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COMMENT

Divergent effects of systolic blood pressure amplification on accuracy and precision of cuff blood pressure measurement

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Editorial Commentary on: HTR-2022-0798. Bui TV et al. "Accuracy of cuff blood pressure and systolic blood pressure amplification".

A fundamental structural property of the arterial tree is that arteries reduce in calibre and increase in wall stiffness and relative thickness with distance from the proximal aorta. This geometric and elastic nonuniformity has the effect of increasing the amplitude of the propagating pressure pulse towards the periphery [1]. However, since mean arterial pressure cannot increase from the central aorta to the periphery, and remains essentially constant in large conduit arteries, the increase in pulse amplitude is necessarily associated with a change in the shape of the arterial pressure pulse. This phenomenon was observed in simultaneous invasive measurements of central and peripheral pressure in early studies by Rowel et al. in exercise [2]. A key observation in these experiments was that the degree of pulse amplification and change in waveform morphology was related to the level of exercise and driven largely by changes in heart rate. This property of frequency dependency of pulse amplitude amplification was used in mathematical models to develop a generalised transfer function for the estimation of central aortic pressure from the peripheral (radial) pulse [3]. Although the stiffness and propagation properties are dependent on arterial pressure, due to the non-linear elasticity of the arterial wall, this dependency was shown to have a relatively small effect such that the changes in the invasive measurement of aortic pressure during a Valsalva manoeuvre were reliably tracked by the noninvasive estimation [4]. This confirmed that the model could be reliably used over physiological pressure ranges.

The important clinical implications of the frequency-dependent pulse amplification was effectively illustrated in large cohort studies where higher values of central aortic systolic pressure (SBP) were obtained in those being treated with beta blocking agents compared with calcium antagonists for a similar brachial SBP [5]. The lower heart rate resulting in reduced pulse amplification, hence higher relative SBP at the aortic root for similar brachial SBP, can partly explain the results of the LIFE study where the beta-bocking arm (atenolol) showed a much lower degree of regression of left ventricular hypertrophy compared to the angiotensin receptor blocker arm (losartan) for similar decrease in brachial SBP [6]. This suggested that pulse amplification could have a significant role in the assessment and management of hypertension [7].

In this issue of *Hypertension Research*, Bui et al. [8] address the physiological concept of pressure pulse amplification in the context of the conventional pressure measurement in the brachial artery by an automated oscillometric cuff sphygmomanometer. Their study is based on previous findings of different phenotypes based on the degree of aortic-brachial and brachial-radial pulse amplification explaining the differences in invasively measured aortic SBP [9]. The present study by Bui et al. [8] involved the comparison of invasive aortic and brachial SBP with noninvasive brachial SBP measured by automated oscillometric cuff sphygmomanometer. The study was conducted at five independent research sites in 795 participants (74% male, aged 64 ± 11 years) undergoing diagnostic coronary angiography. There were seven different types of automated brachial cuff devices in the five sites. Pulse amplification was determined from the difference of invasive brachial SBP and aortic SBP.

For the whole cohort, there was a significant difference in brachial cuff SBP (130 ± 18 mmHg) compared to

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invasive brachial SBP (138 ± 22 mmHg). The corresponding invasive aortic SBP was 130 ± 21 mmHg. This suggests that the average value for which the cuff device underestimates brachial SBP (7.6 ± 11.9 mmHg) is similar to the invasive SBP amplification (7.3 ± 9.1 mmHg). The cuff overestimated diastolic pressure (DBP) by 7.4 ± 8.3 mmHg, resulting in an underestimation of brachial pulse pressure by 15.0 ± 12.6 mmHg (21%). Comparison of the difference in invasive and cuff SBP was made in quintiles of amplification and showed an increasing difference with increasing SBP amplification. Using these relationships, when the cuff SBP was corrected for the effects of amplification, the corresponding SBP was 137 ± 21 mmHg, which was very similar to the invasive SBP with a difference of 0.3 ± 11.4 mmHg. In addition, when corrections were applied to the cuff SBP measurements, the concordance of classifications across hypertension threshold increased from 57.4% to 63.7%.

The results of the study are of interest in that they suggest a potential correction to the conventional brachial cuff measurements of blood pressure (BP) to account for the underestimation of SBP and overestimation of DBP. However, it is not clear to what extent the variability of amplification can produce confounding errors in individual measurements, compared to the average values obtained from large cohorts. For example, the corrections applied in this study reduced the mean difference between invasive and cuff brachial SBP from 7.6 to 0.3 mmHg but did not affect the standard deviation (from 11.9 to 11.3 mmHg). This suggests that the effective precision was not improved. This means that there will still be substantial variability between individuals and between measurements under different physiological conditions in the same individuals. The sources of variability are difficult to discern as they are due to concomitant variations of physiological effects and measurement artefact. The authors of the study claim that the SBP amplification explains most of the variance in the accuracy of cuff SBP but with an r^2 value of only 19%. This is consistent with a large standard deviation after correction, possibly because of the confounding effect of various sources of error due to measurement and physiology, and which are much more pronounced in the large spread of SBP values compared to corresponding values of DBP and mean pressure [10].

A potential explanation for the lack of improvement in precision is the possible presence of redundancy. The difference in underestimation of cuff brachial SBP is similar to the difference in aortic and brachial SBP. But if amplification is determined by the algebraic difference of aortic and brachial SBP, it is not surprising that correction for SBP amplification will drive convergence of a mean difference to around zero for the whole cohort, but the variability will still be maintained. This contrasts with corrections made using the frequency dependency of the transfer function which takes in the account the whole waveform to correct for the difference between aortic and peripheral SBP [3, 4]. In a study comparing the difference in aortic and radial SBP [11], applying the transfer function model reduced the mean difference from 15.7 to 0.0 mmHg, and the standard deviation from 8.4 to 4.4 mmHg. The frequency dependency of the transfer function directly affects the degree of the pulse amplification. For example, waveforms that are typical of advanced age will have less high frequency information and will have much less amplification, and so will have a higher aortic SBP for similar brachial SBP [12]. This also takes into account the effect of heart rate, which is the main confounding factor that can change pulse amplification between central aorta and peripheral arteries [2, 3, 5, 7, 12]. The effect of variation of heart rate on amplification was not explicitly considered in the study by Bui et al. and it is not known if this may have also been a factor in the difference between invasive and noninvasive brachial SBP.

In the study of Bui et al. [8] it is not obvious what are the potential mechanisms whereby the SBP value obtained from the oscillogram generated by the volume variations of the brachial artery depends on the value of SBP at a distal site such as the ascending aorta. So, the question remains whether the similarity of the difference between cuff and invasive brachial SBP and invasive aortic and brachial SBP (ie. SBP amplification) is fortuitous, and thus introducing an inherent redundancy in the relationship of accuracy and SBP amplification, or it is an invariant property of the interaction of the operation of the oscillometric technique in the cuff BP device and fundamental arterial design in human adults. If the latter is the case, it should also hold for other human cohorts such as youth, where change in pulse waveform and degree of amplification is much higher than older adults, which was the cohort in this present study. With further studies of this observed phenomenon, it might be possible to extend the application to vascular models [13] or patient-specific corrections [14] for improvement of the precision of BP measurement.

Compliance with ethical standards

Conflict of interest The author declares no competing interests.

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References

1. Taylor MG. Wave transmission through an assembly of randomly branching elastic tubes. Biophys J. 1966;6:697–716.
2. Rowell LB, Brengelmann GL, Blackmon JR, Bruce RA, Murray JA. Disparities between aortic and peripheral pulse pressures

induced by upright exercise and vasomotor changes in man. Circulation. 1968;37:954–64.
3. Karamanoglu M, O'Rourke MF, Avolio AP, Kelly RP. An analysis of the relationship between central aortic and peripheral upper limb pressure waves in man. Eur Heart J. 1993;14:160–7.
4. Chen CH, Nevo E, Fetics B, Pak PH, Yin FC, Maughan WL, et al. Estimation of central aortic pressure waveform by mathematical transformation of radial tonometry pressure. Validation of generalized transfer function. Circulation. 1997;95:1827–36.
5. Williams B, Lacy PS, Thom SM, Cruickshank K, Stanton A, Collier D, et al. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. Circulation. 2006;113:1213–25.
6. Dahlof B, Devereux RB, Kjeldsen SE, Julius S, Beevers G, de Faire U, et al. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. Lancet. 2002;359:995–1003.
7. Avolio AP, Van Bortel LM, Boutouyrie P, Cockcroft JR, McEniery CM, Protogerou AD, et al. Role of pulse pressure amplification in arterial hypertension: experts' opinion and review of the data. Hypertension. 2009;54:375–83.
8. Bui TV, Picone DS, Schultz MG, Peng X, Black JA, Dwyer N, et al. Accuracy of cuff blood pressure and systolic blood pressure amplification. Hypertens Res. 2023. https://doi.org/10.1038/s41440-023-01311-0.
9. Picone DS, Schultz MG, Peng X, Black JA, Dwyer N, Roberts-Thomson P, et al. Discovery of new blood pressure phenotypes and relation to accuracy of cuff devices used in daily clinical practice. Hypertension. 2018;71:1239–47.
10. Pauca AL, Wallenhaupt SL, Kon ND, Tucker WY. Does radial artery pressure accurately reflect aortic pressure? Chest. 1992;102:1193–8.
11. Pauca AL, O'Rourke MF, Kon ND. Prospective evaluation of a method for estimating ascending aortic pressure from the radial artery pressure waveform. Hypertension. 2001;38:932–7.
12. Kobayashi M, Oshima K, Iwasaki Y, Kumai Y, Avolio A, Yamashina A, et al. Older age is associated with greater central aortic blood pressure following the exercise stress test in subjects with similar brachial systolic blood pressure. Heart Vessels. 2016;31:1354–60.
13. Flores Geronimo J, Corvera Poire E, Chowienczyk P, Alastruey J. Estimating central pulse pressure from blood flow by identifying the main physical determinants of pulse pressure amplification. Front Physiol. 2021;12:608098.
14. Liu J, Cheng HM, Chen CH, Sung SH, Hahn JO, Mukkamala R. Patient-specific oscillometric blood pressure measurement: validation for accuracy and repeatability. IEEE J Transl Eng Health Med. 2017;5:1900110.