PAPILLARY MUSCLE-LEFT VENTRICULAR WALL "COMPLEX"

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Objectives. Mitral valve homografts, despite theoretical advantages, are not widely used, in part because of lack of basic information about the three-dimensional geometry of the mitral apparatus. **Methods:** Radiopaque markers were used in the study of eight closed-chest dogs under four conditions: (1) baseline, (2) caval occlusion, (3) tachycardia (atrial pacing), and (4) nitroprusside infusion. Using a cylindrical coordinate system, defined with the origin at the midpoint between the anterior and posterior commissures, and the left ventricular long axis (z-axis), defined by the origin and the left ventricular apex, D_{TIP-MA} (the z-coordinate [millimeters] of the papillary muscle tip), was measured at 10 time points throughout the entire cardiac cycle. $D_{BASE-MA}$ (the z-coordinate of the papillary muscle base) and L_{PM} (the length of the papillary muscle [millimeters]) were also measured. **Results:** D_{TIP-MA} varied slightly with time ($p < 0.001$ by analysis of variance), but the magnitude of change was negligible (<0.9 mm) (e.g., D_{TIP-MA} of the anterior papillary muscle was $20.7 \pm 2.7/20.8 \pm 2.8$ [end-diastolic/end-systolic, mean ± 1 standard deviation]; D_{TIP-MA} of the posterior papillary muscle was $25.8 \pm 4.8/25.5 \pm 4.5$). D_{TIP-MA} was minimally influenced by the above perturbations. $D_{BASE-MA}$ and L_{PM} of each papillary muscle, however, changed throughout the cardiac cycle ($p < 0.001$ by analysis of variance) by about 4 mm, and both parameters were dependent on loading conditions. **Conclusions.** Papillary muscle length changed to keep the D_{TIP-MA} distance constant such that the papillary muscle and left ventricular wall functioned together as a unit ("J-shaped complex"). These results provide a physiologic rationale for measuring D_{TIP-MA} , define its potential surgical usefulness, and imply that using the entire length of the donor's papillary muscle (i.e., maintaining the entire J-shaped complex) is important in operations in which homograft or stentless xenograft mitral valves are used. (J Thorac Cardiovasc Surg 1997;113:292-301)

Use of a mitral valve homograft in the clinical setting was first reported by Senning and Largiadér¹ three decades ago and was supported by encouraging results from laboratory studies.²⁻⁴ Be-

cause of its anatomic features, the mitral homograft may function like a normal mitral valve in preserving valvular-ventricular interaction,⁵⁻⁷ sphincteric motion of the anulus,⁸⁻¹¹ and the saddle shape¹² of the

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Fig. 1. **A,** Marker array in the left ventricle, PMs, and the mitral annulus. **B,** A cylindrical coordinate system. **A,** LV markers were placed subepicardially and annular markers were sewn through a left atriotomy with the animal supported by on CPB. *Black squares* illustrate the markers on the PM bases and *black diamonds* show the markers on the PM tips. **B,** The "midpoint" between the anterior commissure and posterior commissure markers serves as the origin of the reference system; the line from the origin to the LV apex is the z-axis. APM, Anterior PM; PPM, posterior PM; LV, left ventricular; ACOM, anterior commissure; PCOM, posterior commissure.

annulus more completely than other types of valve substitutes. Mitral homograft or stentless xenograft valves have not been widely used in patients, however, because of discouraging clinical results.¹³⁻¹⁸ Problems encountered with mitral homografts include limited durability of the leaflets, chordae tendineae, or papillary muscles (PMs) (including the suture lines^{15,16}) and difficulty with surgical implantation. Such technical challenges (including proper sizing¹⁹ and orientation) may be due in part to lack of basic information about the functional three-dimensional geometry of the mitral valve annulus and subvalvular apparatus. Recently, with improved tissue preservation techniques (e.g., cryopreserved aortic homograft²⁰) and increased experience in reparative mitral surgery,²¹ mitral homografts and stentless mitral xenograft valves are again attracting attention.²²⁻²⁵ In this article we report an interesting, well-regulated spatial relationship between the PM and the mitral annulus, which is termed the *J-shaped complex*. The long descending limb of the J-shaped complex is the left ventricular (LV) wall extending from the annulus to the PM base, and the short ascending limb is defined as the PM. This

quantitative approach may be of clinical utility for homograft sizing and orientation, as well as for planning extensive mitral reparative operations.

Materials and methods

Surgical preparation. Eight healthy adult mongrel dogs (30 ± 5 kg, mean $\pm$ standard deviation) were premedicated with acepromazine (0.01 to 0.05 mg/kg intramuscularly) and atropine sulfate (0.05 mg/kg intravenously). Details of this preparation have been described elsewhere.¹¹ The dogs were anesthetized with sodium thiopental (20 mg/kg intravenously), intubated, and supported with a ventilator. General anesthesia was maintained with inhalational isoflurane at 1% to 2.2%. The left side of the chest was opened in the fifth intercostal space, pneumatic occluders were applied to the inferior and superior venae cavae, and the heart was exposed and suspended in a pericardial cradle. Miniature tantalum radiopaque helices (inner diameter 0.8 mm, outer diameter 1.3 mm, length 1.5 to 3.0 mm) were inserted into the LV wall and interventricular septum at selected sites (Fig. 1, A). Eight markers were placed into the LV subepicardial layer along four equally spaced longitudinal meridians around the circumference of the ventricle (in the anterior, lateral, posterior, and septal walls). Each meridian contained markers at two levels (the basal-equatorial and apical-equatorial planes). A ninth marker was placed

at the LV apex. The septal markers were inserted with the aid of two-dimensional epicardial echocardiographic guidance to place the markers at the desired sites on the right ventricular surface of the interventricular septum. The animal was heparinized (300 IU/kg intravenously), and cardiopulmonary bypass (CPB) was instituted; a 14F or 16F arterial cannula was inserted into the right femoral artery, and a two-stage venous cannula was inserted into the right atrium and inferior vena cava. CPB was instituted with a roller pump and bubble oxygenator. The ascending aorta was crossclamped, and the heart was arrested with 500 ml of cold crystalloid cardioplegic solution delivered in an antegrade fashion. Topical cold saline solution was used subsequently to keep the LV myocardial temperature less than 16° C. The left atrium was opened, exposing the mitral apparatus. As illustrated in Fig. 1, *A*, one miniature gold marker was sewn to the largest tip of each PM and one tantalum marker to the base of each PM. Four miniature tantalum markers were then sutured to the mitral annulus, one near each commissure and one each on the midanterior and midposterior annulus. An implantable Königsberg pressure transducer (P 4.5X, Königsberg Instruments, Inc., Pasadena, Calif.) was placed in the LV lumen via the apex for subsequent monitoring of LV chamber pressure. The aortic cross-clamp was released, the left atriotomy closed, the heart defibrillated if needed, and the dog weaned from CPB. Heparin was reversed with protamine sulfate, and the pericardium was loosely reapproximated. Chest tubes were placed, and the incision was closed. The animals recovered in the intensive care unit of the Stanford University Department of Laboratory Animal Medicine. Oxymorphone hydrochloride (Numorphan, 0.05 to 0.2 mg/kg intravenously, DuPont Merck Pharma, Mantec, Puerto Rico) was used to minimize discomfort.

Experimental protocol. Seven to 12 days after the operation (9 ± 2 days [mean ± 1 standard deviation]), the animal was taken to the experimental animal cardiac catheterization laboratory for hemodynamic and videofluorographic data acquisition. Mild sedation was accomplished with diazepam (5 mg intravenously) and supplemental ketamine (5 mg/kg intravenously), as needed. A micromanometer-tipped catheter (MPC-500, Millar Instruments, Inc., Houston, Tex.) was advanced via a left femoral artery cutdown to the ascending aorta for arterial pressure monitoring. To minimize reflex sympathetic and parasympathetic responses that can occur in conscious animals, autonomic blockade was accomplished with esmolol (infused intravenously at a rate of 0.25 to 0.4 mg/kg per minute, titrated to reduce the heart rate to below 120 min^{-1}) and atropine sulfate (0.02 to 0.04 mg/kg intravenously). UL-FS49 (Boehringer-Ingelheim, Ridgefield, Conn.), a highly specific negative chronotropic agent that does not change the QT interval, inotropic state, or systolic or diastolic blood pressure,²⁶ was also administered (4 to 6 mg intravenously in a single dose) to lower the heart rate further, if needed. Data were obtained in normal sinus rhythm in all but one dog, which was atrially paced at 120 min^{-1} because of rate-dependent atrioventricular dissociation. Baseline hemodynamics and biplane videofluoroscopic data recordings were obtained during

steady-state conditions and during preload reduction by vena caval occlusion. Subsequently, data were taken during tachycardia by atrial pacing. Finally, data were acquired during afterload reduction induced by intravenous infusion of sodium nitroprusside.

All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" prepared by the National Academy of Sciences and published by the National Institutes of Health (DHEW [NIH] publication No. 85-23, revised 1985). The study was approved by the Stanford University Medical Center Laboratory Research Animal Review Committee and conducted according to Stanford University policy.

Data acquisition. All imaging studies were conducted with the animal in the right lateral decubitus position with the use of a Philips Optimus 2000 biplane Lateral ARC 2/Poly DIAGNOST C2 system (Philips Medical Systems International B.V., Best, The Netherlands) with the image intensifiers in the 7-inch cine mode. The 45-degree right and left anterior oblique biplane images were recorded simultaneously at a rate of 60 frames/sec on Sony U-Matic 5800 3/4-inch videocassette recorders (Sony Electronics, Inc., Montvale, N.J.). The analog LV pressure signal was digitized and recorded on each individual video image by means of a custom intelligent video controller (Control Video Corp., Campbell, Calif.); the upstroke of the R wave of the electrocardiogram was detected electronically and also digitally encoded on the videotape as an end-diastolic timing marker. At the completion of the study, images of grids containing 1 cm squares and biplane images of a three-dimensional helical phantom of known dimensions were recorded to determine radiographic distortion and magnification factors. The two-dimensional coordinates of each marker in each projection (x,y and y,z) were digitized frame by frame with the use of a semi-automated, computerized myocardial marker detection system (Hewlett-Packard RS/20 [Palo Alto, Calif.], equipped with Matrox MVP/AT/NP image processing boards [Dorval, Quebec, Canada]) and custom image-processing and digitization software developed in our laboratory.²⁷ The data from the two views were corrected for X-ray magnification and distortion and were merged with the use of custom software to yield the three-dimensional coordinates of each marker every 16.7 msec, as previously described.^{28, 29}

During data acquisition, two channels of analog data (LV pressure and surface-lead electrocardiogram) were acquired and digitized simultaneously at 240 Hz with the use of a 486-based microcomputer (486-33, JDR Microdevices Inc., San Jose, Calif.) with a high-speed data acquisition card (DT 3831-G, Data Translation Inc., Marlboro, Mass.) controlled by commercially available software (Labtech Control 3.2.0, Laboratory Technology Corp., Wilmington, Mass.). These analog signals were simultaneously recorded on a multichannel recorder at a paper speed of 25 mm/sec. The 240 Hz pressure data were temporally aligned with the 60 Hz marker-derived geometry data by frequency-corrected convolution of the 240 Hz and 60 Hz signals.

Fig. 2. Time-course of change as a function of time of the z-coordinate of the tip and base of the anterior PM (APM) and the length of the APM throughout one cardiac cycle during the following conditions: [1] baseline, [2] reduced LV preload by vena caval occlusion (VCO), [3] lowered reduced LV afterload by infusion of sodium nitroprusside (SNP), and [4] tachycardia using atrial pacing. The X axis of each graph indicates the 10 time points during the cardiac cycle, and the Y axis depicts the z-coordinate of the APM tip or base and the length of the APM (millimeters, mean $\pm$ 1 standard deviation). Systole and diastole were both divided into five equal time interval segments. The leftmost panel indicates baseline conditions; sequentially moving to the right, VCO, SNP, and pacing data are portrayed. Note that the APM tip z-coordinate was nearly constant throughout the cardiac cycle and that this pattern did not change during the various LV loading conditions; conversely, the z-coordinate of the APM base as well as APM length varied considerably with time during the cardiac cycle and during VCO. * $p < 0.005$ versus baseline at the individual time points denoted (paired t tests).

Data analysis.

Hemodynamics. To minimize the effects of intrathoracic pressure variation, only end-expiratory beats were selected for analysis. The time derivative of the LV pressure signal was computed to determine peak positive LV dP/dt (dP/dt_{max}) and peak negative LV dP/dt ($-dP/dt$). For each cardiac cycle, the time of end-diastole was defined as the videofluoroscopic frame containing the R-wave marker of the electrocardiogram, and end-systole was defined as the videofluoroscopic frame immediately before the frame that contained the point of maximum negative LV dP/dt . LV ejection fraction was calculated as $([EDV - ESV]/EDV) \cdot 100\%$, where EDV and ESV are end-diastolic and end-systolic LV volumes. The method used to compute LV volume has been described previously.¹¹

Distance between the PM tip and mitral anulus. The distances between the PM tip/base and the mitral anulus were measured as the z-coordinate by means of the cylindrical coordinate system¹¹ shown in Fig. 1, B. The origin was placed at the midpoint between the anterior and posterior commissures of the mitral anulus, and the LV long axis (z-axis) was directed toward the LV apex; the three-dimensional location of each marker is expressed as the distance to the anulus (z, millimeters), radial position from the z-axis (r, millimeters), and angle (Λ , degrees; $\Theta = 0$ degrees precisely between both commissures on the

midposterior anulus, $\Theta = +90$ degrees at the anterior commissure and $\Theta = -90$ degrees at the posterior commissure). We also measured the length of each PM as the distance between markers at the PM tip and base.

Statistical analysis. All data are reported as mean $\pm$ 1 standard deviation. The z-coordinate and PM length were compared with respect to time (i.e., at 10 time points during the cardiac cycle) by means of multivariate analysis of variance, with dog identification entered as a dummy variable to control for between-animal variability differences. Post hoc testing was performed by means of Bonferroni's correction. The z-coordinate of the PM markers and the true three-dimensional PM length were compared between baseline and each intervention (such as caval occlusion, nitroprusside infusion, and pacing) by t tests for paired observations. Statistical significance was inferred if the corrected p value was less than 0.05. For all statistical analyses, SYSTAT (version 5.02, SPSS Inc., Chicago, Ill.) was used.

Results

Hemodynamics. Average heart rates were 108 ± 11 , 103 ± 9 , 108 ± 14 , and $131 \pm 5 \text{ min}^{-1}$ during baseline, caval occlusion, nitroprusside infusion, and

Fig. 3. Relative change (expressed as a percentage) with respect to time of the z-coordinate of the tip and base of the anterior PM (APM) and the length of the APM in one representative cardiac cycle during the four conditions (each time point vs end-diastole). Note that the relative change of the z-coordinate of the APM tip oscillated around zero (i.e., was nearly constant); the APM base z-coordinate changed during the cardiac cycle, and the length of the APM varied even more compared with the z-coordinate of the APM base (percent, mean $\pm$ 1 standard deviation). This pattern was similar for all four LV loading conditions: [1] baseline, [2] vena cava occlusion [VCO], [3] sodium nitroprusside [SNP], and [4] tachycardia (pacing).

atrial pacing ($p < 0.001$ vs baseline), respectively. LV end-diastolic volumes (uncorrected for LV wall mass, inasmuch as these were epicardial marker measurements, which included myocardial volume) were 143 ± 16 , 104 ± 13 , 133 ± 11 , and 139 ± 14 ml, respectively ($p < 0.001$, caval occlusion vs baseline; $p < 0.05$, nitroprusside or atrial pacing vs baseline). LV ejection fractions (epicardial) were $21\% \pm 5\%$, $11\% \pm 4\%$, $21\% \pm 4\%$, and $19\% \pm 4\%$, respectively ($p < 0.001$, caval occlusion vs baseline; $p = 0.01$, atrial pacing vs baseline). Maximum LV systolic pressures were 132 ± 23 , 92 ± 20 , 118 ± 19 , and 137 ± 22 mm Hg, respectively ($p < 0.001$, caval occlusion and nitroprusside vs baseline; $p = < 0.041$, atrial pacing vs baseline). $dp/dt_{\max}$ values were 1591 ± 182 , 1161 ± 206 , 1554 ± 206 , and 1700 ± 206 mm Hg/sec, respectively ($p < 0.001$, caval occlusion vs baseline; $p < 0.05$ [$p = 0.018$] atrial pacing vs baseline).

Anterior PM. In baseline conditions, there were statistically significant differences ($p < 0.001$ by analysis of variance) between the 10 time points for the z-coordinate of the PM tip and base, as well as for PM length. The magnitude of change in the anterior PM tip z-coordinate, however, was less than 0.9 mm; this was a small change compared with the

z-coordinate of the anterior PM base or the PM length distance, which varied by about 4 mm (Fig. 2).

With preload reduction (vena caval occlusion), the z-coordinate of the anterior PM base and the anterior PM length both decreased significantly ($p < 0.001$ vs baseline); on the other hand, the z-coordinate of the anterior PM tip did not differ from baseline during systole ($p =$ not significant [p ranging from 0.09 to 0.75 for all time points for vena caval occlusion vs control, t tests for paired observations]) and throughout most of diastole ($p =$ not significant [$0.26 < p < 0.95$]). At one time in mid-diastole, however, there was a significant change versus baseline ($p = 0.02$), but the absolute difference was very small (0.4 mm) and most likely inconsequential (see Fig. 2). With afterload reduction (sodium nitroprusside infusion), the z-coordinate of the anterior PM tip also remained constant ($p =$ not significant [$0.07 < p < 0.996$ vs baseline], except again at one time point in mid-diastole ($p = 0.022$), but the magnitude of the difference was negligible (0.3 mm larger than control). With tachycardia, the z-coordinates of both the anterior PM tip and base were very similar to baseline measurements most of the time ($p =$ not significant [$0.083 < p < 0.43$ vs control]) except for two time points in

Fig. 4. Time-course of change of the z-coordinate of the tip and base of the posterior PM (PPM) and the length of the PPM for one typical cardiac cycle during with the following conditions: [1] baseline, [2] reduced preload by vena caval occlusion (VCO), [3] lowered afterload by infusion of sodium nitroprusside (SNP), and [4] tachycardia by atrial pacing. The z-coordinate of the PPM tip was nearly constant throughout the cardiac cycle compared with the z-coordinate of the PPM base and PPM length, which varied considerably (millimeters, mean $\pm$ 1 standard deviation). As LV loading conditions were altered, this pattern did not change. * $p < 0.005$ versus baseline at the individual time points indicated by t test for paired comparison.

early systole ($p = 0.025, 0.018$) and in mid-diastole ($p = 0.004, 0.025$), where again the actual magnitude of the difference was very small (≤ 0.3 mm); conversely, during caval occlusion, the anterior PM length was shorter than the baseline measurements, especially during systole ($p < 0.005$).

The percent change in the z-coordinate of the anterior PM tip and base and change in anterior PM length are shown in Fig. 3. The percent reduction in anterior PM length was greater during systole (i.e., up to 12%) than the fractional change in the z-coordinate of either the anterior PM base or tip (i.e., maximum changes of 8% and 3.5%, respectively, $p < 0.001$ by analysis of variance).

Posterior PM. The posterior PM behaved like the anterior PM, except that the posterior PM was shorter than the anterior PM and the z-coordinate of the posterior PM tip was greater than that of the anterior PM tip (i.e., located farther from the anulus). During baseline conditions, there was a statistically significant difference (analysis of variance, $p < 0.001$) among the 10 time points for the z-coordinate of the PM tip and base, as well as for PM length (Fig. 4). The magnitude of change in posterior PM tip z-coordinate, however, was again

less than 0.9 mm, compared with a change of almost 4 mm for the z-coordinate of the posterior PM base and the posterior PM length (Fig. 4).

With caval occlusion, the z-coordinate of the posterior PM base and the posterior PM length both decreased significantly ($p = 0.003$ and 0.002 , respectively, vs baseline); on the other hand, the z-coordinate of the posterior PM tip changed significantly only at two time points near mid-systole (see Fig. 4). With nitroprusside infusion, the z-coordinate of the posterior PM tip did not change compared with baseline ($p = \text{not significant}$ [$0.143 < p < 0.824$]) except at two time points in mid-diastole ($p = 0.039$ and 0.042), where the difference was only 0.3 mm. With tachycardia, the z-coordinates of the posterior PM tip and the base and PM length were all very similar to baseline measurements.

The percent change in the z-coordinate of the posterior PM tip and base and the posterior PM length are illustrated in Fig. 5. Percent reduction in posterior PM length was greater during systole (i.e., up to 10%) than the relative change in the z-coordinate of either the posterior PM base or the tip (i.e., up to 5% and 2.5%, respectively, $p < 0.001$ by analysis of variance).

Fig. 5. Fractional or relative change as a function of time of the posterior PM (PPM) tip and base z-coordinates and the PPM length over one representative in one cardiac cycle during the same four LV loading conditions indicated in Figs. 2 to 4 (each time point versus end-diastole). Notice that the relative motion of the z-coordinate of the relative PPM tip was close to zero whereas the PPM base z-coordinate changed considerably more and the PPM length even more over time (percent, mean $\pm$ 1 standard deviation). The pattern seen in the baseline measurements (*left panel*) was similar to that during the other three conditions.

Discussion

The change of the z-coordinate of both PM tips was negligible for practical surgical purposes (e.g., less than 1 mm), when the reproducibility of echocardiography and accuracy of surgical visualization are taken into account. The relatively load-insensitive nature of the PM tip-mitral annulus distance demonstrated in this study may provide a sound physiologic rationale supporting the method of sizing mitral homografts reported by Acar and associates,²⁴ who used the following parameters: (1) the height of the anterior mitral leaflet in diastole, (2) the anteroposterior diameter of the annulus in systole, and (3) the distance between the annulus and the PM tip.

The results of this study show that the length of the PM changed in just the right way to keep the z-coordinate of the PM tip nearly constant with respect to the mitral annulus. To clarify this concept, we propose the notion of a "J-shaped complex" comprising two components working together: a long descending limb (LV wall from the annulus to the PM base) and a short ascending limb (the PM per se). We assume that one of the reasons for the short ascending limb offsetting the motion of the long descending limb in the z direction was the

different curvature between the two limbs. The ascending limb (i.e., PM) has nearly no curvature, but the descending limb (i.e., LV wall) has a curved shape; thus shortening of fibers in the descending limb will not result in proportional shortening in the z-direction. Another reason relates to relatively more systolic thickening of the LV free wall than the PM.

Acar and associates²⁴ described the difficulty associated with implanting a mitral homograft valve in patients who had previously undergone mitral valve replacement, especially when the subvalvular apparatus had been excised during the previous operation. Under these circumstances, the remaining atrophic PM essentially eliminates the ascending limb of the J-shaped complex, and the PM tip cannot maintain a constant distance from the annulus. Acar and colleagues²⁴ also discussed problems stemming from transfixing sutures between the chordae of the homograft through the LV wall. These problems, again, may be related to either absence or dysfunction of the ascending limb of the J-shaped complex, resulting in geometric alterations of the mitral valve that possibly could lead to the development of mitral regurgitation.

Thus, if a recipient's PM is excised²⁵ or if the homograft's PM is sewn to the recipient's PM in a

side-by-side fashion (especially when the homograft is sewn near the base of the recipient's PM¹⁴) or a transpapillary suture is used,¹⁸ the PM-anulus distance may not remain constant and the homograft may be exposed to excessive systolic motion and stresses, possibly resulting in mitral regurgitation; under these conditions, the PM tip may actually move in a similar fashion to the PM base. Even when the homograft is sewn to the tip of the recipient's PM, scar formation around the sutures may have some influence on PM function. The degree of this impact, however, should be much less than that occurring when the homograft is sewn to the recipient's PM mid-body or base or when the recipient's PM is infarcted or partially excised. Therefore care should be taken to try to sew the homograft only to the PM tip. The results of this study suggest the importance of using the entire length (or as much as possible) of the recipient's PM whenever possible to maintain the J-shaped complex.

One limitation of this work is that we studied only normal canine hearts (which are distinctly different from hearts exposed to volume overload from mitral regurgitation¹⁶); however, these observations indicate that future study of the motion and geometry of the components of the J-shaped complex in dilated ventricles both before and after LV remodeling¹⁹ (i.e., before and after surgical correction of mitral regurgitation) should yield even more useful clinical information. If the myocardium is not irreversibly damaged by chronic mitral regurgitation and previous myocardial infarction has not damaged the PM, the J-shaped complex should remain functionally intact, thereby theoretically allowing the distance between the PM tip and the mitral annulus to remain constant both before and after correction of mitral regurgitation.

There are several other limitations to this study. First, we used a normal canine model with PM and chordae tendineae anatomy similar, but not identical to, human anatomy. For example, human PMs (especially the anterior PM) project into the LV chamber more than canine PMs. This projection, however, probably would not alter the constancy of the distance between the PM tip and the annulus because longer PMs shorten and lengthen more. Second, because of the temporal sampling rate limit of the biplane cine x-ray system (60 Hz), it was necessary to reduce the heart rate with esmolol and UL-FS49. The tachycardia studies were conducted at a heart rate of 140 min⁻¹. It is unknown whether the results would be different at a higher or lower

Fig. 6. "J-shaped complex." This complex consists of a descending limb consisting of the LV wall and an ascending limb (the PM). Both limbs should work together in balance to maintain a stable distance between the PM tip and the mitral annulus. If one component sustains injury or ischemia, the functional integrity of the J-shaped complex could be lost and abnormal mitral valve dynamics and geometry result. APM, Anterior papillary muscle; PPM, posterior papillary muscle.

heart rate. Third, we placed the markers on the largest tip of each PM even though multiple PM heads exist; we have no knowledge of the three-dimensional dynamics of the other PM heads.

Despite these limitations, we believe that the information in this current study has potential clinical utility. These data provide physiologic evidence supporting the practice of measuring the distance from the PM tip to the mitral annulus and suggest the importance of using the entire length of a donor's PM whenever possible in mitral homograft surgery, stentless xenograft mitral valve replacement, or extensive mitral valve repair. Further studies in hearts with chronic mitral regurgitation, with or without leaflet prolapse, or those with mitral stenosis and leaflet restriction are warranted.

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Discussion

Dr. Robert W. Frater (Bronx, N.Y.). I cannot find fault with Dr. Komeda's measurements, but I find them counterintuitive. We know, from frequent echocardiographic observations, that the tip of the ventricle unquestionably moves away from the annulus in diastole, presumably taking the PM along, and yet we do not see normal chordal bending. If the annulus-PM distance was constant, the chordae would have to bend for this to happen. We have made a whole series of observations in long-axis echoes of the distance between the tip of the PM and the plane of the annulus, and we find that in fact the tip moves toward the annulus in systole and away in diastole and the difference in length is between 10% and 20%. This does not happen in patients with posterior LV wall damage and happens much less often in hearts that are severely myopathic.

The ejection fractions at the time of study were from 11% to 21%. This suggests to me that this is not an observation in a normal dog but an observation in a dog with a myopathic heart. Could you comment on that?

Dr. Komeda. We measured the distance between the PM tip and the mitral annulus, not between the leaflet tip and the mitral annulus. As you pointed out, if the distance between the PM tip and mitral annulus is constant, something should bend during diastole when the leaflets open; it may be a leaflet, or the junction between the leaflet and chordae, chordae, or their combination. We are currently investigating this question.

When using echocardiography to measure this kind of distance, we must be very careful about which cross-section we are imaging and which precise site we are talking about. We presented a paper pertaining to this issue at the 1995 meeting of the American Heart Association, which will be published in *Circulation*. For instance, if we measure the distance between one point on the posterior annulus near the anterolateral commissure and the posterior papillary tip, it shortens significantly during systole. Similarly, the distance between a posterior annular point near the posteromedial commissure and the anterior papillary tip shortens. That is what we call the "oblique direction," which may be a more physiologic orientation for chordal resuspension during mitral valve replacement. But, if we measure the distance between a posterior annular locus in the vicinity of the posteromedial commissure and the posterior papillary tip, it is constant throughout the cardiac cycle; this was also the case between an annular point near the anterolateral commissure and the anterior papillary tip. We confirmed this observation with both radiopaque marker cinefluoroscopy and two-dimensional epicardial echocardiography, where we knew the exact orientation of the echo cross-section. Therefore measuring the distance between an annular point and a PM tip by transesophageal or transthoracic echocardiography without precisely confirming which cross-section is being imaged can be misleading; this is one of the reasons we measured the distance between the PM tip and a point representing the mitral annular plane.

LV ejection fraction was calculated by means of data derived from the *epicardial* markers. Hence this method did not take into account LV wall thickness or systolic wall thickness, which is a major component of chamber (angiographic or echocardiographic) ejection fraction. When we corrected the ejection fraction estimate for wall thickness, the ejection fraction was approximately 35% and would be considerably higher had LV wall thickening been taken into consideration. Therefore we believe that LV systolic function in this experiment was only slightly depressed.

Dr. Radu C. Deac (Targu-Mures, Romania). The data presented in this study confirm the known shortening of the PM up to 20% but point out the relatively constant distance between the mitral annulus and the tip of the PM. The physiologic significance of this observation has to be elucidated in the future.

Do you have any data about this dimension in ischemic conditions? Using this measurement as the principal dimension of a stentless mitral pericardial valve, we obtained a competent mitral substitute in each of the 23 patients operated on since 1989 in our unit. However, with changed PM-mitral annulus distance in a diseased ventricle, which is smaller in mitral stenosis and variable in mitral regurgitation or mixed mitral valve disease, we had

to attach the graft at a different length than this measured distance, even at a different length for each PM, to obtain a competent valve. With the current technique of allograft and xenograft replacement of the mitral valve, it will be interesting to see how a normal mitral valve will function in a given diseased ventricle.

Dr. Komeda. The geometry of the PMs and mitral annulus in the context of myocardial ischemia and mitral homograft replacement is a key clinical issue.

We presented the geometry of the PMs, mitral leaflets, and mitral annulus with or without ischemic mitral regurgitation (induced by proximal circumflex coronary artery occlusion) at the 1995 meeting of the American Heart Association. During ischemic mitral regurgitation, the z-coordinate of the posterior PM tip did not change; however, and quite unexpectedly, the anterior PM tip (on the nonischemic side) became translocated toward the LV apex because of ventricular dilatation and a normally functioning anterior PM. In the radial direction (*r* values), both the anterior and posterior PM tips were dislocated, but the magnitude of change was much larger on the posterior PMs (or ischemic side). We agree that when a mitral homograft or stentless mitral valve is used for patients with ischemic mitral regurgitation, this perturbed geometry needs to be contemplated.

Dr. Carlos M. G. Duran (Missoula, Mont.). The message is that the distance between the tip of the PM and the annulus does not change during the cardiac cycle under any circumstance. This became obvious to me when I saw the videos of Dr. Van Zwinker, who showed this phenomenon very clearly. The morphologic explanation is the presence of two basal chordae to the anterior leaflet, which we call "stay chordae" and which can be seen very clearly by echocardiography. If I may be allowed a semantic comment, I suggest that instead of calling the ventriculomitral functional unit the "J-shaped complex," rather use the "ventricular loop" as a more appropriate term for a closed and not open complex.

My question for the authors is this: How do you expect this information to help in mitral homograft transplantation? In the three cases we have performed, we have used this principle as the key distance to correctly determine where to suture the donor to the recipient PMs.

Dr. Komeda. You are correct, Dr. Duran. Functionally the PMs and the LV wall make a closed loop. Morphologically, however, there is a gap in this loop between the PM tip and mitral annulus, at least during diastole, when certain chordae may be redundant or slack; this is why we call this a "J-shaped" complex. By the term "complex," we meant not only the PM-annulus continuity, but also the geometric changes modulated by all elements of the entire structure (i.e., PMs and mitral annulus). Concerning your question about applying these findings to clinical mitral homograft surgery, if we can use the entire length of the recipient PM (and assuming it is viable), a relatively stable condition should result. On the other hand, if one resects the tip of the recipient PM or sews the donor's PM to the base of the recipient PM (as done in many previous reports), early failure of the homograft or substantial mitral regurgitation may result, perhaps caused in part by excessive motion or tension, or both.