Importance of Pressure Pulse Amplification in the Association of Resting Heart Rate and Arterial Stiffness

To the Editor:

The study by Whelton et al.1 shows that resting heart rate is independently associated with arterial stiffness. Arterial stiffness was quantified as distensibility and determined from ultrasound measurements of carotid diameter and MRI measurements of aortic diameter. Distensibility, computed from the relative change in carotid diameter and relative change in aortic area for a cardiac cycle divided by the brachial pulse pressure, was related to heart rate that spanned an average range of 50.9 to 78.1 bpm between the first and the last quintile, respectively (Table 1 of Whelton et al.1). Distensibility showed a marked reduction with heart rate for the carotid artery and less so for the aorta. However, with change in heart rate, the pulse pressure that is associated with the measured change in vessel caliber cannot be measured accurately from a peripheral and distal location, such as the brachial artery. The aortic pressure pulse undergoes change in wave shape and amplitude as it travels to peripheral locations, and this increases with heart rate. Using the amplification values reported by Wilkinson et al.2 derived in paced patients, average amplification increases from 18% at 50.9 bpm to 42% at 78.1 bpm. When corrected for heart rate–related pulse amplification, the association virtually disappears for the carotid artery and actually increases for the aorta (Table).

We acknowledge that these are only estimates for the amplification, which does not take age into account, and that a single value of amplification is used for both carotid artery and aorta, although this is not unreasonable because both measurement sites are centrally located and are close to the heart. In addition, the large cohort results in a similar mean age for all heart rate quintiles (mean range, 61.8–62.9 years; Table 1 of Whelton et al.1) and that is also similar to the mean age of 63 years of the cohort of Wilkinson et al.2 from which the heart rate–dependent pulse wave amplification was determined (Table). The best case is when there is no or very little pulse amplification that would be so for very low heart rates or possibly for very old age, in which case the distensibility values using brachial pulse pressure would be valid. In all other cases, even though the centrally located vessel caliber measurements using ultrasound or MRI might be accurate, the heart rate effect is overestimated when using brachial pulse pressure and would be significantly reduced, possibly abolished or even reversed, when distensibility is computed using central aortic pulse pressure.

Disclosures

None.

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Table. Distensibility Corrected for Pulse Amplification

<table>
<thead>
<tr>
<th></th>
<th>Carotid</th>
<th>Aorta</th>
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<tbody>
<tr>
<td>Dist-1 (y)</td>
<td>y = 0.0174HR + 3.63 (r² = 0.95)</td>
<td>y = 0.0067HR + 2.26 (r² = 0.83)</td>
</tr>
<tr>
<td>Dist-2 (y)</td>
<td>y = 0.0012HR + 3.31 (r² = 0.03)</td>
<td>y = 0.007HR + 1.92 (r² = 0.71)</td>
</tr>
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</table>

Reduced slope and very low r² value for the corrected carotid distensibility and relatively similar magnitudes of slope, opposite sign and high values of r² for aortic distensibility compared with uncorrected distensibility values. Dist-1 indicates distensibility determined by Whelton et al.1 using brachial pulse pressure; Dist-2, distensibility corrected for the corresponding amplification related to HR (bpm) from pulse amplification calculated from the data of Wilkinson et al.2 using linear regression between HR and ratio of peripheral/central pulse pressure (their Table 2); HR, heart rate; and r, correlation coefficient for linear regression.
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