Does Reduced Vascular Stiffening Fully Explain Preserved Cardiovagal Baroreflex Function in Older Physically Active Men?

To the Editor:

The article on change in vascular stiffening and baroreflex function by Hunt et al1 contains a serious flaw that may have substantially influenced the results and interpretations. The authors used elegant methods to determine pulsatile carotid artery diameter change and to analyze data, but they related this diameter change to pulse pressure in the finger for determination of carotid artery stiffness. The finger artery pressure waveform is quite different in contour and amplitude to that in the carotid artery, and the relationship between the two varies markedly with age and vasoactive interventions.2 The authors studied change in carotidovagal baroreflex function in young untrained men, older untrained men, and older physically active men given nitroprusside and phenylephrine; no allowance was made for alteration in pressure wave transmission, and the carotid pressure wave amplitude was considered to be identical to the finger pressure wave amplitude under all conditions and at all ages. This is not a reasonable assumption because amplification can vary between unity and over 2.0 under different conditions. We agree with others3,4 that pulse wave amplification must be taken into account when studying carotidovagal baroreflex function by noninvasive methods or serious errors will result in arterial compliance calculations.

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Response

At first blush, the concerns raised by O’Rourke and Nichols appear reasonable. However, more careful scrutiny proves them less so. Pulse wave amplification in the finger can increase carotid stiffness estimates in older individuals. Yet, a correction in our subjects would decrease age- and training-related differences, simply reducing the already poor relation between pulsatile stiffness and integrated carotidovagal baroreflex gain.1 The only available tool to correct for pulse wave amplification is not currently feasible during dynamic baroreflex engagement. Carotid artery tonometry may be useful, but an indirect calibration through the transducer can lead to significant cardiac slowing, attributable to baroreceptor stimulation.2 Even ignoring these considerations, Monahan et al3 suggest differences in augmentation may have inconsequential effect. Although differences in pulse wave transmission are an admitted limitation to this research, our mechanical pressure transduction index derives from changes in systolic pressure and carotid diameter. Pressure changes in the finger accurately reflect changes in the brachial artery,4 which demonstrates augmentation similar to the carotid artery.5 Moreover, our index of the neural baroreflex component is unaffected by pulse wave transmission; thus, its strong relation to carotidovagal baroreflex gain (r=0.71) continues to suggest neural function plays a critical role in determining autonomic function.

On its surface, not accounting for pulse wave amplification appears a serious error, but further consideration shows that it did not substantially influence our results or their implications.

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