Left Basilar Systolic Murmur in Retired Racing Greyhounds

F. Fabrizio, R. Baumwart, M.C. Iazbik, K.M. Meurs, and C.G. Couto

Nineteen of 28 (67%) Greyhounds enrolled in the Blood Donor Program at The Veterinary Teaching Hospital, The Ohio State University (Columbus, OH), had a left basilar systolic murmur. Ten Greyhounds with murmurs and 9 without murmurs were evaluated to gain knowledge about the pathogenesis of this murmur. Echocardiograms were performed without sedation by means of a GE Vivid 7 Echocardiographic System with a continuous ECG; systolic arterial blood pressure (SABP) was measured with an Ultrasonic Doppler Flow detector model 811-B. The mean peak aortic velocity in the Greyhounds with murmurs (2.15 m/s; range, 1.8–2.2 m/s) was significantly higher than in the Greyhounds without murmurs (1.89 m/s; range, 1.6–2.0 m/s) ($P < .001$); there were no significant differences between groups for aortic valve or annulus diameter, fractional shortening, pulmonic velocity, SABP, hematocrit, serum protein concentration, or red blood cell counts. In this study, Greyhounds with soft, left basilar systolic murmurs had mildly (but significantly) higher mean peak aortic velocities than similar dogs without murmurs. In the dogs with murmurs (and higher velocities), we could not identify structural abnormalities, such as valvular lesions or other congenital defects. There was no inverse correlation between the systolic murmur and the higher hematocrit and red blood cell counts observed in this breed. This 1-2/6 basilar systolic murmur is common in Greyhounds, and it does not appear to be of any clinical consequence.

Key words: Aortic velocity; Dog.

T he increasing popularity of rescuing retired Grey- hounds means that veterinarians are likely to evaluate dogs of this breed frequently. Last year, between 12,000 and 18,000 retired racing Greyhounds were adopted in the United States and Canada (Guccione, National Greyhound Council, personal communication). Therefore, it is important that the physiologic peculiarities of this breed be recognized. For instance, the mean PCV, hemoglobin concentration, red blood cell count (RBC), and whole-blood viscosity are higher, whereas the white blood cell count, neutrophil count, and platelet count are lower in Greyhounds than in other breeds.[1–3] In addition, serum creatinine concentrations and liver transaminase activities are higher in Greyhounds than in other breeds.[1,4]

In our institution, most of the dogs enrolled in the Blood Donor Program are healthy, adult, retired racing Greyhounds. We made the observation that a grade 1-2/ 6 left basilar systolic murmur was ausculted in many Greyhound blood donors, but we could not find any reference to this murmur in the literature, even though numerous articles have been written on cardiovascular physiology in the breed.[5–10]

Several factors can be responsible for the develop- ment of a systolic murmur, including congenital or acquired structural cardiac abnormalities and physio- logic factors, such as alterations in blood viscosity or increases in blood flow velocity.[11]

Unfortunately, auscultation alone cannot differenti- ate between functional murmurs, which are not associ- ated with cardiovascular pathology, and those associat- ed with mild changes in outflow tract abnormalities, such as pulmonic or aortic stenosis. Therefore, for many years, clinicians have been challenged by the task of distinguishing physiologic murmurs from those associ- ated with clinically relevant cardiac diseases.

The purposes of this study were (1) to determine the prevalence of and characterize the murmur commonly heard in Greyhounds; (2) to rule out the presence of structural cardiac abnormalities (eg, valvular lesions or other congenital defects) as the cause of this murmur; and (3) to determine if the presence of the murmur correlates with any of the other breed-specific clinico- pathologic features.

Materials and Methods

Participating dogs were selected from the pool of 28 adult, healthy, retired racing Greyhounds enrolled in the Blood Donor Program at the Transfusion Medicine Service, Veterinary Teaching Hospital, College of Veterinary Medicine, The Ohio State University (Columbus, OH).

All dogs underwent a physical examination and cardiac auscultation. Nineteen of 28 (67%) Greyhounds had a 1-2/6 left basilar systolic murmur. From this pool, a representative sample population of 10 dogs with soft (1-2/6) systolic left basilar murmurs and 9 dogs without heart murmurs were selected for additional evaluation.

CBCs were performed in a standard fashion with a Cell Dyn 3500R[a]; serum biochemical profiles were performed on a Hitachi 911 Automatic Analyzer.[b] Inclusion criteria for the study were results of a CBC and profile within the reference range for the breed in an apparently healthy adult Greyhound. All of the dogs were evaluated a minimum of 1 week after blood donation. Additionally, according to standard echocardiographic criteria, no valvular lesions, congenital defects, or cardiomyopathies were identified in any of the dogs.

Echocardiographic examinations and indirect blood pressure measurements were performed in all dogs. A phonocardiogram was performed for illustrative purposes in 4 dogs with murmurs and in 3 dogs within the control group.

Echocardiographic Examination

All the echocardiograms were performed without sedation with a GE Vivid 7 Echocardiographic System[c] with a continuous ECG. Echocardiographic recordings were performed by one of the authors (R.B.), and all raw data were captured digitally to maintain fidelity for offline measurements. A 2.5-MHz transducer was used for color and spectral Doppler examinations, whereas the 2-dimensional (2-D) examination was performed with a 5-MHz transducer. Dogs were examined in right-lateral recumbence, acquiring the images parasternally and subcostally from the dependent right side. For all velocity recordings, the cursor was aligned close to parallel to the flow according to concurrent 2-D and color flow Doppler echocardiography, and optimal signal strength was obtained by observing the real-time spectral display and listening to the audio signal of the Doppler system.

The left ventricle was imaged from the right intercostal (parasternal) position at the papillary muscles to obtain a short-axis tomogram. M-mode measurements of the left ventricular (LV) diastolic diameter (LVD), LV systolic diameter (LVS), and LV wall thicknesses (free wall and septum) in diastole and systole were obtained. All parameters were measured at end diastole and end systole from at least 3 cardiac cycles. The transducer was then angled craniodorsally, bringing the mitral valve into plane. End-point to septal separation (EPSS) was measured from the tip of the early diastolic motion of the anterior mitral leaflet to the septal wall.

The 2-D examination of the LV outflow tract (LVOT) was performed from the right parasternal long- and short-axis views. The valvular and supravalvular regions of the aorta were evaluated subjectively for evidence of narrowing and abnormal morphology within each view. The diameter of the aortic annulus was measured at end systole; it was obtained from the right parasternal long-axis view as the distance from the point where the anterior aortic cusp meets the ventricular septum to the point where the posterior cusp meets the anterior mitral leaflet. The aortic diameter from 3 cardiac cycles was averaged.

Color flow Doppler was used to identify systolic turbulence in the LVOT, aortic insufficiency, and mitral regurgitation by the right parasternal long-axis view. Spectral Doppler of the LVOT was conducted from the subcostal view. The peak LVOT velocity was determined from the subcostal view by continuous-wave Doppler. Pulmonic velocities were recorded with pulsed-wave Doppler from the right parasternal short axis. The mean peak velocity measurement was obtained from 5 cardiac cycles. A heart rate was recorded along with each velocity measurement and was determined from the R-R interval immediately preceding the recorded spectral signal.

Systolic arterial blood pressure (SABP) was measured with an Ultrasonic Doppler Flow detector[d] and a 4.0-cm-wide cuff in the right forearm, with the dog lying in left-lateral recumbency on the floor.

Data Analysis

Because the distribution of data was approximately normal, a 2-sample Student's t-test was used to compare variables between dogs with and without murmurs. Correlation coefficients were determined by the Pearson method.[e] Significance was defined at $P < .05$.

Results

Dogs with Murmurs

A left basilar systolic murmur was ausculted in 19 of the 28 dogs (67%) enrolled in the Blood Donor Program. Ten dogs were selected on the basis of the presence of
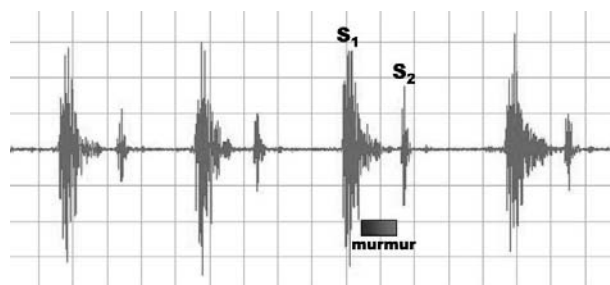

Fig 1. Phonocardiogram of a Greyhound with a typical soft basilar systolic murmur.

a systolic, 1-2/6 left basilar systolic heart murmur; there were 3 spayed females and 7 castrated males, with a mean age of 6.6 years (range, 4–11 years). Their mean body weight was 33.2 kg (range, 28–40.2 kg). All dogs were in either sinus rhythm or sinus arrhythmia and had no evidence of atrial or ventricular ectopic complexes, as determined from the continuous ECG during echocardiographic evaluation. The mean heart rate was 118 beats/min (range, 83–156 beats/min). The mean platelet count for this group was 206×10^9/L (range, $152–239 \times 10^9$/L), whereas the mean hematocrit (Hct) was 56% (range, 49–64%), the mean RBC was 8.1×10^9/L (range, $6.9–9.0 \times 10^9$/L), and the mean serum protein concentration was 6.3 g/dL (range, 6.0–7.5 g/dL). The mean indirect systolic blood pressure was 130 mm Hg (range, 110–140 mm Hg). A pictorial representation (phonocardiogram) of the decrescendo, ejection type of murmur heard is shown in Figure 1.

Subjectively, there was no evidence of structural narrowing or abnormalities of the LVOT or pulmonic valve. The mean LVOT diameter was 2.46 cm (range, 2.3–2.72 cm) (Table 1). The LV free wall (LVFW) thickness in diastole was significantly higher than in the dogs without murmurs ($P = .01$). The mean aortic velocity measured from the subcostal view was 2.15 m/s (range, 1.8–2.2 m/s). The mean pulmonic velocity was 1.25 m/s (range, 0.99–1.49 m/s). One dog had trivial mitral regurgitation, and no dogs had aortic regurgitation.

Dogs without Murmurs

Nine dogs were selected on the basis of the absence of an auscultable murmur; we had originally targeted 10 dogs for this group but could not find the 10th dog without a murmur in a reasonable time frame. There were 4 spayed females and 5 castrated males, with a mean age of 6.4 years (range, 2–9 years). Their mean body weight was 31.2 kg (range, 23.7–36 kg). All the dogs were either sinus rhythm or sinus arrhythmia and had no evidence of atrial or ventricular ectopic complexes at the time of their echocardiographic examination. The mean heart rate was 121 beats/min (range, 91–138 beats/min). The mean platelet count for this group was 187×10^9/L (range, $169–220 \times 10^9$/L), whereas the mean Hct was 57% (range, 43–66%), the mean RBC was 8.4×10^9/L (range, $5.9–8.0 \times 10^9$/L), and the mean serum protein concentration was 6.5 g/dL

Table 1. M-mode echocardiographic measurements in Greyhounds with and without aortic murmurs.[a]

	Dogs with Murmurs	Dogs without Murmurs	P Value
IVSd (cm)	1.55 ± 1.17	1.47 ± 1.20	0.11
LVIDd (cm)	4.42 ± 0.41	4.60 ± 0.41	0.10
LVFWd (cm)	1.37 ± 0.13	1.25 ± 0.08	0.01[b]
IVSs (cm)	1.96 ± 0.36	1.88 ± 0.47	0.33
LVIDs (cm)	2.89 ± 0.41	3.14 ± 0.42	0.15
LVFWs (cm)	1.79 ± 0.30	1.84 ± 0.18	0.3
FS (%)	33.6 ± 6.3	32.1 ± 6.1	0.30
EPSS (cm)	0.39 ± 0.11	0.46 ± 0.10	0.08

IVSd/s, interventricular septum thickness in diastole/systole; LVIDd/s, left ventricular internal dimension during diastole/systole; LVFWd/s, left ventricular free wall thickness during diastole/systole; FS, fractional shortening; EPSS, end-point to sepal separation.
[a] Mean±SD.
[b] Significant difference between groups.

(range, 5.9–8.0 g/dL). The mean indirect blood pressure was 135.7 mm Hg (range, 120–145 mm Hg).

Subjectively, there was no evidence of structural narrowing or abnormalities of the LVOT or pulmonic valve. The mean LVOT diameter was 2.46 cm (range, 2.16–2.81 cm) (Table 1).

The mean aortic velocity from the subcostal view for this group was 1.89 m/s (range, 1.6–2.0 m/s). The mean pulmonic velocity was 1.12 m/s (range, 0.82–1.46 m/s). One dog had trivial mitral regurgitation, and no dogs had aortic regurgitation.

The study and control groups were comparable with respect to age ($P = .44$) and body weight ($P = .14$); the sex distribution was also similar. The Greyhounds with murmurs had significantly higher aortic velocity (AV) than those in the control group ($P < .001$) (Fig 2). No significant differences were found in the aortic valve annulus diameter ($P = .49$), fractional shortening ($P = .3$), pulmonic velocity ($P = .07$), or blood pressure ($P = .17$) between the 2 groups. As stated above, the LVFW thickness in diastole was higher in the dogs with murmurs ($P = .001$). There was no significant difference between the 2 groups with regard to heart rate either ($P = .41$).

No significant differences were found between the Greyhounds with the murmur and those in the control group for Hct ($P = .47$), serum protein concentration ($P = .32$), or RBC count ($P = .35$). No significant correlation was observed between AV and Hct ($r =$

Fig 2. Box and whisker plots of aortic velocities (AVs) in Greyhounds and non-Greyhounds (aortic velocity expressed in m/s) ($P = .001$).

0.133), AV and platelet count ($r = -0.034$) or between AV and plasma protein concentration ($r = -0.028$).

Discussion

In this study, Greyhounds with soft, left basilar systolic murmurs had mildly (but significantly) higher aortic velocities than similar dogs without murmurs (Fig 2). In the dogs with murmurs and higher velocities, there was no evidence of a recognizable structural abnormality, such as valvular lesions or other congenital defects. Additionally, no correlation was found between the systolic murmur and the higher Hct and RBC observed in this breed.

The Reynolds number provides insight into the many factors that can contribute to the development of a heart murmur. It can be defined by (Radius × Velocity × density)/viscosity.[11] The results of this study suggest that there is no statistically significant difference in radius between the dogs with and without heart murmurs in that there was no significant difference in aortic valve annulus diameter between groups.

Blood viscosity plays a major role in the genesis of physiologic murmurs, because it is in the denominator of the equation used to calculate the Reynolds number.[12] Bodey and Rampling[3] found that whole-blood viscosity correlates well with the Hct in different breeds and that other rheologic factors, such as cellular deformability and plasma viscosity, do not differ significantly among breeds; not surprisingly, in that study, Greyhounds had the highest blood viscosity of all breeds examined.

In patients with anemia, the combination of decreased blood viscosity due to a low Hct and an increased stroke volume can result in a physiologic murmur.[11,13,14] However, because there was no difference in the Hct (which is the main determining factor in blood viscosity) between the 2 groups of dogs of our study, the murmur observed in Greyhounds is not likely to be associated with the rheologic characteristics of the breed. Paradoxically, because Greyhounds have higher blood viscosity than dogs of other breeds, it should be assumed that their likelihood of developing a murmur would be lower than in other breeds with a similar AV
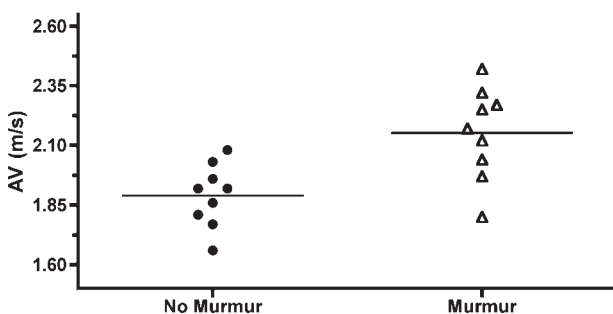
or vessel radius, because viscosity is in the denominator in the formula to calculate the Reynolds number. Therefore, other factors, such as changes in blood density, which are highly unlikely, or differences in stroke volume that could contribute to an increase in aortic velocity should be considered.

Previous studies have determined that Greyhounds have larger and heavier hearts than dogs of other breeds.[7–9,15,16] These findings have been variably attributed to work hypertrophy, genetic factors, or an increase in blood volume resulting in an increased cardiac workload. However, the heart weight:body weight ratios were the same in trained Greyhounds and in those not allowed to exercise since puppyhood, strongly suggesting a genetic component to the relative cardiomegaly in the breed.[7] On the basis of the methodology of this study, we were not able to determine if the Greyhounds with murmurs had larger hearts than those without murmurs. We did not evaluate the heart weight:body weight ratios in these patients, and although the LVFW thickness in diastole was significantly higher in the dogs with murmurs, this finding was not observed for interventricular thickness or the left ventricular posterior wall in systole.

The statistically higher LVFW thickness in diastole in the Greyhounds with murmurs is difficult to interpret. All dogs were retired from racing, and the likelihood of an athletic heart is low. The increase in thickness could be a compensatory response to increased afterload or hypertension; however, blood pressure was not statistically different between groups. Increased wall thickening could also be due to a fixed or dynamic obstruction such as aortic stenosis or systolic anterior motion of the mitral valve. None of these were identified on echocardiographic examination; however, mild subaortic stenosis may not necessarily be detected on 2-D echocardiographic evaluation.

Although Doppler echocardiography has been documented to be a reliable procedure for the diagnosis of moderate-to-severe subvalvular aortic stenosis (SAS), cardiac catheterization with direct pressure measurements and angiography remain the gold standard for the antemortem diagnosis of SAS.[17] Therefore, because of the noninvasive nature of our study, we cannot completely rule out the presence of mild SAS as the cause of the systolic murmur in this group of Greyhounds. However Greyhounds are not considered at high risk for this congenital defect.[18]

A potential limitation of this study is that the aortic valve diameter was only measured at the annulus. Subtle subvalvular or supravalvular aortic diameter changes that were not identified in the current study could potentially exist, but they were not reported in any of the previous echocardiographic or invasive cardiovascular physiology studies conducted in the breed.[6–10]

Blood viscosity was not specifically measured because of technical reasons; only the Hct and serum protein concentration were evaluated, and, as discussed above, Hct is the main determinant of blood viscosity, contributing to >90% of the viscosity measurement.[3] The data we obtained do not provide definitive evidence of whether blood viscosity contributes to the dynamics involved in murmur genesis, but as previously discussed, high viscosity should decrease the likelihood of turbulent flow in Greyhounds.

This study did not attempt to prove a direct cause-and-effect relationship between high AV and murmur generation, because that would require an invasive approach. However, based on data extrapolated from humans,[11,19] the high AV in the Greyhounds with murmurs may be comparable to the increased ventricular velocity described by Spooner et al[20] in human patients with systolic murmurs. Moreover, the data from Spooner et al correlate well with invasive studies in humans that have demonstrated that turbulent flow occurs in the aortic root when Reynolds numbers are in the range of 5,700–10,000.[20,21]

In conclusion, the results of this study suggest that although a soft (1-2/6) murmur is common in adult Greyhounds, it does not appear to be associated with an obvious congenital defect or any CBC differences unique to this breed. Further studies are required to understand the genesis of the systolic murmur in these dogs.

Footnotes

[a] Cell Dyn 3500R, Abbott Diagnostics, Abbott Park, IL
[b] Hitachi 911 Automatic Analyzer, Roche Laboratories, Indianapolis, IN
[c] GE Vivid 7 Echocardiographic System, General Electric, Horten, Norway
[d] Model 811-B Ultrasonic Doppler Flow detector, Parks Medical Electronics Inc, Aloha, OR
[e] GraphPad Prism, Graph Pad Software, San Diego, CA

Acknowledgment

References

1. Porter J, Canaday WR Jr. Hematologic values in mongrel and Greyhound dogs being screened for research use. J Am Vet Med Assoc 1971;159:1603–1606.
2. Doxey DL. Cellular changes in the blood as an aid to diagnosis. J Small Anim Pract 1966;7:77–89.
3. Bodey AR, Rampling MW. A comparative study of the haemorreology of various breeds of dog. Clin Hemorreol Microcirc 1998;18:291–298.
4. Feeman W, Couto CG, Gray TL. Serum creatinine concentrations in retired racing Greyhounds. Vet Clin Pathol 2003;32:40–42.
5. Schneider HP, Truex RC, Knowels JO. Comparative observations of the hearts of mongrel and Greyhound dogs. Anat Rec 1964;149:173–179.
6. Rippe JM, Pape LA, Alpert JS, et al. Studies of systolic mechanics and diastolic behavior of the left ventricle in the trained racing greyhound. Basic Res Cardiol 1982;77:619–644.
7. Pape LA, Price JM, Alpert JS, Rippe JM. Hemodynamics and left ventricular function: A comparison between adult racing

greyhounds and greyhounds completely untrained from birth. Basic Res Cardiol 1986;81:417–424.

8. Snyder PS, Sato T, Atkins CE. A comparison of echocardiographic indices in the nonracing, healthy Greyhound to reference values from other breeds. Vet Radiol Ultrasound 1995;36:387–392.

9. Page A, Edmunds G, Atwell RB. Echocardiographic values in Greyhounds. Aust Vet J 1993;70:361–364.

10. Lonsdale RA, Labuc RH, Robertson ID. Echocardiographic parameters in training compared with non-training Greyhounds. Vet Radiol Ultrasound 1998;39:325–330.

11. Murgo JP. Systolic ejection murmur in the era of modern cardiology. What do we really know? J Am Coll Cardiol 1998;32:1596–1602.

12. Sabbah HN, Lee TG, Sten PD. Role of blood viscosity in the production of innocent ejection murmurs. Am J Cardiol 1979;43:753–756.

13. Shapiro E. The mechanism of systolic murmur in anemia. Ann Pract Dig Treat 1951;2:128–133.

14. Da Silva DG. Systolic murmur in anemia. Hospital (Rio J) 1953;44:273–280.

15. Carew ME, Covell JW. Left ventricular function in exercise-induced hypertrophy in dogs. Am J Cardiol 1978;42:82–88.

16. Gunn HM. Heart weight and running ability. L Anat 1989;167:225–233.

17. Lehmkuhl LB, Bonagura JD, Jones DE. Comparison of catheterization and Doppler-derived pressure gradients in a canine model of subaortic stenosis. J Am Soc Echocardiogr 1995;8:611–620.

18. Buchanan JW. Prevalence of cardiovascular disorders. In: Fox PR, Sisson D, Moïse NS, eds. Textbook of Canine and Feline Cardiology: Principles and Clinical Practice, 2nd ed. Philadelphia, PA: WB Saunders; 1999:457–470.

19. Spooner PH, Perry P, Brandenburg RO, Pennock GD. Increased intraventricular velocities. An unrecognized cause of systolic murmur in adults. J Am Coll Cardiol 1998;32:1589–1595.

20. Stein PD, Sabbah NH. Turbulent blood flow in humans: its primary role in the production of ejection murmurs. Circ Res 1976;38:513–525.

21. Stein PD, Sabbah NH. Aortic origin of innocent murmurs. Am J Cardiol 1977;39:665–671.