Mechanical Pressure Versus Intrinsic Effects of Hypertension on Large Arteries in Humans

Ricardo Armentano, Alain Simon, Jaime Levenson, Nguyen Phong Chau, Jean Louis Megnien, and Ricardo Pichel

Brachial artery diameter and compliance were measured in 23 normotensive control subjects and 49 hypertensive patients. The results were compared in isobaric conditions by a modeling analysis extrapolating from the measured data a short segment of the pressure–diameter and pressure–compliance curves in the artery. A logarithmic diameter–pressure function was used as well as measurements of brachial artery blood pressure and lumen diameter (by pulsed Doppler), and of brachial-to-radial pulse wave velocity (by mechanography). The measured values of diameter and compliance in the hypertensive patients were 109% and 63%, respectively, of the control group values. By extrapolating the data via the model at the same pressure level in all subjects (the average level of mean blood pressure of the two groups), the isobaric values of diameter and compliance in the hypertensive patients were 107% and 81%, respectively, of the control group values. Overall, measured isobaric diameters and measured compliance correlated with systolic, diastolic, and mean blood pressure values (p<0.001), whereas isobaric compliance correlated only with systolic (p<0.05) and pulse (p<0.01) pressure values. Thus, the increased diameter and reduced compliance of the brachial artery observed in hypertensive humans cannot be attributed solely to the stretching effect of elevated blood pressure, but also to intrinsic alteration of the arterial walls. These could represent either adaptive structural or functional changes secondary to the chronic increase in arterial pressure, or primary abnormalities of the vessel wall. (Hypertension 1991; 18:657–664)

Effects of hypertension on large arteries has become an important field of investigation in hypertensive disease and has received considerable attention recently. Indeed, the large artery changes associated with hypertension may suggest a trend toward early atherosclerotic lesions. Reduction in arterial compliance is a well-known alteration observed in large arteries of hypertensive subjects. It may, in the long term, affect cardiovascular function, particularly by increasing systolic blood pressure and creating an extra load on the heart. Increase in lumen diameter is another large artery change that occurs in sustained hypertension. However, little is known of the physiopathological mechanisms of this increase in large artery diameter and decrease in compliance, particularly its precise relation with elevated blood pressure. It seems likely that pressure elevation per se may be a common factor responsible for increased arterial diameter and decreased arterial compliance. Increasing blood pressure stretches and dilates arteries and reduces their distensibility. The latter effect is explained by the fact that, for the same arterial segment, compliance is a nonlinear function of blood pressure. Therefore, unless the blood pressure is controlled at the same level, it is impossible to know whether the increase in arterial diameter and decrease in compliance are a mechanical consequence of high blood pressure or an intrinsic effect of hypertension on the arterial wall. Therefore, any method used to address this basic question must compare subjects with and without elevated blood pressure at the same level of pressure. We propose in the present study a new technique to derive, via a model, arterial diameter and compliance as functions of pressure. The technique is based on noninvasive measurements of brachial artery diameter (pulsed Doppler) and pulse wave velocity (strain gauge mechanography) and on a logarithmic representation of the diameter–blood pressure curve in the brachial artery. The technique was applied to a group of men with sustained essential hypertension, and the data were compared with those of normotensive men of similar age. This...
The internal lumenal diameter of the brachial artery was determined by a transcutaneous pulsed Doppler7,11 (Echovar Doppler, Alvar Electronic, Montreuil, France).

The system and method of operation have been described in detail previously.7,11 We will describe herein some pertinent characteristics. The system has two original features: first, a double transducer probe to adjust the incidence angle at 60±1° between the ultrasonic beam and the arterial axis; second, a range-gated time system of reception of emitted pulses. By electronically adjusting the delay and duration of reception, it was possible to focus the sample volume of the Doppler signals to 0.04 cm and to advance the sample in successive 0.04-cm steps across the artery. Synchronization with an electrocardiogram enabled the step advance to be automatically started at the QRS complex for every other cardiac pulse. The number of pairs of peaks of the velocity profiles allowed us to calculate the brachial artery diameter by the formula

\[ D = N \times 0.04 \times 0.866 \]

where \( D \) is brachial artery diameter, \( N \) is number of pairs of peaks, and 0.866 is the sine of 60° (the angle between the beam direction and the arterial axis). To improve the reliability of the method, the Doppler probe was fixed throughout the investigation over the course of the artery by a stereotaxic device placed above the arm. Measured diameter was defined as the average of at least two consecutive measurements on each probe and expressed in centimeters. The variability of the method was 7±2%.

Pulse wave velocity along the brachial artery was measured by means of two transcutaneous pulse mechanographic transducer heads (Gould Electronic, Cleveland, Ohio) fixed to the skin over the most prominent parts of the brachial and radial arteries.7 The foot-to-foot pulse wave velocity was calculated as the ratio between the distance separating the two transducers and the time interval separating the feet of the brachial and radial waves. For this purpose, the brachial and radial waves were simultaneously recorded (ES2000 recorder, Gould Electronic, Ballainvilliers, France) on 150 mm/sec speed paper. The foot of each wave was defined as the point obtained by extrapolating the wave front downward to intersect the straight line, extrapolating the last part of the preceding diastolic curve. Measurement of time interval was made in at least 10 pairs of pulses, and the mean value was used to estimate pulse wave velocity (expressed in m/sec). Reproducibility of the method was 8±3%. Using the formula of Bramwell and Hill,13 we derived the following equation for the calculation of arterial compliance, where \( C_m \) (in cm/mm Hg) corresponds to the prevailing pressure:

\[ C_m = \frac{(1334 \cdot D_m)}{(2\rho \cdot PWV^2)} \]  

\( D_m \) is the measured diameter, \( D \) is the blood density (\( \rho = 1.06 \)), and \( PWV \) is pulse wave velocity. The calculation of compliance according to this equation did not refer, as classically, to a change in volume over a change in pressure, but to a change in diameter over the change in pressure (dD/dP); therefore, it describes a segmental diametrical, not a volumic, index of arterial distensibility.

### Table 1. Clinical Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normotensive subjects (n=23)</th>
<th>Hypertensive patients (n=49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>39±14</td>
<td>43±9</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>68±9</td>
<td>72±9</td>
</tr>
<tr>
<td>Pm (mm Hg)</td>
<td>96±7</td>
<td>122±12*</td>
</tr>
<tr>
<td>Dm (cm)</td>
<td>0.463±0.064</td>
<td>0.503±0.059†</td>
</tr>
<tr>
<td>PWV (m/sec)</td>
<td>8.8±1.6</td>
<td>11.6±1.2*</td>
</tr>
<tr>
<td>Cm (10⁻⁴ cm/mm Hg)</td>
<td>3.97±1.01</td>
<td>2.51±0.73*</td>
</tr>
<tr>
<td>En (cm⁻¹)</td>
<td>27.8±6.4</td>
<td>36.3±13.2†</td>
</tr>
</tbody>
</table>

Values are mean±SD. Pm, measured mean blood pressure; Dm, measured brachial artery diameter; PWV, pulse wave velocity; Cm, measured brachial artery compliance; En, normalized elastance.

*\( p<0.001 \), †\( p<0.05 \), ‡\( p<0.01 \).

A comparison was performed at a similar pressure derived via the model, that is, in isobaric conditions.

### Methods

#### Subjects

Twenty-three normotensive men (17–62 years of age) and 49 ambulatory male patients (24–63 years of age) with mild-to-moderate hypertension (defined as the sum of diastolic pressure plus one third of the difference between systolic and diastolic pressures. This difference between systolic and diastolic pressures.

The internal lumenal diameter of the brachial artery was determined by a transcutaneous pulsed Doppler7,11 (Echovar Doppler, Alvar Electronic, Montreuil, France).

The system and method of operation have been described in detail previously.7,11 We will describe herein some pertinent characteristics. The system has two original features: first, a double transducer probe to adjust the incidence angle at 60±1° between the ultrasonic beam and the arterial axis; second, a range-gated time system of reception of emitted pulses. By electronically adjusting the delay and duration of reception, it was possible to focus the sample volume of the Doppler signals to 0.04 cm and to advance the sample in successive 0.04-cm steps across the artery. Synchronization with an electrocardiogram enabled the step advance to be automatically started at the QRS complex for every other cardiac pulse. The number of pairs of peaks of the velocity profiles allowed us to calculate the brachial artery diameter by the formula

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Pulse wave velocity along the brachial artery was measured by means of two transcutaneous pulse mechanographic transducer heads (Gould Electronic, Cleveland, Ohio) fixed to the skin over the most prominent parts of the brachial and radial arteries.7 The foot-to-foot pulse wave velocity was calculated as the ratio between the distance separating the two transducers and the time interval separating the feet of the brachial and radial waves. For this purpose, the brachial and radial waves were simultaneously recorded (ES2000 recorder, Gould Electronic, Ballainvilliers, France) on 150 mm/sec speed paper. The foot of each wave was defined as the point obtained by extrapolating the wave front downward to intersect the straight line, extrapolating the last part of the preceding diastolic curve. Measurement of time interval was made in at least 10 pairs of pulses, and the mean value was used to estimate pulse wave velocity (expressed in m/sec). Reproducibility of the method was 8±3%. Using the formula of Bramwell and Hill,13 we derived the following equation for the calculation of arterial compliance, where \( C_m \) (in cm/mm Hg) corresponds to the prevailing pressure:

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### Estimation of Pressure–Diameter and Compliance Relations

Our method required a mathematical expression for the relation between arterial pressure and diam-
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**FIGURE 1.** Schematic representation of diameter-pressure curve (slashed line) extrapolated via logarithmic model for brachial artery of one subject. Curve passes through the (Dm, Pm) point of measure (●) and is tangent to line (arrow) whose slope (dDm/dPm) is equal to the measured compliance (Cm). Point of curve extrapolated to pressure (Pi) (○) enables calculation of isobaric diameter (Di) corresponding to Pi. Dm, measured diameter; Pm, measured mean blood pressure.

D = D₀ + (D₀/β) log(P/P₀)  \( (2) \)

where D₀ is the diameter corresponding to a standard pressure (P₀) and β is a constant representing the curvature index of the curve. Specifically, the logarithmic curve of this equation was mathematically defined by two parameters only: a definite point of the curve and the slope of the curve at the same point (Figure 1). The point used for defining the diameter of the pressure curve was constituted by the measured diameter and the corresponding measured mean blood pressure. The slope of the curve at this point was the value of the measured arterial compliance (Cm = dDm/dPm), calculated according to equation 1. So, the logarithmic segment of curve that obeyed equation 2 and passed through the working point (Dm, Pm) with a slope to this point equal to Cm was determined by substituting the working point (Dm, Pm) for the (D₀, P₀) point in equation 2 and by calculating the β index. From equation 2 it followed, by differentiation of the measured diameter with respect to (Pm), that [dDm/dPm = Cm = Dm/β · Pm]. This could be written as [Dm/β = CmPm], and inserting this in equation 2 gives:

D = Dm + (CmPm) log(P/Pm)  \( (3) \)

Thus, the compliance-pressure curve was deduced as the first derivative function of the diameter-pressure curve (dD/dP) according to the equation

C = CmPm/P  \( (4) \)

Elastance, the reciprocal of compliance (dP/dD), was calculated as E = P/CmPm, where E is elastance. This last equation enabled us to define a "normalized elastance" (En) \([En = dP/(P · dD)]\) as a constant independent of pressure

En = 1/CmPm  \( (5) \)

Equations 3 and 4 defined the segments of the diameter-pressure and compliance-pressure curves from which diameter and compliance were extrapolated from the measured data at the same level of pressure (Pi) in all subjects, that is, in isobaric conditions (Figure 1). Pi was chosen as the average level of mean blood pressure of the two groups [(mean control pressure + mean hypertensive pressure)/2]. This value was close to the average of mean blood pressure of each group, as well as to the standard pressure of 100 mm Hg used for defining the logarithmic diameter-pressure curve in previous reports.\(^{15-18}\)

**Experimental Validation of the Logarithmic Model**

The purpose of this section is to describe the methodology used to test experimentally the validity of the logarithmic model chosen for representing the in vivo diameter-pressure relation in the human brachial artery. Three mongrel dogs were anesthetized with enflurane 1.5%. The left femoral artery...
was dissected free of surrounding tissue. Two tourniquets were placed at the distal and proximal portions of the vessels to isolate an arterial segment of approximately 10 cm. A cannula was inserted via a collateral branch of this segment for infusion or withdrawal of fluid inside the lumen. Another cannula was inserted in the vessel lumen and connected to a Statham transducer for monitoring the instantaneous pressure change inside the femoral segment. Two piezoelectric crystals were fixed diametrically opposed on the adventitia of the midpart of the femoral segment and connected to a sonomicrometer for monitoring the instantaneous external diameter change of the femoral segment. Then, the vessel segment was isolated by the tourniquets and flushed free of blood, and enough fluid was infused into the vessel to verify by visual inspection that there were no leaks.

Once the vessel was completely emptied, it was filled rapidly by hand injection of 1 ml of physiological solution using an appropriate-sized syringe. During the inflation period, the instantaneous changes in pressure and diameter were monitored on an oscilloscope and recorded on an FM tape recorder (model 3968A, Hewlett-Packard, Palo Alto, Calif.). Using an IBM-PC XT personal computer, the correlation between instantaneous diameter and pressure signals demonstrated the instantaneous diameter–pressure relation for the in situ femoral artery of the dog (Figure 2, top panel). The curves obtained in all three dogs were curvilinear. When the data were analyzed on a semilogarithmic scale, with the diameter on the y axis and the logarithmic transformation of pressure on the x axis, the curve became linear above a threshold of pressure of 37, 32, and 35 mm Hg, respectively, for dogs 1, 2, and 3 (see arrows, Figure 2, bottom panel). Above this threshold, the linear correlation coefficients on a semilogarithmic scale were 0.998, 0.999, and 0.988, respectively, for dogs 1, 2, and 3 (Figure 2, bottom panel).

These data were obtained in a canine large artery similar in size to the human brachial artery. They provide an experimental basis for using with a reasonable confidence a logarithmic curve to represent the passive diameter–pressure relation in the human peripheral muscular large artery, exclusive of the lower pressure range.

**Statistical Analysis**

Group data were expressed as mean±SD. Comparison of arterial parameters between normotensive and hypertensive groups was made by the Student's t test. Differences were considered significant if p<0.05. Correlations were performed by least-squares regression.

**Results**

Compared with the normotensive group, the hypertensive group had larger artery lumen diameters (p<0.05), higher pulse wave velocity (p<0.001), higher normalized elastance (p<0.01), and lower arterial compliance (p<0.001) (Table 1).

Figure 3 demonstrates the pressure–diameter and pressure–compliance curves in the normotensive and hypertensive groups for mean blood pressures ranging from 95 to 125 mm Hg. The curves of the hypertensive group corresponding to diameter and compliance were located, respectively, above and below the curves of the normotensive group.

Isobaric comparison of diameter and compliance at the Pi pressure showed that diameter remained higher (p<0.05) and compliance lower (p<0.01) in the hypertensive group than in the normotensive group (Figure 4).

Table 2 shows correlations in the normotensive and hypertensive groups between measured and isobaric values of diameter and compliance and systolic, diastolic, mean, and pulse values of prevailing blood pressure. Measured and isobaric diameters were both correlated positively with pressure (p<0.001), except for pulse pressure. Measured compliance was negatively correlated to systolic, diastolic, mean, and pulse pressures (p<0.001). Isobaric compliance was correlated to systolic (p<0.05) and pulse (p<0.01) pressures, but not to diastolic and mean pressures. Normalized elastance was correlated to systolic (p<0.05) and pulse (p<0.001) pressures, but not diastolic and mean pressures. Figures 5 and 6 show the individual data points of the correlations between measured and isobaric diameter and compliance and mean blood pressure. Last, no correlation existed between isobaric diameter and isobaric compliance in the normotensive (r=-0.38) or in the hypertensive (r=0.12) groups.
Discussion

In this study, we propose an original, noninvasive modeling method for estimating the mechanical pressure dependence of diameter and compliance of the human large artery in vivo. To this end, we used a nonlinear model for representing the diameter–pressure relation in the brachial artery. We chose a logarithmic relation because it could be mathematically defined by two parameters only, a point of the curve and the slope of the curve at this same point. The point of the curve was the \((D_m,P_m)\) point of measure, with \(D_m\) as the measured brachial artery diameter and \(P_m\), the measured prevailing mean blood pressure. The slope of the curve at this point \((dD_m/dP_m)\) was the segmental arterial compliance \(C_m\) measured at the prevailing pressure of the subject. This index of compliance was different from the classical volumic compliance, defined as a change in volume over a change in pressure. Instead, it expressed the change in diameter over the change in pressure. We chose this unusual compliance index to use as the slope of the diameter–pressure curve. Finally, we determined the compliance–pressure curve as the first derivative function of the diameter–pressure curve. Thus, only three variables, diameter, pressure, and compliance, measured noninvasively in the brachial artery in resting conditions, were needed to define in a relatively easy manner the nonlinear mechanical diameter–pressure and compliance–pressure curves, and thus to derive isobaric values of diameter and compliance at the same pressure level in all subjects. However, there are several considerations regarding assumptions that underlie these methods that need to be addressed and deserve further discussion.

At first, estimations of isobaric values of diameter and compliance were based on the use of a logarithmic model for representing the diameter–pressure curve. This model has been previously applied to human large arteries and validated in in vitro and in vivo studies.\(^{14-18}\) To test more fully its validity, we used an experimental procedure to obtain the pressure–diameter curve for the in situ femoral artery of dogs, in vivo, during acute changes in distention pressure. The passive pressure–diameter curve of unactivated canine femoral artery in...
baseline conditions was curvilinear (Figure 2), but the curve was a pure logarithmic function only above a threshold of pressure of about 30 mm Hg. Indeed, above this threshold, the correlation between the diameter and the logarithmic transformation of pressure was absolutely linear. However, from the results obtained in our experimental conditions, we were not able to identify if the logarithmic diameter–pressure curve took into account the influence of smooth muscle activation. Further studies are necessary to demonstrate how varying degrees of smooth muscle activation may affect the diameter–pressure relation.

Thus, canine large artery data provided an accurate and reliable basis to use a logarithmic function for representing the passive response of the limb artery diameter to change in pressure, independently of the smooth muscle participation and exclusive of the low pressure ranges. Accordingly, we extrapolated not the entire logarithmic diameter–pressure curve, but only a relatively short segment of the curve from the mean blood pressure of the subject. The pressure $P_i$, until which the curve was extrapolated, was the same for all subjects. It was equal to the average level of the mean pressure of normotensive and hypertensive groups. As the number of subjects in both groups was different, the $P_i$ value was...
This means that the model was extrapolated more for the control group than for the hypertensive group. Nevertheless, the Pi value was sufficiently close to the mean blood pressure of any subject that the logarithmic extrapolation was small and performed on a segment relatively linear in semilogarithmic scale. Moreover, it has been shown that the nonlinear pressure-volume curves are fairly close regardless of the parabolic, exponential, or logarithmic function used to represent the curves within the pressure range of our extrapolation.22 On the basis of these arguments, the logarithmic model could be reasonably considered as accounting for the mechanical passive pressure dependence of diameter, and consequently of compliance, within a limited pressure range and without information on the vascular smooth muscle participation. Finally, we preferred using this modeling approach to giving an antihypertensive drug to patients to reduce their blood pressure to a normal level. Indeed, antihypertensive drugs may have an intrinsic effect on arterial smooth muscle, and therefore this approach is not advisable.

A second consideration that could limit our methodology was related to the technical difficulty of accurately determining arterial diameter or pulse wave velocity and to the validity of the formula of Bramwell and Hill13 to calculate arterial compliance. The accuracy of the brachial artery diameter measurement depended on the precision of the location of the proximal and distal arterial walls with a Doppler sample volume.7,11 This volume was reduced to 0.04 cm and was displaced by 0.04 cm gradations. The accuracy of the method has been previously tested in vitro in our laboratory by performing the regression of the diameter measured by the echocardiographic Doppler method against the true diameter of calibrated tubes. The intercept of this regression, which gave an estimation of the error of measurement, was less than 8% of the brachial artery diameter. This error represented a systematic overestimation of diameter due to sample volume size. When comparing data in subjects, the systematic error was eliminated and did not affect the statistical evaluation of the diameter differences.

Concerning pulse wave velocity, we used a technique previously described7,10,22 that included measurements in at least 10 pairs of pulses. The method had excellent short-term reproducibility. However, the capacity of pulse wave velocity to give an in vivo estimation of arterial compliance must be considered with respect to the reflected waves. Indeed, the use of the formula of Bramwell and Hill13 to deduce arterial compliance from pulse wave velocity implied that reflections were negligible.24 On the other hand, it has been shown that even in short arterial segments, such as in the forearm, propagation coefficients were modified by reflected waves.23 In this case, alterations in pulse wave velocity might result, not only from changes in compliance of large arteries, but also from changes in compliance of arterioles downstream.

The present study confirmed, in accordance with previous reports, that large artery diameter was significantly higher1-3,7,30 and compliance lower1-4,7,23,26,30 in hypertensive patients compared with normotensive subjects. The new finding was that increased diameter and decreased compliance persisted when hypertensive and normotensive groups were compared at the same level of pressure. When comparing the two groups at prevailing pressures and at the same pressure via the model, we found that brachial artery diameter was 9% higher at prevailing pressure and 7% higher at isobaric pressure in the hypertensive group than in the normotensive group. Therefore, the increase in arterial diameter due to mechanical stretching by pressure elevation was negligible. Likewise, arterial compliance was 37% lower at prevailing pressure and 19% lower at isobaric pressure in the hypertensive group than in the normotensive group. Therefore, the decrease in arterial compliance due to the mechanical stretching of pressure elevation was only about half of the measured decrease in compliance.

We also developed in our work an index that characterized the elastic modulus of the brachial artery independently of arterial pressure. This index, called “normalized elastance,” expressed the slope of the linear relation predicted by the model between the elastance (dP/dD) and the distention pressure. The elevation in normalized elastance found in hypertensive patients compared with normotensive controls confirmed that the large artery rigidity of hypertensive patients was not dependent only on pressure elevation. Thus, our findings suggest that the abnormalities in large artery diameter and compliance observed in human hypertension are not only due to a mechanical change of arterial walls stretching by high blood pressure. They agree with our previous suggestion that the decrease in forearm arterial compliance in hypertensive patients com-
pared with normotensive subjects cannot be explained solely on the basis of the elevated pressure.\textsuperscript{30}

More recently, it has been demonstrated in subjects undergoing cardiac catheterization that the lower aortic compliance observed in hypertensive patients could not be attributed solely to the higher absolute level of blood pressure, since compliance in the hypertensive group remained lower than in the normotensive group at nearly equal pressures.\textsuperscript{2} Thus, intrinsic factors independent of any mechanical pressure effect likely participated in the large artery changes of hypertension. Among them, the adaptive response of the vessel wall to the chronic elevation in pressure might play a role.\textsuperscript{1,5,6} We tried to analyze this role in the present study by performing correlations between isobaric values of diameter and compliance and the prevailing blood pressure. We found that isobaric diameter and compliance had different relations to the mean and pulsatile components of pressure. After correction for the mechanical influence of pressure, isobaric diameter remained dependent on the magnitude of mean blood pressure, whereas isobaric compliance lost its correlation to mean blood pressure but remained dependent on the amplitude of pulsatile pressure. This suggests that the pulsatility of blood pressure could participate in the intrinsic reduction of arterial compliance to a greater extent than the chronic elevation of mean blood pressure.\textsuperscript{1,6} However, in the present study, it was not possible to ascertain to what extent these intrinsic arterial changes were secondary to chronic high blood pressure or also represented primary abnormalities. Last, further investigations are needed to examine whether the alterations in large artery diameter and compliance are due to changes in arterial wall structure, function, or both elements.

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References

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