Clinical Overview of Sleep-Disordered Breathing

Sleep-Disordered Breathing (SDB)

The term "sleep-disordered breathing" (SDB) refers to a variety of breathing disorders that can occur during sleep. SDB has become the general term used to describe many disease states that manifest breathing abnormalities during sleep. These include obstructive sleep apnea (OSA), central sleep apnea (CSA), and hyperventilation or hypoventilation occurring in patients suffering from chronic respiratory diseases (chest wall, neuromuscular, ALS, or lung diseases, such as Chronic Obstructive Pulmonary Disease) or chronic heart failure. The most common form of SDB is OSA, which is characterized by repetitive episodes of upper airway obstruction. While the mechanisms of SDB differ, a basic explanation of obstructive sleep apnea provides the best introduction to this group of diseases and their significance.

Sleep-disordered breathing (SDB) is characterized by apneas and hypopneas.

- Apnea: a cessation of airflow for ≥ ten seconds with or without oxygen desaturation
- Hypopnea: 50% or greater decrease in flow for ≥ 10 seconds or more with or without oxygen desaturation.
- In a sleep study, polysomnography (PSG), events less than 10 seconds in duration are typically scored as events if accompanied with ≥ 3% oxygen desaturation

Apneas and hypopneas result from upper airway occlusion, either full or partial, or from a loss of the autonomic drive to breathe (central component free of upper airway obstruction).

Breathing resumes when the patient has a brief sleep arousal, of which they usually have no memory. In severe cases, patients may have up to 100 events per hour, resulting in severe daytime symptomatology. Disease severity is usually classified according to the apnea/hypopnea index (AHI). Measured during a sleep study, AHI refers to the number of apneas and hypopneas per hour of sleep.

AHI	SDB Severity
5-14	Mild
15-29	Moderate
30 or more	Severe

Signs and Symptoms

OSA has been called the "snoring disease," but even OSA patients do not always snore, so SDB may not be the obvious cause of a patient's symptoms. Following are the most common symptoms:

- Excessive daytime somnolence (EDS)
- Tiredness
- Witnessed apneas
- Snoring
- Gasping attacks, restless sleep
- Irregular breathing during sleep
- Morning headaches
- Depression / moodiness
- Obesity
- Sexual dysfunction

In addition to AHI, oxygen desaturation and excessive daytime sleepiness indicate the severity of SDB. Physicians should diagnose and prescribe treatment in accordance with the symptoms and the sleep study results; however, subjective and objective findings do not always match. In some cases, patient complaints can differ greatly from the findings of the sleep study. The necessity of treatment to prevent disease progression, patient compliance with prescribed treatment, and patient satisfaction can become complicated in these situations.

Prevalence OSA

OSA is the most common form of SDB. The American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) patient information states that 45% of normal adults snore occasionally and 25% are habitual snorers. A frequently referenced study by Young et al reports that as many as 29% of adult males and 9% of adult females have some degree of obstructive sleep apnea/hypopnea syndrome (OSAHS).[1] Using a criterion of an AHI of 15 events per hour with EDS, numerous studies report a 4% incidence of OSA in adult males and 2% in adult females. The prevalence of OSA peaks around the age of 48 years and is either less common in youth and old age or recognized less often.

OSA is found in all population groups, from newborns to the elderly. While obesity may increase the risk of OSA in some patients, it is not the cause of OSA. People of all body types have OSA. While women enjoy some hormonal protection, once they go through menopause their incidence is the same as men's.

Associated Risks

Researchers continue to develop an understanding of the risks created by and associated with SDB. Associated risks include the following:

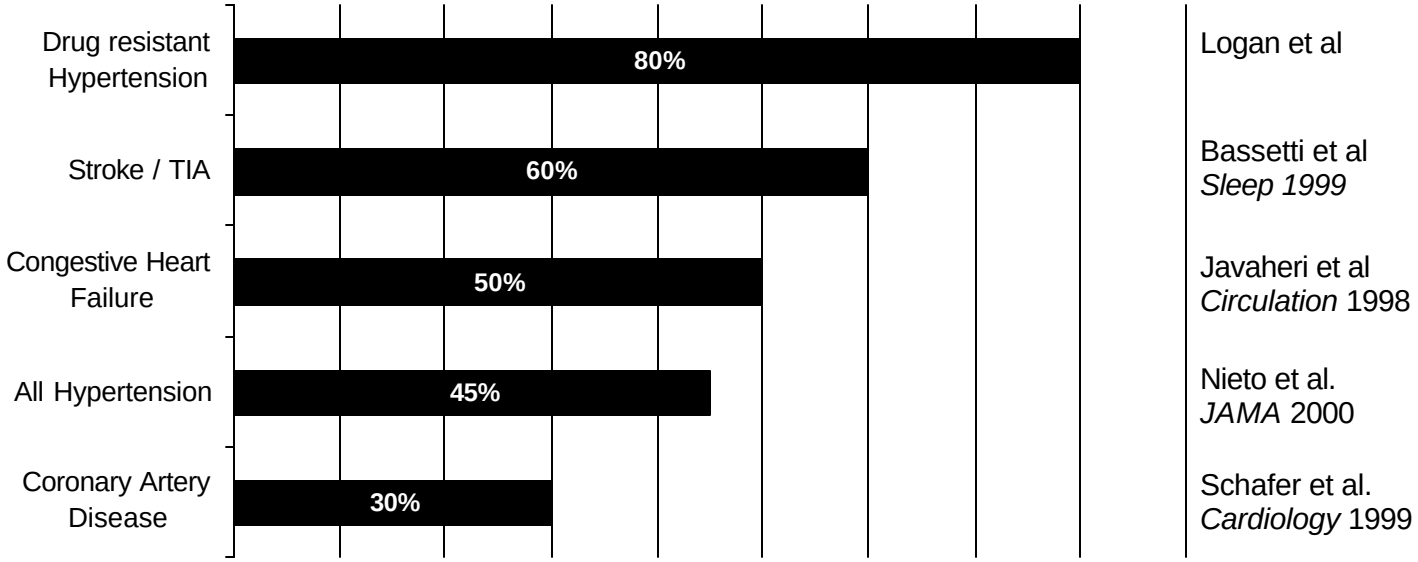
- Hypertension
- Stroke
- Congestive heart Failure (CHF)
- Traffic accidents

SDB and Cardiovascular Disease

Research from the past several years indicates SDB as a cause of hypertension, the primary risk factor for cardiovascular disease. This knowledge among medical researchers has finally found its way into screening and treatment guidelines. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7) identifies sleep apnea as a cause of hypertension. For more information about JNC7 go to

http://www.nhlbi.nih.gov/guidelines/hypertension/index.htm

While the exact relationship between SDB and CVD demands further investigation, much of the basic relationship and the levels of prevalence are clear. Sleep arousals related to obstructive events are associated with increases in blood pressure and heart rate.[2,3] More importantly, a number of studies now suggest that untreated SDB may be associated with significant cardiovascular responses. Studies have also demonstrated that the decrease observed in blood pressure during sleep in normal subjects does not occur in patients with SDB.[4,5]



SDB apneic events cause an impairment of gas exchange that ultimately leads to severe hypoxia and hypercapnia and an increase in inspiratory drive. During each apnea, there is a progressive increase in sympathetic nerve activity. This increase in nerve activity leads to vasoconstriction, increasing peripheral vascular resistance in the systemic and pulmonary vasculature, and a progressive rise in arterial pressure. Patients with SDB have been found to have higher levels of sympathetic activity (both nerve activity and catecholamine levels) while awake when compared to non-apneic people.[6] This chronic increase in daytime sympathetic nerve activity has been linked to the development of daytime hypertension and cardiac arrhythmias.[7] Additionally, changes in intrathoracic pressure as a result of ineffective respiratory efforts influence venous return, ventricular filling, arterial and cardiopulmonary baroreflexes, and the release of atrial natriuretic peptide.[8,9]

Studies have also demonstrated increased sympathetic nerve activity in patients with SDB, and researchers suggest that this mechanism contributes to the development of hypertension in SDB patients.[10,11] Controversy persists about the mechanism responsible for the hemodynamic changes, but the hypoxemia and arousals characteristic of SDB are considered the primary causes.

Though unanswered questions exist, researchers know that SDB has a profound association with hypertension independent of all other risk factors.[12] SDB is associated with systemic hypertension in middle age and older individuals of both sexes (after controlling for potential confounders).[13] Most significantly, Peppard et al have shown that SDB increases the risk of hypertension and cardiovascular morbidity in the general population (independent of known confounding factors) and established that untreated SDB causes for hypertension.[14]

Chronic hemodynamic consequences of SDB include nocturnal hypertension, diurnal hypertension, and pulmonary hypertension. Additionally, some evidence indicates that left ventricular mass is greater in patients with SDB than in age-matched controls.[15] One study of 147 men and women demonstrated an increase in hypertension in patients with an AHI greater than 5 events per hour as compared to a control group matched for obesity, age, and sex.[16]

Traffic Accidents

Researchers have known for some time that sleepy drivers cause a large number of traffic accidents. Researchers have also known that individuals with SDB have an increased risk of involvement in traffic accidents. Two studies looked specifically at the probability of traffic accidents for people with sleep apnea syndrome (SAS). Teran-Santos et al found that individuals with SAS are over six times more likely to be involved in a traffic accident.[17] Horstmann et al found that individuals with SAS are 15 times more likely to be involved in a traffic accident.[18] These findings highlight yet another risk that individuals with SDB may not recognize.

Increased Mortality and Health Care Costs

Evidence also points to increased mortality rates for patients with untreated SDB. Using the U.S. age-adjusted survival curve, Partinen et al reported a five-year decrease in survival for patients with untreated SAS (compared to patients treated by tracheostomy).[19] Partinen et al demonstrated decreased survival in SAS patients with an AHI of greater than 20 events per hour. This difference proved most evident in patients less than 50 years of age. Research also indicates that individuals with untreated SAS utilize more resources within the healthcare system.[20, 21, 22]

Diagnostic Testing of SAS

Most insurance carriers require a full polysomnography (PSG) for diagnosis of SAS. The American Academy of Sleep Medicine accredits sleep labs and provides a lab locator on their Web site (http://www.aasmnet.org/listing.htm).

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