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# THE RELATION OF PULSATILE PRESSURE AND FLOW IN THE PULMONARY VASCULAR BED

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The flow of blood in the pulmonary artery is pulsatile but it can be analysed, as can systemic arterial flow (McDonald, 1960), by resolving it into two components. These are a steady, or mean, forward flow throughout the cardiac cycle and an oscillatory flow which fluctuates about that mean. The ratio of the mean pressure drop between the pulmonary artery and the left atrium to the mean flow has been extensively studied and is defined as the vascular resistance. By a similar analogy with the electrical terms for alternating current, the relationship between the oscillatory pressure and the oscillatory flow at the origin of the pulmonary trunk may be described in terms of the fluid input impedance.

This latter concept has been developed at length in previous reviews (McDonald & Taylor, 1959; McDonald, 1960). The term input impedance can only be precisely applied to a simple harmonic oscillation at a given frequency. Thus, if the pressure and flow created by the heart are measured, a Fourier, or harmonic, analysis of both curves is necessary. In the series of experiments reported here we have imposed sinusoidal oscillations, generated by a pump, on to a steady flow in perfused rabbit lungs. While more artificial than the study of flow from the heart, the technique has enabled us to make measurements with greater ease and precision over a more varied range of frequencies.

Preliminary communications on these topics have been presented to the Physiological Society; that is, on wave velocity (Caro & McDonald, 1960) and on the input impedance (Bergel, Caro & McDonald, 1960).

## METHODS

A series of twenty-four rabbits (2·3–4·6 kg) were anaesthetized with I.V. pentobarbitone (Nembutal, Abbott Laboratories; 30 mg/kg body wt.) and heparinized (1 ml. Roche Liquemin containing 5000 i.u.). A rib was resected and the thorax widely excised. The animal was bled to death. For perfusion of the pulmonary bed a somewhat angled glass

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cannula was introduced into the pulmonary artery through an incision in the right ventricle; it was held by a ligature just distal to the pulmonary valves. The cannula had a side arm (bore *ca.* 3 mm) which was connected to a manometer. A second cannula was inserted into the left atrium through an incision in the left ventricle and connected to a constant-level reservoir. Normally this was held so that the atrium was at atmospheric pressure. A diagram of the apparatus is shown in Fig. 1. Perfusion was carried out with isotonic saline solution in nearly all experiments. In some, Dextran (Dextraven, Bengers Ltd.) was substituted in an effort to retard the onset of pulmonary oedema, but was abandoned because it was without effect.

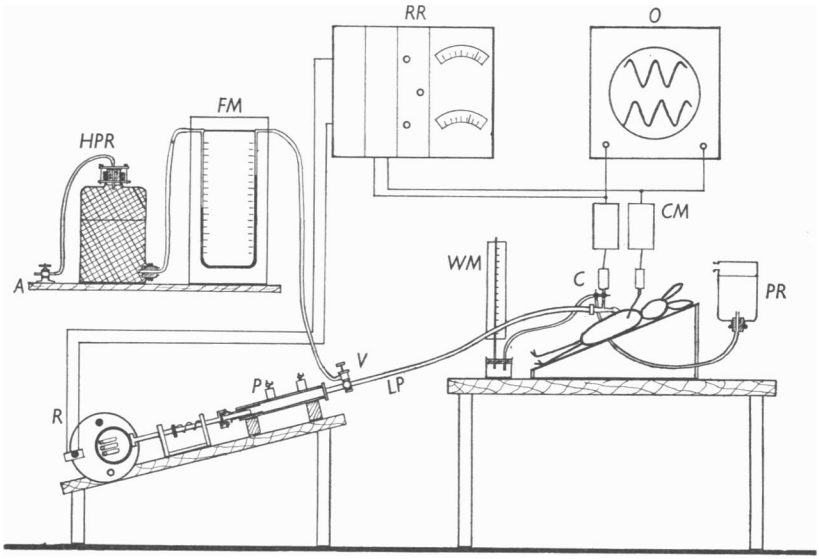


Fig. 1. A diagram of the apparatus used for measuring the input impedance and the wave-velocity in the pulmonary arterial bed of a rabbit. *A* compressed air; *C* cannula; *CM* capacitance manometers; *FM* flowmeter; *HPR* high-pressure perfusion reservoir; *LP* lead pipe; *O* oscilloscope; *P* pump; *PR* perfusion reservoir; *R* reservoir; *RR* resolver relays, controls and meters; *RV* reducing valve; *WM* water manometer.

*Oscillatory flow.* This was generated by a cam-driven sinusoidal pump designed by Taylor (1957*b*, 1959), using the barrel of a 5 ml. all-glass syringe. The output is known to contain not more than 2–3 % of second harmonic. Pump frequencies were held steady by the use of a velodyne motor. With 4:1 reduction gearing the frequency range studied was from 3 to 20 c/s, and a later change enabled us to study the range 1–13 c/s; at frequencies below 3 c/s a flywheel on the motor shaft improved the evenness of running. The stroke volume was varied between 0·08 and 0·72 ml. in various experiments. The linear displacement of the piston was measured by means of a vernier caliper; a calibration curve relating this to the volume displacement was made with a micro-burette, and shown to be a straight line.

In order to ensure that the oscillatory flow created by the pump was identical to that at the pulmonary cannula the two were connected by a 1 m length of lead piping (ext. diam. 12·5 mm; wall thickness 2 mm) with brass adaptors at either end. To reduce the likelihood of air bubbles becoming trapped the apparatus was cleaned with Teepol each day and the animal was arranged on a stand so that the pipe ran up at an angle of about 25° throughout. Any bubbles moved by flushing therefore became visible in the glass pulmonary cannula.

The pump assembly was made of Perspex and contained taps where bubbles could be removed. These precautions are important, for an air bubble in the lead pipe can markedly alter the amplitude and phase of the flow at the cannula relative to that created by the pump. Bubbles close to the manometer will, of course, introduce marked distortion in the pressure recording. At the higher frequencies pressures which are subatmospheric may easily be created at the pump and air sucked in—even when the joints appear to be tight.

*Steady flow.* Steady perfusion was created from a 10 l. glass reservoir enclosed in expanded metal and kept at an absolute pressure of 2 atm from a compressed-air source. This was linked to the lungs through a screw-reducing valve (Bassett Lowke) at a proximal end of the lead piping. The large pressure drop across this valve was designed to ensure that no oscillatory flow passed into the reservoir. Steady flow was measured by recording the pressure drop across a capillary tube (length 22 cm; bore 2 mm) inserted in the high-pressure line. This pressure drop was measured by a mercury U-tube sealed to the two ends. A calibration curve was made of the pressure difference against known volume flows—owing to inlet length effects this relation was not linear. In nearly all experiments flow was adjusted so that the mean pressure in the pulmonary artery was 10 mm Hg.

The fact that no oscillations were observed on the steady-flow meter indicates that none of the pump-generated flow was leaking back into the reservoir. An additional check was made by measuring the input impedance of a length of rubber tube clamped at the far end. This was done first with the valve completely closed and then with the valve open the usual amount but with the reservoir at atmospheric pressure. No difference could be detected under these unfavourable conditions (for the impedance of the tube was higher than that of the pulmonary bed and the impedance of the valve would obviously be raised by subjecting the far side to an extra pressure of 1 atmosphere); hence it was assumed that no appreciable distortion of recorded values occurred.

A second small perfusion reservoir, whose height could be varied, was used for maintaining perfusion of the lungs during setting up and while refilling the high-pressure reservoir. This was connected to the side arm of the perfusion cannula. It was also used for manometer calibration in conjunction with a water manometer which could be switched into the circuit.

*Inflation of the lungs.* In the earlier experiments the lungs were inflated to a measured pressure and this was held constant throughout a run over the whole frequency range of the pump. When it was found that the impedance varied very little with the degree of inflation of the lungs, cyclical positive pressure inflation from a respiratory pump (Palmers Ltd) was substituted for static inflation. The impression was gained that the onset of oedema occurred later when cyclical inflation was used. Lung pressure was measured throughout with a water manometer.

### *Measurement of pressure*

As the rate of the oscillatory flow was determined by the stroke and frequency of the pump, the only variable that needed to be recorded, in order to measure the input impedance, was the pressure at the input into the pulmonary vascular bed. This was measured with a capacitance manometer (Southern Instruments Ltd) attached directly through a three-way metal tap (connecting with the subsidiary perfusion reservoir) to the wide glass side arm of the pulmonary cannula. As the 400 mm Hg head was normally used the resonant frequency of the manometer and connexions was about 400 c/s and the damping was about 0·02. The maximum frequency imposed was 20 c/s, therefore there was no appreciable distortion of either amplitude or phase. The sensitivity of the manometer was increased in all experiments with small stroke volumes by placing an additional amplifier between the manometer and resolver.

The output of the manometer was displayed on a Cossor 1049 two-beam oscilloscope but this was principally used as a monitor to ensure that there was no deviation from a sinusoidal wave form. Measurements of pressure were made through an harmonic resolver (Taylor,

1957*b*, 1959). In principle this depends on two half-wave rectifications of the wave by two two-pole chopper relays activated by two sets of contacts 180° apart on a wheel driven off the cam-shaft of the pump. The two sets of contacts were 90° out of phase with each other and two damped centre-reading meters then read the integral value of the cosine and sine Fourier components of the pressure wave. The method of calculating the modulus and phase of the impedance from these readings is discussed below. The resolver was arranged so that it could be switched to the input of a second manometer (when wave velocity was being measured); other contacts were fixed on the resolver wheel so that any second and third harmonics of the pump frequency could be detected and measured.

*Measurement of wave velocity*

The apparent phase velocity of a pressure wave is defined by the phase shift of a single harmonic component over a known distance. Pressure was measured, as described above, at the origin of the pulmonary artery and simultaneously with an additional capacitance manometer from a site in a peripheral pulmonary artery. By the use of the resolver the phase shift of the wave between these points could be determined.

The method of cannulating a peripheral pulmonary artery was as follows: A measured length of 1·0 or 1·5 mm bore stiff nylon tubing, threaded over a somewhat longer piece of catgut, was passed along the arterial bed until it wedged in a peripheral artery. The tubing was then forced through the lung surface and drawn out to the exterior until its proximal end lay at the point where it had wedged, when it was secured by an exterior ligature. The catgut was then seized at the pulmonary valves and pulled out of the arterial tree; measurement of its length minus the length of the nylon tube then gave the distance between the origin of the pulmonary artery and the proximal tip of the peripheral catheter. In the first two experiments on the wave velocity the distance was measured externally, but these values are not regarded as being so accurate as those measured with the catgut.

A second capacitance manometer was attached to the nylon tubing which was cut off at as short a distance as was feasible. Nevertheless the resonant frequency was less than that of the proximal manometer. With the stiffest membrane (400 mm Hg head) it was *ca.* 300 c/s or higher; with a less stiff membrane (200 mm Hg head) it was 150–180 c/s with a damping *ca.* 0·1 or smaller. This is still over ten times the highest frequency used to measure wave velocity, and so is regarded as tolerable (McDonald, 1960, Ch. 11). The expected phase error at 15 c/s might, however, be as much as 0·1 radians. As the measured phase shift at this frequency was often well over $2\pi$ radians no attempt was made to correct for phase error in the distal manometer.

*Pressure measurement in pulmonary veins*

A length of nylon tube, similar to that used in the arterial measurements, was passed through a small incision in the left atrial appendage into a pulmonary vein and advanced until its tip was seen to lie in the region of the confluence of the lobar veins. Pressure recording was the same as for recording in the peripheral arteries. The phase shift between the origin and the veins was recorded but phase velocities could not be determined, as the length of the path through the capillary bed could not be measured.

*Sources of error*

The most potent source of error in impedance measurements is the presence of air bubbles. The precautions taken to eliminate these have been described. Nevertheless, bubbles did occasionally lodge in the lead pipe. If this occurred during a run there was usually an unexpected change in the phase of the pressure readings, or possibly a drop in the amplitude of the pressure oscillation. Whenever bubbles were discovered the previous readings of the run were discarded.

A second source of error is introduced by the fact that the vascular bed is a non-linear system (Womersley, 1957). This is shown by the fact that a virtually pure sinusoidal input

gives rise to an appreciable amount of second and even third harmonic. This problem has only been investigated very superficially in the present work. The main reason for this was the great complication it would have added to the work. In addition, faults developed in the contacts for higher harmonics on the resolver. In practice we accepted any results in which the output appeared sinusoidal on the monitor CRO. This would tolerate up to 10 % of second harmonic.

The development of higher harmonics is due in large part to the oscillatory dilatation of the vessels and hence to the amplitude of the oscillatory pressure developed. As the oscillatory pressure increases with frequency, with a constant stroke on the pump, these errors are much greater at the upper end of the range. In practice we found very little distortion provided that the amplitude of the oscillatory pressure did not exceed the mean pressure. In early experiments with relatively large stroke volumes the pressure swings reached over 150 % of the mean pressure and the wave form became grossly distorted. The inclusion of these results appears to account for the larger scatter in the input impedance at high frequencies.

The way to obviate this was to use a much smaller stroke volume. This raises the converse difficulty that the pressure oscillations were very small at low frequencies, e.g. if they are $\pm 10$ cm $H_2O$ at 16 c/s then they may only be $\pm 0{\cdot}5$ cm $H_2O$ at 1 c/s. Such pressure oscillations were in fact measured with the aid of the additional amplifier in the manometer output. When measured with a manometer designed to work over the range 0–400 mm Hg this naturally raises a question of accuracy. In fact, both calibration studies and the fact that the reproducibility of results were best where the pressure readings were lowest (usually around 3–4 c/s) suggest that this manometer was recording these small oscillations accurately.

The calibration of the manometers has been discussed above. Taylor (1959) has also noted that the response time of the relays in the resolver, although only 1·9 msec, introduces an appreciable phase error—*ca.* 0·25 radians at 20 c/s. As the absolute phase was not considered in any detail this correction has not been made. In measuring wave velocity these phase errors will be the same for each recording point and hence not appear in the phase difference. The principal source of error here is in measuring the distance between the recording points. The methods used for doing this have been detailed above. Estimates made by the catgut method and external measurement might differ by as much as 1 cm and thus in a 4 cm pathway might cause a 25 % error. In practice the value obtained by the catgut method was accepted. In the two experiments where external measurements only were used the results may well be 15–20 % too high (they were the two highest velocities recorded).

The measured wave velocity will be the sum of the true wave velocity and the mean flow velocity (McDonald & Taylor, 1959). Over the main part of the arterial bed the mean velocity of flow is probably not more than 3–5 % of the wave velocity; in the pulmonary trunk, however, flow velocity may be as much as 30 % of the measured wave velocity and allowance for this would be needed.

The transmission of vibrations from the pump to the proximal manometer was very difficult to eliminate because of the rigid connexions. It was minimized by constructing all supports on the operating table of tubular scaffolding. The vibrations in the input pressure record were of a very much higher frequency than the pump oscillation and so were not recorded by the resolver. The manometer used to measure the peripheral arterial or venous pressure was suspended independently and as it was attached to a flexible catheter was not subject to vibration. Considerable care was taken to see that none of the small venous pressure oscillations were due to this cause by showing that not only were they not in phase with the input but that the phase lag increased progressively with frequency. Hence it was due to a transmission time.

*Calculation of results*

The units for impedance in this study have been dyn.sec/cm$^5$. That is the pressure (dyn/cm$^2$) divided by the volume flow (cm$^3$/sec). This unit has been adopted in spite of the reasons advanced by Taylor (1959) and McDonald (1960) for using the ratio of pressure to average linear velocity of flow—making the unit dyn.sec/cm$^3$. This has been done because of the difficulty of measuring the true diameter of the pulmonary trunk with a glass cannula in it, in order to derive the linear velocity from the measured volume flux. Cotton (1960) has also argued that the volume flux is a better analogue of electrical current than is velocity. Either system is internally consistent.

The rate of volume flow ($Q$) is determined by the amplitude of the pump oscillation ($V$) and the frequency ($f$ c/s). The amplitude means the maximum deviation from the mean position, i.e. the pump puts out $+V$ ml. on forward stroke and withdraws to $-V$ ml. Under these circumstances

$$|Q| = 2\pi f \times V, \tag{1}$$

where $|Q|$ is the amplitude or modulus of the volume flux. As the measured stroke volume ($V_s$) is from maximum forward to maximum back, i.e. $2V$, for calculation

$$|Q| = \pi . f . V_s. \tag{2}$$

The pressure records made on the resolver are the integrated values of the half wave-rectified cosine and sine components. The values actually recorded on meters, $A'$ and $B'$, are related to the true amplitudes by the following equations (Taylor, 1957*b*):

$$A' = 2/\pi(A_1 + \tfrac{1}{3}A_3 + \tfrac{1}{5}A_5 \ldots), \tag{3}$$

$$B' = 2/\pi(B_1 + \tfrac{1}{3}B_3 + \tfrac{1}{5}B_5 \ldots), \tag{4}$$

where $A_1$, $A_3$ and $A_5$ are the Fourier cosine components of the first, third and fifth harmonics and $B_1$, etc., the corresponding sine terms. As we are dealing with a single harmonic oscillation the terms for the third and higher odd-numbered harmonics are negligible. Therefore to get the correct value of the Fourier terms $A_1$ and $B_1$ the meter readings $A'$ and $B'$ have to be multiplied by $\frac{1}{2}\pi$. They are also, of course, multiplied by a manometer calibration term to convert to actual pressure units.

Given the cosine and sine terms of the pressure this may be handled as a complex number but it is easier to convert into modulus and phase form (McDonald, 1960, App. 1). The amplitude, or modulus $M$ is given by Pythagoras's theorem

$$M = (A^2 + B^2)^{\frac{1}{2}}, \tag{5}$$

and the phase $\phi$ by

$$\phi = \tan^{-1} B/A. \tag{6}$$

The pressure, $P$, then is $M\cos(\omega t - \phi)$. It is necessary to determine the correct quadrant for $\phi$ by the signs of the cosine and sine terms, e.g. if both $A$ and $B$ are positive $\phi$ lies between 0° and 90° (0 and $\frac{1}{2}\pi$ radians); if $A$ is negative and $B$ positive it lies between 90° and 180° and the angle is $180° - \phi$. As the phase of the flow is fixed at 0° by the resolver setting, the phase of the pressure is also the phase of the input impedance.

As the modulus of the flow is simply determined by eqn. 2 the modulus of the impedance $|Z|$ is $|P|/|Q|$, or from eqns. 5 and 2

$$|Z| = M/(\pi . f . V_s). \tag{7}$$

To measure the wave velocity we have a known distance between measuring points, $\Delta x$. The modulus and phase are recorded at each point as described above. Then the difference in phase between the two points, $\Delta\phi$, represents the time (expressed as a fraction of a cycle) for the wave to travel between the two points. The apparent phase velocity $c'$ averaged over this interval then will be

$$c' = \frac{2\pi . f . \Delta x}{\Delta\phi} \tag{8}$$

when $\Delta\phi$ is measured in radians. If it is measured in degrees then 360 should be substituted for $2\pi$.

*The interpretation of these results* is made on precisely the same basis as that of oscillatory flow in the systemic arterial system (McDonald, 1960, Ch. 10). In a system of branching tubes the occurrence of wave reflexion is inevitable. In such a situation the input impedance will not only be determined by the calibre of the vessels and their elastic properties but also on the length of the system from the origin to the main reflecting sites. The unit of length that we are concerned with is the wave-length, i.e. the wave velocity divided by the frequency in c/s. The type of reflexion that we meet in a system subdividing into smaller branches is that known as 'closed-end', or 'in-phase'. This is the type of reflexion that occurs when the impedance increases across a junction (when completely closed, of course, the terminal impedance becomes infinite). As it is known that fluid resistance increases with subdivision of the vessels, so also does fluid impedance increase, although this effect is small in the large vessels and only becomes marked in small arteries and arterioles.

Thus, close to a 'closed' end the incident and reflected pressure waves are nearly in phase and sum together. Flow, on the other hand, is small so that the impedance is high. At a quarter-wave-length distance the incident and reflected waves are 180° out of phase and tend to cancel each other out, so that pressure oscillation is at a minimum while flow is at a maximum. The input impedance is, therefore, at a minimum. Maxima and minima thereafter succeed each other at quarter-wave-length intervals. With a system of fixed anatomical length the wave-length is dependent on the frequency of the oscillation, so that the first minimum to be found can be used, if the wave velocity is known, to define the distance at which the main reflexions are occurring. Because the waves are damped in travel the nearer we get to the origin the larger are the incident waves and the smaller the reflected waves, so that successive cancellation and summation of the waves produce smaller fluctuations.

A demonstration of these effects was made on a simple model consisting of two rubber tubes. The proximal tube connected to the pump was 120 cm long and had a bore of 0·6 cm; its wave velocity was *ca.* 1440 cm/sec. Into its end was fitted a thicker-walled tube of 0·35 cm bore which had a wave velocity of 2230 cm/sec. The distal tube was completely closed at its far end but was 7·5 m long to minimize the effect of reflexions from that end. The modulus of the input impedance measured in it is illustrated in Fig. 6. It can be seen from the values given that the proximal tube was one wave-length long at *ca.* 12 c/s and at 3 c/s there is a minimum in the amplitude of the impedance representing the quarter wave-length condition. There follows a maximum at 6 c/s (half wave-length) and another minimum at 9 c/s (three-quarter wave-length). This is very similar to the behaviour of the input impedance in the pulmonary bed, except that there is a much bigger variation between the maximum and the minima in the model because it has a much higher reflexion coefficient (see p. 439).

Similar considerations affect the phase shift of the wave and hence the apparent phase velocity. This is too complex to consider here, but we may briefly distinguish the true phase velocity, $c_0$, in a very long elastic tube which is solely determined by the elastic properties of the wall and assumes that the fluid has no viscosity. When filled with a viscous fluid we have the complex wave velocity, $c$, but except for very small vessels this may be taken as close to $c_0$. Finally, in the presence of reflexions we have the apparent phase velocity, $c'$, which is in excess of $c$ and $c_0$ in regions where the impedance is high and is below the true value where the impedance is low. This is only true for the 'spot' velocity. If the velocity is measured over a long interval the variations in apparent phase velocity are averaged out and a value approximating to the true velocity is found. For this reason the values of velocity for frequencies representing approximately a half wave-length and longer are chosen as representative of the true velocity, $c_0$. This averaging of phase velocities over the whole system was verified (but not illustrated) in the model described above. The phase velocity of these higher frequencies also approximates most closely to the 'foot-to-foot' velocity of a compound wave, such as that of the natural pulse wave (McDonald & Taylor, 1959).

## RESULTS

The input impedance was measured in 52 experimental runs on 24 animals. In addition, in 15 runs on 10 of these animals a second pressure record was made in a peripheral artery from which the wave velocity could be measured. Although, chronologically, these wave-velocity measurements were among the later experiments it is simpler and more logical to consider these results first.

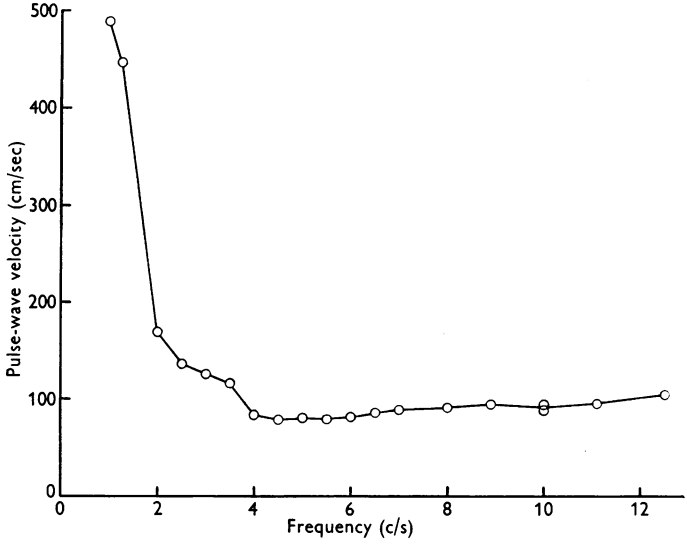


**Fig. 2.** The variation in apparent phase velocity in the pulmonary arterial tree with frequency in a single experiment. In this experiment the distal catheter was in the right lower lobe and its tip was 3·7 cm from the pulmonary valves.

### *Wave velocity in the pulmonary arterial bed*

The apparent phase velocity derived from a single experiment is shown in Fig. 2. It will be seen that it has a value of 500 cm/sec at 1 c/s, but this value falls steeply with increasing frequency until it reaches a value of 85 cm/sec at 4 c/s and thereafter remains relatively stable. There is a slight rise above 10 c/s and the velocity at 12·5 c/s is 105 cm/sec. In this experiment the catheter was in the right lower lobe and the interval between it and the pulmonary valves was 3·7 cm.

As explained under 'Interpretation of results' in Methods (p. 432), the high apparent phase velocities at low frequencies are due to the presence of reflexions occurring at less than one quarter wave-length distance and so give no measure of the true wave velocity. At higher frequencies, however, the interval is a considerable fraction of a wave-length and the effect of

reflexions is averaged out. The flat part of the curve is, therefore, taken as the characteristic wave velocity of the system over the interval measured.

A summary of all results of the 15 experiments is shown in Fig. 3, which displays the average value together with the S.E. of the mean. Results are also averaged for mean values of the frequency, e.g. the values at 4 c/s are for all readings in the range 3·6–4·5 c/s. The intervals over which these were measured ranged from 3·2 cm (when the catheter was in the middle lobe) to 7·0 cm (when in the tips of the upper or lower lobes). The mean value of

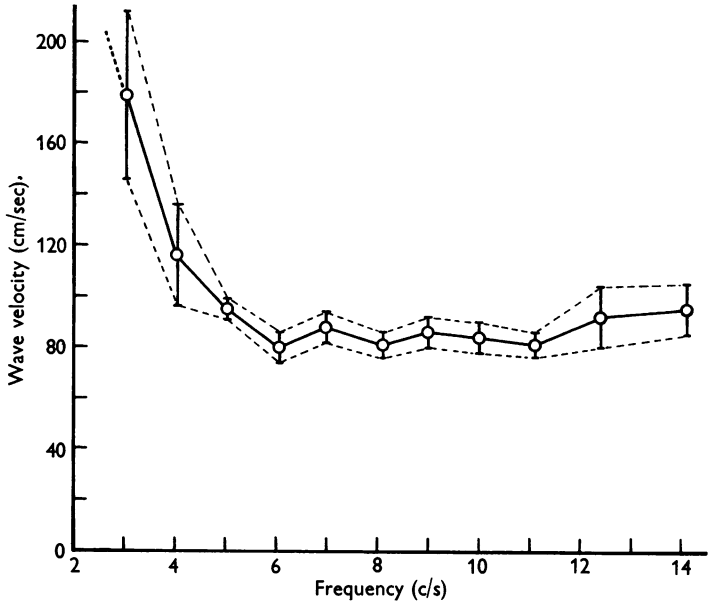


Fig. 3. Graph summarizing all measurements of wave velocity in 15 experiments. The mean values are shown by the open circles and full line; vertical bars show S.E. of mean. Each point averages all values at a mean frequency over a range of 1 c/s, e.g. the point at 4 c/s includes all results between 3·6 and 4·5 c/s. The mean interval between measuring points was 4·3 cm (range 3·2–7·0 cm). The mean wave velocity between 6·0 and 11·1 c/s was 83·3 cm/sec ± 5·7 S.E.

the interval was 4·3 cm. In the whole series it will be seen that a relatively stable value is not reached until 6 c/s and at low frequencies the apparent velocity is much higher. The mean value for the frequency range 6–11·1 c/s is 83·3 cm/sec ± 5·7 S.E. From 11·1 to 14 c/s the mean value is 93·3 cm/sec ± 11·1 S.E.

Velocities measured in arteries in the upper and lower lobes tended to be higher than those measured in arteries in the middle lobes, where the path length was shorter. In the latter, stable values were not reached until a rather higher frequency and this is attributed to the fact that a short

interval did not exceed a quarter wave-length until these higher frequencies were reached. This is the main reason for the large scatter at 3 and 4 c/s in Fig. 3 for the values of all experiments over widely differing intervals.

The lower wave velocity observed in the artery to the middle lobe is probably due to the wave velocity being lowest in the main pulmonary artery and becoming progressively higher as the arteries subdivide (see below). As the wave velocity measured over the whole interval is a weighted mean of the velocities in each successive segment, the relatively shorter second- and third-order arteries to the middle lobe will not increase the mean velocity as much as the longer small arteries in the upper and lower lobes.

Some support for this suggestion was given by measurements of the phase velocity made after the catheter had been pulled out for successive short intervals from the pulmonary valves. No great weight can be attached to these results because the accuracy of measuring small intervals in an easily extensible organ such as the lung is not great, and the technique was only used in one animal. The values obtained in this experiment were as follows: with interval 2 cm, i.e. within the pulmonary trunk, mean velocity 55·1 cm/sec; interval 3·2 cm, velocity 61·6 cm/sec; interval 4·2 cm, velocity 66·3 cm/sec; interval 5·5 cm, velocity 79·9 cm/sec. Velocities are mean values for the frequency range from 5 to 9 c/s. The volume flow was 3·3 ml./sec and as the main pulmonary artery was *ca.* 0·6 cm in diameter the linear velocity of the steady flow was *ca.* 12 cm/sec in this artery. From the findings of Patel, Schilder & Mallos (1960) in the dog it would appear that the cross-sectional area of the right and left pulmonary arteries together may be less than that of the trunk. Thus the correction for steady-flow velocity will be at least as high in these vessels. This suggests that the true value (see 'Sources of error' in Methods, p. 430) for wave velocity in the main pulmonary artery is about 45 cm/sec, or almost half that for the whole bed. As even over the full interval we were still only recording in arteries of 0·1–0·15 cm diameter, the flow-velocity correction may still be appreciable and all values recorded may be a little higher than the true wave velocity.

In one experiment the effect on wave velocity of altering the mean pulmonary artery pressure was tested. At 10 mm Hg the mean value of eleven observations in the frequency range 6–10 c/s was 87·6 cm/sec; with the arterial pressure at 20 mm Hg the mean value of the velocity (five observations) was 85·7 cm/sec. This suggested that pulmonary wave velocity did not vary significantly with mean pressure over the limits of what we presumed was the normal range, so that all other observations were made at 10 mm Hg.

The average wave velocity into all parts of the pulmonary arterial bed

may thus be taken as *ca.* 80 cm/sec. This enables us to predict the wave-lengths of oscillations that are imposed on it. Thus at 4 c/s the wave-length is 20 cm and a quarter wave-length will be 5 cm; at 8 c/s these distances will be halved, and so on proportionately. These dimensions are important in interpreting the behaviour of the input impedance.

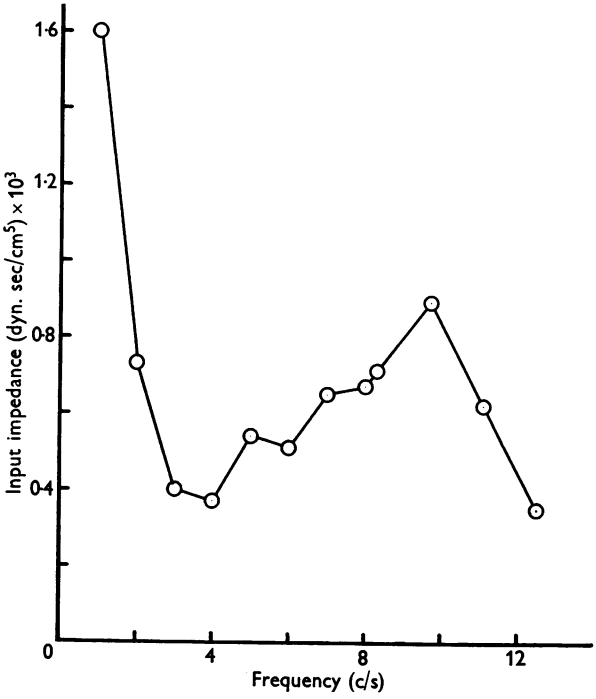


Fig. 4. The values of the modulus of the input impedance in a representative experiment, showing the variation with frequency. Rabbit, 3·1 kg.

*The input impedance of the pulmonary bed*

The results of a single experiment are illustrated in Fig. 4. It can be seen that the highest value of the modulus of the impedance is at 1 c/s, when it is $1{\cdot}6 \times 10^3$ dyn.sec/cm$^5$; with increasing frequency it falls to a minimum value, at 4 c/s, of 0·37 (the true minimum is probably slightly below 4 c/s). Thereafter it rises to another peak value, at 9·5 c/s, of 0·89 and then decreases to 0·35 at 12·5 c/s. From other experiments it can be predicted that this is close to another minimum and that the values would rise with increasing frequency. If the value at 12·5 c/s was a minimum, it can be seen that the peak value at 9·5 occurred at rather more than twice the frequency of the first minimum and that the second minimum fell at a little over three times this frequency. This suggested that the minima were

falling at frequencies representing the quarter and three-quarter wave-length situation and the maximum at a half wave-length for the system.

A graph summarizing the mean results of 52 experimental runs is shown in Fig. 5, together with the S.E. of the means. As in Fig. 3, the values have been averaged for frequency, i.e. the value for 6 c/s includes all results between 5·6 and 6·5 c/s. Considering that there has been no weighting for differences in size of animals or variations in resistance the scatter up to

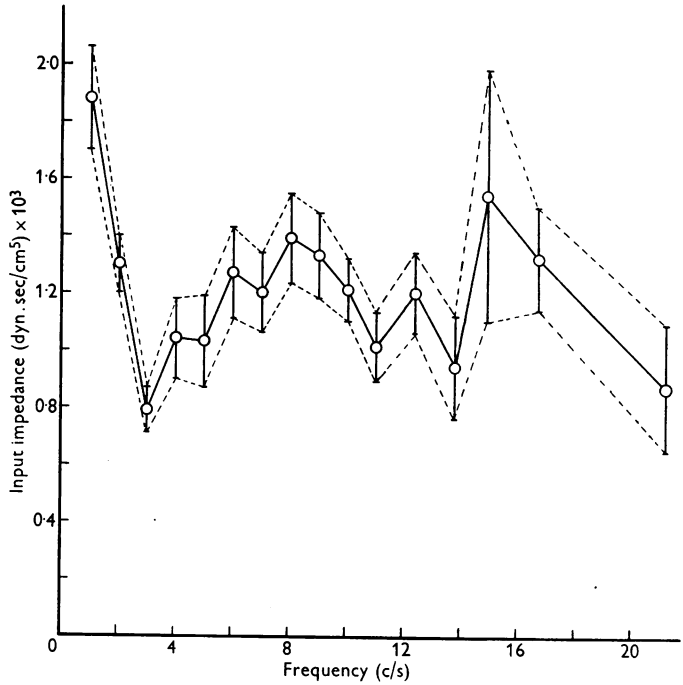


Fig. 5. Graph summarizing all measurements of pulmonary input impedance in 52 experiments on 24 animals. As in Fig. 3 the results have been grouped according to frequency at 1 c/s intervals and the mean frequency taken for each group. Average values of the input impedance are given by the open circles and solid line; vertical bars show the S.E. of mean.

15 c/s is remarkably small. The larger scatter in the higher frequencies may be partly attributed to the fact that fewer experiments were done in this frequency range, and they include a number of results that later standards would have rejected because the oscillatory pressure exceeded the mean pressure (see 'Sources of error' in Methods, p. 430).

From the whole set a similar pattern to that seen in Fig. 4 emerges. The impedance is high at low frequencies and falls to a minimum at 3 c/s. It rises again to a maximum at 8 c/s, falls to another minimum at 13·8 c/s, rises to another maximum at 15 c/s and thereafter decreases up to 21 c/s.

The precise values for the main points of possible interest are given in Table 1.

If the value close to 3 c/s is a minimum in input impedance such as occurs at a quarter wave-length in a system with reflexions then we can derive an estimate of the average length of the system. Taking the average wave velocity of 83·3 cm/sec then a quarter wave-length at this frequency is 6·96 cm. This may be compared with the value of 6 cm that Engelberg & DuBois (1959) took as the average length of the arterial bed in the rabbit lung. If this were a simple system (e.g. Fig. 7) there would be a maximum

TABLE 1. Some mean values of input impedance measured in 52 experiments on perfused rabbit lungs

| Mean frequency (c/s) | Impedance (dyn.sec. $cm^{-5} \times 10^3$) | S.E. of mean | Comment |
|---|---|---|---|
| 1·14 | 1·88 | 0·18 | — |
| 2·99 | 0·79 | 0·08 | minimum |
| 6·05 | 1·27 | 0·16 | ? subsidiary maximum |
| 8·19 | 1·39 | 0·16 | maximum |
| 11·11 | 1·01 | 0·12 | ? subsidiary minimum |
| 12·39 | 1·20 | 0·14 | ? subsidiary maximum |
| 13·81 | 0·94 | 0·18 | minimum |
| 14·87 | 1·54 | 0·44 | maximum |

representing a half wave-length at 6 c/s. Reference to Fig. 5 and Table 1 shows that there is a hint of such a peak here, but that it is overshadowed by the maximum occurring at 8·2 c/s. The three-quarter wave-length frequency would then be 9 c/s, where a minimum should occur (the nearest suggestion of one is at 11·1 c/s) and the whole wave-length maximum at 12 c/s (possibly suggested by the fluctuation at 12·4 c/s).

The maximum in the middle range is at 8·2 c/s, and if this is a half-wave-length maximum then the minimum at 13·8 c/s would fit fairly well as its corresponding three-quarter-wave-length value. This would correspond to a system length of 5·1 cm. This is also quite a reasonable figure of distance to the arterioles compared with the mean distance of 4·3 cm to arteries of 1·0–1·5 mm diameter recorded in the experiments on wave velocity. It is possible that reflexions from the closer arteriolar beds become dominant at the higher frequencies because, owing to the shorter length of path, damping of the waves in travel is less marked. The fact that the wave velocity tends to rise appreciably with frequencies over 10 c/s would also cause the wave-length to increase and so prevent the maxima and minima falling at exact multiples of the quarter-wave-length frequency.

The main conclusion is that the fluctuations of input impedance are due to reflexions. Considering the great range of distances to the peripheral arterial bed from the pulmonary trunk that is present in the lung it is more surprising that a definite pattern can be detected rather than the fact that

maxima and minima do not occur with the exact regularity of a simple model. The type of oscillations seen in such a model is shown in Fig. 6. It consisted of a length of rubber tube terminating in another tube of smaller bore and stiffer walls. The reflexion coefficient was estimated by means of Womersley's (1957) formula and estimated at 0·65. This is much higher than our results suggest for the pulmonary bed (rough calculations indicate a figure of *ca.* 0·25) so that the fluctuations in the amplitude of the impedance in the model are far greater than in the pulmonary bed. The similarities between the two systems are, however, quite easy to recognize.

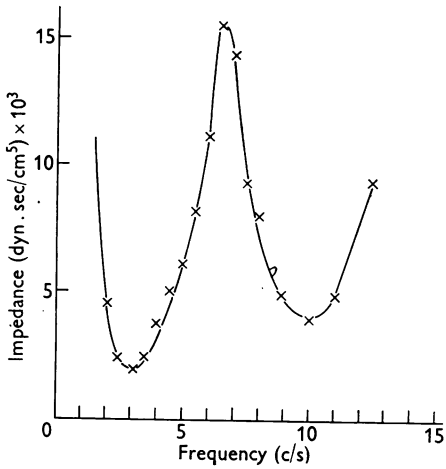


Fig. 6. The behaviour of the input impedance in a simple model with a single reflecting site. This shows marked similarities to the pattern seen in the pulmonary vascular bed (e.g. Fig. 4).

We do not present detailed results of measurements of the phase of the complex fluid impedance because a considerable diversity of pattern was seen in different experiments, and it does not appear that any useful information can be gained from its analysis. In a model with a single reflecting site there is a phase lead at low frequencies which falls to a value close to zero at the quarter-wave-length frequency, i.e. at the minimum of impedance amplitude. Following this the phase lags progressively to a point midway between the minimum and maximum, and returns to zero at the frequency of the next amplitude maximum. It then reverses once more and becomes a phase lead, reverses again at the three-quarter-wave-length frequency, and so on. Minor oscillations of this sort were observed in many experiments but in others they were quite overshadowed by a progressive reduction of phase lead as the frequency increased. Interpretation of results is further vitiated by the fact that, as measurement of

the phase was not originally a matter of interest, insufficient care was taken to ensure that the signs of the Fourier components were accurately recorded.

*Relation of impedance to vascular resistance*

In a previous communication Bergel *et al.* (1960) said that there appeared to be no obvious connexion between the impedance values and those for the pulmonary vascular resistance. This was based on a superficial comparison of the scatter of these two measurements. The measured range of resistance was from 2·0 to 22·0 $\times 10^3$ dyn.sec/cm$^5$. As the mean pulmonary artery pressure was almost always 10 mm Hg this represented flow rates of *ca.* 5–0·5 ml./sec. Thus the lowest resistance values in this range alone may be taken as roughly corresponding to normal values, i.e. a flow of 300 ml./min (Dittmer & Grebe, 1959). All animals were used until there was marked pulmonary oedema when the flow became too small to measure, with any precision, on the meter in use. The resistance was then classed as 'very high'. In the first experiment in which Dextran was used the resistance immediately became immeasurably high, apparently due to embolization of small vessels with agglutinated erythrocytes, as tests on microscope slides showed marked clumping of red cells with the Dextran in use. More normal values of resistance were obtained in later experiments when the vascular bed was perfused with saline solution for several minutes before starting the Dextran infusion.

TABLE 2. Relationship between the amplitude of the input impedance and the vascular resistance of the pulmonary vascular bed at varying frequencies

| Frequency (c/s) | No. of expts. | Regression line ($\|Z\| = a+b(R)$) | Correlation coefficient ($r$) |
|---|---|---|---|
| 1·0 | 8 | 0·483+0·137$R$ | 0·97 |
| 2·0 | 12 | 0·430+0·060$R$ | 0·94 |
| 3·0 | 12 | 0·541+0·018$R$ | 0·59 |
| 4·0 | 12 | 0·652+0·0001$R$ | 0·001 |

The interesting value of impedance to compare with the vascular resistance would be the impedance of the 'resistance' vessels. In so far as we consider the bed as having a partially closed termination, the impedance of these vessels is a terminal impedance. The closest approximation we can make to measuring this is by studying it at low frequencies when the length of the system, in terms of wave-length, is shortest. The correlation between the input impedance and the resistance in all experiments in which measurements were made at 2 c/s or below, and in which the resistance was recordable, is given in Table 2.

It can be seen that a satisfactory degree of fit for the regression lines is only found at 1 and 2 c/s. It will be seen, however, that the slopes show

that the rise in impedance is much less than the corresponding rise in resistance. This implies that the behaviour of some of the resistance vessels does not influence the terminal impedance significantly.

At 1·0 c/s the length of the system was about 1/16 of a wave-length as the impedance minimum fell at 4 c/s (1/4 wave-length) in this selected series. The highest ratio of impedance to resistance at this frequency was 1·13 : 2·13 or 53 %. The average value for the 5 experiments where the resistance was reasonable—below $4 \times 10^3$ dyn.sec.cm$^{-5}$—was 0·98 : 3·23 or 30 %. The ratio at the highest resistance recorded was 3·7 : 22·0 or 17 %. This illustrates the observation that impedance does not vary directly with resistance. Even so, all these values are greater than the corresponding ratio in the femoral bed, which is about 10 % (McDonald, 1960, Ch. 10). As the impedance increases very rapidly with reduction of frequency below 3 c/s it may well be that a value of 53 % at 1 c/s, for example, may represent a true terminal impedance that is quite close to the vascular resistance.

It should be emphasized that most of the changes in resistance we recorded were due to the gradual onset of pulmonary oedema, so that no physiological inferences can be made from these results. The preparation we used was not suitable for the investigation of vasomotor activity. In one experiment we did infuse noradrenaline (a total of 0·5 mg in a total flow in the run of 50 ml.) which caused a rise in the input impedance at 2·8 c/s from 0·6 to $0{\cdot}97 \times 10^3$ dyn.sec/cm$^5$. The resistance, unfortunately, was very high and could not be recorded. No effect on imput impedance could be detected with infusions of acetylcholine.

The virtually complete lack of any relation between impedance and resistance at 3 and 4 c/s emphasizes that in a system with partial reflexions and viscous damping there is no simple relation between its input impedance and the behaviour of its termination. In a simple system an increase in the terminal impedance will cause a reduction in the impedance at the quarter-wave-length minimum so that one would expect a negative slope to a regression line. However, as Table 2 shows, no significant regression line can be drawn from the values we have recorded in the pulmonary bed.

### *Amplitude of pressure in peripheral pulmonary arteries*

In the course of measuring the wave velocity in the arterial bed records were naturally also made of the amplitude of the pressure. Averaged over the whole frequency range the amplitude of oscillation was slightly higher at the periphery than at the origin, but the difference was not statistically significant. Around the frequency of the impedance minimum, however, the amplitude at the origin falls and becomes appreciably less than that in

the periphery (Fig. 7). For this reason we should expect a certain amount of peaking of the fundamental component of the naturally formed pulse wave. The 'peaking' observed is not, however, as marked as in the systemic system. It may be noted that the peripheral pressure was measured with the 'end' of the catheter and would, therefore, tend to be higher than a lateral pressure record because of the additional kinetic energy. With the low flow velocities that we may expect in such small vessels it is unlikely that the kinetic correction would be measurable.

*Oscillatory pressure in pulmonary veins*

Pulsatile venous pressure was recorded in lobar veins through a nylon catheter passed retrogradely from the left atrium. Over the frequency range 3–5 c/s the mean of 7 experiments was 0·62 cm $H_2O \pm 0{\cdot}12$ S.E. In the same experiments the average amplitude at the cannula was 2·52 cm $H_2O \pm 0{\cdot}32$ S.E. Thus the venous oscillation was rather less than 25% of that at the pulmonary valves. However, in many of these experiments resistance was considerably above what might be regarded as normal. It was found that the venous oscillatory pressure varied inversely with the vascular resistance, so that the normal ratio of venous to arterial pressure must be considerably greater than this.

A more interesting estimate of the amount of the arterial oscillation that passed through the capillary bed was given by comparing the oscillation in the peripheral arteries with that in the veins. The results of 6 experiments are shown graphically in Fig. 7, where the pressure oscillations at the origin of the pulmonary artery, in a peripheral artery and in a lobar vein, are compared. The average values at these three sites at the frequency of the impedance minimum were 1·56, 2·05 and 0·42 cm $H_2O$ respectively. In the case with the lowest resistance ($2{\cdot}2 \times 10^3$) the values were 1·55, 2·0 and 0·90 cm $H_2O$. The pulsation in the vein was then 58% of that at the origin and 45% of that in the peripheral arterial bed.

In some early experiments the flow from the atrial cannula appeared to be pulsatile at low frequencies but we thought it probable that this was due to vibration from the pump. No pressure oscillation was detected with the catheter tip in the atrium.

As was stated in Methods, no accurate measurements of wave velocity were possible from artery to vein as the length of the pathway was unknown. When we assumed a distance of double the interval between the origin and a peripheral artery the value obtained was about half that recorded in the arteries, i.e. 30–40 cm/sec. While a reduction of wave velocity during passage through the very narrow resistance vessels is to be expected (McDonald, 1960, Ch. 9) this evidence merely hints that this viscous effect is operating. The presence of this factor is supported by the

fact that the velocity tended to be lowest at low frequencies and climbed to a steady value with increase of frequency. It thus contrasted with the behaviour of the arterial velocity, which was very high at low frequencies (Figs. 3 and 4). The artery–vein velocity pattern is similar to that in a tube where the viscous effects are marked and reflexion is negligible. The main practical value of calculating these approximate velocities to the venous bed was to ensure that the phase shifts observed were consistent with a definite transmission time and that the apparent venous pressure recorded could not have been due to vibrations of the manometer transmitted from the pump or the direct effect of pulsations in contiguous arteries.

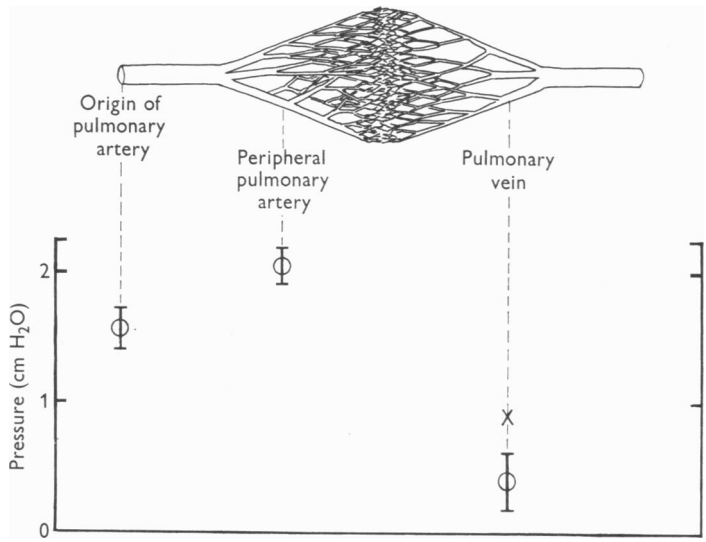


**Fig. 7. A comparison of the amplitude of the oscillatory pressure in various regions of the pulmonary vascular tree. The results are for 6 experiments in which this was measured at three sites. Results are shown for the frequency of the first impedance minimum (3–4 c/s), i.e. when the oscillatory pressure at the origin was least in comparison with that in the peripheral arteries. At double this frequency when impedance is at a maximum the oscillatory pressure at the origin is rather greater than that in the periphery. Mean values are shown by circles with a vertical bar to show the S.E. of mean. The venous pressure oscillation in the case with the lowest resistance ($2{\cdot}2 \times 10^3$ dyn.sec/$cm_5$) is shown by a cross—the corresponding arterial pressures fell on the mean values and are not shown.**

## DISCUSSION

When studying the behaviour of pulsatile flow in a vascular bed it is insufficient to record the form of the pressure wave, although this is the easiest measurement to make. Even if the flow pattern is also measured, or derived indirectly, comparisons of the wave forms of pressure and flow

can only be made descriptively (e.g. Baxter & Pearce, 1951, described waves as 'rounded', 'round-triangular' or 'triangular') unless some form of mathematical analysis is used to extract quantitative data. The method we use is that of describing a pulse by a Fourier series, i.e. a set of harmonic oscillatory components at frequencies which are integral multiples of the pulse frequency. At a single frequency the input fluid impedance is then the ratio of the oscillatory pressure at the origin to the oscillatory flow. This method has been used fruitfully in the systemic arterial bed (McDonald & Taylor, 1959; McDonald, 1960). A greater range of frequencies may be studied by the technique used in the present work where a sinusoidal flow from a pump is imposed on a steady flow through perfused lung. The input impedance of such a system is greatly influenced by the wave-length of the oscillation and this is determined by the frequency (which is known) and the wave velocity. It was therefore necessary to determine this velocity.

*Pulse-wave velocity.* In contrast to the literature on the systemic arteries there appear to have been very few attempts to measure the wave velocity in pulmonary arteries. The value we obtained was 83·3 cm/sec $\pm$ 5·7 S.E. At first sight this appears to be very low, being only about 1/5 of the value of *ca.* 400 cm/sec in the aorta. Intuitively, however, it does not seem unreasonable in view of the much greater distensibility of the pulmonary compared with systemic arteries.

The only figure this can be compared with, in the rabbit, is that of 200 cm/sec derived by Engelberg & DuBois (1959). They assessed the velocity, $c_0$, by means of the modification of the classical Moens–Korteweg formula introduced into this country by Bramwell & Hill (1922), but proposed earlier by Otto Frank, which is:

$$c_0 = \sqrt{\left(\frac{V.\delta P}{P.\delta V}\right)}. \tag{9}$$

The volume distensibility or elastance, $\delta P/\delta V$, was measured for the whole arterial bed after the capillaries had been blocked by emboli (it was quoted as its reciprocal, the compliance). The total volume, $V$, however, had to be derived by the assumption of a simple geometrical relation for the change of volume at each subdivision of the arteries. Furthermore, the equation used is for a single thin-walled elastic cylindrical tube, filled with a non-viscous liquid. It is doubtful whether it can be regarded as more than an approximate solution for a rapidly subdividing system of tubes filled with a viscous liquid. The comparison of their derived value and our measured value implies that there are marked errors (making $V$/compliance some four to five times too large) in their figures.

The reported estimates of wave velocity in the pulmonary bed in other species are also higher than those we recorded. Patel *et al.* (1960) measured

the ratio of the changes in radius and pressure ($\delta R/\delta P$) in the pulmonary artery of the dog and the velocity derived from these we calculate as about 250 cm/sec for their average data. The mean pressure was much higher (22 cm $H_2O$) for their experiments which may account for some of this difference, and there is also a species difference. On the other hand, our results imply that the velocity for the whole bed would be considerably higher than that for the pulmonary trunk. A much earlier measurement by Johnson, Hamilton, Katz & Weinstein (1937) gave a value of 400 cm/sec. The implications of these values are discussed below in regard to speculation on the behaviour of the input impedance.

In man, Fleischner, Romano & Luisada (1948) estimated the main pulmonary arterial wave velocity as 200 cm/sec and the more peripheral arteries to have a velocity of 275 cm/sec. These measurements were made by kymographic techniques which measure delay in dilatation between the pulmonary knob and the right hilar shadow, and between the right hilar shadow and the visible base of the right lung. In view of the possible errors in measuring the precise dimensions of X-ray shadows these figures must be regarded with caution. As recording in the pulmonary artery with a double-lumen catheter is standard practice in many clinics, the taking of suitable pressure records, a known distance apart, should make precise measurements of foot-to-foot wave velocity a relatively easy matter—although none appear to have been made as yet.

Following Engelberg & DuBois (1959), Milnor, Jose & McGaff (1960) have endeavoured to measure the distensibility of the total pulmonary vascular bed in terms of compliance. Their estimation of true pulmonary vascular volume in man is very elegant but it is difficult to see how they can estimate compliance on a single measurement of volume against pressure. Distensibility must be defined as a ratio of the change of volume to the change of pressure. For the lung, which is a closed bag (albeit complicated geometrically) this is a useful measurement. For the vascular bed, which is also geometrically complex but in addition has a large leak at the far end, it would seem very difficult to separate distensibility from flow. In addition, the only values that Milnor *et al.* (1960) can derive are for the whole pulmonary vascular bed, whereas by far the greatest part of the pulsatile flow is on the arterial side of the capillaries. It would appear to us that the elastic properties of the pulmonary arterial bed would be much more simply characterized by measurements of arterial wave velocity. The techniques of studying lung mechanics are not easily applied to those of a vascular bed where both through flow and change of volume are taking place.

*Input impedance*

In the presentation of Results it has been seen that the amplitude of the input impedance is highest at very low frequencies and falls to a minimum value at 3–4 c/s. Thereafter it rises to a maximum value of about 8 c/s and subsequently falls and rises again with increasing frequency. This behaviour is typical of a fluid-filled elastic system in which reflexions are occurring and this is the interpretation we have put on them. It is indeed difficult to see any other way in which this behaviour could be explained.

Reflexion of waves propagated in an elastic tube will occur at any point where the fluid impedance changes. It was shown by Womersley (1957) that at any subdivision into branches one cannot achieve exact matching of the complex impedances on either side of the junction. At branches of the larger vessels, however, he predicted that the discrepancy would be small. Theoretically it is much greater at the subdivision of vessels of the magnitude of the smallest arteries, and experimental evidence in systemic vascular beds indicates that the main reflecting sites are in the region of the arteriolar beds (McDonald, 1960). The value for the average length of the arterial bed derived from our Results lies between 5 and 7 cm, which when compared with the anatomical size of the rabbit lung suggests that in this vascular bed the main site of reflexion is in the minute vessels. This estimate agrees very reasonably with that of 6 cm made by Engelberg & DuBois (1959).

The only other estimate of the impedance of the pulmonary arterial system is also by these last authors. This was derived by computing the behaviour of a simple analogous electrical circuit. It was an oscillatory circuit consisting of an inductance and capacitance, with a resistance connected in parallel across the condenser. The inductance represented the mass of the blood and its value was based on an estimate assuming a certain geometrical arrangement of subdivision of the pulmonary arteries. The capacitance represented the compliance which had been measured. The resistance represented the total vascular resistance of the bed and was regarded as residing principally in the smallest vessels such as the capillaries. Apart from the necessary approximations involved in deriving the dimensions of these components, especially the inductance, the authors regarded the omission of any transmission time effect as being a permissible, first-order approximation. At first sight this may seem reasonable for, taking their assumed wave velocity of 200 cm/sec, a 6 cm system is only 1/10 wave-length at the average pulse frequency they found (200 c/min). In fact this is far from negligible, for at the frequency of the second harmonic the system is 1/5 wave-length which is close to the relative node

at 1/4 wave-length. For the fifth harmonic it is a half wave-length, which by no approximation can be regarded as very short. In fact they calculated the behaviour of their analogue up to $10^4$ c/min when the system is more than 5·5 wave-lengths.

From our data the system is about 1/4 wave-length at 3 c/s (180 c/min) so that the approximation is even less valid. Nevertheless it is interesting to compare their graph with our own (Fig. 8). They found a minimum of impedance at 4·3 c/s (260 c/min), which is not far removed from many of

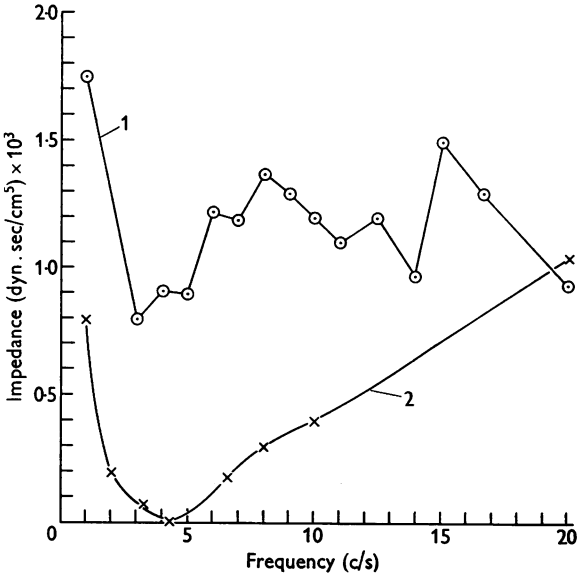


Fig. 8. A comparison between the values of the fluid input impedance in our experiments and those calculated from an electrical analogue by Engelberg & DuBois (1959), and described in the text. Curve 1 (⊙) shows the mean values of all our experiments and is taken from Fig. 5. Curve 2 (×) is a transcription of a part of their graph—it is only approximate, for this graph is small in scale and plotted with a logarithmic abscissa for frequency.

our observed values (e.g. Fig. 4) although rather higher than our over-all average. There is, however, a big discrepancy in the value of the impedance at this minimum for they calculated it to be 8 dyn.sec/cm$^5$ whereas we found the mean to be 800 dyn.sec/cm$^5$. At lower frequencies the values in both curves rise sharply but at 1c/s it is only about $0{\cdot}8 \times 10^3$ in their graph, whereas our mean is over $1{\cdot}8 \times 10^3$. With increasing frequencies above the minimum their graph rises steadily and is quite unlike the curve we find, and up to 20 c/s is always below the values we obtained. It is not felt, therefore, that this rather involved method of deriving the impedance by the use of this analogue circuit is very useful. Even with a circuit in the

form of a transmission line (Taylor, 1957*a*) it has been shown that it cannot fully mimic a fluid-filled system (Taylor, 1959).

Clearly the use of perfusion and an oscillatory pump is of no value in a living animal. If the flow and pressure in the pulmonary artery can be measured the input impedance at the frequencies represented by the harmonic components of these pulses may be determined. As an example we have taken some curves recorded in the cat by Baxter & Pearce (1951).

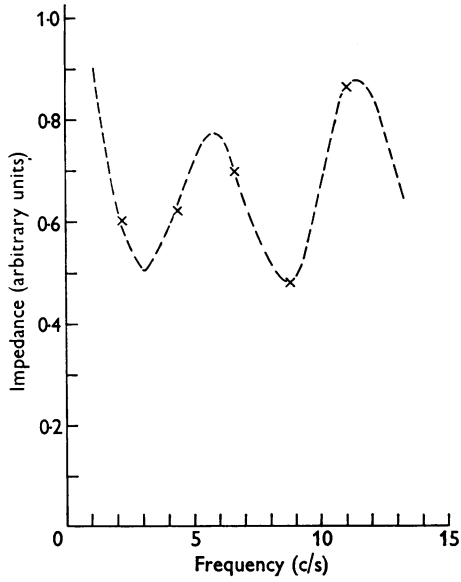


Fig. 9. The input impedance in the cat pulmonary artery derived from simultaneous records of pressure and flow by Baxter & Pearce (1951, Fig. 10). The pulse frequency was 2·2 c/s and the values for the first five harmonic components have been calculated by the use of Fourier analysis. These values are shown by crosses. The dotted line shows an impedance variation with frequency similar to that found in the rabbit and it can be seen that these points might well fall on such a curve. The phases of the harmonic components add support to this view.

These were enlarged and subjected to Fourier analysis. The modulus of the impedance for the first five harmonics in one pair of curves is shown in Fig. 9. As the fundamental frequency was 2·2 c/s the available points are insufficient to delineate the curve with the precision that we could using the pump (Fig. 4), but the line drawn through them based on the rabbit data shows they might well be points on such a curve. That is, the first two harmonics (2·2 and 4·4 c/s) probably fall on either side of a minimum at *ca.* 3 c/s followed by a maximum at 6 c/s and another minimum at 9 c/s with a subsequent rise at 11 c/s. As the size of the lung in the cat is much the same as that of the rabbit, if this is a valid comparison it would imply

that the pulmonary wave velocity is about the same value in both species. It is unfortunately not possible to compare the value of the impedance from Baxter & Pearce's curves with our own, as theirs were not calibrated.

This technique of measuring the input impedance could be employed in man, using the indirect method that Bergel, McDonald & Taylor (1958) used in the dog femoral artery. With the use of a double-lumen catheter with two lateral apertures sufficiently close together, so that they both lay in the main pulmonary artery, the velocity of the pulsatile flow could be derived from the pressure gradient while the pressure in the pulmonary artery could be recorded through one of the channels. Thus with the addition of very little complexity to routine recordings made in cardiac catheterization in many laboratories considerably greater quantitative information could be gained. For whereas the shape of the pressure and flow pulses is determined by both the form of the systolic ejection by the heart and the properties of the vascular bed, the input impedance is determined by the latter alone. How much of the pulmonary bed is involved remains to be discussed.

### *The extent of the pulmonary 'arterial' bed*

Properly speaking the input impedance involves all the system up to the point where oscillatory flow is damped out. As we recorded pressure oscillations in the pulmonary veins this is more extensive than the arterial bed, but this last plays such a dominant part that for the sake of brevity that region determining the input impedance may be called 'arterial'.

As this system terminates in the capillary bed one might expect the terminal impedance to change with the resistance of the bed. As is seen from Results (p. 441), there is a rise in the input impedance at 1 c/s (which is the nearest we were able to get to measuring the terminal impedance) and the resistance. Also, the impedance at this frequency was much closer to the resistance in the pulmonary bed than corresponding values in the femoral bed. In the latter case this was interpreted by McDonald (1960) as indicating that only the proximal arterioles were included in the region which determined the impedance. Nevertheless, the 'terminal' impedance could be increased two to three times by intra-arterial noradrenaline. In the lungs we have studied the changes in 'terminal' impedance were much less than this with equally large changes in the resistance. These changes in resistance, however, were not due to arteriolar constriction but to pulmonary oedema, which presumably exerts a far greater effect on the capillaries and venules than it does on the arterioles. The fact that the impedance does vary quite appreciably with the resistance indicates that the effective end of the arterial system is probably at the distal end of the arterioles or in the capillary bed.

Any reflexions of the oscillations that pass through the capillary bed must be very small because they will be heavily attenuated by that bed. One might expect that such as do occur would tend to be out of phase with the arterial reflexions, for a wave travelling towards the left atrium from the capillaries is moving towards an 'open' end in contrast to the partially closed end of the arterial system.

The length of the arterial system that we are interested in when concerned with oscillatory flow is in terms of wave-length. The main variables determining this are the frequency and the wave velocity. The actual anatomical length is by comparison relatively fixed; this will vary somewhat with respiration and with changing vasomotor activity the effective termination may shift slightly. These variations are very small compared with the range of frequencies represented in the cardiac pulse and probably small compared with changes that may occur in wave velocity, e.g. with alterations of mean pulmonary artery pressure or pathological changes affecting the stiffness of the arterial wall.

Changes in the length of the system, in this sense, will alter the frequency at which the input impedance is at a minimum. This means that the pressure necessary to create a given flow is least at this frequency. As the oscillatory load on the ventricle is then at its smallest it is interesting to note that the frequency for this minimum approximates to that of the normal pulse frequency. Thus the fundamental component of the pulse wave will normally be working against a low impedance and as this is always the largest component it may well be that this fact plays a part in determining the resting pulse rate. The situation is, however, more complex than this implies because the cardiac pulse is a compound wave containing a range of frequencies. Therefore an impedance minimum would be to some extent balanced out by the fact that the second harmonic would be working at an impedance maximum and this is also a component of considerable magnitude (in the example of Fig. 9 its amplitude was 70 % of the fundamental). The values shown in Fig. 5 do indicate, however, that a marked fall in pulse frequency, say to 1 c/s, would cause a great increase in the load on the right ventricle. Any marked increase in stiffness of the pulmonary artery by shifting the minimum to a higher frequency would have a similar effect. Observations on larger species would be of interest in this respect, for a greater anatomical length may be offset by a faster wave velocity (as values quoted for the dog suggest), but in fact the resting pulse frequency is also less. Without more data it is difficult to assess the relative importance of these two factors.

It is possible that the main significance of the frequency-dependence of the input impedance of the pulmonary circulation is to be sought in the balance between right and left ventricles. One exploratory experiment on

the input impedance of the rabbit aorta suggests that the quarter-wavelength minimum is also about 3 c/s, a result which parallels observations in the dog (McDonald, 1960). This dynamic similarity between the systemic and pulmonary circulations is due to the fact that although the anatomical length of the latter is much shorter the wave velocity is correspondingly lower. Thus the impedances against which the right and left ventricles are working are matched and the optimal frequency will be the same for both.

### *Venous pulsation*

The finding of pulsation in the pulmonary veins was to be expected after the observation of Lee & DuBois (1955), using the 'body plethysmograph' technique, that flow in the pulmonary capillaries was pulsatile. It would be interesting to be able to estimate the damping in the capillary bed, but this would be far from precise from our results as the venous pressure was measured at some distance from the capillary bed. As noted above, flow from the capillaries on the venous side is travelling towards an 'open' end which will, in general, cause a reduction in the amplitude of the pressure wave. However, without knowing more about the physical characteristics of the venous bed it is impossible to predict how these changes will vary with frequency. The largest oscillation recorded in a lobar vein was some 45% of that recorded in a small artery in the same lung. It would be interesting to compare these findings with pressure records made with a 'wedged' arterial catheter and from the left atrium. Since the transmitted oscillation diminished as the vascular resistance increased it seems that damping in the small vessels is the major factor in determining the size of venous pulsation.

SUMMARY

1. In perfused rabbit lungs an oscillatory flow has been imposed by a pump with a sinusoidal output. The pressure oscillations were measured at the origin of the pulmonary artery. The ratio of oscillatory pressure and flow was recorded as the input impedance.

2. In some experiments the oscillatory pressure was also recorded in a small artery in one of the lobes of the lung. From the phase shift between the two points the wave velocity was calculated. In 15 experiments the mean value was $83{\cdot}3 \pm 5{\cdot}7$ cm/sec at 6–11 c/s. At lower frequencies it rose markedly; this is attributed to the presence of reflected waves altering the apparent wave velocity.

3. The input impedance was also high at low frequencies and fell to a minimum at 3–4 c/s, where its mean value in all experiments was *ca.* 800 dyn.sec/cm$^5$. Thereafter followed a maximum at *ca.* 8 c/s and another minimum at 12–14 c/s.

4. This pattern is interpreted as due to reflexions arising from the termination of the arterial bed which would cause a minimum when the system is either one quarter or three quarters of a wave-length. The mean length of the arterial system at the wave velocity recorded lies between 5 and 7 cm, which fits reasonably with the anatomical dimensions.

5. The vascular resistance varied considerably in various experiments and the input impedance at 1 c/s varied with it but to a much smaller degree. The implications of this in regard to the main site of reflexions is discussed.

6. Oscillatory pressure in the veins was measured in 7 experiments. The transmission through the capillary bed varied inversely with the vascular resistance. In the lung with the lowest resistance ($2{\cdot}2 \times 10^3$ dyn.sec/cm$^5$) the pulsation in the lobar veins was 45 % of that in a small artery and 58 % of that at the pulmonary valves. These values are for the frequency of the first impedance minimum.

7. It is pointed out that for oscillatory flow the behaviour of the input impedance, which represents a considerable part of the load on the ventricle, is determined by the wave-length of the system, i.e. its wave velocity and the oscillatory frequency. As the pulmonary arterial system has a much lower wave velocity than the systemic arteries, their effective lengths are comparable in spite of the great disparity of their anatomical lengths. The significance of this in matching the optimal working conditions of the right and left ventricles and the part it may play in determining the normal pulse frequency is discussed briefly.

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