TO THE EDITOR: This has been an interesting debate that has narrowed the field of disagreement between the two camps. Despite our title, we described in our first article how the sympathetic perivascular nerves, although not influencing steady-state autoregulation at rest, can be activated to constrict the larger cerebral arteries in response to acutely induced hypertension or hypotension, thereby shifting the limits of autoregulation toward higher blood pressure. Further references in support of this are given by Dr. Lars Edvinsson in his comment (see Ref. 1).

Endogenous rises in blood pressure, e.g., during REM sleep, as described in the comment by Dr. Cassaglia and coworkers (1), also causes cerebral vasoconstriction mediated by the sympathetic nerves, which modulate dynamic autoregulation in this situation. It seems likely that this is also an effect on the larger inflow tract resistance vessels of the cerebral circulation. Furthermore, as commented on by Drs. Levine and Zhang (1), they have shown that dynamic CBF autoregulation is abolished by ganglionic blockade, which has little effect on steady-state autoregulation other than shifting its limits toward lower blood pressure.

The main difference of opinion between van Lieshout and Secher and us seems to be on the participation of the cerebral circulation in the systemic response to changes in cardiac output and orthostasis. To explain how the orthostatic fall in CBF is unchanged by ganglionic blockade but still mediated by the sympathetic nervous system, they invoke the effect of intravenously infused phenylephrine, which, as stated by Dr. Edvinsson in his comment, only will have effect in the cerebral blood vessels if the blood-brain barrier is “leaky.” That was surely not the case in the subjects studied by Zhang and Levine (2). Lowering of cardiac output by lowering central blood volume will lead to some sympathetic activation and constriction of the cerebrovascular inflow tract and hence impair autoregulation, as shown in many previous studies. That hardly justifies introducing a concept of regulation of CBF by cardiac output changes.

REFERENCES