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Research Article

Obstructive Sleep Apnea (OSA) A Daily Nightmare That Can Be Avoided

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Abstract:

We are living with an explosion of cardiovascular diseases in the modern world. It is known that the quality of life has worsened in the world scenario and the control of risk factors, access to medical services and adherence to the proposed treatments is the great challenge for many countries, and its excellence is a privilege of few. The knowledge of traditional risk factors such as high blood pressure, diabetes, obesity, sedentary lifestyle, increased cholesterol for the development of these diseases is known, but other factors such as sleep apnea still need to be further disseminated. A significant portion of the adult population suffers from this condition, which is considered a new risk factor for the development of cardiovascular diseases and for increasing systemic blood pressure. It is up to scientific departments and public agencies related to human health to disclose ways of suspecting this condition of later formalization of the diagnosis and proper treatment. The anamnesis looking for signs of physical fatigue, lack of concentration, drowsiness, irritability, restless sleep and the presence of snoring, associated with increased neck circumference and increased hematocrit are easy to identify. [1] Obstructive sleep apnea (OSA) should be treated as a serious risk factor for the development of cardiovascular diseases and a more intense approach is necessary.

Key words: sleep, obstructive sleep apnea, cardiovascular disease

In case of wholesome individuals awake, the airway can remain open even in the presence of clear anatomical narrowing. The respiratory center is able to compensate effectively by generating sufficient nerve impulses to promote the dilation of the pharyngeal muscles. Respiratory regulation during sleep, however, involves complex physiological mechanisms distinct from those in wakefulness. Once asleep, the stimulation from wakefulness is absent, and metabolic demand becomes the primary factor in determining the minute respiratory volume. The activity of the muscles in the upper airway then relies on local reflexes that respond to mechanical cues, such as the activation of afferent impulses. These afferent signals sent to the respiratory center, such as those triggered by low oxygen (O2) or high carbon dioxide (CO2) levels, increase the resistance load and alter the respiratory rhythm. Both heightened and reduced responses of the respiratory center can lead to abnormal gas exchange, resulting in conditions like hypoxemia, hypercapnia, or sleep-disordered breathing, including OSA.[2]

Apnea is defined by sleep study a ≥90% decrease in the airflow from the baseline value for ≥10 seconds. We further classified apnea in obstructive or central based on the presence or absence, respectively, of respiratory related chest wall excursions. Hypopnea is defined by a ≥30% decrease in the airflow from the baseline value that lasted ≥10 seconds and occurred in conjunction with ≥3% oxygen desaturation. OSA is defined by an AHI ≥15 events / hour, because mild OSA seems to not have significant cardiovascular consequences.[3], [4]

The majority patients with OSA exhibit a higher respiratory drive—the strength of output from the respiratory center—when awake compared to healthy individuals. The respiratory center's response to low oxygen (O_2) and high carbon dioxide (CO_2) levels is similar in OSA patients and healthy individuals. Only patients with a CO_2 retention condition, such as those with obesity hypoventilation syndrome (OHS) during the day, show a reduced response to low O_2 and high CO_2 levels. Individuals with OHS experience elevated $PaCO_2$ levels while awake, resulting in hypercapnic ventilatory drive. Prolonged increases in serum bicarbonate enhance CO_2 buffering capacity, reducing the drop in cerebrospinal fluid pH and subsequently diminishing the hypercapnic ventilatory response.[2]

In addition, a body of evidence shows that the primary effects elicited by OSA during sleep trigger a cascade of intermediate responses, such as increased sympathetic activity not only

during sleep but throughout the 24-hr period, that contributes to high blood pressure (BP). OSA can also contribute to increase the risk of developing HTN, contribute to poor BP control, and to blood vessel and heart remodeling.[3] The main features of OSA with intermittent hypoxia, recurrent arousals and intrathoracic pressure changes are all likely contributors to the pathogenesis of cardiovascular disease.[5] Intermittent hypoxia (IH) is a hallmark feature of OSA and substantial evidence points to a central role of this factor in the pathophysiology of cardiometabolic diseases. [5]

Furthermore, intrathoracic pressure fluctuations appear to play a key role in the development of atrial fibrillation in individuals with OSA. [5] However, the impact of increased respiratory effort on cardiovascular mortality remains insufficiently studied. The limited physiological data available indicate that fluctuations in intrathoracic pressure during respiratory events lead to heightened activity in the sympathetic nervous system, which may, in turn, accelerate arterial stiffening and remodeling of arterial walls. Furthermore, current evidence suggests that respiratory effort also plays a role in elevating nocturnal blood pressure.[6]

There is a pathophysiological basis for the link between heightened respiratory effort during sleep and hypertension. Obstructive events in sleep stem from the partial or complete collapse of the upper airway. These disruptions in airway openness lead to repeated forced inspirations against the blocked airway, causing significant negative shifts in intrathoracic pressures. These pronounced pressure fluctuations not only activate the sympathetic nervous system but also induce notable shear stress and remodeling of blood vessel walls. The effect of increased respiratory effort is under-researched and likely underestimated as a risk factor for hypertension associated with OSA.[6]

Recurrent arousals are a characteristic feature in OSA leading to sleep fragmentation and subsequent excessive daytime sleepiness as the most debilitating daytime symptom in these patients. Their occurrences depend on the arousal threshold of the individual subject but typically, arousals arise as a result of the interrupted ventilation with subsequent hypoxia, hypercapnia and increased respiratory effort in order to restore ventilation. The arousals are associated with repetitive substantial blood pressure rises. Each forced inspiration against an obstructed airway during an apnea episode generates substantial negative intrathoracic pressure. These repeated pressure shifts lead to increased venous return, causing overload of the right ventricle. Additionally, the lower pressure within the heart chambers compared to the surrounding extra thoracic structures raises the left ventricular afterload, which impairs both systolic and diastolic function and causes chronic stretching of the left atrium. [5]

Overnight laboratory polysomnography (PSG) is the diagnostic gold standard for diagnosing OSA. PSG studies analyze positional changes and body movements while sleeping, electroencephalogram (EEG) activity, respiratory rate, quality of breathing, oxygen saturation, BP, chest wall movement, and heart rate. While PSG remains the traditional standard for OSA diagnosis, modern home portables sleep monitoring are increasing in popularity among both prescribers and patients. Portables sleep are more convenient, less invasive and nearly half the cost of PSG and the diagnostic sensitivities between the two tests are statistically equal.[7]

Portable monitors have emerged as a streamlined alternative to PSG for select cases, addressing the technical complexities typically associated with PSG, which is generally confined to sleep clinics and specialized facilities. Portables sleep monitoring function as devices for testing OSA that can be operated outside clinical settings. Depending on the type, are use simplified methods and fewer connections to gather the necessary physiological data for detecting respiratory events, often requiring little to no assistance from a trained technician. Portables sleep monitoring are generally divided into three main categories: II, III, and IV, and classified by the number of channels they use to record essential physiological parameters.[8]

The sleep study type I corresponds to the PSG itself, that also known as standard PSG, employs a minimum of 7 channels, including EEG, chin EMG, EOG, ECG, airflow, effort, and oxygen saturation. Although less prescribed, sleep study type II utilizes the same number of channels as sleep study type I, but is intended for unattended use outside of the laboratory setting. Patients are initially set up with a sleep study type II device in the clinic and then sent home, where they initiate data acquisition on the device upon going to bed. Alternatively, the setup can be conducted directly at the patient's home. Sleep study type III utilizes at least 4 channels, incorporating two respiratory parameters such as airflow and respiratory movements, a cardiac parameter such as ECG or heart rate, and oxygen saturation. Sleep study type IV, on the other hand, utilizes one or two channels, typically focusing on heart rate and oxygen saturation or solely airflow.[8]

For example, a study showed that portable sleep monitoring type IV, for detecting desaturations, is a reliable method for tracking and potentially aiding in the diagnosis of OSA and determining the severity of OSA in patients with suspected OSA, as well as for determining its severity among patients with suspected OSA.[9] Prior to considering the use of portables sleep monitoring for OSA diagnosis, a pre-diagnostic clinical examination is typically conducted. During this examination, the clinician evaluates the patient's sleep history and assesses their respiratory and cardiovascular health. Symptoms such as regular loud snoring, nocturnal gasping or choking may indicate increased risk of OSA. PMs II-IV are recommended for home sleep apnea studies in symptomatic patients who do not suffer from potential respiratory muscle weakness, severe cardiopulmonary disease, or critical illness or immobility.[8]

Therefore, it is of great importance that the healthcare community is aware of the symptoms and consequences that OSA can cause when left untreated so as not to delay the start of treatment for patients. In this sense, new technologies are being used to facilitate diagnosis in a safe and effective way, to direct the best course of action for each patient.

In conclusion, we understand that there is a lot of difficulty in managing risk factors for the development of cardiovascular diseases and for their treatment. New risk factors such as depression, pollution, traffic, insecurity, financial gain, and domestic violence are being studied such as OAS as already discussed in the text. A collective and efficient effort should always be the objective of health agencies and daily medical practice to reduce the number of deaths or complications of cardiovascular diseases, which are the leading cause of death worldwide. [10]

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