OSA Related to Hypertension and Heart Failure

The more severe the sleep apnea, the higher a patient's nocturnal and daytime blood pressure.

BY CAROLINE HELWICK
Contributing Writer

NEW ORLEANS — Obstructive sleep apnea appears to contribute importantly to both the development and severity of hypertension and may play a role in heart failure as well. The good news is that regular use of continuous positive airway pressure not only treats the apnea but also lowers blood pressure in some patients, according to speakers at the annual meeting of the American Society of Hypertension.

Obstructive sleep apnea (OSA) has been observed in approximately 40% of persons with treatable hypertension, compared with approximately 25% of men and 10% of women in the general population, according to Dr. David Calhoun of the department of medicine at the University of Alabama at Birmingham.

"A number of studies suggest that nocturnal blood pressure may be a better predictor of cardiovascular outcomes than daytime elevations in blood pressure, so there is growing interest in what is happening during the night, especially when blood pressure fails to decrease. One factor in this is obstructive sleep apnea," Dr. Calhoun said at a press conference on the topic.

Others have found a dose-dependent increased risk of developing hypertension in relationship to OSA. In a prospective evaluation of normotensive patients, those with the most severe OSA at baseline had more than twice the risk of developing hypertension over 4 years (JAMA 2000;283:1829-36).

"This confirmed the relationship between OSA and hypertension, establishing sleep apnea as a potential cause," he said.

Other studies also have found that the more severe the sleep apnea, the higher a patient's nocturnal and daytime blood pressure, as well. One important study documented the overall prevalence of OSA (defined as more than 10 events per hour) to be 83% among persons with drug-resistant hypertension, including 96% among men and 65% among women.

"You are seemingly at much higher risk of having sleep apnea if you have difficult-to-control hypertension. And it suggests that having sleep apnea contributes to difficulties in treating hypertension," Dr. Calhoun noted.

OSA also has been associated with heart failure, according to Dr. Alexander G. Logan of Mount Sinai Hospital, Toronto, and the University of Toronto. In the Sleep Heart Health Study (Am. J. Resp. Crit. Care Med. 2001;163:19-25), persons with sleep-disordered breathing had an odds ratio of 2.38 for developing heart failure, as well as an increased risk of stroke and coronary heart disease, vs. those without. Many other studies have also shown an increased risk of OSA in persons with heart failure, he said.

In normotensive patients, those with the most severe OSA at baseline had more than twice the risk of developing hypertension over 4 years.

Treatment of OSA with continuous positive airway pressure (CPAP) may help some patients, a number of studies have shown, the speakers said.

Although the data may not be "very compelling," according to Dr. Calhoun, randomized studies have shown that about 5 hours of CPAP per night is associated with small reductions in mean arterial pressure and about 10 mm Hg reduction in systolic and diastolic pressures. In one study of patients with resistant hypertension, regular use of CPAP for 2 months was associated with substantial reductions in 24-hour, daytime, and nocturnal blood pressures (Eur. Respir. J. 2003;21:241-7), a finding that established CPAP as an important adjunct to treatment of patients with resistant hypertension, he said.

"The benefit of CPAP appears to be strongest in nocturnal blood pressures," he added. "CPAP appears to help restore the 'dipping' pattern (10% decrease in blood pressure) overnight."

Dr. Calhoun believes this translates into cardiovascular benefits. French investigators found fewer cardiovascular events among hypertensive patients who adhered to CPAP for 5 years vs. those patients who discontinued CPAP (Eur. Heart J. 2004;25:728-34). Event rates were 24% vs. 58%, respectively.

Dr. Logan noted that in medically treated heart failure patients with OSA, the use of CPAP reduces blood systolic blood pressure, partly as a result of a decrease in sympathetic vasoconstrictor tone; improves left ventricular systolic function; improves baroreflex sensitivity; decreases the frequency of ventricular premature beats; and improves the quality of life in hypersomnolent patients. ■

Sleep Apnea Disrupts Classic Circadian Pattern of Sudden Cardiac Death

BY BRUCE JANCIN
Denver Bureau

SNOWMASS, COLO. — Individuals with obstructive sleep apnea exhibit a striking alteration in the typical day-night pattern of sudden cardiac death, underscoring the sleep disorder's potency as a risk factor for nocturnal cardiovascular events, Dr. Bernard J. Gersh said.

It's well established that the peak hours of sudden cardiac death (SCD) in the general population are 6 a.m. until noon, and that the fewest such deaths happen from midnight to 6 a.m. However, this diurnal pattern is reversed in people with obstructive sleep apnea (OSA), Dr. Gersh, professor of medicine at the Mayo Clinic, Rochester, Minn., noted at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

OSA is associated with numerous pathophysiologic changes that provide potential mechanisms promoting arrhythmias and SCD during sleep.

He cited a study by his colleagues, Dr. Apoor S. Gami and coworkers at the clinic, who reviewed the death certificates and medical records of 112 Minnesotans who underwent polysomnography and later died suddenly from cardiac causes. SCD occurred between midnight and 6 a.m. in 46% of the 78 people with OSA, compared with 21% of those who didn't fulfill criteria for OSA. Persons with OSA had a 2.6-fold greater risk of SCD between midnight and 6 a.m. than in the other 18 hours of the day.

By comparison, a large meta-analysis of studies examining the morning excess of SCD in the general population showed that only 16% of SCDs occurred between midnight and 6 a.m. (Am. J. Cardiol. 1997;79:1512-6). And that 16% figure is surely an overestimate, since it included some individuals with undiagnosed OSA, Dr. Gersh noted at the conference, which was cosponsored by the American College of Cardiology.

In the Minnesota study, severity of OSA correlated directly with the relative risk of SCD occurring from midnight to 6 a.m. Individuals with an apnea-hypopnea index of 40 or more were 40% more likely to experience SCD between midnight and 6 a.m. than were those with mild to moderate OSA as reflected in an apnea-hypopnea index of 5-39 (N. Engl. J. Med. 2005;352:1206-14).

Dr. Gersh observed that OSA is associated with numerous pathophysiologic changes that provide potential mechanisms promoting arrhythmias and SCD during sleep. These include nocturnal hypoxemia, hypercapnia, a tremendous increase in sympathetic nerve activity, hypertensive surges, endothelial dysfunction, vascular oxidative stress, inflammation, hypercoagulability, and markedly elevated left ventricular wall stress.

In contrast, normal individuals experience decreased sympathetic activity during sleep. Their risk not only of SCD but also of onset of acute MI is at a nadir during the 6-hour period beginning at midnight. The peak in the incidence of these events from 6 a.m. until noon is believed to be related to increased coagulability and sympathetic drive.

Dr. Gersh noted that in a separate study by Dr. Gami and coworkers—presented last fall at the American Heart Association annual meeting—they reported that onset of acute MI in individuals with OSA followed the same pattern of increased incidence during the hours of sleep as did SCD. Onset of MI occurred between midnight and 6 a.m. in 32% of individuals known to have OSA and just 5% of those without OSA. ■

Too Much, Too Little Sleep Doubles Mortality

COLORADO SPRINGS — Change in sleep duration during midlife is associated in a U-shaped fashion with risk for death more than a decade later, Dr. Francesco Cappuccio reported at a conference of the American Heart Association.

The major driver of increased mortality among individuals at the low end of the sleep duration continuum is an excess of cardiovascular deaths, while in long sleepers the increase in mortality is due to noncardiovascular causes, according to the results of the Whitehall II study, said Dr. Cappuccio of Warwick Medical School, Coventry, England.

Whitehall II is a prospective cohort study of 10,308 white-collar British civil servants who were 35-55 years old when enrolled in the study in 1985-1988.

The Whitehall II analysis of the impact of changes in sleep duration included data on baseline sleep patterns in 7,729 participants and changes in those patterns over the next 5 years. Participants then were followed for mortality through 2004.

Cardiovascular mortality was 2.4-fold higher among subjects who slept an average of 6-8 hr/night at baseline but cut their sleep duration to 5 hr/night or less over the next 5 years' follow-up, compared with those who held fast to the 6- to 8-hour pattern. The findings held after adjustment for potential confounding factors.

In subjects who increased their sleep duration from 7 to 8 hr/night at baseline to 9 or more, there was an adjusted 2.1-fold increase in noncardiovascular mortality.

Short sleep duration is known to be associated with hypertension, weight gain, and diabetes, all of which increase cardiovascular risk. The relationship between long sleep and increased mortality is unclear. Hypotheses include possible links with depression and cancer-related fatigue, Dr. Cappuccio added.

—Bruce Jancin