Dipping Deeper Into the Ambulatory Arterial Stiffness Index

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Letter to the Editor

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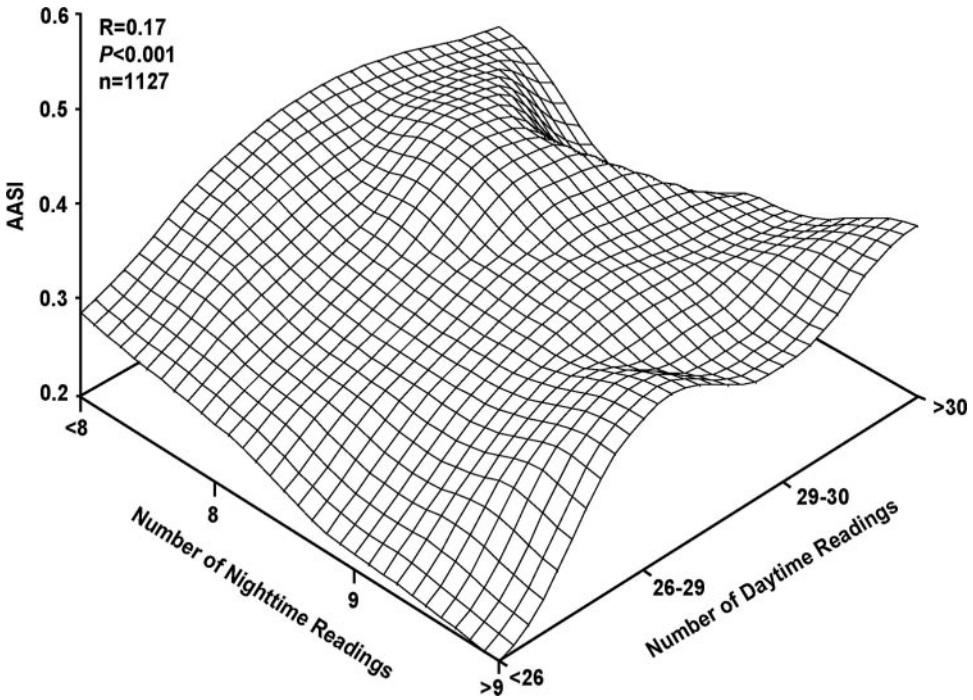
To the Editor:

Methodologic and conceptual issues seriously weaken the conclusions of Schillaci et al[1] on the ambulatory arterial stiffness index (AASI), as published in the May 2007 issue of *Hypertension*.

Schillaci et al[1] reported that, in 515 untreated patients, AASI depended on the nocturnal blood pressure fall. We confirmed this observation in our Flemish population study.[2] The correlation coefficients were similar to those in the report by Schillaci et al[1]: −0.24 versus −0.28 for systolic blood pressure (2-sided *P* value computed by Fisher's *Z* transformation, 0.42), and −0.39 versus −0.46 for diastolic blood pressure ($P=0.11$). However, the ambulatory recording of 1 of the representative patients of Schillaci et al[1] included ≈25 nighttime and ≈35 daytime readings. The night:day ratio of the number of blood pressure readings was therefore 0.71, whereas in our studies,[2,3] it was ≈0.30. As shown in the Figure, this ratio influences estimates of AASI. Furthermore, in our 166 Chinese volunteers,[3] in whom we measured AASI and pulse wave velocity (PWV) within 24 hours, the correlation coefficients with the percentage fall in nocturnal blood pressure were similar for PWV and AASI (MTEST statement in the PROC REG procedure of the SAS package, version 9.1.3; $P>0.54$), amounting to −0.54 and −0.49 for systolic dipping and to −0.56 and −0.57 for diastolic dipping.

Schillaci et al[1] did not seek survival of AASI in the multivariate-adjusted association with left ventricular mass index. In Table 3 of their report,[1] they introduced not independent but highly intercorrelated predictors, bound to remove AASI from the model. The correlation between the daytime systolic pressure and the nocturnal fall in systolic blood pressure is close, because computation of the latter requires use of the former. Schillaci et al[1] did not report the t-to-enter for AASI and the variable, probably the daytime systolic blood pressure, that excluded AASI to remain in the model. More importantly, Schillaci et al[1] failed to demonstrate that, with similar adjustments applied as for AASI, the association between left ventricular mass index and PWV remained significant.

In line with our first report on AASI,[3] Schillaci et al[1] found significant ($P<0.001$) association between PWV and AASI, although the correlation coefficient was lower (0.28 versus 0.51; $P=0.0039$). Schillaci et al[1] must be aware of Bland and Altman's[4] recommendations for assessing concordance between 2 measurements and our analyses complying with these recommendations.[5] Nevertheless, the Italian investigators did not go beyond reporting a correlation coefficient as measure of agreement. Moreover, it is conceptually wrong in the assessment of concordance between measurements to adjust for common determinants underlying the measured trait. In our Chinese volunteers,[3] mean arterial pressure removed the association between PWV and AASI. That accounting for common determinants weakens the correlation between PWV and AASI actually corroborates that these 2 measurements reflect arterial stiffness.



Association of the AASI with the number of daytime and nighttime readings in 1127 subjects randomly recruited from a Flemish population.[2]

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In conclusion, we confirmed that AASI is inversely correlated with the nocturnal fall in blood pressure, especially in ambulatory recordings with a disproportionately large number of nighttime readings. We concur with the idea that AASI is an indirect measure of arterial stiffness.[3,5] However, we disagree with inappropriate or unnecessary adjustment in regression models,[1] and we regret that the information on the correlations of left ventricular mass index with AASI and PWV was incomplete.[1]

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Disclosures

None.

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