Transmission of Pulsatile Blood Pressure and Flow through the Isolated Lung

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ABSTRACT
A horizontal isolated lung preparation has been used to examine the transmission of pulsatile blood pressure and flow. Arterial and venous pressure and flows were measured with pressure transducers and electromagnetic flowmeters. After a sudden increase in pulmonary arterial pressure, delays of several seconds were observed before venous flow began to increase. Flow then increased slowly over a period of 1 to 9 seconds to a new steady level. The transmission of pulsatile flow was independent of the outlet pressure and identical for perfusion in the forward and reverse directions. Flow transmission fell from about 75% at a frequency of 0.03 cps to approximately 10% at a frequency of 1 cps. Pressure transmission down to the collapsible vessels was measured when alveolar pressure exceeded venous pressure. At 0.1 cps approximately 50% of the incident pressure wave was transmitted, and at 1 cps this was reduced to 30%. It was concluded that the lung passed well only the low-frequency components of the applied input and that in a vertical lung only about 30% of the mean to peak pressure pulse would be transmitted down to the small vessels and therefore affect the distribution of blood flow.

ADDITIONAL KEY WORDS
regional lung perfusion
pulmonary capillary flow pulse
wave transmission
input impedance

Measurements made with the body plethysmograph in man (1) and in dogs (2) have demonstrated that pulmonary capillary blood flow is markedly pulsatile. Flow pulsations have been seen by direct observation though not consistently (3). These experiments were carried out to extend the above observations and to examine in more detail the frequency dependence of the pressure and flow transmission within and through the pulmonary vascular bed.

An additional reason for our interest is that pulmonary artery pressure is pulsatile and this may affect the distribution of blood flow in the vertical lung. A model has been suggested (4) to explain the increasing flow toward the base of the isolated lung under conditions of steady perfusing pressure. Briefly, at moderately high lung volumes the lung may be divided into three zones of flow. In zone I, where pulmonary artery pressure is less than pericapillary pressure (approximately alveolar pressure) there is no flow. In zone II, where pericapillary pressure is less than pulmonary venous pressure, flow is governed by the usual arterial-venous pressure difference. In zone III, venous pressure is greater than pericapillary pressure, and flow is governed by the arterial-pericapillary pressure difference. In this scheme, flow rises to the level at which pulmonary artery pressure is equal to pericapillary pressure when...
the lung is perfused with nonpulsatile flow. We extended this work to examine the effects of periodic fluctuations in pressure and flow on a horizontal lung which was subject to pressures appropriate to each of the above zones. The opportunity was taken to study the lung preparation under conditions of low flow rates and with pressures appropriate to those near the top of the vertical lung. Measurements were also made of the proportion of the input pulsatile pressure wave transmitted to the smaller vessels of the lung and of the input impedance of the lung at various frequencies.

Methods

A horizontal isolated lung preparation was used. Conditions in either zone II or zone III of the vertical lung were studied by varying the relationships between pressures in the pulmonary artery, pulmonary vein, and alveoli. The experimental preparation has already been described (4) and will be briefly reviewed here. A greyhound was anesthetized with a barbiturate given intravenously, and the left lung was excised and placed on a horizontal plate in a Lucite box. Glass cannulas were tied into the left pulmonary artery, left main bronchus, and left atrium, and these projected through the front of the box. They entered the lung approximately 4 cm above its lower margin. The isolated lung was ventilated with intermittent negative pressure and perfused with venous blood at 37°C pumped from a donor mongrel dog. For each measurement the lung was held at a transpulmonary pressure of 10 cm H₂O. The mean height of the lungs at this transpulmonary pressure was 6.2 cm. The mean lung weight of 29 preparations was 140 g (excluding cannulas), which increased by an average of 12% (range 1% to 28%) in the course of the experiment, which lasted 5 to 6 hours. The lungs were weighed in a collapsed state (transpulmonary pressure zero) with cannulas in place at the beginning and the end of each experiment. The blood vessels were drained to the same extent before each weighing.

Figure 1 is a schematic diagram of the preparation and the equipment used to develop pulsatile pressures and flows. The oscillatory flow and pressure were generated by a cam-driven sinusoidal pump similar to that described by Taylor (5). The pump was driven by a controlled-speed motor (Servomex) at frequencies between 0 and 4 cps via a reduction gear box (30:1 or 100:1). In some experiments it was necessary to superimpose the pulsatile flow on a given steady flow of blood. This came from a high-pressure bottle and passed through a constriction which ensured that no oscillatory flow returned to the reservoir. Pressure swings of about ±10 cm H₂O were generated by the pump. Stroke volume did not exceed 35 ml. In some experiments larger pressure swings were needed, and these were generated by suspending a blood reservoir from a wire which passed over a pulley on the ceiling and was attached to the end of a crank arm rotated by the pump motor. The arterial and venous pressures were recorded with strain-gauge manometers (Consolidated Electrodynamics, England, 0-75 cm Hg). The manometers recorded pressures, via thin side-hole polythene catheters, from points at which the glass cannulas were tied into the lung vasculature. For each experiment the frequency response of the manometers was determined by the pressure-transient method (6). The damped, natural frequency of the manometers as used was in the region of 100 cps with relative damping of about 0.2. The measurements of transmission were made at frequencies between 0 and 4 cps with a manometer sensitivity of about 1.9 cm H₂O/cm deflection. Arterial and venous flows were measured with a two-channel electromagnetic flowmeter (Medicon M4000) using cannulating flow transducers of 5-mm lumen diameter fitted into the end of the glass cannula. The relative positions of the flow transducers are shown in Figure 1. Sensitivities were about 1.5 ml/sec/cm deflection. The arterial and venous glass cannulas had an internal diameter of 7 mm and 9 mm, respectively, and were 10 and 12 cm long. The manometers and flowmeters were calibrated under conditions of steady flow and pressures at the beginning and end of each experiment and were checked during the study. The frequency response of the flowmeter was determined electrically. Amplitude response was reduced to 90% at 10 cps and a phase shift of 4°/cps was present. While the sensitivity of these transducers was constant there was evidence of a
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small zero drift during the experiment, for which correction was made. It should be noted that the measurement of the amplitudes of the flow waves is not affected by zero drift.

There were three experimental designs: step function experiments, flow transmission experiments, and pressure transmission experiments. In every experiment the lung was ventilated in the same fashion before any measurements were made. It was first expanded twice to a transpulmonary pressure of 20 cm H₂O and then ventilated between 5 and 10 cm H₂O for 30 to 60 seconds. It was then held at 10 cm H₂O transpulmonary pressure for each measurement. Between measurements the lung was ventilated at 6 breaths/min and perfused with blood from the donor dog.

STEP FUNCTION EXPERIMENTS

In these experiments the pulmonary artery pressure was initially placed below alveolar pressure at the bottom of the lung so that no flow occurred. The arterial pressure was then increased suddenly by opening a clip in the line to a raised reservoir above the top of the lung. The form of the step increase in pressure is shown in the top curve of Figure 2. The rise time of the pressure was about 250 msec, and the overshoot was less than 5% of the pressure change. Arterial and venous pressures and flows were recorded prior to and following the sudden increase in arterial pressure. Initial arterial pressures were varied between ~13 and +13 H₂O in relation to the hilum. The final arterial pressure was usually 20 cm H₂O and constant for each lung.

FLOW TRANSMISSION EXPERIMENTS

To measure the frequency dependence of flow transmission through the lung, a sinusoidal pulmonary artery pressure was so arranged that arterial pressure exceeded alveolar pressure at all times. Mean pulmonary arterial pressure was approximately 10 cm H₂O above alveolar pressure at the top of the lung with a peak-to-peak amplitude of approximately 8 to 10 cm H₂O at 1 cps and 4 cm H₂O at 0.04 cps. The arterial and venous flow oscillations were recorded at each frequency with the venous pressure at one of two levels. For the first series of experiments the venous pressure was placed 10 cm below the plane of the pulmonary artery, thus the lung was under zone II conditions. The venous pressure was then suddenly increased to a new steady level 10 cm above the plane of the pulmonary artery, placing the lung under zone III conditions, where venous pressure is greater than pericapillary pressure. The whole experiment was then repeated while the lung was perfused in the reverse direction, from vein to artery. In this case the arterial pressure was placed either 10 cm below or above the plane of the pulmonary artery. Measurements were thus made of the flow oscillations transmitted through the lung under conditions simulating zones II and III during both forward and reverse perfusion. The amplitude of the flow oscillations varied from about 20 ml/sec at 1 cps to 2 ml/sec at 0.04 cps. Mean flow was approximately 4 ml/sec.

In a further series the relationship between mean venous flow and the frequency of the sinusoidal arterial pressure pulse was examined. In these experiments mean pulmonary artery pressure was itself too low to cause flow although the peak pressure rose well above the top of the lung. These experiments were repeated with increasing mean pulmonary artery pressures.

PRESSURE TRANSMISSION EXPERIMENTS

The experiments so far described were concerned with measurements at the input and output of the lung. To examine the effective pressure transmission down to the collapsible vessels of a lung under zone II conditions, that is where the pulmonary venous pressure is less than pericapillary pressure, the following experimental design was used. Initially, a steady flow was passed...
through the lung, generating a steady pulmonary artery pressure (see inset Fig. 9). This flow was then suddenly stopped, and the fall in pulmonary artery pressure recorded continuously. This fall is indicated by the solid line in the inset; pressure fell and equilibrated at a value slightly below the bottom of the lung. This pressure, at which flow stopped, was taken as a measure of pericapillary pressure for it is the pressure at which small vessels in the region of the capillaries close. The experiment was repeated with pressure oscillations superimposed on the same steady pressure. Steady flow was then arrested by clamping the line to the pressure bottle. Pressure again fell (Fig. 9, inset, dotted line) but in all experiments flow ceased when the mean of the pressure swings was below the final pressure reached in the steady pressure experiments. The simplest assumption here is that flow completely ceases through the collapsible vessels when their peak internal pressure during the cycle is less than their perivascular pressure. The difference in the two final mean pressures is equal to the magnitude of the mean to peak effective pressure pulse transmitted down to the collapsible vessels.

**Results**

**STEP FUNCTION EXPERIMENTS**

Figure 2 is a photograph of an actual record and illustrates the pattern of pressures and flow after a sudden increase in pulmonary artery pressure. Following the sudden rise in arterial pressure there was a significant delay before venous flow increased, and thereafter it rose slowly to a new steady level. On the arterial side the flow suddenly rose and overshot before it decreased to a new constant level. The slow increase in the venous pressure and flow in spite of the sudden increase in pulmonary artery pressure suggests that the lung was passing only the low frequency components of the input function.

In Figure 3 the results of six experiments are shown in which the volume of blood which passed into the lung before venous flow commenced is plotted against the initial pulmonary artery pressure measured with reference to the plane of the pulmonary artery. The average final pulmonary artery pressure was 20 cm H$_2$O. The injected volume was obtained by integrating the pulmonary arterial flow curve from time zero to the time at which venous flow increased. The curve shows a diminishing injected volume with increasing initial pulmonary artery pressure. Two conditions of venous pressure are shown. The upper line represents the lung placed under zone II conditions, that is with venous pressure less than pericapillary pressure at the bottom of the lung. The lower line is that of the lung during zone III conditions, that is with venous pressure just above pericapillary pressure at the top of the lung. The standard deviation of the mean is shown for four points on the diagram.

Similar curves are shown in Figure 4 where the delay before venous flow changed is plotted against the initial pulmonary artery pressure.
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It can be seen that delays as long as 1.8 seconds occurred at initial arterial pressures of about –14 cm H$_2$O which reduced to 400 msec at high starting pressures (+15 cm H$_2$O). The delay varied with changes of venous pressures and was further reduced to about 200 msec when venous pressure was just above pericapillary pressure at the top of the lung. Standard deviations of the mean are given at four points. The difference between the zone II and zone III curves is significant (P < 0.02).

FLOW TRANSMISSION EXPERIMENTS

In Figure 5 original records of two flow transmission experiments are illustrated. In the left-hand panel, the venous flow amplitude is very small compared to the arterial flow amplitude. The frequency is 1.1 cps. The right-hand panel shows the marked increase in the venous flow amplitude in the same lung when the frequency was decreased to 0.04 cps. At this frequency approximately 67% of the arterial flow amplitude is passed to the venous side. The lung is under the same conditions except that the frequency has been reduced.

In Figure 6 the percentage of the input flow amplitude which appeared at the output of the lung is plotted against frequency. Both input and output flow were sinusoidal. Each point represents the mean value of four measurements which lie within an interval of one tenth of 1 cps. Four curves are shown representing the forward transmission on four preparations and the reverse transmission on three preparations under two conditions of venous pressure. The lung was placed in the conditions of zone II or zone III by holding the venous pressure 10 cm H$_2$O below or above the plane of the pulmonary artery. The general shape of the curves is the same under all conditions and the transmission decreased as frequency increased to 1 cps. At very low frequencies (less than 0.1 cps) approximately 70 to 80% of the input flow pulse was transmitted to the output side of the lung, whereas at 1 cps this was reduced to about 10%. For the forward perfusion experiments comparisons were made between the transmissions at the same frequencies up to 0.6 cps during zone II and III conditions, and no significant difference was demonstrated. This means that changing the output pressure did not alter the percentage of the input flow pulse transmitted to the output of the lung. Similar comparisons were made during perfusion from vein to artery, and again no significant difference between the transmission during zone II or zone III conditions was evident. In Figure 7 the
The left panel shows an original record of flow transmission at a frequency of 1.1 cps. The venous pressure was less than alveolar pressure (zone II), and flow was in the forward direction from artery to vein. Note the extremely small magnitude of the venous flow wave. The right panel shows the flow transmission in the same lung under the same conditions of venous pressure except that the frequency was reduced to 0.04 cps. Note the much greater flow transmission, approximately 67% in this instance.

The ordinate shows the percentage of the input flow pulse which appears on the output side of the lung. Transmissions for both zones have been combined to compare the transmission in the forward and reverse directions. No significant difference exists between the transmission in these two directions, (P > 0.05).

In all these experiments the outflow tube from the pulmonary artery or veins was plastic, 0.9 cm diameter and 170 cm long. For the purpose of comparing transmission in different zones and directions no correction is necessary for the presence of this tube. However, the fluid impedance represented by a column of blood of these dimensions will reduce flow transmission through the lung, and our results in Figures 6 and 7 have been corrected for this effect.
The ordinate expresses the flow transmission as the percentage of the input flow pulse which is transmitted to the output side of the lung. The solid symbol represents the transmission in the forward direction irrespective of venous pressure. The open symbol represents transmission in the reverse direction, that is, from vein to artery, irrespective of outlet pressure. There was no significant difference between the flow transmissions in the forward and reverse directions.

Corrected for this effect. The pressures at the downstream end of the pulmonary vessels could not be held constant but oscillated by an amount sufficient to drive fluid through the tube, thus altering the pressure gradient across the lung. It is possible to correct for this knowing the hydraulic impedance of the lung and of the tube. The latter was calculated from the equations of Womersley (7) and agreed well with actual measurements. The necessary correction increases flow transmission by approximately the same amount at all frequencies used, namely by 8% absolute.

Some experiments were done in which the output tube was shortened by 50%; this increased transmission through the lung by the expected figure of 4% absolute.

In another series of experiments the mean pulmonary artery pressure was placed at different levels below the top of the lung. In Figure 8 one example is shown in which the mean pressure was placed successively at levels —8 cm, —5 cm, and +1 cm in relation to the hilum. Average venous flow was measured for each of these steps and plotted against the frequency of the applied sinusoidal pressure wave. The peak pulmonary artery pressure was approximately constant over the frequency range at each mean pressure. The

**Figure 7**

The ordinate expresses the flow transmission as the percentage of the input flow pulse which is transmitted to the output side of the lung. The solid symbol represents the transmission in the forward direction irrespective of venous pressure. The open symbol represents transmission in the reverse direction, that is, from vein to artery, irrespective of outlet pressure. There was no significant difference between the flow transmissions in the forward and reverse directions.

**Figure 8**

The mean flow passing through a lung plotted for three different values of mean arterial pressure. Peak arterial pressure is above the top of the lung. Note that flow falls with increasing frequency and there is a frequency above which no flow occurs for mean pulmonary artery pressures of —5, —8 cm H₂O. The pump frequency at which mean flow is zero increases as the mean arterial pressure increases. Lung bottom is —4 cm below the hilum.
The percentage of the input pressure amplitude which is transmitted down to the collapsible vessels of the lung. The abscissa is the frequency of the sinusoidal input pressure pulse. The inset in the upper right-hand corner is a schematic diagram of the experimental design (see text). Effective pressure transmission falls with increasing frequency of the input pressure pulse. The different symbols represent experiments on different lungs.

Three curves are for venous flow at each level of mean pulmonary artery pressure. It can be seen that for mean pressures well below the hilum of the lung there was a frequency above which no flow occurred. At higher mean pressures this frequency increased. When the mean pressure was above the hilum of the lung, some mean flow was always evident. We postulated that with increasing frequency the pressure oscillations reaching the small vessels of the lung were too small to open them. The pressure transmission experiments were designed to determine the fraction of the input pressure pulse which reached these vessels.

**Pressure Transmission Experiments**

In Figure 9 the percentage of the input pressure oscillation transmitted to the collapsible vessels is plotted against frequency on a logarithmic scale. The effective pressure transmitted to the collapsible vessels was reduced as frequency was increased. At a frequency of 0.03 cps only about 70% of the input pressure amplitude was transmitted to these vessels, and this fell to between 20 and 30% at 1 cps. Thereafter there was evidence of a slower fall up to the maximum frequency used (4 cps). In these experiments the venous pressure was placed well below the bottom of the horizontal lung such that the unit was in zone II. The different symbols represent experiments on 5 lungs.

**Input Impedance**

In both the flow and the pressure transmission experiments reported above the oscillatory arterial pressures and flows were measured. From these the ratio of arterial pressure amplitude to arterial flow amplitude, that is, the modulus of the input impedance of the lung, was calculated together with the phase difference between arterial pressure and flow. These values are plotted against frequency on a logarithmic scale in Figure 10. The divisions on the abscissa indicate the frequency range for each mean value. The figures in brackets are the numbers of points from which the mean was calculated. The

![Figure 9](image)

![Figure 10](image)
vertical lines show ± 1 SD of the mean at three frequencies. With diminishing frequency the impedance rose to a maximum value between 0.1 and 0.2 cps and thereafter decreased as the frequency was further reduced. The difference between the maximum input impedance and that at the lowest frequency is significant (P > .05). These figures have been corrected for the frequency response of the flowmeters. Alterations of transmural pressure in the major pulmonary arteries from +10 to +30 cm H$_2$O did not consistently alter the input impedance.

Discussion

In these experiments we did not find evidence to suggest that our results were affected by the size of the input pressure or flow pulses. Thus we propose to analyze the system as if it were linear in accordance with the observations of Bergel and Milnor (8), with the important exception that flow ceases at all points where local perivascular pressure exceeds pulmonary artery pressure.

The pressure-flow characteristics of the horizontal lung preparation have previously been studied under conditions of steady perfusing pressure. We used the same preparation to analyze the pulsatile transmission of pressure and flow with different relationships between arterial, venous, and alveolar pressures. As a basis for interpretation, consider three vascular regions in the horizontal lung. These, the arterial, collapsible, and venous sections, are shown in Figure 11. The arterial and venous segments are distensible though stiffness varies along their length (9). The diameter of the individual vessels decreases towards the collapsible segment. The collapsible unit includes the capillaries but extends further out into the elastic small vessels. Observations on frozen lung sections in this laboratory reveal that in zone 1 where pulmonary artery pressure is less than pericapillary pressure most of the vessels of diameter less than about 30 μ are closed. The pressure surrounding the collapsible vessels is approximately that in the alveoli, but it can be less, depending on the volume history of the lung (10). In the above experiments the lung was taken through an identical volume history before each measurement. The pressure around the arterial and venous segments will vary between pleural pressure for the larger vessels and interstitial pressure, which is probably more negative than pleural pressure, for the smaller vessels surrounded by lung parenchyma (11). The final diameter of the lung vessels depends on the balance between transmural pressure, elasticity, and smooth muscle tone.

Three factors at least will combine to affect the relationship between input and output waveforms of pressure or flow in the lung. The first is the delay due to transmission of waves down a tubular system at a finite velocity. This velocity depends on the elastic characteristics of the vessels, their size, the frequency components in the imposed disturbance, and the nature of the contained fluid. Where the fluid is viscous and the wall exhibits visco-elasticity, as is the case here, the amplitude of the waves will be reduced as they travel. If in addition there exist sites of reflection within the system, transmission velocity and attenuation will be altered. These effects have been intensively studied in relatively large vessels, but less is known of transmission in vessels of arteriolar and capillary size (6).
Secondly, transmission will depend on the distribution of path-lengths and transit times in the bed, for waves which have travelled by different routes may well arrive at the venous end out of step, and the resulting waveforms will be due to summation of the individual waves. Taylor (12) has discussed the properties of such a system in a computer study of the input impedance and transmitting properties of a randomly branching arterial system.

Thirdly, it is postulated that at some point in the pulmonary system vessels close and open under the influence of the local transmural pressure. The delays occurring in the opening and closing process, together with any variation in the opening and closing pressures at any level, will further influence the transmitting properties of the system.

Although there is good evidence that the small vessels collapse when arterial pressure is less than that in the alveoli, it is likely that they do not all require the same arterial pressure to open them. Thus the proportion of vessels open at any time will depend on the pulmonary arterial pressure. No transmission can take place through a closed vessel but once it is open its transmitting properties will be determined by the factors mentioned.

All these effects are presumably influencing the results reported here, but it is not possible to distinguish between them.

**STEP FUNCTION EXPERIMENTS**

When the horizontal lung has a low venous pressure the volume of the venous segment is small. If arterial pressure is less than alveolar pressure there is no flow through the lung, and the vessels exposed to alveolar pressure collapse. The initial volume of the arterial segment decreases as the starting pulmonary artery pressure is lowered. With the sudden step increase in arterial pressure the arterial segment must be filled and the pressure must increase sufficiently to overcome the alveolar pressure which closes the collapsible vessels. When flow begins the collapsible segment is first filled, and then blood flows into the venous side. With increasing starting pressures, the injected volume (Figure 3) becomes less before venous flow appears because the initial volume of the arterial segment is greater.

In a similar fashion, the delay (Figure 4) decreases as the initial starting pressure increases. The delay depends on the pulse-wave velocity and on the time required to open the collapsed vessels where all vessels in the lung are collapsed as in the case at very low initial pulmonary artery pressures. Further, in vessels whose compliance is a function of pressure the pulse-wave velocity will likewise vary with internal pressure. At initial pressures above zero there is a significant difference ($P < 0.02$) between the corresponding delay with high and low venous pressures. The shortest delay seen in these experiments was 0.5 seconds or approximately 0.1 seconds each in the arterial and venous segments. Suppose the mean path length through the lung from artery to capillaries is 15 cm, a distance compatible with measurements on casts of pulmonary vessels (13) and from estimations based on pulmonary artery impedance (8). However, lowering venous pressure by about 10 cm H$_2$O and putting the lung under zone II conditions increases the delay to 0.4 seconds. Assuming the artery is unaltered (delay 0.1 seconds), a delay of 0.3 seconds occurs in 15 cm of vein implying a velocity of only 50 cm/sec. This seems unlikely and would mean that the distensibility of the vein wall had been reduced by a factor of 9 since pulse-wave velocity is proportional to the square root of distensibility. Caro (9) has published figures showing the distensibility of rabbits' pulmonary veins and, although venous distensibility changes with pressure, the alteration is less than ninefold over the range of interest here. The most reasonable conclusion is that in passing from conditions of zones II to III the collapsible vessels themselves have altered in shape or mechanical properties and transmit pressure waves with less delay.

A further feature of interest in these experiments is the smooth and gradual increase in venous pressures and flows. The sharp fea-
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tures of the input functions have become lost, suggesting great attenuation of the higher frequency components of the input pulse.

FLOW TRANSMISSION EXPERIMENTS

The results of the flow transmission experiments are shown in Figures 5-7. The rapid fall in flow transmission with increasing frequency is notable. These measurements refer to the flow pulse transmitted from the entrance to the pulmonary artery to the exit at the left atrial cuff of the preparation where the venous pressure is constant. Two features of these graphs are important: the fact that raising the venous pressure from below to above alveolar pressure has no marked effect on the relation between frequency and transmission, and the observation that forward and reverse perfusion lead to an identical flow transmission at any frequency. In either direction the overall flow transmission falls off rapidly with increasing frequency. The fact that raising the venous pressure has no effect on the damping of the flow pulse suggests that the transmission characteristics of the downstream portion is not altered by this increase in the venous pressure. Such would be the case if the alteration in the damping due to the downstream compliance were offset by an opposite change in the inertance. Increasing the venous pressure may reduce the compliance of the venous compartment and hence increase its impedance to pulsatile flow. However, an increase in cross-sectional area of the segment will reduce the contribution of fluid inertia, and it is suggested that these effects may cancel each other.

The flow transmission in the forward and backward directions are identical (Figure 7) which suggests a symmetry in the transmission properties of the lung in these experiments. Morphologically, gross symmetry exists in the lung. The elastic properties of the smaller vessels (ca. 750 μ in diameter) are similar in the venous and arterial segments but they diverge as one moves away from the capillary bed (9). It is also likely that the collapse point moves to the downstream end of the collapsible vessels in each case so that insofar as these collapsible vessels contribute to the damping this will result in symmetry of transmission properties.

One possible error which must be considered is the effect of pulmonary edema on the transmitting properties of the lung. The mean increase in weight of the lung was 13% during the experiments which lasted 5 to 6 hours. Low-frequency measurements were made early and late in the experiments as well as high-frequency measurements. In some experiments measurements at one particular frequency were repeated after several hours. In no instance was there any suggestion that the duration of the perfusion affected the transmitting properties of the lung. Furthermore, there was no consistent increase in vascular resistance of the horizontal lung preparation during the studies reported. On occasions small amounts of interstitial edema were seen after several hours of perfusion but alveolar edema was never seen. The pattern of transmission in those lungs which gained the most weight was similar to those in which there was little increase in weight.

Under conditions of steady flow and pressure the pulmonary bed shows virtually the same resistance when perfused forward or in reverse (14). However, studies have been reported by Caro and associates (15) in which the transmission of pressure wave was found to be markedly asymmetrical under certain conditions. It was argued that such asymmetry could be the result of asymmetrical disposition of capacitance and inertance on either side of the resistance vessels. Thus the effect was best seen at relatively high intravascular pressures which might be expected to distend the veins and so render them less extensible than the arteries (9). Such an effect was not seen in the flow transmission experiments reported here. There are a number of reasons which might explain this. Caro et al. (15) measured pressure-wave transmission by snaring the arteries or veins, thus closing the system hydraulically. This is in contrast to our experiments in which the line was open, and pressure oscillations at the termination were minimal. In the present...
The results of this measurement have important consequences in terms of the distribution of blood flow in the vertical lung. Under conditions of nonpulsatile perfusing pressure this distribution has been explained in terms of a lung of three zones. The upper boundary between zone I and zone II is given by the level at which pulmonary artery pressure is equal to alveolar pressure. If our findings apply to the vertical lung, this boundary should move up and down the lung with each pulmonary arterial pressure cycle. At a frequency of 1 cps we predict that the maximum height of the no-flow boundary will not be the peak arterial pressure but about 30% of the mean to peak pulmonary artery pressure above the mean. Thus it is not likely that the capillaries or vascular units in the upper zone of the lung will open and close up to the level of peak arterial pressure during each cycle. It also follows that the highest level at which blood flow can be detected in the vertical lung will, in the presence of normal pulmonary artery pressure waves, be somewhat above that of the mean arterial pressure.

Records of the pulmonary capillary flow pulse in the dog (3) and in man (1) have been published. In the absence of simultaneous pulmonary artery flow measurements, it is not possible to determine directly the flow transmission to the capillaries. However, we have made a comparison of the capillary flow curve recorded in man with the N₂O technique and a pulmonary artery flow wave recorded in man with a catheter tip electromagnetic flow transducer (16). Both curves were subjected to Fourier analysis and scaled by assuming equal mean flows. The calculated transmission of flow waves for the first five harmonics were 51, 27, 13, 13, and 14% (fundamental 1.3 cps). It can be seen that transmission falls with frequency though less steeply at higher frequencies. Calculated flow transmissions were greater than the corresponding pressure transmissions (Fig. 9).
between the pulmonary artery and the entrance to the pulmonary capillaries. It should be remembered that in the excised lung experiments pressure transmission was estimated from the end point of total capillary closure, but the capillary flow pulse measurements relate to a lung in which the collapsible vessels are probably fully open (patient horizontal). This might further increase the magnitude of the flow pulse, though the identical transmission found in zones II and III (Fig. 6) suggests that this effect is small.

Recently Wiener and co-workers (17) have published a computer analysis of pressure and flow transmission through a lung model. This was based on measurements made in intact dogs, and it is possible that differences in the preparations explain some of the discrepancies between their results and ours. Our measurements support the idea that the flow pulse is transmitted through to the venous side of the lung but not to the same extent as they computed in their Figure 8 or suggested by their Figure 6. Furthermore, we interpret the small peak flow rate of the "precap" and "postcap" of their Figure 4 and the reduction in amplitude of the first two harmonics at the level of the microcirculation (Figure 8) to mean that the flow pulse is substantially attenuated at this level. This interpretation is not supported by the markedly pulsatile nature of the capillary flow in their Figure 6. More recently Pinkerton (18) has commented upon "the marked attenuation of pressure and flow pulses which were observed . . . after their transmission through the entire pulmonary circulation" in the anesthetized dog.

PULMONARY ARTERIAL IMPEDANCE AND PHASE

In the course of these experiments a number of measurements were made of the arterial impedance in the isolated lung. These measurements may be compared to those of Bergel and Milnor (8) which show the same general pattern down to a frequency of approximately 0.1 cps. The behavior of the impedance at lower frequencies is shown in Figure 10. Here it seems to reach a maximum and then return to a lower value at a frequency of 0.03 cps. This result is at variance with that found by Taylor (19) from a consideration of the input impedance of an assembly of randomly branching elastic tubes. The plot of phase angle of impedance against frequency is, however, more like that predicted and measured by Taylor (19, 20). A number of experiments were carried out on the effects of increasing mean pulmonary artery pressure on vascular impedance. It was seen that for a range of vascular pressures from +10 to +30 cm H2O there was no effect on the shape or value of the impedance curve with frequency. This result is in accord with that reported by Bergel and Milnor (8).

The fall in input impedance below 0.1 cps is hard to explain. It would be expected that the impedance would rise smoothly towards its zero frequency value, the resistance. Any anomalous feature should also be reflected in some alteration of the phase of the impedance which was not seen. If this is a real fall in impedance it might be due either to phasic vascular muscle activity at these low frequencies or to a transition of arterial wall elasticity from a static to a dynamic value. This transition certainly takes place at very low frequencies (21).

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