roid hormone overproduction. It is the most common cause of hyperthyroidism, with 20-50 cases per 100 000 persons annually. The incidence peaks at 30-50 years. The annual incidence of Graves' ophthalmopathy is 18/100 000 women and 3/100 000 men yearly. The diagnostic work-up of hyperthyroidism is presented below (Smit and Hegedus, N Engl J Med, 2016). Treatment of hyperthyroidism is initiated by an antithyroidal drug (methimazole/carbimazole is first-line, propylthiouracil is an alternative option). The final choice of treatment (12—18 months of antithyroid drug therapy, radioiodine or total thyroidectomy) should be individually tailored. Patients should be advised to stop smoking. In this session, we will discuss these different treatment options, treatment of hyperthyroidism during pregnancy, as well as the diagnosis and treatment of Graves' ophthalmopathy.

KEYWORDS: Graves' disease, thyroid-hormone receptor antibodies, hyperthyroidism.

ADRENAL INSUFFICIENCY

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Adrenal insufficiency (AI) is a condition associated with decreased secretion of steroid hormones from adrenal cortex resulting in a decrease of their biological effects on cells, tissues and organs of human body. Primary AI is a consequence of destruction of the entire adrenal cortex resulting wit loss of both, glucocorticoid and mineralocorticoid activity. By contrast, secondary adrenal insufficiency reflects an inability to sufficiently stimulate adrenal cortex by ACTH that results mainly in glucocorticoid deficiency whereas mineralocorticoid secretion is largely preserved. Among peripheral (primary) causes autoimmune destruction of adrenal cortex is most frequent in developed countries. Other possible peripheral causes of AI are: infections, bilateral metastases, bilateral adrenalectomy, adrenoleukodystrophy, amyloidosis, hemochromatosis, vascular causes (bilateral adrenal hemorrhage or thrombosis). Central (secondary) causes of adrenal insufficiency are: structural lesions of the hypothalamus or pituitary gland (tumours, infiltrating disorders, irradiation, lymphocytic hypophysisits) and some other rare conditions. One of most frequent causes in clinical practice is functional suppression of HPA axes caused by exogenous glucocorticoids. Proper replacement therapy is essential for patients' survival and also for maintaining their quality of life a normalizing morbidity and mortality. Treatment of acute AI should be performed and ICU setting with a close monitoring of patients. It has to start with immediate intravenous application of 100 mg of hydrocortisone followed by daily dose 200-400 mg continuously or divided in 3-4 partial doses. At the same

time volume resuscitation with intravenous saline infusion and hypoglycaemia correction with intravenous glucose has to be carried out. Treatment of chronic AI of all causes consists of oral administration of glucocorticoid, commonly hydrocortisone in basal doses in approximate dose of $10-15 \text{ mg/m}^2$. Basal dose has to be adjusted before and during stress conditions properly. In primary AI patients usually require an addition of mineralocorticoid (Fludrocortisone in oral dose of 0.5-2 mg daily). On the other hand in cases with secondary AI hydrocortisone could be replaced by selective glucocorticoid in equipotent dose (e.g. prednisone or prednisolone). Proper and careful education of patients is essential and patients have to be equipped with steroid emergency card.

KEYWORDS: adrenal insufficiency, replacement therapy, exogenous glucocorticoids.

MALE HYPOGONADISM

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The scientific and medical topic of male hypogonadism is one of the most growing and discussable problems in endocrinology in our days. Hypogonadism in male patients defined as testosterone level decrease in serum associated with specific symptoms and/or signs can be observed in case of abnormal changes in testes and/or pituitary such as Klinefelter syndrome, Kallmann syndrome and also in male patients with idiopathic, metabolic or iatrogenic disorders resulting in androgen deficiency. Among the classical reasons pointed above the growing number of hypogonadal elderly men are now in focus of clinicians. The main guidelines' for diagnosing and treatment options of hypogonadal adolescents and adults will be discussed. Also the introduction into Russian Guidelines for diagnosing and treatment of testosterone deficiency will be discussed during the talk.

KEYWORDS: male hypogonadism, Klinefelter syndrome, Kallmann syndrome, testosterone deficiency.

BONE BIOLOGY AND FUTURE TARGETS FOR OSTEOPOROSIS TREATMENT

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Novel therapeutic approaches to osteoporosis have not only provided better treatment modalities but also shed new light on the cellular and molecular mechanisms by which trabecular and cortical bone skeletal homeostasis is regulated. Skeletal homeostasis is ensured by the balanced activities of bone resorption and bone formation in bone remodeling. Osteoclasts are responsible for the resorption of bone, but also for the local recruitment