**Letter to the Editor**

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

We thank Immink et al for their interest in our research. We fully agree that the concept of the cerebral blood flow (CBF) regulation is highly complex; thus, such exchange of viewpoint is clearly welcomed and advantageous to our understanding of this topic. The letter by Immink et al invites us to respond to 2 critical questions: are our findings novel, and is the cerebrovascular system under adrenergic control? As explicated below, our short replies are “yes” and “in certain situations” to these questions.

**Are Our Findings Novel?**

Although the concept of “imperfect” cerebral autoregulation has been reported, as mentioned in our article, no studies have clearly quantified cerebral autoregulation over a wide range of blood pressures in the same human individuals. Nevertheless, unfortunately, the literature is rife with referrals to this “plateau phenomenon,” and the idea that there is an independent relationship between blood pressure and CBF remains part of clinical teaching and “knowledge.” Therefore, based on clear experimental data, we feel that our statement of a paradigm shift is warranted. The main implication of this concept, because of the relatively linear relationship between pressure and flow, is appreciation of the critical importance of the arterial baroreflex in buffering changes in blood pressure and, thus, CBF. Perhaps this is not a new concept, rather just one that has been misplaced over time, in part, because of a landmark review on this topic. We do acknowledge, however, that our findings were perhaps most gracefully described in 1890 by Roy and Sherrington, “The higher the arterial pressure, the greater is the amount of blood which passes through the cerebral blood-vessels and vice versa.”

**Is the Cerebrovascular System Under Adrenergic Control?**

The extent to which CBF is controlled via cerebral sympathetic activity remains unclear. As such, we cannot discount that changes in adrenergic activity may, in part, explain the reduction in frontal cerebral oxygenation saturation observed after phenylephrine infusion. However, the seminal work of Greenfield and Tindall conducted in conscious humans clearly showed that internal carotid artery blood flow does not alter for 20 seconds after injection of relatively high doses of norepinephrine directly into the internal carotid artery. This finding corroborates with studies showing that, when directly observed, middle cerebral artery caliber remains constant during intravenous phenylephrine infusion. More importantly, they indicate that the blood-brain barrier is an effective mechanism that prevents intravascular catecholamines from binding to \( \alpha \)-adrenergic receptors of cerebral arterioles. Obviously this does not preclude perivascular sympathetic nerves from modulating CBF, although it is important to acknowledge that human pial arteries may only be sparingly innervated with low sensitivity to transmural electric field stimulation and topical noradrenaline.

We welcome future correspondence, debate, and experimental studies to help better define the cerebrovascular pressure-flow relationship as, more correctly, both a complex concept and complex phenomenon!

**Disclosures**

None.

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