Sleeping and resting respiratory rates in dogs with subclinical heart disease

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Objective—To characterize sleeping respiratory rates (SRRs) and resting respiratory rates (RRRs), collected in the home environment, of dogs with subclinical heart disease that could result in left-sided congestive heart failure.

Design—Prospective cross-sectional study.

Animals—190 adult dogs with subclinical left-sided heart disease.

Procedures—Most dogs had mitral valve disease or dilated cardiomyopathy of various severities. Clients collected ten 1-minute SRRs or RRRs during a period ranging from 1 week to 6 months. Clinicians provided echocardiographic and medical data on each patient.

Results—The within-dog mean SRR (SRR_{mean}; 16 breaths/min) was significantly lower than the within-dog mean RRR (RRR_{mean}; 21 breaths/min). Seven dogs had SRR_{mean} and 33 dogs had RRR_{mean} > 25 breaths/min; 1 dog had SRR_{mean} and 12 dogs had RRR_{mean} > 30 breaths/min; these dogs mostly had a left atrial (LA)-to-aortic ratio > 1.8. Dogs with moderate LA enlargement had a significantly higher SRR_{mean} than did other dogs. However, median SRR_{mean} for each of 4 levels of LA enlargement was < 20 breaths/min; median RRR_{mean} for each of 4 levels of LA enlargement was < 25 breaths/min. Both within-dog SRR and RRR remained stable for 10 consecutive measurements. Treatment with cardiac medications or presence of pulmonary hypertension was not associated with SRR_{mean} or RRR_{mean}.

Conclusions and Clinical Relevance—Results suggested that dogs with confirmed subclinical left-sided heart disease of various severities generally had SRR_{mean} < 25 breaths/min, which was infrequently exceeded at any time, and that SRR and RRR remained stable, regardless of individual within-dog SRR_{mean} or RRR_{mean}. (*J Am Vet Med Assoc* 2013;243:839–843)

Left-sided CHF is a syndrome characterized in dogs by development of pulmonary venous congestion and edema or pleural effusion secondary to severe left heart disease.[1] Clinical signs of L-CHF in dogs include dyspnea or varying degrees of tachypnea. These can be subtle and difficult to detect early in the course of developing L-CHF but can progress to marked signs.[1] Diagnosis of L-CHF often relies on results from several tests and traditionally requires confirmation of severe left-sided cardiac disease, radiographic evidence of pulmonary interstitial or alveolar opacity, and consistent clinical signs.

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Abbreviations

CHF	Congestive heart failure
CV	Coefficient of variation
DCM	Dilated cardiomyopathy
LA	Left atrium
LA:Ao	Left atrial-to-aortic ratio
L-CHF	Left-sided congestive heart failure
MMVD	Myxomatous mitral valve disease
RRR	Resting respiratory rate
RRR_{mean}	Within-dog mean of resting respiratory rate measurements
SRR	Sleeping respiratory rate
SRR_{mean}	Within-dog mean of sleeping respiratory rate measurements

Previous investigators reported RRR to be the most sensitive and specific single diagnostic test for identifying L-CHF as a cause of respiratory tract–related clinical signs associated with heart disease and an independent variable in predicting L-CHF in multivariable regression analysis.[2] Another study[3] revealed that resolution of the L-CHF resulted in reduction in respiratory rate into pre-CHF ranges. Thus, respiratory rate has the potential of being a sensitive, albeit nonspecific, indicator of developing or recurring CHF in dogs. Healthy dogs have SRR_{mean} < 25 breaths/min.[4] Continued, frequent monitoring of SRR or RRR could allow more timely therapeutic intervention in dogs and cats with severe subclinical heart disease (eg, those with marked LA en-

largement) or known prior history of L-CHF. Many veterinary cardiologists currently recommend that clients monitor respiratory rates in canine and feline cardiac patients with subclinical disease in their home environment to help determine the onset of L-CHF or to monitor effectiveness of treatment. Anecdotally, veterinary cardiologists have suggested that an SRR < 30 breaths/min likely excludes L-CHF as a cause of clinical signs; however, to the best of the authors' knowledge, no data exist confirming this threshold value in dogs with subclinical left-sided heart disease.

Therefore, the purpose of the study reported here was to characterize SRRs and RRRs, collected in the home environment, of dogs with subclinical heart disease (mostly MMVD and DCM) that could result in L-CHF. The hypothesis was that dogs with these subclinical left-sided heart diseases, as defined by a lack of diuretic requirement, would have SRR < 30 breaths/min, similar to that of apparently healthy dogs.

Materials and Methods

Dogs—Veterinary cardiologists were recruited to request their clients to collect SRR and RRR data from dogs evaluated in the course of their clinical work by use of a standardized collection form. Clients were invited to participate in data collection without prejudice. Clients returned completed forms to participating cardiologists who provided echocardiographic and pertinent medical information on the patients. Data collection began in April 2010 and ended in March 2012. Participants were recruited from various regions of North America, Europe, and Asia. All data were entered into an online data acquisition system through a public website[a] and stored on a central server prior to analysis.

Institutional approval was not sought because this study was purely observational, voluntary, and noninvasive. To characterize the disease present and to ascertain its severity, a comprehensive echocardiographic evaluation of each dog was performed by the participating veterinary cardiologist prior to any SRR or RRR data collection as part of a complete clinical cardiac evaluation in subclinically affected dogs.

Excluded were dogs with a history of L-CHF, dogs receiving diuretics, dogs with other severe systemic diseases, and dogs that were < 6 months old. Cardiologists were asked to provide linear measurements of the LA and aorta as described[5,6] and to determine whether pulmonary hypertension was present (as measured via tricuspid valve regurgitation velocity, when present, and defined as a tricuspid valve regurgitation velocity > 3.2 m/s).[7] Additionally, the cardiologists performing the examination were asked to detail any medications (cardiac or noncardiac) the dogs were receiving at the time of the examination and whether any comorbidities existed that were likely to affect respiratory rate. Finally, the cardiologists were asked to provide information about thoracic radiographs obtained at the time of examination and recruitment into the study. In dogs with moderate or severe LA enlargement for which radiographs were not obtained as part of the clinical evaluation, the period from the end of SRR data collection to the first prescription of diuretics was evaluated to confirm the presumptive diagnosis of subclinical disease.

Study design—Participants collected 10 SRR measurements from their dogs in their home environment during deep sleep by counting breaths for 1 minute, with instructions to avoid measuring SRR when dogs were in active motor sleep (paddling and twitching). No more than 2 measurements/d were allowed (therefore, the most rapid data acquisition would take 5 days), and there was no upper limit to the time over which collection could occur. During periods of data collection, dogs were to be kept in a thermoneutral environment, meaning that extremes of heat and cold should be avoided, but temperature ranges that would be acceptable were not specified, and participants did not record ambient room temperatures at the time of collection. Measurements of RRR were performed in a similar manner, except that dogs were awake and recumbent. Participants were requested not to measure RRR if the dog was panting or had recently been exercised. Participants recorded date of birth (approximated if not known), sex and reproductive status, body weight, body condition score, breed, and respiratory rates.

Statistical analysis—The SRR_{mean}, RRR_{mean}, SD, and CV were calculated as well as the maximum and minimum within-dog SRR and RRR for each dog's set of 10 SRR and RRR measurements. These within-dog variables were examined by means of box-and-whisker plots to describe the data distribution for the SRR and RRR means, SD, maxima, minima, and CVs for the entire study sample.

A 2-way ANOVA was used to examine the effect of LA size and use of cardiac medications on SRR and RRR after confirming normality of SRR and RRR data by use of a Shapiro-Wilk test. Bonferroni-adjusted pairwise comparisons were performed for each factor in the 2-way ANOVA that was found to be significant at $P < 0.05$. The SRR and RRR data required log transformation to achieve a normal distribution. The relationship between LA:Ao and log(SRR) or log(RRR) was examined by use of linear regression. Left atrial size was categorized by assigning an ordinal scale on the basis of quantiles of LA:Ao measurements as follows: LA:Ao ≤ 1.6 = 1 (clinically normal), 1.6 < LA:Ao ≤ 1.9 = 2 (mild LA enlargement), 1.9 < LA:Ao < 2.3 = 3 (moderate LA enlargement), and LA:Ao ≥ 2.3 = 4 (severe LA enlargement). These categories were based on data from a recent study.[b] It was then determined whether SRR and RRR differed between dogs receiving cardiac medication and those not receiving cardiac medication via Mann-Whitney *U* tests. Similarly, whether dogs receiving medication had larger LAs than those not receiving medication was evaluated via Mann-Whitney *U* tests.

To examine the possibility of significant increases in SRR over the collection period for each dog, the intradog coefficients (slopes) of the regressed data were evaluated by use of a 1-sample *t* test. A slope of zero would suggest that there was no systematic increase in SRR over the collection period, indicating stable subclinical disease.

Within-dog SRR_{mean} with RRR_{mean} and the within-dog coefficients of variation for both SRR and RRR of all dogs from which RRR was acquired were evaluated with Wilcoxon signed-rank tests. All analyses were performed with statistical software.[c,d] For all comparisons, $P \leq 0.05$ was considered significant.

Results

Twenty-two cardiologists from 3 continents (11 countries: United States, Israel, Italy, Sweden, Taiwan, United Kingdom, Canada, Denmark, Netherlands, Bulgaria, and Slovenia) provided data on 190 dogs with subclinical heart disease. Data were not complete for some dogs. Breeds represented by at least 5 dogs included Cavalier King Charles Spaniel (n = 31), Maltese (27), mixed-breed (22), Pekinese (13), Dachshund (12), Shih Tzu (9), Toy Poodle (7), Miniature Schnauzer (6), and Chihuahua (5). Dogs had a median age of 128 months (range, 22 to 251 months [date of birth for 1 dog was not available]) and included 28 sexually intact females, 58 spayed females, 65 sexually intact males, and 39 neutered males. The dogs had a median weight of 8 kg (17.6 lb; range, 1 to 78 kg [2.2 to 171.6 lb]); 18 were considered thin, 148 were considered of healthy body condition, and 24 were considered overweight. One-hundred seventy-four dogs had MMVD, of which 115 had no LA enlargement, 30 had mild LA enlargement, 23 had moderate LA enlargement, and 8 had severe LA enlargement. Ten dogs had DCM, of which 7 had no LA enlargement and 3 had mild LA enlargement. Three dogs had mitral valve dysplasia, of which 2 had no LA enlargement and 1 had severe LA enlargement. Two dogs had patent ductus arteriosus, of which one had no LA enlargement and the other had moderate LA enlargement. One dog had HCM with no LA enlargement. Twenty-one dogs had pulmonary hypertension. Sixty-five dogs were receiving cardiac medications, of which 59 were receiving an angiotensin-converting enzyme inhibitor, 24 were receiving pimobendan, and 2 were receiving β-adrenergic receptor antagonists.

Thoracic radiographs were obtained in 94 of 190 (50%) dogs at the time of enrollment in the study as part of the routine cardiac evaluation, including 19 of 33 (58%) dogs with moderate to severe LA enlargement. None of the remaining 14 dogs with moderate to severe LA enlargement required diuretics for at least 3 months after the end of data collection. The SRR and RRR data collection periods ranged from 5 to 178 days, with a median collection period of 15 days. Two dog owners provided < 10 SRR measurements for their dogs; however, their data were included in the analysis. Two dog owners provided < 10 RRR measurements for their dogs (these data were included in the analysis), and 16 dog owners did not provide any RRR measurements.

SRR measurements—Dogs with subclinical heart disease had a median SRR_{mean} of 16 breaths/min (range, 9 to 33 breaths/min; **Figure 1**). Within-dog regression coefficients (slopes) did not differ from zero (P = 0.4), with a mean slope of –0.02, indicating that measurements did not change significantly over time.

Seven dogs had $SRR_{mean} \geq 25$ breaths/min, and one of these dogs had $SRR_{mean} \geq 30$ breaths/min. The severities of LA enlargement in these 7 dogs, determined by the quantile assignments, were as follows: 3 had moderate LA enlargement (LA:Ao, 2.1, 2.0, and 2.0), 3 had mild LA enlargement (LA:Ao, 1.9), and 1 had no LA enlargement (LA:Ao, 1.2). Ten dogs had individual SRR measurements ≥ 30 breaths/min.

RRR measurements—Resting respiratory rate measurements were available for 174 dogs. Dogs had a median within-dog RRR_{mean} of 21 breaths/min (range, 10 to 43 breaths/min; Figure 1). Within-dog regression coefficients (slopes) did not differ from zero (P = 0.08), with a mean slope of –0.06 for all dogs, indicating that measurements did not change significantly over time. Thirty-three dogs had $RRR_{mean} \geq 25$ breaths/min, and 12 of these dogs had $RRR_{mean} \geq 30$ breaths/min. Of these 12 dogs, 3 had no LA enlargement, 4 had mild LA enlargement, 4 had moderate LA enlargement, and 1 had severe LA enlargement as determined with the quantile assessment of LA size. Forty-three dogs had at least 1 RRR measurement ≥ 30 breaths/min.

Comparison of SRR and RRR—Dogs had lower SRR_{mean} than RRR_{mean} ($P < 0.001$). Only 2 of 174 dogs in which both SRR and RRR were obtained had $SRR_{mean} > RRR_{mean}$: one dog had SRR_{mean} of 24 breaths/min and RRR_{mean} of 23 breaths/min, and the other dog had SRR_{mean} of 13 breaths/min and RRR_{mean} of 12 breaths/min. Within-dog SRR CV did not differ from RRR CV (P = 0.5) because within-dog variability of SRR was identical to that of RRR (mean and median SRR CV and RRR CV were 11%).

Associations between cardiac medications or LA size and SRR or RRR—When the associations between cardiac medications or LA size and SRR_{mean} were examined, an association between LA size and SRR_{mean} was detected ($P < 0.003$; **Figure 2**) but no association with

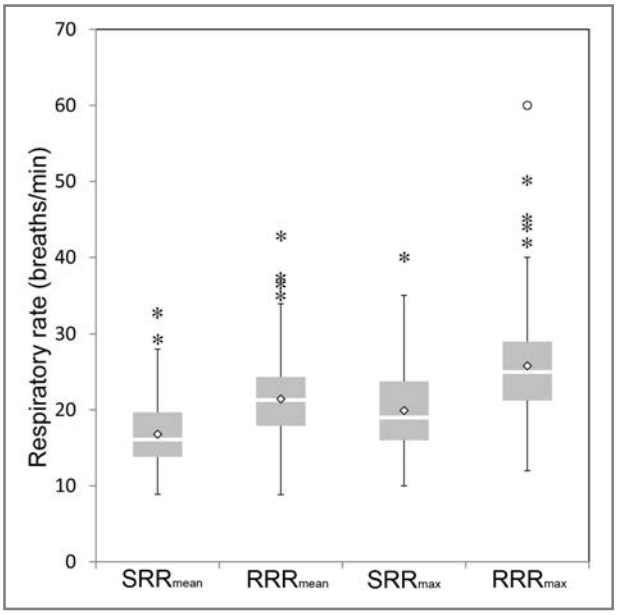

Figure 1—Box-and-whisker plots of SRR_{mean}, RRR_{mean}, SRR_{max} (within-dog maximum SRR), and RRR_{max} (within-dog maximum RRR) of 190 dogs with subclinical heart disease. In each plot, the white line denotes the median value, the diamond denotes the mean value, the shaded box denotes the interquartile range (IQR [25th to 75th percentile]), an asterisk denotes a value > 1.5 times the IQR greater than the 75th percentile, and a circle denotes a value > 3 times the IQR greater than the 75th percentile. Owners of 16 dogs did not provide RRR measurements.

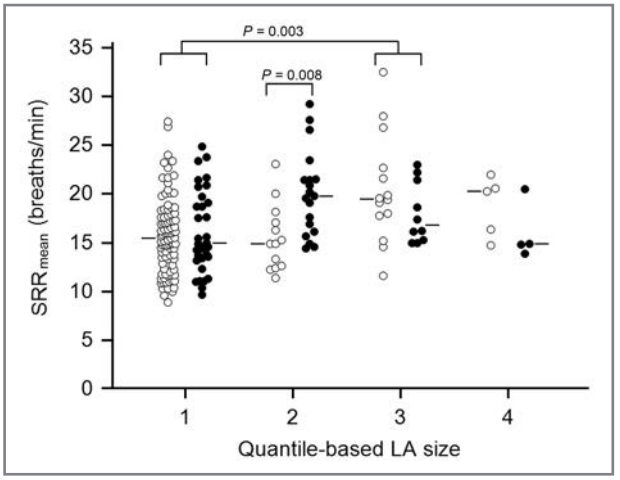

Figure 2—Dot plot of 190 dogs with subclinical cardiac disease indicating the relationship between SRR_{mean} and disease severity as estimated by dividing LA:Ao measurements into quantiles: 1 = no LA enlargement (LA:Ao ≤ 1.6), 2 = mild LA enlargement (1.6 < LA:Ao ≤ 1.9), 3 = moderate LA enlargement (1.9 < LA:Ao < 2.3), and 4 = severe LA enlargement (LA:Ao ≥ 2.3). Pairs of dot plots within each quantile represent dogs receiving cardiac medications (black circles) and dogs not receiving cardiac medications (white circles). Horizontal lines beside each dot plot represent median values.

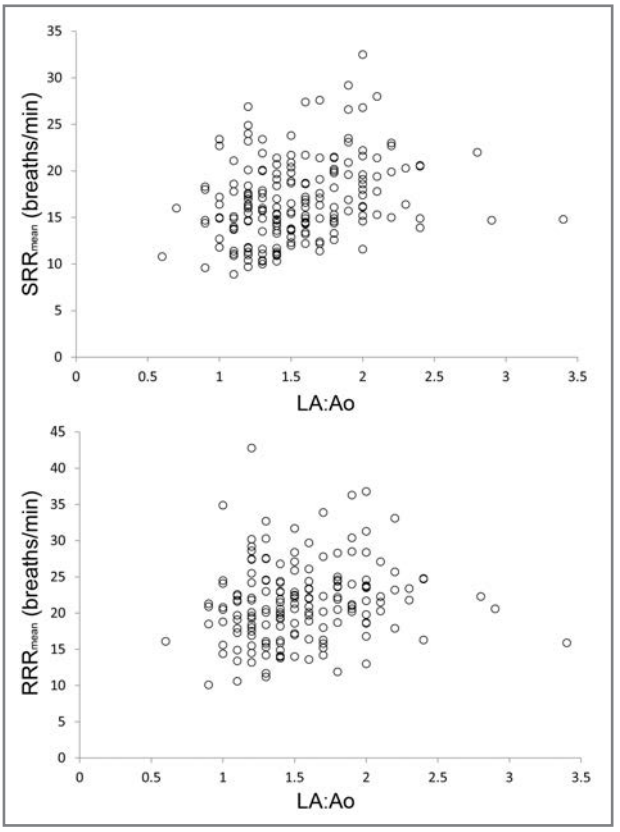

Figure 3—Scatterplots of SRR_{mean} (A) and RRR_{mean} (B) values plotted against LA:Ao measurements in 190 dogs with subclinical cardiac disease.

cardiac medications was detected ($P = 0.93$). Multiple comparisons revealed that dogs with moderate LA enlargement had higher SRR_{mean} than did dogs with no LA enlargement ($P = 0.003$). Nonmedicated dogs with mild LA enlargement had lower SRR_{mean} than did medicated dogs with mild LA enlargement ($P = 0.008$). Linear regression of log(SRR) against LA:Ao revealed a weak positive relationship between LA size and SRR (adjusted $r^2 = 0.08$; $P < 0.001$; **Figure 3**).

There were no associations between LA size ($P = 0.06$) or cardiac medications ($P > 0.8$) and RRR. Linear regression of RRR against LA:Ao revealed a weak positive relationship between LA size and RRR (adjusted $r^2 = 0.03$; $P = 0.03$; Figure 3).

Dogs receiving cardiac medications had larger LA (as determined by both LA:Ao and LA quantiles) than dogs not receiving cardiac medications ($P < 0.001$ and $P = 0.009$, respectively). However, dogs receiving cardiac medications did not have higher SRR or RRR than dogs not receiving cardiac medications ($P = 0.07$ and $P = 0.34$, respectively).

Twenty-one dogs were identified as having pulmonary hypertension on the basis of tricuspid valve regurgitant velocity ranging from 3.24 to 5 m/s. However, only 4 of those dogs were classified as having moderate LA enlargement, and 1 was classified as having severe LA enlargement. The within-dog SRR_{mean} of all dogs with pulmonary hypertension was < 20 breaths/min.

Discussion

The present study provided comprehensive evaluation of SRRs and RRRs in dogs with subclinical heart disease that could ultimately result in L-CHF. These results might provide guidelines for clinicians that prescribe at-home monitoring of respiratory rates in dogs with subclinical heart disease for timely detection of progression to L-CHF. The data apply to the full spectrum of severities of subclinical heart disease that would potentially result in L-CHF.

Dogs with subclinical heart disease had an SRR_{mean} that infrequently exceeded the upper limit of SRR_{mean} in healthy dogs of 25 breaths/min and rarely exceeded 30 breaths/min.[4] Dogs with subclinical heart disease had a median SRR_{mean} of 16 breaths/min, similar to that of healthy dogs (14 breaths/min).[4] However, some dogs (5%) occasionally had individual SRR > 30 breaths/min. Furthermore, within-dog SRR did not vary substantially from day to day for most dogs, as evidenced by the low within-dog CV. A small, significant (but clinically unimportant) increase in SRR_{mean} with increasing LA size was found. Somewhat surprisingly, only dogs with moderate LA enlargement, but not severe LA enlargement, had marginally higher SRR than did dogs with mild or no LA enlargement. This was most likely a function of the small sample size of the group with severe LA enlargement (type II error), in which only 9 dogs had an LA:Ao ≥ 2.3, and less likely a type I error with the dogs that had moderate LA enlargement because median SRR_{mean} was similar for both the moderately enlarged LA group and the severely enlarged LA group. The differences were of a clinically irrelevant magnitude (1 to 2 breaths/min). Finally, no effect of cardiac medications on SRR_{mean} was found, although some dogs that received cardiac medications had more severe disease (as evidenced by larger LA size) than those not receiving cardiac medications.

The data may provide clinically useful information for clinicians who use SRR as a biomarker for L-CHF

in dogs with subclinical heart disease. Dogs with severe left-sided heart disease and evidence of tachypnea during sleep warrant closer inspection, as these dogs might have mild clinical signs of L-CHF. Moreover, on the basis of the minimal within-dog SRR variability observed in the present study, owners of dogs with subclinical heart disease can be encouraged by clinicians to determine their dog's SRR range while the dog has stable subclinical heart disease and to routinely monitor their dog's SRR to ascertain stability over time. Such a strategy might permit owners to detect deterioration in their dog's clinical status as evidenced by a gradual increase of SRR, or by SRR > 30 breaths/min, and allow more timely intervention by the clinician.

As expected, RRR_{mean} was slightly higher than SRR_{mean} in the dogs in this study. The median RRR_{mean} was 21 breaths/min but had a similarly small degree of within-dog variability, compared with that seen for SRR.

The study did not evaluate the sensitivity and specificity of SRR in the diagnosis of CHF. However, in dogs examined by clinicians, an in-clinic SRR or RRR that is less than the reference limit is likely to rule out L-CHF as a cause of clinical signs, but a high SRR is not specific because noncardiac causes of tachypnea, such as pneumonia, heatstroke, stress, and pain, might be present. Equally important is that an SRR within reference range does not exclude the presence of heart disease, but only reduces the probability of the dog having L-CHF.

This study had several limitations typical of such cross-sectional studies. Participants were not asked to record ambient temperatures during the period of data collection. However, data collection extended over 3 continents, multiple climatic zones, and multiple seasons within each region, so the authors believe that these numbers can be applied confidently to dogs across the globe. It was requested that the dogs be in a thermoneutral environment (ie, not too hot and not too cold), although this was a subjective assessment by the owners. It is possible, however, that in some instances, higher SRRs were recorded while the dogs were exposed to hot conditions (eg, a dog sleeping in front of a warm fire).

Cardiologists were not asked to systematically train clients to record SRRs and RRRs in their dogs. However, in the authors' previous study[4] of healthy dogs, SRRs and RRRs collected by trained individuals (veterinarians and veterinary students) did not differ from those collected by untrained dog owners. Furthermore, the similarity in SRRs and RRRs among dogs in the present study would suggest that untrained dog owners measured SRRs and RRRs similarly. Finally, correspondence with several cardiologists involved in the study about training clients indicated that they routinely demonstrate how RR should be counted. Thus, we are confident that the rates obtained by most owners were accurate.

Left atrial size was assessed by use of a quantile-based analysis by means of the LA:Ao provided by the cardiologists from a right parasternal short-axis view. Although no standards exist for ascribing LA size by use of LA:Ao cutoff values, results of 1 study[c] suggest that most cardiologists agree with these limits.

Only a small number of dogs had severe subclinical disease. Thus, whether the results are robustly extrapolatable to the population of dogs with severe subclinical disease warrants additional evaluation. Additionally, although it is possible that some dogs with moderate or severe subclinical disease might have had subtle signs of CHF at the time of data collection, overt pulmonary edema was not observed on thoracic radiographs in 19 of 33 dogs, and none of the remaining 14 dogs required diuretic administration for control of clinical signs of CHF for at least 3 months. Thus, we are confident that the population truly had subclinical disease at the time of data collection.

Most of the dogs in the present study had MMVD, with a smaller percentage having DCM. Although the specific disease (MMVD vs DCM) would not be expected to cause a different effect on SRR, the greater percentage of dogs with MMVD suggests that our results are most valid for that population.

The data suggest that dogs with subclinical heart disease generally have SRR_{mean} < 25 breaths/min in the home environment; the data are easily obtained by most clients and provide clinicians with a strong basis on which to make their assessment of respiratory rate. Dogs with repeated or consistent SRR_{mean} > 30 breaths/min might warrant additional investigation to determine whether or not L-CHF or respiratory disease exists.

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