Capitol Sleep Medicine Newsletter

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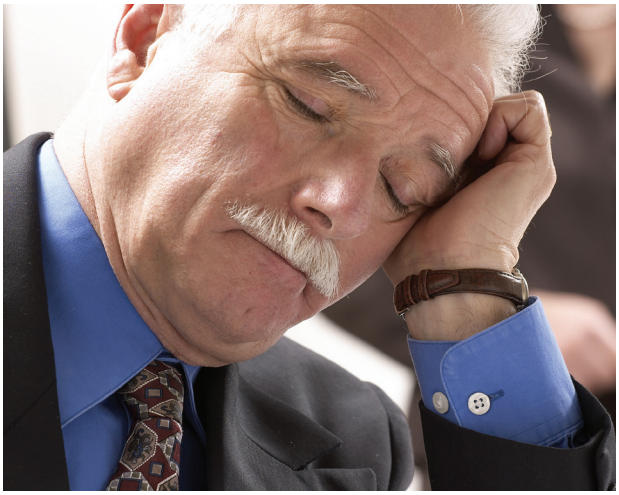
The Sleeping Heart



Obstructive sleep apnea (OSA) is a common medical condition that has been documented to occur in approximately 5% to 15% of the population. However, more recent estimates from the 2005 National Sleep Foundation Sleep in America poll indicate that as many as one in four American adults could benefit from evaluation for OSA.[1]

The clinical spectrum of obstructive sleep apnea (OSA) related cardiovascular disease (CVD) comprises systemic arterial hypertension (prevalence: 40-60%), pulmonary hypertension (20-30%), coronary artery disease (20-30%), congestive heart failure (5-10%), and stroke (5-10%). Epidemiologic data have clearly shown that cardiovascular risk is increased in OSA independent of confounding factors such as obesity and concomitant metabolic disease. In recent years, the pathophysiology of OSA related CVD has been further elucidated showing that apart from the well-known sympathetic activation, increased oxidative stress and pro-inflammatory changes seem to play major roles. Furthermore, studies using high resolution ultrasonography have demonstrated endothelial dysfunction and enhanced atherosclerosis in these patients. Therapy of OSA by continuous positive airway pressure (CPAP) ventilation exerts cardioprotective effects. It has been shown to restore endothelium-dependent vasodilation, lower 24-h blood pressure, eliminate nocturnal heart rhythm disorders, and improve left ventricular function. Furthermore, long-term CPAP therapy leads to a reduction in important clinical endpoints such as the rates of myocardial infarction and stroke.[2]

Patients with congestive heart failure (CHF) have an increased risk of sleep disordered breathing. In a recent study of 56 elderly patients with CHF, 67.9% had sleep disordered breathing. Of these, 53.6% had OSA, 7.1% had central sleep apnea and 39.2% had mixed sleep apnea. The apnea-hyponea index was closely related to left ventricular end diastolic function.[3]

In a very recent study of patients with severe OSA, the use of CPAP for a daily average of just 3 hours was sufficient to decrease the diastolic BP of hypertensive OSA patients.[4] In patients with OSA, treatment with CPAP improves baseline endothelial NO release and stimulates endothelium dependent vasorelaxation in the systemic circulation. This is a potential mechanism for improving systemic and vascular function in patients with OSA treated with CPAP.[5]

Pulmonary hypertension is correlated with OSA. In one study, 20.7% of OSA patients without any other lung or heart disease developed at least mild pulmonary hypertension which was partially or completely reversed after 6-month CPAP treatment.[6]

Patients with OSA have an increased risk of arrhythmias. Patients with atrial fibrillation (AF) and severe left ventricular impairment have an increased likelihood of sleep disordered breathing (particularly central sleep apnea).[7] Of considerable clinical importance is that patients with AF as well as untreated OSA have a higher recurrence of AF after cardioversion than patients without OSA. Appropriate treatment with CPAP in OSA patients is associated with lower recurrence of AF.[8]

A recent study in patients with reduced left ventricular ejection fraction and life-threatening ventricular tachyarrhythmias treated with an implantable cardioverter-defibrillator had a significantly higher occurrence of ventricular arrhythmias in association with disordered breathing.[9]

Clinicians should be aware that OSA may be a risk factor for the development of cardiovascular disease.

Happy Halloween!

[1] Chest. 2006 Sep;130(3):780-6
[2] Med Klin (Munich). 2006 Apr 15;101(4):321-7.
[3] Nan Fang Yi Ke Da Xue Xue Bao. 2006 Jun;26(6):847-8.
[4] J Hypertens. 2006 Oct;24(10):2091-9.
[5] Thorax. 2006 Jun;61(6):491-5. Epub 2006 Mar 14.
[6] Respiration. 2001;68(6):566-72.
[7] Chest. 2005 Oct;128(4):2116-22.
[8] Circulation. 2003 May 27;107(20):2589-94. Epub 2003 May 12.
[9] Chest. 2002 Aug;122(2):558-61.