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Invited Review Article

Associations between central and brachial blood pressure in patients with hypertension and aortovascular disease: Implications for clinical practice

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ABSTRACT

Central blood pressure (CBP) measurements, compared to brachial blood pressure (bBP), offer a superior predictive accuracy for aortovascular disease outcomes. This emphasises the distinctiveness of central hemodynamic metrics such as CBP, measuring the pressure directly exerted from the cardiac muscle to the major arteries, and provides a more direct assessment of cardiovascular workload than bBP, which measures the pressure against peripheral artery walls. This review synthesises findings evaluating the correlation between CBP and key aortovascular disease markers. Thoracic aortic aneurysm (TAA) growth is a crucial aspect of aortovascular assessment. CBP more accurately correlates with arterial stiffness (AS), the growth of TAA, and cardiovascular diseases, offering a more dependable prediction of aortovascular diseases, adverse cardiovascular events (CVE) and organ damage compared to bBP. The incorporation of CBP into routine clinical practice could enhance aortovascular assessments and therapeutic strategies when compared to bBP, particularly through a deeper understanding of aortic wave dynamics, which could fundamentally alter aortovascular diagnostics and treatment. In conclusion, integrating CBP into aortovascular and cardiovascular risk management is encouraged. Further research is necessary to substantiate these aspects and explore the operative implications of CBP in clinical settings.

Introduction

Blood pressure (BP) measurement is a fundamental aspect of aortovascular risk assessment in all patients and plays a critical role in evaluating and mitigating cardiovascular risk. Elevated BP level is indicative of hypertension, a condition universally recognised as the primary risk factor for aortovascular disorders. The prevalence of hypertension is increasing, driven by an ageing population and the

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widespread adoption of unhealthy lifestyles.[1,2] According to the 2015 Global Burden of Disease analysis, the prevalence of elevated systolic blood pressure grew by 3.2 % from 17.3 % in 1990 to 20.5 % in 2015[3] and is still rising, affecting around 33 % of adults worldwide in 2019.[4] This underscores the imperative need for an advanced understanding and thorough assessment of BP values within the clinical realm.[5,6]

Since its incorporation into clinical practice more than a century ago, BP measurement has traditionally been derived from the brachial artery[7]. This includes readings obtained in diverse settings, including office, home, and ambulatory settings. In contrast, there is a growing recognition of central BP (CBP) measurements as a valuable tool for refining the categorisation of hypertension status and as a reliable predictor of vascular stiffness, target organ damage, and cardiovascular morbidity, in comparison to bBP.[8,9]

Historically, the assessment of CBP required cardiac catheterisation, thereby increasing its complexity.[10] However, recent advances have introduced a range of non-invasive devices for CBP measurement.[11] Comparative investigations have been conducted to evaluate the performance of invasive and non-invasive devices, showing that non-invasive CBP measurements can achieve levels of accuracy comparable to their invasive counterparts.[12]

Non-invasive techniques for measuring CBP have the potential to serve as more accurate and specific tools for evaluating aortic diseases.[13] These devices can directly capture the pressure wave exerted on the aortic wall following the contraction of the left ventricle. The left ventricle contraction causes a pressure load in the proximal part of the aorta, transmitted across the entire aorta.[12] The transmission velocity is relative to the arterial/vascular stiffness, which depends on structural (elastic compensation) and dynamic features (sympathetic activity). The load from the left ventricle contraction generates pressure on the wall of the aorta, which is transmitted as a pulse wave through the arterial tree. At each branching point (bifurcation), this pulse wave reflects and combines with the incoming wavefront. The overlap between these forward and reflected wavefronts forms the augmentation index (AIx), defined as the ratio of the increase in pressure from the initial systolic peak to the reflected wave peak, expressed as a percentage of the pulse pressure. Therefore, the morphology of pulse waves in different segments of the aorta differs from those in the periphery due to the influence of reflected waves, leading to distinct properties. The viscoelastic properties of large blood vessels are key determinants of the timing and interaction between forward and backward wave coupling, and these properties can be quantitatively assessed through the measurement of pulse wave velocity (PWV).[12] The latter is a measurement that quantifies the distensibility of arteries, precisely the speed at which pressure waves propagate through the blood vessels.

This narrative review will discuss the differences between central and peripheral blood pressure and their implications for aortovascular disorders, cardiovascular health, and organ damage.

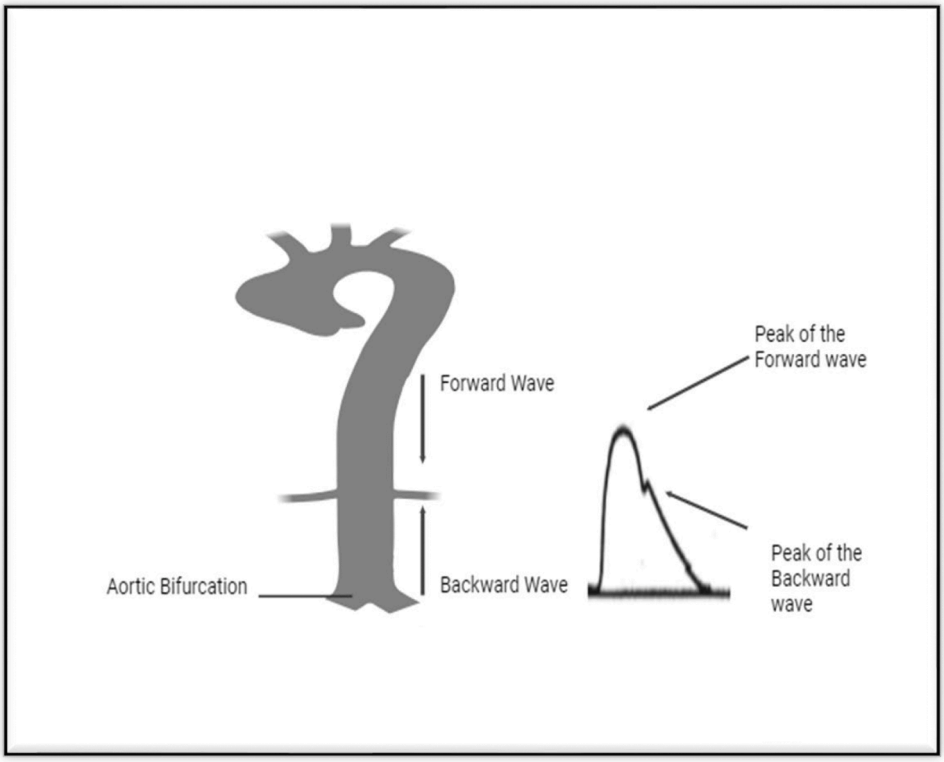

Fig. 1. Schematic Diagram of Aortic Wave Reflection Contributing to the Augmentation Index. This figure illustrates the propagation of the forward pressure wave from the heart along the aorta and the reflection of the backward wave at the aortic bifurcation. The interaction of these waves at the central aorta enhances the systolic peak, contributing to the augmentation index, a quantifiable measure of arterial stiffness and a predictor of cardiovascular risk.

Differences between CBP and bBP

There is often a discrepancy between blood pressure measurements taken at the brachial artery and those taken at the aorta.[14] In the younger population who had been diagnosed with hypertension, there exists a variation of up to 40 mmHg between brachial systolic blood pressure (bSBP)and central systolic blood pressure (cSBP), a phenomenon called BP amplification. Indeed, the bSBP tends to be higher than the cSBP, while the diastolic and mean arterial pressures exhibit relatively stable values.[15]

The disparities in arterial characteristics can be attributed to the varying structural makeup of the arterial walls[16]. The structural changes in arterial walls are crucial in determining arterial elasticity and stiffness, influenced by factors like ageing, hypertension and several other risk factors, often referred to as arteriosclerosis.[17,18]

Structural proteins such as collagen and elastin have a critical role in determining arterial elasticity, with collagen providing support and elastin enabling artery flexibility. Smooth muscle cells regulate vascular tone, and intima-media thickness and fibrosis changes can contribute to AS. Chronic inflammation can further exacerbate AS by causing endothelial dysfunction and fibrosis. Understanding these structural changes is essential for addressing AS and improving cardiovascular health through targeted interventions.[18] The matching between forward and backward waves along the arterial tree and the velocity of the pulse values travelling in central and peripheral arteries determines the extent of the amplification of pulse pressure.[15] The extent of this amplification exhibits significant intra- and inter-individual variation, which is impacted by factors such as age, gender, ethnicity, heart rate, body size, and pathophysiological alterations that affect the vasculature and its tone.[19]

Sex-based disparities in the morphology of central and peripheral arterial pressure waveforms persist consistently across the lifespan.[19] However, with advancing age, these disparities progressively diminish, and the amplification observed in the upper limbs is reduced to a minimal level.

The discrepancy in wave reflection indices is also attributed to differences in stature rather than variations in PWV. The proximity of the place where wave reflection occurs in relation to the heart is found to be closer in females Fig. 1, which can be attributed to their relatively shorter average body height compared to males.[19] Furthermore, ethnic comparisons reveal that African individuals have increased wave reflections and reduced upper limb amplification compared to Caucasian counterparts after correcting for age and sex.[20,21]

Due to the considerable diversity in the determinants affecting PP amplification, it is impractical to forecast this amplification only based on bBP measurements using a single prediction model.[12] Therefore, direct measurement is required for precise evaluation.

The clinical importance of this variability in clinical settings has been emphasised through extensive cohort studies, as seen in Table 1. These studies[8,22–29] have shown significant differences in the gap between cSBP and bSBP across all age groups, this establishing that CBP plays a crucial role in the assessment of hypertensive and aortovascular disorders.

There are diverse methods for measuring or estimating CBP.[12] The conventional method entails the utilisation of intra-arterial catheterisation for the purpose of assessing blood pressure within the ascending aorta. Nevertheless, the utilisation of this invasive methodology, which necessitates specialised proficiency, proves to be unfeasible for regular screening objectives and is presently restricted to the adjustment of non-invasive measurement instruments.

In the last two decades, there has been a significant increase in the availability of non-invasive equipment capable of capturing pressure waveforms from peripheral sites such as the radial, brachial, or femoral arteries.[30] These waveforms are calibrated using bBP measurements obtained using a cuff sphygmomanometer.

Table 1
overview studies comparing central and peripheral blood pressure in different outcomes.

Study reference	Population (mean age)	Summary of findings	Central vs. Peripheral
Wassertheurer et al. 2015[22]	CKD stages 2–4,(59.9)	cSBP predicts mortality better than bSBP.	cSBP > bSBP
Rouxinol-Dias et al. 2018[28]	Mixed BP status patients, (55)	CBP or ABPM abnormalities correlate with AS	CBP = ABPM for predicting AS
Kim et al. 2019[23]	Adults without CVD, (64.5)	CBP is linked more closely to LV health than baPWV.	CBP > baPWV, for LV diastolic funtion
Chuang et al. 2019[8]	General community adults> 19 years old (49.4 male-64.3 female)	CBP uncover more hypertension cases under new guidelines.	CBP>bBP for hypertension diagnosis
Zuo et al. 2020[24]	Elderly hypertensives, (61)	CBP and CPP are superior predictors of CV events in the elderly.	CBP > bBP, for predicting CV events
Khatir et al. 2021[25]	CKD patients	CBP is more sensitive to treatment effects than bBP.	CBP > bBP, for treatment monitoring
Rooprai et al. 2022[26]	TAA patients, (62)	cSBP is linked to larger aneurysms and faster growth.	cSBP > bSBP for aneurysm monitoring
Liu et al. 2022[29]	Hemodialysis patients, (52.6)	Ambulatory PP outperforms CPP in prognosis.	Ambulatory PP > CPP
Abdelmegid et al. 2023[27]	Hypertensive patients, (55.29)	cSBP correlates more with LVH than bSBP.	cSBP >bSBP for LVH correlation

CBP (Central Blood Pressure) and **bBP** (Brachial Blood Pressure). **AS** (Arterial Stiffness) CVE (Cardiovascular Events). **LVH** (Left Ventricular Hypertrophy). **PWV** (Pulse Wave Velocity) and **TIP–FCS** (Thrombogenic Potential-Flow Cytometry Score). **ABPM** (Ambulatory Blood Pressure Monitoring), and **baPWV** (Brachial-Ankle Pulse Wave Velocity). **cSBP** (Central systolic Blood Pressure),**bSBP** (brachial systolic blood pressure). **PP** (Pulse pressure) Ambulatory **PP** (Ambulatory Pulse Pressure**). CPP** (Central Pulse pressure).

Associations between CBP, AS, and TAA

AS is widely acknowledged as an autonomous and direct prognostic determinant for cardiovascular and aortic disorders, playing a crucial role in the early detection of functional and structural alterations in the arterial wall.[17,31] Functional changes, such as endothelial dysfunction and sympathetic activation, can lead to compromised vasodilation and vasoconstriction, which are considered key drivers in the progression of atherosclerosis and CVE.[16] These endothelial and medial layer function alterations are pivotal factors contributing to vascular dysfunction, especially in the context of hypertension and cardiovascular conditions. On the other hand, structural modifications in the arterial wall, specifically increased AS due to changes in the medial layer, are implicated in vascular damage and endothelial dysfunction.[16]

The measurement of carotid-to-femoral pulse wave velocity (cf-PWV) is widely recognised as the primary non-invasive method for assessing AS.[32] One study by Rooprai et al.[26] investigated central hypertension in patients with TAA revealed a significant correlation between CBP and aneurysm growth in patients with TAA. Higher cSBP and central pulse pressure (CPP) were independently associated with larger aneurysm size at baseline and faster aneurysm growth over time, suggesting that CBP measurements, rather than bBP readings, may provide valuable prognostic information regarding the progression of TAA.[26] Similarly, a study by Boczar et al.[33] involving 137 non-operated participants with TAA revealed that combining aortic size with arterial hemodynamics significantly impacted the assessment of future aneurysm expansion. Individuals with large aneurysms (>40 mm) and abnormal aortic function experienced notably faster aneurysm growth rates, with a mean growth of 1.2 mm/year. cSBP showed a positive correlation with aneurysm expansion; for every 10 mmHg increase in cSBP, there was a corresponding 0.3 mm/year increase in aneurysm growth rate.[34]

These findings underscore the importance of a comprehensive evaluation integrating aortic size and hemodynamic parameters in the risk assessment and management of TAA, providing valuable insights for refining surveillance strategies and treatment approaches in clinical practice. A comparative study was conducted to examine gender differences in the progression of aneurysm growth and aortic stiffness. The study showed a significant correlation between carotid-femoral pulse wave velocity (cf-PWV), an indicator of aortic stiffness, and the annual growth rate of aneurysms. Notably, the study highlighted that females, particularly those with degenerative TAA, exhibit faster aneurysm growth compared to males.[33]

AS and CBP are critical in influencing the accelerated growth of TAAs, with a pronounced effect on the female population.[33,35] AS, indicative of aortic wall health and closely linked to CBP, plays a pivotal role in the pathophysiology of TAAs, particularly in females. The intricate interplay between AS and CBP underscores the importance of considering these hemodynamic parameters in the progression of TAAs, especially in female patients.[33] Estimated aortic pulse wave velocity (e-PWV) which is an indirect determination of aortic stiffness, and was independently associated with TAA growth.[36] Importantly, e-PWV showed a stronger association with TAA expansion than traditional clinical variables, with a hazard ratio for the association between e-PWV and TAA growth being 0.24 (95 % CI: 0.09–0.40) in males and 0.15 (95 % CI: 0.05–0.24) in females [ref].

Sex-specific analyses demonstrated that the relationship between e-PWV and TAA growth was stronger in females compared to males, suggesting that incorporating e-PWV measurements into risk assessment models could enhance prognostication and improve personalised management strategies for patients with TAA.[36] Oestrogen is a fundamental female sex hormone that maintains aortic wall elasticity by regulating collagen deposition and elastin production. Age-related changes in oestrogen levels may compromise the protective effects of oestrogen on the aortic wall, leading to increased AS and CBP in females.[33]

These sex-specific correlations between AS, CBP, and TAA growth highlight the complex interaction among haemodynamic factors, hormonal influences, and aneurysm development in female patients.

AS and aortic repair

Exploring the impact of aortic surgery on AS, as measured by PWV, is a crucial area of investigation within aortovascular medicine. The postoperative increase in AS poses challenges related to aortic compliance and the long-term risk of CVE. Understanding the factors influencing changes in AS and optimising surgical strategies to mitigate these effects are essential for improving patient outcomes and reducing cardiovascular risks associated with aortic repair.

Hori et al.[37] Investigated the impact of aortic arch replacement on PWV following surgery. The findings indicated increased PWV observed among patients, regardless of the surgical techniques employed. The research noted a substantial rise in PWV from preoperative to postoperative assessments. Patients who received prosthetic graft replacement exhibited a distinct increase in PWV post-surgery. Similarly, individuals undergoing the frozen elephant trunk procedure demonstrated elevation in PWV after the operation.[37]

Consequently, this increase in PWV prompts an elevation in systolic pressure due to the augmented arrival of the backward wave from the peripheral vessels to the ascending aorta during systole, suggesting potential implications for AS and cardiovascular health in the postoperative period. Investigating different locations of the TAA, a study by Salvi et al.[38] On postoperative and mid-term hemodynamic changes after ascending aorta replacement revealed that patients with aneurysms had a reduced pressure curve slope before surgery (pulsus tardus). After surgery with polyester grafts, there was a significant increase in the PP slope postoperatively, which normalised in the mid-term. Interestingly, aortic stiffness remained unchanged postoperatively and in the mid-term. These results challenge the belief that replacing the ascending aorta with a rigid prosthesis would negatively impact downstream hemodynamics, highlighting the complex relationship between prosthetic materials and systemic circulation.[38]

CBP as a marker of CVE

CBP have greater significance in cardiovascular disease pathophysiology than bBP. Accumulating scientific and clinical data suggests that CBP have a critical predictive role in cardiovascular outcomes and all-cause mortality.[39] Various studies have investigated the capacity of CBP to predict the likelihood of future fatal and nonfatal CVE, such as myocardial infarction, stroke, revascularisation, and aortic syndromes, as well as overall mortality.[31]

Importantly, a comprehensive meta-analysis of 11 longitudinal studies by Vlachopoulos et al.[31], encompassing a cohort of 5648 individuals, unveiled compelling associations between central haemodynamic parameters and cardiovascular outcomes. This found that an increase of 10 mmHg in cSBP is linked to an 8.8 % higher risk of CVE. Similarly, an increase of 10 mmHg in CPP is associated with a 13.7 % higher risk. Furthermore, a 10 % rise in the central AIx was correlated with a significant 31.8 % increase in the risk of CVE.

Three studies consistently demonstrate the importance of central hemodynamic parameters in predicting CVE and mortality.[40–42] CBP metrics, particularly cSBP and CPP, consistently emerged as more robust predictors of adverse cardiovascular outcomes compared to traditional bBP measurements. For example, Li et al.[41] showed that 10 mmHg increase in cSBP was associated with a 10 % higher risk of CVE. In contrast, a 10 mmHg rise in CPP correlated with a 22 % increased risk of all-cause mortality. Moreover, a 10 % increase in the central AIx was linked to an 18 % higher risk of composite CVE and a 19 % increased risk of all-cause mortality. All these results highlight the independent predictive value of central pressures and AIx for both CVE and all-cause mortality.[31]

Predictivity of CBP versus bBP

While some studies emphasised the impact of aortic wave timing, precisely the peak of the aortic forward wave, on cardiovascular risk and left ventricular mass, other studies underscored the independent predictive value of central aortic augmentation index and PP in forecasting CVE and all-cause mortality.[40,42] These highlight the clinical significance of integrating CBP assessments and understanding aortic wave dynamics in risk assessment for cardiovascular diseases, suggesting their potential utility in improving prognostic evaluation and guiding clinical management strategies.

Whether CBP possesses an additional and distinct capacity for prediction compared to bBP remains unanswered. The meta-analysis by Vlachopoulos et al. indicated that CPP demonstrated a borderline improved predictive capability in relation to future CVE and all-cause mortality when compared with peripheral PP.[31] Hence, central hemodynamic parameters, such as CPP, may offer valuable insights into the risk assessment of cardiovascular outcomes. The difference observed underscores the potential importance of considering CPP as a more robust predictor of clinical events compared to peripheral PP.

To obtain compelling and significant data about the comparison between central and peripheral pressures, it is necessary to study large populations due to the robust correlation between these two factors. The slight yet present advantage of CBP over bBP was confirmed by a comprehensive meta-analysis by McEniery et al. of individual data from 22,433 participants across 15 studies.[43] While both cSBP and bSBP showed similar predictive ability for myocardial infarction, central pressure exhibited a statistically significant superiority in predicting stroke, particularly in individuals under the age of 61. After mutual adjustment in the study, the analysis provided valuable insights into the relationship between CBP and cardiovascular outcomes. The associations between cSBP and stroke, after adjusting for bSBP, were stronger in individuals under 61 years of age (1.87 compared to 1.09). These findings suggest that while both CBP and bBP pressure exhibit comparable associations with future CVE when examined independently, cSBP may emerge as a more robust predictor of future stroke, particularly among younger individuals.

Relevance of CBP parameters for end-organ damage

In older adults and individuals with chronic hypertension, the heart, brain, and kidneys are acknowledged as crucial organs significantly impacted by pulsatile damage. These central organs are sensitive to the adverse effects of increased cSBP and PP, which may vary from the bBP typically measured in the brachial artery.

Central haemodynamic indices have been demonstrated to have an independent association with end-organ damage and the development of cardiovascular disease. For instance, coronary artery disease is linked to an elevated aortic AIx.[44] The late systolic augmentation of the central pressure waveform is related to an elevation in the left ventricular mass index, while CBP serves as a separate indicator for left ventricular wall thickness.[45,46] Furthermore, when compared to bBP, CBP exhibits a stronger correlation with critical surrogate endpoints, such as vascular hypertrophy and the degree of carotid atherosclerosis.[47,48]

Several studies have provided evidence indicating that CBP values may have a stronger association with surrogate markers of disease in central organs, including the heart, brain, and kidneys.[49–51] These investigations assume that CBP represents the pressure perceived by central organs as their perfusion pressure. Consequently, CBP should exhibit a stronger correlation with indicators of tissue damage.

One study on blood pressure and cognitive function revealed that elevated cSBP, increased CPP, and reduced PP amplification were linked to diminished cognitive performance across various cognitive domains in a cohort of independently living adults aged 20–82.[50] These findings underscored that CBP and PP amplification serve as valuable indicators of cognitive ageing, capturing aspects of cognitive function not accounted for by traditional bBP measurements.[50]

Other studies demonstrate slightly greater associations between CBP and central indicators, such as carotid intima-media thickness and left ventricular mass, compared to bBP.[23,27,46] These studies support the potential significance of monitoring changes in CBP during therapy.[25] A meta-analysis demonstrated that cSBP is marginally but consistently more effective than bSBP in predicting

end-organ damage, such as carotid intima-media thickness, PWV, and left ventricular mass index, with the exception of albuminuria.[52] Nevertheless, given the robust associations observed between CBP and bBP, it most of the studies lacked adequate statistical power to establish that assessing central pressure provides significant clinical insights into the presence or extent of target organ damage beyond what can be determined by measuring bBP alone. New evidence from interventional studies focusing on interventions for patients with increased CBP rather than bBP values could shed light on this issue. Specifically, the use of spironolactone showed that it might have more beneficial effects in reducing left ventricular mass, CBP, and bBP.[53]

Conclusion

This review focusing on the comparison between CBP versus bBP as predictors of aortovascular and cardiovascular diseases has highlighted the distinct predictive capacity of central hemodynamic parameters, such as cSBP and PP. These parameters are instrumental in forecasting adverse cardiovascular outcomes and all-cause mortality. Incorporating CBP assessments, coupled with a nuanced understanding of aortic wave dynamics, could significantly enhance risk assessment strategies for aortovascular conditions. The demonstrated superiority of CBP in correlating with TAA growth underscores a crucial area for additional research. These findings suggest a need for re-evaluating standard aortovascular risk assessment models to incorporate central hemodynamic parameters, potentially leading to more precise and effective therapeutic interventions. Future studies are essential to explore the practical implications of implementing CBP measurements in routine clinical settings, which could fundamentally alter the approach to aortovascular healthcare.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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