

SCREENING FOR HYPERTROPHIC CARDIOMYOPATHY IN YOUNG ATHLETES

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ABSTRACT

Background For more than 20 years in Italy, young athletes have been screened before participating in competitive sports. We assessed whether this strategy results in the prevention of sudden death from hypertrophic cardiomyopathy, a common cardiovascular cause of death in young athletes.

Methods We prospectively studied sudden deaths among athletes and nonathletes (35 years of age or less) in the Veneto region of Italy from 1979 to 1996. The causes of sudden death in both populations were compared, and the pathological findings in the athletes were related to their clinical histories and electrocardiograms. Cardiovascular reasons for disqualification from participation in sports were investigated and follow-up was performed in a consecutive series of 33,735 young athletes who underwent preparticipation screening in Padua, Italy, during the same period.

Results Of 269 sudden deaths in young people, 49 occurred in competitive athletes (44 male and 5 female athletes; mean [\pm SD] age, 23 ± 7 years). The most common causes of sudden death in athletes were arrhythmogenic right ventricular cardiomyopathy (22.4 percent), coronary atherosclerosis (18.4 percent), and anomalous origin of a coronary artery (12.2 percent). Hypertrophic cardiomyopathy caused only 1 sudden death among the athletes (2.0 percent) but caused 16 sudden deaths in the nonathletes (7.3 percent). Hypertrophic cardiomyopathy was detected in 22 athletes (0.07 percent) at preparticipation screening and accounted for 3.5 percent of the cardiovascular reasons for disqualification. None of the disqualified athletes with hypertrophic cardiomyopathy died during a mean follow-up period of 8.2 ± 5 years.

Conclusions The results show that hypertrophic cardiomyopathy was an uncommon cause of death in these young competitive athletes and suggest that the identification and disqualification of affected athletes at screening before participation in competitive sports may have prevented sudden death. (N Engl J Med 1998;339:364-9.)

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MOST sudden deaths in athletes are due to cardiovascular disease.¹⁻¹⁰ Atherosclerotic coronary artery disease is the most common cause of sudden death in athletes over 35 years of age.^{2,4,5} Hypertrophic cardiomyopathy has been implicated as the principal cause of cardiac arrest in younger competitive athletes, accounting for about one third of fatal cases in the United States.^{3,7-9} The early identification of this

abnormality by screening of athletes before they participate in competitive sports might prevent sudden death, but the cost effectiveness of this strategy is still controversial.^{11,12} A national program for systematic preparticipation screening of all young competitive athletes has been in place in Italy for more than 20 years. The present study addressed the effects of this strategy in terms of the prevention of sudden death from hypertrophic cardiomyopathy in the Veneto region of Italy.

METHODS

Since 1971 Italian law has required that every athlete undergo an annual clinical evaluation to obtain approval to participate in competitive sports.¹³⁻¹⁵ We evaluated the effects of this community-based screening strategy through a prospective investigation of the causes of sudden death in both competitive athletes and nonathletes 35 years of age or younger in the Veneto region of Italy from 1979 to 1996 and an assessment of the cardiovascular reasons for disqualification in a large series of young competitive athletes who underwent preparticipation screening during this time in the Padua area. A competitive athlete was defined as "a participant in an organized sports program requiring regular training and competition."⁹

Sudden Death in Young Athletes and Nonathletes

The Veneto region of Italy covers an area of 18,368 km². During the study period, the population was stable and averaged 4,379,900, according to census data. There were 2,009,600 persons 35 years of age or less, defined as "young" in this paper. Nearly all residents were white, and the population was ethnically homogeneous. According to the Sports Medicine Data Base of the Veneto region, the rate of participation in competitive athletics among young people was 9.6 percent.

A prospective clinicopathological study of sudden death in young people has been carried out in the Veneto region since 1979.¹⁶ The sudden infant death syndrome was excluded from this investigation. The medical centers participating in this project (see the Appendix) serve 94.4 percent of the population. In all cases of sudden death in young people that occurred from 1979 to 1996, an autopsy was carried out by the local pathologist or medical examiner at one of these medical centers. Sudden death was defined as unexpected death occurring as a result of natural causes in which loss of all functions occurred instantaneously or within six hours of the onset of symptoms or collapse. After noncardiac causes of death were ruled out, all the hearts were fixed in formalin and forwarded to the Institute of Pathological Anatomy of the University of Padua for detailed morphologic assessment, according to a previously described protocol.¹⁶ The subject's clinical history and athletic activity and the circumstances surrounding the cardiac arrest were investigated in each case. Causes of sudden death in competitive athletes and nonathletes were compared to assess which conditions were significantly associated with

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cardiac arrest during athletic activity. The pathological findings in athletes were related to their clinical history and electrocardiographic findings in order to establish why the underlying disease had not been suspected at the preparticipation screening.

Preparticipation Cardiovascular Screening of Young Competitive Athletes

From 1979 to 1996, a consecutive series of 33,735 young athletes (28,539 male and 5196 female athletes; mean [±SD] age, 19±5 years) underwent 73,718 preparticipation cardiovascular evaluations at the Center for Sports Medicine in Padua. Screening for cardiac disease was part of a more comprehensive medical evaluation that included a general clinical history taking, physical examination, orthopedic examination, spirometry, and urinalysis. The initial cardiovascular protocol included family and personal history taking, physical examination with determination of blood pressure, basal 12-lead electrocardiography, and limited exercise testing (with the Montoye step test). Additional tests, such as echocardiography, 24-hour ambulatory Holter monitoring, or submaximal exercise testing, were requested for subjects who had positive findings (discussed below) at the initial evaluation.

The family history was considered positive if one or more close relatives had had a premature heart attack (i.e., at 50 years of age or less) or sudden death or if there was a family history of coronary artery disease, cardiomyopathy, Marfan's syndrome, the long-QT syndrome, severe arrhythmias, or other disabling cardiovascular diseases.

The personal history was considered positive if the subject had had chest pain or discomfort, syncope or near-syncope, or irregular heartbeat or palpitations on exertion, or if the subject had had shortness of breath or fatigue on exertion that was out of proportion to the degree of physical effort.

Positive physical findings included musculoskeletal and ocular features suggestive of Marfan's syndrome, diminished and delayed femoral-artery pulses, mid- or end-systolic clicks, a second heart sound that was single or widely split and fixed with respiration, marked heart murmurs (any diastolic and systolic grade 2/6 or higher), irregular heart rhythm, and brachial blood pressure greater than 145/90 mm Hg on more than one reading. The electrocardiogram was considered positive according to accepted criteria^{11,17-20} if one or more of the findings listed in Table 1 were present.

Hypertrophic cardiomyopathy was suspected at the initial screening in young people with a suggestive personal or family history, positive physical findings, or positive electrocardiographic findings. The definitive diagnosis was subsequently based on the echocardiographic demonstration of a hypertrophic, nondilated left ventricle (wall thickness, ≥13 mm), in the absence of another cardiac or systemic disease that could cause hypertrophy of the magnitude present in that person.²¹

The distinction between hypertrophic cardiomyopathy and athlete's heart was based on echocardiographic and clinical features, such as the magnitude and distribution of thickening of the left ventricular wall, the dimension of the left ventricular cavity, the presence or absence of electrocardiographic abnormalities, the type of sport played, and the results of deconditioning.²²⁻²⁷ The criteria for hypertrophic cardiomyopathy included a high degree of left ventricular hypertrophy (wall thickness, >16 mm) with an unusual distribution (heterogeneous, asymmetric, or sparing the anterior septum); a left ventricular cavity of normal size (<45 mm); the presence of striking electrocardiographic abnormalities (a marked increase in voltages, prominent Q waves, and deep, negative T waves); training in athletic disciplines other than endurance sports, such as rowing, canoeing, cycling, and swimming; and the persistence of hypertrophy after six months of deconditioning.²²⁻²⁷

Subjects were disqualified from competitive athletic activity when clinically relevant cardiovascular abnormalities were recognized. The Italian guidelines for assessing athletic risk are similar to those of the 16th and 26th Bethesda Conferences,^{28,29} although the Italian criteria for sports eligibility are more restric-

TABLE 1. CRITERIA FOR A POSITIVE 12-LEAD ELECTROCARDIOGRAM.*

P wave
Left atrial enlargement: negative portion of the P wave in lead V ₁ ≥0.1 mV in depth and ≥0.04 sec in duration
Right atrial enlargement: peaked P wave in leads II and III or V ₁ ≥0.25 mV in amplitude
QRS complex
Frontal-plane axis deviation: right ≥+120 degrees or left -30 degrees to -90 degrees
Increased voltage: amplitude of R or S wave in a standard lead ≥2 mV, S wave in lead V ₁ or V ₂ ≥3 mV, or R wave in lead V ₅ or V ₆ ≥3 mV
Abnormal Q waves ≥0.04 sec in duration or ≥25 percent of the height of the ensuing R wave, or QS pattern in two or more leads
Right or left bundle-branch block with QRS duration ≥0.12 sec
R or R' wave in lead V ₁ ≥0.5 mV in amplitude and R:S ratio ≥1
ST segment, T waves, and QT interval
ST-segment depression or T-wave flattening or inversion in two or more leads
Prolongation of QT interval corrected for the heart rate >0.44 sec
Rhythm and conduction abnormalities
Premature ventricular beats or more severe ventricular arrhythmia
Supraventricular tachycardia, atrial flutter, or atrial fibrillation
Short PR interval (<0.12 sec) with or without delta wave
Sinus bradycardia with resting heart rate ≤40 beats per minute and increasing to <100 beats per minute during limited exercise testing
First-degree (PR ≥0.21 sec, not shortening with hyperventilation or limited exercise testing), second-degree, or third-degree atrioventricular block

*The criteria are from Friedman,¹⁷ Romhilt and Estes,¹⁸ Morris et al.,¹⁹ and Savage et al.²⁰

ive.³⁰ Follow-up data were obtained from office visits, telephone interviews, or written questionnaires and were available for all subjects who were disqualified because of cardiovascular problems.

Causes of Death

At autopsy, hypertrophic cardiomyopathy was diagnosed when the subject had macroscopic cardiac hypertrophy, defined according to population-based criteria for normal cardiac weight; either asymmetric or symmetric thickening of the septum and free wall, in the absence of other cardiac causes of hypertrophy, such as hypertensive, valvular, or congenital heart disease; and microscopic evidence of myocardial disarray involving a substantial portion of the interventricular septum.³¹⁻³⁴ Arrhythmogenic right ventricular cardiomyopathy was diagnosed when there was gross or histologic evidence of regional or diffuse full-thickness replacement of the myocardium of the right ventricular free wall by fat and fibrous tissue, in the absence of other known cardiac or noncardiac causes of death.¹⁶ Obstructive atherosclerotic coronary artery disease was diagnosed when one or more major epicardial coronary arteries showed cross-sectional narrowing of 70 percent or more.³⁵ Myocarditis was diagnosed according to the Dallas criteria,³⁶ on the basis of the presence of an inflammatory infiltrate in the myocardium with degeneration or necrosis of adjacent myocytes. Mitral-valve prolapse was diagnosed when there was increased thickness, floppiness, and redundancy of the leaflets, intercordal hooding, and billowing of the leaflet toward the left atrium.³⁷ Sudden death was classified as unexplained if there was no macroscopically or microscopically apparent structural heart disease or other identifiable cause of death.

Statistical Analysis

Continuous variables were expressed as means ±SD. The chi-square or Fisher's exact test was used to assess the significance of

differences between subgroups. The relative risk of sudden death (the ratio of the risk of sudden death among competitive athletes to the risk among nonathletes) and 95 percent confidence intervals were calculated with the Stata 5.0 statistical package (Stata, College Station, Tex.). A two-tailed P value of less than 0.05 was considered to indicate statistical significance.

RESULTS

Sudden Death in Young Competitive Athletes

From 1979 to 1996, 269 sudden deaths occurred in people 35 years of age or less in the Veneto region (0.8 per 100,000 persons per year): 49 among competitive athletes (1.6 per 100,000 per year) and 220 among nonathletes (0.75 per 100,000 per year). The estimated relative risk of sudden death among athletes as compared with nonathletes was 2.1 (95 percent confidence interval, 1.5 to 2.9; $P < 0.001$).

The 49 athletes (44 male and 5 female athletes) ranged in age from 11 to 35 years (mean, 23 ± 7 years) and had participated in a variety of sports: soccer (22 subjects); basketball (5); swimming (4); cycling (3); rugby, running, gymnastics, tennis, skiing, judo, and volleyball (2 subjects each); and weight lifting (1). Fourteen athletes had previously had palpitations on exertion, syncopal episodes, or both; 16 had had recorded electrocardiographic abnormalities or rhythm and conduction disturbances. In 40 cases, sudden death occurred during sports activity (35 cases) or immediately afterward (5 cases).

The most common causes of sudden death in the athletes were arrhythmogenic right ventricular cardiomyopathy (11 subjects, 22.4 percent), atherosclerotic coronary artery disease (9 subjects, 18.4 percent), and anomalous origin of a coronary artery from the contralateral aortic sinus (6 subjects, 12.2 percent) (Table 2). Arrhythmogenic right ventricular cardiomyopathy ($P = 0.008$) and anomalous origin of a coronary artery ($P < 0.001$) were the only cardiovascular conditions that were associated with sudden death significantly more often in athletes than in nonathletes. Hypertrophic cardiomyopathy caused only 1 sudden death among the athletes (2.0 percent), whereas it was the cause of 16 sudden deaths in the nonathletic population (7.3 percent). Three of the 16 nonathletes with hypertrophic cardiomyopathy died suddenly during mild exertion unrelated to participation in sports. None of the nonathletes who died suddenly from hypertrophic cardiomyopathy had been screened before death and excluded.

As summarized in Table 3, clinical findings indicative of cardiovascular disease had been detected at preparticipation screening in 82 percent of the athletes who died of arrhythmogenic right ventricular cardiomyopathy, as compared with 22 percent of those who died of atherosclerotic coronary artery disease ($P = 0.02$) and 25 percent of those who died of congenital anomalies of the coronary arteries ($P = 0.02$).

TABLE 2. CAUSES OF SUDDEN DEATH IN ATHLETES AND NONATHLETES 35 YEARS OF AGE OR LESS IN THE VENETO REGION OF ITALY, 1979 TO 1996.

CAUSE	ATHLETES	NONATHLETES	TOTAL
	(N=49)	(N=220)	(N=269)
	number (percent)		
Arrhythmogenic right ventricular cardiomyopathy	11 (22.4)	18 (8.2)*	29 (10.8)
Atherosclerotic coronary artery disease	9 (18.4)	36 (16.4)	45 (16.7)
Anomalous origin of coronary artery	6 (12.2)	1 (0.5)†	7 (2.6)
Disease of conduction system	4 (8.2)	20 (9.1)	24 (8.9)
Mitral-valve prolapse	5 (10.2)	21 (9.5)	26 (9.7)
Hypertrophic cardiomyopathy	1 (2.0)	16 (7.3)	17 (6.3)
Myocarditis	3 (6.1)	19 (8.6)	22 (8.2)
Myocardial bridge	2 (4.1)	5 (2.3)	7 (2.6)
Pulmonary thromboembolism	1 (2.0)	3 (1.4)	4 (1.5)
Dissecting aortic aneurysm	1 (2.0)	11 (5.0)	12 (4.5)
Dilated cardiomyopathy	1 (2.0)	9 (4.1)	10 (3.7)
Other	5 (10.2)	61 (27.7)	66 (24.5)

* $P = 0.008$ for the comparison with the athletes.

† $P < 0.001$ for the comparison with the athletes.

Disqualifying Cardiovascular Conditions at Preparticipation Cardiovascular Screening

Of the 33,735 young athletes who were screened at the Center for Sports Medicine in Padua, 1058 were disqualified from participation in competitive sports for medical reasons because of the following types of conditions: cardiovascular in 621 (58.7 percent), orthopedic in 134 (12.7 percent), ophthalmic in 130 (12.3 percent), neurologic in 46 (4.3 percent), respiratory in 37 (3.5 percent), nephrologic or urinary in 34 (3.2 percent), otorhinolaryngologic in 22 (2.1 percent), endocrinologic in 22 (2.1 percent), and other in 12 (1.1 percent). The most frequent cardiovascular conditions causing disqualification were rhythm and conduction abnormalities (38.3 percent), systemic hypertension (27.1 percent), and valvular diseases, including mitral-valve prolapse complicated by clinically significant ventricular arrhythmias, mitral-valve regurgitation, or both (21.4 percent) (Table 4). Among the rhythm and conduction abnormalities, ventricular arrhythmias accounted for 19.5 percent of the total number of disqualifications, supraventricular tachycardia, atrial flutter, or fibrillation for 7.6 percent, the Wolff-Parkinson-White syndrome for 7.1 percent, complete left bundle-branch block or right bundle-branch block and left-axis deviation for 1.9 percent, second-degree atrioventricular block for 1.6 percent, and the long-QT syndrome for 0.6 percent. Hypertrophic cardiomyopathy was identified in 22 young athletes (0.07 percent of those screened) and accounted for 3.5

TABLE 3. CLINICAL FINDINGS AT PREPARTICIPATION SCREENING OF ATHLETES WHO DIED SUDDENLY OF ONE OF THE LEADING THREE CARDIOVASCULAR CAUSES.

FINDING	CAUSE OF DEATH		
	ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (N=11)	ATHEROSCLEROTIC CORONARY ARTERY DISEASE (N=9)	CORONARY ARTERY ANOMALY* (N=8)
	number (percent)		
Family history of sudden death from heart disease	2	0	0
Palpitations on exertion	6	1	0
Syncope	5	1	1
Chest pain	0	0	0
ST-segment or T-wave abnormalities	9	0	1
Ventricular arrhythmias	6	0	2
One or more of the above	9 (82)	2 (22)†	2 (25)†

*This group includes both patients with anomalous origin of a coronary artery and those with myocardial bridges.

†P=0.02 for the comparison with those with arrhythmogenic right ventricular cardiomyopathy.

percent of the cardiovascular causes of disqualification. Less frequent cardiovascular reasons for disqualification included congenital, rheumatic, and ischemic heart disease, as well as pericarditis.

Hypertrophic Cardiomyopathy

Of the 33,735 athletes initially screened, 3016 (8.9 percent) were referred for echocardiographic evaluation because of the family history, abnormal physical findings, or electrocardiographic abnormalities. Twenty-two (20 male and 2 female athletes; mean age, 20±4 years; range, 16 to 28) had definite evidence of hypertrophic cardiomyopathy on echocardiographic examination. These 22 athletes were referred for echocardiographic study because they had one or more of the following findings: a positive family history in 3; cardiac murmur in 2; one or more electrocardiographic changes in 16 (73 percent), consisting of repolarization abnormalities in 14, elevated voltages in 11, and abnormal Q waves in 5; and premature ventricular beats in 5. The maximal thickness of the left ventricular wall was 19±3 mm (range, 16 to 24), and the end-diastolic diameter of the left ventricular cavity was 43±2 mm (range, 39 to 46). No significant differences in the degree of left ventricular hypertrophy were seen before and after deconditioning.

Follow-up

Four of the 621 athletes who were disqualified for cardiovascular causes died during a mean follow-up period of 8.2±5 years (range, 1.3 to 16.8). One athlete with mild mitral-valve prolapse complicated by complex ventricular arrhythmias died suddenly of natural causes; the other three athletes, who had atri-

TABLE 4. CARDIOVASCULAR CONDITIONS CAUSING DISQUALIFICATION FROM COMPETITIVE SPORTS IN 621 ATHLETES IN PADUA, 1979 TO 1996.

CONDITION	No. (%)
Rhythm and conduction abnormalities	238 (38.3)
Systemic hypertension	168 (27.1)
Valvular diseases (including mitral-valve prolapse)	133 (21.4)
Hypertrophic cardiomyopathy	22 (3.5)
Others	60 (9.7)

al septal defect, ventricular septal defect, and a bicuspid aortic valve with regurgitation, died of nonnatural causes (drug abuse, a car accident, and suicide, respectively).

None of the 22 athletes who were disqualified because they had hypertrophic cardiomyopathy died during follow-up. Two patients with paroxysmal atrial fibrillation were treated, one with a beta-blocker and the other with amiodarone. In both cases, the treatment was effective in restoring and maintaining sinus rhythm. Another asymptomatic patient with a family history of sudden death was treated with amiodarone after 24-hour Holter monitoring documented the presence of nonsustained ventricular tachycardia.

DISCUSSION

A structural cardiac abnormality was found at autopsy in most of the cases of sudden death in young competitive athletes that have been previously re-

ported.¹⁻¹⁰ Several cardiovascular disorders were implicated, including hypertrophic cardiomyopathy,^{3,7-9} congenital anomaly of a coronary artery,^{6,9} Marfan's syndrome,^{3,8,9} atherosclerotic coronary artery disease,^{6,8} and arrhythmogenic right ventricular cardiomyopathy.^{6,16} In the present study, sudden death in young competitive athletes was related to the same underlying disorders, but the prevalence of each cause of death differed substantially from that previously reported. Studies from the United States have consistently found that hypertrophic cardiomyopathy was the most common cause of cardiac arrest in young competitive athletes (up to 30 percent).^{3,7-9} In this Italian study, hypertrophic cardiomyopathy caused only one death among the athletes but caused sudden death in the nonathletic young population with a frequency similar to that found in the United States.⁷ Moreover, we found a high prevalence of arrhythmogenic right ventricular cardiomyopathy and premature atherosclerotic coronary artery disease among both groups.

The low prevalence of hypertrophic cardiomyopathy among young competitive athletes who died suddenly was most likely the result of the systematic preparticipation screening that has been in practice in Italy for more than 20 years.¹³⁻¹⁵ Indeed, two of our main findings provide indirect evidence that screening reduces sudden death from hypertrophic cardiomyopathy. First, the prevalence of hypertrophic cardiomyopathy among young nonathletes who died suddenly was similar in our study (7.3 percent) and in the study of Burke et al.⁷ in the United States (3 percent). Among young athletes who died suddenly, however, the prevalence of hypertrophic cardiomyopathy was very different in the two studies (2 percent vs. 24 percent). This pattern suggests a selective reduction in sudden death from hypertrophic cardiomyopathy among the competitive athletes who underwent systematic preparticipation screening.

Second, screening of 33,735 young competitive athletes in the Padua area identified and disqualified 22 athletes with hypertrophic cardiomyopathy, thus protecting them from the risk entailed by athletic activity. In the United States, hypertrophic cardiomyopathy is present in approximately 0.2 percent of young adults who are screened by echocardiography. The prevalence is higher among blacks (0.24 percent) than among whites (0.10 percent).³⁸ The prevalence of 0.07 percent among the white athletes in the Veneto region is thus similar to that reported among young white persons in the United States, although the screening in Italy was done mainly by electrocardiography rather than by echocardiography. This shows that the screening program based largely on the electrocardiogram was an efficient means of detecting hypertrophic cardiomyopathy in the population of young athletes.

None of the athletes who were disqualified from

participation in sports because of hypertrophic cardiomyopathy died during the follow-up. This finding further supports the conclusion that systematic preparticipation screening might reduce mortality through the detection of hypertrophic cardiomyopathy in athletes and their disqualification from competitive sports.

Although echocardiography is the main diagnostic tool for the clinical recognition of hypertrophic cardiomyopathy, it is very expensive and impractical for large-scale screening of athletes.^{11,12} Twelve-lead electrocardiography has been proposed as a more cost-effective alternative. In the present study, a combination of electrocardiography, clinical history taking, and physical examination successfully selected athletes as candidates for echocardiography. Of the 33,735 athletes initially screened, 3016 (8.9 percent) were referred for echocardiographic evaluation, and 22 were eventually found to have evidence of hypertrophic cardiomyopathy and were disqualified from sports. Thus, more than 90 percent of the screened population did not undergo echocardiography, resulting in a considerable cost savings.

The present study confirms that the identification at preparticipation screening of young athletes with coronary artery disease is limited by the scarcity of warning signs and the low sensitivity of both baseline and exercise electrocardiography in detecting signs of myocardial ischemia.^{39,40} In contrast, athletes who died of arrhythmogenic right ventricular cardiomyopathy often had a history of syncopal episodes, electrocardiographic abnormalities consisting of inverted T waves in the right precordial leads, and ventricular arrhythmias with a left bundle-branch block pattern. Nonetheless, these athletes were not identified at preparticipation screening, because this disease is not widely recognized as a cause of sudden death during sports activity. The results of the present study suggest that the finding of even isolated premature ventricular beats at cardiovascular screening, with morphologic features of left bundle-branch block associated with right precordial T-wave abnormalities on the electrocardiogram, with or without a history of syncopal attacks, should suggest the possibility of an underlying arrhythmogenic right ventricular cardiomyopathy and lead to further testing, such as echocardiography.

APPENDIX

The following medical centers participated in the Research Project on Juvenile Sudden Death in the Veneto region of Italy: the Center for Sports Medicine, National Health Service, Padua; the Institutes of Pathological Anatomy and Forensic Medicine, University of Padua; and the departments of pathological anatomy of the following institutions: Civil Hospital, Vicenza; Civil Hospital, Treviso; Civil Hospital, Castelfranco Veneto; Civil Hospital, Dolo; University of Verona; Civil Hospital, Conegliano; Civil Hospital, Belluno; Civil Hospital, Camposampiero; Civil Hospital, Chioggia; Civil Hospital, Feltre; Civil Hospital, Piove di Sacco; Civil Hospital, Thiene; Civil Hospital, Cittadella; Civil Hospital, Este; Civil Hospital, Mestre; Civil Hospital, Montebelluna; Civil Hospital, Mirano; Civil Hospital, Arzignano; Civil Hospital, Battaglia Terme; Civil Hospital, Noale;

Civil Hospital, Valdagno; Civil Hospital, Venice; Civil Hospital, Bassano; Civil Hospital, San Donà di Piave; and Civil Hospital, Asolo.

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