

Cushing's syndrome

Template:Infobox - disease Cushing's syndrome is caused by the long-term excessive action of *high levels of cortisol* in the body.

🔗 For more information see *Glucocorticoids*.

The so-called Cushing's Trias is a circulatory response to intracranial hypertension

Etiopathogenesis

Depending on whether Cushing's syndrome is linked to high or low ACTH levels, we divide it into ACTH-dependent and ACTH-independent, which makes sense in diagnosis and therapy.

- **ACTH dependent Cushing's syndrome'** (secondary Cushing's syndrome).
 - **Central Cushing's syndrome:** increased production of ACTH in the pituitary gland most commonly pituitary adenoma (sometimes referred to as **Cushing's disease**), causing abnormal high adrenal stimulation.
 - **Ectopic Cushing syndrome:** paraneoplastic production of ACTH, most often by small cell lung carcinoma or bronchial carcinoid.
- **ACTH independent Cushing's syndrome'** (primary Cushing's syndrome).
 - **Peripheral Cushing's syndrome** is an overproduction of glucocorticoids in the adrenal cortex, most often caused by a unilateral tumor of the adrenal gland (adenoma or carcinoma), rarely the involvement is bilateral.
 - **Iatrogenic Cushing's syndrome** during long-term glucocorticoid therapy.



Speeches



Cushing's disease has many manifestations due to an excessive concentration of corticosteroid hormones in the body.

- **Central obesity:** There is an increase in appetite and body weight, obesity occurs with a maximum of fat in the abdomen and face, on the other hand, the limbs tend to be very thin (muscle atrophy). The face has a characteristic round shape (*moon-shaped face*), and there is an increased amount of fat on the neck (*buffalo hump*). The patient may thus have the appearance of a chestnut stick figure (skinny limbs, enlarged abdomen and face)

- **Skin:** Stretch marks appear on the skin of the abdomen as a result of reduced skin resistance (glucocorticoids reduce collagen production). Stretch marks are purple in color. The skin is atrophic (parchment skin), easily injured, and recurrent skin infections and subcutaneous bleeding are also characteristic.
- **Bones:** High levels of glucocorticoids reduce the proliferation of osteoblasts (thus the density of bone mass), osteoresorption is activated and osteoporosis occurs.
- **Glucose metabolism:** Insulin resistance develops up to diabetes mellitus type 2. The so-called "early steroid diabetes" can also develop, which usually disappears after stopping corticoids.^[1]
- **Protein metabolism:** Cortisol has a catabolic effect on protein metabolism, leading to *steroid myopathy* (muscle pain and muscle atrophy), which mainly affects the girdle muscles.
- **Blood pressure:** Secondary arterial hypertension – thanks to excess cortisol, mineralocorticoid receptors are activated and at the same time the sensitivity of the vascular wall to pressor effects increases.
- **Psychic:** Depression, concentration and memory disorders, fatigue, emotional lability.
- **Other symptoms:** Decreased libido in both sexes, in women, in addition, **androgenic changes** (hirsutism, menstrual disorders up to amenorrhea, infertility, acne) may occur. If the high level of corticoids in the body is additionally combined with the use of anti-inflammatory drugs with acetylsalicylic acid, there is a significant risk of developing gastric ulcers.

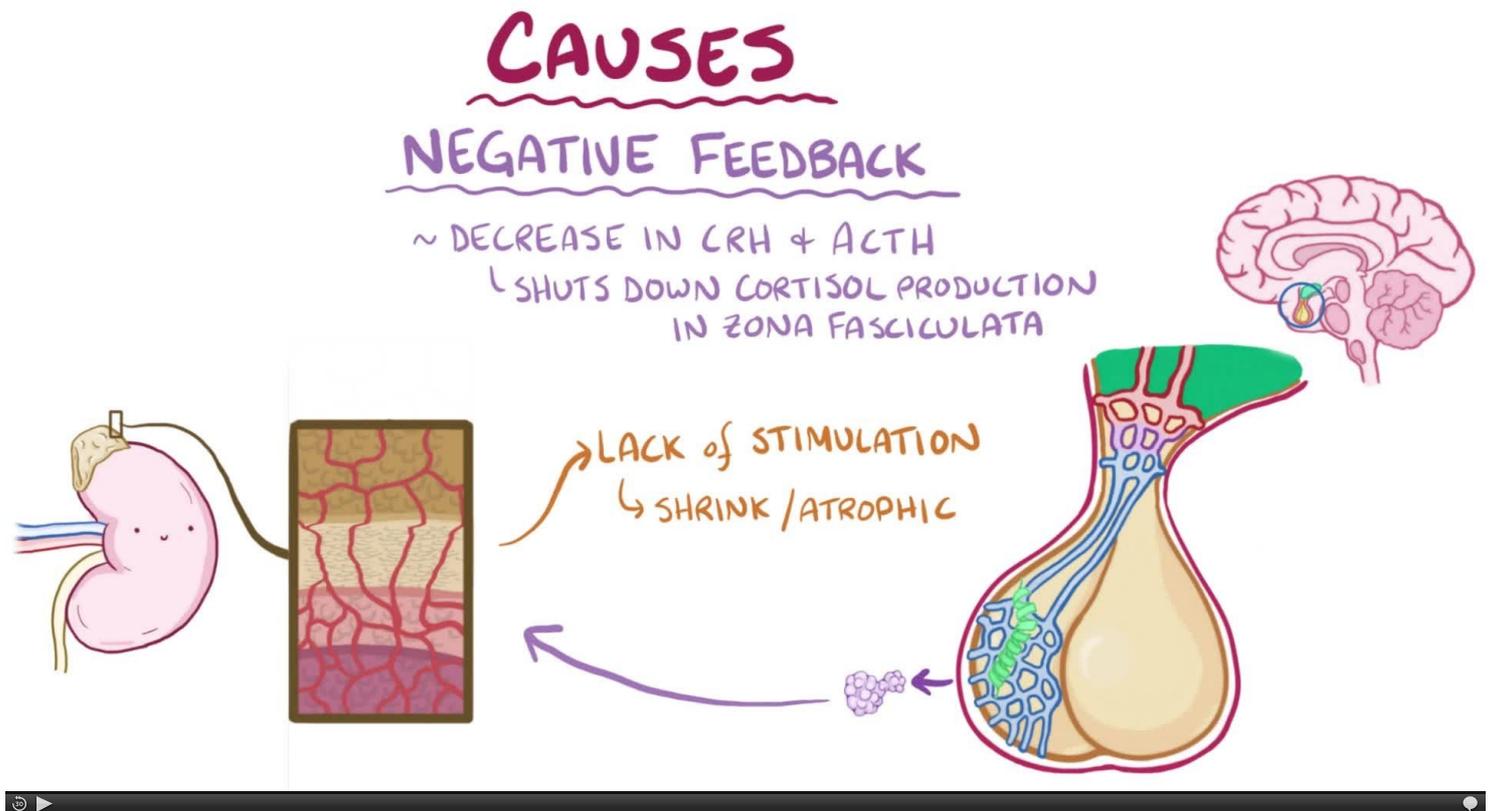
Diagnosics

- Laboratory determination of free cortisol waste in urine in 24 hours,
- examination of serum cortisol,
- dexamethasone test (stronger to distinguish between central and peripheral forms),
- MRI (adenoma diagnosis).

Treatment

- **Pituitary adenomas**
 - Neurosurgical therapy: removal of pituitary adenoma.
 - Stereotactic radiation therapy (in case of unsuccessful surgical therapy): Leksell's gamma knife.
- **Inhibition of steroidogenesis:** ketoconazole, metyrapone.
- **Ectopic (paraneoplastic) Cushing's syndrome:** removal of the primary tumor.
- **Primary (peripheral) Cushing's syndrome:** adrenalectomy (in case of bilateral adrenalectomy damage, hypocorticalism is treated with glucocorticoid supplementation), possibly combined chemotherapy.
- **Iatrogenic Cushing's syndrome:** if not necessary, stop administering high doses of corticoids, or at least reduce them. This of course applies on the condition that the doctor evaluates the adverse effects of corticoids as more significant than their beneficial effects in the given patient.

Video



Links

Related Articles

- Pituitary adenoma
- Conn's syndrome
- Glucocorticoids
- Pituitary function test
- Endocrine hypertension

Source

- ŠTEFÁNEK, Jiří. *Medicine, diseases, studies at the 1st Faculty of Medicine, UK* [online]. [cit. 2011-04-14]. <<https://www.stefajir.cz/index.php?q=cushinguv-syndrom>>.

References

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- CZECH, Richard, et al. *Intern*. 1. edition. Prague : Triton, 2010. 855 pp. pp. 327-329. ISBN 978-80-7387-423-0.

References

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