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Epidemiology

Reference Values for SphygmoCor Measurements in South Africans of African Ancestry

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Background: Measurements of blood pressure (BP) together with applanation tonometry at the radial and femoral arteries allow for reproducible assessments of various indexes of arterial stiffness, including peripheral (PP_p) and central (PP_c) pulse pressures, peripheral (AI_p) and central (AI_c) augmentation indexes, and aortic pulse wave velocity (PWV). In the absence of an outcome-driven and ethnicity-specific reference frame, we defined preliminary diagnostic thresholds for subjects of African descent living in Africa, using the distributional characteristics of these hemodynamic measurements.

Methods: We randomly recruited 347 subjects from a South African population of African origins. The PP_p was the average difference between systolic and diastolic BP measured five times consecutively at one home visit. For measurement of PP_c, AI_p, AI_c, and PWV, we used a high-fidelity micromanometer interfaced with a laptop computer running the SphygmoCor software. For analyses we selected 185 subjects without hypertension, diabetes, and previous or concomitant cardiovascular disease.

Results: Mean age (33.5 years) was similar in 77 men and 108 women. The PP_p, PP_c, AI_p, AI_c, and PWV significantly increased with age. The 95th prediction bands of this relation at age 30 years, approximated to 70 mm Hg for PP_p, 50 mm Hg for PP_c, 100% for AI_p, 40% for AI_c, and 8.0 m/sec for PWV. The aforementioned thresholds would need adjustment by approximately 2.5 mm Hg, 4.0 mm Hg, 10%, 6%, and 1.0 m/sec, respectively, for each decade that age differs from 30 years.

Conclusions: Pending validation in prospective outcome-based studies 70 mm Hg for PP_p, 50 mm Hg for PP_c, 100% for AI_p, 40% for AI_c, and 8.0 m/sec might be considered as preliminary thresholds to diagnose increased arterial stiffness in young adult subjects of African descent. Am J Hypertens 2006;19:40–46 © 2006 American Journal of Hypertension, Ltd.

Key Words: Pulse pressure, pulse wave velocity, augmentation index, reference value.

Cardiovascular disease is declining in developed countries, but it is increasing in developing nations such as South Africa. Large artery stiffness has been recognized as an independent risk factor for cardiovascular disease.[1,2] Measures of arterial stiffness have been recommended in the risk stratification for cardiovascular disease.[3] About a decade ago, O'Rourke and other investigators developed applanation tonometry into a simple and reproducible method of assessing various indexes of arterial stiffness.[4–6] A validated algorithm permits transformation of peripheral arterial to central aortic waveforms.[7–9] Analysis of the shape and timing of measured waveforms allows for peripheral and central pulse pressures and peripheral and central augmentation indexes to be calculated.[10] Although several studies have been published describing the distribution and correlates of arterial stiffness determined using applanation tonometry in groups of European origins,[11–13] to our knowledge, nor-

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mal values have not been determined in groups of African descent. This is particularly pertinent considering the higher arterial stiffness values obtained using more complex measurement techniques in African-Americans as compared to Americans of European descent.[14] The aim of the present study was therefore to describe the distribution of arterial characteristics determined using applanation tonometry in South Africans of African ancestry and to provide a preliminary proposal for normal values of arterial stiffness in this population, based on the distribution of arterial characteristics in normotensive subjects without concomitant disease.

Methods

Study Population

The African Project on genes in Hypertension (APOGH) is in progress and is being conducted according to the principles outlined in the Helsinki declaration for investigations in human subjects. The Human Research Ethics Committee (Medical) of the University of the Witwatersrand approved the protocol (approval number: M02-04-72). Participants gave informed, written consent. We recruited a random sample of nuclear families of South African ancestry (Nguni and Sotho chiefdoms) living in the metropolitan area of Johannesburg. We invited family members to take part in the study, if at least one or two offsprings with a minimum age of 16 years and one or both parents were available for examination. Of the 347 participants enrolled in the study, we eliminated 9 from analysis because the recorded peripheral or central augmentation index was of insufficient quality. Pulse wave velocity (PWV) could not be measured in 35 subjects because they had bradycardia or were too obese. To generate a healthy sample, we excluded a total of 153 subjects because of hypertension ($n = 135$) or diabetes ($n = 22$), or because they had previous or concomitant cardiovascular disease, including coronary heart disease, heart failure, transient ischemic attack, or intermittent claudication ($n = 6$). The overall number of participants statistically analyzed totaled 185 for the peripheral and central pulse pressures and the peripheral and central augmentation indexes and 159 for PWV.

Clinical Measurements

Trained observers visited the participants at their homes for measurement of brachial blood pressure (BP). The participants were seated and asked to rest for 5 min. The observers measured the participants' sitting BP five times consecutively. Systolic and diastolic (phase V) BP were determined to the nearest 2 mm Hg according to the recommendations of the European Society of Hypertension.[15] In most participants standard cuffs were used, which had an inflatable bladder with a length of 22 cm and a width of 12 cm. If arm circumference exceeded 31 cm, larger cuffs with a 31 by 15 cm bladder were used. For analysis, we averaged the five BP recordings obtained at the home visit. Hypertension was defined as a BP of at least 140 mm Hg systolic or 90 mm Hg diastolic or as the use of antihypertensive drugs. We administered a standardized questionnaire to obtain information on each participant's medical history, smoking habits, intake of alcohol, and use of medication. Height and weight were measured with the participants in a standing position wearing indoor clothes and no shoes. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters.

Pulse Wave Analysis

Participants were invited for an examination at a locally organized clinic. After they had rested for 15 min in the supine position, we recorded during an 8-sec period the radial waveform at the dominant arm by applanation tonometry. We used a high-fidelity SPC-301 micromanometer (Millar Instrument, Inc., Houston, TX) interfaced with a laptop computer running SphygmoCor software, version 6.21 (AtCor Medical Pty. Ltd., West Ryde, New South Wales, Australia). We discarded recordings when the systolic or diastolic variability of consecutive waveforms exceeded 5%, or the amplitude of the pulse wave signal was less than 80 mV. We calibrated the pulse wave by manual measurement (auscultation) of BP immediately before the recordings. From the radial signal, the SphygmoCor software calculates the aortic pulse wave by means of a validated and population-based generalized transfer function. The radial augmentation index was defined as the ratio of the second to the first peak of the pressure wave expressed in percent. The aortic augmentation index was the difference between the second and the first systolic peak given as a percentage of the aortic pulse pressure. Peripheral and central pulse pressure were defined as the difference between systolic and diastolic BP derived from the brachial BP measured at the subjects' homes or from the aortic pulse wave. Aortic PWV was measured by sequential recordings of the arterial pressure waveform at the carotid and femoral arteries. Distances from the suprasternal notch to the carotid sampling site (distance A) and from the suprasternal notch to the femoral artery (distance B) were measured. Pulse wave velocity distance was calculated as distance B minus distance A. Pulse transit time was the average of 10 consecutive beats. Aortic PWV was calculated as the ratio of the distance in meters to the transit time in seconds.

Statistical Analysis

Database management and statistical analyses were performed with SAS software, version 6.12 (SAS Institute Inc., Cary, NC). The central tendency and spread of the data are reported as mean ± SD. Departure from normality was evaluated by Shapiro-Wilk's statistic[16] and skewness by the computation of the coefficient of skewness (ie, the third moment about the mean divided by the cube of the

Table 1. Characteristics of the study population

	Men	Women	*P*
Number	77	108	
Age (yr)	33.4 ± 15.2	33.5 ± 13.1	.90
Anthropometric measurements			
Height (cm)	168.1 ± 7.5	158.3 ± 7.2	<.01
Weight (kg)	67.9 ± 14.2	69.7 ± 16.5	.40
Body mass index (kg/m^2)	23.9 ± 4.4	27.8 ± 6.1	<.01
Peripheral hemodynamic measurements			
Systolic pressure (mm Hg)	126.4 ± 24.1	121.2 ± 14.7	.07
Diastolic pressure (mm Hg)	80.0 ± 7.2	79.1 ± 10.8	.50
Pulse pressure (mm Hg)	46.5 ± 23.2	42.1 ± 10.2	.10
Augmentation index (%)	74.7 ± 17.5	78.0 ± 24.1	.30
Pulse rate (beats/min)	56.9 ± 7.9	67.6 ± 11.8	<.01
Central hemodynamic measurements			
Systolic pressure (mm Hg)	116.0 ± 21.9	112.0 ± 15.7	.10
Diastolic pressure (mm Hg)	80.8 ± 7.3	80.0 ± 10.8	.50
Pulse pressure (mm Hg)	35.2 ± 20.0	32.0 ± 9.9	.10
Augmentation index (%)	21.6 ± 12.6	22.5 ± 15.5	.60
Aortic pulse wave velocity (m/sec)	5.8 ± 1.9	5.7 ± 1.5	.60
Subjects with characteristics			
Smoking habits (%)	27 (35.5)	3 (2.7)	<.01
Alcohol intake (%)	29 (38.2)	17 (15.6)	<.01

Values are mean ± SD or number (%). $P \leq .05$ indicates significant gender difference.

standard deviation).[17] The normal distribution was used to determine the significance of the coefficient of the skewness.[17] Our statistical methods also included the large sample z-test and the χ^2 statistic to compare means and proportions as well as single regression.[18]

Results

Characteristics of the Participants

Table 1 gives the characteristics of the participants by gender. Mean age was 33.5 years. Women had a higher BMI than men, with 64 women (59.3%) and 31 men (40.3%) being overweight (BMI ≥25 kg/m^2). Of the 185 participants, 27 men (35.5%) and 3 women (2.7%) were smokers and 29 men (38.2%) and 17 women (15.6%) reported alcohol consumption. Among smokers, median tobacco use was 8 cigarettes per day (range, 2 to 20 cigarettes). Among regular drinkers, median alcohol consumption was 6.2 g/d (range, 1 to 26 g/d). Although the peripheral and central BPs tended to be higher in men than in women, none of the gender differences in the hemodynamic measurements reached statistical significance with the exception of pulse rate, which was 10.7 beats/min faster in women.

Distribution of Hemodynamic Measurements

In all subjects, the distributions of the peripheral and central pulse pressures departed from normality and were positively skewed ($P < .001$). The coefficients of skewness were 4.5 and 4.1, respectively. Similarly, the peripheral and central augmentation indexes were not normally distributed ($P < .0001$) with coefficients of skewness amounting to 1.1 and −0.3, respectively. Fig. 1 shows the distributions of these hemodynamic measurements. In all 185 subjects, pulse pressure averaged 43.9 mm Hg (95% confidence interval [CI], 40.5–47.3) peripherally and 33.3 mm Hg (95% CI, 30.3–36.3) centrally. The augmentation indexes averaged 76.6% (95% CI, 74.9–78.3) peripherally and 22.2% (95% CI, 20.6–23.8) centrally. Pulse wave velocity averaged 5.8 m/sec (95% CI, 5.6–5.9). Additional statistics for the peripheral and central pulse pressures and the peripheral and central systolic augmentation indexes and PWV are presented by age groups in Tables 2 and 3.

Proposal for Diagnostic Thresholds

To determine diagnostic thresholds for men and women combined, we rounded the 95th prediction bands (Figs. 2, 3, and 4) for the approximate mean age of the participants

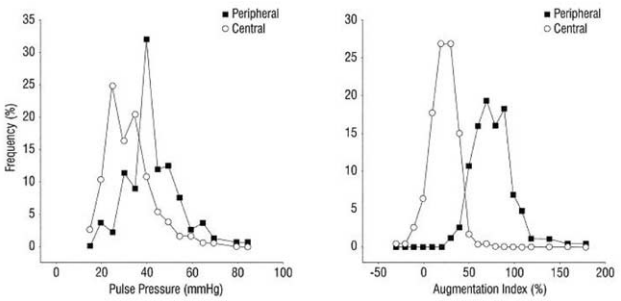

FIG. 1. The distribution of peripheral and central pulse pressures and peripheral and central augmentation indexes in 185 healthy subjects.

Table 2. Distribution of peripheral and central pulse pressures and peripheral and central augmentation indexes by age in healthy subjects

	Peripheral				Central			
Age (yr)	**<30**	**30–49**	**≥50**	**All**	**<30**	**30–49**	**≥50**	**All**
Pulse pressure (mm Hg)								
N	97	61	27	185	97	61	27	185
Mean	42.2	43.1	51.9	43.9	29.9	34.1	43.9	33.3
SD	17.0	18.0	12.2	17.0	14.7	14.8	11.1	15.0
P5	24.0	26.0	36.0	26.0	18.1	18.2	29.0	18.2
P10	30.0	30.0	40.0	30.0	19.9	19.8	34.1	20.3
P50	40.0	40.0	50.0	42.0	27.3	32.2	40.3	31.1
P90	52.0	54.0	66.0	56.0	39.1	48.8	59.5	46.8
P95	62.0	58.0	68.0	64.0	41.8	52.3	63.7	52.6
Augmentation index (%)								
N	97	61	27	185	97	61	27	185
Mean	66.3	84.8	95.1	76.6	15.3	27.5	34.9	22.2
SD	16.0	20.5	22.2	21.6	13.2	11.2	10.3	14.3
P5	43.4	58.9	68.0	48.3	−7.8	11.9	18.6	−0.5
P10	48.5	65.2	71.7	52.2	−0.3	13.7	19.3	4.3
P50	62.6	81.5	89.9	74.9	16.0	26.2	34.2	23.4
P90	86.9	107.8	113.6	101.2	32.5	40.6	44.9	39.2
P95	97.0	120.3	123.3	111.9	35.5	44.9	49.8	42.2

N, SD, P5, P10, P50, P90, and P95 indicate number of subjects, standard deviation, and percentiles.

(30 years) downward to the nearest value ending in zero, or integer for PWV. This procedure yielded the following thresholds: 70 mm Hg for peripheral pulse pressure, 50 mm Hg for central pulse pressure, 100% for the peripheral augmentation index, 40% for the central augmentation index, and 8.0 m/sec for PWV. Peripheral and central pulse pressures (Fig. 2), the peripheral and central augmentation indexes (Fig. 3), and PWV (Fig. 4) increased with age. Per decade of life, the changes in these hemodynamic measurements approximated to 2.5 mm Hg and 4.0 mm Hg for the peripheral and central pulse pressures, to 10% and 6% for the peripheral and central augmentation indexes, and to 1.0 m/sec for PWV. Thus, for these age-dependent hemodynamic measurements, the aforementioned thresholds need adjustment by approximately 2.5 mm Hg, 4.0 mm Hg, 10%, 6%, and 1.0 m/sec, respectively, for each decade that age differs from 30 years.

Table 3. Distribution of pulse wave velocity (m/sec) by age in healthy subjects

Age (yr)	**<30**	**30–49**	**≥50**	**All**
N	84	52	23	159
Mean	5.0	6.2	7.6	5.8
SD	1.2	1.2	2.5	1.7
P5	3.8	4.5	3.5	3.8
P10	4.0	4.8	5.5	4.1
P50	5.0	5.9	7.3	5.5
P90	6.2	7.8	9.6	7.8
P95	6.6	7.9	11.5	8.8

N, SD, P5, P10, P50, P90, and P95 indicate number of subjects, standard deviation, and percentiles.

Discussion

In the present study, we determined the distributional characteristics of various measures of arterial stiffness in South Africans of African descent by means of applanation tonometry. In the absence of an outcome-driven reference frame, our study suggests that at age 30 years arterial stiffness might be abnormally increased if the following thresholds are exceeded: 70 mm Hg for the peripheral pulse pressure, 50 mm Hg for the central pulse pressure, 100% for the peripheral augmentation index, 40% for the central augmentation index, and 8.0 m/sec for aortic PWV. These thresholds would need adjustment by 2.5 mm Hg, 4 mm Hg, 10%, 6%, and 1.0 m/sec, respectively, for each decade that age differs from age 30 years.

Prospective studies, in which pulse pressure was analyzed as a continuous variable, have demonstrated that

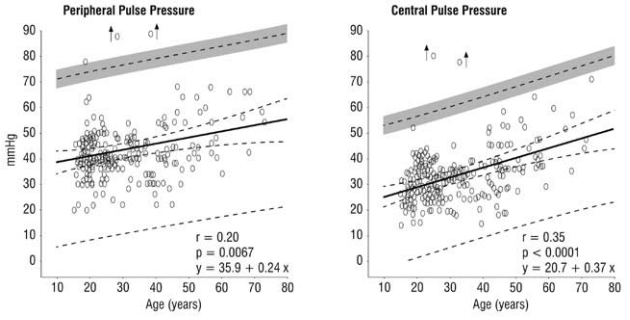

FIG. 2. Relation of the peripheral and central pulse pressures with age in 185 healthy subjects. Each panel shows the regression line and the 95% prediction bands for mean and individual values of the augmentation indexes. The shadowed area represents the transition between normal and elevated values.

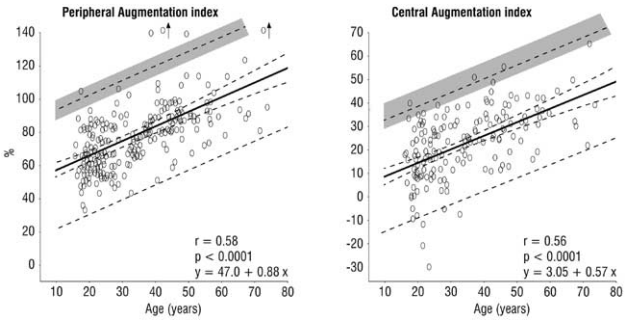

FIG. 3. Relation of the peripheral and central augmentation indexes with age in 185 healthy subjects. Each panel shows the regression line and the 95% prediction bands for mean and individual values of the augmentation indexes. The shadowed area represents the transition between normal and elevated values.

peripheral pulse pressure has prognostic significance. Asmar and colleagues[19] studied 61,724 consecutive subjects, 49% women, between 16 and 90 years old. They found that in women as well as men the mean value of pulse pressure across the age range was close to 50 mm Hg. These investigators suggested 65 mm Hg as a diagnostic threshold, which they determined either by adding two standard deviations to the mean or from the 95th percentile.[19] This 65 mm Hg threshold, and ours of 70 mm Hg, is in close agreement with values of the peripheral pulse pressure (63 mm Hg, 65 mm Hg,[20] or 68 mm Hg[21]) previously reported to be associated with cardiovascular morbidity and mortality. Franklin and colleagues[22] also noted that in middle-aged and older subjects, pulse pressure was an important predictor of cardiovascular risk. Both a high systolic and a low diastolic BP were associated with adverse outcomes.[22]

Arterial stiffness and the velocity of the reflected arterial wave are the main determinants of the peripheral and central augmentation indexes. Wave reflection occurs at sites of changes of arterial impendence along the arterial tree, such as branching points or atherosclerotic plaques. The peripheral and central augmentation indexes increase with age and mean arterial pressure,[10] and are inversely related to heart rate[23,24] and body height.[25] Systolic augmentation is influenced by PWV. With higher PWV, the reflected waves return earlier in the aorta. Age is the main determinant of PWV. Few studies have described the distribution of these hemodynamic measurements in the population at large or in healthy reference groups. Mitchell and colleagues[13] studied carotid femoral PWV and systolic augmentation at the level of the carotid artery in 188 men and 333 women in the Framingham Heart Study Offspring Cohort, who were free from cardiovascular disease, hypertension, dyslipidemia, obesity, and smoking within the past 12 months. Mean age was 56.6 years. From the data of Mitchell and co-workers,[13] we calculated mean values plus two standard deviations. For carotid femoral PWV, this threshold was 12.7 m/sec in men and 12.0 m/sec in women.[13] The corresponding estimates of the carotid augmentation index were 33% and 37%, respectively.[13] As the mean age of the group studied by us was lower, we cannot make direct comparisons with the data obtained by Mitchell and colleagues.[13] With respect to comparisons based on ethnic group, our data on augmentation index when compared to that obtained in Czech or Polish populations of similar mean age (peripheral augmentation index: 64%, 67%; central augmentation index: 14%, 15%, respectively)[26] agree with the current literature[14] that arterial stiffness is increased in peoples of African ancestry in comparison to peoples of European descent. To our knowledge, the present study is the first conducted in subjects of African descent using the simple and reproducible technique of applanation tonometry. One limitation of our study however, is the relatively small sample size, which precluded us from studying gender differences, including the determination of thresholds, in detail. Nevertheless, the summary statistics suggest that the peripheral and central pulse pressures might be on average 4 mm Hg and 3 mm Hg higher in men than in women. Although the present study was limited by relatively small sample sizes, our findings might be readily extrapolated, because our study was population-based. Our population demographics are similar to those previously reported[27,28] and hence our data are representative of South Africans of African ancestry.

We used a high-fidelity pressure transducer to increase the accuracy of the recorded pressure waveforms. Only one trained observer obtained all vascular measurements. Pulse wave analysis was used to assess central pulse pressure and central augmentation index. Such an approach may have led to a small degree of error in central pressure estimation, but the transfer function involved has previously been validated.[29,30] However, as the transfer

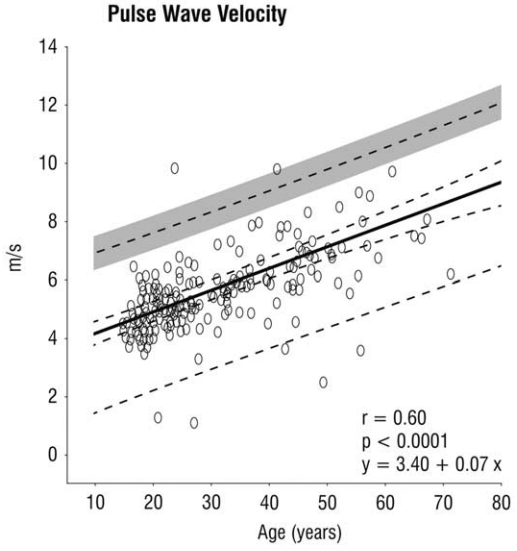

FIG. 4. Relation of the pulse wave velocity with age in 159 healthy subjects. Each panel shows the regression line and the 95% prediction bands for mean and individual values of pulse wave velocity. The shadowed area represents the transition between normal and elevated values.

function has not been validated in peoples of African ancestry we have not discussed the possible prognostic significance of our central pulse pressure data. In contrast to the Framingham investigators,[13] we did not exclude smokers from our reference sample. Smoking may increase the stiffness of large arteries and wave reflection.[31] However, we do not believe that smoking had a large impact on the present findings, because few women smoked and among smokers the median tobacco use was only 8 cigarettes a day.

In conclusion, pending validation in prospective outcome-based studies, 70 mm Hg for peripheral pulse pressure, 50 mm Hg for central pulse pressure, 100% for peripheral augmentation index, 40% for central augmentation index, and 8.0 m/sec for PWV might be considered as preliminary thresholds to diagnose increased arterial stiffness in young adult subjects of African descent. The aforementioned thresholds need adjustment for gender and age and require additional studies in reference populations with larger sample sizes. Such a study is currently in progress within the framework of the African Project on Genes in Hypertension.

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