Is Isolated Systolic Nondipping Pattern Related To Prevalent Subclinical Organ Damage?

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A growing body of evidence from hypertensive cohorts and population-based studies supports the view that an elevated nocturnal blood pressure (BP) or a reduced nocturnal BP fall (i.e., nondipping pattern) is associated to metabolic disorders, subclinical target organ damage, and more importantly a worse cardiovascular prognosis over and beyond the information provided by average 24-h ambulatory BP. The multifactorial pathogenesis of altered BP circadian rhythm remains to be fully elucidated. Dysfunction of the autonomic nervous system including an excess of sympathetic activation, impaired vagal activity, and disturbed baroreflex sensitivity as well as alterations in the circadian pattern of salt excretion (i.e., predominant night-time natriuresis) have been proposed to account for the nondipping pattern. Finally, it is worth noting that sympathetic withdrawal during sleep fully lowers night-time BP only when cardiovascular structure is unimpaired. Thus, target organ damage in nondippers may be not simply regarded as a marker of reduced nocturnal BP fall but as a factor blunting the functional vasodepressor influence of sleep.

The cross-sectional study by Syrseloudis et al. in the current issue of the Journal adds further evidence to the known clinical importance of night-time pressure, by showing that even isolated systolic BP nondipping status is a harmful condition strongly correlated to cardiac and extracardiac organ damage. Hypertensive individuals with isolated nondipping systolic BP had significantly higher left ventricular mass index, pulse wave velocity, and urinary albumin excretion than their counterparts with isolated diastolic or combined systolic and diastolic nondipping patterns. The study further showed that in isolated systolic nondipper patients: (i) alterations in left ventricular structure were paralleled by impaired left ventricular diastolic indices as reflected by lower transmitral early-to-late velocity ratio and prolonged isovolumic relaxation time; (ii) the extent of subclinical organ damage, categorized as the presence of one or more markers, was more prevalent after accounting for differences in age, body mass index, serum C protein, and 24-h systolic BP.

A major challenge in identifying patients with nocturnal BP nondipping pattern relies in determining the effective night-time period and the reproducibility of such classification in reflecting a true clinical trait. In the present study, Syrseloudis et al. used narrow clock-fixed time intervals for definition of daytime and night-time, which excludes the transition periods between wakefulness and sleep in the evening and in the morning. In addition, they properly classified the patients according to their nocturnal concordant BP patterns based on two ambulatory BP monitoring sessions carried out within 1 week. As a consequence of this more accurate definition of nocturnal BP status, it is likely that these patterns based on two concordant ambulatory BP monitoring periods represent true clinical BP phenotypes associated with a different degree of organ damage.

This study indicates that nocturnal isolated systolic hypertension, reflecting an increased night pulse pressure, may be associated to prevalent cardiac, macrovascular, and renal damage. From a clinical standpoint it suggests (despite its cross-sectional nature does not permit any conclusion on the mechanism(s) of this relationship), that an effective systolic BP control during sleep time may have a key role in cardiovascular protection.

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