Effect of endurance training on cardiac morphology in Alaskan sled dogs

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Stepien, R. L., K. W. Hinchcliff, P. D. Constable, and J. Olson. Effect of endurance training on cardiac morphology in Alaskan sled dogs. J. Appl. Physiol. 85(4): 1368–1375, 1998.—The cardiac morphology of 77 conscious Alaskan sled dogs before and after 5 mo of endurance training (20 km/day team pulling a sled and musher) was studied using two-dimensional and M-mode echocardiography. Subgroups included dogs with at least one season of previous training (“veterans”) and dogs undertaking their first season of training (“rookies”). Training resulted in a significant (P < 0.05) decrease in resting heart rate (−15%) and significant increases in interventricular septal thickness (systole, 15%; diastole, 13%), left ventricular (LV) internal dimension in diastole (LVIDd, 4%), LV free wall thickness in systole (9%) and diastole (LVWd, 9%), and left atrial diameter (5%) in all dogs, but the increase in LVWd was greater in rookies (16%) than in veterans (7%). Training increased end-diastolic volume index (8%), LV mass index (24%), and heart weight index (24%) and decreased the LVIDd-to-LVWd ratio (−6%) but did not alter cardiac index. We conclude that increased LV mass attributable to LV dilation and hypertrophy is associated with endurance training in Alaskan sled dogs. Disproportionate LV wall thickening accompanying LV dilatation suggests that cardiac morphological changes are due to volume and pressure loading. These training-induced changes are similar to those documented in human athletes undertaking combined isometric and isotonic training and differ from studies of dogs trained on treadmills.

Left ventricular hypertrophy; echocardiography; athlete's heart; exercise; dog

The term “athletic heart” is used to describe the morphological and functional cardiac changes that occur in human athletes as a result of strenuous repetitive exercise. Electrocardiographic changes in trained human athletes are indicative of enhanced vagal tone and left and right ventricular enlargement (3, 38, 49, 54, 71). Cross-sectional and longitudinal studies of cardiac structure and function have found variation in echocardiographic changes induced by training. In general, endurance training has been associated with increased end-diastolic volume (EDV), increased stroke volume (SV), and development of a resting bradycardia (4, 19, 37). Morphological changes include increased left ventricular (LV) diameter and increased LV wall (LVW) and interventricular septal (IVS) thickness (17, 31), resulting in increased LV mass (LVM). When LVW thickness increase is marked, the resting echocardiogram of endurance athletes may be confused with some types of myocardial disease (25, 31, 43).

Athletic training is associated with increases in plasma volume in humans (11) and dogs (30, 33). Increased LVW thickness associated with athletic training has been attributed to compensatory hypertrophy, whereby increased LV volume leads to an increase in LVW stress and then to compensatory hypertrophy of the LVW (law of Laplace) (21). The type of exercise training appears to have an effect: isometric exercise (e.g., weight lifting) reportedly tends to result in concentric LV hypertrophy, whereas isotonic exercise (e.g., endurance running) tends to result in eccentric LV hypertrophy (35). Types of training that involve both isometric and isotonic aspects (e.g., rowing and cycling) have been reported to lead to LV changes that are both eccentric and concentric in nature (15–17), although these findings are not consistent in all studies (65).

Despite the historical use of dogs in athletic competition, game hunting, and sheep herding and as models for studying the effects of training on the heart, there have been conflicting reports regarding the effect of exercise on cardiac morphology and function in dogs (2, 6, 46, 48, 60, 61, 64, 70). Training has been consistently associated with significant decreases in resting heart rate (HR) and decreased HR response to exercise or atropine challenge in dogs (2, 30, 48, 61, 64, 70). Morphological changes include increased (46, 60, 70) or unchanged (48) LVM after training in non-greyhound breeds of dogs. LV diameter and LVW measurements were available in one study of mongrel dogs; LVW thickness increased with no change in LV EDV in response to training (70). Studies of greyhounds (a breed selected for racing ability) have documented greater LV internal diameter (LVID) and LVW and IVS thickness than other dog breeds of similar body weight (6, 41, 56). Variable effects on cardiac function in exercised dogs have been documented. Functional studies have reported no change in ejection fraction (EF) after training (30), increase in SV and cardiac output response to submaximal exercise testing after training (61), and significant increase in SV but not cardiac output at rest but improved function during maximal exercise (2), and increase in SV, EDV, and stroke work but no change in EF after training (48). These variable effects may be attributable to the type and duration of exercise training. Notably, no studies describe changes in dogs similar to those seen in human athletes who undertake both isometric and isotonic exercise (e.g., canoeists, cyclists, and rowers), no studies have de-
tallowed the echocardiographic findings associated with prolonged endurance training in dogs, and no studies involved a large population of exercising animals.

The purpose of this study was to investigate the effect of prolonged, strenuous endurance training on cardiac morphology of a large, relatively uniform population of Alaskan sled dogs. Alaskan sled dogs are mongrel dogs purpose bred for endurance and speed in races that may include daily runs of up to 160 km. Elite Alaskan sled dogs compete in the Iditarod Trail Sled Dog Race, an endurance event covering ~1,760 km in <10 days. Alaskan sled dogs provide a unique model of athletic training; they are genetically and nutritionally homogenous, they undergo long training periods of spontaneous exercise, and large numbers of similarly trained dogs can be studied at a single time point. Because Alaskan sled dogs perform extreme forms of endurance exercise while pulling a driver and laden sled, we speculated that exercise-training-induced changes in these dogs would be prominent and reflective of both isotonic and isometric exercise.

MATERIALS AND METHODS

Seventy-seven Alaskan sled dogs (51 males and 26 females) used for competitive, professional sled dog racing from five kennels were studied before (early September) and after (mid-March) 5 mo of training. Fifty-four dogs (70%) had trained at least one previous season (‘‘veterans’’). Veterans consisted of 32 males, 5 neutered males, 16 females, and 1 neutered female; median age in September was 4 yr (range 2–9 yr). Twenty-three dogs (30%, 14 male, all intact) had had no previous training (‘‘rookies’’); median age of rookies in September was 1 yr (range 7–18 mo). At the time of the September examination, all dogs had undergone 3 mo of rest from heavy training and had been subjected to <80 km of light training over the 2 wk before examination. Exercise during the 5-mo training period consisted of teams of 10–18 dogs pulling sleds containing a musher for ~3,886 ± 1,650 (SD) km, equivalent to a mean training distance of ~20 km/day. The dogs were trained and raced at ~16 km/h, resulting in an exercise intensity level of 30–40% of maximal O2 consumption (20), depending on snow conditions, air temperature, number of dogs in the team, and stage of training. Dogs were examined at their home kennel after 24 h of rest. All examinations took place between 9 AM and 7 PM.

Age and weight (kg) were recorded for each animal. All animals underwent routine thoracic auscultation; if a cardiac murmur was detected, the intensity and point of maximal intensity of the murmur were recorded. Cardiac murmurs were graded on a scale of 0–6 by a single examiner with the operator blinded to the dog’s identity. All values were measured to the nearest millimeter and were taken according to accepted echocardiographic standards for the dog (5, 40, 50, 63); recorded values represented the average of three consecutive beats. The measured beats were selected on the basis of quality of the recording and presence of a regular cardiac rhythm. The following parameters were recorded for each dog: IVSd thickness in systole and diastole (IVSd), LV free wall thickness in systole and diastole (LVWd), LV end-systolic volume (ESV, ml), and SV index (SVI, ml/kg body wt), EDV (ml), and EDV index (EDVI, ml/kg body wt) were calculated using a modified cube method that has previously been used to estimate ventricular volume in the dog (40, 62).

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V = (7.0(2.4 + d))^3
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where \(V\) is LV volume (ml) and \(d\) is LV diameter (cm). SV (ml) and SV index (SVI, ml/kg) were calculated as EDV – ESV and EDVI = ESVI, respectively, and cardiac index (CI, ml/min·1·kg\(^{-1}\)) was calculated as SVI × HR, where HR was derived from the mean of three consecutive R-R intervals on the electrocardiogram recorded during each dog’s measured M-mode echocardiogram. EF (%) was calculated as [(EDV – ESV) × 100]/EDV, and fractional shortening (FS, %) was calculated as [(LVWd – LVIDd) × 100]/LVIDd. LVM (g) and LVM index (LVMI, g/kg) were calculated according to the method of Devereux and Reichek (13).

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LVM = 1.04[(LVIDd + LVWd + IVSd) – (LVIDd)]^3 – 13.6^3\]

Total heart weight (HW, g) was estimated according to the data of Herrmann (22) as HW = LVM/0.46. LVM as percentage of body weight (LVM%BW) and heart weight as percentage of body weight (HW%BW) were calculated for each group.

Statistics. Data were analyzed using a two-way ANOVA for repeated measures. Factors in the design were previous training status (rookie vs. veteran) and time (September (preseason) vs. March (postseason)). Post hoc testing was performed using a Student-Newman-Keuls test. The level of significance was set at \(P < 0.05\) for all comparisons. Values are means ± SD. The means of rookie and veteran dogs were reported separately when a significant training status vs. time interaction was detected. Overall means were reported when no interaction was detected. Frequency of cardiac murmurs was compared using a \(\chi^2\) analysis (\(P < 0.05\)).

RESULTS

Body weight for rookie dogs increased significantly over the training period (9%, \(P < 0.05\)), whereas that of the veteran dogs did not (–2%; Table 1). Training resulted in a significant decrease in resting HR (15%) and a significant increase in heart size evident as increases in LVIDd (4%), IVSd (13%), IVS thickness in systole (15%), LVW thickness is systole (9%), and LA diameter (5%). Although rookies and veterans demonstrated significant increases in LVWd (16 and 7%, respectively), LVWd of veterans was significantly greater than that of rookies before 5 mo of training. After training, there was no difference in LVWd between the groups. There was a disproportionately greater increase in LVWd than in LVIDd (LVWd decreased significantly), but IVSd/LVWd was unchanged. Heart size, as estimated by calculated LVM, increased significantly with training (24%), as did the related values LVMI (24%), LV%BW (25%), and HW%BW (24%). The increase in heart size was associated with significant increases in EDVI (8%) and ESV (5%). Cardiac output, CI, SV, FS, and EF did not change significantly with training.
There was a significant effect of training on the occurrence and intensity of cardiac murmurs (Fig. 1). Twelve dogs (16%) had auscultable cardiac murmurs before training, and 35 dogs (45%) had cardiac murmurs after training (P < 0.05). There was an overall increase in murmur intensity, with one dog (1%) having a grade 3 or greater murmur before training and 19 (24%) dogs having such murmurs after training. Murmurs were systolic in timing and were loudest over the left base of the heart; some dogs had lower-intensity murmurs audible from the right side of the thorax.

**DISCUSSION**

We investigated the effect of prolonged, strenuous endurance training on the cardiac morphology of a large, relatively uniform population of Alaskan sled dogs. Five months of strenuous training (20 km/day) resulted in modest but significant increases in measured LV diameter, LVW, and IVS thickness, an increase in calculated LVM, and a decrease in resting HR, but no change in echocardiographically derived measures of LV function. Previous studies using forced-exercise animal models have been criticized, because they may have limited applicability to voluntarily exercising humans (45). Moreover, in human studies, questions regarding the effect of body conformation on the interpretation of echocardiographic data have been raised (45). Our use of working Alaskan sled dogs as a model of spontaneous exercise avoids some of these confounding factors. The dogs used in our study were of relatively uniform genetic background, temperament, nutritional status, and body conformation. The exercise training involved a heavy training schedule of ~20 km/day but was not free running; in all cases, dogs were teamed in harness to pull a sled or training wagon. It was not possible to standardize the load or team size across the training season.

Increased LVIDd, LVWd, and IVSd is a consistent finding in human endurance athletes (19, 31, 37, 49, 54) and dogs after athletic training (70). LV chamber enlargement is attributed to increases in blood volume secondary to the increased demand of working skeletal muscle; a 30% increase in blood volume has been...
documented after as little as 6 wk of training in beagles (30). LVW thickening is thought to be an adaptation of the myocardial cells to reduce wall stress associated with chamber dilation (21). In our dogs, the increase in LVWd in response to athletic training was proportionately greater than the increase in LVIDd, and LVW thickening was concentric in nature (LVWd and IVSd proportionally increased), suggesting that LVW thickening did not occur as an adaptation to LV dilation alone.

Previous studies have shown that exercise involving upper and lower body extremities of humans results in LV dilation with more LVW and IVS hypertrophy than would be expected as compensatory changes due to the increased wall stress of dilation (15, 34). Our findings, therefore, suggest that sled dogs, like rowers, canoeists, and cyclists, are exposed to an isometric load (i.e., pulling against resistance) as well as the isotonic load of running. Alaskan sled dogs sustain marked increases in systolic blood pressure during exercise (occasionally to >300 mmHg) while exhibiting only mild increases in mean arterial pressure (66), findings similar to those of rowers (7). In rowers, increases in systolic pressure were attributed to the influence of repetitive Valsalva maneuvers. Although quadrupeds have been used to study the physiological effect of exercise, the physiological effects of running in a quadruped may differ from that in bipedal runners. Addition of arm exercise to leg-only exercise (as is obligatory with quadruped animals) has been shown to increase aerobic demand in humans (23). Our findings suggest that echocardiographic changes noted in the trained Alaskan sled dogs are more typical of a “combined-load” human athlete than a pure runner. The combination of quadruped locomotion and the load of pulling a sled and musher during training appears to result in a significant isometric load in addition to the isotonic training of endurance running. Comparison to nonpulling dogs was not studied, so the exact contribution of the isometric load to the morphological changes documented is unknown. In some human athletes, “combination-training” sports may result in LVW thickening exceeding ranges considered to be indicative of primary myocardial hypertrophy (44). Because the changes in LVW and IVS thickness and LVID resulted in measurements that were typically at the upper end of the normal reported range for dogs of this size (40), the changes seen in our study animals are unlikely to be confused with pathological hypertrophy.

The LVWd was significantly thicker in veteran than in rookie dogs at baseline but was not different after training, and the increase in LVWd was proportionately greater in rookie dogs (16%) than in veteran animals (7%); this may indicate a true difference in hypertrophic response but more likely reflects lack of complete regression of hypertrophy induced by previous seasons’ training. Because age-matched sedentary controls were not available, the effect of age alone on the LV measurements of these dogs is unknown.

The significant increase in EDVI and LA size noted in our dogs is consistent with an increase in preload. Ventricular compliance was not measured in the present study; studies performed in human athletes and racing greyhounds suggest that hypertrophy associated with athletic training does not change or may increase diastolic compliance and function (6, 8, 14, 24, 26, 27, 47, 68). Mitral insufficiency cannot be ruled out as a cause for increased LA diameter and increased LVIDd in these dogs, but no gross valve abnormalities were noted in any dog during echocardiographic examination.

An ~24% increase in the calculated LVM occurred over 5 mo of training. Although growth may account for increases in heart mass in young animals (rookie dogs’ body weight increased significantly compared with that of veterans), there was no difference in relative increases in LVM between the two groups. This indicated that growth alone was not responsible for the observed increase in LV. The increases in LVMI were based on increased LV diameter and LVW thickness. LVM%BW (0.76% after training) was well above the ratio reported by Herrmann (0.37%) in 200 mongrel dogs (22). Part of this discrepancy may be due to inclusion of only one-half of the IVS in Herrmann’s measurement of LVM, but we are confident that an actual increase due to LV hypertrophy occurred on the basis of the documented increases in the variables used to calculate LVM.

Although the calculated HW%BW in Alaskan sled dogs before training is comparable to values commonly reported for the greyhound (41, 53, 59), caution must be used in interpreting calculated LV volume and LVM estimates as indicative of true volume or mass. We chose to use an LV volume estimation formula proposed by Teichholz and colleagues (62) that has previously been used in clinical studies of dogs (40) and the methods of Devereux and Reichek (13) to estimate...
LVM. Many methods of LVM and LV volume estimation using two-dimensional or M-mode echocardiographic measurements have been proposed in humans and dogs (9, 12, 14, 19, 29, 31, 37, 52, 55, 57, 69). Use of formulas involving longitudinal measurements was impractical in this study because of the technical difficulties associated with obtaining reliable and repeatable measures of LV longitudinal dimensions in awake, unsedated dogs (40). We chose to use the methods of Devereux and Reichek to estimate LVM because of the good correlation between mass calculated from LV M-mode measurements and postmortem findings in humans; also the calculation did not require use of an LV length measurement and was therefore rapidly and easily obtained in unsedated dogs. The intent of this study was to document any change in cardiac morphology or LV function resulting from 5 mo of training. Therefore, no attempts could be made to validate estimates as indicators of true LVM or LV volume. M-mode estimates of LV volume or LVM may over- or underestimate true volume or mass (32, 69), but systematic error in estimation would be expected to affect pre- and posttraining values equally, suggesting that the documented increase in LV volume and LVM is genuine.

The increase in estimated LVM in trained Alaskan sled dogs is less than the 45% increase in LVM sometimes seen in human endurance athletes (31) but markedly greater than the 9% increase in LVM seen after treadmill training in mongrel dogs (70). The dogs in this study consistently exercised for greater distances and over a much longer time period than the treadmill-trained dogs studied by Wyatt and Mitchell (70); these prolonged periods of intense exercise may have contributed to the more marked increase in LVM seen in our study. Caution must be used in comparing the data because of different methods of calculating LVM, but the increase in wall thickness seen in our dogs (9%) was greater than that noted in the dogs studied by Wyatt and Mitchell (7%); this difference may be the result of the combined load of running and pulling a sled in our study population, whereas Wyatt and Mitchell used running alone as training.

The degree of change in LVM that might be expected in a previously untrained athlete may have been partially obscured by the inclusion of many veteran dogs in this study. Detraining is reported to lead to relatively rapid regression of athletic cardiac hypertrophy (25, 31), but in some cases, some amount of measurable hypertrophy is thought to persist (42, 51). Although sled dogs are not intensively trained in the off-season, they are typically not sedentary animals and do not constitute a completely nonathletic control group. Persistence of previously acquired LV hypertrophy may account for the significantly higher LVWd documented in veteran dogs as opposed to rookie dogs at the first observation point, but veteran dogs also tended to weigh more than rookie dogs in September. The degree of difference between increases in LVM in our study population and the increases in LVM due to training in humans may be due to differences in training levels or methods of measurement. Alternatively, the canine heart and the untrained human heart may not hypertrophy with endurance training to a similar degree, inasmuch as dogs have an HW%BW (0.8%) that exceeds the overall mean for most mammals (0.59%) (58).

Calculated resting FS and calculated EF did not change, despite increases in LV cavity size. Mean FS before (23%) and after (24%) training was at the low end of the range reported by some authors for normal mongrel dogs [25 ± 5% (SD) (40)] and below the normal range reported by other authors [29–55% (5), 39 ± 6% (28), and 24–57% (36)] but similar to that reported for racing greyhounds [range 19–34% (6)]. Calculated EF was within the normal range for dogs (40). The low-to-normal functional indexes in our dogs at rest may reflect the physiological capability of the trained athlete to maintain resting cardiac output at a lower HR because of increased LV EDV (51).

CI in our dogs was unchanged by training but was higher than previously reported values for resting mongrel dogs (32, 61). Our population of dogs may have a higher CI than mongrel dogs, or this finding may be due to higher “resting” HR or may be an artifact of volume calculation based on echocardiographic assumptions (62). Mean HR decreased significantly after training but at both time points was higher than resting HR previously reported for conscious, restrained dogs (32, 61) and for conscious, standing Alaskan sled dogs (10); this elevated HR may have contributed to a higher resting CI. Sled dogs have been reported to have HR varying from 120 to 150 beats/min in the excited state (66); our findings are comparable. The stable calculated CI after training was expected; CI in athletes is typically unchanged from the pretraining values at rest but increases markedly when HR increases during exercise (4).

Resting HR decreased significantly with training, in agreement with previous studies in various species (1, 2, 10, 17, 70, 71). The decreased resting HR associated with athletic training in this study is likely to be an effect of increased vagal tone and decreased sympathetic tone (10, 71). Changes in autonomic tone may be direct or occur as adaptations to increases in SV but ultimately maximize diastolic filling and increase efficiency of cardiac performance (71). The 15% decrease in mean HR noted in these dogs is less dramatic than that seen in other studies (10, 70). HR in this study were recorded with the dogs restrained in lateral recumbency for the echocardiogram; restraint for an unfamiliar procedure may have induced an anxiety-related sinus tachycardia in some dogs, obscuring differences in resting HR. In addition, 54 of the dogs studied had been trained in previous seasons; these dogs may have retained some effects of previous training (42).

The percentage of dogs with an auscultable cardiac murmur increased from 16 to 45% after training. These findings are comparable to previous studies of Alaskan sled dogs (10), in which ~40% of highly trained Alaskan sled dogs auscultated had cardiac murmurs, and studies of human dynamic athletes, in which systolic murmurs were noted in 30–50% of trained individuals (25). Systolic murmurs occurring in athletes have been...
attributed to increases in $SV$ associated with athletic training (39). In our study, $SV$ tended to increase with no change in aortic diameter, suggesting that, in the trained athletic dog, left basilar ejection murmurs might be due to “relative stenosis” of the outflow tract. Outflow obstruction or mitral insufficiency was not systematically ruled out in these animals, but there was no direct evidence of gross valvular or outflow tract abnormalities on the two-dimensional echocardiogram of any dog, and significant cardiovascular abnormality seems unlikely in these high-performance athletes.

Limitations of this study include lack of an untrained control group and limitations of the technical aspects of echocardiography. Because of logistics of working with large numbers of privately owned animals, a truly comparable sedentary control group of Alaskan sled dogs was not available over the long time course of this study. However, the repeated-measures design of our study allowed us to detect relatively small changes in some variables.

The repeatability and precision of echocardiographic measurements in the detection of small changes associated with athletic training have been questioned (45). The inherent error associated with use of echocardiography to measure changes in cardiac morphology is a concern in any study involving small changes in echocardiographic measurements, and magnification of any variability or error occurs when measured values are used in formulas to calculate additional values. In our study, significant increases in some calculated variables (e.g., LVMI) are a reflection of significant changes in each underlying variable, but in some cases, statistically insignificant changes became statistically significant with mathematical manipulation to indexes (e.g., ESV). On the basis of other echocardiographic studies of dogs, the mean percent error in standard LV measurements is $\pm 4–6\%$ (40, 69), and most clinical echocardiograms have a spatial resolution of $\sim 1\text{ mm}$ with use of a 3.5-MHz transducer (67). Therefore, although echocardiography would not be expected to be a reliable method for differentiating an athletic canine subject from a sedentary one, our results indicate that echocardiography allows detection of a modest physiological response to endurance training. Another limitation concerns the intra- and interobserver variability of the echocardiographic examination. We eliminated interobserver variability by having one person record all echocardiograms on videotape, and we attempted to minimize intraobserver variability and bias by blinding the measurer to the status of the dogs.

Conclusion. LV and LA dilation, LV free wall and septal thickening, and increased LVMI result from endurance training in Alaskan sled dogs. Indicators of LV function did not change in the resting animal. Increases in LV systolic pressure related to pulling a load may play a role in modulation of the LVW and IVS hypertrophy that accompanied the LV dilation expected from running training alone, making this model of exercise comparable to human athletes undergoing combined isometric and isotonic training. The documented morphological changes, although unlikely to be of clinical significance in any individual dog, are indicative of a physiological response to a sustained exercise load in these spontaneously exercising dogs.

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REFERENCES


