Outcomes of Cardiac Screening in Adolescent Soccer Players


BACKGROUND

Reports on the incidence and causes of sudden cardiac death among young athletes have relied largely on estimated rates of participation and varied methods of reporting. We sought to investigate the incidence and causes of sudden cardiac death among adolescent soccer players in the United Kingdom.

METHODS

From 1996 through 2016, we screened 11,168 adolescent athletes with a mean (±SD) age of 16.4±1.2 years (95% of whom were male) in the English Football Association (FA) cardiac screening program, which consisted of a health questionnaire, physical examination, electrocardiography, and echocardiography. The FA registry was interrogated to identify sudden cardiac deaths, which were confirmed with autopsy reports.

RESULTS

During screening, 42 athletes (0.38%) were found to have cardiac disorders that are associated with sudden cardiac death. A further 225 athletes (2%) with congenital or valvular abnormalities were identified. After screening, there were 23 deaths from any cause, of which 8 (35%) were sudden deaths attributed to cardiac disease. Cardiomyopathy accounted for 7 of 8 sudden cardiac deaths (88%). Six athletes (75%) with sudden cardiac death had had normal cardiac screening results. The mean time between screening and sudden cardiac death was 6.8 years. On the basis of a total of 118,351 person-years, the incidence of sudden cardiac death among previously screened adolescent soccer players was 1 per 14,794 person-years (6.8 per 100,000 athletes).

CONCLUSIONS

Diseases that are associated with sudden cardiac death were identified in 0.38% of adolescent soccer players in a cohort that underwent cardiovascular screening. The incidence of sudden cardiac death was 1 per 14,794 person-years, or 6.8 per 100,000 athletes; most of these deaths were due to cardiomyopathies that had not been detected on screening. (Funded by the English Football Association and others.)
SUDDEN CARDIAC DEATH IN A SEEMINGLY healthy athlete invariably prompts discussion about possible preventative screening strategies. The absence of a systematic registry of deaths in young athletes (<35 years old) has been a major obstacle to ascertaining the precise incidence of sudden cardiac death in this group; estimates vary between 0.5 and 13 deaths per 100,000 athletes, depending on the population studied and methods of data collection used. Furthermore, data on outcomes in adolescent athletes who have been screened for cardiovascular disease in a well-defined cohort are lacking.

The English Football Association (FA) has run a mandatory cardiac screening program for adolescent athletes in the United Kingdom since 1997, with a total of more than 11,000 athletes screened since its inception. The aim of this study was to determine the incidence and causes of sudden cardiac death in this well-defined population of previously screened soccer players.

METHODS

SCREENING PROGRAM

Between January 1, 1996, and December 31, 2016, a total of 11,168 soccer players at clubs affiliated with the FA underwent mandatory cardiovascular screening. The program encompassed all youth academy players (15 to 17 years of age) across the 92 professional clubs in the soccer league system who had excelled within the preceding 5 years. All such high-ranking players were offered a formal remunerated scholar contract (usually at the age of 16 years) with a view to progressing to a professional senior career. Written informed consent for screening was obtained from each player by the team doctor. Written informed consent from a parent or guardian was required for athletes younger than 16 years of age, in accordance with the FA governance department.

Athletes underwent assessment with a health questionnaire (Fig. S1 in the Supplementary Appendix, available with the full text of this article at NEJM.org), physical examination, 12-lead electrocardiography (ECG), and echocardiography. Mobile screening units staffed by accredited sonographers visited the clubs to conduct the evaluations, and the results were reviewed by an expert regional cardiologist who was a member of the FA-approved cardiology consensus panel. A formal report was sent to the FA medical department, in which each athlete was classified in one of three categories on the basis of the evaluation: normal; further evaluation needed, if an abnormality was detected that required further investigation to confirm or refute the presence of cardiac disease; or cardiac disease detected. The last category was subclassified into disorders that are associated with sudden cardiac death or those encompassing congenital septal and valvular conditions.

Further investigations were performed at regional specialist centers. Athletes with T-wave inversion, abnormally enlarged cardiac dimensions, or a low ejection fraction and those fulfilling echocardiographic criteria for left ventricular noncompaction underwent maximal exercise stress testing, 24-hour Holter monitoring, and cardiovascular magnetic resonance imaging (MRI). Athletes with a prolonged QT interval underwent exercise stress testing and 24-hour Holter monitoring, and athletes with a Wolff–Parkinson–White ECG pattern were risk-stratified on the basis of an exercise test and an electrophysiological study. From 2012 onward, there was sufficient evidence indicating that among black athletes, anterior T-wave inversion was a common variant that was not associated with structural heart disease or serious cardiac arrhythmias; therefore, asymptomatic black athletes with this repolarization abnormality did not undergo further investigation unless the echocardiogram was abnormal.

In 2013, we ceased to investigate athletes who fulfilled echocardiographic criteria for left ventricular noncompaction but had a normal ECG and normal cardiac function. The results of all the investigations were reported back to the FA headquarters and reviewed independently by the first and last authors.

DIAGNOSIS OF DISORDERS ASSOCIATED WITH SUDDEN CARDIAC DEATH

The diagnosis of hypertrophic cardiomyopathy was based on a left ventricular wall thickness of 15 mm or greater in any myocardial segment on echocardiography or cardiovascular MRI in the absence of another condition capable of producing left ventricular hypertrophy. The diagnosis of dilated cardiomyopathy was considered if the patient had a dilated left ventricle (a left ventricular end-diastolic dimension of >59 mm in males and >53 mm in females) and a reduced ejection fraction.
fraction (<52% in males and <54% in females).\textsuperscript{10,11} The diagnosis of arrhythmogenic right ventricular cardiomyopathy was based on published criteria.\textsuperscript{12,13} The diagnosis of long-QT syndrome was based on a corrected QT (QTc) interval of 500 msec or greater or of 470 to 490 msec in association with notched T waves in at least three leads, paradoxical prolongation of the QT interval with exercise, or a positive genetic test.\textsuperscript{14,15} Identification of the Wolff–Parkinson–White ECG pattern was based on findings of a short PR interval and a slurred upstroke to the QRS complex.

**RECOMMENDATIONS FOR FOLLOW-UP ASSESSMENTS OR EXCLUSION FROM PLAY**

Some of the screened athletes had abnormal T-wave inversion but structurally normal hearts, and others had borderline-abnormal cardiac dimensions but no other features to support the diagnosis of cardiomyopathy. These athletes were investigated with ECG and echocardiography performed every year and cardiovascular MRI performed every 2 years. Athletes with congenital valvular abnormalities or septal defects were monitored with annual ECG and echocardiography.

Athletes with cardiac disorders that are associated with sudden cardiac death were advised not to compete and were discharged into the care of the National Health Service. Decisions to disqualify such athletes were made by the FA cardiology consensus panel after discussions in accordance with current exercise recommendations of the European Society of Cardiology and the American Heart Association.\textsuperscript{16–18} The decision was relayed to the player in the presence of the player’s parent or guardian and the club doctor by the regional cardiologist.

**OUTCOMES**

Calculation of the follow-up period per athlete was based on the number of years of competition within the FA, which was determined from the FA registry of players. Deaths among athletes were ascertained through the development of a database that was compiled from voluntary reports to the FA. A second method to ascertain the number of deaths was through a secure survey that was sent to health professionals at each of the 92 FA-affiliated clubs, asking specifically about deaths from any cause. In addition, regular Internet searches had been performed since 2005, with the use of three different search engines (Google, Yahoo, and MSN search [Bing]) and at least 16 keywords (student, athlete, collapsed, died, death, heart, cardiac, arrest, attack, soccer, running, school, unknown, college, defibrillator, and saved).

Death certificates were obtained from the U.K. government for all deceased persons in the cohort to ascertain the causes of death, which were categorized broadly as accidental, suicide, drug-related, cancer, or cardiac causes. Autopsy data were available in all cases of sudden cardiac death, and diagnoses were based on previously established pathological criteria in conjunction with consultation with an expert cardiac pathologist.\textsuperscript{19}

Data on survival status during the screening program over the 20-year period among athletes who had diagnoses of cardiac disorders that are associated with sudden cardiac death were obtained from attending cardiologists, most of whom were part of the expert FA consensus panel.

**RESULTS**

**FURTHER EVALUATION AFTER SCREENING**

The mean (±SD) age of the 11,168 soccer players who underwent cardiovascular screening was 16.4±1.2 years; 10,581 (95%) of the athletes were male. In the entire cohort, 830 athletes (7%) underwent further investigation after preliminary assessment. Among these athletes, 104 (0.9%) reported symptoms that were deemed noncardiac in origin after assessment by a cardiologist. Among the remaining 726 athletes, 292 (3% of the total cohort) underwent investigation for T-wave inversion (anterior [153], inferior or lateral [114], or widespread [25]). A total of 25 athletes (0.2%) underwent investigation for a prolonged QTc interval. A further 409 athletes (4%) underwent cardiovascular MRI because of ventricular remodeling consistent with cardiomyopathy.\textsuperscript{9} Specifically, 229 athletes (2%) had a left ventricular wall thickness of 13 mm or greater; 106 (0.9%) had an enlarged left ventricular cavity with a borderline-low ejection fraction (of 50 to 52%)\textsuperscript{30}; 80 athletes (0.7%) had a suspected right ventricular regional wall-motion abnormality, and 19 (0.2%) had increased left ventricular trabeculation and a mildly reduced ejection fraction (of ≤50%).\textsuperscript{6} Twenty-five athletes had abnormal T-wave inversion and enlarged cardiac dimensions.
Outcomes of Cardiac Screening in Soccer Players

Figure 1. Outcomes in the Football Association Cardiac Screening Program.

11,168 Adolescent athletes underwent screening
10,581 Were male
587 Were female

830 Underwent further evaluation

287 Did not have abnormality detected at further evaluation
42 Had disorder associated with sudden cardiac death
5 Had hypertrophic cardiomyopathy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
3 Had long-QT syndrome
2 Had coronary artery anomaly
3 Had bicuspid aortic valve with either aortic-root enlargement or severe aortic regurgitation
26 Had Wolff–Parkinson–White electrocardiographic pattern

10,625 Did not have abnormality detected
225 Had other disorder
68 Had bicuspid aortic valve
62 Had atrial septal defect
13 Had ventricular septal defect
29 Had aortic regurgitation
24 Had mitral-valve prolapse
18 Had patent ductus arteriosus
9 Had pulmonary stenosis
2 Had trisomy

501 Underwent continued clinical surveillance

276 Had no diagnosis
23/ Died from cardiac-related cause
23/ Died from any cause

68 Had bicuspid aortic valve
62 Had atrial septal defect
13 Had ventricular septal defect
29 Had aortic regurgitation
24 Had mitral-valve prolapse
18 Had patent ductus arteriosus
9 Had pulmonary stenosis
2 Had trisomy

501 Underwent continued clinical surveillance

23 Died from any cause
3 Died from cardiac-related cause

15 Died from noncardiac cause
5 Had cancer
7 Had road traffic accident
2 Had drug overdose
1 Committed suicide

6 Died from cardiac-related cause
1 Had hypertrophic cardiomyopathy
1 Had idiopathic left ventricular hypertrophy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
1 Had sudden arrhythmic death syndrome

1 Committed suicide
2 Died from hypertrophic cardiomyopathy
1 Had idiopathic left ventricular hypertrophy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
1 Had sudden arrhythmic death syndrome

225 Had other disorder
68 Had bicuspid aortic valve
62 Had atrial septal defect
13 Had ventricular septal defect
29 Had aortic regurgitation
24 Had mitral-valve prolapse
18 Had patent ductus arteriosus
9 Had pulmonary stenosis
2 Had trisomy

23/ Died from cardiac-related cause
23/ Died from any cause

15 Died from noncardiac cause
5 Had cancer
7 Had road traffic accident
2 Had drug overdose
1 Committed suicide

6 Died from cardiac-related cause
1 Had hypertrophic cardiomyopathy
1 Had idiopathic left ventricular hypertrophy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
1 Had sudden arrhythmic death syndrome

1 Committed suicide
2 Died from hypertrophic cardiomyopathy
1 Had idiopathic left ventricular hypertrophy
2 Had arrhythmogenic right ventricular cardiomyopathy
1 Had dilated cardiomyopathy
1 Had sudden arrhythmic death syndrome
<table>
<thead>
<tr>
<th>Condition, Sex, and Age</th>
<th>Race</th>
<th>History and Examination</th>
<th>ECG Result</th>
<th>Echocardiography Result</th>
<th>LGE on Cardiac MRI</th>
<th>Exercise Test Result</th>
<th>Genetic Test Result</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Negative TWI (leads II, aVF, V2–V6)</td>
<td>LVWT 16 mm (asymmetric septal hypertrophy)</td>
<td>Yes</td>
<td>Normal</td>
<td>MYBPC3 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 15 yr White</td>
<td></td>
<td>Negative TWI (V2–V6), LAD, isoelectric ST segments</td>
<td>LVWT 15 mm (asymmetric septal hypertrophy)</td>
<td>No</td>
<td>Normal</td>
<td>MYBPC3 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Negative TWI (leads II, III, aVF), ST depression</td>
<td>Apical hypertrophy</td>
<td>No</td>
<td>Normal</td>
<td>MYH7 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 16 yr Black</td>
<td></td>
<td>Negative TWI (V1–V5), ST depression</td>
<td>LVWT 16 mm (asymmetric septal hypertrophy)</td>
<td>Yes</td>
<td>Normal</td>
<td>Negative</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 16 yr Mixed</td>
<td></td>
<td>Negative TWI (leads V4–V6), isoelectric ST segments</td>
<td>Apical hypertrophy</td>
<td>No</td>
<td>Normal</td>
<td>Negative</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>ARVC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Palpitations TWI (leads V1–V3)</td>
<td>Reduced LV systolic function; dilated and aneurysmal RV</td>
<td>Yes</td>
<td>Ventricular ectopy of LBBB morphology</td>
<td>Negative</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 17 yr White</td>
<td></td>
<td>Negative Normal</td>
<td>Aneurysmal RV with hypokinetic free wall</td>
<td>No</td>
<td>Ventricular ectopy of LBBB morphology</td>
<td>PKP2 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>DCM</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Dyspnea TWI (leads V1–V4), ST depression</td>
<td>LVEDD, 61 mm; EF, 45%</td>
<td>Yes</td>
<td>LV ejection fraction did not increase with exercise</td>
<td>Negative</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>LQTS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F, 16 yr White</td>
<td></td>
<td>Negative QTc, 510 msec</td>
<td>Normal</td>
<td>NA</td>
<td>QTc, &gt;500 msec</td>
<td>KCNQ1 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 15 yr White</td>
<td></td>
<td>Negative QTc, 503 msec</td>
<td>Normal</td>
<td>NA</td>
<td>QTc, &gt;500 msec</td>
<td>Negative</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Negative QTc, 490 msec</td>
<td>Normal</td>
<td>NA</td>
<td>Paradoxical increase in QTc during recovery</td>
<td>KCNQ1 mutation</td>
<td>Advised not to play</td>
<td></td>
</tr>
<tr>
<td>CAA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M, 16 yr White</td>
<td></td>
<td>Negative Normal</td>
<td>Left coronary artery arising from right sinus of Valsalva</td>
<td>NA</td>
<td>Positive for myocardial ischemia</td>
<td>NA</td>
<td>Underwent corrective surgery and returned to play</td>
<td></td>
</tr>
<tr>
<td>M, 15 yr White</td>
<td></td>
<td>Negative Normal</td>
<td>Right coronary artery arising from left sinus of Valsalva with adverse course</td>
<td>NA</td>
<td>Normal</td>
<td>NA</td>
<td>Underwent corrective surgery and returned to play</td>
<td></td>
</tr>
</tbody>
</table>
A total of 42 athletes (0.38%) were found to have cardiac disorders that are associated with sudden cardiac death (Fig. 1 and Table 1). Five (0.04%) had a diagnosis of hypertrophic cardiomyopathy, 2 (0.02%) of arrhythmogenic right ventricular cardiomyopathy,12 and 1 (0.01%) of dilated cardiomyopathy. Three athletes (0.03%) had a diagnosis of long-QT syndrome. Two athletes (0.02%) had a diagnosis of an anomalous origin of a coronary artery, and 3 (0.03%) were found to have a bicuspid aortic valve associated with either aortic-root enlargement of 50 mm or greater (1) or severe aortic regurgitation (2). A total of 26 (0.23%) athletes had the Wolff–Parkinson–White ECG pattern (Table S1 in the Supplementary Appendix).

Among the 42 athletes with cardiac disorders that are associated with sudden cardiac death, 2 (5%) had symptoms, 1 (2%) had an abnormality detected on cardiac examination, and 1 (2%) had both symptoms and an abnormality detected on examination (Table 2). Abnormal ECGs were obtained in 36 of these athletes (86%), and abnormal echocardiograms in 12 (29%).

There were 225 athletes (2%) with other cardiac disorders, including congenital septal and valvular abnormalities (Fig. 1). The diagnoses in all of these athletes were made with the use of echocardiography; 48 of the athletes (21%) had an abnormal ECG, and 74 (33%) had an abnormality detected on examination (Table 2).

**DETECTION OF CARDIAC DISEASE**

**INTERVENTIONS AND OUTCOMES**

Athletes with a diagnosis of cardiomyopathy (8) or long-QT syndrome (3) were advised against participation in competitive soccer. None of the athletes with cardiomyopathy were deemed to have a sufficiently high-risk profile to warrant a prophylactic implantable cardioverter–defibrillator.7,20,21 The 3 athletes with long-QT syndrome began treatment with beta-blockers. Both athletes with anomalous coronary-artery origins underwent corrective surgery and returned to play. The 3 athletes with bicuspid aortic valves underwent surgical intervention for aortic-root dilatation (1) or severe aortic regurgitation (2) and returned to play (Table 1). All 26 athletes with the Wolff–Parkinson–White ECG pattern underwent risk stratification, and 24 underwent ablation before returning to play. The remaining 2 athletes with the Wolff–Parkinson–White pattern were deemed
to have low-risk accessory conduction pathways that were not ablated, and they continued to compete (Table S1 in the Supplementary Appendix).

Of the 42 athletes who, during screening, received diagnoses of cardiac disorders that are associated with sudden cardiac death, 40 (95%) were alive at the end of the study period. Two athletes with a diagnosis of hypertrophic cardiomyopathy continued to compete despite medical advice and died subsequently during intensive exercise.

Among the 225 athletes with other cardiac disorders, 7 with an atrial septal defect and 1 with a ventricular septal defect had hemodynamic indications to warrant percutaneous closure before returning to play. The remaining 217 athletes (97%) had mild disease and were permitted to compete. We could not establish a cardiac diagnosis in 276 athletes (2%) who had abnormal T-wave inversion or borderline abnormal cardiac dimensions. All these athletes continued to compete but remained under clinical surveillance.

<table>
<thead>
<tr>
<th>Condition</th>
<th>No. of Athletes</th>
<th>No. of Athletes with Abnormal Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any cardiac condition</td>
<td>267</td>
<td>6 76 84 237</td>
</tr>
<tr>
<td>Condition associated with sudden cardiac death</td>
<td>42</td>
<td>3 2 36 12</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>5</td>
<td>0 0 5 5</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>2</td>
<td>1 0 1 2</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>1</td>
<td>1 0 1 1</td>
</tr>
<tr>
<td>Coronary-artery anomalies</td>
<td>2</td>
<td>0 0 0 2</td>
</tr>
<tr>
<td>Bicuspid aortic valve–associated disease*</td>
<td>3</td>
<td>1 2 0 3</td>
</tr>
<tr>
<td>Long-QT syndrome</td>
<td>3</td>
<td>0 0 3 0</td>
</tr>
<tr>
<td>Wolff–Parkinson–White ECG pattern</td>
<td>26</td>
<td>0 0 26 0</td>
</tr>
<tr>
<td>Other cardiac condition</td>
<td>225</td>
<td>3 74 48 225</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>68</td>
<td>1 32 15 68</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>62</td>
<td>1 6 26 62</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>29</td>
<td>0 16 2 29</td>
</tr>
<tr>
<td>Mitral-valve prolapse</td>
<td>24</td>
<td>0 12 3 24</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>18</td>
<td>0 1 1 18</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>13</td>
<td>0 3 1 13</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>9</td>
<td>1 4 0 9</td>
</tr>
<tr>
<td>Cor triatriatum</td>
<td>2</td>
<td>0 0 0 2</td>
</tr>
</tbody>
</table>

* Bicuspid aortic valve–associated disease includes bicuspid aortic valve with either aortic-root enlargement or severe aortic regurgitation.

**ALL-CAUSE MORTALITY**

Of the 92 professional clubs, 86 (93%) responded to the questionnaire regarding all-cause mortality. During a mean follow-up period of 10.6±8.3 years, there were 23 deaths. The causes of death included road traffic accidents (7 deaths [30%]), cancer (5 [22%]), drug overdose (2 [9%]), and suicide (1 [4%]). Cardiac disorders accounted for 8 deaths (35%), all of which were sudden and occurred during exercise (Fig. 2).

**SUDDEN CARDIAC DEATHS**

Autopsy data were available for all sudden cardiac deaths. Among the athletes who died from cardiac disorders, the mean time between screening and sudden death was 6.8 years (range, 0.1 to 13.2). Cardiomyopathies were the most common cause of death and accounted for 7 of 8 (88%) sudden cardiac deaths (Table 3). There were a total of 118,351 person-years of follow-up over the 20-year study period. The resulting overall incidence of sudden cardiac death among ado-
lescent soccer players was 1 per 14,794 person-years, or 6.8 per 100,000 athletes.

Of the 8 sudden cardiac deaths, 6 (75%) were in athletes who had normal findings during preliminary screening (Fig. 1 and Table 3). Variation in test interpretation could have accounted for the normal findings. Therefore, 50 deidentified ECGs and corresponding echocardiograms from the cohort were analyzed by two independent experts in inherited cardiac diseases who were unaware of the outcomes. The ECGs and echocardiograms were from 45 athletes whose results had been interpreted as normal, including the 6 decedents, and 5 athletes with abnormal results, including 1 athlete each with a diagnosis of arrhythmogenic right ventricular cardiomyopathy, dilated cardiomyopathy, hypertrophic cardiomyopathy, long QT interval, and the Wolff–Parkinson–White ECG pattern. Blind reading showed 100% agreement between the two reviewers for the 6 decedents with normal results (Table 3).

**COST OF DIAGNOSIS**

On the basis of the U.K. National Health Service tariffs, the cost of preliminary investigation with consultation (£160 [$213 in U.S. dollars]), ECG (£25 [$33]), and echocardiography (£72 [$96]) would amount to £257 ($342) per athlete, resulting in an initial cost of screening 11,168 athletes of £2,870,176 ($3,817,334). The cost of further investigating 830 athletes was £375,587 ($499,531), with a total estimated outlay of £3,245,763 ($4,316,865). The cost to detect serious cardiac disease associated with sudden cardiac death (42 athletes) was £77,280 ($102,782) per case, and the cost to identify any cardiac disorder (267 athletes) was £12,156 ($16,167) per case.

**DISCUSSION**

We report the outcomes of cardiovascular screening of adolescent soccer players, determined with the use of data from the FA in the United Kingdom. The prevalence of disorders associated with sudden cardiac death in young athletes was 0.38%, which is similar to that reported in other screening programs. Congenital septal and minor valvular disorders were detected in an additional 2% of the athletes, leading to an overall prevalence of 2.4% for all cardiac conditions.

In this cohort, electrical diseases accounted for 29 (69%) of the 42 cases of cardiac disorders that are associated with sudden cardiac death, whereas the primary cardiomyopathies accounted for only 8 (19%) of these cases. Anomalous coronary-artery origins accounted for 2 (5%) of the cases but were almost certainly underrepresented because of the limitations of echocardiography in detecting this type of disorder. The remaining 3 (7%) of the 42 cases were due to advanced aortic-valve disease. History, findings on physical examination, and ECG were abnormal in 7%, 5%, and 86%, respectively, of athletes with cardiac disorders associated with sudden cardiac death.

Hypertrophic cardiomyopathy was the most commonly detected cardiomyopathy, and all 5 athletes in whom this condition was detected had an abnormal ECG and echocardiogram. The prevalence of hypertrophic cardiomyopathy among these elite adolescent soccer players was 1 in 1861 athletes (including 1 case not detected by screening), which is considerably lower than that reported in the general population, raising the possibility that athletes with a more advanced phenotype at this age may have been selected out of competition because of reduced cardiorespiratory capacity. Age-related penetrance of hypertrophic cardiomyopathy is also an important consideration, since in many affected persons the disease is not expressed during adolescence.

The prevalence of arrhythmogenic right ventric-
Idiopathic left ventricular hypertrophy
Hypertrophic cardiomyopathy
Hypertrophic cardiomyopathy
Hypertrophic cardiomyopathy
Hypertrophic cardiomyopathy
Hypertrophic cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Hypertrophic cardiomyopathy
Sudden arrhythmic death syndrome

Table 3. Characteristics of Athletes with Sudden Cardiac Death.

<table>
<thead>
<tr>
<th>Athlete No.</th>
<th>Sex and Age</th>
<th>Race*</th>
<th>Years from Screening to Death</th>
<th>Diagnosis</th>
<th>Initial Screening Result</th>
<th>Blind Reading (Reviewer 1)</th>
<th>Blind Reading (Reviewer 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 16.8 yr</td>
<td>Black</td>
<td>0.1</td>
<td>Idiopathic left ventricular hypertrophy</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>2</td>
<td>M, 16.6 yr</td>
<td>Mixed</td>
<td>1.0</td>
<td>Hypertrophic cardiomyopathy</td>
<td>Abnormal ECG and echocardiogram</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>3</td>
<td>M, 16.6 yr</td>
<td>Black</td>
<td>3.3</td>
<td>Hypertrophic cardiomyopathy</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>4</td>
<td>M, 16.3 yr</td>
<td>Black</td>
<td>7.7</td>
<td>Dilated cardiomyopathy</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>5</td>
<td>M, 17.0 yr</td>
<td>White</td>
<td>7.9</td>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>6</td>
<td>M, 17.2 yr</td>
<td>White</td>
<td>9.7</td>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>7</td>
<td>M, 15.7 yr</td>
<td>White</td>
<td>11.5</td>
<td>Hypertrophic cardiomyopathy</td>
<td>Abnormal ECG and echocardiogram</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>8</td>
<td>M, 16.8 yr</td>
<td>White</td>
<td>13.2</td>
<td>Sudden arrhythmic death syndrome</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

* Race was reported by the athlete or the parent or guardian.
cardiovascular screening of a large cohort of adolescent soccer players in the United Kingdom. Diseases associated with sudden cardiac death, the majority of them electrical cardiac disorders, were identified in 0.38% of participants. The incidence of sudden cardiac death among these previously screened athletes was approximately 1 per 14,800 person-years, or 6.8 per 100,000 athletes. Most of these deaths were due to cardiomyopathies that were not detected on screening.

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APPENDIX

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OUTCOMES OF CARDIAC SCREENING IN SOCCER PLAYERS


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