Letter by Monfredi et al Regarding Article, “Physical Activity and Heart Rate Variability in Older Adults: The Cardiovascular Health Study”

To the Editor:

Circulation recently published the work of Soares-Miranda et al., in which physical activity and heart rate variability (HRV) in older adults were evaluated. The authors showed that greater leisure activity, walking distance, and walking pace are associated with higher HRV, which they interpreted as better autonomic function. We have reservations concerning this conclusion. First, physical activity lowers resting heart rate (HR). It is established that time- and frequency-domain measures of HRV are nonlinearly dependent on HR, with low HRV being intrinsically linked to a higher HR, and vice versa. We argue that HRV is a nonlinear surrogate of HR. Soares-Miranda et al recognized the dependence of HRV on HR, and stated that HR was evaluated as a “potential confounder or mediator” of HRV in a separate analysis by “adding a multiplicative interaction term and assessing the statistical significance of the Wald test.” They state: “Results were . . . not appreciably altered” by “further adjustment for baseline HR.” However, they do not give the mean HRs of the different groups at different time points. Furthermore, there are no details of how HRV was corrected for differences in HR; we assume they made a linear correction, which will be inadequate to correct for the nonlinear dependence of HRV on HR. The importance of HR on observed changes in HRV can be estimated: Zaza et al have independently shown that the natural log of standard deviation of normal-to-normal beats—log(SDNN)—changes by 0.16 to 0.17 for every 10 beats per minute (bpm) change in HR; our own data yield a similar value. It follows that the ratio of SDNN in 2 different conditions is an exponential function of the difference in HR (ie, \( \frac{SDNN_2}{SDNN_1} = e^{-(HR_2 - HR_1) / 58.8} \)). This equation shows that the modest increase in SDNN associated with activity in the study of Soares-Miranda et al (eg, from 104.8–115.5 ms in Figure 2) could be explained by a modest and plausible bradycardia (5.7 bpm decrease in the example given). Secondly, Soares-Miranda et al conclude that greater levels of physical activity lead to “more favorable indices of HRV” through reduction in sympathetic nerve activity and increased vagal activity. Previously, we have argued that there is no substantive evidence that resting bradycardia developing after physical activity is related to increased vagal activity. Instead, Soares-Miranda et al should be afforded the opportunity to review the raw data to satisfy themselves that proper correction has been made.

Disclosures

None.

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References


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