Influence of Obstructive Sleep Apnea on Endothelial Function in Obese Patients

To the Editor:

We read with great interest the article by Ades et al.1 in a recent issue of CHEST (December 2011). In their article, Ades et al. demonstrated that weight loss is associated with improvement in endothelial-dependent flow-mediated dilatation (FMD) in overweight and obese patients with coronary heart disease. Moreover, the study pointed out that greater FMD is related to greater weight reduction in a dose-dependent way. The authors remarked on the causative relationship between several conditions and the impairment of FMD.

In this regard, we would like to highlight the important role of obstructive sleep apnea (OSA) in obese patients. Indeed, OSA can directly worsen endothelial function (EF), as reported by Kraicz and colleagues.2 Later, in 2005, Altin et al.3 showed that OSA is responsible for increasing the intima-media thickness. More recently, Chung and colleagues4 demonstrated that the degree of nocturnal hypoxemia in OSA is positively correlated with both arterial stiffness and endothelial impairment. The primary mechanism underlying endothelial abnormalities in OSA is represented by repetitive episodes of hypoxia/reoxygenation associated with transient cessation of breathing during sleep. The hypoxia/reoxygenation cycle promotes the generation of reactive oxygen species and inflammation and leads to a reduction in nitric oxide availability. In addition, sleep fragmentation/dep- rivation and genetic susceptibility for vascular manifestations of OSA contribute to worsening EF.5 On the other hand, our group has recently evaluated the presence of changes in FMD among obese patients with OSA after treatment with CPAP. We showed significant improvement of FMD in patients treated with CPAP for >3 months vs patients not treated. It is important to underline that the two study groups had similar BMI. Besides, a noteworthy finding is that the FMD values in patients treated for >3 months are similar to those in healthy subjects without cardiovascular risk factors.6 In another article, published December 2011 in CHEST, Akinnusi and colleagues7 also demonstrated an improvement of EF in obese patients with OSA after CPAP. They found a reduced expression of lectin-like oxidized low-density lipoprotein receptor 1 together with apoptotic circulating endothelial cells in patients with OSA after 8 weeks of CPAP therapy. In this study, patients' BMI at baseline was the same as post-CPAP. Taken together, these results suggest that, on one hand, weight loss can reduce the endothelial damage in obese patients; on the other hand, treatment with CPAP can improve EF among obese patients with OSA, regardless of weight reduction.

In conclusion, we would like to stimulate discussion about the role of weight loss in the improvement of OSA, a polysomnographic examination performed both before and after weight loss may be helpful, to know the relative contribution of OSA in the improvement of EF.

REFERENCES