Morning Surge, Dipping, and Sleep-Time Blood Pressure as Prognostic Markers of Cardiovascular Risk

To the Editor:

Verdecchia et al recently reported lowest cardiovascular disease (CVD) risk in hypertensive patients of the first quartile of the early morning blood pressure (BP) surge (MBPS) determined by a single before-treatment 24-hour ambulatory BP monitoring (ABPM), defining their results as unexpected, novel, and at variance with previous investigations. Nonetheless, findings of prospective MBPS investigations are inconsistent. On the contrary, numerous studies consistently show, first, an association between blunted sleep-time relative BP decline and increased incidence of fatal and nonfatal CVD events, and, second, the asleep BP mean is a better predictor of CVD events than the awake or 24-hour BP means. Israel et al evaluated the prognostic value of MBPS and the dipping classification of 2627 patients referred for a single 24-hour ABPM study, finding that increased MBPS is significantly associated with decreased mortality. The recently reported Monitorización Ambulatoria para Predicción de Eventos Cardiovasculares (MAPEC) study of 3344 subjects, with baseline BP ranging from normotension to sustained hypertension, and evaluated at least annually by 48-hour ABPM for a median duration of 5.6 years, found that a large MBPS was associated with a significantly lower CVD risk, in line with the lower risk associated with increased BP dipping. Furthermore, when the asleep BP mean was jointly incorporated with either the MBPS or awake BP mean in the same adjusted Cox regression model, only the asleep BP significantly predicted both total and major CVD events (CVD death, myocardial infarction, and stroke); findings were identical for the 607 MAPEC study patients with type 2 diabetes mellitus.1

A major limitation of all previous ABPM-based prognostic studies, except the MAPEC study, is reliance on only a single baseline profile at the time of participant inclusion, without accounting for potential changes in ambulatory BP level and patterning during follow-up as a consequence of treatment, aging, target organ damage, and so on, and thereby assuming the 24-hour ABPM pattern did not change during follow-up. Furthermore, in the absence of periodic multiple ABPM evaluations, it was impossible to evaluate if or how modification of potential prognostic ABPM parameters, increased sleep-time relative BP decline, altered MBPS, and reduced sleep-time BP mean impacted CVD risk. Cox regression analysis of the yearly or more frequent 48-hour ABPM follow-up data of the MAPEC study indicates that progressive decrease in the asleep BP mean was the most significant predictor of survival. Changes in SD, MBPS, and other investigated ABPM-derived parameters during follow-up were less, or not at all, significantly associated with reduced/increased CVD risk. Corroboration of these findings is currently being examined in the multicenter Hygia Project (www.clinicaltrials.gov, NCT00741585) designed to evaluate prospectively CVD risk by annual or more frequent 48-hour ABPM studies. The above cited reports, surprisingly omitted in the discussion by Verdecchia et al, markedly limit the assumed novelty of their findings; moreover, prospective intervention trials involving systematic periodic ABPM evaluation have not only already been reported but are also currently being performed.

Disclosures

None.

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