

# Technique and outcome of mitral valve replacement in dogs

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**M**itral regurgitation (MR) secondary to degenerative mitral valve disease (DMD) is the most common heart disease in dogs, accounting for about 40% of cases in this species.<sup>1</sup> Dogs with dilated cardiomyopathy (DCM) may have severe MR as a component of their disease. Progressive congestive heart failure (CHF) and death are the inevitable consequences of severe MR despite optimal medical treatment.

Mitral valve replacement offers the possibility of complete correction of MR and reversing the course of CHF. Except for 2 case reports<sup>2,3</sup> and a series in clinically normal dogs,<sup>4</sup> mitral valve replacement has been largely unexplored as a treatment option for dogs with MR. The purpose of the study reported here was to describe a technique for and assess outcome of mitral valve replacement with a mechanical prosthesis in dogs with naturally occurring severe MR.

## Procedures

Dogs were considered candidates for mitral valve replacement if they had severe mitral regurgitation, were in CHF, and had an apparent absence of serious noncardiac disease. Severe MR was defined as turbulent flow in > 50% of the area of the left atrium as revealed by color-flow Doppler echocardiography (either right parasternal long axis or left apical 4-chamber views).<sup>5,6</sup> Congestive heart failure was defined as a requirement for long-term treatment with furosemide to control or prevent recurrence of cardiogenic pulmonary edema. Selection of dogs that met the inclusion criteria for mitral valve replacement was based on the owners' acceptance of the inherent risks and financial obligation of surgery.

Left ventricular and atrial dimensions were obtained by M-mode echocardiography from right parasternal and left ventricular outflow views.<sup>7,8</sup> Left ventricular diastolic volume index (LVDVI) and left ventricular systolic volume index (LVSVI) were calculated by cubing the left ventricular diastolic and systolic dimensions, respectively, and dividing the resulting value by body surface area.<sup>9</sup> The left atrium-to-aorta (LA:Ao) ratio and degree of MR were evaluated, as described.<sup>5,7</sup> Differences in echocardiographic values or indices before and after surgery were determined by use of the Wilcoxon signed rank test.<sup>a</sup> Values of  $P < 0.05$  were considered significant.

In this study, the basic protocols for anesthesia and cardiopulmonary bypass used were those performed previously by this group.<sup>10-14</sup> Arterial cannulation for

cardiopulmonary bypass was either in the left femoral or right carotid artery, depending on the size of the dog. A thoracotomy was performed via the right fifth intercostal space. Venous drainage was performed via direct cannulation of both vena cavae with angled cannulae. Cardiopulmonary bypass was instituted, and the dogs were cooled to approximately 28°C (esophageal temperature). Mitral valve replacement was performed during hypothermic cardiac arrest with the ascending aorta cross-clamped. Cold (4°C [39.2°F]) cardioplegic solution was administered via a cannula placed in the aortic root to initiate and maintain cardiac arrest; the solution was administered every 20 minutes while the aorta was cross-clamped. The solution used to induce cardioplegia was a crystalloid-sanguinous mixture.<sup>13</sup>

The mitral valve was approached via an incision in the left atrium that was dorsal to the interatrial groove, ventral to the right pulmonary veins, and within the pericardial space as described for mitral valve repair.<sup>14</sup> The septal leaflet of the mitral valve and its associated chordae tendineae were excised (Figure 1). The mural leaflet of the mitral valve and its associated chordae tendineae were preserved. Valve-sizing devices provided by the valve manufacturer<sup>b</sup> were used to determine the size of the valve prosthesis that would fit easily into the mitral orifice. Mattress sutures of 3-0 polyester

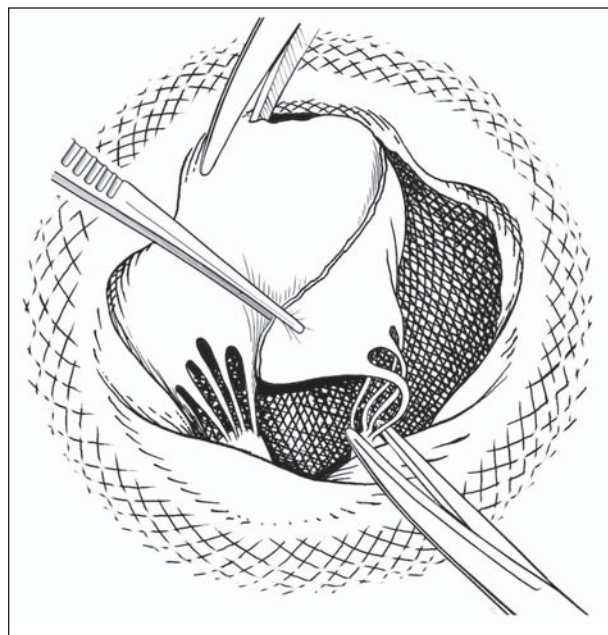


Figure 1—Illustration of excision of the mitral septal leaflet and associated chordae tendineae through a left atrial approach (dorsal view) to the mitral valve in a dog. A 2- to 3-mm apron of leaflet is left intact at the annulus. Chordae tendineae are divided at the papillary muscle. The mitral mural leaflet and chordae tendineae are not excised.

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reinforced with pledgets<sup>6</sup> were preplaced through the mitral annulus from the atrial to the ventricular side of the annulus and then through the prosthetic sewing ring from the ventricular (outflow) to the atrial (inflow) side of the prosthesis (Figure 2). Over the mural portion of the valve, mattress sutures incorporated several bites of the mural leaflet to reef or plicate the leaflet as sutures were tightened.<sup>15</sup> The valve was seated into the annulus with the valve leaflets orientated at right angles to the normal anatomic orientation of the native leaflets. The sutures were tied (Figure 3). The atriotomy was closed with 4-0 suture in a continuous mattress pattern oversewn with a simple continuous pattern. Air was removed from the left side of the heart with a vent cannula that was temporarily passed through the atriotomy and valve prosthesis. The aortic cross clamp was removed. Internal electrical defibrillation (20 to 50 J) was used if needed to restore sinus rhythm. Cardiopulmonary bypass was discontinued. A thoracostomy tube was placed. The thoracotomy was closed in routine fashion. A high-volume, low-pressure, cuffed tracheostomy tube<sup>d</sup> was introduced via a temporary tracheostomy.

Ventilatory support consisting of spontaneous ventilation with inspiratory pressure support (2 to 8 cm H<sub>2</sub>O), supplemental oxygen (F<sub>IO</sub><sub>2</sub>, 40% to 60%), and positive end-expiratory pressure (5 to 10 cm H<sub>2</sub>O) was administered until the morning after surgery. Levels of ventilatory support were determined by monitoring arterial blood gas analysis. Dobutamine (2

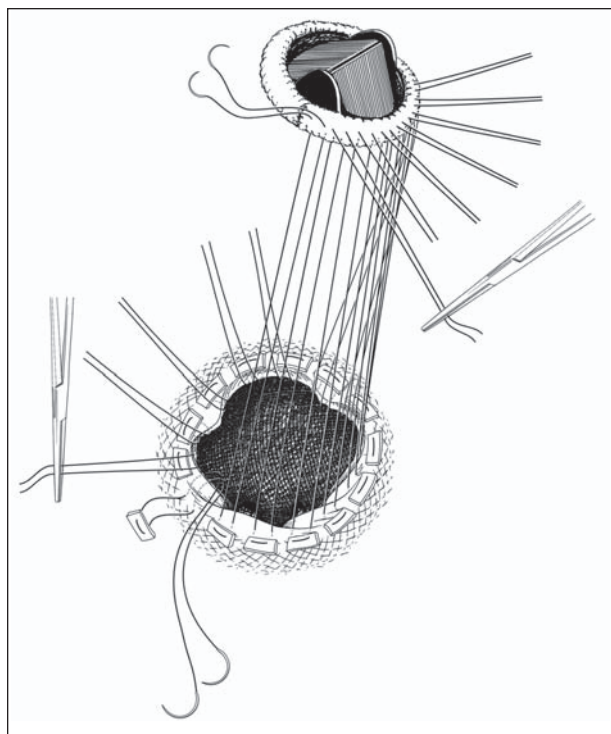


Figure 2—Illustration of the placement of pledget-reinforced mattress sutures into the mitral annulus of a dog. Sutures are passed through the annulus from the atrial to the ventricular side of the valve. Over the mural portion of the valve, mattress sutures incorporate 2 to 3 bites of the mural leaflet to reef or plicate the leaflet when the sutures are tightened. Mattress sutures are placed through the sewing ring of the valve prosthesis from the ventricular (outflow) to the atrial (inflow) side and tagged with a hemostat.

to 10 µg/kg/min [0.9 to 4.5 µg/lb/min], constant rate infusion) was administered, if necessary, to maintain adequate arterial blood pressure and cardiac output. Adequacy of cardiac output was determined by evaluation of heart rate, arterial pulse pressure, blood lactate concentration, venous O<sub>2</sub> saturation, and calculation of O<sub>2</sub> extraction. Lidocaine (70 µg/kg/min [32 µg/lb/min], constant rate infusion) was administered to suppress ventricular tachycardia for 24 to 48 hours after surgery. Heparin (100 U/kg [45 U/lb], SC, q 8 h) was administered after postoperative bleeding had ceased and the thoracostomy tube had been removed (generally 24 to 36 hours after surgery). Warfarin administration (0.05 to 0.2 mg/kg [0.02 to 0.09 mg/lb], PO, q 24 h) was started 1 day after heparin administration and continued for the life of the dog. Heparin administration was discontinued 2 to 3 days after starting warfarin administration. The total weekly dose of warfarin was periodically adjusted on the basis of results of measurement of prothrombin time and calculation of the international normalized ratio (INR). The goal of warfarin administration was to maintain the INR from 2.5 to 3.5.<sup>16</sup>

## Results

Eight dogs underwent mitral valve replacement between July 1998 and November 1999. The cause of mitral regurgitation was DMD in 7 dogs and DCM in 1 dog. Median age at the time of surgery was 10 years (range, 8 to 12 years). Median weight of dogs was 10.1 kg (22.2 lb). Range of weights was 4.3 to 32.2 kg (9.5 to 70.8 lb), respectively. Prior to surgery, all dogs were receiving furosemide (2.9 to 10.2 mg/kg/d [1.3 to 4.6 mg/lb/d], PO, divided q 8 to 12 h) and enalapril (0.5 mg/kg [0.23 mg/lb], PO, q 12 h). Other cardiac drugs administered prior to surgery included digoxin (n = 3 dogs); spironolactone (3); hydralazine (2); and amlodapine, hydrochlorothiazide, and sotalol (1 each). Median

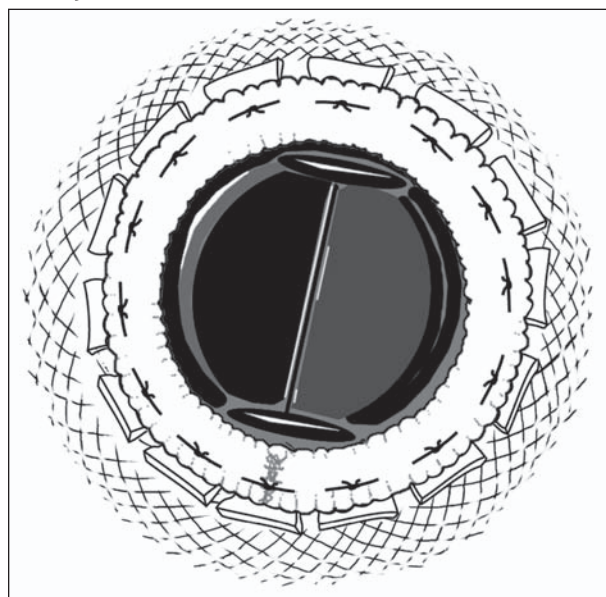


Figure 3—Illustration of a mitral valve prosthesis after placement in a dog. The prosthesis is seated into the mitral annulus, and mattress sutures are tied tightly to secure the prosthesis and prevent leakage around the prosthesis.

duration of CHF prior to surgery was 7 months (range, 5 to 12 months). Prior to surgery, mean  $\pm$  SD value for LVDVI was  $205.5 \pm 90.5$  mL/m<sup>2</sup>, mean LVSVI was  $39.7 \pm 31.9$  mL/m<sup>2</sup>, mean LA:Ao ratio was  $2.66 \pm 0.4$ , and mean fractional shortening was  $46.3 \pm 13.3\%$ . Seven dogs had concurrent trivial to mild tricuspid regurgitation.

All dogs underwent mitral valve replacement with a bileaflet, tilting-disk, mechanical prosthesis<sup>d</sup>; size ranged from 19 to 26 mm. One dog died during surgery after several failed attempts to wean from cardiopulmonary bypass. Obstruction of left ventricular outflow by an oversized valve prosthesis (19-mm valve in a 4.5-kg [9.9-lb] dog) was the suspected cause of an inability to wean from cardiopulmonary bypass. Seven dogs survived surgery and were discharged from the hospital. Surviving dogs with DMD had resolution of CHF and did not require furosemide administration after surgery. The dog with DCM still required diuretic administration to control CHF after surgery. Echocardiography performed 5 to 7 days after surgery revealed a significant decrease in LVDVI but no difference in LVSVI, compared with indices obtained before surgery (Figure 4). Mean LA:Ao ratio decreased ( $P = 0.046$ ) to  $1.73 \pm 0.65$  after surgery. Mean fractional shortening decreased ( $P = 0.018$ ) to  $26.7 \pm 9.2\%$  after surgery.

Median survival after surgery was 4.5 months (range, 0.75 months to 5.25 years). Six dogs died from acute severe medically refractory pulmonary edema. Three of these dogs had thrombosis of the valve prosthesis as confirmed by histologic examination. In each dog, thrombosis involved the prosthetic hinge mechanism and caused loss of motion of 1 or both prosthetic valve leaflets. Valve prosthesis thrombosis was considered the suspected cause of fatal pulmonary edema in the other 3 dogs on the basis of acute onset and medically refractory course ( $n = 3$  dogs), disruption of warfarin administration in the 72-hour period prior to onset of pulmonary edema (3), and results of gross

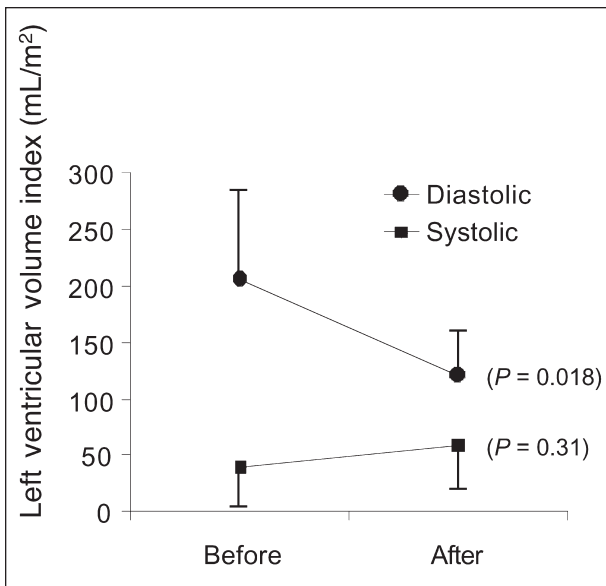


Figure 4—Left ventricular diastolic and systolic volume indices (mean  $\pm$  SD) before and after mitral valve replacement in 7 dogs. *P* values indicate comparisons of values obtained before versus after placement.

postmortem examination (1). One dog with DCM died during attempted electrical cardioversion of atrial fibrillation. On postmortem examination, this dog had metastatic pulmonary neoplasia.

## Discussion

Mitral valve replacement resolved CHF in 6 of 6 surviving dogs with DMD despite the presence of severe left atrial and ventricular dilation, systolic dysfunction, and relatively long-standing CHF. The only dog with DCM in this series survived surgery and had palliation of CHF despite severe systolic dysfunction (LVSVI, 101 mL/m<sup>2</sup>) at the time of surgery. The only surgery-related death was likely the result of the smallest available valve prosthesis (19 mm) being too large for the smallest dog (4.3 kg) in this series. The dramatic decrease in left atrial and ventricular dimensions within days of surgery reflected the immediate hemodynamic benefit that dogs in this study gained from complete correction of severe MR. These results suggest that dogs with severe MR tolerate mitral valve replacement surprisingly well, even relatively late in the course of CHF, and defy predictions that dogs need surgery early in the course of their disease to survive the surgery.<sup>17</sup> Although the number of dogs in each series was small, the immediate outcome (ie, surgery survival and acute resolution of CHF) was better in dogs that underwent mitral valve replacement than in dogs that underwent mitral valve repair.<sup>14</sup> Perfect correction of MR, which was seldom achieved in dogs that underwent valve repair, was the most likely reason for a more favorable immediate outcome in dogs that underwent valve replacement.

Fractional shortening decreased significantly after mitral valve replacement. However, this was primarily the result of decreased left ventricular diastolic dimension rather than increased systolic dimension; thus, worsened systolic function was not the cause of decreased fractional shortening after surgery. Fractional shortening decreased because of a profound decrease in preload caused by sudden correction of MR-induced volume overload. Mitral valve repair preserves systolic function better than mitral valve replacement in human patients.<sup>18</sup> Such a benefit was not apparent in dogs because systolic function after surgery was similar in dogs in this study, compared with dogs that underwent mitral valve repair.<sup>14</sup> One of the reasons cited for improved systolic function after mitral valve repair is that chordal-papillary muscle continuity is maintained during mitral repair, and this in turn retains diastolic and systolic support to the left ventricular wall.<sup>15,18,19</sup> For many years, the standard technique for mitral valve replacement included excision of both valve leaflets; consequently support of the left ventricular wall by the mitral valve apparatus was lost. It is now standard practice to preserve either 1 or both mitral leaflets to maintain chordal-papillary muscle continuity and preserve ventricular function after surgery.<sup>15,19</sup> In the dogs in our study, the mural leaflet was retained and reefed into the mattress sutures securing the valve prosthesis. This was done to maintain chordal-papillary muscle continuity and may account, in part, for relatively well-preserved systolic



function after surgery. In humans, it is recommended, on the basis of end-systolic diameter or volume, that patients with chronic primary MR receive surgery before deterioration of ventricular function becomes severe.<sup>18</sup> Otherwise, progressive left ventricular dysfunction and death from CHF may occur despite surgery.<sup>18,20</sup> One dog in our study had severe left ventricular systolic dysfunction (ie, LVSVI > 90 mL/m<sup>2</sup>) before surgery and did in fact have progressive heart failure after surgery. On the basis of recommendations in humans<sup>18</sup> and our previous experience with mitral valve repair,<sup>14</sup> dogs with MR should ideally have surgery before LVSVI becomes > 70 mL/m<sup>2</sup>.

Despite a favorable immediate outcome, long-term outcome after mitral valve replacement with a mechanical prosthesis was disappointing. Although 1 dog lived longer than 5 years, most dogs in this series survived < 1 year after surgery. The cause of death was thrombosis of the valve prosthesis, either confirmed or suspected, in all but 1 dog that survived surgery. Clinical experience in humans has clearly established the need for lifetime anticoagulation in patients who undergo heart valve replacement with a mechanical prosthesis.<sup>21</sup> The current recommendation in humans is that all patients with a mechanical prosthetic valve in the mitral position receive orally administered warfarin to maintain the prothrombin time-based INR from 2.5 to 3.5.<sup>21</sup> By use of this standard, the incidence of thrombosis in humans with a mechanical valve in the mitral position is approximately 1 to 3 incidents/100 patient years, depending on the study.<sup>18,21</sup> In the absence of an established recommendation for anticoagulation in dogs, the guideline for humans was chosen for dogs in this study. Unfortunately, valve thrombosis occurred despite the best efforts of attending veterinarians to maintain anticoagulation with warfarin. Whether this failure was caused by comparative inexperience with warfarin treatment in dogs, a greater propensity of dogs to undergo prosthetic valve thrombosis, or both is a matter of speculation. Results of interviews with attending veterinarians suggested that most dogs in this study had either inadequate anticoagulation or a disruption in warfarin administration within a 72-hour period preceding valve thrombosis. A common reason for the disruption in warfarin administration was concern that the dog might be either hemorrhaging internally or at risk for eminent hemorrhage. In retrospect, it is perhaps worth noting that none of the dogs in this study had major hemorrhage while receiving warfarin. It is unclear whether increased experience and a greater appreciation of the importance of warfarin administration could improve the late-term outcome in dogs that undergo mitral valve replacement with a mechanical prosthesis in the future. Given the favorable immediate outcome of dogs in this study and the certainty of an unfavorable outcome without surgery, mitral valve replacement is likely worth further exploration as a treatment option for dogs with severe MR. Improving late-term outcome will depend on finding strategies to prevent prosthetic valve thrombosis or to explore valve

prostheses that do not require lifetime anticoagulation therapy (eg, glutaraldehyde-fixed tissue valves).

- a. StatView for Windows, version 5.0.1, SAS Institute Inc, Cary, NC.
- b. St Jude Medical Prosthetic Heart Valve, Saint Paul, Minn.
- c. Ti-Cron with 3 × 3-mm TFE polymer pledgets, Sherwood, Davis & Geck, St Louis, Mo.
- d. Shiley low-pressure, cuffed tracheostomy tube, Nellcor, Pleasanton, Calif.

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