The Cerebrovascular Pressure-Flow Relationship: A Simple Concept But a Complex Phenomenon

To the Editor:

We read with interest the article by Lucas et al1 on mean middle cerebral artery blood velocity (MCA \( V_{\text{mean}} \)) and cerebral tissue oxygenation during changes in blood pressure. We may question whether the conclusion by the authors, “that cerebral blood flow (CBF) and oxygenation are not independent of changes in blood pressure requires a paradigm shift in the concept of cerebral autoregulation,” is new or even indisputably supported by the data provided.

The authors’ proposed new paradigm shift was highlighted in 1983 by Heistad and Kontos2 stating that CBF is not independent from blood pressure. In 1998, Panerai3 pointed out that a completely flat pressure-flow relationship would require feedback gains much greater than those normally observed in biological systems.

When attempting to determine the blood pressure-CBF relationship, the assumption is that the cerebrovascular resistance responds exclusively to changes in blood pressure. However, the pharmacologically induced changes in blood pressure in the study by Lucas et al4 are accompanied by shifts in other hemodynamic parameters relevant for cerebral autoregulation. Sodium nitroprusside, for instance, reduces blood pressure by massive vasodilatation. Whether cerebral and systemic resistance vessels dilate to a similar extent is, however, unknown. If not, a “cerebral steal” may preferentially direct blood flow to the low resistance systemic vascular bed rather than the relatively high resistance cerebral bed.4 This could be a reasonable alternative explanation for the reported reduction in MCA \( V_{\text{mean}} \). In addition, reflex tachycardia in response to nitroprusside increases cardiac output.3 There is accumulating evidence, including from the authors’ own work,5 that changes in cardiac output affect MCA \( V_{\text{mean}} \) independently from blood pressure. Vice versa, the \( \alpha \)-agonist phenylephrine induces reflex bradycardia with a reduction in cardiac output. The authors suggest that phenylephrine has no significant cerebrovascular effect, seemingly neglecting the significant sympathetic innervation of the cerebral vessels.6 The reported increase in MCA \( V_{\text{mean}} \) with \( \alpha \)-stimulation may, therefore, be explained by maintaining flow through a vessel of smaller diameter by cerebrovascular constriction in parallel with its effect on other vascular beds, or flow may be reduced explaining the progressive reduction in cerebral tissue oxygenation.

The postulate by Heistad and Kontos2 on the absence of a plateau is confirmed in daily life by the reduction in CBF, MCA \( V_{\text{mean}} \), and cerebral tissue oxygenation associated with standing up when the decline in cerebral perfusion pressure is hardly more than trivial. New data of the last 2 decennia made us aware that determination of the blood pressure-CBF relationship in humans continues to remain difficult, because deliberately induced changes in blood pressure are inevitably accompanied by changes in flow and resistance that each independently have the potential to modify CBF and oxygenation.

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