Cardiovascular Adaptations to Prolonged Physical Effort

By George L. Beckner, M.D., and Travis Winsor, M.D.

Studies of the cardiovascular system were made among 26-mile marathon runners after at least five years of physical training. Studies made with the subjects at rest showed an electrocardiogram characterized by a vertical or semi-vertical electrocardiographic position of the heart in the chest, bradycardia, high voltage of the QRS complexes and high T and U waves. T waves which were low with respect to the height of the R waves were not encountered. Etiologically the heart was enlarged. Immediately after running 26 miles the transverse diameter of the heart was decreased considerably.

The recognition of the acute and chronic physiologic adaptations of the cardiovascular system which occur as a result of intensive, prolonged physical exertion is of importance because the changes which result are similar in many respects to those seen with disease. Barach, Gordon, and coworkers and Branwell and Ellis have pointed out that cardiac enlargement is a usual finding among marathon runners and is not indicative of cardiac disease. The present study confirms this observation and illustrates certain electrocardiographic deviations from normal which resemble findings encountered in patients with cardiac disease. It is the purpose of this report to describe the acute and chronic changes occurring in the cardiovascular system as a result of prolonged physical exertion among well-trained marathon runners.

Methods and Materials

One hundred and sixty-five male marathon runners with more than five years of physical training and 40 male nonrunners were studied. The average height of the runners was 68 inches (65 to 76) and of the controls 65.5 inches (59 to 71). The runners' ages averaged 27.9 years (18 to 48) and the controls' 25.5 years (18 to 42). The average weight of the runners was 142 pounds (119 to 168) and of the controls 148 pounds (102 to 175).

Studies were made before and after running 26, 18 or 10 miles on city streets during the day in an average temperature of 78 F. (70 to 90 F.) and humidity of 35 per cent (20 to 50). Studies made before the races were carried out usually from 1 to 24 hours prior to running and were repeated from one to five minutes after or in certain instances at longer periods up to 56 hours. The subjects of the control group consisted of males in sedentary occupations (airplane pilots) in an industry which required frequent physical examinations. Two hundred electrocardiograms were taken on the runners and 50 on the nonrunners. Forty tracings were taken with a string galvanometer and the remainder with the Viso-Cardieter. All electrocardiograms were taken with the patient in the prone position. The various electric axes of the electrocardiogram were analyzed in a similar manner in the two groups from amplitudes rather than area, with the aid of a hand lens when indicated. The amplitude of all waves was measured in millimeters and the duration in seconds. The electrocardiographic position of the heart in the chest was determined according to the method of Wilson and his coworkers. The corrected Q-T interval was determined, using a modified Bazette formula as described by Taran and Szilagyi. Venous pressures were recorded with the Phlebomanometer. Electrolyte studies on blood samples drawn before and after the races were carried out by a well established commercial laboratory using standard technics. The cardiac size of both groups was measured from teleoentgenograms using the technics of Hodges and Eyster and Ungerleider and Gunber. The transverse, broad, long and great vessel measurements were made directly from the teleoentgenogram and the cardiac area was calculated from the formula A = π/4 × L × B.

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* Cambridge Instrument Co., New York, N. Y.
† Sanborn Co., Cambridge, Mass.
‡ Courtesy of The Clinical Laboratory, Hammack & Maner, Los Angeles, Calif.
Results

General Observations. The average weight loss for 40 runners running 26 miles was 5.1 per cent of the body weight; the average loss for eight runners running 18 miles was 3.4 per cent of body weight; while the average loss for 56 runners running 10 miles was 2.7 per cent of body weight. Although the weight loss was not linear the approximate loss was 1 per cent of body weight for each five miles run. The blood pressure in three runners averaged 126/75 before the race and 90/60 after the race. The antecubital venous pressures in 10 runners was normal before the race but after running averaged 210 per cent above the resting level. The pressures remained elevated for 30 minutes after running after which they returned to the resting levels. Abdominal compression resulted in a decrease in the venous pressure in all cases before and after the race. The pulse rate for the runners before the race averaged 57 beats per minute (40 to 72) and for the controls 65 beats per minute (55 to 85). After the race the pulse rate of the runners averaged 91 beats per minute (76 to 108).

Electrolyte changes occurring in 10 runners are shown in figure 1. Of particular interest are the great reduction in the blood chloride and the increase in blood nonprotein nitrogen. Five of the subjects with low blood chloride had muscle cramps soon after they stopped running; it was felt that the low blood chloride may have been responsible for these cramps. The serum potassium level after running did not deviate from the resting level significantly. There was no positive correlation between the increased height of the T waves in the electrocardiogram after exercise and the serum potassium encountered immediately after the race. The potassium level was followed for 8, 21 and 30 hours after running in two subjects. In these subjects the serum potassium rose above the values found immediately after the race and it was noted that as the serum potassium rose the T waves became smaller.

Electrocardiographic Findings. The electro-

![Graph](http://circ.ahajournals.org/)

**Fig. 1.** Effect of running 26 miles on the blood chemistry of 10 runners. The average resting values are shown in the underlined figures and the percentage deviation from the resting value is shown in the open figures. The low serum chloride could be correlated with the presence of muscle cramps which occurred after the race.
cardiographic position of the heart in the chest before and after the race was determined: In 94.3 per cent the position was semivertical or vertical before and after the race. In 5.7 per cent the position was intermediate before and after the race. In no instance was the position horizontal or semihorizontal.

The amplitude and duration of the P waves in lead II were studied in the two groups (table 1). P waves of normal height and duration were present in the nonrunners and in the runners before the race, but after the race these waves increased an average of 66.6 per cent in amplitude and 8.1 per cent in duration. The average magnitude of the electric axes of the P waves for the two groups at rest was 1.3 and 1.3 units, respectively, and the average direction 50 degrees and 50 degrees, respectively. After running, the average magnitude increased 61 per cent to 2.1 units, and the direction moved to the right 15 degrees, to 65 degrees. An increase in the magnitude of the P wave vector similar to that which occurred in the runners after exercise was not produced in three normal subjects by holding the breath in deep inspiration.

The P-R interval of the runners at rest averaged 0.16 second (0.14 to 0.20) and of the nonrunners 0.17 second (0.15 to 0.21). After running, the P-R interval decreased or showed no change except in one runner whose P-R interval increased from 0.16 to 0.21 second (fig. 2).

The duration of the QRS complexes in lead II was compared in both groups. The average for the runners at rest was 0.086 second (0.070 to 0.100, S.D. 0.011) as compared with 0.074 (0.050 to 0.100, S.D. 0.017) for the nonrunners. After running, the duration was less than the resting value in all cases.

The direction and magnitude of the mean electric axes of the QRS complexes of the nonrunners and runners were determined. The average direction for the nonrunners was 54 degrees (30 to 102) as compared with 66.8 degrees (−5.0 to 95.0) for the runners before the race. After the race the average was 69.1 degrees (−5.0 to 69.0). The magnitudes of the nonrunners averaged 9.0 units (2.3 to 16.7); those for the runners before the race averaged 14.1 units (7.6 to 39.0). After the race the average magnitude was 15.2 units (6.5 to 38.5). Thus the direction of the axis of the QRS complexes was more to the right and the magnitude

![Table 1](image1)

![Fig. 2](image2)

![Fig. 3](image3)
TABLE 2.—Amplitude of R Waves in Lead II among Runners and Nonrunners. R Waves are High among the Runners at Rest and Increase Slightly after a 29 Mile Run.

<table>
<thead>
<tr>
<th></th>
<th>Nonrunners</th>
<th>Runners</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Mean</td>
<td>12.0</td>
<td>16.2</td>
</tr>
<tr>
<td>Min</td>
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<td>9.0</td>
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<tr>
<td>Max</td>
<td>22.0</td>
<td>40.0</td>
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<tr>
<td>S.D. ±</td>
<td>5.0</td>
<td>6.5</td>
</tr>
<tr>
<td>C.V. ±%</td>
<td>41.6</td>
<td>39.0</td>
</tr>
</tbody>
</table>

The highest R waves of the six precordial leads for nonrunners and runners is shown in table 3. The tallest R waves occurred in V₄ in 72 per cent of the runners and in 70 per cent of the nonrunners. For the nonrunners the maximum R wave was 31.0 mm. Ten per cent of the tracings taken of the runners at rest exceeded this value. The average height of the R waves in the nonrunners was 18.6 mm, while in the runners at rest it was 24.1 mm., or an average increase of 29.5 per cent. After running, the mean height of the R waves increased 7.1 per cent.

The amplitude of the deepest S waves in the six precordial leads is shown in table 4. The deepest S wave of the runners at rest occurred in lead V₂ in 60 per cent; in V₁ in 20 per cent; and V₃ in 20 per cent with a similar distribution after running. Among the nonrunners the deepest S wave occurred in lead V₂ in 69 per cent; in V₁ in 11 per cent and in V₃ in 20 per cent. The average for the control group was 12.7 mm. whereas the average for the runners at rest was 55.9 per cent greater, being 19.8 mm. After running, the average increase in depth of the S wave over the resting tracing was 9.6 per cent. The maximum depth of the S waves for the nonrunners was 29.0 mm. This value was exceeded by 12 per cent in the tracings of the runners at rest (figs. 4 and 5).

The sum of the amplitudes of R + S of lead II is shown in table 5. The mean value for the nonrunners is 13.4 mm. while that for the runners at rest is 17.2 mm. (28.3 per cent greater). After running, the mean was 19.2 mm., an increase of 11.6 per cent over the resting value. The maximum R + S amplitude

was greater in the runners at rest than in the nonrunners. Running resulted in a further increase in magnitude with a slight additional shift to the right. In three normal subjects electrocardiograms were taken with the breath held in inspiration. The magnitude of QRS did not approach the values in the runners at rest.

The configuration of the QRS complexes in V₁ was within normal limits in all the 165 runners except the three shown in figure 3. In these there was an R-R prime wave suggestive of a right bundle branch block although the width of the QRS complexes did not exceed the upper limit of normal (0.1 second). These complexes were seen before and after running and were observed at intervals for a period of five years.

The amplitude of the R waves in lead II for the runners at rest averaged 16.2 mm. with a 10.5 per cent increase after running (table 2). This average amplitude was considerably greater than that of the nonrunners which was 12.0 mm. Eighteen per cent of the runners had R waves in lead II which exceeded the maximum (22.0 mm.) of the nonrunners.
for the nonrunners was 22.0 mm.; however, 15 per cent of the runners had R + S amplitudes at rest which exceeded this maximum value (fig. 6).

The greatest R + S voltage of the six precordial leads was studied by adding the height of the R wave to the depth of the S wave of the precordial lead showing the largest amplitude (table 6). Among the nonrunners the highest R + S wave amplitude occurred in V4 in 40 per cent; in V3 in 30 per cent; in V2 in 20 per cent and in V5 in 10 per cent of subjects. Among the runners both at rest and after the race the highest R + S amplitude occurred in V2 in 35 per cent; in V3 in 30 per cent; and in V5 in 5 per cent of subjects. The mean R + S amplitude for the nonrunners was 25 mm. while for the runners at rest it was 34.7 mm. or 38.8 per cent greater than the nonrunners. After running, the voltage increased an average of 10.9 per cent. The maximum R + S voltage for the nonrunners was 47 mm. Ten per cent of the tracings taken on the runners at rest exceeded this value (figs. 5 and 6). High voltage persisted for five years after cessation of training in six patients in whom this study was made.

The sum of the height of the R wave in V1 and the depth of the S wave in V5 was determined. The average value for the runners at rest was 7.5 mm. (2.0 to 21.0, S.D. 3.3) while after the race it was 9.2 mm. (4.0 to 16.0, S.D. 2.4). Eighteen per cent of the runners' tracings taken at rest revealed amplitudes in excess of 10.5 mm. which is the upper limit of normal given by Sokolow and Friedlander. Forty-two per cent of the runners had values above this after the race.

The sum of the amplitudes of the S waves in V1 plus the R waves in V5 (Sv1 + Rv5) was determined likewise before and after the race. The mean value for the runners' tracings taken at rest was 33.0 mm. (18.0 to 64.0, S.D. 9.0)
while after the race it was 35.4 mm. (18.0 to 60.0, S.D. 10.0). Thirty-three per cent of the tracings taken at rest showed amplitudes which exceeded the upper limit of normal given by Sokolow and Friedlander of 35.0 mm., whereas 44 per cent exceeded this value after the race.

The amplitude of the T waves in lead II averaged 2.8 mm. in the nonrunners while the average for the runners at rest was 4.22 mm. or 50.7 per cent greater than the nonrunners (table 7). After running, the T waves increased 14.9 per cent. The maximum T wave was 5.0 mm. for the nonrunners. Eighteen per cent of the runners’ tracings taken at rest showed T waves which exceeded this value.

The height of the tallest T waves in the precordial leads is shown in table 8. In 63 per cent of the runners and in 54 per cent of the nonrunners the tallest T waves occurred in V3. The average height of the tallest T wave of the precordial leads in the nonrunners was 7.7 mm. as compared with 9.2 mm. in the runners at rest or an average increase of 19.3 per cent. The tallest T wave averaged 12.6 or 36.9 per cent greater after a race than in the tracing taken before the race. The maximum T wave of the precordial leads for the nonrunners was 14 mm.; 16 per cent of the electrocardiograms taken of the runners at rest exceeded this value.

The direction and magnitude of the T wave vectors were calculated for the two groups. The direction of the vector for the nonrunners averaged 38.0 degrees (0 to 76) as compared with 38.4 degrees (0 to 82) for the runners. After a race, these values averaged 45.7 degrees (0 to 87). The magnitude of the T wave axis for the nonrunners was 2.6 units (1 to 5)
Table 8.—Amplitude of Tallest T Waves in Premordial Leads. Sixteen Per Cent of Runners Showed Tracings at Rest which Exceeded the Maximum for the Nonrunners of 14.0.

<table>
<thead>
<tr>
<th></th>
<th>Nonrunners</th>
<th>Runners</th>
</tr>
</thead>
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<tr>
<td></td>
<td>Before</td>
<td>After</td>
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<tr>
<td>Mean</td>
<td>7.7</td>
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</tr>
<tr>
<td>Min</td>
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<td>2.0</td>
</tr>
<tr>
<td>Max</td>
<td>14.0</td>
<td>17.0</td>
</tr>
<tr>
<td>S.D. ±</td>
<td>1.5</td>
<td>3.6</td>
</tr>
<tr>
<td>C.V. ±%</td>
<td>36.0</td>
<td>38.0</td>
</tr>
</tbody>
</table>

as compared with 3.6 units (1.2 to 6.5) for the runners before the race. After a race, the value was 3.9 units (2.0 to 6.5). Thus the direction of the T wave axis of the two groups was similar at rest but the magnitude was greater in the runners. After a race, the axis shifted to the right with no significant change in the magnitude. Typical T wave changes after running 26 miles are shown in figure 7.

The S-T segments in the runners were within normal limits before and after running in all subjects except one (fig. 8). This runner had 1.5 mm. negative S-T segment shifts in V2 and

V1 immediately after running 23 miles. The runner had unusual fatigue but no chest pain. Subsequent tracings showed a return of these segments to the base line in one hour.

The intrinsicoid deflection was measured in tracings made with the string galvanometer on 20 nonrunners at rest and on 20 runners. In lead V1 the nonrunners showed a mean time of 0.014 second (0.005 to 0.030, S.D. 0.003) compared with the runners whose resting tracings showed values of 0.030 (0.020 to 0.050, S.D. 0.003). After the race the runners had values of 0.031 second (0.020 to 0.050, S.D. 0.008). In V6 the nonrunners showed an average of 0.038 second (0.020 to 0.050, S.D. 0.005) compared with the tracings of the runners taken at rest of 0.049 second (0.040 to 0.060, S.D. 0.008). Thus the intrinsicoid deflection time was longer in the runners in V1 and in V6 than in the nonrunners.

The corrected Q-T interval (Q-Tc) was measured in both groups: The mean value before running was 0.40 second (0.33 to 0.45, S.D. 0.044). After running the value was 0.41 second (0.36 to 0.47, S.D. 0.036). The nonrunners averaged 0.40 second (0.35 to 0.45). Thus the Q-T interval was not abnormal before or after running.
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Table 9.—Transverse, Broad, Long and Great Vessel Diameters as well as Cardiac Areas in 40 Runners and Nonrunners

<table>
<thead>
<tr>
<th>Great Vessel Diameter</th>
<th>Transverse</th>
<th>Broad</th>
<th>Long</th>
<th>Area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non runners</td>
<td>Runners</td>
<td>Non runners</td>
<td>Runners</td>
</tr>
<tr>
<td>Mean</td>
<td>121</td>
<td>129</td>
<td>112</td>
<td>117</td>
</tr>
<tr>
<td>Min.</td>
<td>104</td>
<td>106</td>
<td>110</td>
<td>105</td>
</tr>
<tr>
<td>Max.</td>
<td>139</td>
<td>143</td>
<td>115</td>
<td>132</td>
</tr>
<tr>
<td>S. D. ±</td>
<td>12</td>
<td>12</td>
<td>0.8</td>
<td>6</td>
</tr>
<tr>
<td>C. V. ±%</td>
<td>10</td>
<td>9</td>
<td>0.7</td>
<td>5</td>
</tr>
</tbody>
</table>

The ventricular gradient was calculated for both groups. The direction for the nonrunners averaged 53 degrees (2 to 89) and for the runners at rest 61 degrees (0 to 84). After running the average was 66 degrees (7 to 87). The magnitude for the nonrunners averaged 11.3 units (4.4 to 17.8) as compared with the runners at rest which averaged 17.4 units.
Fig. 10. Cardiac shadow of a runner before and after running 26 miles. The cardiac shadow is small after running compared with the resting size.

(10.3 to 45.8). After running the magnitude averaged 18.6 units (9.8 to 43.2). Thus in the runners the gradient vector was slightly more to the right than in the nonrunners and the magnitude of the gradient averaged 53.9 per cent greater in the runners at rest than in the nonrunners.

The positional relationships between the QRS, G, T and H axes were calculated for both groups. The anatomic axis (H) for nonrunners averaged 42 degrees (28.0 to 59.0) as compared with that for the runners, which was slightly more vertical averaging 44 degrees (32.0 to 57.0). A comparison of the various vectors with each other showed that among the runners: (1) the T axis was to the left (negative) of the QRS axis by an average of 29 degrees while the average for the nonrunners was 26 degrees to the left; (2) the average QRS axis was to the right of the average gradient by 6 degrees as compared with 5 degrees for the nonrunners; (3) the average magnitude of the gradient was longer than the magnitude of the QRS complex by 3 units as compared with 2.4 units for the nonrunners, and (4) the average gradient was to the right (plus) of H by 17 degrees, the average for nonrunners being 19 degrees. These findings may be interpreted as indicating that the QRS complexes and T waves are in normal relationship to each other and are likewise in normal relationship to the anatomic position of the heart in the chest.

Teleoroentgenographic Findings. The cardiac size was measured from posteroanterior teleoroentgenograms in both groups (table 9). The transverse measurements of the runners averaged 6.6 per cent greater than the controls. Likewise the actual transverse measurements of the runners exceeded the predicted values by 5.8 per cent. The prediction was based on the height and weight according to the method of Ungerleider and Gubner.9 The average broad measurements of the heart of the runners exceeded the average of the nonrunners by 4.5 per cent. The average long measurements of the runners exceeded that of the nonrunners by 7.9 per cent. The average cardiac area of the runners exceeded that of the nonrunners by 13.0 per cent and the runners showed a 15.8 per cent increase over that predicted from their height and weight.7 The average great vessel diameter of the runners exceeded the normal by 5.8 per cent. A typical teleoroentgenogram of a 45 year old veteran runner taken with the subject at rest is shown in figure 9.

The cardiac size in 10 subjects was com-
comparison before and after they had run 26 miles. Five of these had teleorontgenograms and five had x-ray kymograms. In all subjects the cardiac shadow was considerably smaller after the race than before. A typical example is shown in figure 10.

**DISCUSSION**

The cardiovascular adaptations to prolonged physical training are as follows: The electrocardiograms taken with the runners at rest show definite changes which fall outside the generally accepted limits of normal as well as outside the values seen in the nonrunners who served as controls. The P waves of the electrocardiograms taken on the runners at rest were of normal size and configuration and their electric axes were shifted slightly to the right. The duration of the QRS complexes at rest was increased, which is in keeping with the findings of others. The intrinsicoid deflection times were prolonged in V₁ and V₆, which suggests hypertrophy of the right and left ventricles. High voltage of the QRS complexes in the standard and precordial leads was exceedingly common, which indicates also ventricular hypertrophy. This appears to be a physiologic adaptation to intensive prolonged physical training of the endurance type and is not a sign of disease as suggested by normal ventricular gradients in all cases. The increased voltage of QRS often was present in both standard and precordial leads, indicating that the etiology was related to the heart and that the voltage was not due to the position of the heart in the chest. In general the electrocardiographic position was semivertical and the electric axis of the QRS complex was shifted slightly to the right. The ventricular gradient was well within normal limits indicating that the relationship of QRS to T was within normal limits. The U waves were large and superimposed often on the downstroke of the T wave. These waves were in keeping with cardiac enlargement. The ventricular gradient was shifted slightly to the right which is in keeping with the somewhat more vertical position of the heart of the runners than of the controls. The high voltage of QRS and of the T waves and more vertical position of the heart has been described by others. The sum of the amplitude of Sv₁ and Rv₆ normally does not exceed 35 mm. and an increase over this has been advocated as a sign of left ventricular enlargement. By this guide left ventricular enlargement occurred in 33 per cent of the runners. The sum of Rv₁ and Sv₆ normally does not exceed 10.5; however, this value was exceeded in 18 per cent of the runners. This suggests right ventricular enlargement. The resting cardiac rate was slower generally in the runners than in the nonrunners. The entire electrocardiographic picture of slow rate, high voltage of QRS, T and U, ventricular gradient of increased magnitude and normal relationships of the electric axes to each other is characteristic of the athlete who has had prolonged, intensive training. This picture remains present for at least five years after training has ceased. In 3 of 40 runners an R-R prime wave was found in lead V₁ which remained for at least five years. It seems probable that this resulted from right ventricular strain and may represent a harmful effect of endurance running.

The acute cardiovascular adaptations to prolonged physical effort as seen in this series are based on findings seen immediately after running 26 miles and are as follows: The P waves increased in amplitude and duration in lead II in our series and in the studies of others and rightward deviation of the P-wave axis took place. These changes were not the result of the inspiratory position of the diaphragm or the increased volume of air in the lungs, as waves of this type could not be produced in the nonrunners by holding the breath in the inspiratory position. The auricular T waves were exceptionally large and were opposite in direction to the P waves in those runners whose P waves were exceptionally large. The P-R interval decreased in all cases except one in which it was increased. An increase such as this after exercise compared with the resting is said to be indicative of disease. In the runners, however, the absolute value of 0.22 second was not exceeded; thus it is not certain that this is due to disease. The duration of the QRS complexes decreased in all cases after exercise. This is a normal re-
spontaneous response to exercise. The voltage of the QRS complexes increased fairly consistently after exercise, and this is usual after prolonged exercise. Again this degree of increased voltage could not be duplicated by the nonrunners by holding the breath in inspiration. This suggests that the cause of the high voltage was cardiac rather than extracardiac. The electric axis of QRS complexes deviated to the right after exercise. The RS-T segments measured from the P-R level just before the QRS complexes exceeded the limit of normal in one runner. This subject appeared disoriented and unusually exhausted after running but had no chest pain. The infrequency of this finding suggests that this is a pathologic response to exercise. The T-waves showed large increases in amplitude after severe exercise, which is in keeping with the findings of others. The changes were greatest in the leads with the tallest QRS complexes. This increase in the voltage of T was the most conspicuous result of exercise in many cases. The changes in the blood potassium did not explain these changes. These waves were not accompanied by abnormal gradients, and thus were not considered to be the result of ischemia. Others have reported inverted T waves in leads II and V₄, V₅, and V₆ with intensive exercise but this was not noted in this series. The T axis moved to the right. The Q-Tc interval changed little as a result of running. In one case the Q-Tc exceeded the upper limit of normal of 0.45 second. In no other instance was the degree of prolongation of Q-Tc comparable to that seen in patients with heart disease after exercise.

Teleoroentgenograms of the hearts of the runners at rest showed the presence of cardiac enlargement in a significant percentage of subjects. The greatest increase above normal of the cardiac measurements was in the cardiac area. Both the long and broad diameters of the heart were increased likewise above normal. In general the x-ray films revealed a cardiac silhouette which was long and extended to the left. The pulmonary artery and pulmonary vascular markings were considerably increased in diameter and density. The great vessel shadows were large. Immediately after running 26 miles the cardiac shadow was considerably smaller.

Summary

The electrocardiographic, radiologic, electrolytic and other findings are described in 165, 26-mile marathon runners and 40 normal control nonrunners. There was a characteristic electrocardiographic picture which consisted of a slow cardiac rate with high voltage of QRS complexes, T and U waves. The picture is characteristic of right and left ventricular enlargement without evidence of cardiac disease. Radiologically the heart was enlarged in its broad, long and transverse diameters before the race and was considerably smaller immediately after running. The recognition of this picture and its relation to previous physical training is important in the differential diagnosis of cardiac enlargement.

Acknowledgments

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Sumario Español

Los hallazgos electrocardiográficos, radiológicos, electrolíticos y otros se describen en 165 corredores del maratón de 26 millas y en 40 controles normales no corredores. Se encontró un cuadro electrocardiográfico característico que consistió de un pulso bajo con alto voltaje de los complejos QRS y las ondas T y U. El cuadro es característico de engranecimiento ventricular derecho e izquierdo sin evidencia de enfermedad cardíaca. Radiológicamente el corazón se encontró engranecido en su diámetro ancho, transverso y largo antes de la carrera y considerablemente más pequeño luego de la carrera. El reconocimiento de este cuadro y su relación a previo entrenamiento físico es importante en el diagnóstico diferencial de engranecimiento cardíaco.

References

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