Doppler echocardiography–derived evidence of pulmonary arterial hypertension in dogs with degenerative mitral valve disease: 86 cases (2001–2005)

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Objective—To determine the prevalence of Doppler echocardiography–derived evidence of pulmonary arterial hypertension (DEE-PAH) in dogs with mitral valve disease (MVD) classified according to the International Small Animal Cardiac Health Council (ISACHC) heart failure classification scheme and various echocardiographic and Doppler indices of MVD severity.

Design—Retrospective case series.

Animals—617 dogs examined from 2001 to 2005 with MVD in ISACHC classes I to III.

Procedures—Dogs were examined echocardiographically. Criteria used for systolic and diastolic DEE-PAH were detection of high tricuspid ($\geq$ 2.5 m/s) and telediastolic pulmonic ($\geq$ 2.0 m/s) valvular peak regurgitant jet velocities, respectively, by use of continuous-wave Doppler echocardiography.

Results—86 (13.9%) dogs with MVD had a diagnosis of DEE-PAH. Severity and prevalence of DEE-PAH increased with ISACHC class (3.0%, 16.9%, 26.7%, and 72.2% prevalences for ISACHC classes Ia, Ib, II, and III, respectively). A significant correlation between systolic or diastolic pulmonary arterial pressure and left atrial-to-aortic diameter ratio (LA/Ao) was detected. Doppler echocardiography–derived evidence of pulmonary arterial hypertension was detected in 18 dogs with values of LA/Ao within reference range, all of which had moderate (n = 2 dogs) or severe (16) mitral valve regurgitation on color Doppler imaging.

Conclusions and Clinical Relevance—The prevalence and degree of DEE-PAH were related to the severity of MVD. Changes associated with DEE-PAH may be detected in early stages of the disease, but only in dogs with severe mitral valve regurgitation. (*J Am Vet Med Assoc* 2006;229:1772–1778)

ABBREVIATIONS

PAH	Pulmonary arterial hypertension
MVD	Mitral valve disease
DEE-PAH	Doppler echocardiography–derived evidence of PAH
ISACHC	International Small Animal Cardiac Health Council
2D	Two-dimensional
LA/Ao	Left atrial-to-aortic diameter ratio
LAA	Left atrial area
ARJ	Area of the regurgitant jet signal
DPAPG	Diastolic pulmonary artery-to-right ventricle pressure gradient
SPAP	Systolic pulmonary arterial pressure

Pulmonary arterial hypertension is defined as high diastolic or systolic pulmonary arterial pressure and may lead to right ventricular concentric or eccentric hypertrophy, right atrial enlargement, and right-sided heart failure.[1,2] In humans, PAH is a well-known condition that occurs sporadically as an idiopathic disorder (primary PAH) but is more commonly a complication of other diseases (secondary PAH).[2,3] Primary PAH has been described only rarely in dogs.[4,5] In dogs, heartworm disease is one of the most well-known causes of secondary PAH.[6] However, findings from 1 study[7] indicated that PAH could also be a complication of other common respiratory tract and cardiac diseases, suggesting that PAH in dogs is more common than has been supposed.

Degenerative MVD is characterized by valvular degeneration and is the most common acquired cardiac disease in small-breed dogs.[8-11] The condition results in mitral valve insufficiency, which may lead to left atrial and ventricular dilatation and, eventually, to development of pulmonary edema. Pulmonary arterial hypertension is typically described as a late complication of MVD that leads to failure of the right side of the heart. However, the true prevalence of PAH associated with MVD in dogs is unknown, and screening dogs with MVD for PAH may have prognostic and therapeutic implications.[12,13]

The aims of the present study were to retrospectively assess the prevalence of DEE-PAH in dogs with MVD and determine whether prevalence depends on the clinical class of the disease (ie, ISACHC classification[14]). We also undertook to determine the correlation between pulmonary arterial pressure and certain Doppler echocardiographic indices that reflect the severity of valvular disease (eg, size of regurgitation jets and left atrium).

Criteria for Selection of Cases

Medical records of 636 dogs that underwent complete echocardiographic and Doppler examination

from January 2001 through December 2005 at the Cardiology Unit of the veterinary school at Alfort and in which a diagnosis of MVD (treated or not) was made by a trained observer (VC, APN, VG, FJS, or CCS) were retrospectively reviewed. Records of dogs with a diagnosis of systolic DEE-PAH, diastolic DEE-PAH, or both were included in the study. Thoracic radiographs were evaluated by a board-certified specialist. Dogs with MVD were excluded from the study if evidence of concurrent respiratory tract disease was detected on thoracic radiographs. Dogs with clinical signs of MVD were included only if thoracic radiographs had been obtained and there was radiographic evidence of congestive heart failure (eg, pulmonary edema and vascular congestion) but no abnormalities compatible with respiratory tract disease. Dogs with no clinical signs were included in the study, whether radiographic examination was performed or not. Obese dogs were excluded from the study. The degree of heart failure was classified according to ISACHC recommendations (**Appendix**).[14]

Procedures

Diagnosis of MVD—The diagnosis of MVD was made on the basis of the following criteria: left-sided systolic apical murmur of late appearance (> 1 year of age), no history of infectious disease, and echocardiographic and Doppler evidence of MVD (eg, irregular and thick mitral valve leaflets observed on the right parasternal 4-chamber view and a color-flow regurgitation jet indicating systolic mitral valve insufficiency in the left atrium on the left parasternal 4-chamber view).

Diagnosis of DEE-PAH—All ultrasonographic examinations included color Doppler analysis of tricuspid and pulmonary valvular flow for detection of regurgitant jets. When a regurgitant jet was detected, maximal regurgitant flow velocity was determined by use of continuous Doppler mode and only high-velocity values (ie, ≥ 2.5 m/s and ≥ 2.0 m/s for tricuspid and pulmonic valve regurgitant jets, respectively) were recorded. Detection of high tricuspid valve (≥ 2.5 m/s in the absence of pulmonic valve stenosis) or telediastolic pulmonic valve (≥ 2.0 m/s) regurgitant jet velocities with continuous-wave Doppler imaging was considered to be diagnostic for systolic or diastolic DEE-PAH, respectively. These 2 velocity thresholds correspond to high pulmonary arterial pressures (reference ranges,[1] 15 to 25 mm Hg and 5 to 10 mm Hg during systole and diastole, respectively).

Echocardiographic and Doppler examinations—Echocardiographic and Doppler examinations were performed in standing awake dogs undergoing continuous ECG monitoring with ultrasound units[a] equipped with 7.5- to 10-, 5- to 7.5-, and 2- to 5-MHz phased-array transducers according to described and validated methods.[15]

Ventricular measurements were obtained from the right parasternal location during 2D-guided M-mode echocardiography according to recommendations of the American Society of Echocardiography[16]; measurements were compared with reference range values.[17] Measurements of the aorta and left atrium were obtained with a 2D method, and LA/Ao was calculated and compared with reference range values according to a described technique.[18] The left and right end-diastolic atrial diameters were compared at the level of the atrioventricular valves in the 2D right parasternal 4-chamber view. The right atrium was considered to be dilated if the diameter was greater than or equal to the diameter of the left atrium.[17] In dogs with left atrial dilatation, right atrial chamber size was assessed subjectively according to criteria such as abnormal degree of sphericity.

Quantification of mitral valve regurgitation in dogs with MVD—Mitral valve regurgitation was assessed semiquantitatively by measuring the size of the systolic color-flow jet originating from the mitral valve and spreading into the left atrium in the left apical 4-chamber view. By use of a previously described technique,[19] images were analyzed frame by frame to compute the maximum area of the regurgitant jet signal. The LAA was measured via computerized planimetry in the same frame in which the maximum ARJ was seen, and the ARJ/LAA ratio was calculated. By means of this Doppler method, color-flow signals revealed mild (ARJ/LAA < 30%), moderate (30% ≤ ARJ/LAA ≤ 70%), or severe (ARJ/LAA > 70%) mitral valve regurgitation. This method yields a quantitative assessment of the severity of mitral valve regurgitation, which correlates with Doppler regurgitant volume and effective regurgitant orifice area.[19] To limit intra- and interobserver variation, all observers were trained for at least 2 years in Doppler imaging technique and the same ultrasound machines with the same settings were used in all dogs. To confirm the reliability of the technique used, a validation protocol[b] involving the observer who performed the most examinations in the study (VC) was performed concurrently with the reported work. Intra- and interday variation in color Doppler variable measurements was determined by performing 36 Doppler examinations in 6 nontreated dogs that had MVD but no clinical signs on 4 days during a 2-week period. Those dogs included 3 Poodles, 1 Beagle, 1 Yorkshire Terrier, and 1 Shih Tzu. Mean ± SD age of those dogs was 11.5 ± 2.4 years (range, 9 to 15 years), and mean ± SD weight was 7.6 ± 4.0 kg (16.7 ± 8.8 lb), with a range of 3.1 to 14.4 kg (6.82 to 31.7 lb). On a given day, 3 dogs were examined 3 nonconsecutive times. Each variable was measured 3 times during 3 consecutive cardiac cycles by use of the same frame; mean values were used to determine within- and between-day variation by means of a general linear model. The within- and between-day coefficients of variation of ARJ/LAA were < 10% (7.1% and 8.2%, respectively), confirming that repeatability and reproducibility of the Doppler technique used to assess mitral valve regurgitation were high.

Doppler evaluation of PAH—Doppler echocardiography–derived evidence of systolic and diastolic PAH was determined via described techniques.[7,20] Color-flow Doppler examination of the right ventricular outflow tract obtained from the right parasternal short axis view was used to detect diastolic pulmonic valve regurgitant flow, which was assessed quantitatively with

continuous-wave Doppler examination. A peak telediastolic pulmonic valve regurgitant flow velocity ≥ 2m/s was considered to be indicative of diastolic DEE-PAH. The maximal regurgitant flow velocity measured was incorporated into the modified Bernoulli equation ($\Delta P = 4 \times \text{velocity}^2$) to calculate the DPAPG across the pulmonic valve.

Color-flow Doppler examination of the tricuspid valve was performed by use of the left apical 4-chamber view. When present, systolic tricuspid valve insufficiency was confirmed and quantitatively assessed in continuous-wave Doppler mode. A peak tricuspid valve regurgitant flow velocity ≥ 2.5 m/s was considered to be indicative of systolic DEE-PAH. Application of the modified Bernoulli equation to the maximal velocity of tricuspid valve regurgitant flow was used to calculate the systolic pressure gradient across the tricuspid valve.[20] Systolic pulmonary arterial pressure was calculated by adding the estimated right atrial pressure to the systolic right ventricle-to-right atrium pressure gradient. The estimated right atrial pressure was 5 mm Hg in dogs with a nonenlarged right atrium, 10 mm Hg in dogs with an enlarged right atrium but no right-sided heart failure, and 15 mm Hg in dogs with right-sided heart failure.[1] Dogs were categorized as having mild (SPAP = 30 to 50 mm Hg), moderate (SPAP = 51 to 75 mm Hg), or severe (SPAP > 75 mm Hg) systolic DEE-PAH. Dogs with no detectable pulmonic or tricuspid valve regurgitant flow were considered to be free of systolic or diastolic DEE-PAH or to have PAH that could not be estimated noninvasively.

Statistical analysis—Data were expressed as mean ± SD or percentages. The Pearson product moment correlation was used to detect correlations between SPAP or DPAPG and LA/Ao. A 1-way ANOVA, followed by a Student *t*-test with Bonferroni correction if necessary, was used to compare SPAP and DPAPG among dogs in the 3 ISACHC classes. For all comparisons, *P* < 0.05 was considered significant.

Results

Prevalence and characteristics of DEE-PAH in dogs with MVD—From January 2001 to December 2005, MVD was diagnosed in 636 dogs, 105 of which had DEE-PAH. The other 531 dogs had either no pulmonic or tricuspid regurgitant flow jet or low-velocity regurgitant jets. Of the 105 dogs with DEE-PAH, 19 were excluded because of concurrent respiratory tract

disease (tracheal collapse [n = 7]); tracheal collapse and overweight (2); chronic bronchopneumopathy (7); chronic bronchopneumopathy and overweight (1); and suspected pulmonary thromboembolism (2) that could have contributed to development of PAH. Therefore, only 86 dogs with DEE-PAH of the 617 with MVD (13.9%) were used in statistical analyses. Thoracic radiographs were available for 70 of the 86 dogs (81.4%) with DEE-PAH, including dogs with (n = 61) and without (9) clinical signs. No evidence of bronchointerstitial, alveolar, or tracheal disease was detected.

Treatment at the time of diagnosis was known for 82 of the 86 dogs with DEE-PAH. Fifty-five of those 82 dogs received ≥ 1 treatment for heart failure, including angiotensin-converting enzyme inhibitors such as benazepril, enalapril, imidapril, and ramipril (n = 49 dogs [89.1%]); furosemide (25 [45.5%]); spironolactone (18 [32.7%]); pimobendan (8 [14.5%]); and digoxin (4 [7.3%]).

Among the 86 dogs with DEE-PAH, both diastolic and systolic DEE-PAH were detected in 15 dogs. In 62 of the 86 dogs, systolic DEE-PAH was confirmed but the absence of diastolic pulmonic valve regurgitant flow did not permit assessment of DPAPG. Similarly, in 9 of the 86 dogs, diastolic DEE-PAH was confirmed, but the absence of systolic tricuspid valve regurgitant flow did not permit assessment of SPAP. In the 77 dogs with systolic DEE-PAH, mean ± SD SPAP was 56.0 ± 21.4 mm Hg (range, 30 to 123 mm Hg). Systolic DEE-PAH was considered to be mild in 43 of the 77 dogs (55.8%; mean ± SD SPAP, 40.6 ± 5.8 mm Hg), moderate in 18 dogs (23.4%; SPAP, 62.0 ± 8.9 mm Hg), and severe in 16 dogs (20.8%; SPAP, 90.4 ± 12.0 mm Hg). In the 24 dogs with diastolic DEE-PAH, mean DPAPG was 21.4 ± 5.8 mm Hg (range, 16 to 35 mm Hg).

Characteristics of dogs with DEE-PAH—As expected, this group was composed primarily of aged (11.3 ± 2.3 years; range, 4 to 16 years) male (n = 61 [70.9%]) small-breed dogs weighing < 10 kg (< 22 lb; n = 72 [83.8%]). Only 10 (11.6%) and 4 (4.7%) of the 86 dogs with DEE-PAH were of medium-sized (10 to 25 kg [22 to 55 lb]) or large breeds (> 25 kg), respectively.

Clinical findings in dogs with DEE-PAH—Twenty-one of the 86 (24.4%) dogs with DEE-PAH had no clinical signs and were classified as ISACHC class Ia (**Tables 1 and 2**). Four other dogs had chronic cough-

Table 1—Number (%) of dogs with various clinical and echocardiographic findings among 86 dogs with MVD and DEE-PAH.

Variable	No. of dogs (%)
Clinical signs	
No cardiorespiratory clinical signs	21 (24.4)
Cough	43 (50.0)
Exercise intolerance	13 (15.1)
Dyspnea	21 (24.4)
Ascites	12 (14.0)
Syncope	6 (7.0)
Echographic findings	
Right atrial and/or ventricular enlargement	28 (32.6)
Pleural effusion	2 (2.3)
Pericardial effusion	10 (11.6)
Ascites	12 (14.0)
No echocardiographic evidence of PAH	58 (67.4)

Table 2—Prevalence (No. [%]) of PAH and values of SPAP and DPAPG in 617 dogs with MVD by ISACHC heart failure classification and severity of mitral valve regurgitation.

| Variable | No. of dogs | Dogs with DEE-PAH | |
		No. (%)	SPAP/DPAPG (mean ± SD mm Hg)
ISACHC class			
Ia	367	11 (3.0)	46 ± 17.8/16*
Ib	83	14 (16.9)	56.5 ± 20.0/21.2 ± 6.0
II	131	35 (26.7)	52.4 ± 19.9/20.8 ± 4.9
III	36	26 (72.2)	65.0 ± 22.6†/22.7 ± 6.9
Mitral valve regurgitant jet size			
Mild (ARJ/LAA < 30%)	238	0	NE/NE
Moderate (30% ≤ ARJ/LAA ≤ 70%)	102	2 (1.3)	41.5 ± 5.0 NE
Severe (ARJ/LAA > 70%)	277	84 (19.9)	56.3 ± 21.5 21.4 ± 5.8

*Diastolic DEE-PAH was detected in only 2 dogs and was 16 mm Hg in each dog. †Significantly ($P < 0.05$) different from values in ISACHC classes Ia, Ib, and II.
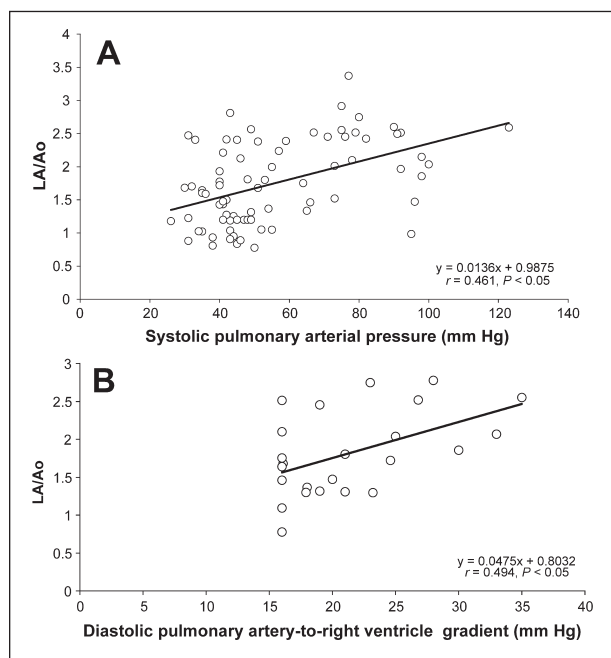NE = Not evaluated.

Figure 1—Correlation between LA/Ao and SPAP in 77 dogs with systolic DEE-PAH (A) and between LA/Ao and DPAPG in 24 dogs with diastolic DEE-PAH (B) among 86 dogs with DEE-PAH and mitral valve disease.

ing (considered to be secondary to left mainstem bronchial compression by the enlarged left atrium on the basis of radiographic findings) and were categorized in ISACHC class Ib. The remaining 61 dogs had clinical signs and were categorized in ISACHC classes II or III. A chronic cough (> 1 month in duration) was the most common clinical sign (39/61 [63.9%]) in those dogs.

A significant ($P < 0.01$) increase in prevalence of DEE-PAH was observed with increasing ISACHC class: prevalences of 3.0%, 16.9%, 26.7%, and 72.2% were detected for ISACHC classes Ia, Ib, II, and III, respectively (Table 2). Similarly, SPAP was significantly ($P < 0.05$) higher in the ISACHC class III group than in dogs

in other ISACHC classes. Values of DPAPG among the 4 ISACHC classes were not significantly different.

Echocardiographic and Doppler examinations— In dogs with DEE-PAH, the most common echocardiographic abnormalities attributable to PAH were dilatation of the right heart chambers (28/86 [32.6%]) and pericardial, pleural, or peritoneal effusion (24/86 [27.9%]; Table 1). A significant ($P < 0.05$) positive correlation was found between left atrial size (assessed by the LA/Ao ratio) and both SPAP ($r = 0.46$) and DPAPG ($r = 0.49$; Figure 1). Among the 617 dogs with MVD, none with a mild regurgitant jet size (n = 238) had DEE-PAH (Table 2). Conversely, most dogs with DEE-PAH (84/86 [97.7%]) had severe mitral valve regurgitation, characterized by an ARJ/LAA value > 70% (mean ± SD, 100 ± 7.8%). The other 2 dogs had moderate ARJ/LAA values (55% and 60%). Interestingly, DEE-PAH was identified in 18 dogs with LA/Ao in reference range (1.0 ± 0.14; range, 0.73 to 1.2); all of those dogs had moderate (2/18 [11.1%]) or severe (16/18 [88.9%]) mitral valve regurgitation (ARJ/LAA, 80 ± 17%; range, 55% to 100%). Mean ± SD SPAP in those 18 dogs was 44.6 ± 14.4 mm Hg (range, 30 to 95 mm Hg), and diastolic DEE-PAH was detected in 2 of those dogs (DPAPG, 16 mm Hg for each).

Discussion

For the last 5 years, color-flow continuous- and pulsed-wave Doppler-mode imaging has routinely been performed during all echocardiographic examinations at the Cardiology Unit of the National Veterinary School of Alfort. We reviewed this cardiology database to analyze the prevalence and characteristics of DEE-PAH in 617 dogs with MVD. To our knowledge, this is the first report in which DEE-PAH was extensively investigated in a large population of dogs with MVD or in which the influences of heart failure score and valvular dysfunction on DEE-PAH prevalence and severity were analyzed.

Compared with other species, the dog has low pulmonary vascular resistance and reactivity.[21] However, results of the present study indicated that PAH in dogs

is more common than has previously been supposed: DEE-PAH was detected in nearly 15% of 617 dogs with MVD, and the disease was advanced (ISACHC class III) in > 70% of those dogs.

Pulmonary artery pressures can be accurately assessed by means of catheterization of the chambers on the right side of the heart. The chief limitations associated with this technique are high cost, lack of availability, technical expertise, and requirement for general anesthesia. With advances in Doppler echocardiography, reliable information about pulmonary arterial pressure can now be obtained noninvasively in humans and conscious dogs.[20,22,23] In the past decade, the frequency with which DEE-PAH has been diagnosed in humans has increased as a result of increased availability of Doppler echocardiography and awareness of the condition. Pulmonary arterial hypertension is a well-known and important complication of numerous human cardiorespiratory and systemic diseases.[2,3,24] Severe chronic PAH is an independent predictor of death[12] and can be difficult to treat successfully.[13] In veterinary medicine, the most studied cause of PAH is heartworm disease[6]; few reports have focused on the importance of other causes.[7] For example, only 22 cases of DEE-PAH unrelated to heartworm disease were reported in a retrospective study[25] that included data from a 10-year period at the University of Illinois veterinary teaching hospital. Results of a more recent study[7] suggested that, as is true in humans, DEE-PAH can develop in dogs as a complication of common cardiopulmonary diseases; in that study, heartworm disease accounted for < 10% and MVD accounted for > 30% of 53 dogs with DEE-PAH.

The present study also revealed that DEE-PAH may develop in early stages of MVD. Nearly a third of the dogs with DEE-PAH had ISACHC class I disease. Humans with PAH may have few clinical signs until the severity of disease becomes life-threatening, prompting physicians to screen patients for PAH in earlier stages of predisposing diseases.[24] Most (67.4%) of the dogs with DEE-PAH in the present study did not have right-sided heart enlargement on echocardiographic examination. The absence of change in the right heart chambers was not surprising because most (55.8%) dogs had mild DEE-PAH. These findings indicated that the absence of clinical signs of right-sided heart dilatation in a dog with MVD does not necessarily rule out PAH and also confirmed the value of screening for PAH with Doppler echocardiography in dogs with MVD.

The prevalence and severity of DEE-PAH increased significantly with progression of MVD in dogs of the present study, as indicated by the significant increase in prevalence with clinical class and higher SPAPs in dogs in the end stages of the disease (ISACHC class 3). Similarly, Doppler echocardiography–derived data indicated that the greater the incompetence of the mitral valve, the more common and severe the PAH. None of the 218 dogs with MVD that had a mild regurgitant jet had DEE-PAH. The minimum ARJ/LAA value measured in the 86 dogs with DEE-PAH was 55%, and nearly all (97.7%) of those dogs had severe mitral valve regurgi-

tation, characterized by values of ARJ/LAA > 70%. Moreover, LA/Ao, which indirectly reflects the degree of mitral valve dysfunction, was significantly correlated with both SPAP and DPAPG.

Although a significant correlation was observed between the degree of left atrial dilatation and SPAP or DPAPG in dogs with DEE-PAH, PAH was also identified in 18 dogs with MVD in which LA/Ao was within reference range. In that group of dogs, PAH was considered mild (mean SPAP, 45 mm Hg). All of those dogs with PAH had clinically relevant mitral valve regurgitation (mean ARJ/LAA, 80%; minimum ARJ/LAA, 55%). Whether dogs with mild PAH are prone to developing moderate to severe PAH and right-sided heart failure should be investigated in future prospective studies because such findings would call into question the benefit of early treatment in those dogs.

As a retrospective study, this report had several limitations. Aged small-breed dogs are predisposed to development of bronchointerstitial pulmonary disease, which may be undetectable because the condition is clinically silent or because changes are masked by pulmonary edema on thoracic radiographs. Even when no abnormal findings are detected radiographically, mild pulmonary disease or mild tracheal collapse cannot definitively be ruled out if radiographic examination is not performed, and both of those conditions can contribute to high pulmonary arterial pressure. Moreover, the influence of mild obesity, a common finding in aged small-breed dogs, could not be thoroughly evaluated because of the difficulty in assessing this factor retrospectively. Because of these limitations involving weight and clinically or radiographically undetectable respiratory tract disease, a cause-and-effect relationship between MVD and PAH could not be confirmed from our results. A relationship is suspected, however, on the basis of the increased prevalence and severity of PAH with progression of MVD.

Technical limitations also affected the study. First, pulmonic or tricuspid valve regurgitant flow is not always detected in dogs with PAH; hence, the true prevalence of the disease may be underestimated.[26,27,c] This may explain the fact that DEE-PAH was not detected in all dogs with ISACHC class III disease. A simultaneous increase in pulmonary arterial pressure would be expected in dogs with marked volume and pressure overload of the left atrium, as that change represents a necessary adaptation to the pathologic condition.[1] Our study focused on DEE-PAH, and the sensitivity of Doppler examination in detecting PAH has yet to be determined. It is probable that some of the 531 dogs with MVD but not DEE-PAH had PAH that could not be detected because of an absence of measurable pulmonic and tricuspid valve regurgitant flow. The prevalence of DEE-PAH in dogs with MVD likely underestimates the actual prevalence of PAH. Second, estimation of right atrial pressure may represent another limitation of Doppler estimation of SPAP. In dogs with left atrial dilatation, right atrial dilatation was subjectively assessed and the assessment depended on the observer's experience. However, because the estimated right atrial pressure ranged

from 5 to 10 mm Hg (in the absence of right heart failure) by use of this method, the importance of this bias was considered limited. In humans, right atrial pressure estimation may also be made on the basis of the degree of collapse of the inferior vena cava during inspiration.[28] Lastly, SPAP and DPAPG in dogs with DEE-PAH may have been underestimated because of concurrent treatment with diuretics or angiotensin-converting enzyme inhibitors. The efficacy of treatment with the latter has been proven in a canine model of percutaneously induced mitral valve regurgitation.[29] In that model, mean pulmonary arterial pressure remained within reference range in dogs treated with ramipril but increased significantly in untreated dogs.

Doppler echocardiograpic evidence of PAH is not an uncommon finding in dogs with MVD and is more frequently detected in dogs with severe mitral valve regurgitation. Although the prevalence and severity of PAH are closely related to progression of MVD, PAH may be detected in early stages of the disease, even in dogs without clinical signs of the condition. Whether or not detection of DEE-PAH at any time during the course of MVD is associated with an increased mortality rate should be investigated in future studies. The impact of treatment for PAH at all stages of valvular disease should also be determined. Prospective studies should be undertaken to determine whether early treatment of dogs with MVD that are at risk for or that have already developed PAH may aid veterinarians' ability to prevent progression of MVD, improve clinical outcome, and prolong life.

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Appendix

International Small Animal Cardiac Health Council scheme used to classify 617 dogs with MVD.

ISACHC class	Clinical characteristics	Other characteristics
Ia	Detectable heart disease (murmur, arrhythmia, or cardiac enlargement) without clinical signs of heart failure	No clinical signs of compensation
Ib	Detectable heart disease (murmur, arrhythmia, or cardiac enlargement) without clinical signs of heart failure	Radiographic or echocardiographic evidence of decompensation detected
II	Clinical signs of heart failure are evident at rest or with mild exercise and range from exercise intolerance to mild dyspnea or ascites	Home treatment is indicated
III	Clinical signs of heart failure are immediately obvious and range from severe ascites and dyspnea to cardiogenic shock	Home treatment or hospitalization is necessary

Selected abstract for *JAVMA* readers from the American Journal of Veterinary Research

Evaluation of retinal images for identifying individual dogs

Juliet R. Gionfriddo et al

AJVR

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Objective—To determine whether vessels in the ocular fundus changed over the lifetime of Beagles and whether any changes were substantial enough to likely preclude positive identification of individual dogs by use of their retinal vascular patterns.

Animals—18 Beagles.

Procedures—Fundic photographs of both eyes of 18 Beagles taken at 1 or 3, 5, and 7 or 9 years of age were digitalized. Photographs were analyzed by use of 2 software programs. One was used to determine vessel numbers and widths and the other to determine the locations of the 3 largest vessels. Measurements were compared over time periods in the life of each dog. Only observations made at baseline (1 or 3 years of age) and again at 5 and 9 years of age were included in the statistical analysis, as these points were common to all dogs.

Results—No significant changes in numbers or locations of the blood vessels were detected over time. Widths of the vessels decreased significantly as the dogs aged.

Conclusions and Clinical Relevance—The ocular fundus of Beagles changed over each dog's lifetime in that the retinal blood vessels became smaller but did not change in number or location. Results suggest that digitalized retinal images can likely be used to identify dogs over their lifetimes. (*Am J Vet Res* 2006;67:204–207)
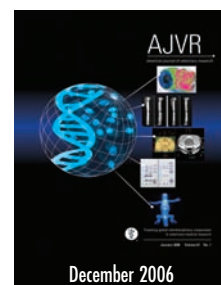
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