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Current medical concepts in obstructive sleep apnea

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Obstructive sleep apnea (OSA) is the most common form of sleep-disordered breathing. OSA is characterized by repetitive upper airway closure causing intermittent hypoxia. The prevalence of OSA has significantly increased with the global epidemic in obesity. Despite the recognition of the serious health consequences of OSA, many patients with OSA remain underdiagnosed. This article provides a review of the epidemiology, the physiologic mechanisms, and the current nonsurgical interventions for OSA.

Historical perspective

Despite the high prevalence of sleep apnea in the general population, the recognition of this condition is relatively new. Although early images suggestive of sleep apnea appeared in antiquity, the first formal medical descriptions were delayed until Broadbendt's clinical description of periodic breathing in heart failure in 1877 and the report of obstructive respiratory events during sleep in the mid-20th century[1]. The subsequent explosion of information on sleep apnea has been enabled by polysomnography, which serve as the diagnostic tool for obstructive sleep apnea (OSA) and the caliper for metrics of severity. The development of effective and acceptable treatments was dominated by device therapies starting with continuous positive airway pressure (CPAP) in 1981.

The more narrow syndrome of obstructive sleep identified in these founding descriptions of disordered breathing are now incorporated within a complex constellation of sometimes coexisting conditions. The prevalence and scope of these diverse forms of sleep-related breathing disorders[2] have also expanded because of increase in obesity, narcotic use, and recognition of its presence in medical literature among the population (Table). The complexity of sleep-related breathing disorders is classified[2] within the group of highly prevalent conditions that are implicated in serious but treatable health problems. Treatment of sleep apnea has become largely non-pharmacologic with emphasis on device therapy and surgical procedures in selected cases. This discussion of the current understanding of the medical aspects of sleep apnea provides a context for surgical practice in sleep medicine.

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Causes of changing prevalence

Obesity

In the United States, 2 decades ago, 68.2% (or ~154.7 million) adults above the age of 20 years had a body mass index (BMI) > 25 kg/m^2. Approximately 4% of middle-aged men and 2% of middle-aged women had OSA, based on apnea or hypopnea index (AHI) of >5 and excessive sleepiness during the day.[3] Since then, the global prevalence of OSA has sharply increased along with the surge in obesity worldwide. In the United States, more than one-third

Table Diversity of sleep-related breathing disorders

Obstructive sleep apnea
Central sleep apnea
Primary
Cheyne-Stokes
High altitude
Neurologic/medical conditions
Periodic breathing
Treatment-Emergent (positive airway pressure)
Hypoventilation
Congenital
Idiopathic
Narcotics
Hypoxemia
Cardiopulmonary disease
Hypoventilation

of adults are now obese[4]—a clear contributor to the increased prevalence rates of OSA.

There is robust evidence from both population- and community-based studies that body weight plays a major role in the development of OSA. OSA is seen in more than 50% of obese patients with mean BMI higher than 40 kg/m^2.[5] The linear relationship between weight and the severity of OSA is well established and bi-directional, with increases in body weight leading to increased severity of OSA and, at the same time, OSA contributing to weight gain.

Generally, an excess of body fat is more frequently associated with metabolic abnormalities when compared with a high level of lean body mass. BMI is widely used by the scientific community to assess obesity. However, BMI cannot distinguish between the lean or fat mass, assess the distribution of fat and the change of this distribution with age, or take into account the changes that occur in people who begin a diet accompanied or not by exercise. Recent studies[6-8] suggest that waist height ratio is likely to be the best anthropometric index to assess the association of adiposity with cardiovascular risk or overall mortality. The stronger predictors of the severity of OSA in obese patients are neck circumference in men and BMI in women.

Using the data from the Wisconsin Sleep Cohort Study, Peppard and Young estimated the prevalence of sleep-disordered breathing (SDB) in the United States for the periods 1988-1994 and 2007-2010.[9] In their study, SDB was modeled as a function of age, sex, and BMI, and estimates were extrapolated to US BMI distributions estimated using data from the National Health and Nutrition Examination Survey. Peppard and Young reported that approximately 13% of men (aged 30-70 years) and 6% of women (aged 30-70 years) have moderate to severe OSA (AHI $\geq$ 15), and another 14% of men and 5% of women have an AHI $\geq$ 5, along with symptoms of daytime sleepiness.[9]

In addition to obesity, age contributes to OSA, with an increased risk of OSA in the elderly population.[10] In the United States, by 2050, the number of older adults (aged $\geq$65 years) is expected to be more than double, rising from 40.2 to 88.5 million.[11] With the increase in the aging population, the number of obese older adults will likely grow, thereby increasing the prevalence of obesity-related chronic medical conditions, including OSA. Bixler et al[3] demonstrated that the prevalence of OSA significantly increased with age among both men and women. Fat deposition in the parapharyngeal region, structural changes in parapharyngeal area, and soft palate elongation are some of the proposed mechanisms causing upper airway obstruction in the elderly population.[12,13]

Comorbidities

Compared to the general population, the prevalence of OSA is much higher ($\geq$50%) in patients with cardiac and metabolic disorders, and this adds complexity in the management of these patients. In addition, OSA and hypertension often coexist. Roughly 50% of OSA patients suffer from hypertension,[14] and approximately 30% of patients who have hypertension also have concurrent OSA, which is often undiagnosed.[15,16] OSA is also associated with several cardiovascular disorders that include coronary artery disease, congestive heart failure, hypertension, cardiac arrhythmias, and stroke, which further increase morbidity and mortality.

Both obese patients and OSA patients have elevated circulating levels of leptin, which has been associated with the development of cardiac hypertrophy, a recognized risk for heart failure.[17,18] Patients with heart failure can have different patterns of SDB, including OSA, central sleep apnea (CSA), and Cheyne-Stokes respiration, all associated with increased mortality.[19]

OSA is also more prevalent in patients with congestive heart failure than in the general population.[20] Based on a prospective study that included 700 patients with heart failure, Oldenburg et al[21] reported that 76% of the patients with heart failure had SDB; about 40% patients had CSA, while 36% had OSA. As a result, a number of treatments have been investigated, with varying results. While many therapies may improve the severity of SDB, only positive pressure ventilation has been shown to improve cardiac function. Newer forms of positive pressure ventilation, such as adaptive servo-ventilation, appear to be even more effective in correcting CSA.[22]

Narcotics

The use of opioid medications for management of chronic pain has increased over the past 2 decades. Caudill-Slosberg et al[23] reported that the prescriptions of opioids for chronic pain have increased from 8% in 1980 to 16% in 2000. They also reported an increase in the prescription of potent opioids such as morphine, hydromorphone, and oxycodone for management of chronic pain from 2%-9%. Patients who are on chronic opioids have irregular breathing pattern with central apneas during sleep