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Obstructive Sleep Apnea Syndrome in Endocrinologist Practice

Kurbonova Nozima Sobirjonovna
Assistant of the department of General endocrinology and pediatric endocrinology, Samarkand State Medical University, Samarkand, Republic of Uzbekistan

Yusupov Asilbek Otabek o'g'li, Uzoqov Abdikarim Juma og'li,
Bekmurodova Charos Shurat qizi, Ikromov Fayozbek Akmal o'g'li
Student of group 418 of the term pediatric faculty of Samarkand State Medical University, Samarkand, Republic of Uzbekistan

Abstract: It is known that breathing disorders during sleep can be observed in patients with various endocrine pathologies. The development of various sleep apnea has been described in patients complicated by obesity, hypothyroidism, acromegaly, diabetes mellitus, especially diabetic autonomic neuropathy, as well as hypercorticism and polycystic ovary syndrome. The presented review article describes the pathogenetic and pathophysiological mechanisms, clinical manifestations and treatments for obstructive sleep apnea in patients with various endocrinopathies.

Key words: Apnea, snoring, endocrine disorders, obesity.

Introduction. Respiratory disorders in a dream have been known since ancient times. Already in the works of Hippocrates, you can find the first mention of the phenomenon of sleep apnea. Respiratory disorders during sleep were first described in the medical literature in 1936, but detailed study of them only began in the second half of the 20th century, when episodes of respiratory arrest were recorded during sleep when recording electroencephalograms and respiration in patients with Pickwick syndrome [1].

Currently, the prevalence of respiratory disorders during sleep is growing exponentially, which is mainly associated with an obesity epidemic, which is known to be the main predisposing risk factor for the development of this type of disease [2-4].

According to the International Classification of sleep disorders (2005) [31], the spectrum of breathing disorders during sleep includes obstructive sleep apnea syndrome in adults and children, central sleep apnea syndromes (including Cheyn Stokes breathing, infant sleep apnea, etc.), sleep-related hypoventilation syndromes (idiopathic, congenital, etc.), other non-specific respiratory disorders in sleep. Obstructive sleep apnea syndrome (osa) is the most studied form of sleep breathing disorder due to its high prevalence and severe clinical and medico-social consequences [5-7].

Obstructive sleep apnea syndrome is repeated episodes of respiratory arrest during sleep due to the closure of the lumen of the upper respiratory tract, as a result of their increased ability to fall, with preserved movements of the respiratory muscles, often a decrease in the level of oxygen saturation of the blood (desaturations) [8-13].

According to the American Academy of Sleep Medicine [14], obstructive Apnea is a respiratory arrest episode with airflow lasting ≥90% ≥10 seconds. with subsequent movements of the respiratory muscles aimed at restoring breathing. Hypopnea is an incomplete respiratory arrest episode lasting at least 10 seconds. with a decrease in air flow ≥30% with a decrease in blood oxygen saturation ≥4% or with a decrease in air flow ≥50% with a decrease in blood oxygen saturation ≥3% or activation

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reactions/awakenings recorded in the electroencephalogram, which is necessary to increase the tone of the pharyngeal dilator muscles and open the lumen of the upper respiratory tract [15-18].

The most suspicious clinical signs and signs for the presence of obstructive sleep apnea syndrome, daytime excessive drowsiness with rest / active activity, normal, frequent high snoring, stopping breathing during sleep and feeling episodes of suffocation, fatigue, as well as arterial hypertension, increased night urination, decreased libido, depressed mood, headache, concentration disorders and emotional and personal disorders [19-22]. Alcohol consumption or the use of sedative drugs before bedtime leads to a worsening of apnea symptoms. The severity of obstructive sleep apnea is called the respiratory impairment index (IDR), obtained according to a night polysomnographic study, which reflects the degree of deviation from normal respiratory physiology during sleep. IDR is determined by the number of episodes of obstructive Apnea and hypopnea during one hour of sleep, and does not meet the criteria of Apnea-hypopneus (called RERA, respiratory effort-related arousals) in combination with the activations associated with respiratory movements recorded on the electroencephalogram. The most common concept regarding the number of episodes of apnea-hypopneus during sleep, which should be considered pathological necessary to form a clinical symptom complex is obstructive sleep apnea syndrome, based on research from the Stanford Sleep Research Center, a typical picture of obstructive sleep apnea syndrome is formed when there are more than five episodes of apnea / hypopne during an hour's sleep [23-27].

The most favorable conditions for the formation of obstructive apnea-hypopne in patients obstructive sleep apnea syndrome occurs in the first to second stages of the non-REM sleep phase and REM sleep [28-30]. Multiple recurring episodes of respiratory dysrhythmia during sleep lead to changes in the structure of sleep-the duration of the deep stages of the non-REM sleep phase, as well as the duration of the REM phase, decreases.

The prevalence of obstructive sleep apnea syndrome is on average 1-2% in the total population, according to the above criteria, according to epidemiological studies [31-34]. Up to 25% of men and 9% of middle-aged women are thought to have an episode of respiratory arrest ≥5 times in an hour during sleep.

The prevalence of obstructive sleep apnea syndrome is higher in elderly, hypertensive patients with coronary heart disease and morbid obesity than in the general population. Many population studies show that obstructive sleep apnea syndrome is two to three times more common in men than in women [35].

The mechanisms of such differences are still unclear, but suggest that female sex hormones (estrogens and progesterone) may play an important protective role [36], which is confirmed by the low prevalence of respiratory failure during sleep in women of reproductive age and its increase after menopause. However, it has been shown that injecting men with estrogen and progesterone does not cause a decrease in the Apnea-hypopnea index (IAH) [37-39]. In addition, androgens can help increase the ability of the upper respiratory tract to collapse, which is confirmed by the frequent development of sleep apnea syndrome in women with polycystic ovary syndrome, as well as Dexter et al. about complete loss of sleep apnea in a woman after surgical removal of a testosterone-producing ovarian tumor [40-43]. In addition, anatomical differences in the upper respiratory tract, differences in the activity of the muscles of the chin-tongue during wakefulness and the risk of developing the structure of fat deposits may explain the high risk.obstructive sleep apnea syndrome in men.

A number of researchers also believe that there may be a certain genetic predisposition to develop obstructive sleep apnea syndrome. Candidate genes responsible for development of obstructive sleep apnea syndrome, are currently not identified and are under study. One epidemiological study showed a significant correlation between the presence and presence of the Apoe allele epsilon-4 (a protein involved in the development of Alzheimer's disease and cardiovascular disease).obstructive sleep apnea syndrome, a similar correlation was found only in middle-aged patients [44-47].

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According to in a genome study of people with and without obstructive sleep apnea syndrome found that there are several candidate regions on chromosomes 1p, 2p, 12p, 19p associated with high IAH. Chromosomes 2p, 7p, and 12p were found to contain regions associated with high body mass index (BMI).

One study found that various genetic variants of il - 6 (specifically allele C carriers) may contribute to the development of obstructive sleep apnea syndrome even in patients who do not have excess body weight [48-51]. In addition to the genetic predisposition predicted to develop obstructive sleep apnea syndrome, there may be a number of additional factors that contribute to the development of phenotypic structural features of the upper respiratory tract, contributing to the development of sleep apnea syndrome.

The problem obstructive sleep apnea syndrome is of certain importance in the practice of an endocrinologist. It is known that respiratory disorders during sleep can develop in patients with various endocrinopathies, and the relationship between endocrine pathology and respiratory disorders during sleep is not as simple as it seems at first glance. On the one hand, a violation of breathing during sleep leads to spontaneous hormonal changes, in particular-to the activation of the sympathetic nervous system along with the release of cortical and medulla hormones of the adrenal glands, continues during the day even in the absence of apnea episodes and leads to serious clinical consequences; sleep to disorders of somatotropic hormone secretion due to On the other hand, patients with certain endocrine disorders such as obesity, Type 1 diabetes (T1D) and type 2 diabetes (T2D), hypothyroidism, acromegaly, Cushing's syndrome, hyperandrogenism may develop sleep breathing disorders., and lead to a deterioration in their clinical course [52-56].

To improve quality and duration of life, it is important to develop strategies for early diagnosis, control and treatment of respiratory disorders during sleep in patients with various endocrine disorders.

For the first time, the development of Central and obstructive sleep apnea was described in patients with endocrinopathy such as acromegaly and hypothyroidism.

Later, there were reports of high prevalence of respiratory disorders during sleep in diabetic patients, especially complicated by diabetic autonomic neuropathy; in obese patients; Cushing's syndrome and hyperandrogenia due to hormonal active formation of ovaries or adrenal glands.

The pathogenesis of obstructive sleep apnea in various endocrinopathies is complex, but its main component is anatomical changes in the upper respiratory tract and impaired neuromuscular control of the upper respiratory tract during sleep.

Obesity and obstructive sleep apnea.

Obesity is one of the most important and independent risk factors for the development of sleep apnea. In The Study, Vgontzas A. with co-authors. [57] of the 250 obese patients who did not complain of sleep disorders, 40% of men and 3% of women identified the pathological number of episodes of sleep apnea. The prevalence of sleep apnea among morbid obese patients reaches 40-90%.

Studies have shown that an increase in BMI to 1-SD is associated with a quadrupling risk of developing Apnea [58] and a 10% increase in body weight is associated with a sixfold increase in the risk of developing obstructive sleep apnea syndrome over the next four years. Of particular importance is the type of abdominal obesity, which, according to a number of studies, has a high risk of developing obstructive sleep apnea syndrome. IDR [59] has been shown to increase with increasing Ot / ob index.

The development of respiratory disorders in a dream in obesity is associated with a number of effects, the main of which is the anatomical narrowing of the pharyngeal lumen of the upper respiratory tract due to the accumulation of fat in the lateral pharyngeal pockets, a change in the elastic properties of the pharyngeal walls due to their fat infiltration. Changes in bone and soft tissue structures (neck

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enlargement, tongue enlargement, airway narrowing, nasal obstruction, etc.) make a certain contribution, which leads to an increase in the ability of the upper respiratory tract to collapse. A decrease in lung volume during sleep and an increase in diaphragm load due to obesity in the abdomen contribute to the formation of a periodic decrease in the upper respiratory tract, i.e.the emergence of episodes of obstruction [60-66].

Mechanoreceptors, mainly localized in the larynx, respond to changes in airway pressure during wakefulness, increasing the activity of the pharynx dilator muscles and keeping the airways in an open position [67].

Obesity and obstructive sleep apnea syndrome have a close relationship. As mentioned above, stopping breathing at night, changing the structure of sleep, leads to a decrease in the deep stages of non-REM sleep and a violation of the production of insulin-like growth factor 1, a somatotropic hormone that plays a role in the balance between fat and muscle mass of the human body. In an adult, a lack of growth hormone leads to a change in more fat mass compared to muscle mass, i.e.contributes to the development of obesity in the abdomen, which forms a kind of vicious circle.

Many researchers say that the metabolic cluster for obstructive obese sleep apnea is very characteristic, including arterial hypertension, dyslipidemia, type of abdominal obesity, impaired glucose tolerance, a symptom complex described as a "metabolic syndrome". Given the role of obstructive sleep apnea syndrome as an independent risk factor for cardiovascular disease and the frequent presence of sleep breathing disorders in patients with metabolic syndrome, Ian Wilcox proposed in 1998 to combine obstructive sleep apnea with other known risk factors and convert metabolic X syndrome into Z syndrome [68-71].

According to a large number of studies, a decrease in body weight was found to be very effective in controlling respiratory disorders during sleep in obese patients.

Thus, a decrease in body weight by only 10 percent is accompanied by a 2,5-fold decrease in IDR [72]. However, obese patients have obstructive sleep apnea syndrome, a number of factors that make it difficult to lose weight (serious lipid metabolism disorders, hormonal imbalances, etc.). The appointment of drug therapy for obesity in combination with lifestyle changes (rational nutrition, optimal mode of physical activity) increases the expected therapeutic effect on such patients. Timely appointment of CPAP therapy (constant positive airway pressure, auxiliary ventilation with the creation of constant positive airway pressure) allows you to accelerate the rate of weight loss and achieve the necessary metabolic control.

Diabetes and obstructive sleep apnea

It is known that breathing disorders during sleep are more common in patients with T2D. In addition, given the totality of developmental risk factors (abdominal obesity), T2D and obstructive sleep apnea often accompany each other.

Experimental data in human and animal models showed that decreased sleep due to hypoxia and its breakdown negatively affects obstructive sleep apnea syndrome, glucose metabolism. Studies have shown that the prevalence of glucose tolerance in obese patients is about 29% of obstructive sleep apnea syndrome (IAH<5), while obstructive sleep apnea syndrome in obese patients with IAH is>15-36% [73-75], Confirming the role of obstructive sleep apnea syndrome as an independent risk factor for insulin resistance and carbohydrate metabolism disorders.

In addition to the generally recognized factors that determine the development of insulin resistance in obese patients, the hyperactivity of the sympathetic nervous system is one of the main pathogenetic connections that are accompanied by respiratory disorders during sleep, leading to the development of carbohydrate metabolism disorders in patients.obstructive sleep apnea syndrome through activation of glycogenolysis and gluconeogenesis and glucagon secretion.

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The development of diabetic autonomic neuropathy in diabetic patients may be prone to developing obstructive sleep apnea syndrome. According to Keller et al., prevalence reaches 30% of obstructive sleep apnea syndrome in patients with diabetic neuropathy [17]. A number of researchers have shown the likelihood of cardiorespiratory arrest in patients with diabetic autonomic neuropathy [25], some of which have resulted in death.

The cause of this phenomenon is a violation of the vegetative joint of respiratory regulation, namely: vagus denervation of the lungs with a violation of volume-dependent respiratory reflexes, afferent and efferent denervation of carotid glomeruli with a violation of the reaction to hypoxia, receptor denervation of the mucous membrane of the upper respiratory tract.reflex responses to changes in flow and air pressure. Therefore, the assessment of the presence of respiratory disorders during sleep in patients with SD is of important clinical importance before surgery, increasing the likelihood of cardiorespiratory arrest in patients of this category when leaving anesthesia.

Many studies have shown the opposite effect of SRAR therapy on glycemic control, but most have proven that regular use of SRAR therapy with effective therapeutic pressure can improve insulin sensitivity after a long period of time.

Thyroid disorders and obstructive sleep apnea syndrome.

Another form of endocrine pathology, in which breathing disorders often develop during sleep, is hypothyroidism, especially when mycedematous edema occurs. There is much evidence of a high prevalence of obstructive sleep apnea syndrome in patients with hypothyroidism (50-100%), however, attempts to detect a significant prevalence of thyroid hypofunction in patients with respiratory disorders during sleep have failed (only 1-3% of diagnosed patients had obstructive sleep apnea hypothyroidism) [1]. Thus, the prevalence of hypothyroidism in patients is not higher than that observed in the general population of obstructive sleep apnea syndrome. In this regard, according to most researchers, not all patients who contact somnological offices require an assessment of thyroid function.

It is advisable to study thyroid function in patients whose severity of disorders identified by polysomnographic examination does not explain the severity of the clinical picture of the disease, as well as in patients with low efficacy of crap therapy.

Perhaps the most common violation of breathing during sleep in patients with hypothyroidism is called upper respiratory resistance syndrome (manifestation of snoring if there are no clear episodes of upper respiratory obstruction and no activation reactions on the electroencephalogram or episodic floulimitation that results in arousal). obstructive sleep apnea syndrome, but studies proving a similar assumption, no. It should be noted that the clinical picture has similar characteristics as obstructive sleep apnea and hypothyroidism syndrome, which can lead to a low assessment of the clinical condition of patients.

The main pathogenetic factors for the development of sleep disorders in patients with severe hypothyroidism are obstruction of the upper respiratory tract and impaired chemoreception.

Obstruction of the upper respiratory tract is caused by the deposition of mucopolysaccharides and protein extravasation in the tissues of the face and tongue, pharynx and larynx structures, which leads to an increase in the size of the neck and tongue, the appearance of musinous (myxedematous) edema, which leads to thickening of the larynx and pharynx walls.it eventually leads to a narrowing of the lumen of the upper respiratory tract. Certain importance is attached to the deterioration of the contractile properties of the dilator muscles of the upper respiratory tract, the loss of transverse lines due to the development of dysthyroid myopathy [23].

Levothyroxine therapy has been shown to be effective in reducing IDR despite the lack of significant weight loss. However, in patients with hypothyroidism accompanied by obesity, levothyroxine therapy leads to clinical improvement, but does not lead to complete loss of obstructive sleep apnea syndrome. More than half of patients with obesity and hypothyroidism, at least until the clinical signs

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of hypothyroidism disappear, need to support cap therapy or surgical treatment in ENT organs after the appointment of replacement therapy, and then, 6-12 months after the start of therapy, a polysomnographic reassessment of the condition is necessary. Some patients require continued CPAP therapy even after clinical remission of hypothyroidism [25].

The likelihood of developing obstructive sleep apnea syndrome increases in patients with large-sized euthyroid goiter due to high airway compression, especially when lying on the patient's back during sleep. At the same time, the pathogenesis of the development of sleep disorders in patients with euthyroid goiter is more complicated than simply the effects of compression. With the development of swelling of the upper respiratory tract, a violation of venous return from the head and neck, dysfunction of the muscles adhering to the hypoid bone, and the loss of the normal caudal tract of the upper respiratory tract during breathing can serve as auxiliary mechanisms of development.obstructive sleep apnea syndrome in patients of this category. After thyroid resection, the condition of patients improves significantly [6, 20].

Acromegaly and obstructive sleep apnea syndrome.

According to population studies, the prevalence of sleep breathing disorders with acromegaly, mainly obstructive Genesis, exceeds the total population and reaches 60-70% (according to various authors – 19-93%).

Obstructive respiratory disorders in a dream due to thickening of the mucous membrane of the upper respiratory tract and bronchi occur in 25% of women and 70% of men with acromegaly. Patients with acromegaly had a 1.6–3.3-fold increased risk of death in obstructive sleep apnea syndrome in 25% of cases [9].

Many studies have shown that independent predictors of the development of sleep disorders in patients with acromegaly are process activity, age, and thumb circumference index size (more than 8.5 cm) as a measure of assessing soft tissue hypertrophy [22]. Rosenow F. etc. patients with achromegaly had obstructive sleep apnea syndrome with somatotropic hormone and insulin-like growth factor-1 levels much higher than those with achromegaly without apne episodes [26]. However, a number of studies have found no significant correlation between STH levels and the presence of obstructive sleep apnea syndrome [13].

The main diseases of breathing during sleep in acromegaly are respiratory disorders during obstructive sleep [19]. In this category of patients, the development of episodes of obstruction is associated with increased tongue (macroglossia) and para swelling of Retrofaringeal soft tissues due to increased extracellular water and cell mass due to hyperplasia and connective tissue growth [22]. Differences in craniopharyngeal morphology of patients with acromegaly in the presence and absence of apnea episodes have not been identified.

With acromegaly, there is also the possibility of developing central respiratory disorders in sleep, the pathogenesis of which is much more complicated. Narrowing of the lumen of the upper respiratory tract can lead to reflexive suppression of the activity of the respiratory center; increased growth hormone levels, and possibly defects in the somatostatinergic pathway (somatostatin is a major inhibitor of growth hormone release and a peptide involved in modulating Central respiratory control in both the hypothalamus and brainstem) can overdo the respiratory center fan reaction to carbon dioxide.breathing leads to arrest. It is also possible to develop a mixed variant of breathing disorders during sleep with acromegaly, with phenomena based on the two conditions described above [14].

Sleep breathing disorders in patients with acromegaly significantly increase the risk of serious cardiovascular and respiratory complications and ultimately the risk of cardiovascular death, the leading cause of death in patients with acromegaly [25].

It should be noted that the control of respiratory disorders during sleep in patients with acromegaly is of particular importance due to data on the possible development of asphyxia in the early

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postoperative period after transfenoidal adenomectomy due to the tamponade of the nasal cavity and swelling of the upper respiratory tract [22, 25].

The use of octreotide, according to a number of works, is necessary at the stage of preoperative preparation of patients of this category [22].

Adequate etiotropic treatment of acromegaly leads in many cases to regression of existing respiratory disorders. Changes in the number of apnea are associated with a decrease in visseromegaly, especially the tongue, which is accompanied by a decrease in the level of growth hormone as a result of treatment, Harris reports A. etc., achieved in 61% of patients mainly through extracellular dehydration [15]. However, a number of patients with irreversible structural changes due to pharyngeal wall hypertrophy and / or fibrosis and deformity of the lower jaw may develop refractory obstructive sleep apnea syndrome [22], so timely diagnosis and treatment are important for Obstructive Sleep Apnea Syndrome. In addition, a number of patients with acromegaly may have the duration of respiratory disorders during sleep after etiotropic treatment, and therefore polysomnography is recommended for patients in the high risk group (elderly, overweight, with SD) after radical treatment of acromegaly to improve control of existing diseases. possible supportive treatment with the method of creating constant positive pressure in the respiratory tract (CPAP therapy) with periodic correction of the required level of therapeutic pressure [22].

Cushing's syndrome and obstructive sleep apnea.

The likelihood of developing respiratory disorders during sleep in patients with Itenko-Cushing's disease and Cushing's syndrome is primarily associated with obesity, which has its own characteristics: adipose tissue accumulates in the mediastin, head, neck and retroperitoneal region according to the centripetal type, which leads to narrowing of the lumen of the upper respiratory tract, increased external load on the walls of the pharynx, as well as

Glucocorticoid myopathy can develop, which leads to dysfunction of the pharyngeal dilator muscles (primarily the chin-tongue and chin-hypoid muscles), which makes a certain contribution to the development of respiratory disorders during sleep. In addition, the development of respiratory dysrhythmia has been described in patients with yatrogenic Cushing's syndrome.

There is no data on the possibilities of treating respiratory disorders during sleep in patients with Itenko-Cushing's disease and Cushing's syndrome, but etiotropic (operative) treatment in this case has been shown to be very effective [22].

Polycystic ovary syndrome and obstructive sleep apnea syndrome.

A potentially significant contribution has recently been shown in obstructive sleep apnea syndrome to the risk of developing impaired glucose tolerance, hypertension and other diseases in polycystic ovary syndrome (SPI), which affects about 5-8 percent of women [8]. The abundance of androgens, subnormal levels of estrogens and the presence of visceral obesity, which affect both nervous control of breathing and the mechanics of the upper respiratory tract, can explain the increased risk of development.obstructive sleep apnea syndrome in sleep patients [25]. A number of researchers speculate that testosterone can increase the ability of the upper respiratory tract to collapse, while its effect on controlling ventilation is controversial [22]. While the exact mechanisms of excessive androgen exposure remain unclear, data on the development of testosterone has been collected in the literature on obstructive sleep apnea syndrome [22].

In clinical practice, the possibility of respiratory failure in obstructive Genesis Sleep is important in patients with uncontrolled arterial hypertension, with the exception of adrenal pathology or other causes.

Obstructive sleep apnea syndrome, which was previously indicated in 40% of patients, reveals high blood pressure during waking up, in addition, in 40% of patients with refractory hypertension [24], which is 20-35% of patients with hypertension in the general population, with obstructive sleep

apnea. It has recently been confirmed that 83% of patients with uncontrolled hypertension have osa, despite combination therapy (three drugs or more) at optimal therapeutic doses [18]. Characteristic features of arterial hypertension associated with obesity and obstructive sleep apnea include the predominance of isolated diastolic hypertension, increased heart rate and blood pressure variability, and the absence of a nocturnal drop in blood pressure. The importance of the problem lies in the fact that the US National Joint Committee placed obstructive sleep apnea syndrome at the top of its list of causes of secondary arterial hypertension [29].

About the effect of obstructive sleep apnea syndrome to the rhythm of melatonin secretion-the hormone epiphysis, which is involved in the regulation of the sleep – wake cycle, and also has antioxidant and anti-inflammatory properties, is especially important for patients.obstructive sleep apnea syndrome-little known. A number of studies have shown peak melatonin secretion in patients with high IAH [5], however, other studies have shown that patients lack a nocturnal peak of its secretion in obstructive sleep apnea syndrome, which may be partly due to the difficulty of patients achieving a normal sleep-wake cycle [16].

The endogenous synthesis of melatonin is thought to be due to the stimulation of b-adrenoreceptors with endogenous norepinephrine (75% of night melatonin levels) and the interaction of catecholamines with pinealocytes ' A1-adrenoreceptors [5]. Obstructive sleep apnea shown for patients is characterized by increased activity of the sympathetic nervous system with increased levels of catecholamines, and although peripheral and Central levels of norepinephrine are not always related, increased levels of melatonin are observed in patients.obstructive sleep apnea syndrome at night, when norepinephrine levels are high. A significant change in melatonin secretion against the background of SRAR therapy was found mainly in the second half of the night, when the best oxygenation of the blood was achieved and the level of norepinephrine decreased [16].

Currently, a lot of data has been collected on the negative clinical and medico-social consequences of obstructive sleep apnea, which is primarily due to the high risk of cardiovascular diseases (arterial hypertension, cardiac arrhythmias, myocardial infarction, pulmonary hypertension), which still occupy a leading place in the composition of the causes of death. While there are effective controls and treatments for respiratory disorders in obstructive Genesis Sleep, at least 82% of men and 93% of women and 80-90% of severe cases remain undiagnosed obstructive sleep apnea [36].

To improve hormonal and metabolic indicators, patients should be identified early obstructive sleep apnea syndrome and treat them on time. In some cases, the treatment of joint diseases-improving nasal breathing, compensating for hypothyroidism, etiotropic treatment of acromegaly, Cushing's syndrome – leads to a clear clinical improvement in the condition of patients.

Treatment obstructive sleep apnea syndrome.

Treatments depend on the severity of the disorders in which obstructive sleep apnea syndrome has been identified. Optimal therapeutic tactics of treatment obstructive sleep apnea syndrome involves mandatory lifestyle changes with any severity (decreased body weight, restriction of alcohol and sedatives).

A decrease in body weight with" metabolic syndrome "has been shown to be the most effective therapeutic measure that allows stopping the cycle of" obesity – obstructive sleep apnea syndrome – insulin resistance –obesity " [24]. Positional therapy with a change in body position during sleep, avoiding sleeping on your back can alleviate the condition of patients. Creating a raised head position (30 degrees or higher) reduces tongue squatting in the back position; the downward displacement of fluid in the body, as a result, at the level of the nose and pharynx, the swelling of the mucous membrane decreases, their Lumen increases, and, accordingly, snoring is slightly weakened.

In the case of mild obstructive sleep apnea syndrome oral instruments made of soft plastic that keep the upper airways in an open position can be used by moving the lower jaw forward or keeping the

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mouth open(in most cases, it is necessary to consult a dentist-orthodontist to choose the device correctly); perform a series of surgical operations ON ENT organs; as well as

The method of choice for moderate to severe severity of the syndrome is the long-term use of continuous positive airway pressure therapy (CPAP therapy) or two-level positive airway pressure therapy (BiPAP therapy, bilevel positive airway pressure). Performing surgical procedures to alleviate the symptoms a little is possible if the patient tolerates cap therapy or if there is insufficient therapy with cap.

Continuous. Positive Airway Pressure Treatment (CPAP therapy)-a non – invasive treatment method by creating constant positive air pressure in the patient's Airways-is the gold standard of therapy.obstructive sleep apnea syndrome and is used.

The same air supply is carried out through a mask (nose, oronasal), the upper respiratory tract remains open at night during the use of the instrument, and the patient's flat and calm breathing is ensured.

At all stages of sleep, the minimum therapeutic pressure level, which eliminates episodes of Apnea and hypopneas in any state of the body, eliminating sleep breakdown, snoring and desaturation, is determined by pressure titration during a night polysomnographic examination [28]. The effectiveness of therapy depends on the correct choice of mask, the patient's operating conditions of the device (at least 70% night per week, at least four hours per night) [28]. A number of studies not only show an improvement in breathing during sleep in patients with obstructive sleep apnea syndrome against the background of regular CPAP therapy with effective therapeutic air pressure, but also an improvement in cardiometabolic parameters, as well as the Prevention of the risk of developing cardiovascular events.

Thus, obstructive sleep apnea syndrome is an important medical problem. The presence of obstructive sleep apnea syndrome is a more frequent finding in patients with endocrine pathology than previously assumed, and is one of the most important problems in this category of patients due to its significant impact on quality of life and prognosis.

In recent times, obstructive sleep apnea syndrome has gained special importance due to the epidemic of obesity, T2DM, serious clinical and medico-social consequences. Treatments for Obstructive Sleep Apnea Syndrome have been identified in general. However, further research is needed to develop strategies to improve the control of endocrine disorders associated with respiratory disorders during sleep.

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