Diurnal Blood Pressure Changes in Stroke Subtypes

To the Editor:

We read with interest the recent article by Metoki and colleagues investigating the relationship between the patterns of circadian blood pressure (BP) variation and the magnitude of the morning BP surge (MBPS) with the incidence of stroke subtypes. The authors reported that a disturbed nocturnal BP decline (nondipping/inverse-dipping) was associated with cerebral infarction (CI), whereas an excessive nocturnal BP fall (extreme-dipping) and an exaggerated MBPS were related to intracerebral hemorrhage (ICH).1

However, the former results were deviant from those of Kario et al who documented inverse-dipping as an independent predictor of ICH in hypertensive patients.2 Our group, after having assessed cross-sectionally the diurnal BP changes in unmedicated hypertensive patients with primary ICH and control subjects, has also documented an independent association of nondipping/inverse-dipping with ICH even after adjustment for baseline characteristics and ambulatory BP levels. In contrast, the prevalence of extreme-dippers did not differ significantly in the 2 subgroups.3

In view of the former inconsistent observations, some limitations from the study of Metoki et al are worth pondering. For one, the authors do not provide information concerning the probable causes of ICH and do not clarify whether they included cases with secondary causes of ICH (vascular malformations, long-standing anticoagulation, coagulopathy, presumed cerebral amyloid angiopathy) in the outcome events of their cohort. Second, although excessive alcohol consumption has been identified as an independent risk factor for ICH,4 this important confounder was not included in the analyses. Third, the prevalence of obesity and obstructive sleep apnea, which have been associated with diminished nocturnal BP dipping, was not investigated. Thus, it may be argued that the barely statistically significant relationship ($P=0.04$) of inverse-dipping/nondipping with an increased risk of ICH, may have been confounded by the aforementioned 2 factors. Fourth, recent evidence has suggested that the different circadian BP patterns in treated hypertensive patients are not related to a significant increase in nocturnal physical activity, but may be associated with the absence of 24-hour therapeutic coverage in most nondipper patients.5 However, the impact of the treatment time on the circadian BP profile and its potential interaction with CI or ICH was not investigated by Metoki et al. Last and most important, no data are presented regarding the time of stroke onset in different stroke subtypes. Since ICH cases are expected to cluster during the MBPS in the subgroup of patients characterized by an excessive nocturnal BP decline followed by a sharp morning BP rise, the prevalence of the different circadian BP profiles in patients experiencing ICH during the MBPS period would be of great interest in the present cohort.

The findings of Metoki and colleagues draw attention to the potential role of the inherent diurnal rhythms of BP variation in the pathogenesis of different stroke subtypes. However, before embarking in a randomized, placebo-controlled trial to investigate whether the therapeutic modulation of these abnormal circadian BP profiles could be beneficial, the prognostic implications of the blunted or exaggerated nocturnal BP fall in the incidence of stroke subtypes remain to be further clarified.

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