Correlation of Visco-Elastic Properties of Large Arteries with Microscopic Structure

V. EFFECTS OF SINUSOIDAL FORCINGS AT LOW AND AT RESONANCE FREQUENCIES

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ABSTRACT

Nine aortas from recently killed dogs were sectioned into 21 or more ring segments supported horizontally on two hooks in Ringer's solution. One hook oscillated sinusoidally from .01 to 21 Hz to stretch the segments 1.2% in excess of 4 or more mean strain levels from 5 to 100%. The segments were kept at 4 temperature levels (0°, 20°, 37°, 60°C). The other hook was coupled to a force transducer. At frequencies below 1 Hz, the force registered was sinusoidal with the same frequency as the stretch which it led, unless the specimen contained demonstrably contracted smooth muscle; then the stress was nonlinear and lagged behind the strain at frequencies below .05 Hz. As frequencies rose above 1 Hz, the force amplitude rose to a maximum, resonating at frequency $\omega_0$, which was higher at higher initial strains. Concurrently the phase shift increased to 90° at another frequency $\omega_0'$. When $\omega_0' > \omega_0$, viscous losses were appreciable, in agreement with other measurements and implicating either muscle or elastin. These two wall components could be distinguished from each other by responses to drugs and to changes in temperature. Absolute dynamic modulus, storage modulus, loss modulus, phase shift, and loss angle measured from stress-strain loops compared favorably with similar measurements published for other visco-elastic materials and with viscous and elastic constants obtained from stress-relaxation experiments on aorta.

ADDITIONAL KEY WORDS

sinusoidal forcings muscle nonlinearities resonance

loss modulus storage modulus

visco-elasticity dog aorta elastin collagen

absolute dynamic modulus phase shift

Visco-elastic constants already obtained from step-function-induced stress-relaxation curves (1, 2) are theoretically sufficient (3) to permit predictions of the responses to other forcings (4-6). Step-function forcings have not been adequate, however, for determining whether stress-relaxation can be described correctly by an exponential curve with a single time constant (7) nor for obtaining the total value of an elastic constant which is related to the loss modulus (1). It is not clear whether this difficulty derives from material properties or from the fact that a true step-function stretch or release is not realizable, since it takes a finite amount of time to go from one length to another. This question can be settled only with sinusoidal forcings which span frequencies far below resonance (visco-elastic region) or are demonstrably in the resonance region.

The present work succeeded in inducing sinusoidal circumferential stretch of arteries at frequencies from .01 to 21 Hz which spanned the visco-elastic as well as resonance behavior. Suitable measurements were taken
from the stress-strain loops produced at low frequencies to give the frequency dependence of the absolute dynamic modulus, of the storage and loss moduli, and of the phase shift. These values were checked against the maximum dynamic modulus and viscous losses measured by the resonance response. In this way, the visco-elastic properties of at least 21 aortic regions of nine dogs were described in the frequency domain, whereas stress-relaxation studies previously reported (1-3) gave properties in the time domain. Suitable transfer functions given in the glossary made it possible to bridge from one domain to the other. What is more, the frequency studies revealed some unsuspected nonlinear behavior of smooth muscle which could influence the response of composite materials like aorta to complex forcings like the blood inflow curve during systole (4-6).

Methods

The aortas were removed from nine recently killed dogs. Each aorta was sectioned into rings 0.3 cm long and studied immediately for the behavior in Ringer's solution with and without 4 μg/ml of phenylephrine hydrochloride to test the responses of smooth muscle. The same segments were then kept at 4°C in Ringer's solution with pH controlled at 7.2 for at least 24 hours before being tested for the behavior of elastin and collagen (2). Each aorta provided about 27 segments. One aorta, prepared to contain only elastin according to the method of Hass (8), was also sectioned into rings and tested like the intact aortas. Adventitia removed by microdissection (2) provided arterial collagen rings.

Each ring was supported horizontally by two hooks in the bath. Temperature was controlled at four levels: 0°, 20°, 37°, and 60°C to use the thermal response for identifying the behavior of individual wall components (2, 9-11) to sinusoidal forcings for comparison with the responses to step-function forcings (1, 2). Otherwise the segments were studied at 37°C. The rings were stretched to 6 or 7 strain levels (5% to 100%) by separating the hooks to register an initial mean force, m.

The sinusoidal motion of one of the supporting hooks initiated sinusoidal stretch (less than 1% strain in excess of the mean force, m) of the specimen. The other hook was attached to a Statham strain gauge (frequency response, 350 Hz) which registered the force induced by the sinusoidal stretch. The gauge output was fed into a damped resonant frequency (12). (See Glossary for definition of terms.)

To help identify the wall component under stretch and therefore responsible for these measurements, the amplitude, B, of the sinusoidal response to a strain of constant amplitude, A, at ω = 0.1 was recorded continuously, superimposed on the mean force, m, while temperature was varied over the available range (Fig. 1). The thermal response, already shown to be unique for each of the three wall components, elastin, muscle, and collagen (5-4, 10, 11), was used to identify the stretched component.

From the relation between |G|_{max} and |G|_{min} and \( a_1, a_2 \) and \( η \) in glossary equations 3 and 4, it was possible to transfer from the time to the frequency domain. The parameter \( t_η \) is needed for this transformation and is obtainable more easily for muscle which has a linear circumference-force relationship (1) than for elastin or collagen which are linear only after all fibers are stretched (1).

Results

All specimens of the dog aorta (except those containing contracted smooth muscle) responded to a sinusoidal stretch by developing
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FIGURE 1
Time course of response of aorta 14 cm from valve. \( \theta \) = temperature; \( \epsilon \) = displacement at frequency \( \omega = 0.1 \text{ Hz} \); \( \sigma \) = force. Three mean force levels (and therefore strain levels) were tested. (A) \( m = 20 \text{ g} \); rise in temperature from 20 to 38°C. Mean force and force amplitude increase at first; but later both mean force and amplitude drop markedly, like other smooth muscle (15). (B) \( m = 156 \text{ g cm}^{-1} \); rise in temperature from 15 to 37°C. Mean force and force amplitude increase just as elastin would. (C) \( m = 220 \text{ cm}^{-1} \); rise in temperature from 18 to 37°C. Mean force increases because of a shortening of elastic lamellae but amplitude decreases, because of the thermal response of collagen. (D) The same conditions as in C, but the medium is 30% ethylene glycol instead of Ringer's solution and temperature rise was from 18 to 51°C. There is no change in the length of elastic fibers, but the amplitude of the force drops. The time scale is shorter and the amplitude gain lower than in A, B or C.

A sinusoidally varying force that was of the same frequency but preceded the stretching function by an angle \( \phi \). The behavior of the aortic wall at frequencies below resonance had to be distinguished from the behavior in the resonance region.* At resonance, all aortic regions behaved essentially alike at all levels of circumferential strain, whether muscle was contracted or relaxed. That is, the amplitude of the stress increased, then decreased, as the frequency of the strain increased, with the maximum amplitude occurring at \( \omega_r \). Concurrently, the strain lagged behind the stress by a phase angle \( \phi \) reaching 90° at \( \omega_{05} \), which was always greater than or equal to \( \omega_r \). The higher \( \omega_r \), the larger was the dynamic modulus; the greater the difference between \( \omega_{05} \) and \( \omega_r \), the larger the viscous losses. The modulus and losses were comparable in magnitude to those computed from stress-relaxation curves. Theoretically it is possible to compute \( |G|_{\text{max}} \) and \( \eta \) from the form of the resonance curve. However, this form was not reliable in these experiments because the apparatus was not accurate at \( \omega > 20 \text{ Hz} \) and because the visco-elastic region at \( \omega < \omega_r \) (13) often extended into the resonance region.

At frequencies below resonance, the behavior depended on the state of the muscle, the level of the circumferential strain, and the aortic region being tested. Muscle, elastin, and collagen, tested separately, behaved distinctly, while aortic wall behaved like any one of these components, depending on the circumferential strain or the wall composition determined chemically or histologically (1). In general, the behavior resembled standard linear solids as long as \( \omega < \omega_r \) (13).

Elastin.—A sinusoidal stretch of pure aortic elastin induced a sinusoidal force which led the strain representing a loss angle \( \phi \). The loss angle increased to a maximum, then decreased to zero, before resonance set in. \( G_2 \) was maximal where \( \phi \) was maximal at a frequency, \( \omega^* \), which is a function of the viscous losses, as glossary expression 7 for \( \eta \) indicates. When viscosity was low, \( \omega^* \) was high and entered the resonance region so that \( \phi \) was not zero at the end of the visco-elastic region. Nevertheless, \( G_1 \) and \( G_2 \) could be computed as illustrated in Figure 2. A \( |G|_{\text{min}} \) occurred at lowest frequencies and a \( |G|_{\text{max}} \) at the frequency where \( \phi \) returned to zero.

Adventitial Collagen.—A sinusoidal stretch induced a sinusoidal force with no phase shift until the high frequencies where resonance

*It is not yet clear whether the particular behavior designated here as "resonance" is due alone to the properties of the material or also to peculiarities of the apparatus, but studies to investigate the source are in progress.

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set in (Fig. 3). This behavior is elastic rather than visco-elastic, and the modulus was much higher than for elastin.

Smooth Muscle.—Guinea pig bladder and highly muscular aorta (between the renal arteries, for example) at low strains responded to a sinusoidal stretch with a periodic, but not sinusoidal, force which lagged behind the

(A) Aorta 14 cm from valve; $\epsilon = 1.4$; $m = 75$ g cm$^{-1}$, muscle relaxed. Plots of $|G|$, $G_e$, $G_s$, tan $\phi$ versus frequency. The response was sinusoidal. Up to $\omega = 0.7$ Hz, the material behaves like a standard linear solid (13, 14). "Resonance" occurred at $\omega_r = 8$; 90° phase shift at $\omega_{90} = 11$. The location of $\omega^*$ is noted. It is where $G_s$ is maximal. (B) Aortic elastin. "Resonance" occurred at $\omega_r = 8$; 90° phase shift at $\omega_{90} = 11$.

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strain at lowest frequencies (Figs. 4 and 5). When strain leads stress, a unique “gain angle” results. The gain angle reverted to a loss angle which increased to a maximum, then decreased to zero as the frequency of the strain was increased. Meanwhile the force amplitude (and therefore |G|) increased from a minimum, giving |G|_\text{min} at the lowest frequencies to a plateau giving |G|_\text{max} when the lag became zero. The storage modulus, G_\text{s}, and the loss modulus, G_\text{L}, could not be validly computed because of the nonlinear behavior.

However, ω^* could give one measure of viscosity from glossary equation 7.

**Intact Aorta.**—(1) If the strain was less than 1.6 and muscle was contracted, then the wall strain led the stress at low frequencies. Such behavior suggested that smooth muscle was being stretched, since it alone of arterial wall components has a gain rather than a loss...
angle at low frequencies. The more muscle, the more likely was the peculiar phase shift to be detected at low frequencies and the larger was the viscous loss computed from glossary equation 5 or 7. This behavior resembled smooth muscle in the other ways described above and is illustrated in Figure 4.

(2) If the strain was less than 1.6 and the muscle was relaxed, or if the strain was between 1.6 and 1.8 (whether muscle were contracted or not), the aorta behaved like elastin (Fig. 2). (3) Aorta strained more than 1.8 behaved at low frequencies like a perfectly elastic material of a much higher modulus than elastin or muscle (Fig. 3). Resonance occurred at much higher frequency than at lower strains and even showed no viscous loss by $\omega_0 = \omega_r$ as long as $\epsilon > 2.0$. However, often at $1.8 < \epsilon < 2.0$, $\omega_0 > \omega_r$ although there was no evidence of viscous loss at low frequencies even at this range of strains. This anomalous behavior is somewhat characteristic of a Voigt material. Otherwise, highly strained aorta behaved like adventitial collagen.

Figures 6, 7, and 8 show how $|G|_{\text{min}}$, $|G|_{\text{max}}$, and $\eta$, obtained from low frequencies at several strain levels, vary down the aorta.

The $a_2$ computed from glossary equations 3 and 4 was always considerably higher than $a_2$ obtained from stress-relaxation curves (3). This finding is in keeping with the fact that the step function inducing the stress relaxation was not infinitely fast, but took a finite time so that some of the peak tension was lost. When that happens, some of $a_2$ is lost (1). The amount lost is greater for high $a_2$, for low $\eta$, and for forcings taking a long time $\tau$ to complete. The $a_2$ from a stress-relaxation curve should be related to the currently computed $a_2$, called here $a_{2\text{p}}$, as

$$a_{2\text{p}} - a_2 = \frac{a_{2\text{p}} \tau}{\eta} a_2.$$
A plot of mean values of \( |C|_{\text{min}} \) (so \( \pm 3.1 \times 10^6 \)) versus distance along aorta from valve. Up to tensions of 100 \( \text{g cm}^{-1} \) \( (m = 100) \), and down 10 cm from valve, \( |C|_{\text{min}} \) was not a function of tension or of distance from valve. Below that distance and above tensions of 100, \( |C|_{\text{min}} \) increased when muscle contracted and when aorta was excessively stretched. Very high mean tensions \( (m = 250) \) were associated with a marked increase in \( |C|_{\text{min}} \) down the aorta, which might reflect increasing collagen down the aorta. Asterisk indicates contracted muscle.

A plot of mean values of \( |C|_{\text{max}} \) (so \( \pm 5.7 \times 10^6 \)) versus distance along aorta from valve. Within 9 cm of the valve, it was not possible to distinguish \( (P = .05) \) significantly among tension levels. Farther down the aorta, \( |C|_{\text{max}} \) increased as long as tensions were greater than 100 \( \text{g cm}^{-1} \). Contracted muscle (asterisk) imparted a higher \( |C|_{\text{max}} \).
and from resonance at the same frequency elsewhere in the apparatus. The last factor was eliminated by obtaining resonance at \( \omega_r = 110 \text{ Hz} \) in springs used in lieu of arterial segments and at \( \omega_r = 2 \text{ Hz} \) in rubber. Errors in segment length (used to compute \( m \)) contributed to errors in \( |C| \) values at \( \omega << \omega_r \), as well as to \( |C|_{\text{max}} \) values at \( \omega = \omega_r \). Therefore this was not likely to be the source of the difference between \( (a_1 + a_2)_1 \) and \( (a_1 + a_2)_2 \). Since \( (a_1)_1 \) values were agreeably close to \( a_1 \) values from stress-relaxation curves, it seemed logical to assume, for the present, that \( (a_1 + a_2)_1 \) was better than \( (a_1 + a_2)_2 \) for computing \( a_2 \) to be considered the "real" \( a_2 \) value. It would be advisable, however, to keep in mind that frequency-dependent wall properties may be responsible for \( (a_1 + a_2)_2 > (a_1 + a_2)_1 \). Since blood ejection from the heart into the aorta may appear as a damped oscillation of 8 Hz during systole and zero frequency during diastole, the properties at resonance \( (\omega_r = 8) \) should be taken into consideration in predictions or examinations of the in vivo wall behavior.

There were obvious discrepancies also between values for \( \eta \) obtained by the three routes used here: (a) stress relaxation, already published (1, 2), (b) from glossary equation 7, and (c) from glossary equation 5. While values for \( \eta_a, \eta_b, \eta_c \) obtained by any one of the three methods did not vary greatly for any given portion of aorta \( (\sigma_0 < 2.1) \), the values obtained by the three methods did differ unacceptably from each other \( (\eta_a = \eta_b + 13.7 \pm 1.84 \text{ SD}) \), contracted muscle \( (\eta_c = \eta_a + 117 \pm 24 \text{ SD}) \). The most likely error is too low values for \( \eta_a \) obtained from stress relaxation, rather than too high values for \( \eta_b \) and \( \eta_c \) since the step-function forcing is not truly realizable. The most one can say, at this time, is that the variations in \( \eta \) with the state of the muscle have been confirmed; \( \eta \) increases markedly when the muscle contracts and is higher in regions of the aorta having a large amount of muscle. In addition, elastin has a lower \( \eta \) than muscle, while collagen seems to have no detectable \( \eta \). Detection of the peculiar muscular property of a gain angle, however, firmly establishes that contracted muscle influences aortic wall properties even at tensions in the high in vivo range (pressures of 175 mm Hg).

**Discussion**

There are no data available to compare with all of the values depicted in the figures except those already published from this laboratory (1, 2), Bergel (16), however, did publish values for dynamic moduli of aortic regions designated as thoracic and abdominal. Since his specimens were studied only several hours after the death of the animal, the muscle probably was viable. However, his lowest specified frequency was 2 Hz, which is close to the resonance frequency \( (\omega_r = 5 \text{ Hz}) \) in the present study where muscle was involved.
and also the resonance frequency ($\omega_r = 25$ radians/second = 4 Hz) published by Lawton (17, 18) for aortic strips. Bergel did not suggest that the peaks appearing at $\omega = 4$ Hz in his four published plots of $|G|_{\text{max}}/|G|_{\text{min}}$ probably indicated resonance. It is possible that resonance was not obvious since he used no frequencies less than 2 Hz. At resonance, not only are dynamic moduli considerably larger than true values, which Schwarzl and Staverman (13) specify must be computed at frequencies less than resonance levels, but the modulus appears to be spuriously frequency dependent. It is surprising, however, that both Lawton and Bergel found no appreciable phase shift at the resonant frequency. Actually, resonance is defined by a 90° phase shift (13), either at or near the frequency associated with maximal amplitude. The present data did discover this correlation. The important fact is that elastin and collagen behaved in the frequency domain very much as time domain studies predicted. The parameters $a_1$, $a_2$, and $\eta$ obtained from stress-relaxation curves are sufficiently close to the same parameters computed from dynamic moduli to confirm the validity of the model (1, 19) upon which these studies are based.

Contracted smooth muscle in the aortic wall produced a unique phase lead of strain over stress or gain angle at low frequencies, even at high circumferential strains ($\epsilon = 1.6$). Actually, the gain occurring in descent signified the prolonged stress relaxation already measured in contracted aortic muscle (1), while the gain in ascent signified recontraction of the previously stretched muscle. Each cycle, therefore, had to take place slowly enough for the recontraction to occur; otherwise the extra long stress relaxation did not occur. At higher frequencies, the muscle behaved more like a linear polymer (14), with a phase lag and an increase in $|G|$.

At the frequencies of the phase lead, the ratio of the force to displacement amplitude ($B/A$) gave a falsely low measure of the minimal absolute dynamic modulus $|G|_{\text{min}}$ lower than obtainable from a static forcellength relationship. Indeed, the $|G|$ curve in Figure 4 has a longer tail at low frequencies than does elastin or other linear polymers. In fact, this nonlinear behavior of smooth muscle makes unreliable all the measurements obtained from a conventional analysis of stress-strain loops. Not only is $a_1$ too low, but so is $\eta$, for $\omega^*$ would be too high. Yet $a_1 + a_2$, computed from $|G|_{\text{max}}$ and $b_1 + a_2$, computed from $\omega_{00}$ are probably more reliable than $a_1 + a_2$ computed from the peak of a stress-relaxation curve of muscular specimens. Muscle would certainly have a lower peak with any realizable step forcing than with an ideal step. Therefore, the several approaches used in these studies are all necessary to evaluate visco-elastic parameters of smooth muscle.

Despite the discrepancies, the present data are internally consistent in several ways. A finding that $|G|_{\text{min}} < |G|_{\text{max}}$ implies that $a_2 > 0$. In that case viscous losses are inevitable. Indeed, $|G|_{\text{min}} < |G|_{\text{max}}$ was always associated with $\omega_{00} > \omega_r$ so that the viscous losses could be computed from glossary equation 5. What is more, stimulation of the muscle simultaneously increased the difference between $|G|_{\text{min}}$ and $|G|_{\text{max}}$ and increased the difference between $\omega_{00}$ and $\omega_r$, showing that the low frequency data ($|G|_{\text{min}}$ and $|G|_{\text{max}}$) were thus far consistent with the resonance frequency data. It does not follow, however, that $|G|_{\text{min}} = G_{\text{max}}$ (which implies that $a_2 = 0$) also must be associated with $\eta = 0$. Indeed, $\eta$ did not always vanish under such circumstances. A finding of $a_2 = 0$ and $\eta = 0$ implies that the material behaves like a Voigt model. Such a material would not stress-relax; and collagen seems to be such a material. Several regions at high tensions behaved like collagen by having a lower modulus at high temperatures (11) and by failing to stress-relax, but did have $|G|_{\text{min}} = |G|_{\text{max}}$, $a_2 = 0$, but a finite, even high, $\eta$ from $\omega_{00} > \omega_r$. At still higher tensions, however, no specimens gave evidence of viscous losses, therein resembling adventitial collagen in every respect. This suggests that medial collagen may be strained at smaller diameters than adventitial and that adventitial collagen may be different from medial
in some respects, although all collagen does seem to have an exceedingly high $|G|$ which is highest at lowest temperatures tested (0°C).

One finding not detected in previous work is a high concentration of muscle in the aorta between the two renal arteries. This region (14 cm from the valve) contracted vigorously in response to phenylephrine hydrochloride, developed the high $a_2$ and $\eta$ (Fig. 4) characteristic of contracted smooth muscle, and its strain curve led its stress curve at low frequencies. Elsewhere along the aorta, the contractility and the strain necessary to give evidence of each component were in keeping with previous findings (1, 2).

One purpose of these studies (1, 2) was to use frequency and time domain measurements to determine to what extent each of the arterial wall components likely to bear tension could actually do so. The studies required the use of temperatures, extensions, and tension ranges not found in vivo, but nevertheless did suggest which components could bear tension without necessarily indicating whether they do bear tension in vivo. In particular, the studies have indicated that certain aortic diameters and regions would be more likely to be associated with the stretching of one of the components than of others. At low circumferential strains, the muscle bears the tension, if it is contracted. At higher strains, muscle contributes something to wall properties, but elastin seems to be primarily involved. At still higher strains, the effect of muscle is undetectable, and the wall behaves more like collagen than like other components. Therefore, in vivo diameter measurements would be necessary to elucidate the in vivo conditions under which a particular component could come into play. Such measurements are difficult to make and might not be necessary if an unassailable correlation can be found between visco-elastic parameters and dimensions, for then, in vivo visco-elasticity might be an indirect way of obtaining in vivo dimensions, provided a suitable algorithm for the conversion was available. Such a parameter has been developed (4-7) and seems promising, but it will depend on accurate knowledge of arterial wall properties and in vivo dimensions for reliable predictions of in vivo behavior. Whatever component happens to be responsible for a given visco-elastic behavior, however, the present studies have quantified the behavior sufficiently well to make it worthwhile to use these parameters in this or similar algorithms.

Definitions and Equations

$A =$ amplitude of sinusoidal strain imposed on specimen (cm).
$B =$ amplitude of sinusoidally varying stress developed as a result of the strain (g).
$\omega =$ frequency of the forcing and of the response (cps, radians/sec, or Hz).
$|G|$ = absolute dynamic modulus (dyne cm$^{-2}$);
$= B/A$.
$|G|_{\text{max}} =$ minimal absolute dynamic modulus found at frequencies below resonance. It is found in these specimens when $\omega = .01$ Hz (dyne cm$^{-2}$).
$|G|_{\text{max}} =$ maximal absolute dynamic modulus found at frequencies below resonance. It is found in these specimens when $\omega = 0.25$ to 1 Hz (dyne cm$^{-2}$).
$\phi =$ phase angle between force curve and stretch curve (degrees).
$G_1 =$ storage modulus (dyne cm$^{-2}$). It is a function of frequency and is minimal and maximal at the same frequencies where $|G|_{\text{min}}$ and $|G|_{\text{max}}$ occur.
$G_2 =$ loss modulus (dyne cm$^{-2}$). It is zero at frequencies where $|G|_{\text{min}}$ and $|G|_{\text{max}}$ are found if the material acts like a standard linear solid.
$G =$ $G_1 + iG_2$ where $i = \sqrt{-1}$; hence $|G|^2 =$ $G_1^2 + G_2^2$.
$\tan \phi =$ $G_2/G_1$.

Standard linear solid is a material which can be represented as a parallel combination of a Maxwell element and an elastic element. Maxwell element is represented as a spring in series with a dashpot.
Elastic element is represented as a spring. Voigt element is represented as a spring in parallel with a dashpot.
$h$ and $L =$ wall thickness and segment length.
$m =$ mean force $\cdot$ cm$^{-1}$ upon which sinusoidal forcing was superimposed (dyne cm$^{-1}$ or g cm$^{-1}$).
$a' =$ the force constant of a single elastic element in a tube wall combining many elements (g force cm$^{-1}$).

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\[ a_1 = \text{the sum of force constants of all elastic elements arranged in parallel in a material combining many elastic elements in tube form. The tube would be 1 cm thick, 1 cm long and } l_{10} \text{ cm in circumference where } l_{10} \text{ is the unstretched length of a single elastic element (g cm}^{-3} \text{ or dynes cm}^{-2}) \]

\[ a_2 = \text{the sum of all force constants of elastic elements in a material of unit dimensions describable by Maxwell elements (g cm}^{-3} \text{ or dynes cm}^{-2}) \]

\[ a_2' = \text{total obtainable } a_2 \]

\[ \eta = \text{the sum of the viscosities in all dashpots in a similar material (poise cm}^{-1}) \]

\[ l_{10} = \text{the unstretched length of longest elastic elements of force constant } a' \text{ (cm); therefore is the zero force intercept of a linear portion of static circumference-force relationship}. \]

\[ l_{20} = \text{the unstretched length of elastic elements of force constant } a'' \text{ (cm)} \]

\[ l_{1}, l_{2} = \text{the corresponding stretched lengths} \]

\[ l_{20} = l_{10} - l_{20}, l_{3} = l_{1} - l_{2} \]

Here \( l_{10} \) is taken as the unstretched length of the elastic lamellae with the largest circumference. These would be the most wrinkled when the aorta was empty and would be the last to be stretched when the aorta was expanded (cm). Then

\[ G_{\min} = a_1 l_{10} \]

\[ G_{\max} = (a_1 + a_2) l_{10} \]

\[ \sigma = \text{stress (dyne cm}^{-2} \text{)} \]

\[ \epsilon = (l_1 - l_{10})/l_{10} = \text{strain} \]

\[ \omega_r = \text{frequency where } B \text{ is maximal under a sinusoidal forcing at } \omega > 1 \text{ Hz (Hz)} \]

\[ \omega_{90} = \text{frequency where the phase difference between stress (force) and strain (stretch) is } 90^\circ \text{ (Hz)} \]

hence

\[ \eta = 2m \sqrt{(2 \pi \omega_{90})^2 - (2 \pi \omega_r)^2} \]  \hspace{1cm} (5)

\[ G_{\max} = \frac{2 \pi \omega_{90} m}{h} \]  \hspace{1cm} (6)

\[ \omega^* = \text{frequency where } C_2 \text{ is maximal (Hz)} \]

\[ \eta = \frac{a_1 a_2}{2 \pi \omega^* (a_1 + a_2)} \]  \hspace{1cm} (7)

\[ \tau = \text{time constant of a so-called step-function forcing. It would be zero for a true step function} \]

\[ (a_1)_{1}, (a_1 + a_2)_{1} = a_1 \text{ and } (a_1 + a_2) \text{ computed from data at frequencies in the viscoelastic region} \]

\[ (a_1)_{2}, (a_1 + a_2)_{2} \text{ = computations from data at frequencies in the resonance region} \]

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References


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