Elastic Modulus of the Radial Artery Wall Material Is Not Increased in Patients With Essential Hypertension

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Abstract
Hypertension is known to decrease arterial distensibility and systemic compliance. However, the arterial tree is not homogeneous, and it has been shown that the medium-size radial artery does not behave like the proximal, elastic, large, common carotid artery. Indeed, radial artery compliance in hypertensive patients (HTs) has been shown to be paradoxically increased when compared with that in normotensive control subjects (NTs) at the same blood pressure level. To determine whether this increase was due to hypertension-related hypertrophy of the arterial wall, radial artery functional and geometric parameters from 22 NTs (mean ± SD, 44 ± 11 years) were compared with those from 25 age- and sex-matched never-treated essential HTs (48 ± 12 years) by using a high-precision ultrasonic, echo-tracking system coupled to a photoplethysmograph (Finapres system), which allows simultaneous arterial internal diameter, intima-media thickness, and finger blood pressure measurements. When the values for HTs were compared with those of NTs at their respective mean arterial pressures, HTs had similar internal diameter (2.50 ± 0.56 versus 2.53 ± 0.32 mm, mean ± SD) and greater intima-media thickness (0.40 ± 0.06 versus 0.28 ± 0.05 mm, P < .001) measurements and increased arterial wall cross-sectional areas (3.79 ± 1.14 versus 2.45 ± 0.57 mm², P < .001).

Hypertension is known to decrease arterial distensibility and systemic compliance and has been reported for pressure-dimension experiments as well as ring and strip studies of human and animal large arteries in vivo and in vitro. However, the arterial tree is not homogeneous, and it has been shown that the radial artery, a muscular, medium-size artery, does not behave like the common carotid artery, a proximal, elastic large artery. Indeed, radial artery distensibility and compliance in hypertensive patients are not significantly different from those of normotensive control subjects when the two populations are compared at their mean arterial pressures (MAPs). Furthermore, when the two groups are compared at the same blood pressure (BP; ie, 100 mm Hg) by means of distensibility- and compliance-pressure curves, it appears that distensibility (D₁₀₀) and compliance (C₁₀₀) in hypertensives are not significantly lower than in normotensives, being either unchanged or higher. These findings contrast with the well-known decrease in proximal large-artery compliance due to hypertension and suggest that adaptive changes occur in hypertensive, medium-size arteries. The unchanged (or higher) radial artery distensibility (D₁₀₀) and compliance (C₁₀₀) values observed in hypertensives compared with normotensives may be due to hypertension-related hypertrophy of vascular smooth muscle, among other factors. Because smooth muscle is more prominent in medium-size than large arteries, this characteristic may decrease the relative amount of the less extensible connective tissue in the vessel wall, thus favoring a "structural" increase in compliance. Compliance of cerebral arterioles as well has been shown to be increased in stroke-prone spontaneously hypertensive rats (SHRs) despite hypertrophy of the vessel wall. In never-treated hypertensive patients, we have demonstrated increased intima-media thickness at the radial artery site by using a method described and validated by Tardy et al and our group. Compared with age- and sex-matched normotensives, hyperten-
sives have a significantly higher intima-media thickness and cross-sectional area of the radial artery wall, thus confirming in vivo the reported increases in arterial intima-media thickness in hypertensives.

In a recent article Mulvany suggests that normal compliance and distensibility can be achieved only if the elastic modulus (a measure of elasticity independent of geometry) of the wall components is reduced. Indeed, Baumbach et al and Mulvany have shown that the elastic modulus of artery wall material for a given wall stress decreases in small arteries from SHR compared with normotensive controls. To our knowledge, whether the elastic modulus of medium-size artery wall material is reduced in hypertensive humans has not been determined.

To obtain information concerning the human vasculature in vivo, we noninvasively determined radial artery intima-media thickness and pulsatile changes in diameter and BP. Using these data, we assessed (1) the elastic response of the artery via distensibility- and compliance-pressure curves and (2) the elastic properties of artery wall material via the incremental modulus of elasticity (Eint). The main finding of this study is that at the same BP or circumferential stress, the elastic modulus of radial artery wall material is not increased in hypertensive patients compared with that obtained in age- and sex-matched normotensives.

Methods

Subjects and Patients

Twenty-two normotensive subjects (range, 25 to 64 years; mean ± SD, 44±11 years) and 25 untreated patients with mild or moderate essential hypertensive (range, 28 to 72 years; mean ± SD, 48±12 years) were included in the study. Normotension was defined by BP values <140/90 mm Hg at repeated visits. Hypertension was defined as mild to moderate by supine diastolic pressures (DBPs) of 95 to 114 mm Hg on three consecutive visits. All essential hypertensive patients included in this study had never received any antihypertensive treatment. No patient had signs or symptoms of hypertensive complications, and none had valvular heart disease, major arrhythmia, carotid artery stenosis, or noncardiovascular diseases. The study was approved by the Ethics Committee of Broussais Hospital, and all patients gave written informed consent. The principal clinical characteristics of the subjects and patients are given in Table 1.

The investigation was performed between 9 and 11 AM in a controlled environment kept at 22±2°C. BP was measured with a mercury sphygmomanometer and a cuff adapted to their arm circumference after the subjects had been recumbent for at least 15 minutes. Systolic blood pressure (SBP) was defined by the pressure at which phase 1 Korotkoff sounds appeared and DBP by the pressure at which they disappeared (phase V). MAP was calculated as DBP plus one-third pulse pressure. Radial artery parameters (internal diameter and intima-media thickness) were measured simultaneously with the noninvasive determination of finger BP by a Finapres system (see below). Two separate determinations of BP, at the site of the brachial and digital arteries, were used and are presented in the tables (brachial BP) and figures (finger BP).

Measurement of Radial Artery Diameter and Intima-Media Thickness

The ultrasound system used in this study has been described and validated for measurement of radial artery internal diameter and intima-media thickness. A high-resolution, pulse-echo tracking device (NIMS 02, SMH: Capital Medical Services) was used to acquire backscattered radio frequency (RF) data from the radial artery at the wrist. The probe consisted of a 10-MHz strongly focused piezoelectric transducer (5.2-mm diameter, 11-mm focal length) operated in the pulse-echo mode. The -10-dB beam width is 0.3 mm at the focal point, and the depth of field at -10 dB is 5 mm. A stereotactic arm permits motion of the transducer at x, y, and z coordinates in micrometric steps to place the probe perpendicular to the arterial axis, i.e., at its largest cross-sectional dimension. The transducer was positioned so that its focal zone was at the center of the artery and the backscattered echoes from both anterior and posterior walls could be visualized. A typical RF signal was then displayed on a computer monitor interfaced to the transducer system. Arterial diameter and posterior intima-media thickness were measured when a "double peak" RF signal received from the arterial walls was obtained. These signals are visible only when the ultrasound beam crosses the axis (center) of the vessel; they are characterized by an initial high-amplitude signal followed by a relatively silent acoustic zone and then a second, high-amplitude signal. To measure intima-media thickness of the posterior wall, trackers were positioned on the leading edge of echoes as illustrated in the upper part of Fig 1. Movements were tracked for 60 seconds, and the RF signal was sampled at 100 MHz over 8 bits. Software tracker movements were computed at 250 Hz, averaged five times, and then stored at a 50-Hz repetition frequency on a 200-megabyte hard disk for further data processing. Data were derived by computing the analytic signal according to previously described methods that are based on Fourier transformation.

Finally, diameter and intima-media thickness were determined by multiplying time measurements by the approximate speed of sound in tissue (1.54 mm/µs) and expressed in micrometers. All data processing was performed using software developed by Asulab and installed in a 486/33-MHz AT computer. The pulse length of the 10-MHz ultrasound system was 0.1 µsecond at 6 dB and corresponded to a practical axial resolution of 160 µm for absolute intimal thickness or intima-media thickness measurements and 2.5 µm for these parameters during systolic-diastolic changes.

Radial artery parameters were studied with the subject in the supine position and at least 20 minutes rest. The radial artery was studied 2 cm from the wrist in the nondominant arm after the forearm had been extended and secured comfortably on a splint. For each subject, five measurements of systolic-diastolic variations in internal diameter and posterior wall intima-media thickness over several cardiac cycles were
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POSTERIOR WALL
INTERNAL DIAMETER

MEDIA-ADVENT ITIA
LUMEN-INTIMA INTERFACES

INTIMA-MEDIA THICKNESS.

FIG 1. Typical radiofrequency (RF) signal of a human radial artery. The probe is placed perpendicular to the arterial axis at its largest cross-sectional dimension. The transducer is positioned so that its focal zone is at the center of the artery and the backscattered echoes from both anterior and posterior walls can be visualized. To measure internal diameter and intima-media wall thickness of the posterior wall, trackers are positioned on the leading edges of echoes, as illustrated by the filled arrows.

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recorded (an example from a normotensive subject is shown in Fig 2). Only one observer (J.-J.M.) was involved in the study.

Repeatability of In Vivo Measurements

Repeatability of radial artery diameter and intima-media thickness measurements was investigated in 10 subjects (5 normotensives and 5 hypertensives) by calculating the repeatability coefficient (RC), as defined by the British Standard Institution23 according to the formula

\[
RC^2 = \Sigma D_i^2 n
\]

where \(n\) is the sample size and \(D_i\) the relative difference within each pair of measures. This coefficient is the SD of the estimated difference between two repeated measurements. RC for intraobserver repeatability (comparison of two determinations obtained at a 10-minute interval by the same observer) for radial artery diameter and intima-media thickness was 97 and 47 \(\mu m\), respectively. These values were small when compared with the actual values of radial artery diameter and intima-media thickness: 2367±443 and 399±61 \(\mu m\), respectively, and with the difference between normotensives and hypertensives. These measurements therefore seemed sufficiently repeatable for use in cross-sectional clinical studies.

Calculation of Arterial Distensibility and Compliance

The echo-tracking system described above was coupled to a commercially available plethysmograph (Finapres system, Ohmeda, BOC Group Inc) to allow simultaneous BP measurement. From the two simultaneous and continuous recordings of arterial diameter and BP, the computerized data acquisition system derived the cross-sectional pressure curve, fitted it by using an arctan model with three independent parameters, and then calculated the cross-sectional compliance– and distensibility-pressure curves. Distension (change in volume) of an artery during a cardiac cycle depends on the elastic characteristics of the vessel wall and surrounding tissue and the local pulse pressure. This relation can be expressed in terms of either compliance, when the change in volume is expressed in absolute values, or distensibility, when the change in volume is expressed as a percentage of the baseline value. Because changes in arterial volume are mainly due to changes in arterial lumen cross section, compliance may also be defined as the change in arterial cross section induced by a change in arterial pressure. Because the elastic properties of the arterial wall are a function of BP, compliance is determined over the systolic-diastolic range of pressures (ie, pulse pressure) and is therefore characterized by a compliance-pressure curve or a distensibility-pressure curve. Compliance and distensibility calculated at 100 mm Hg (ie, \(C_{100}\) and \(D_{100}\)) were arbitrarily chosen as reference points for comparing normotensives and hypertensives.

Calculation of Circumferential Stress and Elastic Modulus

Circumferential wall stress (\(\sigma\), in dynes per square centimeter \(\cdot 10^2\)) was calculated as \(MAP \times D/2h\), where \(MAP\) is mean arterial pressure, \(D\) the mean lumen diameter, and \(h\) the combined thickness of the media and intima. \(MAP\) was converted from millimeters of mercury to kilopascals (kPa; 1 mm Hg=0.133 kPa). The wall to lumen ratio was calculated as \(2h/D\).

In contrast to compliance, which provides information about elasticity of the artery as a hollow structure, the incremental modulus of elasticity (\(E_{inc}\)) provides direct information on the elastic properties of the wall material, independently of the vessel geometry. By definition, for a right cylindrical vessel
with wall stiffnesses that are equal in all directions, $E_{\infty}$ is the slope of the stress-strain curve and can be defined by

$$E_{\infty} = \frac{\sigma_{\infty}}{\varepsilon_{\infty}} = \frac{d_0}{d_0} \left( \frac{\delta \sigma}{\delta \varepsilon} \right)$$

where $\varepsilon = (d - d_0)/d_0$ is strain and $d_0$ the diameter at zero transmural pressure. For in vivo measurements, $d_0$ is generally unknown, which prevents calculation of strain and consequently, estimation of the modulus of elasticity. Therefore, we applied Hooke's law, which determines the behavior of thick-walled tubes, to the calculation of the $E_{\infty}$, a procedure that does not require knowledge of the unloaded state (zero distending pressure). $E_{\infty}$ is calculated (see "Appendix") as

$$3 \times \left( \frac{LCSA}{WCSA} \right)$$

distensibility

where LCSA is the lumen cross-sectional area calculated as a function of BP as described above, WCSA is the mean wall cross-sectional area, and distensibility is a function of BP, calculated as described above. $E_{\infty}$, pressure curves were then calculated. In contrast to wall thickness, WCSA is not influenced by BP because of the incompressibility of the arterial mass. WCSA, expressed in square millimeters, was calculated as $m(R_e^2 - R_i^2)$, where $R_e$ is the mean internal radius plus mean intima-media thickness (millimeters) and $R_i$ is the mean internal radius (millimeters). Elastic modulus was also expressed as a function of circumferential wall stress.

**Statistical Analysis**

All values were averaged and expressed as mean±SD, except in figures for which mean±SEM was used for clarity. BP and arterial parameters were compared in normotensive and hypertensive subjects with unpaired Student’s $t$ test. To compare the diameter-pressure curve of hypertensives with that of normotensives, we first calculated the area under the curve for each subject. Then the areas under the curve of hypertensives were compared with those of normotensives by unpaired Student’s $t$ test, as though they were raw data. Areas under the curve were calculated within the overlapping section of the normotensive and hypertensive curves, ie, within the section of pulse pressure common to normotensives and hypertensives (95 to 130 mm Hg). The same procedure was followed for comparison of distensibility-, compliance-, or elastic modulus–pressure curves and the elastic modulus–circumferential stress curve between normotensives and hypertensives.

**Results**

Table 2 shows the arterial parameters of normotensive subjects and hypertensive patients. At respective MAPs, radial artery intima-media thickness and WCSA were greater in hypertensives than normotensives ($P<.05$), whereas there was no difference in internal end-diastolic diameter. Circumferential wall stress was not significantly different between the two groups.

As BP increased from diastolic to systolic values, radial artery distensibility and compliance increased in a curvilinear fashion (Figs 4 and 5). Distensibility and compliance were greater in hypertensive patients than normotensive subjects when compared for the range of BP common to both groups. This observed upward shift of the curve in hypertensives was statistically significant ($P<.05$, Figs 4 and 5). Distensibility and compliance calculated at 100 mm Hg were significantly greater in hypertensives than normotensives ($P<.05$), whereas distensibility and compliance calculated for their respective MAPs were not significantly different (Table 2).

In the whole population, WCSA was positively correlated with age and MAP (Table 2). $C_m$ was significantly correlated with $WCSA$ ($P<.02$), independently of age and MAP (Fig 6).

Elastic modulus increased in a curvilinear fashion as BP increased from diastolic to systolic values (Fig 7). The $E_{\infty}$-pressure curve was significantly shifted downward in hypertensives ($P<.01$). $E_{\infty}$ was significantly reduced in hypertensives ($P<.01$) when they were compared with normotensives at the same level of BP, ie,
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1227

20
15
10
5
0
-5
-10
-15
-20

ARTERIAL PRESSURE (mmHg)

ARTERIAL DISTENSIBILITY (kPa×10⁻³)

Fig 4. Mean radial artery distensibility-pressure curves in 22 normotensive (NT) subjects (○) and 25 hypertensive (HT) patients (●). The curve was significantly shifted upward in HT (P<.05). Values are mean±SEM.

100 mm Hg (Table 2). $E_{oc}$ increased in a curvilinear fashion as circumferential stress increased from diastolic to systolic values (Fig 8). The $E_{oc}$-circumferential stress curves in hypertensives were not significantly different from those in normotensives. In addition, the $E_{oc}$ in hypertensives was not significantly different from that in normotensives when both groups were compared at the same level of circumferential stress (Fig 8).

Discussion

This study provides the first noninvasive determination of the elastic characteristics of conduit artery wall material in hypertensive patients. The present investigation was designed to evaluate (1) the elastic response of the radial artery via distensibility- and compliance-pressure curves and (2) the elastic properties of the radial artery wall material via $E_{oc}$. The main finding of this study is that $E_{oc}$ of the radial artery wall material is not increased in hypertensive patients compared with that obtained at the same level of BP or circumferential stress in age- and sex-matched normotensives.

Consideration of Methods

The method used in this study is completely noninvasive, because it uses RF signals to measure internal artery diameter and intima-media thickness and photoplethysmography to continuously record BP. The ultrasound device is characterized by a high precision of internal arterial diameter and intima-media thickness measurements, thus permitting reliable calculation of arterial LCSA and WCSA.13-15 This precision is indispensable for detecting changes in arterial geometry that occur within the cardiac cycle, for establishing pressure-diameter curves, and for deriving distensibility-, compliance-, and $E_{oc}$-pressure curves. In a previous study,14 we validated the ultrasonic measurement of the radial artery intima-media thickness by determining the correlation between histological and ultrasonic measurements in 15 radial artery segments, as obtained from the distal end of wrist-elbow harvest for coronary artery bypass grafting in patients with coronary heart disease. A positive correlation was observed between ultrasonic and histological measurements ($r=.618, P<.014$), and the difference between ultrasound and histology measurements was 41±66 μm, with higher measurements found by the ultrasonic device. In a subgroup of 11
patients, we determined the correlation between in vivo ultrasonic measurements of radial artery intima-media thickness at the preoperative stage and in vitro ultrasonic measurements of intima-media thickness obtained postoperatively in the same arterial segments. In vitro WCSA was correlated with in vivo WCSA ($r=0.929$; $P<0.0001$). These results, together with the good repeatability of in vivo measurements, indicate that intima-media thickness measurements can be used for cross-sectional clinical studies, provided the former are done by an experienced investigator following a standardized protocol.

The Finapres method is one of the most commonly used noninvasive systems for continuous assessment of BP variations during the cardiac cycle. We used this system in conjunction with the ultrasound device to determine the diameter-pressure curve within the systolic-diastolic range. Several studies have shown that BP measured indirectly in a finger by the Finapres method is similar to that measured simultaneously in a brachial or a radial artery under resting conditions. Imholz et al. showed that Finapres SBPs and DBPs, on average, differed by -3.5 and -4.4 mm Hg, respectively, from intrabrachial pressures. Compared with intraradial BP, Finapres SBPs and DBPs differed, on average by +1.2 and +2.9 mm Hg, respectively. Although the reliability of the Finapres method to detect BP changes during phenylephrine infusion has recently been challenged, it is generally accepted as a valid and reliable method for estimating forearm BP under resting conditions.

In normotensives, the character of the pulse waveform recorded at the site of the radial artery with the Finapres system was not much different from that in mild hypertensives, in contrast to the marked changes that hypertension induces at the site of the common carotid artery by means of pulse-wave amplification. The lack of change in the finger pulse waveform between normotensives and hypertensives was not unexpected, because changes in pulse waveform are mainly due to wave reflections along the arterial system from distal to proximal sites. Because the digital artery is very close to distal reflection sites, a pulse wave amplification may not have occurred.

To compare the $E_\infty$ of arterial wall components in hypertensives to that in normotensives for the same BP level, we needed to calculate $E_\infty$ in hypertensives over a range of BP that overlapped that of normotensives. We chose to calculate the $E_\infty$-pressure curve over the systolic-diastolic range rather than from the end points of the pressure-diameter curve generated during one cardiac cycle under varying levels of BP. Indeed, the maneuvers used to vary BP can affect the pressure-diameter curve, particularly through changes in baroreflex activity and radial artery vasomotor tone.

To determine $E_\infty$, we applied Hooke's law for thick-walled tubes. We considered that the general case of a thick-walled tube was more appropriate for a vessel like the radial artery, whose wall to lumen ratio is close to 0.3 (Table 2). Previous studies using Young's modulus have generally been performed in larger, more proximal, and more elastic arteries like the carotid artery or the thoracic aorta, which are characterized by a smaller wall to lumen ratio (close to 0.1). In addition, the application of Hooke's law does not require knowledge of the unloaded (unstretched) state.

**Considerations of Findings**

**Arterial Compliance**

Previous studies have demonstrated that sustained hypertension decreases large-artery distensibility and compliance. This has been reported for pressure-dimension experiments as well as ring and strip studies of human and animal large arteries both in vivo and in vitro. Thus, in contrast to large arteries, our findings suggest normal distensibility at the site of medium-size arteries during sustained hypertension, despite hypertrophy of the arterial wall. A likely explanation for the difference in our findings and those of previous studies is that the arteries examined in these studies were of different sizes. Composition of the arterial wall varies with vessel size, and the effect of hypertension on the conduit artery may vary with vessel caliber. For instance, previous studies on forearm arterial distensibility have not documented a decrease.

Using determinations of pulse-wave velocity measured on the human forearm enclosed in an airtight box, Gribbin et al. and Smulyan et al. showed that, for the same mean transmural pressure, normotensive and hypertensive subjects had the same pulse-wave velocity and therefore the same distensibility as that derived from the Bramwell and Hill formula. Whereas distensibility of the basilar artery and branches of the posterior cerebral artery has been reported to be reduced in SHRs, compared with normotensive Wistar-Kyoto rats, the incremental distensibility of cerebral arteries was significantly greater in older SHRs than in Wistar-Kyoto rats. Possible mechanisms that might explain the increase in distensibility for a given level of BP are unknown. However, the fact that we observed a significant relationship between $C_{100}$ and $WCSA$ suggests that arterial wall hypertrophy may be associated with these mechanisms.

**Elastic Modulus**

We have shown that at MAP, $E_\infty$ in hypertensives is not significantly increased compared with that in normotensives. Moreover, the $E_\infty$ is significantly reduced in hypertensives when both groups are compared at the...
same level of BP (ie, 100 mm Hg). However, the \( E_{\text{ec}} \) pressure curve depends not only on the properties of the wall material but also on the way in which the material is arranged. Therefore, to provide information that is dependent on the properties of the material only, we calculated \( E_{\text{ec}} \) for a given circumferential stress. Under these conditions, no significant increase in \( E_{\text{ec}} \) was observed in hypertensives. That the \( E_{\text{ec}} \) of the radial artery wall material was not increased in hypertensive patients compared with that in normotensives at the same level of BP or circumferential stress is apparently contrary to the common observation that the stiffness of arterial wall material is increased in hypertension.\(^2\)\(^{3}\)\(^{4}\)\(^{5}\)\(^{6}\) It should be emphasized, however, that several studies have shown that arterial wall stiffness is heterogeneous and that alterations at peripheral sites may not invariably reflect those that occur centrally. For example, using autopsy samples, Learoyd and Taylor\(^6\) observed that in the case of the thoracic aorta, the values of Young’s modulus calculated for a given BP were increased in old compared with young individuals. For peripheral arteries, this relation was reversed, with the younger vessels having the greater \( E_{\text{ec}} \). However, such a comparison may not be an appropriate means of comparing changes in arterial wall mechanics because of the stress dependence of arterial wall mechanics. In animals, Baumbach et al\(^1\) and Mulvany\(^2\) showed that the \( E_{\text{ec}} \) of the wall materials for a given wall stress was decreased in small arteries from SHRs compared with normotensive controls.

**Implications of Findings**

Our results suggest that the elastic response of the radial artery is maintained despite hypertrophy of the arterial wall. An advantage of hypertrophy is that an increase in wall thickness presumably attenuates increases in wall stress that accompany increases in intravascular pressure. We suggest that at the site of distal, muscular, medium-size arteries, a second advantage of hypertrophy could be the maintenance of “normal” compliance despite the increase in intravascular pressure at a given circumferential stress in hyper-

\[ E_{\text{ec}} \]

can be derived from Equation 3 as

\[ E_{\text{ec}} = \frac{p}{A_{\text{ec}}} \]

where \( p \) is distending pressure, \( A_{\text{ec}} \) is the elastic modulus, \( b \) is the internal radius, and \( a \) is Poisson’s ratio.

The mechanism that might explain the lack of increase in \( E_{\text{ec}} \) at a given circumferential stress in hypertensives remains purely speculative. Mulvany\(^1\) has suggested that this could be an alteration in the tissue properties of the individual wall components or in the relative proportions of these components in the wall. As suggested by Baumbach et al\(^1\), reduction in the \( E_{\text{ec}} \) of the vascular wall material could be a method that the vasculature uses to maintain its distensibility characteristics despite the relatively increased intima-media thickness required by intravascular pressure. Whether the \( E_{\text{ec}} \) of wall material remains unchanged in muscular, medium-size arteries from other vascular territories of hypertensives is not known. If this were the case, it would suggest that such a distal adaptive mechanism could at least partially compensate for the hypertension-induced decreased distensibility and compliance of proximal large arteries. The contribution of distal, muscular, medium-size arteries toward central aortic compliance remains controversial. Because of their large number, distal, muscular, medium-size arteries may represent a substantial part of the arterial compliance of the whole arterial tree, despite their low compliance.\(^9\)

Indeed, a model of the arterial circulation suggests that distal processes contribute significantly (>35%) to central aortic compliance.\(^7\)

In addition, maintenance of “normal” compliance at the site of distal, muscular, medium-size arteries, despite the decrease in proximal compliance, leads to reduction of the compliance gradient between proximal and distal arteries. Because the compliance gradient between different parts of the arterial tree has been reported to generate wave reflections,\(^4\)\(^5\)\(^6\) which increase pulse pressure and cardiac afterload, we suggest that this decrease in compliance gradient could be a means by which the vasculature attenuates wave reflections and pulse pressure at central arterial sites.

**Appendix**

The relationship between pressure \( P \) and the lumen cross-sectional area \( LCSA \) was fitted with the model of Langewouters et al,\(^1\)\(^4\)\(^5\) which uses an arctan function and three optimal-fit parameters:

\[ LCSA = a \left( \frac{\pi}{2 + \tan^{-1} \left( \frac{P - B}{\gamma} \right)} \right) \quad \text{with} \quad LCSA = \frac{\pi D^2}{4} \]

where \( D \) is the internal diameter for a right cylindrical vessel. The three parameters \( a, \beta, \) and \( \gamma \) fully characterize the diameter-pressure curve.

Local arterial cross-sectional compliance (\( C \)) for a right cylindrical vessel is defined by the change in lumen cross-sectional area (\( 8LCSA \)) for a given change in intravascular pressure (\( \delta P \)).

\[ C(P) = \frac{8LCSA}{\delta P} \]

Because of the nonlinearity of the \( LCSA-P \) curve, \( C \) decreases as BP increases. To determine \( C \) for a given level of BP, we established the \( C-P \) curve over the systolic-diastolic range. This was done by deriving the equation for the \( P-LCSA \) curve.\(^1\) By using Equation 1, the following analytic form was obtained for local arterial compliance:

\[ C = \frac{\alpha}{\gamma + [(P - \beta)/\gamma]^2} \]

Arterial cross-sectional distensibility (Distensibility) is the \( C \) value normalized for \( LCSA \) and is defined by

\[ \text{Distensibility} = \frac{1}{LCSA} \cdot \frac{8LCSA}{\delta P} \]

We applied Hooke’s law,\(^1\)\(^2\)\(^4\) which determines the behavior of an elastic, homogeneous, isotropic material, to the calculation of the incremental circumferential elastic modulus. This procedure does not require knowledge of the unloaded (unstretched) state. The incremental elastic modulus is calculated from the following classic formula:

\[ \Delta b = \frac{p b}{E_{\text{ec}}} \cdot \frac{a^2(1 + \nu) + b^2(1 - 2\nu)}{a^2 - b^2} \]

where \( \Delta b \) is the change in inner radius from baseline in response to an increase in distending pressure, \( p \) is distending pressure, \( E_{\text{ec}} \) is the elastic modulus, \( b \) is the inner radius, \( a \) is the outer radius, and \( \nu \) is Poisson’s ratio.

This formula is valid for thick-walled tubes composed of isotropic material. For incompressible material (\( \nu = 0.5 \); References 4 and 25), \( E_{\text{ec}} \) can be derived from Equation 3 as
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Thus pulsatile change in inner radius. The Ab

p(b) can be written as

\[ p(b) = p(b_0) + \frac{\partial p}{\partial b} \Delta b \]

where \( p(b) \) is the internal diameter, \( D \) is the distance between two points on the arterial wall, \( \Delta b \) is the pulsatile change in inner radius, and \( b_0 \) is the initial radius.

Therefore, the compliance of the arterial wall can be calculated as

\[ E_{wa} = \frac{p(b) - p(b_0)}{\Delta b} \frac{1}{1 + \frac{LCSA}{WCSA}} \]

where \( LMC \) is the mean wall cross-sectional area and \( \Delta b \) is the pulsatile change in inner radius. \( p(b) \) can be written as

\[ p(b) = p(b_0) + \frac{\partial p}{\partial b} \Delta b \]

Since \( p(\Delta b) = \frac{\partial p}{\partial b} = 0 \), where \( D \) is the internal diameter,

\[ E_{wa} = \frac{p(b)}{\Delta b} \frac{1}{1 + \frac{LCSA}{WCSA}} \]

Since \( \frac{\partial p}{\partial b} = LCSA \frac{p(b)}{LCSA} \), the compliance is calculated as

\[ E_{wa} = \frac{3}{2} \frac{1}{LCSA} \frac{1}{WCSA} \]

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References

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