Degenerative mitral valve disease (DMD) is a common condition of aging dogs. The incidence of DMD is reported to be as high as 58% in dogs 9 years or older. Congenital mitral valve dysplasia (CMD) is a less frequently occurring condition that is associated with young large-breed dogs. Valve prolapse as a less frequently occurring condition that is associated with young large-breed dogs.

Evaluation of techniques and outcomes of mitral valve repair in dogs

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Objective—To describe surgical techniques for and assess outcome of treatment of mitral regurgitation in dogs.

Design—Uncontrolled prospective study.

Animals—18 dogs with naturally occurring mitral regurgitation.

Procedure—All dogs weighed > 5 kg (11 lb) and had severe mitral regurgitation, congestive heart failure (CHF), and no serious noncardiac disease. Left ventricular volume indices, left atrial size, and degree of mitral regurgitation were determined echocardiographically before and after repair. Surgical techniques included circumferential annuloplasty, placement of artificial chordae, chordal fenestration and papillary muscle splitting, and edge-to-edge repair. Factors predictive for surgery survival and resolution of CHF were determined.

Results—12 dogs survived surgery. Factors predictive for surgery survival included weight > 10 kg (22 lb) and CHF of less than 6 months' duration. In 9 dogs, CHF resolved for a median period of 1 year (range, 4 months to 3 years) after surgery. One dog had stable CHF at 12 months. One dog died as a result of progressive CHF; another was euthanatized for noncardiac disease. Valve prolapse as a less frequently occurring condition that is associated with young large-breed dogs.

Conclusion and Clinical Relevance—Mitral valve repair may resolve CHF in dogs with severe mitral regurgitation, particularly in dogs that weigh > 10 kg and are treated within 6 months of the onset of CHF. (J Am Vet Med Assoc 2004;224:1941–1945)
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 or crystalloid-amino acid solution.

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 (Fig 1). Mitral valve leaflets and the atrial wall were inspected for jet lesions that might have indicated regions of maximal mitral regurgitation. The assessment of reflux of cold physiologic saline (0.9% NaCl) solution that was vigorously infused into the left ventricle was also used to demonstrate areas of mitral regurgitation. Valve leaflets and chordal apparatus were inspected by use of a nerve hook. In most dogs, leaflet prolapse was repaired with artificial chordae fashioned from CV-5 polytetrafluoroethylene (PTFE) suture in a horizontal mattress pattern (Fig 2). An edge-to-edge repair was also used to correct leaflet prolapse in some dogs (Fig 3).

Restrictive leaflet motion in dogs with CMD was corrected by fenestration between fused chordae tendineae and longitudinal splitting of the papillary muscle between chordal attachments in an attempt to functionally lengthen the chordal apparatus. Circumferential annuloplasty to reduce the size of the mitral orifice was performed in all dogs. In most dogs, annuloplasty was accomplished by use of a partial ring fashioned from PTFE patch material and placed in the mural portion of the annulus between points of attachment of the septal leaflet to the cardiac trigone (Fig 4). The length of the partial ring was estimated from the circumference of valve orifice sizes matched to the area of the septal leaflet. In 2 dogs, annulop-
Results

Eighteen dogs underwent mitral valve repair between March 1999 and May 2003. The cause of mitral regurgitation was CMD in 4 dogs (median age, 16 months; range, 6 to 24 months) and DMD in 14 dogs (median age, 11 years; range, 6 to 13 years). Median weight of the dogs was 13.4 kg (29.5 lb; range, 6.3 to 40 kg [13.9 to 88 lb]). Median duration of CHF prior to inclusion in the study was 6 months (range, 2 to 16 months). Among the study dogs, the daily dosage of furosemide before surgery was 2 to 15 mg/kg/d (0.9 to 6.8 mg/lb/d). Prior to surgery, all dogs were also receiving either enalapril (0.5 mg/kg [0.23 mg/lb], PO, q 12 h) or benazepril (0.5 mg/kg, PO, q 24 h). Other cardiac drugs being administered at time of surgery included digoxin (7 dogs), spironolactone (4), amlopidine (3), hydrochlorothiazide (2), and mexiletine (1).

The primary mechanism of mitral regurgitation in dogs with CMD was restrictive motion of 1 or both valve leaflets (fused and short chordae) in 3 dogs and prolapse of the septal valve leaflet in 1 dog (absence of chordae). All dogs with DMD had prolapse of the septal leaflet with or without prolapse of the mural leaflet caused by chordal stretching or rupture, or both. Secondary dilation of the mitral valve annulus was considered a contributing cause of mitral regurgitation in all dogs. In 13 dogs (11 with DMD and 2 with CMD), tricuspid regurgitation was detected (from trivial to moderate severity). Prior to surgery, mean LVDVI value ± SD was 224.3 ± 99.2 cm3/m2 (range, 65.5 to 504.4 cm3/m2) and mean LVSVI was 56.6 ± 36.5 cm3/m2 (range, 13.9 to 129.2 cm3/m2). At that time, mean LA:Ao ratio was 2.33 ± 0.42.

A partial PTFE ring annuloplasty was performed in 16 dogs. The length of the ring was 33 to 70 mm corresponding to an estimated ideal valve diameter of 16 to 30 mm. Continuous PTFE suture annuloplasty was performed in 2 dogs with DMD. All 14 dogs with DMD had PTFE artificial chordae repair. A single PTFE artificial chorda was placed between the septal leaflet and the cranialateral papillary muscle in 2 dogs or between the septal leaflet and the caudal medial papillary muscle in 4 dogs. Eight dogs had 2 PTFE artificial chordae placed in the septal leaflet, 1 to each papillary muscle. Two dogs with DMD had an additional edge-to-edge repair. In 1 dog, the repair was placed centrally between the septal and mural leaflets, and in the other dog, the repair was placed between the caudal medial portions of the leaflets. In the dog with CMD that had congenital absence of chordae, an edge-to-edge repair between the caudal medial portions of the leaflets was performed without artificial chordae repair. The 3 other dogs with CMD underwent chordal fenestration and splitting of papillary muscles to increase leaflet motion and coaptation.

Twelve dogs (4 of 4 dogs with CMD and 8 of 14 dogs with DMD) survived surgery and were discharged from the hospital. Causes of surgery-related death in 6 dogs were a tear in the atrioventricular groove (n = 1), disseminated systemic thrombosis (1), and low-output heart failure (4). Body weight > 10 kg was predictive for survival of surgery (P = 0.021). Four of 6 dogs that did not survive surgery weighed < 10 kg. Less than 6 months’ duration of CHF (P = 0.019) and < 120 minutes’ duration of cardiac arrest (P = 0.027) were pre-
predictive for avoiding low-output left ventricular failure as cause of surgery-related death.

Nine dogs (2 of 4 dogs with CMD and 7 of 8 dogs with DMD) had resolution of CHF with a median follow-up period of 1 year (range, 4 months to 3 years) at the time of this report. Values of LVDVI < 250 cm³/m² and LVSVI < 70 cm³/m² before surgery were both predictive for resolution of CHF ($P = 0.018$ and $0.046$, respectively). At 12 months after surgery, 1 dog with DMD still required treatment with furosemide but had had no apparent progression of CHF. One dog with CMD with severe left ventricular dilatation (LVDVI value, 504.4 cm³/m²) developed atrial fibrillation and died of complications related to progressive heart failure at 6 months after surgery. This dog also had dehiscence of the mid-portion (approx 50%) of its ring annuloplasty that was surgically repaired with butressed mattress sutures 4 months after the original surgery. One dog with DMD was euthanatized 1 month after surgery for a reason unrelated to cardiac disease.

After surgery, 2 dogs with DMD had trivial or no mitral regurgitation, 5 dogs (2 with CMD and 3 with DMD) had mild mitral regurgitation, and 5 dogs (2 with CMD and 3 with DMD) had moderate mitral regurgitation. Left ventricular diastolic volume index decreased significantly ($P = 0.003$) from 226.9 ± 117.7 cm³/m² before surgery to 134.9 ± 70.4 cm³/m² at 6 months after surgery (n = 10). Left atrium-to-aorta ratio decreased significantly ($P = 0.007$) from 2.3 ± 0.51 before surgery to 1.75 ± 0.46 at 6 months after surgery (n = 10). Left ventricular systolic volume index was not significantly ($P = 0.094$) different before and 6 months after surgery (467 ± 39.5 cm³/m² and 343 ± 38.3 cm³/m², respectively; n = 10). Two dogs had died or been euthanatized prior to the evaluation at 6 months after surgery.

Discussion

Mitral valve repair was associated with resolution of CHF in 9 of 12 dogs that survived surgery and 9 of 18 dogs that were treated, as judged by the discontinuation of administration of the diuretic agent without development of pulmonary edema and decreases in left ventricular and left atrial dilatation detected via objective assessment. Although this was not a controlled clinical trial in that it did not have nonsurgically treated cohort, it is widely accepted that dogs with severe mitral regurgitation and diuretic-dependent CHF have an invariably progressive course of disease with most of those dogs surviving < 1 year after onset of CHF.24 Dogs treated in the study of this report are known to have remained free of diuretic-dependent CHF for periods of up to 3 years after mitral valve repair. Therefore, on the basis of historical experience, it appears that successful mitral valve repair dramatically altered the clinical course of severe mitral regurgitation.

Improving the success rate of mitral valve repair in dogs would require decreasing the proportion of surgery-related deaths (6 of 18 dogs in the study of this report) and increasing the success rate of surgical repairs in surviving dogs (9 of 12 dogs in the study of this report). Because of the complexity of the mitral valve repair surgery and the limited previous experience with open-heart surgery in veterinary medicine, it is reasonable to expect that both the aforementioned factors might change with increased experience with the surgical procedure and thereby improve its success rate. Additionally, the establishment of realistic case selection criteria will likely play an important role in improving the overall success rate of the procedure in the future. In the study of this report, small dogs (weight < 10 kg) had a much higher risk of surgery-related death. Furthermore, long-standing CHF (duration > 6 months) or a long period of cardiac arrest during surgery were predictive of fatal low-output heart failure, the most important cause of surgery-related death in the study of this report. Failure to resolve CHF after surgery was predicted by both severe left ventricular dilatation (LVDVI > 250 cm³/m²) or severe left ventricular systolic dysfunction (LVSVI > 70 cm³/m²). Although strict recommendations for selection criteria for mitral valve repair in dogs will have to await a broader experience, our findings have suggested that mitral valve repair is more likely to be successful if dogs undergo the procedure early in the course of CHF and before left ventricular dilatation or systolic dysfunction become severe. Such a conclusion would be consistent with current recommendations for mitral valve repair in humans.25-27 However, on the basis of experience to date, it is important to note that small dogs are at increased risk for an unfavorable outcome, even though they represent a large percentage of dogs with DMD.

It is widely accepted that dilatation of the mitral valve annulus associated with progressive left heart dilatation contributes to progression of mitral regurgitation regardless of inciting cause.22 The hemodynamic benefit of external circumferential annuloplasty has been demonstrated in dogs with experimental mitral regurgitation.28 In 2 case reports,10,11 of mitral valve repair in dogs, favorable results were apparently achieved without circumferential annuloplasty. In the authors’ opinion, circumferential annuloplasty plays an important role in definitive surgical correction of mitral regurgitation. Furthermore, in the event that repair of the primary leaflet abnormality cannot be accomplished, annuloplasty likely decreases the magnitude of mitral regurgitation and thereby could be expected to have at least a palliative effect. Indeed, some dogs in the study of this report had resolution of CHF despite retaining moderate mitral regurgitation after surgery. Estimation of the orifice size of the circumferential annuloplasty was based on the circumference of valve sizers matched to the approximate area of the sepal leaflet. Early in our experience with surgical repair techniques in the study of this report, we did not allow for bunching or puckering of the ring as it was sutured in position. This tended to cause overcorrection of annulus size and higher than ideal transvalvular pressure gradients in some dogs after surgery. We increased estimated ring lengths by approximately 20% to 25% to overcome this effect. Circumferential annuloplasty was not extended across the intertrigonal area of the annulus at the base of septal leaflet to avoid possible injury to the aortic valve. In humans, it is known that this portion of the annulus does not dilate, and experience has indicated that it is not necessary to extend the annuloplasty across the base of the septal leaflet.22,23,24,25,26,27,28,29

In mitral valve repair in dogs with leaflet prolapse, we focused on repairing the sepul leaflet because of its prominent size and area. When possible, our preferred
method of repair was placement of artificial chordae. Results of an experimental study in dogs indicated that systolic function decreases when chordae tendineae are transected. In addition, replacement of ruptured chordae in humans reduces stress on the remaining chordae during valve closure and thus decreases the risk of further chordal rupture. Artificial chordae sutures were placed prior to but were not tied until after annuloplasty was performed. Appropriate artificial chordal length was judged with the intention of preventing prolapse of the free edge of the leaflet beyond the annulus. This was easier to judge after annuloplasty had restored a more normal annulus size. The edge-to-edge procedure has been reported as the sole method of mitral valve repair in humans. In our study, 2 dogs had edge-to-edge repair performed as an adjunctive procedure to artificial chordal repair and 1 dog underwent edge-to-edge repair as a sole method for correction of leaflet prolapse. Each of these dogs had good to excellent repairs without evidence of stenosis (based on mitral flow half-times; data not reported).

Although congenital versus acquired degenerative disease was not predictive of outcome in our study, it was difficult to surgically correct restrictive leaflet motion. Fenestration of the chordae and splitting of the papillary muscle increased the range of leaflet motion, but normal leaflet motion was not achieved. All 3 dogs with restrictive leaflet motion retained moderate mitral regurgitation after surgery. Dogs with CMD also appeared to have more severe left ventricular dilatation and systolic dysfunction at the time of surgery. On the basis of these results, a palliative rather than curative effect may be a more reasonable expectation for mitral valve repair in dogs with congenital dysplasia. Nevertheless, in the study of this report, successful mitral valve repair did result in resolution of CHF and decreased left heart dilatation and likely altered the clinical course of dogs with severe mitral regurgitation. On the basis of our data, dogs with DMD that weigh > 10 kg in which mitral valve repair can be performed within 6 months of onset of CHF are the best candidates for successful surgical intervention.

References