Associations Between Echocardiographic Arterial Compliance and Incident Cardiovascular Disease in Blacks: The ARIC Study

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BACKGROUND
Systemic arterial compliance is sometimes derived by echocardiographic stroke volume to pulse pressure ratios. Few studies have assessed echocardiographic arterial compliance in blacks or its associations with explicit, rather than composite, cardiovascular disease (CVD) outcomes.

METHODS
We analyzed a subset (n = 1,887) of blacks (mean age = 59±6 years; 65% women) in the Atherosclerosis Risk in Communities study who were free of prevalent CVD and were imaged by echocardiography in 1993–1995. Arterial compliance was calculated by the aortic velocity time integral and brachial pulse pressure ratio (VTI/PP). Associations between VTI/PP and subsequent CVD (defined as first incident stroke, coronary event, or heart failure) were modeled by Cox regression after controlling for demographics, anthropometry, and cardiac risk factors. For comparison, CVD hazard ratios were also calculated for pulse pressure quartiles.

RESULTS
Over a mean follow-up of 13±4 years, 237 subjects (12%) developed coronary disease, 322 (16%) developed heart failure, and 180 (9%) experienced a stroke. Hazard ratios contrasting lowest with highest quartiles of VTI/PP were 2.3 (95% confidence interval (CI) = 1.7–3.1) for composite CVD, 2.1 (95% CI = 1.3–3.2) for coronary disease, 2.5 (95% CI = 1.7–3.6) for heart failure, and 2.7 (95% CI = 1.6–4.5) for stroke. Hazard ratios contrasting widest with narrowest pulse pressure quartiles were 1.7 (95% CI = 1.3–2.2) for composite CVD, 1.6 (95% CI = 1.0–2.4) for coronary heart disease, 1.8 (95% CI = 1.2–2.6) for heart failure, and 2.3 (95% CI = 1.3–3.9) for stroke.

CONCLUSIONS
In blacks, the VTI/PP ratio has stronger associations with both composite and individual CVD outcomes than does pulse pressure.

Keywords: arterial compliance; blood pressure; cardiovascular disease; echocardiography; hypertension.

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Systemic arterial compliance, the systolic to diastolic change in volume divided by the change in pressure, is a measure of vascular function that may be used to predict cardiovascular outcomes. The gold standard method of measurement, quantifying stroke volume and aortic pulse pressure (PP) invasively by thermodilution and catheter manometers, is a validated index of total arterial compliance.[1,2] However, noninvasive estimates of systemic arterial compliance can be derived by the ratio of echocardiographic stroke volume and brachial arterial PP (SV/PP).[2]

Although noninvasive estimates of arterial compliance are well suited for prospective screenings and longitudinal epidemiologic studies, most observations relating arterial compliance and cardiovascular disease (CVD) have been drawn from cross-sectional analyses. Age,[3,4] hypertension,[5] concentric left ventricular hypertrophy,[6] diastolic left ventricular dysfunction,[7–9] hypercholesterolemia,[10] fasting insulin,[7,11] and Framingham risk score[12] have all been associated with echocardiographic arterial compliance. Prospective analyses of echocardiographic arterial compliance have primarily been limited to Europeans but have predicted mortality in patients with diabetes[13] and fatal and nonfatal cardiovascular events in hypertensives[14,15] and in the general population.[8] To date, few studies have assessed echocardiographic systemic arterial compliance in blacks, and none have analyzed cardiovascular events as explicit, rather than composite, outcomes. We analyzed associations between echocardiographic arterial compliance and incident stroke, heart failure, and coronary events in a cohort of blacks free of prevalent CVD and followed prospectively by the Atherosclerosis Risk in Communities (ARIC) study. We also compared the risks of cardiovascular outcomes

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associated with arterial compliance with those associated with PP and examined model performances to compare the predictive validity of echocardiographic arterial compliance and PP models.

METHODS

Initiated in 1987, the ARIC study is an ongoing, prospective epidemiological cohort based on 4 US areas.[16] A population-based sample (n = 3,732) of blacks aged 45–65 years at the study onset was recruited from Jackson, Mississippi, with informed consent and approval by the University of Mississippi Institutional Review Board. Study retention at the Jackson site was excellent, with 94% of surviving participants completing annual surveys in 2010.

During the period 1993–1995, the Jackson site offered echocardiograms to all study participants. A total of 2,445 echocardiograms were performed by certified sonographers following a standardized protocol. After digitization from VHS tapes, all echocardiograms were analyzed and quantified by a single cardiologist. Quality assurance was ascertained by a random sample of repeat echocardiograms, and intra-sonographer correlation coefficients for each echocardiography variable were calculated.

Left ventricular and left atrial dimensions were quantified from 2-dimensional images in the parasternal long-axis view. Septal wall thickness (IVSTd), the left ventricular inner dimension (LVIDd), and the posterior wall thickness (PWTd) were measured at end-diastole. The left ventricular inner dimension was again measured at end-systole (LVIDs), as was the left atrial diameter. Left ventricular mass was determined by the following equation: LV mass = $0.8 \times 1.04 \times ((\text{LVIDd} + \text{PWTd} + \text{IVSTd})^3 - \text{LVIDd}^3)) + 0.6$g[17] and was indexed to body surface area. Relative wall thickness was calculated by (IVSTd + PWTd) / LVIDd. Fractional shortening, a simple measure of left ventricular function, was calculated by (LVIDd – LVIDs)/ LVIDd × 100. Left ventricular outflow tract (LVOT) diameters were measured in mid-systole, and the cross-sectional area (CSA) was quantified. The transmitral early (E) and late (A) diastolic flow velocities were assessed by pulse wave Doppler, and the E/A ratio was calculated as a measure of left ventricular diastolic function. Systolic flow through the LVOT was interrogated by pulse wave Doppler, and signals were traced by planimetry to calculate the velocity-time integral (VTI). The stroke volume to PP ratio was quantified by the following equation: SV/PP = (VTI × CSA)/PP, with PP derived by subtracting the diastolic from systolic blood pressure. Because 75% of echocardiograms were missing LVOT diameter measurements, a simplified echocardiographic arterial compliance index (VTI/PP) was calculated using the VTI as a surrogate for stroke volume.[18]

All clinical covariables, with the exception of creatinine, were ascertained the day of the echocardiogram. Seated blood pressures were measured by random-zero mercury manometers before imaging. Three measurements were made, with the average of the second and third recorded for analysis. Standardized, 12-lead electrocardiograms were performed and assigned a Minnesota code by the ARIC ECG Computing Center.[19] Fasting cholesterol and glucose were analyzed by ARIC Central Laboratories. Creatinine values were ascertained within 3 years of the echocardiogram, and glomerular filtration rate was estimated using the Chronic Kidney Disease Epidemiology Collaboration formula.[20]

CVD was determined at the baseline visit in 1987–1989 by self-report and validated computer algorithms,[21–23] and thereafter by clinical exams, annual surveys, and hospital surveillance. All survey responses and hospital discharge codes indicative of cardiovascular events were verified by physician review of the medical records. The diagnostic criteria and adjudication have previously been described..[24–26] For the purposes of our analysis, prevalent CVD was defined as stroke, heart failure, or coronary heart disease before the echocardiogram in 1993–1995, whereas incident CVD was classified by subsequent events. Coronary heart disease was considered myocardial infarction, coronary artery bypass graft, percutaneous coronary intervention, or electrocardiogram (ECG) suggestive of myocardial infarction. Stroke was defined by thrombotic, cardioembolic, or lacunar infarctions and subarachnoid or intracerebral hemorrhage. Heart failure diagnoses included both heart failure with preserved and reduced ejection fraction. Hypertension was defined by a systolic blood pressure >140 mm Hg, a diastolic blood pressure >90 mm Hg, or antihypertensive medication use. Diabetes was considered a fasting glucose ≥126 g/dl or use of diabetic medications.

Statistical analysis

All analyses were performed using SAS version 9.3 (SAS Institute, Cary, NC). Normality was verified by Komolgorov–Smirnov testing, Q–Q plots, skew, and kurtosis. Categorical variables were compared by χ^2 testing and continuous variables by analysis of variance. Unadjusted correlations between VTI/PP and continuous variables were assessed by Pearson correlation. Hazard ratios (HRs) for incident CVD associated with VTI/PP quartiles and PP quartiles were assessed using Cox regression, after controlling for age, sex, body mass index, smoking, total cholesterol, hypertension, diabetes, heart rate, and glomerular filtration rate. Adjusted HRs associated with VTI/PP quartiles and PP quartiles were also calculated individually for incident stroke, incident heart failure, and incident coronary disease. Proportional hazards assumptions were verified by plotting Martingale residuals and assessing deviations of observed suprema from 1,000 simulated paths by Kolmolgorov–Smirnov testing. No Cox models were found to violate proportional hazards. Model performances were assessed by the −2 log likelihood, a global measure of fit,[27] and by the c-index, an extension of the receiver operating characteristic c-statistic, which accounts for censoring.[28–30] A complete-case sensitivity analysis was performed among study participants with measured LVOT diameters to compare VTI/PP and SV/PP associations with CVD. Finally, in a subset analysis, the risk of composite CVD associated with previously determined cutpoints[13] of SV/PP indexed to body surface area (SVi/PP) was determined using Cox regression adjusting for age, sex, smoking, total cholesterol, hypertension, diabetes, heart rate, and glomerular filtration rate.

RESULTS

Of the 2,445 study subjects evaluated with echocardiography, 2,059 were free of prevalent CVD and had adequate image quality. Of these, 1,887 had measureable VTI/PP ratios. The mean age at the time of the echocardiogram was 59 ± 6 years, and the majority of study participants (65%) were women. Study participants were followed for a mean 13 ± 4 years. Over this time frame, 237 (12%) developed coronary heart disease, 322 (16%) developed heart failure, 180 (9%) experienced a stroke, and 505 (25%) died. A total of 544 (26%) developed incident CVD, when defined as a composite outcome of first incident coronary event, heart failure, or stroke.

The VTI/PP index was divided into true quartiles of increasing arterial compliance, as follows: Q1 <0.33, Q2 = 0.33–0.409, Q3 = 0.41–0.499, and Q4 ≥0.50. Similarly, PP values were divided into true quartiles of increasingly wider PP, as follows: Q1 <46 mm Hg, Q2 = 46–55.9 mm Hg, Q3 = 56–65.9 mm Hg, and Q4 ≥66 mm Hg. Clinical, demographic, and echocardiographic values stratified by VTI/PP quartiles are shown in Table 1 and Table 2.

In unadjusted analyses, increasing VTI/PP (indicating greater arterial compliance) was positively correlated with left ventricular fractional shortening and mitral Doppler E/A ratio and inversely correlated with age, blood pressure, left ventricular mass index, and heart rate. Significant correlations were not observed between VTI/PP and total cholesterol or between VTI/PP and glomerular filtration rate (Table 3).

Lower VTI/PP values (indicating decreased arterial compliance) were associated with poor prognoses. As shown by the

Table 1. Baseline (1993–1995) demographics and clinical characteristics of study participants from the Atherosclerosis Risk in Communities study Jackson site who were free of prevalent cardiovascular disease, stratified by velocity time integral and brachial pulse pressure ratio quartiles

Characteristic	Q1: <0.33 (n = 472)	Q2: 0.33–0.40 (n = 471)	Q3: 0.41–0.49 (n = 473)	Q4: ≥0.50 (n = 471)
Demographics				
Age, y, mean ± SD	60 ± 6	59 ± 6	58 ± 5	57 ± 5
Female sex, no. (%)	274 (59)	287 (62)	299 (64)	341 (73)
Medical history				
Hypertension, no. (%)	341 (73)	266 (57)	247 (53)	211 (45)
Diabetes, no. (%)	130 (28)	94 (20)	84 (18)	81 (17)
Body mass index, kg/m^2, mean ± SD	29 ± 6	30 ± 6	30 ± 6	31 ± 6
Current smoker, no. (%)	110 (24)	102 (22)	72 (15)	77 (16)
Clinical values, mean ± SD				
Systolic blood pressure, mm Hg	159 ± 19	144 ± 14	135 ± 14	123 ± 12
Diastolic blood pressure, mm Hg	86 ± 12	83 ± 10	82 ± 10	80 ± 9
Pulse pressure, mm Hg	73 ± 15	61 ± 10	53 ± 10	42 ± 9
Total cholesterol, mg/dl	207 ± 39	210 ± 40	205 ± 37	208 ± 41
Glomerular filtration rate, ml/min/1.73 m^3	68 ± 16	69 ± 14	71 ± 14	70 ± 12

All differences are statistically significant across quartiles at α = 0.05 level, except total cholesterol and glomerular filtration rate.

Table 2. Baseline (1993–1995) echocardiography values for study participants from the Atherosclerosis Risk in Communities study Jackson site who were free of prevalent cardiovascular disease, stratified by velocity time integral and brachial pulse pressure ratio quartiles

Echocardiography variables	Q1: <0.33 (n = 472)	Q2: 0.33–0.40 (n = 471)	Q3: 0.41–0.49 (n = 473)	Q4: ≥0.50 (n = 471)
Left ventricular mass index, g/m^2	134 ± 40	123 ± 30	117 ± 29	112 ± 26
Relative wall thickness	0.57 ± 0.14	0.56 ± 0.13	0.54 ± 0.12	0.56 ± 0.12
Fractional shortening, %	33 ± 9	34 ± 9	35 ± 8	36 ± 8
Left ventricular velocity time integral, cm	20 ± 4	22 ± 4	24 ± 4	25 ± 5
Left ventricular stroke volume, ml	70 ± 17	79 ± 19	81 ± 21	83 ± 19
Left atrium diameter, cm	3.4 ± 0.6	3.3 ± 0.5	3.3 ± 0.5	3.3 ± 0.5
Mitral E/A ratio	0.99 ± 0.28	1.02 ± 0.27	1.07 ± 0.27	1.09 ± 0.28
Heart rate, beats/min	69 ± 11	67 ± 10	66 ± 10	66 ± 10

All differences are statistically significant across quartiles at α = 0.05 level, except left atrium diameter and relative wall thickness.

Kaplan–Meier analysis, decrements of VTI/PP quartiles were progressively associated with a greater cumulative incidence of CVD (Figure 1). Associations remained robust in multivariable Cox regression models controlling for age, sex, body mass index, smoking, total cholesterol, hypertension, diabetes, heart rate, and glomerular filtration rate. Using the highest VTI/PP quartile as the reference, the lowest quartile was associated with an adjusted HR of incident CVD of 2.3 (95% confidence interval (CI) = 1.7–3.1). When cardiovascular events were analyzed separately, contrasts of the lowest with highest VTI/PP quartiles were significantly associated with adjusted HRs of incident coronary disease (HR = 2.1; 95% CI = 1.3–3.2), incident heart failure (HR = 2.5; 95% CI = 1.7–3.6), and stroke (HR = 2.7; 95% CI = 1.6–4.5). PP quartiles were likewise associated with CVD outcomes but only marginally associated with incident coronary disease. The HRs contrasting the widest with narrowest PP quartile were 1.7 (95% CI = 1.3–2.2) for composite CVD, 1.8 (95% CI = 1.2–2.6) for heart failure, 2.3 (95% CI = 1.3–3.9) for stroke, and 1.6 (95% CI = 1.0–2.4) for coronary heart disease (Table 4). Comparisons of composite CVD risks associated with VTI/PP and PP quartiles are shown in Figure 2. All cardiovascular outcomes modeled by VTI/PP resulted in a lower −2 log likelihood than models predicted by PP, indicating better global fit; however, c-index values did not statistically differ.

Table 3. Pearson correlations with continuous velocity time integral and brachial pulse pressure ratio

Variable	Correlation	*P* value
Age, years	−0.19	<0.0001
Systolic blood pressure, mm Hg	−0.67	<0.0001
Diastolic blood pressure, mm Hg	−0.20	<0.0001
Total cholesterol, mg/dl	−0.001	0.10
Glomerular filtration rate, ml/min/1.73 m^3	0.04	0.10
Body mass index, kg/m^2	0.08	0.0006
Left ventricular mass index, g/m^2	−0.25	<0.0001
Fractional shortening, %	0.13	<0.0001
Mitral E/A ratio	0.15	<0.0001
Heart rate, beats/min	−0.14	<0.0001

To compare VTI/PP with SV/PP, a more widely used index of echocardiographic arterial compliance, a complete-case sensitivity analysis was performed, using a subset (n = 519) of echocardiograms with measured LVOT diameters. The adjusted HRs of CVD contrasting lowest with highest quartiles were 1.8 (95% CI = 1.1–2.9) for VTI/PP and 1.7 (95% CI = 1.0–2.9) for SV/PP, showing close agreement between the 2 estimates of arterial compliance.

We also examined associations between incident CVD and SVi/PP dichotomized into low (<0.60) and preserved (≥0.60) arterial compliance. Low arterial compliance was significantly associated with incident CVD (HR = 1.5; 95% CI = 1.1–2.1). In this subset, we observed that 50 (10%) participants were classified with low arterial compliance despite

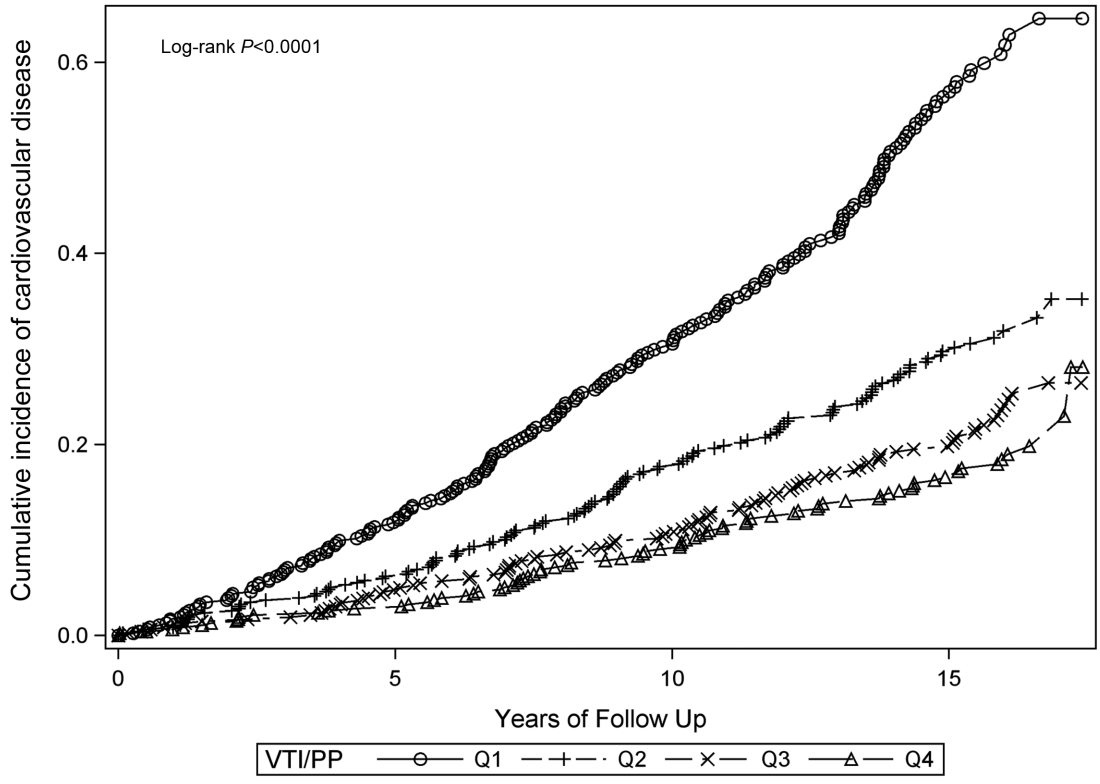

Figure 1. Kaplan–Meier analysis of velocity time integral and brachial pulse pressure ratio quartiles and cumulative incidence of composite cardiovascular disease from 1993 to 2010. Abbreviations: Q1, lowest arterial compliance; Q4, highest arterial compliance.

Table 4. Adjusted hazard ratios for incident coronary disease, heart failure, and stroke associated with VTI and brachial PP ratio quartiles and PP quartiles

Associations with VTI/PP quartiles	Hazard ratio (95% CI)
Incident coronary disease	
QTL 1, lowest arterial compliance	2.1 (1.3–3.2)*
QTL 2	1.4 (0.9–2.2)
QTL 3	1.1 (0.6–1.7)
QTL 4, highest arterial compliance	—
Linear trend, per 1-unit lower VTI/PP quartile	1.3 (1.1–1.5)*
Incident heart failure	
QTL 1, lowest arterial compliance	2.5 (1.7–3.6)*
QTL 2	2.0 (1.3–2.9)*
QTL 3	1.3 (0.9–2.0)
QTL 4, highest arterial compliance	—
Linear trend, per 1-unit lower VTI/PP quartile	1.4 (1.2–1.5)*
Incident stroke	
QTL 1, lowest arterial compliance	2.7 (1.6–4.5)*
QTL 2	1.2 (0.7–2.2)
QTL 3	1.7 (1.0–2.9)
QTL 4, highest arterial compliance	—
Linear trend, per 1-unit lower VTI/PP quartile	1.3 (1.1–1.6)*
Associations with PP quartiles	**Hazard ratio (95% CI)**
Incident coronary disease	
QTL 1, narrowest PP	—
QTL 2	0.8 (0.5–1.3)
QTL 3	1.4 (0.9–2.2)
QTL 4, widest PP	1.6 (1.0–2.4)
Linear trend, per 1-unit higher PP quartile	1.2 (1.1–1.4)*
Incident heart failure	
QTL 1, narrowest PP	—
QTL 2	1.0 (0.7–1.5)
QTL 3	1.4 (0.9–2.1)
QTL 4, widest PP	1.8 (1.2–2.6)*
Linear trend, per 1-unit higher PP quartile	1.2 (1.1–1.4)*
Incident stroke	
QTL 1, narrowest PP	—
QTL 2	1.1 (0.6–2.0)
QTL 3	1.7 (1.0–3.0)
QTL 4, widest PP	2.3 (1.3–3.9)*
Linear trend, per 1-unit higher PP quartile	1.4 (1.2–1.6)*

Adjusted models controlling for age, sex, body mass index, smoking, hypertension, diabetes, total cholesterol, glomerular filtration rate, and heart rate.

Abbreviations: CI, confidence interval; PP, pulse pressure; VTI, velocity time integral.

* Associations significant at α = 0.05 level

being normotensive, indicating the possibility of pseudonormalized blood pressure.

DISCUSSION

This is the first study to examine echocardiographic arterial compliance in an all black cohort and the first to analyze its associations with individual, rather than composite, CVD outcomes. In our study population of blacks free of prevalent CVD, we observed significant associations between VTI/PP and cardiovascular endpoints after adjusting for demographics, anthropometry, and cardiac risk factors. When analyzed as separate endpoints, incident coronary disease, heart failure, and stroke remained associated with VTI/PP quartiles. PP quartiles were likewise associated with CVD but with a lesser magnitude of effect and were only marginally associated with incident coronary heart disease.

As with other studies of echocardiographic arterial compliance, we noted correlations between VTI/PP and left ventricular remodeling and indices of diastolic dysfunction. In the Cardiovascular Health Study, which included echocardiograms from 1,215 study participants from the general US population, lower SV/PP was independently associated with concentric left ventricular geometry.[6] Lower SV/PP was likewise associated with a lower transmitral E/A ratio, an indicator of impaired left ventricular relaxation, in a general population of Swedish men.[8] Correlations of echocardiographic arterial compliance with measures of diastolic properties of the left ventricle have also been observed using strain rate echocardiography, a more sophisticated technique for assessing abnormal left ventricular relaxation and segmental diastolic dysfunction.[9] In our analysis of blacks free of prevalent CVD, lower VTI/PP was correlated with greater left ventricular mass index and decreased transmitral E/A ratios, suggesting a baseline relationship between arterial compliance and left ventricular hypertrophy and diastolic dysfunction.

After a prospective follow-up of approximately 13 years, we observed a higher incidence of CVD, as either a composite or separate outcome of heart failure, stroke, and coronary disease, in participants with lower VTI/PP values. This is consistent with results from a Swedish cohort of 470 men, which reported lower SV/PP associated with a composite outcome of fatal and nonfatal CVD after a mean follow up of 7 years.[8] Likewise, in a study of 294 hypertensive patients followed prospectively for 10 years, lower SV/PP (indexed to body surface area) and lower values for the %SV/PP (calculated as the ratio of measured to expected SV/PP derived by regression models) were associated with an increased risk of composite cardiovascular events and mortality.[14] However, these studies did not examine associations with individual CVD outcomes and were primarily white cohorts.

PP quartiles have previously been shown to predict CVD in epidemiological studies[31,32] and, not surprisingly, were associated with incident CVD in our analysis as well. However, we observed that VTI/PP quartiles were associated with an even greater incremental risk. An invasive study measuring PP by brachial artery cannulation and stroke volume by the Fick method also reported significant associations between

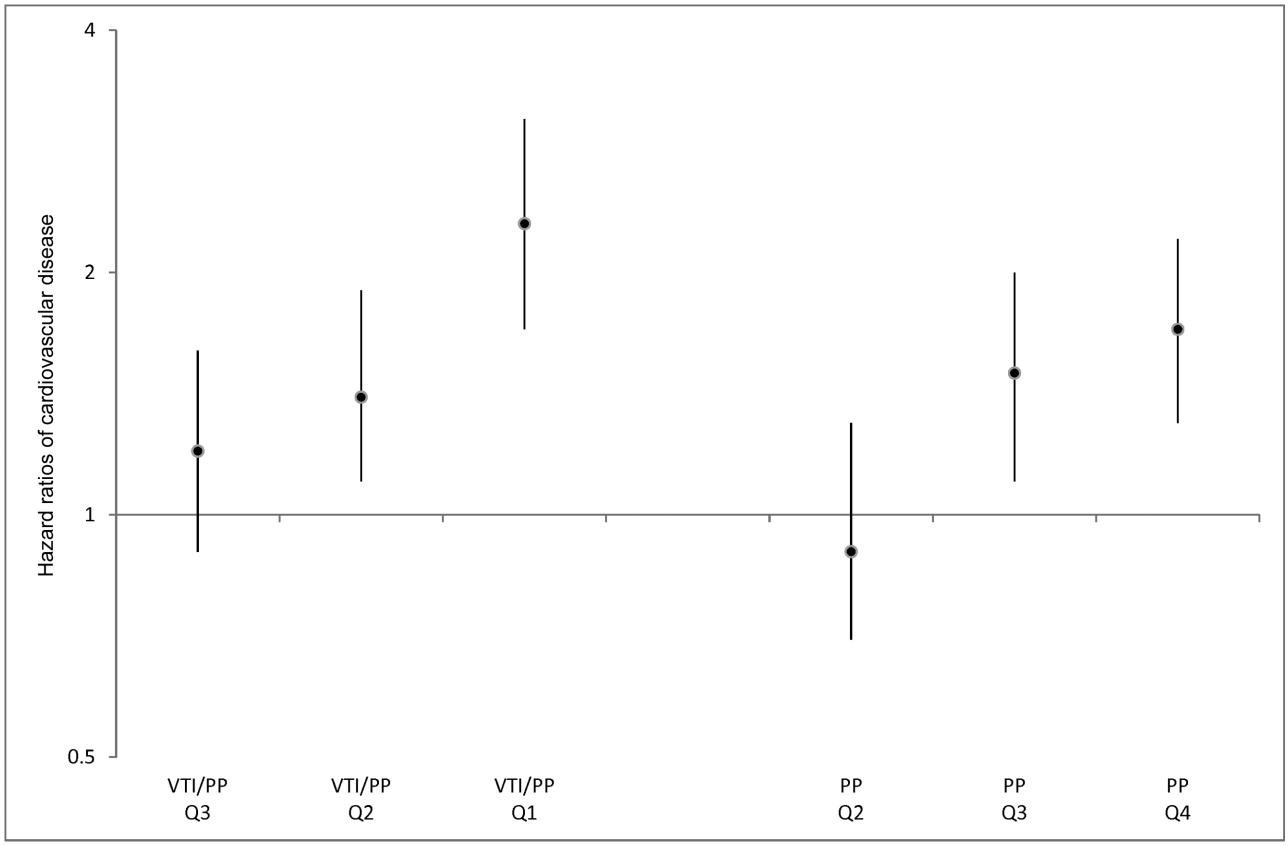

Figure 2. Adjusted HRs of incident cardiovascular disease (stroke, coronary disease, or heart failure) associated with quartiles of VTI/PP ratio (reference = Q4, greatest arterial compliance) and quartiles of PP (reference = Q1, narrowest PP). Abbreviations: HRs, hazard ratios; LCL, lower confidence intervals; PP, partial pressure; VTI, velocity time integral; UCL, upper confidence intervals.

PP/SV ratios and incident CVD but found no associations between PP and composite CVD in adjusted analyses.[15] In contrast, we observed significant associations between VTI/PP quartiles and composite CVD, incident heart failure, and incident stroke, but the association with incident coronary disease was marginal at best. This is consistent with a recent analysis from the REGARDS study, an observational cohort that included 9,621 blacks, which reported an adjusted HR of incident coronary disease of 1.61 (95% CI = 1.00–2.58), when contrasting widest with narrowest PP quartiles in blacks.[33]

Several methods for measuring systemic arterial compliance by echocardiography have been described. All are based on the 2-element Windkessel mathematical function, which models the elastic storage property of the aorta.[34] During systole, the aorta buffers the stroke volume by expanding, and in diastole, recoils slowly to eject the stored blood, allowing nearly continuous peripheral flow throughout the cardiac cycle.[34] From this model, total arterial compliance is derived invasively by the stroke volume to aortic PP ratio.[1,2] Some investigators have used tonometry-derived aortic PP as a noninvasive estimate of central PP.[10,12,35] However, echocardiographic arterial compliance indices are frequently derived using brachial PP as a surrogate for aortic PP,[7–9,13,14] as with our study. In comparative analyses, estimates of echocardiographic arterial compliance using tonometry-derived central PP and brachial PP have been shown to have similar associations with cardiovascular risk.[36]

The echocardiographic stroke volume used to calculate systemic arterial compliance has also been derived by a number of methods. In older echocardiograms antedating Doppler imaging, stroke volume has been estimated by left ventricular m-mode measurements, using either the z-method or the method of cubes with Teichholtz correction.[3,6] More commonly, stroke volume is estimated by Doppler echocardiography as the product of the left ventricular outflow tract velocity time integral (VTI) and its cross-sectional area.[37] In the seminal work by Lewis *et al.*, echocardiographic VTI × CSA stroke volumes were shown to be highly correlated (r = 0.95) with gold-standard stroke volumes measured invasively by thermodilution.[38] Although the correlation between VTI and invasive stroke volume was not reported, calculations based on the published raw data yield a Spearman correlation coefficient of 0.87 in study participants without aortic regurgitation. In the Strong Heart Study, which examined 1,935 American Indians by echocardiography, the VTI did not correlate well with the Teichholz stroke volume;[39] however, the Teichholz method is limited by cardiac orientation and is not the gold-standard measurement of stroke volume. Approximately 75% of the ARIC study echocardiograms were missing measurements of LVOT diameters. Accordingly, we derived arterial

compliance by the VTI/PP ratio, which did not differ greatly from SV/PP quartiles in associated risks of CVD in a complete-case analysis.

Our study has a few weaknesses that deserve mention. Because this is a longitudinal study with echocardiograms performed nearly 20 years ago, generalizations to contemporary populations may be questionable. Certainly, several advances in cardiovascular medicine have been realized over this time frame, which could influence both arterial compliance and incidence of cardiovascular events. We also were unable to analyze the effects of specific antihypertensive therapies on echocardiographic arterial compliance. As mentioned previously, our study relied on brachial PPs for the arterial compliance derivations and was missing LVOT diameters in the majority of echocardiograms. However, we believe this does not greatly detract from the associations between echocardiographic arterial compliance and incident CVD. Our study also has several strengths. The ARIC study Jackson site provides a large sample of blacks imaged by echocardiography and followed prospectively for over 15 years. Study participant retention was excellent, and clinical values were collected by standardized protocols with quality assurance ascertainment.

In conclusion, echocardiographic arterial compliance may be a useful adjunct for cardiovascular risk stratification. Echocardiograms are noninvasive and routinely performed in medical practice, and indices of arterial compliance are easily calculated by the ratio of stroke volume (or alternatively VTI) to brachial PP. We observed that in blacks, echocardiographic arterial compliance is associated with both composite and individual CVD outcomes and is more closely associated with these outcomes than is PP.

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DISCLOSURES

The authors declared no conflict of interest.

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