Flawed Measurement of Brachial Tonometry for Calculating Aortic Pressure?

To the Editor:

The article by Segers et al1 on aortic-radial wave transmission and amplification casts doubt on the present approach to the calculation of central aortic pressure from the radial artery tonomographic waveform; this assumes that brachial and radial pressures are substantially identical. Findings of high amplification (≈8 mm Hg1 between brachial and radial systolic pulse pressure) cannot support the present practice of calibrating the radial pressure wave from brachial cuff pressures and could strengthen the view that indices of pressure waves that do not depend on cuff pressure (eg, amplification, as a percentage, or augmentation index) might be preferred to estimations of central aortic pressures.

We are uncomfortable about this, believing that central pressures, so determined, have proved useful in trials such as REASON (Preterax in Regression of Arterial Stiffness in a Controlled Double-Blind), CAFE (Conduit Artery Function Evaluation substudy of the Anglo-Scandinavian Cardiac Outcomes Trial [ASCOT]), and Strong Heart Study and that invasively recorded pressure waves2 do not show the minor pressure increase between carotid and brachial sites and the claimed >2-fold greater increase between brachial and radial arteries.1 Authors relied on brachial tonometry, but they had difficulties with this. In 18%, recordings were impossible, and in the remainder, a “highly skilled . . . operator” was required “who qualitatively judged” waveforms as reliable. The problem with such “judgment” is that it is theoretically and practically flawed. The requirements for accurate tonometry cannot be met at the brachial site, because the artery lies under the stiff bicipital aponeurosis, and its anterior surface cannot reliably be flattened under the sensor. Applanation tonometry is accurate at carotid and radial sites, because the artery can be applanated against the radial bone or vertebral bodies and ligaments behind.3,4 We and others have been happy to use radial and carotid tonometry, because we have been able to record similar noninvasive and invasive waveforms consistently in radial and carotid arteries.3,5 However, we have not been consistently able to record similar waveforms in the brachial artery, where the tonometric brachial pulse is usually blunted compared with the normally peaked intra-arterial trace.1,4 Our transfer function studies have shown almost identical modulus values between the aortic-to-brachial as between the aortic-to-radial artery.5

Our concerns will remain until Segers et al1 are able to show that there is greater amplification of invasively recorded pressure waves between brachial and radial arteries than between the central aorta and brachial artery and that brachial tonometry can accurately measure pulse waveforms at the brachial artery. We have been unable to confirm either. The consequence of the approach by Segers et al, also used by others, is that there is little or no difference in calculated systolic or pulse pressure between the central and brachial arteries1 instead of the ≈10 mm Hg universally agreed on for invasive studies and shown in trials such as REASON, CAFE, and Strong Heart Study.

Disclosures

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