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Velocity and pressure distributions in the aortic valve

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The distribution of pressure in normal and stenosed aortic valves is investigated experimentally with a rigid-walled model placed in a pulsatile water-tunnel, and the experiments are complemented by a one-dimensional solution of the unsteady inviscid-flow equations. In the normal valve, convectively fed vortices are formed in the aortic sinuses; the vortices aid cusp positioning and the prevention of jet formation during valve closure. Aortic valve stenosis is shown to prevent the generation of vortices, causing the formation of a turbulent jet, with reduction of the pressure difference between the inlets (ostia) of the coronary arteries and the ventricle. This pressure difference is calculated for man resting and exercising, and for various degrees of stenosis.

Introduction

The aortic valve is on the outlet side of the left ventricle, the pump which supplies oxygenated blood to the body. It consists of three non-muscular cusps, about 0·1 mm thick attached to the outlet pipe, the aorta (diameter 25 mm in man), along three equally spaced generators and around the upstream edge of each cusp. Permanent dilatations of the aorta, matching each cusp, are called sinuses, and from the sinuses spring the two coronary arteries which supply oxygenated blood to the heart muscle.

An account of experiments and calculations on the closure mechanism of the aortic valve was given by Bellhouse & Talbot (1969). They used a rigid-walled model of the valve, which was placed in a pulsatile water-tunnel, and showed that the model valve was controlled by a fluid feed-back system incorporating a stagnation point at the downstream end of each sinus, and a vortex within it, and that three-quarters of the valve's closure was accomplished during forward flow, so that very little reversed flow was required to seal it. A steady-state solution of the flow in the sinuses was obtained, and they assumed that the solution was valid for a substantial part of the closure phase of the valve. No pressure measurements were reported.

In a preliminary communication, Bellhouse, Bellhouse & Reid (1968) using the same model valve but with pipes to simulate coronary arteries, suggested that the ostia, or inlets, of the coronary arteries always lie within the sinuses since those positions permit optimal coronary flow. Their assertion was supported by measurement of flow through the simulated coronary arteries. In addition, they considered the effect on coronary flow of a narrowed aortic valve opening, a frequent clinical condition called aortic stenosis. In this case, the valve cannot open fully and a jet is formed so that free-stream stagnation pressure is not attained at the coronary ostia during forward flow (systole), and the coronary arteries could experience retrograde flow during that part of the cycle. Their experiment took no account of the variable resistance of the coronary arteries, nor were pressure measurements or calculations reported.

The purpose of the present paper is to extend the theory of the aortic valve, so that pressure distributions in the normal and stenosed situations may be calculated for a range of heart rates, peak velocities and aortic dimensions.

Steady flow through the valve

In steady flow, the cusps were seen to project slightly into the sinuses, by about 10 % of the aortic radius, a. A strong vortex, with maximum velocity close to aortic velocity u_1, formed in each sinus, as in figure 1. The thrust exerted on the cusps by the vortices was matched by increased pressure on the aortic

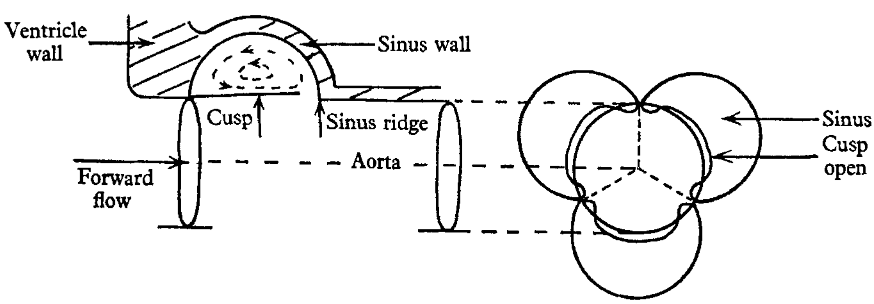

FIGURE 1. Aortic valve in open position.

side of the cusps resulting from the slight divergence of the flow. Bellhouse & Talbot calculated that the maximum velocity in the sinus, q was given by

$$q^2 = \frac{2\alpha^2 u_1^2}{25}, \tag{1}$$

where u_1 is the aortic velocity and α is a constant, and found that the average pressure exerted on the cusps by the vortices, $\bar{p}_s$, was given by

$$\frac{\bar{p}_s - p_1}{\frac{1}{2}\rho u_1^2} = 1 - 0{\cdot}0672\alpha^2, \tag{2}$$

where p_1 is the pressure at the aortic root (figure 2), and ρ is density. They chose to evaluate α from (2), but a more satisfactory procedure might be to take $q = u_1$ in (1), which gives $\alpha^2 = 12{\cdot}5$. This is justified by velocity measurement in both steady and pulsatile flow.

In steady flow the cusps assume the shape of part of a cone, as in figure 2. The continuity and Bernoulli equations for flow between the cusps are

$$u_1 y^2 = u_1 a^2$$

and

$$p_1 + \tfrac{1}{2}\rho u_1^2 = p + \tfrac{1}{2}\rho u^2,$$

provided the flow is uniform at any cross section. These equations give

$$\frac{p-p_1}{\frac{1}{2}\rho u_1^2} = 1-\frac{a^4}{y^4}, \quad \text{where} \quad y = a+\frac{r-a}{L}x. \tag{3}$$

Integrating (3) over the cusp surface, S, and writing the averaged value of p as

$$\bar{p} \equiv \frac{1}{S}\int_S p\,dS,$$

we obtain

$$\frac{\bar{p}-p_1}{\frac{1}{2}\rho u_1^2} = 1-\frac{1}{\lambda^2} \tag{4}$$

where $\lambda = r/a$.

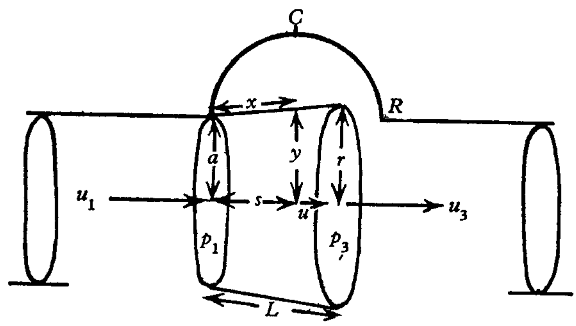

FIGURE 2. Equilibrium cusp position.

Since the cusps are balanced in steady flow, the average pressures on the sinus and aortic sides of the cusps are equal. From (2) and (4) we have $\lambda^2 = 1{\cdot}1905$ and $\lambda = 1{\cdot}091$, which correspond to the downstream edges of the cusps projecting into the sinuses by 9·1 % of the aortic radius.

Pulsatile flow

Bellhouse & Talbot (1969) reported measurements in pulsatile flow in which cusp position and aortic velocity were recorded simultaneously. Velocity was measured with a heated thin-film gauge and displayed on an oscilloscope with its screen in the plane of the valve. Measurements were made from individual frames of a ciné film of the valve and the oscilloscope screen shot through a downstream viewer. Whenever the cusps projected into the sinuses their downstream edges would have been invisible, since the aortic wall limited the field of view. In pulsatile flow, the acceleration vanishes at peak velocity and we assume that the cusps reach their steady-flow position at peak systole. From the ciné film (but replacing measurements of λ^2 when the cusps were invisible with $\lambda^2 = 1{\cdot}1905$ at peak systole) figure 3 was drawn.

Since the valve had to support a pressure difference when closed, the cusps were pushed back towards the ventricle during diastole. For the first 53 msec of systole they were pushed forward without opening, they opened in 47 msec to parallel with the aortic wall, then opening further to their equilibrium position at peak systole. They remain wide open ($\lambda^2 \geqslant 1$) for 197 msec, before closing down to $\lambda^2 = 0{\cdot}28$ by the end of systole, taking a further 67 msec.

If the cusps offer negligible obstruction to the accelerating flow during the opening phase, then the flow through the valve would be identical to the flow of an inviscid fluid in a parallel tube. The assumption is valid if, in the time taken for the valve to move from its unloaded position to parallel with the aortic wall, the aortic flow has moved forward by the length of the cusp.

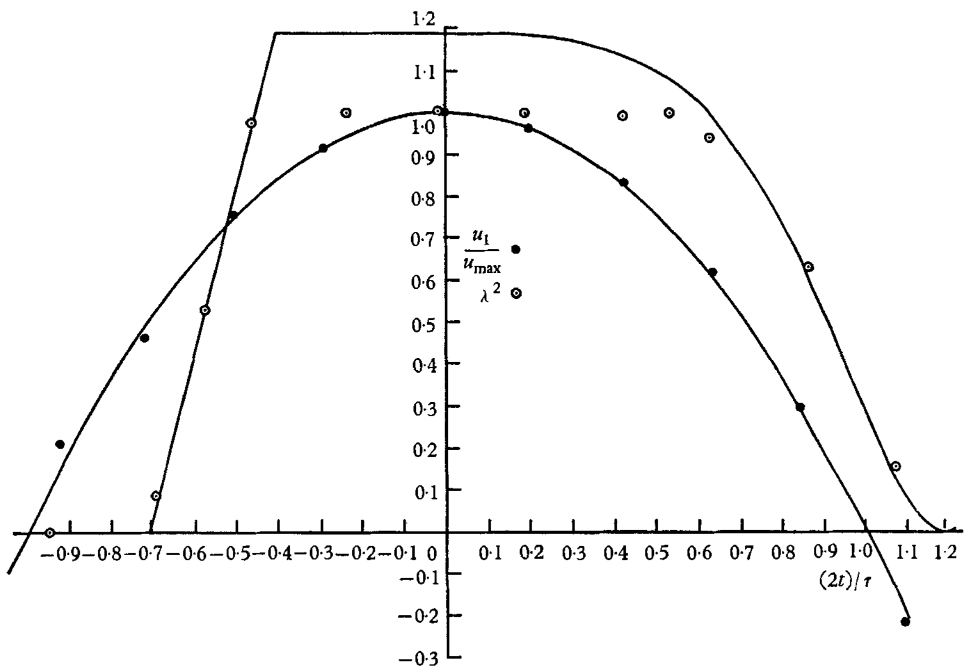

FIGURE 3. Aortic velocity and valve opening area as functions of time.

From figure 3, denoting the beginning and end of the rapid opening phase by t_1 and t_2, we have the distance moved by the forward flow in the time interval (t_1, t_2)

$$\int_{t_1}^{t_2} u_1\, dt = 2{\cdot}17\,\text{cm},$$

which is close to the measured cusp length of 1·9 cm. From the inviscid, unsteady, one-dimensional momentum equation

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial s} = -\frac{1}{\rho}\frac{\partial p}{\partial s}, \tag{5}$$

where u_1 is aortic velocity, and s axial distance, we obtain, in the notation of figure 4

$$p_2 - p_1 = -2\rho a\, du_1/dt, \tag{6}$$

$$p_R - p_1 = \tfrac{1}{2}\rho u_1^2 - 2\rho a\, du_1/dt, \tag{7}$$

where p_R is the pressure at the sinus ridge, which we assume is a stagnation point.

Once the cusp tips open beyond the aorta the sinus vortices are formed, and these persist for the remainder of systole. Since the maximum velocity in the vortex equals the aortic velocity u_1, we assume that the steady-state vortex solution obtained by Bellhouse & Talbot holds throughout systole, except for the opening phase, and that the sinus ridge is maintained at free-stream stagnation pressure $p_2 + \frac{1}{2}\rho u_1^2$.

In the notation of figure 5, Bellhouse & Talbot (1969) show that the sinus pressure (p_s) and velocity (q) distributions are given by

$$\frac{p_s - p_2}{\frac{1}{2}\rho u_1^2} = 1 - \left\{1 - 2\left(\frac{r}{a}\right)^2 + \left(\frac{r}{a}\right)^4 + \sin^2\theta\left(3\left(\frac{r}{a}\right)^2 - 2\left(\frac{r}{a}\right)^4\right)\right\}, \tag{8}$$

and

$$q^2 = u_1^2\left\{1 - 2\left(\frac{r}{a}\right)^2 + \left(\frac{r}{a}\right)^4 + \sin^2\theta\left(3\left(\frac{r}{a}\right)^4 - 2\left(\frac{r}{a}\right)^2\right)\right\}. \tag{9}$$

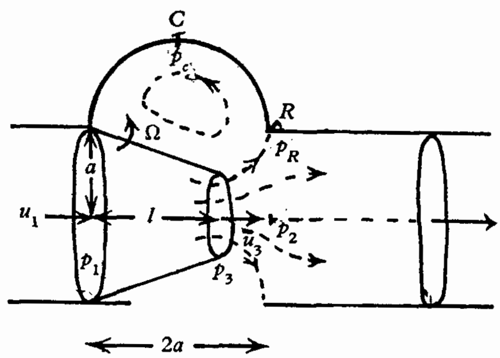

FIGURE 4. Flow pattern during closure of the valve.

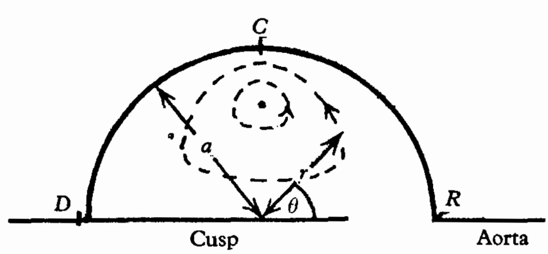

FIGURE 5. Sinus vortex model.

From (8) and (9), we find that at C, where $r = a$ and $\theta = \frac{1}{2}\pi$, $q = u_1$ (which is supported by velocity measurements) and the sinus pressure at the point, p_c, equals p_2.

The valve starts to close at peak systole, when the aortic velocity begins to decrease under the adverse pressure gradient in the aorta. As the cusps move towards their closed position, the aortic streamlines converge at the cusp tips but immediately spread under the action of the sinus vortices, which persist throughout the closure phase, so that the velocity profile level with the sinus ridge is found to be flat at all phases of the cycle. Although equations (6) and (7) may be adequate near peak systole, account should be taken of the convergence and divergence of the streamlines if the pressure distribution in the valve is to be calculated for late systole.

Axial pressure distribution during peak and late systole

We suppose that the cusps lie along a truncated-cone curved surface, drawn in figure 4. The cusps have angular velocity Ω, the velocity through the cusps is u at an axial distance s from the root, and the pressure at that cross section is p. These quantities are given the suffix 1 at the valve root, 2 at the sinus ridge and 3 at the cusp tips. Corresponding to the axial distance s, is a distance along the cusps x, and the radius of the cross section y. We assume that p and u are uniform at each cross section. From continuity

$$u = \frac{a^2 u_1}{y^2} - \frac{2\Omega x^2}{y^2}\left(\frac{a}{6}+\frac{y}{3}\right) \tag{10}$$

and

$$u_3 = \frac{1}{\lambda^2} u_1 - \frac{a}{3\lambda^2}\left(\frac{L}{a}\right)^2 \Omega(1+2\lambda). \tag{11}$$

From the momentum equation, (5), and (10)

$$\frac{p_1 - p_3}{\rho} = \frac{l}{\lambda}\frac{\partial u_1}{dt} + \tfrac{1}{2}u_3^2 - \tfrac{1}{2}u_1^2 - \frac{al}{3\lambda}\left(\frac{L}{a}\right)^2 \frac{d\Omega}{dt}, \tag{12}$$

where

$$\left(\frac{l}{a}\right)^2 = \left(\frac{L}{a}\right)^2 - (1-\lambda)^2.$$

In the region between the cusp tip and the sinus ridge the velocity changes from u_3 to u_1, and we assume that the velocity satisfies a power law of the form

$$\frac{u-u_1}{u_3-u_1} = \left\{\frac{2a-s}{2a-l}\right\}^n. \tag{13}$$

The boundary conditions at the cusp tip ($u = u_3$ when $s = l$) and at the ridge ($u = u_1$ when $s = 2a$) are satisfied for all positive n. Integrating the momentum equation

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial s} = -\frac{1}{\rho}\frac{\partial p}{\partial s}$$

between $s = l$ and $s = 2a$, with u substituted from (13), we obtain

$$\frac{p_3 - p_2}{\rho} = \tfrac{1}{2}u_1^2 - \tfrac{1}{2}u_3^2 + (2a-l)\frac{du_1}{dt} + \frac{(2a-l)}{n+1}\left(\frac{\partial u_3}{\partial t} - \frac{du_1}{dt}\right). \tag{14}$$

From (11)

$$\frac{\partial u_3}{\partial t} = \frac{1}{\lambda^2}\frac{du_1}{dt} - \frac{a}{3\lambda^2}(1+2\lambda)\left(\frac{L}{a}\right)^2\frac{d\Omega}{dt}. \tag{15}$$

From (12), (14) and (15)

$$\frac{p_2 - p_1}{\rho} = -\frac{du_1}{dt}\left\{\frac{l}{\lambda} + (2a-l)\left(1 + \frac{1-\lambda^2}{(n+1)\lambda^2}\right)\right\} + \frac{d\Omega}{dt}\left\{\frac{al}{3\lambda}\left(\frac{L}{a}\right)^2 + \frac{a(2a-l)}{3\lambda^2(n+1)}\left(\frac{L}{a}\right)^2(1+2\lambda)\right\}. \tag{16}$$

From figure 3, taking time $t = 0$ at peak systole, where u_{max} is the peak systolic velocity and τ the duration of systole,

$$\left.\begin{aligned} \frac{u_1}{u_{max}} &= 1-\left(\frac{2t}{\tau}\right)^2 \quad \text{for} \quad -\frac{\tau}{2} \leqslant t \leqslant \frac{\tau}{2} \\ \text{and} \qquad \lambda &= 1\cdot09-0\cdot56\left(\frac{2t}{\tau}\right)^4 \quad \text{for} \quad 0 \leqslant t \leqslant \frac{\tau}{2}. \end{aligned}\right\} \tag{17}$$

The axial pressure differences, p_2-p_1, were calculated from (16) and (17) for the latter half of systole, with n zero, unity and infinity respectively, and with $\tau = 0\cdot4$ sec, $u_{max} = 71\cdot9$ cm/sec, $a = 1\cdot27$ cm, $L/a = 1\cdot5$.

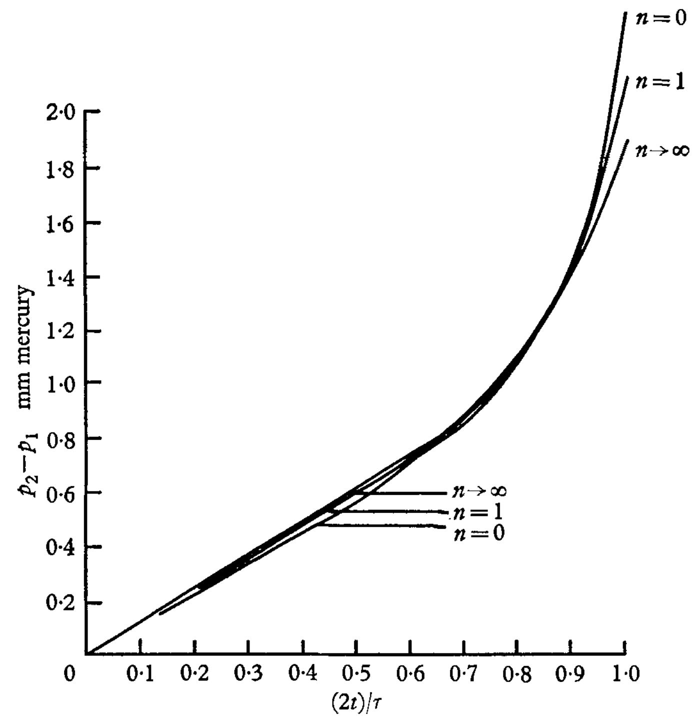

FIGURE 6. Calculation of the axial pressure difference p_2-p_1 for different velocity interpolations.

The differences between the calculations (shown in figure 6) are small, and n is taken to be unity.

If we assume that the forms of $u_1(t)$ and $\lambda(t)$ given in (17) are universal, then the pressure difference p_2-p_1 may be calculated from (16) and (17) for various operating conditions of the valve, given only the value of peak velocity and the duration of systole.

The measurement of differential pressure and aortic velocity

To test the theory in the preceding sections, pressure tappings were made at C, D and R in the wall of the valve, as in figure 5. Stiff plastic tubes linked the pressure holes, two at a time, to a differential pressure gauge of high sensitivity and large frequency response (Kistler type 7251). The rise and fall in pressure at a point was about 200 mm of mercury, the differences to be measured were about 1 mm of mercury. Aortic velocity was measured with a thin-film heated

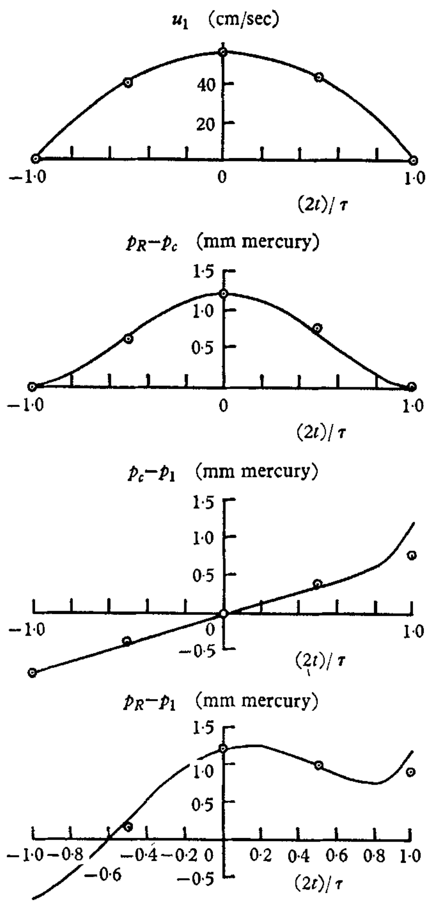

FIGURE 7

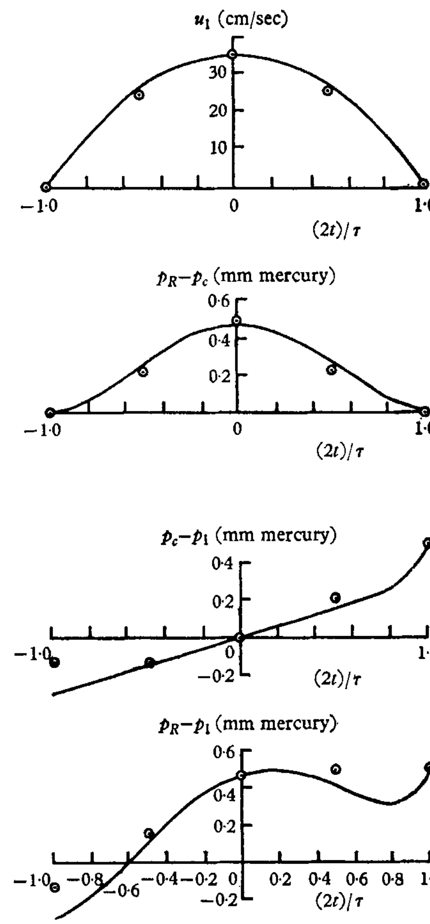

FIGURE 8

FIGURE 7. Pressure and velocity measurements compared with theory for $\tau = 0{\cdot}575$ sec, $u_{max} = 56{\cdot}7$ cm/sec.
FIGURE 8. Pressure and velocity measurements compared with theory for $\tau = 0{\cdot}91$ sec, $u_{max} = 35{\cdot}0$ cm/sec.

element cemented into a stainless-steel tube of outside diameter just under 1 mm. Three different settings of the pulsatile flow rig were used:

$$\text{(i)}\quad u_{\max} = 56{\cdot}7\,\text{cm/sec},\quad \tau = 0{\cdot}571\,\text{sec},$$
$$\text{(ii)}\quad u_{\max} = 35{\cdot}0\,\text{cm/sec},\quad \tau = 0{\cdot}910\,\text{sec},$$
$$\text{(iii)}\quad u_{\max} = 56{\cdot}7\,\text{cm/sec},\quad \tau = 1{\cdot}170\,\text{sec}.$$

It was not possible to eliminate drift of the pressure gauge when its full sensitivity was used. Therefore one point on each pressure curve has been assumed: $p_R - p_c$

is taken at the beginning of systole, $p_c - p_1$ is taken zero at peak systole, and $p_R - p_1$ is taken equal to $p_R - p_c$ at peak systole.

The measurement of velocity presented no difficulties, since the flow was laminar. In figures 7, 8 and 9 the results of the velocity and pressure measure-

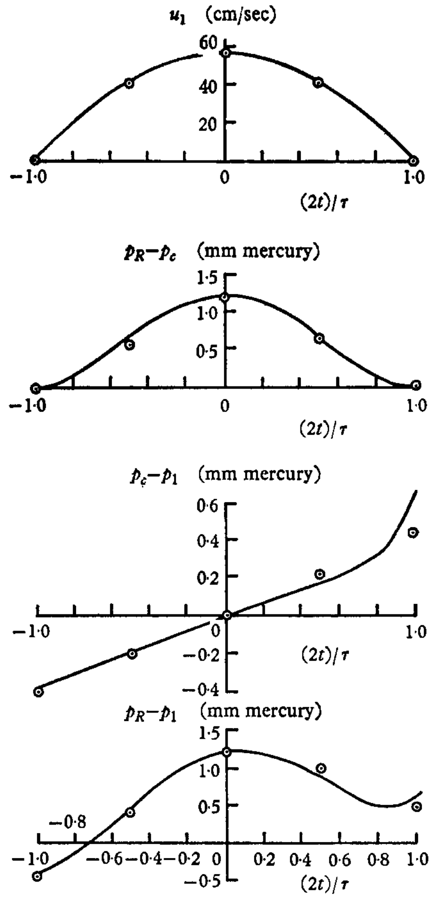

FIGURE 9. Pressure and velocity measurements compared with theory for $\tau = 1{\cdot}170$ sec, $u_{\max} = 56{\cdot}7$ cm/sec.

ments are compared with calculations using (16), (17) and the following deductions from (8):

$$\left.\begin{aligned} p_c &= p_2, \\ p_R - p_2 &= p_R - p_c = \tfrac{1}{2}\rho u_1^2, \end{aligned}\right\} \tag{18}$$

together with the values of peak systolic velocity and the duration of systole. The records were continuous, but only five points were taken from each, since they were sufficient to indicate the measure of agreement between theory and experiment.

Stenosed valve

In the normal valve, vortices are formed in the sinuses when the aortic flow is intercepted by the sinus ridge. The vortices help position the cusps at peak systole, and aid valve closure in the latter half of systole by preventing jet formation as the cusps close. In addition, regions of stagnant blood behind the cusps (which would be favourable to clotting) are avoided, and since the dynamic head is recovered at the sinus ridge, it is an ideal site for the coronary ostia. If the normal flow pattern in the aortic root is destroyed by eliminating the sinuses, a substantial reduction in the efficiency of the valve results. This occurs because no vortices form, since there is no sinus ridge to generate them, and the valve stays in its open position until the end of systole, when it is closed by reversed flow alone. Bellhouse & Bellhouse (1968) describe an experiment with a sinus-less valve in which the cusps remained aligned with the aortic wall until well into diastole, with a resultant reduction in efficiency of about 20 % compared with the normal valve.

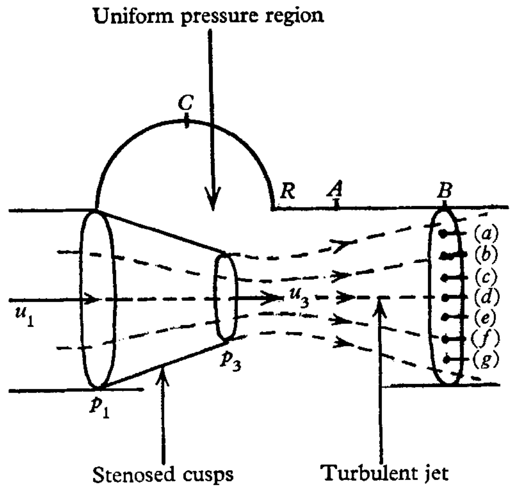

FIGURE 10. Flow pattern in valve with 75% stenosis.

If the cusps become attached to each other at their downstream edges, the valve cannot open fully (aortic valve stenosis) and a turbulent jet results, with consequent blood trauma. Since the valve remains in a nearly closed position, no substantial increase in reversed flow each pulse is likely to be observed. The pressure drop across the valve during forward flow will increase, and energy will be lost in the turbulent jet, so the heart will have to perform more work if the valve is stenosed. In addition, since the jet is unlikely to be intercepted by the sinus ridge, the sinus pressure during systole could be reduced. Since the inlets to the coronary arteries are placed within the sinuses, the reduced inlet pressure relative to the ventricle imply reduction in coronary artery flow. Bellhouse *et al.* (1968) simulated coronary artery flow with aortic stenosis, but since the variable resistance of the coronary arteries is difficult to simulate, their measurements bore little resemblance to what would be measured *in vivo*, and were suitable just to compare flows for normal and stenosed valves in a qualitative way. Since the pressure difference between the coronary ostium and the ventricle is unaffected by the resistance of the coronary arteries, measurements in the model should be a good approximation to what would be measured *in vivo*.

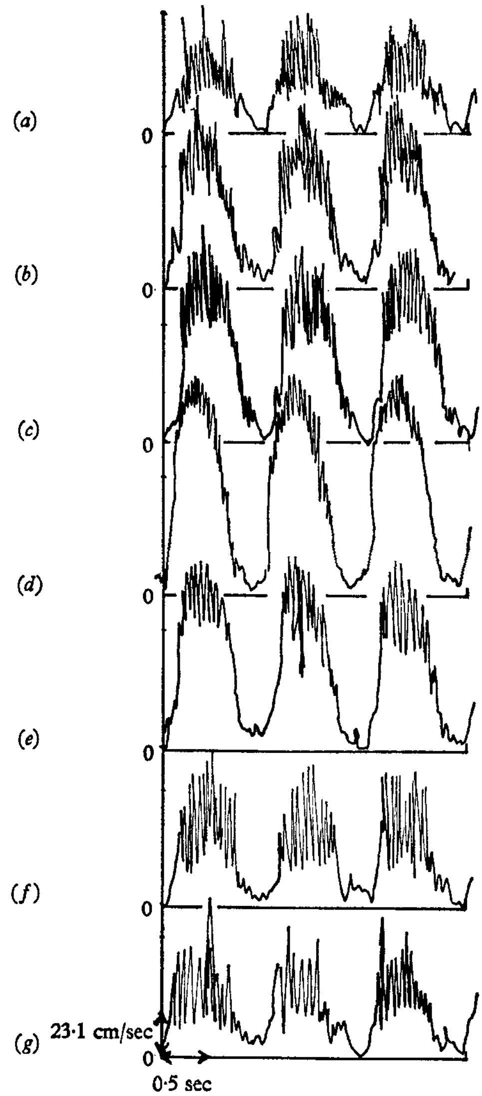

FIGURE 11. Velocity distribution in the turbulent jet of a stenosed valve, one diameter downstream from the sinus ridge.

The cusps in the model valve were glued together so that a stenosis of about 75 % resulted, with the cusps forming the curved surface of a truncated cone. In figure 10 the flow pattern in the stenosed valve is shown. A turbulent jet was apparent throughout systole, with a region outside the jet maintained at uniform pressure. This was confirmed by differential pressure measurements within the sinuses and at points downstream from the sinus ridge. The downstream extent of the separated region is uncertain, since although A in figure 10 was within this region for all of systole, B was within the region for only part of systole, which implied variable spreading of the jet during the cycle.

A velocity traverse at B, at positions (a) to (g) marked in figure 10, is shown in figure 11, in which the turbulent jet is apparent. A velocity traverse upstream of the valve showed the profile to be flat at all stages of the cycle, and the flow to be laminar.

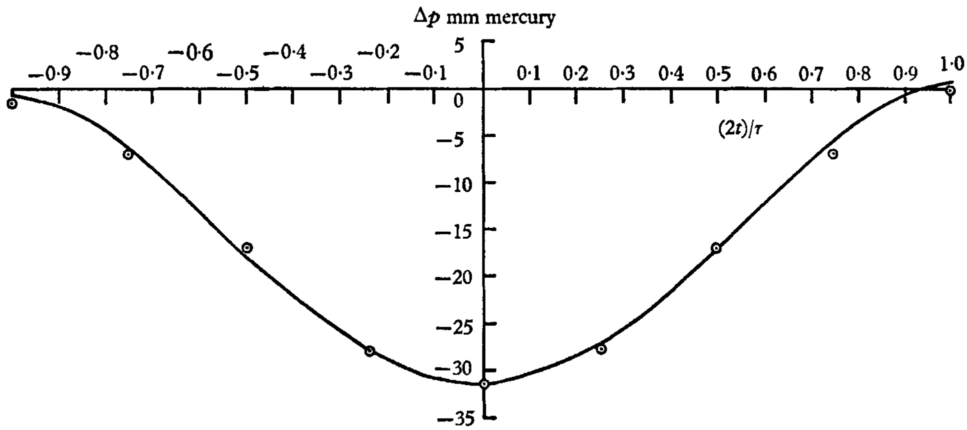

FIGURE 12. Measurements of pressure difference, Δp, between a coronary ostium and the ventricle, compared with theory, for a valve with a 78·8% stenosis, τ = 1·0 sec, u_{max} = 62·4 cm/sec.

We assume that the stenosed valve opens early in systole, into the shape shown in figure 10, and remains in that position until closed by reversed flow. If the velocity and pressure is uniform at each cross-section within the cusps, the pressure difference between cusp tip and root is given, from (12), by

$$\frac{p_3-p_1}{\rho a} = -\frac{l}{\lambda a}\frac{du_1}{dt}+\frac{u_1^2}{2a}-\frac{u_3^2}{2a}. \tag{19}$$

By continuity, $u_3 = u_1/\lambda^2$.

From (19) and (20),

$$\frac{p_3-p_1}{\rho a} = -\frac{l}{\lambda a}\frac{du_1}{dt}-\frac{u_1^2}{2a}\left(\frac{1}{\lambda^4}-1\right). \tag{21}$$

The form of the velocity pulse given in (17) was found to agree with that measured in the stenosed valve. For a systolic duration of 1·0 sec, and peak velocity upstream of the valve of 62·4 cm/sec, the pressure difference between the separated region and the ventricle, $p_3 - p_1$, was measured. The pressure difference reached −30 mm of mercury at peak systole, which was more than sufficient to permit drift-free operation of the pressure gauge. The diameter of the circle formed by the cusp tips was half the aortic diameter, giving a stenosis of 75 %. Since the streamlines converged within the cusps, a vena contracta probably formed a short distance from the cusp tips. In this case the stenosis would exceed 75 %, and if an effective stenosis of 78·8 % is assumed, the pressure differences calculated from (21) are in good agreement with the measurements, as can be seen from figure 12.

Pressure differences in human aortic valves

During diastole the aortic valve is closed, and diastolic coronary ostia pressures will be unaffected by ostia positions provided they are on the aortic side of the cusps. However, the coronary ostia always lie within the sinuses, and in fleet animals, are placed close to the sinus ridge (Bellhouse *et al.* 1968).

Gregg (1963) measured left coronary flow in unanaesthetized dogs, and was able to demonstrate the existence of systolic coronary flow which, in exercise, equalled and often exceeded diastolic flow, and he showed that there was significant systolic flow within the heart-muscle itself.

The theory developed above may be used to calculate coronary ostium pressure relative to the ventricle for man, for rest or exercise and for a normal or stenosed valve, taking account of ostium position. We assume that the forms of $u_1(t)$ and $\lambda(t)$ given in (17) are universal in the normal valve, and that $u_1(t)$ is the same shape in the stenosed valve. From (16) the difference in pressure between the sinus centre and the ventricle may be computed. The addition of the dynamic head gives the pressure difference between the sinus ridge and the ventricle. These are, respectively, the least and most favourable ostium positions found in man. For a stenosed valve (21) is used, with $\lambda^2 = 0{\cdot}75$ for a 25 % stenosis, and $\lambda^2 = 0{\cdot}25$ for a 75 % stenosis.

At rest, the cardiac output in man is taken to be 5 l/min, his heart rate 70/min, the proportion of the cycle occupied by systole $\frac{1}{3}$ and the diastolic pressure 80 mm mercury. In exercise, the cardiac output rises to 30 l/min, heart rate rises to 180/min, systole occupies $\frac{1}{2}$ of the cycle, and diastolic pressure rises to 100 mm mercury. Taking the aortic diameter to be 2·5 cm, the peak systolic velocity and duration of systole may be deduced, using the velocity pulse shape of (17). At rest these are 74·0 cm/sec and 0·286 sec, and in exercise 295·5 cm/sec and 0·167 sec.

Although the forms of $u_1(t)$ and $\lambda(t)$ may not be invariant for wide changes of peak velocity or heart rate, a modest extrapolation can be justified on the basis of the experiments varying both parameters, shown in figures 7, 8 and 9.

Calculations of systolic pressure difference between a coronary ostium and the ventricle were performed for the sinus ridge and sinus centre positions for a normal valve, and for a 25 and 75 % stenosed valve. The ostium of the stenosed valve could be at any sinus position, since the sinus was part of the uniform pressure region outside the jet. The calculations were plotted, as in figure 12, and averaged throughout systole, by measuring the area under the curve.

The averaged results are shown in table 1, the third columm being the mean systolic pressure difference. The assumed diastolic pressure difference is shown in column four, and the pressure difference averaged over the cycle is shown in column five. At rest, there appears to be little to choose between the sinus ridge or sinus centre for ostium position, and the stenosis has to be severe to reduce the mean pressure significantly.

In exercise, the differences are important, and it is apparent that an ostium on the sinus ridge is advantageous. Even a small stenosis will cause a substantial reduction in pressure difference, and a man with a 75 % stenosis might well be incapable of exercise. This could be the explanation of the symptom of angina pectoris associated with effort, when the coronary arteries are normal, but the aortic valve stenosed.

Ostium position	Condition of valve	Mean systolic Δp mm mercury	Diastolic Δp mm mercury	Mean Δp over cycle mm mercury
Man resting				
Sinus ridge	Normal	+1·08	80	53·7
Sinus centre	Normal	−0·01	80	53·3
Ridge or centre	25 % stenosis	−0·86	80	53·0
Ridge or centre	75 % stenosis	−16·17	80	47·9
Man exercising				
Sinus ridge	Normal	+17·48	100	58·7
Sinus centre	Normal	−0·11	100	49·9
Ridge or centre	25 % stenosis	−13·53	100	43·2
Ridge or centre	75 % stenosis	−260·5	100	−80·3

TABLE 1. Calculated mean pressure difference, Δp, between a coronary ostium and the ventricle for a man resting and exercising, for two ostia positions and normal and stenosed valves.

Conclusion

A method of calculating the pressure distribution in the aortic valve when perfused with a pulsatile flow has been developed, using inviscid, one-dimensional equations for momentum and continuity. The calculations have been confirmed by differential pressure measurements in a model of the aortic valve over a range of pulsation frequencies and peak flow rates.

It has been confirmed that the mechanism of the normal aortic valve depends on a convectively fed vortex in each sinus; the vortices help position the cusps at peak systole, and assist in the prevention of jet formation during valve closure.

Some effects of aortic valve stenosis have been shown to be the formation of a turbulent jet, the elimination of the vortex pattern, and the reduction of the pressure at the coronary ostia with respect to the ventricle, during systole. The method of calculation of pressure in the stenosed valve has been confirmed by experiment. Calculations of pressure difference between the coronary ostia and the ventricle have been made for man, both resting and exercising, for various ostia positions and for stenosed valves. The results are shown to be in accord with anatomical and physiological observations, and to offer an explanation of a clinical paradox.

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