How Is Slow Wave Sleep Related to Hypertension?

To the Editor:
I recently read with interest the article by Fung et al. I note that in the online Data Supplement (available at http://hyper.ahajournals.org) to this article it is reported that, among the parameters measured, was bilateral leg movements. However, data regarding periodic leg movements in sleep (PLMS) was not reported. PLMS are among the most common sleep disorders in the age group (>65 years) studied in this article. PLMS occur most prominently during the first third of the sleep period. The first third of the sleep period generally includes the majority of slow-wave sleep (SWS). PLMS may disrupt sleep and SWS by producing brief arousals. As PLMS increase with age, SWS decreases.2 From a sleep analysis point of view, it may be argued that effects of any PLMS were included in the analysis, because arousals from all sources were apparently scored. However, PLMS have also been associated with so-called “autonomic arousals” that are not easily scored visually.3 These autonomic arousals are associated with brief increases in both heart rate and blood pressure. However, the effect of PLMS on these cardiovascular parameters are unknown.4 For these reasons, the total number of PLMS or the PLMS index might provide an additional parameter to be considered or may well indeed be related to SWS reduction. Can the reader look forward to a further analysis of the data with PLMS as a factor?

In addition, the data on sleep disordered breathing do not break down the respiratory disturbance index between rapid eye movement (REM) and non-REM sleep. This also appears to be a potential confounding variable that might have reduced the significance of the author’s finding. Subjects whose sleep-disordered breathing is primarily confined to REM sleep would seem more likely to have less interrupted non-REM sleep and thus more SWS. The presence of a high respiratory disturbance index (in REM) and high SWS would certainly act to reduce the significance of the findings.

I am not convinced by these data that the reduction of SWS, in and of itself, is somehow related to hypertension. SWS can be reduced or completely eliminated by benzodiazepines and other sedative/hypnotics. Yet, I am not aware that the absence of SWS in this massive population of patients results in hypertension. A significant reduction or elimination of SWS can also occur secondary to many forms of sleep-fragmenting disorders, including sleep apnea and PLMS, or could result from normal aging of the brain. It would appear reasonable that the source of the SWS reduction is most likely associated with hypertension.

Disclosures
None.

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References

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