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Pulmonic Stenosis in Dogs: Balloon Dilation Improves Clinical Outcome

M. Stafford Johnson, M. Martin, D. Edwards, A. French, and W. Henley

Medical records of 81 dogs with severe pulmonic stenosis from 2 referral centers were examined retrospectively. Forty dogs underwent balloon valvuloplasty (BV), which was performed by 1 operator, whereas 41 did not. The mean age at latest follow-up was 41.5 months. A statistical comparison of the clinical outcome and survival was performed. Dogs revealing clinical signs at presentation showed a 16-fold increase in risk of death compared with asymptomatic dogs ($P < .001$). Statistical analyses demonstrated that an increase of 1 mm Hg in transstenotic pressure gradient (PG) at presentation was associated with a 3% increase in hazard rate ($P < .001$). Thirty-seven dogs survived BV with a median reduction in PG of 46%. The median preoperative PG was 120 mm Hg, and median PG 24 hours postoperatively was 55 mm Hg with a median of 55 mm Hg 6 months post-BV. Twenty (49%) of the non-BV (NBV) dogs remained asymptomatic at last follow-up. Fourteen (34%) of the NBV dogs died or were euthanized because of heart disease related to pulmonic stenosis. Twelve of these dogs died suddenly, whereas only 1 of the BV dogs died suddenly. After adjusting for PG, clinical signs at presentation, and age, BV or dilation was associated with a 53% reduction in hazard rate ($P = .005$). This study indicates that BV, when performed by an experienced operator, appears to be successful both in alleviating clinical signs and in prolonging survival in dogs with severe pulmonic stenosis.

Key words: Bulldog; Sudden death; Tricuspid valve regurgitation; Valvuloplasty.

Pulmonic stenosis generally is considered the third most common congenital cardiac defect in dogs.[1] Dogs with a Doppler-derived pressure gradient (PG) over 80 mm Hg have been classified as having severe stenosis and are at risk for developing clinical signs of exercise intolerance, syncope, or sudden death.[1] Balloon valvuloplasty (BV) currently is the best available therapy for this condition and has been recommended in severe cases with moderate or severe right ventricular hypertrophy (RVH) or when affected dogs are symptomatic.

Only 1 previous report[2] compared clinical signs and survival times in dogs undergoing BV (n = 18) with those that did not (NBV, n = 6). The investigators identified a reduction in clinical signs in dogs undergoing BV but did not demonstrate a significant difference in survival times between the 2 groups. Another study compared survival in dogs undergoing BV (n = 25), NBV (72), and dogs undergoing open surgical repair of their pulmonic stenosis (30) and found increased survival over 2 years in BV dogs versus NBV dogs.[1] Others have described the results of BV without comparison with an NBV population.[3–8] This study compares the progression of clinical signs and survival in 40 BV dogs with 41 NBV dogs.

Materials and Methods

Medical records of the Veterinary Cardiorespiratory Center (VCRC) and the University of Edinburgh (UE) between January 1993 and January 2003 were searched for cases of severe pulmonic stenosis. All dogs were presented either for investigation of an asymptomatic heart murmur or for clinical signs of exercise intolerance, collapse, or both. Inclusion criteria for the study included the presence of severe pulmonic stenosis with a PG > 80 mm Hg diagnosed on Doppler echocardiography. Dogs with concurrent cardiac defects causing cardiomegaly or clinical signs were excluded, but dogs with defects that were not considered clinically relevant were included. Dogs with tricuspid valve regurgitation (TVR) were included.

A total of 88 dogs were selected, but 7 were excluded. Specific reasons for exclusion from the study included concurrent clinically significant cardiac defects (n = 6) and absence of adequate follow-up (1). Three dogs had concurrent patent ductus arteriosus, 1 had concurrent moderate aortic stenosis, and 2 had moderate-sized ventricular septal defects. Evidence of left ventricular hypertrophy or dilatation was present in all 6 dogs.

Echocardiography[b] was performed in a standard manner from both right and left parasternal views at both centers.[9] The PG was assessed from the right parasternal short axis and left cranial parasternal views. No attempt was made to distinguish dogs with hypoplasia of the annulus from dogs without hypoplasia. Also, the presence of commissural fusion of the pulmonary valves versus thickened immobile valve leaflets was not recorded in this retrospective study. Data were recorded as subvalvular, valvular, or supravalvular stenosis. Subvalvular stenosis was defined as fixed rather than dynamic subvalvular obstruction. More proximal (midventricular) obstruction or double-chamber right ventricle was not present in any of these dogs. The presence of pulmonic or TVR was noted in each dog. The degree of RVH was recorded as mild, moderate, or severe. Mild hypertrophy was defined as subjectively increased right ventricular wall thickness with absence of paradoxical motion of the interventricular septum on the right parasternal short-axis view. Moderate hypertrophy was defined as mild paradoxical motion of the interventricular septum. Severe RVH was defined as marked paradoxical motion of the septum on this view.

BV was advised in all dogs with severe stenosis regardless of the morphologic appearance of the stenosis and whether hypoplasia of the annulus was present. Dogs undergoing BV were treated with beta blockers (atenolol 1 mg/kg PO q12h) for at least 2 weeks before BV. BV was performed via the left jugular vein as described in earlier reports by using fluoroscopic guidance.[4–7,10] A balloon with a mean diameter of 1.33 (range, 1.2–1.5) times the diameter of the pulmonary valve annulus as measured on angiography was used to perform balloon dilation. Beta blockers were administered for a minimum of 6 months after BV until the initial revisit was scheduled. Drug therapy was ceased at that time unless the results of BV were considered un-

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satisfactory and there was evidence of ongoing dynamic right ventricular outflow obstruction because of persistent RVH.

In Bulldogs, in addition to the above procedures, coronary angiography via the femoral artery was carried out before BV to identify dogs with an aberrant coronary artery associated with subvalvular stenosis in this breed. This abnormality would represent a contraindication to BV if identified.

The decision not to perform BV was dictated by the owners, usually because of financial reasons. Some of these dogs were maintained long term on atenolol 1 mg/kg PO q12h. NBV dogs were rechecked annually whenever possible, and echocardiography was repeated. For dogs that were not returned, follow-up was performed via telephone conversation with the owners and primary veterinary surgeons. Long-term follow-up was defined as follow-up of 6 months or more.

Statistics

A preliminary examination of the data used frequency tables and the mean plus range for different variables. Cox regression analysis was used to determine any association between time to death associated with heart failure (measured from the time of presentation until death from heart failure, including sudden death or euthanasia associated with heart failure) and BV, before and after adjusting for potential risk factors including PG, RVH, clinical signs, sex, and age.[11] PG, RVH, and clinical signs all were measured at the time of presentation, whereas BV was considered a time-dependent variable with dogs treated after presentation contributing time at risk to both the treatment (BV) and the nontreatment (NBV) groups. In addition, a binary variable indicating whether the dog had ever been treated with beta blockers was considered for inclusion in the Cox regression analysis.

Variables were selected for inclusion in the model if they significantly improved the fit (likelihood ratio chi-square statistic $P < .05$). Two-way interaction terms were tested between all main-effect variables. The relationship between survival time and treatment (BV), after adjusting for other risk factors, was displayed with plots similar to Kaplan-Meier curves but for time-dependent variables.[12] Smoothing splines were used to explore the functional form of the relationship between time to death associated with heart failure and the continuous variables (age and PG), after accounting for confounding from other variables.[c] The fit of the selected Cox regression model[13]was assessed by plotting deviance residuals against time to identify poorly fitting points.[12]

Results

Sixty-three dogs from the VCRC and 18 dogs from the UE were included in the study. All 40 dogs that underwent BV were selected from the VCRC and had BV performed by a single experienced operator (M.M.). The remaining 41 dogs did not undergo BV. Thirteen of these dogs were maintained long term on atenolol 1 mg/kg PO q12h.

TVR varying from mild to severe was identified in 32 dogs (39%). Nineteen were from the NBV group and 13 were from the BV group. Other additional cardiac defects were noted in 8 dogs. Five had a suspected patent foramen ovale. These were diagnosed during right ventricular angiography preceding BV when trivial right-to-left interatrial shunting was observed. No atrial septal defect was apparent on 2-dimensional echocardiographic examination in these dogs. Two dogs had a small patent ductus arteriosus, and 1 had a small ventricular septal defect with concurrent mild subaortic stenosis.

Three Bulldogs were diagnosed with subvalvular stenosis, and 1 Cocker Spaniel had supravalvular stenosis. The remainder had valvular stenosis. Three Bulldogs underwent BV. The first, performed early in the series, did not undergo coronary angiography and died during BV because of the unrecognized presence of an aberrant coronary artery that ruptured. In the remaining 2 dogs, an aberrant coronary artery was not identified and BV was performed successfully.

Table 1. Breeds affected with severe pulmonic stenosis in this series.

West Highland White Terrier	10
Cocker Spaniel	8
Labrador Retriever	8
Bulldog	7
Cavalier King Charles Spaniel	6
Bullmastiff	5
Yorkshire Terrier	5
German Shepherd Dog	4
Boxer	3
Crossbreeds	3
Shih Tzu	3
Border Terrier	2
Fox Terrier	2
Miniature Schnauzer	2
Rough Collie	2
Border Collie, Bichon Frise, French Bulldog, Parson Russell Terrier, Lurcher, Newfoundland, Pekingese, Great Pyrenees, Staffordshire Bull Terrier, Soft Coated Wheaton Terrier, Whippet	1 each breed

Twenty-six different breeds, including crossbreeds, were represented (Table 1). The West Highland White Terrier was most commonly represented, followed by the Cocker Spaniel, Labrador Retriever, Bulldog, and Cavalier King Charles Spaniel. Sex distribution was 39 females (48%) and 42 males (52%).

Although the majority of dogs were younger than 12 months old at first presentation, the age distribution of the study population skewed greatly to the right, with a small number of dogs much older than the rest.

Twenty-six dogs (32%) were asymptomatic at presentation and have remained so at last follow-up at a median age of 50 months (range, 5–144 months). Of these, 20 were from the NBV group and 6 were from the BV group. Median PG for this population was 90 mm Hg (range, 80–170 mm Hg).

Forty-two dogs (52%) were symptomatic with exercise intolerance, syncope, or both. Thirty-three were from the BV group and 9 were from the NBV group. The median age of onset of clinical signs was 12 months (range, 2–70 months). Age of onset varied between the 2 populations, with BV dogs showing signs at a median age of 6 months and NBV dogs at 12 months. Median PG for symptomatic dogs was 110 mm Hg (range, 80–200 mm Hg). The median age at last follow-up was 28 months (range, 3–120 months).

Sudden death was noted in 13 dogs (16%). One occurred 2 months post-BV, whereas 12 occurred in the NBV group. These are described later. Median PG for dogs experiencing sudden death was 150 mm Hg (range, 140–200 mm Hg).

Thirty-seven dogs survived BV and 3 died during the procedure. In the 37 surviving dogs, a median reduction in PG of 46% was observed. The median preoperative PG was

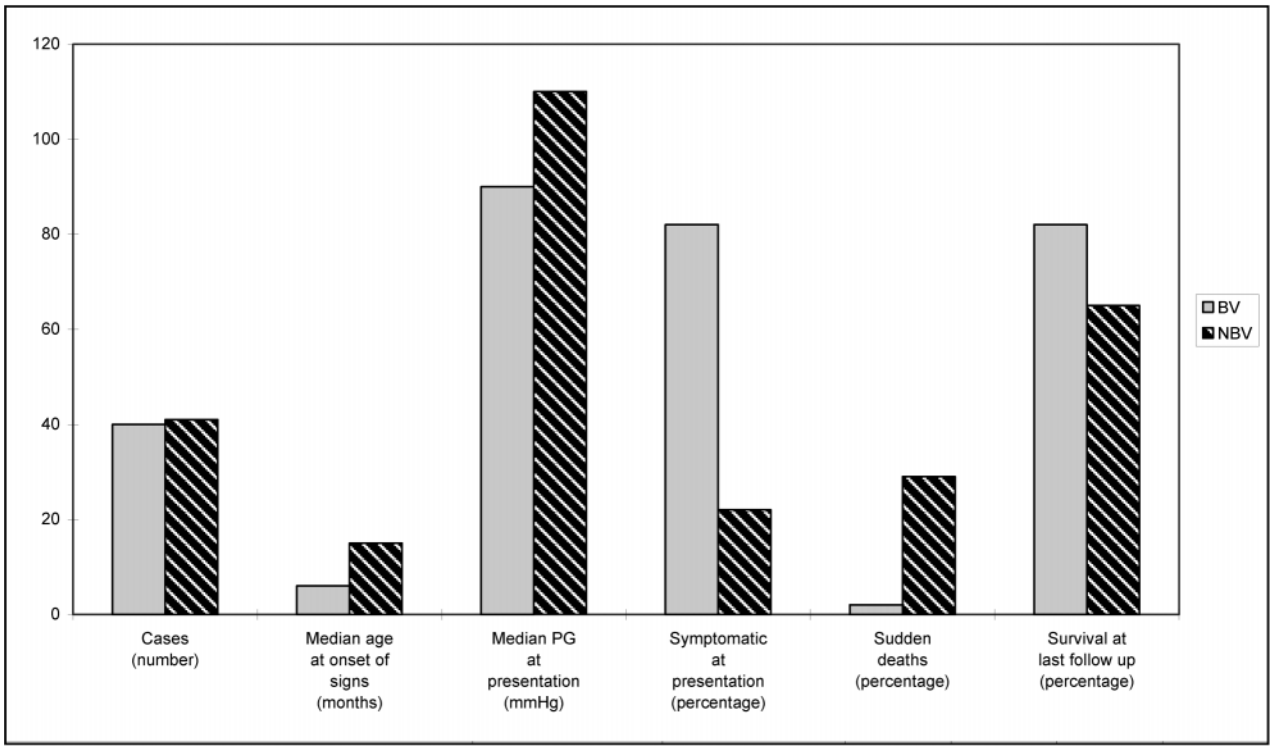

Fig 1. A comparison between BV dogs and NBV dogs at presentation and at last follow-up.

120 mm Hg, and median PG 24 hours postoperatively was 55 mm Hg with a median of 55 mm Hg at 6 months post-BV.

Long-term follow-up was available for the 37 surviving BV dogs, and the median age at last follow-up was 32 months (range, 12–96 months). Of these 37 dogs, 30 became asymptomatic and 3 remained asymptomatic (range, 12–94 months). One of the dogs that became asymptomatic developed right-sided congestive heart failure 8 years after BV at 10 years and was euthanized at that time. Of the remaining 4 dogs, 1 had continuing right-sided congestive heart failure and died 4 months post-BV, 1 experienced restenosis and had ongoing right heart failure until euthanasia at 18 months post-BV, 1 had ongoing syncopal episodes with sudden death after 2 months, and 1 died of unrelated causes after 4 months.

Follow-up was available for all 41 NBV dogs with a median age at last follow-up of 46 months. Twenty of these dogs (49%) remained asymptomatic at this time, and 14 of the symptomatic dogs (34%) died or were euthanized because of heart disease related to pulmonic stenosis. Of these dogs, 1 died of right heart failure, 1 was euthanized because of severe clinical signs, and the remaining 12 died suddenly with (n = 8) or without (4) previous clinical signs. Eight dogs demonstrated signs of collapse before death. Death occurred at a median age of 9 months for these 12 dogs (range, 3–65 months). The clinical features of BV and NBV are compared in Figure 1.

Table 2 shows the univariable Cox regression models used to assess associations between individual risk factors and time to death from heart failure. When considered independently of other risk factors, BV did not have a significant treatment effect ($P = .48$). The significant risk factors from the univariable models were PG, TVR, RVH, and clinical signs (including congestive heart failure) at presentation ($P = .001$, .019, .002, and .010, respectively). Two dogs with BV had missing values for clinical signs at presentation and were therefore excluded from the corresponding model. Age, sex, and use of beta blockers were not significantly associated with survival time ($P = .330$, .057, and .620, respectively). Although age was nonsignificant as a linear effect in the univariable analysis, no assessment was made of nonlinear effects at this stage (see below).

Table 3 shows the final multivariable Cox regression model for risk factors associated with death from heart failure. An increase of 1 mm Hg in PG at presentation was associated with a 3% increase in hazard rate ($P < .001$). Dogs revealing clinical signs at presentation had a 16-fold increase in risk of death compared with asymptomatic dogs ($P < .001$). The relationship between age at presentation and risk of death associated with heart failure was assessed by fitting a multivariable Cox regression model with age represented by a smoothing spline. This analysis showed that age had a nonlinear relationship with risk ($P < .05$): Risk decreased with age up to approximately 24 months, after which risk started to increase with age (Fig 2). The plot suggests that age has an approximately quadratic relationship with risk of heart failure, and it was included in this form in the final multivariable model. After adjusting for PG, clinical signs at presentation, and age, BV was associated with a 53% reduction in hazard rate ($P = .005$). Figure 3a,b show separate survivor curves for time to death from heart failure before and after adjustment for the risk factors in the multivariable model. These plots illustrate

Table 2. Univariate results for risk factors calculated by Cox regression analysis.

Risk Factor	No. of Dogs	Coefficient	Standard Error	Hazard Ratio	Lower Confidence Interval	Upper Confidence Interval	*P* value
Age	80[a]	−0.017	0.017	0.98	0.95	1.02	.330
Beta blockers	80[a]	−0.101	0.202	0.90	0.61	1.34	.620
BV	80[a]	−0.147	0.209	0.86	0.57	1.30	.480
PG	80[a]	0.020	0.006	1.02	1.01	1.03	<.001
Male dogs	80[a]	0.404	0.212	1.50	0.99	2.27	.057
Clinical signs	78[a,b]	1.190	0.460	3.27	1.33	8.06	.010
TVR	80[a]	0.461	0.197	1.59	1.08	2.33	.019
RVH	78[a,b]	1.450	0.233	4.28	1.71	10.70	.002

BV, balloon valvuloplasty; PG, pressure gradient at presentation; TVR, tricuspid valve regurgitation; RVH, right ventricular hypertrophy.

[a] One dog whose age was not included at BV was excluded from the Cox regression analysis.

[b] Two dogs whose clinical signs were not included at presentation and RVH were excluded from the corresponding univariable Cox regression models.

how the beneficial effect of BV on survival time becomes apparent after adjustment for the confounding variables. As before, 2 dogs were excluded from the final multivariable model because of missing data for clinical signs at presentation.

Although both TVR and RVH were significant risk factors in the univariable analysis, neither was significantly associated with survival time in the multivariable analysis after adjustment for PG ($P = .793$ and .560, respectively). An indicator variable for institute was included in the multivariable model after adjusting for BV and was not found to be significant ($P = .41$).

Plotting deviance residuals against time identified 2 dogs that were poorly fitted by the selected Cox regression model. Both died within 1 month of presentation, with 1 undergoing BV and the other untreated. Refitting the final model after excluding these 2 dogs did not have a material impact on the model coefficients.

Discussion

One previously published paper compared a small population of BV dogs and NBV dogs and demonstrated a reduction in clinical signs but not increased longevity in dogs after BV.[2] In our study, comparing a larger population of dogs, we documented an improvement in clinical signs and survival in dogs undergoing BV, with a 53% reduction in hazard rate ($P = .005$). Additionally, there was only 1 sudden death in the BV group compared with 12 in the NBV group.

Only 1 experienced operator from 1 center performed BV to minimize variability in results because of operator experience or technique. Records of 18 additional NBV dogs from another center (UE) were used to balance the numbers in the 2 populations. The NBV dogs from both centers did not undergo BV because of owner constraints and were not explicitly selected according to the presence or absence of clinical signs or echocardiographic appearance of the stenosis. However, it is apparent from the data that dogs with clinical signs were more likely to receive BV, which, if not considered in the analysis, could have led to underestimation of the benefits of BV: 85% of the BV dogs were symptomatic on presentation compared with 51% of the NBV dogs. Owners whose dogs display clinical signs caused by pulmonic stenosis may be more likely to elect an invasive procedure for their dogs than are owners whose dogs display no outward signs of being affected. Financial constraints are also a factor.

The inclusion in this study of data from 2 institutes with all treated dogs from the same institute (VCRC) could have resulted in confounding of the relationship between BV and survival time. But because untreated dogs were represented at both institutes, an assessment of the institute effect for untreated dogs could be conducted, thus avoiding complete confounding. The nonsignificant effect of institute suggested that untreated UE dogs and untreated VCRC dogs could be combined into a single untreated category for both institutes. We were not able to assess whether the effect of BV varied by institute, and the analysis of this study is

Table 3. Cox regression model indicating that balloon valvuloplasty (BV), pressure gradient (PG) at presentation, clinical signs at presentation, age, and the quadratic function of age (Age^2) were significantly associated with survival time of dogs with severe pulmonary stenosis.

	Coefficient	Standard Error	Hazard Ratio	Lower Confidence Interval	Upper Confidence Interval	*P* value
Age	−0.188	0.058	0.828	0.740	0.927	.001
Age^2	0.003	0.001	1.003	1.001	1.004	.002
BV	−0.750	0.267	0.472	0.280	0.797	.005
PG	0.025	0.007	1.025	1.012	1.039	<.001
Clinical signs	2.746	0.690	15.575	4.029	60.205	<.001

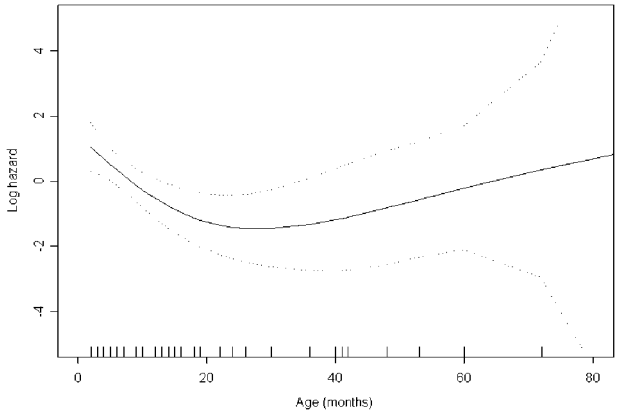

Fig 2. Graphic representation of the functional form of age at presentation, modeled with smoothing splines in a multivariable Cox regression model for risk of death associated with heart failure, including sudden death, with time measured as number of months after first presentation. The plot shows the smoothed, fitted log hazard and 95% confidence intervals, with rug plots (vertical lines at the bottom of the figure) representing the age at presentation of individual dogs, indicating that the risk of death decreases with age at first and then increases after 30 months.

based on the assumption that the BV treatment protocol at the VCRC is sufficiently representative so that the estimated effect of BV can be generalized to other centers, including the UE.

In observational studies, the above types of selection effects are common. We have tried to reduce their impact on the estimated treatment effect by adjusting for measured covariables such as age, PG, and clinical signs. Even so, the risk of residual confounding from other unmeasured variables remains.

The West Highland White Terrier, Labrador Retriever, and Cocker Spaniel all were commonly presented breeds in common with a previous UK study,[2] and all are popular breeds in the UK. No sex predisposition was identified, in keeping with another study,[14] but 2 other studies demonstrated a male predisposition.[2,3]

TVR, varying from mild to severe, was seen in 39% of dogs compared with 46% in another study.[14] Associated right atrial enlargement varied from mild to marked. Many of these dogs may have represented concurrent tricuspid valve dysplasia, which has been recognized in association with pulmonic stenosis.[1]

A patent foramen ovale was diagnosed during right ventricular angiography before performing BV. Contrast echocardiography was not performed routinely in NBV dogs, and some of these dogs also may have had a patent foramen ovale, which was not detected.

It was not possible to classify the detailed nature of the stenosis in this retrospective study. A recent study[3] has shown the value of differentiating between dogs with or without hypoplasia of the pulmonary annulus before BV. A better prognosis after BV may be given in dogs without hypoplasia, and this finding may influence the decision to perform BV. In the future, this factor should be taken into consideration before undertaking BV.

It is important to perform coronary angiography in Bulldogs before BV. Failure to document the presence of an aberrant coronary artery caused the death of the first Bulldog undergoing BV in our clinic.

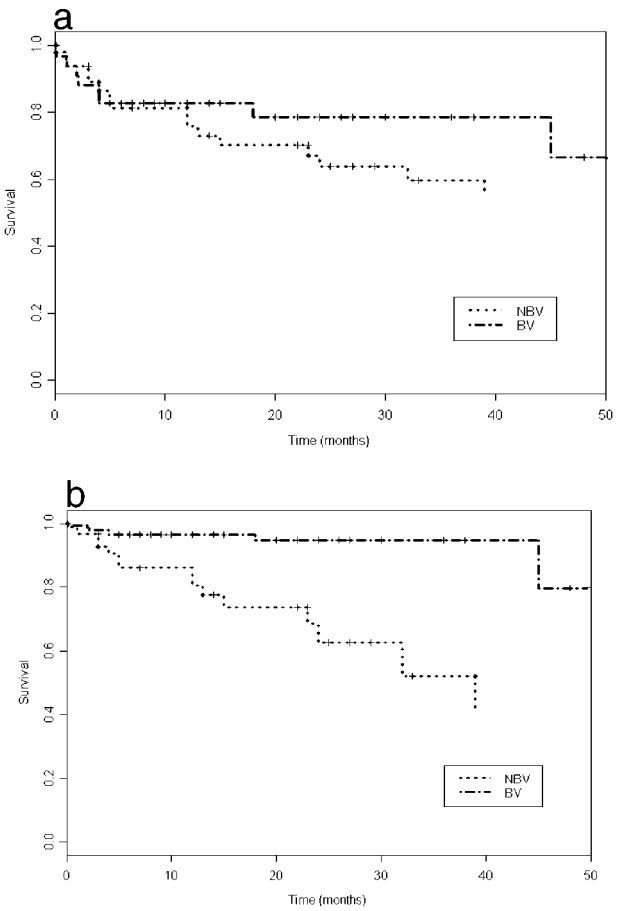

Fig 3. Survivor curves comparing time to death associated with heart failure, including sudden death for dogs treated with balloon valvuloplasty (BV), and dogs in the nontreatment group (NBV). **(a)** Without adjustment for other variables. **(b)** Following adjustment of the model for clinical signs, gradient pressure, and age at presentation. The crosses represent censoring times (ie, the times at which dogs were lost to follow-up). The survivor curves are not displayed beyond 50 months because of the small number of dogs followed up for this length of time. No deaths were in the NBV group beyond 40 months, and only 2 deaths were in the BV group beyond 50 months.

In some dogs placed on atenolol before BV, PG decreased by the time of intervention. Whether a reduced PG could be beneficial in NBV dogs on long-term atenolol still is speculative.

Although the BV and NBV populations of dogs in this study differed in some respects, the median PG at presentation was similar for both groups. However, more dogs in the NBV group (n = 19) had TVR than did dogs in the BV group (13). Dogs in the BV group developed clinical signs at a younger age than did dogs in the NBV group.

The increased risk of death caused by severe pulmonic stenosis in young dogs was not surprising, and an increase of 1 mm Hg in PG at presentation was associated with a 3% increase in hazard rate. The most severely affected pups are most likely to exhibit clinical signs or die of their condition at a young age. The risk decreased over the first 30 months of life. Reasons for increased risk again after this time period are unclear.

The proportion of dogs that eventually becomes symptomatic because of pulmonic stenosis is not known at this time. Symptomatic dogs in general presented with a higher median PG than did asymptomatic dogs. However, overlap occurred between these 2 groups, and it is not possible to determine from the presenting PG which individuals will become symptomatic. Fourteen dogs in the NBV group, all with PG below 100 mm Hg and with mild to moderate RVH, have remained apparently asymptomatic for as long as 84 months. It is likely that a proportion of these would show increased exercise tolerance after BV, but in all instances the owners have not perceived a problem. These dogs are currently monitored primarily via telephone contact with the owner.

The drop in PG post-BV was similar to that reported in several previous studies.[2,3,15] Hemodynamic improvement and a reduction in clinical signs after BV has been documented previously in humans and dogs.[8,16–19]. Despite the greater proportion of symptomatic dogs in the BV group at presentation, this group improved symptomatically post-BV. In this group, 15% of dogs were asymptomatic on presentation, increasing to 72% after successful BV. In the NBV group, 49% presented as asymptomatic. Dogs in this group were more likely to exhibit sudden death, with or without previous clinical signs. Several dogs had been scheduled for BV but died before the procedure could be performed. In total, 12 dogs in the NBV group died suddenly versus 1 dog in the BV group.

The results of the final multivariable Cox regression model showed that BV had a significant protective effect on survival times. This treatment effect was not significant in the univariable model because of the confounding effect of the variable clinical signs at presentation: Dogs in the BV group were more likely to have clinical signs at presentation than were dogs in the nontreatment group, and clinical signs were associated with increased risk of heart failure, thus counterbalancing the protective effect of BV.

Although this study found BV to be significantly protective for death associated with heart disease, the dogs in this study were selected by convenience sampling. The estimated protective treatment effect may have been influenced by other unmeasured confounders. A randomized clinical trial would provide the ''gold standard'' design for confirming the efficacy and clinical usefulness of BV as an intervention to improve the survival of dogs with severe pulmonic stenosis. In addition, some of the improvement in survival time attributed to surgery may have been attributable to superior postdiagnosis management.

In this study, failures were defined as dogs that died or were euthanized because of heart failure. However, euthanasia is not an end-point determined exclusively by disease pathology; it also will depend on factors that include clinical status of the dog as well as financial constraints and attitudes of the owner. The classification of 3 euthanized dogs as equivalent to those dying naturally of heart failure may have introduced some bias into the estimation of the survival function and median survival time. However, any bias in the estimate of the treatment effect is likely to be negligible because of the small proportion of dogs that was euthanized. An alternative approach would have been to censor the euthanized dogs, but this would have introduced a form of informative censoring and would have reduced the power of the study.[20]

Ideally, all dogs should be followed until the point of death, whether by natural causes or euthanasia. It is not currently known what proportion of BV dogs may ultimately die of their heart disease in comparison with NBV dogs. To document such outcomes would require longer follow-up of these dogs.

In conclusion, BV was associated with a 53% reduction in hazard rate after adjustment for the significant risk factors, PG, age, and clinical signs at presentation, with only 1 sudden death in the BV group compared with 12 in the NBV group. This study has shown that BV, when performed by an experienced operator, appears to be successful both in alleviating clinical signs and in prolonging survival in dogs with severe pulmonic stenosis.

Footnotes

[a] Ewey DM, Pion PD, Hird DW. Survival in treated and untreated dogs with congenital pulmonic stenosis [abstract]. J Vet Intern Med 1992; 6:114

[b] Esaote Biomedica Challenge 7000 with 2.5–7.5 MHz mechanical probes; Esaote Biomedica UK Ltd, Huntingdon, Cambridgeshire, UK

[c] S-PLUS 6 (Insightful 2001) Insightful Corp, Seattle, WA

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References

1. Kienle RD. Congenital pulmonic stenosis. In: Kittleson MD, Kienle RDE, eds. Small Animal Cardiovascular Medicine. St Louis, MO: Mosby; 1998:248–260.

2. Ristic JME, Marin CJ, Baines EA, Herrtage ME. Congenital pulmonic stenosis—A retrospective study of 24 cases seen between 1990–1999. J Vet Cardiol 2001;3:13–19.

3. Bussadori C, De Madron E, Santilli RA, Borgarelli M. Balloon valvuloplasty in 30 dogs with pulmonic stenosis: Effect of valve morphology and annular size in initial and 1-year outcome. J Vet Intern Med 2001;15:553–558.

4. Brownlie SE, Cobb MA, Chambers J, et al. Percutaneous balloon valvuloplasty in four dogs with pulmonic stenosis. J Small Anim Pract 1991;32:165–169.

5. Martin MWS, Godman M, Luis Fuentes V, et al. Assessment of balloon pulmonary valvuloplasty in six dogs. J Small Anim Pract 1992;33:443–449.

6. McIntosh Bright J, Jennings J, Toal R, Hood ME. Percutaneous balloon valvuloplasty for treatment of pulmonic stenosis in a dog. J Am Vet Med Assoc 1987;191:995–996.

7. Sisson DD, MacCoy DM. Treatment of congenital pulmonic stenosis in two dogs by balloon valvuloplasty. J Vet Intern Med 1998;2: 92–99.

8. Thomas WP, De Lellis LA, Sisson D. Balloon dilation of congenital outflow obstruction in dogs: Mid-term results. Proceedings of the 8th ACVIM Forum, Washington DC, 1990;907–909.

9. Boon JA. Pulmonic stenosis. In: Manual of Veterinary Echocardiography, 1st ed. Baltimore, MD: Williams & Wilkins; 1998:397–404.

10. Buchanan JW, Anderson JH, White RI. The 1st balloon valvuloplasty: An historical note. J Vet Intern Med 2002;16:116–117.

11. Cox DR. Regression models and life tables (with discussion). J Royal Stat Soc 1972; B74:187–220.

12. Venables WN, Ripley BD. Modern Applied Statistics with S-Plus, 2nd ed. New York NY: Springer-Verlag; 1997.

13. Therneau TM, Grambsch PM. Modeling Survival Data: Extending the Cox Model. New York NY: Springer-Verlag; 2000:107–111.

14. Fingland RB, Bonagura JD, Myer CW. Pulmonic stenosis in the dog: 29 cases (1975–1984). J Vet Med Assoc 1986;189:218–226.

15. Gordon SG, Miller MW, Baig S. A retrospective review of balloon valvuloplasty for the treatment of pulmonic stenosis in 50 dogs. First International Symposium for Veterinary Cardiology, Scientific Proceedings, Prague, Czechoslovakia, June 14–15, 2002.

16. Rao PS, Mohammed DF, Solymar L, Mardini MK. Long-term results of balloon pulmonary valvuloplasty of valvar pulmonic stenosis. Am Heart J 1988;115:1291–1296.

17. Echigo S. Balloon valvuloplasty for congenital heart disease: Immediate and long-term results of multi-institutional study. Pediatr Int 2001;43:542–547.

18. Gupta D, Saxena A, Kothari SS, Juneja R. Factors influencing late course of residual valvular and infundibular gradients following pulmonary valve balloon dilatation. Int J Cardiol 2001;79:143–149.

19. Shrivastava S, Krishna Kumar R, Saxena A, Das G. Determinants of immediate and follow-up results of pulmonary balloon valvuloplasty. Clin Cardiol 1993;16:497–502.

20. Binns SH, Johnson MS, Martin MWS, Day MJ. Survival analysis of 124 cases of canine pericardial disease: Censoring and prognostication. In: Proceedings of the Society for Veterinary Epidemiology and Preventive Medicine, Warwick, UK, April 2003.