

THE RELATION OF PULSATILE PRESSURE TO FLOW IN ARTERIES

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This paper is concerned with the recording of the phasic changes in arterial flow during each cardiac cycle, and the investigation of the pressure oscillations generating this flow. The relation of pressure to flow is 'the central problem in haemodynamics' (Burton, 1952), and has never been satisfactorily resolved for arterial flow, as its oscillatory nature makes the application of Poiseuille's law invalid. The study has been confined here to the femoral artery of the dog. Direct measurements of flow have been made by following the movement of injected bubbles of oxygen recorded by high-speed cinematography (McDonald, 1952*a*). Pressure gradients have been measured by simultaneous recording of arterial pressure at two points in the artery and also by electrical differentiation of the output of a capacitance manometer. Flow curves have been calculated from the gradients using equations derived by Womersley (1954), the theory of which is presented in his accompanying paper (Womersley, 1955). General agreement between observed and calculated flow curves has been found and so establishes the validity of the theoretical derivation based on general hydrodynamic principles and also justifies the method of recording flows. Preliminary accounts have been presented to the Physiological Society both of the flow curves (Helps & McDonald, 1953) and of the derivation of flow from the pressure gradients (Helps & McDonald, 1954).

METHODS

Anaesthetic. I.V. pentobarbitone sodium (Abbott) was used in all experiments; initial dose was 30 mg/kg and thereafter as required.

The rectal temperature was recorded by a mercury thermometer in some experiments.

Mean linear flow velocity. The method using a Kodak high-speed camera operating between 1400 and 1800 frames/sec has been described in detail (McDonald, 1952*a*). The bubble fills the tube and travels at the mean linear velocity across the tube. The only modification is that pure oxygen has been used instead of air to reduce the risk of cumulative gas embolism. Analysis of the film has also been made easier by using a G.B. Bell-Howell projector with a reversing mechanism.

Pressure recording. Capacitance manometers (Southern Instruments Ltd.) were used, recording through a 10 cm (approx.) length of 1 mm bore polythene tubing inserted into branches of the femoral artery and adjusted so that the ends lay flush with the wall of the main vessel. The frequency response of these instruments has been measured by Dawes, Mott, Widdicombe & Wyatt (1953). Zero drift was checked at intervals but was never troublesome. The manometer output was led to a Cossor 1049 double-beam oscilloscope and recorded with time-base inhibited with film moving at 2.5 or 5 in./sec as a routine, but 10 in./sec was also used in many of the pressure-gradient studies to separate the traces adequately. In two animals the pressure was recorded on an Ediswan pen-writer, but only values for systolic and diastolic pressure have been taken from these records and no account taken of the form of the pulse wave.

Pressure gradients. Direct measurements of pressure gradient were made by recording simultaneously with two capacitance manometers on the two beams of the oscilloscope. This produced two similar pulse waves separated by the pulse-wave transmission time (Fig. 1A). The record, on film, was then projected in a photographic enlarger and the two waves traced on to graph paper. The difference in pressure between the two recording points was then measured graphically and divided by the distance between the recording points to give the pressure difference per cm of artery (Fig. 1B). This subtraction could more conveniently be performed by feeding both manometer outputs to opposite sides of a two-sided amplifier, but this was not done owing to lack of suitable apparatus. The distance apart of the recording points depended on available branches, but was usually some 3–6.5 cm. The greater distance gives a better separation of traces but also distorts the form of the gradient slightly so that the shorter distance was preferred. The brightness of the beam was kept at a minimum consistent with a clear record so that the trace record should be as thin as possible, as with projection thick traces introduce considerable errors. As the pressure gradient is only a small fraction of the total pulse-pressure the calibration of the manometers needed to be as accurate as possible. As the A_1 and A_2 amplifiers on the Cossor oscilloscope have different gains, and are calibrated in steps, the exact matching of the 2-beam deflexions was often very tedious. Calibrating pressures were produced by a pressure bottle with a mercury manometer attached. The saline (0.9% NaCl solution) flow in the manometer heads was set before calibration and not changed afterwards. Heparinization of the animal was also used, but even so slight degrees of clotting in the cannula was considered to be the main cause of the distortion of wave form that often developed during an experiment. A three-way tap was inserted between the manometer head and the cannula and used for checking zero drift, for recalibrating and for flushing out the cannula every 15–30 min.

Differential coefficient of the pressure. The limiting form of the pressure gradient as the two recording points are brought together, is the space-differential coefficient of the pressure wave (Fig. 1C). Therefore in the last series of experiments when flows were also being recorded, the output from the single manometer in use was put through a differentiating circuit and displayed on one beam of the oscilloscope while the normal pulse wave was displayed on the other beam. The calibration of the differential curve presents some difficulty for the oscilloscope record is of the time differential dp/dt and to derive dp/dz (the space differential) we have to use the relation

$$\frac{dp}{dz} = -\frac{1}{c} \frac{dp}{dt},$$

where c is the pulse-wave velocity; hence to measure the wave velocity two manometers are needed. In practice the values for the differential given were estimated by direct calibration against gradients measured with two manometers as described above.

RESULTS

I. Measurements of flow velocity

Measurements of flow recorded by cinematography were made in one femoral artery of each of six dogs and observed without filming in three others. In two dogs the film records were not good enough for accurate analysis owing to thickness of the arterial wall, but qualitative observations of the phasic flow pattern could be made. Typical flow curves are shown in Figs. 2 and 3, and the main values for flow presented in Table 1 for the four animals in which it was measured. Only results for 'normal' animals are presented here, i.e. anaesthetized animals without any other alteration of circulatory conditions.

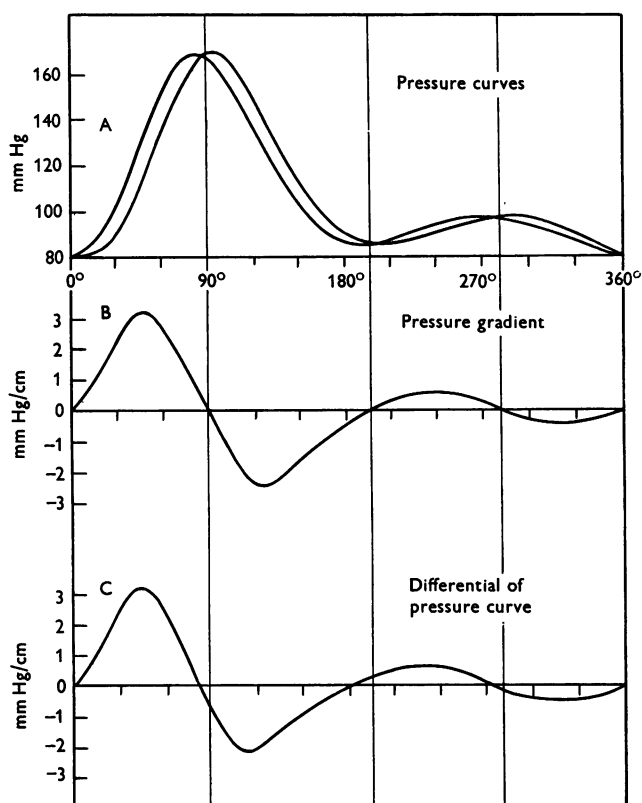


Fig. 1. A: simultaneous pressure recording at two points in the femoral artery 3.5 cm apart. In this case the shape of the pulse wave is identical at both points. B: the pressure gradient derived by subtracting the pressures at each point, at 15° intervals, and dividing by the distance between the manometric cannulae. C: the time-differential of the first pulse-pressure curve in A. The small differences between B and C are discussed in the text. The abscissa in this figure and in all subsequent ones except Fig. 2 is calibrated in degrees of arc of the cardiac cycle. As the cardiac cycle varied from 320 to 380 msec, degrees and msec may be taken as approximately equal.

Pulse frequencies varied from 2.6 to 3/sec. All the abscissae (except Fig. 2) are presented as fractions of a cycle, i.e. in degrees of arc to facilitate comparison with calculated curves, but degrees and msec may be taken as approximately equal.

The characteristic flow curve is seen to fall into four phases: (i) a fast forward flow due to cardiac systole reaching a peak velocity of about 100 cm/sec at 45° and falling to zero at about 105°, (ii) a back-flow phase following immediately afterwards reaching a more rounded peak velocity of 30 cm/sec and falling off more gradually than the systolic phase, (iii) a forward flow following

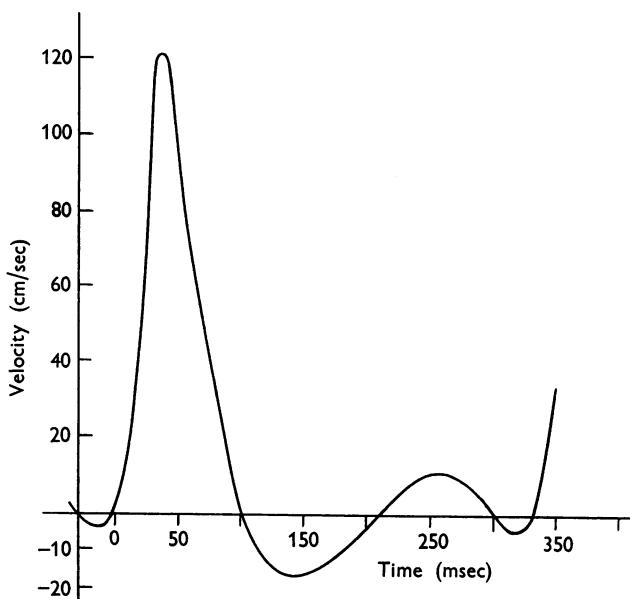


Fig. 2. The mean linear velocity of flow in the femoral artery of the dog plotted against time during one cardiac cycle.

the back flow, starting at 210° and ending at 330° with a maximum velocity of 20 cm/sec, (iv) a short and small back flow immediately preceding systole. Throughout the cardiac cycle there is, therefore, a double oscillation discernible in the flow pattern although it is heavily and unsymmetrically damped. The damping in the diastolic portion of the cardiac cycle may cause the obliteration of the terminal small back flow and more rarely there may be no diastolic forward flow. The flow pattern is then more closely similar to that seen in the rabbit aorta (McDonald, 1952*a*).

The back-flow phase was seen in all nine dogs. Even without a high-speed film the eye can see the moment of arrest and reversal of direction of a bubble. It can be seen with the first bubble of an injection so that the question of the

back-flow phase being due to gas embolism in the small distal vessels may be eliminated. Also, as demonstrated below, a back-flow phase is predicted from the observed pressure gradient in the artery.

The timing of flow reversal is of interest. It was not possible to record a central pulse during these experiments so that the time of closure of the aortic valves was not measured, but the estimated length of systole from records such as those of Woodbury & Hamilton (1937) is 35% of the cardiac cycle, or about 130° on the scale of Fig. 3. Reference to Table 1 shows that in all cases the

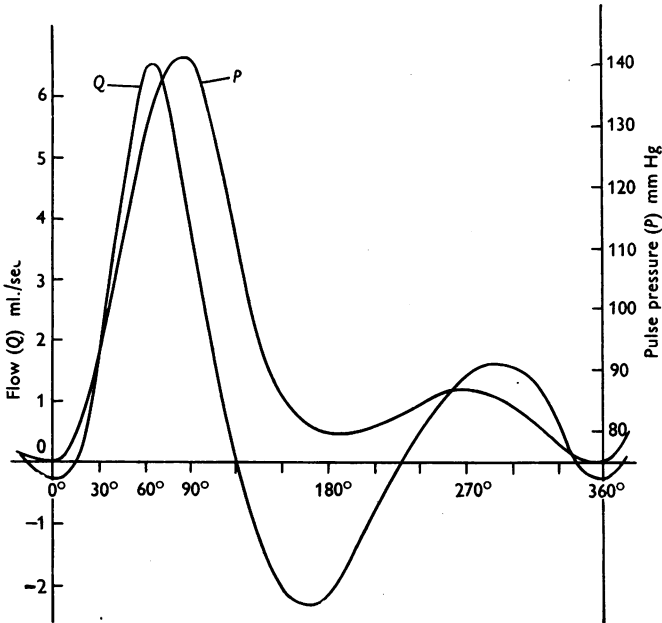


Fig. 3. A volume flow curve derived from high-speed film analysis and the pulse pressure on a comparable scale. Although superficially similar in appearance the fact that the peak flow occurs before the pressure peak shows that there is no direct relation between these curves.

reversal of flow occurs before this point and forward flow is shorter in duration than systole. It is of interest to note that it was the observation of such early back flow that provided the stimulus for investigating the pressure gradient that led to the experiments reported below.

II. Measurements of pressure gradient

Attempts have been made in the past, e.g. Machella (1936), to correlate the form of the flow curve with that of the pulse pressure curve. Fig. 3, however, shows that the phase relationship of flow in the femoral artery is quite different, as the peak of flow falls during the rising phase of the pulse curve and the pressure is greatest when flow is almost zero. This is not surprising as the

arterial pressure curve represents changes occurring throughout the system, whereas the flow must be related to differences in pressure along the artery. Pressure gradients have, therefore, been measured by simultaneous recording at two points along the artery and subtracting them as described above (see 'Methods') and as illustrated in Fig. 1A, B. When the pressure wave is transmitted without change the pressure gradient is positive (referred to the direction of flow from the heart to the periphery) during the rising phase of the pulse wave. This is true for all pressure waves propagated in this way because of the delay in the wave reaching the downstream recording point. During the falling phase of the curve the gradient is negative for the same reason. A similar alternation of positive and negative gradient occurs as a result of the secondary diastolic pressure wave. Expressed in terms of pressure/unit length of artery the positive gradient rises from zero to a value of about 3 mm Hg/cm at 45° and falls to zero again at 90°. The negative phase reaches a maximum of about 2.5 mm Hg/cm at 120° and lasts until 180–210°. The succeeding diastolic gradients are more variable and have peak values of 0.5 mm Hg/cm or less. These values cannot be regarded as critical because of their small dimensions compared to the total pressure changes and the impossibility of calibrating the individual manometers to within fractions of a mm Hg.

A divergence from these figures that was commonly seen was the observation of a negative gradient greater than the preceding positive peak. This was associated with an increased pulse pressure recorded distally in the artery; this phenomenon has been recorded by many workers in the arteries of the hind-limb, e.g. Hamilton (1947) in comparing the pulse wave in the femoral and dorsalis pedis arteries. The maximum negative pressure gradients in this case were between 3.5 and 4.5 mm Hg/cm compared to a positive gradient of 3.0–4.0 mm Hg/cm. The alteration of wave form actually within the short length of artery observed was, however, small and may be due to manometric errors. An enhanced negative peak of this kind would theoretically be associated with a larger back flow than has been observed (see 'Results', section IV). The possible causes of this discrepancy, which was only seen in some of the direct pressure-gradient recordings, are considered in the 'Discussion'.

The pulse-wave velocity in the dog femoral artery was usually about 7 m/sec, but it was not measured precisely owing to the errors inherent in recording from two points so close together.

III. *Differential coefficient of the arterial pressure*

The curve obtained by differentiating the output of the manometer against time is similar to that of the pressure gradient (Fig. 1C). In all cases the negative peak has been of smaller magnitude than the positive peak, corresponding to the observation that the rising phase of the pulse wave is steeper

than the falling phase. The time-differential pressure curve differed from the pressure-gradient curve, as measured with two manometers, in that the peak was always reached slightly earlier. This difference is half the transmission time of the pulse wave from one recording point to the next. Comparison of the zero point between the positive and negative waves in Fig. 1 will demonstrate this, for in the differential curve the zero is the crest of the pulse wave while in the gradient curve the zero is the point of crossing of the two pulse curves which is approximately the mid-point between the crests of the two waves. Apart from the slight difference in phase of the two curves there were commonly slight differences of amplitude, especially in the diastolic portion of the curve where the differential curve was often smaller in dimensions than gradient curves taken under similar conditions. Comparisons are only relative as direct simultaneous comparison has not been made between the two curves.

IV. Calculation of flow from the pressure gradient

The theoretical derivation of the relation between an oscillating pressure and the resulting flow is described in detail by Womersley (1955). A graphical analysis of one such calculation is shown in Figs. 4 and 5. The ordinates of the pressure-gradient curve are measured at 15° intervals. In Fig. 4 the sine waves of the first four harmonics derived by a Fourier analysis of the pressure gradient are shown, both separately and in their summated form. Each harmonic is of the form $M \cos (nt + \phi)$, where M is the modulus and ϕ the phase shift or argument, and n the frequency of the harmonic. Each pressure wave generates a corresponding flow curve also of sine wave form but differing in amplitude and lagging in phase (phase shift ϵ) owing to the inertia and viscosity of the blood (Fig. 5). The flow curve is represented by the equation

$$Q = \frac{MR^4}{\mu} \frac{M'}{\alpha^2} \sin (nt + \phi + \epsilon), \quad (1)$$

where Q is flow in ml./sec, M and ϕ are the modulus and argument of the harmonic of the pressure gradient and n is again its frequency. α is a non-dimensional constant where

$$\alpha = R \sqrt{\frac{2\pi f \rho}{\mu}}, \quad (2)$$

and M' and ϵ are derived from tables calculated by Womersley (1955) using Bessel functions; both are dependent on α . R is the radius, f the pulse frequency, μ the viscosity (0.04 P) and ρ the density (1.054) of the blood.

The separate flow curves for each harmonic shown in Fig. 5 are shown successively and summated in Fig. 6. With the aid of the tables of M'/α^2 and ϵ the calculations involved are simple but, without mechanical aid, lengthy and tedious. It will be noted that contribution of each harmonic of the pressure

wave becomes progressively damped so that while four harmonics show appreciable errors when summed in the pressure curve, the higher harmonics make no appreciable difference to the flow curve. In practice six harmonics have been calculated in all cases but the approximation derived from the first four is always very close to the final curve.

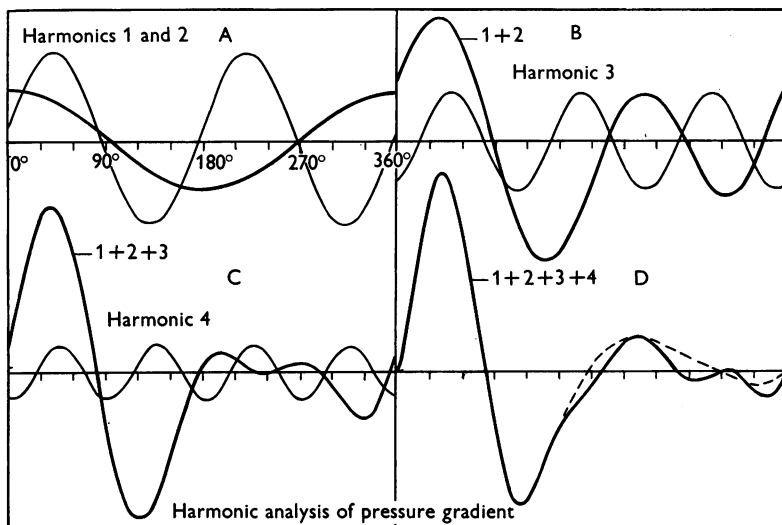


Fig. 4. The first four Fourier components of the pressure gradient of one experiment in the form $M \cos (nt + \phi)$. A: 1 and 2 are the sine waves representing the first, or fundamental harmonic ($n = 1$) and the second harmonic ($n = 2$). The modulus (M) of the second harmonic is 1.32 and that of the fundamental is 0.78. ϕ is $+0^\circ 39'$ for the first harmonic and $-82^\circ 45'$ for the second. B: 1 + 2 represents the sum of the first two harmonics represented in A and the third harmonic (3) is superimposed ($M = -0.74$ and $\phi = +26^\circ 30'$). C: 1 + 2 + 3 is the sum of the first three harmonics and the fourth is superimposed ($M = -0.41$, $\phi = -16^\circ 39'$). D: 1 + 2 + 3 + 4 is the sum of four harmonics. The broken line shows the observed curve where it is not fully represented by four harmonics—the fifth and sixth harmonics are not shown but make the two curves virtually identical.

The relation of flow to the generating pressure is shown more clearly in Fig. 7. Considering one cycle, the arbitrary point of zero pressure is seen to coincide with a small back flow. The phase lag between zero pressure and zero flow is 10° . The positive peak of the pressure gradient is at 35° and falls to zero at 78° while the flow accelerates to a peak at 65° and falls to zero at 112° , by which time the pressure is almost at its negative peak (116°). Thus we see that the application of positive pressure causes an acceleration of the blood but only after a time lag due to its inertia. Once it is flowing fast the inertia tends to keep it moving, but in fact a rapid deceleration is caused by the fact that the pressure gradient becomes negative. The continuing negative gradient then induces a retrograde flow of blood which is in turn decelerated and reversed

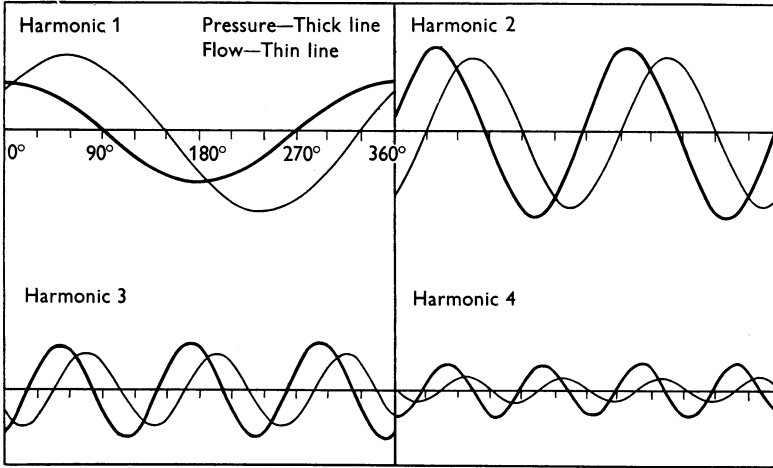


Fig. 5. The flow terms for each harmonic of the pressure gradient are shown together. Note that there is a phase shift (ϵ) due to the inertia of the fluid. 1 is the fundamental, 2, 3 and 4 the next three harmonics. ϵ is $31^\circ 14'$ in 1, $19^\circ 57'$ in 2, $15^\circ 50'$ in 3 and $13^\circ 29'$ in 4. The composite modulus (see Equation 1) is 2.4 in 1, 2.33 in 2, 1.12 in 3 and 0.34 in 4 and the curves show how the resultant flow is progressively damped in the higher harmonics. The ordinates are on an arbitrary scale.

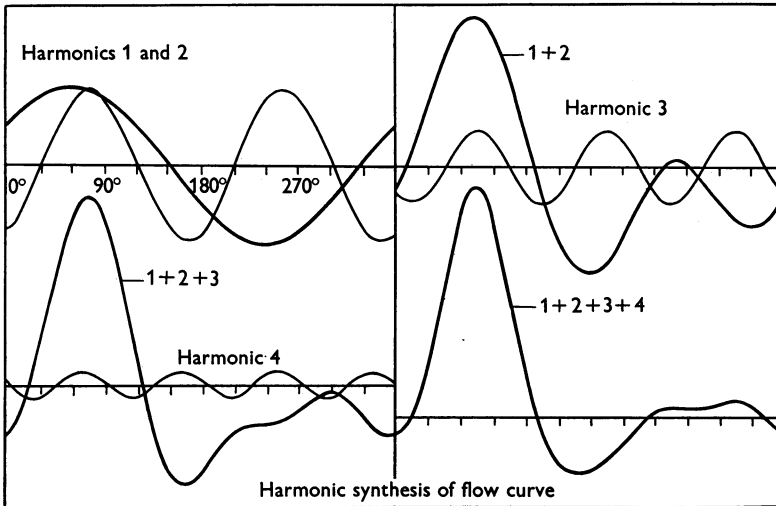


Fig. 6. The flow components shown in Fig. 5 are here shown singly and then summed successively as the pressure components were in Fig. 4. In the final summation $1+2+3+4$ the zero line has been moved to represent the addition of the mean flow, as the sine waves only represent the oscillating part of the flow. Because of the damping of the flow in the higher harmonics the flow due to the fifth and sixth harmonics is too small to show on this graph.

to a forward flow by the positive pressure gradient in diastole. The phase difference is greatest at this point and it can be seen from Fig. 7 that the end of back flow is some 55° after the corresponding end of the pressure gradient. With the slower changes of pressure in the diastolic portion of the curve the flow comes progressively more in phase until the end of the cycle is reached.

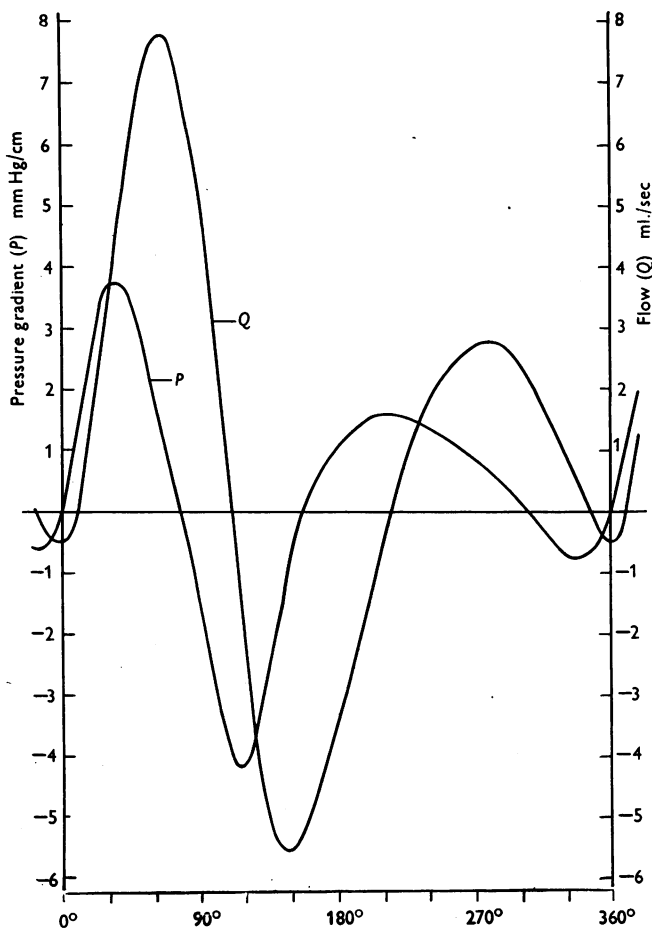


Fig. 7. The pressure gradient in one experiment with the flow generated by it are shown here superimposed. Note the variations in phase lag between pressure and flow at various parts of the cycle—this is discussed fully in the text.

The flow in any given direction is thus influenced, not only by the magnitude of the applied pressure but by the length of time that the pressure is applied and by the other pressure oscillations during the cycle. The pressure gradient necessary to produce a steady Poiseuille-type flow equal to the peak forward flow would be 1.1 mm Hg/cm.

The calculation of flow, based as it is on a summation of harmonic terms, only gives the oscillating values and over a whole cycle these will sum to a mean of zero. In fact there is, of course, a net forward flow in the artery and this is calculated separately and added to the final curve. This mean flow term is calculated from the mean positive pressure over the whole cardiac cycle using Poiseuille's law. This flow is of the order of 1 ml./sec and requires a mean pressure gradient along the artery of some 0.13 mm Hg/cm. The approximation has been made that this is linear throughout the cycle and its addition is shown in Fig. 6 by lowering the zero-flow line. The necessity for providing a mean positive pressure gradient has provided a check on the accuracy of the gradient curves, for in some cases the net gradients were negative and these curves were rejected as certainly showing manometric errors.

A further approximation is made in the calculation in that the diameter of the artery is treated as constant, i.e. as though it were a rigid tube. This approximation is, in part, only apparent because the actual pressure gradient measured is derived from observations in the living animal. This means that only the short length of artery observed is treated as a rigid system. Furthermore, the dimension of the expansion at the end of systole is almost certainly less than 5% (McDonald, 1953 and unpublished observations) and at the time of the maximal expansion flow is at a minimum, or is retrograde (Fig. 3). The errors due to this approximation and to neglecting the velocity of the propagation of the pulse wave will be published in a future paper, but are certainly small in comparison with the errors inherent in the manometric measurements and in the estimation of the diameter of the artery. A summary of results of the calculated flow values is presented in Table 2 for comparison with the observed values in Table 1. Direct comparison by measuring the pressure gradient and the flow at the same time have not yet proved possible.

V. *Calculation of flow from the time-differential of the pressure curve*

The derivation of the time-differential by passing the manometer output through a differentiating circuit has been described above (Methods). It is necessary to assume that the pulse velocity remains constant and that the time-differential bears a constant relation to the space-differential or pressure gradient. The calculation of the oscillating flow from the differential is carried out in precisely the same way as for the pressure gradient. The mean flow term, however, cannot be derived from the differential as it is only measured at one point. In the results presented the mean flow was derived from the actual observed value as the differential could be recorded simultaneously with recording of the arterial flow. The results are included in Table 2, and a comparison between observed and calculated flow is shown graphically in Fig. 8.

TABLE 1. Observed flows

Expt. no.	Wt. (kg)	B.P. (mm/Hg)	Pulse frequency per sec	Peak systolic forward flow (ml./sec)	Time end of forward flow ($^{\circ}$ Arc)	Peak back flow (ml./sec)	Time end of back flow ($^{\circ}$ Arc)	Peak diastolic forward flow (ml./sec)	Peak diastolic back flow (ml./sec)	Mean flow (ml./sec)
<i>D</i> 3 (Fig. 2)	12.0	$\frac{180}{110}$	3.0	8.3	72	2.0	180	0.6	0.1	0.7
<i>D</i> 4	12.0	$\frac{205}{125}$	2.6	10.0	83	2.0	166	0	1.0	0.4
<i>E</i> 19	10.7	$\frac{170}{100}$	3.1	8.4	108	1.2	216	0.7	0.3	1.1
<i>F</i> 12	7.7	$\frac{180}{105}$	2.8	6.5	98	2.3	170	1.0	0.5	0.75
<i>F</i> 23 (Fig. 8)	10.8	$\frac{160}{90}$	2.7	6.5	104	2.3	210	1.6	0.3	0.86

TABLE 2. Calculated flows

Expt. no.	Wt. (kg)	B.P. (mm/Hg)	Pulse frequency per sec	Peak systolic forward flow (ml./sec)	Time end of forward flow ($^{\circ}$ Arc)	Peak back flow (ml./sec)	Time end of back flow ($^{\circ}$ Arc)	Peak diastolic forward flow (ml./sec)	Peak diastolic back flow (ml./sec)	Mean flow (ml./sec)
<i>PG</i> 61	10.0	$\frac{175}{105}$	3.0	7.5	98	4.2	215	2.2	—	0.5
<i>PG</i> 65	12.3	$\frac{170}{95}$	2.8	7.65	105	5.4	240	1.6	—	0.5
<i>DF</i> 1 (Fig. 7)	10.3	$\frac{215}{150}$	2.8	7.7	102	5.5	206	2.8	0.5	0.9
<i>F</i> 23 (Fig. 8)	10.8	$\frac{160}{90}$	2.7	7.0	112	1.8	218	0.4	0.5	1.0

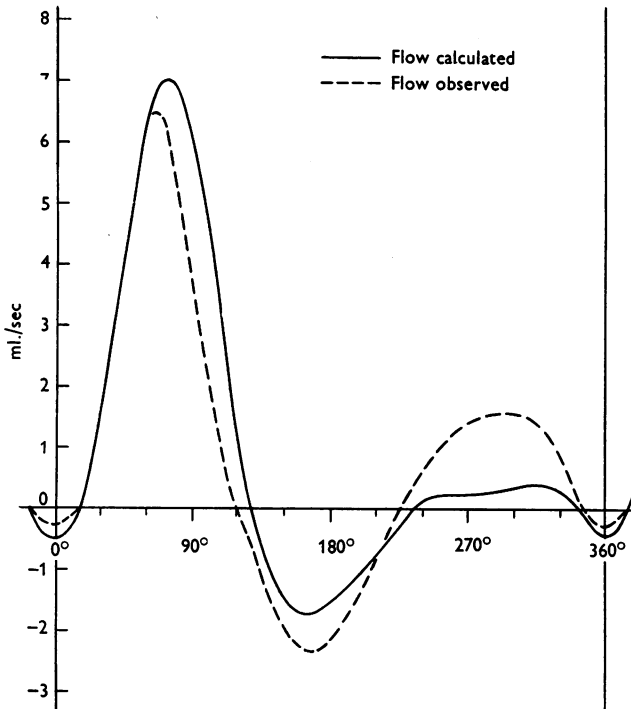


Fig. 8. The flow measured by high-speed cinematography is compared to a flow calculated from the time-differential of pressure in the same experiment. The two sets of values are within the limits of observational error.

DISCUSSION

The experiments reported here have been limited deliberately to the dog femoral artery under standard conditions in order to simplify the investigation of the relation of pressure and flow. Haemodynamic investigations in the past have tended to treat the arterial system as a whole and from this has arisen theories such as the 'windkessel' of Frank or the system of standing waves of Hamilton. Although they have been fruitful in their application, these general theories involve such considerable assumptions in their approximation of the arterial tree to a simple system of elastic tubes that it is difficult to apply rigorous physical analysis to them. In the present work my aim has been to start, as it were, at the other end of the problem and analyse the behaviour of flow in one artery with the hope that the results that emerge may be extended later to include wider aspects of the circulation. This has only been possible on account of the mathematical analysis, and continual collaboration of Mr J. R. Womersley (Womersley, 1954, 1955).

Observed flow in the femoral artery

The femoral artery was chosen initially because more reports were available on the phasic flow pattern in this artery of the dog than on any other. Nevertheless, these reports vary so much in detail that it is first necessary to consider the probable fluctuations of the arterial flow during the cardiac cycle. The high-speed cinematograph method I have used has the advantage that the artery is intact and the flow unimpeded by a meter and that the oxygen bubbles injected have a very small inertia. Its main disadvantage lies in the difficulty of analysing the films of the movement of the bubbles. The pattern recorded is constant, however, and as detailed above in 'Results', it consists of a fast forward flow during systole followed immediately by a back-flow phase which in turn is followed by a forward flow in mid-diastole which flow is usually terminated by a brief back flow. For comparison with previous workers this will be considered with the flow records of Shipley, Gregg & Schroeder (1943) using an orifice meter and Richardson, Denison & Green (1952) and Richards & Williams (1953) both using electromagnetic flowmeters. All are agreed on the initial fast forward flow, although the dimensions vary. With a mean flow of 1 ml./sec (60 ml./min) which is close to my observed means, Shipley *et al.* (1943) record a peak flow of 250–380 ml./min while Richardson *et al.* (1952) record 125 ml./min. My peak value is 400–500 ml./min and this is also reported by Richards & Williams (1953), their range being 415–452 ml./min in normal animals but their mean flow is high (170–195). The end of this flow occurs at about 105° (the whole cycle being treated as 360°) in my observations; this is difficult to compare with the published records of the electromagnetic flowmeter although one of Richards & Williams's (their fig. 8*b*) would appear to be

as early, or earlier, than this. Shipley *et al.* suggest that it ends later. This timing is of interest because, as mentioned in 'Results' (I), and previously by Helps & McDonald (1953), this suggested that the duration of forward flow in the femoral artery is less than the duration of systolic ejection. The ensuing back-flow phase is most in dispute. Shipley *et al.* find, as I do, that it is a constant phenomenon. The present experiments, however, indicate that it is commonly one-third of the velocity of the peak forward flow, whereas Shipley *et al.* report it as much smaller. All the workers using the electromagnetic flowmeter report that no back flow occurs unless there is marked vasoconstriction such as that due to adrenaline. The present experiments and the previous reports agree that forward flow occurs in diastole, although I am the only one to report a fall to zero or back flow at the end of diastole (Shipley *et al.* fig. 1, show one curve falling to zero).

Thus it will be seen that the oscillating type of flow reported in this paper is similar in shape but not in dimensions to that of Shipley *et al.* (1943), but differs fundamentally from that described by Richardson *et al.* (1952) and Richards & Williams (1953) in that they deny the existence of back flow. The function of the physical analysis may be regarded at the outset as being a means of deciding the true phasic pattern in addition to elucidating the haemodynamic conditions.

The pressure that generates the flow

From a consideration of the types of flow recorded in the femoral artery and discussed above it is apparent that the shape of the flow curve is quite dissimilar to that of the pulse-pressure curve (Fig. 3). A consideration of first principles makes it apparent that no particular correlation is to be expected as flow will be related to the pressure gradient along the artery and not to the pressure level. To obtain this gradient it is necessary to record at two points and measure the pressure change between them. The measurement of such actual gradients has been made by Green & Gregg (1940) in the coronary system, by taking the difference of aortic and intra-ventricular pressures, but the technique does not appear to have been used by other workers. Fig. 1 shows that any wave of the type of the pulse wave when transmitted along a pipe will give rise to an oscillating pressure gradient such as has been recorded above. The factors determining the magnitude of the positive and negative oscillations involve, in addition, distortion of the wave form and a fall in the mean pressure. This latter becomes an important factor as the pulse wave passes into small arteries, and this will enhance the positive phases and reduce the negative phases. The amplitude of the wave may be reduced as it passes distally and this will have much the same effect as the fall in mean pressure. If, however, the height of the pulse wave increases as it passes distally the negative phase of the gradient will be markedly increased. This is the cause of

the phenomenon that was frequently observed in the measured pressure gradients, namely that the negative peak was greater in magnitude than the positive one. The closer together the recording points were the smaller was this distortion and in the differential coefficient this phenomenon was never seen. As the flow is being recorded over a finite length of artery the gradient taken over that length is more accurate than the time-differential taken at one point. In fact if the wave is distorted during transmission, then the time-differential coefficient is not a limiting form of the gradient. This must be set against the fact that the flow calculated from the differential is in some cases in better agreement with observed flows than those calculated from the pressure gradient. This discrepancy is discussed below.

The calculated flow curves

The mathematical relation between pressure and flow derived by Womersley (1955) contains no theoretical innovations but it is original in that it is put in a form that can be computed easily. An essentially similar theoretical approach was used by Lambossy (1952*a*) in a study of the principles of condenser manometers (Lambossy 1952*b*). The approximations made (elasticity and transmission time) were detailed above in 'Results' (IV) and the magnitude of the error they introduce is known to be relatively small (Womersley, to be published). Discrepancies between the calculated and observed curves may arise on the one hand from errors in the mathematical theory and from the approximations that have been made, and on the other from observational errors in the measurement of the pressure gradient, the size of the artery and the viscosity of the blood. When it is appreciated that an error in the gradient of 1.0 mm Hg/cm at the peak value or an error of 0.1 mm in the estimation of the radius of the artery will give rise to a change of flow of some 30% it is seen that technical limitations are extremely important. A comparison of Tables 1 and 2 and Figs. 7 and 8 shows that in all cases the phase relations of forward flow and the beginning and end of back flow are in good agreement. This phase relationship provides the practical test for the mathematical theory and bears out its validity. The dimensions of the flow show more discrepancy, but the values for the forward flow show that calculated and observed are within 10-15% of each other. In view of the sensitivity of the calculated flow to minute errors in the measurement of the radius of the artery (the most inaccurate of all the physical dimensions) this relatively close agreement may be taken as fortunate. The error in measurement of observed flow is probably at least 15% at peak values.

The size of the back flow shows much more discrepancy and this is of more significance because observational errors would tend to apply throughout the cycle, so that if the calculated forward flow is of similar size to the observed then the back flow should correspond in the same way. The observed variation

is from that shown in Fig. 8, where the calculated back flow (based on the time-differential of the pressure) is slightly less than the observed, to that shown in Fig. 7, where the calculated back flow is about 100% larger than any observed by the cinematograph technique. These large back flows are all derived from curves where the negative peak is as large or larger than the positive peak of the pressure gradient. This phenomenon, as noted above, is related to a recorded increase in the pulse wave as it passes distally. This distortion of the wave may be a manometric artifact in the femoral artery, although it is known to occur in the limb vessels generally, e.g. Hamilton (1947). On the other hand, it may be a mathematical artifact because the Fourier coefficients, although representing the recorded curve, may not represent the true pressure gradient. Secondly, the calculated curve may be correct and the observed one wrong because the bubble that is being filmed may not record the full amount of the back flow. Thirdly, it is perhaps possible that the nature of the flow has changed from laminar to turbulent at this point, but the calculated flow is based on an assumption of laminar flow and hence gives a value that is too large. The stability of the fluid is being investigated, but it is known from dye-injection studies in the rabbit aorta (McDonald, 1952*b*) that a disturbance of flow pattern occurs in late systole, and studies of the velocity profile in the dog femoral artery show that much higher rates of shear occur in this type of oscillating flow than occur in a Poiseuille-type flow of similar Reynolds's number and may cause a breakdown of laminar flow (Hale, McDonald & Womersley, to be published). Whatever the cause of this quantitative discrepancy, there is no doubt that the back flow is always present. With reference to the first section of the 'Discussion' it would therefore seem to be established that recording methods used by other workers which do not record such a back flow are inaccurate.

Agreement between calculated and observed rates of flow in the diastolic phase is also relatively poor. The differences noted most often are in the size of the peak value (e.g. Fig. 8) or, in the calculated curves, a failure of the flow to return to zero at the end of diastole. This second type of flow was not seen in any film records taken but, as it was recorded by Shipley *et al.* (1943), it may have occurred. The differences in flow value in diastole may be attributed to manometric errors, as here a small error will produce the largest percentage error in the calculation.

To summarize, the calculation of flow from the pressure gradient agrees excellently with the phase relations of the flow patterns, while as regard its dimensions it predicts the systolic flow well but shows discrepancies in back flow and diastolic forward flow. This suggests that the flow records of Shipley *et al.* (1943), using an orifice meter, show the general shape of the curve well but produce some damping, especially of the fast forward flow. In view of the high resistance of this meter this is not altogether surprising. It is of interest

that a calculation of the flow from their pressure curve has been made by using graphical differentiation and the resultant flow curve has a larger forward flow peak than their meter recorded. The cause of the distorted recording of the electromagnetic flowmeter is harder to explain but is probably to be found in the nature of the oscillating flow. The phase change varies across the pipe (Hale, McDonald & Womersley, in preparation; Lambossy, 1952*a*) and this could very easily invalidate the calibration at steady flow states even though Kolin (1952) has shown that it is valid for laminar and turbulent flow. The possibility that the induced e.m.f. damps the flow according to Lenz's law does not appear to have been studied.

The a.c. theory of flow

Although the pressure-flow relation derived by Womersley (1955) may be regarded as being in an elementary form at present, some general inferences from it may be made. In a simple way we may regard the oscillating arterial pressure gradient generating a flow with a phase lag as similar to the relation between an alternating voltage and current. Hence we may refer to the 'a.c. theory of flow' as governing arterial blood flow. On the same analogy the relation of pressure and flow that obeys Poiseuille's law is the 'd.c. theory of flow'. Thus under conditions of branching where there is a steady flow the flow in each branch may be derived simply, and it is usual to talk of their *resistance*, and the summation of the action of resistances is comparable to the application of Ohm's law in a network of conductors. With an oscillating pressure in a branching system, however, there will be the phase relationship as well as the dimensions of the flow to consider and the effect of branching is analogous to the sum of *impedances*. Unfortunately the theory governing fluid flow under these conditions is considerably more complex than that governing the flow of alternating current at normal low frequencies, where the size of the conductor may be disregarded, although Womersley points out that the theory is fundamentally the same as that determining the flow of very high-frequency currents. This has the important result that the necessary Bessel functions have already been computed and tabulated. The second complicating factor, of course, is the fact that the heart generates a complex wave form so that in place of the single sinusoidal wave created by the electrical engineer we have to consider a sum of many harmonic components.

To appreciate the factor of the size of the vessel on the pressure-flow relationship one must study the behaviour of the non-dimensional constant α (Equation 2). α varies as the radius of the artery and as the square root of the pulse frequency, but this latter is the same throughout the arterial system of any one animal. Therefore, α will vary directly with the radius. Hence as a parent vessel branches the value of α becomes smaller. This has the effect (Womersley, 1955, fig. 2) of bringing the flow more in phase with the pressure

gradient and its dimensions approximate to that of a Poiseuille-type flow. Such measurements have been made for the saphenous artery where α is approximately 1.0, compared with 3.4 in the femoral artery (McDonald, to be published).

The application of the theory to larger arteries, especially the aorta, has not yet been attempted as no reliable observations on the flow in such arteries in the dog are available to check the calculated results. The work of Hamilton & Remington (1947) on the measurement of the cardiac output from a central aortic pressure pulse is of great interest in this respect. In a sequence of papers these workers and their colleagues have established an essentially empirical formula that relates various subdivisions of the pulse contour with the volume ejected. Under normal conditions, i.e. the conditions under which the formula was derived, prediction is fairly good but when conditions are altered the correlation becomes poor (Remington, 1952). This work has roused much interest and has been criticized, notably by Peterson (1952, 1954). This worker has devised an apparatus for delivering known volumes of fluid into the aorta at various times in the cardiac cycle and finds that identical injections by the syringe do not produce identical, or even predictable pulse curves. The basic error both in Hamilton's theory and in Peterson's criticism lies in the treatment of a single cardiac cycle as an isolated event when the pulse is an essentially periodic function continuously in action over a long period of time. The oscillations of one cycle are modified by previous ones and will in turn influence those that follow it because the system has not attained equilibrium at the end of a cycle as the flow curves (Figs. 2, 3, 7 and 8) show. Peterson's elaborate experiments are in fact simulating extra-systoles and it is only to be expected that the resulting curves will be very different according to the relation of the injection to the exact time in the cardiac cycle that it occurs. Unfortunately the present hydrodynamic theory cannot yet be applied to the complex conditions of the arch of the aorta. A considerable determination of physical constants such as Young's modulus, Poisson's ratio and density of the arterial wall needs to be made. Furthermore, the stability of the stream close to the input at the aortic orifice is not accurately known, but it is hoped that these problems can be solved in the future. For, although the pressure-flow relationship in the femoral artery may not be of equal practical importance to that of measuring cardiac output, the satisfactory solution of the simpler case will enable us to gain a much better understanding of the fundamental physical relationships of the arterial system. Only from such an understanding can the solution of such problems as the prediction of cardiac output from pressure recording be attempted with any confidence as to its validity.

SUMMARY

1. The phasic changes in blood flow in the dog femoral artery have been measured by high-speed cinematography.

2. The pressure gradient in the artery has also been measured by simultaneous recording with two manometers a short distance apart. The gradient oscillates, with an initial positive peak of about 3 mm Hg/cm followed by a negative peak of similar dimensions and thereafter by a smaller positive and negative wave.

3. The flow in the artery due to this pressure gradient has been calculated by an application of standard hydrodynamic theory. Agreement between observed and calculated flows was within the limits of observational error. This suggests that phasic flow recording which differs markedly from the results recorded by high-speed cinematography, e.g. electromagnetic flowmeters, is definitely inaccurate.

4. The flow in the femoral artery oscillates in the same way as the pressure gradient but with a phase lag which varies throughout the cycle. A large forward flow (7-15 times the mean flow rate) occurs during systole followed by a smaller back-flow phase and a subsequent forward flow during diastole.

5. The differential coefficient of the pulse-pressure curve may also be used to calculate the flow, and hence a relation between this curve and the flow generated has been established.

6. The implications of the physical relation between pressure and flow in the femoral artery in regard to the dynamics of the arterial system are discussed.

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