What role for hypercapnia in obstructive sleep apnea?

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TO THE EDITOR: The question raised by Wang et al. (5) in their recent viewpoint is important inasmuch as apneic events in obstructive sleep apnea (OSA) are associated with both hypoxia and hypercapnia. Given the cerebrovasculature is more sensitive to hypercapnia than hypoxia (2), it is surprising, as Wang et al. (5) indicate, that the impact of OSA-related hypoxemia on neurocognitive consequences of OSA has received greater attention than the impact of OSA-related hypercapnia. Because changes in cerebral blood flow have been linked to those in EEG (5), the increase in EEG delta/alpha ratio during hypercapnia (combined or not with hypoxia) suggested by Wang et al. (5) could be interpreted as an indirect measure of cerebrovascular reactivity to CO2 (CVR). However, the CVR response to hypercapnia in OSA remains equivocal. Although we (3) failed to observe any difference in CVR between patients with severe OSA and matched (age and BMI) healthy controls, others report a lower CVR in moderate-to-severe OSA compared with age-matched controls (4). Because an impaired CVR may contribute to cognitive impairment (1) and untreated OSA is associated with earlier onset cognitive decline, we propose that the cerebral circulation may hold the mechanistic key linking OSA and neurocognitive impairment. Altogether, although the role of OSA-related hypoxemia should not be trivialized, the importance of hypercapnia in the neurocognitive consequences of OSA warrants further research.

DISCLOSURES
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J.V.B. conception and design of research; J.V.B. interpreted results of experiments; J.V.B., G.E.F., and A.E.B. drafted manuscript; J.V.B., G.E.F., and A.E.B. edited and revised manuscript; J.V.B., G.E.F., and A.E.B. approved final version of manuscript.

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