The Relationship of Obesity and Obstructive Sleep Apnea

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- Sleep-disordered breathing • Obesity
- Medical weight-loss therapy • Bariatric surgery
- Dietary weight loss • Sibutramine

Obesity is a chronic disease that has become epidemic in the United States and worldwide. Approximately 127 million adults in the United States are overweight (body mass index [BMI] 25.0–29.9) and 60 million are obese (BMI >30.0).[1,2] The World Health Organization predicts that by 2015, approximately 700 million adults will be obese (at least 10% of the world's projected population).[3] Obesity is a major risk factor for various disorders, including obstructive sleep apnea (OSA), a sleep-related breathing disorder characterized by recurrent upper-airway obstruction during sleep, which results in a cycle of hypoxemia, increased respiratory effort, and frequent arousals. In addition to being a risk factor for OSA, obesity may be a consequence of OSA. This article reviews the role of obesity in the development of OSA and discusses the two-way relationship between obesity and OSA. It also reviews the evidence on the effects of treatment of both disorders on their interrelationship.

DEFINITION OF OBSTRUCTIVE SLEEP APNEA

An apnea is defined as the cessation of airflow for at least 10 seconds in the presence of thoracoabdominal ventilatory efforts. A hypopnea is a reduction in airflow of at least 30% with a decrease in oxygen saturation of 2% or more for at least 10 seconds in the presence of thoracoabdominal ventilatory efforts.[4] The apnea–hypopnea index (AHI) is the sum of apneas and hypopneas per hour of sleep. An AHI of 5 or more per hour is established as a criterion for the diagnosis of OSA according to the criteria of the American Academy of Sleep Medicine.[4] However, there are various definitions of hypopnea. A survey of American Academy of Sleep Medicine–accredited sleep centers found that no two laboratories used the same definition of hypopnea,[5] and differences include various degrees of reduction in airflow and changes in thoracoabdominal movement, associated oxygen desaturation, and arousal. Such differences have important implications for the diagnosis of OSA and the standardization of research results. Recent population studies correlating the AHI with cardiovascular disease have helped to standardize the definition of hypopnea. The Sleep Heart Health Study, which was a large, multicenter trial designed to relate cardiovascular disease with polysomnographic findings, defined hypopnea as a 30% reduction in airflow or chest wall movement from baseline movement for at least 10 seconds and accompanied by oxygen desaturation of 4% or greater.[6]

The evidence for the prevalence of OSA derives from pooled data from four large studies that used similar in-laboratory monitoring techniques, diagnostic criteria, and sampling methods. From these data, it is estimated that in Western countries 24% of men and 15% of women have OSA and 4% of men and 2% of women have OSA with symptoms of sleepiness.[7]

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RISK FACTORS AND HEALTH CONSEQUENCES OF OBSTRUCTIVE SLEEP APNEA

Many risk factors for OSA have been identified in addition to obesity, including increasing age, being male, abnormal craniofacial morphology, nasal obstruction, genetic factors, and endocrine abnormalities such as thyroid disorders.[8,9] However, the true difference in risk between the two sexes is not clear. In women, menopausal status is likely a factor, with postmenopausal women being at a much higher risk for OSA than premenopausal women.[10] Some studies suggest that black Americans are at greater risk than whites.[11] There are many cardiovascular[12] and other consequences of OSA (**Table 1**). Peppard and colleagues[13] found that patients who had mild OSA had a 42% greater risk for developing hypertension in 4 years compared with patients who did not have OSA and who were matched for body habitus, age, sex, smoking status, and alcohol use. Patients with more severe OSA had a much higher risk of having hypertension at a 4-year follow-up. OSA has also been strongly associated with cardiac death.[23] The relative risk of sudden death from cardiac causes during sleep was 40% higher in patients who had severe OSA (AHI $\geq$40/hour) than in patients who had mild to moderate OSA (AHI 5–39/hour). In a large cross-sectional study, OSA was associated with an increased prevalence of self-reported heart failure and stroke.[19] OSA has also been shown to be independently associated with coronary artery disease after adjustment for traditionally considered risk factors.[15] Mooe and colleagues[25] showed that sleep-disordered breathing in patients who had coronary artery disease was associated with a worse long-term prognosis. They found that patients who had an AHI of 10 per hour or less had a 62% relative increase and a 10.1% absolute increase in the composite endpoints of death, stroke, and myocardial infarction. OSA has been detected in more than 50% of patients who had an acute stroke and patients after recovery from stoke, indicating that OSA had been present before the stroke.[26] In a recent, large, prospective cohort study, Yaggi and colleagues[17] found that OSA increases the risk of stroke or all-cause mortality (hazard ratio 1.97), independent of other cerebrovascular risk factors, including hypertension.

Table 1
Medical consequences of obstructive sleep apnea

Cardiovascular Consequences	Noncardiovascular Consequences
Hypertension[13]	Motor vehicle accidents[14]
Coronary artery disease[15]	Impaired cognitive performance[16]
Stroke[17]	Depression[18]
Heart failure[19]	Occupational accidents[20]
Arrhythmias[21]	Poor exercise tolerance[22]
Death from cardiac causes[23]	Poor marital relationship[24]

OBESITY AS A RISK FACTOR FOR OBSTRUCTIVE SLEEP APNEA

The BMI is a reliable measure of body fat and body fat mass. A normal BMI is between 18.5 and 24.9. A person who has a BMI of 25 to 29.9 is considered to be overweight, and a person who has a BMI of 30 or more is considered to be obese; there are three classes of obesity based on the BMI (**Table 2**). The prevalence of obesity in the United States has been increasing for decades (**Fig. 1**). About 60 million adults (30% of the adult population) are obese, twice the percentage from 1980.[27,28]

Obesity can affect the structure and function of the upper airway. The upper airway has three primary functions: swallowing, phonation, and breathing. In breathing, various forces promote airway collapse and airway patency. The two primary forces that tend to collapse the airway are the intraluminal negative pressure generated by the diaphragm during inspiration and the extraluminal tissue pressure, which is the pressure resulting from tissues and bony structures surrounding the airway. These forces are counterbalanced primarily by the action of the pharyngeal dilator muscles, although longitudinal traction on the airway resulting from lung inflation likely contributes as well. Upper-airway narrowing seems to be an important factor in OSA. Obesity can cause narrowing of the pharynx because of the effects of subcutaneous and periluminal fat

Table 2
Classification of obesity in adults

BMI	Body Characteristics	Obesity Class
25.0–29.9	Overweight	—
30.0–34.9	Obesity	I
35.0–39.9	Obesity	II
$\geq$40.0	Extreme obesity	III

Data from National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. NIH publication no. 98-4083. National Institutes of Health, 1998.