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Near-Infrared Spectroscopy for the Assessment of Vascular Responsiveness of the Brain: A Screening Method for Cerebrovascular Morbidity in Obstructive Sleep Apnea

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Introduction: Obstructive Sleep Apnea Syndrome (OSAS) is an important risk factor for the development of cardiovascular morbidity and mortality. Compromised cerebral blood flow (CBF) has been postulated to account for the cerebrovascular morbidity associated with OSAS. Near-infrared Spectroscopy (NIRS), a non-invasive, portable, costeffective methodology that allows transcranial and continuous real-time measurements of brain tissue oxygenation and hemodynamics is the only modality that provides information on brain oxygenation. Hence, NIRS is an important tool for the assessment of cerebrovascular health. Methods: A. Subjects: Twenty subjects (25-74yrs) participated in the study. Eight (29-74yrs) had OSAS (AHI>20), while twelve healthy nonsnorers (25-54yrs) constituted the control group.B. Instrumentation and Monitoring: Arterial blood oxygen saturation (SaO2) and heart rate (HR) were monitored via pulse oximetry (N-200, Nellcor Inc., Pleasanton, CA), and the breathing rate with a respiratory strain gauge (Resp-EZ, Sleepmate, New-Life Technologies, Midlothian, VA). The NIRS parameters, such as oxy-([O2Hb]), deoxy-([HHb]), total hemoglobin ([tHb]) concentrations, and tissue hemoglobin oxygen saturation (SO2) were monitored by a frequency-domain tissue oximeter (OxiplexTS, ISS Inc., Champaign, IL).C. Experimental protocol: All subjects were instructed to perform breath-holding exercises while vascular reactivity and cerebral hemodynamic responses to hypoxia and consequent hypercapnia (carbon dioxide is a vasodilator in the brain) was determined. The protocol consisted of: 5-7 minutes normal breathing; 10-30/seconds of breath holding (at FRC); Resumption of normal breathing and repetition of breath holding (3-5 times).

Results: Appropriate analysis schemes have been applied to differentiate cerebrovascular reactivity between the two groups and characterize the subjects. In healthy subjects, the brain responds to hypoxia and hypercapnia by vasodilation and opening of the capillary bed. Normals in our study showed a prompt decrease in [HHb] and an increase in [O2Hb] and [tHb]. These changes correlated with an increase in CBF to meet the oxygen demands of the brain. The decrease in [HHb] represents the wash-out effect from the increased CBF. In subjects with OSAS, the hemodynamic response to hypoxia was significantly delayed, reduced or even absent (p<0.040).

Conclusions: Using simple breath holding exercises and a novel methodology, we showed that cerebrovascular reactivity to hypoxia and hypercapnia is compromised in OSAS sufferers. Our results suggest that

compromised brain microvasculature in OSAS may lead to structural and functional impairment and diminished or loss of ability to vasodilate. This finding may explain the increased cerebrovascular morbidity seen in OSAS. NIRS provides non-invasive, transcranial, real-time measurements of cerebral oxygenation and hemodynamics. It gives direct information on cerebrovascular health by monitoring the response of the brain to hypoxic insults. NIRS can provide a cost-effective screening method for individuals at risk for the cerebrovascular consequences of OSAS.

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