Effects of endurance training on standard and signal-averaged electrocardiograms of sled dogs

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Objective—To determine the effect of endurance training on QRS duration, QRS-wave amplitude, and QT interval.

Animals—100 sled dogs in Alaska.

Procedure—Dogs were examined in early September (before training) and late March (after training). During the interim, dogs trained by pulling a sled with a musher (mean, 20 km/d). Standard and signal-averaged ECG were obtained before and after training.

Results—Endurance training significantly increased mean QRS duration by 4.4 milliseconds for standard ECG (mean ± SEM; 62.3 ± 0.7 to 66.7 ± 0.6 milliseconds) and 4.3 milliseconds for signal-averaged ECG (51.5 ± 0.7 to 55.8 ± 0.6 milliseconds) without changing body weight. Increase in QRS duration corresponded to a calculated increase in heart weight (standard ECG, 23%; signal-averaged ECG, 27%). Signal-averaged QRS duration was correlated with echocardiographically determined left ventricular diastolic diameter for the X orthogonal lead (r = +0.41), Y orthogonal lead (r = +0.33), and vector (r = +0.35). Training also increased QT interval (234 ± 2 to 249 ± 2 milliseconds) and R-wave amplitude in leads II and rV_2, increased peak-to-peak voltage and S-wave amplitude in the Y orthogonal lead, and decreased Q-wave amplitude in the Y orthogonal lead.

Conclusions and Clinical Relevance—Electrocardiographic changes reflected physiologic cardiac hypertrophy in these canine athletes in response to repetitive endurance exercise. The QRS duration increases in response to endurance exercise training and, therefore, may be of use in predicting performance in endurance activities. (*Am J Vet Res* 2000; 61:582–588)

Endurance training induces major cardiovascular adaptations that are manifest as auscultable cardiac murmurs, bradyarrhythmias, an increased R-wave amplitude in selected electrocardiographic leads, increases in QRS duration and QT interval, and echocardiographic evidence of cardiac chamber enlargement.[1-6] These changes are believed to reflect functional and morphologic adaptations of an athlete's heart in response to sustained endurance exercise rather than evidence of cardiovascular disease.[3] Of interest is whether measurement of these functional and morphologic changes provides a useful method for predicting performance in endurance activities.

Endurance-trained sled dogs provide an excellent model for examining cardiovascular adaptations to endurance exercise.[7,8] We have reported[7] that elite canine endurance athletes have an increased frequency of auscultable cardiac murmurs, increased R-wave amplitude in selected electrocardiographic leads, and increased QRS duration and QT interval, compared with untrained dogs. We also reported[9] that free-ranging gray wolves (*Canis lupus*), the archetypical endurance athlete, have a markedly longer QRS duration and QT interval than captive wolves. The increase in QRS duration concomitant with endurance training and activity is of particular interest, because QRS duration was correlated with heart weight in 49 humans (r = 0.43),[10] 155 Greyhounds (r = 0.73),[11] and 55 horses (r = 0.92)[12]; thickness of the **left ventricular (LV)** wall in 49 humans (r = 0.66)[10]; and cardiac transverse diameter in 41 humans (r = 0.46).[5] Analysis of those findings suggests that QRS duration may be correlated with endurance performance, because cardiac size is correlated with maximal cardiac output and, therefore, maximal oxygen consumption and endurance performance.[1] Support for this hypothesis is provided by results of prospective studies that indicated QRS duration accounts for 18% of the variation in race performance in sled dogs[13] and 9 to 25% of the variation in race performance in horses.[12,14,15]

The concept that QRS duration is of predictive value for endurance activities remains controversial and has not been widely accepted by human and veterinary cardiologists.[16-21] The QRS duration is dependent on ventricular size (and, therefore, age and body weight), ventricular activation sequence, heart rate, autonomic tone, electrolyte status, and effects of cardioactive drugs or pathologic cardiac changes.[7,11,12,14,15,22-24] Although there is a clear dependence of QRS duration on age and body weight in mammals,[25] it is believed that QRS duration in mature animals is not altered by endurance training.[4,26] On the basis of our results from cross-sectional studies in sled dogs[7] and gray wolves,[9] we hypothesized that a longitudinal study would confirm that QRS duration increases with endurance training. Accordingly, our major hypothesis was that endurance training increases QRS duration in sled dogs. An addi-

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tional hypothesis was that endurance training would induce other changes in the ECG such as an increase in QT interval, R-wave amplitude, or prevalence and duration of late potentials. Finally, we examined the correlation between QRS duration and echocardiographic measurement of LV internal diastolic diameter and estimation of LV mass to confirm that increased QRS duration reflects an increase in cardiac size.

Materials and Methods

Dogs—A group of 118 sled dogs, comprising 67 mature dogs (≥ 3 years old) and 51 young dogs (1 to 2 years old), were available for examination in Alaska in early September 1994 (before training) and in late March 1995 (after training). Young dogs had not been extensively trained prior to initial examination, whereas mature dogs had all undergone ≥ 1 complete season of endurance training. The sled dogs were representative of elite canine endurance athletes, and 58 raced as members of sled dog teams in the Iditarod Trail race during the study period.

At the time of initial examination in September, all dogs had been rested from heavy training for ≥ 3 months and performed < 80 km of light training during the 2 weeks prior to examination. Each dog performed endurance training and racing during the study period, ranging from 673 to 6,034 km (mean ± SEM, 3,886 ± 165 km), which was equivalent to 20 km/d. Training and racing involved teams of 4 to 16 dogs pulling a sled with a musher at an approximate speed of 16 km/h, depending on snow conditions, ambient temperature, number of dogs in the team, and stage of training. The training speed represented an exercise intensity of 30 to 40% of maximum oxygen consumption.[27]

Dogs were examined ≥ 24 hours after exercise during daylight hours, and they were fed and provided water in accordance with their normal schedule on the day of examination. Standard and signal-averaged ECG were recorded with the dogs in standing position, because examinations frequently were conducted outside and because of our experience that unsedated sled dogs resist restraint in lateral or sternal recumbency. Data recording in this manner also permitted the heart to be physiologically positioned within the thorax.[28] Electrocardiograms were recorded at ambient temperatures between –23 and –7 C. Echocardiograms were recorded for 77 dogs, and those results were reported elsewhere.[29]

Standard electrocardiography—Electrocardiograms were obtained with all dogs in a standing position, the forelimbs and hind limbs perpendicular to the ground, all limbs separated, and the head and neck parallel to the ground and in the same plane as the long axis of the body. The ECG leads were attached with alligator clips to each dog's skin at the palmar aspect of the left and right forelimb over the olecranon, the cranial aspect of the left and right hind limb over the patellar ligament, the left sixth intercostal space at the chondrosternal junction (V_2 or CV_6LL), the left sixth intercostal space at the costochondral junction (V_4 or CV_6LU), the right fifth intercostal space near the edge of the sternum (rV_2 or CV_6RL), and the dorsal spinous process of the seventh thoracic vertebra (lead V_{10}). Points of electrode attachment were saturated with a solution of 70% alcohol, and ECG were recorded by use of an electrocardiographic unit[a] with a frequency range of 0.5 to 150 Hz. A high-pass filter of 0.5 Hz was used to minimize baseline wander, which was commonly seen in trained sled dogs. When directed, the electrocardiographic unit recorded and stored, in digitized form at 500 Hz with 16-bit precision, signals from leads I, II, III, aVR, aVL, aVF, V_2, V_4, rV_2, and V_{10} obtained throughout a 10-second period. This analog-to-digital conversion procedure is adequate for accurate computer processing of the ECG.[30] The resultant ECG was printed (25 mm/s). After thorough inspection of the calculated amplitude and duration values, data were extracted from the digital record for subsequent analysis.

The ECG used an averaged representative beat from each lead to determine the onset and end of P, QRS, and T waves in each lead.[31] Durations of the QRS wave and PQ or QT intervals were determined to the nearest millisecond for each lead. Values for QRS duration, PQ interval, and QT interval used for final data analysis were determined from the ECG, using a weighted mean for the 10 recorded leads. This method of analysis produces values for P-wave and QRS duration and amplitude and QT-interval duration identical to those obtained by cardiologists,[32] with a smaller coefficient of variation for repeated measures, particularly the QRS duration.[32,33] Therefore, the analysis procedure was the most appropriate method for detecting small changes in QRS duration. The value for **QT interval corrected for heart rate (QT_c)** was determined, using Bazett's formula as follows:

$$QT_c = QT/\sqrt{(R\text{-}R)}$$

with QT and R-R intervals measured in seconds.[34] Because QT and QT_c are dependent on heart rate, we also calculated QT_{index}, which provides a measure of duration of electrical systole independent of heart rate such that $QT_{index} = (QT + QT_c)/2$.[7] Mean electrical axis of ventricular depolarization in the frontal plane was estimated by plotting the net value for QRS complexes from leads I and III and determining the direction of the resultant vector.

Signal-averaged electrocardiography—Signal-averaged ECG were obtained, using the orthogonal lead system developed by McFee and Parungao.[35] This is the only lead system specifically designed for the thorax of dogs that meets the conditions of mutual orthogonality of axes and parallelism with the corresponding anatomic axes,[28,35] and it has been used successfully in other studies in dogs.[28,36,37] The technique is reproducible with only minor day-to-day variations.[28]

Each dog was brought into a quiet area and encouraged to stand on a rubber mat. Forelimbs and hind limbs were placed perpendicular to the ground, all limbs were separated, and the head and neck were positioned parallel to the ground and in the same plane as the long axis of the body. Electrode paste was applied to the skin over the sites of electrode placement, but the hair was not clipped, because dogs had to train, race, and sleep in ambient temperatures as low as –60 C. Alligator clip electrodes were applied[28,35,36] as follows:

X lead (left to right): 4 electrodes; 1 positive electrode placed at the left 3rd and another at the left 6th intercostal spaces at the costochondral junction, and 1 negative electrode placed at the right 3rd and another at the right 6th intercostal space at the costochondral junction.

Y lead (cranial to caudal): 2 electrodes; 1 positive electrode placed on the lateral aspect of the left hind limb in the region of the stifle joint, and 1 negative electrode placed on the lateral aspect of the left side of the neck.

Z lead (dorsal to ventral): 4 electrodes; 1 positive electrode placed at the dorsal spinous process of the 6th thoracic vertebrae, and 3 negative electrodes placed, respectively, at the right and left sternal junctions of the 7th ribs and on the ventral aspect of the manubrium at a distance necessary to create an equilateral triangle centered immediately ventral to the heart.

Ground electrode: cranial aspect of the stifle joint on the right hind limb.

Electrode potentials were averaged (X and Z leads), using precision resistors (± 2%). A representative template beat was selected from the X lead axis, and subsequent beats were signal averaged until a mean noise value of 4 μV for the 3 orthogonal leads was attained. Reduction of noise by signal

averaging is proportional to the square root of the number of averaged cycles.[38]

Analysis of results of preliminary studies indicated that 4 μV was the lowest noise value that could be reasonably and consistently achieved in unsedated sled dogs in standing position with unclipped hair.

The 3 orthogonal lead voltages were amplified and recorded, using band-pass filtering, from 0.05 to 300 Hz and digitized at 2 kHz with 16-bit precision. The offset potential was determined from the offset voltage of the midpoint of the PR segment. Orthogonal lead voltages then were reset to zero (based on the offset potential), using custom-designed software, and the Q-, R-, and S-wave amplitude and peak-to-peak voltage amplitude were determined. Peak-to-peak amplitude was the larger of the sum of the modulus of Q- and R-wave voltages or the sum of the modulus of R- and S-wave voltages. Instantaneous spatial magnitude (in millivolts) was calculated from the orthogonal lead voltages as follows:

$$\text{spatial magnitude} = \sqrt{(X^2 + Y^2 + Z^2)}$$

Instantaneous value for spatial magnitude in millivolts represents the signal-averaged, time-varying vector lead amplitude.

Signals for signal-averaged orthogonal and vector leads were filtered by use of a high-pass frequency of 40 Hz to reject the low-frequency activity in the ST segment and, thereby, enhance the frequency component of terminal ventricular activation associated with late potentials. The QRS duration then was determined by manual inspection of the filtered orthogonal and vector leads, without knowledge of each dog's identity, age, or training status. Duration of late potentials was defined as the time that the terminal portion of the QRS complex for the filtered orthogonal or vector lead remained < 40 μV. A late potential was defined as being evident when the duration of the late potential for a filtered vector lead exceeded 18 milliseconds and QRS duration exceeded 58 milliseconds.[39] These values approximate half of the values defined in human studies.[39]

Echocardiography—Two-dimensional and M-mode echocardiography was performed on 77 of the dogs. The LV internal diastolic diameter was determined and the LV mass calculated, as described elsewhere.[29]

Statistical analyses—All continuous data were reported as mean ± SEM and represent data from all dogs. Continuous data were analyzed by use of repeated-measures ANOVA. Data were tested for normality by calculating the Shapiro-Wilk statistic, and variables with a nonnormal distribution or unequal variances were log transformed before ANOVA was performed. Whenever the F ratio for main effects (age, 2 categories; training, 2 categories) or their interaction term was found to exceed the critical value ($P < 0.01$), appropriate comparisons were made, also using a value of $P < 0.01$. When the Shapiro-Wilk statistic indicated that the transformation procedure failed to produce an approximately normal distribution, data were compared by use of nonparametric methods (Friedman test and appropriate comparisons by the Wilcoxon signed rank test). All post hoc tests of values before and after training were conducted on paired data. A value of $P < 0.01$ was selected for all analyses because of the number of potentially correlated statistical comparisons made from the same data set.

Correlation between QRS duration (from standard and signal-averaged electrocardiography) and LV internal diastolic diameter or $\sqrt[3]{}$LV mass (from echocardiography) was determined, using values from the before-training data, because this provided the largest data set. The $\sqrt[3]{}$LV mass was used, because a strong linear association exists between LV chamber length and $\sqrt[3]{}$heart weight,[10] and exercise-induced cardiac chamber enlargement will increase LV chamber length, thereby producing a longer pathway for ventricular depolarization. This will result in increased QRS duration, assuming there has not been a change in myocardial conduction velocity.[40]

Results

Dogs—Of the 118 dogs originally examined before training, 100 were available for examination at the end of the training season. Eighteen dogs were not available for examination, because they were injured, died, or had been sold to other sled dog mushers. Fifty-eight dogs ran in the 1995 Iditarod Trail race in teams that finished 2nd, 6th, 10th, 15th, and 23rd (57 teams competed in the race). The Iditarod Trail race is widely regarded as the premier long-distance sled dog race in the world, and the 1995 race was completed in record time. There was not a detectable change in body weight with training (**Table 1**).

Table 1—Effect of training on body weight and electrocardiographic findings in 100 sled dogs

Variable	Before training	After training	*P* value
Body weight (kg)	23.6 + 0.3	23.9 + 0.4	NS
Heart rate (beats/min)	120.7 + 2.1	116.9 + 2.3	NS
PR interval (ms)	114.7 + 1.7	114.4 + 1.4	NS
QRS duration (ms)	62.3 + 0.7	66.7 + 0.6	< 0.001
QT interval (ms)	234.3 + 1.9	248.9 + 2.2	< 0.001
QT_c (ms)	333.0 + 2.2	346.1 + 2.6	< 0.001
QT_{index}	283.6 + 1.7	297.5 + 1.9	< 0.001
Mean electrical axis (°)	56 + 4	59 + 3	NS

Values are mean ± SEM. NS = Not significant. QT_c = QT interval corrected for heart rate, using Bazett's formula: $QT/\sqrt{(R–R)}$

Table 2—Effect of training on the amplitude of Q, R, and S waves in semiorthogonal leads (I, aVf, V_{10}), chest leads (V_2, V_4, rV_2), and lead II and on the *P*-wave amplitude in lead II in 100 sled dogs

Lead	Before training	After training	*P* value
Semiorthogonal leads			
Lead I			
Q wave (mV)	0.44 + 0.04	0.41 + 0.04	NS
R wave (mV)	1.48 + 0.07	1.62 + 0.08	NS
S wave (mV)	0.00 + 0.00	0.00 + 0.00	NS
Lead aVf			
Q wave (mV)	0.60 + 0.03	0.54 + 0.04	NS
R wave (mV)	2.40 + 0.06	2.63 + 0.08	NS
S wave (mV)	0.09 + 0.02	0.11 + 0.02	NS
Lead V_{10}			
Q wave (mV)	1.07 + 0.03	1.00 + 0.03	NS
R wave (mV)	0.49 + 0.02	0.52 + 0.03	NS
S wave (mV)	0.00 + 0.00	0.00 + 0.00	NS
Chest leads			
Lead V_2			
Q wave (mV)	0.03 + 0.01	0.02 + 0.01	NS
R wave (mV)	2.67 + 0.08	2.97 + 0.09	NS
S wave (mV)	0.64 + 0.04	0.66 + 0.04	NS
Lead V_4			
Q wave (mV)	0.15 + 0.02	0.20 + 0.02	NS
R wave (mV)	3.97 + 0.09	4.23 + 0.14	NS
S wave (mV)	0.53 + 0.04	0.53 + 0.05	NS
Lead rV_2			
Q wave (mV)	0.01 + 0.01	0.00 + 0.00	NS
R wave (mV)	1.58 + 0.06	1.82 + 0.06	0.004
S wave (mV)	0.04 + 0.01	0.04 + 0.01	NS
Lead II			
P wave (mV)	0.34 + 0.01	0.39 + 0.01	0.004
Q wave (mV)	0.73 + 0.04	0.73 + 0.05	NS
R wave (mV)	3.00 + 0.07	3.30 + 0.09	0.008
S wave (mV)	0.04 + 0.01	0.04 + 0.01	NS

See Table 1 for key.

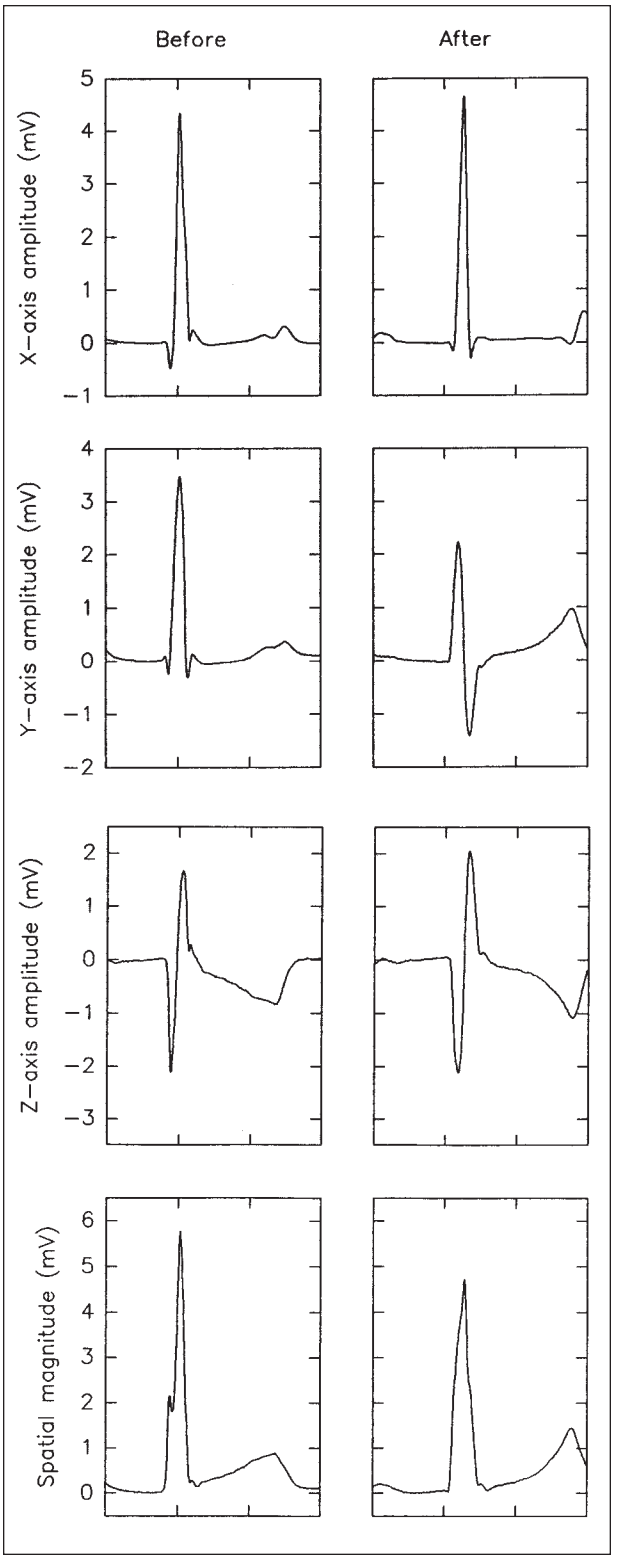

Figure 1—Representative signal-averaged QRS complex obtained from the orthogonal leads (X, Y, and Z axes) and vector (spatial magnitude) of a mature dog before and after endurance training. The x-axis is time (in 100-millisecond divisions).

Standard electrocardiography—Electrocardiograms were obtained from the 100 dogs (40 young, 60 mature) that were available before and after training.

Table 3—Effect of training on signal-averaged electrocardiographic values in 100 sled dogs

Variable	Before training	After training	*P* value
Number of beats averaged	102 + 8	117 + 7	NS
Noise value (μV)	3.83 + 0.06	4.02 + 0.02	NS
QRS duration (ms)			
X lead	51.1 + 0.6	56.6 + 0.6	< 0.001
Y lead	49.4 + 0.7	53.2 + 0.6	< 0.001
Z lead	48.7 + 0.6	53.9 + 0.6	< 0.001
Vector	51.5 + 0.7	55.8 + 0.6	< 0.001
Late potential duration (ms)			
X lead	6.2 + 0.5	10.6 + 1.2	< 0.001
Y lead	4.9 + 0.6	6.7 + 1.2	NS
Z lead	4.8 + 0.5	7.2 + 0.5	NS
Vector	3.2 + 0.4	5.3 + 0.5	0.005
QRS complex amplitude (mV)			
X peak-to-peak	4.16 + 0.16	4.58 + 0.17	NS
Y peak-to-peak	3.03 + 0.08	3.71 + 0.10	< 0.001
Z peak-to-peak	3.63 + 0.10	3.97 + 0.13	NS
X lead amplitude (mV)			
Q wave	0.56 + 0.04	0.70 + 0.06	NS
R wave	3.56 + 0.12	3.79 + 0.13	NS
S wave	0.17 + 0.03	0.16 + 0.02	NS
Y lead amplitude (mV)			
Q wave	0.44 + 0.06	0.03 + 0.01	< 0.001
R wave	2.40 + 0.08	2.58 + 0.08	NS
S wave	0.37 + 0.04	1.09 + 0.06	< 0.001
Z lead amplitude (mV)			
Q wave	2.43 + 0.07	2.54 + 0.09	NS
R wave	1.20 + 0.07	1.43 + 0.07	NS
S wave	0.0 + 0.0	0.0 + 0.0	NS
Peak spatial magnitude (mV)	4.56 + 0.12	4.68 + 0.12	NS

See Table 1 for key.

Studied variables were not significantly affected by age status of dogs; therefore, data for young and mature dogs were pooled for additional comparisons between values before and after training. Paired comparisons were only made on the 100 dogs for which ECG had been obtained before and after training.

Training significantly increased mean QRS duration by 4.4 milliseconds and significantly increased mean QT interval, QT_c interval, and QT_{index} by 14.6, 13.1, and 13.9 milliseconds, respectively (Table 1). Changes in heart rate, PR interval, and mean electrical axis were not observed.

Training significantly increased the P-wave amplitude in lead II and R-wave amplitude in leads II and rV_2 (**Table 2**). The R-wave amplitude appeared to increase in all other leads, but these increases were not significant.

Signal-averaged electrocardiography—Acceptable signal-averaged ECG recordings (**Fig 1**) were not obtained from all 100 dogs because of suboptimal skin surface preparation at the site of electrode placement and frequency of movement in standing unsedated dogs. Specifically, acceptable data for the X, Y, and Z orthogonal leads and the vector, respectively, were obtained from 94, 67, 79, and 62 dogs before training and 95, 94, 91, and 91 dogs after training. There was approximately a 10-fold reduction of noise as a result of signal averaging (**Table 3**).

Because age did not significantly affect variables in the study, data for young and mature dogs were pooled and comparisons were made between values for before and after training. Training increased QRS duration by 5.5, 3.8, 5.2, and 4.3 milliseconds in the X, Y, and Z

orthogonal leads and the vector, respectively (Table 3). Peak-to-peak voltage was increased in the Y lead, because of an increase in the S-wave amplitude, which was accompanied by a decrease in Q-wave amplitude (Fig 1). Peak-to-peak voltage in the X and Z leads appeared to increase, but not significantly. Changes in spatial magnitude were not observed.

Duration of late potentials was mildly increased in the X lead and vector after training (Table 3). Late potentials were not detected in any dogs before training, whereas late potentials were evident in 1 mature dog after training (duration of late potentials, 26 milliseconds; QRS duration 72 milliseconds). That dog had frequent unifocal single ventricular premature complexes before and after training but had trained and raced well.

Association between QRS duration and LV internal diastolic diameter—The QRS duration had a significant positive, but weak, correlation with LV internal diastolic diameter for the X orthogonal lead ($r = +0.41$, $P < 0.001$), Y orthogonal lead ($r = +0.33$, $P = 0.007$), and vector ($r = +0.35$, $P = 0.006$). The QRS duration also was positively correlated with the Z orthogonal lead, but not significantly ($r = +0.25$, $P = 0.049$).

Relationship between QRS duration and $\sqrt[3]{}$(LV mass)—The QRS duration was positively, but weakly, correlated with $\sqrt[3]{}$(LV mass) for the X orthogonal lead ($r = +0.34$, $P = 0.007$), Y orthogonal lead ($r = +0.35$, $P = 0.004$), and vector ($r = +0.35$, $P = 0.006$). The QRS duration was positively correlated, but not significantly, with $\sqrt[3]{}$(LV mass) for standard electrocardiography($r = +0.31$, $P = 0.020$) and the Z orthogonal lead ($r = +0.24$, $P = 0.045$).

Discussion

Major findings of the longitudinal study reported here were that endurance training of sled dogs was accompanied by increases in QRS duration, QT interval, QT_c interval, QT index, and R-wave amplitude in leads II and rV_2, peak-to-peak voltage and S-wave amplitude in the Y orthogonal lead, P-wave amplitude in lead II, and a decreased Q-wave amplitude in the Y orthogonal lead. Our finding that values before training were similar in mature and young dogs suggested that the observed changes were reversible, because all mature dogs had undergone ≥ 1 full season of endurance training followed by a 3-month deconditioning period before the initial examination. We believe that these changes reflect physiologic and reversible hypertrophy of the canine athlete's heart in response to endurance training.

Most longitudinal and cross-sectional studies in which cardiac effects of endurance training were evaluated revealed a reversible increase in heart weight, stroke volume, and maximal cardiac output, decreased resting heart rate, unchanged PR interval, increased LV chamber diameter and wall thickness, and increased myocardial cell diameter and myocardial capillary density.[2,11,41-43] The extent of these changes are related to the intensity and duration of the training program and the species involved.[2] Studies in which the association between QRS duration and endurance training in mature animals was examined have all been cross-sectional.[7,9,12,14,15,22-24,26] We believe that the study reported here represents the first longitudinal study in animals to examine the effect of endurance training on QRS duration.

We used 2 methods to determine QRS duration in this study (ie, standard ECG and signal-averaged ECG). Although both techniques produced similar increases in mean QRS duration with endurance training (standard ECG, 4.4 milliseconds; signal-averaged ECG, 4.3 milliseconds), the measured QRS duration was longer when we used standard electrocardiography. We attributed this difference to technical issues regarding accurate determination of the junction point, which marks the end of the QRS complex and start of the ST segment. Merging of the terminal part of the QRS complex with the ST segment is common in the ECG of dogs, making computer algorithm determination of the end of the QRS complex problematic.[30] The mean QRS durations measured in this longitudinal study (before training, 62.3 milliseconds; after training, 66.7 milliseconds) agreed closely with measurements we obtained in a cross-sectional study[7] in which we used another computer algorithm ECG system[b] and another sled dog population (untrained sled dogs, 62.5 milliseconds; endurance-trained sled dogs, 66.1 milliseconds).

The QRS duration in humans varies with heart weight in accordance with the following equation: QRS duration in milliseconds = $10 \times \sqrt[3]{}$(heart weight in grams).[10] Applying this equation to dogs, an increase in mean QRS duration from 62.3 to 66.7 milliseconds (standard ECG) or from 51.5 to 55.8 milliseconds (signal-averaged ECG) corresponded to an estimated increase in heart weight of 23 or 27%, respectively. These estimates agreed closely with the 24% increase in heart weight calculated from echocardiographic measurements in the same sled dog population.[29] Moreover, QRS duration for the X and Y orthogonal leads and vector was positively correlated with LV internal diastolic diameter and $\sqrt[3]{}$(LV mass), as estimated by use of echocardiography.[29] Accurate determination of QRS duration is required if inferences are to be made concerning changes in heart size and weight, because small increases in QRS duration (4 milliseconds) correspond to relatively large increases in heart size and weight (25% increase). This may be best appreciated by examining the small range for QRS duration in mammals (eg, mouse, 22 milliseconds; elephant, 160 milliseconds), relative to the large range for body weight in mammals (eg, mouse, 23 g; elephant, 6,650,000 g).[25,39] The requirement for accurate determination of QRS duration may limit the utility of QRS duration as a predictor of endurance performance.

The QT and QT_c intervals of human endurance athletes are prolonged by 10 to 15%, with the degree of prolongation being training-dependent.[6] In the study reported here, QT and QT_c intervals were prolonged by 6 and 4%, respectively, compared with values obtained before training. Lengthening of the QT interval can be caused by changes in heart rate, cardiac size, autonomic tone, serum electrolyte concentration (particularly

calcium and potassium ions), and administration of drugs that alter duration of cardiac action potentials.[7,44] Effects of cardioactive drugs or abnormal serum electrolyte concentrations were discounted as reasons for the prolonged QT interval, because drugs had not been administered, and serum electrolyte concentrations were assumed to be within the reference ranges at the time electrocardiography was performed on these apparently healthy dogs. Heart rate effects were also discounted as possible causes for the prolonged QT interval, because heart rate was not significantly different before and after training, and analysis of the index independent of heart rate (QT_{index}) indicated that electrical systole was significantly longer after training. Therefore, endurance training induced a prolonged QT interval because of cardiac enlargement, changes in autonomic tone, or both. Clinical importance of a prolonged QT interval in endurance athletes remains uncertain,[6] despite the association between prolongation of QT interval and sudden death in humans with and without coronary artery disease.[45]

Endurance training usually decreases the resting heart rate as a result of changes in autonomic tone (specifically increased vagal tone and decreased sympathetic tone).[6,13,43] Our failure to observe a decrease in heart rate with training in this study was attributed to difficulty in obtaining a true resting state, because ECG were usually obtained after echocardiographic examination, and dogs were not restrained for electrocardiography by the musher. A significant reduction in heart rate was observed after endurance training when the dogs had echocardiography performed, with the dogs being restrained by the musher in lateral recumbency.[29] Mean heart rates for endurance-trained sled dogs in this study (117 beats/min) were significantly higher than those obtained when sled dogs were restrained by the musher (102 beats/min)[7] and much higher than those obtained during sleep (40 to 60 beats/min).[8]

Signal-averaged electrocardiography can be useful as a screening test in predicting the risk of spontaneous ventricular tachycardia, ventricular fibrillation, and sudden cardiac death in humans and dogs with focal areas of myocardial ischemia.[38,39] Signal-averaged echocardiography is based on the hypothesis that abnormal ventricular activation can be detected on the body surface, and that this abnormality is associated with an increased incidence of life-threatening ventricular arrhythmias, because prevalence and duration of late potentials recorded by signal-averaged ECG are correlated with the incidence of sudden cardiac death in humans with myocardial infarction.[46] For signal-averaged ECG to reveal a late potential, it must be of sufficient amplitude and extend beyond the typical QRS complex into the ST segment. Late potentials will not be detected if they are of short duration, are of similar amplitude to the amount of residual noise, are in the ventricular apex, or are concurrent with bundle-branch block.[38] Signal averaging to a noise value of 0.3 μV maximizes the ability to detect late potentials; however, reducing noise to such values can be difficult in humans. We found it extremely difficult to signal-average to a noise value of < 4 μV in standing unsedated sled dogs because of movement and suboptimal preparation of electrode sites. Because of the high initial noise values in this study (approx 40 μV), signal averaging to 0.3 μV would have required approximately 20,000 beats. Therefore, we selected a noise value of 4 μV for this study, which required signal averaging approximately 110 beats. This noise value may have obscured some low-amplitude late potentials.

Human endurance athletes frequently have electrocardiographic evidence of LV enlargement.[1,3,6] Using a semiorthogonal lead system, we reported elsewhere[7] that canine endurance athletes also have electrocardiographic evidence of LV enlargement attributable to exercise-induced hypertrophy of the ventricular septum and LV free wall. Echocardiographic examination of the sled dogs in our study indicated thickening of the ventricular septum and LV free wall as well as generalized cardiac enlargement.[29] The orthogonal lead system used in our longitudinal study indicated that peak-to-peak voltage tended to increase in the X, Y, and Z leads, which is consistent with generalized cardiac enlargement. The only significant changes in amplitude were in the Y lead, manifest as an increase in peak-to-peak voltage and S-wave amplitude and a decrease in Q-wave amplitude. These changes indicate that endurance training increases terminal cephalad-directed forces, suggesting hypertrophy of the base of the left and right ventricular free wall and intraventricular septum, because those sections of the heart of dogs was activated last in an apicobasilar manner.[47] The electrocardiographic changes were consistent with the echocardiographic findings that endurance training induced LV free wall and septal thickening as well as LV chamber enlargement.[29] An identical change is observed in endurance-trained human athletes.[6] The finding that endurance training did not change peak spatial magnitude is consistent with generalized cardiac enlargement and other studies in dogs[37] that indicate there is not a correlation between spatial magnitude and heart weight.

[a]Pagewriter XLi, M1700A, Hewlett Packard Medical Products, Andover, Mass.

[b]Mortara EL-1, Mortara Instrument, Milwaukee, Wis.

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