THE MITRAL VALVE
A Pluridisciplinary Approach

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16 Analysis and Interpretation of the Normal Mitral Valve Flow Curve*

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INTRODUCTION

Traditionally, left ventricular inflow has been calculated indirectly from measurements of volume derived from cardiometry, roentgenography, myocardial segment length, or internal dimension changes. Ventricular filling has also been inferred from hemodynamic parameters which reflect the state of ventricular function, e.g., cardiac output, left atrial pressure, and left ventricular diastolic pressure. Neither method is as precise as the direct measurement of phasic transmitral flow, which offers the possibility of more meaningful physiological studies in the experimental animal.

In this report we formulate a simple mathematical approach with which to analyze the pressure-flow relation in the chambers of the left

heart and to present the results of such measurements. Our focus is analytical and our emphasis is conceptual rather than quantitative.

METHODS

Large mongrel dogs were anesthetized with pentobarbitol 30 mg/kg i.v. and placed on cardiopulmonary bypass. An intracardiac electromagnetic flow probe* was sutured to the mitral annulus in the supra-annular position and a cuff probe placed on the ascending aorta. Left ventricular, left atrial, and aortic pressures were measured with catheter-tip strain gauge transducers† and recorded along with the ECG, LV dp/dt, and intracardiac phono on an oscillographic recorder.‡

THEORY

Following the approach of Spencer and Greiss,[1] who studied aortic outflow, we describe the atrioventricular pressure difference in terms of resistance and inertance (Fig. 1, upper insert). That is, the total pressure difference is proportional to the sum of the rate of change of flow, and of the flow. In the lower insert, the differential equation is solved for the condition of no pressure difference, i.e., during diastasis, and it is seen that under these conditions flow decays exponentially toward zero. The properties of the first equation are well known and are shown schematically in the figure. The A-V gradient reversal and the start of inflow occur simultaneously; flow peaks after the pressure difference peaks and before it reaches zero; flow decelerates exponentially with equalization of pressure; and an atrial contraction imparts additional momentum to the fluid so that flow reaches zero after the A-V pressure crossover. Compliance is significant only during systole when energy is stored in the viscoelastic valvular appartus during closure and released during opening.

RESULTS

An oscilloagraphic record from a dog with moderate to rapid heart rates is shown (Fig. 2). At all three heart rates there is a rapid

* Carolina Medical Electronics.
† Millar.
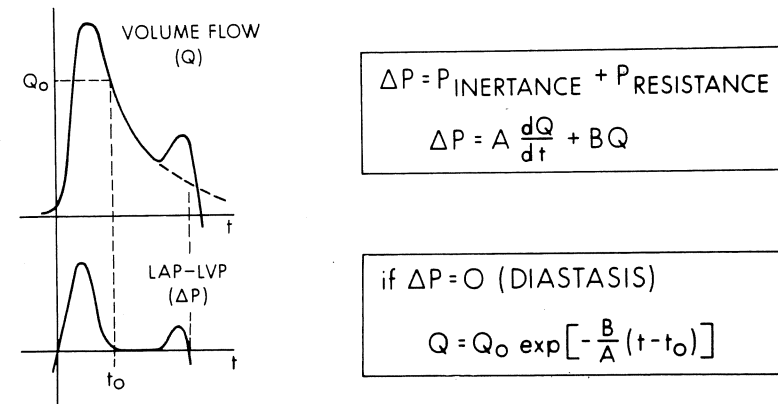‡ Electronics for Medicine.

$$\Delta P = P_{INERTANCE} + P_{RESISTANCE}$$

$$\Delta P = A \frac{dQ}{dt} + BQ$$

if $\Delta P = 0$ (DIASTASIS)

$$Q = Q_0 \exp\left[-\frac{B}{A}(t - t_0)\right]$$

Figure 1. The equation of motion for the fluid as it crosses the mitral valve and its rationale in the upper right. The equation is solved for flow as a function of time during diastasis when the pressure difference is zero. The curves on the left are schematic depictions of a pressure-flow relationship which obeys the formulation of the equations.
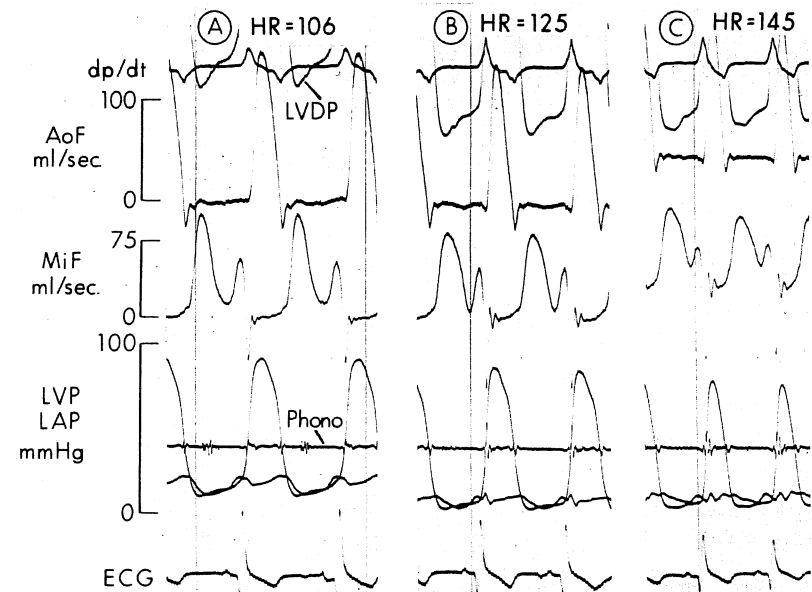

Figure 2. An original oscillographic record from a normal dog at three heart rates. AoF = Aortic Flow; MiF = Mitral Flow; HR = Heart Rate; LVP, LAP = Left Ventricular and Left Atrial Pressures; ECG = Electrocardiogram; Phono = Phonocardiogram; dp/dt = Derivative of LVP; LVDP = Left Ventricular Diastolic Pressure at high sensitivity (not used in this study).

rate of fall of ventricular pressure and a rapid early filling phase which peaks at the time the LVP reaches its nadir; the three mitral flow curves have a bicuspid wave-form with a clear atrial component; the diastolic filling period is greater than 50% of the total period; approximately 25% of the mitral inflow occurs by the time ventricular relaxation is completed; and the flow oscillation at valve closure coincides with the first heart sound and the atrial "C" wave.

In contrast with Figure 2 the hemodynamic data from another dog at equal or slower heart rates (Fig. 3) show a slowed rate of relaxation, particularly in the late isovolumic relaxation period; a depressed early filling phase; a LVDP which reaches its minimum as late as end diastole; a monocuspid mitral flow wave-form with an indistinguishable atrial component; and a shortened diastolic filling period (less than 30% of the total).

In Figure 4, the data illustrate the effect of a premature atrial contraction on an otherwise normal condition. The P wave in the ECG occurred before the completion of ventricular depolarization and the atrial contraction coincided with the early rapid filling so that

peak mitral flow increased. Because of the decreased filling period, however, the filling volume was reduced.

The atrial contribution to filling is analyzed (Fig. 5) in the record of a dog with a transient sequence of A-V dissociation, with regular sinus rhythm leading to some nodal and some ectopic ventricular contractions. The mitral inflow in beats 1 and 2 are similar in magnitude and duration, except that beat 2 has an atrial component and beat 1 does not. If the deceleration phase in beat 2 is extrapolated to follow the same shape as beat 1 (i.e., exponential), then the atrial contribution is the shaded area above the dashed curve. That area represents the difference in filling volume between the two beats and is clearly the area which would have existed if there were no atrial contraction in beat 2, and the filling period remained unchanged. Beats 6 and 7 also have an initially similar wave-form, except that in this case there is no atrial contraction in beat 7, and its diastolic period
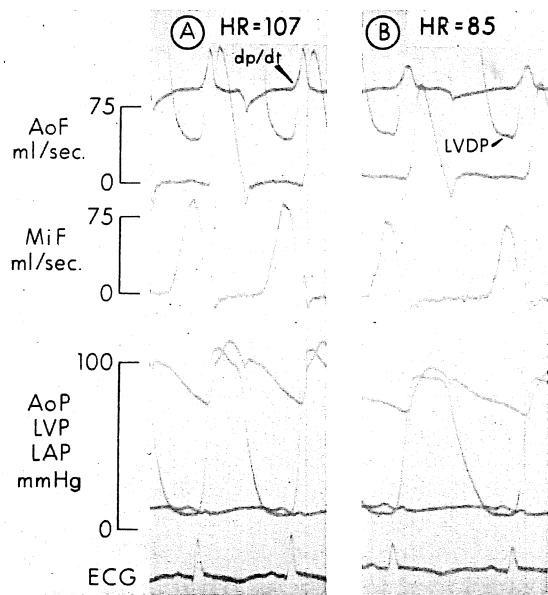

Figure 3. An oscillographic record at moderate and low heart rates from a dog with depressed contractility illustrating mitral wave-forms different from Figure 1. AoP = Aortic Pressure.
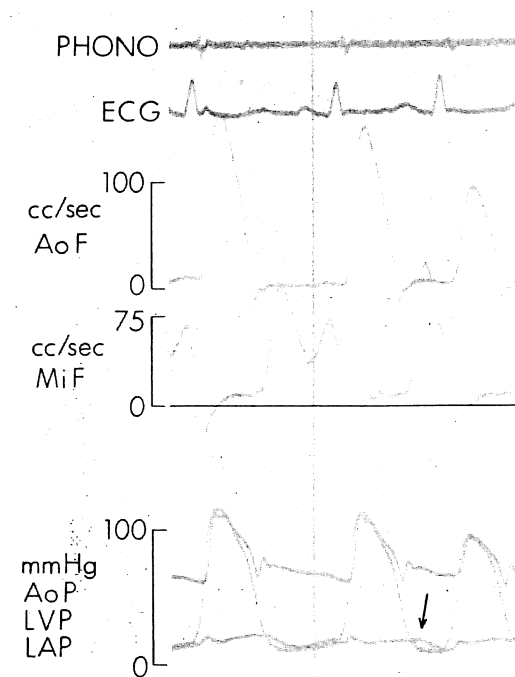

Figure 4. A normal record illustrating the effect of an atrial premature contraction.

is shortened. Thus, in contrast to 1 and 2, the difference in filling volume between beats 6 and 7 is the entire shaded area in beat 6, not just the area above the extrapolated curve.

Mid-diastolic reversal of the A-V pressure difference is illustrated in Figure 6. In Panel A, cinefluorograms of the delineated mitral
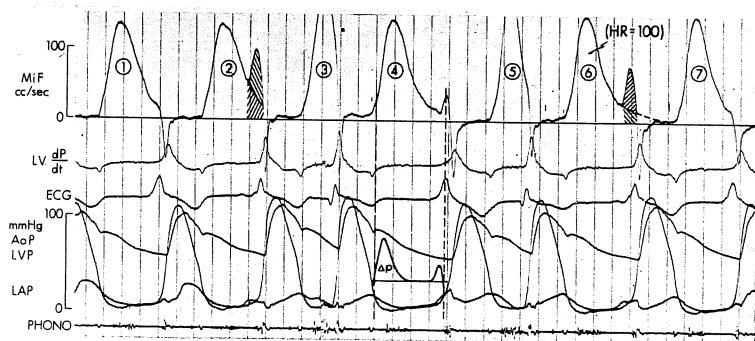

Figure 5. A record of a dog with A-V dissociation illustrating the differences between beats with and without an atrial contraction. The dashed curves are flow extrapolations based on the theory. They and the shaded areas are discussed in the text. In beat No. 4, the pressure difference has been superposed to illustrate the temporal relations between pressure difference and flow.
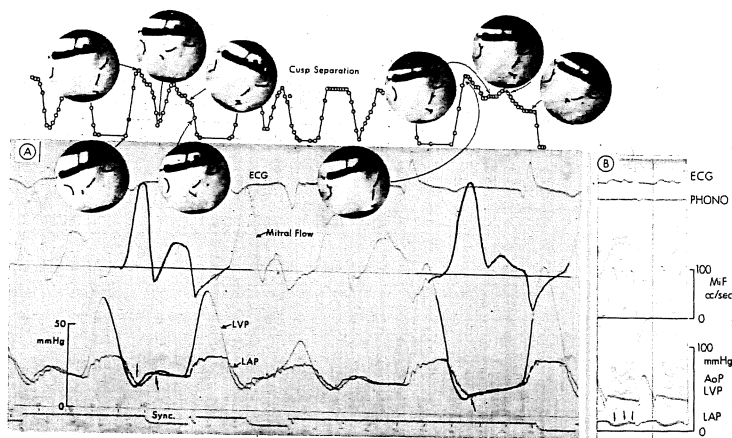

Figure 6. A record from a dog with a failing heart produced by volume overload (Panel A), illustrating the effect on mitral flow of reversal of the pressure gradient during diastole. Some of the traces have been darkened for illustrative purposes. The mitral cusps were delineated with radiopaque sutures. The effects of mid-diastolic gradient reversal under normal conditions are shown in Panel B. The arrows indicate the points of pressure cross-over.

cusps and a plot of cusp separation are superposed on the hemodynamic data. In the first beat the pressure difference becomes negative rather than zero during diastasis (between the two arrows) and flow decelerates rapidly, bringing the valve to closure. The gradient then becomes positive (second arrow) and forward flow resumes in the absence of an atrial contraction. The last beat in Panel A is similar to the first, except that the gradient does not oscillate during diastastis, there is no second wave of forward flow, and the valve remains widely open. In Panel B, we see another example of pressure oscillations where the second pressure cross-over coincides with an atrial contraction leading to an unusually large second peak. The long P-R interval in Panel B results in an atrial relaxation and pressure reversal (third arrow) before ventricular contraction, and a significant amount of backflow occurs during valve closure.

DISCUSSION

These results are consistent with those found in calves;[2] in the chronic unanesthetized dog;[3] in dogs and in humans with catheter tip probes in the mitral orifice;[4,5] and our previous work on dogs.[6,7] The data demonstrate clearly that the flow field has the resistive and inertial properties described in the equation (Fig. 1). The phasic pressure-flow characteristics at valve opening, valve closing, and during diastasis substantiate the fluid dynamic principles developed herein. Particularly impressive are those mid-diastolic events with exponential deceleration (Fig. 2, Panel A; Fig. 5, beats 1, 4, 6, 7), and with rapid deceleration due to an adverse gradient (Fig. 6, Panels A, B). Both are predicted by the theory. Particular emphasis should be placed on the finding (substantiated by theory) that mitral flow continues for approximately 25 msec after the A-V pressure crossover, so that the gradient reversal should not be used to infer cessation of flow and valve closure.

Figures 3-6 are not all "typical" results. Indeed, they have been selected as examples of pressure-flow relations which may not be normal or common, but which can be interpreted by a simple mathematical formulation. By presenting such diverse hemodynamic conditions, we have demonstrated that while the pressure-flow relations may change in pathology or unusual circumstances, the physical laws describing these events do not. Therein lies the power of analysis.

These results indicate that small pressure differences across the normal mitral valve (differences which might mistakenly be called

artifacts) lead to large changes in the flow wave-form and valve motion. This suggests that mitral flow is a sensitive indicator of small variations in the left atrial or ventricular pressures, which, in turn, reflect the diastolic properties of the two cardiac chambers. Mitral flow measurements can thus be used to study ventricular properties.[8]

The finding that flow decays exponentially during diastasis (if $\Delta p = 0$) leads us to propose a new method for defining the atrial contribution to ventricular filling. In those beats where mitral flow has started to decelerate before atrial systole, an exponential extrapolation will divide the flow wave-form into a part which would have existed had the atrium not contracted, and a part due solely to atrial contraction (Fig. 5). This approach chooses each beat as its own control and assumes a constant filling period, but has the disadvantage of not considering the specific condition which led to the loss of the atrial contraction—for example, sinus vs. nodal rhythm.[2] With this approach, we calculate an average atrial contribution of approximately 10%. Choosing the entire area under the curve during atrial contraction (Fig. 5, shaded areas of beats 2 and 6) gives an average contribution of approximately 20%. The smaller figure leads us to speculate about reports concerning dramatic increases in cardiac output after the restoration of normal atrial rhythm. We suspect that it is not the atrial contribution which is important, but rather the elimination of asynchronous, ineffective ventricular ectopic contractions which lead to the improvement in cardiac output. More thought and work are certainly required here.

Other interesting questions which arise from the measurement of mitral flow include: the relation between ventricular relaxation rate and mitral flow rate; and the contractile properties of the left ventricle during isovolumic contraction, when, because of fluid inertia, a significant portion of the period is not actually isovolumic, and the ventricle is contracting against an increasing preload.

Finally, although the lumped parameter method cannot describe the distributed forces due to fluid motion, our results are consistent with those of others who have used different fluid dynamic approaches. The vortex system presented by Bellhouse,[9] and the stable flow patterns described by Taylor and Wade,[10] can exist only if the dissipative forces are viscous. Inertial losses are symptomatic of a highly disturbed or turbulent system. The exponential decay of flow during diastasis supports the finding of a stable vortex system. The breaking jet theory of Henderson and Johnson[11] is likewise consistent with our findings, because the closing volume tends to be minimized when the ventricle contracts while inflow is still occurring.

CONCLUSION

The successful measurement of phasic transmitral flow along with related hemodynamic parameters has revealed significant new insights into atrioventricular dynamics and has encouraged continued investigations in this area.

ADDENDUM

Inasmuch as good flowmeter frequency response is vital for the study of temporal relations and dynamic events, we include the following short addendum.

The damping characteristics of two Carolina Medical Electronics flowmeters are illustrated in the oscillographic record (Fig. 7). Records were taken during a steady state so that the stroke volume in each series was constant. "Time Constant" and "Hz Response" are terms used by the manufacturer to describe the damping characteristics; they are inversely proportional to each other. In the new flowmeter (Model 501), more high frequency components and noise were passed as the frequency response was increased, but the wave-forms were essentially unchanged in area and magnitude. In the older flowmeter (Model 322), on the other hand, there was unacceptable

UPPER PANELS: FLOWMETER MODEL 501, HZ RESPONSE: 3, 30, 100 (LEFT TO RIGHT)

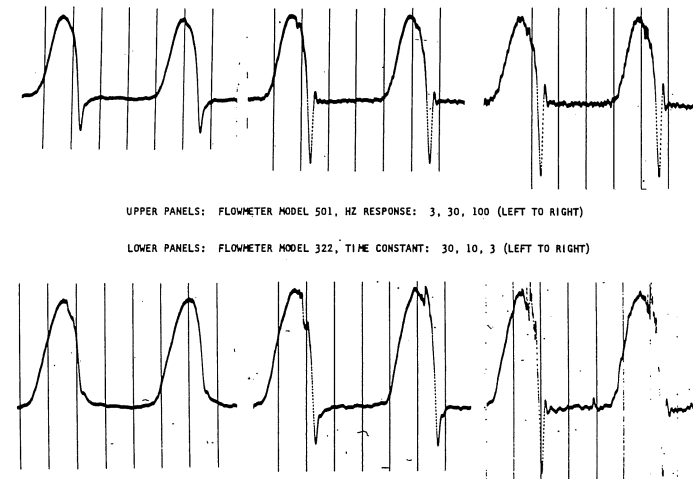LOWER PANELS: FLOWMETER MODEL 322, TIME CONSTANT: 30, 10, 3 (LEFT TO RIGHT)

Figure 7. Oscillographic records illustrating the phasic flow patterns for different frequency-response characteristics of the flowmeter. Further discussion in the Addendum.

distortion at the highest time constant. These records also indicate that it is not always necessary to conduct elaborate frequency response tests. If, upon increasing the frequency response, the high frequency components appear in the trace, and there is no further change in shape of the wave-form, we can conclude that there is no distortion or frequency-dependent time delays in the flowmeter.

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DISCUSSION: PART III

Dr. Wieting: There is only one question I would raise concerning Dr. Bellhouse's work: the rubber bag he showed us took on a more spherical configuration than I feel the natural ventricle has. Also, there is a small vortex which develops behind the posterior leaflet in all the studies I have done, both in a ventricular and in a cylindrical chamber.

Dr. Bellhouse: I don't think that small changes in the shape of the ventricle have much effect on the ventricular vortex. However, in one experiment, we shaped the ventricle so that there was a much bigger region behind the posterior cusp than behind the anterior cusp. In this case the main vortex strength lay behind the posterior cusp, with the result that the posterior cusp closed sooner than the anterior cusp.

Dr. Yacoub: I enjoyed Dr. Bellhouse's presentation. Can I ask him how he explains the fact that echocardiography shows simultaneous movement of the anterior and posterior cusps?

Dr. Bellhouse: The marked asymmetry of the vortex in my model of the left ventricle might be greater than in the physiological ventricle; this would tend to exaggerate differences in movements of the anterior and posterior cusps.

Dr. Gabe: Can I just ask Dr. Bellhouse what he feels is the influence the chordae have on the vortices? Do they reduce the energy of the vortices in any way?

Dr. Bellhouse: I think that any obstruction within the ventricle will reduce the strength of the vortex, but that the losses in vortex strength produced by the chordae tendineae and trabeculae carnae will be small.

Dr. Taylor: I think we can help resolve this apparent difference in the valve cusp movement observed by echocardiography, and those just demonstrated by Dr. Bellhouse in his model studies and older descriptions based on cineradiography. Unlike the models where a rubber 'ventricle' of uniform compliance is used, the left ventricle has a variable compliance, the septal region being stiffer than the free wall: this results in a rotation of the valve annulus relative to the anterior chest wall during diastolic filling. In our original measurements on cusp movement based on endoscopic cinephotographic records, we chose as our reference line the plane joining the commis-

sures, and obtained pictures similar to those shown by Dr. Bellhouse. Because of the apparent inconsistency of these with echocardiographic data, we have recently remeasured our original endoscopic records using as a reference point the anteroseptal wall, which corresponds to the echo reference line: this had produced records similar to clinical echocardiograms. It is all a question of the reference plane from which measurements are made.

On the point raised by the previous speaker, using a side-viewing telescope with our endoscope, we have observed flow between the chordae feeding the vortex associated with the anterior cusp, and the chordae do not appear to have a major influence on disturbing flow patterns or in generating secondary vortex trains.

Dr. Wright: Dr. Bellhouse, if we consider the topography of the heart, the outside is smooth, the inside is covered with knobs and excrescences. Do you think these can act as dampers?

Dr. Bellhouse: Yes.

Dr. Yellin: I would like to offer the possibility of an alternate approach to the function of the chordae, the role of vortices, the movement of the cusps, and flow patterns. The following two slides are the results of studies done by Dr. Peskin of New York University. We see in Figure 1 a model of the left heart without an aortic valve, since we are looking only at events during diastole. The equation of motion for the fluid and the heart wall has been solved numerically with a digital computer. What you see is not a simulation but rather a mathematical solution which is presented in a simulated form.

With ventricular relaxation, a pressure difference is set up from the atrium to the ventricle and the valve cusps swing out widely towards the septum and posterior wall. Therefore, at the beginning of diastole, the fluid momentum is directed toward the walls of the ventricle rather than the apex. In the next frame the jet is forming, the valve is moving toward closure, and a vortex is forming at the tip of the valve before the fluid reaches the apex and sweeps up the walls. If this solution is accurate, then, the size of the ventricle will have little to do with early vortex formation. The vortex is due to friction at the cusp surface.

The existence of chordae which tend to restrain the cusp from swinging open too far will help the vortex pattern. In this solution you can see a slight indentation in the apex where unseen elastic chordae connect the valves to the ventricle. Later in time a very well developed vortex is seen and the valve moves toward closure. Flow

measurements indicate that the time required for the fluid to reach the apex in the dog heart is long compared with echo observation of cusp motion, so that the valve moves toward closure in vivo before the large vortex forms.

There is a continuation of the flow patterns (Fig. 2). There is a long diastasis so that flow is dying down although a small vortex still exists. Now the atrial contraction accelerates flow again, strengthens the vortex, and brings the valve towards closure. Finally, with ventricular contraction, closure is completed. Note the stagnation point just above the cusp margins where the fluid on one side moves into the ventricle and on the other into the atrium so that there is no backflow. Fluid inertia is carrying it into the ventricle rather than allowing backflow in the atrium.
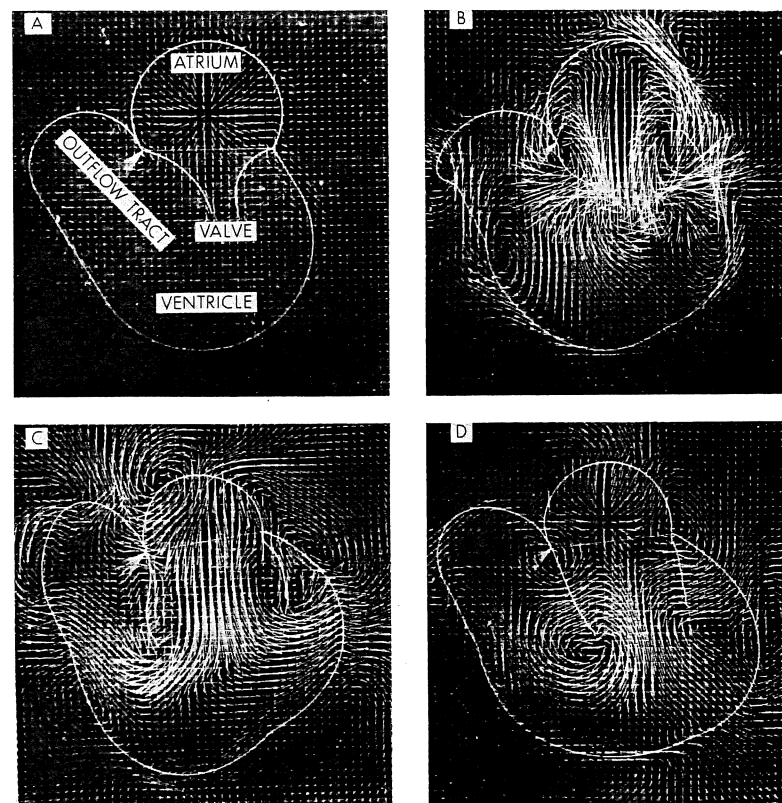

Figure 1. A mathematical solution to mitral flow dynamics and closure of the valve (see text).

Now this approach is admittedly speculative, but it is consistent with observations and should be given serious consideration. For example, the contraction of the jet as it goes through the valve indicates that the valve can move toward closure without in any way altering the movement of the main stream. This will not be seen in a model where the inlet is a straight tube such as Dr. Bellhouse has used, because there would not be any inward direction of the flow which has been directed by the tube toward the apex. This is one explanation for the vortices closing the valve in his model which may not be required in the natural valve.

Dr. Gabe: I wonder if I could ask Dr. Yellin a question about possible artifacts in this sort of flow measurement. It looks as if the flow through the mitral valve is going to be inertia-dominated. The
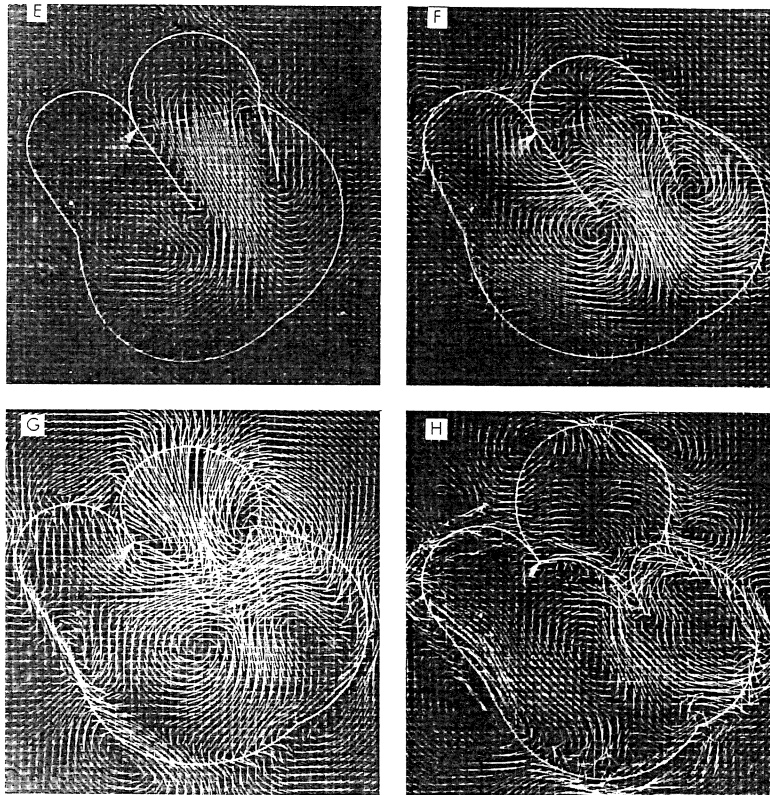

Figure 2. Continuation of flow patterns (see text).

method involves placing a rigid electromagnetic cuff above the valve which I suppose is about the length of the mitral valve. If the mitral valve inertance is roughly doubled in this kind of way, isn't it going to produce a significant artifact in the pressure gradient?

Dr. Yellin: I am not sure what you mean by inertance of the valve.

Dr. Gabe: In this context I mean the length of the valve multiplied by the density of the blood and divided by the cross-sectional area.

Dr. Yellin: It is a good question and the best answer I can give is that it is the inertia of the blood that dominates the system, not of the valve. In effect you are saying that we are creating a jet or tube of flow which is being accelerated and decelerated and, therefore, has inertia. You want to know whether a cuff probe would increase the size of the tube. The only way I can answer that is to say that we have done orifice flow studies and found that unless you get to a small diameter orifice (2,3,4 mm) the inertial term, except at opening and closing, is insignificant. We have also used electromagnetic probes at the mitral orifice as small as 12 mm diameter (i.e., much smaller than the annulus) and as large as 18.3 mm with the same initial pressure difference, so that we conclude that the probe does not increase the inertial term.

Dr. Rutishauser: Dr. Nolan, would you agree that the phases I, IV, and V may be due to the position of the probe above the valve and do not really mean that blood actually crosses from the left ventricle to the left atrium or vice versa?

Dr. Nolan: You have raised a point that disturbed us for several years. Phases I, IV, and V of mitral valve flow are always found with a normal mitral valve. These phases of flow correspond to fluctuations in the atrial and ventricular pressures. In addition, we discovered that certain pathological states, such as mitral regurgitation, could eliminate some of these phases. We concluded from these observations that these minor fluctuations in flow represent motion of the mitral leaflets when the valve is closed. Perhaps the problem is one of semantics. We do not believe that there is an actual flow of blood from ventricle to atrium during these phases but rather movement of the leaflets which in the closed position serve to partition the two chambers.

Dr. Yacoub: I have a question for Dr. Nolan. In phase III, forward flow is ascribed to atrial contraction, and this interpretation has been commonly used by all speakers. Yet, Dr. Yellin has shown

that this forward flow wave or hump can occur in the absence of atrial contraction. In one of Dr. Laniado's slides where the dog had atrial fibrillation, there was still a late hump simulating phase III or atrial contribution. One of our patients in London who was in atrial fibrillation had a definite phase III. Are we right in ascribing this forward flow during end diastole to atrial contribution?

Dr. Nolan: Several of the other participants may also wish to answer your question. I did not refer to phase III as the atrial contribution to mitral valve flow, but I did state that phase III flow occurs during atrial contraction. Our experiments did not allow us to determine the percentage of ventricular filling which can be attributed to atrial contraction. However, we were able to demonstrate one situation in which atrial contraction augmented ventricular filling by 20%.

While I have the floor, I would like to ask Dr. Yellin a question. In the last slide that you showed, you interpreted the secondary acceleration of mitral flow as having occurred in the absence of atrial contraction. However, I had thought that the electrocardiogram demonstrated an early nodal rhythm, in which case the secondary accelleration may have been due to an abbreviated atrial contraction.

Dr. Yellin: In the last slide there is nothing on the electrocardiogram to indicate any electrical activity of the ventricle. We often see it in failure states of our dogs when we overload them with volume in order to maintain cardiac output. We also see it in late states of massive mitral regurgitation, and I think this is one of the areas that has good clinical application. I think it is worth further discussion.

Dr. Laniado: Dr. Yacoub's observation that some patients who are in atrial fibrillation demonstrate in the echocardiogram an "A" wave (resembling an atrial contribution to mitral flow) is true. We have often seen, in dogs with atrial fibrillation, that during long diastole the pressure gradient across the mitral valve may reverse following a rapid transfer of blood from the left atrium into the left ventricle during the early filling phase. The reversal of the pressure gradient rapidly decelerates mitral flow and in some cases may even induce reversal of flow through the open mitral valve. This in turn will induce a second pressure-gradient reversal now in favor of the atrium, coming at the end of diastole and simulating atrial contraction in its effect on flow and cusp motion.

Dr. Yellin: Let me add just one thing to that. Just because the mitral valve was open, and the atrium and the ventricle are one

chamber, does not mean that there cannot be pressure oscillations between the two of them, and that these pressure oscillations will not produce flow. In fact what sometimes happens is that the ventricle can be a resonating system of very low frequency and the gradients can reverse and can cause flow to go back and forth. We occasionally see flow reversal as regurgitation during these conditions because these are acutely dilated ventricles and the valve is held open during the time the gradient reverses; therefore, you can get regurgitation. If the ventricle is not distended, the valve will close during that period of time and you won't get regurgitation.

Dr. Kalmanson: I'd like to answer Dr. Yacoub's question. We have carried out a series of recordings of mitral valve flow velocity in patients in sinus rhythm and in patients with atrial fibrillation. In patients in sinus rhythm, we consistently found a temporal relationship between the end diastolic forward flow wave and the P wave of the ECG. This holds true also for patients with a long P-R interval. On the other hand, in patients with atrial fibrillation, during diastoles of short length, there is no spur of end-diastolic forward flow waves. These only occur during long diastoles. Furthermore, contrary to what happens in patients with sinus rhythm, the shape or pattern as well as the duration of these waves varies from one diastole to the other. It is, therefore, logical to discriminate between regular and stereotyped end-diastolic forward-flow waves of patients in sinus rhythm, which can reasonably be ascribed to atrial contraction, and anarchical and intermittent humps that occur during long diastoles of atrial fibrillation. These may well be due to pure oscillations caused by pressure-gradient reversal as Dr. Yellin just suggested. Evidence which supports this hypothesis is given by the frequent occurrence of flow reversal between the two successive diastolic forward flow waves and even after the end-diastolic A wave in case of prolonged P-R interval.

Now, I would like to put a question to Dr. Folts. First Dr. Folts is to be warmly congratulated for having shown his recording of mitral flow volume in patients, obtained preoperatively, since it is the very first time that such data have been determined in man. I was a little disconcerted, however, when I saw the postoperative recording in your patient with mitral regurgitation. Indeed, as expected, the systolic-regurgitation flow wave disappeared after operation, but the diastolic-filling flow wave failed to decrease, as it is known to do after surgical cure. Do you think there might be some artifact due to your technique?

Dr. Folts: If so, I don't know where it would be, since the same probe was used before and after, and put in the same position. The only alteration has been the repair of a portion of the torn commissure and the insertion of a mitral ring. So, if it is an artifact, I don't know where it would come from.

Dr. Reneman: I should like to ask Dr. Folts two questions. First of all do you have any idea about the uniformity of the magnetic field in this C-shaped probe you showed us? We have noticed that the magnetic field is usually not uniform in C-shaped probes, so that they are more sensitive to changes from axisymmetric velocity profiles than can be expected from the weight function alone. Therefore, differences in the velocity profile before and after surgery can easily lead to unexpected volume flow readings after the operation. Secondly, can you give us some information about the sensitivity of your probe and the amperage with which you are feeding it? I expect the sensitivity to be low because of the large air gap.

Dr. Folts: I don't know exactly what the amperage is. Carolina Medical Electronics flowmeters have a constant current source for probes of that size that is the same for aortic probes and this mitral probe. It is calibrated by using dialysis tubing. Another way is to make a clay mold around it as some people have done for calibration purposes. The iron filing field distribution seems pretty uniform and is very comparable to a flowprobe constructed for a cuff around an aorta or some other large bore vessel.

Dr. Angell: I wonder whether the formation of vortices discussed in the earlier papers has any influence on the occurrence of that forward flow phase or whether it has anything to do with ventricle compliance.

Dr. Yellin: I am not sure I understand how the compliance question relates to the vortices. I know nothing about vortices in the intact heart; we just haven't measured them. Dr. Taylor has and he sees a stable pattern as he described. I'll make one comment on compliance, since you raised the question. In the studies we have done in the dog, roughly 25% of the ventricular inflow occurs while the ventricle is still relaxing, i.e., down to what the cardiologist calls the 0 point, 25% of the volume has already entered the ventricle and, therefore, I caution you against using the stroke-volume as determined from cardiac output to measure compliance. That is number one. Number two—the ventricle is a visco-elastic chamber and more

force is required for rapid distension, so slow filling or filling in mitral stenosis which is uniform will give you a different calculation for volume compliance than rapid filling. There are several other problems which we really can't go into at the moment.