A Recipe for Reducing Blood Pressure Variability: Adding Blood Flow to the Mix

To the Editor:

An important medical advancement during the last 3 decades has been the development of accurate but noninvasive blood pressure monitoring devices. The successful application of such technology has lead to growing recognition that identifying elevated blood pressure variability (BPV) across a wide range of time scales may be useful in predicting poorer health outcomes. Thus, with great interest we read the article by Matsui et al1 showing that day-by-day BPV was lower in patients treated with an angiotensin II receptor blocker/calcium channel blocker combination compared with those treated with an angiotensin II receptor blocker/diuretic combination. This study raises the exciting possibility of potential methods for treating elevated BPV.

In light of treatment possibilities, we believe that regional blood flow must also be considered. Taking the brain as an example, the classic model of cerebral autoregulation (CA) misleadingly portrays that cerebral blood flow is maintained constant across a wide range of cerebral perfusion pressures (60–150 mm Hg). This concept implies that mechanisms normally involved in systemic blood pressure control are relatively unimportant for cerebral blood flow in the presence of intact CA. However, blood pressure is not a static entity that can be described purely in steady-state terms; blood pressure is highly dynamic, and we now know that the capacity of CA to counterregulate against changes in blood pressure depends on the time scale of the presenting stimuli. For example, the slower, lower-frequency components of blood pressure are more effectively buffered than faster, higher-frequency components. Although BPV is an important determinant of cerebral blood flow variability, what is not clear is whether BPV alone can be taken to reflect cerebral blood flow variability given that a myriad of mechanisms (including CA) function to stabilize cerebral perfusion. This is a critical consideration given that conditions like ischemic stroke are fundamentally attributed to inadequate perfusion rather than blood pressure, per se.

In the accompanying editorial, Parati and Bilo2 noted the need to identify the mechanisms responsible for day-by-day BPV and the changes in BPV elicited by treatment interventions. We agree, and further add that greater understanding is also required regarding the impact of any such interventions on both short- and long-term perfusion variability of target organs. The detrimental effects of calcium channel blockade on renal autoregulation were documented a decade ago.3 Recently, we showed that cerebrovascular-specific calcium channel blockade (nimodipine, 60 mg) can render the cerebral microcirculation more vulnerable to blood pressure variations, which might explain why nimodipine appears to have a detrimental effect on outcome after ischemic stroke despite its antihypertensive effects.4 Although nimodipine is infrequently indicated in the treatment of hypertension, other classes of calcium channel blockers are widely used with little understanding of their effects on the cerebral circulation. Therefore, whereas the addition of calcium channel blockers may benefit BPV, there is reason to consider that these benefits may be negated or mitigated by impaired autoregulation. The findings of Matsui et al1 encourage us to continue our investigation on the effects of a range of calcium channel blockers on CA.

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