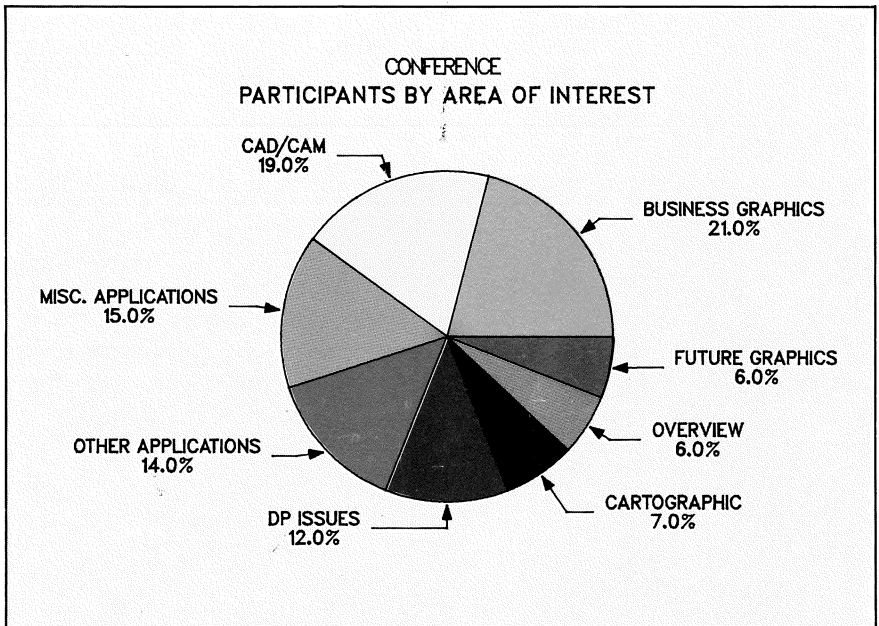


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A COMPUTER TEST CHAMBER FOR MODELLING NATURAL AND PROSTHETIC MITRAL VALVES

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ABSTRACT

We have developed a computational method for modelling the *in vivo* fluid dynamics of the mitral valve. A digital computer provides a discrete time-step solution of the equations of motion of blood in the heart. The fluid properties, the muscular properties and initial geometry of the contractile chamber, and the mechanical constraints of the valve under consideration are specified as input. The subsequent motions of the fluid, chamber, and valve are computed simultaneously, taking into account the complex interaction between the moving fluid and the flexible boundaries of the chamber and valve. This approach permits prediction of the hemodynamic performance of a wide variety of valves, natural and prosthetic, under normal or pathological physiological conditions. Performance is judged by graphical presentations of both some clinically-familiar physiological variables and some not so familiar fluid mechanical variables which would be very difficult to obtain *in vivo*. In particular we make use of a computer-generated movie of the fluid, chamber and valve motions throughout the computation.

INTRODUCTION

We have developed a computational method for modelling the *in vivo* fluid dynamics of the mitral valve. A digital computer is used to solve the coupled equations of motion of the blood, the muscular heart wall and the valve. The results of the computation represent a prediction of how a specified valve, natural or prosthetic, will perform under specified physiological conditions.

These results have both clinical and engineering significance. The hemodynamic performance of the mitral valve determines both the trans-valvular pressure difference needed to sustain the required cardiac output and the pressure in the pulmonary circulation. Detailed knowledge of the distributions of velocity and pressure within the ventricle is important in assessing disease states and in assuring proper placement of instrumentation in the ventricle.

Engineering development of heart valve prostheses is complicated by the coupling of the moving blood, heart wall and valve. Because the motion of the occluder influences and is itself influenced by the fluid motion, simple predictions of valve performance are not possible. The evaluation of an existing design is made difficult by the relatively limited data available in clinical or *in vivo* experimental situations, and by the inflexibility of present pulse-duplicators. In addition, the experimental optimization of a proposed valve design requires the time-consuming and expensive manufacture of prototype valves with variations in design parameters.

Theoretical models have been, up to now, almost exclusively restricted to flows past stationary valves. Clearly, the pattern of flow past a stationary object differs greatly from the pattern produced by an object moving with the fluid. Even if the correct flow rate and valve motion as functions of time are known from experimental data, the predicted flow pattern will be incorrect. Moreover, for an untested design, the flowrate and occluder motion are both unknown in advance, precluding any possibility of a correct flow pattern prediction.

The model we have developed is not restricted by the limitations described above. Only the fluid properties, the muscular properties and

initial geometry of the contractile chamber, and the mechanical constraints of the valve of interest are specified. The subsequent coupled motions of the fluid, chamber and valve are computed simultaneously. The results of this approach can be used to optimize the parameters of a given design without actually fabricating a prototype for each set of parameters. It can also be used to study the sensitivity of a given valve to changes in the physiological situation in which the heart and valve must operate.

In fairness it must be pointed out that, due to limitations in computer speed and storage, our results are presently restricted to a two-dimensional model and to a Reynolds no. of about 1/25 the normal physiological value. Despite this, the agreement between the results of our computations and *in vivo* experimental results, which is described below, is excellent. We believe, based in part on the results outlined here, that this is the most fully developed model for application to the analysis of valve performance and design currently available.

The methods we employ and much of the results described below are given in detail elsewhere (Peskin (1977), Peskin and McQueen (1980), McQueen, Peskin and Yellin (1981)). We obtain two types of results: those which can be compared directly with the results of animal experiments, and those which would be very difficult to obtain *in vivo*. The first type provides a check that we have successfully established accurate model physiological conditions for our computations. The second provides new information which may motivate new lines of physiological investigation. Both types will be presented here. In most the utility of the graphical presentations shown, which are precisely those we use ourselves, is self-evident. Consequently the discussion of this aspect is usually brief and simple, except where more detail is demanded.

ANATOMY OF THE MODEL

The anatomy of interest, shown in Fig. 1a & 1b, is a cross-section of the heart using a plane which bisects the two major leaflets of the mitral valve. This plane cuts through the left ventricular outflow tract and the aortic valve, and it passes between the two papillary muscles which are located on either side of the plane. The tips of the papillary muscles give rise to the chordae tendineae which insert on the margins of both valve leaflets. Despite their not being visible in this view, the papillary muscle and chordal forces on the leaflets have a component in the plane of interest.

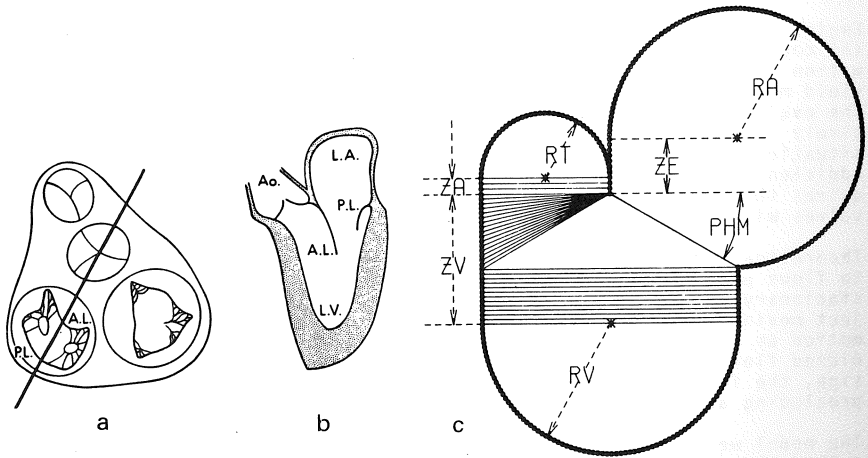


Fig. 1. Anatomy. (a) Plane of interest. (b) Cross section of the heart in the plane of interest. (c) Model test chamber without valve.

The initial geometry of our test chamber, which models the left heart (less the mitral and aortic valves), is shown in Fig. 1c. The chamber is constructed from a series of circular and straight line segments which roughly approximate the shape of the heart. This somewhat unrealistic shape is used for simplicity and could be altered as necessary. The boundary is represented by a series of closely spaced points immersed in fluid and connected by links representing the elastic and muscular fibers of the heart wall. The long links which cross the chamber represent the the components of forces from circumferential (i.e. out-of-the-plane) fibers. These are required in our two-dimensional model to prevent the chamber from becoming circular under internal pressurization.

Our model of the mitral valve is shown in Fig. 2a. Initially the leaflets are two circular arcs, tangent to each other at their free margins. The valve is equipped with adjustable parameters controlling the details of its geometry. As with the chamber, the leaflet shapes are somewhat simplified and arbitrary. However, the relatively few parameters required to specify the geometry facilitate systematic studies of the functional anatomy of the valve. Like the real valve, our model is asymmetric, and its larger (anterior) leaflet opens towards the ventricular outflow tract, while its smaller (posterior) leaflet opens towards the posterior wall. The papillary muscles and chordae are modelled by the Y-shaped apparatus, shown in Fig. 2b, linking the apex to the tips of the leaflets. This represents the component of the chordal restraining forces in the plane of interest.

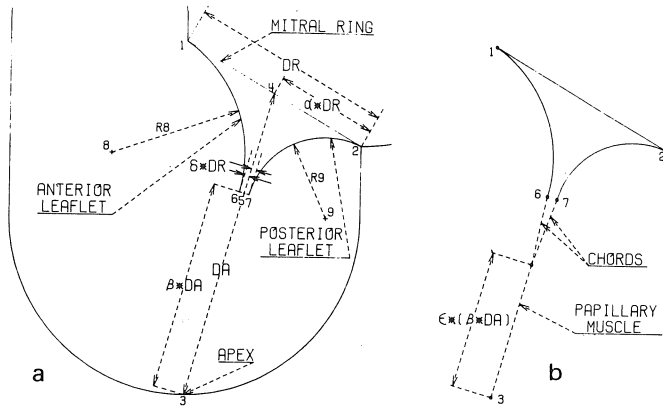


Fig. 2. (a) Natural valve model in chamber. (b) Chordal restraint model.

Models of three prosthetic valves (caged-ball, flat pivoting disc, and curved pivoting disc) are shown in Fig. 3. Unlike the flexible natural valve leaflets, the occluders of the prostheses retain their original shape while moving. Rigidity of the ball is maintained by way of links similar to bicycle wheel spokes. Rigidity of the discs is maintained by bending resistant links. Other constraints such as the ball-valve cage and the pivoting mechanism of the discs are appropriately constructed, but are not shown in the Figures.

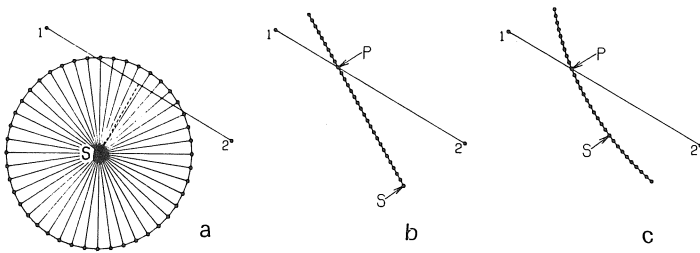


Fig. 3. Model prostheses. (a) Caged ball. (b) Flat and (c) Curved pivoting discs.

Pulmonary venous return is modelled using a pressure-controlled source in the atrium. The aortic valve is not present, and our results are limited to ventricular diastolic filling, the period in which the dynamic behavior of the mitral valve is most significant.

RESULTS

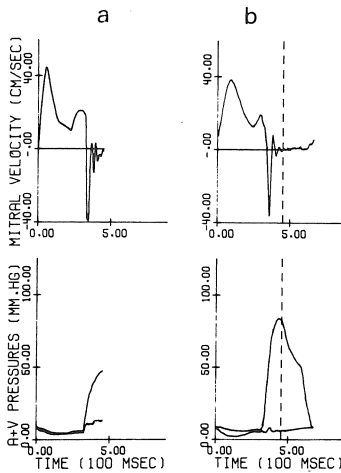


Fig. 4. Comparison of (a) computed and (b) canine experimental results.

Fig. 4 compares computed and experimental records of velocity and pressure. The experimental data, taken from the canine laboratory of E.L.Yellin (see Laniado, et.al. (1973) for experimental methods), is normally available to us in the graphical form shown (redrawn from strip-chart recordings). A simple by-eye comparison indicates the degree to which our computation successfully models actual physiology. We feel that the agreement between computed and experimental mitral velocities is excellent. Computed and experimental pressures are also similar, except that the computed atrioventricular pressure difference is noticeably smaller than the experimental, indicating a somewhat less stenotic model valve. This may be a consequence of the two-dimensional nature of the model, or may be due to a stenotic effect of the electromagnetic flowmeter placed around the mitral ring in the experimental preparation. In any event, comparisons between different model valves should be meaningful.

Fig. 5 shows the streamline patterns for the model natural valve at selected evenly-spaced times during ventricular diastole, atrial systole and early ventricular systole. Bear in mind that streamlines necessarily cross moving boundaries; a boundary with no streamlines crossing it is at rest. Regions where streamlines are relatively closely spaced is one in which the velocity is relatively large.

In frame (1) of Fig. 5 the valve is opening. The jet of blood is intense only between the leaflets. The jet does not extend to the apex at this early time. In frame (2) the valve has reached its maximally open position. The tips of the leaflets are restrained by the chordae, and the papillary muscle tension causes a slight indentation at the apex. Frame (3) shows vortex formation at the cusp margins. The vortex streamlines are closing the valve. This closure movement persists in frame (4) as the vortices are shed. By frame (5), the vortex-dominated flow pattern of mid-diastole is well established, and the valve is essentially at rest. As the vortex flow-pattern develops, the jet of blood gradually extends, frames (3)-(8), towards the apex of the left ventricle. Atrial systole strengthens the jet and the vortices. In frame (13) the valve is just beginning to close under the influence of the vortices when ventricular systole intervenes, frame (14). The flow pattern of frame (14) shows backflow at the level of the mitral ring with forward flow persisting at the level of the cusp margins. The valve is effectively closed in frame (15).

Fig. 6 shows the pressure contours (isobars) of the natural valve at times corresponding to those of Fig. 5. The characteristic minima of pressure at the centers of the shed vortices are simple consequences of centrifugal force. The apparent thickness of the chamber walls is due to the large pressure difference across the tensed curved boundary.

Fig. 5 & 6 are notable not only because similar results would be very difficult to obtain in vivo, but also for the remarkable amount of descriptive information contained in a fairly compact graphical form.

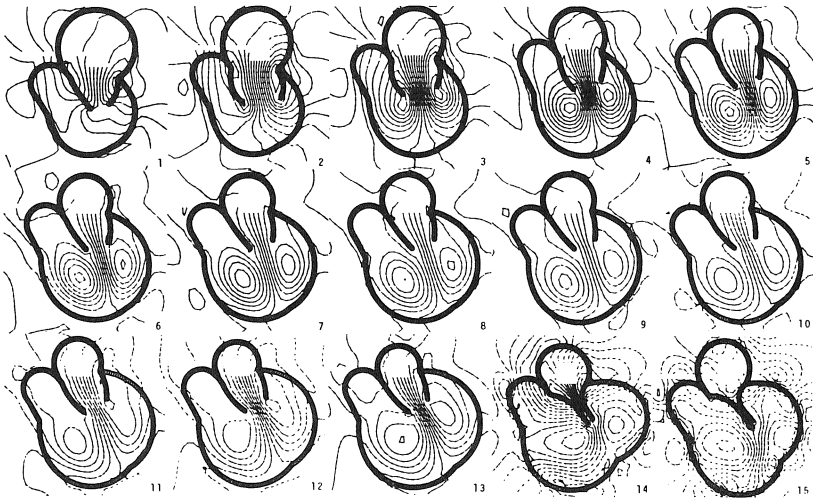


Fig. 5. Streamlines of the natural valve

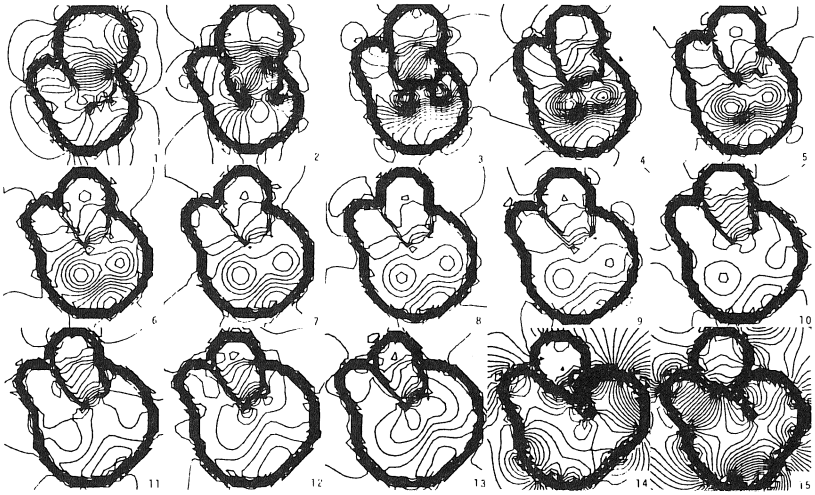


Fig. 6. Pressure contours of the natural valve

Figs. 7 and 8 show comparisons of streamlines and pressure contours for natural and various prosthetic valves at corresponding times for the same physiological test conditions. Frames c and d show the effect of a change in pivot point location on the performance of the flat pivoting disc valve. In frame d the pivot point is closer to the center of the valve; its angle of opening is significantly smaller, which results in a reduction of flow and an increase in transvalvular pressure difference. We have found that the maximum angle of opening of such a valve depends on the pivot point location, and is determined solely by fluid mechanical forces. That is, in the absence of any constraint built into the valve mechanism, the disc opens to an angle less than 90° and closes successfully during ventricular systole. Furthermore there is a pivot point which maximizes flow through the smaller of the two openings on either side of the pivot point. This choice of pivot point is optimal for reducing stagnation and the possibility of resulting thromboembolism in the "secondary flow".

Frame e shows the effect of adding a small amount of curvature to the disc pivoted as in frame c. Both the maximum angle of opening and the secondary flow can be increased. We have found, however, that too much curvature produces a valve which can open to more than 90° , with the result that the disc continues to open until stopped by contact with the ventricular wall; closure of the valve occurs very slowly with accompanying massive regurgitation into the left atrium.

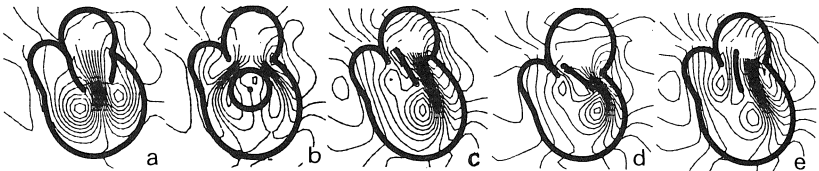


Fig. 7. Streamlines for various valves. (a) Natural valve. (b) Caged ball. (c) Flat pivoting disc. (d) Flat pivoting disc pivoted closer to the center. (e) Curved pivoting disc pivoted as in (c).

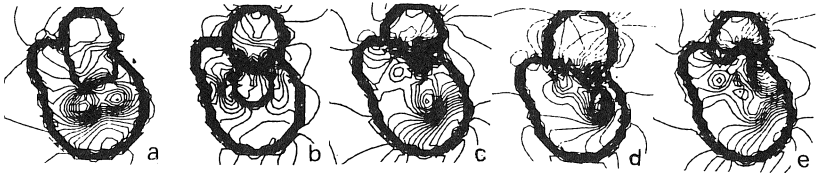


Fig. 8. Pressure contours for valves as in Fig. 7.

Fig. 9 shows a summary comparison of some measures of performance for the natural valve and the caged-ball, flat pivoting disc (two different pivot points) and curved pivoting disc prostheses. The data include mitral velocities, atrial and ventricular pressures, phonocardiograms and echocardiograms (or angles of opening, as appropriate). Presentations of this type allow for rapid evaluation of relative valve performance.

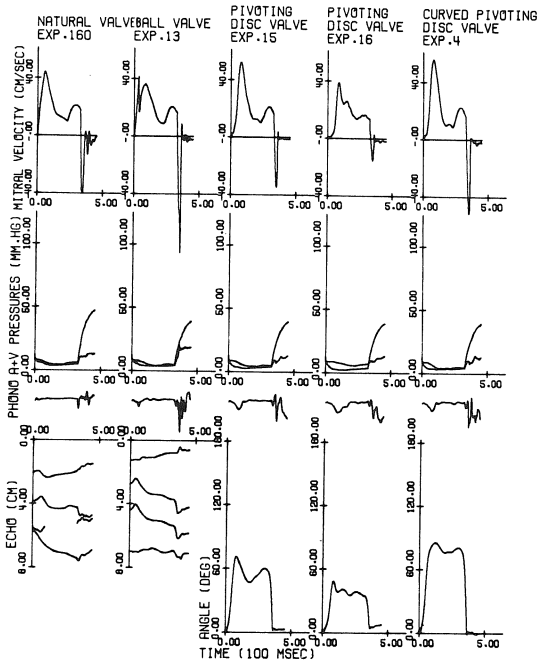


Fig. 9. Comparison of the natural valve and some prosthetic valves.

COMPUTER-GENERATED MOVIES

During each step of the solution of the equations of motion, the computer determines the current location within the domain of the computation of all points on the chamber boundary and on the valve. In addition, the positions of a set of "fluid markers", points placed initially in an orderly array in the fluid and subsequently moving with the fluid, are determined. These points are displayed at their current positions on a computer-controlled cathode ray tube, and the image is photographed by a movie camera. The resulting series of film frames forms a motion picture of the fluid, chamber and valve as the solution

(and hence model diastolic filling) advances. The period of filling (approximately 0.4 seconds) is divided into 640 discrete time-steps, and a film frame is generated every other step. The movie, projected at 24 frames/second, is a slow motion image moving at a speed reduction of about 3/100. Along one side of the image of the chamber are plotted the updated time-histories of mitral flow, atrial and ventricular pressures, and a simulated valve echocardiogram. The overall effect of the movie is that of a strip-chart recording combined with a rather sophisticated cineangiogram of the fluid and chamber motions. Thus the dynamic behavior of the model system can be immediately compared with and used to illuminate clinically-familiar (computed) data describing the system "physiology".

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