Possible Difference in the Sympathetic Activation on Extreme Dippers With or Without Exaggerated Morning Surge

To the Editor:

We read with great interest the report by Grassi et al\(^1\) that sympathetic activation is responsible for the nocturnal blood pressure (BP) dipping status in hypertensive subjects. We agree that reverse dipping status is characterized by a marked sympathetic activation. We have reported previously that an \(\alpha\)-adrenergic blocker, doxazosin, markedly affects the nocturnal BP dipping status of hypertensive subjects, with apparently greater reduction of nighttime, as well as daytime, BP in reverse dippers.\(^2\) In contrast, the finding that doxazosin had no significant effects on nighttime BP in extreme dippers suggests that \(\alpha\)-adrenergic tone falls, at least during the nighttime, in extreme dippers. However, we disagree with the authors that there was no correlation between extreme dipper status and morning sympathetic activation, as determined using direct measurement of muscle sympathetic nerve activity performed in the morning.

First, there are at least 2 possible patterns of morning BP surge (MBPS) in extreme dippers, one with exaggerated MBPS and another with no exaggerated MBPS. The pathological mechanisms of these variations and their clinical use remain unclear, but the former may be closely associated with morning sympathetic activation.\(^3\) Our previous report\(^4\) showed that 24% of extreme dippers had exaggerated MBPS (\(\geq 55\) mm Hg: top decile of the population), defined as the difference between the mean systolic BP during the 2 hours after waking and the average of 3 readings centered on the lowest systolic BP taken while patients were asleep. The differences in study population between the present study by Grassi et al\(^1\) (mean age: 47 years) and our previous study\(^4\) (mean age: 72 years) suggests that there may have been substantial variation of MBPS among extreme dippers between the 2 studies.

Second, the measurements of sympathetic activation in this study were performed in the morning with subjects in the supine position, which means that the orthostatic changes of sympathetic activation remained unclear. Previously, we observed orthostatic increases in BP and plasma norepinephrine levels in extreme dippers,\(^5\) both of which contributed to orthostatic hypotension. In fact, orthostatic hypertension, defined as a systolic BP increase \(\geq 10\) mm Hg, was found in 72% of extreme dippers, and the BP change was selectively abolished by an \(\alpha\)-adrenergic blocker.\(^5\) These findings suggest that enhanced sympathetic activation or vascular reactivity may occur in extreme dippers at least partly because of the effects of posture change (ie, standing rather than supine measurement). However, the present study excluded subjects who showed a substantial difference in systolic BP of more than \(\pm 5\) mm Hg between sitting and standing. Thus, further studies will be needed to clarify the above hypothesis.

Disclosures

None.

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