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_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 al1 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 cerebrovascular bed.4 This could be a reasonable alternative explanation for the reported reduction in MCA V_mean. In addition, reflex tachycardia in response to nitroprusside or α-agonist phenylephrine induces reflex bradycardia with a reduction in cardiac output.5 The postulate by Heistad and Kontos2 on the absence of a CBF mean independently from blood pressure.98.172) and J.T. (NHS grant 2006.B027), and the Dutch Diabetes Foundation sponsored Y.-S.K. (2004.00.00).

Disclosures

None.

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