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Women Have Significantly Greater Difference Between Central and Peripheral Arterial Pressure Compared to Men: The Bogalusa Heart Study

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Abstract

Background—Gender differences in the relationship between central and peripheral BP are not well described. We sought to investigate gender differences between central systolic blood pressure (cSBP) and peripheral systolic blood pressure (pSBP) in adults in the Bogalusa study population.

Methods—This study enrolled adults in a cross sectional survey conducted in 2007–2010. BP was measured with a standard cuff and Omron applanation tonometer. Data were available from 876 participants.

Results—Participants were 57.9% female and 42.1% male (mean age 43.5 years ± 4.4). Mean (SD) for cSBP-pSBP was 1.0 (6.9) for males and 7.4 (5.2) for females ($p<0.001$). Augmentation index (AI) was higher in women (men: 70.8±14 vs. women: 85.5±13; $p<0.01$), as well as augmentation index standardized to heart rate (HR) of 75 (AI@75) (men: 68.5±13 vs. women: 84.4±11.8; $p<0.01$).

Conclusions—Female participants had greater difference between cSBP and pSBP than males. This suggests that given similar peripheral BP females might be at higher risk for developing target organ damage. Women in this study had higher AI, which may contribute to the difference found between cSBP and pSBP. These findings may explain why women have more age-related left ventricular hypertrophy, and poorer prognosis following myocardial infarction compared to males.

Keywords

Blood pressure; Gender differences; Central blood pressure; Augmentation Index

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Dr. Giles is a consultant and investigator for Forest Laboratories. Dr. Sander is a speaker for Forest Laboratories.

Introduction

Although men have a higher overall prevalence of hypertension than women(1), women have a higher risk of hypertensive target organ damage than age-matched men. Women have more left ventricular hypertrophy (LVH) on echocardiography compared to men (2–4), and female gender is an independent predictor of both microalbuminuria and LVH. Additionally, while both men and women develop LVH with increasing age, women develop more age-related LVH(5,6), and the impact of LVH on adverse cardiovascular events may be greater in women than in men(7). Women have also shown increased risk of death by stroke, especially at younger ages(8), and have higher mortality risk after myocardial infarction (MI) than men(9,10).

The above observations are based on the measurement of systemic arterial blood pressure (BP) in the brachial artery. While peripherally measured BP is a valuable predictor of cardiovascular events, it is reported that non-invasive measurement of central arterial pressure more accurately predicts adverse cardiovascular events and target organ damage(11,12) and that elevations in central arterial pressure are more closely linked to hypertensive target organ damage than peripheral measurements(13).

It has been suggested that gender differences in target organ damage may be due to differences in pulsatile vascular load(3). Until around age 50 women tend to have lower peripheral BP than men(14,15); however, in this same age group women show greater target organ damage than men. Thus, we speculated that there might be significant differences between men and women in measured central versus peripheral BP. Therefore, we analyzed data from adult males and females enrolled in the Bogalusa Heart Study to describe differences between central and peripheral BP in women compared to men. Further, we sought to determine if other measured parameters would assist in explaining the postulated difference.

Methods

Study Cohort

Data were available for 876 participants in the Bogalusa Heart Study. The Bogalusa Heart Study is a long-term study of cardiovascular disease risk factors in children and adults in the semirural, multi-racial community of Bogalusa, Louisiana. The study began in 1973 by screening children ages 5–17 for cardiovascular risk factors and has 20 cross sectional follow up surveys to date with a total cohort of over 16,000 individuals. Data for the present study were obtained from a cross-sectional survey that enrolled participants in 2007–2010. Participants were excluded if any relevant screening data were missing or if they were currently using any antihypertensive medications. All participants in this study gave informed consent for examination. Study protocols were approved by the Institutional Review Board of the Tulane University Medical Center.

General Examination

Standardized protocols were used by trained examiners. Anthropometric and BP measurements were made in replicate and the mean values were used for analysis. Peripheral BP measurements were obtained using mercury sphygmomanometers on the right arm of participants in a relaxed, sitting position with three replicates each performed by two randomly assigned nurses. The first and fourth Korotkoff phases were used to determine systolic and diastolic BP, respectively. Hypertension was defined as systolic BP of 140 mmHg or higher or diastolic BP of 90 mmHg or higher. Central BP measurements were estimated using an Omron HEM-9000AI device (Omron Healthcare Co., Ltd., Kyoto, Japan). Each participant underwent 4 cuff BP measurements (based on the cuff-oscillometric

principle) and 4 radial artery pressure waveform readings acquired using the HEM9000AI radial applanation tonometer. The readings were averaged to calculate each variable listed. Inflection points of the peripheral pulse waveform that corresponded to early (pSBP1) and late systolic BP (pSBP2) were obtained by derivatives of the original waveform. The Omron device then estimates central systolic BP from the pSBP2 value using linear regression(16,17). The Omron device contains software to derive augmentation index (AI), as well as augmentation index standardized to heart rate 75 (AI@75), as described by Richardson, et al (18). Body mass index (BMI) calculated as weight in kilograms divided by the square of height in meters, was used as a measure of overall adiposity. Information on smoking status was obtained from questionnaires. Those who smoked at least one cigarette per week during the past year were considered current smokers.

Laboratory Analyses

Participants were instructed to fast for 12 hours before screening, and compliance with fasting was determined by an interview on the morning of examination. Serum cholesterol and triglycerides (Trig) were determined enzymatically on the Hitachi 902 Automatic Analyzer (Roche Diagnostics, Indianapolis, IN)(19). Insulin resistance status was assessed as homeostasis model assessment of insulin resistance (HOMA-IR) according to the formula described: insulin (μU/mL) × glucose (mmol/L)/22.5(20). Plasma high sensitivity C-reactive protein (CRP) levels were measured by latex particle-enhanced immunoturbidemetric assay on a Hitachi 902 Automatic Analyzer (Roche Diagnostics, Indianapolis, IN, USA).

Statistical Analysis

All analyses were conducted using the SAS software package version 9.2 (SAS Institute, Cary, NC). Continuous variables are expressed as mean +/− standard deviation (SD) unless otherwise indicated. Analysis of covariance (ANCOVA) was used to assess contrasts in race and sex groups for difference in central versus peripheral systolic BP and other cardiovascular (CV) risk factor variables after controlling for age. Post-hoc test with Bonferroni adjustment for multiple comparisons were used where appropriate. Normality of distribution was assessed by the Kolmogorov-Smirnov test. Because Trig, insulin, HOMA-IR and CRP were not normally distributed, log transformation was used to improve normality. All analyses were performed on transformed data where appropriate. Multivariable adjusted linear regression models were used to examine the total and sex-specific independent effects of CV risk factors and hemodynamic variables on measures of difference in central versus peripheral systolic BP and augmentation index standardized to heart rate of 75 bpm. Independent variables for all models included: age, heart rate (HR), height, smoking, HOMA-IR, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), Trig, CRP, race and gender. For gender-specific models, the effect of gender as an independent variable was excluded. All p values were two tailed and adjusted for covariates where appropriate. The level of significance for hypothesis testing was set at 5% ($\alpha = 0.05$).

Results

Participant characteristics are shown in Table 1. There was no significant difference in age between men and women (men: 43.9±4.3 vs. women: 43.2±4.5). Women had lower peripheral systolic BP (pSBP) (men: 123.3±14.7 mmHg vs. women: 115.9±15.1 mmHg; $p<0.01$), and diastolic BP (men: 78.4±10.6 mmHg vs. women: 73.6±11.1 mmHg; $p<0.01$). Despite differences in peripheral BP, there was no significant difference in central systolic BP (cSBP) (men: 124.4±16.3 mmHg vs. women: 123.4±17.5 mmHg; $p>0.05$). Augmentation index (AI) was higher in women (men: 70.8±14 vs. women: 85.5±13; $p<0.01$), as well as augmentation index standardized to heart rate (HR) of 75 (AI@75) (men:

68.5±13 vs. women: 84.4±11.8; p<0.01) (Figure 2). Women tended to have more favorable lipid profiles, higher CRP, and lower insulin levels while HOMA-IR levels were similar between men and women (Table 1).

Overall, women showed a greater difference between cSBP and pSBP than men (women: 7.4±5.2 vs. men: 1.0±6.9; p<0.001) (figure 1). The difference between cSBP-pSBP remained greater in white women compared to white men (women: 7.0±5.1 vs. men: 0.88±6.5; p<0.01), as well as in black women compared to black men (women: 8.3±5.3 vs. men: 1.4±7.8; p<0.01).

Because augmentation index is highly correlated to cSBP, multivariable analysis of independent correlates to cSBP-pSBP was conducted excluding AI@75 and AI (table 2). After adjusting for other variables table 2 shows a gender effect consistent with that in figure 1 in total sample as well as in blacks and whites. Multivariable analysis of independent correlates to AI@75 was also performed and showed a gender effect consistent with that in figure 2 in the total sample as well as in blacks and whites (table 3).

Discussion

In this study women showed significantly greater increases in central systolic BP from peripheral systolic BP when compared to men (figure 1). This was true for both black and white women and was independent of age. Our study also shows that in multivariable analysis of independent predictors of cSBP-pSBP that gender is a strong determining factor in differences between central and peripheral pressures (table 2). Thus, it appears that peripherally measured BP underestimates central systolic BP in women compared to men. Importantly, while the women in this study had a mean pSBP of 115.9±15.1 mmHg, a level which could be characterized as “ideal”, their corresponding cSBP was in a range consistent with “prehypertension” (123.7±16.6 mmHg) (21–23). This contrasts with the males in this study whose pSBP (123.3±14.7 mmHg) was almost identical to their cSBP (124.4±16.3 mmHg), with both values consistent with prehypertension. These findings suggest a scenario wherein peripheral BP underestimates central BP, a more potent predictor of target organ damage, and therefore underestimates risk of hypertensive target organ damage in women but not men.

The finding of an increased augmentation index in women compared to men offers a possible explanation for the difference between peripheral and central BP in men compared to women. Augmentation index is a measure of reflected waves and is considered a measure of arterial “stiffness” (24,25). Typically central blood pressure is affected by both a forward pulse wave generated during ventricular contraction and a reflected wave generated at arterial branch points and other areas of impedance mismatch. In normal arteries, the reflected wave arrives centrally during diastole and therefore does not significantly augment central systolic pressure. However, when arteries are stiffened the reflected wave arrives centrally earlier, augmenting the forward wave and increasing the systolic pressure(26–28). In this manner an increased augmentation index, due to increases in vascular stiffness, could be a factor in raising the central systolic BP. Therefore, the higher AI in women in this study may represent decreased arterial elastance than men and may be responsible for the enhancement of central aortic pressure.

Higher AI has been shown to predict adverse cardiovascular events (29–31) and is associated with increases in hypertensive target organ damage (32–34). Consistent with previous reports, women in this study had significantly higher AI (AI in men: 77.8±14 vs. women: 91.3±14.2; p<0.01) and AI@75 than men (men: 68.9±13 vs. women: 84.9±11.5; p<0.01) (6,35,36,37,38). Age is a primary predictor of increasing AI, but heart rate and body

height also contribute (39,40). Results of multivariable analysis in our data indicate that these considerations are not sufficient to explain gender differences in AI, and that gender itself is a strong determining factor of AI@75. This suggests gender differences in arterial structure and function may account for differences in peripheral versus central BP in the present study.

The importance of correctly assessing the central BP was shown by the Conduit Artery Functional Evaluation (CAFE) study, a large sub-study of the Anglo- Scandinavian Cardiac Outcomes Trial (ASCOT)(41). CAFE was one of the first studies to monitor central arterial pressures in a major clinical trial because it had previously erroneously been assumed that peripheral BP accurately predicted central BP. However, the CAFE trial demonstrated that anti-hypertensive medications with varying mechanisms of action differentially affected central and peripheral BP. In this study, while participants had similar control of brachial artery BP, there were significant differences in central pressure between treatment groups, and those with lower central pressures saw a significant reduction in adverse outcomes. Investigators in the CAFE trial were the first to link improved central arterial pressures to reduced adverse cardiovascular events, all-cause mortality and evidence of target organ damage such as renal impairment. The CAFE study was closely followed by an analysis of central versus peripheral pressures in the Strong Heart Study (SHS)(11). Investigators of the SHS found that central pulse pressure obtained by radial applanation tonometry more accurately predicted carotid artery hypertrophy, extent of atherosclerosis and adverse cardiovascular events than did brachial pulse pressure.

A strength of our study is the large number of participants, and high participation by females allowing for gender comparisons of BP. Our study was able to compare commonly used peripheral BP measurements to non-invasive pressure measurement to aid in the interpretation of central to peripheral BP relationships. Weaknesses in this study include a relatively narrow age range of participants, which made it difficult to determine whether our parameter of cSBP-pSBP changed with age. Also, because this is a cross-sectional study we were unable to observe changes over time, and were unable to reliably correlate our measure of cSBP-pSBP with changes in organ function consistent with target organ damage. Finally, our use of the Omron HEM-9000AI device, while shown to provide accurate estimation of pressure amplification that is proportional to other similar devices (18,42), could not be correlated with invasive measurements, the gold standard measure of central pressure.

We conclude that the assessment of peripheral versus central arterial BP is related to gender and to indices of arterial structure and function. Recognition of these differences should be incorporated in clinical management and in evaluation of clinical trial data. In the future non-invasive assessment of central blood pressure may be commonly measured and therefore will offer a useful tool in the clinical evaluation of blood pressure (43).

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Abbreviations

LVH	left ventricular hypertrophy
MI	myocardial infarction

BP	blood pressure
pSBP1	early systolic inflection point of radial artery peripheral arterial waveform
pSBP2	late systolic inflection point of radial artery peripheral arterial waveform
AI	augmentation index
AI@75	augmentation index standardized to heart rate of 75 beats per minute
BMI	body mass index
Trig	triglycerides
HDL-C	high density lipoprotein- cholesterol
LDL-C	low density lipoprotein, cholesterol
HOMA-IR	homeostasis model assessment of insulin resistance
CRP	C-reactive protein
cSBP	central systolic blood pressure
pSBP	peripheral systolic blood pressure
DBP	diastolic blood pressure
HR	heart rate

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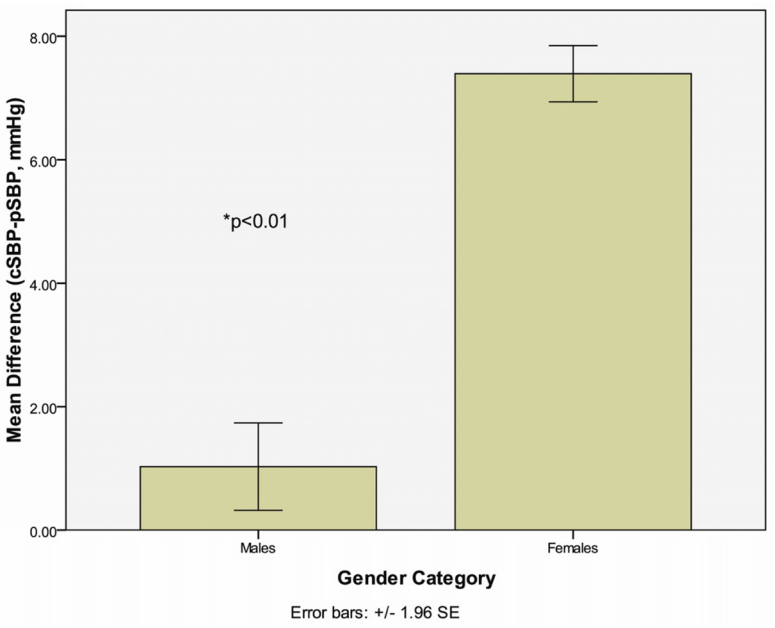

Fig 1.
Difference Between cSBP and pSBP by Gender

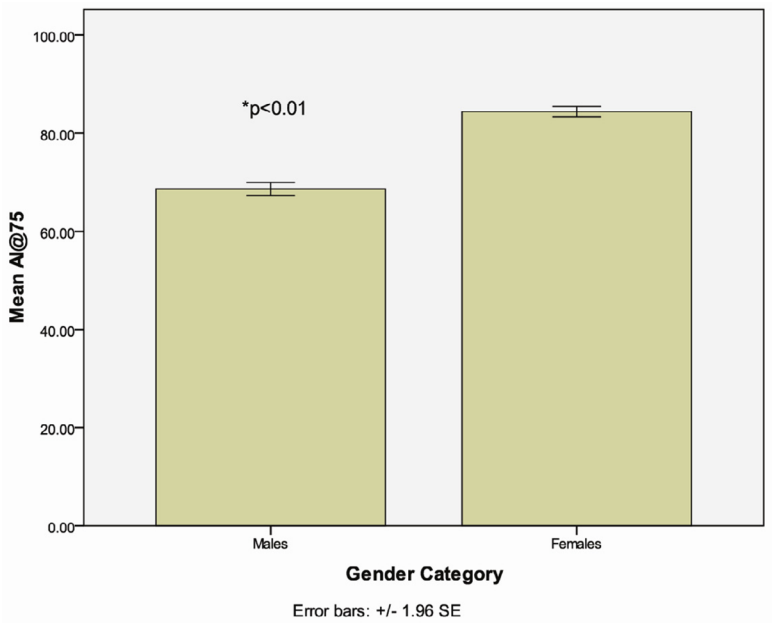

Fig 2.
Augmentation Index Corrected to Heart Rate of 75bpm by Gender

Table 1

Risk factors and hemodynamic variables by total and gender groups

	Total (n= 876)	Males (n= 369)	Females (n=507)	p-value*
Age (yrs)	43.5 (4.4)	43.9 (4.3)	43.2 (4.5)	NS
SBP (mmHg)	119.1 (15.4)	123.3 (14.7)	115.9 (15.1)	<0.01
DBP (mmHg)	75.6 (11.1)	78.4 (10.6)	73.6 (11.1)	<0.01
cSBP (mmHg)	123.8 (17)	124.4 (16.3)	123.4 (17.5)	NS
Heart Rate (bpm)	71.4 (10.9)	70 (11.4)	72.5 (10.4	NS
Weight (Kg)	88.4 (23.3)	97.4 (22.4)	81.4 (21.8)	<0.01
Height (m)	1.7 (0.1)	1.8 (0.1)	1.6 (0.1)	NS
BMI (Kg/m^2)	30.7 (7.5)	30.7 (6.6)	30.8 (8)	NS
AI@75	77.8 (14.5)	68.5 (13)	84.4 (11.8)	<0.01
AI (%)	79.3 (15.2)	70.8 (14)	85.5 (13)	<0.01
Tot Chol (mg/dL)	190.7 (39)	193.5 (41.4)	188.7 (37.1)	<0.01
HDL-C (mg/dL)	46.9 (14.4)	42.6 (14.1)	50 (13.8)	<0.01
LDL-C (mg/dL)	124.8 (34.2)	128.2 (36.5)	122.4 (32.2)	<0.01
Trig (mg/dL)¥	109 (89)	122 (103)	99 (74)	<0.01
CRP (mg/L)¥	1.3 (2.6)	0.9 (1.8)	1.8 (3.4)	<0.01
Insulin (iU/mL)¥	10.1 (11.4)	10.9 (11.3)	9.6 (11.3)	<0.01
HOMA-IR¥	2.3 (3.1)	2.5 (3.1)	2.2 (3)	NS

Data shown in mean and standard deviation (SD), unless otherwise indicated

¥ Median and interquartile range

* Comparison by analysis of covariance, controlling for age.

Table 2

Independent correlates of difference between *cSBP and pSBP* by mutivariable linear regression analyses.

	Model 1: Total (n= 876)		**Model 2: Whites (n= 601)**		**Model 3: Blacks (n= 275)**	
	β	***p* value**	**β**	***p* value**	**β**	***p* value**
Age	0.14	<0.001	0.14	<0.001	0.12	0.09
HR	-0.22	<0.001	-0.24	<0.001	-0.18	<0.01
Height	-0.22	<0.001	-0.23	<0.001	-0.20	0.04
Smoking (Y/N)	0.13	<0.001	0.10	<0.01	0.17	<0.01
HOMA-IR ¥	-0.04	0.35	-0.03	0.51	-0.04	0.57
HDL-C	0.08	0.05	0.07	0.21	0.08	0.31
LDL-C	-0.02	0.59	0.03	0.54	-0.11	0.13
Trig ¥	-0.02	0.62	-0.04	0.41	0.02	0.82
CRP ¥	0.02	0.60	0.04	0.40	-0.01	0.87
Race (W/B)	-0.08	0.03	-	-	-	-
Gender (F/M)	0.18	<0.001	0.17	0.01	0.24	0.02
R^2	0.22		0.22		0.22	

R^2, variance explained

β standardized regression coefficient

¥Log transformed to improve normality

Table 3

Independent correlates of *AI@75* by mutivariable linear regression analyses

	Model 1: Total (n= 876)		**Model 2: Whites (n= 601)**		**Model 3: Blacks (n= 275)**	
	β	***p* value**	**β**	***p* value**	**β**	***p* value**
Age	0.20	<0.001	0.22	<0.001	0.14	<0.01
HR	-0.06	0.04	-0.10	<0.01	0.02	0.638
Height	-0.33	<0.001	-0.35	<0.001	-0.24	<0.01
Smoking (Y/N)	0.14	<0.001	0.14	<0.001	0.13	<0.01
HOMA-IR ¥	-0.08	0.01	-0.09	0.02	-0.10	0.08
HDL-C	0.07	0.02	0.03	0.34	0.13	0.03
LDL-C	0.01	0.62	0.02	0.54	0.01	0.90
Trig ¥	0.04	0.20	0.02	0.62	0.08	0.16
CRP ¥	0.06	0.03	0.12	<0.001	-0.05	0.40
Race (W/B)	-0.09	<0.001	-	-	-	-
Gender (F/M)	0.28	<0.001	0.26	<0.001	0.41	<0.001
R^2	0.43		0.44		0.41	

R^2, variance explained

β standardized regression coefficient

¥Log transformed to improve normality