УДК 616.24-008.444-02:616.1/.8-036.12
https://doi.org/10.20538/1682-0363-2025-1-154-163

Sleep disordered breathing and its impact on the course of chronic non-communicable lung diseases

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ABSTRACT

The lecture synthesizes and analyzes the findings of research concerning the impact of sleep disordered breathing (SDB) on the progression of the most prevalent chronic non-infectious lung diseases (CNLDs). SDB, including conditions, such as snoring, sleep hypoventilation syndrome, and obstructive and central sleep apnea syndrome, constitutes a significant medical concern due to its high prevalence and adverse health consequences. SDB is regarded as an independent risk factor for the development and progression of a range of CNLDs. Timely diagnosis and management of SDB may serve as an effective preventive measure against severe manifestations and complications associated with this group of diseases.

Keywords: sleep disordered breathing, snoring, apnea, hypoxia, chronic non-communicable lung diseases

Conflict of interest. The authors declare the absence of obvious or potential conflicts of interest related to the publication of this article.

Source of financing. The authors state that they received no funding for the study.

For citation: Bespalova I.D., Mitrichenko U.M., Koshchavtseva Yu.I., Kapitanova D.V., Badmaev A.Z., Agaeva S., Zhukovskaja O.V., Kolmakova V.M., Belyakova T.V., Teteneva A.V., Bukreeva E.B., Boyarko V.V., Nesterovich S.V., Vinokurova D.A., Kalyuzhin V.V. Sleep Disordered Breathing and Its Impact on the Course of Chronic Non-Communicable Lung Diseases. *Bulletin of Siberian Medicine.* 2025;24(1):154–163. https://doi.org/10.20538/1682-0363-2025-1-154-163.

Нарушения дыхания во сне и их влияние на течение хронических неинфекционных заболеваний легких

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РЕЗЮМЕ

В лекции обобщены и проанализированы результаты исследований, касающихся изучения влияния нарушений дыхания во сне (НДС) на течение наиболее распространенных хронических неинфекционных забо-

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леваний легких (ХНЗЛ). Нарушения дыхания во сне, такие как храп, синдром гиповентиляции во сне, синдром обструктивного и центрального апноэ сна, представляют собой актуальную медицинскую проблему ввиду их высокой распространенности и неблагоприятных последствий для здоровья. Нарушения дыхания во сне рассматриваются как независимый фактор риска развития и прогрессирования целого ряда ХНЗЛ. Своевременная диагностика и коррекция НДС может быть эффективной мерой профилактики тяжелого течения и осложнений этой группы заболеваний.

Ключевые слова: нарушения дыхания во сне, храп, апноэ, гипоксия, хронические неинфекционные заболевания легких

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии финансирования при проведении исследования.

Для цитирования: Беспалова И.Д., Митриченко У.М., Кощавцева Ю.И., Капитанова Д.В., Бадмаев А.З., Агаева С., Жуковская О.В., Колмакова В.М., Белякова Т.В., Тетенева А.В., Букреева Е.Б., Боярко В.В., Нестерович С.В., Винокурова Д.А., Калюжин В.В. Нарушения дыхания во сне и их влияние на течение хронических неинфекционных заболеваний легких. *Бюллетень сибирской медицины.* 2025;24(1):154–163. https://doi.org/10.20538/1682-0363-2025-1-154-163.

INTRODUCTION

The beginning of the twenty-first century has set new challenges for medical science and clinical medicine related to the epidemic level of prevalence of chronic non-communicable diseases and the need to search for mechanisms underlying their progression and effective methods of elimination. A special place in the profile of morbidity is occupied by chronic non-communicable lung diseases (CNLDs), which represent a serious problem for the health care systems of most countries. CNLDs are characterized by persistent inflammation, damage to the lung parenchyma, and progressive deterioration of the functional activity of external respiration, which underlies disability and high mortality and contributes to a significant decrease in the quality of life.

The accumulation of clinical and experimental knowledge, the development of modern medical technologies, and a new branch of practical medicine – somnology, have made it possible in recent years to identify new aspects of the pathogenesis of a number of pathological conditions and to consider sleep disordered breathing (SDB) as one of the mechanisms underlying the progressive course and complications of chronic non-communicable diseases. SDB is a heterogeneous group of syndromes characterized by periodic or persistent changes in the breathing pattern during sleep, which may include episodes of apnea (short-term pauses in breathing), hypopnea (decreased respiratory activity), and other disorders. These conditions lead to hypoventilation, hypoxemia and can be associated with various somatic symptom and mental disorders, which, along with the high prevalence, determines their medical significance. According to the mechanism of occurrence, obstructive apnea/hypopnea, caused by upper airway closure, and central apnea/hypopnea, associated with collapse of the respiratory center, are distinguished [1].

The most studied type of SDB is obstructive sleep apnea (OSA) – a condition in which episodes of a lack of pulmonary ventilation with pauses in breathing during sleep for more than 10 seconds (apnea/hypopnea) are recorded, accompanied by snoring, periodic pharyngeal collapse, hypoxemia, excessive daytime sleepiness, and gross fragmentation of sleep [2]. The prevalence of OSA in the Western population is 5–7% of the entire population over 30 years of age. Severe forms of the disease affect about 1–2% of this group. In people over 60 years of age, the frequency of OSA increases significantly and is about 30% in men and about 20% in women. The prevalence of clinically pronounced SDB reaches 15% in patients with internal diseases and increases with cooccurring comorbidities.

According to a number of studies, the incidence of OSA in overweight patients exceeds 30%, reaching 50–98% in patients with morbid obesity

[3]; the prevalence of OSA and other forms of SDB are observed in 35–80% of patients with arterial hypertension (AH) [4], 38–65% of patients with coronary heart disease (CHD) [5], 38–72% of patients with a previous stroke [6], 35–40% of patients with heart failure (HF), and 56–74% of patients with rhythm disturbances [4]. Given the current trend towards population aging and the obesity pandemic, a steady increase in the prevalence of SDB is expected, since obesity and old age are recognized as the main risk factors for OSA [7].

Unfortunately, the level of awareness of patients and primary care specialists about the problem of SDB is low; OSA often remains undiagnosed in a large part of the population due to the low specificity of complaints and the unavailability of instrumental diagnostic methods [8].

The disappointing values for morbidity, disability, and mortality from respiratory diseases are largely determined by the epidemic level of prevalence of metabolic syndrome components in the general population and a high cardiovascular risk in different population groups [9, 10]. In view of the fact that SDB is an independent risk factor for cardiovascular diseases [1], there is a need to consider it as a significant mechanism for a severe course and development of complications in the most common CNLDs.

SLEEP DISORDERED BREATHING AND BRONCHIAL ASTHMA

Despite significant advances in the diagnosis and treatment of bronchial asthma (BA), high morbidity and low control levels in the presence of comorbid pathologies, including SDB, require studying the mechanisms underlying syntropy and finding effective treatment approaches. The incidence of OSA in patients with BA is higher than in the general population, regardless of body mass index (BMI), gender, age, or smoking status [11–13]. According to various data, the prevalence of SDB in patients with BA varies from 23 to 46% and depends on the severity of the disease. The prevalence of OSA in patients with BA ranges from 19 to 60% in mild BA and reaches up to 95% in severe asthma.

BA was shown as an independent risk factor for habitual snoring, which is the mildest form of SDB. The wide range of epidemiological parameters is associated with the peculiarity of the study design, with the use of different diagnostic criteria for pathological conditions, and with patient inclusion criteria. Researchers from Saudi Arabia revealed high prevalence of BA in patients with SDB (35.1%), which is also significantly higher than in the general population. Patients with OSA with BA had higher BMI and greater apnea / hypopnea index compared to patients with OSA without BA, while BMI > 35 kg / m^2 was a significant predictor of BA in patients with OSA [14]. The authors believe that such high prevalence of the association of two diseases (OSA and BA) cannot be a coincidence and is determined by the pathophysiology of the diseases [15].

OSA and BA have some common characteristics. Both diseases are obstructive respiratory diseases, but with different mechanisms and anatomy of obstruction. In patients with co-occurring OSA and BA, there is obstruction of both upper and lower airways during sleep. Both pathological processes have the same comorbidities, such as obesity, allergic rhinitis, and gastroesophageal reflux disease (GERD). It was also noted that smoking, obesity, GERD, and allergic rhinitis should be considered as important risk factors for OSA in patients with BA. A comparative analysis of parameters characterizing sleep quality in groups of patients with BA demonstrated that patients with OSA had higher BMI, higher incidence of allergic rhinitis, a more severe course, and worse predicted forced expiratory volume in the first second (FEV_1). This category of patients has poor sleep quality, which is often associated with high morbidity and mortality. Researchers agree that BA and OSA are characterized by a bidirectional interaction.

On the one hand, the severity and duration of BA affect the predisposition to OSA. The mechanisms of this phenomenon are considered to be systemic inflammation and neuroimmune interactions due to their involvement in the control of breathing, as well as the negative effects of inhaled glucocorticoids (ICS) on smooth muscles and fat content, changing the anatomy of the upper respiratory tract. On the other hand, OSA affects

airway inflammation, promotes their remodeling and dysfunction in such a way that it determines resistance to standard therapy, which explains the relationship between OSA and BA with worse clinical outcomes at all stages of medical care. Moreover, the prevalence of OSA correlates not only with the duration and severity of the disease, but also with the dosage of glucocorticoids taken [15]. Thus, the absence of OSA treatment can lead to increased ICS therapy, which, in turn, will accelerate this vicious circle and contribute to irreversible dysfunction of the lower respiratory tract [16, 17].

There is growing evidence of a relationship between SDB and BA based on common pathophysiological factors and mutual influence. The exact mechanisms by which these diseases interact are not fully understood. SDB is believed to stimulate inflammatory responses through hypoxia, hypercapnia, and sleep fragmentation, leading to a reversible increase in C-reactive protein (CRP) levels and TNFα production and is associated with airway collapse. At the same time, the level of both proinflammatory cytokines usually decreases after CPAP therapy, positively affecting the course of BA, pulmonary function parameters, and quality of life.

OSA, in turn, aggravates nocturnal manifestations of BA due to reflex bronchoconstriction associated with upper airway irritation during snoring. Inflammatory infiltration of the upper airways in BA, increased fat deposition in the pharyngeal walls due to steroid use [18], or the presence of comorbidities, such as obesity, lead to a decrease in the cross-sectional diameter of the upper airways. The frequent association of BA with allergic rhinitis, nasal polyps, and adenoid hypertrophy contributes to airflow resistance and creates high negative pressure during inspiration, which increases the risk of upper airway collapse [19]. BA is thought to affect pharyngeal muscle function either directly by affecting neural sensory pathways due to inflammation, or indirectly by muscle weakness due to ICS therapy [15]. Pharyngeal muscle myopathy increases the ability of the upper airways to collapse, increasing the risk of OSA. Japanese scientists established that the severity of OSA, estimated by the value of the apnea/hypopnea index in BA, is determined by the thickness of the mediastinal adipose tissue and the severity of bronchial hyperreactivity [20].

The practical significance of studying the mechanisms of mutual aggravation of the two diseases is determined by the fact that BA with co-occurring SDB is characterized by a low level of control, has a worse prognosis, is associated with a high risk of repeated hospitalizations due to exacerbation, and, as a result, is associated with high treatment costs [21, 22].

Currently, there is an obvious need to diagnose SDB in patients with BA, especially in cases with refractory BA, frequent night attacks, concomitant obesity, GERD, and atopic rhinitis. Timely diagnosis of SDB in patients with BA and appropriate treatment will stop the vicious circle of OSA and eliminate associated adverse effects of basic therapy, which will also help reduce cardiovascular risk in this category of patients, improve their quality of life, and naturally reduce the economic burden of medical care.

SLEEP DISORDERED BREATHING AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a progressive disease characterized by persistent airflow limitation, which is a consequence of the chronic inflammatory response of the airways and lung tissue to the effects of inhaled harmful particles or gases. COPD is an urgent medical and social problem, being, according to experts from the World Health Organization, one of the leading causes of death in the world, ranking third.

SDB and sleep disorders are an extremely common and often underestimated problem in patients with COPD. It is assumed that each form of SDB in COPD is associated with adverse clinical outcomes, including an increased risk of exacerbations, hospitalizations, cardiovascular events, decreased survival, and deterioration in the quality of life [23]. Due to the high incidence of OSA, much attention has been paid by researchers to the study of co-occurring COPD and OSA, which is called overlap

syndrome. Given the high prevalence of COPD and OSA separately, researchers have suggested that the coexistence of both disorders may arise solely based on chance association [24]. The prevalence of overlap syndrome in the general population has been reported to range from 1 to 3.6% [25].

However, this figure increases significantly when the prevalence of overlap syndrome is assessed in patient populations from specialized clinics for the diagnosis and treatment of OSA or COPD. Studies including patients with diagnosed OSA have shown that the prevalence of overlap syndrome ranged from 7.6 to 55.7%. The presence of comorbid OSA in populations with established COPD has also been assessed, and again a wide prevalence range from 2.9 to 65.9% was observed [25]. However, smaller studies have reported significantly higher prevalence of overlap syndrome, which may indicate the possibility of incidental findings or higher-risk patient cohorts [26]. The reasons for these contradictory results are unclear, but they may reflect differences in the study populations, recording methods, and diagnostic methods for OSA and COPD.

Intermittent upper airway obstruction in OSA may worsen the course of COPD, leading to more pronounced hypoxemia and hypercapnia, which, in turn, accelerates the development of pulmonary hypertension and chronic respiratory failure [27]. Chronic inflammation and airway remodeling in COPD create the prerequisites for the development of OSA by reducing the tone of the upper airway muscles and increasing their collapsibility. There is a correlation between the severity of obstructive respiratory disorders and sleep disorders in patients with overlap syndrome [28]. Patients with COPD have a deterioration in sleep quality due to a decrease in its efficiency and reduction in the REM phase, which is an additional factor in the potential association with OSA [29].

The manifestations of systemic inflammation and oxidative stress observed in COPD and OSA indicate a deep pathogenetic relationship between these pathologies and their impact on the development of cardiovascular diseases [30]. Systemic inflammation is a major factor in the pathogenesis of atherosclerosis, and intermittent (periodic) hypoxemia associated with recurrent episodes of apnea/hypopnea in OSA significantly affects this inflammatory response. Hypoxemia, both intermittent and persistent, is more pronounced in co-occurring OSA and COPD compared to each disease separately and, therefore, is expected to increase the inflammatory response. Patients with overlap syndrome have higher sympathetic and lower parasympathetic activity compared to patients with OSA or COPD alone.

Thus, it can be expected that cardiovascular diseases will be more common in patients with a combination of OSA and COPD. Retrospective studies have shown higher prevalence of AH, diabetes mellitus, metabolic syndrome, and atrial fibrillation in patients with co-occurring OSA and COPD compared to patients with OSA alone [31, 32]. From a cardiovascular perspective, the importance of recognizing concomitant OSA in patients with COPD is supported by a study in a rodent model, which found that cardiovascular changes caused by chronic intermittent hypoxia can be reversible under normoxia [33].

The overlap syndrome is associated with a more severe disease course, a high risk of exacerbations, hospitalizations, and mortality compared to the isolated course of each of these diseases. In particular, a research team from Uzbekistan showed that patients with COPD in the presence of OSA are characterized by an increase in the intensity of dyspnea, the severity of obstructive disorders, and a decrease in exercise tolerance. In addition, the course of the disease was accompanied by an increase in the number of exacerbations requiring hospitalization [34].

According to the latest data, different phenotypes of COPD suggest the participation of diverse, different pathophysiological mechanisms in the formation of SDB [35]. In particular, patients with a predominantly bronchitis phenotype of COPD are most often diagnosed with OSA, which leads to an increase in mortality rates, the risk of cardiovascular complications, hospitalizations, and the frequency of exacerbations [36]. In addition, there is growing evidence that hyperinflation of the lungs associated with emphysema reduces the likelihood of developing OSA [37]. However, in practice, the vast majority of patients with COPD have a combination of emphysema and chronic bronchitis, and thus the likelihood of developing OSA is likely to depend on the balance of protective and contributing factors in individual patients [24].

Therefore, early diagnosis of SDB in patients with COPD allows for identifying concomitant pathologies,

which, in turn, contributes to the development of adequate treatment strategies and improvements in the patient's quality of life. In addition, a correct assessment and monitoring of sleep can contribute to more accurate control of clinical manifestations of COPD. Consequently, the diagnosis of SDB in this category of patients becomes an integral part of a comprehensive approach to treatment and health maintenance.

SLEEP DISORDERED BREATHING AND INTERSTITIAL LUNG DISEASES

Interstitial lung diseases (ILD) are parenchymatous lung diseases characterized by a chronic, sometimes rapidly progressive course and a high mortality rate. ILD includes about 130 nosological entities of known and unknown etiology. One of the most unfavorable diseases of this group is idiopathic pulmonary fibrosis (IPF). However, with the development of inflammation and fibrosis of the pulmonary interstitium and air spaces, other interstitial diseases can acquire a progressive course and be very close to IPF in severity, progression of respiratory failure, and mortality prognosis.

The patient's life expectancy from the moment of IPF diagnosis is 2 to 5 years. Hypoxic vasoconstriction, obliteration, and remodeling of the vascular endothelium contribute to the development of pulmonary hypertension, which is an unfavorable prognostic sign for the course of ILD. The most common cause of death in this group of patients is progressive respiratory failure [38]. Factors that determine the course and prognosis of patients with ILD include age, forced vital capacity (FVC), diffusing capacity of the lungs for carbon monoxide (DLCO), and exercise tolerance [39, 40].

It is known that due to ventilation dysfunction and gas exchange limitations, SDB is a very common concomitant pathology of ILD, varying from 45 to 90% depending on the diagnostic methods used. Most of the studies on co-occurring ILD and SDB are devoted to IPF and lung damage in systemic connective tissue diseases. Despite the small number of studies, it was shown that OSA and nocturnal hypoxemia are associated with progression and adverse outcomes of the disease. In the study by N.I. Laz et al., 69 patients with ILD identified by high-resolution chest computed tomography were divided into groups with and without OSA. Patients were assessed using the STOP-BANG questionnaire, Epworth Sleepiness Scale, and nocturnal polysomnography to diagnose and classify SDB. More than half of the patients (60.9%) had SDB, of which 57.1% had OSA, the incidence of mild OSA was only 21.7% [41].

In a prospective study of 46 patients with ILD that lasted for 18 years, a multivariate regression analysis showed that exercise desaturation (hazard ratio (HR) 8.2; 1.8–36.5 95% confidence interval (CI); $p = 0.006$) and apnea/hypopnea index ≥ 30, namely the threshold for severe OSA (HR 7.5; 1.8–30.6; $p = 0.005$), were the only independent variables associated with disease progression [42].

In another prospective observational study, 102 patients with ILD who did not have daytime hypoxemia underwent a home sleep study for 1 year. Nocturnal hypoxemia was defined as ≥ 10% of the total sleep time with $SpO_2 < 90\%$, and OSA was detected if the apnea/hypopnea index was ≥ 15 events / hour. Nocturnal hypoxemia was detected in 20 (19.6%) of them, and OSA in 32 (31.4%). Nocturnal hypoxemia was associated with a significant deterioration in the quality of life and a higher risk of death from all causes within one year (HR 8.21; 95% CI 2.4–28.1; $p < 0.001$). A similar association was not found for OSA [43].

According to some authors, treatment of SDB in patients with IPF can improve the quality of life and disease prognosis. In a prospective pilot study, 50 patients with IPF and SDB were systematically monitored and received CPAP therapy and/or nocturnal oxygen therapy depending on the type of SDB. Sleep studies revealed some type of SDB in 70% of patients: OSA – in 36% of cases, central sleep apnea – in 22% of cases, and nocturnal hypoxemia – in 12%. Over the course of one year of therapy, polysomnography revealed an improvement in the morphological parameters of IPF, while no significant changes in the functional parameters were noted. The authors conclude that episodes of apnea/hypopnea in patients with IPF contribute to recurrent traction lung injury and enhance fibrotic changes [44].

Future prospective randomized studies with a longer follow-up period will allow to study in detail the mechanisms of interaction between ILD and SDB, assess their impact on the quality of life of patients, and develop effective methods for treating this combined pathology.

SLEEP DISORDERED BREATHING AND RESPIRATORY CANCER

Recent studies have shown that there is a link between SDB and an increased risk of cancer development and progression [45]. According to modern scientific data, there are a number of mechanisms that contribute to this link. The key pathogenetic factor is the presence of intermittent hypoxia at night [46]. Hypoxia is an important component of carcinogenesis; it can enhance the malignant properties of tumor tissue: promote more aggressive tumor growth, active proliferation, invasion and metastasis, reduce the effectiveness of radiation therapy or chemotherapy; increase the frequency of cancer recurrence and mortality [47].

In 2012, according to the Wisconsin Cohort Study, which lasted more than 20 years, a link was shown between SDB and cancer mortality, and this link remained significant after adjusting for possible concomitant variables, including age, gender, smoking, BMI, physical activity, diabetes mellitus, waist circumference, and sleep duration [48]. A study by N. Marshall et al. published in 2014 showed that moderate or severe OSA (apnea/hypopnea index > 15) was associated with a relative risk of 2.5 for cancer incidence and 3.4 for cancer mortality [49]. A later Israeli cohort study of 5,243 patients found that patients under 45 years of age with severe OSA had significantly higher incidence of all types of cancer than the general population [50]. It was shown that the presence of severe OSA was associated with a 15% increased risk of developing cancer of various localizations compared to those who did not have OSA [51].

The association between SDB and lung malignancies deserves special attention. A meta-analysis published in 2022, which included seven large studies, showed that the presence of SDB was independently associated with higher incidence of lung cancer [adjusted odds ratio (OR): 1.28; 95% CI 1.11–1.47; $p < 0.001$; 12 = 37%] [52]. The results indicate not only high incidence of malignant neoplasms in the context of SDB, but also wide prevalence of SDB among cancer patients. It was found that the prevalence of OSA among this category of patients was 46% (95% CI, 27–67), and in patients with OSA, the incidence of cancer was 1.53 (95% CI 1.01–2.31) times higher than in patients without OSA, and it depended on the severity [53]. There are data confirming significant prevalence of SDB among patients with tumors of both upper and lower respiratory tract.

Thus, according to the results of a cross-sectional study conducted by Spanish researchers among 66 patients with a confirmed diagnosis of lung cancer, the overwhelming majority (80%) were diagnosed with OSA (apnea/hypopnea index > 5) during the examination, and 50% had moderate or severe OSA (apnea/hypopnea index > 15) [54]. A recent study by a team of scientists from New Delhi also demonstrated high prevalence of SDB in patients with lung cancer. The researchers set themselves the goal of establishing the prevalence of SDB in patients with newly diagnosed lung cancer. Among 30 such patients, SDB and OSA were confirmed in 66.6 and 56.6% of patients, respectively, using polysomnography [55].

SDB is quite common in patients with tumors of the head and neck (namely, the upper respiratory tract – nasopharynx, oropharynx, larynx). The high prevalence of OSA before treatment in patients with head and neck tumors can be explained, on the one hand, by structural abnormalities due to growing tumor tissue with airway obstruction; on the other hand, the development/worsening of OSA occurs during treatment due to structural changes in the upper respiratory tract due to surgery and/or radiation therapy [56]. The authors suggest that the main reason for the worsening of OSA in patients after radiation therapy is a decrease in the function and control of the pharyngeal dilator muscle, which can affect the compliance and resistance of the upper airways [57]. It was found that the clinical cancer outcome (recurrence of the disease or mortality) in patients with head and neck tumors was significantly associated with the apnea/hypopnea index [58].

Recent publications emphasize the importance of further research on the development of OSA in patients with respiratory cancer aimed at identifying the mechanisms and developing effective pathogenetically substantiated methods of correction [59]. Timely diagnosis and treatment of SDB will reduce the potential risk of developing cancer and help improve the prognosis and course of existing tumor processes.

CONCLUSION

Thus, SDB should be considered as a risk factor for a severe course and complications of socially sensitive chronic non-communicable lung diseases. The prevalence of SDB of varying severity in

respiratory diseases is extremely high, especially in the context of comorbid pathology. In the context of the obesity epidemic and the trend towards population aging, a widespread increase in the prevalence of SDB is expected in the coming years. Timely diagnosis and elimination of SDB can be an effective measure for preventing a severe course and complications of this group of diseases.

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Received 13.08.2024;
approved after peer review 10.09.2024;
accepted 12.09.2024