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REVIEW

Central pressure should not be used in clinical practice

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Abstract The heart, brain and kidneys are key targets of pulsatile damage in older people and in patients with longstanding hypertension. These central organs are exposed to central systolic and pulse pressures, which may differ from the corresponding peripheral pressures measured in the brachial artery. Studies employing the generalized transfer function as a means to estimate central pressure have demonstrated a large difference between central and peripheral systolic and pulse pressure that diminishes with age but remains substantial even in octogenarians. As a result of this persistent difference, some have advocated that central pressure may represent a more robust indicator of risk for target organ damage and major cardiovascular disease events. From the perspective of risk prediction, it is important to acknowledge that a new technique must add incremental predictive value to what is already commonly measured. Thus, in order to justify the added complexity and expense implicit in the measurement, central pressure must be shown to add significantly to a risk factor model that includes standard cardiovascular disease risk factors. A limited number of studies have shown marginally better correlations between central pressure pulsatility and continuous measures of target organ damage in the heart. A similarly limited number of prospective studies in unique cohorts have suggested that central pressure may provide marginally better risk stratification, although no reclassification analysis has been published. Thus, currently available evidence does not provide sufficient justification for widespread adoption and routine use of central pressure measurements in clinical practice.

Contents

Introduction 9
Technical limitations of devices that measure central pressure 9

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Key misconceptions and limitations of central blood pressure 10
Evidence that central blood pressure provides additive prognostic information 11
Moving blood pressure research out of the 19th century 12
Conflict of interest statement 12
References 12

Introduction

Because of variable effects of timing and amplitude of wave reflections in the arterial system, peripheral blood pressure as conventionally assessed in the arm can differ from central blood pressure in the proximal aorta. As a result of this difference, recent consensus statements have suggested that central pressure may be a better marker of cardiovascular disease risk than the conventional blood pressure from which central blood pressure is derived.[1,2] The hypothesis that central blood pressure should be a better marker of risk for cardiac complications of hypertension seems well founded. In the presence of a potentially large and highly variable relation between central and peripheral blood pressure, central pressure should provide a better measure of hemodynamic load on the heart and therefore should be a better indicator of cardiac risk. However, only a few studies have demonstrated differing relations of central and peripheral pressures with cardiac structure and function and no study has demonstrated that knowledge of central pressure meaningfully reclassifies risk. Furthermore, the concept that "central pressure" is a better indicator of central hemodynamic stress has been extrapolated to other target organs, such as the brain and kidneys, which lie a considerable distance from the heart and proximal aorta. Whether imputed or measured proximal aortic pressure is truly relevant to structure and function in these more distal locations within the arterial tree remains incompletely elucidated. Before central blood pressure can be recommended for widespread clinical usage, a number of critical technical limitations of currently available devices need to be resolved. Then, using properly validated, robust measures of central pressure, it will be necessary to demonstrate that knowledge of central pressure meaningfully reclassifies risk.

Technical limitations of devices that measure central pressure

A number of commercially available devices purport to measure central blood pressure. However, results from various devices vary widely and consensus on an optimal method to impute central pressure is lacking. Critically, methods used to calibrate peripheral waveforms that are used to derive the central pressure waveform are controversial and have a major effect on estimates of central pressure.[3,4] Various approaches to calibration contribute to variable errors in estimated differences between central and peripheral pressures that exceed the actual differences in pressure between the 2 locations.[5–7]

One approach for estimating central pressure involves use of a generalized transfer function, which is applied to a peripheral pressure waveform in order to obtain a surrogate for the central pressure waveform.[8] The transfer function is essentially a low pass (smoothing) filter that compensates for the boost in high frequency components of the pressure waveform as it travels from central aorta to the brachial or radial artery where the waveform is recorded by using a cuff or tonometer. Studies that used invasive peripheral waveforms have shown that central pressure can be estimated using such an approach.[9] However, noninvasive devices generally measure systolic and diastolic blood pressure in the brachial artery and then use those values to calibrate the peak and trough of a radial pressure waveform (Fig. 1). Because of variable amplification of the pressure waveform as it travels from the brachial to the radial recording site, the calibration of the radial waveform with brachial systolic and diastolic pressure leads to underestimation of radial systolic, mean and pulse pressure, whereas diastolic pressure is comparable between brachial and radial sites.[4,10] Since the radial waveform is improperly calibrated, the derived aortic pressure waveform will have systolic, mean and pulse pressures that are too low. When the underestimated values for central systolic and pulse pressure are then compared to brachial cuff pressure, the pressure difference is overestimated by an amount equal to pressure amplification between brachial and radial recording sites (Fig. 1). In order to avoid calibration errors, either a brachial waveform, which is obtained at the same location as cuff pressure, should be used as the source waveform from which to estimate central pressure or the radial waveform should be calibrated to brachial mean and diastolic pressures. The latter approach requires a brachial pressure waveform, which can be acquired by tonometry or by using the oscillometric pressure waveform recorded from a properly fitted and properly inflated brachial cuff. Using brachial blood pressure and a formula to estimate brachial mean pressure is not acceptable because the shape (or K) factor of the brachial pressure waveform is highly variable. In addition, the maximum amplitude algorithm, which is commonly employed in oscillometric devices to estimate mean arterial pressure, has limitations that may be related to arterial stiffness.[11–13]

An alternative approach for estimating central pressure involves finding the inflection point or peak created by the reflected wave in a properly calibrated brachial or radial pressure waveform. This landmark has been referred to as "SBP2." Since flow in the aorta is low during late systole, pressure gradients in the arterial system are relatively small. Furthermore, since the late (reflected wave) pressure peak represents the dominant peak in most adults from midlife onward, the reflected wave peak recorded in the periphery (SBP2) may represent a surrogate for central aortic systolic pressure in older adults.[14–17] However, devices that utilize the SBP2 approach based on a radial