TO THE EDITOR: Pancheva and colleagues (2) have suggested that we had difficulties explaining the results in our recent paper (4) as a consequence of “two misconceptions.” On the contrary, we believe there is incontrovertible proof that these are not misconceptions. First, cerebral vessels respond to changes in arterial PCO2. Fathi and colleagues (1) at the National Institutes of Health demonstrated that isolated human cerebral endothelial cells produce nitric oxide, a potent vasodilator, in direct proportion to PCO2. This provides the mechanistic basis for the cerebrovascular responses to hyper- and hypocapnia that are firmly established in the literature. In our recent work, we affirmed the role of the progressive decline in PCO2 as individuals approach syncope to increase critical closing pressure while resistance area product only decreased when mean arterial pressure began to fall. These CO2-linked processes contribute simultaneously with myogenic, metabolic, and autonomic mechanisms to regulate cerebral blood flow.

Second, with reference to the capillary pump-mechanism and arteriovenous pumps postulated by Pancheva et al. (2), we would state that these can only be tested when they have published peer-reviewed data to support the concepts. Pancheva et al. suggested that the heart would “elevate venous blood against gravity indirectly by hydraulic mutual induction.” Unfortunately for this concept, veins are compliant, storing blood when pressure distends the vessel walls and collapsing when suction is applied as would be the case with elevated heart rate especially during a passive upright tilt with lower body negative pressure. L. B. Rowell(3) explained the basic concepts in Human Cardiovascular Control on page 44 when he stated “the consequent negative pressure would collapse the great veins feeding the right atrium.”

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS
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