COMMENTARY

Impaired Nocturnal Cerebral Hemodynamics during Long Obstructive Apneas: The Key to Understanding Stroke in OSAS Patients?

Commentary on Pizza et al. Nocturnal cerebral hemodynamics in snorers and in patients with obstructive sleep apnea: a near-infrared spectroscopy study. SLEEP 2010;33:205-210.

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TWENTY-FIVE YEARS AGO, PARTINEN AND PALOMAKI (1985) PUBLISHED A SHORT NOTE IN THE LANCET REPORTING THAT THERE WAS AN UP TO 40 TIMES higher risk for stroke in snoring inhabitants of the country of Iceland relative to non-snoring Icelanders.[1] It inspired not only a greater number of research groups to investigate linkages between stroke and sleep apnea, but also had importance for the future of sleep medicine. To this day, the increased risk of stroke is one of the reasons—besides excessive day time sleepiness and increased accident risk—that public and private health insurance companies are willing to bear the cost for sleep apnea diagnostic procedures and treatment, because the financial consequences of nontreatment versus treatment would be higher than for many other medical morbidities caused by sleep disordered breathing (SDB). The risk of stroke is also likely to be one of the primary reasons that many elderly patients with sleep apnea agree to accept CPAP and oral appliance treatments.

Therefore ongoing research on the connection between stroke and SDB is of crucial importance for the awareness of SDB in public health policy and in the general population.

The direct link between SDB and high arterial blood pressure (BP), established through the Sleep Heart and Health Study,[2] suggests a straightforward cause for the increased risk of stroke in SDB. That is, high BP increases the risk for an apoplectic insult or in the long run arteriosclerosis and ischemic infarction. However, there may be additional contributors to the effects of SDB on stroke risk, including the effects of the former on intrathoracic pressure changes and cerebral hemodynamics.

In the 1990s, several groups started to measure cerebral blood flow via transcranial Doppler to look if cerebral hemodynamics were altered in sleep apnea patients; some concentrated on the average blood flow during day and night, and some on the blood flow during the actual apneic event, with the challenge of making measurements during the patient's sleep.[3-6] These studies established that cerebral blood flow was altered in sleep apnea and that the autoregulation of cerebral blood flow and cerebral arterial pressure is partly disabled, probably because the reaction time to the peripheral pressure and flow changes in the major arterial vessels is too long. However, these studies reached somewhat different conclusions because of their different approaches. While some suggested that blood flow was altered independent of apneic events,[5] thus leaving unanswered the question of whether thoracic pressure swings during SDB influenced blood flow in the major cerebral arteries, others reported a direct relation of cerebral blood flow to apneic events and the duration of these events.[6] Moreover, all of these earlier studies suffer from relying on relatively small numbers of homogenous subjects, and, in some cases, from the technical challenge of obtaining clean Doppler signals while patients were asleep. Since my laboratory contributed to this work, I would have to admit that the requirement to keep the Doppler probe in place in the medial cerebral artery while the patient slept could be at best characterized as "adventurous."

Optical measurements, such as near infrared spectroscopy (NIRS) and functional MRI have the advantage over transcranial Doppler that NIRS and MRI measurements are somewhat less disturbed when the subject moves, and they have shown the impact of altered hemodynamics in the brain during low brain oxygenation in sleep apnea patients.[7-9] Several studies with NIRS repeatedly reported an impairment of brain tissue oxygenation in sleep apnea patients. However these investigations lacked a diversified group of subjects and a focus on single events.[10,11] Fortunately, there is new evidence that fills some of these important gaps in our understanding of the relationship between obstructive events during sleep and cerebral hemodynamics.

This issue of SLEEP contains a report by Pizza and colleagues[12] on who used NIRS to investigate nocturnal cerebral hemodynamics in snorers and in patients with obstructive sleep apnea. The work has important advantages over former studies in that it evaluated cerebral hemodynamics and oxygenation of brain tissue in single and varied events in different patient groups. The authors report a significant impact on cerebral hemodynamics in severe sleep apnea patients and during obstructive apneas, but not in snorers and those with hypopneas. Thus they failed to confirm early concerns that every snorer was at higher risk for stroke.[1] However, they did confirm the earlier studies that suggested there was a relationship between reduced blood flow and tissue oxygenation relative to apnea duration.

The results of Pizza and colleagues[12] provide additional new information on the effects of sleep apneas on cerebral hemodynamics. Perhaps most importantly, they point to a reason to be concerned that patients with longer-duration apneas may be at greater risk for stroke. This is an important discovery that now

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needs to be confirmed prospectively and in an even larger number of patients. NIRS methodology may offer a way to obtain such data on a large number of sleeping patients essential for such a study.

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