

# Canine dilated cardiomyopathy: a retrospective study of signalment, presentation and clinical findings in 369 cases

**OBJECTIVE:** To review the clinical and diagnostic findings and survival of dilated cardiomyopathy from a large population of dogs in England.

**METHODS:** A retrospective study of the case records of dogs with dilated cardiomyopathy collected between January 1993 and May 2006.

**RESULTS:** There were 369 dogs with dilated cardiomyopathy of which all were pure-bred dogs except for four. The most commonly affected breeds were dobermanns and boxers. Over 95 per cent of dogs weighed more than 15 kg and 73 per cent were male. The median duration of signs before referral was three weeks with 65 per cent presenting in stage 3 heart failure. The most common signs were breathlessness (67 per cent) and coughing (64 per cent). The majority of dogs (89 per cent) had an arrhythmia at presentation and 74 per cent of dogs had radiographic signs of pulmonary oedema or pleural effusion. The median survival time was 19 weeks.

**CLINICAL SIGNIFICANCE:** Dilated cardiomyopathy occurs primarily in medium to large breed pure-bred dogs, and males are more frequently affected than females. The duration of clinical signs before referral is often short and the survival times are poor. Greater awareness of affected breeds, clinical signs and diagnostic findings may help in early recognition of this disease which often has a short clinical phase.

M. W. S. MARTIN, M. J. STAFFORD  
JOHNSON AND B. CELONA\*

*Journal of Small Animal Practice* (2009)  
50, 23–29  
DOI: 10.1111/j.1748-5827.2008.00659.x

Veterinary Cardiorespiratory Centre, Thera House, Waverley Road, Kenilworth CV8 1JL

\*Via G. Arico 3, Messina, Sicily, Italia

## INTRODUCTION

Dilated cardiomyopathy (DCM) is a well-recognised cause of heart failure in dogs. With the increasing availability of ultrasound in general practice, the ability to diagnose DCM is increasing. The clinical signs and presentations are variable and usually the prognosis is grave. In practice, diagnosis is made on the basis of clinical findings and echocardiographic examination as opposed to histopathology following post-mortem, which is now rarely

permitted by owners. There is a long pre-clinical phase in dogs with DCM and those that present with clinical signs likely represent a small portion of the total number of dogs in the population that have DCM. In clinical practice, and except in breed screening schemes, dogs with DCM will generally be diagnosed when they develop clinical signs or present with a clinical abnormality on examination.

DCM affects a wide range of medium to large sized pure-bred dogs; mixed and small breed dogs are uncommonly affected. The most commonly affected breeds vary between publications and represent populations from different countries; there have been no publications representative of DCM in dogs from England. The last retrospective study of a significant number of dogs (189) was in 1997 (Tidholm and others 1997) from Sweden. Most other publications have been breed specific: cocker spaniels (Gooding and others 1982), Newfoundlands (Tidholm and Jonsson 1996), great Danes (Meurs and others 2001), boxers (Baumwart and others 2005), Portuguese water dogs (Sleeper and others 2002) and dobermanns (Calvert and others 1982).

The purpose of this study was therefore to review the findings of dogs with clinical DCM in England and augment our currently limited publications of this important disease in dogs. The medications used and prognostic factors are the subject of a subsequent paper.

## MATERIALS AND METHODS

Case records of dogs referred to the Veterinary Cardiorespiratory Centre, Kenilworth, England, were searched retrospectively, between January 1993 and May 2006, for a diagnosis of DCM. Variables for clinical presentation and diagnostic tests (electrocardiography, radiography, Doppler echocardiography and

clinical pathology) were recorded. Dogs were also categorised into severity of heart failure based on the recommendations of the International Small Animal Cardiac Health Council (ISACHC) (1999). The following clinical pathology variables were recorded in the database: haematocrit and serum concentrations of total proteins, urea, creatinine, sodium and potassium.

All dogs with a diagnosis of DCM, as described below, were recorded. Only dogs that were referred with clinical signs or a clinical abnormality were included in the database, and dogs identified as having preclinical DCM, such as for breed screening, were excluded. The diagnosis was primarily made on Doppler echocardiographic examination in conjunction with the clinical presentation, breed, thoracic radiographic (dorsoventral and right lateral views) and electrocardiographic (standard six limb leads) findings. The echocardiographic diagnosis was made using the accepted criteria of a dilated left ventricle with reduced contractility of the left ventricular (LV) chamber (Kittleson 1998, Sisson and others 1999). LV measurements were compared with published normal values for specific breeds in the first instance. For breeds in which there were no such publications, the results were compared with those from breeds of a similar size and weight and/or bodyweight tables (Boon 1998). LV diameter in diastole and systole were retrospectively indexed to bodyweight using the method described by Cornell and others (2004) to determine the usefulness of this indexing method. The indices for LV diastolic and systolic M-mode diameters are given by  $LVDd/(BW^{0.294})$  and the  $LVDs/(BW^{0.315})$ , and these were compared with the 97.5 per cent confidence intervals given for the index in the Cornell document ( $LVDd=1.27$  to  $1.85$ ;  $LVDs=0.71$  to  $1.26$ ).

Dogs were excluded if there was gross left atrial (LA) dilation (that is significantly larger than the diameter of the left ventricle in diastole) that could infer primary mitral valve disease (Kittleson 1998), a fractional shortening greater than 25 per cent, suspicion of myocarditis; if there was a tachycardia-induced myocardial failure that could mimic the phenotype of DCM; if there was congenital disease or if there was

another acquired disease. Boxer dogs with arrhythmogenic right ventricular cardiomyopathy (ARVC) that did not meet the inclusion criteria for DCM were not included in this study.

Assessment of cardiomegaly on thoracic radiography was based on a combination of subjective experience and the use of the vertebral heart scale system (Buchanan and Bucheler 1995).

The cause of death was noted as died or euthanased related to heart disease or non-cardiac-related death/euthanasia. All deaths were assumed to be cardiac related, unless they were clearly known to be non-cardiac. The survival time was counted to the nearest week from the date of referral. Overall survival time (to death or euthanasia because of cardiac reasons) was compared using the Kaplan-Meier method (Procedure lifetest or phreg, SAS Online Doc Version 8; SAS Institute Inc., 1999). Cases which died or were euthanased for non-cardiac reasons, or which were lost to follow-up, were included in the survival analysis up until the last time point at which they were alive and then were thereafter censored in the analysis. Basic descriptive statistics (mean and median) were performed using Microsoft Excel.

## RESULTS

A total of 369 dogs met the criteria for DCM. There were 35 different breeds of which nearly all were pure-bred dogs, except for four crossbreed dogs. The most commonly presented breeds (Table 1) were dobermanns (59), boxers (53), great Danes (38), cocker spaniels (30), German shepherd dogs (GSDs) (24), Saint Bernards (20) and Labradors (20). The median bodyweight (interquartile range) was 37 kg (29 to 57), ranging from a 8 kg border collie puppy to a 87 kg great Dane  $\times$  mastiff. Only 18 dogs (4.7 per cent) weighed less than 15 kg.

Seventy-three per cent (281) of dogs were male and 27 per cent were female giving a ratio of 2.7:1 (male:female). The neutered status of the dogs was not recorded in the database. For the most common breeds ( $n > 14$  dogs), there was some variation in the male:female ratio (Fig 1), for example great Danes and GSDs had a ratio of

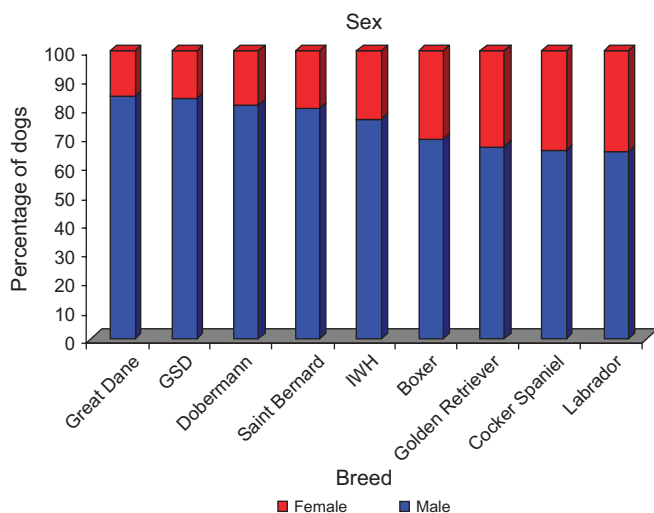
**Table 1. The number of each of the breeds of dog diagnosed with dilated cardiomyopathy**

Breed of dog	Number
Dobermann	59
Boxer	53
Great Dane	38
Cocker spaniel	30
German shepherd dog	24
Saint Bernard	20
Labrador	20
Irish wolfhound	17
Golden retriever	15
Newfoundland	14
Old English sheepdog	9
Weimaraner	6
Deerhound	6
Rottweiler	5
Flat-coated retriever	5
English bull terrier	5
Bull mastiff	5
Dalmatian	4
Springer spaniel	4
Staffordshire bull terrier	3
Border collie	3
Gordon setter	3
Neapolitan mastiff	3
Dogue de Bordeaux	3
Crossbreed	3
Cavalier King Charles spaniels	2
Leonberger	2
Belgian shepherd, border terrier, Pyrenean mountain dog, red setter, Bouvier des Flandres, standard schnauzer, standard poodle, saluki, mixed breed	1

approximately 6:1, dobermanns and Saint Bernards had ratios of approximately 5:1 and boxers, golden retrievers, cocker spaniels and Labradors had ratios of approximately 3:1.

The median age at presentation (interquartile range) was 80 months (57.5 to 102.5) and ranged from a three month old border collie to a 14.8-year-old cocker spaniel. The majority of dobermanns presented between five and 10 years of age with a peak incidence at seven years (Fig 2). Boxers had bimodal peaks at six and nine years of age (Fig 2). There were no obvious age patterns with the other commonly presented breeds ( $n > 14$  dogs).

The median (interquartile range) duration of signs before referral was three weeks (one to six weeks). Eight per cent of dogs (30) were in stage 1 heart failure (ISACHC), 27 per cent (99) in stage 2 and 65 per cent (238) in stage 3 heart failure. The most common presenting signs were breathlessness (67 per cent), cough



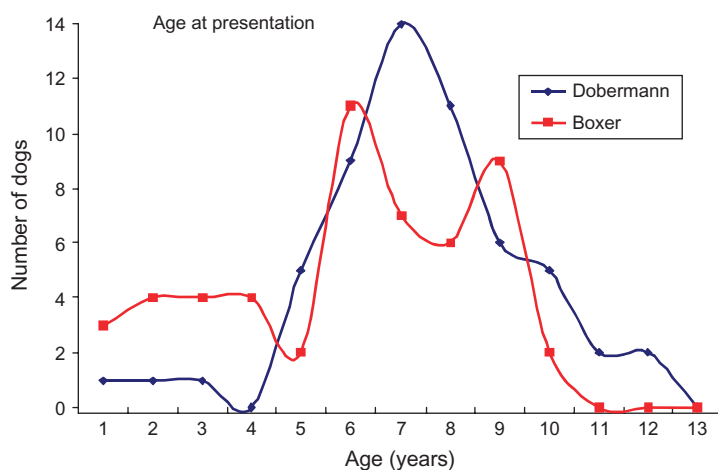
**FIG 1.** Bar chart showing the ratio of male compared with female dogs for the most commonly seen breeds (n>14 dogs included)

(64 per cent), exercise intolerance (48.8 per cent), weakness (39.2 per cent), reduced appetite (35.7 per cent), weight loss (30.2 per cent), collapse (26 per cent), dullness and lethargy (20 per cent). There were some differences in the clinical presentation between breeds, for example in boxers collapse (70 per cent) was the most common presentation, in golden retrievers reduced appetite (60 per cent) and in great Danes weakness (55.3 per cent) was the most common. However, in many breeds, coughing and breathlessness were the dominant clinical signs at presentation. Exercise intolerance was common in GSDs (61 per cent), great Danes (58 per cent), Saint Bernards (58 per cent) and Irish wolfhounds (IWH) (53 per cent), and weakness was

common in great Danes (58 per cent), Saint Bernards (52 per cent) and GSDs (48 per cent).

The most common clinical findings were weak pulse (39 per cent), murmur (33 per cent), mucosal pallor (16 per cent), ascites (15 per cent) and a gallop sound (10 per cent). Of the dogs with a murmur recorded (n=120), 56 per cent were grade 1, 15 per cent were grade 2, 24 per cent were grade 3 and 5 per cent were grade 4.

Electrocardiograms were recorded in 349 dogs (94.6 per cent). The mean heart rate (sd) was 175 per minute (44). The mean heart rate (range) tended to increase with class of heart failure: ISACHC 1=152/minute (90 to 200), ISACHC 2=163/minute (75 to 300) and ISACHC 3=176/min-



**FIG 2.** Graph showing the distribution for age at presentation for dobermanns and boxers

ute (100 to 290). Atrial fibrillation (AF) was present in 45 per cent, ventricular premature complexes (VPCs) in 31 per cent and supraventricular premature complexes (SVPCs) in 9 per cent of dogs. AF was most common in the giant and large breed dogs: IWH (94.1 per cent), great Dane (78.9 per cent), Newfoundland (77 per cent), GSD (73 per cent) and Saint Bernard (72 per cent) and least common in cocker spaniels (8 per cent). VPCs were most common in boxers (52.8 per cent) and dobermanns (44 per cent). SVPCs were most commonly seen in boxers (25 per cent) and GSDs (14 per cent). Only 11 per cent of dogs did not demonstrate an arrhythmia, and of these, all had either QRS enlargement pattern consistent with cardiomegaly or ST depression.

Thoracic radiographs were available in 98 per cent of dogs. Cardiomegaly was evident in 289 (80 per cent) and congestive signs (pulmonary oedema or pleural effusion) in 267 (74 per cent). Of the 10 breeds most commonly presented (n>12 dogs), the median percentage (range) of dogs with cardiomegaly was 75 per cent (67 to 89) and with pulmonary oedema or pulmonary venous congestion was 75 per cent (53 to 91).

Echocardiographic examination confirmed the diagnostic features of DCM as defined in the *Materials and methods*. The median fractional shortening percentage (interquartile range) was 12.7 per cent (8.8 to 17.5). The distribution of the FS per cent for each of the groups: 0 to 5, 5 to 10, 10 to 15, 15 to 20 and 20 to 25 per cent was 6, 28, 30, 22 and 14 per cent, respectively. The median ratio of LA diameter to LV diastolic diameter (interquartile range) was 0.98 (0.90 to 1.08). The median Cornell index (interquartile range) for LVDd was 2.11 (1.93 to 2.39) and for LVDs was 1.79 (1.52 to 1.92); these values were outside the normal range based on the Cornell index (LVDd=1.27 to 1.85; LVDs=0.71 to 1.26).

The mean and median clinical pathology variables for all dogs were all within the normal range. There were no significant trends noted, although some individual dogs did have abnormal values. Haematocrit was recorded in 248 dogs and was low in 24 (9.7 per cent) dogs, all of which were in stage 2 or 3 ISACHC

heart failure. Serum urea and creatinine were recorded in 301 dogs and only small changes were noted with increasing class of heart failure, with the upper range value changing the most. The mean (range) concentrations of serum urea were ISACHC 1=5.76 (3.0 to 15) mmol/l, ISACHC 2=6.50 (2.2 to 18) mmol/l and ISACHC 3=8.41 (1.0 to 38) mmol/l. For serum creatinine, mean (range) concentrations were ISACHC 1=109.5 (69 to 208)  $\mu$ mol/l, ISACHC 2=107.8 (52 to 235)  $\mu$ mol/l and ISACHC 3=117.8 (12 to 442)  $\mu$ mol/l.

Of the 369 dogs in total, 12 were excluded from the survival analysis because there were insufficient data available for the statistical analysis, leaving a total of 354 dogs available for the survival analyses. Of the 354 dogs, 180 (51 per cent) were euthanased for cardiac reasons, 142 (40 per cent) died or were euthanased for non-cardiac reasons. A total of 20 dogs (6 per cent) were alive at the last recorded time, and for three dogs (0.8 per cent), the outcome was unknown. Survival data from these 23 dogs were included in the survival analyses up to the last time point at which the dog was known to be alive and were thereafter censored. The median (interquartile range) survival time (to death or euthanasia) for all dogs with DCM was 19 weeks (four to 60 weeks) and the mean survival (sd) was 50.2 weeks (82.7). The survival rate at one year was 28 per cent and at two years was 14 per cent (Fig 3).

## DISCUSSION

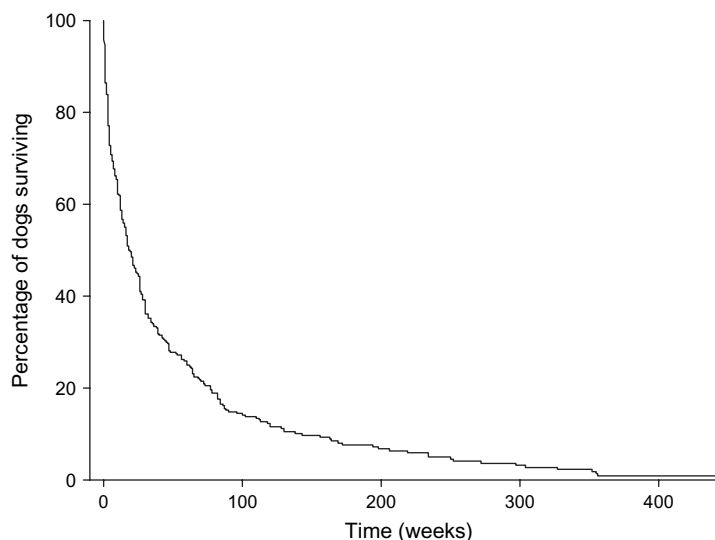
This retrospective review of 369 dogs with DCM not only highlights the similarities with some previous publications but also demonstrates the variation in clinical presentation within breeds. The last publication of a large number of dogs with DCM was a decade ago by Tidholm and others (1997) describing 189 dogs with DCM in Sweden. Other publications included 37 dogs in Colorado (Monnet and others 1995), 76 dogs in Italy (Borgarelli and others 2006) and a more recent study of 62 dogs in Sweden (Tidholm 2006).

In our study, there were 35 different breeds of dogs and only four crossbreed dogs; Tidholm and others (1997) described 38 different breeds. There is a strong tendency for certain breeds to have a higher frequency of DCM. In our study based in England, dobermanns (16 per cent) and boxer (14 per cent) were the two most commonly affected breeds. In North America, the dobermann dominated, ranging from 32 to 57 per cent of affected dogs (COVE Study Group 1995, Monnet and others 1995, Sisson and Thomas 1995, Kittleson 1998). In Sweden, it was the Newfoundland 23 to 26 per cent (Tidholm and others 1997, Tidholm 2006), in Italy it was the great Dane 46 per cent (Borgarelli and others 2006) and in Slovenia it was the dobermann (39 per cent) (Petric and others 2002). These differences in predominance of certain breeds probably reflect the popularity of these breeds in these countries.

The majority of dogs in our study were large breed dogs, and the median bodyweight was 37 kg, very similar to the results of Tidholm (2006) who documented dogs with DCM to have a mean bodyweight (sd) of 36.2 kg (12.7) and the COVE study in which the dogs with DCM ranged in weight from 14.4 to 68.6 kg. Less than 5 per cent of dogs in this study weighed less than 15 kg which is similar to previous publications.

The majority of publications (Monnet and others 1995, Calvert and others 1997, Tidholm and others 1997, Meurs and others 2001, Borgarelli and others 2006, Tidholm 2006) confirmed that DCM occurred predominately in males, and in this study, there was a ratio of 2.7:1 (male: female). However, the sex ratio did vary between breeds with great Danes, GSD, dobermanns and Saint Bernards having a male: female in excess of 5:1. In one report, describing DCM in Dalmatians, all dogs were male (Freeman and others 1996). There were only four Dalmatians in our study, and all were male. However, there have been some publications, which did not demonstrate a sex predisposition in dogs with DCM (O'Grady and others 1992, Baumwart and others 2005). The reason for this difference is unclear. It should be pointed out that sex bias in DCM in our study was in a population of dogs that presented with clinical DCM, whereas previous screening studies have failed to demonstrate a sex bias in pre-clinical DCM. Thus, it may be that while both sexes carry the genotype for DCM, there is a predominance of males that develop heart failure.

The majority of dogs presented at six to eight years of age, similar to Monnet and others (1995), Tidholm and others (1997), Calvert and others (1997), Thomas (1987) and Tidholm (2006). In



**FIG 3.** Kaplan-Meier survival curve in dogs diagnosed with dilated cardiomyopathy. The graph shows the percentage of dogs surviving (not reaching the endpoint of death or euthanasia) versus time since referral

great Danes, the median age at presentation was five years, as found also by Meurs and others (2001) and Borgarelli and others (2006). In IWHs in Germany, the mean age was 4.7 years (Vollmar 1999), whereas in England it was 6.4 years for males and seven years for females (Brownlie and Cobb 1999), similar to this study with a median age of 6.8 years. In boxer dogs, there was a bimodal age of presentation at six and nine years, very similar to results reported by Harpster (1983) who documented peaks at six and 10 years.

The median duration of signs before referral was less than six weeks for the majority of dogs (mean three weeks). The majority of dogs (67 per cent) presented in stage 3 heart failure, similar to other publications (Monnet and others 1995, Borgarelli and others 2006). The most common presenting signs were breathlessness, coughing and exercise intolerance, similar to Tidholm (2006). The most common clinical signs reported in previous studies were depression, cough and inappetence (Tidholm and others 1997); syncope, weight loss and exercise intolerance (Monnet and others 1995) and in great Danes cough, exercise intolerance and weight loss (Meurs and others 2001). In this study, boxers with DCM primarily presented with collapse (70 per cent) whereas Harpster (1983) and Baumwart and others (2005) reported syncope was a presenting sign in 35 per cent of boxers. The most common clinical signs in golden retrievers were reduced appetite (60 per cent) and in great Danes was weakness (55 per cent). Sudden death as the initial presentation was not recorded in this study, as such cases do not reach referral; however, this is reported to be a common presentation for dobermanns in up to 30 per cent of cases (O'Grady and O'Sullivan 2004). There were 8 per cent of dogs presented in ISACHC class 1 heart failure, that is without clinical signs. These were dogs that were referred with a clinical abnormality detected by the referring veterinarian such as a murmur, gallop sound or arrhythmia and found to have DCM on echocardiographic examination.

A murmur, grade 3 or less, was detected in only 33 per cent of dogs, that is the majority of dogs did not have an audible murmur recorded. This seems a low per-

centage and might represent under-reporting in our clinical notes. However, similar values, ranging from 25 to 56 per cent, were reported by Monnet and others (1995), Tidholm and others (1997), Baumwart and others (2005) and Tidholm (2006). However, Borgarelli and others (2006) reported a prevalence of grade 3 or less murmurs of 76 per cent.

The mean heart rate based on an ECG recording was 175/minute with an increasing rate with higher class of heart failure, as was also found by Boswood and Murphy (2006). AF was recorded in 45 per cent of dogs and tended to be more common in the large and giant breed dogs (IWH, great Dane, Newfoundland, GSD and Saint Bernard), similar to previous reports which ranged from 30 to 71 per cent (Monnet and others 1995, Calvert and others 1997, Tidholm and others 1997, Meurs and others 2001, Tidholm 2006). Brownlie and Cobb (1999) noted that 46 per cent of 39 IWHs on screening had AF but by the time they presented with the onset of congestive heart failure, all had AF.

VPCs were seen in 31 per cent of dogs, most commonly in boxers and dobermanns. Because these were the most commonly presented breeds seen, this is probably why the overall incidence of VPCs was higher in this study than other reports, which ranged from 14 to 27 per cent (Monnet and others 1995, Tidholm and others 1997, Tidholm 2006). In boxers, VPCs were noted in 58 per cent of dogs by Baumwart and others (2005). SVPCs were fairly uncommon in our population of dogs, occurring in only 9 per cent (most commonly seen in boxers and GSDs). The great majority of dogs (89 per cent) were presented with an arrhythmia, with only 11 per cent that did not, compared with 20 per cent reported by Tidholm and others (1997). Even in these 11 per cent of dogs without any arrhythmia, there were abnormalities in the waveform consistent with cardiomegaly or ischaemia. Based on these data, it would seem that a completely normal ECG might be a sensitive rule out for clinical DCM in dogs; however, it would be prudent to assume this not necessarily the case in practice.

Thoracic radiographs demonstrated cardiomegaly in 80 per cent and congestion

(pulmonary oedema or pleural effusion) in 74 per cent of dogs. The prevalence of pulmonary oedema in other reports ranged from 73 to 97 per cent (Monnet and others 1995, Calvert and others 1997, Borgarelli and others 2006, Tidholm 2006). In boxers, Baumwart and others (2005) reported pulmonary oedema in only 50 per cent of dogs.

As defined by the echocardiographic criteria, all dogs had dilated left ventricles consistent with DCM; additionally the LVDd to LA diameter ratio was approximately 1.0. In contrast, dogs with primary mitral valve disease usually have a LA diameter in excess of the LVDd diameter (Kittleson 1998). The Cornell index for LVDd and LVDs diameter was higher than the normal range as defined by Cornell and others (2004), and this is supportive of the Cornell index being useful in the diagnosis of DCM.

There were no significant findings between serum urea concentrations and severity of heart failure; however, a study by Boswood and Murphy (2006) did document a significant increase in urea with class of heart failure. The increase of serum urea but not serum creatinine concentrations with higher stage of heart failure suggests a prerenal azotaemia secondary to the heart failure. While these results represent findings at the time of initial referral examination, the influence of prior medications such as furosemide by the referring veterinarian cannot be excluded. The haematocrit was low in 9.7 per cent of dogs, and all these dogs were in class 2 or 3 heart failure. The reason for the low haematocrit in this small number of dogs is unclear; however, it could be associated with the fluid retention that occurs in congestive heart failure and thus haemodilution. Boswood and Murphy (2006) did not document a statistically significant development of anaemia in dogs with DCM. The lack of findings in this study is probably because it is retrospective; proper assessment of clinical pathological findings would require a controlled prospective study.

In this study of dogs with DCM, the median (interquartile range) survival time was 19 weeks (four to 60 weeks) and the mean (sd) survival was 50.2 weeks (82.7). This difference indicates that the survival times were not normally distributed and

therefore the median survival time is the most robust index. The survival rate at one year was 28 per cent and at two years was 14 per cent. Tidholm and others (1997) reported a median survival time of 27 days (3.8 weeks) and a mean of 175 days (25 weeks) with a survival rate at one year of 17.5 per cent and at two years of 7.5 per cent. In Tidholm's Study (1997), 9 per cent of dogs received an ACE inhibitor, whereas in this study 93 per cent received an ACE inhibitor. More recently, Tidholm (2006) documented a median survival time of 126 days (18 weeks) and survival rates of 34 per cent at one year and 20 per cent at two years, similar to the findings in this study. Monnet and others (1995) documented a median survival time of 65 days (9.3 weeks), with 37.5 per cent survival at one year and 28 per cent survival at two years of which 37 per cent of dogs received an ACE inhibitor. Curiously, Borgarelli and others (2006) documented an unusually high median survival time of 671 days (95 weeks), which is difficult to explain.

The cause of death of the dogs in this study was euthanased in 51 per cent of dogs, in comparison to values ranging from 74 to 84 per cent of in Sweden (Tidholm and others 1997, Tidholm 2006), presumably because of intractable clinical signs. Natural death occurred in 40 per cent of dogs, which is likely to have either been because of progressive fulminant congestive failure or a malignant arrhythmia. Calvert and others (1997) reported that half of dobermanns were euthanased and half died, and that 20 per cent died suddenly within the first three months. Petric and others (2002) documented the cause of death in dobermanns to be CHF in 33 per cent, euthanasia in 29 per cent and sudden death in 33 per cent of dogs. In contrast, Borgarelli and others (2006) found that only 3.2 per cent of dogs were euthanased, although 50.8 per cent were still alive at the end of the study, and it seemed that the majority died of cardiac-related death (39.7 per cent). It is possible that the attitudes of the pet-owning public in Italy may view euthanasia differently to other cultures.

The main limitations of this study are that it is retrospective and accuracy of the data relies on case records being reliably

maintained. There is the possibility of some false-positive diagnoses being made in any study of a disease in which there are not definitive criteria for ante-mortem diagnosis, however such numbers are likely to be small. Additionally, the relatively large number of dogs reviewed (n=369) should help overcome the weaknesses inherent in any retrospective study. Holter monitoring was not performed in many of these dogs, particularly those seen many years ago, and there were insufficient number to warrant inclusion of Holter data. Additionally, because the dogs in this study were selected using echocardiographic criteria for DCM, this approach excluded a number of boxers with ARVC described by Baumwart and others (2005).

In summary, dogs with DCM in England represent a wide range of medium to large breed pure-bred dogs with dobermanns and boxers being the most common. The affected breeds were mostly greater than 15 kg in bodyweight, and males were affected significantly more frequently than females. The duration of clinical signs, before referral, was often short (one to six weeks) with exercise intolerance, breathlessness and coughing being the most common signs. Most dogs did not have an audible murmur, and in those that did the murmur was rarely louder than grade 3. AF was a common arrhythmia in the larger and giant breeds, while VPCs were more common in dobermanns and boxers. The majority of dogs had radiographic evidence of cardiomegaly and congestion, and on echocardiography, the Cornell index for LV diameters appeared useful for assessment of LV dilation. The median time to 50 per cent survival was 19 weeks, with the survival rates at one year being 28 per cent and at two years 14 per cent.

### Acknowledgements

The authors thank Drs Jon King and Guenther Strehlau of Novartis Animal Health, Basel, Switzerland, for assistance with the survival analyses; Craig Cornell of University of Davis for advice on using his method of allometric scaling and the referring veterinarians for the referral of these cases.

### References

- BAUMWART, R. D., MEURS, K. M., ATKINS, C. E., BONAGURA, J. D., DEFRADESCO, T. C., KEENE, B. W., KOPLITZ, S., LUIS FUENTES, V., MILLER, M. W., RAUSCH, W. & SPIER, A. W. (2005) Clinical, echocardiographic, and electrocardiographic abnormalities in Boxer with cardiomyopathy and left ventricular systolic dysfunction: 48 cases (1985-2003). *Journal of the American Veterinary Medical Association* **226**, 1102-1104
- BOON, J. A. (1998) Appendix IV: echocardiographic reference values. In: *Manual of Veterinary Echocardiography*. Ed J. A. Boon. Williams & Wilkins, Baltimore, MD, USA. pp 453-473
- BORGARELLI, M., SANTILLI, R. A., CHIAVEGATO, D., D'AGNOLO, G., ZANATTA, R., MANNELLI, A. & TARDUCCI, A. (2006) Prognostic indicators for dogs with dilated cardiomyopathy. *Journal of Veterinary Internal Medicine* **20**, 104-110
- BOSWOOD, A. & MURPHY, A. (2006) The effect of heart disease, heart failure and diuresis on selected laboratory and electrocardiographic parameters in dogs. *Journal of Veterinary Cardiology* **8**, 1-9
- BROWNLIE, S. E. & COBB, M. A. (1999) Observations on the development of congestive heart failure in Irish Wolfhounds with dilated cardiomyopathy. *Journal of Small Animal Practice* **40**, 371-377
- BUCHANAN, J. W. & BUCHELER, J. (1995) Vertebral scale system to measure canine heart size in radiographs. *Journal of the American Veterinary Medical Association* **206**, 194-199
- CALVERT, C. A., CHAPMAN, W. L. & TOAL, R. L. (1982) Congestive cardiomyopathy in Doberman pinscher dogs. *Journal of the American Veterinary Medical Association* **181**, 598-602
- CALVERT, C. A., PICKUS, C., JACOBS, G. J. & BROWN, J. (1997) Signalment, survival and prognostic factors in Doberman pinschers with end-stage cardiomyopathy. *Journal of Veterinary Internal Medicine* **11**, 323-326
- CORNELL, C. C., KITTLESON, M. D., DELLA TORRE, P., HAGGSTRÖM, J., LOMBARD, C. W., PEDERSEN, H. D., VOLLMAR, A. & WEY, A. (2004) Allometric scaling of M-Mode cardiac measurements in normal adult dogs. *Journal of Veterinary Internal Medicine* **18**, 311-321
- COVE STUDY GROUP (1995) Controlled clinical evaluation of enalapril in dogs with heart failure: results of the Cooperative Veterinary Enalapril Study Group. *Journal of Veterinary Internal Medicine* **9**, 243-252
- FREEMAN, L. M., MICHEL, K. E., BROWN, D. J., KAPLAN, P. M., STAMOULIS, M. E., ROSENTHAL, S. L., KEENE, B. W. & RUSH, J. E. (1996) Idiopathic dilated cardiomyopathy in Dalmatians: nine case (1990-1995). *Journal of the American Veterinary Medical Association* **209**, 1592-1596
- GOODING, J. P., ROBINSON, W. F., WYBURN, R. S. & CULLEN, L. K. (1982) A cardiomyopathy in the English Cocker Spaniel: a clinico-pathological investigation. *Journal of Small Animal Practice* **23**, 133-149
- HARPSTER, N. K. (1983) Boxer cardiomyopathy. In: *Kirk's current veterinary therapy, VIII: small animal practice*. Ed R. W. Kirk. W. B. Saunders, Philadelphia, PA, USA. pp 329-337
- INTERNATIONAL SMALL ANIMAL CARDIAC HEALTH COUNCIL (ISACHC) (1999) Appendix A: recommendations for diagnosis of heart disease and treatment of heart failure in small animals. In: *Textbook of Canine and Feline Cardiology*. Eds P. R. Fox, D. Sisson and N. W. Moise. 2nd edn. Saunders, Philadelphia, PA, USA. pp 883-901
- KITTLESON, M. D. (1998) Primary myocardial disease leading to chronic myocardial failure. In: *Small Animal Cardiovascular Medicine*. Eds M. D. Kittleson and R. D. Kienle. Mosby, St Louis, MO, USA. pp 319-346
- MEURS, K. M., MILLER, M. W. & WRIGHT, N. A. (2001) Clinical features of dilated cardiomyopathy in Great Danes and results of pedigree analysis: 17 cases (1990-2000). *Journal of the American Veterinary Medical Association* **218**, 729-732

- MONNET, E., ORTON, E. C., SALMAN, M. & BOON, J. (1995) Idiopathic dilated cardiomyopathy in dogs: survival and prognostic indicators. *Journal of Veterinary Internal Medicine* **9**, 12-17
- O'GRADY, M. R. & O'SULLIVAN, M. L. (2004) Dilated cardiomyopathy: an update. *Veterinary Clinics of North America* **34**, 1187-1207
- O'GRADY, M. R. & HORNE, R. (1992) Occult dilated cardiomyopathy: an echocardiographic and electrocardiographic study of 193 asymptomatic Doberman pinschers (Abstract). *Journal of Veterinary Internal Medicine* **6**, 112
- PETRIC, A. D., STABEJ P. & ZEMVA M. D. (2002) Dilated cardiomyopathy in Doberman pinschers: survival, causes of death and a Pedigree Review in a related line. *Journal of Veterinary Cardiology* **4**, 17-24
- SISSON, D. D. & THOMAS, W. P. (1995) Myocardial diseases. In: Textbook of Veterinary Internal Medicine. Eds S. J. Ettinger and E. C. Feldman. 4th edn. W. B. Saunders, Philadelphia, PA, USA. pp 995-1032
- SISSON, D., O'GRADY, M. & CALVERT, C. A. (1999) Myocardial disease of the dogs. In: Textbook of Canine and Feline Cardiology. 2nd edn. Eds P. R. Fox, D. Sisson and N. S. Moise. W. B. Saunders, Philadelphia, PA, USA. pp 581-619
- SLEEPER, M. M., HENTHORN, P. S., VIJAYASARATHY, C., DAMBACH, D. M., BOWERS, T., TUSKENS, P., ARMSTRONG, C. F. & LANKFORD, E. B. (2002) Dilated cardiomyopathy in juvenile Portuguese Water Dogs. *Journal of Veterinary Internal Medicine* **16**, 52-62
- THOMAS, R. E. (1987) Congestive cardiac failure in young Cocker Spaniels (a form of cardiomyopathy?): details of eight cases. *Journal of Small Animal Practice* **28**, 265-279
- TIDHOLM, A. (2006) Survival in dogs with dilated cardiomyopathy and congestive heart failure treated with digoxin, furosemide and propranolol: a retrospective study of 62 dogs. *Journal of Veterinary Cardiology* **8**, 41-47
- TIDHOLM, A. & JONSSON, L. (1996) Dilated cardiomyopathy in the Newfoundland: a study of 37 cases (1983-1994). *Journal of the American Animal Hospitals Association* **32**, 465-470
- TIDHOLM, A., SVENSSON, H. & SYLVEN, C. (1997) Survival and prognostic factors in 189 dogs with dilated cardiomyopathy. *Journal of the American Animal Hospitals Association* **33**, 364-368
- VOLLMAR, A. C. (1999) Use of echocardiography in the diagnosis of dilated cardiomyopathy in Irish Wolfhounds. *Journal of the American Animal Hospitals Association* **35**, 279-283