Alterations with Age in the Viscoelastic Properties of Human Arterial Walls

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In contrast to the large amount of information available on the histological structure of arteries, relatively little attention has been paid to the physical constants of the arterial wall. Measurements of this nature are in fact of more value to the physiologist than even detailed qualitative descriptions of wall structure. This is because the pulsatile pressure flow relationships in arteries are intimately related to these wall constants, which must therefore be known with some precision if an adequate understanding of circulatory dynamics is to be achieved. Although Bergel has studied the physical properties of dog arteries in considerable detail, the present investigation was undertaken in order to make similar observations on human vessels.

Following Bergel's technique, the circumferential incremental Young's modulus \( E \) of the arteries was measured under both static and dynamic conditions. The latter parameter is a complex quantity, because there is a phase difference between the pressure and diameter oscillations.

As in the dog, a progressive increase in \( E \) was found with increasing distance from the heart. For any given vessel under dynamic conditions, values of \( E \) rose over the low frequency range, (up to 3 cycles/sec) and then remained nearly constant with frequency.

Methods

Arterial diameter was measured photoelectrically. The artery was held vertically in the centre of a horizontal light beam which was brought to a focus on a photoelectric cell, (IP21 Amalgamated Wireless Australasia) the output of which was determined therefore by the diameter of the artery. Except for the addition of a few modifications given below, the apparatus employed was essentially the same as that used by Bergel. The light source showed too much drift when driven by a 12 volt battery so this was replaced by a d-c power supply stable to ± 5 mv. Immediately after each experiment the instrument was calibrated with a range of standard rods. The calibration was linear in all cases. Additional refinements were introduced also into the resolving circuit described by Taylor. The commutator wheel with mechanical contacts was replaced by a photoelectric switching device which permitted reading Fourier coefficients up to the 3rd harmonic. Fifty-nine arterial segments taken from 12 subjects were studied. Details of the origin of these segments may be found in table 1.

The arteries were removed at postmortem and stored frozen in physiological saline. Bergel has shown that prolonged storage in this way does not change the physical properties significantly. Any grossly atheromatous vessels were discarded. Since all arteries retract longitudinally on removal from the body, the length of each segment was measured carefully while it was still in the body. The segment was then held stretched to this length while the experimental observations were being made. Before making the measurements the arteries were inflated several times to pressures up to 250 mm Hg to eliminate the excessive hysteresis found on first inflation (Remington). The arterial wall is assumed to have a Poisson's ratio of 0.5, its volume remains constant under stress, so that wall thickness may be calculated at any pressure provided the volume is known. Wall volume was found for each specimen by measuring its loss of weight when suspended in distilled water. Knowing the wall thickness, the external radius, and the internal

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TABLE 1

Details of Origin of Arterial Segments on Which Both Static (S) and Dynamic (D) Measurements Were Made

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Subject</th>
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<th>Abdominal</th>
<th>Thoracic</th>
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<td>1</td>
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<td>M.B.</td>
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<td>1</td>
<td>1</td>
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<tr>
<td>19</td>
<td>M</td>
<td>J.A.</td>
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<td>1</td>
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</tbody>
</table>

Totals “young” 7(5)* 5(5) 5(4) 3(3) 5(5) 4(4) 6(5) 3(3) 5(3) 3(3)

| 36  | M   | R.E.   | 3        | 1         | 1        | 1         | 1        | 1         | 1        | 1         |
| 44  | F   | E.T.   | 2        | 1         | 1        | 1         | 1        | 1         | 1        | 1         |
| 45  | F   | E.R.   | 2        | 1         | 1        | 1         | 1        | 1         | 1        | 1         |
| 45  | F   | N.S.   | 2        | 1         | 1        | 1         | 1        | 1         | 1        | 1         |
| 50  | F   | U.L.   | 1        |           |          |           |          |           |          |           |
| 52  | F   | J.H.   | 2        | 2         | 1        | 2         | 1        | 2         | 1        | 2         |

Totals “old” 11(6) 7(5) 4(3) 2(2) 8(3) 3(2) 3(3) 1(1) 5(4) 2(2)

*Numbers in parentheses indicate the number of subjects contributing to any given total of arterial segments. Dynamic measurements were made on arterial segments which had been used for static measurements.

Pressure, \( E \) for static conditions \( (E_s) \) was calculated at increments of 10 mm Hg over the pressure range, 30 to 240 mm Hg. The formula used was that derived by Bergel\(^2\) from Love,\(^7\) viz.:

\[
E_s = \frac{P_3 - P_1}{R_3 - R_1} \cdot \left( \frac{2(1 - \sigma^2)}{R_1^2} \right) \left( \frac{R_3^2 - R_2^2}{R_2^2 - R_0^2} \right)
\]

Where \( E \) is the circumferential incremental Young’s modulus, \( P \) is the internal distending pressure, \( R_1 \) is the inside radius, \( R_0 \) is the outside radius and \( \sigma \) is Poisson’s ratio which is assumed\(^8\) to be 0.5. Subscripts 1, 2, 3 refer to successive observations of the quantity concerned made at pressure intervals of 10 mm Hg.

For dynamic studies all vessels were held at a mean pressure of 100 mm Hg and submitted to oscillations of pressure of ± 5 to 15 mm Hg about that mean. These pressure oscillations were produced by means of a cam-driven syringe producing a sinusoidal volume change, which was run at frequencies 1 to 10 cycles/sec. The outputs of the pressure transducer and the photocell measuring diameter were amplified and resolved independently up to 3rd harmonic. The modulus of the 1st harmonics of pressure and diameter were then calculated, together with the phase difference between them. In these experiments the 1st harmonic was corrected only for the element of 3rd it contained, as the content of higher harmonics was negligible. The values were substituted into the above formula to calculate \( E \) for dynamic conditions which is a complex quantity \( (E_c) \). The manometer behaviour was analysed by the method of Hansen,\(^9\) it was shown to have an undamped natural frequency of 135 cycles/sec with a damping factor of 0.055. Appropriate amplitude and phase corrections for the pressure oscillations were introduced into the computations, all of which were done using the SILLIAC computer at the University of Sydney.

Results

In order to obtain information on the effect of aging on the physical properties of arteries, the results have, in general, been considered in two groups, viz., “young” less than 35 years old, and “old” over 35 years. These groups consisted usually of approximately similar numbers of arteries as indicated in the legend with each figure.

Static Measurements

(i) Longitudinal Retraction

This is the amount by which a segment of vessel shortened on removal from the body, expressed as a percentage of the length of the segment in situ. In figure 1 the percentage retraction has been plotted at each site for the two groups. The “young” arteries show a significantly greater retraction than the “old” and in both groups retraction increases progressively towards the periphery.

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Instead of absolute values for wall thickness \( h \), the ratio of wall thickness to mean diameter (relative wall thickness, \( h/2R \)) has been graphed in figure 2 for the two groups over the pressure range 50 to 150 mm Hg. “Young” and “old” arteries differed in an interesting manner. In the “old” the relative wall thickness increased progressively in the more peripheral vessels whereas the “young” showed a fairly constant wall thickness ratio except for a slight increase in the femoral.
Considerable difference between the "young" and "old" groups was found. The distribution of the curves in the "young" subjects indicates a rise in modulus at any given pressure, when tested progressively in a peripheral direction down the arterial tree. This latter feature, however, is not present in the older subjects. In fact the reverse situation holds to some extent since the "old" thoracic vessels are stiffer than the more peripherally situated arteries. They are also stiffer than the "young" thoracic aortae but the peripheral vessels (iliac and femoral) in the "old" have a lower \( E_s \) than the same vessels in the "young." Although these findings show considerable scatter there is sufficient evidence to indicate that the "old" arterial tree has undergone significant changes since it was "young."

(iv) Degree of Extension

The effect of varying the degree of longi-

\[ \text{FIGURE 3} \]

Average values for circumferential incremental Young's modulus of arterial segments of "young" subjects graphed against internal pressure in mm Hg. Site from which segments were taken is indicated by the legend. Scatter about the mean is indicated by the limits of \( \pm 2 \) standard errors of the mean (SEM). In all arteries the SEM decreased with decreasing pressure. Numbers of each segment studied are as given for "young" subjects in figure 2.

\[ \text{FIGURE 4} \]

Average values for circumferential incremental Young's modulus of arterial segments of "old" subjects graphed against internal pressure in mm Hg. Site of origin of the segments is as indicated by the legend in figure 3. Numbers of each segment studied are as given for old subjects in figure 2.

Vessels. These findings are relevant to the aging process as will be discussed below.

(iii) Static Circumferential Incremental Young's Modulus \( (E_s) \)

The results of the computations of \( E_s \) over the pressure range 50 to 150 mm Hg are graphed in figure 3 for "young" and in figure 4 for "old" subjects. All vessels showed an increase in \( E_s \) as the distending pressure rose. This is a reflection of the nonlinear relationship (concave to the pressure axis) which exists between the pressure and the diameter of all arteries. It has been shown by Roach and Burton\(^{10} \) that this is due to the progressive transfer of the circumferential wall stress from the readily stretched elastin fibres to the very inextensible collagen fibres as the pressure and hence the diameter increases. Thus, at small degrees of distension the value of \( E_s \) is determined largely by elastin and when greatly distended it is mainly due to collagen.
tudinal extension of the artery was explored also in several specimens. Figures 5 and 6 show $E_s$ for an iliac artery graphed against pressure, and against external radius, respectively, when the artery is held at each of three different lengths. It is apparent that the form of the $E_s$ versus pressure relationship does not depend greatly upon the degree of

**FIGURE 5**

Circumferential incremental Young's modulus, $E_s$, (Y.M., ordinate) plotted against internal pressure for a human iliac artery (subject 45 years old) at each of three grades of extension expressed as a percentage of the unstretched length.

**FIGURE 6**

Circumferential incremental Young's modulus, $E_s$, (Y.M., ordinate) plotted against external radius (abscissa) for the same artery as in figure 5 at the same three grades of extension.
longitudinal stretch, but figure 6 shows clearly that the value of $E_a$ at any given external radius is increased considerably by stretching the specimen lengthwise. The Laplace equation ($T = PR$ where $T$ = circumferential wall tension, $P$ = internal distending pressure and $R$ = radius), though strictly applicable only to tubes with infinitely thin walls, provides a basis for understanding the effect of longitudinal stretching on the circumferential modulus of an artery.

Since the fibrous and muscular elements of the arterial wall are arranged in longitudinal helices, stretching a specimen lengthwise will, at any given internal pressure, reduce the radius. Because of the Laplace relationship a reduction in $R$ will reduce the circumferential tension developed in the wall if the internal pressure is kept constant. This will tend to offset any increase in circumferential wall tension due directly to longitudinal stretching of the helically wound fibres.

Since the modulus of elasticity depends on the circumferential strain, and hence on the stress, which is due to the wall tension, it can be seen that increasing longitudinal stretch need not necessarily produce a rise of $E_a$ at any given pressure. It may in fact cause a reduction of $E_a$ if the effect of the reduction in the radius more than compensates for the increased circumferential tension produced by the stretching. Bergel\(^5\) has made an observation which shows this latter effect and a suggestion of such behaviour can be seen in figure 5.

However, if $E_a$ is graphed against external radius the situation is very different. Since a higher pressure is required to maintain a given radius, much higher circumferential wall tensions are developed when the lengthwise extension is increased and the values of $E_a$ at a given radius show a marked increase as the extension of the specimen increases.

**Dynamic Measurements**

The results of dynamic studies have been graphed in figures 7 and 8. The parameters computed were the phase lag ($\phi$) of diameter behind the pressure and the circumferential incremental Young's modulus which, under dynamic conditions, is a complex quantity ($E_c$ complex, $E_a$).

Following Hardung,\(^1\) the real and imaginary parts of this complex number have been expressed independently. The former is the dynamic elastic component $E_D$ and the latter the viscous retarding force ($\eta\omega$). $\eta$ is the viscous constant and $\omega$ the angular frequency.

These are derived simply from the complex elastic modulus $E_a$ by the following relationships:

$$E_D = E_c \cos \phi$$
$$\eta\omega = E_c \sin \phi$$

If the arterial wall is conceived to be represented by a simple Kelvin-Voigt element consisting of a spring and dashpot in parallel then $E_D$ represents the spring constant and $\eta$ the viscous constant for the dashpot.

The ratio of the dynamic modulus to the static modulus has also been graphed. ($E_D/E_a$ is this modulus ratio). Frequency dependence of the dynamic constants is evident particularly over the range 1 to 3 cycles/sec. From 3 to 10 cycles/sec there is little change in the parameters graphed. All vessels showed an increase in the relative stiffness, $E_D/E_a$, and the phase angle, $\phi$, with frequency. This was most marked in the femoral arteries, particularly in the "young" group. All vessels showed a decrease in viscosity with increasing frequency, indicating that thixotropic behaviour occurs in arterial walls.

**Pulse Wave Velocity**

The rate of travel of a pressure wave in a nonviscous fluid within an elastic tube of infinite length is given by the Moens equation\(^1\)

$$v = \frac{Eh}{2\pi R^2}$$

where $E = \text{elastic modulus of the wall}$

$h = \text{wall thickness}$

$R = \text{mean radius of the tube}$

$\rho = \text{density of fluid}$.

In considering pressure waves in arteries, $\rho$ is the density of blood and as it is very
close to unity (1.055) it may be ignored. All the other quantities on the right-hand side of the equation have been measured and it is therefore possible to derive a theoretical value for pulse wave velocity. A comparison may then be made with measurements of the foot-to-foot velocity made in vivo since this provides a good approximation to the true wave velocity. The degree of correspondence between these two velocities gives some indication of the confidence with which the experimental measurements of the wall constants made in vitro may be applied to the arterial tree in vivo.

Accordingly, in patients who, for other reasons, were required to undergo retrograde
aortic catheterization, recordings of the arterial pulse wave were made at 10 cm intervals from the aortic valve to the femoral artery.* From these records measurements of the foot-to-foot velocity, using the brachial artery pressure pulse as a reference, were made in the thoracic aorta, abdominal aorta and iliac arteries. Unfortunately the site of femoral puncture was too high in the groin to permit such measurements in the femoral artery. It should be mentioned also that none of the arterial segments used in this study was taken from these patients.

Some explanation is required with respect to the values actually substituted into the Moens equation. Since the foot of the pulse wave is produced by the high frequency components (10 to 20 cycles/sec) the value which should be substituted for $E$ in the Moens equation is the dynamic modulus for

*These recordings were supplied by Dr. A. D. Jose, Research Director, Hallstrom Institute of Cardiology, Royal Prince Alfred Hospital, Sydney. We acknowledge gratefully his ready co-operation in this regard.
oscillations of this frequency at a mean pressure equivalent to the average of the mean blood pressures of the group of patients studied. This was approximately 80 mm Hg. Unfortunately no such measurements are available since all the data presented here were obtained from dynamic measurements made at a mean pressure of 100 mm Hg for frequencies up to 10 cycles/sec. However, values of $E_s$ at 80 mm Hg have been determined. Some observations have also been made on the dynamic constants at different mean pressures and these show that the values of $\phi$ and $E_D/E_s$ remain relatively unchanged for a given vessel over the pressure range 50 to 150 mm Hg. Therefore measurements of $E_D/E_s$ obtained at 100 mm Hg may be applied to values of $E_s$ at 80 mm Hg in order to calculate $E_D$ at 80 mm Hg.

Although values of $E_D/E_s$ are known only up to 10 cycles/sec these show very little progressive rise above 3 cycles/sec. Little error would therefore be introduced by using such values at 10 cycles/sec to calculate $E_D$ at 10 to 20 cycles/sec. The best approximation to the correct value of $E$ to be substituted in the Moens equation is therefore given by:

$$E = \text{average } E_s \text{ at } 80 \text{ mm Hg} \times \text{average } E_D/E_s \text{ for 10 cycles/sec at 100 mm Hg.}$$

Since $h/2R$ at 80 mm Hg has been measured directly, calculation of pulse wave velocity could be made.

The comparison of the measured and theoretical pulse wave velocities is shown in figure 9. Despite the considerable scatter in the direct measurements the correlation between the two graphs indicates at least that the experimental results are of the correct order of magnitude, particularly as they also agree well with other reported figures.14, 15 The appropriate separation of "old" and "young" groups adds further confirmation.

**Discussion**

The findings in the present series of experiments are qualitatively similar to the data on dogs' arteries presented by Bergel.5, 3 Both studies show an increasing $E_s$ in all vessels with an increase in pressure, and, under dynamic conditions, the frequency dependence of the various parameters is similar. Since the dogs Bergel used were a uniform population of young animals they should be compared with the "young" humans. Such comparison reveals several differences. The human arteries had wall constants which were two or three times greater than the values observed in dogs. There was also a progressive increase of wall stiffness in the more peripherally situated arteries. This latter feature is not nearly so apparent in the dog although its thoracic aorta is less stiff than the other vessels.

**Figure 9**

Average values for pulse wave velocity calculated as described in text compared with average of direct measurement of foot-to-foot velocity made in a different group of five "young" subjects and five "old" subjects. Standard errors of the means of the measured values for "young" and "old" respectively were: thoracic aorta 1.1, 1.8; abdominal aorta 0.64, 1.0; iliac arteries 0.55, 0.72.
Under dynamic conditions the behaviour of all parameters was, again qualitatively similar to that found by Bergel. The quantitative differences in the measurements of $E_d$ and $\eta \omega$ are simply a reflection of the static differences already discussed. However, the graphs for $E_d/E_s$ and $\phi$ are independent of the absolute value of the wall modulus and permit making a direct comparison. The correspondence between the two sets of observations is very good. Both studies show a relatively sharp increase of $\phi$ and $E_d/E_s$ in the low frequency range (1 to 3 cycles/sec) which is most marked in the femoral artery in man and the carotid artery in the dog. Bergel has discussed the relationship between these parameters and the muscle content of the various arteries. He concludes that the muscle present in an artery is chiefly responsible for its viscosity and this explains the greater phase lag in the dog carotid and femoral. This contention seems logical and is applicable also to the present studies even though the actual experimental results differ.

In man the femoral artery, a very muscular vessel, is by far the most viscous, having a greater phase lag than any of the other arteries studied. The human carotid artery showed, in the present studies, a distinctly lower value for $\phi$ which was of the same order as that for the thoracic aorta. Here a qualitative species difference may be manifest. Although on histological examination Bergel found the dog carotid to be a muscular artery, standard texts\textsuperscript{16} classify the human common carotid with the elastic vessels exemplified by the thoracic aorta. It is also very likely that the high modulus ratio exhibited by the femoral artery is another consequence of its large content of smooth muscle. The time constants of stress relaxation in smooth muscle are known to be long (Abbott and Lowy\textsuperscript{17}) so that a muscular organ would be expected to exhibit a considerably higher elastic modulus under dynamic conditions even at low frequencies. The nonviability of the muscle in these experiments probably would not greatly affect the results since Remington and Alexander\textsuperscript{18} have shown that the viscoelastic properties of dead kitten bladder are very similar to those exhibited by the living tissue. The above effects would be most marked in arteries which had the highest muscle content, i.e., femoral in man and both femoral and carotid in dog.

Perhaps the most interesting finding in this series of experiments is the marked difference between “old” and “young” arteries. The values for $E_d$ in the “young” showed a progressive increase with distance from the heart. However in the “old,” $E_d$ in thoracic aorta was considerably higher than in the “young” and the change with distance from the heart was quite different, there being lower values of $E_d$ in the more peripheral arteries.

The following interpretation of these findings is offered. Two well known changes accompany aging of the cardiovascular system. These are dilation of the thoracic aorta\textsuperscript{19} and increased thickness of arterial walls.\textsuperscript{20} These changes are exemplified by the measurements of the external vessel diameters and wall thicknesses seen in figure 10. Here the average values for external radius and relative wall thickness at different sites down the arterial tree are graphed for “young” and “old” subjects. It is clear that old vessels become dilated and thickened but these two phenomena differ in degree at different sites. The proximal arteries are more prone to dilation and the peripheral arteries to wall thickening.

Bergel\textsuperscript{5} has shown, and our observations confirm, that for a given artery, $E_d$ is related more to the circumference of the vessel than to the internal distending pressure. However the change in wall stiffness with age cannot be explained solely on the basis of dilation. At 100 mm Hg, $E_d$ of the “young” thoracic aortae averaged 7.5 \times 10^6 dynes/cm\textsuperscript{2} and for the “old” the average was 16.6 \times 10^6 dynes/cm\textsuperscript{2}. It was not until the pressure reached 135 mm Hg that the average modulus of the “young” aortae approached 17 \times 10^6 dynes/cm\textsuperscript{2} but this was associated with an increase in external radius of only 5 to 10%. At 100
mm Hg the average radius of the "old" vessels was 20% greater than that in the "young" (see fig. 10). If the "young" vessels had been distended till their radius increased by 20% the modulus would have been many times greater than the $16.6 \times 10^6$ the "old" arteries exhibited at this radius. Thus, in spite of a higher elastic modulus it appears that the "old" aorta has a considerably weaker wall than the "young" one.

To confirm this conclusion, a graph for each portion of the arterial tree has been constructed in which the average value of $E_s$ at 100 mm Hg for each subject is plotted against the subject's age (fig. 11). In the thoracic aorta there is a progressive increase of $E_s$ but the other vessels show either a decrease (iliac) or no significant change with age. To eliminate the effect of the greater degree of distention of the "older" thoracic vessels the values of $E_s$ in the thoracic aortae at an external radius of 1.0 cm are plotted against age and show a significant decrease (fig. 11-thoracic (b)). There is little dilation with age in the other parts of the arterial tree and evidence for a decrease of $E_s$ at 100 mm Hg in the iliac artery. Although no significant regression is seen with age in the abdominal, femoral and carotid arteries, these graphs are not inconsistent with the hypothesis that all arteries become weaker with age.

Since Cleary has shown that no significant changes appear with age in the relative amounts of the fibrous proteins (collagen and elastin) constituting the wall of any artery, one is forced to conclude that some qualitative deficiency occurs in the wall elements. This causes a reduction in the tensile strength of the wall with age and permits considerable dilation of the "old" thoracic aortae. The distribution of the stresses in the walls of these vessels loads the collagen fibres relatively more than in the "young" and produces the higher elastic moduli of the former.

In the peripheral vessels this dilation is prevented by the laying down of large additional amounts of collagen and elastin. The proportions of these two elements is maintained unchanged as evidenced by Cleary's measurements but the greater mass of tissue causes marked thickening of the wall. These fibrous proteins may be of inferior quality but nevertheless permit the wall tension to be widely distributed and hence the stress on any single wall element is considerably reduced.

There is strong histological evidence to sup-
VISCOELASTICITY OF HUMAN ARTERIES

Static circumferential incremental Young's modulus $E_s$, at a pressure of 100 mm Hg (thoracic (b) excepted) graphed against the age of the subject. Thoracic (b) shows $E_s$ for thoracic aortae measured at an external radius of 1.0 cm graphed against the age of the subject. Each point represents the mean value of all the arterial segments taken from the particular site in any given subject. Subject CC (11 yrs) has been excluded from thoracic (b) since her aorta never achieved an external radius of 1.0 cm at any pressure. Regression lines, where significant, have been drawn. Regression coefficients and their significance ("t" test) are as follows: Thoracic (a): $+0.36, 0.01 > P > 0.001$. Thoracic (b): $-0.29, 0.01 > P > 0.001$. Iliac: $-0.65, 0.05 > P > 0.02$. If the regression lines are calculated for all the arterial segments the values given above become: thoracic (a): $+0.36, 0.05 > P > 0.01$. Thoracic (b): $-0.34, 0.001 > P$. Iliac: $-0.48, 0.02 > P > 0.01$.

Support the above thesis of a weakening of the wall with age. General disorganization of the wall elements accompanied by fragmentation of elastin and local hyaline degeneration of collagen have been described.\(^2\)\(^2\)\(^3\) Also the newly deposited elastin fibres are known to have slightly abnormal staining properties.\(^2\)\(^2\) Although other studies\(^2\)\(^4\) on the aging aortae of rabbits indicate a weakening of the arterial wall with age, the above observations seem to be at variance with the conclusions, reached by Roach and Burton\(^2\)\(^5\) from their measurements on human iliac arteries. They showed, in a large series of measurements, that the "elastance," which was derived directly from pressure volume curves increased progressively with age. Since by their definition "elastance" is equal to Young's modulus ($YM_t$), they inferred that $YM_t$ also increased with age because their histological sections of undistended vessels showed a constant wall thickness in all age groups. To account for these findings they deduce that the collagen content per unit mass in iliac arteries must increase by about 60% during life. As Cleary's observations on the collagen and elastin content in human arterial walls do not confirm this, some other interpretation of Roach and Burton's findings must therefore be sought.

It would be surprising indeed if our findings agreed precisely with those of Roach and Burton, since the "Young's modulus" measured in each case differed. They attempted to calculate a true Young's modulus whereas we preferred the incremental modu-
lus. This could account for significant quantitative differences but the gross differences in directional change with age require further explanation. The most likely cause for the difference lies in the assumption Roach and Burton made regarding the wall thickness ($t$). They used values of $t$ measured microscopically on undistended specimens to derive the wall modulus at higher pressures, e.g., 100 mm Hg. This assumption, that the wall thickness remains constant with pressure would introduce considerable error particularly in the young vessels which probably retract more (circumferentially) in the undistended state. At higher pressures therefore they would have much thinner walls and hence much higher moduli than those authors conclude. Another source of difference between the two sets of observations is the fact that our specimens were held stretched at the length they exhibited in vivo, whereas Roach and Burton allowed their specimens to contract and expand longitudinally with changes in internal pressure. This would accentuate the effects due to the change in wall thickness discussed above. It is suggested that the combination of these factors is sufficient to produce opposite trends with age in the two sets of observations and that the concept of a weakening of the wall elements due to degeneration with age is more consistent with the histological and biochemical evidence.

The dynamic modulus ($E_D$) of the “old” group showed no differences from the “young” which could not be attributed to the changes in $E_s$ already discussed. Although the differences in the other dynamic parameters may not be statistically significant they do suggest that the “young” peripheral arteries have a greater viscosity than the “old” ones. This, as explained above, is consistent with a higher smooth muscle content which is not unlikely in the younger arteries. It is not clear why $\rho c$ is higher in the “old” thoracic aorta unless this also is a result of its greater degree of circumferential stretch.

In spite of the rather gross differences in the values for $E$ and $h/2R$ used for “old” and “young” arteries the computations of pulse wave velocity (PWV) showed a satisfactory agreement with direct measurements of foot-to-foot velocity even to the extent of demonstrating the acknowledged increase in PWV with age.26 The general agreement between the calculated and the observed values supports the validity of the experimental measurements of the wall parameters although the differences between “young” and “old” PWV may not be statistically significant.

It is now pertinent to look at the arterial tree in toto and consider the physiological appropriateness of the wall properties with which this study is concerned. In order for the cardiovascular system to perform optimally, certain theoretical requirements in its design must be met. These have been discussed by Taylor,27 who pointed out the necessity in such a system for the matching of impedance both proximally (heart to proximal aorta) and distally (terminal arterial tree to peripheral arterioles).

In circulatory dynamics the impedance of any given section of the arterial tree is a measure of the resistance it offers to the oscillatory flow of blood. It can be expressed quantitatively as the ratio of the pressure rise inside the vessel to the volume of blood flowing into it. In the absence of flow measurements the pulse wave velocity gives a good indication of vascular impedance, being directly related to it in any given vessel. For the work of the heart to be a minimum it is necessary that the thoracic aorta should offer a low impedance to the outflow of blood from the heart. This will occur if it is an easily distensible tube having a relatively thin wall of low coefficient of elasticity and hence a low pulse wave velocity. Such is the case in the “young” aorta but the “old” aorta is considerably stiffer. However this is partly compensated for by dilation, which, due to the vessel’s larger initial volume, permits the heart to discharge its stroke volume into it without excessive rise in pressure, thus maintaining a relatively low impedance. The pulse wave velocity measurements confirm this,
showing only a small increase in the “old” thoracic aortae.

At the other end of the system, the impedance of the femoral artery should, to fulfill theoretical requirements, match the high impedance of the arterioles. This is desirable because such a situation will minimize the reflected components of the pulse wave, which if large, would produce circumstances unfavourable to the heart. At the other end of the system, the impedance of the femoral artery should, to fulfill theoretical requirements, match the high impedance of the arterioles. This is desirable because such a situation will minimize the reflected components of the pulse wave, which if large, would produce circumstances unfavourable to the heart.

Thus in “young” femoral arteries the wall modulus is high, which in addition to a slight increase in wall thickness ratio $h/2R$ ensures a high impedance. In the “old” femoral, in association with the general weakening of the fibrous elements, the wall becomes very greatly thickened which prevents excessive dilation and hence a relatively high impedance is maintained. The rise in the pulse wave velocity towards the periphery in both groups supports these deductions.

**Summary**

The circumferential incremental Young’s modulus was measured in 59 major arteries of both “young” (less than 35 years of age) and “old” (greater than 35 years of age) subjects. Dynamic measurements of wall elasticity and viscosity were made which indicated a high viscosity in the femoral arteries. This was attributed to their high content of muscle.

Although the “young” group showed, at any given pressure, an increasing wall stiffness towards the periphery, in “old” arteries, an opposite trend was found. When dimensional changes (radius and wall thickness) in the “old” group were considered it was apparent that at all sites the arterial wall tissue became weaker with age. Nevertheless, as a consequence of these dimensional changes the impedance characteristics of the old arterial tree still retained the nonuniformity (an increase towards the periphery) of the “young” which has considerable haemodynamic advantage.

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