Forward and Backward Transmission of Pressure Waves in the Pulmonary Vascular Bed of the Dog

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ABSTRACT

Pressures were measured in lobar pulmonary arteries, veins, and left atria of anesthetized open-chest dogs. Observations were made before and after snaring the lobar vessels and before and after infusing dextran to raise the mean pressure. Pressure waves were subjected to harmonic analysis. Forward (artery to vein) and backward (vein to artery) transmission (output/input pressure) and transmission ratios (forward/backward transmission) were calculated for each harmonic.

At low frequencies, forward transmission was increased by dextran, but backward transmission was unaffected. At low frequencies and high mean pressures the bed was nonsymmetrical, forward transmission being several times greater than backward at the same mean pressure (transmission ratio > 1).

The findings were analyzed with the aid of simple models. Nonsymmetry at low frequencies and high mean pressures appears to be due to arterial exceeding venous compliance. The findings and resulting theory are pertinent to the understanding of the shape and amplitude of arterial "wedge"-pressure traces.

ADDITIONAL KEY WORDS nonsymmetry of pressure wave transmission pulmonary artery compliance pulmonary vein compliance pulmonary artery wedge pressure pulmonary capillary pressure Fourier analysis

Theoretical studies suggest that pressure wave transmission in a vascular bed will be influenced by the relative compliances of the arteries and veins. A number of studies have been made of the compliance of the entire pulmonary bed (1), but little work has yet been done on the separate elastic properties of the arteries and veins (2, 3).

The relationship of pressure to radius was essentially linear in pulmonary arteries in excised rabbit lungs (3). By contrast, this relationship was markedly nonlinear in rabbit pulmonary veins, which became virtually inextensible at mean pressures above 10 cm H2O (3).

We studied the forward and backward transmission of pressure waves in the pulmonary circulation of the dog and found transmission to be nonsymmetrical. The mechanisms have been analyzed with the aid of simple models.

Theory

We wish to analyze forward and backward propagation of pressure waves in the pulmonary circulation. We have to simplify and consider the pulmonary circulation as a closed system, having zero mean flow, to which pressure oscillations are applied either at the ar-

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Model consisting of two rubber tubes joined by a rigid resistance tube for study of effect of non-symmetry of compliance and inertance on propagation of pressure waves.

The models should be considered as functional rather than structural representations. Their main deficiency is that they are lumped constructions, consisting of only a few discrete elements, whereas the pulmonary circulation is a distributed system having appreciable length relative to wave length.

Consider a model (Fig. 1) consisting of two rubber tubes which represent the compliance, or volume distensibility, of the pulmonary artery (C₀) and vein (Cᵥ). These are connected by a narrow rigid tube which represents the resistance (R) of the small pulmonary blood vessels. We wish to know the relationship between the pressures in these two chambers (P₀ and Pᵥ) when the outlet of the "vein" is closed and a pressure is applied to the "artery," or vice versa. We assume linear relationships between pressure and volume, and pressure and flow, and ignore fluid inertance. Then, with the vein outlet closed:

\[ P₀ = P_a - QR, \]

where Q is the volume flow rate of fluid through R, and

\[ Q = P_a/Z, \]

where Z is the impedance of R and Cᵥ in series. Thus:

\[ Z = R + (1/iωCᵥ), \]

where i is the imaginary operator and \( ω \) is angular frequency in radians/sec.

It follows that

\[ P₀ = P_a - [P_a - iωCᵥ/(1 + iωCᵥ)]R. \]

Therefore forward transmission, \( T_f \), the proportion of input (artery) pressure appearing in the downstream (vein) chamber, is given by

\[ T_f = P_a/P₀ = 1/(1 + RωCᵥ). \]  (1)

Thus, \( T_f \) is unity when \( ω = 0 \), diminishes with increase in frequency, and is independent of the value of \( Cᵥ \).

Similarly, if the pressure is applied to the vein, it follows that \( T_b \) (backward transmission) is given by

\[ T_b = P_a/P_v = 1/(1 + RωC₀). \]  (2)

We define the transmission ratio (\( T_r \)) as the ratio of forward to backward transmission, hence

\[ T_r = T_f/T_b = (1 + RωC₀)/(1 + RωCᵥ). \]  (3)

\( T_r \) will have a value of unity only when \( C₀ = Cᵥ \), or when \( ω = 0 \). If, for example, \( C₀ > Cᵥ \), \( T_r \) will have a value greater than one and it will tend to its limiting value of \( C₀/Cᵥ \). To summarize, with asymmetry of compliance, transmission will always be greater with propagation from a more to a less compliant chamber.

We have so far ignored the effects of fluid inertance (\( L \)), a property opposing change of volume flow rate. The inertance of a fluid column is proportional to density and length and inversely related to cross-sectional area. In this simple exposition, we ignore the complex variation of the inertance of a viscous fluid with frequency, kinematic viscosity, and tube radius (4). It follows from the relationship of fluid inertance to tube dimensions that the dominant inertance of the model shown in Figure 1 will be that of the fluid in the single narrow resistance tube. By contrast, in the pulmonary bed, the short "resistance vessels" have a very large combined cross-sectional area and are expected to contribute less inertance than the larger arteries and veins.

The inclusion in a model of inertances associated with the artery and vein leads to ex-
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FREQUENCY (CYCLES/SEC)

FIGURE 2

Transmission in inertially symmetrical models. Top, results in model with symmetry of compliance. Bottom, nonsymmetry of compliance caused nonsymmetry of transmission. All models exhibit resonance.

The transmission ratio was unity at very low frequencies, rose at 1.5 cycle/sec to a plateau of about 3.5 (approximately the ratio of arterial to venous compliance) and thereafter rose progressively with increase of frequency.

In the other model the artery again had the resistance. The artery and vein had the same dimensions, and as a consequence their inertances were equal. This model was studied when the compliances of artery and vein were equal and again after halving the compliance of the vein by increasing wall thickness without changing internal dimensions. In the other model, the compliances and inertances of the artery and vein were unequal.

Pressure was measured in the artery and vein (Fig. 1) in the same way as in the animal experiments (see below). Static compliances of artery and vein were determined by clamping the resistance tube at the mean working pressure level and injecting 0.4 ml water into each chamber. One end of the model was connected by rigid tubing to a pump which delivered a sinusoidal flow of water, while the other was closed with a tap. Flow oscillations were applied over the frequency range 0.25-5.0 cycle/sec. The procedure was repeated after connecting the pump to the other end. Mean pressure was the same in all comparable experiments, and pressures were at all times greater than atmospheric.

RESULTS

Inertially Symmetrical Models

The transmission was about unity at 0.25 cycle/sec in all experiments (Fig. 2). In the symmetrically compliant model (Fig. 2, top) it rose to about 1.3 at 1 cycle/sec (indicating some degree of resonance), and thereafter fell with increasing frequency. As expected $T_a = T_b$ at all frequencies, i.e. $T_r = 1$.

In the model with asymmetry of compliance (Fig. 2, bottom) $T_a > T_b$ above 1 cycle/sec. Stiffening the vein increased $T_a$ but had almost no effect on $T_b$. This is to be expected from equations 1 and 2. $T_r$ rose with frequency up to 2 cycle/sec and then appeared to reach a final value of about 2.3. The value expected from direct measurements of compliance was 2.0 (equation 3).

Inertially Nonymmetrical Models

Two models were studied. In the first, the compliance and inertance of the artery exceeded those of the vein. The transmission ratio was unity at very low frequencies, rose at 1.5 cycle/sec to a plateau of about 3.5 (approximately the ratio of arterial to venous compliance) and thereafter rose progressively with increase of frequency.
the higher compliance, but this time the lower inertance. The transmission ratio was again unity at very low frequencies, rose to about 13 at 2 cycle/sec and thereafter fell again towards unity. The ratio $C_d/C_w$ was in this case approximately 5. The behavior of this particular model qualitatively most closely resembled that seen in the dog lung.

**Animal Experiments**

**METHODS**

Eighteen unselected adult mongrel dogs weighing 18 to 25 kg were studied. They were anesthetized with sodium pentobarbital (Abbott), 0.5 ml/kg i.v. The chest was widely opened through the left fourth intercostal space. The animals were then ventilated with positive pressure, using a tracheal cannula and a Smith-Clarke respirator. End-expiratory pressure was 5 cm H$_2$O.

Measurements were usually made on the left lower lobe of the lung (Fig. 3). A fine polyethylene catheter was inserted into a small branch of the lobar pulmonary artery and another into a small branch of the vein. They were arranged so that their open ends were flush with the wall of the parent vessel. A third catheter was inserted into the left atrium.

Loose snares of monofilament nylon were positioned around the lobar artery and vein upstream and downstream from the respective catheters (Fig. 3). Observations were made with cyclical respiration suspended and with the lungs inflated with oxygen to a steady pressure of 5 cm H$_2$O. Vascular pressures were measured in the control state and after infusing approximately 500 ml of 6% dextran in saline (Dextraven, Glaxo). The dextran was used to raise mean pulmonary venous pressure to the level of mean pulmonary artery pressure in the control state, thus making possible measurements of forward and backward transmission at about the same mean pressure. Measurements were made with snares loose, 10 sec after snaring either lobar artery or vein, and 10 sec after the snare was released. Cyclical ventilation was resumed between each run.

Pressures were measured with Statham P23Gb or C.E.C. (Devices Ltd.) strain gauge pressure transducers. The dynamic characteristics of the transducers, together with the attached catheters, were determined by the pressure transient method (4) at the beginning and end of the experiment. The damped natural frequency was usually about 100 cycle/sec and the relative damping ($\beta$) was 0.1 to 0.2. These factors were taken into account when the pressure waves were being subjected to harmonic analysis.

The output from each pressure transducer was led to a galvanometer (natural frequency 300 cycle/sec) in an ultraviolet light recorder (S.E. 2000, S.E. Laboratories, Feltham, Middlesex). Records were made at a paper speed of 25 cm/sec. For purposes of static pressure calibration and of flushing catheters, all manometers were connected via stopcocks to a single junction tap which could be switched to a zero reservoir, a pressure reservoir or a flushing bottle containing heparin in saline (5000 U/500 ml). The surface of the zero reservoir was level with the main pulmonary artery.

**Measurement of Records and Computation**

One or two cardiac cycles were selected in each experiment. Pressure ordinates were measured at intervals of 0.01 sec and the values, together with the static and dynamic calibration factors of the pressure transducers and other relevant parameters, were transferred to punched cards. The analyses were performed by an IBM 7094 digital computer. The analyses consisted of conversion of all ordinates to pressure, Fourier analysis of each line to 10 harmonics of the heart rate, and the calculation of the moduli and phases of each harmonic after correction for the dynamic performance of the manometer system.

When the period chosen for analysis did not contain an integral number of time lines (1/100 sec), the reading for the first line differed from that of the N + 1 time line (where N is the number of readings). The difficulty was overcome by dividing the difference between the two readings by N, and adjusting all intermediate readings by the appropriate fraction of the difference.

**RESULTS**

Because in several experiments we were unable to obtain three satisfactory pressure records and excluded from analysis animals that did not receive dextran, the results in
only 7 of the 18 dogs are considered. Even then parts of the records of dog 9 and 10 had to be omitted.

The mean pressure in the pulmonary artery, vein and left atrium before dextran was the same but after dextran the arterial pressure rose to equal pulmonary venous pressure. However, before dextran, with the arterial snare, the arterial pressure fell to equal pulmonary arterial pressure in all dogs. In all cases there was a pressure difference of several cm H$_2$O (mean 31.5 ± 2.6 cm H$_2$O).

In Table 1, with the vein snared, blood pressure was the same in open-chest dogs 17 and 18 with dextran infusion. Contrast without venous snare, with pulmonary artery occluded, and with pulmonary artery occluded and with pulmonary vein occluded, and with pulmonary artery occluded and without dextran are shown. The curves were traced from actual records and are mounted to permit direct comparison of wave forms and time relations. Comparison of forward transmission (vein snared) before dextran with backward transmission (artery snared) after dextran shows nonsymmetry at approximately the same mean pressure.

### TABLE 1

<table>
<thead>
<tr>
<th>Dog</th>
<th>Before dextran</th>
<th>After dextran</th>
</tr>
</thead>
<tbody>
<tr>
<td>17</td>
<td>21.27</td>
<td>11.41</td>
</tr>
<tr>
<td>12</td>
<td>22.41</td>
<td>9.30</td>
</tr>
<tr>
<td>Mean</td>
<td>19.32</td>
<td>6.29</td>
</tr>
</tbody>
</table>

*Records obtained after bleeding animal following dextran infusion. †Mean gradient.

P.A. = pulmonary artery; P.V. = pulmonary vein; L.A. = left atrium.

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**FIGURE 4**

Pressure waves in pulmonary artery, vein, and left atrium of open-chest dog 17 before and after dextran in dogs 13 and 12 are presumably due to manometer drift. The mean pressure in the pulmonary artery, vein, and left atrium before dextran was the same but after dextran the arterial pressure rose to equal pulmonary venous pressure. However, before dextran, with the arterial snare, arterial pressure fell to equal pulmonary arterial pressure in all dogs. In all cases there was a pressure difference of several cm H$_2$O (mean 31.5 ± 2.6 cm H$_2$O).
2.6) between the pulmonary artery and vein. Where mean venous pressure exceeded alveolar pressure (5), the presumptive explanation is that pressure equilibration between artery and vein was incomplete at the time of measurement.

Table 1 shows that in 3 of the 5 dogs (13, 17, 18) mean vascular pressure with artery snared after dextran was approximately equal to mean vascular pressure with vein snared before dextran. This pressure difference was relatively small in the other 2 animals.

Pressure waves recorded from pulmonary artery, pulmonary vein, and left atrium (dog 17) are shown in Figure 4 before and after infusion of 500 ml dextran. The records were taken in the control state, i.e. before snares, with artery occluded and with vein occluded. The static and dynamic calibration of the three pressure transducers was approximately the same so that the amplitude and time relations of the three curves can be compared. Mean pressures are shown in cm H2O.

Before dextran (control) the arterial wave needs no comment. The venous and atrial waves resemble each other. The venous snare caused a change in the shape of the venous wave, which came more to resemble the arterial wave but was delayed and attenuated. Its amplitude is about half that of the arterial wave and it appears smoothed, suggesting loss of high frequency components in transmission.

When the pulmonary artery was snared, there was a striking change in the arterial wave. This became virtually featureless apart
from a few small high frequency oscillations. Comparing the traces with artery and vein occluded, there was greater forward (artery to vein) than backward (vein to artery) transmission of pressure. However, because of the lower mean vascular pressure with artery snared (8.5 cm H$_2$O as compared with 25.7 cm H$_2$O), vascular resistance could have been higher during backward transmission, thus accounting for the nonsymmetry of transmission.

To exclude this possibility, dextran was infused so that mean vascular pressure with artery snared would be approximately equal to mean pressure with vein snared before dextran. The pressure waves at the three sites after the infusion are shown in Figure 4. With the vein occluded, there was greater forward transmission than there had been with the vein occluded before dextran. However, with the artery occluded there was little more backward transmission than with the artery occluded before dextran. Comparison of vein occluded before dextran with artery occluded after dextran shows nonsymmetry of transmission, even though the two records were obtained at virtually the same mean pressure (25.7 and 23.3 cm H$_2$O).

The results in Figure 5 are based on harmonic analysis of the pressure traces in 5 dogs. They are averaged with respect to modulus of pressure and frequency at each harmonic. At low frequencies, dextran caused a large increase in forward transmission (0.02 < P < 0.025, at the frequency of the fundamental). By comparison, the infusion had no significant effect on backward transmission.

Figure 5 also shows the relationship of transmission to frequency. Both before and after dextran, artery-to-vein transmission fell with increase of frequency. This explains the rather smoothed appearance of the venous waves with vein occluded (Fig. 4). Vein-to-artery transmission appears to increase at the frequency of the third and fourth harmonics, both before and after dextran (Fig. 5), but the reason for this is not known.

With the artery snared, transmission from left atrium to vein (6 dogs) averaged about 75%, and this did not vary significantly with frequency or after dextran.

The ratio of forward transmission before dextran to backward transmission after dextran, i.e. the transmission ratio, has been plotted for the first five harmonics of the pulse (5 dogs) (Fig. 6). The ratio exceeds unity for all dogs at the frequency of the fundamental and in four dogs also at the frequency of the second harmonic. Thereafter, with increase of frequency it tends to approach or fall below unity. In dog 17 (Fig. 4), forward transmission at the frequency of the second harmonic is 6.6 times greater than backward transmission measured at almost the same mean pressure (approximately 24 cm H$_2$O, Table 1). Judging from a very small series (dogs 13, 17, 18) in which mean vascular pressure was almost the same during forward and backward transmission, nonsymmetry was greater the higher the mean pressure (Fig. 6 and Table 1).

The harmonic content of the pressure waveforms (7 dogs) without snares, before and after dextran, can be seen in Figure 7. The moduli have been averaged for each harmonic. As previously observed, the moduli of the arterial pressure wave diminish with frequency (6-8). The atrial waves contain relatively more high frequency components than left atrium to vein (6 dogs) averaged about 75%, and this did not vary significantly with frequency or after dextran.

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Harmonic content of pressure waveforms in pulmonary artery, vein, and left atrium without snares before and after dextran (average values 7 dogs). Mean pressure is shown at harmonic 0. Note that second and higher harmonics are relatively richer in atrium and vein than in pulmonary artery. Dextran increased size of venous and atrial moduli but did not affect arterial moduli.
do the arterial waves (9) and so do the venous waves. Dextran, while it increased mean pressure, had no effect on the absolute magnitude of the arterial moduli. However, it significantly increased the magnitude of the venous and left atrial moduli.

**Discussion**

We have made a number of assumptions concerning the linearity of resistance, compliance and iner-tance in the pulmonary bed. Provided vascular pressure exceeds alveolar pressure (5), there appears to be a reasonably linear relationship between pressure gradient and flow (10, 11). It has also been shown that pulmonary vascular resistance is independent of the direction of perfusion (12).

The assumption of linear compliance is justified for rabbit pulmonary arteries (3) and for the main pulmonary artery of the dog (13), though linear pressure-radius and pressure-volume relationships are clearly not equivalent. However, the pulmonary veins of the rabbit have nonlinear pressure-radius relationships (3) and the present findings suggest that the same is true for the dog. Nevertheless, the assumption of linearity may not be violated when pressure oscillations are relatively small, as they were in these experiments. Similar reasoning justifies the assumption of constant fluid iner-tance in compliant tubes subjected to pressure oscillations.

These assumptions are further supported by observations on the hydraulic input impedance of the canine pulmonary vascular bed which showed linearity of behavior over a wide range of frequencies and pressures (8). Although the pulmonary veins may have nonlinear pressure-volume relationships, the relatively small amplitude of the pressure moduli (Fig. 7) may still justify the use of Fourier analysis.

The most striking finding was nonsymmetry of forward and backward transmission of pressure waves. This was obvious even in raw data (Fig. 4). Nonsymmetry of this sort does not appear to have been previously reported. The explanation is undoubtedly complex and our analysis is an oversimplification since it is based on a lumped system. Nonetheless we think it likely that the nonsymmetry, occurring particularly at the frequency of the lower harmonics of the pulse and at high mean pressures, was due mainly to inequality of pulmonary arterial and venous compliance.

Our analysis indicates that iner-tance will become dominant only at high frequencies and that transmission in either direction will be determined by the compliance of the chamber downstream of the resistance. We found that backward transmission was unaffected by a rise of mean pressure caused by dextran, while forward transmission was enhanced.

From these findings we argue that the compliance of the pulmonary arteries was unaltered by the rise of mean pressure, while that of the pulmonary veins was reduced. Linear relationships have already been demonstrated between pressure and radius for the main pulmonary artery of the dog (13) and for pulmonary arteries of various sizes in isolated rabbit lungs (3). No data are available for canine pulmonary veins, but the compliance of rabbit pulmonary veins was markedly reduced at high mean pressure (3).

If this conception of the mechanical properties of the canine pulmonary vascular bed is correct and holds also for man (14), it helps in the interpretation of pressure waves recorded with a cardiac catheter in the pulmonary artery wedge position. Pressure waves may usually be detected under these conditions in man. However, they are usually undetectable beyond an occluding balloon in one of the main pulmonary arteries (15) or distal to a snared lobar artery (14).

The vascular segment between the tip of a wedged catheter and the resistance vessels will have a low compliance because of its small absolute volume, and possibly also because of the increased wall stiffness of small arteries (3). Both factors will enhance transmission of pressure waves into the relatively iner-tant closed segment. By contrast the greater compliance of the major part of the...
normal pulmonary arterial bed will serve to attenuate atrial pressure oscillations.

The act of wedging a cardiac catheter will not only remove pressure waves transmitted from the pulmonary artery but will also enhance retrograde pressure wave transmission. Normal capillary pressure oscillations are unlikely therefore to resemble those recorded with a wedged catheter.

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