

## Noninvasive Ventilation in Acute Ischemic Stroke

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### Editorial

The healthy human brain is able to regulate its blood flow according to any acute and chronic changes in various parameters. Cerebral autoregulation mechanisms protect the brain, and are dependent on myogenic, neuronal, endothelial and metabolic factors [1].

In patients with acute ischemic stroke with proximal arterial occlusions, cerebral autoregulation is often hampered. These patients are prone to developing cerebral steal phenomenon in which the unaffected vessel “steals” the blood away from the diseased vessel, and further aggravates the ischemia in the affected territory. This phenomenon was aptly coined as “Reversed Robin Hood Syndrome”, i.e. “rob the poor to feed the rich” [2]. Sleep-disordered breathing, in the form of obstructive, central or mixed sleep apneas, may occur in 50-70% of patients with acute ischemic stroke. Accordingly, sleep-wake disturbances, insomnia, disturbances of wakefulness, sleep-related movement disorders and parasomnias are found in 10-50% of these patients [3].

However, the commonest type of sleep-disordered breathing in acute ischemic stroke is obstructive sleep apnea. Central sleep apnea has also been described in patients with bilateral strokes with disturbed consciousness and even in unilateral strokes with preserved consciousness. The latter is observed especially due to involvement of the insula, cingulate cortex and thalamus [4]. These conditions may cause fluctuations in the oxygen and carbon dioxide concentrations in the blood. Carbon dioxide and oxygen are key stimulating vasodilators in cerebral autoregulation [1]. However, in patients with acute ischemic stroke and persisting proximal arterial steno-occlusive disorders, cerebral autoregulation may be impaired and sleep-related disorders will have detrimental consequences – by causing hypercapnia, cerebral steal phenomenon, and infarct expansion.

In order to combat this vicious cycle of hypoventilation-hypercapnia-intracranial steal phenomenon induced by various sleep-related breathing disorders, the use of noninvasive ventilation (NIV) has been proposed. NIV has been found to be safe and well-tolerated during the acute phase of ischemic stroke [5,6]. Jens et al even showed a trend toward improvement in National Institute of Health Stroke Scale score (NIHSS) when continuous positive airway pressure (CPAP) was instituted within 24-hours of acute ischemic stroke [5]. Similar trend toward greater neurological improvement with BiPAP was reported in a recent study by Tsvigouis et al. [6]

Two ongoing trials are currently exploring the benefits of noninvasive ventilation in acute ischemic stroke- the Sleep Apnea

Cardiovascular Endpoints (SAVE) Trial [7], and the Reversal of the Neurological Deficit in Acute Stroke with the Signal of Efficacy Trial of Auto-BPAP to Limit Damage from Suspected Sleep Apnea (Reverse-STEAL) [8]. Both trials are multi-center, prospective, randomized-controlled trials.

The SAVE Trial aims to recruit 5000 patients with moderate to severe OSA and established coronary or cerebrovascular disease, randomize them to CPAP and standard care for 12 months or standard care alone, to determine the effects of CPAP in addition to standard care in the prevention of coronary and cerebrovascular disease [7].

On the other hand, the Reverse-STEAL Trial aims to recruit 60 patients with suspected or confirmed acute ischemic stroke presenting within 24-hours of stroke onset with proximal arterial occlusions, and randomizing them to receive auto-BiPAP and standard care for 48 hours, or standard care alone. The investigators’ hypothesize that early auto-BiPAP positively affects short-term clinical outcomes in AIS patients [8].

With the high prevalence of sleep-disordered breathing in acute ischemic stroke patients, especially when associated with persisting proximal cerebral arterial occlusions, NIV appears to be a well-tolerated, safe and promising adjuvant therapy in the armamentarium of a stroke neurologist.

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