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Correlation of arterial blood pressure and compliance with left ventricular structure and function in the very elderly

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Abstract

There are very little data on the relationship between systolic blood pressure (SBP), diastolic blood pressure (DBP), arterial compliance and left ventricular structure and function, particularly left ventricular hypertrophy (LVH) in the very elderly (>75 years). SBP and arterial stiffness increase with age, and the question is: which of the two is the main stimulus to LV hypertrophy? This is a cross-sectional study to compare blood pressure and arterial stiffness measures with regard to their correlations with echocardiographic parameters of LV structure and function, controlling for age and cardiovascular risk factors, in a very elderly population. Arterial stiffness was determined by radial pulse waveform using pulse contour analysis. LV dimensions were measured by transthoracic M-mode echocardiography, and diastolic function by tissue Doppler measurements of diastolic mitral annular velocities. There were 179 subjects, all male, with a mean age of 81.8 years. Using age-adjusted partial correlations, SBP, DBP and mean arterial pressure (MAP) were correlated with parameters of LV structure and function. Correlation coefficients were: SBP v. left ventricular mass index (LVMI), $r = 0.246$; SBP v. early diastolic mitral annular velocity (MAV), $r = -0.179$; DBP v. LVMI, $r = 0.199$; DBP v. MAV, $r = -0.199$; MAP v. LVMI $r = 0.276$; and MAP v. MAV, $r = -.206$, all with $p < 0.05$. However, neither capacitative nor reflective arterial compliance was significantly correlated with any parameter of LV structure and function. After controlling for age and ten cardiovascular and metabolic risk factors, the correlation between blood pressure and the measured LV parameters was substantially unchanged, as was the lack of correlation between indices of arterial compliance and the LV indices. Arterial blood pressure is correlated with LV structure and function in the very elderly, but arterial stiffness, as measured by diastolic pulse contour analysis, is not.

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Keywords

Blood pressure; blood pressure; arterial stiffness; arterial compliance; myocardial remodeling; left ventricular hypertrophy; elderly

Introduction

Numerous studies have shown that left ventricular hypertrophy (LVH) is an important risk factor for cardiovascular events, particularly myocardial infarction and heart failure.¹ The most important modifiable cause of LVH is uncontrolled hypertension.²

Mainly as a result of the findings of the Framingham study,³ the paradigm of antihypertensive treatment has shifted from the control of diastolic blood pressure (DBP) towards that of systolic blood pressure (SBP). Subsequently, large clinical trials^{4,5} have shown that certain antihypertensive drugs improve cardiovascular outcomes beyond these drugs' effects on the brachial blood pressure. It has been hypothesized that this due to these agents' effects on central, rather than peripheral blood pressures.⁵ One possible mechanism for the magnitude of the difference between central and peripheral blood pressures relates in general to arterial compliance, a measure of arterial stiffness. Myocardial remodeling, particularly LVH, is one of the proposed intermediary mechanisms for the effect of arterial stiffness on cardiovascular events.^{6,7} The conventional model, therefore, is increased arterial stiffness, associated with increased pulse wave velocity, augmentation of the systolic pressure by a reflected pressure wave, the elevated systolic pressure increasing left ventricular (LV) output impedance, causing a pressure-induced increase in the dimension of cardiac myocytes, with consequent LVH. However, while it is true that in older people arteries are stiffer and LVH is more common,⁸⁻¹⁰ the correlation between these variables is often poor and they may occur independently.^{11,12} Also, there are no studies that have evaluated the relationship of arterial blood pressure, arterial stiffness and LVH in the very elderly, defined as those over 75 years of age. The objective of this study was to compare various measures of arterial blood pressure and indices of arterial stiffness with regard to their correlations with echocardiographic measurements of LV structure and function, particularly LVH, controlling for age, cardiovascular risk factors, and metabolic factors, in a very elderly population in a clinical setting.

Methods

Subjects were recruited from the outpatient lists in the Computerized Patient Records System of the James J. Peters Veterans Affairs Medical Center in Bronx, New York. The preliminary screening criteria were cognitively-intact ambulatory patients over 75 years old. The absence of dementia was verified with the Clinical Dementia Rating scale, and age- and education-adjusted norms of the Mini-Mental State Examination. Only male patients were included in the study. Informed consent, based on guidelines for enrollment of human subjects in medical research in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the Medical Center, was obtained from each subject prior to participation in the study. The study included 179 consecutive patients who completed a clinical examination, pulse contour analysis, and an echocardiographic study from October 2005 to October 2008.

A clinical examination was performed in a scheduled well-patient visit. The subject provided a medical history and underwent anthropometric measurements and physical examination. Blood chemistry values were obtained from the latest test results within the past year, as recorded in the Computerized Patients Records System.

Arterial pressures were measured using the HDI/PulseWave CardioVascular Profiling Instrument (Hypertension Diagnostics). After at least five minutes of rest, with the patient in the supine position with head inclined up 30 degrees, left brachial blood pressure was measured oscillometrically. Tracing of the arterial waveforms from the right radial artery was performed using a calibrated stainless-steel tonometer with a connected ceramic piezoelectric element. A computer-based diastolic pulse contour analysis of radial artery pulse waveforms was performed by evaluating the average diastolic pressure curve by a non-linear parameter-estimating algorithm using a third-order, four-element Windkessel model of the circulation. The diastolic decay of a waveform was quantified for the large artery elasticity index (LAEI), representing capacitive arterial compliance (C1) and for the small artery elasticity index (SAEI), representing reflective arterial compliance (C2) (Appendix A). The values of the C1 and C2 indices are weighted averages of the values obtained from waveforms recorded over 30 seconds.¹³⁻¹⁶ Mean arterial pressure was derived from the waveform analysis (after calibration with the blood pressure obtained by oscillometry). Arterial pressures and compliance measurements are listed in Appendix A.

On the same day, two-dimensional echocardiography was performed using Sequoia C512 sonography instrumentation (Acuson). LV dimensions were measured by M-mode. Early mitral filling and diastolic mitral annular velocities were determined by tissue Doppler imaging. The presence or absence of segmental wall motion abnormalities was assessed subjectively by a single observer who was blinded to any other information about the subject.

Seven anatomical and functional echocardiographic parameters, were recorded in the study; these were calculated based on the recommendations of American Society of Echocardiography,¹⁷ or were used in other studies. The anatomical parameters were LV mass index (LVMI), LV hypertrophy index (LVHI),¹⁸ and LV mass/height^{2.7} (LV/HT^{2.7}).¹⁹ Functional parameters were systolic – ejection fraction (EF), and presence of segmental wall motion abnormality, or diastolic – early diastolic mitral annular velocity (MAV) and the mitral peak velocity of early filling to early diastolic mitral annular velocity ratio (E/E' ratio).^{20,21} Equations for the calculation of parameters of myocardial structure and function are listed in Appendix B.

In addition to age, eight cardiovascular risk factors included in the study were a history of diabetes mellitus (verified by measurement of glycosylated hemoglobin), dyslipidemia (elevated triglyceride or low-density lipoprotein, low high-density lipoprotein), coronary artery disease, chronic kidney disease (based on serum creatinine), cerebrovascular accident, and cigarette smoking (pack years smoked). Levels of blood urea nitrogen and serum bicarbonate were also included as two additional metabolic parameters. These ten cardiovascular risk factors may be associated with measures of arterial pressure and indices of arterial stiffness and with parameters of LV structure and function, and hence are included in the analysis as control variables. Hypertension was not included as a control variable since measures of arterial pressure were evaluated in the analysis.

Statistical analysis

Partial correlation analyses were performed to evaluate the relationship of each parameter of LV structure and function by each measure of arterial pressure or index of stiffness, controlling only for age, and age with other cardiovascular risk and metabolic factors. The method of Meng, et al.²² for comparing correlation coefficients was used to compare correlations of two measures of arterial pressure with the same echocardiographic parameter. Statistical analyses were performed using SPSS version 14. A p-value of <0.05 was considered significant and is shown in boldface.

Results

The number and percentages of subjects in the study sample with clinical conditions conferring coronary disease risk, as well as the number and percentages of those on cardiovascular medications, are shown in Table 1. Means and standard deviations of anthropometric measurements, clinical laboratory values, measures of arterial pressure, indices of arterial stiffness, and parameters of left ventricular structure and function are listed in Table 2.

Table 3 shows the partial correlation coefficients and the p-values, controlling for age, for the correlations between measures of arterial pressure (SBP, DBP, MAP, PP) or indices of arterial stiffness (LAEI, SAEI) and seven parameters of left ventricular structure and function (LVMI, LVHI, LV/HT^{2.7}, EF, SWMA, MAV and the E/E' ratio). SBP was significantly correlated with three, DBP with six, MAP with three, and PP with three. Reflective arterial compliance (SAEI) was significantly associated only with mitral annular velocity. Capacitive arterial compliance (LAEI) was not significantly associated with any parameter of left ventricular structure or function.

Table 4 presents the results of analyses similar to those in Table 3, but controlling for ten cardiovascular risk and metabolic factors in addition to age. DBP and MAP were significantly correlated with four LV structure and function parameters and SBP was significantly correlated with three, and pulse pressure with two. Neither measure of arterial compliance was significantly correlated with any LV parameter. Controlling for these additional variables did not substantially change the partial correlations, although there were some changes in the significance status at the $p = 0.05$ level.

Discussion

A computer-based third order, 4-element modified Windkessel model of the circulation was used to match the diastolic pressure decay of the tonometrically obtained waveforms, to quantify changes in arterial waveform morphology, and thus to derive C1 and C2. This methodology has been validated in many studies,^{16, 23-26} supporting the conclusion that C1 and C2 are measures of large (capacitance) and small (resistance) artery compliance, and that C2 is, at least in part, a measure of arterial wave reflectance.²⁷ In addition, a recent publication from the Multi-Ethnic Study of Atherosclerosis (MESA) group demonstrates the remarkable predictive value for cardiovascular events of measurements made by diastolic pulse contour analysis.²⁸

There have been other studies of reduction of large and small artery elasticity using the same methodology as in our study, and also in the elderly. These have shown an association with endothelial dysfunction,²⁹ and with blood pressure,³⁰ but “elderly” in these studies have been a relatively young cohort, with mean ages of 64 years and 72 years. Weinberger et al.³¹ compared normotensive ($n=81$, mean age 47 years) and hypertensive ($n=194$, mean age 57 years) subjects. A significant age-related decrease in large and small vessel compliance was seen, but only in all hypertensive subjects and in normotensive women. All of our cohort were men, and, while 81% had hypertension, this was well-controlled with a mean blood pressure of 136/70 mm Hg.

The results of the present study demonstrate that the measures of arterial pressure showed significant relationships with echocardiographic parameters of LV structure and function, but the indices of arterial stiffness did not. This discrepancy was observed whether controlling for age or for ten cardiovascular risk and metabolic factors in addition to age; results were quite similar when not controlling for age or risk factors in this elderly male sample above 75 years of age (results not shown). Among the four measures of arterial

pressure, the MAP correlated most strongly with the LV parameters, followed by SBP and DBP. These correlations were small to moderate, probably accounting for a small component of the variation. However, other predictors, namely those related to arterial compliance, showed no significant correlation with the different measures of LV structure or function.

Forward conducting pulse waveforms peak during maximal ejection of blood and start to decline as ejection decreases. Pulse waves are also reflected from locations of impedance mismatch. The summation of these waves establishes the composite arterial waveform representing a complex interaction of signals of different frequencies at any point in the arterial system.²⁶ Subsequently, as the ventricles relax and the forward pressure decreases, the pressure waveform declines until the end of ventricular relaxation, when the nadir pressure is the DBP. As a person ages – because of the increase in arterial stiffness and therefore pulse wave velocity – the reflected pulse wave increases in magnitude and occurs at a progressively earlier time during the cardiac cycle, until the forward and reflective waves fuse; the peak pressure achieved by this fusion is the SBP.³² There is thus an augmentation of the SBP with a corresponding decrease in the DBP, thought to be the main pathophysiologic mechanism for the development of isolated systolic hypertension in older persons.

Since the augmented SBP in older people with stiff arteries represents the LV afterload, and since increased afterload is a stimulus to LV hypertrophy, the finding of an absence of a correlation between the indices of vascular stiffness and indices of LV structure and function was unexpected. There are, however, several possible and plausible explanations for the lack of correlation of the measures of compliance with LV mass. These include the relatively low and constant compliance of small vessels in very old people, the lack of correlation between small-vessel compliance and blood pressure, and the “protected survivor” effect. These are discussed below.

While, in general, there is a significant inverse correlation between age and both large artery compliance and small artery (oscillatory) compliance, the regression line describing this relationship seems to flatten out in individuals over the age of 60-70 years. Oscillatory compliance (C2) values, representing small vessel stiffness, are very low (<0.05 ml/mm Hg) and do not vary very much with age in subjects over the age of 75 years.³³ Thus there is relative uniformity of oscillatory compliance at very low levels in the very elderly, and any variability in LV mass in this population will not correlate well with compliance.

It is also noteworthy that McVeigh et al,³³ who derived large and small vessel compliance data in healthy subjects from intra-arterial brachial artery waveforms (n=115) and from radial artery tonometry (n=212), found that the fall in small artery or oscillatory compliance with age was independent of systolic blood pressure in both groups. This might provide a further explanation of our finding of a correlation between blood pressure and LV mass, with no such correlation with small artery compliance.

Another explanation of these results is the protected survivor effect, in which a minority of individuals possess inherited protection from risk factors, which for the majority would pose a threat throughout life. At early and middle ages, the population consists of individuals in whom one response to increased vascular stiffness is LV hypertrophy. However, those who have survived to a late age without LV hypertrophy and any of its fatal consequences, may have done so because they are more resistant to the hypertrophic effect of the increased LV output impedance associated with stiff arteries. We have seen this effect in action in a number of other studies in cognitively intact very elderly subjects, in whom homocysteine,³⁴ C-reactive protein,³⁵ hemoglobin A_{1c},³⁶ and total and low-density

lipoprotein levels³⁷ were all positively correlated with cognitive function, in vivid contrast to the situation in younger individuals.

Review of the literature also revealed other studies in which LV hypertrophy was closely correlated with arterial blood pressure but not with measures of vascular compliance. In 1315 normotensive or untreated hypertensive individuals older than 30 years of age, SBP was an independent predictor of left ventricular mass. However, indices of arterial stiffness, including carotid pulse wave velocity, aortic compliance, arterial elastance and carotid augmentation index did not independently predict left ventricular mass.³⁸ In another study of 255 normotensive and untreated hypertensives aged 25-88 years, SBP and DBP correlated with left ventricular mass and arterial elastance index correlated with endocardial and midwall fractional shortening but not with LV mass.³⁹ In yet another study of 276 mostly middle-aged normotensives and untreated hypertensives, the MAP was shown to be an independent predictor of LV mass and wall thickness, but carotid artery stiffness index, elastic modulus and arterial compliance index were not associated with LV mass.⁴⁰ While direct comparison is difficult because of the differences in study sample, parameter studied, conditions of data collection and use of covariates in the analysis, most studies have consistently shown the usefulness of SBP and DBP in predicting myocardial remodeling. The correlations of different indices of arterial stiffness with parameters of LV structure and function have not been consistent.

Confining this study to the very elderly is both an advantage and a limitation. The advantage is that it is the first such study in the very elderly, but the limitation is that, since age is an important confounding factor in the analysis of measures of arterial stiffness, this study was limited to a narrow age range of the very elderly. In this limited range of very elderly subjects, age was not predictive of measures of arterial pressure, indices of arterial stiffness and parameters of LV structure and function. Clearly, conclusions from this study are not necessarily applicable to populations of a different age group. Also, correlation analysis makes no assumption concerning the causal relationship between measures of arterial pressure and parameters of LV structure and function. The study is cross-sectional, which limits the possibility of establishing a cause-effect relationship.

We conclude that measures of arterial blood pressure are associated with LV structure and function, but the indices of arterial stiffness by diastolic pulse contour analysis are not. The relationship between SBP and LV mass is as predicted, since SBP is the output impedance, and therefore the afterload, of the LV. The absence of significant correlation between arterial stiffness and LV mass can be explained by the relatively low and constant compliance of small vessels in very old people, the lack of correlation between small-vessel compliance and blood pressure, and the “protected survivor” effect, and is also consistent with at least some of the published reports in the literature.

Appendix A

Arterial Pressure and Compliance Measures

$$\begin{aligned} \text{SV (ml)} &= -6.6 + 0.25 \times (\text{ET} - 35) - 0.62 \times \text{HR} + 40.4 \times \text{BSA} - 0.51 \times \text{age} \\ \text{CO (L/min)} &= \text{SV} \times \text{HR} \times 1000 \\ \text{SVR (dynes} \times \text{sec} \times \text{cm}^{-5}) &= \text{MAP} / \text{CO} \\ \text{P(t)} &= A_1 e^{-A_2 t} + A_3 e^{-A_4 t} \cos(A_5 + A_6 t) \\ \text{LAEI (ml/mmHg} \times 10) &= 2A_4 [(A_2 + A_4)^2 + A_3^2] / \text{SVR} A_2 (2A_4 + A_2)(A_4^2 + A_3^2) \\ \text{SAEI (ml/mmHg} \times 100) &= 1 / \text{SVR} (2A_4 + A_2) \end{aligned}$$

Where SV = stroke volume, ET = ejection time (ms.); HR = heart rate/min⁻¹; BSA = body surface area (m²); CO = cardiac output (ml/min); SVR = systemic vascular resistance; MAP = mean arterial pressure (mm Hg); LAEI = large artery elasticity index/capacitive arterial compliance; SAEI = small artery elasticity index/reflective arterial compliance. A1 (mmHg), A3 (mmHg) and A6 (rad) represent conditions during start of diastole; A2 (sec-1) – the dominant exponential nature of the curve; A4 (sec-1) – damping of pressure oscillation; and A5 (sec-1) – frequency of pressure oscillation.

Appendix B

Parameters of myocardial remodeling

$$\begin{aligned} \text{LVMI} &= \{0.8 \times (1.04[(\text{Dd} + \text{PWT} + \text{IVST})^3 - (\text{Dd})^3] + 0.6\}) / \text{BSA} \\ \text{LVHI} &= [(\text{PWT} + \text{IVST}) / 2] / (\text{Dd} / 2) \\ \text{LV} / \text{ht cm}^{2.7} &= \{0.8 \times 1.04[(\text{Dd} + \text{PW} + \text{VS})^3 - (\text{Dd})^3] + 0.6\} / \text{ht in cm}^{2.7} \end{aligned}$$

EF; ejection fraction

WMA; presence of wall motion abnormalities;

MAV; early diastolic mitral annular velocity

E/E'; ratio of mitral peak velocity of early filling/early diastolic mitral annular velocity

Where: LVMI = left ventricular mass index (g/m²)

LVHI = left ventricular hypertrophy index (cm)

Dd = left ventricular end-diastolic diameter (cm)

PWT = posterior wall thickness (cm)

IVST = interventricular septal thickness (cm)

BSA = body surface area (m²).

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Table 1

Number (percentage) of patients with a history of clinical conditions, and on cardiovascular medications. N = 179

Clinical condition:	Percentage
Coronary artery disease	67 (37.4%)
Hypertension	145 (81.0%)
Diabetes	42 (23.5%)
Cerebrovascular accident	22 (12.3%)
Chronic kidney disease	18 (10.1%)
Smoking	130 (72.6%)
Cardiovascular Medications:	
ACE-inhibitors	63 (35.2%)
Angiotensin-receptor blockers	34 (19.0%)
β -Blockers	95 (53.1)%
α -Blockers	34 (18.9)%
Diuretics	89 (49.7%)
Calcium-channel blockers	54 (30.1%)
Nitrates	19 (10.6%)
Aspirin	95 (53.0%)
HMG-CoA-reductase inhibitors	115 (64.2%)

Table 2

Clinical characteristics of the study sample.

	Variable (units)	Mean±Standard Deviation
Anthropometric Measurements	Age (y)	81.8±4.3
	Weight (kg)	79.3±12.8
	Height (m)	1.7±0.07
	Body mass index (kg/m ²)	26.5±3.6
	Body surface area (m ²)	1.9±0.18
Clinical Laboratory Values	Glycosylated hemoglobin -Hb _{A1c} (%)	5.9±1.0
	Triglyceride (mg/dL)	131.1±68.0
	High density lipoprotein - HDL (mg/dL)	49.4±13.9
	Low density lipoprotein – LDL (mg/dL)	94.0±27.0
	Creatinine (mg/dL)	1.2±0.42
	Blood urea nitrogen – BUN (mg/dL)	23.9±9.8
	Bicarbonate (mEq/dL)	27.00±3.1
	Cigarettes smoked (pack years)	24.5±25.8
Measures of Arterial Pressure and Stiffness	Diastolic blood pressure – DBP (mm/Hg)	70.8±10.1
	Systolic blood pressure – SBP (mm/Hg)	136.5±18.3
	Mean arterial pressure – MAP (mm/Hg)	97.7±13.1
	Pulse pressure – PP (mm/Hg)	65.7±14.3
	Large artery elasticity index – LAEI (ml/mmHg × 10)	12.7±5.4
	Small artery elasticity index – SAEI (ml/mmHg × 100)	3.5±2.2
Parameters of Left Ventricular Structure and Function	Left ventricular mass index – LVMI (gm/m ²)	136.5±39.2
	Left ventricular mass/height ^{2.7} - LV/ht ^{2.7} (gm/m ^{2.7})	60.1±18.0
	Left ventricular hypertrophy index - LVHI	0.46±0.13
	Mitral annular velocity – MAV (cm/sec)	14.3±4.6
	Ejection fraction – EF (%)	64.1±11.5
	Segmental wall motion abnormality- SWMA (yes=1, no=0)	0.28±0.45
	Mitral peak velocity of early filling to early diastolic mitral annular velocity (E/E' ratio)	4.43±1.64

Table 3

Partial correlations of measures of arterial pressure and indices of arterial stiffness predicting parameters of myocardial remodeling controlling for age (N = 179).

Arterial measures		Parameters of LV Structure and Function						
		LVMi	LVHI	LV/HT ^{2.7}	EF	SWMA	MAV	E/E' ratio
SBP	r	.246	.087	.241	.114	-.069	-.179	.115
	p	.001	.247	.001	.130	.359	.017	.126
DBP	r	.199	.155	.206	.172	-.151	-.199	.003
	p	.008	.039	.006	.021	.044	.008	.968
MAP	r	.276	.143	.267	.117	-.066	-.206	.139
	p	.0002	.058	.0003	.121	.385	.006	.064
PP	r	.179	.005	.168	.028	.016	-.093	.147
	p	.017	.942	.025	.715	.834	.216	.050
LAEI	r	-.036	-.086	-.039	-.011	.059	.046	.014
	p	.634	.254	.603	.882	.437	.539	.856
SAEI	r	.025	-.046	.039	-.077	.068	.163	-.049
	p	.744	.546	.608	.307	.366	.030	.519

Abbreviations: SBP – systolic blood pressure, DBP – diastolic blood pressure, PP – pulse pressure, MAP – mean arterial pressure, LAEI – large artery elasticity index, SAEI – small artery elasticity index, LVMi – left ventricular mass index, LVHI – left ventricular hypertrophy index, LV/HT^{2.7} – left ventricular mass(g)/height (cm)^{2.7}, EF – ejection fraction, SWMA – presence of segmental wall motion abnormality, MAV – early diastolic mitral annular velocity, E/E' ratio – Mitral peak velocity of early filling to early diastolic mitral annular velocity ratio, r – partial correlation coefficient, p – p-value

Table 4

Partial correlations of measures of arterial pressure and indices of arterial stiffness predicting parameters of myocardial remodeling controlling for age and ten cardiovascular risk and metabolic factors (N = 179).

Arterial measures		Parameters of LV Structure and Function						
		LVMi	LVHI	LV/HT ^{2.7}	EF	SWMA	MAV	E/E' ratio
SBP	r	.267	.093	.266	.051	-.031	-.144	.106
	p	.0005	.232	.0005	.508	.687	.063	.170
DBP	r	.227	.194	.246	.117	-.093	-.164	.034
	p	.003	.012	.001	.131	.231	.034	.664
MAP	r	.295	.151	.293	.066	-.036	-.166	.152
	p	.0001	.051	.0001	.387	.642	.032	.049
PP	r	.196	-.006	.182	-.010	.020	-.078	.115
	p	.011	.935	.018	.902	.800	.312	.139
LAEI	r	-.038	-.107	-.050	.023	.040	.034	.013
	p	.622	.166	.521	.770	.603	.661	.871
SAEI	r	.037	-.060	.040	-.504	.064	.144	-.049
	p	.638	.437	.602	.484	.408	.063	.529

Abbreviations: SBP – systolic blood pressure, DBP – diastolic blood pressure, PP – pulse pressure, MAP – mean arterial pressure, LAEI – large artery elasticity index, SAEI – small artery elasticity index, LVMi – left ventricular mass index, LVHI – left ventricular hypertrophy index, LV/HT^{2.7} – left ventricular mass/height in centimeters^{2.7}, EF – ejection fraction, SWMA – presence of segmental wall motion abnormality, MAV – early diastolic mitral annular velocity, E/E' ratio – Mitral peak velocity of early filling to early diastolic mitral annular velocity ratio, r – partial correlation coefficient, p – p-value. The ten cardiovascular and metabolic factors were hemoglobin A1C, triglyceride, high density lipoprotein and low density lipoprotein, creatinine, blood urea nitrogen and bicarbonate levels, number of cigarettes smoked in pack years, presence of coronary artery disease and occurrence of stroke or transient ischemic attack.