates follicle maturation and ovulation readiness in women. In men, this hormone ensures the development and function of seminiferous tubules and seminiferous tubules, and the process of spermatogenesis in Sertoli cells.
-

Prolactin in blood serum. Macroprolactin

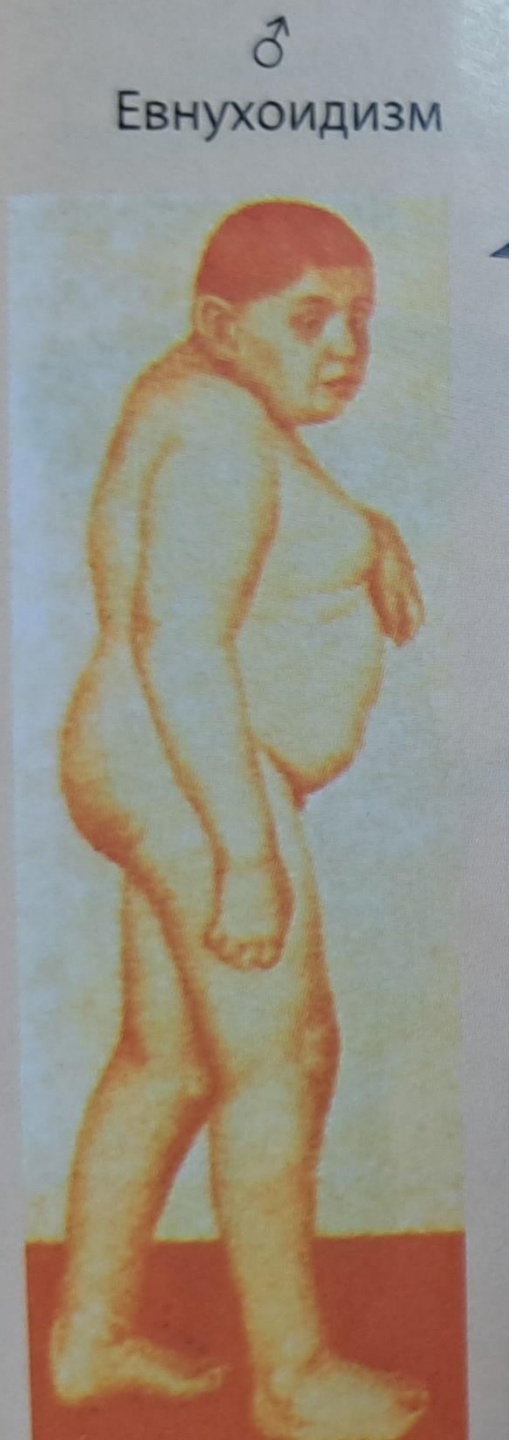
The normal level of and 58-475 mME/l in ma

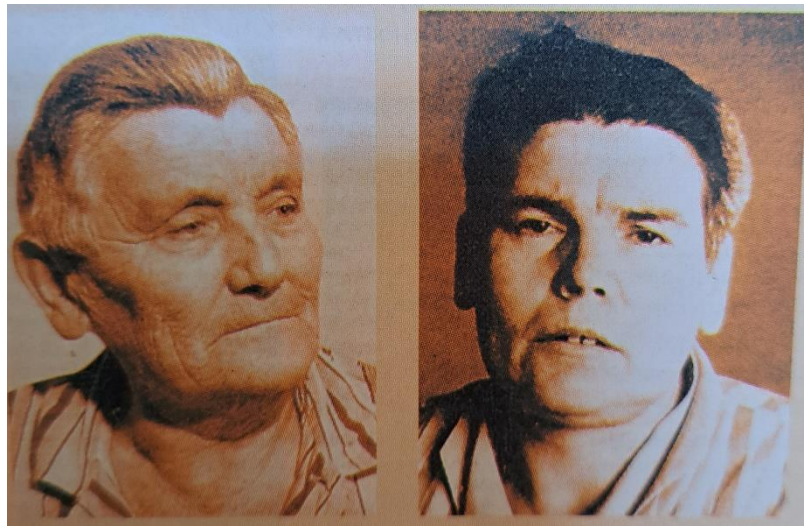
Prolactin is synthesized in the lactogenic cells of the anterior lobe of the hypophysis. Its synthesis and secretion are carried out under the stimulating-inhibiting influence of the hypothalamus. In addition to the hypophysis, the decidual membrane (because amniotic fluid contains prolactin) and the endometrium also synthesize prolactin. Together with estradiol, prolactin in female affects the growth and function of the mammary glands, causing lactation. The role of this hormone in male is unknown. Normally, macroprolactin is 0-10% of total prolactin.

Since macroprolactin does not have biological activity, its excess amount in the blood - macroprolactinemia is not accompanied by clinical symptoms and it's a laboratory diagnostic phenomenon that does not require treatment. A macroprolactin level of more than 30% in the blood gives reason to talk about macroprolactinemia. The following are taken as the basis for the macroprolactin test:

- After the precipitation of immune complexes, the result of the level of prolactin in the blood is up to 40% of the initial indicator
- After the precipitation of immune complexes, the result of the level of prolactin in the

Hipofizar Hipoqonadizm



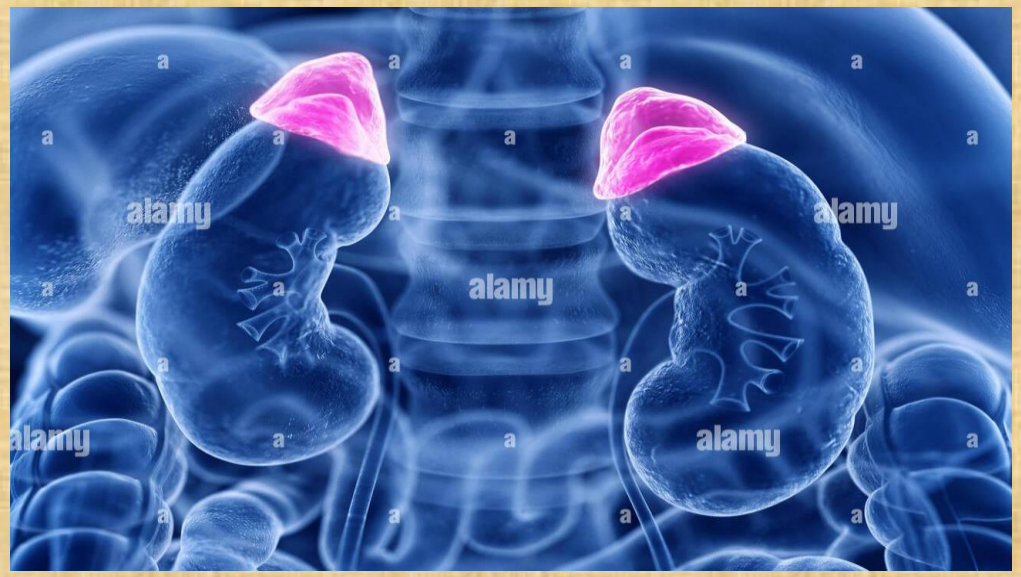


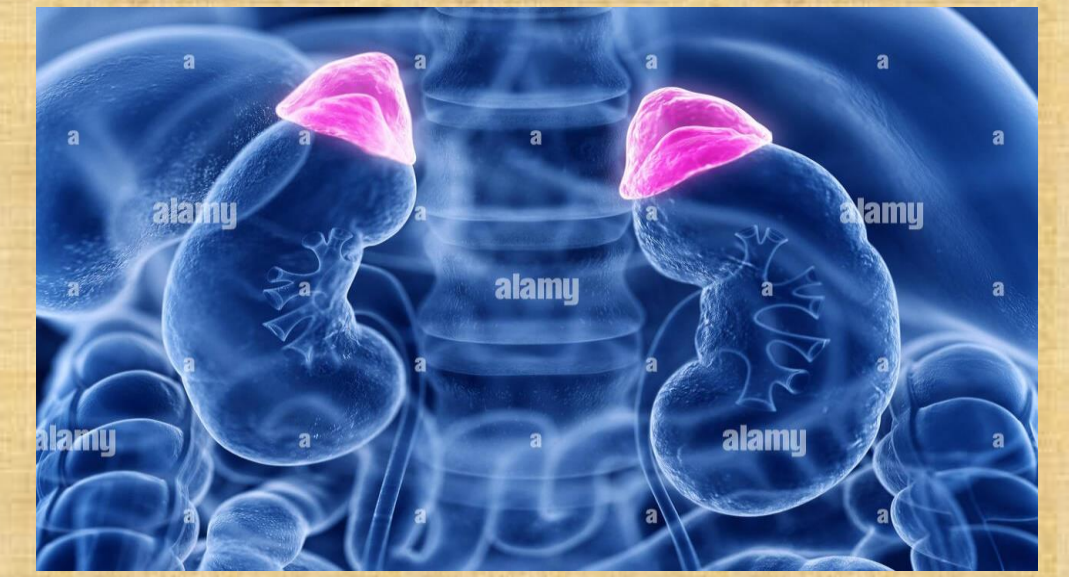
Hypothalamus- hypophysis - indicators characterizing the adrenal gland system

- The anterior lobe of the hypothalamus hypophysis and the cortex of the adrenal gland are functionally united in the hypothalamus-hypophysis-adrenal gland system.
- The adrenal gland is composed of a shell and a brain substance that perform various functions. Histologically, the cortical substance consists of 3 layers - peripheral glomerular, middle layer and the lowest reticular layer. Only aldosterone is secreted from the glomerulus. The other two layered and reticular layers form a functional complex and secrete the main hormones of the adrenal cortex (glucocortoids and androgens).

The adrenal gland

al





The following syndromes are distinguished during diseases of the adrenal cortex:

- - Hypercorticism:
- - Itsenko-Cushing’s disease (hypothalamus-hypophysis disease);
- - Cushing's syndrome - corticosteroma (malignant or benign) or bilateral small-nodular dysplasia of the cortex substance of the adrenal gland;
- - ACTH-ectopic syndrome: tumors of bronchi, pancreas, thymus, liver, ovaries secreting ACTH or corticotropin-releasing hormone;
- - Feminization and virilization (synthesis of estrogen or androgens)
- - Hypocorticism:
- - Primary
- - Secondary
- - Tertiary
- - Dysfunction of the adrenal cortex
- Adrenogenital syndrome (AGS)

Cortisol in the blood

The concentration of free cortisol in the blood is normally at 8.00-200-700 nmol/l, at 20.00 55-250 nmol/l. The difference between morning and evening concentrations should be more than 100 nmol/l. During adrenal insufficiency, the level of cortisol in the blood decreases. During adrenal gland deficiency I and II, the amount of cortisol, free cortisol, 17-oxycorticosteroid (17-OKS) in urine also decreases. In non-acute adrenal insufficiency, the concentration of cortisol in the blood Should not decrease due to the weakening of the metabolism of the hormone. In such doubtful cases, functional tests are performed with ACTH preparations. These include a single intramuscular injection of corticotropin and an intravenous injection of synactin. In healthy people, the level of cortisol increases more than 2 times after conducting these calculations. Absence of reaction to the injection of preparations into the body indicates the presence of I deficiency of the adrenal gland.

Adrenal gland response to ACTH injection is preserved during type II adrenal insufficiency. However, long-term adrenal insufficiency leads to atrophy of the adrenal medulla, in which it loses its ability to increase glucocorticoid secretion against ACTH administration.

An increase in the concentration of cortisol in the blood is observed during Itsenko-

Free cortisol in urine

The concentration of free cortisol in urine is normally 30-300 nmol/l or 15-30 nmol/l daily creatinine. Cortisol that is not bound to blood plasma proteins (free) is filtered from the renal glomeruli and excreted in the urine. Free cortisol in blood plasma is one of the main biologically active forms of the hormone. The concentration of free cortisol in the daily urine directly reflects the level of free cortisol in the blood. The determination of the level of free cortisol in the daily urine is the main test for the diagnosis of adrenal gland hyperfunction. When evaluating this test, it is necessary to take into account that the concentration of the hormone will increase during physical load in obese patients.

The level of free cortisol in the urine of patients with renal failure is reduced and does not reflect the measures of cortisol secretion. Most patients with Itsenko-Cushing's syndrome have an increased level of free cortisol in their urine. A very high level of free cortisol in the urine confirms the diagnosis of carcinoma of the adrenal gland.

17-oxyketosteroids in urine

The concentration of 17-oxyketosteroids (17-OKS) in urine is normally 5.2-13.2 µmol/day.
17-oxyketosteroids are metabolites of steroid hormones, cortisol (F), cortisone (E) 11-deoxycortisol (S), tetrahydrocortisol (THF), tetrahydrocortisone (THE) tetrahydro-11-deoxycortisol (THS).

The main component of urinary 17-oxyketosteroids are tetrahydrometabolites of cortisol. I should be noted that the daily urinary excretion of 17-oxyketosteroids is not diagnostically significant as it depends on the patient's weight and other factors.

Urinary excretion of 17-oxyketosteroids is reduced in chronic adrenal insufficiency. In suspicious cases, a test with ACTH preparations is carried out. Excretion of 17-oxyketosteroids by 1.5 times or more on the first day after ACTH injection, and even more on the 3rd day, indicates the reserve function of the adrenal cortex, and the diagnosis of adrenal cortex I deficiency is denied.

Increased urinary excretion of 17-oxyketosteroids is observed in Itsenko-Cushing's disease Cushing's syndrome, alimentary-constitutional and hypothalamo-hypophysis obesity. Liddle's dexamethasone test is performed for the differential diagnosis of Itsenko-Cushing's disease and obesity. A decrease in urinary excretion of 17-oxyketosteroids by 50% more than the norm is considered against Itsenko-Cushing's disease. At this time, the level of 17-oxyketosteroids

Urinary 17-ketosteroids (17-KS)

17-ketosteroids (17-KS) in urine are normal: female 20-40 years old: 5-14 mg/day, male: 20-40 years old 9-17 mg/day, after 40 years of age, a decrease in 17-KS excretion is observed.

Urinary 17-ketosteroids (17-KS) are metabolites of androgens secreted from the retina of the adrenal gland and the gonads. Only a small part of the 17-ketosteroids excreted in the urine (17-KS) originates from the precursors of glucocorticoids. **Evaluation of 17-ketosteroids in urine (17-KS) serves to evaluate the functional activity of the adrenal cortex**. A decrease in 17-ketosteroids excreted in urine is observed in chronic insufficiency of the adrenal cortex, and its increase is observed in androsteroma, Itsenko-Cushing's disease or Cushing's syndrome, in congenital hyperplasia of the adrenal cortex.

For congenital hyperplasia of the cortex substance of the adrenal gland, it is important to note the increase in the excretion of 17-ketosteroids in the urine, as well as the increase in the ACTH activity of the plasma, the lower limit of

methasone test (test with 8 mg of dexamethasone, **Liddle's large test**) when a **small c**
ve, it is performed to determine the exact form of pathological hypercorticism, i.e. for
Itsenko-Cushing's disease and Cushing's syndrome. After examining the baseline level
rine 17-oxycorticosteroid (17-OKS), patients are prescribed **dexamethasone** orally fo
). Urine 17-oxycorticosteroid (17-OKS) is taken again on the 3rd day of taking the dru
ACTH in the blood is determined at 8 o'clock in the morning on the 4th day of taking d
ion of 8 mg of dexamethasone causes suppression of cortisol and ACTH concentration
agnosed with hypophysis hypercortisolism, as Itsenko-Cushing's disease patient.
sult reflects that the main problem lies in the anomaly of the hypothalamo-hypophysis
, which goes with maintaining the feedback mechanism. The adrenal form of hypercor
ndrome, is characterized by a decrease in the level of ACTH, an increase in the level o
d in the urine. Absence of delay in the level of all three indicators (ACTH, cortisol, 17
tic for ectopic ACTH secretion syndrome.
oxical reaction may occur in adrenal medullary cancer (increased levels of cortisol and

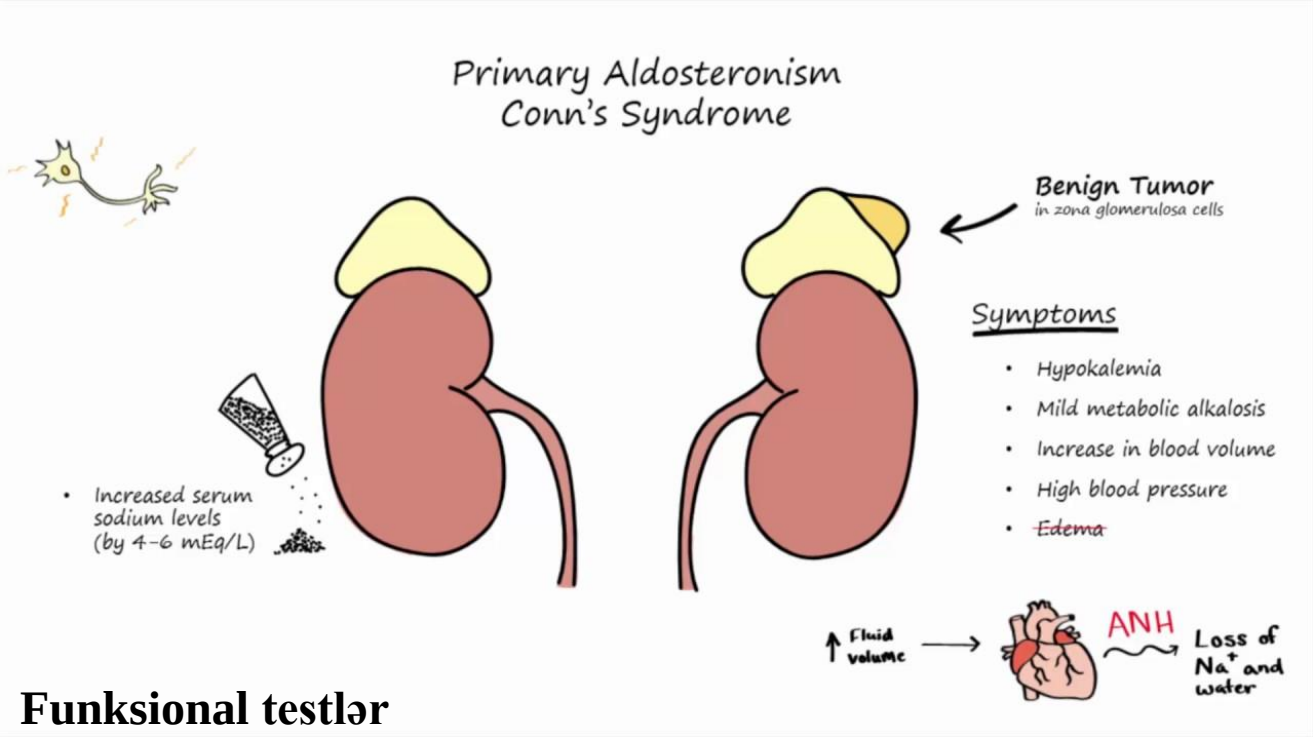
ACTH stimulation test during adrenal gland insufficiency.

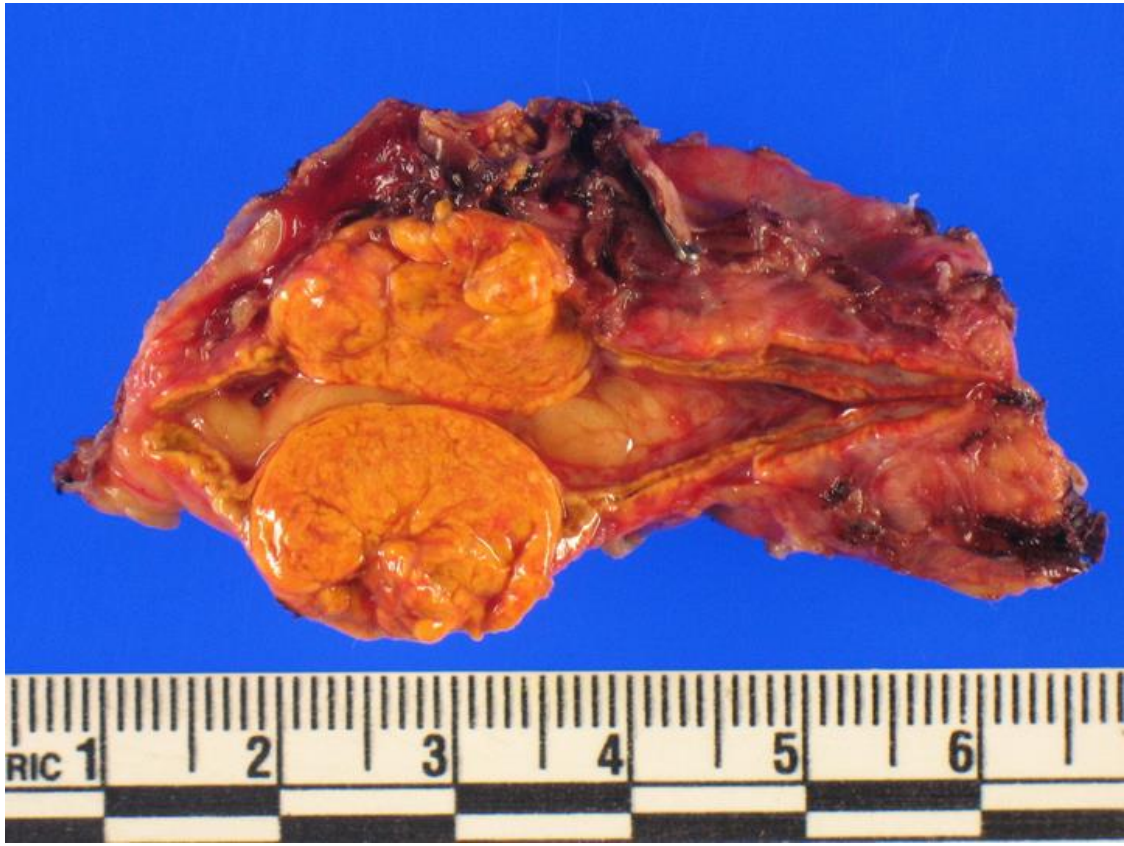
It allows to detect the hidden form of adrenal gland insufficiency, to carry out differential diagnosis between adrenal gland insufficiency I and II insufficiency. At present, ACTH synthetic analogue "Synacthen" or its prologized form " Synacthen -depot" is used for the test.

A short trial with " Synacthen ". It is performed to evaluate the reserve function of the adrenal gland.

Hipofiz-böyrəküstü vəzi sisteminin bəzi xəstəliklərinin laborator -diaqnostik kriteriyaları

Aldosteromalar, adenokarsinoma, hormonal qeyri-fəal şişlər, subklinik I-li aldosteronizm





Funksional testlər

Deksametazonla (2 və 8 mq) funksional test

2. Verospironla sınaq

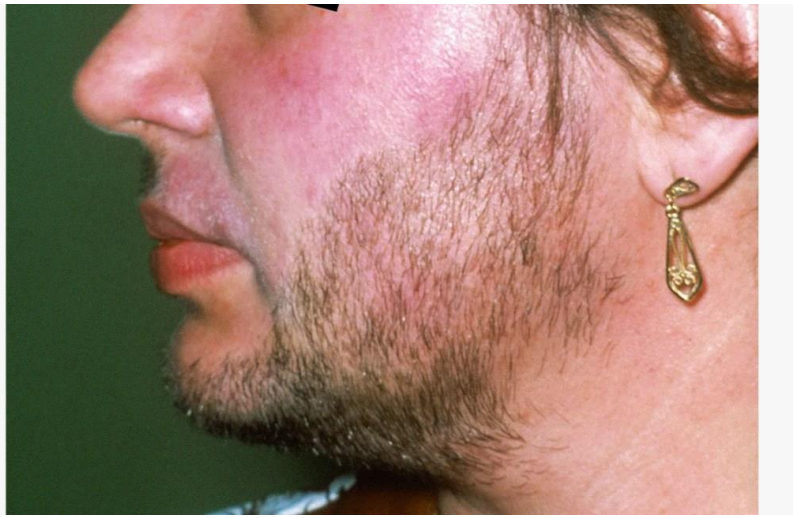
3. Ortostatik sınaq

4.Sinakten-depo sınağı (böyrəküstü vəzin qabıq maddəsinin anadangəlmə hiperplaziyasının silinmiş formasına şübhə olduqda aparılır)

Congenital hyperplasia of the adrenal cortex with a defect of 21-hydroxylase

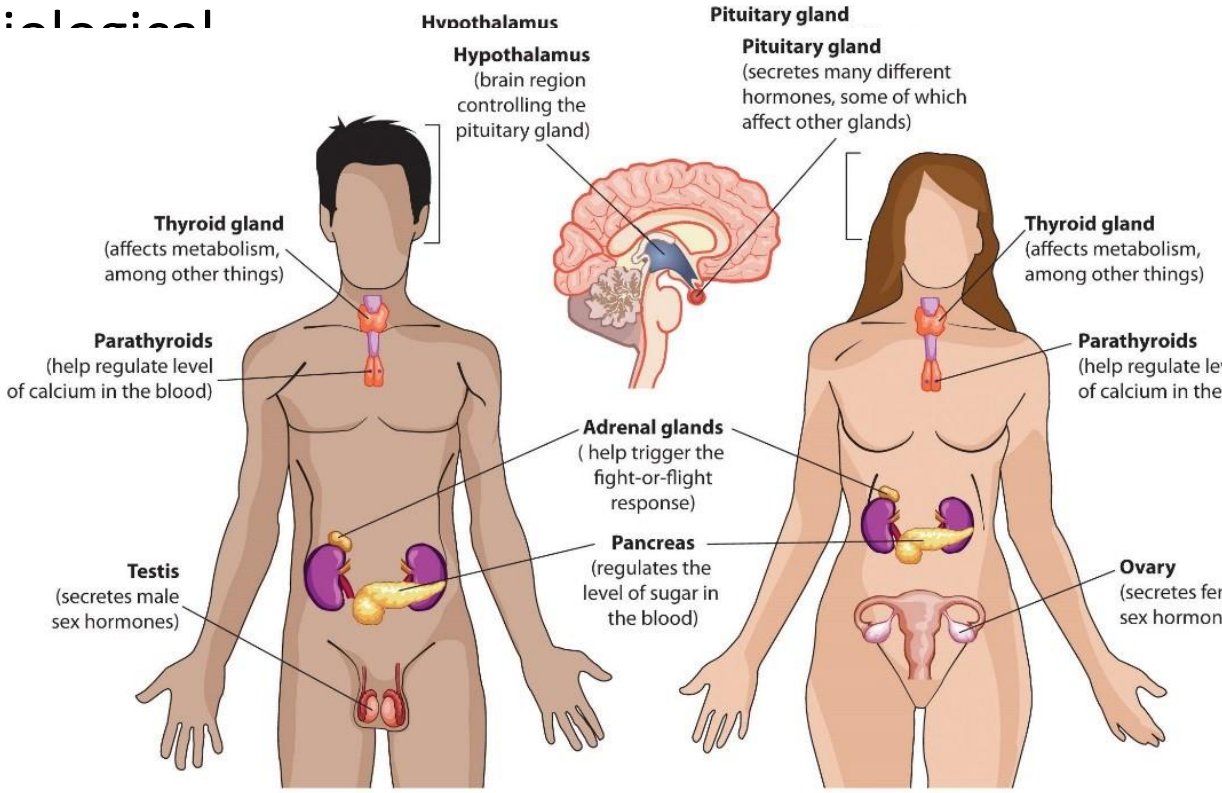
is diagnosed based on the clinical signs of Viral syndrome, with a blood level of 17-OHP exceeding 5 mg/ml in the early follicular phase of the menstrual cycle.

Studying the level of 11-deoxycorticosterone (DOC), 11-deoxycortisol (S), which are substrates of 11 β-hydroxylase, **plays a major role in the laboratory diagnosis of the form of congenital hyperplasia of the adrenal gland with a deficiency of 11 β-hydroxylase.** Information about these hormones in the blood can be obtained by the **method of doing high performance liquid chromatography** of corticosteroids.



The level of estradiol in the blood

The main representative of estrogens is estradiol, which has the highest biological activity. Estrone is obtained from estradiol in an enzymatic way, and its b
activity is not high and its level increases
pregnancy. At this time, estrone is synthesiz
dehydroepiandrosterone-sulfate formed in the
cortex of the fetus. Thus, estrone is an indica
characterizes the state of the fetus. In the fema
estradiol is produced in the ovaries, granulosa
membrane of the follicles. After the begir
pregnancy, the synthesis of estrogen is car
massively by the couple. Adrenal gland and p
sebaceous glands are other organs where
synthesis is carried out. Determination of estradiol concentration is important in evaluating the function of the ovaries (by aromatizing androgens). There is no exact



Testosterone in the blood

Testosterone is an androgen hormone, which ensures the emergence of secondary sexual characteristics in men. The main source of testosterone is the Leydig cells of the seminiferous tubules. Testosterone stimulates spermatogenesis, the growth and function of the gonads, and ensures the development of the genitals and testicles. In particular, it has an anabolic effect on bones and muscles. Testosterone stimulates erythropoiesis by directly affecting the bone marrow and activating the synthesis of erythropoietin in the kidneys. Testosterone provides libido and potency. Testosterone synthesis is regulated by luteinizing hormone secreted from the hypophysis gland. It is the only hormone that ensures sexual maturity in male. Blood concentration increases after physical activity. Since the determination of free testosterone does not depend on the concentration of steroid-binding globulin (SBG), there is an indication for the determination of this hormone in diseases accompanied by an increase in SBG (hyperthyroidism, hyperestrogenia,


Thank
You