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

In our analysis, demonstrating an independent and inverse association between resting heart rate and arterial distensibility in the Multi-Ethnic Study of Atherosclerosis (MESA) study, distensibility was measured using the noninvasive imaging modalities of carotid ultrasound and cardiac MRI. On the basis of these measurements, we calculated carotid and aortic distensibility using well-known equations that incorporate pulse pressure as measured from the routine resting brachial artery blood pressure. Avolio et al point out that this peripheral measurement of pulse pressure may be an overestimate of the central aortic pulse pressure, especially at higher heart rates. They propose the use of a model derived from pulse wave analysis work by Wilkinson et al to correct for differences in pulse pressure between the peripheral and the central arteries. Using this model, they think that the association between resting heart rate and distensibility would disappear for the carotid artery and would reverse for the aorta (higher heart rate would be associated with greater distensibility [less stiffness]).

We agree that it would be ideal to measure the pulse pressure directly within the carotid and the aortic arteries. However, this would require invasive measurement techniques that are not feasible in a large population-based study and is a recognized limitation of these distensibility measurements. Pulse wave analysis is indeed an interesting novel methodology that uses peripheral wave forms and a generalized transfer function to estimate the central aortic pressures. However, it is important to note that in the study by Wilkinson et al, pulse wave analysis is conducted at different heart rates using a permanent pacemaker transiently to alter heart rate. A distinction should be drawn here between epidemiologically derived resting heart rate and near-term variability in heart rate. An individual who likely has a chronically elevated resting heart rate may over time develop structural changes to their vasculature that are not reflected in the measurements derived from short-term increases in heart rate.

The pulse wave analysis derived estimates of central pulse pressure, and their effects on the association between resting heart rate and arterial distensibility proposed by Avolio et al are an interesting concept but not without their own limitations, including well-documented differences according to sex and an inverse relationship with height. We encourage further research investigating this association to determine whether these estimates of central amplification accurately predict directly measured values at differing physiological resting heart rates.

Disclosures

None.

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Hypertension. 2013;62:e47; originally published online October 21, 2013; doi: 10.1161/HYPERTENSIONAHA.113.02263

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