

COMMENTARY

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# Alternative Interpretations on the Case for Non-invasive Central Aortic Pressure Monitoring

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The derivation and application of estimates of central blood pressure continues to generate discussion and debate with published commentaries expressing a range of views regarding the use of central blood pressure (BP) in the management of hypertension. The fundamental point of issue should, however, not be related to device specifics but, assuming a reliable estimate is available, be on the clinical place of central BP estimation and of appropriate use in management of cardiovascular disease, is it under- or over-utilised?

While there has now been a long history of publications on the pros and cons of central BP in clinical practice [1–3], in recent years, new evidence has helped to clarify interpretation of some major issues in the field, whereas other issues remain unresolved. This article expands on some of these issues, and in doing so, presents an alternative interpretation to that previously presented by Keston et al. [4] on the case for non-invasive central aortic pressure monitoring.

No doubt central (or local organ) BP is the pathophysiologically relevant metric. However, as opposed to the abundance of clinical trial evidence in support of standard brachial cuff BP, there is a lack of prospective

outcome studies regarding non-invasively measured central BP. Possibly the main hindrance to proposing any need for general usage of non-invasive central BP are the recent and substantive population studies and individual patient meta-analysis showing that major cardiovascular outcomes have similar strengths of association with central and brachial cuff BPs [5, 6]. As Keston et al. [4] acknowledge, studies of central BP (relatively small, retrospective and usually in samples of convenience) have varied in relative prognostic result; however, in no study has traditional brachial BP not shown the expected prognostic benefit.

To our knowledge, the only signal for elevated cardiovascular risk identified involving central BP may reside with a ‘central hypertension’ phenotype in which cuff BP is controlled (e.g. systolic BP < 130 mmHg) but, due to low systolic BP amplification, central BP is above a ‘central hypertension’ threshold (e.g. systolic BP > 120 mmHg) [7]. This phenotype occurred with low prevalence (3.7%) and was more likely among women taking beta-blockers, within the International Database of Central Arterial Properties for Risk Stratification ( $n = 5576$ ) [7]. The true prevalence of central hypertension is probably higher than that identified because of error in the transfer function at low levels of systolic BP amplification [8]. Existence of a central hypertension phenotype could explain residual cardiovascular risk experienced among some people with controlled hypertension, and thus a theoretical opportunity to follow a different treatment pathway than that guided by cuff BP (e.g. increase anti-hypertensive medication in spite of controlled cuff BP).

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However, this speculation remains to be tested in rigorous clinical trials.

Brachial cuff BP measurement has moved from the basic physics of a column of mercury. After more than a century of actuarial and epidemiological evidence based on this simple technique BP management, in the post-mercury era, predominantly utilises automated devices that contain proprietary variations of increasingly sophisticated, but subtly different, signal processing approaches. Notably, many automated brachial cuff BP devices provide systolic BPs similar to invasively recorded central aortic values [9], thereby effectively functioning as ‘central BP devices,’ without directly applying specialised central BP algorithms or techniques. This occurs due to systematic underestimation of intra-arterial brachial systolic BP for reasons yet to be clarified but probably related to the “oscillometric” waveform processing employed within automated cuff BP devices [10]. There are many other approaches and devices to estimate central BP besides the transfer function method mentioned in the current article [4]. Lack of direct comparability between devices and potentially different device precision makes any blanket statement about applicability of central BP somewhat dubious and difficult to apply in clinical practice [11]. There would need to be good evidence to introduce further “black-box” influences and since it is well established that non-invasive central BP results are device-dependent [11, 12], this additional level of variability without any evidence of benefit would be poor practice.

It has been repeatedly reported that while group average results are similar, there is considerable variation in central BP difference, both positive and negative, when compared to invasively measured central BP in an individual. It cannot be taken for granted, therefore, that any difference in classification between brachial cuff BP assessment and derived central BP must be due to issues relating to the brachial cuff BP measurement rather than to true differences. Brachial cuff BP measurement is an indicator of group risk that is well founded but does not guarantee a personalised prognosis. For example, many people with elevated brachial BP do not have cardiovascular events, whereas others with “normal” BP do. While discordant brachial and non-invasive central BP may involve different sensitivities and specificities, on a community level, there is no evidence based on hard clinical outcomes that would provide reason to consider central BP to be more personalised than brachial cuff BP.

There is a basic paradox in the application of the transfer function recommended by Kesten et al. [4]. The need for a transformed central BP is predicated on magnitude and morphological changes in pressure propagation from central to brachial artery sites. If this forward

transformation was uniform, any central characteristic would be entirely illustrated at the brachial artery. However, it is accepted that the forward transformation is not uniform, and therefore, brachial artery characteristics may not be indicative of central artery characteristics—if this is true, it is difficult to reason that an inverse transformation (brachial to central) of a non-constant (forward) transfer function can be represented as the constant uniform transfer function supplied in commercial devices. Thus, the paradox is, if transformation is uniform backwards, why is it not uniform forwards? If it is uniform in a forward direction, no information content of the central BP is lost in the brachial artery waveform. Brachial cuff BP, logically, is, therefore, as effective a prognostic index as transfer function derived central BP.

Published studies of directly measured central BP in the catheterisation laboratory have failed to show prognostic superiority over brachial cuff BP [13]. These are large studies, admittedly more relevant to secondary than primary prevention, and informative for clinical practice despite design limitations of cuff BP being recorded under variable conditions and not simultaneous with invasive BP [13].

So how should treatment be applied on the basis of central BP? The concept of “spurious hypertension” is talked about—particularly in young males. The condition is diagnosed when non-invasive central BP (taken as accurate) is normal and brachial cuff BP (also taken as accurate) is high [14, 15]. This self-fulfilling diagnosis cannot be verified (except by invasive study) and is predicated on a true measure of central BP, with no way of being certain this is correct in a given individual. Even if correct, the long-term untreated implications of discordant central—brachial systolic BP difference in young adults remains unknown. A potential for doing harm either by under treatment or with unnecessary treatment clearly exists, but yet again, we are faced with large evidence gaps on the merits of cardiovascular risk managed by central BP.

The case for or against non-invasive central aortic pressure monitoring perhaps does depend on one’s point of view, but the real issue is not whether we can derive a parameter designated as central BP (which is easy), but do we need it and should we bother. To date, there is no evidence with hard clinical outcomes that patients or their doctors benefit from knowledge of non-invasive (or invasive) central BP, nor suffer by not knowing central BP. The speculated health economic benefits of central BP monitoring [4] are also yet to be robustly modelled. Overall, the burden of hypertension needs to continue to be treated on the basis of high-quality evidence.

We have detailed an array of additional complexity and issues for consideration on central BP monitoring. Despite many concerns there still may reside a role

for the expert clinician well versed in the limitations of both central and cuff BP technology to use central BP in special circumstances, for example to refine medication titration to achieve optimal BP lowering at lowest dose for patients concerned with taking medications [16], or possibly to help identify increased BP risk despite controlled cuff BP where there may be evidence of target organ damage—the so called central hypertensive phenotype.

There is evidence demonstrating a potential dissociation of the central and peripheral BP effects of some anti-hypertensives with those most likely to produce a greater central than peripheral BP lowering being potent resistance vessel vasodilators. While this difference can be impressive benefit is uncertain as it has been conjectured that the central BP lowering effect may be associated with peripheral damage.

Establishing any benefit from central BP monitoring would require presentation of convincing evidence from high-quality trials, and until such time cuff BP will continue to remain the clinical standard. Currently, the likely best use for assessment of central BP is in small intensively instrumented invasive studies to obtain improved knowledge of basic physiological mechanisms [17] or in large ambulatory pharmaceutical or other interventional studies demonstrating group effects (e.g. ASCOT and beta-blockers) [18], not in individualising hypertension management unless superior accuracy and precision of central BP beyond standard cuff BP can be achieved.

#### Abbreviation

BP Blood pressure

#### Author Contributions

JDC and JES contributed equally to this work.

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None.

#### Availability of Data and Materials

Data will not be made available as all source data for this commentary is referenced and available from standard sources.

#### Declarations

#### Conflict of Interest

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#### Ethical Approval and Consent to Participate

Not applicable.

#### Consent for Publication

Not applicable.

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#### References

1. Cameron JD. Comparison of noninvasive devices for assessing central blood pressure parameters: what to compare, when and why. *J Hypertens.* 2013;31:27–31.
2. Sharman JE. Central pressure should be used in clinical practice. *Artery Res.* 2015;9:1–7.
3. Mitchell GF. Central pressure should not be used in clinical practice. *Artery Res.* 2015;9:8–13.
4. Kesten S, Qasem A, Avolio A. Viewpoint: the case for non-invasive central aortic pressure monitoring in the management of hypertension. *Artery Res.* 2022;28:128–39.
5. Huang QF, Aparicio LS, Thijs L, et al. Cardiovascular end points and mortality are not closer associated with central than peripheral pulsatile blood pressure components. *Hypertension.* 2020;76:350–8.
6. Lamarche F, Agharazii M, Madore F, Goupil R. Prediction of cardiovascular events by type I central systolic blood pressure: a prospective study. *Hypertension.* 2021;77(2):319–27.
7. Cheng YB, Thijs L, Aparicio LS, et al. Risk stratification by cross-classification of central and brachial systolic blood pressure. *Hypertension.* 2022;79(5):1101–11.
8. Bui TV, Picone DS, Schultz MG, et al. Comparison between cuff-based and invasive systolic blood pressure amplification. *J Hypertens.* 2022;40:2037–44.
9. Picone DS, Schultz MG, Otahal P, et al. Accuracy of cuff-measured blood pressure: systematic reviews and meta-analyses. *J Am Coll Cardiol.* 2017;70:572–86.
10. Sharman JE, Tan I, Stergiou GS, et al. Automated “Oscillometric” blood pressure measuring devices: how they work and what they measure. *J Hum Hypertens.* 2023;37:93–100.
11. Narayan O, Casan J, Szarski M, et al. Estimation of central aortic blood pressure: a systematic meta-analysis of available techniques. *J Hypertens.* 2014;32:1727–40.
12. Papaioannou TG, Karageorgopoulou TD, Sergeantanis TN, et al. Accuracy of commercial devices and methods for noninvasive estimation of aortic systolic blood pressure: a systematic review and meta-analysis of invasive validation studies. *J Hypertens.* 2016;34:1237–48.
13. Laugesen E, Knudsen ST, Hansen KW, et al. Invasive aortic pulse pressure is not superior to cuff pulse pressure in cardiovascular risk prediction. *J Hypertens.* 2021;39:607–13.
14. Lurbe E, Redon J. Isolated systolic hypertension in young people is not spurious and should be treated. *Hypertension.* 2016;68:276–80.
15. McEniery CM, Yasmin, Wallace S, et al. Increased stroke volume and aortic stiffness contribute to isolated systolic hypertension in young adults. *Hypertension.* 2005;46:221–6.
16. Sharman JE, Marwick TH, Gilroy D, et al. Randomized trial of guiding hypertension management using central aortic blood pressure compared with best-practice care: principal findings of the bp guide study. *Hypertension.* 2013;62:1138–45.
17. Leung MCH, Meredith IT, Cameron JD. Aortic stiffness affects the coronary blood flow response to percutaneous coronary intervention. *Am J Physiol-Heart Circulat Physiol.* 2006;290:H624–30.
18. Williams B. Differential impact of blood pressure-lowering drugs on central arterial pressure influences clinical outcomes—principal results of the conduit artery function evaluation (Cafe) study in ascot. *Circulation.* 2005;112:3362.