Noninvasive Evaluation of Ventricular Hypertrophy in Professional Athletes

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SUMMARY Athletes often exhibit ECG findings which are considered to be abnormal. Therefore, we used noninvasive graphic methods to study 42 active professional male basketball players, ranging in age from 21 to 31 years, without clinically evident heart disease. Of the 42, 11 (25%) met the Romhilt-Estes ECG voltage criteria for left ventricular hypertrophy, and 12 (29%) satisfied VCG criteria for left ventricular enlargement; nine (21%) had left ventricular hypertrophy by both methods. In 33 subjects (79%) the 0.04 sec vector in the horizontal plane was anterior, and 29 of these exhibited one or more standard criteria for right ventricular hypertrophy by ECG, while 18 satisfied VCG criteria for right ventricular enlargement; the ECG and VCG were concordant for right ventricular hypertrophy in 16 subjects (38%). Submaximal treadmill exercise tests (Bruce protocol) were normal in eight athletes, while in one subject ventricular premature beats occurred during the test. In 24 of 25 athletes (96%) from whom phonocardiograms were obtained a third heart sound was recorded, while in 14 (56%), a fourth heart sound was present. Of the 14 athletes who had a fourth heart sound, 12 (86%) had either ECG or VCG evidence of ventricular hypertrophy. Only four of 23 athletes had an increased cardiothoracic ratio (> .50) on routine chest X-ray.

Ten athletes and ten control subjects matched for height, weight and body surface area had echocardiograms satisfactory for analysis. The left ventricular end-diastolic dimension in the athletes averaged 53.7 ± 1.3 (SE) mm compared with a value of 49.9 ± 0.7 mm in the control subjects (P < 0.02), and was increased (≥ 56 mm) in four. Left ventricular posterior wall thickness averaged 11.1 ± 0.6 mm, compared with a value of 9.8 ± 0.5 mm in the control subjects (P < 0.05), and was increased (≥ 11 mm) in six athletes. The right ventricular end-diastolic dimension averaged 20.8 ± 1.1 mm compared with a value of 12.9 ± 2.2 mm in the controls (P < 0.004), and was increased (≥ 23 mm) in four athletes. No athlete or control subject exhibited paradoxical septal motion. In the athletes, ejection fraction (cube method) averaged 79 ± 2.0% and mean Vcf averaged 1.13 ± 0.04 circ/sec; these values did not differ from those of the control subjects. Thus, both right and left ventricular enlargement (“physiological hypertrophy”) are often present in the well-trained athlete, but left ventricular performance remains normal in the basal state in such individuals. We conclude that these individuals represent a selected subgroup of subjects who are variants of normal.

CONTINUING CONTROVERSY surrounds the concept of the so-called “athlete’s heart.” The effects of training and of the repeated stress of exercise produce a variety of alterations described under the vague designation “athletic heart syndrome,”1-6 and have led to the suggestion that the well-trained athlete’s heart is diseased. Since no comprehensive studies of left ventricular performance in such individuals are available, we sought to examine the cardiac status of professional basketball players by a variety of noninvasive techniques.

Methods

Forty-two professional basketball players were studied over three years. A routine pre-employment annual history and physical examination were performed in the training camp by the team physician. The average age was 24.6 years (range 21 to 31), and the average height was 6'5" (range 5'10" to 6'10"). A control group of ten individuals, who were matched for age, sex, height, weight and body surface area with the group of athletes, and who had satisfactory ultrasound studies was also studied.

A standard 12-lead ECG, supplemented by a V3R lead and an inspiratory rhythm strip, was obtained in all athletes and in the control subjects. Recordings were made in the supine position during quiet respiration. In nine athletes and in all control subjects a treadmill exercise test was performed employing a modified Bruce protocol.7 (Except for the ECG, it was not possible to obtain all studies in all the athletes since participation in the other studies was purely voluntary.)

The diagnosis of right ventricular hypertrophy by ECG depended on the presence of one or more of the following criteria described by Sokolow and Lyon:8 intrinsicoid deflection in V1, greater than 0.04 seconds, R/S ratio in V1, greater than 1, R in V1 of 7 mm or more, S in V1 less than 2 mm, R in V1 plus S in V5 or V6 greater than 10.5, R/S ratio in lead V5 or V6 equal to or less than 1, R in aVr 5 mm or more, and right axis deviation greater than or equal to 110 degrees.

The ECG diagnosis of left ventricular hypertrophy was based on the “point-score” system of Romhilt and Estes.9 In each instance where the diagnosis of left ventricular hypertrophy was made, there was either 20 mm of QRS voltage in the limb leads or 30 mm of QRS voltage in precordial leads V1, V2, V5 or V6. In addition, an intrinsicoid deflection in lead V5 or V6 exceeding 0.05 sec usually was present. These ECG abnormalities are frequently observed in individuals with left ventricular volume overload.10

A Hewlett-Packard Vector Programmer Model 1507-A was utilized to obtain a standard Frank lead system vectorcardiogram (VCG). The VCG diagnosis of right ventricular hypertrophy, derived from criteria of Chou et al.,11 was made if in the horizontal plane more than 70% of the loop area was anterior and rightward, or if more than 20% of the loop area was posterior and rightward. For the VCG diagnosis of left ventricular hypertrophy in these young men, the criteria of Pipberger et al. were utilized.12

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A phonocardiogram was obtained in 25 athletes and in all ten of the control subjects using an Elena-Schonander Mingograph-34 with a Siemens-Elena E-117E transducer and amplifier system. An apex diagram and an apical recording of the heart sounds at 50 Hz were obtained in the supine position. An indirect carotid arterial tracing was obtained along with a recording of the heart sounds at 100 Hz at the upper left sternal border. Twenty-three athletes and all control subjects had a posteroanterior chest film available for analysis.

Echocardiograms were obtained with a Picker Echoline instrument, employing a 2.25 MHz transducer, 1/2 inch in diameter, in the M mode interfaced to a Honeywell strip chart recorder. High-quality recordings satisfactory for analysis were obtained from ten athletes and all the control subjects. All measurements were recorded in the supine position to avoid the artifactual increase in the right ventricular end-diastolic dimension that would be obtained in the left lateral decubitus position. For right ventricular and septal measurements the angulated transducer was pointed inferiorly and laterally as described by Popp and coworkers. Conveniently, this chord also corresponds to that routinely used in our laboratory for the measurement of the mean velocity of circumferential fiber shortening (Vcf). For each calculation, five cycles were measured and averaged. The dimension criteria employed were modified from those described by Feigenbaum. The upper limits of normal for each measurement were taken as right ventricular end-diastolic dimension ≥ 23 mm; left ventricular septal thickness ≥ 14 mm; left ventricular end-diastolic dimension ≥ 36 mm; posterior left ventricular wall thickness ≥ 11 mm. Chi-square analysis and comparison by the Student’s t-test were performed wherever appropriate.

Results

Electrocardiographic and Vectorcardiographic Studies

All athletes had ECGs and VCGs available for analysis and in many subjects duplicate records were obtained one or more years apart. All ECGs were abnormal in some respect (table 1). The most common abnormalities were sinus bradycardia, sinus arrhythmia, junctional rhythm and first-degree A-V block. Rhythm disturbances often were exaggerated with an inspiratory rhythm strip, and one athlete had ventricular premature beats (< 5 per 100 cycles). As seen in table 1, ST-segment elevation (early repolarization), biphasic T waves and abnormal T-wave inversion were frequently seen in the ECG and in the VCG.

Although 33 of 42 athletes (79%) had a 0.04 second vector which was still anterior in the horizontal plane, only 29 (69%) met ECG criteria for right ventricular hypertrophy. The criterion most frequently satisfied in the scalar ECG was a delayed intrinsicsoid deflection in lead V1. Eighteen athletes (43%) met VCG criteria for right ventricular hypertrophy, usually by the anterior and rightward location of more than 70% of the loop area in the horizontal plane. Thirty-eight percent (16 of 42) had right ventricular hypertrophy by both techniques while only 11 (26%) failed to show right ventricular hypertrophy by either method.

Twenty-six percent (11 of 42) of these trained athletes met ECG criteria and 12 (29%) met VCG criteria for left ventricular hypertrophy. Nine (21%) had left ventricular hypertrophy by both methods, while two-thirds of the group met neither VCG nor ECG criteria for left ventricular hypertrophy.

Nine athletes performed submaximal treadmill exercise. Each athlete who exercised had ST-segment elevation of the early repolarization type in the resting tracing and five had sinus bradycardia, bradyarrhythmias or junctional rhythms. All of these changes normalized with exercise. One athlete had ventricular premature beats before, during and after the exercise test.

Phonocardiographic and X-ray Studies

In 25 basketball players in whom phonocardiograms were obtained, all but one individual had a third heart sound recorded and 14 (56%) had a fourth heart sound. Twelve of 14 players with an S3 gallop had ECG or VCG evidence of ventricular hypertrophy. Nine of the ten members of last year’s team (the exception was a rookie guard) had a recordable S4, such as that illustrated in figure 1.

The posteroanterior chest films showed that four of 23 athletes (17%) had an increased cardiothoracic ratio (greater than 0.50).

Ultrasound Studies

Figure 2 depicts the measurement of standard echocardiographic dimensions in two athletes. The results of echo dimensional analysis are shown in table 2 and figure 3. Five of ten athletes had a right ventricular end-diastolic dimension of 23 mm or more and the left ventricular end-diastolic dimension was increased in four. Left ventricular posterior wall thickness was increased in six of ten athletes. Left ventricular septal thickness was 14 mm or more in six athletes. As seen in table 2, when echographic measurements of the control group were compared with the athletes, there was a significant difference in right ventricular end-diastolic dimension, left ventricular end-diastolic pressure and posterior wall thickness. Although the septal thickness in the control group showed no significant difference compared with the athletes, the average dimension in both groups exceeded 11 mm. In addition, the septal/posterior wall ratio was 1.3 or more in four of the athletes and in five of the con-

<table>
<thead>
<tr>
<th>Table 1. Electrocardiographic Abnormalities</th>
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<tr>
<td>Rhythm</td>
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<tr>
<td>Sinus arrhythmia and bradycardia</td>
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<tr>
<td>WAP, ectopic atrial rhythm</td>
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<tr>
<td>Junctional rhythm</td>
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<td>1st A-V block</td>
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<td>Wenckebach periods</td>
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<tr>
<td>Repolarization abnormalities*</td>
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<td>ST elevation ≥ 1 mm</td>
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<td>Biphasic or inverted T waves (excluding leads III and V1)</td>
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*Excluding 3 with IRBBB.
Abbreviations: WAP = wandering atrial pacemaker; IRBBB = incomplete right bundle branch block.
FiguRe 1 The simultaneously obtained electrocardiogram (ECG), apexcardiogram (ACG) and phonocardiogram recorded at the left ventricular apex in a 23-year-old, 67th professional basketball player. A fourth heart sound (S4) is recorded coincident with the "A" wave of the ACG and a third heart sound (S3) is recorded coincident with the rapid filling wave (RFW) of the ACG.

Table 2. Echocardiographic Measurements of Athletes and Controls

<table>
<thead>
<tr>
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<th>Athletes (N = 10)</th>
<th>Control subjects (N = 10)</th>
<th>P values</th>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>25.4 ± 1.1*</td>
<td>26.5 ± 1.1 NS</td>
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<tr>
<td>Height (in)</td>
<td>76.4 ± 1.4</td>
<td>76.7 ± 0.8 NS</td>
<td></td>
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<tr>
<td>Weight (lb)</td>
<td>192.1 ± 7.3</td>
<td>192.5 ± 2.5 NS</td>
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<tr>
<td>BSA (m²)</td>
<td>2.19 ± 0.06</td>
<td>2.20 ± 0.02 NS</td>
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<tr>
<td>HR (beats/min)</td>
<td>53.4 ± 2.0</td>
<td>65.9 ± 2.6 0.001</td>
<td></td>
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<tr>
<td>IVEDD (mm)</td>
<td>20.8 ± 1.1</td>
<td>12.9 ± 2.2 0.004</td>
<td></td>
</tr>
<tr>
<td>Septal thickness (mm)</td>
<td>13.7 ± 0.5</td>
<td>12.8 ± 0.6 NS</td>
<td></td>
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<tr>
<td>Septal/posterior wall</td>
<td>1.2 ± 0.1</td>
<td>1.3 ± 0.1 NS</td>
<td></td>
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<tr>
<td>LVESD (mm)</td>
<td>53.7 ± 1.3</td>
<td>49.9 ± 0.7 0.02</td>
<td></td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>31.9 ± 1.5</td>
<td>31.1 ± 0.8 NS</td>
<td></td>
</tr>
<tr>
<td>LVESD thickness (mm)</td>
<td>11.1 ± 0.6</td>
<td>9.8 ± 0.3 0.05</td>
<td></td>
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<tr>
<td>LVEDV (ml)</td>
<td>157 ± 11</td>
<td>126 ± 5 0.02</td>
<td></td>
</tr>
<tr>
<td>SV</td>
<td>123 ± 8</td>
<td>95 ± 4 0.007</td>
<td></td>
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<tr>
<td>EF (%)</td>
<td>79 ± 2</td>
<td>76 ± 1 NS</td>
<td></td>
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<tr>
<td>Mean Vcf (circ/sec)</td>
<td>1.13 ± 0.04</td>
<td>1.18 ± 0.05 NS</td>
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*All data are reported as mean ± SE. Abbreviations: BSA = body surface area; HR = heart rate; IVEDD = right ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; LVESD = left ventricular end-diastolic volume; SV = stroke volume; EF = ejection fraction; Vcf = rate of circumferential fiber shortening; NS = not significant.

trol subjects. The athletes' left ventricular end-diastolic volume averaged 157 ml and stroke volume averaged 123 ml. These values were significantly increased compared to the control group (table 2). The average ejection fraction of 0.79 was not significantly different from the control subjects. Mean Vcf averaged 1.13 circ/sec in the athletes, which is within the range of normal; this value was not significantly different from the control group.

Discussion

Since 1896, when the Greek government established the marathon (40 km run) as an Olympic event, physicians have studied the physiologic effects of severe exertion and have debated the possible pathologic consequences of their findings. The original observations of an irregular, slow pulse and cardiomegaly diagnosed by percussion have been confirmed by ECG and X-ray techniques. The term "athlete's heart" was coined to describe the findings of bradycardia, often associated with atrial arrhythmias, nodal rhythm or Wenckebach periods, repolarization abnormalities, left and right ventricular hypertrophy, intermittent systolic murmurs, S1, gallop and increased cardiothoracic ratio observed in athletes engaged in vigorous competitive sports. It has frequently been suggested that the hearts of these athletes have become diseased and that the repeated stress of exercise has impaired their cardiac performance. However, the effects of vigorous exercise in youth upon subsequent longevity remain controversial.

Cardiac catheterization has not been performed in such athletes since they usually do not exhibit symptoms to justify invasive diagnostic measures. Since the availability of echocardiography makes possible the noninvasive measurement of left ventricular performance, we sought to compare the previously described features of the so-called "athlete's heart syndrome" with standard clinical techniques (ECG, VCG, chest X-ray, phonocardiogram) as well as with ultrasound measurements of left ventricular performance and dimensions.

ECG and VCG Studies

The resting scalar ECG of endurance athletes has been intensively studied. Beckner and Winsor reported that 33% of their marathon runners satisfied ECG voltage criteria for left ventricular hypertrophy and 18% satisfied such criteria for right ventricular hypertrophy. Once the marathon runner discontinued endurance exercise, however, the changes of right ventricular hypertrophy regressed in all but three while the excessive voltage characteristic of left ventricular hypertrophy persisted. Consistent with these data is the observation that increased heart size by X-ray has been a prominent finding among older athletes even when the heart rate has increased. In our study, the incidence of left ventricular hypertrophy was similar to that reported by Beckner and Winsor (26 vs 33%).

Professional basketball players in our study exhibited a higher incidence of right ventricular hypertrophy by ECG and VCG than previously documented in reports describing other endurance athletes. For example, Arestila and Koivikko, who also used the Frank lead system to study 46 endurance athletes, reported that eight (17%) had a one-half area vector which was anterior in the horizontal plane. By
FIGURE 2  Left) Echocardiogram in a 31-year-old, 6'1" athlete who has been playing professional basketball for eight years. The interventricular septum is thickened (16 mm). The right ventricular end-diastolic dimension (RVEDD) is enlarged (24 mm). The left ventricular posterior wall (LVPW) also is slightly thickened (11.4 mm), while the left ventricular end-diastolic dimension (LVEDD) and the left ventricular end-systolic dimension (LVESD) both are normal. This athlete's ECG met criteria for right ventricular hypertrophy, while his VCG satisfied criteria for left ventricular hypertrophy. Both an S₃ and an S₄ were recorded in this individual. Right) Echocardiogram in a 23-year-old, 6'11" professional basketball player. The left ventricular (LV) posterior wall is thickened (13.5 mm), the LV end-diastolic dimension (EDD) is enlarged (36 mm) and the right ventricular (RV) EDD is also abnormal (23 mm). This athlete's ECG met criteria for both right and left ventricular hypertrophy. In each of the above examples, both the ejection fraction and mean Vcf were normal.

In contrast, this finding was present in 50% of the athletes in our series. Of interest was the observation that athletes released from the team had a reduced frequency of persistent anterior forces. As indicated above, it is known that anterior forces tend to regress with lack of training.4, 30

In addition to ventricular hypertrophy, it has been shown previously that athletes often tend to exhibit cardiac arrhythmias, including sinus bradycardia, sinus arrhythmia, junctional rhythm, ectopic atrial rhythm, Wenckebach periods and repolarization abnormalities. The electrocardiographic findings in the athlete have been recently reviewed, 4 and the subjects in our study demonstrated many of the same phenomena reported previously. Thus, on the basis of our own studies and from a review of the literature, it appears that these professional basketball players are similar to other endurance athletes and have biventricular hypertrophy of the volume overload type according to standard criteria and commonly exhibit atrial arrhythmias and repolarization abnormalities.

The maximal oxygen uptake has been utilized as a method for determining the level of training in athletes,31-34 and had it been available in our subjects we would have permitted direct comparison of these professional basketball players with cross-country skiers, long-distance runners, speed skaters, orienters, runners and cyclists.33 However, these professional basketball players volunteered only for basal, noninvasive studies. As indicated in the Results, these individuals had a low resting heart rate,32, 33, 38 increased stroke volume and ultrasound evidence of left and right ventricular hypertrophy compared with tall control subjects.

FIGURE 3  Dimension data from ultrasound recordings in ten professional athletes. RVEDD = right ventricular end-diastolic dimension; LVEDD = left ventricular end-diastolic dimension.

[Diagram showing echocardiogram dimensions and data]
addition, the resting stroke volume of these athletes was considerably greater than that of 25 normal subjects of average size ranging in age from 21 to 29 years recently studied by ultrasound in our laboratory in the basal state (123 vs 91 ml, \( P < 0.001 \)). These observations are consistent with the hypothesis that the professional basketball players we studied were well-trained athletes.

Whether a variety of constitutional variables could account for our results deserves comment. To control for the young age of our subjects we employed the criteria of Pipberger et al. These same investigators found no effect of height on the orthogonal ECG and VCG but noted increased anterior forces in the presence of a decreased anteroposterior diameter. In a recent report Van Ganse and associates examined the role of such variables in a group of 30 cyclists compared to controls matched for age, height, sex and weight and confirmed an increased incidence of bradycardia and increased voltage suggestive of biventricular hypertrophy among the athletes. In our study, comparison with a matched group of tall control subjects suggests that body habitus does not account for the differences observed with respect to ventricular hypertrophy. Since many of the athletes were black, the question of race also requires consideration. Published scalar ECG data among a variety of tribal and ethnic subgroups in South Africa have revealed that it is unusual for the net voltage to exceed 26 mm in any of the precordial leads. Such a voltage increase occurred in only 11.7\% of the Bantu (aged 20–29) studied by Walker and Walker. The percentage in our series is higher (26\%), even though we employed a more stringent criterion (30 mm of QRS voltage) which tends to reduce false positive results. To account for some of the ECG alterations observed among African blacks, the survival value of athletic ability should be considered. Thus, some of the individuals described in the study of Walker and Walker may have been at the same level of physical fitness as many endurance runners.

Repolarization abnormalities occur among blacks to a greater extent than among whites. Athletes of all races have been shown to exhibit repolarization abnormalities similar to the patterns seen in young black Americans. In the earliest published examples of marathon runners' ECGs, repolarization changes were evident in examples 2, 4 and 5. The incidence of ST elevation greater than 1 mm (early repolarization) by ECG or VCG (77\%) and of T-wave abnormalities (not including T-wave inversion in leads V\(_1\) or III) (33\%) are greater than expected for race. Thus, it is unlikely that racial characteristics alone can explain our results.

**Phonocardiographic Studies**

The presence of "curious prolongation of the first heart sound, not unlike the crescendo murmur of mitral stenosis" and of a "reduplicated first sound at the apex" was reported in marathon runners who participated in the 1928 Olympic Games. However, this observation has not been further defined or confirmed. In our series an S\(_4\) gallop which coincided with the "A" wave of the apexcardiogram was recorded in 56\% of our subjects (fig. 1). Often this gallop sound was audible, even in training camp. Third heart sounds were, of course, quite frequently recorded and were easily audible as previously described. Using the same techniques, fourth heart sounds were not recorded in our tall control group. Whether the mechanism of production of the fourth heart sound is related to heart rate, to P-R interval or to intrinsic changes in the diastolic properties of the left ventricle is highly speculative. Both an S\(_4\) and an S\(_5\) gallop were present in the five athletes with left ventricular hypertrophy. In these athletes the possible pathological significance of an S\(_4\) also remains unclear.

**Echocardiographic Studies**

The ultrasound data substantiate the presence of biventricular hypertrophy in these athletes and demonstrate that in the basal state these individuals exhibit normal left ventricular performance. Both the left ventricular and right ventricular end-diastolic dimensions were increased. When average left ventricular end-diastolic dimension was compared with the average right ventricular end-diastolic dimension, neither ventricle seemed to predominate (table 2). Nevertheless, both dimensions were significantly greater than those recorded in the control group as well as in the normal subjects reported by Popp and coworkers. Despite the presence of an increased right ventricular end-diastolic dimension in five of the ten athletes and the ECG and VCG incidence of approximately twice as much right as left ventricular hypertrophy, the interventricular septum moved in the normal fashion during both systole and diastole.

Septal thickness was increased beyond previously published upper limits in both the athletes and the matched tall control group. Although septal/posterior wall thickness ratios were greater than 1.3 in four athletes and in five control subjects, the mitral valve echo was entirely normal in these individuals. Thus, despite the presence of what has previously been described as a criterion for asymmetric septal hypertrophy, there is no evidence to support a diagnosis of hypertrophic obstructive cardiomyopathy in any of our subjects. Whether these data can be explained as a consequence of the technical limitations of choosing septal and posterior wall measurements in the same chord used to measure both the right and left ventricular end-diastolic dimensions will require further examination. In this connection, the possibility of measuring an increased posterior wall thickness by using an oblique beam and thereby producing an artificially decreased ratio has been commented on by Henry and coworkers. Conversely, in tall, thin people with narrow chest diameters, it is possible that the ultrasound beam passes obliquely through the septum leading to an artificially increased ratio. These observations suggest that caution should be used in assessing asymmetrical septal hypertrophy in athletes and in young, tall, normal males. Caution in the application of currently available echo criteria for asymmetric septal hypertrophy has also been suggested by Wise and coworkers and by Shah.

Our observations concerning left ventricular ultrasound dimensions are similar to those reported by Morganroth and associates in endurance athletes. However, these investigators did not measure right ventricular dimensions in their subjects, and described increases in left ventricular posterior wall thickness only in athletes who performed
isometric exertion. Recent data reported in trained dogs tend to confirm our own observations. Wyatt and Mitchell noted an increase in end-diastolic left ventricular wall thickness and in the maximum QRS magnitude in dogs exercised on a treadmill over a 12 week period.47

Our data are consistent with the views of Grande and Taylor29 who proposed that “physiological hypertrophy” of the heart represents a useful mechanism of adaptation in the athlete. Compared to the tall control group, mean Vcf and ejection fraction in this group of athletes showed no difference despite the lower heart rate of the athletes which tends to reduce mean Vcf.48,49 In addition, the values for mean Vcf were within the previously published range of normal19 and were consistent with the data obtained in a recent study using young, normal volunteers.49

As indicated earlier, previous investigations have suggested that right ventricular forces may be reduced in athletes who cease to exercise,4,50 and animal studies have documented a decrease in left ventricular wall thickness after cessation of exercise.51 By contrast, other reports suggest that despite an increased heart rate after discontinuation of exercise, ECG and X-ray evidence of left ventricular enlargement persist.28 In the future, serial ultrasound studies in athletes should be employed to resolve and clarify these issues.

Summary and Conclusions

We conclude that right and left ventricular enlargement presumably due to “physiological hypertrophy” are common findings among professional basketball players and that gallop rhythm is frequent among such individuals. Increases in right and left ventricular internal dimensions determined by ultrasound methods are also common and a septal/posterior wall thickness ratio greater than 1.3 can often be documented. These findings are not unusual in well trained athletes and may lead to the erroneous diagnosis of heart disease in such individuals. However, we have demonstrated that such athletes exhibit normal left ventricular performance in the basal state. These echocardiographic data support the contention that despite ECG, VCG and phonocardiographic findings usually considered to be abnormal, the athletes we studied are free of organic cardiovascular disease. The ultrasound data suggest that caution should be employed in the application of currently proposed ultrasound criteria for asymmetric septal hypertrophy. We further suggest that the “athletic heart syndrome,” rather than being a pathological state, should be regarded as a normal variant. In this context, the stimulus to hypertrophy from the repeated stress of competitive sports has elicited a normal response in cardiac muscle resulting in hypertrophy that is balanced between the two ventricles and that corresponds to the load induced by the exercise level. The rate at which this “super normal” state then regresses with cessation of the repetitive exercise stress remains to be determined, as do the effects, if any, of such regression on cardiac performance.

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