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The authors are correct in stating that central blood pressure (CBP) is not routinely performed in the United States, however its value is significant and proven over many studies. Central pressure has been shown to more strongly relate to vascular disease and cardiovascular outcomes than traditional blood pressure measures [1-3]. It also can distinguish between the effects of different hypertension medications when traditional blood pressures do not [4]. No study elucidated this more clearly than the ASCOT-CAFÉ studies. ASCOT was a prospective randomized study of atenolol +/- thiazide versus amlodipine +/- perindopril, demonstrating a 16% advantage of the amlodipine arm in CV outcomes. However, brachial blood pressure did not account for the differences in outcomes. A substudy of ASCOT, the CAFÉ study, evaluated central blood pressure and demonstrated that while the groups had nearly identical systolic brachial blood pressures (133.9 vs 133.2, p=ns), the two groups had significant differences in central blood pressure. The Amlodipine arm had significantly reduced central blood pressure compared with atenolol, (121 vs 125 mm Hg, p<0.0001). Thus central blood pressure differentiated between the two treatment arms where brachial pressure did not. While this certainly doesn't prove that the entirety of benefit of being on amlodipine/perindopril is a significant reduction in CBP, it certainly adds to the existing data that higher CBP is associated with more events, and clinicians can use this information to more closely monitor those patients.

Large cohort studies have identified several roles for CBP. For individual patients, central pressure cannot be accurately predicted from brachial pressure, therefore direct measure is required to use this prognostic marker. Central systolic pressures can differ substantially between patients with the same brachial pressures. Central pressure is a better measure than peripheral pressure of the load on target organs such as the heart, brain, and kidney. We have learned that an individual's CBP would be more indicative of hypertension-related risks to those target organs. As guidelines continue to call for more aggressive therapies in high risk patients, clinicians need tools to identify those patients who are at highest risk, and where changes in therapy can effect event reduction. This is most paramount in our 'treatable' cardiovascular risk factors, such as high blood pressure and elevated serum cholesterol. So, the practical answer is, when patients are perceived or measured to be at high cardiovascular risk, determining the central effects of the therapies we are using should lead to improve outcomes, a goal for all clinicians.