HHS Public Access

Author manuscript

J Clin Hypertens (Greenwich). Author manuscript; available in PMC 2017 December 01.

Published in final edited form as:

J Clin Hypertens (Greenwich). 2016 December ; 18(12): 1222–1227. doi:10.1111/jch.12898.

Hemodynamic Correlates of Blood Pressure in Older Adults: The ARIC Study

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Abstract

The primary aim of the present study was to identify the hemodynamic correlates of both steady and pulsatile blood pressure in community-dwelling older adults. In 3,762 adults aged 70–89 years, we observed that significant hemodynamic determinants of systolic blood pressure included arterial stiffness as measured by aortic pulse wave velocity, stroke volume (via echocardiography), arterial wave reflection, left ventricular ejection time, and upstroke time. The strongest influence was exerted by arterial stiffness. The steady state component of blood pressure, mean arterial pressure, was associated with both cardiac index and total peripheral resistance (TPR), but was more strongly associated with TPR. Results were similar when participants taking antihypertensive medications were excluded from analyses. The overall findings suggest that mean arterial pressure is associated strongly with TPR and that significant hemodynamic correlates of systolic blood pressure included arterial stiffness, stroke volume, and arterial wave reflection.

Keywords

aging; hypertension; arterial compliance; systemic hemodynamics

Introduction

Arterial pressure increases progressively with advancing age, resulting in a high prevalence of essential hypertension. This rise in blood pressure with age is a major contributor to age-related increases in numerous cardiovascular disorders[1]. While systolic blood pressure rises continuously, diastolic blood pressure plateaus and tends to decline after 50–60 years of age[2]. Accordingly, pulse pressure increases markedly with advancing age, resulting in a high prevalence of isolated systolic hypertension[3].

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Conflict of Interest

Nothing to disclose.

Although the trend of age-associated increases in blood pressure is well established, it remains unclear what hemodynamic factors determine blood pressure levels in older adults. Arterial blood pressure can be divided into both steady state and pulsatile primary components. The steady state component of blood pressure is represented by mean arterial pressure and is a critically important cardiovascular measure from the physiological standpoint, as it is the effective pressure that determines perfusion to the vital organs. Mean arterial pressure is determined exclusively by cardiac output and total peripheral resistance as governed by Ohm's law. The hemodynamic factors that influence the pulsatile component on the other hand, are much more complex. Systolic blood pressure is governed by a number of hemodynamic factors, including arterial stiffness, stroke volume, and left ventricular ejection fraction, whereas the primary hemodynamic determinants of diastolic pressure include total peripheral resistance, heart rate, arterial stiffness, and systolic blood pressure. The relative contribution of each hemodynamic factor is currently unknown, especially in older adults, as most of the available evidence is derived from circulatory modeling studies or comparisons with a single hemodynamic variable[4–6].

We evaluated a comprehensive number of hemodynamic determinants of blood pressure in Atherosclerosis Risk in Communities (ARIC) Study cohort. The availability of comprehensive tonometric measures in ARIC provided an added opportunity to separately interrogate correlates of both peripheral and central blood pressure. The latter approach is clinically important in light of increasing evidence that central, compared with peripheral, blood pressure may be more predictive of cardiovascular and other morbid outcomes[7]. Accordingly, the primary aim of the present study was to characterize the hemodynamic determinants of steady state and pulsatile blood pressure in community-dwelling older adult participants of the ARIC study.

Methods

Subjects

The ARIC Study is an ongoing, population-based longitudinal study involving four US communities (Forsyth County, NC, Jackson, MS, Minneapolis, MN, and Washington County, MD). A total of 6,533 participants (65% response rate from 10,036 eligible participants) attended ARIC study visit 5 (in years 2011 to 2013) and underwent a standardized examination[8]. For the present analyses, we excluded participants with missing information on blood pressure, arterial stiffness, and/or echocardiography, BMI ≥40 kg/m^2, major arrhythmias (Minnesota codes 8-1-3, 8-3-1, and 8-3-2: ≥10% atrial and ventricular premature beats, atrial fibrillation or flutter), peripheral vascular disease (aortic aneurysms, abdominal aorta ≥5 cm, history of aortic or peripheral revascularization or presence of an aortic graft, aortic stenosis), other major cardiovascular disease (history of coronary artery disease, heart failure, or stroke), and moderate or greater aortic regurgitation. Participants who self-identified as Asian and African American from Minnesota and Maryland field centers were excluded due to small numbers. After exclusions, the final analytic sample included 3,762 participants. Institutional review boards approved the study protocol at each field center and participating institution, and all study participants provided written informed consent.

Measurements

Participants were asked not to consume food or drinks and to refrain from tobacco and vigorous physical activity after midnight prior to the visit or for 8 hours prior to the visit. Participants were also asked to bring all prescription and nonprescription medications taken within 2 weeks. Blood samples were obtained following a standardized venipuncture protocol and were assayed in ARIC central laboratories. Diabetes was defined as fasting glucose ≥126 mg/dl, non-fasting glucose ≥200 mg/dl, antidiabetic medication use, or self-reported diagnosis of diabetes.

Brachial blood pressure (systolic, mean, and diastolic blood pressure) was measured twice with the participants in the supine position using oscillometric automated sphygmomanometer (VP-1000 Plus, Omron Healthcare, Kyoto, Japan), and the average measurement was used for analyses. Stroke volume and cardiac output were calculated based on 2D echocardiographic measurements (IE33, Philips Healthcare, Andover, MA) performed with excellent reproducibility in our core laboratory, as previously described[9]. Echocardiographic measures were indexed to body surface area, where appropriate. Total peripheral resistance was calculated as mean arterial pressure divided by the cardiac index. Carotid-femoral pulse wave velocity, an index of arterial stiffness, and carotid artery pressure waveforms were obtained using an automatic vascular screening device (VP-1000 Plus, Omron Healthcare, Kyoto, Japan) as previously described[10] with excellent reproducibility[11]. Carotid and femoral arterial pressure waveforms were acquired for 30 seconds by applanation tonometry sensors attached on the left common carotid artery (via neck collar) and left femoral artery (via elastic tape around the hip). Augmentation index, an index of arterial wave reflection, carotid systolic pressure, ejection time, and upstroke time were obtained from the carotid pressure waveform analyses[5]. Augmentation index measured by this machine is strongly associated with that obtained with SphygmoCor[12]. Vascular and cardiac measurements were performed on different days.

Statistical analyses

All statistical analyses were conducted using SAS version 9.4 (Cary, NC). Associations between blood pressure and hemodynamic variables were evaluated using multivariable linear regression and partial correlational analyses. Adjusted models included age, sex, race, body mass index, and current smoking status. Separate analyses were performed in the total sample of 3,762 participants and in the subgroup of 1,204 participants not taking antihypertensive medications at the time of the examination. A two-sided $P<0.05$ was considered statistically significant.

Results

The average systolic blood pressure was 137±17 mmHg and a majority of participants (68%) were taking antihypertensive medications at the time of the examination (Table 1).

Table 2 displays multivariable-adjusted associations of both brachial and carotid systolic blood pressure measures with hemodynamic variables. All the hemodynamic variables examined were significantly associated with both brachial and carotid systolic blood

pressure except for ejection fraction. For both brachial and carotid systolic pressure, the hemodynamic variables that contributed the most variation to systolic pressure (as represented by the partial R^2 value) were arterial stiffness followed by augmentation index and ejection time. The results were similar when the analyses were repeated after excluding the participants taking antihypertensive medications.

In an attempt to determine if the associations between blood pressure and hemodynamic parameters are affected by age, the study cohort was divided according to the age categories that approximate tertiles (<75 years, 75 to <80 years, and ≥80 years) (Table 3). The strength of associations between arterial stiffness and systolic BP became weaker with increasing age while associations with stroke volume and augmentation index became stronger.

When analyses were repeated using pulse pressure, the overall results were similar to those observed for systolic blood pressure (Table 4). Ejection time was the most prominent hemodynamic determinant of variation in both brachial and systolic pulse pressure in the total study sample, followed by arterial stiffness. Stroke volume, in addition to augmentation index, was also among the hemodynamic measures that was observed to contribute significant variation to measures of pulse pressure. When the participants were divided into age tertiles, contribution of ejection time to pulse pressure became greater with increasing age (Table 5).

The multivariable-adjusted hemodynamic correlates of mean blood pressure are shown in Table 6. We observed relatively small contributions of CI and TPR to variation in MBP in regression models that adjusted for all the clinical covariates. Mean blood pressure was associated with both cardiac index and total peripheral resistance; however, total peripheral resistance was the primary hemodynamic determinant of variation in mean blood pressure. The results were similar when the participants who had been taking antihypertensive medications were excluded from these analyses.

Discussion

Arterial blood pressure progressively increases with advancing age, resulting in a high prevalence of essential hypertension in the population at large. Indeed, in our community-based study sample of predominantly older adults, the prevalence of hypertension was over 70%. As implied by the term “essential” hypertension, the physiological factors that contribute to the steady rise in blood pressure in aging adults remain largely unknown. Thus, in the present study, we interrogated the distinct steady state and pulsatile components of blood pressure and examined the hemodynamic correlates of these components measured both peripherally and centrally.

The steady state component of blood pressure is characterized by mean arterial blood pressure and is determined by cardiac output and peripheral resistance via the Ohm’s law. Of these two factors, total peripheral resistance displayed the more dominant influence on mean arterial pressure in the present sample of community-dwelling older adults. These results are consistent with previous small-scale cross-sectional studies showing that the elevation in mean arterial pressure with aging is related to an increase in total peripheral resistance

because cardiac output typically declines[5, 6]. The steady state blood pressure component based on Ohm's law is useful in gaining physiological insight. However, it may not be appropriate to apply to an aging population because mean arterial pressure does not increase much with adult aging due to age-related declines in diastolic blood pressure that offset corresponding increases in systolic blood pressure. Furthermore, in clinical practice, hypertension is typically defined in terms of systolic and diastolic blood pressure, and mean blood pressure is usually not even calculated.

In the present study, we included a variety of hemodynamic measures that have been described as physiological correlates of systolic blood pressure, and we examined the associations between potential hemodynamic determinants and noninvasively-measured systolic blood pressure. We observed that most of the hemodynamic determinants, including arterial stiffness, stroke volume, arterial wave reflection, left ventricular ejection time, and upstroke time, were significantly related to brachial systolic pressure. The only hemodynamic measure that did not display significant associations with systolic blood pressure was left ventricular ejection fraction, possibly due to attrition-related sampling bias (i.e., ARIC study participants who died prior to visit 5 were more likely to have had reduced ejection fraction).

An increase in the stiffness of the large elastic arteries located in the cardiothoracic (central) circulation (e.g., aorta and carotid artery) has been implicated as the primary mechanism underlying the age-associated increase in systolic blood pressure and pulse pressure[13, 14]. Indeed, the strongest relation with systolic blood pressure was observed with arterial stiffness as measured by pulse wave velocity. The increase in central artery stiffness observed with adult aging likely occurs because of changes in both functional and structural determinants within the vascular wall[13, 15]. However, age-related increases in arterial stiffness do not appear to be dependent on the presence of clinical atherosclerotic disease. The stiffening of arteries with advancing age has been observed in a rural Chinese population in whom the prevalence of atherosclerosis is very low[16, 17] and in rigorously-screened U.S. men and women[18–20], as well as in beagle dogs who are resistant to atherosclerosis[21]. Interestingly, when the study cohort was divided into approximate tertiles, the strength of associations between arterial stiffness and systolic BP became weaker with increasing age while associations with stroke volume and augmentation index became stronger. These results suggest that the role of arterial stiffness as a primary determinant of pulsatile blood pressure component may get diminished with advancing age.

To date, studies of the hemodynamic determinants of blood pressure have largely focused on peripheral (i.e., brachial) blood pressure. Thus, the determinants of central blood pressure have been inferred but not established. One of the strengths of the present analyses is the inclusion of central (i.e., carotid) blood pressure assessment. Central blood pressure is more directly related than peripheral blood pressure to cardiac afterload and coronary perfusion during diastole[7]. Accordingly, central blood pressure is considered a more accurate and robust cardiovascular prognostic marker than conventional brachial blood pressure and is differentially affected by antihypertensive medications[22, 23]. We observed that hemodynamic correlates of central systolic pressure included arterial stiffness, stroke volume, arterial wave reflection, left ventricular ejection time, and upstroke time. The strengths of these

associations were fairly similar to those observed for peripheral (i.e., brachial) blood pressure.

Strengths of the present study include its very large sample size involving older adults as well as comprehensive measures of hemodynamic factors. However, there are also a number of limitations that should be emphasized. First, the cross-sectional nature of the present analyses cannot provide any information regarding causality or longitudinal changes. Second, a major confounding factor for the present analyses was the high prevalence of anti-hypertensive medication use. Therefore, we performed separate analyses in the subset of individuals not taking anti-hypertensive medications and observed very similar results to those from the analyses of the total sample. However, it should be noted that there are a number of co-existing conditions that we could not account for fully with statistical analyses. Conversely, we were not able to address the effects of certain anti-hypertensive medications. Third, the present sample was primarily composed of older adults; thus, the extent to which our results can be extended to younger populations is unknown. Finally, the strengths of associations between blood pressure and hemodynamic factors were modest, likely due in large part to the fact that all measurements were performed non-invasively at a single point in time in this large epidemiologic cohort; as such, our results should be interpreted with caution.

In conclusion, the findings of the present study in community-dwelling older adults indicate that mean arterial pressure is associated strongly with cardiac output and particularly with systemic vascular resistance. Significant hemodynamic determinants of systolic blood pressure included arterial stiffness, stroke volume, arterial wave reflection, left ventricular ejection time, and upstroke time with the strongest influence exerted by arterial stiffness. We also showed that these factors similarly impacted central BP. Understanding physiological factors that determine components of blood pressure should lead to better prevention and treatment strategies for the epidemics of hypertension.

Acknowledgments

The Atherosclerosis Risk in Communities Study is carried out as a collaborative study supported by National Heart, Lung, and Blood Institute contracts (HHSN268201100005C, HHSN268201100006C, HHSN268201100007C, HHSN268201100008C, HHSN268201100009C, HHSN268201100010C, HHSN268201100011C, and HHSN268201100012C). This work was also supported by NIGMS grant T32 GM74905 (ELM), NHLBI cooperative agreement NHLBI-HC-11-08 (SDS), grants R00-HL-107642 (SC) and K08-HL-116792 (AMS), a grant from the Ellison Foundation (SC), and grant 14CRP20380422 from the American Heart Association (AMS).

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Table 1

Characteristics of the study participants

Characteristic	Total Sample (N=3,762)
Age (year)	75±5
Women (%)	63
Black (%)	21
Body mass index (kg/m^2)	28±4
Obesity (%)	30
Diabetes (%)	23
Total cholesterol (mg/dl)	4.9±1.0
LDL cholesterol (mg/dl)	2.8±0.9
HDL cholesterol (mg/dl)	1.4±0.4
Triglycerides (mg/dl)	1.4±0.7
eGFR (ml/min)	71.7±16.1
Antihypertensive medication (%)	68
Current smoker (%)	5
Heart rate (bpm)	65±10
Systolic BP (mmHg)	137±17
Diastolic BP (mmHg)	73±9
Mean BP (mmHg)	100±13
Carotid systolic BP (mmHg)	144±22
Carotid AI (%)	19.5±16.0
cfPWV (cm/sec)	1146±295
SV index (ml/m^2)	28.1±6.0
CO index (ml/min/m^2)	1801±391
TPR (U)	0.03±0.01
LVEF (%)	66±10
Reduced LVEF <30% (%)	0.05

eGFR=estimated glomerular filtration rate, BP=blood pressure, AI= augmentation index, cfPWV=carotid-femoral pulse wave velocity, SV=stroke volume, CO=cardiac output, TPR=total peripheral resistance, LVEF=left ventricular ejection fraction.

Values are shown as mean±SD or percent frequency.

Table 2

Multivariable-adjusted hemodynamic correlates of brachial and carotid systolic pressures

Hemodynamic	Total sample (n=3,762)			Non-medicated (n=1,204)		
Parameter	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**
Brachial systolic pressure						
cfPWV	0.02 (0.0009)	0.134	<0.0001	0.02 (0.002)	0.121	<0.0001
SV index	1.71 (0.30)	0.009	<0.0001	1.80 (0.50)	0.011	0.0004
AI	4.50 (0.29)	0.067	<0.0001	4.17 (0.49)	0.060	<0.0001
LVEF	−0.05 (0.29)	0.000009	0.86	−0.66 (0.49)	0.002	0.18
ET	3.37 (0.28)	0.040	<0.0001	2.35 (0.51)	0.017	<0.0001
UT	−2.15 (0.28)	0.016	<0.0001	−2.25 (0.50)	0.017	<0.0001
Carotid systolic pressure						
cfPWV	0.02 (0.001)	0.089	<0.0001	0.02 (0.002)	0.091	<0.0001
SV index	2.97 (0.38)	0.016	<0.0001	2.65 (0.63)	0.014	<0.0001
AI	4.83 (0.37)	0.045	<0.0001	4.22 (0.62)	0.039	<0.0001
LVEF	0.70 (0.36)	0.001	0.05	0.11 (0.61)	0.00003	0.86
ET	5.94 (0.34)	0.072	<0.0001	4.53 (0.63)	0.040	<0.0001
UT	−2.35 (0.36)	0.011	<0.0001	−2.54 (0.62)	0.014	<0.0001

cfPWV=carotid-femoral pulse wave velocity, SV=stroke volume, LVEF=left ventricular ejection fraction, AI=augmentation index, ET=ejection time, UT=upstroke time.

All analyses were adjusted for age, sex, black race, BMI, diabetes, and current smoking status. Coefficients represent change in systolic pressure per 1-SD change in the hemodynamic parameter.

Table 3

Multivariable-adjusted hemodynamic correlates of brachial and carotid systolic pressures (total sample, N=3,762), by age group

	Age <75 years			Age 75 to <80 years			Age ≥80 years		
Parameter	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**
Brachial SBP									
cfPWV	0.03 (0.001)	0.158	<0.0001	0.02 (0.002)	0.123	<0.0001	0.02 (0.002)	0.094	<0.0001
SV index	1.32 (0.40)	0.006	0.0009	2.04 (0.57)	0.013	0.0004	2.55 (0.69)	0.020	0.002
AI	3.82 (0.38)	0.051	<0.0001	4.73 (0.55)	0.074	<0.0001	5.93 (0.70)	0.101	<0.0001
LVEF	−0.95 (0.40)	0.003	0.02	0.99 (0.52)	0.004	0.06	0.69 (0.63)	0.002	0.27
ET	2.94 (0.38)	0.029	<0.0001	3.25 (0.52)	0.038	<0.0001	4.48 (0.59)	0.075	<0.0001
UT	−1.62 (0.39)	0.009	<0.0001	−2.11 (0.53)	0.016	<0.0001	−3.35 (0.63)	0.039	<0.0001
Carotid SBP									
cfPWV	0.03 (0.002)	0.106	<0.0001	0.02 (0.002)	0.080	<0.0001	0.02 (0.002)	0.071	<0.0001
SV index	2.42 (0.51)	0.011	<0.0001	3.21 (0.72)	0.019	<0.0001	4.25 (0.81)	0.036	<0.0001
AI	4.03 (0.49)	0.033	<0.0001	5.07 (0.72)	0.049	<0.0001	6.35 (0.85)	0.073	<0.0001
LVEF	−0.63 (0.52)	0.0007	0.22	2.19 (0.66)	0.011	0.0009	1.75 (0.75)	0.007	0.02
ET	5.57 (0.48)	0.060	<0.0001	5.79 (0.66)	0.069	<0.0001	6.72 (0.70)	0.108	<0.0001
UT	−1.63 (0.50)	0.005	0.001	−2.56 (0.67)	0.014	0.0002	−3.53 (0.77)	0.028	<0.0001

SBP=systolic blood pressure, cfPWV=carotid-femoral pulse wave velocity, SV=stroke volume, LVEF=left ventricular ejection fraction, AI=augmentation index, ET=ejection time, UT=upstroke time.

All analyses were adjusted for age, sex, black race, BMI, and current smoking status. Coefficients represent change in systolic pressure per 1-SD change in the hemodynamic parameter.

Table 4

Multivariable-adjusted hemodynamic correlates of brachial and carotid pulse pressures

Hemodynamic	Total sample (n=3,762)			Non-medicated (n=1,204)		
Parameter	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**
Brachial pulse pressure						
cfPWV	0.01 (0.0006)	0.083	<0.0001	0.01 (0.001)	0.069	<0.0001
SV index	2.22 (0.20)	0.030	<0.0001	1.90 (0.34)	0.024	<0.0001
AI	2.14 (0.20)	0.030	<0.0001	1.90 (0.33)	0.026	<0.0001
LVEF	0.74 (0.20)	0.004	0.0002	0.04 (0.33)	0.00001	0.90
ET	4.03 (0.18)	0.112	<0.0001	3.11 (0.33)	0.063	<0.0001
UT	−1.31 (0.19)	0.011	<0.0001	−1.29 (0.34)	0.011	0.0001
Carotid pulse pressure						
cfPWV	0.01 (0.001)	0.042	<0.0001	0.014 (0.002)	0.045	<0.0001
SV index	3.51 (0.31)	0.032	<0.0001	2.73 (0.49)	0.023	<0.0001
AI	2.42 (0.31)	0.016	<0.0001	2.03 (0.48)	0.014	<0.0001
LVEF	1.38 (0.30)	0.005	<0.0001	0.45 (0.48)	0.0007	0.36
ET	6.51 (0.27)	0.122	<0.0001	5.20 (0.48)	0.082	<0.0001
UT	−1.46 (0.30)	0.006	<0.0001	−1.30 (0.49)	0.005	0.008

cfPWV=carotid-femoral pulse wave velocity, SV=stroke volume, LVEF=left ventricular ejection fraction, AI=augmentation index, ET=ejection time, UT=upstroke time.

All analyses were adjusted for age, sex, black race, BMI, diabetes, and current smoking status. Coefficients represent change in pulse pressure per 1-SD change in the hemodynamic parameter.

Table 5

Multivariable-adjusted hemodynamic correlates of brachial and carotid pulse pressures (total sample, N=3762), by age group

	Age <75 years			Age 75 to <80 years			Age ≥80 years		
Parameter	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**	**Coef. (SE)**	**Partial R^2**	**P-value**
Brachial PP									
cfPWV	0.01 (0.0009)	0.100	<0.0001	0.01 (0.001)	0.077	<0.0001	0.01 (0.001)	0.072	<0.0001
SV index	1.78 (0.27)	0.020	<0.0001	2.62 (0.38)	0.044	<0.0001	3.03 (0.48)	0.051	<0.0001
AI	1.59 (0.26)	0.018	<0.0001	2.50 (0.38)	0.043	<0.0001	2.88 (0.52)	0.044	<0.0001
LVEF	0.21 (0.28)	0.0003	0.44	1.43 (0.35)	0.016	<0.0001	1.24 (0.45)	0.010	0.006
ET	3.66 (0.25)	0.093	<0.0001	3.72 (0.034)	0.104	<0.0001	5.06 (0.40)	0.178	<0.0001
UT	−0.84 (0.26)	0.005	0.002	−1.68 (0.36)	0.021	<0.0001	−2.03 (0.46)	0.026	<0.0001
Carotid PP									
cfPWV	0.02 (0.001)	0.055	<0.0001	0.01 (0.002)	0.033	<0.0001	0.01 (0.002)	0.041	<0.0001
SV index	2.88 (0.42)	0.22	<0.0001	3.94 (0.59)	0.040	<0.0001	4.73 (0.69)	0.057	<0.0001
AI	1.80 (0.40)	0.010	<0.0001	2.66 (0.60)	0.019	<0.0001	3.34 (0.75)	0.027	<0.0001
LVEF	0.47 (0.43)	0.0005	0.28	2.60 (0.54)	0.021	<0.0001	2.04 (0.65)	0.013	0.002
ET	6.16 (0.38)	0.107	<0.0001	6.18 (0.52)	0.113	<0.0001	7.26 (0.57)	0.169	<0.0001
UT	−0.77 (0.41)	0.002	0.06	−2.21 (0.56)	0.014	<0.0001	−2.14 (0.65)	0.014	0.001

PP=pulse pressure, cfPWV=carotid-femoral pulse wave velocity, SV=stroke volume, LVEF=left ventricular ejection fraction, AI=augmentation index, ET=ejection time, UT=upstroke time.

All analyses were adjusted for age, sex, black race, BMI, and current smoking status. Coefficients represent change in pulse pressure per 1-SD change in the hemodynamic parameter.

Table 6

Multivariable-adjusted hemodynamic correlates of mean blood pressure based on Ohm's law

Hemodynamic	Total sample (n=3,762)			Non-medicated (n=1,204)		
Parameter	Coef. (SE)	Partial R^2	P-value	Coef. (SE)	Partial R^2	P-value
Cardiac index	0.74 (0.21)	0.003	0.0005	1.11 (0.38)	0.007	0.003
TPR	6.29 (0.21)	0.151	<0.0001	6.29 (0.39)	0.178	<0.0001

TPR=total peripheral resistance

All analyses were adjusted for age, sex, black race, BMI, and current smoking status. Coefficients represent change in mean blood pressure per 1-SD change in the hemodynamic parameter.