sensing mechanisms (i.e., renal perfusion), stronger correlations between ARR and central BP, compared with brachial BP, may be expected. This was not observed in the Tomaschitz et al. (1) study.

With each cardiac ejection, there is generation of forward traveling pressure waves that move at high speed (e.g., 5 to 20 m/s) from proximal elastic large arteries (e.g., the aorta and carotid artery) distally through increasingly less compliant vasculature (e.g., the brachial and radial arteries). Some of these waves are reflected back to the heart. Overall, the result is amplification of the pressure pulse such that central SBP is always lower than brachial SBP, whereas brachial diastolic blood pressure (DBP) remains similar or is slightly lower (e.g., 1 to 3 mm Hg) than central DBP (5). In patient populations similar to that reported by Tomaschitz et al. (1), directly recorded central SBP is expected to be approximately 10 to 17 mm Hg lower than peripheral SBP (6,7), but this is not the case as presented by Tomaschitz et al. (1). Indeed, in places throughout the text, brachial SBP is lower than central SBP and central DBP is up to 10 mm Hg lower than brachial DBP.

What explains this nonphysiologic discordance between brachial and central BP? Could this be a result of different methodologies and time points at which brachial and central BPs were acquired? For correct data interpretation, this requires clarification. In any case, perhaps the relationship between ARR and central and brachial BP may be best explored using noninvasive central BP methods combined with traditional upper arm BP. This approach also would enable scrutiny of the relationship between BP amplification and ARR and, importantly, would negate the confounding effects associated with measuring BP in the coronary catheterization laboratory.

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REFERENCES

1. Tomaschitz A, Maerz W, Pilz S, et al. Aldosterone/renin ratio determines peripheral and central blood pressure values over a broad range. J Am Coll Cardiol 2010;55:2171–80.
2. McEniery CM, Yasmin, McDonnell B, et al. Central pressure: variability and impact of cardiovascular risk factors. The Anglo-Cardiff Collaborative Trial II. Hypertension 2008;6:1476–82.
3. Sharman JE, Stowasser M, Fassett RG, Marwick TH, Franklin SS. Central blood pressure measurement may improve risk stratification. J Hum Hypertens 2008;12:838–44.
4. Vlachopoulos C, Aznaouridis K, O'Rourke MF, Safar ME, Baou K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. Eur Heart J 2010;15:1865–71.
5. Nichols WW, O'Rourke MF. McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles. 5th edition. London: Hodder Arnold, 2005.
6. Chen CH, Nevo E, Fetics B, et al. Estimation of central aortic pressure waveform by mathematical transformation of radial tonometry pressure. Validation of generalized transfer function. Circulation 1997;9:1827–36.
7. Sharman JE, Lim R, Qasem AM, et al. Validation of a generalized transfer function to noninvasively derive central blood pressure during exercise. Hypertension 2006;47:1203–8.

Reply

We thank Dr. Sharman for his interest in our paper regarding the associations of the aldosterone-to-renin ratio with arterial blood pressure (BP) (1). We appreciate his insightful comments on the differences between peripheral and central BP levels in the LURIC (Ludwigshafen Risk and Cardiovascular Health) study.

Dr. Sharman correctly stated that systolic BP, as the pulsatile component of BP, is amplified with increasing distance from the aortic root, resulting in higher peripheral than corresponding central BP levels. In fact, in LURIC we observed slightly lower peripheral systolic BP levels (141 ± 24 mm Hg) that were measured on the morning of the day when coronary angiography was scheduled (and that constituted the mean of 3 measurements), than central aortic systolic BP values (143 ± 30 mm Hg) that were determined invasively once during routine diagnostic cardiac catheterization later in the day. Peripheral diastolic BP measurements (81 ± 11 mm Hg) were higher than aortic diastolic BP levels (71 ± 12 mm Hg), and here the same limitations apply regarding frequency and timing of the 2 measurements.

The pressure amplification phenomenon generally is more pronounced in the younger age group and diminishes with aging because of the development of progressive aortic stiffening (2). The LURIC cohort represents an elderly population, presumably with increased mean vascular stiffness, as can be deduced from the large percentage of participants exhibiting coronary artery disease (78%). We therefore believe that in addition to the above considerations, a less pronounced pressure amplification might have accounted for the differences seen in diastolic BP levels. Furthermore, ongoing antihypertensive drug treatment, which was noted for 86.8% of the LURIC participants, additionally might have influenced the relationship between central and peripheral BP values because peripheral BP measurements were obtained earlier in the day than invasive BP measurements (2).

We agree with Dr. Sharman that noninvasive measurements of central BP are an appropriate approach. However, it has been recommended in a consensus document pertaining to central BP measurements that central BP ideally should be determined invasively (3). Although it has been shown that noninvasive, that is, tonometric, methods also may measure central BP accurately, inaccurate measurement of cuff pressure, heart rate, height, age, ongoing medication, and observer-dependent factors may constitute important confounders of this method (3,4). We therefore believe that invasive central BP measurement during cardiac catheterization was a reliable method for estimating aortic BP values, despite the fact that the scheduling of peripheral and aortic BP measurements at 2 different time points was suboptimal. We nevertheless found it important to report both central and peripheral BP datasets to demonstrate that there is a strong relationship between the aldosterone-to-renin ratio and BP, independent of the method used for measurement of BP. The concordant finding with both methods strengthens our findings and underlines the important role of inappropriate aldosterone levels in the pathogenesis of arterial hypertension and cardiovascular disease (5,6).

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REFERENCES

1. Tomaschitz A, Maerz W, Pilz S, et al. Aldosterone/renin ratio determines peripheral and central blood pressure values over a broad range. J Am Coll Cardiol 2010;55:2171–80.
2. Williams B, Lacy PS. Central aortic pressure and clinical outcomes. J Hypertens 2009;27:1123–5.
3. Agabiti-Rosei E, Mancia G, O'Rourke MF, et al. Central blood pressure measurements and antihypertensive therapy: a consensus document. Hypertension 2007;50:154–60.
4. Zuo JL, Li Y, Yan ZJ, et al. Validation of the central blood pressure estimation by the SphygmoCor system in Chinese. Blood Press Monit 2010 Mar 30 [E-pub ahead of print].
5. Tomaschitz A, Pilz S, Ritz E, Obermayer-Pietsch B, Pieber TR. Aldosterone and arterial hypertension. Nat Rev Endocrinol 2010;6:83–93.
6. Tomaschitz A, Pilz S, Ritz E, Meinitzer A, Boehm BO, Marz W. Plasma aldosterone levels are associated with increased cardiovascular mortality: the Ludwigshafen Risk and Cardiovascular Health (LURIC) study. Eur Heart J 2010;31:1237–47.