Results of Screening a Large Group of Intercollegiate Competitive Athletes for Cardiovascular Disease

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To determine the feasibility of detecting cardiovascular disease in a large group of young competitive athletes, a prospective screening evaluation of intercollegiate student athletes was undertaken at the University of Maryland. Initial clinical screening (including personal and family history, physical examination and 12 lead electrocardiogram) was performed in 501 athletes. Ninety of these subjects had positive findings on one or more of the three studies and agreed to further cardiologic evaluation. The vast majority (75 [84%] of 90) had no definitive evidence of cardiovascular disease, although 1 athlete had mild systemic hypertension and 14 (15%) had echocardiographic evidence of relatively mild mitral valve prolapse that had not been previously suspected.

In three athletes with relatively mild ventricular septal hypertrophy (14 to 15 mm), it was not possible to discern with absolute certainty whether the wall thickening was a manifestation of hypertrophic cardiomyopathy or secondary to athletic conditioning ("athlete heart"). Therefore, this screening protocol identified no athletes with definite evidence of hypertrophic cardiomyopathy, Marfan’s syndrome or other cardiovascular diseases that convey a significant potential risk for sudden death or disease progression during athletic activity. This failure to identify such diseases could have been due to a lack of sensitivity of the screening tests or to the low frequency with which these diseases occur in youthful healthy athletes. A systematic preparticipation screening program (such as the present one) does not appear to be an efficient means of detecting clinically important cardiovascular disease in young athletes.

The highly conditioned competitive athlete epitomizes the most healthy segment of our society. Nevertheless, occasionally youthful (or older) competitive athletes may die suddenly, often during athletic activity (1–9). Because such catastrophes are totally unexpected, they convey a particularly tragic and alarming connotation to the community. Previous studies (1,2) have shown that sudden death in young athletes is due to covert cardiovascular disease in the vast majority of instances, usually a congenital malformation of the heart or great arteries and most commonly hypertrophic cardiomyopathy. Consequently, there has been increasing interest in defining procedures by which those athletes at risk for sudden death due to cardiovascular disease can be prospectively identified before such catastrophes occur (10).

It has been estimated (11) that >25 million children and young adults participate in organized competitive athletic activities in the United States annually. Hence, it is obvious that a periodic individualized and comprehensive medical examination for each participant in organized sports is beyond the capabilities of the current medical care system. Although programs designed to screen large numbers of athletes for cardiovascular disease have been attempted sporadically, such examinations have varied considerably in approach and design and are frequently superficial and designed only to fulfill a perceived legal requirement. Indeed, it remains to be determined whether the screening of large groups of athletes for cardiovascular disease is a practical or cost-efficient endeavor. For this reason, the present prospective screening study was conducted in a large and well-defined group of youthful intercollegiate athletes.

Methods

Study design. The University of Maryland in College Park, Maryland is a member of the National Collegiate Athletic Association (Division I), with a large and successful intercollegiate athletic program involving about 500 to 600
male and female athletes during each academic year. All student athletes are asked by the University to undergo an annual evaluation at the University Health Center to determine their medical fitness for competition.

Students who expressed an interest in participating in an intercollegiate sport between September 1984 and June 1985 were evaluated and constitute the present study group. Initial evaluation at the University Health Center included: 1) personal and family history, 2) physical examination (including blood pressure determination), and 3) 12 lead scalar electrocardiogram (ECG). History and physical examination were performed by at least one of the three clinicians who are permanent members of the Health Center staff. Athletes who expressed a positive response to the history or showed an alteration on the physical examination or ECG (Table 1) were referred to the Cardiology Branch of the National Institutes of Health for a noninvasive cardiovascular evaluation (including M-mode and two-dimensional echocardiography, history, physical examination, ECG and chest radiograph).

**Characterization of study group.** A total of 540 athletes constituted the initial study group. However, 27 athletes refused to participate in the protocol and 12 others disclosed that they had previously undergone a noninvasive cardiologic evaluation (including echocardiography) by a private physician of their choice. Hence, 501 athletes participated in the preparticipation screening evaluation and constituted the final study group. Athletes ranged in age from 17 to 30 years (mean 19.3); 357 (71%) were men. The majority were white (381 [76%]); 114 (23%) were black, and 6 (1%) were Asian or Hispanic. They were involved in 14 sports, although football contributed the largest number (152 [30%]) (Table 1).

**Echocardiography.** An Advanced Technology Laboratory (ATL) Mark 500 mechanical sector scanner with a 3 MHz transducer was used to perform the two-dimensional echocardiographic studies. Images of the heart were obtained in a number of cross-sectional planes, using standard transducer positions (12). M-mode echocardiograms were recorded with a dedicated Irex System II ultrasound unit equipped with a 2.25 MHz transducer, or were derived from the two-dimensional image under direct anatomic visualization.

Methods for imaging the ventricular septum and posterior left ventricular free wall with M-mode echocardiography have been described previously (13,14). Ventricular septal thickness was measured at the onset of the R wave of the ECG at the cross-sectional level where wall thickness was maximal; thickness of the posterior free wall was measured during the same phase of the cardiac cycle and at the same level. Other cardiac dimensions were assessed according to the criteria of the American Society of Echocardiography (14).

Mitrail valve echograms were recorded with M-mode echocardiography using an ultrasound window at the left sternal border (usually the third intercostal space) with the transducer perpendicular to the anterior chest wall and mitral valve. A late systolic or pansystolic (nadir in mid-systole) displacement ≥3 mm of one or both mitral leaflets posterior to the line of mitral leaflet coaptation connecting its closure (C) and opening (D) points was regarded as evidence of mitral valve prolapse (15–17). On the two-dimensional echocardiogram, mitral prolapse was considered to be present if portions of the mitral leaflets were displaced during systole beyond the plane of the mitral anulus (superiorly into the left atrium) on the parasternal long-axis view or on both the long-axis and apical four chamber views (18–20). A qualitative judgment was made regarding overall valve size and leaflet thickening or redundancy.

**ECG.** The standard 12 lead ECG was performed utilizing a Burdick EK-5A instrument with the patient in the supine position during quiet respiration. The ECG was considered abnormal if one or more of the criteria detailed in Table 2 were achieved.

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### Table 1. Distribution of Sports Participated in by 501 Intercollegiate Athletes

<table>
<thead>
<tr>
<th>Sport</th>
<th>No. of Athletes</th>
<th>% of Overall Group</th>
<th>No. (%) Referred for Cardiovascular Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Football</td>
<td>152</td>
<td>30</td>
<td>29 (32%)</td>
</tr>
<tr>
<td>Track and field/cross-country</td>
<td>51</td>
<td>8</td>
<td>22 (25%)</td>
</tr>
<tr>
<td>Lacrosse</td>
<td>38</td>
<td>11</td>
<td>8 (9%)</td>
</tr>
<tr>
<td>Soccer</td>
<td>43</td>
<td>8</td>
<td>12 (13%)</td>
</tr>
<tr>
<td>Field hockey</td>
<td>0</td>
<td>7</td>
<td>5 (6%)</td>
</tr>
<tr>
<td>Wrestling</td>
<td>24</td>
<td>5</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Basketball</td>
<td>11</td>
<td>4</td>
<td>5 (6%)</td>
</tr>
<tr>
<td>Swimming and diving</td>
<td>12</td>
<td>4</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Tennis</td>
<td>6</td>
<td>3</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Gymnastics</td>
<td>0</td>
<td>0</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Volleyball</td>
<td>0</td>
<td>2</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Baseball</td>
<td>11</td>
<td>2</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Golf</td>
<td>9</td>
<td>2</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>
Table 2. Criteria for a Positive History, Physical Examination and 12 Lead Electrocardiogram

History (personal and family)
1. Prior denial of medical approval for athletic participation, military service or life insurance
2. Occurrence of any of the following during the last 5 years
   - Near fainting, fainting or “blacking out”
   - Chest pain, pressure or discomfort occurring more than once
   - Irregular heartbeat or palpitation
   - Shortness of breath or fatigue out of proportion to the degree of exertion undertaken
3. Prior identification of elevated blood pressure
4. Prior medical illness requiring a physician’s care and lasting >1 week (depending on circumstance)
5. Close relative (parent, sibling, aunt, uncle or grandparent) who experienced a “heart attack,” disabling cardiovascular disease or sudden death before 50 years of age

Physical examination
1. Grade ≥2/6 systolic murmur
2. Any diastolic murmur
3. Second heart sound single or widely split and fixed with respiration
4. Elevated brachial blood pressure (>145/90 mm Hg on two or more readings)
5. Findings suggestive of mitral valve prolapse (mid-systolic click or late systolic murmur)
6. Irregular heart rhythm
7. Physical features suggestive of Marfan’s syndrome*

Electrocardiogram
1. R wave ≥30 mm in lead V5 or V6 or in a standard lead
2. S wave ≥30 mm in lead V1 or V2
3. ST segment depression or T wave flattening or inversion in two or more leads
4. Abnormal Q waves†
5. PR interval ≥0.25 or <0.12 second
6. Second or third degree atrioventricular block
7. Left or right atrial enlargement
8. QRS frontal plane axis deviation: right axis ≥120° and left axis ≥30°
9. Prolonged QT interval (corrected for heart rate) (21)
10. Right or left bundle branch block or Rsr' pattern in lead V1 or V2
11. Premature ventricular complexes

*These features included: 1) greater than average height for age with excessively long and thin limbs (arm span exceeding height); 2) looseness and hyperextensibility of joints; 3) arachnodactyly; 4) history of visual problems; 5) sparsity of subcutaneous fat; 5) pectus carinatum or excavatum. †Q (or QS) waves were considered abnormal if ≥0.04 second in duration or ≥3 mm deep and were present in two or more leads (Q or QS waves present only in leads V1 and V2 were not considered abnormal). ‡Left atrial enlargement was diagnosed if the product of the depth and duration of the negative portion of the P wave in lead V1 was greater than −0.03. §Right atrial enlargement was diagnosed if P waves in leads II and III or V1 were peaked and ≥2.5 mV in amplitude.

Results

Screening protocol. The results of the preparticipation screening protocol are summarized in Figure 1. Of the 501 study subjects, 102 had positive findings on one or more of the initial screening studies (history, physical examination and ECG); 83 athletes had alterations on one study alone (ECG, 57; physical examination, 16; history, 10) and 19 had alterations on two studies (physical examination and ECG, 6; history and ECG, 8; history and physical examination, 5). Of these 102 athletes, 12 chose not to cooperate further with the protocol, but the remaining 90 athletes (75 men and 15 women) underwent noninvasive cardiologic evaluation and testing (including echocardiography) and, therefore, constitute the final study subset. Abnormal physical findings, positive responses to the personal and family history and ECG alterations in these 90 athletes are summarized in Table 3.

Athletes referred for noninvasive evaluation. Of the 90 athletes referred for evaluation, 75 showed no definitive evidence of cardiovascular disease, and had normal left ventricular wall thickness (≤12 mm) (Fig. 2). Six of these 75 athletes had incidental echocardiographic findings that were considered to be of little or no clinical relevance, including 2 with a prominent right ventricular muscle band (moderator band) and 3 with left ventricular bands (false tendons); the latter structures were identified on parasternal long-axis or apical four chamber views as thin and discrete linear echoes evident throughout the cardiac cycle within the cavity extending from the mid-portion of the ventricular septum to the free wall (or papillary muscles) (22,23). One other athlete demonstrated chaotic motion of a small portion of the distal mitral valve apparatus, which had the appearance of a ruptured chorda tendinea, but this was not associated with mitral valve prolapse or regurgitation.

Three of these 75 athletes without definitive evidence of
Total No. Athletes

Figure 1. Flow diagram showing results of the preparticipation screening investigation for a cohort of competitive intercollegiate athletes studied at the University of Maryland. C-V = cardiovascular; HCM = hypertrophic cardiomyopathy; MVP = mitral valve prolapse.

cardiovascular disease showed a mild increase in ventricular septal thickness of 14 to 15 mm that was confined to the anterior and basal portion of septum; the left ventricular free wall was of normal thickness (≤12 mm); septal to posterior free wall thickness ratio was 1.2 in 2 of these athletes and 1.5 in the other. These findings were associated with left atrial enlargement (42 to 48 mm) in all three athletes; left ventricular end-diastolic dimension was normal in two (47 and 52 mm) and increased in one (58 mm) and each had a normal end-systolic dimension (31 to 38 mm). Systolic anterior motion of the mitral valve was absent in all three athletes. Each of the three athletes with septal thickening had been referred for noninvasive evaluation because of a soft systolic ejection murmur, but had a negative family history for heart disease and a normal ECG.

Fourteen (15%) of the 90 athletes had echocardiographic evidence of relatively mild mitral valve prolapse, demonstrated by M-mode echocardiography alone in 4, by twodimensional echocardiography alone in 2 and by both techniques in 8; the pattern of prolapse was pansystolic in each of the 12 athletes in whom it was identified by M-mode echocardiography. In 13 of the 14 athletes, the mitral valve leaflets were thin and pliable; only 1 had a true anatomic abnormality in which the valve appeared to be increased in size and the leaflets thickened and redundant. Two of the 14 athletes had auscultatory findings suggestive of prolapse (mid-systolic click), but none had evidence of mitral regurgitation on the basis of the auscultatory or Doppler examination. Only 1 of the 14 athletes with echocardiographic evidence of mitral valve prolapse reported cardiac symptoms that potentially were manifestations of mitral prolapse syndrome (that is, chest pain, syncope, dyspnea).

One athlete had a history of systemic hypertension and proved to have relatively mild elevation in blood pressure on physical examination (140 to 155/90 to 100 mm Hg).

In each of the 90 athletes referred for noninvasive cardiovascular evaluation, it was possible to identify by twodimensional echocardiography the origin of the left main coronary artery in its proper anatomic position (emanating from the left sinus of Valsalva) and thereby exclude an anomalous origin of the left coronary artery from the anterior sinus of Valsalva (24).

Cardiac dimensions obtained with M-mode echocardiography in the 90 athletes are summarized in Table 4. No athlete showed an increase in transverse aortic dimension. Left atrial, right ventricular and left ventricular end-diastolic dimensions for the group were typical of trained athletes (25,26). Several showed increased cavity dimensions, including 16 (18%) with a left ventricular end-diastolic dimension of 56 to 61 mm, 11 (12%) with a left atrial size of 41 to 48 mm and 11 (12%) with a right ventricular dimension of 31 to 38 mm. In each athlete, chest radiographs showed no cardiac or pulmonary abnormalities.

Athletes evaluated noninvasively outside the study protocol. Twelve university athletes (not in the group of 501) had been examined for cardiovascular disease by a private physician before the present screening study was initiated. A noninvasive evaluation including echocardiography showed no evidence of cardiovascular disease in 11 of the 12, whereas the other athlete proved to have systemic hypertension (blood pressure 180/130 mm Hg).

Discussion

Sudden death in athletes. Previous studies (1–3) have documented that sudden unexpected death may occur occasionally in young competitive athletes. Such catastrophes, although uncommon, are usually due to congenital cardiovascular disease (most commonly hypertrophic cardiomyopathy), but also to anomalies of the coronary arteries or a ruptured aorta as a manifestation of Marfan’s syndrome (1,2). These findings have stimulated physicians concerned with the medical care of athletes to consider methods whereby such cardiovascular diseases might be detected before catastrophes occur (10). Such information would make it possible to withdraw selected athletes from the potentially high risk circumstances of competitive athletics (27).

Limitations of preparticipation screening. There are many potential problems that make it difficult to establish successful and economically feasible large-scale screening
programs for the purpose of identifying cardiovascular disease in asymptomatic individuals. Truly comprehensive community-based screening is probably impractical because it would essentially require each athlete to undergo a non-invasive cardiologic evaluation. Given the large number of participants in intercollegiate athletic programs, all but the least expensive screening effort would be beyond the financial resources of most institutions. Hence, the present study constitutes an attempt to design a simple, practical and inexpensive protocol that might be readily applicable to other scholastic or collegiate settings for the purpose of detecting those cardiovascular diseases known to cause sudden death or produce significant morbidity in young athletes. Such diseases include hypertrophic cardiomyopathy and Marfan's syndrome, but also other congenital cardiac diseases and systemic hypertension.

**Design and findings of present screening protocol.** With these priorities in mind, the present protocol was structured to rely initially on the personal and family history, physical examination and 12 lead ECG. Because echocardiography was not used as an initial screening test in this study, we cannot be certain of its utility in a large group of asymptomatic young athletes. Intuitively, we would have expected echocardiography to enhance our sensitivity for the detection of certain cardiovascular abnormalities (such as hypertrophic cardiomyopathy and aortic dilation) (28,29); however, this test would also have been prohibitively expensive and generally impractical for the study of >500 athletes. The stress exercise ECG would also have important limitations as a primary screening test because of its expense, the high frequency with which a false positive ST segment response occurs in athletes (30) and the fact that coronary heart disease is very uncommon in young athletes (1,2,31).

In this investigation of >500 intercollegiate athletes, we identified no athlete with definitive evidence of cardiovascular disease of major clinical consequence (with the possible exception of 1 individual with relatively mild systemic hypertension). However, in 3 of those 90 athletes who were referred for further testing, hypertrophic cardiomyopathy could not be excluded with certainty on the basis of the echocardiographic findings. Each of these individuals had a mildly increased left ventricular wall thickness of 14 to 15 mm confined to the anterior portion of the ventricular septum (in the absence of systolic anterior motion of the mitral valve and evidence of obstruction to left ventricular outflow). Hence, although echocardiographic findings in these three athletes resembled a mild morphologic expres-
Table 3. Positive Family and Personal History and Electrocardiographic (ECG) Alterations in 90 Athletes Referred for Cardiovascular Evaluation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Positive Physical Examination \n(24 athletes)</th>
<th>Positive Family History \n(7 athletes)</th>
<th>Positive Personal History \n(13 athletes)*</th>
<th>ECG Alterations \n(65 athletes)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic murmur</td>
<td>19</td>
<td>Marfan’s syndrome</td>
<td>8</td>
<td>LVD†</td>
</tr>
<tr>
<td>Systolic click</td>
<td>2</td>
<td>Premature death</td>
<td>1</td>
<td>Symmetric T wave</td>
</tr>
<tr>
<td>Irregular rhythm</td>
<td>3</td>
<td>or “heart attack”</td>
<td>1</td>
<td>inversion</td>
</tr>
<tr>
<td>Chest pain</td>
<td>8</td>
<td>Near syncope</td>
<td>1</td>
<td>Heart murmur</td>
</tr>
<tr>
<td>LV diastolic dimension (mm)</td>
<td>2</td>
<td>Exertional dyspnea</td>
<td>3</td>
<td>Other ST-T</td>
</tr>
<tr>
<td>Right ventricular dimension (mm)</td>
<td>2</td>
<td>Irregular heartbeat</td>
<td>3</td>
<td>LAE</td>
</tr>
<tr>
<td>Posterior free wall thickness (mm)</td>
<td>2</td>
<td>Hypertension</td>
<td>2</td>
<td>Q waves</td>
</tr>
<tr>
<td>Ventricular septal thickness (mm)</td>
<td>10.5</td>
<td>or “heart attack”</td>
<td>6</td>
<td>LAD</td>
</tr>
<tr>
<td>Aortic root dimension (mm)</td>
<td>29.1</td>
<td>Hypertension</td>
<td>2</td>
<td>RBBB</td>
</tr>
<tr>
<td>Left atrial dimension (mm)</td>
<td>35.5</td>
<td>Hypertension</td>
<td>2</td>
<td>PVCs</td>
</tr>
<tr>
<td>Right ventricular dimension (mm)</td>
<td>24.2</td>
<td>Hypertension</td>
<td>2</td>
<td>PVCs</td>
</tr>
</tbody>
</table>

*Some athletes had more than one abnormality; four other athletes with an abnormal ECG on initial screening are not included in this analysis because they did not participate in the noninvasive cardiovascular evaluation.
†Based on increased voltages (that is, R wave in lead V3 or V4, or in a standard lead ≥30 mm or S wave in lead V1 or V2 ≥30 mm. ¶Systemic hypertension was not verified on subsequent examinations in one of these athletes.

LV = left ventricular.

Table 4. M-Mode Echocardiographic Dimensions in 90 Intercollegiate Athletes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal thickness (mm)</td>
<td>10.5</td>
<td>7 to 15</td>
</tr>
<tr>
<td>Posterior free wall thickness (mm)</td>
<td>10.1</td>
<td>7 to 14</td>
</tr>
<tr>
<td>Septal/free wall thickness ratio</td>
<td>1.0</td>
<td>0.7 to 1.5</td>
</tr>
<tr>
<td>LV diastolic dimension (mm)</td>
<td>50.9</td>
<td>41 to 61</td>
</tr>
<tr>
<td>Aortic root dimension (mm)</td>
<td>29.1</td>
<td>21 to 35</td>
</tr>
<tr>
<td>Left atrial dimension (mm)</td>
<td>35.5</td>
<td>26 to 48</td>
</tr>
<tr>
<td>Right ventricular dimension (mm)</td>
<td>24.2</td>
<td>12 to 38</td>
</tr>
</tbody>
</table>

LV = left ventricular.
and cardiovascular disease (44). In particular, a wide variety of ECG alterations are known to occur commonly in trained athletes (45–49), many of which may mimic those abnormalities observed in patients with structural heart disease. Indeed, almost 15% of the athletes in this study met our criteria for an abnormal ECG, but ultimately proved to have no evidence of underlying cardiovascular disease. Of note, the number of such “false positive” results will be influenced largely by the particular criteria arbitrarily assigned to define an abnormal ECG; for example, had we used QRS voltage ≥25 mm as a criterion for abnormality, we would have substantially increased the absolute number of abnormal ECGs, the vast majority of which would have represented additional false positive results.

The history and physical examination also have intrinsic limitations as screening tests for cardiovascular disease in a group of young athletes. For example, whereas the physical examination could be expected to detect virtually all patients with aortic valve stenosis because of the characteristic loud heart murmur, most patients with hypertrophic cardiomyopathy would not be identified by examination alone because the majority of patients with this disease have its nonobstructive form and, therefore, have a soft heart murmur or none at all (33,50).

Another possible limitation concerns the relatively small subset of 27 athletes (constituting 5% of the overall screening cohort) who refused to participate in the protocol. It is possible that these athletes did not cooperate with the study because they suspected or knew that they had cardiovascular problems. On the other hand, such lack of compliance is a well recognized risk of undertaking any large population study. Unfortunately, neither the University of Maryland nor the clinical investigators involved could actually require from the student athletes absolute cooperation with the screening protocol.

Conclusions. Designing a practical, cost-efficient and effective approach for the mass screening of large groups of young athletes is a formidable task. The present screening program initially utilizing history, physical examination and the 12 lead ECG identified no individuals with definitive evidence of cardiovascular diseases known to cause sudden death in young athletes, such as hypertrophic cardiomyopathy or Marfan’s syndrome with aortic dilation (although 15% of those ultimately referred for echocardiography had evidence of relatively mild mitral valve prolapse). These results may be explained by the low frequency with which many cardiovascular diseases occur in a group of young athletes or by the lack of sensitivity on the part of the examiner in detecting important cardiovascular disease, and it was also responsible for a large number of false positive observations.

References

6. Waller BF, Roberts WC. Sudden death while running in conditioned runners aged 40 years or over. Am J Cardiol 1980;45:1292–300.


