options of chromaffin tumors. It emphasizes important aspects of preparing for surgery, and discusses prognosis.

KEYWORDS: pheochromocytoma, adrenal tumors, hypertension, management.

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THYROID DISORDERS ASSOCIATED WITH IODINE DEFICIENCY IN PRACTICE OF ENDOCRINOLOGISTS

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Iodine deficiency (ID) impairs thyroid hormone production and has many adverse effects throughout the human life cycle. The most serious effect of ID is mental impairment in children, adolescents and adults. Goiter is the most visible and well known effect of insufficient iodine nutrition. Management of goiter and other thyroid disorders caused by ID is an important part of routine clinical practice of endocrinologists. Moreover, in dealing with thyroid disorders the clinicians should be well aware of changing patterns of iodine intake to make necessary amendments to their clinical practices. Effective goiter prevention program (combination of massive use of iodized salt and distribution of iodine supplements in vulnerable groups of population) was conducted in the USSR until 1990 and reduced goiter prevalence to nearly sporadic level. Collapse of iodized salt production in 1991—1992 led to a significant increase in goiter morbidity, especially in areas with severe ID. It took nearly half decade before this negative trend had been realized and another 10 years or more before situation had improved in the former USSR countries that had adopted universal salt iodization (USI) strategy. However, this progress has been much less spectacular in Russia and Ukraine that are still relying only on a voluntary use of iodized salt. In Russia, certain regions (Moscow, Tyumen, St.-Petersburg) with move advanced voluntary salt iodization programs may have median UIC in children in the optimum range (100—300 mcg/l). In other regions and, especially, in rural areas ID still remains widespread. Several sub-national surveys conducted in Russia regions (oblasts) in the past 10—15 years showed mild-to-moderate ID (median UIC in the range of 40 to 80 mcg/l). This uneven pattern of iodine nutrition provides another challenge to endocrinologists who should adapt their clinical strategy in dealing with thyroid disorders to potential status of iodine deficiency (or sufficiency) in the given territory. Thus, major benefits of increasing iodine intake though salt iodization in populations with mild-to-moderate ID are decrease in prevalence of goiter, thyroid autonomy and thyrotoxicosis in adults and increase in IQ in children. In the situation of optimum iodine nutrition populations, especially children, are better protected from radioactive iodine exposure in case of nuclear accident. These benefits occur at the expense of a small increase in the prevalence of subclinical hypothyroidism in adults that could be minimized by avoiding excessive iodine intakes.

KEYWORDS: iodine deficiency; goitre; universal salt iodization.

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THYROID CANCERS: THE STATE OF THE ART MANAGEMENT

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Thyroid nodules are a common clinical problem, and differentiated thyroid cancer (DTC) is becoming increasingly prevalent. High-resolution ultrasound can detect thyroid nodules in 20—70% of randomly selected individuals, with higher frequencies in the elderly. The main clinical problems concerning thyroid nodules are US-based categorization of the malignancy risk and indications for US-guided fine-needle aspiration biopsy (FNA), cytological classification of FNA samples, the roles of immunocytochemistry and molecular testing applied to thyroid FNA, therapeutic options, and follow-up strategy. Recent advances in research on thyroid carcinogenesis have yielded applications of diagnostic molecular biomarkers and profiling panels in the management of thyroid nodules. Differentiated thyroid cancer (DTC), which includes papillary and follicular variants, comprises the vast majority (>90%) of all thyroid cancers. Most of the detected tumours are very small and have unknown clinical importance and malignant potential. 25% of the new thyroid cancers diagnosed in 1988—1989 were less than 1 cm compared with 40% of the new thyroid cancer diagnoses in 2008—2009. This tumour shift can be explained due to the increasing use of neck ultrasonography or other imaging very often without clear clinical indications and switch last clinical recommendations to less aggressive initial treatment with organ-saving in patients with thyroid microcarcinomas. Nevertheless clinical controversy still exists in many areas of thyroid cancer management. The management of very rare medullary thyroid cancer is now generally based on molecular testing of RET-proto-oncogen mutations. The main directions for further research in the field of thyroid cancer and nodules are optimizing molecular markers for diagnosis, prognosis, and therapeutic targets, improvement of the risk stratification and understanding of the risks and benefits of DTC initial treatment options.

KEYWORDS: thyroid nodules, differentiated thyroid cancer, fine-needle aspiration biopsy.

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GRAVES' DISEASE

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Graves' disease is an autoimmune disease where activating thyroid-hormone receptor antibodies cause thy-

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.

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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 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.

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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.

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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