Low arterial compliance in young African-American males

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Departments of 1Rehabilitation Medicine and 2Psychiatry, 3Mailman School of Public Health, College of Physicians and Surgeons and 4Teachers College, Columbia University, New York 10032; and 5Department of Medicine, Howard University, Washington, District of Columbia 20059

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Zion, Adrienne S., Vernon Bond, Richard G. Adams, Deborah Williams, Robert E. Fullilove, Richard P. Sloan, Matthew N. Bartels, John A. Downey, and Ronald E. De Meersman. Low arterial compliance in young African-American males. Am J Physiol Heart Circ Physiol 285: H457–H462, 2003. First published May 8, 2003; 10.1152/ajpheart.00497.2002.—Hypertension remains a common public health challenge because of its prevalence and increase in co-morbid cardiovascular diseases. Black males have disproportionate pathophysiological consequences of hypertension compared with any other group in the United States. Alterations in arterial wall compliance and autonomic function often precede the onset of disease. Accordingly, our purpose was to investigate whether differences in arterial compliance and autonomic function between young, healthy African-American males without evidence of hypertension and age- and gender-matched non-African-American males. All procedures were carried out noninvasively following rest. Arterial compliance was calculated as the integrated area starting at the well-defined nadir of the incisura of the dicrotic notch to the end of diastole of the radial artery pulse wave. Power spectral analysis of heart rate and blood pressure variability provided distributions representative of parasympathetic and sympathetic modulation and sympathovagal balance. Baroreflex sensitivity (BRS) was calculated using the sequence method. Thirty-two African-American and twenty-nine non-African-American males were comparable in anthropometrics and negative family history of hypertension. t-Tests revealed lower arterial compliance (5.8 ± 2.4 vs. 8.6 ± 4.0 mmHg·s; P = 0.0017), parasympathetic modulation (8.9 ± 1.1 vs. 9.7 ± 1.1 ln ms2; P = 0.0063), and BRS (13.7 ± 7.3 vs. 21.1 ± 8.5 ms/mmHg; P = 0.0007) and higher sympathovagal balance (2.9 ± 3.2 vs. 1.5 ± 1.1; P = 0.03) in the African-American group. In summary, differences exist in arterial compliance and autonomic balance in African-American males. These alterations may be antecedent markers of disease and valuable in the detection of degenerative cardiovascular processes in individuals at risk.

arterial hypertension; autonomic nervous system; baroreflex

HYPERTENSION (HT) in African-American males continues to be a major health challenge because of the staggering financial costs related to medical and disability expenses. The increased prevalence of morbidity and mortality due to HT in this group is among the highest in the world (63). Not only does HT occur more frequently, but it also presents at an earlier age and causes increased complications of cardiovascular diseases compared with white Americans (57). Although the general prevalence of hypertension has decreased among all genders and ethnic groups in the United States, recent reports indicate that HT now ranks as the 13th leading cause of death in the United States, having moved up from its former 15th place. A variety of environmental, behavioral, and biological factors have been proposed to account for the racial differences in the prevalence and severity of HT.

Arterial HT is associated with structural and functional changes in the cardiovascular system. These changes involve the conductance in large arteries as well as the resistance in a small artery (31). In animal models, arterial compliance was attenuated in animals with HT compared with a normotensive group (12). Specifically, carotid arteries were stiffer due in part to increased smooth muscle mass (36). In humans, abnormalities in the diastolic waveform contour, as evidenced by a reduced area under the dicrotic notch and identified as reduced compliance, have been recognized in the Framingham and other studies as a possible marker for cardiovascular morbidity and mortality and in the etiology of arterial disease, HT (42, 50), stroke (2, 33, 52), diabetes (41), and atherosclerosis (32). Increases in arterial wall stiffness lead to an augmentation in the stress-strain relationship, elevating blood pressures and accelerating HT, ultimately inducing atherosclerosis. Alterations in arterial compliance may precede the onset of clinically apparent disease and help to identify individuals at risk before the onset of disease (10, 25). As with arterial wall compliance, there are conditions in which baroreflex sensitivity (BRS) and autonomic function are impaired: coronary artery disease, stroke (2), as demonstrated in the Framingham study (52), atherosclerosis, hypertension (26), diabetes (4, 20), alcoholic neuropathy (28), and in smokers (65). In addition, autonomic function becomes altered and BRS declines as a natural part of the aging process.

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process (9, 14, 17, 44). Underlying age-related changes that are thought to alter baroreflex mechanism include arterial stiffening and a reduced cardiovascular responsiveness to adrenergic stimulation (38). Strong evidence, accounting for racial differences in blood pressure, appears to point toward a decrease in vasodilation during mental and physical stress in normotensive African-American men, which results in an attenuated buffering of blood pressure pulsatility (19, 21).

Therefore, the rationale of this study was to determine whether differences in arterial compliance and autonomic function exist in young male normotensive African-Americans with a negative family history of HT compared with a similar group of non-African-Americans.

METHODS

Thirty-two African-American volunteers were compared with a similar group of 29 non-African-American males recruited from the staff and student body of Columbia and Howard Universities. Before enrollment, potential subjects were screened for general medical history, physical fitness levels, and ancestral history. Subjects were excluded if they had any systemic medical illness, allergies that required medications, currently smoked, or had a positive family history of HT. In accordance with the Institutional Review Boards, subjects provided written informed consent before being tested. Via self-report, individuals with two parents or two grandparents of African descent were assigned to the African-American (AA) group, and those who did not have any parent or grandparent of African descent were assigned to the non-African-American (NAA) group.

Subjects arrived at the laboratory between the hours of 7:00 AM and 10:00 AM Tuesday through Friday after an overnight fast. No testing took place on Mondays, because there may be an exaggerated increase in sympathetic modulation at the start of the work week compared with other days (61). Caffeinated products were not consumed before the testing protocol. After anthropometric measurements were taken, subjects were instrumented with electrocardiograms (Max 2000; Marquette Instruments, Marquette, WI), and beat-by-beat radial blood pressure (BP) (Colin tonometer; Colin, San Antonio, TX) and respiratory recording (YSI temperature probe; Yellow Springs Instrument, Yellow Springs, OH) data were captured via an analog-to-digital conversion board (ATMIO-16; National Instruments, Austin, TX) and stored on a computer. After a 15-min seated equilibration period in which BP, heart rate, and respiratory rate fluctuated <5%, 5 min of resting data were acquired in accordance with published recommendations (60) and sampled at 500 Hz. As previously mentioned, the area under the diastolic pressure waveform of the radial artery was utilized to estimate compliance (37) (Fig. 1). This method of pulse wave analysis has been shown to be a valid and reliable method for the early detection of vascular disease (10). (For a more detailed description of the method as well as its shortcomings, the reader is referred to Ref. 39.) Our laboratory has used a similar contour analysis method in the past to assess arterial compliance (13–15). Specifically, the analysis included two successive 10-beat radial BP waves that were extracted from the beat-to-beat BP recordings. A well-defined nadir of the incisura of the dicrotic notch and the end of diastole delineated the area of integration (37). The resultant values were averaged and yielded an estimate of arterial compliance. Power spectral density analysis of heart rate variability (HRV) and blood pressure variability (BPV) was used to derive measures of autonomic modulation (55). A priori power spectra of R-R intervals within the 0.15- to 0.4-Hz range were defined as the high-frequency (HF) component of HRV (denoted as $\text{HF}_{\text{RR}}$), representing primarily parasympathetic modulation. The low-frequency (LF) component of HRV (0.04–0.15 Hz) is a mixture of both parasympathetic activity and sympathetic activity. Unlike parasympathetic activity, the sympathetic activity is not easily separated from the power spectrum of HRV (27). Sympathovagal balance was computed as the ratio between the LF and HF spectra of HRV (49). LF BP modulation in the 0.04- to 0.15-Hz range represents sympathetic vasomotor activity (48, 51). All spectral data were log transformed to remove skewness and minimize the large standard deviations customarily present in these data. Digitized R-R intervals and in-phase systolic peaks determined spontaneous BRS by a modification of the sequence technique (5), shown to have a high correlation to invasive methods (26). After these assessments were completed, maximum oxygen consumption ($\text{VO}_{2 \text{max}}$) capacity tests were performed by using open-circuit spirometry (Max 1 metabolic system; Physiodyne, Farmingdale, NY) according to a standard incremental cycling protocol (7). Classic criteria were used to determine whether a maximal effort had been achieved (7).

Statistical analyses. Independent $t$-tests were performed for all variables pertaining to the hypotheses. As a secondary analysis, a discriminant analysis was computed based on Fisher’s procedures to determine the relative strength of the arterial compliance, autonomic variables, and BRS in distinguishing the members of one racial group from another and in correctly predicting the racial group to which each sample participant belonged. Data are presented as means ± SD. Probability for all analyses was set at $P < 0.05$.

RESULTS

All subjects were American born, listed English as their primary language, and had no familial history of
HT. The AA group consisted of 32 males, and the NAA group consisted of 29 males. The racial distribution of the NAA group was 55% Caucasian (16/29), 31% Asian (9/29), and 14% Hispanic of non-African descent (4/29). Descriptive physical characteristics of study subjects by group are presented in Table 1. Mean ages, heights, weights, and body mass indexes were similar in both groups, and there were no differences between groups for VO2max values, resting heart rates, systolic BP (SBP), and respiratory rates. Results of arterial compliance, autonomic modulation, and BRS (t-tests) are presented in Table 2. The AA group had significantly lower arterial compliance (5.8 ± 2.4 vs. 8.6 ± 4.0 mmHg·s; P = 0.0017), HFRR (8.9 ± 1.1 vs. 9.7 ± 1.1 ms²; P = 0.0063), and BRS (13.7 ± 7.3 vs. 21.1 ± 8.5 ms/mmHg; P = 0.0007) and a higher LF/HF ratio (2.9 ± 3.2 vs. 1.5 ± 1.1; P = 0.03) than the NAA group. The LFRR and LFSBP components (sympathetic modulation) failed to reach significance (Table 2). The test of significance (F value) was used to determine whether the discriminant function equation was able to predict an independent variable more accurately than by chance alone. Arterial compliance, HFRR, LFSBP, LF/HF, and BRS were the variables in the overall model. Probability value was P < 0.0001. The significance of the univariate F ratio (7.67) indicated that when the predictors were considered individually, all except LFSBP modulation significantly differentiated race. The ANOVA of discriminant scores revealed no within-group differences. The F ratio for between-group differences was 41.18 (P = 0.00). Finally, to further assess the accuracy of the analysis, we computed discriminant scores for each sample participant and compared each participant’s predicted racial group with his actual race. Approximately 77% of the cases were correctly classified (P < 0.05).

Our major results demonstrate that the AA group had lower arterial compliance, HFRR (parasympathetic modulation), and BRS and a higher LF/HF power ratio (sympathovagal balance). Within the discriminant analysis model, baroreflex sensitivity was the single best predictor of race and was closely followed by arterial compliance.

DISCUSSION

The results of this new investigation demonstrate that young, normotensive African-American males have differences in arterial compliance and autonomic modulation compared with a similar group of non-African-American males. The loss of arterial compliance has been proposed as a possible mechanism in the initiation, progression, and etiology of HT and as a prognostic marker of cardiovascular disease (2). Once arterial wall degeneration is initiated, a positive feed back loop may be established whereby degeneration leads to pressure increases, ultimately leading to further vascular degeneration (46). In physiological conditions such as aging or HT, increased wall thickness and luminal diameter of large arteries reduce vascularity of bodily organs and tissues (54). As the artery dilates, wall tension and pulsatile stresses increase and accentuate arterial wall degeneration that has already occurred. Assessments of large artery stiffness in HT are complicated by the role of BP in determining values of both pulse pressure and diameter changes. If the index used does not remove the effects of intraindividual variation in BP, a residual association with HT is not unexpected (2). In this investigation, all subjects for both groups were normotensive and of similar heights; thus changes in arterial compliance were not dependent on elevations in BP or height. The method used here to derive arterial compliance measured the area under the radial artery pressure wave during diastole. We chose the diastolic phase because nearly two-thirds of the cardiac cycle comprises diastole, and, as such, any change in compliance during this phase will have a pronounced effect on overall BP. In addition, and unlike the brachial artery, the radial

Table 1. Physical characteristics of subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>African-American</th>
<th>Non-African-American</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>32</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Age, yr</td>
<td>21.6 ± 2.6</td>
<td>23.9 ± 3.6</td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>179.5 ± 6.7</td>
<td>176.0 ± 5.3</td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77.7 ± 10.4</td>
<td>76.8 ± 11.7</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.1 ± 2.9</td>
<td>24.7 ± 3.9</td>
<td></td>
</tr>
<tr>
<td>VO2max, ml·kg⁻¹·min⁻¹</td>
<td>34.0 ± 5.8</td>
<td>36.7 ± 3.7</td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>68.8 ± 8.8</td>
<td>69.6 ± 9.6</td>
<td></td>
</tr>
<tr>
<td>SBP mmHg</td>
<td>114.9 ± 12.6</td>
<td>119.2 ± 13.3</td>
<td></td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>76.7 ± 7</td>
<td>74 ± 9</td>
<td></td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>89 ± 4</td>
<td>89 ± 3</td>
<td></td>
</tr>
<tr>
<td>RR, breaths/min</td>
<td>12.5 ± 2.8</td>
<td>12.4 ± 2.7</td>
<td></td>
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</tbody>
</table>

Values are means ± SD. No significant differences were found between any of the variables. BMI, body mass index (weight/height); VO2max, maximum oxygen consumption; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial blood pressure; RR, respiratory rate.

Table 2. Arterial compliance, autonomic modulation, and BRS results

<table>
<thead>
<tr>
<th>Variable</th>
<th>African-American</th>
<th>Non-African-American</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial compliance, mmHg·s</td>
<td>5.8 ± 2.4</td>
<td>8.6 ± 4.0</td>
<td>0.0017</td>
</tr>
<tr>
<td>Heart rate variability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HF, ln ms²</td>
<td>8.9 ± 1.1</td>
<td>9.7 ± 1.1</td>
<td>0.0063</td>
</tr>
<tr>
<td>LF, ln ms²</td>
<td>9.7 ± 1.5</td>
<td>10.4 ± 1.7</td>
<td>0.12</td>
</tr>
<tr>
<td>LF/HF</td>
<td>2.9 ± 3.2*</td>
<td>1.5 ± 1.1</td>
<td>0.03</td>
</tr>
<tr>
<td>Blood pressure variability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LF, ln mmHg²</td>
<td>5.07 ± 1.5</td>
<td>5.40 ± 1.0</td>
<td>0.15</td>
</tr>
<tr>
<td>BRS, ms/mmHg</td>
<td>13.7 ± 7.3</td>
<td>21.1 ± 8.5</td>
<td>0.0007</td>
</tr>
</tbody>
</table>

Values are means ± SD. HF, high-frequency component; LF, low-frequency component; BRS, baroreflex sensitivity. *P ≤ 0.05.
artery is a resistance artery composed of smooth muscle and largely devoid of atherosclerosis. Moreover, because of the group similarities in body habits, \( V_O^2_{max} \), resting BPs, heart rates, and respiration, we eliminated confounding influences often present in the measurement of arterial compliance.

**Arterial compliance.** In the current investigation, the lower arterial compliance was seen in the presence of an augmentation in sympathovagal balance. This augmentation has been shown to further reduce distensibility of small, medium, and large arteries, resulting in a tonic restraint of elastic and resistance type vessels (3, 6, 11, 24, 43). Independently and in combination, the loss of arterial compliance and alterations in autonomic function are associated with increased HT risk. The age-associated increase in arterial stiffness manifests as a progressive increase in SBP (35). Arterial stiffening (decreased distensibility) leads to changes in the arterial blood pressure contour (62) and increases in pulse pressure (47). Stiffening of the arterial tree has been recognized for more than a century from clinical and pathological studies as a major risk factor. Although arterial stiffness was originally regarded as a benign concomitant of the aging process, it is now acknowledged that additional factors exert significant influence on arterial stiffness, including ethnic and genetic differences as well as dietary and activity habits (62).

**BRS.** An additional finding of this study was the attenuation in BRS in the AA group. The baroreceptor response to sustained pressure on the arterial side of the circulation produces an afferent connection to the vasomotor and cardioinhibitory areas, and the efferent pathways from these areas constitute a reflex feedback mechanism that operates to stabilize the BP and heart rate. When the arterial system is less compliant, this results in a continuous increased stretch of the baroreceptors, causing a downregulation, i.e., a loss of sensitivity, in turn altering autonomic function by lowering parasympathetic modulation and increasing sympathetic outflow. Support for this attenuation in BRS has been observed in normotensive relatives of hypertensive patients (18, 59). Furthermore, the loss of BRS has been shown to be a major risk factor for cardiovascular morbidity and mortality in postmyocardial infarction patients (34, 53). We are not aware of any comparative data of BRS between young, normotensive African-American and comparable non-African-American males. Moreover, differences in baroreflex sensitivity here emerged as a significant discriminator between the races.

**Parasympathetic modulations.** Efferent autonomic signals are modulated by BRS, which that in turn are tethered to viscoelastic elements in the arterial walls. Our findings indicate an attenuation of HFRR or parasympathetic modulation in the AA group compared with the NAA group. The decrease in parasympathetic activity at rest may be a physiological consequence of the associated changes in arterial compliance and BRS. We are aware of the cross-sectional nature of this investigation and therefore are unable to delineate the temporal sequence of these parameters. However, it is clear that the loss of parasympathetic modulation is a powerful predictor of cardiovascular morbidity and mortality in cardiac patients (34, 53, 56).

**Sympathetic modulations.** Unlike parasympathetic activity, the sympathetic activity is not easily separated from the power spectrum of HRV (27). Therefore, sympathovagal balance is a better concept used to isolate sympathetic activity that recognizes both reciprocal and nonreciprocal parasympathetic and sympathetic influences on heart rate by computing the ratio between the LF and HF spectra (49). This LF/HF ratio in the AA group was found to be less favorable compared with the NAA group. An altered vascular reactivity has been one postulated mechanism for the high incidence of HT in black men (1, 29). This may be explained by genetic differences linked to polymorphic variations in \( \alpha \)-adrenergic receptors having greater peripheral vascular sensitivity to norepinephrine (22, 40) and/or attenuations in \( \beta \)-adrenergic receptor-mediated vasodilation (58, 64). Environmental loci responsible for affecting autonomic balance suggest that the diminished vascular reactivity exhibited in African-Americans is due to chronic stressors such as perceived racisms, discriminations, and unfair treatment that lead to observable differences in physiological function (8). In addition, reduced sources of social support in this population are linked to an increased risk for the development of cardiovascular disease (16). Our findings demonstrate a loss of parasympathetic modulation rather than an augmentation of sympathetic modulation. Regardless of what branch of the autonomic nervous system has shifted, the outcome leads to a disturbance in sympathovagal balance, a common denominator in the future development of HT (29, 30).

This cross-sectional investigation provides for the first time evidence that arterial wall compliance and autonomic function differ between African-American and age-matched non-African-American men. The findings in this cross-sectional investigation may be suggestive that the establishment of clinical disease may occur much earlier than believed. On the basis of public health statistics, it is conceivable that these subjects may develop HT at a future point in their lives. Accordingly, when preclinical markers of disease risk are verified in young asymptomatic African-Americans, aggressive behavioral adaptations should be made early to minimize the progression and onset of cardiovascular disease. As a consequence of early recognition and effective therapy, hypertensive disease is rarely a problem in the young (45). The methods to evaluate arterial compliance and autonomic function utilized in this study were safe, noninvasive, and caused no discomfort to the subjects. They are relatively inexpensive and may easily be incorporated into a clinical site physical examination. Thus preclinical detection can make early interventions possible. Because health outcomes are shaped by racial inequality (23), results from this study could raise awareness toward the initiation of a screening and detection pro-
gram using noninvasive assessments to target individuals at risk.

DISCLOSURES

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