Influence of Flow and Pressure on Wave Propagation in the Canine Aorta

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ABSTRACT

Data on wave speed acquired from 20 anesthetized dogs showed that the thoracic aorta was essentially nondispersive for small artificially generated pressure waves traveling in the downstream or the upstream direction and having frequencies between 40 and 120 Hz. The amplitude of these waves decayed exponentially with the distance traveled. The attenuation was independent of frequency and pressure if the distance was measured in wavelengths. The logarithmic decrement of the downstream waves ranged between 0.7 and 1.0, whereas that of the retrograde waves was between 1.3 and 1.5. The discrepancy in the attenuation for the two directions appeared to be due to the taper of the thoracic aorta. Simultaneous measurements of the transmission times of waves traveling downstream and upstream indicated that small pressure perturbations were convected with a speed that was approximately equal to the mean flow velocity. The speed of such perturbations depended strongly on the aortic pressure level at which they were generated. For normal pressure pulses generated by the heart, the speed of small perturbations at systole might be 30% higher than that at diastole. Theoretical studies have shown that such changes in wave speed due to variations in pressure and flow produce marked nonlinear effects in hemodynamics.

KEY WORDS nonlinear properties of aorta phase velocity convection of pressure signals wave transmission characteristics dissipative mechanisms elastic behavior of blood vessels retrograde waves

■ Experimental studies of the dispersion and the attenuation of small artificial pressure waves (1) indicate that the aortas of anesthetized dogs should exhibit nonlinear properties with respect to large-amplitude pulse waves like those generated by the heart. By superimposing finite trains of sinusoidal pressure waves on the naturally occurring pressure waves, it has been shown that the speed of the sine waves increases markedly from diastole to systole (1). This rise in speed probably results from an increase in the elastic modulus of the wall of the aorta with pressure and from a more rapid convection of the pressure perturbations by the higher blood flow rates during systole. A closer examination of the propagation characteristics of small wave trains induced at approximately the same pressure levels during systole suggests a definite dependence of the wave speed on the variation in blood flow rate over the cardiac cycle. Measurable changes in the dispersive properties of blood vessels with pressure and flow imply that the natural pulse wave should be accompanied by nonlinear phenomena; the significance of these phenomena has probably been widely underestimated, especially in analyses of the propagation of cardiac pressure and flow pulses over large distances in the arterial tree. Evidence for this underestimation has been established in a recent theoretical investigation of flow pulses and shock waves in arteries (2, 3).

To establish a quantitative basis for studies of such nonlinear phenomena, we carried out in vivo experiments designed to separate the changes in wave speed due to flow from those due to variations in aortic pressure. Nonlinear effects might be insignificant when the propagation of pulse waves over a distance of a few vessel diameters is studied. However, in assessing the changes in the characteristic features of the cardiac pulse during propagation over large distances, nonlinearities cannot be disregarded, since their effects are cumulative. This fact must be kept in mind, especially when the

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purpose of the measurement is the diagnosis of disease patterns on the basis of relatively small changes in cardiovascular parameters.

Theoretical Considerations

On the basis of intuition and theoretical analysis (4), we expected small pressure signals to be convected whenever they were induced in an artery in which the blood was flowing. Convection is defined as the velocity imparted to a wave due to the fluid motion relative to the body. With flow and aortic pressure varying considerably and simultaneously during each cardiac cycle, the significance of their combined and individual effects on the speed of pressure perturbations might vary with the cardiac phase. Each effect should therefore be examined individually. Such an examination is possible when the relationship between the mean flow velocity and the convection speeds of small pressure signals in the downstream (positive) and the upstream (negative) directions in an arterial segment is known. To be fully successful in separating the two effects, the transmission times of signals traveling downstream between two given locations along the vessel and of those traveling upstream between the same two locations must be measured in a sufficiently simultaneous manner. By performing these measurements at various instants during the cardiac cycle, the variation in the wave speed with the aortic pressure and also with the mean flow velocity as a function of the cardiac phase can be obtained.

Theoretical studies of waves in elastic tubes containing a streaming fluid (4) have shown that the speed of convection should, for practical purposes, be equal to the mean flow velocity, $\bar{U}$, as long as $\bar{U}$ is small compared with the wave speed, c_0, measured at zero mean flow and as long as the mean flow rate in the vessel segment examined is approximately constant during the transmission time of the perturbations. Accordingly, the quasi-instantaneous wave speeds, $c^D(p)$ and $c^U(p)$, measured at any transmural pressure, p, for the downstream and the upstream directions, respectively, can be expressed in terms of $c_0(p)$ and $\bar{U}$ by

$$c^D(p) = c_0(p) + \bar{U}, \quad (1)$$

$$c^U(p) = c_0(p) - \bar{U}. \quad (2)$$

Solving these equations for $c_0(p)$ and $\bar{U}$,

$$c_0(p) = \tfrac{1}{2}[c^D(p) + c^U(p)], \quad (3)$$

$$\bar{U} = \tfrac{1}{2}[c^D(p) - c^U(p)]. \quad (4)$$

Consequently $c_0(p)$—the instantaneous wave speed at zero flow and at the pressure p—and $\bar{U}$—the instantaneous mean flow velocity—can be determined if the corresponding values of $c^D(p)$ and $c^U(p)$ are known. In view of the continuous variations in $\bar{U}$ and in the transmural or aortic pressure, p, during each cardiac cycle and considering that $c^D(p)$ and $c^U(p)$ in Eqs. 1–4 represent instantaneous speeds, waves travelling upstream and downstream between the two points of observation must be simultaneously produced, and their speeds within a time interval during which the transmural pressure and flow remain essentially constant must be recorded.

Methods

The experimental setup, instrumentation, recording system, and data analysis were basically the same as those previously reported (1). However, two electromagnetic impactors were used in the present study to generate pressure signals in the thoracic aortas of 20 anesthetized dogs as illustrated in Figure 1. Mature mongrel dogs weighing 20–40 kg were anesthetized with sodium pentobarbital (30 mg/kg, iv). They were kept in a supine position throughout the experiment and ventilated as described in the earlier publication (1). Each impactor could induce pressure perturbations in the form of finite trains of sine waves with frequencies

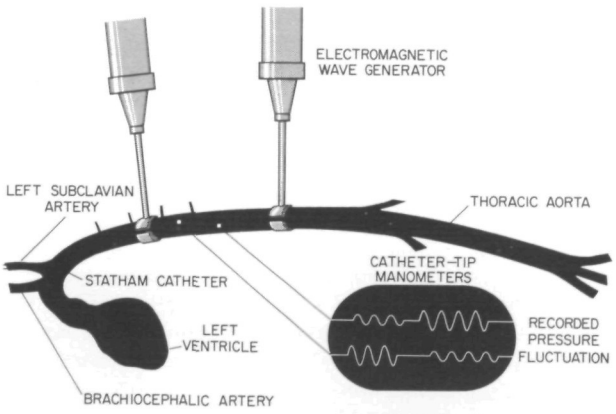

FIGURE 1

Arrangement of the experimental apparatus used in the dual-impactor experiments. Two electromagnetic impactors produced waves traveling upstream and downstream in a single segment of the aorta by controlled external indentation of the aortic wall. The number, the frequency, and the amplitude of the sine waves in each train could be selected independently for each of the wave generators. The two catheter-tip manometers were fixed together as a unit with a preselected separation. Manipulation under the fluoroscope permitted positioning of the manometers near the center of the flow stream.

between 20 and 200 Hz by indenting the aorta externally 1–3 mm in a controlled manner. By using sine waves with frequencies above 20 Hz, we can assume that the pressure and the flow conditions remain essentially unchanged over the duration of one period. Thus, the transmission characteristics of a single sine wave were a measure of the quasi-instantaneous mechanical properties of the vessel segment and the mean flow rate. To satisfy the criterion of small pressure perturbations and to preserve well-defined sinusoidal signals for an accurate determination of the transmission time between the two manometers, the amplitude of the sine waves was restricted to a range between 1 and 3 mm Hg and the frequency was limited to values between 40 and 120 Hz.

The pressure signals generated by the proximal impactor were recorded by Bytrex catheter-tip manometers as the signals traveled downstream, and the signals produced by the distal wave generator were recorded by the same transducers as they propagated in the upstream direction. The pressure manometers were separated by a fixed distance, usually 5 cm. The proximal impactor was inserted into the thoracic cavity through an opening in the left fourth intercostal space and positioned on the aorta a few centimeters below the left subclavian branch. The distal impactor was placed on the thoracic aorta a few centimeters above the diaphragm through an opening at the eight or the ninth intercostal space. Generally the distance between the wave generators was on the order of 15 cm. Fluoroscopic examination provided a check on their separation.

The two catheter-tip manometers were inserted through the femoral arteries and located fluoroscopically. Since the recordings from these manometers contained signals propagating in both upstream and downstream directions, the problem of identifying the signals traveling in each direction was eliminated by having the proximal and the distal impactors generate trains of three and four sine waves, respectively, or by having them produce sine waves of differing frequencies. Sample recordings at a frequency of 70 Hz are shown in Figure 2.

Normally, data were acquired over four to ten contiguous cardiac cycles for each frequency studied. To arrive at an independent measurement of the flow velocity in the thoracic aorta, a Pieper flowmeter (5) was used in some of the experiments immediately after the dual-impactor procedures were completed.

Maneuvers were carried out to make measurements over pressure ranges beyond the normal pulse pressure. Such pressures were achieved by mechanically clamping the aorta or by occluding it with an intra-aortic balloon catheter. The balloon-tipped catheter was inserted through the right carotid artery and advanced into the descending aorta. Inflation of the balloon for 3–5 seconds blocked the flow into the descending aorta, and the pressure in the thoracic aorta rapidly fell below the diastolic level. Pressures above the systolic level were produced in the test region by brief aortic clamping a few centimeters above the diaphragm. Data on wave speed were recorded continuously during these procedures.

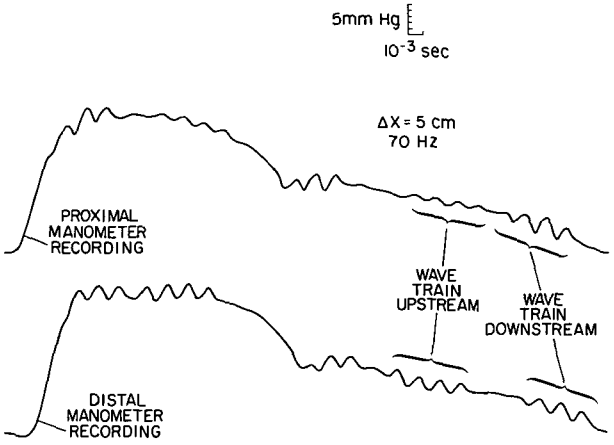

FIGURE 2

Typical recordings from the two pressure manometers between the two impactors. In this case trains of four sine waves were recorded as they propagated upstream, and trains of three sine waves were recorded as they traveled downstream. $\Delta x =$ distance between the two catheter-tip manometers.

Results

Measurements of transmission time during diastole verified that the speeds of downstream-propagating signals were slightly higher than those of upstream-traveling (retrograde) waves. Accordingly, we inferred that the convection speed and consequently the flow velocity were relatively small during diastole. Actual recordings of the flow velocity with the aid of the Pieper gauge fully corroborated this inference. As already shown for downstream waves (1), there was no noticeable dispersion of the retrograde waves when the frequency of the sine waves was varied between 40 and 120 Hz. In contrast, the attenuation of the retrograde waves was considerably stronger. However, their attenuation pattern was also characterized by an exponential decay of the amplitude; when we measured this decay on a per wavelength basis, it was independent of frequency and pressure, as in the case of downstream-traveling waves (1). Although the downstream waves had logarithmic decrements of 0.7–1.0, the values for the retrograde waves were on the order of 1.3–1.5.

Typical results of simultaneous measurements of the wave speeds in the upstream and the downstream directions as a function of the instantaneous aortic pressure are given in Figure 3. For each of the five frequencies the data shown were acquired during five to ten consecutive cardiac cycles. Singling out the data points obtained during the early phase of systole when the flow velocity and

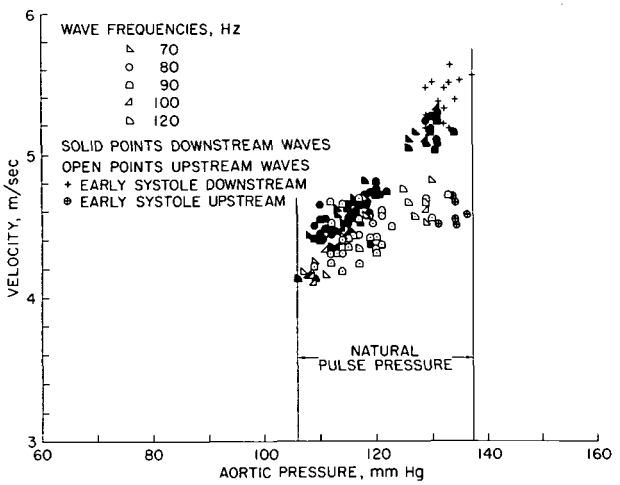

FIGURE 3

Wave speed–pressure data for upstream and downstream waves showing the increase in speed due to pressure and flow. Note the sharp increase in speed differences for signals recorded during early systole when flow reached a maximum. These data are from dog 3 but are representative of data obtained from all the other dogs. The transmission time of the waves could be measured to a fraction of a millisecond on the high-speed optical oscillograph recordings. Slight variations in the wave speeds measured for either the upstream or the downstream propagation direction at a given pressure were presumably due to slight pressure and flow fluctuations associated with the short series of contiguous cardiac cycles from which the data were obtained. $\Delta x = 5$ cm.

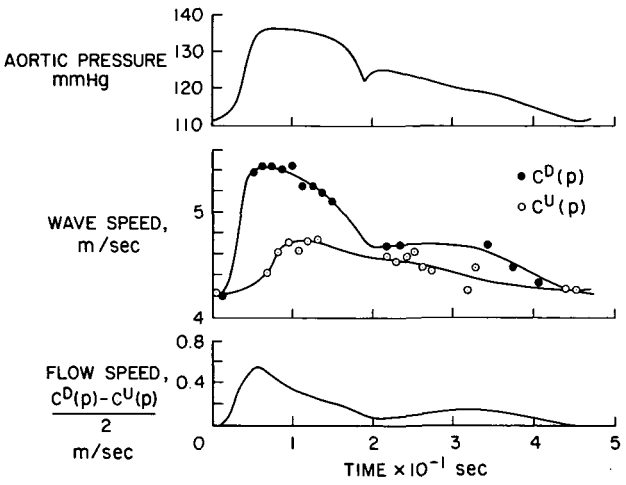

FIGURE 4

Typical flow patterns determined from measurements of wave speed in the thoracic aorta of a dog. **Top:** *Natural pulse wave defining cardiac phase at which the data shown in the lower two graphs were obtained.* **Middle:** *Upstream wave speeds and downstream wave speeds measured at different instants of the cardiac cycle. The upstream and the downstream data correspond to two heart beats a few seconds apart but with matching pressure patterns.* **Bottom:** *Calculated mean flow velocity, $\bar{U}$. $c^D(p)$ = downstream wave speed, $c^U(p)$ = upstream wave speed.*

thus the convection speed were expected to be high, we found that the difference in wave speed was approximately 1 m/sec for the two directions (Fig. 3). At elevated cardiac outputs this difference was as much as 2 m/sec. The variations in signal speed for either direction, which can be noticed in Figure 3 at any pressure level, were in part due to beat-to-beat fluctuations in the flow velocity and in part due to the fact that during each cardiac cycle a given pressure level was at times associated with different flow rates as the aortic pressure rose or fell. This latter fact is illustrated in Figure 4, which shows typical results from experiments in which trains of 10–30 sine waves with a frequency of 70 Hz or more were superimposed on the natural pulse. Determination of the transmission times of individual sine waves within these long trains showed a systematic variation in the upstream and the downstream speeds and also in the mean flow velocity, $\bar{U}$, defined by Eq. 4. Since it was very difficult to distinguish between upstream- and downstream-traveling waves when the trains were very long, we alternately induced waves in each direction during two or more consecutive pressure pulses of identical shape and determined the corresponding speeds. Recordings of the flow pulses with the Pieper catheter-tip flowmeter demonstrated that successive flow pulses during the resting phase of the respiratory cycle were sufficiently identical to justify this procedure. The flow pattern shown at the bottom of Figure 4 and deduced from Eq. 4 is quite similar to the flow pulses obtained with the Pieper gauge; however, no direct comparison was possible, since no simultaneous measurements of wave speeds and flow velocity were made because the Pieper gauge together with the two catheter-tip manometers would have effectively reduced the cross-sectional area of the aorta by approximately 30% and, accordingly, would have altered the mean flow and the wave transmission characteristics. Table 1 gives the results for $\bar{U}_{max}$ and $c_0(p)$ obtained in four dogs.

Extending the normal aortic pressure range by blocking the flow into the descending aorta or by clamping the thoracic aorta above the diaphragm allowed the wave speeds in the upstream and the downstream directions to be determined for aortic pressures from approximately 50 to 220 mm Hg. The corresponding wave speeds are plotted in

TABLE 1

Maximum Mean Flow Velocity, Wave Speed at Zero Flow, and Pressure in Four Dogs

Expt.	$\overline{U}_{max}$ (cm/sec)	c_0 (p) (m/sec)	Pressure (mm Hg)
1	100 ± 10	8.2 ± 0.4	169/133
1	120 ± 12	8.7 ± 0.4	164/133
2	50 ± 5	5.0 ± 0.3	158/131
3	50 ± 5	5.0 ± 0.3	137/106
4	80 ± 8	5.6 ± 0.3	143/118

Values are means ± SD. Results for two different trials are shown for dog 1.

Figure 5 as a function of the instantaneous aortic pressure. With the blockage of aortic flow, the convection speeds can be assumed to be rather small. Indeed, the differences in the wave speeds for the upstream and the downstream directions were insignificant under this condition. The data in Figure 5 were recorded at different times and using sine waves of three different frequencies. Sizable fluctuations in the wave speeds far beyond the measurement error (1) occurred at higher pressures for both directions. It is possible that the baroreceptor reflexes were initiated by these large pressure changes. This fact might account for the velocity variations shown in Figure 5 at chosen pressure levels, particularly those above the normal systolic values.

The values of $c_0(p)$ calculated using Eq. 3 agreed within a few percent with the values of

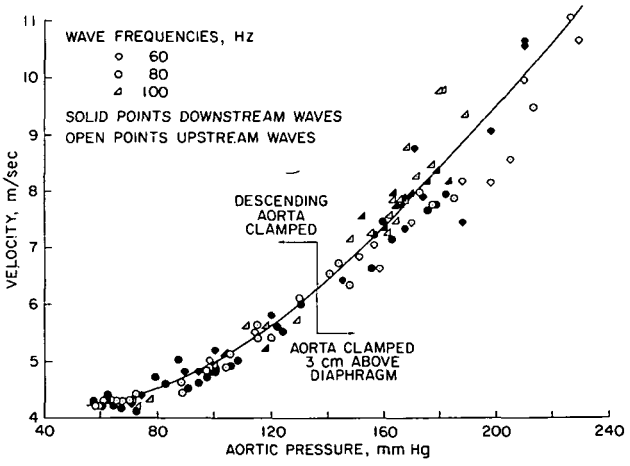

FIGURE 5

Wave speed–pressure data measured during aortic occlusion in dog 1. Pressures lower than normal were obtained by occlusion above the test segment, and elevated pressures were obtained by occlusion below the test segment. Each point represents the average of a peak and a successive valley of a sine wave. $\Delta x = 5$ cm.

$c_0(p)$ measured during aortic occlusion. Again, since the occlusion procedures occurred late in each experiment, slight changes in the smooth muscle response of the aorta might have caused minor discrepancies in the measured and the calculated values of $c_0\,(p)$.

Discussion

Data on wave speed from 20 anesthetized dogs showed that sine waves with frequencies between 40 and 120 Hz were convected with a speed approximately equal to the mean flow velocity. This finding is in substantial agreement with mathematical studies of waves propagating in elastic vessels filled with a streaming fluid. Since no simultaneous wave speed and flow velocity measurements were made, this statement cannot be given in more precise form. However, flow measurements performed with a Pieper gauge on the same dog and at the same aortic pressure levels at which the wave speeds were determined lent ample support to the conclusion as worded. Waves traveling in either direction exhibited similar exponential decay patterns for their amplitudes. If the attenuation was computed as a function of distance in wavelengths, λ, it was independent of frequency. This relationship can be expressed as

$$A = A_0 e^{-k\frac{\Delta x}{\lambda}} \qquad (5)$$

where A_0 is the amplitude at a reference point along the aorta, A is the amplitude of the wave after it has propagated the distance Δx from the reference point, and k is the logarithmic decrement. For the retrograde waves, k assumed values between 1.3 and 1.5 in different dogs. The corresponding values of k for the downstream waves normally ranged from 0.7 to 1.0 and thus were considerably smaller than those for the upstream waves. In each case the standard deviation was on the order of 0.15. The direction dependency of the attenuation appeared to be connected with the aortic taper, which causes signals propagating in the direction of decreasing diameter to be amplified and which causes waves traveling in the opposite direction to be reduced in amplitude. Therefore, the actual attenuation of sinusoidal pressure waves was characterized approximately by the mean value of the logarithmic decrements for the two directions, i.e., by a value between 1.0 and 1.25. For the frequencies considered, such k values implied that the attenuation of the waves could not be accounted for by the

viscosity of the blood but must be primarily due to the viscoelasticity of the vessel wall (6, 7).

From the variations in wave speed with aortic pressure shown, for example, in Figure 5 and the convection results illustrated in Figure 4, we concluded that, for normal aortic pressure levels and for signals like the pulse wave itself, the increases in wave speed due to pressure and those due to convection should be about equally significant as contributors to nonlinear phenomena affecting hemodynamics in long segments of arteries (2, 3). With aortic pressures fluctuating between 80 and 120 mm Hg, the wave speed, $c_0(p)$, at negligible flow will increase by about 1.2 m/sec, and the mean flow velocity should vary between 0 and 1.2 m/sec. At higher aortic pressure levels, the pressure effect on the wave speed should begin to dominate the nonlinear aspects. Combining the two effects, we arrived at wave speed changes of approximately 30% or more between diastole and systole. In cases of aortic insufficiency, the nonlinear aspects could lead to formation of shock waves which manifest themselves in terms of pistol shot sounds (2, 3).

If we adopted a mathematical model for the mechanical behavior of blood vessels which treated the elastic properties of the vessel wall as being independent of stress, we would predict on the basis of this model that the wave speed would decrease with pressure. This finding follows from the fact that the ratio of wall thickness to radius, h/a, decreases as the pressure rises. Thus, according to the Moens-Korteweg equation,

$$c^2 = (E/2\rho)(h/a), \qquad (6)$$

where ρ is the density of blood and E is the effective elastic modulus, the wave speed, c, would also decrease. The progressive increase in wave speed with aortic pressure must therefore be attributed to a massive change in the effective elastic modulus, E, of the aortic wall with stress. Hence, for Eq. 6 to yield higher speeds with pressure, E must be strongly stress dependent (8, 9).

References

1. Anliker, M., Histand, M.B., and Ogden, E.: Dispersion and attenuation of small artificial pressure waves in the canine aorta. Circ Res 23:539–551, 1968.
2. Anliker, M., Rockwell, R.L., and Ogden, E.: Nonlinear analysis of flow pulses and shock waves in arteries: I. Derivation and properties of mathematical model. Z Angew Math Phys 22:217–246, 1971.
3. Anliker, M., Rockwell, R.L., and Ogden, E.: Nonlinear analysis of flow pulses and shock waves in arteries: II. Parametric study related to clinical problems. Z Angew Math Phys 22:563–581, 1971.
4. Morgan, G.W., and Ferrante, W.R.: Wave propagation in elastic tubes filled with streaming liquid. J Acoust Soc Am 27:715–725, 1955.
5. Pieper, H., and Paul, L.: Catheter-tip gauge for measuring blood flow velocity and vessel diameter in dogs. J Appl Physiol 24:259–261, 1968.
6. Jones, E., Anliker, M., and Chang, I.D.: Effects of viscosity and constraints on the dispersion and dissipation of waves in large blood vessels: I. Theoretical analysis. Biophys J 11:1085–1120, 1971.
7. Jones, E., Anliker, M., and Chang, I.D.: Effects of viscosity and constraints on the dispersion and dissipation of waves in large blood vessels: II. Comparison of analysis with experiments. Biophys J 11:1121–1134, 1971.
8. Patel, D.J., Janicki, J.S., and Carew, T.E.: Static anisotropic elastic properties of the aorta in living dogs. Circ Res 25:765–779, 1969.
9. Bergel, D.H.: Properties of blood vessels. *In* Biomechanics: Its Foundations and Objectives, edited by Y. C. Fung, N. Perrone, and M. Anliker. Englewood Cliffs, New Jersey, Prentice-Hall, Inc., 1972, pp 105–139.