Central aortic pressure and clinical outcomes

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Conventional brachial blood pressure (BP) measurement by sphygmomanometry is simple and has remained the gold standard for the measurement of BP for over a century. Moreover, it is well established that brachial BP levels are predictive of cardiovascular morbidity and mortality. When we measure brachial BP, we do so with the assumption that the pressure measured over the brachial artery accurately reflects the pressures at the ‘business end’ of the circulation, notably the larger ‘central arteries’, that is, the aorta. Intuitively, it seems reasonable to assume that if we could routinely and reliably measure pressures in the central arteries, these central aortic pressures should be more predictive of target organ damage and clinical outcomes than brachial BP. On the other hand, if brachial BP measurements always faithfully reflected the pressures in these central arteries, then there would be no need to consider measurement of central aortic pressures and the measurement of brachial BP would suffice. Alas, brachial BP is not the perfect surrogate for central aortic pressure, and brachial BP levels do differ from those recorded contemporaneously for the central circulation, either directly at cardiac catheterization or derived noninvasively by analysis of the radial pressure waveform.

Mean BP and diastolic BP change little from the aortic root to brachial artery and represent the tonic component of BP. In contrast, systolic BP is the more dynamic, pulsatile pressure component and is amplified with increasing distance from the aortic root. This systolic and pulse pressure amplification process is principally a function of the timing of pressure wave reflections in the circulation. These reflections are more likely to augment systolic and pulse pressure as the pressure wave moves from the aortic root and closer to reflecting sites at the periphery [1]. This amplification phenomenon is more pronounced in younger people with healthy conduit arteries who thus have greater brachial systolic and pulse pressures relative to their corresponding central aortic pressures. Aortic:brachial pulse pressure amplification can be quite marked in young healthy people, and a typical brachial:aortic ratio is 1.5. The amplification process diminishes with ageing, principally due to aortic stiffening and an increased pulse wave velocity. Thus, with ageing or aortic stiffening or both, central aortic pressures are closer to the brachial pressures but are rarely the same [2].

In addition to the important impact of age and aortic stiffness on the relationship between brachial and central aortic pressures, there is also the important impact of drug treatment to consider. After all, brachial BP is used to monitor the efficacy of BP-lowering treatment. Recent studies have clearly demonstrated differential effects of different BP-lowering therapies on brachial and central aortic pressures [3,4]. Thus, ageing, vascular disease and drug treatment can all influence the relationship between central aortic and brachial pressures. Put simply, if a group of people with varied ages and health status all had identical brachial BP, there would be a wide scatter in the corresponding central aortic pressures. So, does it matter?

If it is accepted that brachial BP measurement is not always the perfect surrogate for central aortic pressures, then the question follows as to whether central aortic pressures should be measured more routinely in clinical practice. The answer to this question has two important strands; first, can central aortic pressure be measured in routine clinical practice? Second, the only point in measuring central aortic pressures would be if the measurement provided more accurate information than brachial BP about the patients’ risk and a better assessment of their response to treatment. Techniques have been developed to derive central aortic pressures and related haemodynamic indices noninvasively from tonometrically derived radial or carotid waveforms, calibrated to brachial BPs. Such techniques are continuously being refined and could readily be applied to routine clinical practice. Accordingly, much interest has now been focused on the relationship between central aortic BP and clinical outcomes. In particular, defining whether central aortic pressures are a better predictor of target organ damage, cardiovascular events and mortality when compared with conventional brachial BP measurements.

Recently, there have been a number of clinical outcomes studies published, relating central aortic pressures to clinical outcomes. Most studies have used noninvasive radial or carotid tonometry to acquire waveforms to derive central aortic pressures. Some studies have used invasive direct measurements of central aortic pressures, acquired during routine diagnostic cardiac catheterization. The
mindful of the fact that brachial BP is clearly a function of and their relatively small size.

The most recent study by Wang et al. [10] was reported in this journal. It reported the results of a community-based longitudinal study of 1272 Chinese who underwent measurement of brachial BP and noninvasive central pressure (carotid) measurements at baseline, along with measurements of target organ damage. These were related to total and cardiovascular outcomes after 10 years of follow-up. Cross-sectional analysis at baseline showed that central pressures (central systolic and pulse pressures) were more strongly related to target organ damage [left ventricular hypertrophy (LVH), carotid intima–media thickness and estimated glomerular filtration rate (eGFR)] than their brachial counterparts. Moreover, central aortic systolic pressure was the most predictive of all. In the longitudinal analyses of mortality, after adjustment for various cardiovascular risk factors, only central aortic systolic pressure remained as an independent predictor of cardiovascular mortality. These findings support those of the previous recent studies outlined in the table and a number of consistent themes emerge: central aortic systolic pressure or pulse pressure or both have invariably been shown to be more strongly related to vascular damage, target organ damage, cardiovascular outcomes or mortality whenever they have been compared to brachial BP parameters in the same study; this conclusion seems to apply irrespective of whether the central pressure indices have been acquired from pulse wave analysis or direct invasive measurement; this conclusion also applies across different ethnic groups, including American Indians and Chinese; there is variance as to whether central systolic or central pulse pressure is most strongly predictive in individual studies, which we suspect reflects the demographics of the different studies and their relatively small size.

It is remarkable that these findings have emerged at all, mindful of the fact that brachial BP is clearly a function of central aortic pressure and that the studies cited have been relatively small (~1–2000) with regard to the study of clinical outcomes. This would suggest that central aortic pressure is a powerful and potentially more robust predictor of risk than brachial BP. What is now required is more data. While brachial BP measurement has stood the test of time, time, science and technology move on. It is clear that brachial BP is not always a faithful record of the central aortic systolic and pulse pressure load in individual patients and that this relationship can be further undermined by the differential effects of drug treatments on pressure amplification. To better clarify whether central aortic pressure indices are more accurate predictors of risk and the response to BP-lowering drug therapy, we need to embed central aortic pressure measurements into future large clinical outcome studies testing therapeutic interventions, and larger-scale longitudinal epidemiological studies of clinical outcomes. This is happening and if the evidence cited above holds true, which we think it will, then the move to preferentially measure central aortic pressures in routine clinical practice will follow and will drive the necessary technological developments to allow it to happen.

Recent published studies reporting analyses relating brachial and central aortic pressure indices to major clinical outcomes. The dominant pressure variable is shown after adjustment for other variables in multivariate regression analyses. CV, cardiovascular; R, central pressure derived from radial pulse wave analysis; C, carotid pulse wave analysis; D, direct invasive measurement. *Identifies data expressed per 10 mmHg change in pressure.

Table 1 summarizes recent studies that have evaluated brachial and central aortic pressure relationships with major clinical outcomes.

References


