Determination of the True Phase Velocity of Arterial Pressure Waves in Vivo

By Robert H. Cox

ABSTRACT
Results of simultaneously recorded pressure, diameter, flow, and differential pressure from an arterial segment were studied by Fourier analysis. The method was applied to pressure propagation in the femoral artery of anesthetized dogs. Experimental values of phase velocity compared favorably with values predicted from a theoretical wave propagation model. The phase velocity was generally constant for frequencies over 35 rad/sec. At frequencies below 20 rad/sec, it decreased rapidly with decreasing frequency. The frequency variations of the true and apparent phase velocities were significantly different. The characteristic impedance of the femoral artery was a weak function of frequency and nearly independent of the mean arterial pressure. High frequency values of the local fluid impedance above 50 rad/sec were significantly lower than those of the characteristic impedance. It was concluded that the method can be used to obtain reliable values of phase velocity in the physiological frequency range from in vivo measurements.

KEY WORDS linearized wave propagation models pulse wave velocity arterial viscoelasticity apparent wave velocity wave reflection Fourier analysis characteristic impedance anesthetized dogs

The velocity at which pressure waves propagate in the arterial system has been of considerable interest since the work of Thomas Young (1) because it reflects the physical properties of both the blood and the blood vessel. In fact, it has been used as a measure of the wall elasticity in the arterial system (2). Three principal methods have been used to measure this velocity in arteries in vivo: one is based on the propagation of natural pressure waves, another is based on the propagation of artificially induced transients superimposed upon the natural pressure wave, and the last is based on the propagation of trains of superimposed sinusoidal pressure waves.

A number of investigators have attempted to measure the velocity at which certain features of the natural pressure wave propagate through the arterial system, e.g., the dicrotic notch, the foot and peak of the pressure pulse (for reviews see refs. 3-5). Studies based on these methods have not met with a great deal of success because of resolution problems and an inherent subjectivity (4, 6). The reflections caused by geometric and elastic nonuniformity and by branching, as well as the effects of harmonic dispersion and selective damping (7), contribute to the poor reproducibility of these methods.

Landowne (8) developed a method to study the propagation of pressure waves in intact human arteries using pressure transients generated by externally induced impacts. This method, however, has been criticized by Anliker as not being unique because of the uncontrolled nature of the pressure transient and the possible nonlinear effects that could result from large amplitude pressure disturbances (9). This criticism is supported by...
Landowne's own data showing different transmission times for the onset of the impulse and the peak of the impulse (8).

Anliker et al. (9) developed a method for measuring the transmission characteristics of arteries from the propagation of small sinusoidal pressure oscillations superimposed upon the natural pressure wave. This method uses short trains of sine waves generated by an electronically driven impactor. This method is restricted, however, to frequencies that are much higher than those of physiological interest, i.e., over 25 Hz. Their results show that in the range of frequencies considered (40-200 Hz) there is little or no dispersion (frequency dependence) in the phase velocity in the canine descending aorta. This finding indicates that in this range of frequencies the viscoelastic properties of the wall are independent of frequency. They found, in addition, that the aorta exhibited strong attenuation characteristics that they attributed primarily to dissipative mechanisms in the wall. On the basis of the coupled nature of the phase velocity and attenuation characteristics of wave propagation in viscoelastic materials, these two statements are somewhat conflicting (10).

As a result of the limitations of these methods, it was the principal objective of the work described here to develop a method for measuring the propagation velocity of pressure waves in vivo, using the physiological pressure pulse. A method was developed to derive values of true phase velocity from the apparent phase velocity of pressure waves using a basic equation developed by Wiener et al. (11) and the predictions of a theoretical model developed by me (10). This method was applied to the study of the propagation of pressure waves in the canine femoral artery. With this method, the frequency dependence of the phase velocity for physiological frequencies was investigated to gain some insight into the magnitude of the viscoelastic properties of the wall.

There was an additional objective in this study related to the validity of linearized wave propagation models. It has been shown by a number of authors, beginning with Witzig (12), that such models predict that the effect of blood viscosity is to lower the magnitude of the phase velocity of pressure waves at low frequencies from a high frequency asymptotic value. This viscosity effect has not been demonstrated in vivo. In fact, the apparent phase velocity measured in vivo at low frequencies shows very large increases (13). The demonstration of this effect would add strong credibility to the validity of the application of linearized wave propagation models to arterial blood flow (14).

Methods

ANALYTICAL

The similarity in the mathematical formulation describing pressure propagation in arteries and the propagation of electromagnetic waves in transmission lines has led several investigators to apply the techniques and terminology of the latter to the description of arterial blood flow (11, 15-17). For example, Taylor (15) has used measurements of input impedance \(Z_x\) and propagation constant \(\gamma\) from the propagation of sinusoidal pressure waves in long rubber tubes to compute values of longitudinal fluid impedance \(Z_f\) and transverse wall admittance \(Y_{lc}\). Noordergraaf and his co-workers (18) have used transmission line methods to develop an analog representation of the human arterial tree.

A number of theoretical physical models of pressure propagation and blood flow in the arterial system have been developed (10, 19, 20). These models are based on linearized wave propagation theory and consider the propagation of harmonic pressure waves through a Newtonian fluid contained within a tube. The various models differ principally in the equations used to represent the motion of the tube wall and predict values of longitudinal fluid impedance and propagation constant from values of the physical properties of the tube and fluid (e.g., fluid viscosity and density, wall density and geometry, etc.). Using the methods of transmission line theory it is possible to compute two additional
theoretical quantities, the transverse wall admittance and the characteristic impedance (16). The wall admittance can be related to the theoretical quantities \( Z_t \) and \( \gamma \) by the equation, \( Y_w = \gamma^2 / Z_t \) (15). Similarly, the characteristic impedance can be given by \( Z_0 = Z_f / \gamma \). These four quantities described above \((Z_t, \gamma, Y_w, \text{and} \ Z_0)\) are based on the theoretical models and can be called the theoretical values of these hemodynamic variables.

To obtain experimental values of the transmission line variables from an arterial segment, simultaneous measurements of intra-arterial pressure, flow, differential pressure, and external wall displacement must be made (14). The various transmission line variables can be defined in terms of these experimental variables.

**SYMBOLS**

- \( a \) = Internal wall radius
- \( b \) = External wall radius
- \( c \) = Pressure phase velocity
- \( D \) = Mean arterial diameter
- \( D_n \) = Nth harmonic of Fourier analysis of diameter
- \( E^* \) = Complex viscoelastic modulus of arterial wall
- \( E \) = Effective elastic modulus of the artery
- \( L \) = Distance between pressure measurements for differential pressure
- \( P \) = Intra-arterial pressure
- \( P_\text{m} \) = Mean value of intra-arterial pressure
- \( P_n \) = Nth harmonic of intra-arterial pressure
- \( \Delta P \) = Intra-arterial differential pressure
- \( Q \) = Arterial blood flow rate
- \( Q_\text{m} \) = Mean flow rate
- \( R \) = Effective viscous modulus of artery wall
- \( R_p \) = Poiseuille resistance of the artery segment
- \( Y_w \) = Transverse wall admittance
- \( Z \) = Mean value of local fluid impedance, i.e., resistance
- \( Z_f \) = Longitudinal fluid impedance
- \( Z_0 \) = Characteristic impedance of artery
- \( Z_a \) = Local fluid impedance
- \( \alpha \) = Attenuation coefficient
- \( \gamma \) = True propagation constant
- \( \gamma^* \) = Apparent propagation constant
- \( \delta \) = Effective radius-wall thickness ratio = \( \frac{2\alpha^2}{b^2 - a^2} \)
- \( \epsilon \) = Radial wall strain, external diameter divided by mean diameter
- \( \omega \) = Angular frequency

The longitudinal fluid impedance is computed from the ratio of the drop of pressure \((\Delta P)\) per unit length of blood vessel \((L)\) to the blood flow rate \((Q)\) as (14)

\[
Z_f = \frac{1}{L} \frac{\Delta P}{Q}
\]

The transverse wall admittance represents the distensible nature of the arterial wall as well as the energy losses associated with viscous properties. The wall admittance has been determined analytically by Horeman and Noordergraaf (21) and is given by

\[
Y_w = j\omega \frac{3\pi a b^2}{E^*(b^2 - a^2)}
\]

where \( \omega \) is the angular frequency, \( E^* \) the incremental viscoelastic modulus of the wall, and \( a \) and \( b \) the internal and external wall radii, respectively. The incremental viscoelastic wall modulus is computed from the Fourier series analysis of the intra-arterial pressure and external diameter from the equation (14)

\[
E^* = \frac{4a^2b}{b^2 - a^2} P_n
\]

where \( P_n \) is the \( n \)th harmonic of the pressure and \( D_n \) is the \( n \)th harmonic of the external diameter.

Experimental values of the characteristic impedance of vascular segments are computed from experimental values of longitudinal fluid impedance and transverse wall admittance by \( Z_0^2 = Z_f / Y_w \).

I call the ratio of the intra-arterial pressure to the blood flow rate at any point in the vascular system the local fluid impedance, \( Z_a \) (my terminology). The latter is equal to \( Z_a = P/Q \). In the ascending aorta, the local impedance has come to be called the systemic input impedance (22).

When the pressure difference (or differential pressure) along a short segment of artery is used to determine the propagation constant, the effects of reflected wave components cannot be eliminated (13). What is measured, then, is the apparent propagation constant \((\gamma^*)\). This is computed from intra-arterial pressure \((P)\) and differential pressure \((\Delta P)\)
according to the relation (14)

$$\gamma_0 = \frac{1}{L} \frac{\Delta P}{\Delta P},$$

(4)

where $j$ is the imaginary number, $\sqrt{-1}$.

Experimental values of the true propagation constant are computed from the relation

$$\gamma^2 = Z_0 Y_w.$$  

An alternate relationship between the apparent and true propagation constant was developed by Wiener et al. (11) and can be written as

$$\gamma = \gamma_0 Z_0.$$  

(5)

Equation 5 demonstrates the relation of $\gamma$ and $\gamma_0$ to the impedances $Z_0$ and $Z_0$.

In this analysis, the independent variables (intra-arterial pressure, etc.) are represented in the form of a finite series of harmonically related components (Fourier series). The consequences and justification of this particular representation have been discussed at length in the literature (14, 22, 23). The independent variables can be represented by the general relation for traveling waves as

$$g(r, x, t) = \sum_{n=0}^{N} G_n(r) \exp[j(n\omega t - \gamma_n x)],$$  

(6)

where $g(r, x, t)$ is any independent variable, a function of $r$ (radial coordinate), $x$ (axial coordinate) and $t$ (time); $G_n(r)$ is that part of the variable that is a function of $r$ only; $n$ is the nth harmonic of the representation; and $N$ is the total number of harmonics. It should be noted that the value of $g(r, x, t)$ for $n = 0$ is the average (mean) value of the variable. The term $\exp[j(n\omega t)]$ describes the sinusoidal nature of each harmonic and the term $\exp[j(-\gamma_n x)]$ describes the dependence of the variable on axial distance, $x$. (The identity of the real and imaginary parts of the propagation constants given in Eq. 6 and 7 should be noted. They may be somewhat different than normally defined.)

The propagation constant is composed of two components, one describing the velocity with which the variable propagates, and the other describing the variation of the amplitude of the variable with the distance $x$. This relation can be represented by (14)

$$\gamma = \frac{\omega}{c} - j\alpha$$  

(7)

where $\alpha$ is the attenuation constant and $c$ is the phase velocity.

From instantaneous measurements of intra-arterial pressure, differential pressure, flow, and external diameter, experimental values of the hemodynamic variables ($Z_0$, $\gamma$, $Y_w$, and $Z_0$) can be computed for comparison to theoretical values.

**EXPERIMENTAL**

A series of experiments was performed on the exposed femoral artery of 20 healthy, adult mongrel dogs anesthetized with a mixture of pentobarbital sodium (15 mg/kg) and Dial-urethane (25 mg/kg diallybarbituric acid and 100 mg/kg urethane). A detailed description of the experimental methods employed in this study has been given elsewhere (14). Briefly, intra-arterial pressure and differential pressure were measured with a Statham P23H differential manometer, which was coupled to the vessel by short polyethylene catheters inserted through muscular side branches of the femoral artery to measure lateral pressure only. Catheters of the same size and length were used in each experiment to standardize the frequency response characteristics of the catheter-manometer system. The pressure transducer was calibrated statically using a precision barometer (Hass Instrument Co.) with a resolution estimated to be ±0.1 mm Hg.

The differential pressure output of the transducer had a static imbalance of 0.8% of the applied pressure. This could introduce an error in the base line of the differential pressure measurement. To eliminate this error, the base line was recorded with both transducers exposed to the mean intra-arterial pressure, by occluding the artery below the distal transducer and above the proximal transducer simultaneously. This served as a mechanical zero for the flow measurement as well. The frequency response characteristics of the transducer were determined by the Hansen method (24) and used to correct the data.
by Fry’s method (25). The frequency responses of the individual pressure outputs were flat within ±2% up to 25 Hz with a phase shift of about 2° at 20 Hz (linear up to that point). The frequency response of the differential output was flat within ±5% up to 20 Hz with a phase shift of 5° at 25 Hz.

Blood flow was measured with a Statham M4000 gated sine wave electromagnetic flowmeter with noncannulating type Q probes. The flowmeter frequency response was measured directly and had an amplitude ratio flat within ±5% to 10 Hz with a phase shift of about 5°/Hz at low frequencies. The flowmeter was calibrated in situ by cannulating the artery distal to the flow probe with stiff polyethylene tubing, whose outflow end was elevated above the probe to ensure continuous electrical contact between the probe and the vessel. The flow rate was varied over a wide range by graded constriction of the outflow tubing. The calibrations were linear and indicated a resolution of about 5 ml/min.

The instantaneous external diameter of the femoral artery was measured with a cantilever type of strain-gauge transducer developed in my laboratory (26). The amplitude ratio of the transducer was flat within ±5% up to 60 Hz with a phase shift of about 0.1°/Hz at low frequencies. The transducer was calibrated using premeasured rods of stainless steel drill stock.

The average value of the inner and outer radii of the arterial wall at the measurement site was obtained by the technique developed by Attinger (27). At the end of the experiment, a segment of artery was clamped between two hemostats rigidly coupled 4 cm apart. In this manner, the vessel segment was maintained at its nominal in vivo length as well as at mean intra-arterial pressure. The segment was fixed in a solution of 10% formaldehyde in physiological saline. The error introduced by this solution in the radii measurements has been documented (27), and measured data were corrected accordingly. The solution reduced the external diameter by about 6% without affecting the internal diameter.

The experimental hemodynamic variables were recorded simultaneously on magnetic tape along with periodic base line checks and calibrations. Data were recorded during control periods as well as during graded, unilateral (right) vagal stimulation, which was performed to obtain a wider range of mean intra-arterial blood pressure and heart rate for a more thorough comparison of experimental and theoretical data. Selected cardiac cycles were subjected to analog-to-digital conversion and subsequent Fourier analysis. The four experimentally recorded variables were then used to calculate values of the hemodynamic variables defined in the previous section.

Using experimental values of the viscoelastic wall modulus and other system parameters (e.g., frequency, wall radii, fluid viscosity, etc.) and the equations of the hemodynamic model (10), theoretical values of the hemodynamic variables were computed on an IBM 360/75 digital computer. All data analysis and computational procedures were performed on this computer.

Results

Figure 1 shows an example of a recording of experimental variables from the femoral artery
of a dog during control conditions. Both the proximal and distal pressures are shown in the figure. Table 1 contains a summary of the average control values of a number of hemodynamic quantities from the 20 animals. Included in the table are values of heart rate, mean intra-arterial pressure, mean external diameter, mean flow rate, mean local fluid resistance or d-c impedance, Poiseuille resistance, effective radius-wall thickness ratio, effective elastic modulus of the wall, and effective viscous modulus of the wall. These last two quantities were computed by the method of Peterson et al. (28) from the equation

$$P(t) = \delta(E\epsilon(t) + R\epsilon\epsilon(t)/dt),$$

where $P(t)$ and $\epsilon(t)$ are the pressure and strain time functions and $d/dt$ is the time derivative. This equation was programmed for solution on a digital computer using least square methods. The average values and standard errors of each variable from all the experiments are given at the bottom of the table.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
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<tr>
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<td>137 ± 0.012</td>
</tr>
<tr>
<td>D (cm)</td>
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</tr>
<tr>
<td>Q (ml/min)</td>
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</tr>
<tr>
<td>Z (10^8 dynes/cm^2)</td>
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</tr>
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</tr>
</tbody>
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**TABLE 1**

Average Values of Hemodynamic Variables for the Canine Femoral Artery

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pressure and heart rate were lowered by a progressive increase in frequency of vagal stimulation with a sine wave having a peak-to-peak voltage of 25 v. Sine wave stimulation was used because it produced less noise pickup in the flow recording than the square-wave form. The standard errors for the abscissa values were too small to be included on the graph.

Figure 4 shows a summary of control values of the amplitude and phase of the local impedance and the characteristic impedance as a function of frequency. The variation of local impedance with angular frequency is shown in Figure 5. The various symbols identify data obtained during different levels of vagal stimulation. Also shown in the upper part of the graph are examples of the pressure

The radius-wall thickness ratio included in Table 1 is given by $2a^2/b^2 - a^2$. This particular function is used because it is the thick-walled cylinder equivalent of the radius-wall thickness ratio which is uniquely defined in a thin-walled tube. This above quantity is used because it is the geometrical constant relating the internal pressure variations to the motion of the external wall in an isotropic, thick cylinder under plane stress (14, 28).

Figure 2 shows a graph of the true and apparent phase velocities as a function of angular frequency. Figure 3 shows the dependence of the first nine harmonics of the true phase velocity on angular frequency at four levels of mean intra-arterial pressure. Blood
Frequency variation of true phase velocity at six different levels of mean pressure. The value of the pressure in mm Hg is given in each graph. The points are the mean and the bars the standard error. The curve shows the theoretical values from the hemodynamic model (10).

\( P_{fa} \) and flow \( Q_{fa} \) in the femoral artery at each level of mean arterial pressure.

A comparison of experimental and theoretical values of the true phase velocity as a function of angular frequency for six levels of intra-arterial pressure is shown in Figure 6. Figure 7 shows a comparison of theoretical and experimental values of characteristic impedance as a function of angular frequency for three levels of intra-arterial blood pressure.

**Discussion**

Almost all of the early analytical work on hemodynamics was concerned with developing equations relating the velocity of propagation of pressure waves in fluid-filled tubes and the physical properties of the tube and fluid (29, 30). These studies in the late nineteenth and early twentieth centuries eventually led to the development of equations relating the velocity of propagation of pressure waves and various different measures of the “elasticity” of the arterial wall. The in vivo application of these equations is complicated by the reflections that occur within the arterial system. Reflections due to the spatial nonuniformity of mechanical and geometrical properties of the wall are spatially continuous in nature, while reflections due to the nonperiodic nature of arterial branching are spatially discrete. As a result of the latter factors, the values of apparent phase velocity measured in vivo are considerably different from the values that

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*Figure 6*

*Figure 7*
would exist if arteries were homogeneous and very long (true phase velocity).

In an attempt to circumvent the problems implicit in using the natural pressure pulse to measure pressure propagation characteristics, a number of investigators have used artificially generated pressure pulses. These artificial methods greatly simplify the determination of pressure phase velocity in vivo in the arterial system. They are, however, not without disadvantages. The problems associated with propagation of impulse transients such as those used by Landowne (2, 8) and by Remington (31) have been discussed previously (9). Although the method of Anliker et al. (9) is more quantitative than the impulse methods and represents perturbations in the true sense, it yields information only at rather high frequencies. The high frequency values do yield data on the high frequency asymptote of the phase velocity but do not supply any information in the frequency range where energy is propagated in the arterial system.

The method that has been developed here can be used to obtain values of phase velocity in the physiological frequency range, because it uses Fourier analysis data of the naturally occurring variables. The application of this method, however, involves a number of implicit assumptions. First, it assumes that the use of Fourier analysis in this application is valid. This subject has been considered in detail by Attinger et al. (23); the method appears to be applicable, at least in the peripheral arteries.

Second, there is the assumption of linearity in the wall stress-strain relationships. It is generally accepted that for large deformations, nonlinear stress-strain relations exist (32). Whether normal cardiac cycle variations in arterial pressure and wall displacement can be considered perturbations in the analytical sense is not known explicitly. Experimental evidence has been presented that both supports (33) and refutes (9) this assumption. It appears that the validity of this assumption improves peripherally along the arterial tree (10). The question of the applicability of the linearized wave propagation model to the analysis of femoral artery hemodynamics is related to this assumption. I previously investigated this question and concluded that such models are applicable to peripheral arteries such as the femoral (14).

Gessner and Bergel (34) developed methods to compute values of pressure phase velocity using the natural arterial variables that are similar to the one developed here. For intact arteries, they developed methods involving the measurement of either two pressures and one flow or three pressures. They applied these techniques to data recorded from the femoral artery of an anesthetized dog and concluded that their data were "too noisy" to describe any frequency dependence (due to the fluid viscosity effects) in the phase velocity. Their analysis emphasized the degree of resolution required to make valid comparisons of experimental data and theoretical predictions from in vivo measurements.

TRUE AND APPARENT PHASE VELOCITIES

The comparison of true and apparent phase velocities shown in Figure 2 for control conditions shows some interesting characteristics. First, the true phase velocity (TPV) shows very little frequency variation (dispersion) above an angular frequency of about 35 rad/sec. This indicates the relative constancy of the viscoelastic properties of the wall in this range of frequencies. The apparent phase velocity (APV), on the other hand, shows a strong frequency dependence over the entire range considered. At both high and low frequencies, APV is greater than TPV, the differences being statistically significant. In the middle frequency range, APV is less than TPV, the difference again being statistically significant. A number of authors have used the high frequency values of APV as a measure of TPV (33, 35, 36). In view of the complex frequency variations of APV, this type of averaging could lead to erroneous results. Averaging the values of APV and TPV in Figure 2 for the fourth to ninth harmonics yields values that are nearly identical on the average (TPV = 1161 cm/sec and APV = 1185 cm/sec). However, the ratio of APV to TPV calculated in this manner from the data of the
20 individual experiments varied from 0.37 to 3.7 with about half being above and half below unity.

Although the data obtained during control conditions showed very little dispersion, this was not the case for data obtained during right vagal stimulation, some of which are shown in Figure 3. At low frequencies, TPV is a very strong function of frequency. This dependence is due to the effects of fluid viscosity on the wall and has been theoretically predicted to occur (10). In addition to the frequency variation, there is also a significant mean arterial pressure dependence. This is also shown in Figure 3, where TPV at a given frequency increases with mean arterial pressure. This effect is due to the dependence of the wall mechanical properties on mean arterial pressure (32).

The development of a method to derive values of TPV from measurements of APV is of little real value unless the results are unique and theoretically predictable. Theoretical values of phase velocity were calculated as previously mentioned for comparison to the experimental values. A comparison of these data is shown in Figure 6 from one experiment at six different levels of mean intrarterial pressure. The agreement between theoretical and experimental values of TPV is reasonably good at all levels of blood pressure. There is a slight tendency for the low frequency, low pressure experimental values to be somewhat lower than the theoretical ones. However, considering the number and complexity of the calculations necessary to obtain the experimental data, the agreement is quite good indeed.

**FLUID IMPEDANCES**

Most of our current knowledge concerning the relationship between the local fluid impedance ($Z_L$) and the characteristic impedance ($Z_0$) has been derived from analytical work on transmission line models both uniform and nonuniform (17, 37). In such models, the local or input impedance oscillates around the characteristic impedance (17, 38). A number of authors have extrapolated these analytical results to the arterial system. In doing this they generally assume that the high frequency values of input impedance can be averaged to yield values of frequency-independent characteristic impedance (39–41). In fact, the frequency independence of the characteristic impedance of arteries has not been shown experimentally. From the theoretical point of view, the existence of viscous losses in the blood and blood vessel, and the presence of frequency-dependent wall viscoelasticity (42) and longitudinal fluid impedance (10) would lead one to expect the characteristic impedance to have some sort of frequency variation.

The comparison of the frequency variation of the local and characteristic impedances for the femoral artery under control conditions is shown in Figure 4. The characteristic impedance is a weak function of frequency, decreasing only slightly in the range of frequencies involved in the graph. The phase angle of the latter is relatively independent of frequency at about $-20^\circ$. The frequency dependence of the characteristic impedance can be exaggerated during vagal stimulation and can be seen in Figure 7 to be a low-frequency phenomenon. The comparison of experimental and theoretical values of characteristic impedance shown in Figure 7 is again reasonably good, both in amplitude and phase.

The relationship between the local and characteristic impedances is of interest also because of its relation to the true and apparent propagation constants and true and apparent phase velocities as given by Eq. 5. If these two impedances are identical, then the true and apparent phase velocities will be equal, which implies that no reflected waves exist in the artery. Such a situation could also occur at specific frequencies or locations because of cancellation of reflected waves.

The data on the frequency variations of the local impedance at different levels of mean arterial pressure show some interesting results. The amplitude of the local impedance falls rapidly from its value at zero frequency (resistance of the bed) to a minimum near 75 rad/sec and thereafter increases slightly. From
about 10 rad/sec to higher frequencies, the values of amplitude and phase of the local fluid impedance are superimposable for the different levels of mean arterial pressure obtained in this experiment. Similar results were obtained in the other experiments with graded vagal stimulations. These observations are rather surprising and would seem to indicate that within this range of pressure the characteristic impedance of the femoral artery segment is a very weak function of mean arterial pressure. This is indeed the case as seen from the data in Figure 7. These results also indicate from Eq. 5 that the relation between true and apparent propagation constants does not change and, by inference, that the local reflection coefficient (ratio of forward to backward waves) does not change. As previously stated, the characteristic impedance is given theoretically by the ratio of the longitudinal fluid impedance to the propagation constant. As the mean arterial pressure falls during vagal stimulation, the vessel radius decreases and the viscoelastic modulus falls. These changes cause both the longitudinal fluid impedance and the propagation constant to increase in such a fashion that their ratio is nearly constant. As a result, the characteristic impedance is also nearly constant and independent of mean arterial pressure over a limited range. It would be anticipated that the reduction in mean pressure that occurs during vagal stimulation will elicit responses from the aortic and carotid mechanoreceptors causing increased sympathetic outflow to the heart and peripheral vasculature. Any such effect could be manifested by changes in the characteristic impedance of the femoral artery as well as by increased heart rate and peripheral resistance and by vagal escape, in general (43). These alterations elicited through the mechanoreceptors and the sympathetic nervous system are associated with rather long time constants on the order of 10–20 seconds (44). With this fact in mind, only data recorded during the early part of the response, where heart period and systolic and diastolic blood pressures were relatively constant, were analyzed. In fact, for Fourier series analysis to be applicable it is necessary that these two conditions exist (23). Also, only relatively small changes in blood pressure were purposely induced in order to ensure, subjectively, only moderate changes in sympathetic outflow. In several experiments, when intense vagal stimulation was performed, and blood pressure fell to shock levels, the local fluid impedance values were not superimposable on similar control data. This seems to indicate that the direct effects of sympathetic nervous system activity can alter the frequency variation of these hemodynamic variables.

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References


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