

Sleep Disturbances in Cardiopulmonary Diseases

Creating awareness among patients is essential.

MANY patients with chronic medical conditions have significant sleep deprivation and disturbances. For example, insomnia patients have a 60-percent increase in health care utilization, and people who sleep less than six hours per night have an increased occurrence of ischemic chest pain.^{1,2}

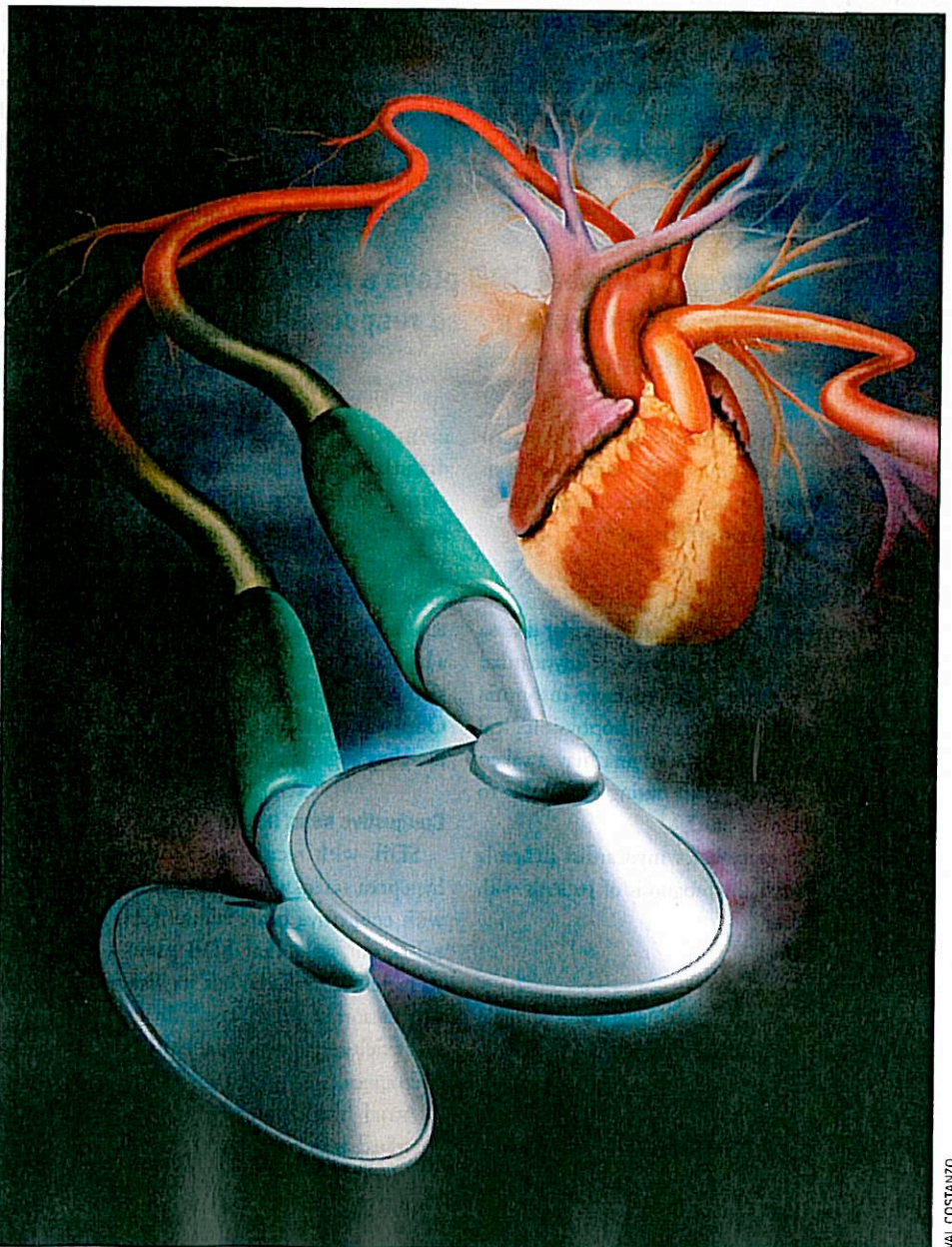
Therefore, it's essential to create an awareness of sleep disturbances and their impact on the quality of life. Health care providers can play an important role in recognizing and educating patients regarding their important conditions.

Sleep disruption is a common disorder affected by several medical conditions with multiple mechanisms. Both insomnia and obstructive sleep apnea are associated with significant comorbid medical conditions — primarily cardiac and pulmonary disease.

Patients with asthma, chronic bronchitis, or emphysema have a higher prevalence of sleep complaints than the general population, while the rates of sleep disturbances increased with patients with respiratory symptoms such as coughing and wheezing.

In addition, OSA has been linked to the development of atherosclerosis, hypertension, cardiac arrhythmias, stroke, congestive heart failure, angina, and sudden death.

Studies show that resultant structural changes to the cardiac muscle are influenced by the severity of sleep apnea, and these effects are reversible if the apneic episodes are eliminated with the use of ▶



continuous positive airway pressure.

Unknown connection

Severe congestive heart failure has been associated with sleep-disturbed breathing, possibly due to paroxysmal nocturnal dyspnea, while patients with insomnia tend to have an increased occurrence of ischemic chest pain.

Although the exact mechanism that links OSA to cardiovascular disease is unknown, there's evidence to suggest that OSA is associated with a group of pro-inflammatory and pro-thrombotic factors that have been identified as key factors in the development of atherosclerosis.³

It's also connected with daytime and nocturnal sympathetic activity. Autonomic changes seen in patients with OSA include increased resting heart rate and blood pressure variability, and decreased R-R interval variability.⁴

Evidence exists not only that SDB plays a role in the pathogenesis of CHF, but it's also responsible for progression of the disease.

Nocturnal oxygen desaturation leads to intermittent nocturnal hypoxemia which has been shown experimentally to accelerate atherosclerosis and increase catecholamine release.^{5,6}

Cardiac ischemia

The fact that patients with cardiovascular disease (angina, congestive heart failure, or myocardial infarction) are more likely to have insomnia can be explained by several pathophysiological mechanisms.⁷ Patients with angina may awaken suddenly during sleep (often during REM) due to fluctuations in cardiac rate.⁸

Hypoxia also exacerbates myocardial ischemia which may worsen the prognosis of patients with cardiovascular disease.⁹

Hypertension

Frequent repetitive apneas and/or hypopneas with oxygen desaturation during sleep found in patients who suffer from OSA may cause intermittent hypoxia, hypercapnia, arousal, and disturbed sleep which may result in increased sympathetic activation, alterations in blood pressure, and vascular atherogenic changes.⁹

Thus, sleep-disordered breathing causes

hemodynamic changes and sympathetic hyperactivity, thereby increasing the risk of thrombotic events and contributing to hypertension. This may in turn influence the development of atherosclerosis. Severe OSA and hypertension are independently associated with increased stiffness of large arteries thereby contributing to ventricular after load.¹⁰

The coexistence of hypertension and OSA is associated with additive effects on arterial stiffness and heart remodeling.

Cardiac arrhythmias

Patients with OSA have a higher frequency of cardiac rhythm disturbances and ST-segment depression episodes than snoring or control subjects. The presence of apnea and nocturnal hypoxemia is associated with an increased prevalence of cardiac arrhythmias.¹¹

Cardiac arrhythmias are a common finding in patients undergoing Holter monitoring, ECG, or polysomnography. ST-segment changes are related to sympathetic tone and sleep fragmentation, whereas most of the rhythm disturbances in patients with OSA are associated with sleep fragmentation, nocturnal hypoxemia, and urinary catecholamine release.

It's uncertain whether OSA causes nocturnal myocardial ischemia in the absence of coronary artery disease, but studies illustrate OSA patients had a higher frequency of nocturnal ST-segment depression episodes than control subjects. Incidentally, OSA was found in nine out of 10 subjects with disabling angina pectoris and nocturnal angina.^{11,12}

Congestive heart failure

SDB, with recurrent episodes of apnea and hypopnea, occurs with increased frequency in patients with congestive heart failure (CHF).¹³ Evidence exists not only that SDB plays a role in the pathogenesis of CHF, but it's also responsible for progression of the disease.¹⁴ In fact, many stable heart failure patients have either OSA or central sleep apnea.

Atrial fibrillation (AF), as obesity, has been described as an emerging epidemic. The proportion of OSA was 49 percent in AF population.¹⁵

There's a concomitant increase in the incidence of AF with obesity, and there's an increased

recurrence of AF in patients with untreated OSA, while use of CPAP can lower the risk of recurrence.

The presence of OSA should be considered in all patients with AF, and screening is warranted in AF patients who are obese or hypertensive.¹⁵

Low left ventricular ejection fraction (LVEF) has been associated with sleep breathing disturbances (e.g., Cheyne-Stokes respiration, OSA) and may cause disruption of sleep due to paroxysmal nocturnal dyspnea.

Given the effectiveness of CPAP in improving cardiovascular end points, study results support the adoption of screening stable heart failure patients with LVEF < 45 percent.¹³

Stroke

The majority of stroke and transient ischemic attack patients have unrecognized and untreated OSA. There's some evidence to suggest that OSA and hypopneas may play a role in the pathogenesis of stroke via several hematologic and hemodynamic changes.¹⁶

OSA has been associated with hypertension, hypercoagulable state, decreased cerebral perfusion, and altered cerebral auto regulation.¹⁵ Often stroke results in pharyngeal dysfunction and subsequent OSA.

Sleep apnea has been found at an alarming rate in patients with acute stroke as well as after neurological recovery, leading some to suggest that it was present before the stroke.

Understanding the link between OSA and stroke may provide a novel preventative and therapeutic approach in the management of stroke.¹⁶

The predominant apnea is of the obstructive type, not central, leading to increased stroke mortality and morbidity. OSA is a modifiable risk factor not only for stroke but also for cardiovascular disease and sudden death.¹⁷ Moreover, chronically disturbed sleep can cause an increase in health care utilization.¹⁸

COPD

Chronic obstructive pulmonary disease is the fourth leading cause of death and afflicts millions of people and generally features progressive irreversible airflow obstruction.

Although sleep is commonly disturbed in these patients, they seldom volunteer sleep-related symptoms.¹⁹ Thus, sleep disorders in this population may continue to be unrecognized while having a significant impact on the quality of their lives.

There are many reasons that sleep may be poor in this patient population. First, sleep onset might be delayed by cough and excessive mucous production, particularly because these symptoms may be exaggerated in the supine position.

Once asleep, these patients are at risk for oxygen desaturation as a result of sleep-related hypoventilation, a reduction in functional residual capacity and increase ventilation-perfusion mismatch.

Such changes are most obvious during rapid eye movements.²⁰

In response to hypoxemia and/or hypercapnia, ventilation increases, as does respiratory effort, resulting in arousal. The severity of hypoxemia during sleep can be predicted by the degree of waking

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ventilatory response to hypercapnia.²¹

Supplemental oxygen therapy during sleep can eliminate significant hypoxemia, but sleep quality may not improve substantially.^{22,23}

Many studies have shown the high prevalence of chronic insomnia in COPD.^{19,24-27}

Researchers reported more than 50 percent of subjects with chronic bronchitis or emphysema have disorder of initiating and maintaining sleep (DIMS), and more than 25 percent had excessive daytime sleepiness.¹⁹

In a later study, both DIMS and excessive daytime sleepiness complaints were related to the presence of respiratory symptoms more strongly than to the diagnosis of lung disease or the degree of airway obstruction.²⁴

In addition, the more symptoms the subject had, the higher the rate of sleep complaints.

Investigators found emphysema was independently associated with early awakening in men.²⁶ Insomnia was a comorbid condition in 17 percent of COPD patients and ranked only third to locomotor disease and hypertension.^{28,29}

Another study confirmed 44 percent of elderly Italians with COPD had nocturnal awakenings, followed by morning tiredness (33 percent), early awakenings (30 percent), and difficulty falling asleep (26 percent).³⁰

Other reports link depression, smoking,

orthopnea, and nocturnal desaturation — all highly prevalent in COPD — to insomnia.^{27-29,31-33}

While insomnia and productive cough are major complaints that impair quality of life and daytime functioning in COPD, insomnia also holds significant implications for pulmonary function.²⁸

The spirometry decline induced by one night's sleep deprivation may have significant impact on health and quality of life in patients with compromised lung function.³⁴

Many of the medications used for the treatment of COPD also can alter the sleep architecture. Several other factors may impair sleep, such as poor sleep hygiene, respiratory symptoms that causes arousals (e.g., cough, dyspnea), hypoventilation, disturbed gas exchange, sleep deprivation due to depression, and poor quality of life.

OSA also can cause sleep disturbances. Reports have instituted high association between

COPD and OSA; prevalence of overlap syndrome varies from 6 percent to 28.5 percent.³⁵⁻³⁸

The OSA-mild COPD association was chance and not pathophysiological linkage, when applying the baseline data from the Sleep Heart Health Study of a large, diverse, middle-aged community population, and defining OSA with an apnea-hypopnea index above 10 or 15 and air flow obstruction as FEV₁/FVC of less than 70 percent.³⁹

However, both the conditions can cause desaturation during sleep, which may lead to sleep fragmentation.

Interstitial lung disease

Patients with interstitial lung disease (ILD) have significant desaturation and altered breathing patterns during sleep. This can lead to major respiratory and sleep disturbances, mainly during REM.

In one study, patients with ILD have a rapid shallow breathing pattern while awake that's thought to be due to activation of lung reflexes.⁴⁰ Researchers studied 11 patients with ILD (five men and six women) during sleep and 11 age- and sex-matched controls.

Sleep quality was worse in patients with ILD compared to controls, with more time in stage 1 (33.7 percent of total sleep time [TST] vs.

13.5 percent) and less time in REM sleep (11.8 percent TST vs. 19.9 percent), and more fragmentation of sleep (13.7 +/- 3.1 arousals/hour and 24.3 +/- 6.0 sleep stage changes/hour vs. 6.9 +/- 1.0 and 12.7 +/- 1.4, respectively).

Patients with ILD with awake SaO₂ less than 90 percent had greater abnormalities in sleep structure than did those with SaO₂ greater than 90 percent. The incidence of apneas and hypopnea periods in patients with ILD was low. Oxygen saturation dropped during REM sleep in patients, especially in those with more severe awake hypoxemia.

Mechanical ventilation

Patients dependent on mechanical ventilation in an intensive care unit sleep poorly. Sleep architecture is abnormal, with occasional disappearance of some stages of sleep, and there are frequent arousals and awakenings.

About half of TST occurs during daytime, and circadian rhythm is markedly diminished or lost. The degree of sleep fragmentation is at least equivalent to that seen in patients with OSA.

Sleep disruption can induce sympathetic activation and elevation of blood pressure, which may contribute to morbidity.

Judgments based on inspection consistently overestimate sleep time and don't detect sleep disruption.

Accordingly, reliable polysomnographic recordings are needed to measure sleep quantity and quality in critically ill patients.

Because most arousals are associated with caregiving activities or noise, staff should consolidate care and decrease environmental noise.⁴¹ Even though abnormalities of sleep are extremely common in critically ill patients, the mechanisms are poorly understood.

Measures to improve the quantity and quality of sleep in critically ill patients include careful attention to mode of mechanical ventilation, decreasing noise, and sedative agents.

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