Evaluation of survival rate and prognostic indicators for surgical treatment of left-to-right patent ductus arteriosus in dogs: 52 cases (1995–2003)

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Objective—To determine factors associated with long-term survival in dogs treated surgically for patent ductus arteriosus (PDA).

Design—Retrospective case series.

Animals—52 dogs treated surgically for left-to-right shunting PDA.

Procedure—Data pertaining to age, breed, sex, body weight, clinical examination findings, type and duration of medical treatment, results of thoracic radiography and echocardiography, and surgical and postoperative complications were collected from records. Follow-up information was obtained from medical records or telephone interviews with owners or referring veterinarians.

Results—22 dogs had mitral valve regurgitation. Mean weight and age were not significantly different between dogs with or without mitral valve regurgitation. Twenty-four (46.2%) dogs had clinical signs related to cardiac insufficiency. Left atrial dilatation was observed in 56.3% of dogs that were radiographed. Sonographic imaging was used to diagnose left atrial dilatation in 23 dogs and left ventricular dilatation in 25 dogs. The 1- and 2-year survival rates were 92% and 87%, respectively. Diagnosis of mitral valve regurgitation before surgery was not associated with the probability of survival. Age, weight, lethargy, preoperative treatment with angiotensin-converting enzyme inhibitors, and right atrial dilatation on radiographs at the time of surgery were negatively associated with probability of survival.

Conclusions and Clinical Relevance—Surgical treatment of PDA was curative in young dogs without clinical signs of heart failure. Surgical correction of PDA should be recommended as early as possible after diagnosis, and mitral valve regurgitation is not a contraindication for surgery. (*J Am Vet Med Assoc* 2005;227:1794–1799)

Patent ductus arteriosus (PDA) is one of the most common congenital heart defects in dogs.[1-4] Patent ductus arteriosus with left-to-right shunting of blood flow induces overcirculation in the lungs and left ventricular and atrial volume overload, resulting in eccentric hypertrophy of the left side of the heart.[5,6] Transformation of left ventricular shape by increased chamber sphericity is the most likely cause of the development of functional mitral valve regurgitation.[7] When the mitral valve becomes incompetent, the left atrium dilates and the likelihood of atrial fibrillation increases.[3,5] Congestive heart failure usually develops between 1 week and 1 year of age.[2,5,6] Closure of the DA is considered curative for affected patients.[8] Closure may be accomplished via surgical occlusion, use of hemostatic clips, or transcatheter embolization.[2,4,6,8-11] If the DA is successfully occluded, it has been posited that heart size may return to normal dimensions and the mitral valve may again become competent.[6] Surgical occlusion yields the best results, in terms of survival rates and minimizing postoperative complications, when the occlusion procedure is performed as soon as possible after diagnosis.[5]

Evaluation of the outcomes in dogs with PDA in previous studies[2,6,8,12-15] reveals that treatment via surgical occlusion is considered curative. However, to the authors' knowledge, studies evaluating cardiac-related death and prognostic indicators in dogs undergoing occlusion surgery for PDA have not been published. The objective of our study was to determine long-term survival rates and factors associated with long-term survival for dogs undergoing surgical ligation of PDA.

Criteria for Selection of Cases

Medical records of all dogs examined because of PDA at the Colorado State University Veterinary Teaching Hospital between 1995 and 2003 were reviewed. Dogs were included in the study if they had a left-to-right shunting PDA and were treated surgically. Dogs treated by means of coil embolization were excluded from the study.

Procedures

Age, breed, sex, body weight, results of clinical examination, type and duration of medical treatment, and surgical and postoperative complications were recorded. Information pertaining to exercise intolerance, lethargy, dyspnea, weight loss, and retarded growth was also included.

Results of radiographic and echocardiographic imaging of the thorax were recorded. Mitral valve regurgitation was diagnosed if there was auscultation of a left apical holosystolic murmur or observation of valvular regurgitation on echocardiography. Perioperative surgical complications were recorded.

Follow-up information was obtained from medical records or via telephone interviews with owners and referring veterinarians. Survival time was defined as the number of days between surgery and the date of death or the last follow-up call. The cause of death was recorded as related or unrelated to PDA. If no information could be collected after the dog left the hospital, it was considered lost to follow-up.

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Statistical analysis—A computer software package[i] was used to perform the statistical analyses. Analysis of variance was used to compare age and weight at the time of initial examination between dogs with and without mitral regurgitation. The χ^2 test was used to compare the percentage of females in the study dogs with that in the general hospital population. Kaplan-Meier actuarial survival analyses were performed to evaluate the long-term survival rate of surgically treated dogs with PDA. A log-rank test was used to compare survival curves of dogs with and without mitral insufficiency. Twenty-seven variables related to signalment, history, mitral regurgitation, results of radiography and echocardiography performed at the time of initial examination, and pre- and postoperative treatments were evaluated by means of Cox proportional hazard analyses for their usefulness as indicators of the probability of survival for dogs with PDA. A **hazard ratio (HR)** and 95% confidence intervals were calculated for each variable. Values of $P < 0.05$ were considered significant. Dogs that were alive at the end of the study or lost to follow-up or that died from a cause unrelated to the PDA or cardiac disease were censored for the Kaplan-Meier actuarial survival analysis and the Cox analysis. Results are given as mean ± SD.

Results

Fifty-two dogs underwent surgery for treatment of PDA. Breeds represented were the Shetland Sheepdog (7 dogs), Toy Poodle (5), Labrador Retriever (5), mixed breed (5), German Shepherd Dog (4), Pomeranian (4), Dachshund (3), Golden Retriever (3), Maltese (3), Cocker Spaniel (2), American Eskimo Dog (1), Bulldog (1), Tibetan Terrier (1), Newfoundland (1), Australian Shepherd (1), Miniature Schnauzer (1), Boston Terrier (1), Chihuahua (1), Great Pyrenees (1), Bernese Mountain Dog (1), and Irish Setter (1). Thirty-seven dogs were female and 15 were males. Mean weight was 10.0 ± 10.1 kg (22 ± 22.2 lb). Mean age at the time of diagnosis was 13.2 ± 24.7 months (range, 1.4 to 109.0 months). There was a significant ($P < 0.001$; adjusted $r^2 = 0.29$) correlation between age and weight.

Twenty-two dogs (14 [63.6%] females and 8 [36.4%] males) had mitral valve regurgitation. Female dogs were overrepresented, compared with the general hospital population (51% female; $P < 0.05$). Mean weight of dogs with mitral valve regurgitation was 7.7 ± 7.9 kg, and mean age at the time of diagnosis was 9.73 ± 20.9 months (range, 1.4 to 97 months). For dogs without mitral valve regurgitation, mean weight was 11.50 ± 11.30 kg (25.3 ± 24.9 lb) and mean age at the time of diagnosis was 16.2 ± 27.8 months (range, 1.5 to 109 months). Mean weight ($P = 0.181$) and age ($P = 0.365$) at the time of diagnosis were not significantly different between dogs with or without mitral valve regurgitation.

Twenty-four (46.2%) dogs had clinical signs related to cardiac insufficiency at the time of surgery. Fifteen (28.8%) dogs were treated with diuretics (n = 14) or **angiotensin-converting enzyme (ACE)** inhibitors (8) at the time of surgery. Of the 22 dogs with mitral valve regurgitation, 8 were treated with a diuretic and 4 were treated with an ACE inhibitor. Seven dogs in which the PDA was causing clinical signs (eg, exercise intolerance, lethargy, or weight loss) received a diuretic and an ACE inhibitor. The left ventricular end-diastolic diameter index was not significantly different between dogs that received an ACE inhibitor (105.9 ± 83.3 mm/m^2) and those that did not (108.4 ± 45.8 mm/m^2; $P = 0.924$). The left ventricular end-systolic diameter index was not significantly different between dogs that received an ACE inhibitor (73.4 ± 50.5 mm/m^2) and those that did not (65.9 ± 28.0 mm/m^2; $P = 0.646$). The ratio of the diameter of the left atrium to the diameter of the aorta was not significantly different between dogs that received an ACE inhibitor (1.8 ± 0.5) and those that did not (1.6 ± 0.2; $P = 0.11$). Shortening fraction was significantly ($P = 0.012$) different between dogs that received an ACE inhibitor (29.9 ± 14.5%) and those that did not (39.3 ± 5.0%).

Two dogs had atrial fibrillation as indicated by an ECG at the time of initial examination, whereas sinus rhythm was detected in the other dogs. The 2 dogs with atrial fibrillation had clinical signs of exercise intolerance, had left atrial dilatation and pulmonary edema on radiographs, and were receiving diuretics and ACE inhibitors. One of the dogs with atrial fibrillation underwent echocardiographic examination. In that dog, shortening fraction was 6.8% and the ratio of the

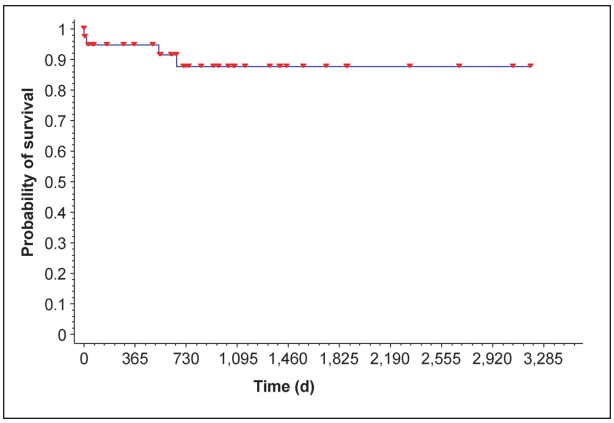

Figure 1—Kaplan-Meier survival curve for 52 dogs treated surgically for patent ductus arteriosis.

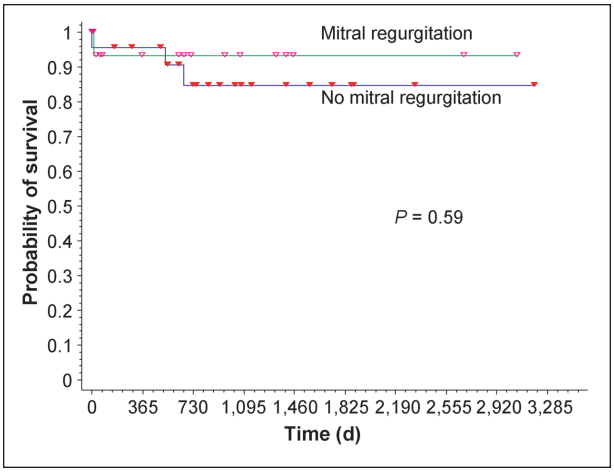

Figure 2—Kaplan-Meier survival curves for 52 dogs with and without mitral valve regurgitation at the time of surgical closure of patent ductus arteriosis.

diameter of the left atrium to the diameter of the aorta was 2.21. Those dogs were in atrial fibrillation after surgery and died 8 and 17 days after surgery.

Radiographs were obtained in 48 dogs. Radiographic abnormalities were detected in 40 (83.3%) of those dogs, with cardiomegaly diagnosed in 32 (66.7%) dogs. Left atrial dilatation was reported in 27 (56.3%) dogs. Right atrial and right ventricular enlargement was reported in 10 and 18 dogs, respectively.

Echocardiography was performed before surgery in 32 dogs. Left atrial dilatation was detected in 23 dogs and left ventricular dilatation in 25 dogs. Dilatation of the right side of the heart was not observed on echocardiography. Mitral regurgitation was observed in 11 dogs.

Eight dogs had acute intraoperative bleeding during dissection of the PDA. All dogs survived the surgical ligation procedure and were discharged from the hospital. Dogs that had bleeding were significantly ($P = 0.018$) older than dogs that did not (32.6 ± 37.2 months and 10.0 ± 21.0 months, respectively).

Postoperatively, 2 dogs (1 with mitral valve regurgitation) had pulmonary edema that required medical attention and 2 dogs (1 with mitral valve regurgitation) had ventricular tachycardia. Two dogs (1 with mitral valve regurgitation) had a continuous murmur after surgery. A left apical holosystolic murmur was auscultated during the immediate postoperative period in 9 of 22 (40.9%) dogs that had mitral valve regurgitation before surgery. No murmur was reported after surgery in dogs that did not have mitral valve regurgitation prior to surgery ($P = 0.001$).

Follow-up information was obtained for 38 dogs (mean follow-up time, 959 ± 790 days; range, 4 to 3,067 days). Fourteen dogs were lost to follow-up after leaving the hospital (1.9 ± 0.7 days). In the long term, 4 dogs were lost to follow-up at 17, 29, 71, and 162 days. Twenty-eight dogs were alive at the time of writing (mean follow-up time, 1,156 days; range, 284 to 3,067 days). Two dogs died for reasons unrelated to cardiac disease at 1,733 and 2,458 days, respectively, after surgery. Four dogs died for reasons related to cardiac disease at 4, 8, 17, and 116 days, respectively, after surgery. The 1- and 2-year survival rates for dogs treated surgically for PDA were 92% and 87%, respectively (**Figure 1**). Diagnosis of mitral valve regurgitation before surgery was not associated with survival rate ($P = 0.603$; **Figure 2**). The 1- and 2-year survival rates were 94% for dogs with mitral valve regurgitation. The 1- and 2-year survival rates were 96% and 85%, respectively, for dogs without mitral valve regurgitation. Six dogs with mitral valve regurgitation and 9 dogs without mitral valve regurgitation had continued medical treatment (eg, an ACE inhibitor, diuretic, or both) after

Table 1—Results of analyses of certain clinical and radiographic variables for usefulness as indicators of the probability of survival for dogs with patent ductus arteriosus.

Variable	Frequency	Hazard ratio	95% CI	*P* value	*P* value likelihood ratio
Signalment findings					
Age at surgery	NA	1.001	1–1.002	0.004	0.004
Sex	NA	4.425	0.393–49.862	0.103	0.111
Weight at surgery	NA	1.081	1.001–1.167	0.048	0.05
Historical findings					
Exercise intolerance	17/52	NC	NC	NC	NC
Lethargy	8/52	7.75	1.078–55.55	0.042	0.058
Retarded growth	7/52	NC	NC	NC	NC
Weight loss	4/52	7.35	0.635–83.33	0.110	0.167
Dyspnea	6/52	1.63	0.36–4.76	0.384	0.436
Mitral insufficiency	22/52	0.559	0.058–5.376	0.614	0.599
Radiographic findings					
Left atrial enlargement	27/48	NC	NC	NC	NC
Left ventricular enlargement	15/48	2.5	0.88–11.11	0.11	0.086
Right atrial enlargement	10/48	13.698	1.404–125	0.024	0.013
Right ventricular enlargement	18/48	NC	NC	NC	NC
Pulmonary edema	11/48	NC	NC	NC	NC
Overperfusion of pulmonary circulation	26/48	1.44	0.511	6.66	0.511
Aneurysmal dilation of the aorta	26/48	0.55	0.122–149	0.293	0.267
Echocardiographic variables					
Left atrial diameter	NA	1.155	0.975–1.368	0.095	0.020
Left atrium/aorta diameter	NA	NC	NC	NC	NC
LVEDV index	NA	0.991	0.9555–1.018	0.606	0.587
LVESV index	NA	1.004	0.960–1.0457	0.892	0.830
Fractional shortening	NA	0.946	0.889–1.007	0.082	0.113
Aortic valve insufficiency	5/32	NC	NC	NC	NC
Tricuspid valve insufficiency	4/32	NC	NC	NC	NC
Pulmonic valve insufficiency	5/32	NC	NC	NC	NC
Treatment					
Diuretic treatment before surgery	14/52	9.345	0.954–90.90	0.055	0.035
ACE inhibitor treatment before surgery	8/52	14.705	1.524–142.85	0.020	0.011
Diuretic treatment after surgery	7/52	6.849	0.956–50	0.055	0.073
ACE inhibitor treatment after surgery	13/52	7.575	0.765–76.92	0.083	0.057

CI = Confidence interval. NC = Not calculable by the computer software because of monotone likelihood. NA = Not available. LVEDV = Left ventricular end-diastolic volume. LVESV = Left ventricular end-systolic volume. ACE = Angiotensin-converting enzyme.

surgery. At the time of follow-up, 3 dogs were being treated with β-adrenergic receptor–blocking drugs, 5 with ACE inhibitors, and 4 with diuretics. One dog (with mitral valve regurgitation before surgery) had exercise intolerance and was coughing and lethargic.

Of the 27 discrete and continuous variables evaluated by means of Cox proportional hazard analyses, 5 were associated with probability of survival (**Table 1**). Arrhythmias could not be evaluated as a risk factor for long-term survival because of the low number of cases with rhythm disturbances.

Discussion

The long-term survival rate after surgical occlusion of PDA was 87%. Four dogs died from cardiac-related disease within 2 years of surgery. There were no deaths related to cardiac disease that were reported after the second year following surgery. Long-term survival rate was associated with age, weight, lethargy, preoperative treatment with ACE inhibitors, and right atrial dilatation on radiographs at the time of diagnosis. However, it was not associated with diagnosis of mitral regurgitation at the time of surgery. Our study population was similar in breed, age, and sex distribution to those in earlier reports.[2,12,13]

The reporting of survival rates from 89% to 95% after surgical ligation of PDA has led to the consideration of surgical correction as a cure for the defect.[8,12,13] Surgical treatment is associated with a better long-term survival rate than medical treatment.[8,12] Our results confirm that finding because only 4 dogs died of cardiac-related disease within 2 years of surgery and no additional deaths related to cardiac disease were reported after that. In 2 earlier studies,[12,13] 4% to 8% of dogs died within a month after surgery and no deaths resulting from cardiac disease were reported in the long term. A study[8] of 37 dogs treated surgically for PDA revealed that the survival rate was 95% at 2 years and 60% at 10 years because dogs continued to die from cardiac-related disease beyond 2 years after surgery. Dogs in that study were older (mean, 18.4 months), and there was a higher prevalence of myocardial disease prior to surgery in that study population than in our study.

Greater age and weight had a negative association with survival in our study. The HR for those 2 variables was 1.001 and 1.081, respectively, which makes their association with survival questionable. In an earlier study,[12] the mortality rate was 22% in dogs that weighed > 23 kg (50.6 lb) and 8% in the overall study population. In that study, the mortality rate in dogs older than 2 years was 18%. In another study[13] involving 201 dogs, age and weight were not correlated with mortality rate. However, in that study, postoperative complications and death occurred in dogs that had hemorrhage during surgery. In the authors' experience, dissection of the DA is technically less difficult in young dogs because the vessel is less friable and the surrounding tissues are less fibrous. These results led to the conclusion that although age was not directly associated with an increased risk of death from PDA, it may increase the risk of intraoperative hemorrhage and subsequent death.[12,13] Another group of investigators evaluated dogs of different age groups (ie, 0 to 6 months, 6 to 12 months, and older than 12 months) and reported that age was not associated with survival rate in that study population.[16] However, dogs that died were older than 46 months old at the time of surgery, and there was a high number of adult dogs with myocardial failure prior to surgery. The high prevalence of heart failure may have been a confounding factor in that study, obscuring age as a risk factor for survival. In another study,[17] the severity of clinical signs of heart failure increased with age in dogs with PDA

Poor cardiac output, arrhythmias, and endocarditis have been proposed as influencing the surgical outcome for ligation of PDA in dogs.[3,16] In other studies,[9,12] heart failure was associated with a 40% mortality rate within 1 week of surgery and there was a 50% mortality rate in dogs with mitral valve regurgitation and atrial fibrillation.[12] Lethargy at the time of surgery appeared to be negatively associated with survival after surgical occlusion of PDA in our study. Other clinical signs associated with cardiac disease did not appear to have a negative association with long-term survival rate, even when they were reported more commonly by owners. Unfortunately, the distribution of the data in our study was such that an HR for exercise intolerance could not be calculated. Impairment of fractional shortening was not associated with lower long-term survival; however, medical treatment with an ACE inhibitor prior to surgery was significantly associated with a worse prognosis for long-term survival. Angiotensin-converting enzyme inhibitors improve the survival rate of patients with heart failure and prevent worsening of heart failure associated with mitral regurgitation.[18-22] In the present study, patients that received an ACE inhibitor at the time of surgery represented a subpopulation of dogs with a lower shortening fraction, suggesting that those dogs had more advanced disease. In another study,[9] 5 dogs were in heart failure; of those, the 2 that died within a week of coil embolization had been receiving medications for heart failure. In a study[8] of dogs in which there was a high prevalence of myocardial failure, a diagnosis of heart failure at the time of initial examination was not associated with survival rate. However, in that study,[8] dogs with heart failure that were treated surgically still had a higher risk of dying from cardiac disease in the long term.

Arrhythmias could not be evaluated as a risk factor in our study because only 2 dogs had atrial fibrillation at the time of initial examination. Atrial fibrillation is associated with atrial dilatation and progression of heart failure.[23,24] The 2 dogs with atrial fibrillation had pulmonary edema on radiographs and were being treated with diuretics and an ACE inhibitor at the time of examination. Those 2 dogs died 8 and 17 days after surgery.

With the exception of right atrial enlargement, radiographic findings did not appear to be prognostic factors in our study. The prevalences of cardiomegaly, left atrial dilatation, enlarged pulmonary vessels, and a prominent aortic bulge in our dogs were similar to previously reported values.[6,15-17] However, none of those findings were negatively associated with survival in our study. Objective criteria, such as the vertebral body-to-heart ratio,[25] are preferred for evaluating heart

size. Right atrial enlargement was a negative prognostic indicator for long-term survival in our study. Right atrial enlargement is not a consistent finding in dogs with PDA, especially when there is severe cardiac enlargement. Different radiographic exposure techniques, differences in cardiac filling, and severe cardiac dilatation may have contributed to that finding.[8,17,26] Dilatation of the right atrium was not observed during echocardiographic examination in our study because it is a subjective evaluation that is not commonly reported. Right ventricular dilatation was observed in 91% of the radiographs obtained in dogs with PDA in 1 study.[15] Right heart enlargement may be a sign of advanced heart failure.

Mitral valve regurgitation was not associated with long-term survival in dogs surgically treated for PDA. In our study, 42.3% of dogs had mitral valve regurgitation at the time of initial examination, a figure that is in the range (29% to 43%) reported for dogs evaluated for PDA closure.[6,8,9,15,16] Mitral valve regurgitation is attributed to dilatation of the mitral valve annulus and tethering of the papillary muscle secondary to left ventricular dilatation.[6,7,15] Mitral valve regurgitation is a recognized risk factor for long-term survival.[16] In a study[8] of 37 dogs, it was reported that evidence of mitral valve regurgitation on echocardiography at the time of initial examination may be associated with a reduction of survival. Mitral valve regurgitation in that study was associated with detection of mitral valve endocardiosis during follow-up examinations. Long-standing mitral valve regurgitation may lead to endocardiosis, a condition associated with a worse prognosis because such lesions are usually nonreversible. In another study,[16] 45% of dogs were 1 year of age or older at initial examination and the mean age was 18.4 months, whereas in our study, only 19.3% of dogs were older than 1 year. In our study, dogs with mitral valve regurgitation were not older than the rest of the study population. In the other study,[16] a high proportion of dogs had myocardial failure at the time of initial examination, indicating more advanced disease in those patients. Results of echocardiographic examinations in earlier studies[6,9] revealed that mitral valve regurgitation regresses in most patients treated surgically with either ligation or coil embolization. In one of those studies,[9] 3 months after PDA closure, the persistence of small jets of regurgitant flow through the mitral valve was observed in only 3 dogs, and 3 dogs that had mitral valve regurgitation and had heart failure at initial examination were not being medicated 1 month after surgery.[9] The percentage of dogs with mitral valve regurgitation was reduced from 38% to 5% after PDA closure in the other study.[6] Correction of PDA likely permits reverse remodeling of the left ventricle, and the mitral valves return to functional competency. The return to normal cardiac dimensions and elimination of mitral valve regurgitation after successful closure of PDA have been reported.[5] Because a diagnosis of mitral valve incompetence in the absence of myocardial failure did not affect long-term survival of dogs treated surgically for PDA in our study, it was presumed that mitral valve regurgitation regressed over time.

The rate of surgery-related complications in our study was low, compared with rates reported in other studies.[6,13,15,16,27] The perisurgical mortality rate in dogs in our study was 0%, despite the occurrence of acute intraoperative bleeding in 8 dogs. Surgeries were performed by experienced surgeons, which contributes to lower surgical mortality rates.[2] In our study, hemorrhage occurred in surgeries on older animals. Dogs that are older or in which the PDA is large may be at higher risk for bleeding during surgery.[6,12,13] It has been hypothesized that the structure is more friable and of larger size in older dogs than in younger dogs. Intraoperative bleeding was controlled by means of vascular clamps placed on the aorta and pulmonary artery. The DA was transected and oversewn with 3-0 or 4-0 monofilament nonabsorbable suture. In our study, none of the dogs that had acute intraoperative hemorrhage died in the first 5 days after surgery, although early deaths were reported by Birchard et al.[13] Different techniques have been recommended to control bleeding at the time of dissection or ligature of the DA.[27,28]

Limitations of our study include the retrospective design, which makes investigators dependent on the details included in records, especially those concerning complementary examinations before and after surgery. Echocardiographic results would have provided more accurate information regarding cardiac function in the dogs. Conclusions regarding the reversibility of systolic dysfunction could not be made from our data. It is known that mitral valve regurgitation is a risk factor for progression of heart failure and the survival of patients with dilated cardiomyopathy[29]; therefore, if mitral valve regurgitation had persisted in our dogs, the survival rate of affected dogs would have been lower than the survival rate of dogs without valvular regurgitation at the time of surgery. We were not able to compare outcomes for different surgical techniques because only 1 technique has been traditionally used at our institution for ligation of PDA. The technique of coil embolization is presently performed, but the number of dogs that had undergone that procedure at the time of the study was too low for those records to be included.

Surgical treatment of PDA was curative in young dogs without clinical signs of heart failure. Mitral valve regurgitation at the time of surgery was not negatively associated with survival rate in the absence of myocardial disease. Preoperative treatment with an ACE inhibitor was associated with poor long-term survival rate. Surgical correction of PDA should be recommended as early as possible after diagnosis, and mitral valve regurgitation is not a contraindication for surgery.

a. StatView, SAS Institute Inc, Cary, NC.

References

1. Tidholm A. Retrospective study of congenital heart defects in 151 dogs. *J Small Anim Pract* 1997;38:94–98.

2. Buchanan JW. Patent ductus arteriosus. *Semin Vet Med Surg (Small Anim)* 1994;9:168–176.

3. Goodwin JK, Cooper RCJ. Understanding the pathophysiology of congenital heart defects. *Vet Med* 1992;87:650–668.

4. Buchanan JW. Patent ductus arteriosus morphology, pathogenesis, types and treatment. *J Vet Card* 2001;3:7–16.

5. Weirich WE, Blevins WE, Rebar AH. Late consequences of patent ductus arteriosus in the dog: a report of six cases. *J Am Anim Hosp Assoc* 1978;14:40–51.

6. Corti LB, Merkley D, Nelson OL, et al. Retrospective evalu-

ation of occlusion of patent ductus arteriosus with hemoclips in 20 dogs. *J Am Anim Hosp Assoc* 2000;36:548–555.

7. Kono T, Sabbah HN, Rosman H, et al. Left ventricular shape is the primary determinant of functional mitral regurgitation in heart failure. *J Am Coll Cardiol* 1992;20:1594–1598.

8. Van Israel N, Dukes-McEwan J, French AT. Long-term follow-up of dogs with patent ductus arteriosus. *J Small Anim Pract* 2003;44:480–490.

9. Sisson D. Use of a self-expanding occluding stent for nonsurgical closure of patent ductus arteriosus in dogs. *J Am Vet Med Assoc* 2003;223:999–1005.

10. Stanley BJ, Luis-Fuentes V, Darke PG. Comparison of the incidence of residual shunting between two surgical techniques used for ligation of patent ductus arteriosus in the dog. *Vet Surg* 2003; 32:231–237.

11. Borenstein N, Behr L, Chetboul V, et al. Minimally invasive patent ductus arteriosus occlusion in 5 dogs. *Vet Surg* 2004;33:309–313.

12. Eyster GE, Eyster JT, Cords GB, et al. Patent ductus arteriosus in the dog: characteristics of occurence and results of surgery on one hundred consecutive cases. *J Am Vet Med Assoc* 1976;168:435–438.

13. Birchard SJ, Bonagura JD, Fingland RB. Results of ligation of patent ductus arteriosus in dogs: 201 cases (1969–1988). *J Am Vet Med Assoc* 1990;196:2011–2013.

14. Smetzer DL, Breznock EM. Auscultatory diagnosis of patent ductus arteriosus in the dog. *J Am Vet Med Assoc* 1972;160:80–84.

15. Van Israel N, French AT, Dukes-McEwan J, et al. Review of left-to-right shunting patent ductus arteriosus and short term outcome in 98 dogs. *J Small Anim Pract* 2002;43:395–400.

16. Van Israel N, French AT, Dukes-McEwan J, et al. Patent ductus arteriosus in the older dog. *J Vet Card* 2003;5:13–20.

17. Goodwin J, Lombard CW. Patent ductus arteriosus in adult dogs: clinical features of 14 cases. *J Am Anim Hosp Assoc* 1992;28: 349–354.

18. Ettinger SJ, Benitz AM, Ericsson GF, et al. Effects of enalapril maleate on survival of dogs with naturally acquired heart failure. *J Am Vet Med Assoc* 1998;213:1573–1577.

19. The CONSENSUS Trial Study Group. Effects of enalapril on mortality of severe congestive heart failure. Results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). *N Engl J Med* 1987;316:1429–1435.

20. Häggström J, Hansson K, Karlberg BE, et al. Effects of long-term treatment with enalapril or hydralazine on the renin-angiotensin-aldosterone system and fluid balance in dogs with naturally acquired mitral valve regurgitation. *Am J Vet Res* 1996;57:1645–1652.

21. Hamlin RL, Benitz AM, Ericsson GF, et al. Effects of enalapril on exercice tolerance and longevity in dogs with heart failure produced by iatrogenic mitral regurgitation. *J Vet Intern Med* 1996;10:85–87.

22. Kvart C, Haggstrom J, Pedersen HD, et al. Efficacy of enalapril for prevention of congestive heart failure in dogs with myxomatous valve disease and asymptomatic mitral regurgitation. *J Vet Intern Med* 2002;16:80–88.

23. Kastor JA. Atrial fibrillation. In: Kastor JA, ed. *Arrhythmias*. Philadelphia: WB Saunders Co, 1994;27–104.

24. Lim E, Barlow CW, Hosseinpour AR, et al. Influence of atrial fibrillation on outcome following mitral valve repair. *Circulation* 2001;104:I59–163.

25. Buchanan JW, Bucheler J. Vertebral scale system to measure canine heart size in radiographs. *J Am Vet Med Assoc* 1995;206: 194–199.

26. Lamb CR, Boswood A, Volkman A, et al. Assessment of survey radiograph as a method for diagnosis of congenital cardiac disease in dogs. *J Small Anim Pract* 2001;42:541–545.

27. Hunt GB, Simpson DJ, Beck JA, et al. Intraoperative hemorrhage during patent ductus arteriosus ligation in dogs. *Vet Surg* 2001; 30:58–63.

28. Hunter SL, Culp LB, Muir WW III, et al. Sodium nitroprusside-induced deliberate hypotension to facilitate patent ductus arteriosus ligation in dogs. *Vet Surg* 2003;32:336–340.

29. Romeo F, Pelliccia F, Cianfrocca C, et al. Determinants of end-stage idiopathic dilated cardiomyopathy: a multivariate analysis of 104 patients. *Clin Cardiol* 1989;12:387–392.