Influences of Physical Training on the Heart of Dogs

By Horace L. Wyatt and Jere H. Mitchell

ABSTRACT

To investigate the effects of physical training on cardiac dimensions and function, eight dogs were exercised for 12 weeks by treadmill running 1 hour/day, 5 days/week. Five dogs were confined in cages as controls for an 8-week period. Heart rates were monitored by telemetry during rest and exercise. Maximum QRS spatial magnitudes were calculated from records of McFee lead electrocardiograms. Left ventricular end-diastolic dimensions were determined radiographically by the bead and clip technique. No statistically significant changes occurred during the control period. Training produced statistically significant decreases in heart rate at rest (72 beats/min to 49 beats/min, \( P < 0.005 \)) and at a standard work load of 6.1 mph on a level treadmill (205 beats/min to 158 beats/min, \( P < 0.005 \)) and statistically significant increases in work load (5.4 mph to 9.1 mph, \( P < 0.005 \)) at a standard heart rate of 194 beats/min. Improvements were rapid during the first 4 weeks of training but gradual during the remaining 8 weeks. Training caused small but statistically significant increases in left ventricular end-diastolic wall thickness (8.7 mm to 9.3 mm, \( P < 0.0025 \)), estimated left ventricular mass (83.6 g to 91.2 g, \( P < 0.01 \)), and maximum (McFee) QRS spatial magnitude (4.0 mv to 4.8 mv, \( P < 0.05 \)).

KEY WORDS standard work load left ventricular end-diastolic dimensions maximum QRS spatial magnitude telemetry exercise bead and clip technique heart rate in trained dogs

The most prominent, well-established effects of intensive physical training in both human (1) and animal (2) studies are the decreases in heart rate at rest and at a given level of submaximum work. However, if the training program consists of exercise of low intensity relative to the subject's previous physical activity, then there may be no change in heart rate (3, 4).

In previous studies, the effects of physical training on heart weight have been assessed by comparing heart weight or the ratio of heart weight to body weight between a group of trained animals and a separate group of control animals. Intensive physical training causes an increase in heart weight in small animals (5). With long training periods, e.g., 6 hours/day, increases in heart weight in small animals can occur within just 1 week (6). In dogs physical training can result in either no change (7, 8) or an increase in the ratio of heart weight to body weight (9, 10).

It is also known that well-trained athletes exhibit electrocardiographic evidence of ventricular hypertrophy; however, few studies have incorporated longitudinal data. Hugenholtz (11) found increases in QRS spatial magnitudes in most members of a group of young men who had trained for rowing competition for 7 months; heart volumes were not determined. Saltin et al. (1) found a small increase in heart volume in five young men who had trained at a high-intensity level for 8 weeks.

There are no previous studies in which serial measurements of both left ventricular dimensions and QRS spatial magnitudes have been recorded in dogs subjected to physical training with each dog acting as its own control. The purpose of the present study was to determine the effects of a 12-week physical training program on heart rate at rest and during exercise, on left ventricular volume and mass, and on QRS spatial magnitude.

Methods

Ten dogs (four females and six males) weighing...
13–25 kg successfully completed the study. After open heart surgery during which beads and clips were placed in the left ventricular myocardium, all dogs were maintained in cages for at least 6 weeks prior to the initial study. Dogs 1–8 were subjected to a physical training period that consisted of treadmill running at approximately 4–8 mph on a 10° incline for 1 hour each day, 5 days each week for 12 weeks. During the first week, the duration of exercise was increased from 30 minutes to 1 hour daily and thereafter the duration remained 1 hour/day. To exclude changes that might result from factors other than physical training, dogs 6–8 were maintained in cages for an 8-week control period immediately preceding the physical training period. Dogs 9 and 10 were also maintained in cages for an 8-week control period.

Heart rates recorded during the daily workouts are listed for each dog in Table 1. In this study, maximum heart rates of the dogs were not measured; however, because many dogs have maximum heart rates slightly above 300 beats/min, a heart rate of 310 beats/min was used as an approximation of the maximum heart rate in each dog. The intensity of exercise in the daily workouts was estimated as a percent value, assuming that the resting heart rate was zero intensity and the maximum heart rate was 100% intensity. These estimates are listed for each dog in Table 1. In seven of the eight dogs, the approximate intensity of exercise was in the narrow range between 39% and 52% of maximum effort. In dog 2, an estimate of 72% was obtained; it is likely that in this dog the true maximum heart rate was substantially higher than 310 beats/min.

Studies were performed on each of the dogs initially and at the end of each control and training period. Each study included heart rate telemetry, electrocardiography, and biplane cinefluorography.

**HEART RATE TELEMETRY**

In the telemetry study, heart rates were determined on the dogs at rest and at a standard work load. Work loads were determined at a standard heart rate. The standard work load and the standard heart rate were arbitrarily set at the beginning of the program at approximately 6 mph on a level treadmill and 195 beats/min, respectively. Work load was always determined as the running speed on a level treadmill.

Resting and exercise heart rates of all dogs were determined using a biotelemetry transmitter (E and M Instruments, model FM–1100-E4) and a receiver (E and M Instruments, model FM–1100-T). The electrical activity of the heart was detected by two silver chloride electrodes placed on each side of the thorax. The electrode wires and the transmitter antennas were taped securely to the dog to decrease interference from movements of the wires and the antennas. The telemetered electrocardiographic signal was recorded on a Sanborn electrocardiograph.

Heart rates were recorded from each dog on at least two separate occasions prior to the beginning of the study, at the beginning of each period, and at 2-week intervals during the training period. Resting heart rates were recorded with the dog lying quietly in the kennel or in a cage inside the exercise room. After the electrodes and the transmitter had been attached to the dog, approximately 0.5–1 hour was allowed for heart rate to stabilize.

After recordings of the resting heart rate had been obtained, the dog was placed on a level treadmill and made to run for approximately 3 minutes, at which time the heart rate had stabilized. A heart rate recording was obtained at the standard work load. The dog was allowed to rest for approximately 5 minutes and then made to run again on a level treadmill at a low speed. The speed was increased by 1 mph at 30-second intervals until the standard heart rate was attained.

**ELECTROCARDIOGRAPHY**

Electrocardiograms (ECG) were recorded during rest from the awake, unanesthetized dog lying on his right side. Front and rear leads were attached with leather straps to prevent movement. Well-type silver-silver chloride electrodes (Beckman) were applied to the skin as specified in the McFee system (12), and the signals were amplified and recorded with a Sanborn 2000 FM tape recorder. The three leads were monitored simultaneously, and records were obtained on a photographic recorder (Electronics for Medicine, model PR-6). From photographic records of the ECG, maximum QRS spatial magnitudes (SM) were calculated using the formula

\[ SM = \sqrt{V_x^2 + V_y^2 + V_z^2} \]  

The maximum QRS deflection in the 1 lead provided a convenient reference point, and, in every case, it was the point of the maximum QRS spatial magnitude.

**BIPLANE CINEFLUOROGRAPHY**

Left ventricular dimensions were analyzed by methods developed in this laboratory (13–15). The dogs were prepared for study at least 6 weeks before.
CARDIAC EFFECTS OF PHYSICAL TRAINING

prior to the beginning of the training program by appropriate placement of six lead beads and two silver-tantalum clips during open heart surgery. The base bead was sutured to the epicardium at the base of the heart just below the left circumflex coronary artery near the junction of the aortic and mitral valve rings. The apex bead was sutured at the apical dimple. The anterior, posterior, septal, and lateral beads were inserted into the left ventricular wall as close as possible to the endocardium at the corresponding locations approximately equidistant from the base and the apex by inserting a hollow needle into the myocardium and using a plunger to fix the beads. The beads outlined a nonprolate ellipsoidal shell of left ventricular myocardium in which the distance between the base and apex beads constituted the major axis \( L_b \) and the distances between the anterior and posterior beads and between the septal and lateral beads constituted the minor axes, \( L_a \) and \( L_s \), respectively. The silver-tantalum clips were sutured to the epicardium directly opposite the anterior and posterior beads to permit determinations of left ventricular wall thickness.

In the biplane cinefluorography study, the dogs were anesthetized with sodium pentobarbital (30 mg/kg, iv) and strapped to the X-ray table in the supine position. Biplane cinefluorograms of the heart were obtained, and left ventricular dimensions were determined.

Left ventricular ellipsoidal cavity volume \( (LVV) \) was calculated using the formula

\[
LVV = \frac{4}{3} \pi abc
\]

where \( a = L_b/2 \), \( b = L_a/2 \), and \( c = L_s/2 \).

The dogs were killed at the end of the final study. Their hearts were removed, left ventricular bead and clip positions were examined, and records were made of percent wall thickness located between the beads and the clips. A better approximation of wall thickness was obtained by correcting for the unmeasured portion of the wall. Following removal of the heart, the left ventricle was weighed, and this value was compared with the estimated left ventricular mass. Left ventricular ellipsoidal external volume \( (ELVV) \) was calculated using the formula

\[
ELVV = \frac{4}{3} \pi abc d e f
\]

where \( d = (L_a + 2WT)/2 \), \( e = (L_s + 2WT)/2 \), \( f = (L_b + 2WT)/2 \), and \( WT \) = left ventricular wall thickness. Left ventricular mass \( (LVM) \) was estimated using the formula

\[
LVM = 1.05(ELVV - LVV)
\]

where 1.05 = density of the myocardium.

Results

Dogs 1–8 finished the 12-week training program. Dogs 6–10 completed an 8-week control period. Results are plotted as mean values in Figures 1 and 2 and as individual values in Figures 3 and 5. Results are summarized in Table 2 as changes in mean values ± SE that occurred during the training period (control vs. trained) and the control period (control 1 vs. control 2). In dogs 6–8, which were subjected to both a control period and a training period, the control measurements immediately preceding the training period were compared with the posttraining measurements. Included in Table 2 are \( P \) values from results of statistical analyses with paired Student's t-tests and the number of dogs from which the means were calculated.

Changes in mean resting heart rate during the training period are plotted for dogs 1–8 in Figure 1. Mean resting heart rate decreased most rapidly during the first 4 weeks of training (from 72 beats/min to 56 beats/min). There was a more gradual decrease during the remaining 8 weeks of training (from 56 beats/min to 49 beats/min). During the 8-week control period, there was an insignificant decrease in mean resting heart rate (from 78 beats/min to 71 beats/min); individual changes were not consistent.

During the first 4 weeks of training, mean heart rate at a standard work load (mean 6.1 mph on a level treadmill) decreased sharply from 205 beats/min to 153 beats/min (Fig. 2). During the next 4 weeks of training, mean heart rate at standard work load increased to 164 beats/min, and, during the last 4 weeks, it decreased again to 158 beats/min. Correspondingly, during the first 4 weeks of training, the mean work load (speed on a level treadmill) at a standard heart rate (mean 194 beats/min) increased sharply from 5.4 mph to 9.1 mph (Fig. 2). During the next 4 weeks of training, mean work load at the standard heart rate decreased to 8.6 mph, and, during the last 4 weeks, it increased again to 8.1 mph. Maximum improvements in exercise heart rate and work capacity were observed after 4 weeks of training. During the control period, there was little or no change in heart rate at a standard work load or in work load at a standard heart rate.

During the training period, the largest individual increases in work load at a standard heart rate were seen in dogs 4 (6 mph to 13 mph) and 5 (5 mph to 12 mph); estimated intensities of daily exercise based on a maximum heart rate of 310 beats/min were 52% and 46%, respectively.

In summary, during the first 4 weeks of training, marked decreases in resting and exercise heart rates and a marked increase in work capacity occurred. During the second 4 weeks of training, a decrease in resting
Effects of Physical Training

<table>
<thead>
<tr>
<th>Time in Weeks</th>
<th>Mean Speed in mph</th>
<th>Mean Heart Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>210</td>
<td>130</td>
</tr>
<tr>
<td>2</td>
<td>194</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>194</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>194</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>194</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>194</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>194</td>
<td>0</td>
</tr>
</tbody>
</table>

Mean values ± 1 SE for heart rate (HR) at a fixed work load and for work load at a fixed heart rate during the training period (eight dogs).

Circulation Research, Vol. 35, December 1974
CARDIAC EFFECTS OF PHYSICAL TRAINING

Figure 3

Individual changes in estimated left ventricular mass during control and training periods.

Mean values for left ventricular end-diastolic volume did not change significantly during either the control or the training period (Table 2).

Estimated left ventricular mass increased consistently during the training period (Fig. 3). During training, mean estimated left ventricular mass increased \( (P < 0.01) \) by 9% from 83.6 g to 91.2 g; this increase was primarily due to a statistically significant increase \( (P < 0.0025) \) in mean left ventricular end-diastolic wall thickness from 8.7 mm to 9.3 mm (Table 2). There was no significant change in estimated left ventricular mass during the control period.

In all dogs except dogs 6 and 10, estimated left ventricular mass corresponded closely to the actual left ventricular mass obtained at the time of death. A linear relation between estimated left ventricular mass and actual left ventricular mass is demonstrated in Figure 4; the correlation coefficient \( (r) \) was 0.79.

The ratio of estimated left ventricular mass (g) to body weight (kg) was calculated for each dog. Mean values are presented in Table 2. There was a statistically significant 7.4% increase \( (P < 0.025) \) in the ratio during the training period (dogs 2-7) from 4.5 to 4.8. During the control period there was a small, insignificant decrease in the ratio from 4.7 to 4.6. In dogs 2-7 during the training period, mean body weight increased from 18.7 kg to 19.1 kg (+2%), accounting for the fact that the ratio of estimated left ventricular mass to body weight increased less (7.4%) during training than did estimated left ventricular mass (9%). During the control period, body weight increased from 19.3 kg to 20.3 kg (+5%).

Electrocardiography

Maximum QRS spatial magnitudes were calculated and results are presented in Figure 5 and Table 2. Data from dog 3 were eliminated because of technical difficulties. With the exception of dog 6, QRS spatial magnitude increased during the training period. The mean value for QRS spatial magnitude increased significantly during training from 4.0 mv to 4.8 mv \( (P < 0.05) \). During the control period, there was an increase in mean QRS spatial magnitude from 4.6 mv to 5.1 mv, but the increase was not statistically significant.

Discussion

It is well known that physical training can cause decreased heart rates in subjects at rest and during exercise at submaximum work loads. The results of this study confirm the findings of other studies (1, 16, 17) that, if the training is severe with respect to the subject's previous physical condition, sub-
Substantial improvements in heart rates and work loads can be produced in as little as 2 weeks. The differences in the results of many programs probably result from differences in the degree of fitness of the subjects prior to the program and from the length and the severity of the training program.

Very few studies have incorporated successive measurements of work load or heart rate changes during training programs. When men were allowed to set their own pace in a low-intensity, 7-month training program, heart rates decreased gradually throughout the program (18). In a training study on dogs, daily treadmill running produced gradual decreases in resting and exercise heart rates throughout a 5-week training period (2). In contrast, in young men heart rate decreased rapidly at a submaximal work load during the first 2 weeks of intensive training, but it decreased only gradually during the remaining 6 weeks (1). In a training study on sedentary blind men who performed high-intensity exercise 3 days/week, there was a rapid increase in work load at a given heart rate during the first 7 weeks but only very gradual increases during the last 8 weeks (19). The latter two studies demonstrate the type of improvements that can be expected with a high-intensity type of training program. The results of the present study agree with the findings of other studies, suggesting that heart rate and work capacity improvements occur rapidly during the first few weeks of strenuous physical training and very gradually for the remainder of the training program (1, 19). In the present study, after the first 4 weeks of training there was little or no change in work capacity and heart rate at submaximum exercise.

Radiologically, enlarged hearts are quite frequently found in athletes and in trained subjects; however, in most cases it is difficult to determine whether there is a concomitant cardiac hypertrophy, especially an increase in ventricular wall thickness. Dilation of the heart allows a greater venous filling and stroke volume output, but hypertrophy of the heart probably enables a greater contractile force to be generated; thus, a greater stroke volume than that predicted by the heart volume alone can be present (20). Changes in radiologically measured heart volume can occur rapidly (8 weeks or less) in men with changes in the chronic level of physical activity (1, 21), but hypertrophy usually requires prolonged training in both men (21) and dogs (9, 10).

In previous studies, the effect of physical training on heart weight in animals has been observed by comparing heart weights between a group of trained animals and a separate group of control animals. In this study, each dog acted as its own control. Left ventricular dimensions were measured from markers placed in the left ventricular myocardium during surgery prior to the study. During a 12-week training period, mean left ventricular end-diastolic wall thickness increased, but mean left ventricular end-diastolic volume did not change. As a result, there was a small but significant (9%) increase in mean estimated left ventricular mass and a 7.4% increase in the ratio of estimated left ventricular mass to body weight.

Hugenholtz (11) found increases in QRS spatial magnitudes in a group of young men who had trained for rowing competition for 7 months; heart volume was not determined. Saltin et al. (1) found increases in heart volume but only small, insignificant increases in QRS spatial magnitudes in five young men who had trained for 8 weeks. The reason for the difference in these results is

\[ \text{ Circulation Research, Vol. 35, December 1974 } \]
CARDIAC EFFECTS OF PHYSICAL TRAINING

unknown; it is possible that in the study of Hugenholtz (11) there was some cardiac hypertrophy after the prolonged training, whereas in the study of Saltin (1) there was a cardiac dilation without hypertrophy after the short-term training.

In the present study, in seven dogs there was a significant (19%) increase in mean maximum QRS spatial magnitude after daily training for 3 months. These results in dogs agree with those found in men by Hugenholtz (11). Mean estimated left ventricular mass did not always change in the same direction as did mean QRS spatial magnitude in the present study. The fact that individual values for spatial magnitude and estimated left ventricular mass did not always change in the same direction could have been due to experimental error in the measurement of the two parameters.

In summary, the present study demonstrated an increase in physical work capacity after 12 weeks of daily physical training in dogs. Heart rates at rest and during submaximum exercise at a fixed work load decreased rapidly during the first 4 weeks of training and gradually during the remaining 8 weeks. Radiographic studies provided suggestive evidence that the physical training induced an increase in left ventricular muscle mass. In most instances, changes in left ventricular mass were paralleled by changes in maximum QRS spatial magnitude.

Acknowledgment

The authors gratefully acknowledge the helpful review and criticism of Dr. Gunnar Blomqvist and the secretarial assistance of Miss Pat Wells.

References

8. EYSTER JAE: Experimental and clinical studies in cardiac hypertrophy. JAMA 91:1881-1884, 1928

Circulation Research, Vol. 35, December 1974
Influences of Physical Training on the Heart of Dogs
HORACE L WYATT and JERE H. MITCHELL

_Circ Res_. 1974;35:883-889
doi: 10.1161/01.RES.35.6.883

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/35/6/883

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation Research_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation Research_ is online at:
http://circres.ahajournals.org/subscriptions/