Motion of both mitral valve leaflets: a cineroentgenographic study in intact dogs

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MOTION STUDIES OF THE MITRAL VALVE leaflets are important for the understanding of the closing mechanisms of the valve during normal and pathologic conditions. Since 1961 extensive and valuable information has been obtained by echocardiography concerning the motion of the anterior leaflet in man (3, 5) and in the open-chest dog (7, 9). Data concerning the motion of both mitral cusps in the open-chest animal have been reported recently (6, 9). Still, there is a lack of systematic data concerning the variations of leaflet motion which might be expected over a wide range of hemodynamic conditions. The intact dog lends itself well to this type of study.

The aims of the present investigation were 1) to provide precise information about the movements and the spatial positions of the two mitral cusps in the intact dog during normal sinus rhythm over a wide range of heart rates; 2) to compare valve leaflet motion following isolated atrial or ventricular contractions with that observed during sinus rhythm; and 3) to correlate leaflet motion with angiography. Measurements were made in five dogs in which small radiopaque markers had been sutured on the atrial surface of both mitral cusps and on the endocardial surface of the apparent mitral annulus 1-11 wk before the studies.

METHODS

Five healthy dogs weighing between 17.3 and 22.2 kg were operated on using a normothermic cardiopulmonary bypass. A left thoracotomy through the fourth intercostal space provided access to the left atrium and the appendage which was incised to expose the mitral valve. Seven perforated lead beads, 2.4-2.7 mm in diameter and 70-115 mg in weight, were used in each dog. One marker was sutured to each cusp. Isolated ventricular contractions closed the valve leaflets completely and symmetric valve closure was ensured by the different rates of leaflet edge approximation. In contrast, atrial closure was slow, partial, and of very short duration.

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left ventricle, and detection of mitral regurgitation. Fluoro-
grams of leaflet motion and angiograms were recorded on
cinefilm at speeds ranging from 100 to 120 frames/s (35
mm film, 3.1 lines/mm resolution). Pressure tracings and
the electrocardiogram were recorded by a multichannel
Visicorder oscillograph (Honeywell, model 1912) at paper
speeds of 150 mm/s. The cinecamera was synchronized
with the oscillographic record by means of an electronic
pulse which marked the exposure of each frame. The delay
in this circuit and that of the electrocardiographic signal

![Image](http://jap.physiology.org/)

**FIG. 1.** Atrial view of mitral orifice and mitral leaflets demonstrating position of seven markers inserted in each dog, left panel. Markers are numbered and tracings of individual bead motion are identified with same numbers in subsequent figures. Bead numbers 1, 2, 3, and 4 are placed at annular level while 5, 6, and 7 are sutured on valve leaflets (see text). Right panel shows projections of beads on television screen with mitral ring in vertical position and parallel to central axis of X-ray beam.

![Image](http://jap.physiology.org/)

**FIG. 2.** Motion of marker placed on free edge of anterior leaflet (heavy solid line) and time intervals of leaflet motion measured in this study. Identical time intervals for posterior leaflet were measured. Z indicates individual marker zero reference position (see text). Peak speeds were determined by taking maximum slopes observed in tracings during opening and closure.

![Image](http://jap.physiology.org/)

**FIG. 3.** Correction of leaflet motion by continuous subtraction of annular movement. Panels on left show uncorrected excursions of three leaflet markers at heart rates of 67 and 107 beats/min and panels on right demonstrate same cardiac cycles after correction for annular movement. Abcissa represents instantaneous linear distance between a marker and its own reference position (Z). Upstroke indicates motion toward ventricle or valve opening and downstroke valve closure. Numbers on right side of every panel identify individual marker motion tracings (solid line is marker 7; interrupted line is marker 3; dotted line is marker 6). All three leaflet markers moved steadily toward ventricle during ventricular contraction due to valve ring displacement. Annulus returned to its original position early in diastole, and during late diastole uncorrected and corrected marker movements were nearly identical.

were found to be less than 0.1 ms. Thus accurate alignment
of the cineframes with the electrocardiogram was possible.

The cineframes were projected with an approximate X6
magnification to reduce the error of measurement. Cross
hairs placed on the image intensifier served as a fixed refer-
ence point in the positioning of each projected image. An
x, y coordinate system was used to determine the spatial
position of the center of every bead in each frame.

The first step in our measurement was to determine in
each cardiac cycle the coordinates of the markers early in
ventricular systole, when the valve was completely closed
(Fig. 2, Z). In the subsequent frames, the position of each
bead was determined as a linear distance from its individual
(Z) reference. The distances thus computed were corrected
for projection magnification and X-ray distortion. In addi-
tion, the movement of the valve ring, determined from the
motion of the four markers placed at the annular level,
TABLE 1. Summary of valve dimension and mitral leaflet motion data, and hemodynamic variables during regular sinus rhythm (27 cardiac beats from six experiments, heart rate 42-184 beats/min)

<table>
<thead>
<tr>
<th>Leaflet</th>
<th>Leaflet Peak Opening Speed cm/s</th>
<th>Maximum Diastolic Valve Opening cm (distance between markers S &amp; T)</th>
<th>Maximum Diastolic Valve Opening cm (distance between markers S &amp; T)/% of complete closure</th>
<th>Duration of Early Diastolic Closure ms</th>
<th>Early Diastolic Valve Closure % of complete closure</th>
<th>Early Diastolic Valve Closure % of complete closure</th>
<th>Time of Complete Leaflet Closure (from Q wave of ECG) ms</th>
<th>Left Ventricular End-diastolic Pressure cmH2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior leaflet</td>
<td>42 ± 10</td>
<td>29 ± 7</td>
<td>1.04 ± 0.21</td>
<td>98 ± 45</td>
<td>49 ± 21</td>
<td>0.92 ± 0.27</td>
<td>110 ± 20</td>
<td>63 ± 16</td>
</tr>
<tr>
<td></td>
<td>24-43</td>
<td>0.70-1.39</td>
<td>0.70-1.39</td>
<td>30-190</td>
<td>7-80</td>
<td>0.42-1.30</td>
<td>67-161</td>
<td>33-117</td>
</tr>
<tr>
<td></td>
<td>51-70</td>
<td>0.70-1.39</td>
<td>0.70-1.39</td>
<td>30-190</td>
<td>7-80</td>
<td>0.42-1.30</td>
<td>67-161</td>
<td>33-117</td>
</tr>
<tr>
<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior leaflet</td>
<td>39 ± 13</td>
<td>21 ± 9</td>
<td>0.55 ± 0.16</td>
<td>83 ± 40</td>
<td>59 ± 25</td>
<td>0.24 ± 0.16</td>
<td>63 ± 19</td>
<td>61 ± 6</td>
</tr>
<tr>
<td></td>
<td>26-60</td>
<td>0.32-0.86</td>
<td>0.32-0.86</td>
<td>27-194</td>
<td>14-89</td>
<td>0.24 ± 0.16</td>
<td>63 ± 19</td>
<td>61 ± 6</td>
</tr>
<tr>
<td></td>
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</table>

Values are means ± SD; range indicated below.

FIG. 4. Mitral valve leaflet motion during regular sinus rhythm at heart rates ranging from 51 to 108 beats/min in same dog (19.1 kg, morphine-pentobarbital anesthesia). In all panels diastolic opening was followed by immediate reclosure of both leaflets, characterized during slow and moderate heart rates (panels 1, 2, and 3) by initial fast movement, a short pause, and resumption of closure; during short diastolic periods (panels 4 and 5) early diastolic closure was minimal. At slow heart rates (panel 1) following early diastolic closure, both leaflets remained practically motionless in semiclosed position until atrial reopening. Heart rate changes did not affect rate of leaflet opening or closure nor their maximal diastolic opening.

was continuously subtracted from the motion of the valve leaflets throughout the cardiac cycle, since it became evident early in the study that concomitant movements of the heart or of the annulus alone might simulate leaflet motion (Fig. 3). Our measurements determining the position of a marker were repeatable with an accuracy of ±0.3 mm.
Maximum slopes of tracings of the markers placed at the free edges of the leaflets were used to calculate peak opening and closing speeds.

Valve leaflet motion, pressures, cardiac output, and angiograms of the left atrium and left ventricle were recorded in each experiment over a wide range of heart rates during normal sinus rhythm (42-184 beats/min). Slow heart rates, i.e., below 70 beats/min, occurred either spontaneously due to the morphine premedication used (three experiments), or were produced by vagal stimulation (three experiments). First and second degree atrioventricular block occurred spontaneously in two experiments and vagal stimulation was used to produce transient block of varying degree in three experiments. Isolated ventricular extrasystoles were produced by stimulating the right ventricular block occurred spontaneously in two experiments and vagal stimulation was used to produce transient block of varying degree in three experiments. Isolated ventricular extrasystoles were produced by stimulating the right ventricle in four experiments.

At the end of the study two dogs were killed and autopy was performed. The left ventricular cavity was opened and the heart was fixed in formalin for histologic examination. The remaining three animals are in good condition 5-8 mo after surgery.

RESULTS

In the two dogs in which autopsy was performed, the inserted beads were found to be placed at their desired locations. A modest amount of fibrous tissue, confined to the endocardium, was present around the beads and no deformity or fibrosis of leaflet tissue or of the mitral ring was apparent. In addition, during regular ventricular contractions, at heart rates studied, no mitral regurgitation was detected by angiography. These findings suggest that the presence of the markers did not compromise valvular motion and function.

Leaflet motion during regular sinus rhythm. Twenty-seven cardiac beats were analyzed in detail (Fig. 2) at heart rates ranging from 42 to 184 beats/min and the data of leaflet motion and valve dimensions are presented in Table 1.

Figure 4 illustrates mitral leaflet motion during different heart rates in the same dog. In diastole both valve leaflets opened and rapidly reached their points of maximal excursion. In most beats measured, valve opening was asymmetrical with the posterior leaflet opening 8-40 ms later than the anterior. This delay was not related to heart rate. Heart rate changes did not significantly affect the opening time of both leaflets (Fig. 2), their peak opening speed, or the maximal valve opening. The minimal linear distance, early in diastole, between the marker placed at the free edge of the anterior leaflet and the ventricular wall was found to average 0.8 ± 0.3 cm (0.5-1.4 cm), and between the marker placed at the free edge of the posterior leaflet and the ventricular wall 0.2 ± 0.1 cm (0.1-0.4 cm). In general the distance between valve cusps and the ventricular wall was greater at slow heart rates and larger diastolic ventricular volumes.

The cusps were never observed to pause at their fully open positions but began to reclose immediately. Diastolic closure was characterized during slow and moderate heart rates by a rapid movement of both leaflets towards the atrium, followed by a short pause, and a resumption of closure (Fig. 4, panels 1, 2, 3).

Peak closing speed of the anterior leaflet during the second part of diastolic closure averaged 10 cm/s and was identical to its rate of closure following isolated atrial contractions. The duration of diastolic closure varied between 30 and 190 ms (Fig. 5) and showed a significant correlation with heart rate (P < 0.005). At the end of diastolic closure the valve was closed 36 ± 23% of its complete closure excursion. In general the valve was closed more completely during slow and moderate heart rates, whereas at faster rates or when atrial contraction occurred early in diastole, leaflet approximation was interrupted by atrial opening (Fig. 4, panels 4, 5; Fig. 6, panel 2).

As a result of atrial contraction, both leaflets reopened, the ventricular excursion being consistently less than those observed early in diastole (Fig. 4; Fig. 6, panel 1) and started to close again immediately.

The onset of closure, measured from the beginning of the P wave of the electrocardiogram (Fig. 2), averaged 110 ± 21 ms and occurred clearly before the QRS complex. Closure appeared to be slower at first and more rapid during the second half with both leaflets reaching their respective closed positions simultaneously. The rate of closure of the anterior leaflet was practically twice the rate of the posterior (Table 1) thus ensuring simultaneous arrival of both cusps at their closed positions. Heart rate changes over the range studied did not significantly affect the closing time, the peak closing speed of both leaflets (Fig. 7), or the time of complete closure, measured from the Q wave of the electrocardiogram (Figs. 2 and 8). This time interval showed remarkably little variation (63 ± 11 ms).

Measurements of the distance between markers 1 and 4 (Fig. 1) placed on the valve annulus provided information about annular size during the cardiac cycle. They indicated that presystolic annular narrowing (12, 13) occurs before or concomitantly with atrial leaflet opening.

Leaflet motion during isolated atrial contractions. Eleven isolated atrial beats were available for analysis and the data are presented in Table 2. Atrial contractions were invariably followed by leaflet opening and immediate reclosure. The onset of closure, measured from the P wave of the electrocardiogram, averaged 118 ± 15 ms and was practically the same as during sinus rhythm. Valve closure was partial and depending on the strength of atrial contraction varied between 40 and 95% of complete closure.
MOTION OF BOTH MITRAL VALVE LEAFLETS

FIG. 6. Mitral leaflet motion during normal (left) and spontaneously prolonged P-R interval (right) at identical heart rates in same dog (17.6 kg, morphine-pentobarbital anesthesia). Diastolic closure of both cusps was diminished when atrial contraction occurred earlier in diastole.

FIG. 7. Relationship of peak closing speed of posterior mitral leaflet and heart rate (regular sinus rhythm, 6 experiments represented by different symbols). Variations in heart rate had no apparent effect on rate of leaflet closure.

FIG. 8. Relationship of time of complete valve closure (measured from Q wave of the electrocardiogram) and heart rate (5 experiments represented by different symbols). Time interval was not affected by heart rate variations.

TABLE 2. Summary of mitral leaflet motion data following isolated atrial contractions (11 atrial beats from three experiments, complete atrioventricular block induced by vagal stimulation)

<table>
<thead>
<tr>
<th>Leaflet</th>
<th>Onset of Leaflet Closure (from P Wave of ECG)</th>
<th>Leaflet Peak Closing Speed</th>
<th>Leaflet Peak Opening Speed</th>
<th>Duration of Leaflet Closure</th>
<th>% Complete Closure</th>
</tr>
</thead>
</table>
| Anterior leaflet | 118 ± 15, 100-140 ms | 8 ± 4, 5-18 cm/s | 2 ± 2, 4-12 cm/s | 27 ± 15 ms, 10-67 ms | 73 ± 15,
| Posterior leaflet | 119 ± 17, 94-147 ms | 5 ± 1, 4-7 cm/s | 5 ± 2, 3-9 cm/s | 25 ± 9 ms, 13-47 ms | 70 ± 12,

Values are means ± SD; range indicated below.
FIG. 9. Comparison of leaflet motion during a regular atrial-ventricular contraction (middle) and during two isolated atrial contractions (17.6 kg dog, morphine-pentobarbital anesthesia, 2:1 atrio-ventricular block). Atrial leaflet closure was slower than during sinus beat, incomplete, and of very short duration.

FIG. 10. Effect of an isolated atrial contraction on mitral cusps (17.6 kg dog, morphine-pentobarbital anesthesia, complete A-V block). Atrial contraction caused leaflet opening, followed by partial transient closure and return to semiclosed valve position.

FIG. 11. Complete closure of mitral cusps by two isolated ventricular contractions (19.7 kg dog, morphine-pentobarbital anesthesia, complete A-V block). Slow idioventricular rhythm (left) and regular ventricular pacing (right). Following diastolic closure (similar to that during slow sinus rhythm), mitral cusps remained motionless during long diastole until complete closure by ventricular contractions. Delayed opening and diastolic closure of posterior leaflet (left) was presumably caused by a decrease in transmural flow and vortex asymmetry.
was reduced, opening of the posterior leaflet was delayed, and diastolic closure was asymmetrical (Fig. 11, left).

**DISCUSSION**

The present study was designed to provide information about the motion of both mitral cusps in the intact animal during sinus rhythm, over a wide range of heart rates, and during isolated atrial and ventricular contractions. This question was explored in intact dogs in which the valve leaflets and the valve annulus were rendered visible by the insertion of small radiopaque markers. This approach permitted direct and precise observations of cusp motion.

During moderate heart rates, the pattern of leaflet motion described by echocardiography in man (3-5) and in the open-chest dog (7, 9), namely partial leaflet approximation following diastolic opening, immediate valve re-opening during atrial contraction, and closure by the combined effect of atrial and ventricular contractions, was reaffirmed by the present experiments which in addition indicated that except for the delayed opening of the posterior cusp, time-motion of both leaflets is similar throughout diastole.

Time intervals of leaflet motion (Fig. 2: opening time, closing time, and time of complete valve closure, measured from the Q wave of the electrocardiogram) varied little and did not appear to be significantly affected by the wide variations in heart rate. Peak opening speed of the anterior leaflet averaged 29 ± 7 cm/s and was similar to values obtained by echocardiography in man (5); it was not significantly affected by heart rate changes and the same was true for the peak closing speed (37 ± 16 cm/s).

The simultaneous closure of the mitral cusps by the contracting ventricle (either during sinus rhythm or isolated ventricular contractions) is apparently ensured by the different rates of leaflet edge approximation. Since at the onset of ventricular contraction the edge of the larger anterior leaflet was seen to be further away from its closed position than that of the posterior, closure at identical rates would result in asymmetry with the posterior cusp arriving at its closed position first, a situation that could result in mitral incompetence.

Diastolic valve closure is thought to be due to vortex formation within the ventricle with intraventricular pressures being higher behind the leaflets than in the area of the valve annulus (2, 11). In the present experiments, it was observed that diastolic closure begins immediately after leaflet opening, suggesting that vortex formation starts during the early phase of ventricular filling. Careful analysis of left atrial angiograms demonstrated that contrast medium entering the ventricle spread behind the leaflets early in ventricular filling while the ventricular cavity was still small. This was coincident with the onset of leaflet movement toward the atrium. The ventricle then gained volume rapidly and flow appeared to travel mainly toward the ventricular apex. This period corresponded to the observed short pause or slowing of leaflet closure. Finally as the blood stream diverged and ran parallel to the ventricular walls, toward the atrioventricular annulus, leaflet closure resumed and continued until blood flow into the ventricle appeared to diminish. The effect of vortex activity throughout this period of diastole was symmetrical and both leaflets started and ended partial closure practically at the same time. Diastolic closing rate of the anterior leaflet in these experiments averaged 10 ± 3 cm/s (range 5-17) and was higher than values obtained by echocardiography in the open-chest dog (7). Whether the concomitant return of the basilar attachment of the valve cusps, i.e., the valve annulus (Fig. 3) to its original position facilitated in any additional way diastolic valve closure could not be determined from our observations.

The duration of diastolic closure varied and showed an inverse correlation with heart rate (Fig. 5). This could support the concept that during slow and moderate heart rates duration of diastolic valve closure and duration of rapid ventricular filling are directly related. This would
not be true during fast heart rates or when atrial contraction occurs early in diastole so that rapid ventricular filling and flow due to atrial contraction merge (8).

Since our observations suggest that the first small quantities of blood entering the ventricle early in diastole do initiate diastolic leaflet closure in a way similar to the aortic valve cusps during ventricular ejection (1, 14), it is unreasonable to expect in the intact heart, during normal ranges of transmitral flow, that the size of the mitral valve orifice would be proportional to volume flow. Indeed remarkably little variation was observed in the individual dog both in the maximal excursions of the leaflets (Fig. 4) and in the distance between them early in diastole (Table 1).

During slow heart rates, after termination of diastolic closure, the cusps remained practically motionless (with the exception of early rebounds or small slow oscillations) in their semiclosed position until subsequent atrial or ventricular contraction (Figs. 4 and 11), indicating equilibrium between the forces acting on the two aspects of the cusps. Continuation of closure or leaflet reopening was never observed, and in the same animal the floating position of the cusps seemed to be constant. It would appear to us that this semiclosed cusp position is probably due to a rapid decay of ventricular vortex activity rather than to tension exerted by the chordae tendineae as suggested previously (10). The latter explanation appears unlikely since, during long diastole, isolated atrial contractions did invariably produce leaflet reopening and closure (Fig. 10).

Valve motion following isolated atrial contractions was characterized by leaflet opening and immediate slow partial closure of varying degree and short duration followed by a slow reopening (Figs. 9 and 10) with the rate of anterior leaflet closure being similar to its peak diastolic closing speed. These observations would suggest a similar closing mechanism for atrial and for diastolic valve closure. Analysis of left atrial angiograms demonstrated the spreading of contrast material behind the valve cusps during isolated atrial contractions. The fact that transient atrial valve closure was at times more complete than during early ventricular filling is possibly related to differences in ventricular size, duration of transmitral flow, and degree of leaflet opening.

During regular sinus rhythm, the onset of valve closure at the end of diastole is caused by atrial contraction. This was evidenced by 1) the beginning of atrialward movement of both leaflets, under all heart rates studied, which occurred before the QRS complex of the electrocardiogram; 2) the onset of closure during sinus rhythm and during isolated atrial contractions, measured from the P wave of the electrocardiogram, was practically identical. Our data do not allow us to answer with certainty the old controversy concerning the importance of atrial systole for competent valve closure, since at the end of diastole and in the absence of atrial contraction the valve was never found to be wide open as might have been expected, and no regurgitation could be detected following valve closure by isolated ventricular contractions. Though it seems unlikely then that under normal transmitral flow conditions atrial contraction is necessary for competent leaflet closure, it may become important when smaller-than-normal quantities of blood enter the ventricle with low velocity, causing a decrease in intraventricular vortex activity and diminished diastolic leaflet approximation.

The observations reported in this study may be true only for the center of the mitral cusps (the sites of marker attachment), and for normal conditions of ventricular diastolic volume, degree of ventricular emptying, and transmitral flow. Delayed valve opening, reduced leaflet excursions, diminished diastolic closure, and asymmetry of opening and closure might be expected to occur under pathologic conditions.

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