Is Sleep-Hypoxia Really Unexpected in Acute Stroke?

To the Editor:

Roffe and colleagues provide an excellent study on a large patient population proving the high prevalence of hypoxia in acute, mostly ischemic stroke patients. They showed that “...many stroke patients who appear normoxic during the day may develop significant hypoxia at night early after the stroke” because stroke patients spent more time in hypoxia and had significantly higher oxygen saturation index (ODI). The authors call attention to the increased danger of hypoxia for developing definitive brain infarctions during brain ischemia, when compensating vasodilatation-capacity of the brain vessels is lost. On the basis of their results, oxygen monitoring of acute stroke patients, especially during sleep, seems to be clearly justified.

The authors state: “because we do not have data on oxygen saturation before the stroke, it is not possible to say whether the difference in oxygen saturation between stroke patients and control subjects is due to the stroke rather than the higher prevalence of smokers, cardiovascular disorders, and sleep apnea in the stroke group.”

Clearing the origin of sleep-hypoxia in acute stroke patients is a crucial question. In our opinion, one of the clues (somewhat neglected by the authors) possibly lighting this problem is hidden among the data of the study. It is the important parameter of the high ODI found in sleep records of stroke patients. Although the length of the desaturation events taken into account is unfortunately not defined in the paper, we presume that the generally used 11-60-second time interval for the minimum of 4% fall in saturation from the baseline just before the drop. The start of the dip is the time point at which the fall begins, and the end of the dip is when the saturation has recovered to the baseline just before the drop.

Frequent shallow oxygen desaturation events are nearly specific markers of sleep apneas, the parameter ODI is also commonly applied in home-screening of sleep apnea syndrome. Consequently it is a probable Nocturnal Hypoxia After Stroke

Response:

We thank Dr Szucs for her comments on our paper “Unexpected Nocturnal Hypoxia in Patients With Acute Stroke.” In response to the queries raised we would like to make the following clarifications.

Oxygen desaturation was defined as a >4% fall in saturation from the baseline just before the dip. The start of the dip is the time point at which the fall begins, and the end of the dip is when the saturation has come back up to baseline minus 1%. The definition of a desaturation we used in this paper does not contain maximum or minimum duration for the event. There is no generally accepted time frame for the definition of desaturations. While our study was performed overnight from 21:00 to 09:00, not all readings were done while the patients were asleep. The baseline oxygen saturation was taken at 21:00 while patients were still awake. Stroke patients had lower oxygen saturation than controls not only during sleep, but also when awake. While stroke patients also had a higher oxygen desaturation index than controls our data suggest that other factors in addition to sleep apnea are responsible for the difference in oxygen saturation between patients and controls. This is also supported by the results of the capillary blood gases taken during the day. The majority of patients had arterial oxygen tensions just below normal or in the low normal range with a mild respiratory alkalosis with high normal or elevated pH, a low normal carbon dioxide tension and normal bicarbonate levels. This pattern would be compatible with respiratory compensation of an oxygen transfer problem. It is likely that hypoxia after stroke has multiple causes.
Letter to the Editor

C. Roffe, MD
S. Sills, RGN
M. Halim, MSc
K. Wilde, PhD
P.W. Jones, PhD
M.R. Allen, MD
P. Crome, MD, PhD
City General Hospital
Stoke-on-Trent, UK


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Anna Szűcs, József Janszky, Zoltán Nagy and György Miglécki

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