Vascular Dysfunction in Sleep Apnea
Not Just a Peripheral Concern

Tomas Konecny, Virend K. Somers

The findings of impaired perfusion of myocardial tissue in OSA patients are in agreement with corroborating evidence from previous smaller studies, which used different imaging modalities. Nguyen et al reported impaired myocardial perfusion reserve in 25 consecutive OSA patients as detected...
by cardiovascular MRI, and Orea-Tejeda et al.\textsuperscript{10} reported perfusion defects on nocturnal single photon emission computer tomography imaging in 14 patients with severe obesity and OSA. Impaired myocardial perfusion or perfusion demand mismatch in OSA provides additional insight into why these patients may be at increased risk of myocardial ischemia and sudden cardiac death, particularly at night, when the hypoxicemic and other stressors are most evident.\textsuperscript{11} The strengths of the study by Butt et al.\textsuperscript{7} include an extensive evaluation of the vascular system with multiple modalities, complete follow-up of patients placed on CPAP treatment, and an innovative design that includes the addition of effectively 2 control groups, healthy subjects without OSA and patients without OSA but with hypertension. It is impressive that sleep studies were used to exclude occult OSA in the hypertensive and control subjects, and all 3 of the groups were free of other comorbidities. Comparison of the OSA patients with non-OSA hypertensive controls enables some perspective on the biological significance of the endothelial dysfunction noted, using the well-documented hypertension-induced vascular dysfunction as a frame of reference. There are also several limitations. As is often the case in studies of OSA, the attempt to separate major comorbidities that could function as confounders (obesity and hypertension) is not completely successful, and some overlap between the study groups occurs, as acknowledged by the authors. In particular, one must note the presence of a significantly elevated systolic blood pressure in the OSA group (supposed to not have any hypertension) and the significant difference in waist circumference between the study groups (although the body mass index was not statistically different). This latter reservation is of particular concern, because central obesity (significantly more marked in the OSA patients) is far superior to body mass index in predicting cardiovascular risk, especially in patients with coronary artery disease.\textsuperscript{12} In mitigation, CPAP improved endothelial function, but we do not know if there was any attenuation in central obesity, as would be expected based on work from Chin et al.,\textsuperscript{13} who showed that CPAP treatment reduced central obesity, even in the absence of significant change in body mass index. Furthermore, the findings regarding CPAP therapy would be more compelling had they been obtained as part of a randomized, controlled study in the OSA patients. These reservations notwithstanding, Butt et al.\textsuperscript{7} help further our understanding of impaired vascular function in OSA patients. Of special interest is their finding of a seemingly diffuse multivessel endothelial dysfunction, affecting both conduit vessels and microvasculature, and involving blood vessels in the limb, skin, and heart. These observations speak to the larger concept of OSA-induced endothelial dysfunction as a systemic disease, with improvement of endothelial function in all of these territories after CPAP treatment. However, whether such improvement translates into a meaningful reduction of adverse clinical outcomes in OSA patients treated with CPAP can ultimately only be established in large randomized clinical trials.

**Sources of Funding**

This publication was supported by NIH/NCCR CTSA Grant Number UL1 RR024150, NIH Grant Number HL 65176, and Grants from the Ministry of Education and Health of the Czech Republic (NT 114015/2011, CZ.1.05/1.1.00/02.0123). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH.

**Disclosures**

V.K.S. received a gift to Mayo Foundation from Phillips Respironics Foundation, is a consultant for Merck, Johnson and Johnson, Resplicardia, ResMed, Sova Pharmaceuticals, and Apexn Medical, and is working with Mayo Health Solutions and their industry partners on intellectual property related to sleep and cardiovascular disease. T.K. has no disclosures.

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Hypertension. 2011;58:352-353; originally published online July 11, 2011;
doi: 10.1161/HYPERTENSIONAHA.111.175976

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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World Wide Web at:
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