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OBSTRUCTIVE SLEEP APNEA: EVALUATION OF BRAIN OXYGENATION AND HEMODYNAMICS BY NEAR-INFRARED SPECTROSCOPY

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Introduction: Obstructive sleep apnea (OSA) is a potentially lethal disease, which leads to hypoxia and hypoxemia. Chronic, recurrent hypoxia during sleep may cause brain injury. Neuropsychological and cognitive deficits, as well as cerebrovascular accidents, including fatal strokes are not uncommon (1). Conventional polysomnography does not provide information on brain oxygenation, an important parameter in subjects with preexisting cardiovascular pathology. Nearinfrared spectroscopy (NIRS), a safe, non-invasive, portable, bedside method, provides real-time transcranial measurements of changes in cerebral oxygenation and hemodynamics. These characteristics make NIRS the ideal tool to study physiological and pathological processes of the brain, in research and clinical settings (2).

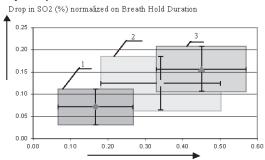
Methods: Thirteen males and eight females (age 22-74 years) participated in the study. Eight were OSA sufferers. Thirteen individuals constituted the control group. One control subject had family history OSA. All OSA subjects and six controls were snorers. Two OSA subjects had severe hypertension and asthma. Arterial blood oxygen saturation (SaO2) and heart rate (HR) were monitored via pulse oximetry, and the breathing rate with a respiratory strain gauge. The NIRS parameters, such as oxy- (O2Hb), deoxy- (HHb), and total hemoglobin (tHb) concentrations, as well as tissue hemoglobin oxygen saturation (SO2) were monitored by a frequency-domain tissue oximeter (OxiplexTS, ISS Inc., Champaign, IL).

Figure 1

Characterization of measured populations by normalized drops in SaO2 and SO2

1-Controls during breath holding

2, 3 -OSA subjects during breath holding and during sleep apnea, respectively



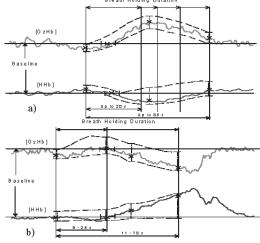
Drop in SaO2 (%) normalized on Breath Hold Duration

Results: We applied NIRS during voluntary breath holding and during daytime napping. Observed changes in SaO2 and SO2 were significantly smaller (p≤0.025) for controls during breath holding than those in SaO2 and SO2 recorded in the OSA group during breath holding and spontaneous sleep apnea events (Fig. 1). Higher levels of arterial and brain deoxygenation were observed for OSA subjects during napping. Different dynamics of O2Hb, HHb, and tHb concentrations and SO2 were detected for control and OSA groups during voluntary hypoxia. We observed that the response to simple breath holding exercises, registered by NIRS, is sufficient to discriminate OSA subjects from healthy controls. The amplitude of the hemodynamic response to hypoxia was significantly larger than the amplitude of the baseline fluctuations in both control and OSA groups (Fig. 2). This response was altered in OSA subjects with a cardiovascular medical history (Fig. 2b).

Figure 2.

Typical time course of oxy- and deoxy-hemoglobin concentrations in brain tissue: a) in eight control subjects, b) in eight OSA subjects

Dashed lines characterize the range of changes in [O2Hb] and [HHb]



Conclusions: From these measurements we can assume that, in healthy individuals with obstructive sleep apnea, there is a protective cerebrovascular response to hypoxia which is likely to prevent eventual brain injury during apnea. In subjects with preexistent cardiovascular pathology, this protective mechanism may be defective. An adequate response may not fulfill the oxygen demands of the brain. Thus, the recurrent hypoxic insult, during sleep, may contribute to the risk for cerebrovascular morbidity. NIRS may be a valuable tool for early detection of cerebral hemodynamic abnormalities in obstructive sleep apnea. Quantitative measurements of oxygenation by NIRS may complement polysomnography for the prevention of hypoxic damage.

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