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ATRIOVENTRICULAR PRESSURE-FLOW DYNAMICS AND VALVE MOTION

E. L. Yellin, Ph.D., S. Laniado, M.D., C.S. Peskin, Ph.D., and R.W.M. Frater, M.D., Departments of Surgery, Medicine and Physiology, Albert Einstein College of Medicine, Bronx, New York

INTRODUCTION

Physical modeling, mathematical modeling, and animal experiments are used in this paper to construct a picture of the mechanisms that govern the behavior of the mitral valve in its normal and pathological states. Our approach has been multifaceted: we hypothesize that the cardiohemic system can be described by lumped parameters having the properties of resistance, inertance and capacitance. With the help of an electric analog we generate solutions to the equations governing this model and qualitatively compare the results with animal experiments. We then look into the "black boxes" of the system in order to delineate the factors which influence the lumped parameters. This is done by solving the one-dimensional equations of motion for average properties; and by solving the Navier-Stokes equation for distributed properties. The results are all complementary, and consistent with each other and with data clinically available from patients.

THE PHYSICAL MODEL AND ELECTRIC ANALOG OF THE MITRAL VALVE

The physical model is represented by the electric analog shown in Figure 1A. The valve is a visco-elastic structure which normally permits only unidirectional flow, but a backflow path is provided in order to model mitral regurgitation. The normal valve, when closed, will store energy due to its compliance; and when open, will permit flow which will be both dissipative and inertial.

The pressure-flow relations across this model can be generated by the operational amplifier circuit show in Figure 1B. An oscillographic record of the driving pressure gradient which includes a simulated "atrial contraction", and the resulting flow, is presented in Figure 2. The value of the analog lies in its ability to corroborate the existence of the physical properties postulated in the governing equations; to predict and test relationships; and to elucidate other possible interactions. In our hands the electric analog is not used to quantify parameters or coefficients, or in matching wave-forms, but rather in studying the dynamics of systems.

EXPERIMENTAL VERIFICATION

The atrioventricular annulus and valve cusps represent an area reduction between two chambers and may be likened to a nozzle or an orifice. One is thus led to consider the equation governing flow across an orifice as a first approximation to the physiological system:

$$\Delta P = KQ^2 \qquad (1)$$

In order to consider the pulsatile character of intracardiac flow, we also examined a pressure-flow relation which included an inertial term and assumed energy losses were proportional to the square of the flow:

$$\Delta P = AQ|Q| + BdQ/dt \qquad (2)$$

where A and B are resistive and inertial coefficients respectively and the flow is multiplied by its absolute value rather than squared as in equation (1) in order to maintain the proper phase between the dissipative aspects of the pressure-flow relation[1]*.

Equation (2) can be derived in a number of ways; we have utilized the one-dimensional equation of motion with the unsteady term retained, and incorporated the fluid mass in an "effective jet length". With this approach, we have shown that at physiological frequencies and flow rates, the system is quasi-steady with regard to discharge coefficient, and, when averaged over time, in regard to local acceleration. Equation (1) is shown to be an accurate relation between the mean pressure and mean square flow; and equation (2) is shown to be an accurate representation of the dynamic relations between pressure and flow. The resistances in both the forward and backflow paths of the model in Figure 1 should thus be non-linear and proportional to the square of the flow.

THE PHYSIOLOGICAL SYSTEM

In order to investigate the pressure-flow dynamics across the natural mitral valve, the left heart of large mongrel dogs was instrumented with an intracardiac electromagnetic flow probe sutured to the mitral annulus. Pressure was measured with Statham gages or Millar catheter tip transducers[2].

Figure 3 is an oscillographic record and reveals the following points of interest: Inflow to the ventricle starts at the moment of gradient reversal (LAP exceeding LVP). The gradient is small and rapidly goes to zero or slightly negative (LVP greater than LAP) thereby decelerating the flow so that it too approaches zero, but at a later time. Note that the

* Superior numbers refer to similarly-numbered references at the end of this paper.

rate of deceleration of flow in Figure 3 is more rapid than in Figure 2. This is due to the fact that in the former, the gradient became negative, whereas in the latter, the gradient went to and stayed at zero. An atrial contraction produces another favorable gradient followed by another period of forward flow, thereby giving the mitral flow trace its bicuspid appearance. A strong ventricular contraction rapidly decelerates the flow and closes the valve several milliseconds after the pressure difference has reversed.

The inertial character of this system is revealed by the fact that flow follows pressure. The resistive character is indicated by the fact that flow reaches its maximum before the gradient reaches zero. The physiological system thus seems to obey the relations described by equation (2).

In our final verification we have opacified the mitral cusps and filmed their motion at 60 frames/sec synchronously with recording the data as before. The results of one experiment are shown in Figure 4, and a frame by frame description follows:
A. The cusps are just beginning to move toward opening as the ventricle starts to relax and decreases the force on the valve. B. The gradient has reversed and blood is starting to move across the valve ring and flow probe, but the cusp margins are still in apposition. C. and D. The cusps open rapidly and widely before flow has peaked. E. The cusps start toward closure and flow is decelerating. F. An atrial systole increases the flow and separates the cusps. Between F. and G. the cusps have started to move toward closure again. G. Ventricular systole at X rapidly decelerates the flow again, applies a force on the cusps directed toward the atrium, and rapidly brings the cusps toward closure. H. At nearly peak systolic pressure the cusps are forced further toward the atrium (compare with A.) and the negative flow is due to this motion.

Figure 5 is a record of a dog with mitral incompetence, elevated LAP, and cardiac arrhythmias. It has been selected because it illustrates the scope and power of the measurement techniques, and the ability of the theory to consistently explain both normal and abnormal physiological function. The first three beats are non-sinus and demonstrate an unusual phenomenon of double regurgitation: once during diastole and once during systole. Note that the cusps follow the flow and the pressure gradient reverses twice during diastole. The last two beats are in normal sinus thythm and there is no double regurgitation. Flow nearly ceases in mid-diastole but the cusps remain more than half open and are closed by the ensuing systole. Note that the flow curve and the cusp motion depend on the shape of the pressure gradient during diastole which differs from the non-sinus beats. The fact that the flow is related to the recorded pressure gradient by the theory presented above substantiates the accuracy of the pressure records. Since it is difficult accurately to measure the pressure gradient when the valve is non-stenotic, we are in essence, saying that the flow trace is a sensitive indicator of the state of the atrioventricular system.

The approaches presented thus far all have in common the fact that they describe the system in terms of lumped parameters. They are phenomenological approaches which reveal the overall system dynamics but not the details. In the concluding section we describe briefly a distributed parameter approach. The results of a numerical method for solving the equations of motion governing the fluid and cardiac tissue are presented below to complement the foregoing analyses [3].

DIGITAL COMPUTER SOLUTION

In order to calculate the flow patterns of blood in the heart we take into account not only the forces which arise in the blood itself, but also the forces which arise in the muscular heart walls and the membranous valve leaflets. The latter forces are determined by the configuration of the heart apparatus in space in a manner which is fixed for the valve and time-dependent for the active muscle. The motion of the cardiac tissue is determined by the condition that it moves at the local fluid velocity. The heart and valve can be idealized as a specialized region of the fluid where extra forces are applied in addition to the usual fluid forces. Based on such considerations we have devised a computer method for constructing approximate solutions to the equations of motion of the blood-valve-heart system. We have applied this method to a two-dimensional representation of a valve of the mitral type between muscular atrial and ventricular chambers. The flexible valve leaflets are restrained by chordae which connect the ventricular apex to the cusp tips. The calculation begins at the end of systole with the atrium large and the ventricle small. First, the ventricle relaxes; later the atrium contracts; and finally the ventricle contracts. The calculation ceases when the valve closes.

The results of a computer experiment are shown in Figure 6 and the sequence of events is described in the captions. The dense network of mesh points results in the ready visualization of streamlines and vortices. We can thus identify streamlines which circulate as a vortex and close the valve without reducing ventricular volume, and those which go from the ventricle to the atrium and reduce the volume, i.e., backflow. Atrial systole serves to impart momentum to the blood in a direction which augments the motion of the circulating vortex and thereby helps close the valve without backflow. Finally, when integrated over a cross-section, the pressure-flow relations are identical with those formulated in equation (2).

CONCLUSION

From the anatomy of the left heart and the physics of flow, we have developed a comprehensive approach to the investigation of atriventricular flow dynamics. Complementary approaches have produced a series of consistent solutions which prove the validity of our model. In particular, we have elucidated the patterns of flow and the overall system dynamics to show that temporal acceleration is highly significant in understanding the instantaneous pressure-flow relations and cusp motion.

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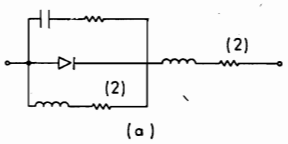
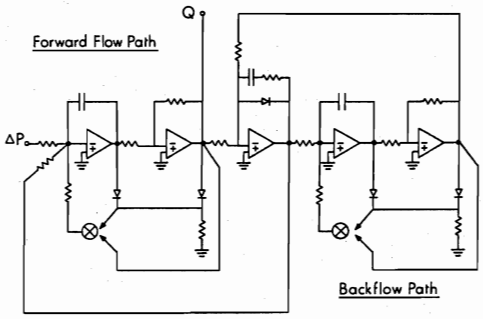


(a)



(b)

Figure 1a. Electric analog representation of the mitral valve. 1b. An operational amplifier circuit which generates solutions to the equations governing the valve model. Pressure difference is the input and flow the output.

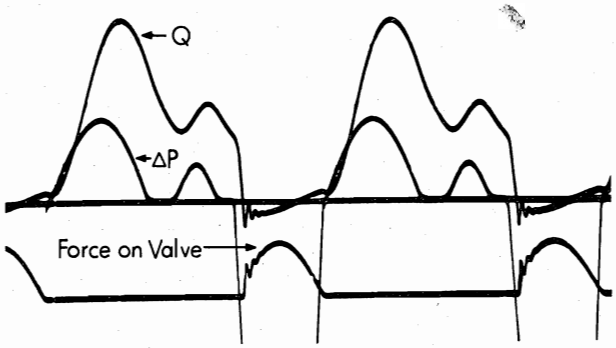


Figure 2. An oscillographic record of a flow waveform generated by the circuit of Figure 1. The bicuspid shape of the applied pressure gradient simulates an atrial contraction. (The force and pressure are on different scales.)

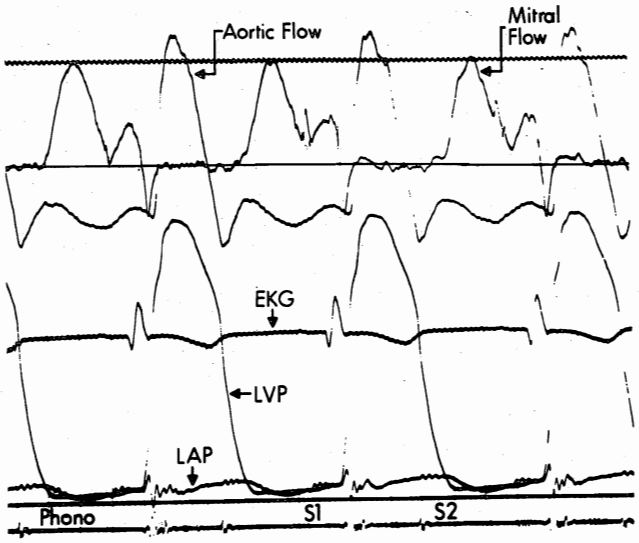


Figure 3. An oscillographic record of the data from an animal experiment. The similarity of wave-forms with Figure 2 is striking.

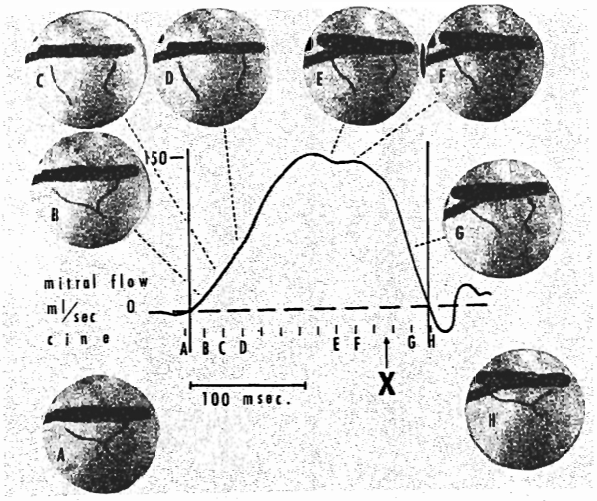


Figure 4. An oscillographic record of the flow from an animal experiment which has been synchronized with cines of mitral cusp motion. The cines are filmed at 60 frames/sec and the paper speed in this record is 200mm/sec. The flow probe appears as the heavy black line in the upper part of each frame. The cusps are delineated with radio-opaque thread running from the AV ring to the cusp margin. Their pliability is evident from the shape they assume during flow and closure. X marks the point in time at which the AV pressure gradient reversed following ventricular contraction.

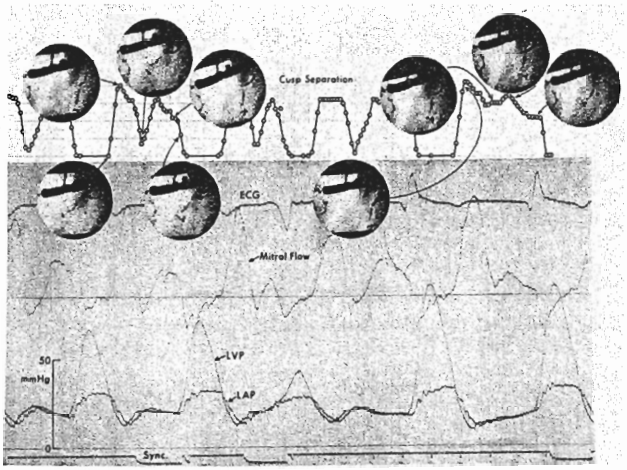
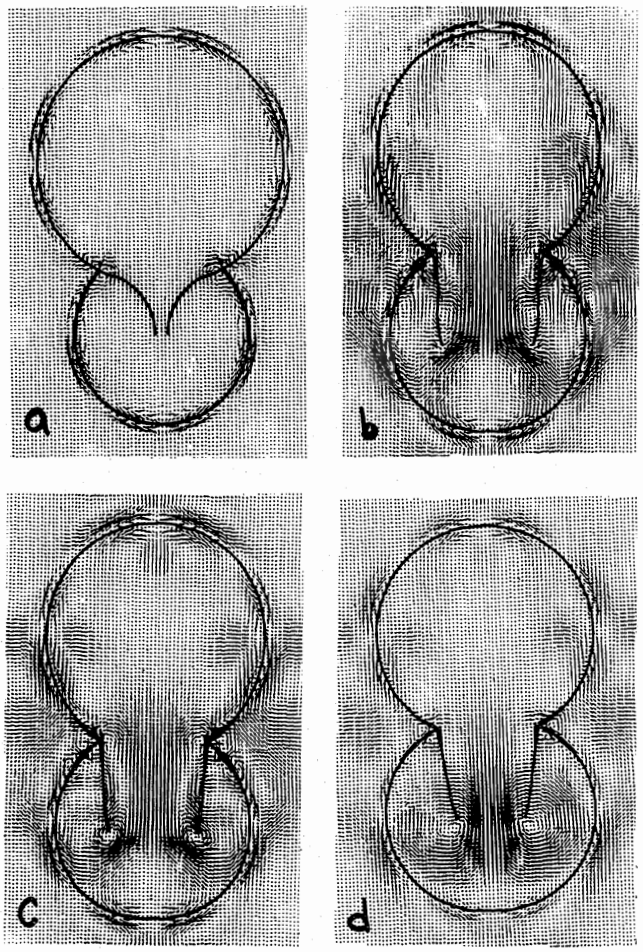


Figure 5. A synchronized cine-oscillographic record of a dog with mitral regurgitation and consequent cardiac failure. The radio-opaque threads have been cut to minimize distortion, thereby giving a different appearance from Figure 4. The thread that was to have delineated the free margin of the anterior cusp has come loose so that the cusps do not appear to be in apposition during closure.



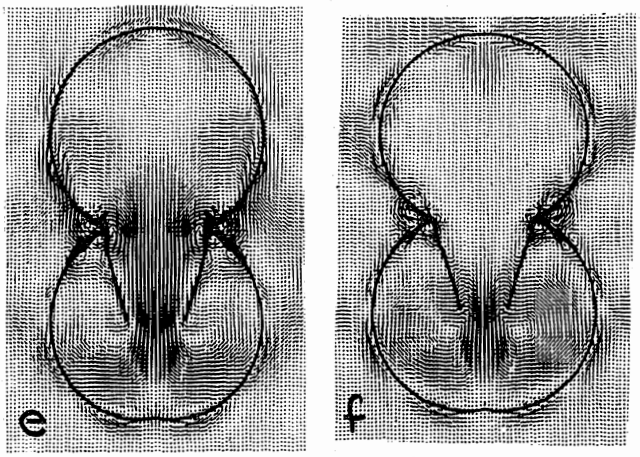


Figure 6. Digital computer solution of flow patterns across a simulated mitral valve. Each line (or point) represents a fluid velocity vector at a mesh point. a) The configuration of the atrium, ventricle and valve at the beginning of diastole when the pressures in both chambers are equal. (The program does not include inflow to the atrium, only outflow, so it appears inordinately large at the beginning of diastole.) b) Valve fully open in early diastole; a vena contracta is evident; and a vortex is forming. c) The vortex has moved to the valve tip and grown stronger so that its streamlines are moving the valve toward closure. d) The jet has "broken" so that there is no flow out of the atrium and a large vortex is rapidly closing the valve. e) An atrial contraction re-establishes the jet and re-opens the valve. f) As in (d), the jet again "breaks" and the valve rapidly moves toward closure.