Pressure Transmission in Pulmonary Arteries
Related to Frequency and Geometry

By Ernst O. Attinger, M.D.

The difficulties in analyzing pulsatile pressure and flow in the circulatory system can hardly be overestimated. Although the so-called Navier-Stokes equation expresses the general relation between pressure gradient and the resulting motion of a viscous Newtonian liquid, its exact solution depends upon the choice of boundary conditions. In pulsatile flow fluid motion varies from point to point and from moment to moment and must therefore be described by a time-dependent three-dimensional vector field. The present state of knowledge in the physical as well as biological sciences is inadequate for an exact solution since we are dealing with a non-Newtonian fluid, in a visco-elastic system of branched, tapered tubes with multiple changes in geometry.

One of the characteristics of the behaviour of the vascular system is the dispersion of the pulse wave during its travel. A number of attempts have been made to analyze this feature with the help of analogues, an approach introduced into hemodynamics by B. H. Weber. The progress of the engineering sciences led to a rapid extension of these methods, with models ranging from rubber tubes to transmission lines and wave guides. Based on a number of simplifying assumptions, Womersley has developed a coherent physical theory of pulsatile flow and his analysis has been applied by McDonald, Taylor, Hardung, and others to the evaluation of experimental results obtained from models and from studies of the peripheral circulation.

Since little is known about the pulsatile behaviour of the pulmonary circulation, this study of the transmission of the pressure pulse in the pulmonary artery of the living dog was undertaken. We will first present a short outline of the basic theory and then examine the experimental results against this background.

THEORY

The theoretical analysis is based on a simplified model of a uniform vascular segment, the behaviour of which can be described by linear differential equations. The justification of basing such a model on a number of assumptions and approximations lies in the tremendous simplification of the analytical treatment. Once the basic usefulness of a model has been shown, it can always be modified and improved as new experimental data becomes available.

The behaviour of the vascular system is determined by its physical parameters: mass, elasticity and viscosity. Neglecting secondary flows, the fluid moves both in radial and axial directions. The response of such a system to a driving force, such as that generated by the heart, will be different at various frequencies, and this frequency dependence is determined not only by its individual components but also by the way in which they are interconnected. The response of a linear system to any driving force can be predicted once its frequency behaviour is known. To analyze the response of the system at various frequencies, one can lump its parameters at one point, which means that the response to an excitation is at every instant identical throughout the system, i.e., one uses simple differential equations to describe the system.

The Windkessel theory is a classical example of this approach. If the error introduced by this approximation is not tolerable, one has...
to describe the model in terms of partial differential equations, where one thinks of the various parameters as distributed throughout the system. As an example, if we take a pulmonary artery of 10 cm length, a heart rate of 120/min and a pulse wave velocity of 200 cm/sec, we observe a phase difference between the two end points of 36° for the fundamental frequency and 180°, or half a cycle length for the fifth harmonic. Since such phase differences are far from negligible for our purposes, we have to resort to an analysis using distributed parameters, i.e., all the variables are a function of distance as well as of time.

The complicated form of pressure and flow pulse are not amenable to an easy analytical approach. However, any periodic function which has at most a finite number of discontinuities, minima and maxima (Dirichlet conditions), can be represented as a linear superposition of sine waves, which are generally more manageable in a mathematical treatment. Such a Fourier analysis is a perfectly valid method of representing a single pulse wave. However, the calculations based on such an analysis require that the system be linear. The vasculature is certainly non-linear in most of its properties; however, the error introduced by assuming linearity is probably less than the measuring error in such experiments.\textsuperscript{5,8,9} The simple harmonic components of the pulse wave are propagated at a definite velocity and one has to consider the relation between the shortest essential wave length and the dimensions of the system in deciding upon the method of analytical approach. If the shortest wave length of the disturbance is very large compared with the maximal dimension of the system acted upon, the maximal phase difference in the component wave between any two points must be very small, and the wave can be considered to have the same instantaneous value at all points in the system, i.e., it can be regarded as lumped at a point.

In order to get a clearer picture of wave mechanics, consider a water wave of simple harmonic shape and travelling at a constant velocity. A cork floating on the wave and restrained from moving horizontally will be observed to bob up and down with simple harmonic motion. It is apparent that the velocity of propagation of the harmonic wave, the wave length (i.e., the distance from crest to crest) and the frequency with which the cork bobs up and down are not independent. In fact, the frequency with which the cork bobs is equal to the ratio of wave velocity over wave length. Hence, the wave, which is propagated at a definite velocity, is not only a simple harmonic function of distance, but at a fixed point represents a simple harmonic function of time as well. Such a behaviour can be described mathematically by an equation of the form:\textsuperscript{6}

\[ \nabla^2 \vec{P} = \frac{1}{c^2} \frac{\partial^2 \vec{P}}{\partial t^2} = 0 \]  

Equation 1 is a three-dimensional wave equation, where \( \vec{P} \) represents the pressure vector and \( \nabla \) the wave velocity. The operator \( \nabla^2 \) stands for the second partial derivatives with respect to the coordinates of the quantity following it. Separating the pressure vector into its three components in the direction of the three spatial coordinates, equation 1 can be expanded into three scalar equations.

Since we are dealing with a cylindrical vessel, we choose the components \( P_r \), \( P_\theta \) and \( P_z \) of the pressure vector in the direction of radius, circumference and longitudinal axis of the vessel. If we now assume that the pressure is uniform throughout any given cross section of the vessel, the radial component \( P_r \) of the vector \( \vec{P} \) is a function only of the axial coordinate \( z \) and the time, \( t \). This assumption, which is implicit in all present methods for measuring blood pressure, is not strictly true, but is a good approximation for our purpose, since the radial dimension of the vessel is less than 2.5% of the wave length at the highest frequencies of interest. Under these condi-

\textsuperscript{5}For details of the derivation the reader is referred to any textbook of electromagnetic field theory.\textsuperscript{11-13}

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The solution of the wave equation becomes:
\[ P(z, t) = f_1(z - vt) + f_2(z + vt) \] (2)
where \( P(z, t) \) is the amplitude of the pressure wave and \( f_1 \) and \( f_2 \) are arbitrary functions.

A function such as \( f_1(z - vt) \) represents a disturbance travelling along the \( z \)-axis with a velocity \( v \), while \( f_2(z + vt) \) corresponds to a wave propagating in the negative \( z \)– direction. The mathematical form of the functions \( f_1 \) and \( f_2 \) depends upon the type of disturbance creating the wave. For sine waves, they will be sine or cosine functions of \( z \pm vt \).

In the presence of friction losses we obtain a damping term in the wave equation:
\[ \frac{\partial^2 P}{\partial t^2} - \frac{1}{\rho} \left( \frac{\partial P}{\partial t} + \begin{pmatrix} \alpha & \beta \end{pmatrix} \begin{pmatrix} P_{\text{d}} \\ P_{\text{u}} \end{pmatrix} \right) \] (3)

For a Newtonian fluid and laminar flow in a linear system the damping term is directly proportional to the viscosity of the conducting medium. The friction losses result in an exponential decay of the wave as it propagates away from the source. The solution for a sinusoidal disturbance \( P_0 \sin \omega t \) in the direction of the positive \( z \)-axis, becomes then
\[ P(z, t) = P_0 e^{-\alpha z} \sin(\omega t - \beta z) \] (4)

This is more conveniently written in exponential form:
\[ P(z, t) = P_0 e^{-\gamma t} \] (5)
where \( \gamma = \alpha + j\beta \) is the propagation-constant
\( \alpha \) = damping constant
\( \beta \) = phase constant

For the case of the superposition of a downstream and an upstream travelling wave, the pressure at any point is given by adding the solutions for each wave. This results in an expression of the form:
\[ P(z, t) = P_0 e^{-\gamma t} - \gamma + P_0 e^{\gamma t} + \gamma \] (6)
where \( P_0 e^{-\gamma t} \) and \( P_0 e^{\gamma t} \) represent the disturbances creating the downstream and upstream travelling wave respectively.

The propagation constant \( \gamma \) is, of course, a function of the physical properties of the conducting medium and is always a complex number. Its real part, \( \alpha \), the damping constant, describes the amplitude variation with distance and its imaginary part, \( \beta \), the phase constant, indicates the way in which the phase of the wave changes, i.e., the velocity at which the transient disturbance is propagated.

The velocity at which a simple harmonic disturbance travels is the phase velocity, mathematically described by:
\[ V_p = \frac{\Delta \Theta}{\Delta t} = \frac{\Delta \omega}{2\pi} \] (7)
where \( V_p \) = phase velocity
\( \Delta t \) = distance over which the phase velocity is measured
\( \Delta \Theta \) = difference in phase angle between the two measurements
\( \lambda \) = wave length
\( f \) = frequency

The phase constant \( \beta \) is inversely proportional to the wave length, and is experimentally evaluated by measuring the phase difference of the wave over a given distance. The damping constant \( \alpha \) is roughly proportional to frequency.14

In a simple elastic system, the phase velocity is related to the physical properties of the system by the Moens-Korteweg equation:
\[ V_p = \frac{E_d}{2p} \] (8)
where \( E \) = modules of elasticity of the vessel wall
\( \rho \) = mass of blood per unit volume
\( R \) = vessel radius
\( d \) = thickness of vessel wall

Theoretically the determination of phase velocity offers an easy experimental approach to the evaluation of the elastic properties of a blood vessel in vivo. As Hardung14 has shown, the values calculated for phase velocity, in rubber tubes and arteries fit, in general, quite well with those predicted from theory, provided the stress and strain distribution is considered in evaluating the modulus of elasticity.

In contrast to the phase velocity, the group velocity expresses the speed at which the compound wave and hence the energy travels. Skillings15 gives the following analogy for the two velocities: the ripples along the back of a caterpillar travel at the phase velocity, whereas the bulk of the beast moves at the group velocity. In general, the group velo-
Superposition of incident and reflected waves proximal to a branch point of a vessel.

Figure 1 shows a diagram of a blood vessel ending in two branches. A steady state sinusoidal pressure generator is assumed at the vessel entrance. The pressure wave drawn with full dots at the bottom of the figure travels for two wave lengths along the vessel to the right and is partly transmitted and partly reflected at the branch point. The dotted line represents the transmitted, the starred line the reflected wave. The line characterized by open circles is the actual pressure wave upstream of the branching point, i.e., the measured pressure, which is the vector sum of the incident and reflected wave. The amount and the phase of reflection are determined by the relation between characteristic and load impedances. Since frictional losses occur, the amplitude of the incident wave decreases along the vessel toward the branch point and the amplitude of the reflected wave decreases toward the origin of the vessel. Because of the frictional losses, no true standing waves can be produced.

In figure 2 the same waves are shown again in form of a polar plot over the wave length just prior to the branch point. Each wave is represented as a vector, i.e., by amplitude and angle. Distance is expressed in terms of angles, one wave length corresponds to 360°.
Pressure Transmission in Pulmonary Arteries

Variation of Pressure Vector Along a Uniform Vessel One Wave Length Long

Figure 2
Polar plot of incident, reflected and resultant wave over one wave length prior to a branch point. Note the ellipsoidal form of the resultant wave and its non-uniform rotation rate as indicated by the cross bars at each eighth of a wave length. 

$P^i =$ pressure vector of incident wave at entrance of vessel

$P^r =$ pressure vector of reflected wave at branch point

$P^o =$ pressure vector of resultant wave at vessel entrance

$P_l =$ pressure vector of resultant wave at branch point

or a full rotation of the pressure vector. The vector $A$ representing the downstream travelling pressure wave starts on the real positive axis (zero phase angle) with a magnitude $P^i$. It rotates uniformly clockwise over 360° during one wave length, decaying exponentially during this travel because of friction-losses. The rate of decay and rotation are determined by the attenuation and phase constant, and at the end of one rotation ($z = l$) its magnitude is $P^i e^{-al}$. The vector $B$ represents the part of the incident wave which is reflected at the branch point at $z = l$. In this diagram its magnitude $P^r$ is 2/3 of the incident wave and has angle 50°. During its travel from the branch point upstream $B$ also decays exponentially and rotates uniformly clockwise. After travelling one wave length its magnitude is $P^r e^{-al}$. Since the reflection coefficient

Figure 3
Changes in amplitude (top), phase (center) and phase velocity (bottom) of the incident and actual compound wave along a uniform blood vessel at varying distances from a reflection site. Without reflection the phase velocity is independent of distance $P_l$. In the presence of reflections the apparent phase velocity oscillates around the true phase velocity. The numbered circles refer to the relations expected between apparent and true phase velocity in the pulmonary vasculature on the basis of a simple model (see text).

$C =$ different from unity and also has an angle, the incident and reflected wave differ both in amplitude and phase. The net pressure (Vector $C$) at any point in the vessel is the vector sum of the incident and reflected wave at that point and its locus describes an elliptic spiral, which varies both in magnitude and in phase as a function of distance. At $z = 0$ its value is $P_o = P^i + P^r e^{-al}$, at the branch point its magnitude is $P_l = P^i e^{-al} + P^r$. Note that although the incident and the reflected vector rotate uniformly, the resultant vector does...
not. This is indicated by the difference in arc length between the cross bars drawn at each eighth of a wave length. Hence its phase change over a wave length is not equal to 360°. This means, of course, that the wave length of the resulting wave is not exactly the same as the wave length of the incident wave. (This same fact is also apparent from figure 1.) As the phase angle increases along the vessel, the two waves will be alternatively in phase and in phase opposition, and thus the magnitude of pressure will ripple as a function of distance. Since at any point the pressure is a sine function of time, the magnitude of the wave goes through maxima and minima, a quarter-wave length apart. Under these conditions, the group velocity also varies with frequency and bears no longer a simple relation to the actual movement of energy.

The relations between amplitude, phase and phase velocity of the incident and the actual wave composed of incident and reflected wave along a uniform vessel are summarized in figure 3. At a given moment the amplitude of the pressure wave decays exponentially and its phase linearly along the vessel. As a result, the phase velocity is constant throughout the course of the vessel. In the presence of a reflected wave the behaviour is characterized by equation 5. At low frequencies, where the length of the bed is considerably greater than a quarter-wave length, the measured phase velocity will be in excess of the true phase velocity. With increasing frequency the observed phase velocity will approach the true phase velocity as an asymptote.

The phase velocity is not only an interesting parameter per se, but enters explicitly into the relations between pressure gradient and oscillatory motion of the blood, as well as those between vessel-expansion and flow within. A better knowledge of its behaviour is essential for the understanding of the behaviour of pulsatile flow. The physiological significance of pulsatile flow is often underestimated, although a recent review summarizes the evidence for its importance for capillary circulation, kidney function and cellular metabolism.15

EXPERIMENTAL

Methods

The experiments were carried out in ten mongrel dogs, weighing between 15 and 20 kg. The animals were anesthetized with Nembutal, intubated and artificially ventilated through a Harvard respiration pump. After thoracotomy, two or three catheters, made from Nylon tubing* (20 cm long, 2.5 mm O. D., 0.4 mm wall thickness), were introduced through small branches of the upper and lower lobe arteries, advanced to the desired measuring points in the main, right or left pulmonary artery, and connected to Statham strain gauges P23D. The results obtained with this system were compared in two experiments with those observed simultaneously from two catheter tip manometers (Dallons catheters), placed at the same measuring points as the Nylon catheters.

Measurements were made with the lungs either

*Polypropylene Nylon tubing, DuPont Form 101, Mfg. by Polymer Corp., Reading, Pennsylvania.
†Manufactured by Dallons Laboratories, El Segundo, California.
influenced by the transpulmonary pressure of 25 cm H2O or deflated (transpulmonary pressure 5 cm H2O). If necessary, succinylcholine was used to eliminate any respiratory movement during the recording periods, which lasted from 20 to 30 seconds. Transpulmonary pressure was maintained at a fixed level by applying the desired pressure from an oxygen tank through an overflow valve to the airways. The valve consisted of a glass tube, connected to a suction of the oxygen line and submersed either 5 or 25 cm below the water level in a suction bottle.

In five animals the chest was closed and sealed after obtaining the open chest measurements. A Nylon tube, connected to a Statham P23D gauge, was left in the pleural cavity through which intrapleural pressure was monitored. Care was taken to expand the lungs fully before sealing the chest wall in order to minimize the effects of pneumothorax. In these cases the overflow valve was adjusted for the value of intrapleural pressure in order to obtain the desired transpulmonary pressure during the recording periods.

In several experiments, lobar arteries were clamped as close to the origin as possible for 10 to 20 seconds. In four experiments additional catheters were introduced through a small lobar vein branch and wedged into the venous side of the capillary system. Through these catheters an estimate of the pressure on the venous side of the capillary system was obtained. This is because only a small outflow segment is obstructed by the catheter, so that the latter transmits the dynamic pressure at the next upstream branch point for an alternate flow path. Weibel has shown that such pathways exist on the venous side through the peribronchial plexus venosus. Although these experiments lasted several hours, no infarction was observed in the cannulated segments. Figure 3 in Lloyd's paper indicates that appropriate drainage exists under these experimental conditions.

The distances between catheter openings were measured at autopsy by threading a marked catheter through the various vessels at a transpulmonary pressure of 15 cm H2O. Since the vascular bed is somewhat longer in the inflated lungs, our results overestimate the phase velocity somewhat for the deflated lungs. This error is probably less than 5% because the change in vessel length is proportional to the cube root of the change in lung volume.

All pressures were recorded simultaneously together with an ECG, intrapulmonary or transpulmonary and peripheral arterial pressure on an 8-channel Sonborn oscillograph at a paper speed of 100 mm/sec. The overall frequency response of the sensing-recording system was flat up to 30 cycle/sec and without phase shift between individual channels, as evaluated by dynamic calibration with a sine pump similar to the one described by Taylor. Since the distances involved and hence phase angles and transit times are much smaller than in the peripheral vasculature, these frequency requirements are quite critical. Static calibration was carried out with a water manometer. The obtained tracings were manually sampled at intervals of 0.01 second and the digitalized data subjected to Fourier analysis, using first an IBM 650 and later an IBM 1620 digital computer.

The Fourier representation of the pressure pulse can be written:

$$p(t) = a_0 + \sum_{n=1}^{\infty} (a_n \cos nt + b_n \sin nt) \quad (9)$$

which can be rearranged to:

$$p(t) = a_0 + \sum_{n=1}^{\infty} \left( a_n \cos (nt - \Theta_n) + b_n \sin (nt - \Theta_n) \right) \quad (10)$$

where:

$$a_n = \frac{1}{\pi} \int_0^{\pi} p(t) \cos nt \, dt$$

$$b_n = \frac{1}{\pi} \int_0^{\pi} p(t) \sin nt \, dt$$

$$\Theta_n = \tan^{-1} \left( \frac{b_n}{a_n} \right)$$

In addition to the sine and cosine coefficients the computer printed out the magnitude Cn and the phase angle \(\Theta_n\). Initially this was done for the first 30 harmonics, establishing the frequency behaviour between 0 and 30 cycle/sec, since the latter value represents the frequency of the tenth harmonic for a heart rate of 150. However, it was found that the harmonics above the sixth were within the noise level of the measuring system and the analysis was consequently reduced to the first six harmonics.

Even the amplitudes of the fifth and sixth harmonics were often below 2 cm H2O. Since we were only able to measure pressures accurately to 0.25 cm H2O with our system, the error associated with the calculation of phase angles and phase velocities for the fifth and sixth harmonics was between 10% and 20%. To correct this, the measurement error for the first four harmonics did not exceed 5%. The revised computer program contained also the calculation of phase angle differences, phase-velocity and attenuation according to equations 6 and 7. At least three consecutive pressure pulses were analyzed for each recording period. Two or three sets of measure-
FOURIER ANALYSIS OF A PRESSURE CURVE IN THE PULMONARY ARTERY

Actual tracing of a pressure pulse in the pulmonary artery and its reconstruction from five harmonics by Fourier analysis.

Results

Figure 4 shows the fit between an actual tracing and its reconstruction from the first five harmonics. An analysis of the figure reveals that 97% of the harmonic content is associated with the first two harmonics and more than 90% with the first five harmonics. The mean square error of the fit was less than 2% for the first four harmonics and decreased only 2/10% when two additional harmonics were added. In other words, the first four harmonics of the Fourier series represent an excellent approximation of the pressure pulse and no significant improvement in the approximation can be expected by the addition of a few more terms in the Fourier expansion.

This most fortunate circumstance could have been predicted from the low amplitudes of the higher harmonics. In table 1 are listed the results obtained from the analysis of 30 pressure pulses in one animal. In this experiment three Nylon and two Dallons catheters were placed into the pulmonary arterial bed. One of the Nypons and one of the Dallons catheters were introduced through a right upper lobe artery into the main pulmonary artery, the other Dallons and a second Nylon catheter through a lower lobe artery into the right pulmonary artery 7.3 cm apart from the first measuring point. The third Nylon catheter was placed through a middle lobe artery into the right pulmonary artery at a distance of 5.6 cm from the proximal and 1.7 cm from the distal measuring point. Except for the second harmonic, the results in columns 3a and 3b are in excellent agreement. The better frequency response of the Dallons catheters as tested by a sine pump was, in our experience, offset by baseline drifts and clotting problems, which did not exist in the other measuring system. The table shows that the apparent phase velocity varied as a function of frequency and lung volume and was significantly larger in the periphery (column 2) as compared to the more proximal part of the pulmonary artery (column 1), both in the inflated and deflated lung. The relatively large standard error in column 2 for all
PressurE tranSMISSION IN PULMONARY ArTERIES

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PHASE VELOCITY IN PULMONARY ARTERY (M551)

Figure 5

Apparent phase velocity in the pulmonary artery of a rabbit as a function of frequency. Measurements from right pulmonary artery at the proximal part of the artery, 5.3 cm apart. The apparent phase velocity is plotted as a function of frequency, both for closed and open chest and inflated and deflated lungs. In all four conditions there is a marked frequency dependence of the apparent phase velocity, particularly at low frequencies. At higher frequencies the values appear to approach an asymptotic value around 200 cm/sec. In general, the apparent phase velocity was larger for the deflated lung as compared to the inflated lung. Figure 5 represents the data obtained in another animal, with the catheters located in the proximal part of the right pulmonary artery. Measurements from the other columns are due to the limitations of the measuring systems with respect to sensitivity and measuring distance as discussed under "Methods."

Figure 6 shows the change in apparent phase velocity for the right pulmonary artery over a distance of 5 cm. The apparent phase velocity is plotted as a function of frequency for closed and open chest preparations, both for inflated and deflated lungs. In all four conditions there is a marked frequency dependence of the apparent phase velocity, particularly at low frequencies. At higher frequencies the values appear to approach an asymptotic value around 200 cm/sec. In general, the apparent phase velocity was larger for the deflated lung as compared to the inflated lung.

TABLE 1

Reproducibility of Measurements-Phase Velocity (cm/sec) in Rt. Pulmonary Artery at Three Sites in One Dog (M552)

<table>
<thead>
<tr>
<th>Measuring site</th>
<th>a</th>
<th>b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distance between measuring points (cm)</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>Frequency cycle/sec</td>
<td>Inflated</td>
<td>Deflated</td>
</tr>
<tr>
<td>2.5</td>
<td>171 ± 10.1</td>
<td>142 ± 5.7</td>
</tr>
<tr>
<td>5.0</td>
<td>242 ± 11.5</td>
<td>181 ± 18.8</td>
</tr>
<tr>
<td>7.5</td>
<td>404 ± 8.6</td>
<td>324 ± 28.7</td>
</tr>
<tr>
<td>10.0</td>
<td>226 ± 9.2</td>
<td>156 ± 13.3</td>
</tr>
<tr>
<td>12.5</td>
<td>216 ± 4.9</td>
<td>156 ± 15.3</td>
</tr>
</tbody>
</table>

*Proven measurements for site 1, 2, 3a were done by Statham strain gauges. Site 3b corresponds to 3a, but two Dallin catheter-tip manometers were used. All measurements were made simultaneously at the different sites. The individual values in each column represent mean values obtained from data of three different arterial cycles.
Change in apparent phase velocity with distance. Each group of 6 bars represents the phase velocities of the first 6 harmonics measured over the distance indicated on the bottom diagram.

Phase velocity with distance in the deflated lung of the same animal. In this instance, the second catheter was withdrawn in steps to the points marked on the diagram of the right pulmonary artery at the bottom of the figure. Each set of six bars represents the apparent phase velocity for the first six harmonics over the indicated distance. The widest variations in phase velocity occurred over the distances involving the major branching points, A, C and D. For the inflated lung, the results were similar, except that the apparent phase velocity was lower and the variation over the measuring distance C had shifted to B. These marked oscillations of apparent phase velocity with frequency can be reduced by proper clamping of major branches. Figure 7 depicts the results from one such experiment. With all the major branches of the left pulmonary artery except two lower lobe arteries clamped, the phase velocity varied only between 320 and 500 cm/sec as a function of frequency over the range studied. Unclamping of the various branches did not significantly alter the phase velocity of the first two harmonics, but introduced a peak velocity of 1050 cm/sec for the fourth harmonic.

The overall results obtained in all ten animals are presented in table 2. The apparent phase velocity for the fundamental frequency is in the order of 260 cm/sec for the inflated and 325 cm/sec for the deflated lung. It varies as a function of frequency with peak velocities at the frequencies of the third and fifth harmonics.

The pulsations of the pressure pulse are transmitted across the capillary bed. Figure 8 shows the amplitude of the various harmonics of the pressure pulse in the pulmonary...
artery of the right middle lobe and from a catheter placed in a pulmonary venous wedge position in the same lobe. The first two harmonics are perfectly well transmitted into the venous side of the capillary bed, particularly in the deflated lung. For the inflated lung the amplitude is lower than in the deflated lung. This difference is most apparent for the pulmonary venous wedge pressure in the closed chest. Since the distance between the two measuring sites was not measurable in this instance, the apparent phase velocity could not be calculated. However, from the data obtained, one can determine the attenuation or inverse gain of the pressure pulse as a function of frequency. The results for the experiment of figure 8 are represented in figure 9, where amplitude and angle of the transfer function are plotted as a function of frequency in the complex attenuation plane. The coordinates of such a locus plot represent the real and the imaginary part of the transfer-function respectively. The frequencies investigated in this particular experiment range from zero to 12 cycle/sec, the heart rate being 2 cycle/sec (fig. 8). Connecting the attenuation for the various frequencies in numerical order by a smooth curve results in a logarithmic spiral. If the pulse were transmitted without attenuation such a plot would represent a unit circle. For the nonpulsatile

![Graph](image)

**Figure 7** Change in apparent phase velocity with frequency with major branches clamped and unclamped.

**Figure 8** Change in amplitude of pressure pulse with frequency. Note the transmission of the first two harmonics into the venous side of the capillary bed, particularly with the lungs deflated.

**Table 2**

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Inflated</th>
<th>Deflated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>355 ± 44</td>
<td>504 ± 76</td>
</tr>
<tr>
<td>2</td>
<td>292 ± 44</td>
<td>350 ± 78</td>
</tr>
<tr>
<td>3</td>
<td>445 ± 126</td>
<td>426 ± 121</td>
</tr>
<tr>
<td>4</td>
<td>259 ± 107</td>
<td>321 ± 88</td>
</tr>
<tr>
<td>5</td>
<td>411 ± 201</td>
<td>468 ± 209</td>
</tr>
<tr>
<td>6</td>
<td>265 ± 94</td>
<td>204 ± 44</td>
</tr>
</tbody>
</table>
Lotus plot of attenuation across the pulmonary capillaries. Note the marked attenuation particularly at the higher harmonics. The coordinate axes represent the real and imaginary axis of the attenuation function $\frac{1}{G}$. The frequencies range from zero to 12 cycle/sec counter-clockwise (see text).

Within the unit circle. For comparison, a locus plot for data obtained simultaneously in the thoracic aorta over a distance of 12 cm is shown at the bottom of the figure. In this case there is a significant attenuation of all harmonic components except the first. The exact shape of such a plot depends not only on the vessel parameters but also on the amount of reflections present, as Ronninger18 pointed out.

The geometry of the larger branches of the pulmonary arterial tree is shown in figures 11 and 12, and the data obtained from 12 vascular casts are summarized in tables 3 to 5. Note that the main pulmonary artery and its two major branches form essentially three adjoining edges of a cube. The left pulmonary artery slopes caudally in a gentle curve while the right continues on a straight course for approximately 4 cm, over which the upper lobe artery arises. It curves then suddenly caudally with an angle of close to 45°. Since we were impressed by the ellipsoidal shape of the casts of the pulmonary artery, we measured the longest diameter and, at a right angle to it, a second (minor) diameter at the points marked on figure 12. The ratio of these diameters was never unity, not even between branching points within the investigated region of the vascular tree. The cross section is therefore not circular and hence we used the approximation formula for the area of an ellipse ($ab$) for the calculation of the cross section, where $a$ and $b$ are the major and minor semiaxes of the ellipse. This, and the higher perfusion pressures employed by Patel et al.,19 may account for the fact that our values are considerably lower than those presented.
### TABLE 3
Cross Section and Diameter Ratios of 12 Canine Pulmonary Arteries at Measuring Sites Listed in Figure 11 (mean and SE)

<table>
<thead>
<tr>
<th>Site</th>
<th>Area mm²</th>
<th>Diameter ratio L/R</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>110.0 ± 11.9</td>
<td>1.24 ± 0.068</td>
</tr>
<tr>
<td>1</td>
<td>45.1 ± 5.4</td>
<td>41.1 ± 4.8</td>
</tr>
<tr>
<td>2</td>
<td>49.9 ± 6.8</td>
<td>52.2 ± 5.6</td>
</tr>
<tr>
<td>3</td>
<td>42.3 ± 8.2</td>
<td>40.5 ± 4.9</td>
</tr>
<tr>
<td>4</td>
<td>46.1 ± 5.7</td>
<td>39.6 ± 6.9</td>
</tr>
<tr>
<td>5</td>
<td>31.0 ± 2.5</td>
<td>30.3 ± 2.8</td>
</tr>
<tr>
<td>6</td>
<td>25.0 ± 2.6</td>
<td>26.6 ± 2.2</td>
</tr>
<tr>
<td>7</td>
<td>14.0 ± 1.3</td>
<td>17.7 ± 2.1</td>
</tr>
<tr>
<td>8</td>
<td>15.4 ± 1.5</td>
<td>15.4 ± 0.7</td>
</tr>
<tr>
<td>9</td>
<td>10.3 ± 0.6</td>
<td>10.5 ± 0.9</td>
</tr>
<tr>
<td>10</td>
<td>8.7 ± 0.7</td>
<td>10.9 ± 1.0</td>
</tr>
<tr>
<td>11</td>
<td>20.4 ± 1.9</td>
<td>19.2 ± 1.7</td>
</tr>
</tbody>
</table>

### TABLE 4
Cross Section Ratios at Branch Points and Lengths of Various Pulmonary Artery Branches (mean and SE)

<table>
<thead>
<tr>
<th>Branch point</th>
<th>Distance-cm</th>
<th>Cross section ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3</td>
<td>3.4 ± 0.1</td>
<td>1.78</td>
</tr>
<tr>
<td>4-8</td>
<td>4.6 ± 0.2</td>
<td>4.3 ± 0.2</td>
</tr>
<tr>
<td>5-9</td>
<td>6.6 ± 0.3</td>
<td>6.1 ± 0.2</td>
</tr>
<tr>
<td>7-10</td>
<td>8.9 ± 0.5</td>
<td>2.21</td>
</tr>
<tr>
<td>11-12</td>
<td>8.5 ± 0.3</td>
<td>7.0 ± 0.3</td>
</tr>
<tr>
<td>UL art.</td>
<td>14.1 ± 0.7</td>
<td>12.8 ± 0.5</td>
</tr>
<tr>
<td>ML art.</td>
<td>15.4 ± 0.5</td>
<td>14.7 ± 0.6</td>
</tr>
<tr>
<td>CL art.</td>
<td>16.5 ± 0.6</td>
<td>16.4 ± 0.5</td>
</tr>
<tr>
<td>LL art.</td>
<td>18.3 ± 0.5</td>
<td>18.3 ± 0.5</td>
</tr>
<tr>
<td>LLI art.</td>
<td>18.7 ± 0.7</td>
<td>19.0 ± 0.7</td>
</tr>
</tbody>
</table>

*Branch points are designated as in figure 11. Distances are measured from pulmonary artery valves either to the various branch points (first five rows) or to the end of the longest lobar branch in each of the 12 cases. UL, ML, CL, LL refer to upper, middle, cardiac and lower lobes, respectively. 11 and 2r stand for left and right pulmonary artery, respectively.*

Reported by these investigators, although the cross section ratios of the sum of the two daughter vessels to the mother vessel are similar for the first branch point, which was the only one studied by them. Calculation of the cross section on the basis of a circular area, the radius of which was obtained by measuring the circumference, showed an average ratio of 0.82 between elliptic to circular cross section, the largest difference being in the right main pulmonary artery (0.72). The spatial orientation of the major and minor axis varied from segment to segment and is probably influenced by the shape of the adjacent structures. Except for the right pulmonary artery no significant difference in cross section...
or diameter ratio due to body position or lung volumes was observed in our limited sample (table 5). Because of changes in flow and transmural pressure such differences may exist in the intact animal under dynamic conditions. Although the diameter ratio of the right pulmonary artery was significantly larger in the supine as compared to the prone position, the calculated cross section appeared to be independent of the body position. The marked flattening of the right pulmonary artery in the supine position is apparently due to the weight of the mediastinal organs. The ratio of the combined cross section of right and left pulmonary artery to the cross section of the main pulmonary artery was less than unity, a finding already reported by Patel. The cross section ratios of the other major branch points (table 4) were rather close to unity and significantly less than the ratio of 1.26 postulated by Fleisch. The volume of the arterial tree was obtained from the cast weight and the specific weight of the cast material. The average volume was 24.3 ± 1.8 ml, which is in excellent agreement with the values reported by Fei et al. in the living dog.

Discussion

We can now attempt to compare the experimental results with those predicted from theory. The superposition of incident and reflected waves results in a compound wave, with nodes and antinodes at intervals of a quarter wave length for each frequency. Our results show that the apparent phase velocity varies both with frequency and distance, as the theory predicts (figs. 5 and 6). However, the agreement between theory and experiment is only qualitative, as the following argument shows. From the average heart rate of 120 per minute and an average true phase velocity of 250 mm/sec, as estimated from the extrapolation of the results in table 2 to an asymptotic value, one obtains a wave length of 125 cm for the fundamental and of 21 cm for the
The arterial bed with an average length of 16 cm (table 4) represents, therefore, 1/8 of a wave length for the fundamental and 3/4 of a wave length for the sixth harmonic. Under these conditions the apparent phase velocity in the investigated segments of the pulmonary vasculature should be higher than the true phase velocity for the first three harmonics and lower for the fourth, fifth and sixth harmonics, as indicated by the numbered circles in figure 3. This is clearly not the case for our experimental results.

On the other hand, one can attempt to explain the dispersion of the pressure pulse by different propagation velocities and selective damping of the various harmonics, associated with mass and friction in the central part of the arterial bed. However, with this assumption the correspondence between theory and experimental results is even worse. In contrast to the pulmonary capillary bed, where all harmonies are markedly attenuated (figs. 8 and 9), there is no damping of the pressure pulse in the pulmonary artery (fig. 10). As a matter of fact, all the harmonics increase in amplitude over the distance measured.

The frequency dependence of phase velocity and input impedance has been demonstrated by a number of investigators for the peripheral and central arteries. However, with the assumption of a model of a simple viscoelastic tube and the frequency dependence of phase velocity and input impedance, it is difficult to arrive at a new explanation for the results obtained. It is, however, not always realized that the similarity between a model of a simple viscoelastic tube and the arterial bed can be misleading. Therefore, a number of investigators have also shown that the frequency dependence of phase velocity and input impedance of the arterial bed is a matter of principle. However, with the assumption of a model of a simple viscoelastic tube and the frequency dependence of phase velocity and input impedance, it is difficult to arrive at a new explanation for the results obtained. It is, however, not always realized that the similarity between a model of a simple viscoelastic tube and the arterial bed can be misleading.

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living vascular system with its multiple branch points may be quite remote. Any of the models proposed describes only a short and uniform vascular segment and cannot account for the complex geometry of the vascular bed (fig. 11). As pointed out in the section on theory, the input impedance of each such segment represents also the load of the previous segment and a mismatch at a junction gives rise to reflections. Since these segments are quite short, reflected waves must arise in very close succession and superimpose themselves upon the downstream travelling wave. Only if the number of reflection sites or the reflection coefficients are small will a separation of the various components be possible. It is apparent that under these conditions the outlined theory is not refined enough to allow a quantitative theoretical evaluation of experimental results in animals. It is, nevertheless, useful for the understanding of wave propagation and reflection, as well as for a qualitative interpretation of our results. The existence of multiple reflection sites does not invalidate the concepts of apparent and true phase-velocity, as applied to pressure pulses in the circulation, although it makes their interrelation much more complex.

Some of the recent experimental results in the peripheral circulation have been interpreted as indicating that the terminations of the large vessels supplying pelvis and thigh are the dominant reflection sites in relation to the aorta.2,3,7,21 Womersley2 calculated the amount of reflection to be 14% at the bifurcation of the aorta and about 7% at the multiple branching of the upper aorta. Taylor4 and Ronniger15 estimated the magnitude of reflection at the termination of the femoral vascular bed at approximately 50% and concluded that the total reflection from the vascular bed is much greater than that from the sites of branching of major arteries. Our experimental design was based on these calculations. It is well known that if the intravascular pressure exceeds the pulmonary capillary pressure, the capillaries collapse. Hence, inflating the lungs with a positive pressure higher than the mean pulmonary artery pressure will transform the pulmonary arterial tree to a state resembling a closed pipe while during deflation of the lungs the system should exhibit a more open-ended type of reflection. Although there was a marked difference with respect to transmission of pulse pressure and attenuation through the capillary bed under these two experimental conditions (figs. 8 and 9), the effects upon the frequency and distance dependence of the apparent phase velocity were considerably smaller than those of clamping the major branches (figs. 5 and 7). It is impossible to determine reflection coefficients on the basis of pressure measurements alone, but the data (figs. 6 and 7) indicate that the primary reflection sites in the pulmonary vasculature are represented by the major branch points. Our values for phase velocity are in good agreement with the 230 cm/sec which Caro and McDonald27 calculated from Patel's19 data. They investigated the frequency behaviour of the pulmonary arterial bed in the rabbit and found the average phase velocity to be about 85 cm/sec at 6-11 cycle/sec, and considerably higher at lower frequencies. The relatively stable value of their apparent phase velocity at higher frequencies may be related to the longer interval (in terms of wave length) over which the measurements were made. Due to an averaging effect, the discrepancies between measured and true phase velocity tend to be reduced if the determination is made over a long distance (fig. 3). There are several reasons why Womersley's9 estimates for reflections in the peripheral circulation are not valid for the pulmonary vascular tree. He calculated the amount of reflection as functions of cross section ratio and the non-dimensional parameter a. The latter is uniquely determined by vessel radius, angular velocity and kinematic viscosity of the liquid. His plots show a minimum of reflection for a cross section ratio between 1.1 and 1.3. This minimum, which represents the optimum for the energy transfer across the junction, is associated with the higher of these
values if the disparity in phase velocity between the original artery and its branches is large and vice versa. According to our data, the branch point of the main pulmonary artery has a cross section ratio of only 0.78. A rough calculation following Womersley's development, but neglecting the throttling effect due to viscosity, yields a reflection coefficient of 55%. Womersley's calculation assumes a circular tube and equal mass loading of all branches. Neither of these assumptions is valid for the pulmonary vasculature (tables 3 and 4), because of the absence of circular symmetry. Since the total cross section of all lobar branches is only some 8% larger than the cross section of the main pulmonary artery (table 3), the velocity of blood flow must remain nearly constant over a large part of the pulmonary arterial bed. The kinetic energy of the blood entering the small vessels is therefore considerably higher than in the peripheral circulation and the energy loss associated with multiple reflections could compensate for this to some extent. For a cardiac output of 2 liters/min the linear velocity of steady flow is about 30 cm/sec in the major pulmonary arteries, a value which has to be deducted from the measured phase velocity, since the latter represents the sum of true phase velocity and mean flow velocity. 3

Furthermore, the presence of right angles and changes in the spatial orientation of the major axis at the branch point may alter the reflection pattern. Although at present no physical theory is available for the wave propagation through a medium of such complex geometry, it is not surprising that the largest variations in phase velocity are encountered in the initial segment of the pulmonary artery (fig. 6). The elliptic shape of the cross section is not due to gravitational forces, as Rodenbeck29 seems to imply, since the spatial orientation of the major and minor semiaxes varies considerably between the various branches. The presence of an elliptic cross section has further implications. According to the Laplace relation, wall tension is proportional to pressure and wall thickness and inversely proportional to the vessel radius for a circular cross section. For the ellipse this problem is much more complicated, since the tangential stress is not only a function of the radius, but also of the angle. This implies either an unequal distribution of the stresses along the circumference of the vessel or structural non-uniformity. The details of the physical relations under these conditions are to be published subsequently.

Barthel30 has estimated the Poiseuille resistance for an ellipse as compared to a circular cross section. From his calculations the ratio of the resistances under these conditions is as follows:

$$R_e = \frac{8\mu L}{\pi A} \cdot \frac{a^2 + b^2}{a^2 b^2} = \frac{2}{\pi r^4} \left( \frac{a^2 + b^2}{a b} \right)$$

where $\mu$ = coefficient of viscosity

$r$ = radius of circular cross section

$a + b$ = the major and minor semiaxes of the elliptic cross section

Hence, for a given pressure gradient, one would expect a smaller flow rate in the elliptic vessel because of its larger flow resistance as compared to the circular vessel. Only for small eccentricities of the ellipse is this difference negligible.

Fry10 has reviewed the application of hydrodynamics to the living cardiovascular system and discussed the simplifying assumptions which would make it possible to derive instantaneous velocity from the axial pressure gradient. Our results indicate that some of these assumptions may not be valid in the pulmonary artery. Consider first the inlet length.31 At the entrance of the vessel the velocity distribution is uniform, although a function of time. As the flow proceeds further down the tube, this uniform velocity profile changes its form gradually. Because of viscous drag, the fluid particles close to the wall slow down and in order to maintain the same rate of flow, the particles near the axis of the tube accelerate. These changes in the velocity profile continue over a certain distance, the inlet length, until finally the profile becomes independent of entrance-effects and we have de-
velocities. Because of the multiple branching close to the heart, the straight part of the pulmonary artery is not long enough for the flow to become free of entrance-effects. Furthermore, the cross section ratio of 0.78 at the first branchpoint as well as the sharp bends in the proximal parts of the system will introduce vortex systems and convective acceleration pressure gradients which cannot be neglected. Changes in the spatial relations of major and minor axes of the different segments and their branches will introduce additional complications in the relation between pressure gradient and flow velocity as predicted by the Navier-Stokes equation. On the basis of the presently existing theories the magnitude of these secondary effects cannot be predicted, and most of these relations will have to be worked out on more realistic models before a comprehensive physical theory can be established.

Summary
The propagation of the pulse wave through the canine pulmonary arterial tree has been evaluated by means of harmonic analysis. The apparent phase velocity varied both with frequency and distance as one would expect in the presence of reflected waves. The phase velocity was about 230 cm/sec for the inflated and 325 cm/sec for the deflated lung. Theoretical considerations as well as experimental results indicate that the major branches represent the primary reflection site for the pulse wave. The pulse pressure is transmitted across the pulmonary capillary bed, although at a high attenuation, and the transmission varies markedly with lung volume.

The dimensions of the pulmonary arterial tree were evaluated from casts. Data for the length and cross sections of the various branches are given. The volume of the arterial tree as represented by the casts was 24 ml. The cross section of the various branches represents an ellipse rather than a circle, and the cross section ratios of the major branch points is considerably less than those postulated for optimal energy transfer. The implications of the geometrical findings for the relation between pressure gradient and flow are briefly discussed.

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References
PRESSURE TRANSMISSION IN PULMONARY ARTERIES

Pressure Transmission in Pulmonary Arteries Related to Frequency and Geometry

ERNST O. ATTINGER

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