

Vitamin C injected intravenously acts as a powerful scavenger of free oxygen radicals. Stress produced by free radicals has many negative effects on well being, including disruption of the vascular system. A recent report from Germany shows that an intravenous injection of 500 mg. of vitamin C in patients with obstructive sleep apnea, a breathing disorder during sleep, normalizes dysfunctional arterial dilation in comparison to a healthy control group. The abstract from that article is included in this volume to illustrate one of the productive directions current research on injectable vitamin C is moving.—*R.D.M.*

## Antioxidant Vitamin C Improves Endothelial Function in Obstructive Sleep Apnea

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**Rationale:** Obstructive sleep apnea (OSA) is associated with oxidative stress, endothelial dysfunction, and increased cardiovascular morbidity and mortality.

**Objective:** We tested the hypothesis that endothelial dysfunction in patients with OSA is linked to oxidative stress.

**Methods:** In the present study, we measured flow-mediated dilation (FMD) of the brachial artery by ultrasound in 10 otherwise healthy, untreated patients with OSA and 10 age- and sex-matched control subjects without sleep-disordered breathing before and after intravenous injection of the antioxidant vitamin C. The investigator performing the FMD measurements was blinded to the status of the patients.

**Results:** When compared with control subjects, baseline FMD was significantly reduced in the patients with OSA. After intravenous injection of 0.5 g vitamin C, vasoreactivity remained unchanged in the control subjects. In the patients with OSA, ascorbate led to an increase in FMD to a level comparable to that observed in the control group.

**Conclusion:** The reduced endothelial-dependent vasodilation in untreated patients with OSA acutely improves by the free radical scavenger vitamin C. These results are in favor of oxidative stress being responsible for the endothelial dysfunction in OSA. Antioxidant strategies should be explored for the treatment of OSA-related cardiovascular disease.

