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ORIGINAL RESEARCH

Central Systolic Blood Pressure and Left Ventricular Mass: Findings From the International 24-Hour Aortic Blood Pressure Consortium

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BACKGROUND: Central systolic blood pressure (cSBP) reflects the pressure experienced by vital organs and may be more closely related to hypertensive cardiac damage than brachial SBP. Twenty-four–hour ambulatory blood pressure monitoring provides additional insight into cardiovascular load beyond office readings. This study investigated the association of 24-hour cSBP, measured noninvasively with a validated device, with left ventricular mass index (LVMi) and LV hypertrophy.

METHODS: We analyzed data from 21 centers worldwide (n=2367; 45.5% women; mean age 49.5 years) participating in the International 24-Hour Aortic Blood Pressure Consortium. Brachial and central 24-hour SBPs were recorded using the Mobil-O-Graph device, calibrated by either systolic/diastolic (cSBP C1) or mean/diastolic (cSBP C2) pressure; LVMi was derived echocardiographically. BP phenotypes (normotension, isolated diastolic and systolic hypertension, and systolic-diastolic hypertension) were based on 24-hour brachial BPs, subdivided by cSBP.

RESULTS: LVMi correlated most strongly with 24-hour cSBP C2 (P<0.001) consistently, across sex, body mass index (BMI), age, and treatment strata. Receiver operating characteristic analysis showed superior prediction of LV hypertrophy for 24-hour cSBP C2 (area under the curve=0.68). Both brachial BP phenotype and central SBP status were independently associated with LVMi (P=0.0045 and <0.001, respectively), without interaction. Discordant classification occurred in 14.4% of participants and was linked to higher LVMi in normotension and lower LVMi in systolic-diastolic hypertension.

CONCLUSIONS: Twenty-four–hour central SBP calibrated with oscillometric mean/diastolic pressure (cSBP C2), is more closely associated with LVMi and LV hypertrophy than brachial SBP. These findings highlight the potential clinical value of incorporating central pressure monitoring into risk assessment and hypertension management.

Key Words: 24-hour blood pressure monitoring ■ central systolic blood pressure ■ echocardiography ■ hypertrophy, left ventricular ■ left ventricular mass ■ multicenter study

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See Editorial by Chen and Cheng.

CLINICAL PERSPECTIVE

What Is New?

- In a large, international cohort using standardized 24-hour ambulatory monitoring, central systolic blood pressure calibrated with oscillometric mean/diastolic pressure demonstrated a stronger and more consistent association with left ventricular mass and hypertrophy than brachial systolic blood pressure.
- Discordance between brachial and central 24-hour systolic blood pressure classifications was common and was independently associated with differences in left ventricular mass across brachial blood pressure phenotypes.

What Are the Clinical Implications?

- Assessment of 24-hour central systolic blood pressure may refine cardiovascular risk stratification and improve detection of hypertension-mediated cardiac damage beyond brachial blood pressure alone.

Nonstandard Abbreviations and Acronyms

bSBP	brachial systolic blood pressure
cSBP	central systolic blood pressure
cSBP C1	central systolic blood pressure calibrated using systolic/diastolic pressure
cSBP C2	central systolic blood pressure calibrated using mean/diastolic pressure
HMOD	hypertension-mediated organ damage
i24ABC	International 24-Hour Aortic Blood Pressure Consortium
IDH	isolated diastolic hypertension
ISH	isolated systolic hypertension
LVM	left ventricular mass
LVMi	left ventricular mass index
LVH	left ventricular hypertrophy
SDH	systolic–diastolic hypertension

Central systolic blood pressure (cSBP) is typically defined as the SBP in the aorta or carotid artery. This measurement is thought to be crucial because it represents the pressure that most vital organs, including the brain, kidneys, and heart, are exposed to. Unlike mean arterial pressure (MAP) and diastolic blood pressure (DBP), which remain relatively constant along the arterial tree, brachial SBP (bSBP) is physiologically higher than cSBP.[1] This so-called SBP amplification can be observed best when both bSBP and cSBP are measured invasively, and is due to the progressive reduction in diameter and increase in stiffness from proximal to distal arterial vessels, along with the effects of wave reflections.[2] Due to high interindividual variability in pressure amplification from central to distal arteries affected by factors like age, sex, heart rate, body height, and cardiovascular risk factors such as dyslipidemia, diabetes, and smoking,[3] cSBP cannot be directly derived from bSBP. The true cSBP can be measured only invasively—something that is not feasible in daily clinical practice. In order to assess cSBP noninvasively, the most common technique is acquisition of peripheral (radial or brachial) pulse waveforms, calibration with brachial BP, and use of transfer formulae applying dedicated mathematics.[4] Over the past few years, several different noninvasive methods and devices have been introduced.[2] These devices have been compared against invasive measurements of cSBP in several studies[5] and some of them[6] may allow reliable estimation of cSBP noninvasively. Importantly, the method of calibration seems to be crucial at this step. For calibration, either MAP/DBP (cSBP C2) or SBP/DBP (cSBP C1) can be used.

Central SBP has clinical potential as a more accurate predictor of cardiovascular events, such as myocardial infarction and ischemic stroke, compared with bSBP. There is ongoing debate on whether cSBP is pathophysiologically more significant for the development of cardiovascular disease.[7] Evidence from several studies suggests that cSBP may be more closely associated with hypertension-mediated organ damage, such as left ventricular hypertrophy (LVH) and carotid atherosclerosis, than bSBP,[8–10] and may better predict cardiovascular outcomes.[8,11] Conversely, other research has not demonstrated a significantly better association of 24-hour cSBP with LVH or diastolic dysfunction.[12] Taken together, these conflicting findings underscore the need for large, harmonized data sets to clarify whether cSBP truly offers incremental clinical value beyond brachial pressure in predicting cardiovascular damage.

The aim of our study was to determine which measure of SBP—central or brachial—shows the closest association with left ventricular mass index (LVMi) and LVH. Additionally, we aimed to provide potential

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explanations for the conflicting results observed in previous studies through subgroup analyses, such as examining different age groups, and finally, to investigate the influence of waveform calibration, a crucial step in the assessment of cSBP, on the results. This latter question has not been answered definitively yet.

METHODS

Data Availability Statement

The data that support the findings of this study are part of the International 24-Hour Aortic Blood Pressure Consortium (i24ABC). Access to anonymized participant-level data is subject to approval by the consortium's steering committee and the data-sharing policies of participating institutions.

Researchers interested in collaborative projects or secondary analyses may contact the corresponding author (Dr Kathrin Danninger, Klinikum Wels-Grieskirchen, Austria; kathrin.danninger@gmx.at) for further information regarding data access procedures.

Study Overview and Participants

The collection of the data for this study was carried out as part of the (i24ABC). The data that support the findings of this study are available from the corresponding author upon reasonable request. The i24ABC is an international collaborative launched in 2017 with the primary aim of deriving reference standards for 24-hour ambulatory central (aortic) BP and pulsatile hemodynamics. Of 26 centers worldwide participating in the consortium, 21 from 14 countries and 3 continents provided all the mandatory data for a total of 2367 participants (Figure S1) and contributed to this analysis (Table S1). The inclusion and exclusion criteria of the respective centers are available in Table S2. Each participating center had collected data according to the principles outlined in the Declaration of Helsinki and had received approval from their local institutional review boards/ethics committees, and each participant had provided written informed consent (for those younger than 18 years written consent was given by a legal guardian). Approval to conduct the consortium itself was obtained from the Tasmanian Health and Medical Human Research Ethics Committee Tasmania (H0015062). The i24ABC consortium is an exclusively academic research undertaking, without any influence or financial support from device manufacturers. Variables used for analysis were collected systematically at each center and were drawn from medical records or from standardized measurements in population-based studies. The data analyzed for this study are available from the i24ABC consortium upon reasonable request.

Clinical Assessments

The clinical assessments of the study participants were carried out at the participating centers. The assessments included a brief clinical history (personal history of diabetes, prior cardiovascular disease, renal disease, smoking habits, drug history) and physical examination, including taking the participants' height and weight (Table 1). Additionally, routine laboratory investigations as recommended in the 2018 European Society of Cardiology/European Society of Hypertension guidelines for the management of arterial hypertension[13] were done, including laboratory parameters such as serum glucose, lipids, and creatinine.

Table 1. Baseline Characteristics of Study Participants

Baseline characteristics	
Number of participants	2367
Men/women	1290 (54.5%)/1077 (45.5%)
Age, y	49.5 (10–104, SD 16.1)
Height, cm	168 (140–200, SD 10.1)
Weight, kg	77 (42–187, SD16.5)
Body mass index, kg/m^2	27.3 (SD 5.9)
Current smoker, n (%)	19.7
Diabetes, n (%)	9.0
Coronary artery disease, n (%)	4.8
Kidney disease, n (%)	4.5
Prior stroke, n (%)	1.7
Fasting glucose, mmol/L	5.50 (SD 1.39)
Serum creatinine, µmol/L	79.6 (SD 41.6)
Total cholesterol, mmol/L	5.20 (SD 1.17)
Low-density lipoprotein cholesterol, mmol/L	3.13 (SD 0.96)
Brachial office SBP, mm Hg	137 (SD 17.7)
Brachial office DBP, mm Hg	89 (SD 12.9)
Brachial 24-h SBP, mm Hg	126 (SD 12.7)
Brachial 24-h DBP, mm Hg	80 (SD 10.5)
Left ventricular mass index, g/m^2	89.9 (SD 29.1)
Presence of left ventricular hypertrophy, %	24.1
Normotensive without antihypertensive drugs, %	29.3
Treated for hypertension, %	31.3
Angiotensin-converting enzyme inhibitor, %	12.4%
Angiotensin receptor blocker, %	11.4%
Calcium channel blocker, %	10.4%
Beta blocker, %	10.1%
Centrally acting antihypertensive, %	1.0%
Alpha blocker, %	0.8%

Values are number of participants (%), mean±SD, or mean (range, SD). Data were available in 1832 participants for current smoking, 1571 for diabetes, 1576 for coronary artery disease, 1571 for renal disease, 1568 for prior stroke, 1890 for fasting glucose, 1798 for serum creatinine, 1877 for total cholesterol, 1771 for low-density lipoprotein cholesterol, and in 1847 for antihypertensive medication. DBP indicates diastolic blood pressure; and SBP, systolic blood pressure.

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Blood Pressure Measurements

Brachial office BP was measured with validated automated oscillometric upper-arm cuff devices in the morning (08.30–12.30) after 5-minute rest under controlled room temperature (22–25 °C) on both arms. The mean of 2 consecutive measurements on the arm with the higher BP was recorded for analysis. Using the commercially available oscillometric brachial cuff–based sphygmomanometer (Mobil-O-Graph NG; I.E.M., Stolberg, Germany), 24-hour ambulatory BP monitoring was performed in all patients during regular daily life, following recent recommendations.[14] cSBP was determined from brachial waveforms, as recorded with the cuff at the level of DBP, calibrated with MAP/DBP (cSBP C2) or SBP/DBP (cSBP C1), and processed with the ARCSolver transfer function. The device has been validated for measurement of brachial BP.[15] Furthermore, the system has been compared against a common tonometric method[16,17] and against invasive pressures, recorded with fluid-filled or gold-standard solid-state pressure sensor-tipped catheters (Millar Instruments, Houston, TX),[18–20] with the majority of results supporting the validity of cSBP, albeit according to criteria established before the current Association for Research into Arterial Structure and Physiology recommendations.[21] Importantly, in the invasive studies, MAP/DBP calibration (C2) resulted in values for cSBP that were closer to invasive cSBP, compared with SBP/DBP calibration (C1).[4]

In the absence of patient diaries for the entire cohort, and similar to a previous publication of this database,[22] nighttime/daytime difference (dipping) was defined as nighttime (01:00–06:00) minus daytime (09:00–21:00) values, expressed as a percentage of daytime SBP.

Echocardiography

All echocardiographic examinations were performed by experienced cardiologists. They followed the recommendations of the American Society of Echocardiography and the European Association of Cardiovascular Imaging.[23] For measurement of LVM, chamber dimensions and wall thicknesses were acquired from the parasternal long- and short-axis views using targeted m-mode echocardiography at the level of the mitral valve leaflet tips at end diastole, with the m-mode cursor positioned perpendicular to the septum and the left ventricular posterior wall. LVM was calculated according to the corrected Devereux formula: LVM (g)=0.8×1.04×{[LV end diastolic diameter (cm)+interventricular septum+posterior wall thickness (cm)]^3–LV end diastolic diameter 3 (cm)}+0.6.[24] In adults, LVH was defined according to the 2018 European Society of Cardiology/European Society of Hypertension guidelines for the management of arterial hypertension[13] with LVM indexed to body surface area, as >115 g/m^2 in men and >95 g/m^2 in women. Body surface area was calculated using the DuBois formula: body surface area(m^2)=0.007184×height (cm) 0.725×weight (kg)$^{0.425}$. In children and adolescents, LVH was defined according to Khoury et al. as >40 g/m^2 in girls and >45 g/m^2 (ie, >95th percentile).[25]

Other Outcomes

Kidney function was quantified, using Chronic Kidney Disease Epidemiology Collaboration formula (estimated glomerular filtration rate=142×min (serum creatinine/κ,1)$^{\alpha}$×max(serum creatinine /κ,1)$^{-1.2}$×0.9938 age×1.012 [if female])[26] in adults and bedside Schwartz formula (estimated glomerular filtration rate =0.413×[height/serum creatinine])[27] in children and adolescents. Kidney function was classified according to Kidney Disease Improving Global Outcomes guidelines.[28]

Statistical Analysis

Normally distributed values are shown as mean±SD, and nonnormally distributed values are shown as median (interquartile range). The associations between LVMi and BPs (24-hour, daytime, nighttime, dipping) were assessed, using Pearson's correlation coefficients, and visualized using scatterplots (dispersion plots) with fitted regression lines and confidence bands. Differences in the strengths of association between BPs and LVMi were compared by z statistics for comparison of correlations within a single sample. A z-test on Fisher z-transformed correlation coefficients was used. Analysis was performed, stratified by sex, age groups (10–29, 30–39, 40–49, 50–59, 60–69, and 70–104 years, respectively), BMI (lean: BMI ≤24.9 kg/m^2, overweight: BMI 25–29.9 kg/m^2, and obese: ≥30 kg/m^2, respectively), and use of antihypertensive drugs. Multiple regression analysis with LVMi as outcome variable, including various SBPs and age, sex, BMI, and use of antihypertensives as covariates was performed, with additional analyses including smoking status and diabetes.

To assess potential nonlinear associations between SBPs and the continuous outcome LVMi, we modeled the relationship using restricted cubic splines fitting piecewise cubic functions and imposing linearity beyond the boundary knots using 2 R approaches (ie, rms and splines package, respectively). In the former case, Wald chi-square test was used to assess the additional contribution of the nonlinear spline components beyond the linear term; in the latter one, likelihood ratio test to compare to simple linear models.

Receiver-operating curve analysis was used to test the ability of SBPs to identify LVH. Areas under the curves were compared according to DeLong et al.[29]

In additional analysis, we investigated the association between BP phenotypes, based on brachial versus

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central 24-hour SBP, and LVMi. Hypertension phenotypes were defined according to average 24-hour brachial BP as normotension, isolated systolic (ISH) and diastolic (IDH) hypertension, and systolic-diastolic hypertension (SDH), using brachial systolic and diastolic threshold at 130 and 80 mm Hg, respectively. Brachial BP phenotypes were further subdivided based on 24-hour cSBP C2 (threshold for central hypertension was 135 mm Hg).[22] We fitted a 2-way ANCOVA (linear model) with LVMi as the outcome, brachial BP phenotype (4 levels) and central status (cSBP normal/elevated) as fixed factors, their interaction, and adjustment for age and sex. Adjusted marginal means with 95% CIs were obtained for each of the 8 factor-level combinations using emmeans. Post hoc contrasts compared discordant versus concordant status within each brachial BP phenotype. For the concordance analysis, we defined "concordant" a priori as cSBP normal for phenotypes without an elevated brachial systolic component (normotension, IDH) and cSBP elevated for phenotypes with an elevated brachial systolic component (ISH, SDH). Within each phenotype, we estimated the adjusted difference in LVMi between discordant and concordant central status using linear contrasts of estimated marginal means. Hypotheses were evaluated with Wald *t* tests and 95% CIs from the *t* distribution.

A 2-sided $P<0.05$ was considered to indicate statistical significance and all analyses were adjusted for multiple testing according to the method of Bonferroni.

All analyses were performed using MedCalc software version 23.0.9 (MedCalc Software Ltd, Ostend, Belgium) and R (R Foundation for Statistical Computing, Vienna, Austria; version 4.4.1).

RESULTS

In total, 2367 participants were included, 45.5% were women, average age 49.5 years (range 10–104 years), 29.3% were normotensive without antihypertensive drugs, 31.3% treated for hypertension. From these study participants, >120 000 individual BP measurements were acquired. Average brachial office BP was 137/89 mm Hg, average brachial 24-hour BP was 126/80 mm Hg. Average LVMi was 89.9 g/m^2, and 24.1% of the study participants had LVH (Table 1).

Correlations Between Blood Pressures and Left Ventricular Mass Index

All BPs were significantly associated with LVMi. Pearson's correlation coefficients for LVMi and office bSBP, 24-hour bSBP, 24-hour cSBP C1, and 24-hour cSBP C2 were 0.20, 0.31, 0.29, and 0.38, respectively (Figure 1). All 24-hour measurements showed significantly closer association with LVMi than office measurements, C2 calibrated cSBP measurements showed significantly closer association with LVMi than brachial and C1 calibrated cSBP measurements, and finally C1 calibrated cSBP measurements were not significantly closer correlated with LVMi than brachial measurements. These results were consistent across the following subgroups: in men and in women; in individuals who were lean, overweight, and obese; and in participants with or without antihypertensive medications (Figure S2A through S2G). We also analyzed the association of LVMi and different SBPs among different age groups and observed consistent results (Figure 2) regarding the superiority of 24-hour cSBP C2. Moreover, the strength of association between all corresponding SBPs and LVMi was stronger in younger patients than in older patients (P value <0.005 for comparison between age group 10 to 29/30 to 39 and age group 70 to 104, respectively). Complementary multiple regression analysis confirmed the results, with partial rs of different SBPs and coefficients of determination R^2 of the models being highest for cSBP C2 (Table S3). Additionally, we analyzed the association of LVMi and diurnal variations of bSBP, cSBP C1, and cSBP C2. We found that, consistent with the 24-hour results, for daytime as well as for nighttime measurements, cSBP C2 showed the closest association with LVMi. There was no association between any of the BP measurements and dipping (Figure S3).

Additional analyses yielded consistent results regarding the absence of nonlinear associations between different SBPs and LVMi (all nonsignificant; data not shown).

Correlations Between Blood Pressures and Kidney Function as Another Index of Hypertension-Mediated Organ Damage

We observed higher values of all SBPs with worse kidney function. Based on Kruskal–Wallis–ANOVA, the numerical relationship between kidney function and SBPs was statistically significant only for 24-hour cSBP C2 (Figure S4).

Receiver-Operating Curve Analysis for Prediction of LVH

In receiver-operating curve analysis, all SBPs were significant predictors of LVH. Areas under the curve were 0.622, 0.654, 0.649, and 0.680 for office bSBP, 24-hour bSBP, 24-hour cSBP C1, and 24-hour cSBP C2 respectively (Table 2, Figure 3). The area under the curve for 24-hour cSBP C2 was significantly larger, as compared with their bSBP and cSBP C1 counterparts ($P<0.0001$ for bSBP, $P=0.001$ for cSBP C1, respectively).

In addition to the different SBPs, age, BMI, sex, and taking antihypertensives (as a proxy to the presence of hypertension) were independently associated with

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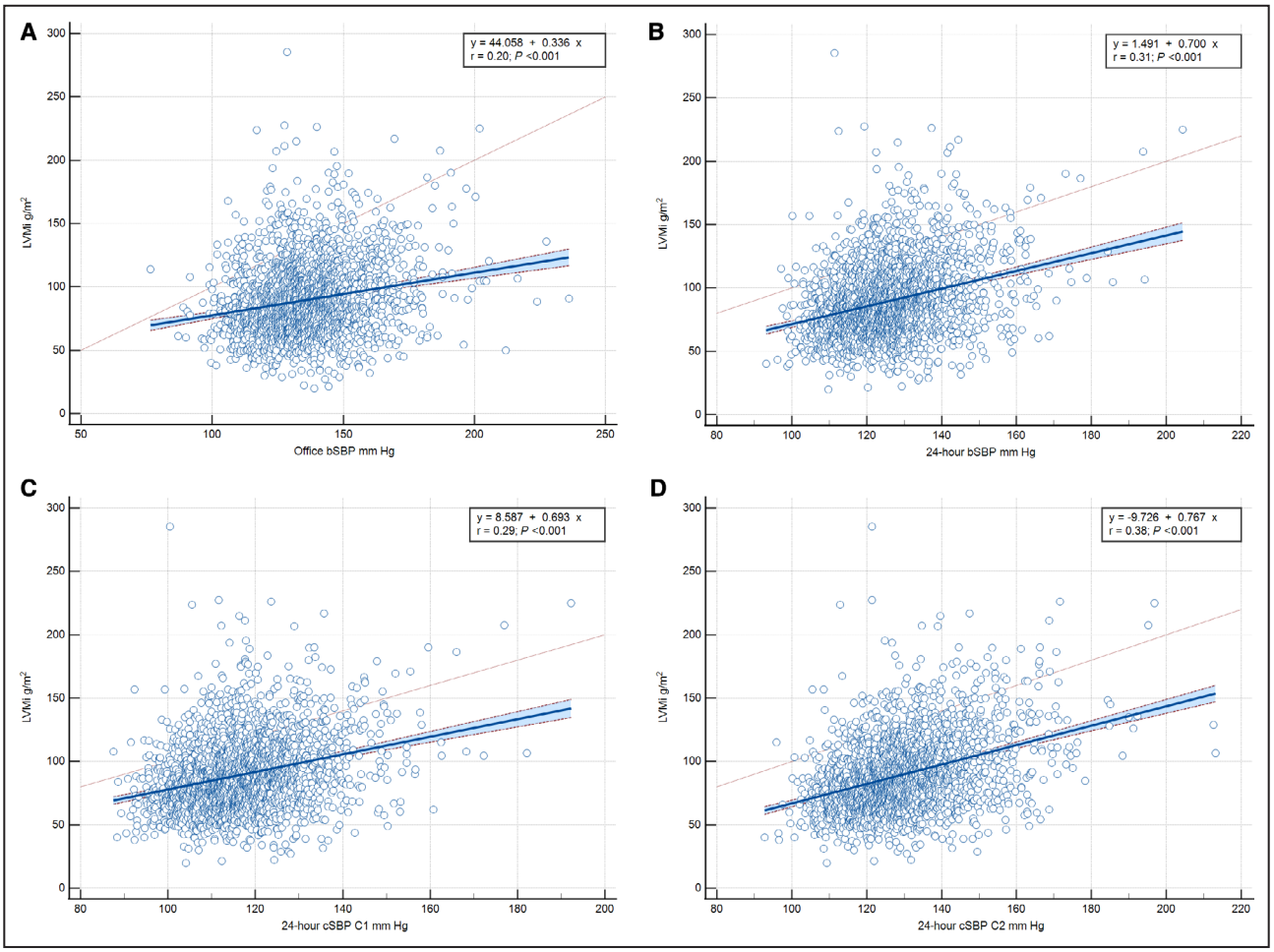


Figure 1. Scatterplots with fitted regression lines illustrating the relationship between left ventricular mass index and different systolic blood pressure measurements.
Based on z-statistics, all 24-h SBPs showed significantly closer association with LVMi than office SBP (P<0.0001 for all comparisons with office bSBP) (**A**), and cSBP C2 showed significantly closer association with LVMi than 24-h bSBP and 24-h cSBP C1 (**B**) (P<0.0001 for both comparisons, respectively). bSBP indicates brachial systolic blood pressure; cSBP C1, central systolic blood pressure calibrated using systolic/diastolic pressure; cSBP C2, central systolic blood pressure calibrated using mean/diastolic pressure; and LVMi, left ventricular mass index.

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LVMi (Table S3). In receiver operating curve analysis, the results of all multiple regression models were significant predictors of LVH. The area under the curve for results of models including 24-hour cSBP C2 was significantly larger, as compared with their bSBP, 24-hour bSBP, and 24-hour cSBP C1 counterparts (P<0.0001 for all comparisons, respectively) (Figure S5).

Results were similar, when diabetes and smoking status, which were not available in all participants, were added to the models (Tables S4 and S5).

Blood Pressure Phenotypes and Association With LVMi

Based on 24-hour brachial BP, 44.3%, 6.9%, 21.8%, and 27.0% of participants were classified as having normotension, ISH, IDH, and SDH, respectively (Table 3). The fraction of discordantly elevated 24-hour cSBP C2 (≥135 mm Hg) in brachial normotension and IDH was 10.6% and 5.0%, respectively, and the fraction of discordantly normal 24-hour cSBP C2 in brachial ISH and SDH was 14.7% and 28.1%, respectively. Sex differences were most striking in brachial normotension with elevated cSBP C2 (male predominance), and brachial ISH with normal cSBP C2 (female predominance). LVMi was higher in all brachial BP phenotypes, if cSBP C2 was elevated (Table 3), with statistically significant differences in brachial normotension and brachial SDH.

In a 2-way ANCOVA with LVMi as the dependent variable, brachial BP phenotype (4 levels) and cSBP status (normal/elevated) as factors, and age and sex as covariates, both brachial phenotype (P=0.0045) and cSBP status (P<0.001) were independently associated with LVMi. The interaction between brachial phenotype and cSBP status was not significant (P=0.57),

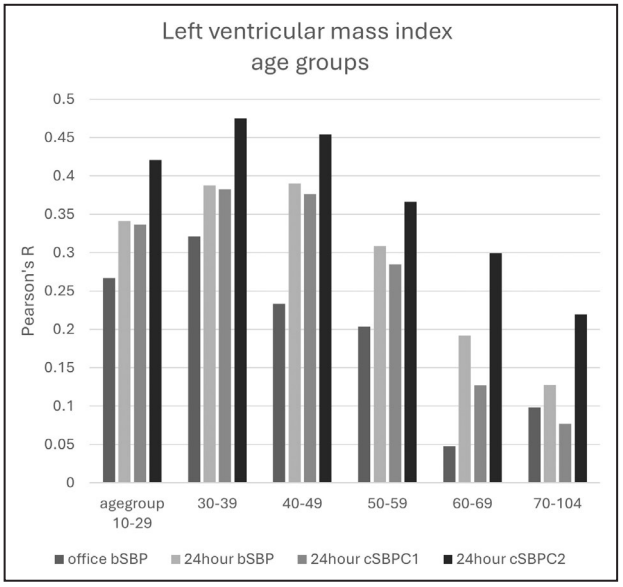


Figure 2. Age-specific correlation of left ventricular mass index with different blood pressure measurements across age groups.
cSBP C1 indicates central systolic blood pressure calibrated using systolic/diastolic pressure; cSBP C2, central systolic blood pressure calibrated using mean/diastolic pressure; and LVMi, left ventricular mass index.

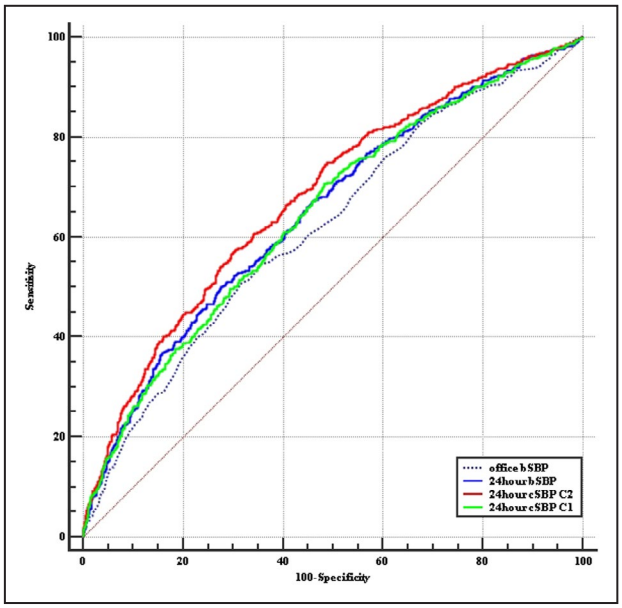


Figure 3. Receiver-operating characteristics curves for different systolic blood pressure measurements in predicting left ventricular hypertrophy, showing AUC—comparisons.
The AUC for 24-h cSBP C2 was significantly larger, as compared with bSBP and cSBP C1 counterparts (P<0.0001 and 0.001, with P values derived with the method of DeLong et al.[29]). AUC indicates area under the curve; bSBP, brachial systolic blood pressure; cSBP C1, central systolic blood pressure calibrated using systolic/diastolic pressure; and cSBP C2, central systolic blood pressure calibrated using mean/diastolic pressure.

indicating that the relationship between cSBP and LVMi did not differ by brachial phenotype (Figure S6).

Within-phenotype comparisons of adjusted LVMi showed that, among participants with normotension, discordant cSBP status (ie cSBP C2 elevated) was associated with higher LVMi (Δ=12.1 g/m^2; 95% CI 6.8–17.4; P=7.45×10^{-6}). Likewise, among those with SDH, discordance (ie, cSBP C2 not elevated) was associated with lower LVMi (Δ=–11.4 g/m^2 [95% CI, –16.1 to –6.82]; P=1.25×10^{-6}). Differences were not statistically significant for IDH (Δ=7.91 g/m^2 [95% CI, –2.58 to 18.4]; P=0.139) or ISH (Δ=–3.80 g/m^2 [95% CI, –15.3 to 7.73]; P=0.518). Positive Δ indicates higher LVMi in the discordant group relative to the phenotype-specific concordant group (concordant defined a priori as cSBP C2 normal for NT/IDH and cSBP C2 elevated for ISH/SDH) (Figure S7).

DISCUSSION

In our international multicenter study, we investigated the relationship of office and 24-hour bSBP and cSBP with LVMi and LVH. Using a large data pool of >2300 study participants, we observed the strongest associations with cSBP, but only for oscillometrically measured MAP/DBP calibration (cSBP C2). Moreover, detection of LVH was best for cSBP C2, and in all brachial BP—based phenotypes, LVMi was dependent on cSBP C2 status. These findings were supported by similar results from analysis for associations with kidney function, another measure of hypertension-mediated organ damage (HMOD).

Table 2. Area Under the Curve and 95% CI Values for Different SBP Measurement Methods in Predicting Left Ventricular Hypertrophy and Statistical Significance (P Values) for Comparisons Among SBP Measurement Methods

	AUC	95% CI	P value vs office bSBP	P value vs 24-h bSBP	P value vs 24-h cSBP C1	P value vs 24-h cSBP C2
Office bSBP	0.622	0.602–0.642		P=0.04	P=0.13	P<0.0001
24 h bSBP	0.654	0.634–0.673	P=0.04		P=1	P<0.0001
24 h cSBP C1	0.649	0.629–0.668	P=0.13	P=1		P=0.001
24 h cSBP C2	0.680	0.660–0.699	P<0.0001	P<0.0001	P=0.001	

P values were adjusted for multiple testing according to the method of Bonferroni. AUC indicates area under the curve; bSBP, brachial systolic blood pressure; cSBP, central systolic blood pressure; and SBP, systolic blood pressure.

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Table 3. Characteristics and Central Systolic Blood Pressure Categories by Brachial 24-Hour Blood Pressure Phenotypes and Their Association With Left Ventricular Mass Index

Brachial phenotype	Central SBP C2	No.	Percentage within phenotype	Age, y mean±SD	Percentage of men	Left ventricular mass index
Normotension	Normal	938	89.4	48.2 (17.8)	43.7	83.2 (81.5–85.0)
	Elevated	111	10.6	49.7 (23.6)	73.9	95.3 (90.4–100.3)
Isolated diastolic hypertension	Normal	491	95	48.3 (11.4)	65.8	84.8 (82.4–87.2)
	Elevated	26	5	53.5 (13.6)	61.5	92.7 (82.5–102.9)
Isolated systolic hypertension	Normal	24	14.7	49.1 (18.8)	37.5	94.1 (83.5–104.8)
	Elevated	139	85.3	56.6 (20.2)	45.3	97.9 (93.5–102.4)
Systolic-diastolic hypertension	Normal	179	28.1	47.6 (12.0)	50.3	90.1 (86.2–93.9)
	Elevated	459	71.9	51.9 (13.7)	64.9	101.5 (99.0–104.0)
Overall		2367				

Central SBP C2 indicates central systolic blood pressure calibrated using mean/diastolic pressure.

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As an important HMOD, LVH has been linked with cardiovascular events,[24] and its regression has been shown to be associated with improved outcomes.[30] Therefore, the relationship with LVMi/LVH may provide important initial clues for the relevance of a novel BP measurement (as cSBP in our case), which later need to be substantiated by the relationship with cardiovascular events, the availability of specific treatments, and the most important proof of concept, that is, the reduction in cardiovascular events by an intervention strategy based on the BP under investigation.

Initial studies,[9,31] summarized 10 years ago,[2] have predominantly shown a closer association between HMOD and cSBP, as compared with bSBP, establishing the narrative that cSBP is the BP actually "seen" by the heart (and other vital organs) and giving rise to large collaborative projects, for instance to establish reference values.[3] Moreover, early longitudinal studies[11,12] seemingly established the superior prognostic value of cSBP over bSBP. These studies were contradicted by other studies, showing little if any advantage of central over brachial pressures with respect to HMOD,[32] even if investigated longitudinally,[33] and with cardiovascular outcomes.[34]

A closer look revealed important technical details regarding the measurement of brachial and central BP, relevant to the research question, and detailed in an Association for Research into Arterial Structure and Physiology position paper on the validation of devices to assess cSBP.[21] Briefly, whereas noninvasive brachial cuff-based measurement is the gold standard for BP measurement in clinical routine due to the overwhelming amount of outcome data generated with this method, this measurement underestimates "true" invasive bSBP.[35] As noninvasive assessment of cSBP needs a noninvasive BP for calibration of waveforms, the use of bSBP (and DBP) imperatively leads to underestimation of cSBP. This might be overcome with the use of oscillometrically measured MAP and DBP for calibration, if MAP can be measured independently and more reliably than bSBP, which may be the case for some (but not all) oscillometric devices.[19] The theoretical reason is that MAP can be directly assessed as the maximum pulsations during cuff deflations, if the oscillometric method is used.[36]

Along these lines, several studies from Greece (SAFAR [Non-Invasive Aortic Ambulatory Blood Pressure Monitoring for the Detection of Tarrget Organ Damage] study),[37] Japan,[20] and a smaller multicenter study[38] have shown that oscillometrically measured MAP/DBP calibrated cSBP (so-called cSBP C2) shows a closer agreement with classical HMOD, as compared with bSBP, with similar trends in studies from Spain[12] and Greece (adolescents).[39] Furthermore, a closer association between coronary atherosclerosis[20] as well as diastolic dysfunction[40] and cSBP C2, as compared with bSBP, was observed.[37] Finally, also the superior prognostic value of cSBP for cardiovascular events was observed only for the C2 calibrated cSBP.[41] In our study, these results are confirmed in the largest sample so far, and extended to meaningful subgroups, with consistent results in men and women; participants across all age groups starting from childhood and adolescence; in individuals who are lean, overweight, and obese; and in those with and without antihypertensive drugs.

Interestingly, the correlation between LVMi and all SBPs was better in younger than in older people. This is in line with a systematic review and meta-analysis by Kollias et al., who investigated the association of central versus brachial BP with HMOD.[10] A possible explanation may be the fact that central-to-peripheral SBP amplification is higher in younger people.[3] Furthermore, in a study using invasively measured cSBP and central pulse pressure (cPP), Rhee et al. found significant relationships between cSBP/cPP and LVMi only in patients below the age of 50 years,[42] again consistent with our own findings.

Discordant BP classification based on bSBP versus cSBP is not uncommon[43,44] and has been observed in the IDCARS (International Database of Central Arterial Properties for Risk Stratification) meta-analysis (5576 patients) in 8.2% of the study population. The classification was based on cSBP C1, which is closely correlated with bSBP, and thus may give a much lower number of cross-classified patients, as compared in our C2-calibration based analysis (which led to cross-classification of 14.4% of participants). Of note, C1-based cross-classified BP phenotypes in IDCARS showed prognostic significance in terms of cardiovascular event prediction. In line, our analysis on BP phenotypes also supports the idea that the measurement of cSBP likely adds to risk stratification in all brachial-BP based phenotypes.

Blood pressure-lowering drugs can have varying effects on bSBP and cSBP. Some antihypertensive medications, like beta blockers, in monotherapy or in combinations, tend to lower bSBP more effectively than cSBP.[45] This discrepancy arises because beta blockers reduce heart rate and alter wave reflection patterns, which can lead to a lesser reduction in cSBP compared with bSBP.[46] On the other hand, calcium channel blockers and angiotensin-converting enzyme inhibitors are more effective in reducing both brachial and cSBP equally. These differences highlight the importance of considering cSBP when assessing cardiovascular risk, as drugs that preferentially lower cSBP may provide greater protection against cardiovascular events.

Strengths and Limitations

The most important strength of our study is the large harmonized data set, collected by 21 centers all over the world, with >2300 study participants, who had their 24-hour BPs measured with the same model BP device. This is about 10 times as many participants as in most of the previous studies on this subject. Next, the possibility to switch the calibration mode post hoc provides a unique opportunity to investigate the value of different calibrations in the same sample. There are, however, also certain limitations to our study. Due to the cross-sectional design, we cannot assume causality for our findings. A further limitation relates to the method used to derive cSBP. The stronger association observed for cSBP C2 may partly reflect the accuracy of oscillometrically measured mean arterial pressure used for waveform calibration rather than central hemodynamics per se. Because cSBP C2 is mathematically derived from brachial waveforms calibrated with MAP and diastolic pressure, some degree of mathematical coupling and collinearity between the calibrated inputs and the derived central pressure cannot be excluded. Consequently, the observed superiority of cSBP C2 over bSBP may result from improved overall blood pressure estimation, or from the inclusion of MAP as a more stable pressure component rather than exclusively from central pressure physiology. Future studies incorporating invasive reference measurements or analytical approaches that disentangle calibration effects from waveform-derived central pressure characteristics are needed to clarify the relative contribution of these factors.

Finally, although LVMi is an established intermediate end point in hypertension, future longitudinal analyses and randomized trials still must test whether cSBP C2 predicts hard cardiovascular outcomes more accurately than bSBP and whether cSBP-guided treatment reduces further cardiovascular events.

CONCLUSIONS

Collectively, these data suggest that incorporating 24-hour central systolic pressure—particularly cSBP C2—could refine cardiovascular risk assessment beyond 24-hour brachial BP alone. Discordance between central and brachial pressures may reveal patients at risk of over- or undertreatment, if decisions rely solely on brachial readings, which has implications for future hypertension guidelines. Prospective studies are needed to determine whether cSBP C2-guided therapy further improves clinical outcomes; until then, 24-hour cSBP C2 appears to be a valuable marker of hypertension-mediated cardiac damage.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Tables S1–S5
Figures S1–S7
STROBE checklist

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