Central Pressure More Strongly Relates to Vascular Disease and Outcome Than Does Brachial Pressure

The Strong Heart Study

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Abstract—Brachial blood pressure is predictive of cardiovascular outcome; however central pressure may better represent the load imposed on the coronary and cerebral arteries and thereby bear a stronger relationship to vascular damage and prognosis. Relations of brachial and central pressures to carotid artery hypertrophy (intimal-medial thickness and vascular mass), extent of atherosclerosis (plaque score), and incident cardiovascular events were examined in the Strong Heart Study. Central pressures were calculated using radial applanation tonometry. Among 3520 participants, central and brachial pulse pressures were more strongly related to vascular hypertrophy and extent of atherosclerosis than were systolic pressures. Central pulse pressure was more strongly related to all 3 arterial measures than was brachial pulse pressure ($r=0.364$ versus 0.309 for plaque score; $P<0.001$ for comparison of Spearman correlation coefficient; $r=0.293$ versus 0.249 for intimal-medial thickness; $P<0.002$; $r=0.320$ versus 0.289 for vascular mass; $P<0.05$). Among the 2403 participants free of clinical cardiovascular disease at baseline, 319 suffered fatal or nonfatal cardiovascular events during mean follow-up of 4.8 ± 1.3 years. After adjustment for age, gender, current smoking, body mass index, cholesterol:HDL ratio, creatinine, fibrinogen, diabetes, and heart rate, central pulse pressure predicted cardiovascular events more strongly than brachial pulse pressure (hazards ratio=1.15 per 10 mm Hg, $\chi^2=13.4$, $P<0.001$ versus hazards ratio=1.10, $\chi^2=6.9$, $P=0.008$). In conclusion, noninvasively-determined central pulse pressure is more strongly related to vascular hypertrophy, extent of atherosclerosis, and cardiovascular events than is brachial blood pressure. These findings support prospective examination of use of central blood pressure as a treatment target in future trials. (***Hypertension*. 2007;50:197-203.**)

Key Words: blood pressure ■ arterial stiffness ■ atherosclerosis

Hypertension, defined as sustained elevation of brachial blood pressure, is a major risk factor for cardiovascular disease, and reduction of brachial blood pressure decreases cardiovascular events, particularly stroke.[1] Available evidence suggests a greater importance of lowering systolic than diastolic pressure.[2,3] In addition, pulse pressure predicts outcome[4–10] and may be more strongly related to cardiovascular events than systolic pressure,[6–8,11] depending on the age of the population studied.[12–14] Although antihypertensive agents differ in their ability to reduce pulse pressure,[15,16] the efficacy of targeting pulse pressure as a treatment goal has not been proven. Furthermore, pulse wave velocity, a measure of vascular stiffness, has been related to cardiovascular risk in hypertensive patients,[17,18] the elderly,[19,20] patients with end-stage renal disease,[21,22] and population-based samples.[23,24] Central aortic pressure can now be reliably determined by noninvasive techniques,[25] and potential evidence of greater prognostic importance of central aortic than brachial pressures has been obtained in treated hypertensive patients.[26] However, the applicability of this observation to population-based samples has not been assessed.

Although mean pressure is relatively similar in different large arteries,[27,28] central aortic and brachial systolic and pulse pressures may differ considerably based on pulse wave velocity, a direct measure of arterial stiffness that influences timing of reflected waves returning from the periphery to the central aorta.[29] The resultant amplification of brachial compared with central pulse pressure is most pronounced in young and nonhypertensive individuals.[29] Central aortic pressures should more accurately reflect loading conditions of the left ventricular myocardium, coronary arteries, and cerebral vasculature and thereby, in theory, better relate to cardiovascular target organ damage and to cardiovascular events than brachial pressures. Likewise, vascular stiffness may better summate chronic damage to blood vessels from aging, hypertension, and diabetes than brachial or even central aortic

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blood pressure. Thus the present study was undertaken to examine the relations of central and brachial blood pressures and conduit arterial stiffness to carotid artery hypertrophy and extent of atherosclerosis, signs of systemic atherosclerosis, and to cardiovascular events in the longitudinal population-based Strong Heart Study.

Methods

Study Population

The study population was drawn from the Strong Heart Study (SHS), a population-based longitudinal study of prevalent and incident cardiovascular disease in American Indians. Details of the study design have been previously published.[30] In brief, members of 13 American Indians tribes in Arizona, North and South Dakota, and Oklahoma were invited to participate in serial evaluations, including personal interview, physical examination, and ascertainment of cardiovascular risk factors and prevalent disease. The baseline examination of the SHS cohort was conducted from 1989 to 1991. The 3rd examination in 1996 to 1999, attended by 88% of surviving cohort members, added carotid ultrasonography and radial artery applanation tonometry to the study protocol. The 3943 participants in the 3rd SHS examination included 3197 members of the SHS cohort and 750 additional family members participating in the SHS pilot family study[31]; 3520 had complete information available regarding brachial and central blood pressures and carotid ultrasonography. Missing data were largely attributable to sonographer unavailability, machine malfunction, or inadequate quality (>5% variability) of radial waveforms. All 3520 participants who underwent carotid ultrasonography and radial applanation tonometry were included in cross-sectional analyses regarding the relations of central and brachial blood pressures to vascular hypertrophy and atherosclerosis. The 2403 of 3197 SHS cohort members who were free of clinically overt cardiovascular disease, including atrial fibrillation, at the 3rd SHS examination were considered in longitudinal analyses.

Blood was drawn at each examination after a 12-hour fast to determine total, low-density and high-density lipoprotein cholesterol, fasting plasma glucose, creatinine, and fibrinogen. Diabetes was defined by the American Diabetes Association criteria[32] as fasting plasma glucose ≥7.0 mmol/L (126 mg/dL) or treatment with insulin or oral hypoglycemic agents. Brachial blood pressure was measured in triplicate in the right arm by cuff and mercury sphygmomanometer after the participant had been resting in a seated position for 5 minutes; diastolic pressure was defined as the disappearance of Korotkoff sounds. The average of the last 2 measurements was used as brachial blood pressure. Pulse pressure was calculated as the difference between systolic and diastolic pressures. Hypertension was defined by Joint National Committee 7 criteria[33] as systolic pressure ≥140 mm Hg, diastolic pressure ≥90 mm Hg or current use of antihypertensive medication.

The occurrence of fatal and nonfatal cardiovascular events was ascertained during follow-up, as previously described.[34,35] Cardiovascular events were determined from medical records, autopsy reports, and informant interviews; all materials were independently reviewed by physician members of the SHS morbidity and mortality committees. Cardiovascular events and causes of death included myocardial infarction, coronary heart disease, sudden death, congestive heart failure, and stroke. Follow-up through December 2003 was 99.8% complete for mortality and 99.2% complete for morbid events. The Indian Health Service Institutional Review Board, Institutional Review Board of the participating institutions, and the participating tribes approved the study. Informed consent was obtained from all of the participants.

Carotid Ultrasonography

All participants underwent carotid ultrasonography using Acuson Sequoia machines equipped with 7 MHz vascular probes on the day of the study visit using a standardized protocol. In brief, the extracranial segments of the left and right carotid arteries were extensively scanned for the presence of discrete atherosclerotic plaque, defined as focal protrusion (intimal-medial thickening) with a thickness exceeding that of the surrounding wall by ≥50%. A plaque score was calculated by the number of left and right segments (common carotid, bulb, internal carotid, external carotid) containing plaque; thus plaque score ranged from 0 to 8. Intimal-medial thickness of the far wall of the distal common carotid artery was measured at end diastole on multiple cycles of M-mode images.[36] Intimal-medial thickness was never measured at the level of a plaque; intimal-medial thicknesses are presented as the average of the left and right values. Carotid cross-sectional area, a measure of vascular volume or mass, was calculated as previously described.[37] All ultrasound studies were performed by research sonographers and interpreted by a single highly experienced cardiologist who was blinded to the clinical characteristics of the participants.

Applanation Tonometry

Radial arterial pressure waveforms were obtained by applanation tonometry using a solid state high-fidelity external Millar transducer; central arterial waveforms and pressures were calculated by the use of the SphygmoCor device using a generalized transfer function (AtCor Medical, Sydney, Australia) and calibration using the brachial mean and diastolic pressures obtained immediately before the procedure. Orientation and pressure applied to the transducer were adjusted to optimize applanation of the artery between the transducer and the underlying tissue. Applanation tonometry has been validated to yield accurate estimates of intraarterial pulse pressure by comparison with simultaneous invasive pressure recordings.[25,38,39] Arterial stiffness was estimated from pressure-diameter relations of the common carotid artery. Minimum (end-diastolic) and maximum (peak-systolic) diameters were obtained from carotid ultrasonography performed immediately before applanation tonometry with the position of the subject and ambient environment unchanged. The arterial stiffness index (beta)[40,41] was calculated according to the formula: $\ln(P_s/P_d)/([D_s-D_d]/D_d)$, where P_s and P_d are aortic systolic and diastolic pressures, respectively, and D_s and D_d are carotid systolic and diastolic diameters, respectively.

Statistical Analyses

Data are presented as percents or mean±standard deviation. Means of continuous variables were compared using Student's *t* test for independent samples or Mann–Whitney *U* test in the setting of skewed distribution. Brachial and central pressures were compared by paired sample *t* test. Categorical variables were compared by χ^2 analysis. Bivariate relations were analyzed using Spearman correlation coefficient. Differences in the strengths of association between central and brachial blood pressures and measures of carotid hypertrophy and atherosclerosis were compared by calculation of z statistics for comparison of correlations within a single sample. Relations of central and brachial blood pressures and arterial stiffness to cardiovascular events were determined in Cox regression analyses with adjustment for age, gender, current smoking, body mass index, total:HDL cholesterol ratio, serum creatinine, fibrinogen, diabetes, and heart rate in all models. Two-tailed $P<0.05$ was considered significant. Statistical analyses were performed with SPSS, version 11.0 (SPSS Inc).

Results

Study Population

Participants (n=3520) who underwent both radial artery applanation tonometry and carotid ultrasonography during the third SHS examination were used in cross-sectional analyses. The average age at the time of examination was 58±14 years (range from 18 to 88 years); 61% were women; body mass index was 31.5±6.8 kg/m^2. Hypertension was present in 48.3% of the population, 49.0% of women and 47.2% of men ($P=0.29$); 70% of hypertensive participants took antihypertensive medications at the time of study. Diabetes mellitus was present in 46.5% of women and 38.1%

TABLE 1. Carotid Ultrasound and Blood Pressure Findings in the Entire Population

Variable	Mean±SD
Intimal-medial thickness, mm	0.72±0.19
Mass, mm^2	15.35±4.56
Plaque, %	56.2
Plaque score, n	1.33±1.61
Brachial systolic pressure, mm Hg	130±19
Brachial pulse pressure, mm Hg	55±17
Central systolic pressure, mm Hg	120±19
Central pulse pressure, mm Hg	41±16
Arterial stiffness index	4.58±2.45

of men ($P<0.001$); 35.3% of men and 25.2% of women were active smokers ($P<0.001$). Carotid ultrasound findings and brachial and central blood pressures are presented in Table 1. Carotid atherosclerosis was present in 53% of women and 60% of men ($P<0.001$). As expected, brachial systolic and pulse pressures were higher than their central aortic counterparts ($P<0.001$ for both comparisons). The ratio of central aortic to brachial pulse pressure increased with age ($r=0.156$, $P<0.001$), consistent with the known diminution of pulse pressure amplification with aging.

Relations of Central Aortic and Brachial Blood Pressures to Carotid Hypertrophy and Atherosclerosis

The relations of central aortic and brachial blood pressures to carotid artery hypertrophy and extent of atherosclerosis (plaque score) are presented in Table 2. The strengths of relations were significantly higher for pulse pressure than for systolic pressure (with the exception of central pressures and vascular mass) and significantly higher for central aortic as opposed to brachial pressures. In addition, the arterial stiffness index was strongly related to intimal-medial thickness, vascular mass, and plaque score, all $P<0.001$. Furthermore, arterial stiffness was more strongly related to carotid hypertrophy and plaque than was brachial systolic pressure and to extent of atherosclerosis than was brachial pulse pressure.

Relations of Central Aortic and Brachial Blood Pressures and Arterial Stiffness to Clinical Outcome

The 2403 participants who were free of overt cardiovascular disease at the third examination were followed for a mean of 4.8±1.3 years during which time 319 suffered incident cardiovascular events, including 67 fatal and 252 nonfatal events (58 myocardial infarction, 54 stroke, 64 congestive heart failure, 120 coronary heart disease, 23 sudden and other cardiovascular deaths). Demographic and cardiovascular disease risk factors in those who did and did not suffer events are compared in Table 3. The 2 groups differed significantly in all variables other than gender, body mass index, and prevalence of current smoking.

The relations of central aortic and brachial systolic and pulse pressures to outcome were examined in separate models, including age, gender, body mass index, current smoking, total:HDL cholesterol ratio, creatinine, fibrinogen, diabetes status, and heart rate (Table 4). All variables other than gender, body mass index, and total:HDL cholesterol ratio were significant predictors of outcome. Age, diabetes, and plasma creatinine were very strongly related to outcome. Pulse pressures strengthened the models in comparison to systolic pressures for both central and brachial pressures. Central pulse pressure was more strongly predictive of cardiovascular events than was brachial pulse pressure both before (hazards ratio [HR]=1.15 per 10 mm Hg, $\chi^2=13.4$, $P<0.001$ versus HR=1.10, $\chi^2=6.9$, $P=0.008$) and after (HR=1.14, $\chi^2=11.7$, $P=0.001$ versus HR=1.09, $\chi^2=5.8$, $P=0.016$) additional adjustment for the presence of carotid atherosclerosis. Furthermore, when both central and brachial pulse pressures were included in the model, brachial pulse pressure ceased to be significant. The arterial stiffness index was also independently related to outcome ($\chi^2=6.1$, $P=0.014$), even after inclusion of carotid atherosclerosis as a covariate ($\chi^2=5.6$, $P=0.018$).

TABLE 2. Relations of Central and Brachial Blood Pressures and Arterial Stiffness to Carotid Hypertrophy and Extent of Atherosclerosis*

Variable	Intimal-Medial Thickness	Vascular Mass	Plaque Score
Brachial SBP	0.196	0.264	0.221
Central SBP	0.257	0.317	0.288
Brachial PP	0.249	0.289	0.309
Central PP	0.293	0.320	0.364
Arterial stiffness index	0.252	0.329	0.353
P value, brachial PP vs brachial SBP†	<0.001	<0.02	<0.001
P value, central PP vs central SBP†	<0.001	ns	<0.001
P value, central vs brachial SBP†	<0.001	<0.001	<0.001
P value, central vs brachial PP†	<0.002	<0.05	<0.001
P value, arterial stiffness vs brachial SBP†	<0.005	<0.001	<0.001
P value, arterial stiffness vs brachial PP†	ns	ns	<0.02

SBP indicates systolic blood pressure; PP, pulse pressure; ns, not significant.

*All correlations $P<0.001$.

†Correlations compared by Z statistics.

TABLE 3. Comparison of Demographic Variables and Cardiovascular Disease Risk Factors in Participants Free of Prevalent Cardiovascular Disease at Baseline Subdivided According to Incident Cardiovascular Events During Follow-Up

Variable	No Events (n=2084)	Events (n=319)	*P* Value
Age, years	62.5±7.5	65.6±7.4	<0.001
Male gender, %	34.8	36.1	0.672
Body mass index, kg/m^2	31.4±6.6	30.8±6.4	0.128
Hypertension, %	50.3	63.0	<0.001
Diabetes mellitus, %	44.1	67.5	<0.001
Current smoking, %	27.5	28.3	0.777
Brachial SBP, mm Hg	131±19	135±23	<0.001
Brachial PP, mm Hg	56±16	62±20	<0.001
Central SBP, mm Hg	121±17	127±22	<0.001
Central PP, mm Hg	41±15	48±18	<0.001
Heart rate, bpm	69±11	71±12	<0.001
Total cholesterol/HDL cholesterol	4.7±1.5	5.0±1.7	<0.003
Creatinine, mg/dL	0.93±0.91	1.33±1.65	<0.001
Fibrinogen, mg/dL	380±119	413±143	<0.001

SBP indicates systolic blood pressure; PP, pulse pressure; HDL, high density lipoprotein.

Because aging lessens pulse pressure amplification and might diminish the predictive advantage of central compared with brachial pulse pressure, we repeated analyses in the younger (<62 years) and older (≥62 years) halves of the population. Aortic pulse pressure (HR=1.12, [CI 1.04 to 1.22], P=0.005), but not brachial pulse pressure (HR=1.06, [CI 0.98 to 1.14], P=0.145), remained predictive of outcome in the older half of the population with diminutions in both hazard ratios compared with the entire group. Hazards ratios for all blood pressures and arterial stiffness were uniformly increased in the younger half of the population compared with the entire group. Additional subgroup analyses based on gender did not substantially alter the findings.

Discussion

Our population-based study demonstrates stronger relations of central aortic than brachial blood pressure with the extent of carotid hypertrophy and atherosclerosis, thereby lending support to the hypothesis that central pressures more accurately reflect the loading conditions on the cerebral vasculature than do brachial pressures. More importantly, central aortic pulse pressure more strongly predicted cardiovascular outcome than did brachial pressures. Our study provides additional support for the greater importance of pulse pressure over systolic pressure[6–8,11–14] in predicting cardiovascular outcome over a broad age range, although the size of the population and number of events do not permit subgroup analyses in different age ranges. In addition, arterial stiffness was as strongly related to carotid hypertrophy and extent of atherosclerosis as was central aortic pulse pressure.

Many studies have examined the importance of brachial blood pressure in relation to target organ damage and clinical outcome. Far fewer studies have examined the relation of central blood pressure and arterial stiffness to preclinical and clinical disease, and very few studies have examined the relative importance of central and brachial blood pressures in their relations to cardiovascular target organ damage or events. Boutouyrie et al found a stronger relation of carotid pulse pressure (r=0.42, P<0.001) than brachial pulse pressure (r=0.27, P<0.001) to carotid intimal-medial thickness in 167 normotensive and hypertensive volunteers,[42] although statistical significance of differences in the strengths of relation was not tested. In a study of 114 men with documented coronary artery disease, the severity of coronary stenosis was independently related to carotid but not brachial systolic and pulse pressures.[43] In a small substudy of the

TABLE 4. Multivariable Cox Models of Relation of Traditional Risk Factors and Central and Brachial Blood Pressures to Cardiovascular Outcome

Variable	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)
Age, year	1.06 (1.04–1.07)*	1.05 (1.04–1.07)*	1.06 (1.04–1.07)*	1.05 (1.03–1.07)*	1.05 (1.04–1.07)*
Male gender	1.13 (0.87–1.45)	1.17 (0.91–1.52)	1.13 (0.88–1.46)	1.22 (0.94–1.58)	1.10 (0.83–1.45)
BMI, kg/m^2	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)
Smoking	1.45 (1.10–1.91)†	1.44 (1.09–1.89)†	1.42 (1.08–1.87)‡	1.39 (1.06–1.83)‡	1.37 (1.01–1.85)‡
Cholesterol:HDL	1.05 (0.98–1.13)	1.06 (0.99–1.13)	1.05 (0.98–1.13)	1.05 (0.98–1.13)	1.09 (1.01–1.18)‡
Creatinine, mg/dL	1.20 (1.12–1.28)*	1.18 (1.11–1.27)*	1.20 (1.12–1.28)*	1.18 (1.10–1.26)*	1.13 (1.03–1.23)‡
Fibrinogen, mg/dL	1.001 (1.000–1.002)†	1.001 (1.000–1.002)†	1.001 (1.000–1.002)†	1.001 (1.000–1.002)§	1.001 (1.000–1.002)‡
Diabetes mellitus	2.48 (1.91–3.22)*	2.44 (1.88–3.17)*	2.47 (1.91–3.21)*	2.41 (1.86–3.13)*	2.42 (1.838–3.22)*
Heart rate, bpm	1.012 (1.001–1.022)‡	1.013 (1.002–1.023)‡	1.013 (1.008–1.143)‡	1.012 (1.001–1.022)‡	1.013 (1.001–1.025)‡
Brachial SBP	1.08 (1.02–1.14)‡				
Brachial PP		1.10 (1.03–1.18)†			
Central SBP			1.07 (1.01–1.14)‡		
Central PP				1.15 (1.07–1.24)*	
Arterial stiffness					1.06 (1.01–1.11)‡

All blood pressures per 10 mm Hg.

BMI indicates body mass index; SBP, systolic blood pressure; PP, pulse pressure.

*P<0.001; †P<0.01; ‡P<0.05; §P<0.005.

pREterax in regression of Arterial Stiffness in a contrOlled double-bliNd (REASON) Project involving 52 hypertensive subjects in whom central blood pressure was determined using applanation tonometry, change in carotid pulse pressure but not brachial pulse pressure was associated with the greater reduction in left ventricular mass detected in the perindopril+indapamide arm as compared with the atenolol treatment arm.[44] Finally, carotid pulse pressure and aortofemoral pulse wave velocity, but not brachial pulse pressure, were independently related to all-cause mortality in patients with end-stage renal disease.[45]

In contrast, brachial pulse pressure but not central pulse pressure was independently related to cardiovascular events in 484 elderly (65 to 84 years old) female hypertensives participating in the Second Australian National Blood Pressure Study.[46] This finding is somewhat surprising given the similarity between brachial and central pulse pressures at baseline (85±17 versus 84±26 mm Hg, respectively), consistent with minimal pulse wave amplification in this elderly population. Likewise, systemic arterial compliance and the augmentation index were not related to outcome; more direct measures of arterial stiffness such as pulse wave velocity and the arterial stiffness index were not examined in the study.

The most important study to date to examine the relative importance of central and brachial blood pressures has been the Conduit Artery Function Evaluation (CAFE) study of the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) hypertension trial.[26] Although brachial blood pressure was reduced to a similar extent in both the atenolol±thiazide and amlodipine±perinopril arms of the CAFE study, central systolic and pulse pressures were reduced to a significantly greater extent by amlodipine-based treatment. Furthermore, both brachial and central pulse pressures were similarly related (χ^2=4.1 for both) to a post hoc–defined composite outcome (new cardiovascular events, cardiovascular procedures, renal impairment) independent of other risk factors. However, it is uncertain whether the more favorable outcome associated with the amlodipine-based arm in the overall ASCOT trial was related to the greater central blood pressure lowering with this regimen.

The findings of the present study, a longitudinal observational study, complement those of the CAFE study, a treatment trial of high-risk hypertensive patients. The number of subjects and mean age are comparable, whereas duration of follow-up was slightly longer in the present study. Similarly, the SHS cohort is high-risk given the high prevalence of diabetes. Fewer events occurred in the CAFE study than in the current study (225 in hypertensives without prevalent cardiovascular disease at baseline versus 319 in the SHS cohort). It is possible that fewer events in the CAFE study explain the comparability of central and brachial pulse pressures in predicting outcome, whereas in the present study, central aortic pulse pressure was a stronger predictor of outcome than was brachial pulse pressure. Alternatively, this difference in findings may relate to inherent differences in the designs and populations of the two studies. Interestingly, mean brachial pulse pressure in the current study (55 mm Hg) is comparable to that of the 2 arms in the CAFE study (55.3 mm Hg in the atenolol arm and 56.2 mm Hg in the amlodipine arm), whereas central pulse pressure was lower in the present study (41 mm Hg versus 46.4 mm Hg in the atenolol arm and 43.4 mm Hg in the amlodipine arm of the CAFE study), indicating a stronger separation between central and brachial pressures in the SHS cohort.

Potential limitations of the current study include the fact that 70% of hypertensive participants (33% of the entire population) were taking antihypertensive medications at the time of study. If anything, such use might be expected to dilute the impact of findings insofar as antihypertensive therapy lowers blood pressure, thereby lessening the significance of bivariate correlations, and improves outcome. Furthermore when antihypertensive medication use was added to the Cox regression models including atherosclerosis, only central aortic pulse pressure remained an independent predictor of outcome (P=0.004). Although not necessarily a limitation, our study was not a randomized treatment trial but rather a longitudinal observational study, including normotensive and hypertensive individuals, examining the relative prognostic importance of central and brachial blood pressures. The greater importance of central aortic pulse pressure and arterial stiffness in relation to target organ damage and outcome noted in the present study may not apply to blood pressure lowering trials, although similarity of data from the CAFE study suggest this might be the case. While our study population is limited to American Indians and results may not be applicable to other populations, the increasing prevalences of obesity and diabetes in the general US population suggest that our findings may be highly applicable to US public health.

In conclusion, noninvasively determined central aortic pulse pressure and arterial stiffness are more strongly related to vascular hypertrophy and extent of carotid atherosclerosis than is brachial pressure. Furthermore central pulse pressure better predicts outcome than does brachial pressure. These findings support the recent call for prospective examination of use of arterial stiffness and central blood pressure as treatment targets in future trials.[47]

Perspectives

Although central blood pressure more directly reflects the load on the heart and coronary and cerebral arteries than does brachial blood pressure and thereby should more directly relate to target organ damage and clinical cardiovascular disease, data to support this hypothesis have been lacking. The recently acquired ability to accurately measure central pressure using noninvasive techniques in population-based samples allows this theory to be tested. The present study provides direct support for the hypothesis that central pressure is more important than brachial pressure in predicting outcome in the Strong Heart Study, an observational study of prevalent and incident cardiovascular disease and their risk factors in American Indians. Whether treatment based on central as opposed to brachial blood pressure favorably alters cardiovascular risk remains to be determined.

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Disclosures

None.

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