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Endothelial function in obstructive sleep apnea and response to treatment.

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Impaired endothelium-dependent vascular relaxation is a prognostic marker of atherosclerosis and cardiovascular disease. We evaluated endothelium-dependent flow-mediated dilation (FMD) and endothelium-independent nitroglycerin (NTG)-induced dilation of the brachial artery with Doppler ultrasound in 28 men with obstructive sleep apnea (OSA) and 12 men without OSA. Subjects with OSA (apnea-hypopnea index; mean \pm SD, 46.0 \pm 14.5) had lower FMD compared with subjects without OSA (5.3 \pm 1.7% vs. 8.3 \pm 1.0%, $p < 0.001$), and major determinants of FMD were the apnea-hypopnea index and age. There was no significant difference in NTG-induced dilation. Subjects with OSA were randomized to nasal continuous positive airway pressure (nCPAP) or observation for 4 weeks. Subjects on nCPAP had significant increase in FMD, whereas those on observation had no change (4.4% vs. -0.8%, difference of 5.2%, $p < 0.001$). Neither group showed significant change in NTG-induced vasodilation. Eight subjects who used nCPAP for over 3 months were reassessed on withdrawing treatment for 1 week. On nCPAP withdrawal, FMD became lower than during treatment ($p = 0.02$) and were similar to baseline values. Our findings demonstrated that men with moderate/severe OSA have endothelial dysfunction and treatment with nCPAP could reverse the dysfunction; the effect, however, was dependent on ongoing use.

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