THE EFFECT OF SOME HEMODYNAMIC FACTORS ON THE BEHAVIOUR OF THE AORTIC VALVE*

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Abstract—To test the validity of a theoretical model of aortic valve closure, based upon the observations in a two-dimensional analogue, the effect of some hemodynamic factors on aortic valve behaviour was studied in open-chest dogs. Direct cinematography was used to record aortic valve movements. The ECG, instantaneous ascending aortic blood flow as well as left ventricular and aortic pressures were registered simultaneously.

The experiments revealed that: (1) at higher stroke volumes complete valve opening was reached earlier in the cardiac cycle; (2) the higher the peak aortic flow the better a circular shape of the completely opened valve was approximated; (3) the higher the systolic aortic pressure the earlier valve closure started in the cardiac cycle; (4) initial valve closure during flow deceleration was faster in the case of a larger systolic aortic pressure drop; (5) the backflow in the aorta increased at a larger end-systolic valve orifice area; (6) a rise in heart rate does not affect the mechanism of valve closure; (7) valve behaviour was not affected significantly by fluid viscosity.

The closing behaviour of the valve as determined in animal experiments under different hemodynamic circumstances showed reasonable agreement with the valve closure as predicted by theory. However, due to the simplifications assumed in the model, the theoretical description must be used tentatively, especially with large pressure changes within the valve orifice during the cardiac cycle, with low peak flows or with high Strouhal numbers.

INTRODUCTION

Recently we have studied aortic valve motion during the cardiac cycle in model experiments as well as in open-chest dogs. Some insight into valvular closing during deceleration of the main stream has been obtained from model studies in a two-dimensional analogue of the aortic valve (van Steenhoven and van Dongen, 1979). This study showed that a simplified quasi-one-dimensional description of the flow in the main stream, and assuming constant pressure on the sinus side of the cusp, gives a good qualitative idea about the initial phase of valve closure. To verify this model, aortic valve motion during the cardiac cycle was studied in open-chest dogs, using direct high-speed cinematography (van Steenhoven et al., 1981). Some findings in this study indicated that the variations in the behaviour of the aortic valve from animal to animal could result from differences in hemodynamic conditions. These differences could also explain some discrepancies between in vivo and model results as far as the closing behaviour of the valve is concerned, because in the latter the influences of some of these hemodynamic variations are not taken into account. Finally, prosthetic valve behaviour was compared with that of the natural aortic valve and a suggestion was made for the application of the aortic valve closing mechanism to the design of a heart valve prosthesis (van Steenhoven et al., 1982).

The aim of the present experimental animal study was to test the validity of the previously mentioned theoretical model under various hemodynamic conditions and to investigate the influence of aortic pressure, aortic flow, heart rate and fluid viscosity on aortic valve behaviour during the cardiac cycle. Since complete valve behaviour cannot be described in one single number, the effect of each variable on valve behaviour cannot be studied independently nor analysed by multiple regression techniques. Therefore, the influence of the hemodynamic variables on valve behaviour was investigated in a comparative way. To study the influence of one factor, we compared the valvular behaviour curves together with the corresponding aortic flow curves of two experiments in which the hemodynamic factors were similar except for the one to be evaluated. Next, the experimentally determined valvular closure was compared with the results obtained in the theoretical model.

METHODS AND MATERIALS

Experimental procedure

Experiments were performed on 15 mongrel dogs of either sex, unknown age and ranging in weight from 25
to 45 kg. The animals were premedicated with Hypnorm® (1 ml/kg body weight i.m.; 1 ml Hypnorm® contains 10 mg fluanison and 0.2 mg fentanyl base). Anesthesia was induced with sodium pentobarbital (10 mg/kg body weight i.v.) and, after endotracheal intubation, was maintained with oxygen–nitrous oxide. Ventilation was kept constant during the experiment with a positive pressure respirator (Pulmonat).

The ECG was derived from limb leads. The chest was opened through the left fifth intercostal space and the heart was suspended in a pericardial cradle. Millar catheter-tip micromanometers (PC 470) were used to measure aortic ($P_a$) and left ventricular pressure ($P_v$). Left atrial pressure ($P_{la}$) was measured with a saline filled catheter and an external pressure transducer (Ailtech). Aortic blood flow ($q_a$) was measured with an electromagnetic flow probe, mounted on the ascending aorta. The probe was connected to a sine-wave electromagnetic flow meter with a carrier frequency of 600 Hz and an upper frequency response of 100 Hz–3 dB (Transflow 600). End-diastolic flow in the aorta was used as zero-reference. The flow probes were calibrated in vitro, previous to the experiments. Qualitatively no significant influence of the fiberscope (see below) on the flow curve could be detected, which indicates that the accuracy of the flow measurement is hardly affected by the presence of the scope. Furthermore, it is assumed that the fiberscope and aortic pressure and flow measurement do not affect valve behaviour. The variables to be measured were recorded on a multi-channel physiological recorder (Schwarzer) and on an electromagnetic tape recorder (Ampex PR 2200). The upper frequency response of the recording system, which is limited by the physiological recorder, was 280 Hz–3 dB.

The technique for high-speed recording of aortic valve movement has been described in detail elsewhere (van Steenhoven, 1979; van Steenhoven et al., 1981). These recordings were made at a film speed of 200 frames/s using a thin (4 mm in diameter) flexible fiberscope. This optical device was placed in front of the valve through the left carotid artery. In water the optical system has an angle of vision of 45°. The coupling of optical and electrical signals was achieved using a 50 Hz timing signal on both film and tape recorder.

This technique requires the replacement of blood by a transparent liquid. Blood was replaced with two roller-pumps, the one connected to the left atrium and the other to the femoral artery. The second pump was essential to maintain peripheral arterial blood pressure at physiological levels. Free outflow occurred through a cannula in the pulmonary artery. A Tyrode solution either with (3.3 g%) or without gelatine was used as the transparent liquid. The fluid had a temperature of 37° and was saturated with a gas mixture consisting of 5% CO2 and 95% O2. A schematic representation of the experimental set-up is given in Fig. 1.

Data processing

A sequence of film frames of five successive heart beats with a regular rhythm was chosen for analysis. An additional condition for the choice was a reasonable degree of uniformity of the instantaneous aortic flow and the aortic and left ventricular pressure curves during these five beats, as far as duration of ejection, maximum values of the parameters and the R–R interval were concerned. These frames were analysed with an analysing projector (Analector, Oude Delft) and drawings were made of the instantaneous cusp positions. From the drawings the instantaneous area of valve opening was measured with a planimeter (Ott 31). For proper evaluation of the valvular behaviour, the orifice area of the aortic valve was calculated as an instantaneous function of time and compared with the instantaneous flow within the valve. The measured aortic flow signal was shifted over about 8 ms because of the position of the flow probe with respect to the valve and the electronic delay in the flowmeter system ($\approx 1.5$ ms). The time shift due to the position of the probe was calculated from the measured distance between flow probe and valve, assuming a pulse wave velocity of 4 m/s (Nichols and McDonald, 1972).

Figure 2 shows a schematic representation of the relations between aortic pressure, aortic flow and valve motion under normal conditions, where $t = 0$ coincides with the onset of the deceleration of aortic flow. Table 1 summarizes the hemodynamic quantities which were measured in the animals. Stroke volume was determined by integrating the electromagnetic flow signal. To reduce the stochastic errors, the influences of artefacts and the effect of physiological
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Fig. 2. Schematic representation of the relationships between aortic pressure (P_a), aortic flow (q_a), and valve behaviour under normal circumstances (a = valvular orifice area fraction; t = closing parameter).

differences from heart beat to heart beat, all those quantities were averaged over five cardiac cycles. The experimental results will be presented as the mean value with the 95% confidence interval of the mean (Table 2). The relation between variables and parameters will be expressed in the correlation coefficient r, as found from linear regression analysis.

The curves of the instantaneous valvular orifice area as well as the corresponding instantaneous aortic flow signals were averaged over five successive heart beats by normalizing these variables to their maximum value. The time scale (the width of the flow curve) was normalized by relating time to the time interval between 25% of the peak flow during acceleration and 5% of the peak flow at the end of deceleration. Next, the instantaneous flow curve following each normalized time step, being 1/80 of the normalized time unit, was averaged over five heart beats. This finally resulted in an average normalized instantaneous flow curve. Because of the coupling to flow, the curve of valve orifice area fraction (a), defined as the ratio of the instantaneous and maximum orifice area, was also of a normalized nature with respect to time. Similar to the procedure followed for the aortic flow, the valve orifice area fraction values were averaged at each time step. Finally both the normalized average flow and the normalized average curve of valve orifice area fraction were restored to physical quantities, i.e. real flow and real time, using the mean values of the selected five heart beats. This resulted in a curve of mean valvular behaviour in relation to a curve of mean instantaneous aortic flow. This averaging procedure has been described and discussed in more detail earlier (van Steenhoven, 1979; van Steenhoven et al., 1981). To get insight in the reliability of the calculation of the mean curves, we determined the 95% confidence interval of the mean, as derived from five successive measurements, averaged over all time steps. For the flow values this resulted in a mean confidence interval of 3.5% of peak flow, whereas for the valve orifice area fraction this value was approximately 0.06.

Comparison with the results obtained in a model of aortic valve closure

The results of the in vivo experiments were compared with those obtained in a theoretical model of aortic valve closure, which is based upon the closing behaviour as observed in a two-dimensional analogue of the aortic valve (van Steenhoven and van Dongen, 1979). During deceleration of the main stream, the following observations were made [Fig. 3(b)].

1. The shape of the cusp does not change very much; it rotates around its attachment line.
2. The main stream velocity profile beneath the cusp remains nearly flat.
3. A region of recirculation is clearly visible behind the cusp. The flow pattern shows some resemblance to the phenomenon of boundary layer separation.
4. A vortex is present in the sinus during the stationary phase. The maximum velocity in the sinus seems to be much lower than that in the aorta.

On the basis of these experimental results, a simplified theoretical model was designed in which the pressure on the sinus side of the leaflet is assumed to be constant and equal to the pressure underneath the free edge of the cusp. From observation 2 it is assumed that potential theory can be applied, which means that the valve behaviour is hardly affected by the viscosity of an inelastic fluid. Two additional assumptions were made: the leaflet is straight and the mean pressure difference across the leaflet, because of its negligible mass, is equal to zero. From this model, an equation is obtained which directly relates the aortic fluid velocity within the valve to the displacement of the leaflet.

For comparison of the theoretical model with the animal experiments, the model has to be extended to the three-dimensional situation. For this purpose, the cusps are assumed to be shaped as a truncated cone and the aorta is assumed to be a rigid tube. As the model only holds for the valve closure during flow deceleration, the predicted valve behaviour will be expressed
Table 1. Definitions of the hemodynamic quantities measured in the experiments

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\eta$</td>
<td>viscosity, which was found to be $10^{-3}$ Ns/m² for a Tyrode solution and $3 \times 10^{-5}$ Ns/m² if gelatine is added</td>
</tr>
<tr>
<td>$HR_{p}$</td>
<td>heart rate as derived from the R–R interval of the ECG</td>
</tr>
<tr>
<td>$P_{a}$</td>
<td>maximum value of the systolic aortic pressure</td>
</tr>
<tr>
<td>$\Delta P_{a}$</td>
<td>aortic pressure drop, defined as the difference between the maximum systolic aortic pressure and the aortic pressure value at the moment of the incisure in the pressure curve $P_{a}$</td>
</tr>
<tr>
<td>$q_{a max}$</td>
<td>peak flow, defined as the maximum of the aortic flow curve</td>
</tr>
<tr>
<td>$q_{a min}$</td>
<td>back flow ratio, defined as the ratio of the maximum back flow early in diastole and the peak flow</td>
</tr>
<tr>
<td>$SV$</td>
<td>stroke volume, which is represented by the area under the aortic flow curve</td>
</tr>
<tr>
<td>$a = \frac{A}{A_{max}}$</td>
<td>valve orifice area fraction, defined as the ratio of the instantaneous valve orifice area $A$ and the maximum orifice area $A_{max}$</td>
</tr>
<tr>
<td>$\lambda^2 = \frac{A}{A(t = 0)}$</td>
<td>closing parameter, which is during the deceleration phase of systolic aortic flow defined as the ratio of the instantaneous valve orifice area and the orifice area at the onset of deceleration $A(t = 0)$</td>
</tr>
<tr>
<td>$t(a = 1) - t(a = 0)$</td>
<td>opening time, defined as the time delay between the moment of onset of valve opening $t(a = 0)$ and the moment that the valve has opened completely $t(a = 1)$</td>
</tr>
<tr>
<td>$q_{a0}(a = 1)$</td>
<td>relative opening flow, defined as the ratio of the instantaneous aortic flow at the moment of complete valve opening $q_{a0}(a = 1)$ and the peak flow</td>
</tr>
<tr>
<td>$a(t = 0)$</td>
<td>mid-systolic valve orifice area fraction, defined as the value of $a$ at the onset of the deceleration of aortic flow</td>
</tr>
<tr>
<td>$a(t_{a0} = 0)$</td>
<td>end-systolic valve orifice area fraction, defined as the value of $a$ at the onset of back flow</td>
</tr>
<tr>
<td>$t(q_{a0} = 0) - t(a = 0)$</td>
<td>closing time delay, defined as the time span between the moment of maximum back flow in the valve and the moment of complete aortic valve closure $t(a = 0)$. The moment $t(a = 0)$ is derived from the point of intersection of the $a-t$ curve and the abcissae $a = 0$</td>
</tr>
<tr>
<td>$r_{o}$</td>
<td>aortic radius just distal of the valve, as measured in the unloaded postmortem heart</td>
</tr>
<tr>
<td>$St = \frac{\pi r_{o} \lambda^2}{q_{a0 max}}$</td>
<td>Strouhal number, where $r$ represents the deceleration time of systolic aortic flow</td>
</tr>
</tbody>
</table>

Next, on the basis of equation (I) the theoretical closing behaviour in terms of the closing parameter $\lambda^2$ was calculated. The mean curves are presented in such a way that theory and experiment can readily be compared.

RESULTS

During perfusion the hemodynamic variables changed as compared with the control situation due to differences in the viscosity of the perfusion liquid, as well as in the inflow rate into both the left atrium and the femoral artery. Because of this dependency of the hemodynamic situation on viscosity and inflow rates, we were able to study aortic valve behaviour under different hemodynamic conditions. In nine experiments the film images were of a quality sufficient for reliable analysis. In these experiments twelve measurements were performed. The hemodynamic data with the 95% confidence interval of the mean of these measurements are listed in Table 2.
In experiment M51-H only one heart beat could be analysed reliably. Therefore, no confidence intervals are given.

In experiment M55 the onset of flow deceleration preceded the moment of complete valve opening, which makes it irrelevant to determine the relative opening flow and the mid-systolic valve orifice area fraction.

The mean values and the 95% confidence intervals of the mean are based on 5 measurements.

Table 2. Hemodynamic data for nine dogs, all quantities being defined in Table 1.1)

<table>
<thead>
<tr>
<th>n</th>
<th>HR</th>
<th>r₀</th>
<th>μa₀</th>
<th>μa₀ - μa₁</th>
<th>q₀</th>
<th>q₀ min</th>
<th>q₀ max</th>
<th>SV</th>
<th>t(l=1)-t(l=0)</th>
<th>q₀(a=1)</th>
<th>q₀(a=0)</th>
<th>al(t=0)</th>
<th>al(q₀=0)</th>
<th>t(q₀=0)</th>
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<td>0.88</td>
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<td></td>
<td></td>
<td></td>
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<td>0</td>
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<td>3</td>
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<td>0.3</td>
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<td>0.16</td>
<td>0.16</td>
<td>2</td>
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<td>2</td>
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<td>11.2</td>
<td>7.1</td>
<td>175</td>
<td>0.06</td>
<td>14.3</td>
<td>2</td>
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<td>5.4</td>
<td>117</td>
<td>0.08</td>
<td>8.8</td>
<td>4</td>
<td>0.11</td>
<td>0.04</td>
<td>20</td>
<td>0.16</td>
<td>20</td>
<td>0.16</td>
<td></td>
</tr>
<tr>
<td>M 50-H</td>
<td>3</td>
<td>65</td>
<td>5.0</td>
<td>11.8</td>
<td>8.2</td>
<td>179</td>
<td>0.01</td>
<td>17.7</td>
<td>28</td>
<td>0.64</td>
<td>0.91</td>
<td>0.09</td>
<td>0.09</td>
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<tr>
<td>M 51-L</td>
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<td>7.8</td>
<td>2.6</td>
<td>99</td>
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<td>222</td>
<td>0.05</td>
<td>21.0</td>
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<td>1.4</td>
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<td>1.4</td>
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<tr>
<td>M 55-L</td>
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<td>212</td>
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<td>9.5</td>
<td>2.4</td>
<td>79</td>
<td>0.54</td>
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<td>50</td>
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<tr>
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<tr>
<td>M 55-H</td>
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<td>5.8</td>
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<tr>
<td>M 56</td>
<td>3</td>
<td>63</td>
<td>4.5</td>
<td>13.9</td>
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<td>0.81</td>
<td>5.5</td>
<td>0.024</td>
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</tr>
</tbody>
</table>

(1) The mean values and the 95% confidence intervals of the mean are based on 5 measurements.

(2) In experiment M49 only such a minor part of the leaflets could be seen during complete valve opening that the geometry could not be determined accurately. For this reason the valve behaviour in this experiment is not considered.

(3) In experiment M51-H only one heart beat could be analysed reliably. Therefore, no confidence intervals are given.

(4) In experiment M55 the onset of flow deceleration preceded the moment of complete valve opening, which makes it irrelevant to determine the relative opening flow and the mid-systolic valve orifice area fraction.
From theory it is found that valve closure is only affected by the aortic velocity and its time derivative. However, it should be noticed that the theory only holds for valve closure during the phase of flow deceleration and that the effects of fluid viscosity and geometrical distortion caused by pressure changes within the valve orifice during the cardiac cycle are neglected. Therefore, the influences on valve behaviour were evaluated for aortic pressure [both the systolic aortic pressure drop (= pulse pressure) and the maximum value of the aortic pressure], peak aortic flow, stroke volume, fluid viscosity and heart rate. Since complete valve behaviour cannot be expressed in one single number, it is impossible to study the effect of each variable on valve behaviour separately. Therefore, we investigated the influences of the hemodynamic factors on valve behaviour in a comparative way through the mean curves of valve behaviour and aortic flow. For comparison two particular experiments were chosen in which the hemodynamic factors were similar except for the one to be evaluated.

In Fig. 4 the influence of the aortic pressure drop on aortic valve behaviour is shown. In case of a large aortic pressure drop, early valve closure during flow deceleration is faster than in the case of a small pressure drop. It is also observed that the differences between theory and animal experiment are of a different nature. The graphs suggest that the agreement between experiment and theory is better in the case of a small systolic aortic pressure drop than in the case of a large one. This impression is supported by the curves shown in Figs 5 and 7, which were also recorded at a relatively small aortic pressure drop.

The influence of systolic aortic pressure on valvular behaviour is illustrated in Fig. 5. The most remarkable effect appeared to be present during the acceleration phase of the aortic fluid. In case of high systolic aortic pressure, the valve began to close early. At the onset of flow deceleration about 16% of closure was already

Fig. 4. Relationship between aortic flow \( q_{ao} \) and aortic valve behaviour (a) at small (left panel) and large (right panel) aortic pressure drops. The solid lines represent the experimental results. Systolic valve closure as determined with equation (1) is represented by the dotted line. Zero time corresponds to onset of flow deceleration.
accomplished. At low systolic aortic pressure, aortic valve closure started later in the cardiac cycle. This significant effect in one experiment could not be confirmed when using all the experiments as indicated by the data in Table 2 where no distinct relationship could be found between \( a(t = 0) \) and \( P_{ao} \) (\( r = 0.64 \)).

The data in Table 2 indicate that the relative opening flow decreases at increasing stroke volume \( (r = 0.82, \text{Fig. 6(a)}) \) or peak aortic flow \( (r = 0.73) \) and that the back flow ratio in the aorta increases at larger end-systolic valve orifice area fractions [Fig. 6(b)].

In one particular experiment (M55) three different values of peak aortic flow were available: one in the low region (M55-L), one in the high region (M55-H) and another in between the two regions (M55-M). The flow curves and the corresponding valvular behaviour curves are shown in Fig. 7. In the three different situations the valve orifice area fraction was determined using the maximum valve orifice area of experiment M55-H. The graphs suggest that the shape of the valvular area, when the valve has fully opened, is related to peak flow and stroke volume. The higher the peak flow the better a circular geometry was approximated (Fig. 8). At very low peak flow values, the valve behaviour was quite different from in other situations. In this condition, the onset of aortic flow deceleration clearly preceded the moment of complete valve opening. It is obvious that under these circumstances, the mechanism of valve closure is disturbed and that as a consequence the theory does not hold any more.
Fig. 7. Relationship between aortic flow ($q_{ao}$) and aortic valve behaviour (a) at different values of peak aortic flow. The solid lines represent the experimental results. Systolic valve closure as determined with equation (1) is represented by the dotted line. Zero time corresponds to onset of flow deceleration. In the three different situations, the valve orifice area fraction was determined using the maximum valve orifice area of experiment M55-H.

Viscosity did not have a relevant effect on valve behaviour [cf. experiments M47 (low viscosity) and M54 (high viscosity) in Figs 5 and 4, respectively]. The influence of heart rate on valve behaviour is illustrated by comparing Figs 5 (M47—low heart rate) and 7 (M55—high heart rate). In these experiments, the values of other variables were comparable (see Table 2). A rise in heart rate is associated with a more frequent opening and closure of the valve. The opening flow is significantly less at low heart rates (see also Table 2). Furthermore, the valve closure during the acceleration phase of aortic flow is absent at high heart rates. On the other hand, the agreement between theory and experiment with regard to valve closure remains fair, suggesting that the mechanism of valve closure is not affected by changes in heart rate.

DISCUSSION

Experimental method

The experimental procedure used provides a valuable method for studying the behaviour of the aortic valve in open-chest dogs (van Steenhoven et al., 1981). The results of each experiment are given as a graphical representation of aortic valve behaviour in relation to aortic flow. The latter variable was chosen since in the theoretical model the aortic flow is our main reference in the evaluation of valve behaviour. In order to reduce stochastic errors, the curves of valvular behaviour, expressed in terms of the valve orifice area fraction $a$, as well as the corresponding aortic flow signal were averaged over five successive heart beats. We are quite aware that as a consequence of the rather complex procedure of data processing the mean curves are of a qualitative rather than a quantitative nature.

To study the influence of one hemodynamic factor, we compared the valvular behaviour curves together with the corresponding aortic flow curves of two experiments in which the hemodynamic factors were similar except for the one to be evaluated. It should be noticed that such an analysis is open to criticism. First of all, interactions between the variables are neglected. Secondly, some differences might be attributed to specific biological differences between the various animals. Thirdly, in animal experiments it is impossible
to change only one variable and to keep all the others constant. The data in Table 2, however, illustrate that the other variables were rather similar for each set of two experiments discussed. Furthermore, the model prediction is quite insensitive for slight variations in $u_o$, and therefore in $a_{max}$. For example, even a variation of a factor of two is not sufficient to explain the large deviation between theory and experiment in the case of a large systolic aortic pressure drop. Therefore, in spite of the limitations, we feel that the procedure is sufficiently sound to get a qualitative picture of the influence of hemodynamic factors on valve behaviour.

**Animal experiments**

The findings in the present study indicate the following. (1) The relative opening flow decreases at increasing stroke volume and peak aortic flow. The opening flow is significantly less at low than at high heart rates. (2) The shape of the valvular area, when the valve has fully opened, is related to peak flow and stroke volume. The higher the peak flow the better a circular geometry was approximated. (3) Early valve closure during the acceleration phase of the aortic fluid is affected by the systolic pressure level. The higher the pressure the earlier valve closure starts in the cardiac cycle. However, this early valve closure is significantly less at high heart rate. (4) The initial phase of valve closure during flow deceleration is influenced by the systolic aortic pressure drop. Initially valve closure is faster in case of a large than in case of a small pressure drop. (5) The backflow ratio in the aorta increases if the end-systolic valve orifice area fraction is larger. (6) A rise in heart rate is associated with a more frequent opening and closure of the valve. (7) Fluid viscosity does not have a relevant effect on arterial valve behaviour.

The opening flow is similarly affected by stroke volume, peak aortic flow and heart rate. Since stroke volume is generally reduced at higher heart rates and lower peak flow values, it is likely that stroke volume is the main determinant of the relative opening flow. This is supported by the observation of Laniado et al. (1976) that at higher stroke volumes, complete valve opening was reached early in the cardiac cycle. The finding that the shape of the valvular area at complete opening is related to peak flow and stroke volume, is supported by the observations of Stein and Munter (1971) and Laniado et al. (1976). The present observation that at physiological peak flow values the orifice has a practically circular geometry confirms previous findings of Hider et al. (1966), Padula et al. (1968), Stein (1971), Thubrikar et al. (1977) and van Steenhoven et al. (1981).

The decrease in valvular orifice area during the flow acceleration phase, as reported here, was also observed in some of the experiments of Laniado et al. (1976). The mechanism involved is not yet clear. Even so the observations made in our study, that the systolic aortic pressure level affects this early closure and why a higher heart rate opposes this effect, is still incompletely understood.

It is possible that the change in valvular orifice area, in particular at the onset of flow deceleration, is affected by the decrease in aortic diameter during this part of the cardiac cycle. Thubrikar et al. (1977) and Brewer et al. (1976), after all, found an expansion of the aortic root during systole of an order of magnitude of 15% at physiological aortic pressure levels. The decrease in aortic diameter is likely to be more pronounced at larger systolic aortic pressure drops. Because of the changes in aortic diameter it is difficult to define the moment where reduction in valvular opening becomes merely dependent on leaflet movement. Therefore, the effects of the other hemodynamic variables were studied at relatively low systolic aortic pressure drops.

The observation that complete valve closure coincides with the moment of maximum backflow in the valve, explains why the backflow is necessary to close the valve completely. It is higher at larger end-systolic valve orifice area fractions.

The present experimental results indicate that fluid viscosity does not affect valve behaviour during the cardiac cycle, which is an extension of our previous findings for the valvular closing phase (van Steenhoven et al., 1981).

**Theoretical prediction of aortic valve closure**

The aim of the present experimental animal study was to test the validity of a theoretical model, based upon the observations in a two-dimensional analogue of the aortic valve. The closing behaviour as calculated from theory and as measured in in vivo experiments under different hemodynamic conditions, generally shows a reasonable agreement in spite of the simplifications used in the model.

It is observed that the difference between theory and animal experiment is affected by the systolic aortic pressure drop. The smaller the pressure drop the better the agreement. This may be explained by neglecting in the model geometrical distortion caused by pressure changes within the valve orifice during the cardiac cycle.

At very low stroke volume and peak flow levels, the theoretical model predicts the valve to close earlier than experimentally found. This is probably caused by a disturbance of the mechanism of valve opening and closure as illustrated by the observation that flow deceleration starts before valve opening is completed (Fig. 7).

The observation that the agreement between theory and experiment with regard to valve closure remains fair at high heart rates, is in agreement with the findings in the analogue (van Steenhoven and van Dongen, 1979). There we found that the model was applicable for Strouhal numbers (see Table 1) lower than 0.2. As shown in Table 2 this condition is satisfied for all the experiments, except for experiment M55-L where, as discussed earlier, valve behaviour was completely disturbed.

In summary we may conclude that there is reason-
able agreement between theory and experiment, suggesting that the model describes the mechanism of valve closure during flow deceleration fairly well. Nevertheless, the model must be tentatively used especially at higher systolic aortic pressure drops and at low peak aortic flows or stroke volumes.

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