Blood Pressure and Arterial Compliance in Young Adults: The Minnesota Children’s Blood Pressure Study

Donna K. Arnett, Stephen P. Glasser, Gary McVeigh, Ronald Prineas, Stanley Finklestein, Richard Donahue, Jay N. Cohn, and Alan Sinaiko

The aim of this study was to assess the relation between blood pressure (BP) and arterial compliance in a healthy sample of young adults. School children (aged 10 to 14 years at entry) were surveyed in 1977 to 1978, and 1207 were followed once to twice yearly until age 23 years. Arterial compliance was measured in 179 adults at the last follow-up visit. The sample included individuals in the upper tertile of systolic BP during the last three follow-up visits and race- and sex-matched individuals in the lower two tertiles. We obtained radial artery waveforms using a calibrated tonometer device and characterized waveform morphology to determine large artery (C₁) and oscillatory (C₂) compliance. Blood pressure was measured using random zero sphygmomanometers. The mean and standard deviation of C₁ was 2.13 ± 0.59 mL/mm Hg and of C₂ was 0.083 ± 0.02 mL/mm Hg. Systolic BP was inversely related to C₁ (P < .001) and C₂ (P < .01) after adjustment for gender, height, weight, insulin, and HDL and LDL cholesterol. After adjustment, a 1 SD change in systolic BP was associated with a −0.30 mL/mm Hg change in C₁ and a −0.008 mL/mm Hg change in C₂. Data from the Minnesota Children’s Blood Pressure Study indicate that systolic BP is inversely related to arterial compliance, particularly C₁ (the large artery, or capacitive compliance). Am J Hypertens 2001;14:200–205 © 2001 American Journal of Hypertension, Ltd.

Key Words: Pediatrics, arterial compliance, blood pressure.

Arterial stiffening is found in patients with hypertension, but it is not clear whether the arterial disease precedes or is a consequence of sustained elevated blood pressure (BP).\(^1,2\) Arterial stiffening increases transiently as BP increases because of the nonlinear distensibility characteristics of the artery. Although acute and reversible stiffening of the large arteries occurs with elevated BP,\(^3,4\) irreversible structural changes also occur with sustained exposure to elevated BP. Hypertension accelerates atherosclerosis, collagen synthesis, and arterial smooth muscle hyperplasia and hypertrophy, thereby increasing arterial stiffness.\(^5–7\) These primary structural changes may lead to permanent arterial stiffening independent of the BP level.

Experimentally induced arterial stiffening increases systolic BP in animal models\(^8,9\); however, longitudinal studies of the relationship between arterial stiffness and hypertension in humans are sparse. The Atherosclerosis Risk in Communities Study reported that increased arterial stiffness predicted the onset of stage II or higher hypertension in middle-aged adults.\(^10\) In contrast, cross-sectional studies observed no association between arterial stiffness and hypertension once the confounding influence of BP was accounted for in the arterial stiffness measurement.\(^11–14\) The latter suggests that the arterial stiffness–hypertension association reflects reverse causality (ie, hypertension caused increased arterial stiffness).

Delineation of the arterial stiffness–hypertension association in humans requires methodologies that permit examination of arterial behavior before development of major structural changes. Large conduit arteries, such as the aorta, serve as capacitors and as cushions, smoothing cardiac pulsation, absorbing the oscillations generated from reflected waves, and directing blood through the organs and tissues in a steady stream. Pulse contour analysis (PCA) provides measurements that capture both capacitive and cushioning (oscillatory) arterial functions. It

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Dr. Jay N. Cohn has commercial interest in the company that makes the instrument used in this study.

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uses the arterial pulse contour to provide an assessment of the large artery (capacitance) behavior and the behavior of smaller arteries that represent the primary source of reflected waves or oscillations in the arterial system. The PCA method has been validated, applied to the study of several populations, and shown associations with a number of clinical endpoints. The pulse waveform is analyzed using a modified Windkessel model. The model includes two compliance elements (generally referred to as $C_1$ and $C_2$) combined with inertia and resistance elements. The decay in the diastolic pressure waveform is determined by an algorithm that consists of the sum of an exponential decay and an exponentially decaying sinusoidal term. The first term accounts for the overall decrease of exponential decay and an exponentially decaying sinusoidal oscillation (generally referred to as $C_2$). The algorithm determined the best set of $A_i$ values for reflecting waves and resonances within the arterial system.

This report presents the results from PCA compliance measurements in 179 normal subjects (mean age, 23.6 years), participating in a long-term study of the natural history of BP. The data describe the relationship of large ($C_1$) and small ($C_2$) artery compliance with BP, weight, height, lipids, and fasting insulin from this sample.

**Methods**

**Study Population**

The Minnesota Children’s Blood Pressure Study was started in the 1977–1978 school year with the BP screening of 10,423 first through third grade children (99% of all children enrolled in those grades) in the Minneapolis Public Schools. After this screening, a cohort was selected for long-term evaluation. Followed were all children in the upper and lower fifth percentiles of the race-specific BP distribution; 50% of the remaining African American children, and one of nine of the remaining white children. There were no exclusion criteria. Written consent for longitudinal evaluation was obtained from 1207 of the 2641 children in the selected cohort.

The 1207 children were examined approximately twice yearly through their grade-school and junior high-school years and once yearly during high school. An examination was conducted within 2 years of post-high-school (PH), at which time BP and anthropometric data were obtained from 817 participants of the original group of 1207. Recontact of the 817 occurred approximately 5 years after the PH visit, and 679 underwent reexamination between 1993 and 1995 (aged 23.6 ± 0.1 years). For this study, a sample was selected as follows: group 1 (high-risk group), included all individuals in the upper tertile of systolic BP of the PH and prior two high school visits; group 2 (low-risk group), included randomly selected individuals from the lower two systolic BP tertiles, matched 2:1 for sex and race with individuals in group 1. A total of 179 subjects, 60 from group 1 and 119 from group 2, were measured for compliance, anthropometric, and biochemical measurements.

**Arterial Compliance Methods**

Radial artery waveforms were recorded with an arterial tonometer sensor array (Nellcor N-CAT model N-500 instrument) using previously described methods. Waveforms were calibrated by the oscillometric method (Hypertension Diagnostics Inc., Minneapolis, MN) with a cuff on the opposite arm and a calibration system internal to the Nellcor device. The tonometry unit contained an array of piezoresistive pressure transducers, each 0.2 mm apart, capable of measuring the relative intra-arterial pulse amplitude with high accuracy in arteries as small as 1.0 mm in diameter. The tonometer sensor array was centered over the radial artery, the hold down pressure was automatically controlled to obtain the optimal waveform, and the calibration was repeated until the waveform was stable. Once stable, 30 sec of analog tracing of the radial artery waveform was digitized at 200 samples per second and stored on a personal computer for compliance analysis (Hypertension Diagnostics, Inc.). A beat-marking algorithm determined the beginning of systole, peak systole, onset of diastole, and end-diastole for each beat during the measurement period. Marked beats were cross-correlated, and beats with a correlation coefficient greater than 0.995 were averaged with the use of the upstroke beat mark as the fiduciary time point for the averaging process. This excluded between 20% and 25% of the beats from a 30–sec sample. We used the averaged beat for analyses.

To derive estimates of arterial compliance, a parameter-estimating algorithm was used to the average beat that divides the total systemic arterial compliance into the large artery ($C_1$, capacitive) or small artery ($C_2$, oscillatory) compliances. The method was previously described. The algorithm used parameters determined from the diastolic portion of the averaged beat by the following equation: $P(t) = A_1 \exp(-A_2 \cdot t) + A_3 \exp(-A_4 \cdot t) \cdot \cos(A_5 \cdot t + A_6)$, where $P(t)$ is the diastolic pressure at time $t$ relative to the aortic valve closure. The first part of the equation represents the overall exponential decay in pressure during diastole. The second part of the equation represents an exponentially decaying envelop of the sinusoidal oscillations occurring in early diastole, as a result of reflecting waves and resonances within the arterial system. The algorithm determined the best set of $A_i$ values for matching the diastolic portion of the averaged beat to this equation. $C_1$ and $C_2$ compliance values were then calculated from the $A_2$, $A_3$, and $A_4$, found with the best fit algorithm, in accordance with the modified Windkessel circuit analysis.

The reliability of the pulse contour analysis derived from measurements conducted on repeat visits on the same individuals ($n = 20$) indicate the reproducibility to be...
equivalent to other noninvasive measurements such as BP. The differences between two studies taken 1 week apart were small \((C_1 = 0.015 \pm 0.38 \text{ mL/mm Hg}; C_2 = 0.007 \pm 0.014 \text{ mL/mm Hg})\).\(^{15}\)

**Other Examination Components**

Seated BP was measured using random zero sphygmomanometers and a standardized protocol.\(^{20}\) Arm circumferences were measured at the midpoint between the olecranon and acromion, and the cuff size selected to be between 38% and 48% of the measured circumference. The position of the arm was adjusted so that the antecubital fossa was at the level of the fourth intercostal space. Duplicate measures of seated BP were obtained after a 5-min rest, with a 30-sec interval between readings. The average of these measurements was used in the analysis. Height and weight were measured without shoes with participants in light clothing. Fasting samples were collected and measured for insulin, HDL cholesterol, and LDL cholesterol.

**Statistical Analysis**

High and low BP groups were combined for analysis as there were no differences in the relationship between BP and compliance between the two sampling groups. Similarly, men and women were analyzed together as the primary association of interest, BP and arterial compliance did not differ between the sexes. Means were calculated for anthropometric, physiologic, and metabolic variables according to quartiles of \(C_1\) and \(C_2\), and differences in the means across quartiles of \(C_1\) and \(C_2\) were assessed using analysis of variance. Multiple linear regression models were calculated for \(C_1\) and \(C_2\) separately, and included systolic BP, sex, height, weight, insulin, and HDL and LDL cholesterol. All analyses were implemented using SAS version 6.1.\(^{22}\) Nonlinear terms for BP were tested and found to be nonsignificant; therefore, the final models included only first order terms for BP.

**Results**

There were 108 young men and 71 young women in the sample (mean age, 23.6 \(\pm\) 0.1 years). The mean of \(C_1\) was 2.13 \(\pm\) 0.59 mL/mm Hg (range, 0.80 to 4.36 mL/mm Hg) and the mean of \(C_2\) was 0.083 \(\pm\) 0.02 mL/mm Hg (range, 0.04 to 0.14 mL/mm Hg). Table 1 summarizes the means of anthropometric and biochemical measurements according to sex-specific quartiles of \(C_1\) and \(C_2\). In bivariate analyses, both systolic \((P < .001)\) and diastolic BP \((P < .02)\) were inversely related to \(C_1\). Height \((P = .04)\) was

**Table 1.** Means and standard deviations of systolic and diastolic blood pressure, height, weight, LDL and HDL cholesterol, and insulin by sex-specific quartile of \(C_1\) and \(C_2\)

<table>
<thead>
<tr>
<th>Variable</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>Overall P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>(C_1) quartile range</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.80–1.89</td>
<td>0.92–1.62</td>
<td>1.89–2.20</td>
<td>1.62–1.81</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>133.7 (11.9)</td>
<td>124.5 (12.0)</td>
<td>124.6 (12.5)</td>
<td>117.1 (12.5)</td>
<td>124.9 (13.5) (&gt; .001)</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>69.9 (8.5)</td>
<td>65.8 (8.1)</td>
<td>66.2 (7.2)</td>
<td>64.9 (7.6)</td>
<td>66.7 (8.0) (&gt; .02)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170.6 (8.0)</td>
<td>172.9 (8.9)</td>
<td>172.9 (9.2)</td>
<td>176.2 (10.2)</td>
<td>173.2 (9.3) (&gt; .04)</td>
</tr>
<tr>
<td>Weight (lb)</td>
<td>168.6 (39.2)</td>
<td>166.3 (42.4)</td>
<td>170.5 (38.4)</td>
<td>176.7 (51.1)</td>
<td>170.6 (42.9) (&gt; .69)</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>104.9 (28.6)</td>
<td>101.0 (25.2)</td>
<td>100.6 (20.6)</td>
<td>102.3 (30.9)</td>
<td>102.2 (26.5) (&gt; .89)</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>44.9 (11.9)</td>
<td>48.6 (14.7)</td>
<td>47.9 (12.2)</td>
<td>48.0 (11.4)</td>
<td>47.4 (12.6) (&lt; .52)</td>
</tr>
<tr>
<td>Insulin (m(^\mu)/L)</td>
<td>20.6 (9.3)</td>
<td>17.9 (10.7)</td>
<td>17.4 (5.9)</td>
<td>19.5 (14.3)</td>
<td>18.8 (10.5) (&gt; .46)</td>
</tr>
<tr>
<td>(C_2) quartile range</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.04–0.07</td>
<td>0.04–0.06</td>
<td>0.07–0.09</td>
<td>0.06–0.08</td>
<td></td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>126.7 (13.3)</td>
<td>123.9 (12.3)</td>
<td>126.4 (15.6)</td>
<td>122.6 (12.3)</td>
<td>124.9 (13.5) (&gt; .42)</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>69.5 (7.5)</td>
<td>66.4 (8.1)</td>
<td>67.4 (8.6)</td>
<td>63.5 (6.9)</td>
<td>66.7 (8.0) (&gt; .005)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171.4 (8.9)</td>
<td>173.1 (8.6)</td>
<td>172.9 (9.4)</td>
<td>175.3 (10.0)</td>
<td>173.2 (9.3) (&gt; .26)</td>
</tr>
<tr>
<td>Weight (lb)</td>
<td>160.6 (50.3)</td>
<td>156.0 (32.6)</td>
<td>174.5 (38.3)</td>
<td>190.5 (41.2)</td>
<td>170.6 (42.9) (&gt; .001)</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>95.7 (22.4)</td>
<td>105.8 (30.1)</td>
<td>98.2 (25.3)</td>
<td>108.9 (26.5)</td>
<td>102.2 (26.5) (&gt; .07)</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>51.5 (12.7)</td>
<td>47.5 (13.6)</td>
<td>46.8 (13.6)</td>
<td>44.0 (9.2)</td>
<td>47.4 (12.6) (&lt; .05)</td>
</tr>
<tr>
<td>Insulin (m(^\mu)/L)</td>
<td>19.9 (16.2)</td>
<td>17.2 (7.5)</td>
<td>19.2 (8.7)</td>
<td>19.0 (7.5)</td>
<td>18.8 (10.5) (&lt; .68)</td>
</tr>
</tbody>
</table>

* F test for overall difference between groups.
modestly associated with \( C_1 \), whereas weight, HDL and LDL cholesterol, and insulin were not. \( C_2 \) was inversely associated with diastolic BP \((P < .005)\) and HDL cholesterol \((P < .001)\) and LDL cholesterol \((P = .07)\). However, \( C_2 \) was not related to systolic BP \((P = .42)\), height \((P = .26)\), or insulin \((P = .68)\) in the bivariate analyses.

Fig. 1 graphically displays sex-adjusted \( C_1 \) and \( C_2 \) according to systolic BP. Adjusted \( C_1 \) decreased consistently across the systolic BP \((P < .001)\). Adjusted \( C_2 \) was not strongly related to systolic BP \((P = .02)\), although \( C_2 \) was highest in the lowest systolic BP quartile compared to the other three.

We calculated multiple linear regression models to estimate the effects of BP once adjusted for sex, anthropometric, and biochemical factors. Models for \( C_1 \) and \( C_2 \) were calculated separately (Table 2). These variables explained 41% of the variation in \( C_1 \) and 48% of the variation in \( C_2 \). Systolic BP remained strongly, inversely associated with \( C_1 \) after adjustment for sex, height, weight, insulin, and HDL and LDL cholesterol. For each SD change in systolic BP, there was a corresponding \(-0.30 \text{ mL/mm Hg}\) change in \( C_1 \) (equivalent to approximately one-half of a SD change in \( C_1 \)). Height and weight were significantly and positively related to \( C_1 \), although the relationship was not as strong as that for systolic BP. HDL cholesterol was modestly associated with \( C_1 \), whereas insulin and LDL cholesterol were not. Results of similar direction were observed for \( C_2 \). Systolic BP was negatively associated with \( C_2 \) once adjusted for other cardiovascular risk factors. For each SD change in systolic BP, there was a \(-0.008 \text{ mL/mm Hg}\) change in \( C_2 \) (about 40% of a SD in \( C_2 \)). Interestingly, insulin was significantly, inversely related to \( C_2 \) in multivariable analysis, but not \( C_1 \).

Because \( C_2 \) was strongly associated with diastolic BP in bivariate analyses, we calculated a multiple linear regression model that regressed \( C_2 \) on the same set of risk factors. The model explained 35% of the variation in \( C_2 \). For each one SD change in diastolic BP, there was a small \((0.004 \text{ mL/mm Hg})\) change in \( C_2 \) (about 20% of a SD in \( C_2 \)). In the diastolic BP model, height, weight, and LDL cholesterol were positively and significantly associated with \( C_2 \), whereas insulin and HDL cholesterol were inversely and significantly associated with \( C_2 \).

**Discussion**

As arterial pressure increases, arterial compliance decreases. Debated is whether reductions in arterial compliance are transient (ie, arteries return to normal levels as BP normalizes) or irreversible. There is considerable evidence to suggest that sustained elevations in BP accelerate atherosclerosis, arterial smooth muscle hyperplasia and hypertrophy, and collagen synthesis, thereby decreasing arterial compliance (perhaps irreversibly).\(^5\)-\(^7\) A prior study of echocardiographically measured aortic stiffness conducted in 11-year-old twins found a strong relationship between aortic stiffness and systolic BP \((r = 0.22\) after adjustment for height and weight).\(^2\) Data from the Minnesota Children’s Blood Pressure Study of young adults also indicate that systolic BP is inversely associated with arterial compliance, particularly \( C_1 \) (the large artery, or capacitive compliance). This relation persisted after adjustment for other cardiovascular risk factors, including HDL and LDL cholesterol, insulin, height, and weight. These results lend support to the hypothesis that abnor-
malities in arterial compliance contribute to the development of essential hypertension for the following reasons. First, participants were measured at 23.6 years of age. Although the large arteries may have undergone subtle atherosclerotic changes, the natural history of atherosclerosis suggests the absence of significant structural changes in the arteries of these young adults.\(^6\) Second, the participants were not hypertensive, and the mean BP was in the optimal BP range (124.9/66.7 mm Hg). Therefore, this inverse relationship between BP and arterial compliance was detected before the onset of clinically apparent essential hypertension. Third, the method of assessment of arterial compliance, the pulse contour analysis, is less sensitive to BP-related measurement error compared to other techniques. Ultrasound or echocardiographic techniques evaluate the change in arterial diameter, area, or volume for a corresponding change in BP; therefore, BP is mathematically integral to the measurement. When calculating associations between BP and ultrasound or echocardiographic indices of arterial stiffness, a residual association between arterial stiffness and essential hypertension is expected.

The peripheral arterial waveform morphology, upon which PCA is based, involves a diminution in the amplitude and duration of the pressure waveform that interrupts the monoexponential decay of diastole. In the waveform, diastole is segmented into two components. The first represents large artery compliance (C\(_1\)), measured as the exponential decay of the waveform. The second (C\(_2\)) represents the small artery compliance that determines peripheral wave reflections, measured as the diastolic fluctuation in the waveform that occur when wave reflections are superimposed on the basic shape of the waveform.\(^6\) Blood pressure was more strongly associated with C\(_1\), large artery compliance, than C\(_2\) in young adults in our study. C\(_2\) was only significantly associated with systolic BP after adjustment, suggesting that the modest relationship between BP and C\(_2\) may be obscured by the relation of C\(_2\) with metabolic or anthropomorphic measures. Prospective studies that track changes in BP, other cardiovascular risk factors, and compliance are needed to understand the complex relationship between these factors, and which factors are the most important in the pathogenesis of reduced arterial compliance.

We present results for BP measured at the time of the assessment of arterial compliance. We also analyzed whether BP measured at baseline and over the course of the child’s development was associated with arterial compliance at young adulthood. The baseline BP and the slope of the individual child’s BP regressed on age were modeled separately in a multivariable linear regression model (data not presented). After adjustment for BP at entry into the study in 1977 to 1978, the individual BP slope significantly predicted C\(_1\) and C\(_2\). Because the amount of variation in C\(_1\) and C\(_2\) explained by the model was slightly lower for the slope of BP over time than for the follow-up BP, we presented results for only the latter study. Nonetheless, because arterial compliance was not measured at baseline, the temporal sequence between the BP increases and the reductions in arterial compliance cannot be established.

Previous studies demonstrate that both C\(_1\) and C\(_2\) decrease with increasing age.\(^13,16\) McVeigh et al.\(^16\) examined PCA in 212 healthy adults aged 21 to 83 years and reported a mean C\(_1\) of 2.05 mL/mm Hg in men and 1.6 mL/mm Hg in women, and a mean C\(_2\) of 0.08 mL/mm Hg in men and 0.06 mL/mm Hg in women. Our findings differ from this report in two ways. First, there was no sex difference in C\(_1\) and C\(_2\) in our study, suggesting that differences in arterial compliance between men and women may express later in life. Scheiken et al.\(^23\) also failed to detect sex differences in aortic stiffness in 11-year-old twins. Second, the mean level of C\(_1\) (2.13 mL/mm Hg) and C\(_2\) (0.083 mL/mm Hg) were higher in the young adults measured in our study using identical measurement techniques, demonstrating that arterial elasticity diminishes with aging.

Fasting insulin was modestly and inversely associated with C\(_2\) after adjustment for BP, lipids, and anthropometric variables: a one standard deviation increase in insulin was associated with a 0.09 mL/mm Hg decrease in C\(_2\) \((P < .001)\). Several studies have demonstrated that diabetics have poorer arterial compliance than nondiabetics.\(^24,25\) A previous study using pulse contour analysis in

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>C(_1) Change</th>
<th>C(_1) P</th>
<th>C(_2) Change</th>
<th>C(_2) P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mm/Hg)</td>
<td>-0.30</td>
<td>&lt;.001</td>
<td>-0.008</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male gender</td>
<td>0.09</td>
<td>.22</td>
<td>0.012</td>
<td>.01</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>0.22</td>
<td>&lt;.001</td>
<td>0.006</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Weight (lb)</td>
<td>0.26</td>
<td>&lt;.001</td>
<td>0.017</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>-0.01</td>
<td>.80</td>
<td>-0.009</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>0.09</td>
<td>&lt;.001</td>
<td>-0.0003</td>
<td>.86</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>0.05</td>
<td>.07</td>
<td>0.0002</td>
<td>.19</td>
</tr>
</tbody>
</table>

\(^*\) C\(_1\) and C\(_2\) regressed on systolic blood pressure, gender, height, weight, insulin, HDL and LDL cholesterol.
diabetics found that reductions in $C_2$ represented a sensitive marker for early vascular abnormalities that occur with diabetes.\textsuperscript{18} Our findings imply that early increases in circulating insulin may exert an effect on oscillatory arterial compliance due small artery susceptibility to insulin resistance.

The sample for the current analysis was selected as part of a population-based sample of school children recruited in 1977. About one-half (46\%) of the participants agreed to be followed longitudinally, and of those 56\% were successfully followed through the post-high school examination. Therefore, our estimates of the relationship between arterial compliance and hypertension may be biased. We consider the magnitude of this potential bias to be small as it would require that the relationship between BP and arterial compliance in the participants lost to follow-up to differ from those observed in this subset. Blood pressure levels in those successfully followed were not significantly different from those that were lost to follow-up; arterial compliance was measured only at the last follow-up visit, precluding making such comparisons for compliance measures.

In conclusion, using the pulse contour analysis technique for ascertainment of arterial compliance, we found a strong, inverse relationship between systolic BP and large artery compliance in healthy, young adults. In contrast, metabolic factors appear to play a more important role than BP in determining small artery compliance. These findings suggest that reductions in large or small arterial compliance occur early in adulthood before the appearance of hypertension.

References