Brief Review

Association of Central and Peripheral Blood Pressures With Intermediate Cardiovascular Phenotypes

Mary J. Roman, Richard B. Devereux

Central blood pressure (BP) has been shown to predict cardiovascular disease clinical outcomes better than brachial BP in studies that demonstrate expected pulse pressure (PP) amplification. The likely underlying mechanism for this observation is a closer relation of central BP to preclinical cardiac and vascular disease because of its more accurate representation of loading conditions on the heart and coronary and cerebral vessels. Over the past 2 decades, proof of concept for this hypothesis has been provided by many cross-sectional analyses. Furthermore, several longitudinal studies in hypertensive patients have provided preliminary evidence to suggest that changes in central BP by pharmacological therapy are more important than decreases in brachial BP in altering cardiac and vascular hypertrophy associated with hypertension. This review will describe studies that have compared central and peripheral BP to intermediate cardiovascular phenotypes (target organ damage).

Although end-diastolic BP is nearly identical throughout the arterial tree, it has been recognized for 50 years that systolic BP varies, at times strikingly, among arterial segments because of the phenomenon of arterial PP amplification. As a result, direct arterial pressure measurements with solid-state catheters have shown that brachial and radial systolic BP is variably higher than concurrently measured central and peripheral BP because of the phenomenon of arterial PP amplification. Although direct arterial pressure measurements with solid-state catheters have shown that brachial and radial systolic BP is variably higher than concurrently measured central arterial BP, the common practice of using conventional sphygmonanometric measurements of brachial BP as pressure load imposed on the heart and on central circulation (including coronary and cerebral arterial trees) may be erroneous. The most commonly used measures of preclinical hypertensive target organ damage, left ventricular (LV) mass and common carotid artery (CCA) geometry, can both be readily and accurately measured using noninvasive techniques, most commonly by the use of ultrasound. Noninvasive central BP has been most commonly derived using either carotid or radial artery applanation tonometry and invasively validated generalized transfer functions.

Carotid Artery Hypertrophy and Atherosclerosis

Numerous studies have compared the relations of central pressure (predominantly PP) versus brachial pressure with carotid wall thickness, lumen diameter, vascular mass, and presence and extent of atherosclerosis. These studies are described below and summarized in Table 1.

In the first comparison of central and brachial PPs as determinants of carotid artery structure, Boutouyrie et al measured right CCA internal diameter and intimal-medial thickness (IMT) in 43 healthy subjects and 124 never-treated hypertensives. Carotid PP was determined from carotid artery applanation, and brachial PP was measured using an oscillometric method. In univariate analyses, CCA diameter and IMT were more strongly related to carotid PP than brachial PP, and in multivariable models, carotid PP was an independent correlate of carotid artery structure, whereas mean brachial pressure and PP were not.

In the much larger, population-based Strong Heart Study of 3520 American Indians, we found both central and brachial PPs to be more strongly related to CCA IMT and mass, as well as the presence and extent of extracranial carotid artery atherosclerosis (all \( P<0.001 \); however, the strengths of association were significantly greater for central PP. This study also found that PP, either brachial or central, was more strongly related to vascular disease than was systolic BP (SBP).

Similarly, in a large community-based Taiwanese population of 1272 normotensive and untreated hypertensive adults, central and brachial PPs were strongly related to CCA IMT (both \( P<0.001 \)), but the strength of association was stronger for central PP compared with brachial PP (\( P<0.05 \)). Comparable to Strong Heart Study findings, PP was a stronger correlate of vascular hypertrophy than was SBP.

In a population-based study of 462 black South Africans living in Johannesburg, CCA IMT was strongly related to central PP derived either by use of a generalized transfer function \(( r=0.49; P<0.0001 )\) or by use of the second shoulder \(( P2)\) of radial waveform \(( r=0.53; P<0.0001 )\). Although associations of IMT with brachial pressures were not provided, the adjustment of central PP for brachial PP or brachial SBP did not influence the strong association with central PP. Furthermore, the authors stated that “brachial BP–target organ relations were abolished” with adjustment for central PP. Again, central PP was more consistently independently related to CCA IMT than was central SBP.

In the European InGenious HyperCar family-based study of the genetics of hypertension, 535 normotensive and mostly in-treatment hypertensive subjects underwent the assessment of cardiovascular target organ damage and radial applanation tonometry. Both central PP and SBP were significantly more strongly related to CCA IMT compared with their brachial...
counterparts.\(^\dagger\) In contrast to most other studies, PP did not bear a strong relation to arterial hypertrophy, compared with SBP. The only study that did not demonstrate superiority of central over brachial pressure is an ancillary study of the Chronic Renal Insufficiency Cohort (CRIC).\(^\dagger\) Among 367 patients with chronic kidney disease studied with carotid ultrasonography and radial applanation tonometry, both central and brachial PPs were strongly and similarly related to CCA IMT and BP measurements was 90 days, in contrast to other studies wherein measurements were performed on the same day.

### LV Hypertrophy

Whether measured by echocardiogram or ECG, LV hypertrophy (LVH) has been shown in numerous population-based samples and clinical studies to be associated with 2-fold higher risks of cardiovascular morbidity and mortality.

### Table 1. Studies Comparing Relations of Central and Brachial Blood Pressures to Carotid Artery Hypertrophy and Atherosclerosis

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>CCA Phenotype</th>
<th>Methods</th>
<th>Central Correlation</th>
<th>Brachial Correlation</th>
<th>Comparison*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Routovryie(^\dagger)</td>
<td>167 HTN plus NL</td>
<td>Right diameter</td>
<td>Carotid(\dagger), USG</td>
<td>PP: 0.33; (P&lt;0.0001)</td>
<td>PP: 0.09; NS</td>
<td>See text</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Right IMT</td>
<td></td>
<td>PP: 0.42; (P&lt;0.0001)</td>
<td>PP: 0.27; (P&lt;0.001)</td>
<td></td>
</tr>
<tr>
<td>Roman(^\dagger)</td>
<td>3520 Al</td>
<td>Mean IMT</td>
<td>Radial(\dagger), USG</td>
<td>PP: 0.293; (P&lt;0.001)</td>
<td>PP: 0.249; (P&lt;0.001)</td>
<td>(P&lt;0.002)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean mass</td>
<td></td>
<td>SBP: 0.257; (P&lt;0.001)</td>
<td>SBP: 0.196; (P&lt;0.001)</td>
<td>(P&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>PP: 0.320; (P&lt;0.001)</td>
<td>PP: 0.289; (P&lt;0.001)</td>
<td>(P&lt;0.005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.317; (P&lt;0.001)</td>
<td>SBP: 0.264; (P&lt;0.001)</td>
<td>(P&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Plaque score</td>
<td>PP: 0.364; (P&lt;0.001)</td>
<td>PP: 0.309; (P&lt;0.001)</td>
<td>(P&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.288; (P&lt;0.001)</td>
<td>SBP: 0.221; (P&lt;0.001)</td>
<td>(P&lt;0.001)</td>
</tr>
<tr>
<td>Wang(^\dagger)</td>
<td>1272 HTN plus NL</td>
<td>Right IMT</td>
<td>Carotid(\dagger), USG</td>
<td>PP: 0.265; (P&lt;0.001)</td>
<td>PP: 0.204; (P&lt;0.001)</td>
<td>(P&lt;0.005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.252; (P&lt;0.001)</td>
<td>SBP: 0.225; (P&lt;0.001)</td>
<td>n/a</td>
</tr>
<tr>
<td>DeLoach(^\dagger)</td>
<td>367 CKD</td>
<td>IMT</td>
<td>Radial(\dagger), USG</td>
<td>PP: 0.36; (P&lt;0.001)</td>
<td>PP: 0.32; (P&lt;0.0001)</td>
<td>Not different</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.29; (P&lt;0.0001)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neisius(^\dagger)</td>
<td>535 HTN plus NL</td>
<td>IMT</td>
<td>Radial(\dagger), USG</td>
<td>PP: 0.426; (P&lt;0.001)</td>
<td>PP: 0.235; (P&lt;0.001)</td>
<td>(P&lt;0.001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.478; (P&lt;0.001)</td>
<td>SBP: 0.417; (P&lt;0.001)</td>
<td>(P=0.01)</td>
</tr>
</tbody>
</table>

AI indicates American Indian; CCA, common carotid artery; CKD, chronic kidney disease; HTN, hypertensives; IMT, intimal-medial thickness; n/a, not available; NL, normotensives; P2, second shoulder of the radial waveform; PP, pulse pressure; SA, South African; SBP, systolic blood pressure; and USG, ultrasound.

*Statistical comparison of central vs brachial correlation.

**Tonometry site.

†Associations remained significant \((P<0.0001)\) after adjustment for either brachial SBP or PP.

### Table 2. Studies Comparing Relations of Central and Brachial Blood Pressures to Left Ventricular Mass and Hypertrophy

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Phenotype</th>
<th>Methods</th>
<th>Central Correlation</th>
<th>Brachial Correlation</th>
<th>Comparison*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Covic(^\dagger)</td>
<td>51 ESRD</td>
<td>LV mass</td>
<td>Radial(\dagger), echo</td>
<td>SBP: 0.56; (P&lt;0.0001)</td>
<td>SBP: 0.35; (P=0.04)</td>
<td>n/a</td>
</tr>
<tr>
<td>Wang(^\dagger)</td>
<td>1272 HTN plus NL</td>
<td>LV mass/BSA</td>
<td>Carotid(\dagger), echo</td>
<td>PP: 0.286; (P&lt;0.001)</td>
<td>PP: 0.219; (P&lt;0.001)</td>
<td>(P&lt;0.005)</td>
</tr>
<tr>
<td>Roman(^\dagger)</td>
<td>3520 Al</td>
<td>LV mass/Ht 2.7</td>
<td>Radial(\dagger), echo</td>
<td>PP: 0.335; (P&lt;0.001)</td>
<td>PP: 0.219; (P&lt;0.001)</td>
<td>(P&lt;0.005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RWT</td>
<td></td>
<td>SBP: 0.396; (P&lt;0.001)</td>
<td>SBP: 0.370; (P=0.001)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>PP: 0.167; (P&lt;0.001)</td>
<td>PP: 0.130; (P&lt;0.001)</td>
<td>(P&lt;0.02)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>SBP: 0.286; (P&lt;0.001)</td>
<td>SBP: 0.250; (P&lt;0.001)</td>
<td>(P=0.005)</td>
</tr>
<tr>
<td>Norton(^\dagger)</td>
<td>678 black SA</td>
<td>LV mass/Ht 2.7</td>
<td>Radial(\dagger), echo</td>
<td>PP: 0.41; (P&lt;0.0001)</td>
<td>PP: 0.389; (P&lt;0.001)</td>
<td>(P&lt;0.005)</td>
</tr>
<tr>
<td>Neisius(^\dagger)</td>
<td>535 HTN plus NL</td>
<td>LV mass/Ht 2.7</td>
<td>Radial(\dagger), echo</td>
<td>PP: 0.385; (P&lt;0.001)</td>
<td>PP: 0.189; (P&lt;0.001)</td>
<td>(P&lt;0.01)</td>
</tr>
<tr>
<td>Wohlfahrts(^t)</td>
<td>657 Czechs</td>
<td>LVH</td>
<td>ECG</td>
<td>SBP: AUC, 0.90±0.02</td>
<td>SBP: AUC, 0.83±0.03</td>
<td>(P&lt;0.05)</td>
</tr>
</tbody>
</table>

AI indicates American Indian; AUC, area under the curve; BSA, body surface area; echo, echocardiography; ESRD, end-stage renal disease; Ht, height; HTN, hypertensives; LVH, left ventricular (LV) hypertrophy; n/a, not available; NL, normotensives; NS, nonsignificant; P2, second shoulder of the radial waveform; PP, pulse pressure; RWT, relative wall thickness; SA, South African; and SBP, systolic blood pressure.

*Statistical comparison of central vs brachial correlation.

**Tonometry site.

†Associations remained significant \((P<0.0001)\) after adjustment for either brachial SBP or PP.
independent of conventionally measured BP and other risk factors.37 Numerous studies, described below and summarized in Table 2, have examined the comparative relations of LV mass to central SBP or PP versus brachial BP.

The first of these studies was performed in a select population of 51 healthy patients with end-stage renal disease undergoing thrice-weekly hemodialysis.14 Echocardiography was performed 10 to 14 hours after dialysis. Predialysis central SBP bore a stronger relation to LV mass \((r=0.56; P<0.001)\) than did brachial SBP \((r=0.35; P=0.04)\). Data are not provided for PPs, indexed LV mass, or for BP recorded after dialysis and thus more closely aligned with potential fluid shifts as well as the timing of echocardiography.

The first large study \((n=1272)\) to examine this issue was the population-based Taiwanese study.13 Central SBP was more strongly related to LV mass index than was brachial SBP, and both systolic pressures were superior to their PP counterparts. This result confirmed earlier findings showing brachial SBP to be a more important stimulus for LVH, compared with brachial PP.19,20Remarkably, similar strengths of association to LV mass/height \(^{2.7}\) were obtained in 3520 participants in the Strong Heart Study.21 This study confirmed the importance of arterial stiffening, using central BP as a marker, in promoting concentric remodeling of the left ventricle (manifest as an increase in LV relative wall thickness).22 In the European InGenious HyperCar study, LV mass/height \(^{2.7}\) was much more strongly related to central than to brachial PP.14 Similar to their findings regarding carotid artery hypertrophy, the strengths of association did not differ between central PP and SBP.

Both standard generalized transfer function–derived central PP and P2-derived central PP were strongly related than brachial PP to LV mass/height \(^{2.7}\) in a community-based South African study,14 and these relations remained strongly independent after adjustment for either brachial PP or SBP. Using a different approach, the same study subdivided participants with normal \((120–129/80–84 \text{ mm Hg})\) or high-normal \((130–139/85–89 \text{ mm Hg})\) brachial BP according to whether their central SBP was normal or elevated \((>112 \text{ mm Hg})\).23 Those with elevated central SBP had significantly greater LV mass index than those with normal central SBP \((44.0±12.1 \text{ versus } 38.2±11.0 \text{ g/m}^{2.7}; P<0.005)\) even after adjustment for important covariates (Figure 1). These findings highlight the greater importance of central BP in promoting cardiac hypertrophy as well as the variable and unpredictable relations between central and brachial pressures.24

In the Czech Post-MONICA Study, the presence of LVH was assessed by ECG using either Sokolow–Lyon or Cornell voltage criteria rather than echocardiography.23 Among 657 participants who had both ECG and radial application tonometry, the presence of ECG-LVH was not related to any BP variable in those <45 years of age. Among older participants, the odds ratios to predict the presence of ECG-LVH were higher for central SBP and PP than those for brachial pressures. In receiver operating characteristic analysis, the area under the curve \((AUC)\) was significantly higher for aortic than brachial SBP \((0.90±0.02 \text{ versus } 0.83±0.03; P<0.05)\) despite the small number \((43; 10\%)\) of participants with ECG-LVH.

### Abnormal LV Diastolic Function

Other studies have assessed whether the parameters of LV diastolic function and, as a corollary, heart failure with preserved ejection fraction (HFPEF) may be more strongly related to central than brachial pressure. One of the first studies to examine this issue was a Mayo Clinic study of older individuals \((≥65 \text{ years})\) at high risk of development of atrial fibrillation.26 Central (derived from radial application tonometry) and brachial PPs, but not carotid-femoral pulse wave velocity (influenced by mean arterial pressure), were independently related to the Doppler echocardiographic grade of diastolic dysfunction as well as left atrial volume index, a marker of chronic LV diastolic dysfunction. There were no significant differences in the strengths of association between central and brachial PPs in this small study of 188 patients.

In a South Korean study of 79 men and 79 women without structural heart disease in whom LV systolic function was normal, simultaneous echocardiography and radial application tonometry were performed.27 Left atrial volume index, again a marker of chronic LV diastolic dysfunction, and estimated LV diastolic filling pressure \((E/E’ \text{ ratio})\) were significantly more strongly related to central as opposed to brachial PP in the entire population \((r=0.22; P<0.003\) for central PP versus \(r=0.18; P=0.014\) for brachial PP; \(E/E’\): \(r=0.30; P<0.001\) for central PP versus \(r=0.17; P=0.030\) for brachial PP). In subgroup analyses, these relations were only present in women, in whom HFPEF is more common.

In a large study \((n=359)\) of patients undergoing diagnostic cardiac catheterization, application tonometry and echocardiography were additionally performed.24 HFPEF was defined as LV ejection fraction >50% with end-diastolic pressure >16 mm Hg and N-terminal pro-brain natriuretic peptide >220 pg/mL. In receiver operating characteristic analyses for the prediction of HFPEF, AUC for brachial PP, \(E/E’ \text{ ratio}, \text{ central PP from tonometry, and invasively determined aortic pulse wave velocity were } 0.816, 0.823, 0.851, \text{ and } 0.867, \text{ respectively (not significantly different). The addition of central PP to } E/E’ \text{ was significantly superior to the addition of brachial PP (AUC, } 0.901 \text{ versus } 0.875; P=0.009 \text{ for comparison).}

### Coronary Artery Disease

Data comparing central and brachial BP relations to the presence and extent of underlying coronary artery disease are limited, in part because of the need for invasive coronary

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**Figure 1.** Comparison of left ventricular (LV) mass index in individuals with normal or high-normal brachial blood pressures subdivided according to whether central systolic blood pressure (SBP) is <112 or ≥112 mm Hg. Data derived from Booysean et al.23
angiography. Studies using noninvasive computed tomo-
graphic angiography are not yet available. Nevertheless, 2
studies have compared central and brachial PPs in this pop-
ulation. The first compared 57 healthy male controls with
114 men with either moderate (50% to 89% maximum ste-
nosis) or severe (≥90% narrowing) coronary artery disease.29
Carotid PP was different in all 3 groups and highest in the
severe group. Carotid SBP (r=0.47; P<0.001) and PP (r=0.45;
P<0.001), but not brachial pressures or pulse wave velocity,
were independent correlates of percent coronary artery nar-
rowing. Although the results were not adjusted for ejection
fraction, all patients had an LV ejection fraction >40%. A
more recent study compared intra-aortic and brachial BPsin
1337 patients undergoing invasive coronary angiography.30
Coronary occlusion and low ejection fraction (≤50%) were
independently associated with lower aortic but not brachial
PP. This inverse relation of PP to vascular and LV disease
highlights the extent to which PP depends on both intrinsic
arterial stiffness and LV stroke volume.

Kidney Disease

The kidneys are distant from central aortic pressure, but they
do arise from the abdominal aorta, and their function might
be differentially impacted by central and brachial pressures,
although perhaps to a lesser extent than the more centrally
located heart and coronary and cerebral arteries. In fact, the
population-based Taiwanese study of 1272 normotensive
and untreated hypertensive subjects found that estimated
glomerular filtration rate was significantly more strongly
related to central PP and central SBP than to brachial pres-
sures.13 However, the correlation coefficients for these asso-
ciations (r=−0.131 to −0.187) were substantially lower than
those for LV mass index and carotid IMT. Similarly, in the
population-based South African study subdividing partici-
pants with normal (120–129/80–84 mm Hg) or high-normal
(130–139/85–89 mm Hg) brachial BP according to whether
their central SBP was normal or elevated (>112 mm Hg), those
with elevated central SBP had significantly lower estimated
glomerular filtration rate than those with normal central SBP
(108±26 versus 115±27 mL/min per 1.73 m²; P<0.005).23
Again, these findings highlight the variable relations of central
and brachial pressures with target organ dysfunction.24

The European InGenious HyperCar study examined the uri-
nary albumin to creatinine ratio as a marker of renal disease.15
There were no differences in the association of this ratio with
central and peripheral pressures, and, again, correlation coeffi-
cients (r=0.226–0.236) were lower than those for LV mass
index and carotid IMT. Taken together, these studies suggest a
lesser importance of central compared with brachial pressure
in promoting more remote target organ vascular damage.

Intervention Studies

Several intervention studies have indicated that treatment-related
reductions in LV mass and carotid artery hypertrophy are related
to differential reduction in central as opposed to brachial pres-
sures. However, the magnitude and clinical relevance of these
findings requires confirmation in larger and longer studies.

The first study to suggest a closer relation of treatment-induced
reduction of central (or local) PP to amelioration of target
organ damage involved 98 hypertensive patients who under-
went carotid applanation tonometry and ultrasound and were
randomized to either cefepirone or enalapril.31 After 9 months,
CCA IMT, carotid PP, and brachial pressures decreased sig-
nificantly and similarly in both groups; however, decreases in
carotid IMT and diameter were related to the decrease in
carotid PP but not in mean brachial pressure, but the relation of
brachial PP to change in carotid IMT was not reported.

A subset of 52 patients in the pRIterax in regression of
Arterial Stiffness in a COntrrolled double-bliNd (REASON)
study, comparing the combination of perindopril and inda-
pamide to atenolol, underwent both echocardiography and
applanation tonometry.32 After 1 year, the combination therapy
resulted in greater decreases in carotid and brachial SBPs and
PPs compared with atenolol. The greater reduction in LV mass
in combination therapy was associated with a greater reduction
in carotid PP but not brachial PP. Similarly, 1 year of nebivo-
lol (n=30) versus metoprolol (n=33) antihypertensive ther-
apy resulted in comparable declines in brachial BP, whereas
nebivolol caused greater reductions in central pressures.33
Nebivolol therapy was also associated with a greater reduction
in LV posterior and relative wall thicknesses (change in overall
LV mass was of borderline significance), which were related to
changes in central but not brachial pressures (Figure 2).

These findings are supported by the Hypertension
Associated Cardiovascular Disease substudy of Anglo-
Scandinavian Cardiac Outcome Trial (ASCOT) comparing
amlodipine-based therapy with atenolol-based therapy.34 In
this cross-sectional analysis of the larger longitudinal inter-
vention study, right carotid artery applanation and echocar-
diography were performed in 259 participants 12 to 18 months
after initial randomization. At the time of study, there were no

Figure 2. A, Correlation between central systolic blood pressure
(BP) change and septal thickness change (r=0.41; P=0.001) for
the whole study group. B, Correlation between central pulse
pressure change and septal thickness change (r=0.32; P=0.01)
for the whole study group. IVS indicates interventricular septal
thickness. Reproduced from Kampus et al33 with permission of
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significant differences in brachial SBP or PP; however, carotid SBP and PP were lower and PP amplification was higher in the amlodipine-based group. LV mass index was lower in the amlodipine-based group and was more strongly related to carotid than brachial SBP. Furthermore, when carotid SBP was added to a multivariable model predicting LV mass, the independent impact of treatment arm was eliminated, suggesting that the alteration in wave reflection associated with amlodipine-based therapy and attendant reduction in central pressure contributed to LVH regression.

Limitations
Studies cited in this review relied on mercury sphygmomanometric or oscillometric measurement of brachial BP, which were then used in the calibration of central waveforms. Inaccuracy of brachial BP determination may dilute the relations of both peripheral and central BPs to target organ damage.35 However, despite this potential systematic error, most studies have demonstrated a superior relation of central over brachial BP to intermediate cardiovascular phenotypes or cardiovascular target organ damage. In conclusion, numerous studies have documented a superior relation of central over brachial BP to intermediate cardiovascular hypertrophy by lowering central pressure for a given brachial pressure require confirmation in larger, longitudinal intervention studies.

Disclosures
None.

References
29. Waddell TK, Dart AM, Medley TL, Cameron JD, Kingwell BA. Carotid pressure is a better predictor of coronary artery disease severity than brachial pressure. Hypertension. 2001;38:927–931.
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Hypertension. 2014;63:1148-1153; originally published online March 24, 2014;
doi: 10.1161/HYPERTENSIONAHA.114.03361
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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