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Assessment of cerebral oxygenation and hemodynamics in obstructive sleep apnea syndrome.

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Abstract: We applied Near-Infrared Spectroscopy for the investigation of cerebral oxygenation during obstructive sleep apnea. We found a relatively large decrease in brain oxygenation during apnea and the peak of deoxygenation occurs after resumption of breathing.

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(170.1610) Clinical applications.

1. Introduction

Near-Infrared Spectroscopy (NIRS), a non-invasive diagnostic tool, offers ideal features for the assessment of oxygenation in tissues such as brain [1]. We exploit this non-invasive technique to evaluate the effects of intermittent hypoxia and hypoxemia on the cerebral oxygenation, during sleep apnea. Near-infrared light penetrates several centimeters into tissues, passing through bony structures. NIRS enables continuous and real time measurements of changes in the hemoglobin oxygenation state and blood volume thus providing information on local tissue oxygenation and hemodynamics. [1, 2, 3, 4].

Obstructive sleep apnea syndrome (OSAS) has increasingly been recognized as a cause of ill health in the community. In the middle-aged work force 2% of women and 4% of men meet the minimal diagnostic criteria for the sleep apnea syndrome (apnea/hypopnea score of 5 or higher and daytime hypersomnolence) [5]. OSAS is described as a potentially lethal disease because it leads to hypoxia and hypoxemia. Since the brain is very sensitive to hypoxia, recurrent decrease of the arterial oxygen saturation in sleep apnea may induce brain injury.

Altered quality of life, daytime sleepiness, neuropsychological dysfunction and cognitive deficits have been associated with OSAS as well as cardiovascular disease, including systemic and pulmonary hypertension, arrhythmias, ischemic heart disease. Cerebrovascular accidents, ranging from transient ischemic attacks to fatal strokes are closely associated to sleep apnea [6,7].

Conventional polysomnography [8] detects sleep apnea in correlation to the various sleep stages and determines arterial oxygen saturation, but does not provide information on brain oxygenation, especially in subjects with preexisting anatomical or functional vascular pathology.

We have developed instrumentation and experimental protocols to determine cerebral hemodynamics during sleep apnea. The instrumentation is based on two sensors of multiple light sources and an optical detector per sensor, to allow differential measurements and the collection of optical data from both cerebral hemispheres.

2. Method

To obtain the absolute brain tissue oxygenation we used the frequency-domain instrumentation we developed (Model 96208, ISS Inc, Champaign, IL). We used a pulse oximeter (N-200, Nellcor Incorporated, Pleasanton, CA), attached to the forefinger, to monitor arterial hemoglobin oxygen saturation and heart rate. We also used a respiratory strain gauge (Resp-EZ, Sleepmate, New Life Technologies, Midlothian, VA) to monitor breathing. We investigated cerebral tissue oxygenation under two hypoxic conditions. First during brief apneic episodes in the course of diurnal napping of an individual with daytime sleepiness and history of sleep apnea, and second during voluntary breath holding of the same subject.

One of the principal problems in NIRS is the construction of a suitable sensor and its attachment to the head. During the pilot studies the subject did not enter deep sleep phases, because the sensor was inflicting discomfort and pain and it was taken off after 30 minutes. An actual problem of sleep studies is that the subjects cannot be instructed to remain motionless and in particular not to move the head during sleep. Thus, our goal is to construct a sensor, which is comfortable, it does not block the blood circulation of the skin, and still it is firmly attached to head.

3. Results and Discussion

Figure 1 summarizes our preliminary findings during sleep apnea. The apneic episodes, in this trial, have duration of approximately 20 seconds (gray area). The arterial oxygen saturation (SaO_2) starts declining at the end of apnea. The drop is of the order of 10%. At the last third of apnea the heart rate increases and the brain tissue oxygenation (SO_2) decreases by 4% concomitantly (Fig. 1a). Episodic changes in the cerebral hemoglobin variables are observed with each apneic event (Fig. 1b). A gradual increase in total (tHb) and deoxy (HHb) hemoglobin concentration is observed at the end of the apneic period. Surprisingly the peak of desaturation shows several seconds after the resumption of breathing and returns at the base value during the first third of the subsequent apnea. Oxy-hemoglobin (O_2Hb) does not seem to present any appreciable changes. This can be explained by the fact that an increase in the cerebral blood flow (CBF) occurs during OSAS. This accounts for the increased cerebral blood volume (CBV) indicated by the increase in tHb concentration. The increase in CBF compensates for reduced SaO_2 and consequently oxygen supply to the brain tissue does not decrease (stable O_2Hb). The result is a smaller decrease in SO_2 as compared to the greater drop in SaO_2 . The delay between the beginning of the decline of these two parameters (last third *versus* the end of apnea period) depends upon the site of measurement (blood travels further to reach the finger than the brain).

Fig.1. Apnea (gray area) during diurnal napping.

Fig.2. Voluntary breath holding (gray area).

From these measurements we can assume that, in this otherwise healthy individual, there is a protective cerebrovascular response to hypoxia, which is likely to prevent an eventual brain injury in sleep apnea. However, in

subjects with already present cardiovascular pathology, namely systemic hypertension, arteriosclerosis, or carotid artery thrombosis, this protective mechanism may be altered. An increase in the CBF may not be adequate to meet with the oxygen demands of the brain. Thus, the recurrent hypoxic insult, during sleep apnea, may contribute to the risk for cerebrovascular morbidity.

Figure 2 reports the findings during voluntary breath holding of the same subject. The apneic episodes last approximately 35 seconds (gray area). The SaO_2 decreases by 20% and SO_2 by 6%. Both parameters show the onset at the second half of the episode. The heart rate shows an increase at the onset of apnea first, and at the resumption of breathing (Fig. 2a). All three hemoglobin variables (Fig. 2b) show episodic changes. Both tHb and HHb increase and their peaks correspond to the resumption of breathing. O_2Hb shows a negligible decrease. It seems that the expected increase of CBF during provoked hypoxia could compensate for reduced SaO_2 .

In order to understand the cerebral hemodynamics and oxygenation in sleep apnea we need to extend our research during both diurnal napping and nocturnal sleep studies associated with polysomnography. The appropriate sensors will enable us to perform differential measurements and investigate the cerebral blood variables during sleep and during induced hypoxia, in subjects diagnosed with OSAS and normal individuals, for the detection of eventual abnormalities.

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