Normobaric Hyperoxia Treatment in Acute Ischemic Stroke

To the Editor:

We read with interest the article by Liu et al, published in the July issue of Stroke.1 The authors demonstrate that early normobaric hyperoxia (NBO) treatment increases the safety of delayed tPA thrombolysis in a rat model of focal cerebral ischemia and can extend its therapeutic time window. They suggest that NBO may inhibit tPA-augmented matrix metalloproteinase (MMP)-9 induction but not ischemic-triggered MMP-9 induction in saline-treated rats. However, the suggestion that the interfering of tPA–MMP-9 is an important underlying mechanism for neurovascular protection of NBO in cerebral ischemia is still debatable. Previous studies show that NBO alone maintains penumbral oxygenation in a rat model of focal cerebral ischemia, reduces infarct volume, and decreases the production of reactive oxygen species, MMP-9, and caspase-8.2 NBO has been shown to improve both tissue O2 availability and cerebral blood flow in experimental focal cerebral ischemia and may reduce the hemodynamic and metabolic disturbances in the penumbra, partly by suppressing perinfarct depolarizations.3 Furthermore, recent clinical studies in acute ischemic stroke also show that NBO is associated with a transient improvement of neurological deficits, MRI abnormalities, and aerobic metabolism, particularly in the penumbral tissue.4–5 Thus, the available preclinical and clinical data suggest that NBO is a promising therapeutic strategy in acute ischemic stroke and may increase the safety of tPA thrombolysis. It seems that the early NBO may also serve as an effective treatment in TIA, considered an ischemic penumbra of varied duration.6

Disclosures

None.

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References
