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Arterial elasticity in American Indian and Caucasian children, adolescents, and young adults

Andrew W Gardner[1] and Donald E Parker[2]

Abstract

We compared arterial elasticity in American Indian and Caucasian children, adolescents, and young adults, and we assessed whether demographic, body composition, and ambulatory activity measures were predictive of arterial elasticity within each group. Fifty-one American Indians and 66 Caucasians between the ages of 8 and 30 years were assessed on large artery elasticity index, small artery elasticity index, body fat percentage, and daily ambulatory activity during 7 consecutive days. American Indians had a higher percentage of body fat than Caucasians ($p = 0.002$), whereas daily ambulatory activity measures were similar ($p > 0.05$). American Indians had a 16% lower large artery elasticity index ($p = 0.007$) and a 19% lower small artery elasticity index ($p < 0.001$) than Caucasians. The regression model for large artery elasticity index included average cadence ($p = 0.001$), fat-free mass ($p < 0.001$), age component (Caucasian only) ($p < 0.001$), and sex ($p = 0.025$). The regression model for small artery elasticity index included fat-free mass ($p < 0.001$), maximum cadence for 30 continuous minutes ($p = 0.009$), race ($p = 0.005$), and average cadence ($p = 0.049$). Between 8 and 30 years of age, elasticity means for the large and small arteries is lower in American Indians than in Caucasians. A smaller difference was observed in children, with a trend to a much larger difference in young adults. Furthermore, greater fat-free mass and higher daily ambulatory cadence are associated with higher arterial elasticity in both American Indians and Caucasians.

Keywords

ambulation; American Indian; arterial elasticity; physical activity

Introduction

Arterial elasticity is a non-invasive marker predictive of cardiovascular events.[1,2] Arterial elasticity is impaired with modifiable cardiovascular risk factors such as smoking,[3–5] hypertension,[1,4,6–9] increased cholesterol,[1,6,7,9] elevated low-density lipoprotein cholesterol,[5,6,9,10] increased triglycerides,[4,5,10] diabetes,[1,11] elevated insulin[4,10] and glucose[7,10] levels, increased high-sensitive C-reactive protein,[7] and increased levels of endothelial biomarkers P-selectin and urinary albuminuria.[4] In contrast, exercise increases arterial elasticity,[9,12–15] with self-reported vigorous physical activity having a protective effect in a longitudinal follow-up of young adults.[16]

American Indians are particularly susceptible to high rates of cardiovascular risk factors such as diabetes,[17,18] hypertension and low levels of high-density lipoprotein cholesterol,[18] cardiovascular disease,[17] and all-cause and cardiovascular mortality.[17,19] Additionally, American Indian adults and children have low levels of physical activity,[20,21] which contribute to the incidence of diabetes[22] and increases the risk of cardiovascular morbidity and mortality.[23–25] Arterial elasticity has not been well studied in American Indians, particularly in youths who have yet to develop cardiovascular complications.

The purposes of this study are (1) to compare arterial elasticity in American Indian and Caucasian children, adolescents, and young adults, and (2) to assess whether demographic, body composition, and ambulatory activity measures are predictive of arterial elasticity within each group. We hypothesized that arterial elasticity is lower in American Indian youth, and that objectively measured daily ambulatory activity is associated with arterial elasticity in both groups.

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Methods

Subjects

A total of 117 healthy subjects between 8 and 30 years of age participated at the General Clinical Research Center and the Children's Medical Research Institute Diabetes and Metabolic Research Program from February 2007 to November 2009. The subjects were recruited by local newspaper advertisements, university email advertisements, and informational flyers distributed in Oklahoma City and surrounding areas. Owing to the potential confounding influences on the study outcome measures, subjects were excluded for the following criteria: (a) being under treatment for hypertension, (b) being under treatment for dyslipidemia, (c) current smoker, (d) insulin- or non-insulin-dependent diabetes mellitus, (e) use of oral contraceptives, and (f) a history of any form of cardiovascular disease, pulmonary disease, renal disease, liver disease, or active cancer. Subjects agreed to participate by signing the informed consent form and child's assent form approved by the Institutional Review Board at the University of Oklahoma Health Sciences Center.

Measurements

Primary outcome measures: large artery elasticity index and small artery elasticity index

Diastolic pulse contour analysis test

Instrumentation. Arterial elasticity measurements were obtained by an HDI/PulseWave™ CR-2000 Research Cardiovascular Profiling System (Hypertension Diagnostics, Inc., Eagan, MN, USA), which analyzes the shape of arterial pressure waves produced by heart beats. This system determines large artery elasticity index and small artery elasticity index by gathering and analyzing a 30-second analog tracing of radial artery waveforms digitized at 200 samples per second using a non-invasive, direct contact acoustic transducer.[6] A beat determination is made using a beat-marking algorithm during the 30-second period,[26] which determines systole, peak systole, onset of diastole, and end diastole. The beat is then incorporated into a parameter-estimating algorithm, and measures of arterial elasticity are calculated from the decline in diastolic blood pressure using a modified Windkessel model.[27] Diastolic pulse contour analysis by the modified Windkessel model separates the diastolic waveform into a declining exponential wave and sinusoidal oscillating wave. The decline in the exponential wave is a function of the elasticity of large arteries (capacitive arterial compliance), and the decline in the sinusoidal oscillating wave is a function of the elasticity of the small arteries (oscillatory or reflective arterial compliance) of the most peripheral vessels.[28]

Procedures. Arterial elasticity measurements were obtained in the morning following an overnight fast of at least 8 hours, prior to engaging in any strenuous physical activity, and following 5–10 minutes of rest in the supine position.[29–32] An appropriately sized blood pressure cuff was placed around the subject's left upper arm, and a rigid plastic wrist stabilizer was placed on the subject's right wrist to minimize wrist movement and stabilize the radial artery during the measurement. An Arterial PulseWave™ Sensor (Hypertension Diagnostics, Inc.) was placed on the skin directly over the radial artery at the point of the strongest pulse, while the arm rested in a supine position. The non-invasive acoustic sensor was adjusted to the highest relative signal strength, and a calibration period of several minutes was performed to obtain stable arterial waveforms. Subsequently, arterial waveforms were recorded for 30 seconds and the diastolic portion was digitized at 200 samples per second to determine large artery elasticity index and small artery elasticity index values,[7] which assess the elasticity of the large and small arteries throughout the arterial system. Measurements were averaged over three continuous 30-second trials.

Outcome measures and procedures. The diastolic pulse contour analysis test was performed to obtain the primary outcome measures of large artery elasticity index and small artery elasticity index. The HDI/PulseWave CR-2000 Research Cardiovascular Profiling System converted the large artery elasticity index value to a whole number by multiplying the units (ml × $mmHg^{-1}$) by 10, and it converted the small artery elasticity index value to a whole number by multiplying the units (ml × $mmHg^{-1}$) by 100. In addition to large artery elasticity index and small artery elasticity index, a battery of other cardiovascular parameters were obtained from the HDI/PulseWave unit including systolic blood pressure, diastolic blood pressure, mean arterial pressure, pulse pressure, heart rate, estimated cardiac output, and systemic vascular resistance. Using the above procedures, the test-retest intraclass reliability coefficient is $R = 0.87$ for large artery elasticity index and $R = 0.83$ for the small artery elasticity index.[30]

Secondary outcome measures

Medical screening. Subjects were evaluated during a medical history and physical examination. Demographic information, pubertal status by Tanner stage, height, weight, body mass index (BMI), cardiovascular risk factors, co-morbid conditions, and a list of current medications were obtained.

Ambulatory activity monitoring

Instrumentation and procedures. Daily ambulatory activity was assessed using a step activity monitor (Step Watch 3; Cyma Inc., Mountlake Terrace, WA, USA), as previously described.[33] Ambulatory activity was measured during 7 consecutive days in which subjects were instructed to wear the monitor during waking hours and to remove it before retiring to bed. The step activity monitor was attached to the right ankle above the lateral malleolus using elastic Velcro straps, and continuously recorded the number of steps taken on a minute-to-minute basis. The accuracy of the step activity monitor exceeds 99 ± 1% in older adults,[33] as well as in children.[34]

Variables obtained from the step activity monitor. The step activity monitor records the number of ambulatory

Table 1. Clinical characteristics and body composition of the subjects

Variables	Caucasians (*n* = 66)	American Indians (*n* = 51)	*p*-value
Age, years	17.9 (5.6)	16.7 (5.8)	0.259
Tanner stage	4.2 (1.1)	4.1 (1.2)	0.876
Weight, kg	66.5 (27.1)	69.2 (25.6)	0.594
Height, cm	166.5 (12.5)	162.5 (12.8)	0.096
Body mass index, kg/m^2	23.6 (8.2)	25.7 (7.9)	0.161
Waist–hip ratio	0.83 (0.07)	0.85 (0.07)	0.107
Body fat percentage, %	22.6 (9.1)	28.4 (10.1)	0.002
Fat mass, kg	16.2 (12.8)	20.6 (12.8)	0.072
Fat-free mass, kg	50.0 (18.0)	47.8 (14.3)	0.477
Sex, % males	58	41	0.079

Values are either means (standard deviation) or percentage of males.

Table 2. Ambulatory activity of subjects recorded during a 7-day monitoring period

Variables	Caucasians (*n* = 66)	American Indians (*n* = 51)	*p*-value
Total ambulatory strides (strides/day)	5281 (1909)	4987 (1967)	0.419
Total ambulatory time (min/day)	346 (92)	329 (104)	0.334
Average cadence (strides/min)	15.2 (3.7)	15.0 (3.0)	0.78
Maximum 1-minute cadence (strides/min)	55.9 (5.3)	54.6 (6.1)	0.196
Maximum 5-minute cadence (strides/min)	44.9 (7.3)	43.2 (8.0)	0.225
Maximum 20-minute cadence (strides/min)	30.5 (8.3)	28.6 (8.1)	0.200
Maximum 30-minute cadence (strides/min)	26.7 (8.4)	24.5 (7.8)	0.157
Maximum 60-minute cadence (strides/min)	19.9 (7.2)	18.3 (6.5)	0.224
Peak ambulatory index (strides/min)	41.8 (7.7)	40.4 (7.7)	0.346

Values are means (SD).

strides taken per minute for each minute throughout a 24-hour period. After downloading data from the step activity monitor to a computer, the software program displays the number of strides taken and the number of minutes spent ambulating each day, from which an average daily cadence is calculated. Any minute in which ambulation occurred (i.e. at least one stride was recorded) was defined as an active minute, and the total number of strides were divided by the total number of active minutes to yield an average cadence of ambulation. The daily ambulatory strides and time are further analyzed by the software program, and are quantified into the maximum cadences for 20 and 30 continuous minutes (i.e. the highest ambulatory cadence for 20 and for 30 continuous minutes each day). All of these outcome measures are recorded and averaged for each day, and then the daily averages are averaged over the 7-day monitoring period. In apparently healthy adults, the test-retest intraclass reliability coefficient for the measurement of total daily strides and total daily minutes of activity over the 7-day period are R = 0.94 and R = 0.91, respectively.[33] The intraclass reliability coefficients for the remaining variables pertaining to daily ambulatory cadences range from R = 0.83 to R = 0.94.[33]

Body-composition assessment. Following an overnight fast of at least 8 hours, body fat percentage, fat mass, and fat-free mass were obtained using a BC-418 eight-electrode bio-electrical impedance analysis device (Tanita Corp., Tokyo, Japan).[35–37] Subjects stood barefoot on the base of the unit, which has two stainless-steel rectangular foot-pad electrodes fastened to a metal platform set on force transducers. During this measurement, subjects held hand-grip electrodes. The system has a total of eight electrodes, two for each hand and foot. All electrodes are connected to a digital circuit board. Age, height, and body type (all classified as standard and none classified as athletic) were entered for each subject for calculation of body fat percentage. The assessment of body composition through bio-electrical impedance using model BC-418 has been validated against measurements obtained from dual-energy X-ray absorptiometry in males and females ranging in age from 6 to 64 years.[36]

Statistical analyses

For each measurement variable the initial comparison between means of two groups was made using an independent *t*-test. Chi-square was used to compare proportions. For both large artery elasticity index and small artery elasticity index the regression on each of the clinical, ambulatory activity, and body composition variables within each group was obtained and compared for the difference in slopes across groups. For both large artery elasticity index and small artery elasticity index the relationship of race and sex was investigated using general linear model (GLM) two-factor ANOVA. A multiple regression model was produced by backward stepwise linear regression, with inclusion $p = 0.05$, and switching $p = 0.10$ for large artery

Table 3. Hemodynamic measures of subjects

Variables	Caucasians (n = 66)	American Indians (n = 51)	p-value
Systolic blood pressure (mmHg)	114 (11)	113 (11)	0.806
Diastolic blood pressure (mmHg)	61 (7)	62 (9)	0.477
Mean arterial pressure (mmHg)	80 (8)	80 (9)	0.836
Pulse pressure (mmHg)	53 (7)	52 (7)	0.267
Heart rate (beats/min)	68 (13)	71 (12)	0.124
Cardiac output (ml/min)	5.8 (1.1)	5.7 (1.1)	0.726
Systemic vascular resistance (dyne × s^{-1} × cm^{-5})	1103 (160)	1181 (306)	0.078
LAEI (ml × $mmHg^{-1}$) × 10	16.7 (5.2)	14.1 (4.7)	0.007
SAEI (ml × $mmHg^{-1}$) × 100	8.9 (2.5)	7.2 (2.6)	< 0.001

Values are means (SD).
LAEI, large artery elasticity index; SAEI, small artery elasticity index.

Table 4. Association between large artery elasticity with clinical, ambulatory, and hemodynamic measures

Variables	Caucasians		American Indians	
	$Slope_1$	R^2	$Slope_2$	R^2
Clinical measures				
Age	0.522†	0.307***	0.193†	0.058
Tanner stage	2.108	0.206***	1.498	0.151**
Weight	0.045	0.054	0.061	0.111*
Height	0.229	0.297***	0.203	0.310***
Body mass index	0.020	0.001	0.068	0.013
Waist–hip ratio	12.663	0.042	−0.715	0.000
Body fat percentage	−0.059	0.011	0.014	0.001
Fat mass	0.019	0.002	0.093	0.064
Fat-free mass	0.168	0.223***	0.145	0.197***
Ambulatory measures				
Total ambulatory strides	0.001	0.044	0.000	0.018
Total ambulatory time	−0.003	0.003	−0.011	0.003
Average cadence	0.535	0.143**	0.433	0.077*
Maximum 1-minute cadence	0.176	0.031	0.104	0.018
Maximum 5-minute cadence	0.215	0.087*	0.102	0.031
Maximum 20-minute cadence	0.204	0.102*	0.086	0.022
Maximum 30-minute cadence	0.190	0.091*	0.083	0.019
Maximum 60-minute cadence	0.167	0.052	0.078	0.012
Peak ambulatory index	0.113	0.027	0.091	0.022
Hemodynamic measures				
Systolic blood pressure	0.018	0.001	−0.005	0.000
Diastolic blood pressure	0.168	0.050	0.044	0.007
Mean arterial pressure	0.016	0.001	0.03	0.003
Pulse pressure	−0.116	0.026	−0.072	0.013
Heart rate	−0.118	0.083*	−0.147	0.145**
Cardiac output	0.405	0.007	1.913	0.200**
Systemic vascular resistance	−0.005	0.027	−0.006	0.163**

*Significant linear regression $p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
†Significant group difference in slopes $p < 0.05$.

elasticity index regressed on the following measures: clinical, ambulatory activity, and body composition set of independent variables, augmented with a race variable, and the significant interaction of race and age that was detected in the initial slope comparisons. A multiple regression model for the small artery elasticity index was produced in a like manner. All analyses were performed using the NCSS statistical package (NCSS Inc., Kaysville, UT, USA).

Results

No significant differences in clinical characteristics were observed between the American Indians and Caucasians ($p > 0.05$), except that the American Indians had a higher percentage of body fat ($p = 0.002$) (Table 1). Furthermore, non-significant trends existed for the American Indians having a greater fat mass ($p = 0.072$) and lower percentage

Table 5. Association between small artery elasticity with clinical, ambulatory, and hemodynamic measures

Variables	Caucasians		American Indians	
	$Slope_1$	R^2	$Slope_2$	R^2
Clinical measures				
Age	0.0923	0.042	0.0205	0.002
Tanner stage	0.526	0.056	0.439	0.043
Weight	0.0438	0.222***	0.0290	0.084*
Height	0.0734	0.133**	0.0948	0.224***
Body mass index	0.1238	0.165**	0.0422	0.017
Waist–hip ratio	10.6556	0.109*	3.9353	0.016
Body fat percentage	0.0756†	0.08*	−0.0313†	0.015
Fat mass	0.0839	0.196***	0.0147	0.005
Fat-free mass	0.0819	0.242**	0.0740	0.170**
Ambulatory measures				
Total ambulatory strides	0.0001	0.012	0.0001	0.001
Total ambulatory time	−0.0009	0.001	−0.0023	0.009
Average cadence	0.1465	0.047	0.044	0.003
Maximum 1-minute cadence	0.0163	0.001	0.0029	0.000
Maximum 5-minute cadence	0.0168	0.002	0.0362	0.013
Maximum 20-minute cadence	0.0491	0.026	0.0556	0.031
Maximum 30-minute cadence	0.0523	0.03	0.0535	0.027
Maximum 60-minute cadence	0.0565	0.026	0.0419	0.011
Peak ambulatory index	0.0234	0.005	0.0441	0.017
Hemodynamic measures				
Systolic blood pressure	0.0312	0.017	−0.1474	0.004
Diastolic blood pressure	0.0228	0.004	−0.0357	0.014
Mean arterial pressure	0.0445	0.000	−0.0124	0.002
Pulse pressure	0.0445	0.017	0.0134	0.001
Heart rate	−0.0431††	0.048	−0.0811††	0.145**
Cardiac output	1.0407	0.2***	1.1452	0.236***
Systemic vascular resistance	−0.0068	0.188***	−0.004	0.23***

*Significant linear regression $p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
†Significant group difference in slopes $p < 0.05$, ††$p < 0.01$.

of males ($p = 0.079$). As shown in Table 2, the groups also were similar ($p > 0.05$) on all measures of ambulatory activity during a 7-day monitoring period. No significant differences in hemodynamic measures were noted between the American Indians and Caucasians ($p > 0.05$), except for the primary outcome measures in which the American Indians had a 16% lower large artery elasticity index ($p = 0.007$) and a 19% lower small artery elasticity index ($p < 0.001$) (Table 3). A non-significant trend also was observed for the American Indians having a 7% higher systemic vascular resistance ($p = 0.078$).

In American Indians, large artery elasticity index was correlated with Tanner stage ($p < 0.01$), weight ($p < 0.05$), height ($p < 0.001$), fat-free mass ($p < 0.001$), average cadence ($p < 0.05$), heart rate ($p < 0.01$), cardiac output ($p < 0.01$), and systemic vascular resistance ($p < 0.01$) (Table 4). In Caucasians, large artery elasticity index was correlated with age ($p < 0.001$), Tanner stage ($p < 0.001$), height ($p < 0.001$), fat-free mass ($p < 0.001$), average cadence ($p < 0.01$), maximum cadence for 5 ($p < 0.05$), 20 ($p < 0.05$), and 30 ($p < 0.05$) continuous minutes, and heart rate ($p < 0.05$). The slope between large artery elasticity index and age was lower in American Indians than in Caucasians ($p < 0.05$), indicating that American Indians had a blunted increase in large artery elasticity index across age.

In American Indians, the small artery elasticity index was correlated with weight ($p < 0.05$), height ($p < 0.001$), fat-free mass ($p < 0.01$), heart rate ($p < 0.01$), cardiac output ($p < 0.001$), and systemic vascular resistance ($p < 0.001$) (Table 5). In Caucasians, the small artery elasticity index was correlated with weight ($p < 0.001$), height ($p < 0.01$), BMI ($p < 0.01$), waist–hip ratio ($p < 0.05$), body fat percentage ($p < 0.05$), fat mass ($p < 0.001$), fat-free mass ($p < 0.01$), cardiac output ($p < 0.001$), and systemic vascular resistance ($p < 0.001$). The slope between the small artery elasticity index and body fat percentage was lower in American Indians than in Caucasians ($p < 0.05$), as was the slope between the small artery elasticity index and heart rate ($p < 0.05$).

For both large artery elasticity index and small artery elasticity index, the GLM ANOVA revealed a significant race main effect ($p < 0.05$), with American Indians having lower values than Caucasians, but not a significant sex main effect or race by sex interaction. The regression models for large artery elasticity index and small artery elasticity index in American Indians and Caucasians combined are shown

Table 6. Regression coefficient summary for independent variables used in regression models for large artery elasticity index (LAEI) and small artery elasticity index (SAEI)

Dependent variables	Predictors	Regression coefficient	95% CI	Partial R^2	p-value
LAEI (ml × mmHg^{-1}) × 10*	Average cadence (strides/min)	0.4633	0.1860 to 0.7405	0.0914	0.001
	Fat-free mass (kg)	0.1506	0.0929 to 0.2082	0.1974	< 0.001
	Age component (Caucasian only)	0.1606	0.0820 to 0.2392	0.1308	< 0.001
	Sex (male)	−1.8744	−3.5097 to −0.2391	0.0452	0.025
	Intercept	0.5992	−4.0070 to 5.2054		
SAEI (ml × mmHg^{-1}) × 100**	Race (Caucasian)	1.2268	0.3864 to 2.0673	0.0713	0.005
	Maximum 30-minute cadence (strides/min)	0.1074	0.0273 to 0.1875	0.0608	0.009
	Fat-free mass (kg)	0.0846	0.0555 to 0.1137	0.2334	0.001
	Average cadence (strides/min)	−0.2360	−0.4714 to −0.0006	0.0350	0.049
	Intercept	4.0291	1.4874 to 6.5707		

*Overall model results for LAEI: $R^2 = 0.3806, p < 0.001$.
**Overall model results for SAEI: $R^2 = 0.3202, p < 0.001$.

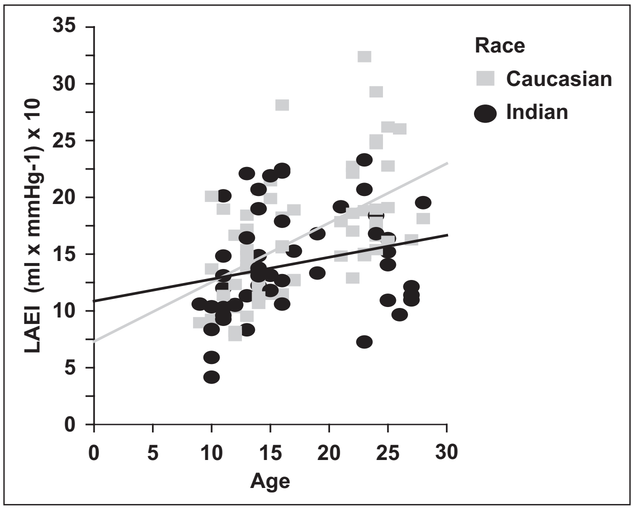

Figure 1. Large artery elasticity index (LAEI) across age in American Indians and Caucasians.

in Table 6. The significant predictors in the multiple linear regression model for large artery elasticity index include average cadence ($p = 0.001$), fat-free mass ($p < 0.001$), age component (Caucasian only) ($p < 0.001$) (Figure 1), and sex ($p = 0.025$). The significant predictors for the small artery elasticity index include fat-free mass ($p < 0.001$), maximum cadence for 30 continuous minutes ($p = 0.009$), race ($p = 0.005$), and average cadence ($p = 0.049$).

Discussion

Ethnicity and arterial elasticity

A novel finding in this study was that arterial elasticity means were lower in American Indians less than 30 years of age than in corresponding Caucasians. It is particularly noteworthy that lower arterial elasticity values were evident in American Indians despite no group differences for systolic blood pressure, diastolic blood pressure, mean arterial pressure, and pulse pressure. Consequently, arterial elasticity, as measured by large artery elasticity index and small artery elasticity index, may be a more sensitive marker of subclinical vascular dysfunction in American Indian youths that is not evident from measurement of the more traditional cardiovascular risk factor of blood pressure. These data suggest that American Indian youths have vascular dysfunction that is manifest as early as childhood, which may increase their susceptibility of vascular dysfunction and cardiovascular events in adulthood. Our results support the findings from the Strong Heart Study[17] that American Indians have greater cardiovascular burden, such as diabetes and hypertension, than participants in the National Health and Nutrition Examination Survey (NHANES) study, which is representative of the general US population.

Body composition and arterial elasticity

Another unique aspect to this study was the determination of body composition and objectively measured ambulatory cadence, and how these parameters were associated with large artery elasticity index and small artery elasticity index within American Indians and within Caucasians. The greater body fat percentage in the American Indians supports previous findings of high rates of obesity in the Strong Heart Study,[20,22] with an associated increased rate of incident diabetes.[22] Of the body composition measurements, fat-free mass was most highly associated with large artery elasticity index and small artery elasticity index in both American Indians and in Caucasians, indicating that increased fat-free mass is associated with increased arterial elasticity. This finding was confirmed in multivariate models, as fat-free mass was a predictor of large artery elasticity index after adjusting for sex, age component (Caucasian only), and average cadence. Similarly, fat-free mass was a predictor of small artery elasticity index after adjusting for race, maximum cadence for 30 continuous minutes, and average cadence. We speculate that greater vascular cross-sectional area accompanies greater fat-free mass, which would lower systemic vascular resistance and increase elasticity for a given blood pressure.

Ambulatory activity and arterial elasticity

A third novel aspect to this study was that daily ambulatory activity was objectively quantified in American Indian and Caucasian youths by measuring cadence on a minute-to-minute basis, as well as total strides taken and minutes spent ambulating. The similar ambulatory activity within the groups suggests that American Indian children, adolescents, and young adults are similar in both the amount and pattern of daily ambulation compared to their Caucasian counterparts. Of the ambulatory measures, average cadence was associated with large artery elasticity index in both American Indians and Caucasians, and the maximum cadences for 5, 20, and 30 continuous minutes were associated with large artery elasticity index in American Indians.

In multivariate models, average cadence was a predictor of large artery elasticity index after adjusting for sex, age component (Caucasian only), and fat-free mass. Maximum cadence for 30 continuous minutes was a predictor of small artery elasticity index, indicating that those who ambulated at higher cadences for 30 continuous minutes had a higher small artery elasticity index. The average cadence also was a predictor of small artery elasticity index, but the association was inverse after adjusting for maximum cadence for 30 continuous minutes, race, and fat-free mass. This is likely due to colinearity of data, as both ambulatory measures are highly correlated. The fact that ambulatory cadence measures were more predictive of large artery elasticity index and small artery elasticity index than total number of strides taken and time spent ambulating each day indicates that the pace (i.e. intensity) of ambulating is necessary to favorably impact arterial elasticity in children, adolescents, and young adults. The implication of this finding is that from the standpoint of increasing arterial elasticity, it may be more valuable for youths to ambulate at a higher cadence for brief periods of between 5 and 30 minutes each day, which would also increase the average daily cadence, than to ambulate for a given number of strides or minutes each day.

Age and arterial elasticity

The current observation that large and small arterial elasticity increase with age from 8 to 30 years, particularly in Caucasians, supports previous reports from our laboratory,[29,38] and from another laboratory utilizing the same methodology of diastolic pulse contour analysis.[39] We previously found that in subjects completely free of cardiovascular risk factors, large and small arterial elasticity increased with age in children, adolescents, and young adults up to age 30, and declined thereafter, particularly after 50 years of age.[29] We replicated this finding in a larger follow-up study of youths and adults, some of whom had cardiovascular risk factors, as large and small arterial elasticity increased up to age 20 and then declined thereafter.[38] The age-related increase in both large and small arterial elasticity is evident even in children younger than in the current study, as age was a strong predictor variable of arterial elasticity in multivariate models in a large cohort of Brazilian children between the ages of 5 and 8 years.[39] Collectively, these findings suggest that elasticity of the macrovasculature and microvasculature increases with growth and physical maturation, with declines not becoming evident until adulthood when cardiovascular risk factors begin to negatively impact large and small arterial elasticity.

The fact that fat-free mass was the strongest predictor of arterial elasticity in the current study supports the notion that growth and physical maturation have positive affects on arterial elasticity in youth. This may explain why we found that BMI was positively associated with small artery elasticity index and obesity was positively associated with large artery elasticity in multivariate models in youths between 9 and 20 years of age,[38] as those with a higher BMI and those with obesity would also have greater fat-free mass as well. Another report using the diastolic pulse contour analysis technique supports the idea of growth and development to favorably impact arterial elasticity,[15] as Tanner stage was positively associated with small artery elasticity index, and height was positively related to large artery elasticity index in children between 9 and 11 years old. Although the diastolic pulse contour technique shows a decline in arterial elasticity with cardiovascular risk factors and with age in adults,[1,4,6–8] which is similar to the results from the established technique of pulse wave velocity, diastolic pulse contour analysis provides an assessment of the elasticity throughout the entire arterial tree rather than in a specific segment in a large conduit artery. It is not clear why the decline in arterial elasticity with age in children and adolescents obtained with pulse wave velocity[40] is not found with diastolic pulse contour analysis. We speculate that the strong and positive relationship between fat-free mass and arterial elasticity with diastolic pulse contour analysis suggests that the greater vascular cross-sectional area supporting the greater fat-free mass decreases vascular resistance and increases systemic arterial elasticity.

Limitations

There are limitations to this study. The cross-sectional research design of this study does not allow causality to be established when examining the relationship between arterial elasticity and the multiple predictor variables. A self-selection bias may also exist regarding study participation. Another limitation is that diastolic pulse contour analysis is a non-invasive technique to determine elasticity of the large and small arteries.[7,26,27] However, this technique has been validated with invasive measures of arterial compliance[26] and provides reliable measurement of arterial elasticity.[30] A limitation is also associated with the assessment of physical activity. Although we directly measured ambulatory activity with a step activity monitor, non-ambulatory activity such as upper extremity movements and resistance training are not recorded by the monitor, and self-reported daily activities was not assessed. However, we believe non-ambulatory activity had minimal impact on the study results because ambulation is a large component of daily activity, and very few subjects indicated during the medical history that they were actively engaged in any type of resistance training exercise. Another limitation is that ethnic determination was by self-report, and the percentage of Indian heritage was not determined. However, no difference exists in fatal and non-fatal cardiovascular disease between those who are full-blooded Indian and those who are not in the Strong Heart Study.[17] A limitation associated with the assessment of body fat percentage is that the bio-electrical impedance analysis technique underestimates fat mass and body fat percentage compared to dual-energy X-ray absorptiometry,[35,37] even though the measures from both techniques are highly correlated.[36] Another limitation is that the measurements were not standardized for menstrual cycle phase. A limitation associated with the statistical models is that other selection methods and criteria might produce slightly different models since some of the variables are highly correlated with others (e.g. BMI and obesity), and substitution of one for the other may not greatly change the model R-square. A final limitation is that the present findings only apply to apparently healthy subjects.

Conclusions

Between 8 and 30 years of age, elasticity means for the large and small arteries is lower in American Indians than in Caucasians. A smaller difference was observed in children, with a trend to a much larger difference in young adults. Furthermore, a greater fat-free mass and higher daily ambulatory cadence are associated with higher arterial elasticity in both American Indians and Caucasians. The clinical implication is that interventions designed to increase fat-free mass and daily ambulatory cadence appear warranted to increase the trajectory of arterial elasticity from childhood to young adulthood, particularly in American Indians. Ambulation at a relatively high cadence for up to 30 minutes each day may be the most efficacious strategy for youths to increase both large and small artery elasticity.

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