HHS Public Access

Author manuscript

J Vet Intern Med. Author manuscript; available in PMC 2015 March 12.

Published in final edited form as:

J Vet Intern Med. 2008 ; 22(4): 924–930. doi:10.1111/j.1939-1676.2008.0121.x.

Echocardiographic Ratio Indices in Overtly Healthy Boxer Dogs Screened for Heart Disease

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Abstract

Background—Boxer dogs are routinely screened by echocardiography to exclude congenital and acquired heart disease. Individuals of a given breed may span a large range of body sizes, potentially invalidating linear regression of M-mode measurements against body weight. Echocardiographic ratio indices (ERIs) provide a novel method of characterizing echocardiographic differences between Boxers and other dog breeds.

Hypothesis—ERIs obtained from overtly healthy Boxer dogs presented for cardiac screening will be different from ERIs established for normal non-Boxer dogs, and those differences will be unrelated to aortic velocity or systolic blood pressure.

Animals—Eighty-one Boxers with no outward clinical signs of heart disease were studied.

Methods—All dogs were examined by 2-dimensional, M-mode, and Doppler echocardiography. M-mode measurements were used to perform ERI calculations, and the indices in Boxers were compared between Boxers with varying severity of arrhythmia and those of normal non-Boxer dogs.

Results—Differences in weight-based ERIs, which reflect increased thickness of the left ventricular free wall (LVW) and interventricular septum (IVS) and smaller aortic size, were found in overtly healthy Boxer dogs compared with normal non-Boxer dogs. ERIs of left atrial and LV cavity size in overtly healthy Boxers were not significantly different from those of non-Boxer dogs.

Conclusions and Clinical Importance—Boxer dogs may have an increased relative thickness of the LVW and IVS that is independent of aortic size, aortic velocity, or arterial blood pressure, and this morphology should be taken into consideration when screening Boxers by echocardiography.

Keywords

Aortic stenosis; Arrhythmogenic right ventricular cardiomyopathy; Cardiology; Echocardiography

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This study was performed at the Foster Hospital for Small Animals at Tufts Cummings School of Veterinary Medicine.

Boxer dogs are routinely screened by echocardiography and ECG to exclude congenital and acquired heart disease.[1–5] Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a common entity in the breed,[4,6] characterized by serious ventricular arrhythmias, syncope, and high risk of sudden death. In a prior study of 188 Boxers assessed by 24-hour ambulatory ECG (AECG), more than 30% of the dogs had more than 100 ventricular premature depolarizations in 24 hours, which has been proposed as a criterion for the diagnosis of ARVC.[4] Although some Boxers also develop overt myocardial failure and dilated cardiomyopathy, the relationship between ARVC and the presence or development of left ventricular (LV) dysfunction is unclear.[7,8]

Subaortic stenosis is also prevalent in the Boxer and is the most common congenital cardiac disease in the breed according to most reports.[1,2] Echocardiography is the least invasive method of diagnosing aortic stenosis, but confirmation of mild or equivocal disease remains problematic because of the high incidence of cardiac murmurs and intrinsically higher aortic velocities documented in Boxers.[9–11] Prior echocardiographic studies have focused on the LV outflow tract of the Boxer and have confirmed a smaller LV outflow tract area and aortic size contributing to the higher aortic velocities and murmurs noted in the breed.[10]

Previous studies have documented significant variability in echocardiographic measurements in dogs of different somatotypes[12] and have demonstrated the limitations of linear regression models in predicting echocardiographic measurements even among dogs of the same breed.12,[a] Specific echocardiographic reference values have been established for several breeds,[13–17] but evaluation of normal echocardiographic variation within a breed remains problematic, particularly for breeds such as the Boxer, which is characterized by large variation in body size.[a] Echocardiographic ratio indices (ERIs) represent a novel, body size-independent method to compare echocardiographic data among patients. They are derived by indexing echocardiographic measurements to a weight-based predicted aortic dimension, which serves as an “internal yardstick” for comparison of cardiac dimensions among individuals of various sizes. Weight-based ERIs have been described for normal dogs[18] and dogs with chronic valvular disease,[19] and may be useful for studying morphologic differences in cardiac structure in dogs within and across various dog breeds. Boxer dogs were excluded from analysis in the previous ERI publications because they were recognized at that time to have M-mode echocardiographic characteristics that may not conform with those of other dogs.[18] In addition, Boxers have been noted to have smaller aortas than non-Boxer dogs,10,[b] which may preclude accurate use of aorta-based ERIs in the breed.

The purpose of the current study was to describe the M-mode echocardiographic characteristics of a group of overtly healthy Boxer dogs screened for heart disease and to compare these values with those of healthy non-Boxer dogs using weight-based ERIs. We also sought to evaluate the relationship between the LV ERIs and arterial blood pressure,

[a]Herrtage ME. Echocardiographic measurements in the normal Boxer. Proceedings 4th Annual ESVIM Congress, Brussels, 1994:172–173

[b]Abbott JA, Duncan R, Clark EG. Aortic valve disease in Boxers with physical and echocardiographic findings of aortic stenosis. Proceeding 19th Annual ACVIM Forum, Denver, 2001:844

aortic velocity, and aortic dimension, and to assess whether any structural differences are found among overtly healthy Boxer dogs with varying degrees of cardiac arrhythmia.

Materials and Methods

Ninety-one adult Boxers with no outward clinical signs of heart disease were evaluated prospectively at the Foster Hospital for Small Animals at Tufts Cummings School of Veterinary Medicine from 2004 to 2006. These dogs were recruited for another study examining the effects of n-3 fatty acid supplementation on arrhythmia frequency in asymptomatic Boxers with ARVC.[6] This study was approved by the Tufts University Institutional Animal Care and Use Committee and informed consent was provided by all owners. Boxers that were outwardly normal according to their owners were screened by physical examination, echocardiographic examination with continuous ECG monitoring throughout the examination, and 2-minute multilead ECG. Dogs found to have any ventricular ectopy during this screening were further studied with Doppler blood pressure measurement and 24-hour AECG. The 24-hour AECG data were evaluated using commercially available software.[c] Arterial blood pressure was measured by the Doppler method[d] with dogs in lateral recumbency, and the mean of 3 systolic blood pressures was recorded. Dogs with no evidence of ventricular arrhythmia during the initial echocardiogram or ECG were designated as Group I and were not evaluated by AECG. For the dogs that were evaluated by AECG, those that had <50 ventricular premature complexes (VPCs) in a 24-hour period on AECG monitoring were designated as Group II, and dogs with >50 VPCs in a 24-hour period on AECG were designated as Group III. Exclusion criteria for the current study included a peak aortic ejection velocity of >2.4 m/s measured either by pulsed-wave (PW) or continuous wave (CW) Doppler echocardiography. Dogs with other identifiable congenital cardiac abnormalities, dogs currently receiving any cardiac drugs, and those with outward signs of cardiac disease identified by the owner (eg, exercise intolerance, collapse, dyspnea) were also excluded.

Echocardiographic examinations consisting of 2-dimensional (2-D), M-mode, color, and spectral Doppler analysis were performed by a single observer (J.R.) with a 5-MHz transducer.[e] All dogs had M-mode echocardiographic data obtained while unsedated and in right lateral recumbency. M-mode recordings were obtained with 2-D guidance with measurements performed from leading edge to leading edge in accordance with the American Society of Echocardiography standards.[20] Measurements included interventricular septum (IVS), left ventricular internal dimension (LVID), and left ventricular free wall (LVW) in diastole (IVSd, LVIDd, and LVWd) and systole (IVSs, LVIDs, and LVWs), as well as standard aortic root (Ao, end-diastole) and left atrial (LA, end-systole) dimensions and the E-point-to-septal separation (EPSS). A transverse 2-dimensional left atrial diameter (LA-2D) was also measured in early ventricular diastole as described by Hansson et al.[21] Most ERI calculations were performed by dividing M-mode measurements by the weight-based predicted aortic dimension (wAo; the character w preceding the parameter denotes

[c]Del Mar Holter Analysis System, Del Mar Reynolds Medical Inc, Irvine, CA
[d]Parks Doppler, Aloha, OR
[e]Sonos 4500 system, Hewlett Packard, Wilmington, DE

weight-based ERI), equivalent to 0.795 (body weight in kg)$^{1/3}$, as reported previously[18,19] (see Appendix 1 for explanation of ERI terminology and calculations). In part I of the study, ERIs from Boxer dogs were compared with those from 47 normal non-Boxer dogs that were >1.5 years of age and used to establish ERI normal values in the original publication.[18] In part II of the study, ERIs were compared among the 3 Boxer study groups.

All cardiac valves and the interatrial and interventricular septa were interrogated by color Doppler. Aortic velocities were obtained from the subcostal or left apical 5-chamber view or both, and the peak velocity was obtained via PW or CW at either location and recorded. Pulmonic velocities were recorded using PW Doppler from the right parasternal short-axis view at the level of the pulmonic valve.

Statistical Analysis

Data were examined graphically. Because some parameters were not normally distributed, all descriptive data are presented as median (range). Data that were not normally distributed were logarithmically transformed before analysis. In part I of the study, continuous data (eg, age, weight, and LA dimension) in the Boxer and non-Boxer groups were compared by an independent *t*-test. In part II of the study, continuous data were compared among the 3 Boxer groups (Groups I–III) by one-way analysis of variance with Tukey's post hoc analysis. Pearson's correlation analysis was used to examine for correlation between ERIs of LV wall thickness and aortic velocity, aortic dimension, or systolic blood pressure. Spearman's correlations were used to compare 2 continuous variables (eg, body weight and raw M-mode measurements or ERIs). Commercial statistical software[f] was used for all analyses and *P*-values < .05 were considered statistically significant.

Results

Of 91 Boxers with no outward clinical signs of heart disease, 81 dogs met the criteria for inclusion in the current study. Two dogs were excluded because of the presence of atrial septal defects based on results of color Doppler interrogation of the interatrial septum. One dog was excluded for having overt myocardial dysfunction (fractional shortening [FS] = 13%), and 7 dogs were excluded for having peak aortic velocities >2.4 m/s. Of the remaining 81 Boxers, 43 were females (32 spayed) and 38 were males (19 castrated). The median age was 6.1 years (range, 2.1–11.0 years). No differences were observed among Boxer Groups I–III with respect to body weight (*P* = .34), sex (*P* = .59), or age (*P* = .17; Table 1). Arterial blood pressure measurement was performed in all Boxers in Groups II and III and in 12 of 44 dogs from Group 1. Median systolic blood pressure was 134 mmHg (range, 120–152 mmHg) for dogs in Group I, 142 mmHg (range, 122–168 mmHg) for those in Group II, and 140mmHg (range, 118–167mmHg) for those in Group III. Intergroup differences in arterial blood pressure were not significant (*P* = .40). Peak aortic velocities were recorded in all 81 dogs. Seventy-one of these dogs had peak aortic velocities recorded by CW Doppler from the left apical 5-chamber view, whereas 27 dogs had a peak aortic velocity recorded by CW from the subcostal position. Seventy-seven dogs had an aortic

[f]Systat 11.0, SPSS, Chicago, IL

velocity recorded by PW from the left apical view. In this study, no differences were identified between CW aortic velocities recorded from the subcostal or left apical positions ($P = .15$). Systolic murmurs were auscultated in 54 dogs (67%), with 34 dogs having I/VI, 18 dogs having II/VI, and 2 dogs having III/VI murmurs over the left heart base. Four dogs had I/VI systolic murmurs at the left cardiac apex. Forty-four dogs had no evidence of ventricular arrhythmia during the initial examination, echocardiogram, or diagnostic ECG, and were not evaluated further by AECG (Group I). AECG was performed in 37 dogs; 12 had <50 VPCs/24 hour (Group II; median, 6 VPCs; range, 1–30 VPCs) and 25 dogs had > 50 VPCs/24 hour (Group III; median, 527 VPCs; range, 66–40,063 VPCs). All raw M-mode echocardiographic measurements (eg, LVIDd, LVIDs, LVWd, LVWs, IVSd, IVSs, Ao, and LA) were significantly correlated with body weight ($r = 0.25–0.42$; $P < .05$). No significant correlations were found between any of the weight-based ERIs and body weight, with the exception of weak correlations of wLVIDd ($r = -0.27$; $P = .02$) and wAo ($r = -0.31$; $P = .004$). There was no significant correlation between body weight and aortic velocity ($r = -0.05$; $P = .66$).

Comparison of ERIs in Boxer and Non-Boxer Dogs

Compared with non-Boxer dogs, significant differences were present in most weight-based ERIs in Boxer dogs (Table 2). Specifically, the wAo ERI was smaller in Boxer dogs ($P < .001$) and weight-based ERIs that reflect increased LV wall thickness in diastole (wIVSd, wLVWd, wLVODd, wWTd, and wWAd) and systole (wIVSs, wLVPWs, wLVODs, wWTs, and wWAs) were significantly larger in Boxers compared with non-Boxers. FS was not statistically different between Boxers and non-Boxer dogs ($P = .13$), but relative indices of fractional wall thickness in systole ($P < .001$) and diastole ($P<.001$), and fractional wall area in systole ($P < .001$) and diastole ($P<.001$), were all significantly larger in Boxers compared with non-Boxer dogs. No significant correlations were noted between magnitude of LV wall thickness in the 81 Boxers and arterial blood pressure, aortic size, or aortic ejection velocity.

Comparative ERIs among Boxer Study Groups

ERIs for the 3 Boxer groups are depicted in Table 3. Raw M-mode measurements were not statistically different among the 3 Boxer groups, with the exception of EPSS, which was greater in Group III compared with Group II ($P = .03$). No significant differences were noted in any weight-based ERIs among groups. FS was lower in Group III compared with Groups I and II ($P = .01$). No significant differences were noted in aortic ($P = .20$) or pulmonic ($P = .053$) ejection velocities among the 3 Boxer groups.

Discussion

The results of this study demonstrate that overtly healthy Boxer dogs have relative increases in LV wall thickness and smaller aortas compared with normal non-Boxer dogs used to establish normal ERIs in a previously reported study.[18] Moreover, most of these measures are not significantly different among Boxer dogs with varying degrees of ventricular arrhythmia, and the magnitude of LV wall thickness appears to be unrelated to arterial blood pressure, aortic size, or ejection velocity.

Although linear regressions against body weight have been published for dogs to describe changes in echocardiographic lengths attributable to varying body size,[22,23] this method is invalid for a sufficiently wide range of body weights because of the nonlinear relationship between length and weight.[18,24–26] This may be true even among individuals of the same breed, especially for those breeds characterized by large heterogeneity of size,[12] thus requiring ERIs or other allometric scaling methods to compare M-mode measurements that are correlated with body weight. The Boxer dogs in this study varied more than 2-fold in their body weight from 42 to 89 pounds. All M-mode echocardiographic measurements were significantly correlated with body weight; however, weight-based ERIs, with the exception of wLVIDd and wAo, were not correlated with body weight. Thus, ERIs were useful not only to correct for body weight and allow comparison of echocardiographic measurements in Boxers versus non-Boxer dogs but also to compare M-mode parameters among individual Boxers of widely different size.

Boxers are routinely evaluated by echocardiography because of the high incidence of cardiac murmurs and the prevalence of congenital defects and cardiomyopathy in the breed.[1–3] Systolic heart murmurs were auscultated in 67% of the overtly healthy dogs in the current study and have been documented in 50–80% of dogs in previous analyses.[1,9,11,27] The results of this study also support the smaller aortic size reported previously.10,[b]

Smaller left ventricular outflow tract (LVOT) areas and increased aortic ejection velocities have been documented in Boxers relative to other dogs.[10] However, the echocardiographic diagnosis of subaortic stenosis is dependent on factors other than aortic velocity, such as the presence of an anatomic obstruction in the LVOT, turbulent flow in the proximal aorta, concurrent aortic regurgitation, or the appearance of LV hypertrophy.[28] Thus, there is no uniform consensus as to a specific cut-off aortic velocity for the diagnosis of subaortic stenosis, and differentiating mildly affected dogs from normal Boxers remains problematic. The aortic velocity limit of 2.4 m/s used to exclude dogs from this study was slightly higher than that used in previous studies that have used cut-off values from 2[1,2,27] to 2.3 m/s[29] to designate aortic stenosis. A wider range of aortic velocities was desirable in this study to allow for assessment of any relationship between echocardiographic measures of LV wall thickness and aortic velocity. Boxers in the current study were found to have significantly increased indices of LV wall thickness in diastole and systole, without a concurrent difference in LV cavity size, compared with normal non-Boxer dogs. LV concentric hypertrophy may result from fixed or dynamic aortic stenosis, systemic arterial hypertension, aortic coarctation, or hypertrophic cardiomyopathy, or may represent a breed-specific variation. The increased LV wall thickness observed in these dogs was independent of aortic velocity, aortic dimensions, and systemic arterial blood pressure measurements, suggesting a breed-specific variation that is independent of aortic stenosis or systemic hypertension.

Prior studies have documented the effects of variations in breed and body conformation, sex, age, and athletic training on M-mode echocardiographic measurements in the dog.[12,30–34] Increased LV wall thickness and LV cavity dimensions have been demonstrated in the Greyhound, and the degree of cardiac hypertrophy is more extensive in actively training Greyhounds.[14,30,31] Moreover, similar increases in LV wall thickness have been

documented in Whippets and Italian Greyhounds, dogs of different size but similar somatotypes.[34] These changes have been attributed variably to training-induced hypertrophy,[30] genetic factors,[30,34] or alterations in blood viscosity resulting in increased myocardial wall stress.[14] The specific conditioning status of these Boxers was not recorded, but no dogs were reported to be actively trained for athletic competition. The increased LV wall thickness in this population of Boxers most likely results from genetic factors, but additional studies are warranted to determine whether the degree of LV wall thickness is affected by activity level, age, or sex and whether the observed changes progress or remain constant over time. Breed-specific differences in logarithmically transformed M-mode measurements were not reported in the study by Cornell et al,[24] which included a group of 75 European Boxer dogs. Additional studies may be warranted to assess whether these findings hold true for a larger population of Boxers and whether differences are found in the cardiac structure of Boxers from different regions of the United States versus those in Europe or elsewhere.

The diagnosis of ARVC, the most common acquired heart disease in the Boxer,[4] is currently based on results of 24-hour AECG, but the number of VPCs used to define ARVC is controversial. Some studies have used a cut-off of >50 VPCs/24 hour, which is the number used in this study to classify Boxers in Groups II versus III.[5] Thirty-one percent of overtly healthy dogs screened for the present study were afflicted with ARVC based on this diagnostic criterion, and this is in agreement with the 36% incidence reported previously by Meurs et al.[4]

All of the dogs in the current study were overtly healthy with no clinical signs of cardiac disease. The Boxer group with >.50 VPCs/24 hour (Group III) had a lower FS compared with dogs in Groups I and II, and an increased EPSS compared with dogs in Group II. This finding suggests the possibility of concurrent myocardial dysfunction in the dogs with documented arrhythmia[7] resulting from primary myocardial disease or tachycardiomyopathy.

The current study has several limitations. Although all Boxers were prospectively evaluated by a single echocardiographer (J.R.), the normal non-Boxer dog ERIs used for comparison in this study were calculated based on retrospectively gathered M-mode data collected by multiple echocardiographers to establish reference ERIs.[18] The echocardiographer in the current study was one of several echocardiographers for the reference ERI study, but use of a single echocardiographer for both the Boxer population and the normal non-Boxer dogs would have been ideal to minimize the effect of interobserver variation.

An additional limitation is the lack of Holter data on the dogs in Group I, which leaves open the possibility that some of the dogs in this group had clinically relevant ventricular ectopy that was not documented during initial screening tests. In addition, we cannot completely exclude causes of ventricular arrhythmia other than ARVC in the Group III dogs, including other spontaneous cardiac, systemic, or abdominal diseases. However, all dogs were overtly healthy, and all Group III dogs were re-evaluated 6 weeks after the time of the original echocardiogram and remained free of outward evidence of concurrent disease. Additional limitations are that blood pressure measurements were not obtained on all dogs and aortic

velocities could not be obtained from the same location in all dogs because of varying chest wall conformations and limited cooperation in some patients. As a result, we cannot exclude the possibility that the aortic velocity of some dogs was underestimated, leading to inclusion of some dogs with mild SAS. Inclusion of dogs with mild SAS might have impacted the measurement of wall thickness, despite the fact that there were no significant correlations between aortic ejection velocity and indices of LV wall thickness. Finally, Doppler indices of diastolic function were not examined in this study, and although diastolic function has been evaluated previously in a population of normal Boxer dogs,[13] future studies investigating the relationship between LV diastolic function and LV wall thickness may be warranted.

The results of this study indicate that M-mode ERIs in Boxer dogs are different from those of non-Boxer dogs, with Boxer dogs having thicker LV walls and smaller aortas than non-Boxer dogs. These findings should be taken into account when screening Boxers for heart disease.

Acknowledgments

This work was supported in part by the Barkley Fund.

Appendix 1. Description and calculation of M-mode echocardiographic ratio indices

Index	Calculation	Description
wAo	Aom/Aow	Index of aortic root dimension
wIVSd	IVSd/Aow	Index of interventricular septal thickness, diastole
wLVIDd	LVIDd/Aow	Index of left ventricular internal dimension, diastole
wLVWd	LVWd/Aow	Index of left ventricular wall thickness, diastole
wIVSs	IVSs/Aow	Index of interventricular septal thickness, diastole
wLVIDs	LVIDs/Aow	Index of left ventricular internal dimension, systole
wLVWs	LVWs/Aow	Index of left ventricular wall thickness, systole
wLA	La/Aow	Index of left atrial dimension
wLVODd	(IVSd+LVIDd+LVWd)/Aow	Index of left ventricular outer dimension, diastole
wLVODs	(IVSs+LVIDs+LVWs)/Aow	Index of left ventricular outer dimension, systole
wWTd	(IVSd+LVWd)/Aow	Index of combined septal and left ventricular wall thickness, diastole
wWTs	(IVSs+LVWs)/Aow	Index of combined septal and left ventricular wall thickness, systole
wΔA	$(LVIDd^2-LVIDs^2)/Aow^2$	Index of change on left ventricular internal area, ie, short-axis stroke area
wWAd	$(LVODd^2-LVIDd^2)/Aow^2$	Index of left ventricular short-axis myocardial wall area, diastole
wWAs	$(LVODs^2-LVIDs^2)/Aow^2$	Index of left ventricular short-axis myocardial wall area, systole
FS	(LVIDd−LVIDs)/LVIDd	Fractional shortening, relative index of internal wall motion
FWTd	WTd/LVODd	Fractional LV myocardial wall thickness, diastole, relative index of LV wall thickness in diastole
FWTs	WTs/LVODs	Fractional LV myocardial wall thickness, systole, relative index of LV wall thickness in systole

Index	Calculation	Description
FWAd	$(LVODd^2-LVIDd^2)/LVODd^2$	Fractional LV myocardial wall area (short axis), diastole, relative index of LV wall thickness in diastole
FWAs	$(LVODs^2-LVIDs^2)/LVODs^2$	Fractional LV myocardial wall area (short axis), systole, relative index of LV wall thickness in systole

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Table 1

Summary data in 81 overtly healthy Boxer dogs.

Variable	Boxers
Weight	63.6 (41.6–89.2) lb 28.9 (18.9–40.5) kg
Age	6.1 (2.1–11.0) years
IVSd	1.21 (0.83–1.61) cm
LVIDd	3.90 (2.90–4.80) cm
LVWd	1.20 (0.90–1.55) cm
IVSs	1.63 (0.81–2.46) cm
LVIDs	2.45 (1.67–3.30) cm
LVWs	1.64 (1.22–2.16) cm
Ao	2.20 (1.82–2.69) cm
LA	2.44 (1.96–3.26) cm
LA2-D	3.48 (2.32–4.20) cm
EPSS	0.30 (0.09–0.72) cm
Aortic velocity (peak)	1.77 (1.14–2.37) m/s
Pulmonic velocity (peak)	1.10 (0.69–1.63) m/s

All data are presented as median (range).

IVS, interventricular septum; LVID, left ventricular internal dimension; LVW, left ventricular wall; Ao, standard aortic root; LA standard left atrial; LA2-D, two-dimensional left atrial; EPSS, E-point-to-septal separation. Letters s and d refer to systolic and diastolic determinations, respectively.

Table 2

Echocardiographic ratio indices for Boxers versus non-Boxer dogs.

ERI Variable	Boxers (n = 81)	Non-Boxers (n = 47)	*P*
wLVIDd	1.58 (1.21–1.90)	1.65 (1.31–1.86)	.378
WIVSd	0.50 (0.36–0.63)	0.43 (0.28–0.58)	<.001
WLVWd	0.49 (0.38–0.58)	0.41 (0.29–0.57)	<.001
WIVSs	0.66 (0.35–1.03)	0.60 (0.44–0.79)	.001
WLVIDs	1.01 (0.68–1.35)	1.08 (0.79–1.37)	.130
WLVWs	0.68 (0.52–0.87)	0.62 (0.43–0.77)	<.001
wAo	0.91 (0.74–1.08)	0.99 (0.81–1.43)	<.001
wLA	1.00 (0.80–1.30)	1.01 (0.80–1.27)	.607
wLVODd	2.55 (2.23–3.05)	2.46 (2.15–2.83)	.002
wLVODs	2.36 (2.05–2.80)	2.27 (1.98–2.58)	.002
wWTd	0.98 (0.75–1.17)	0.86 (0.68–1.12)	<.001
wWTs	1.35 (0.88–1.82)	1.22 (0.98–1.55)	<.001
wΔA	1.48 (0.87–2.20)	1.44 (0.79–2.11)	.767
wWAd	4.08 (2.78–5.78)	3.44 (2.67–5.08)	<.001
wWAs	4.57 (3.13–6.53)	4.06 (3.09–5.17)	<.001
FS	0.37 (0.21–0.51)	0.34 (0.25–0.50)	.130
FWTd	0.38 (0.31–0.48)	0.34 (0.28–0.44)	<.001
FWTs	0.57 (0.39–0.71)	0.53 (0.43–0.66)	<.001
FWAd	0.62 (0.52–0.73)	0.57 (0.48–0.69)	<.001
FWAs	0.81 (0.63–0.91)	0.78 (0.67–0.88)	<.001

All data are presented as median (range).

See Appendix 1 for further explanation of ERI terminology and calculations.

ERI, echocardiographic ratio index; w, weight-based; LVID, left ventricular internal dimension; IVS, interventricular septum; LVW, left ventricular wall; Ao, standard aortic root; LA, left atrial; LVOD, left ventricular outer dimension; WT, wall thickness; DA, change in left ventricular internal area; WA, wall area; FS, fractional shortening; FWT, fractional wall thickness; FWA, fractional wall area. Letters s and d refer to systolic and diastolic determinations, respectively.

Table 3

Comparative echocardiographic ratio indices among Boxer groups.

Variable	Group I (n = 44)	Group II (n = 12)	Group III (n = 25)	*P**
wLVIDd	1.61 (1.21–1.86)	1.55 (1.40–1.88)	1.61 (1.42–1.90)	.865
WIVSd	0.49 (0.36–0.62)	0.50 (0.41–0.63)	0.49 (0.36–0.58)	.315
WLVWd	0.49 (0.39–0.57)	0.49 (0.39–0.58)	0.50 (0.38–0.56)	.838
WIVSs	0.67 (0.48–1.03)	0.66 (0.58–0.91)	0.63 (0.35–0.87)	.171
WLVIDs	0.99 (0.73–1.34)	0.94 (0.68–1.21)	1.06 (0.89–1.35)	.074
WLVWs	0.67 (0.58–0.87)	0.70 (0.58–0.84)	0.67 (0.52–0.79)	.487
WAo	0.92 (0.79–1.05)	0.90 (0.74–1.05)	0.90 (0.76–1.08)	.331
WLA	1.00 (0.87–1.25)	0.98 (0.83–1.15)	1.01 (0.80–1.30)	.446
wLVODd	2.59 (2.23–2.87)	2.51 (2.41–3.05)	2.54 (2.35–3.00)	.953
wLVODs	2.36 (2.05–2.71)	2.35 (2.14–2.80)	2.37 (2.06–2.76)	.868
WWTd	0.98 (0.75–1.14)	1.00 (0.83–1.17)	0.98 (0.76–1.11)	.452
WWTs	1.37 (1.07–1.82)	1.43 (1.15–1.64)	1.32 (0.88–1.61)	.267
wΔA	1.58 (0.91–2.20)	1.48 (1.19–2.12)	1.44 (0.87–1.98)	.320
WWAd	4.17 (2.78–4.98)	4.15 (3.30–5.78)	3.91 (3.05–5.39)	.710
WWAs	4.57 (3.23–6.53)	4.68 (3.77–6.41)	4.51 (3.13–5.97)	.774
FS	0.37 (0.28–0.47)[a]	0.38 (0.28–0.51)[a]	0.33 (0.21–0.44)[b]	.010
FWTd	0.38 (0.34–0.48)	0.39 (.34–0.44)	0.37 (0.31–0.42)	.417
FWTs	0.58 (0.49–0.67)	0.58 (0.50–0.71)	0.55 (0.39–0.64)	.058
FWAd	0.62 (0.56–0.73)	0.63 (0.57–0.69)	0.61 (0.52–0.67)	.418
FWAs	0.82 (0.74–0.89)	0.82 (0.75–0.91)	0.80 (0.63–0.87)	.054

All data are presented as median (range).

Group I, clinically normal Boxers with no arrhythmia recorded during exam; Group II, clinically normal Boxers evaluated via AECG with < 50 VPCs/24 hour; Group III, clinically normal Boxers with > 50 VPCs/24 hour on AECG. ERI abbreviations are as defined in the footnote to Table 2.

* Overall *P*-value for the ANOVA performed comparing the 3 Boxer groups.

Different superscripts indicate significant differences in indices between one or more individual Boxer groups (eg, Group I versus Group II).