Cerebral Hemodynamics in Obstructive Sleep Apnea*

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We have investigated cerebral blood flow velocity (CBVF) in 14 patients with marked obstructive sleep apnea syndrome using transcranial Doppler ultrasonography during sleep. The CBVF increased during apnea, with a mean acceleration of 0.9 cm/s², followed by a rapid decrease during snoring. The same effect was observed by voluntary apnea in healthy subjects, showing a smaller acceleration rate (0.6 cm/s²). These results provide evidence for a normal CO₂ regulation of cerebral vessels during sleep apnea and do not support the notion of cerebral hypoperfusion during sleep being a risk factor for stroke.

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Obstructive sleep apnea syndrome (OSAS) is characterized by everyday sleepiness and a breathing disorder during sleep, with repetitive apneic phases as well as snoring, caused by intermittent upper airway obstructions. Several complications, such as pulmonary and arterial hypertension or cardiovascular disease, have been shown to be related to OSAS. Partinen and Guilleminault demonstrated a higher vascular morbidity and mortality in untreated patients with OSAS. Recently, Palmaeki reported that snoring is a risk factor for sleep-related brain infarction in male patients. In order to study cerebral hemodynamics during sleep, we have investigated cerebral blood flow velocity (CBVF) during apneic episodes in patients with OSAS by means of transcranial Doppler ultrasonography (TCD).

**Materials and Methods**

**Patients**

We examined 14 patients (13 male patients and 1 female patient; mean age, 50±12 years [SD]) who suffered from heavy snoring during sleep and excessive daytime sleepiness. Their medical histories provided no evidence for cerebral stroke or cardiovascular diseases. The findings from neurologic examination, cerebral computed tomographic scan, and Doppler ultrasound investigation of the extracranial arteries were normal in all cases.

**Sleep Studies**

Standard polysomnography for at least two nights monitored the EEG, electro-oculograms, electromyogram, and nasal and oral airflow. Motions of the thorax and abdomen induced by respiration were registered by strain gauges (ZAK). Respiratory frequency and the phase relation between abdominal and thoracic motion were further analyzed according to the method described by Staatsa et al., using a fast Fourier transformation of the respiratory signals. Oxygen saturation and pulse frequency were measured by finger pulse oximetry (Radiometer). Body position and movements were detected by infrared video monitoring.

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**Monitoring CBVF**

A microprocessor-controlled directional pulsed-wave Doppler device (TC 2000S, EME; Überlingen, Germany) operating at 2 MHz was used for ultrasonography of the middle cerebral artery (MCA). The ultrasonic probe was fixed with head tape and gave stable recordings for several hours during sleep.

**RESULTS**

Polysomnography for at least 6 h per night in all patients revealed more than 50 typical repetitive apneic episodes during recording, with cessation of nasal and oral airflow for more than 10 s during non-rapid eye movement sleep. The average apnea-hypopnea index (AHI) was 56/h ± 20/h (n = 14), with a mean total duration of apnea of 36 ± 13 min/h (n = 14). The averaged oxygen saturation of 94 ± 1 percent (n = 14) decreased to 75 ± 15 percent (n = 14) during apnea. Minimum arterial oxygen saturation was 45 percent in one patient, and the average of the maximum desaturation in all patients was 61 ± 14 percent (n = 14). Phase relation between chest wall and abdominal motion more than 45° delay indicated airway obstruction and could shift to 180° during apneic episodes corresponding to paradoxical breathing. After patients had fallen asleep, the average chest wall motion frequency during apnea and snoring was 20/ min ± 3 min (n = 14), in contrast to an average of 16 ± 3 breaths per minute (n = 6) in subjects without OSAS. While patients were awake, the mean CBVF of the MCA was in the range from 40 to 65 cm/s in a depth of 50 mm from the temporal bone. After the patients fell asleep (stage 1 and 2) and obstructive apnea appeared, the mean CBVF increased nearly constantly during the obstructive episodes, with a mean acceleration of 0.9 ± 0.2 cm/s² (Fig 1). The average peak of mean CBVF from all patients during apnea increased to 142 percent compared with the mean CBVF during sleep onset. During apneic episodes exceeding 120 s, CBVF rose to as much as 210 percent of the awake value. Systolic and diastolic CBVF had similar time courses during apnea. After the ventilation effort reached threshold for snoring, CBVF rapidly fell to
measured by means of TCD to the P\textsubscript{CO\textsubscript{2}}. In a previous report, we have shown that the etiology of this CBFV variation in OSAS is based mainly on physiologic vasomotor CO\textsubscript{2} regulation of the cerebral vessels.\textsuperscript{12} The apnea simulated in healthy subjects displayed a similar increase of CBFV with a smaller acceleration. This minor difference may arise from a higher sensitivity of cerebral vessels to CO\textsubscript{2} during sleep in patients with OSAS. The apneic episodes are compensated for in respiration by an increase of ventilation frequency, leading to a faster blood gas exchange during snoring just as in hyperventilation. This could also explain the reduced CBFV after snoring if a slight concomitant hypocapnia is assumed. In conclusion, our results do not support the notion of a higher risk for ischemic brain infarction on hemodynamic grounds in patients with OSAS during sleep. This is in accordance with a report of Gonzalez-Rothi and coworkers,\textsuperscript{13} who found no increased risk of death in patients with OSAS during sleep; however, the vascular stress during the night could be responsible for cerebrovascular alterations, leading to a higher risk of infarction which is difficult to distinguish from the effects of the often accompanying hypertension.

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REFERENCES

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