IN-VITRO STEADY AND PULSATILE FLOW VISUALIZATION OF THE NORMAL MITRAL VALVE

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Abstract

Because of the complex design of the mitral valve, a thorough description of its function is not presently available, even in the non diseased state. The aim of this research, therefore, was to determine the role and importance of critical parameters such as cardiac output, ventricular pressure and papillary muscle position on mitral valve behavior and on the development of the ventricular flow field. Steady and pulsatile flow visualization experiments demonstrated that the main factor which affected the ventricular functions was the papillary muscle position. By controlling regurgitation, it could change the cardiac output as well as the ventricular pressure. A qualitative agreement was found between the color Doppler ultrasound flow field and those obtained by flow visualization.

Introduction

Because of the complex design of the mitral valve, a thorough description of its function is not presently available, even in the non diseased state. The aim of this research, therefore, was to determine the role and importance of critical parameters such as cardiac output, ventricular pressure and papillary muscle position on mitral valve behavior and on the development of the ventricular flow field. In order to obtain this information, steady and pulsatile flow visualization experiments were performed on a normal mitral valve in a model of the left ventricle. By obtaining a picture of the true flow field, it is possible to understand the significance of newly developed techniques such as color Doppler flow mapping, which superimposes visualized velocity patterns on two dimensional images of the cardiac structures in real time.

The mitral valve

The mitral valve, as well as the other cardiac valves, works passively to control the flow of blood through the heart [1,2,3,4,5]. It opens and closes according to the fluid and pressure forces. The mitral valve is located between the low pressure left atrium and the high pressure left ventricle. It is a continuous strip of tissue which surrounds the mitral orifice. Indentations in the tissue divide it into two trapezoidal leaflets. The anterior leaflet, located on the side of the interventricular septum, is twice as large in surface area as the posterior leaflet. At their base, the leaflets form the annulus ring which is the main attachment of the valve to the rest of the heart and undergoes active contraction during systole. The chordae tendineae are collagen fibers which extend from the annulus to the two papillary muscles through the leaflets. They distribute evenly, on all the surface area of the leaflets, the tension applied by the papillary muscles. The papillary muscles, which are anchored in the posterior wall of the ventricle have three functions:

1) They prevent the mitral leaflets from collapsing into the atrium during systole, when the difference of pressure between the atrium and the ventricle is high.
2) They participate in the heart contraction and help maintain a high ejection fraction.
3) They maintain the mitral valve in a posterior position, away from the blood outflow tract, minimizing the flow forces that are applied on the leaflets by the blood.

The mitral valve first opens at the beginning of diastole, when the atrial pressure exceeds that of the ventricle. For the next 100 ms, the leaflets open to their widest position and mitral flow increases while large vortices appear between the anterior leaflet and the interventricular septum. Then, as the pressure gradient between the atrium and the ventricle is slightly reversed, the flow decelerates and the leaflets start to close. About 300 ms into diastole, the atrium contracts, accelerating the flow
passing through the mitral orifice and reopening the leaflets. The flow then decelerates again as the ventricular pressure exceeds the atrial pressure. Just before the onset of systole, the leaflets reach their closed equilibrium position which is determined by the position of the papillary muscles and by the transvalvular pressure gradient.

**Experimental Methods**

Left ventricular model

The left ventricle was simulated by a rigid plexiglass model which used an extracted native human or porcine mitral valve with intact papillary muscle apparatus (Figure 1). Physiologically, the heart contraction starts from the apex and spreads toward the base of the ventricle. Therefore, during systole, the blood streamlines are going from the apex to the aorta, passing between the anterior leaflet of the mitral valve and the septum. This behavior was simulated with a flow input located at the apex of the ventricular model, the shape of which was based on echocardiographic observations of human subjects at the onset of systole. During pulsatile flow experiments, a prosthetic tilting disc valve was used in the aortic position.

Mitral valve

The mitral annulus was sewn on an elliptical plexiglass ring in a way that simulated the native geometry. The papillary muscles were fixed to small plexiglass rings that rotated at the end of metal rods. This prevented any possible twist of the chordae tendineae and allowed the papillary muscles to reach their equilibrium position where they could distribute the tension evenly to all the surface area of the leaflets. The metal rods could be moved in three possible directions to fix the apical/basal, anterior/posterior and in/out position of the papillary muscles.

Steady flow

A steady flow loop was used to simulate peak systolic flow. Steady flow rates of up to 30 l/min, corresponding to a cardiac output of 6 l/min were used. The ventricular pressure was fixed at 100 mmHg.

Pulsatile flow loop

The pulsatile flow system simulated the whole cardiac cycle by duplicating the flow and pressure waveforms of the left heart (Figure 2). A constant level reservoir simulated the atrium and generated a constant pressure, 15 mmHg, that drove the diastolic flow. During diastole, the flow entered the model through the mitral valve and accumulated into a compressible bulb located downstream of the apex orifice. The compression of the flexible reservoir simulated systole. It produced an increase in the ventricular pressure that first closed the mitral valve and then opened the aortic valve. At the end of systole, the compressible bulb expanded, decreasing the ventricular pressure and eventually opening the mitral valve.

A heart rate of 70 beats per minute with a systolic period of 280 ms long was used. The cardiac output varied from 2.5 to 10 l/min while the peak ventricular pressure was varied from 70 to 140 mmHg.

Data acquisition methods

Two techniques of flow visualization were used:

The first was used to obtain a two dimensional map of the velocity flow field. A 1 mm thick plane of light was created by passing a laser beam through a glass rod. This plane of light was positioned at the center of the ventricle, following the long axis direction. It illuminated the full length of the ventricle. Neutrally buoyant particles added to the solution, followed the streamlines and refracted light as they passed through the sheet of light, creating a map of the left ventricular flow field.

The second method was used to obtain information on the mitral valve behavior. A white light was located behind the model and illuminated the mitral valve from the back, displaying precisely its movements. Color Doppler flow maps were obtained using a Toshiba Sonolayer SSH-65A.

Results

Steady flow

Steady flow visualization with a flow rate corresponding to peak systole was first performed. The papillary muscles were located in an apical, posterior, and out position. This resulted in a flat anterior leaflet with most of the flow passing above the valve (Figure 3). The streamline were smooth and followed the shape of the anterior mitral leaflet, indicating a low level of disturbance inside the ventricle.

When the papillary muscles were located in a more anterior position, some of the flow was passing beneath the mitral valve, creating a recirculation region. In spite of this, the flow field between the septum and the valve remained the same. Repeating this experiment for other flow rates did not change the flow field significantly. However, when the flow rate
was low, the main flow exiting the ventricle which defined the outflow tract was small and did not occupy all the space available above the mitral valve. As the flow rate increased, the outflow tract enlarged and eventually occupied nearly all the ventricle.

Pulsatile flow

Pulsatile flow experiments were then performed with the mitral valve positioned in a normal position, the papillary muscles being set into an apical, out, and posterior location. For all the cardiac output and ventricular pressure used, the general flow pattern inside the ventricle was the same.

At the beginning of diastole, when the mitral valve opened, the flow entered the ventricle and formed an inflow tract that was going toward the apex. However, part of this flow was entrained by the fluid which was exiting the ventricle at the end of systole (Figure 4). It created a circular pattern which eventually filled up the entire ventricle and could be seen until the beginning of systole. Then, the flow field changed as the streamlines started to move towards the aorta, passing above the mitral leaflets. During the acceleration phase, the outflow tract was getting wider and the streamlines longer and smoother (Figure 5). This phenomenon reached its maximum at peak systole (Figure 6). Once the flow had stopped to accelerate, some recirculation appeared between the outflow tract and the septum. The volume of this recirculating fluid increased throughout the deceleration phase, reducing the size of the outflow tract. At the end of systole, the flow exiting the ventricle had to pass through the recirculation region (Figure 7). It was this recirculating flow which entrained the incoming mitral flow once diastole had started.

Other pulsatile experiments were performed in order to characterize the effect of the distance between the mitral valve annulus and the papillary muscles on the closure of the valve. When this distance was too large, the leaflets did not close properly and regurgitation occurred. As the distance was decreased, the mitral leaflets closed properly but further decrease of the distance caused the muscles to be too close to the annulus and the valve to prolapse during systole. An increase in ventricular pressure caused the mitral valve to prolapse even more.

For all experiments, a qualitative agreement was found between the color Doppler ultrasound flow fields and those obtained by flow visualization methods.

Conclusion

In this simulation of a normal left heart, it was found that the main factor which affected the ventricular functions was the papillary muscle position. By controlling regurgitation, it could change the cardiac output as well as the ventricular pressure. The techniques used to study the normal case may be extended to study the role of papillary muscle geometry or other variables in diseased conditions. For example, by observing accelerated flow through obstructions such as those observed in hypertrophic cardiomyopathy, areas of aliasing and disturbed flow can be explained on color flow maps.

References

**Figure 1:** The left ventricular model. The mitral annulus is sewn to a large elliptical plexiglass ring. The papillary muscles are attached to the end of metal rods that can be moved in three directions. In pulsatile flow, an artificial aortic valve is inserted in the aortic orifice. Echocardiographic observations are performed through the two ports located in the posterior wall.
Figure 2: The pulsatile flow loop. The constant level reservoir simulates the atrium and produces the driving pressure that generates diastolic flow. The compressible bulb acts like an accumulation chamber. Its compression produces systolic flow.
Figure 3: Steady flow visualization. Peak systolic flow. The mitral valve is in normal position with the papillary muscles set into an apical, out and posterior location.

Figure 4: Pulsatile flow visualization. Diastole. The mitral valve is in normal open position. Part of the flow entering the ventricle through the mitral valve is going toward the apex while the rest is recirculating between the anterior mitral leaflet and the septum.
Figure 5: Pulsatile flow visualization. Systole, Acceleration phase. The mitral valve is in normal closed position. The outflow tract is starting to form. No recirculation can be seen between the outflow tract and the septum.

Figure 6: Pulsatile flow visualization. Peak Systole. The mitral valve is in normal closed position. The outflow tract is clearly defined. Some recirculation can be seen between the septum and the main flow exiting the ventricle.
Figure 7: Pulsatile flow visualization. Systole, Deceleration phase. The mitral valve is in normal closed position. The outflow tract has disappeared while the recirculation region has increased.