



ESC Report

Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol

Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology

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The 1996 American Heart Association consensus panel recommendations stated that pre-participation cardiovascular screening for young competitive athletes is justifiable and compelling on ethical, legal, and medical grounds. The present article represents the consensus statement of the Study Group on Sports Cardiology of the Working Group on Cardiac Rehabilitation and Exercise Physiology and the Working Group on Myocardial and Pericardial diseases of the European Society of Cardiology, which comprises cardiovascular specialists and other physicians from different European countries with extensive clinical experience with young competitive athletes, as well as with pathological substrates of sudden death. The document takes note of the 25-year Italian experience on systematic pre-participation screening of competitive athletes and focuses on relevant issues, mostly regarding the relative risk, causes, and prevalence of sudden death in athletes; the efficacy, feasibility, and cost-effectiveness of population-based pre-participation cardiovascular screening; the key role of 12-lead ECG for identification of cardiovascular diseases such as cardiomyopathies and channelopathies at risk of sudden death during sports; and the potential of preventing fatal events. The main purpose of the consensus document is to reinforce the principle of the need for pre-participation medical clearance of all young athletes involved in organized sports programmes, on the basis of (i) the proven efficacy of systematic screening by 12-lead ECG (in addition to history and physical examination) to identify hypertrophic cardiomyopathy—the leading cause of sports-related sudden death—and to prevent athletic field fatalities; (ii) the potential screening ability in detecting other lethal cardiovascular diseases presenting with ECG abnormalities. The consensus document recommends the implementation of a common European screening protocol essentially based on 12-lead ECG.

Introduction

Considerable interest has been raised regarding the role of pre-participation screening for early identification of those cardiovascular diseases which are responsible for athletic field deaths and for disqualification of athletes at risk, with the expectation that such a strategy may eventually prevent sudden death.¹⁻³ At present, great heterogeneity does exist regarding the medical supervision of competitive athletes, with only a few nations requiring pre-participation medical clearance before participation in official athletic events.⁴⁻⁸

The present article represents the consensus of the Study Group on Sports Cardiology of the Working group on Cardiac Rehabilitation and Exercise Physiology and of the Working Group on Myocardial and Pericardial Diseases of the European Society of Cardiology, which comprises cardiovascular specialists and other physicians from different European countries with extensive clinical experience with young competitive athletes. The document takes note of the 25-year Italian experience of systematic pre-participation screening of competitive athletes³⁻⁵ and focuses on relevant issues, mostly regarding the relative risk, causes, and prevalence of sudden death in athletes, the efficacy, feasibility, and cost-effectiveness of population-based screening for detection of cardiovascular diseases at risk of sudden death during sports, and the potential for preventing fatal events. The main purposes of the consensus document are (i) to reinforce the principle, previously supported by the 1996 American Heart Association consensus panel,² of the need for pre-participation medical clearance of all

young competitive athletes involved in organized sports programmes, on the basis of the proven efficacy of systematic screening by 12-lead ECG (in addition to history and physical examination) to identify hypertrophic cardiomyopathy (HCM) and to prevent sudden death;^{3,9} and (ii) to recommend a common European screening protocol essentially based on 12-lead ECG.

Risk of sudden death during sports performance

Little is known about the risk of sudden death associated with exercise in young competitive athletes, and whether the benefits of sports activity outweigh the hazards of exercise-related fatal events is a clinical dilemma.¹⁰⁻¹⁵ For the purpose of this consensus document, we consider young competitive athletic individuals who are aged 35 years or less and are engaged in a regular fashion in exercise training as well as participating in official athletic competitions.¹⁵⁻¹⁷ Competition is intended as an organized team or individual sport event, placing high premium on athletic excellence and achievement. Characteristics of competitive athletes are their strong inclination to extend themselves physically to their limits and to improve performance.²

Recently, Corrado *et al.*¹⁷ assessed the incidence of sudden death in the athletic and non-athletic young population (12-35 years old) of the Veneto Region of Italy and showed that competitive sports activity enhances by 2.5-fold the risk of sudden death in adolescents and young adults. In this study, young competitive athletes who died suddenly were affected by silent cardiovascular diseases, predominantly consisting of

cardiomyopathies, premature coronary artery disease, and congenital coronary anomalies. Therefore, sports activity was not *per se* a cause of the increased mortality; rather, it acted as a trigger of cardiac arrest upon those underlying cardiovascular diseases predisposing to life-threatening ventricular arrhythmias. Thus, it seems ethically and clinically justifiable that every effort should be made to recognize in good time such diseases that the athlete is at risk, with the perspective that disqualification of affected individuals makes the prevention of athletic field death possible.

Epidemiology of sudden death in young competitive athletes

Incidence

The assessment of the precise frequency with which sudden death occurs in young athletes during organized competitive sports is hampered by the retrospective nature of most analyses. In the past, studies from the United States probably resulted in underestimation of the prevalence of sports-related sudden deaths since they relied on reports from individual schools and institutions, or on media accounts.^{14,15} Recently, a prospective population-based study in the Veneto Region of Italy reported an incidence of sudden death of 2.3 (2.62 in males and 1.07 in females) per 100 000 athletes per year from all causes, and of 2.1 per 100 000 athletes per year from cardiovascular diseases.¹⁷ Reasons for the higher mortality rates found in this Italian investigation, compared with those reported in the USA, include the different underlying pathological substrates which, in part, reflect differences in ethnic and genetic factors as well as the higher mean age and the participation at a higher level of intensity of Italian competitive athletes compared with US high school and college participants.

Gender predilection

Sudden death in athletes shows a clear gender predilection with striking male predominance (male to female ratio up to 10:1).¹⁰⁻¹⁷ The predominance of fatal events in male athletes has been related to the higher participation rate of male compared with female athletes in competitive sports, as well as the more intensive training load and level of athletic achievement of males. More recently, male gender was reported to be, in itself, a risk factor for sports-related sudden death, most likely as a consequence of the greater prevalence and/or phenotypic expression in young males of cardiac diseases at risk of arrhythmic cardiac arrest, such as cardiomyopathies^{18,19} and premature coronary artery disease.²⁰

Causes

Unlike athletes over 35 years of age, in whom atherosclerotic coronary artery disease is by far the most common cause of fatal events,^{2,3} in younger competitive athletes a broad spectrum of cardiovascular causes of sudden death (including congenital and inherited disorders) has been reported.^{3,10-17} HCM has been

implicated as the principal cause of sport-related cardiac arrest, accounting for more than one-third of sudden deaths in the USA;^{10,13-16,21} other causes include anomalous origin of coronary artery from the wrong coronary sinus, arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), myocarditis, premature coronary atherosclerosis, conduction system abnormalities, and Marfan syndrome (Table 1).

Screening protocol in the United States and Italy

Pre-participation cardiovascular screening has traditionally been performed in the USA by means of history (personal and family) and physical examination without 12-lead ECG or other testing, which are requested largely at the discretion of the examining physician.^{1,2,6-8} This screening method has been recommended by the Sudden Death and Congenital Defects Committee of the American Heart Association on the assumption that 12-lead ECG is not cost-effective for screening a large population of young athletes due to its low specificity.² Such a screening strategy, however, has a limited power to detect potentially lethal cardiovascular abnormalities in young athletes. One retrospective analysis on 134 high school and collegiate athletes who died suddenly showed that cardiovascular abnormalities were suspected by standard history and physical examination screening in only 3% of the examined athletes and, eventually, less than 1% received an accurate diagnosis.¹⁵

The addition of 12-lead ECG has the potential to enhance the sensitivity of the screening process for detection of cardiovascular diseases with risk of sudden death. In fact, ECG is abnormal in up to 95% of patients with HCM,²² which is the leading cause of sudden death in the athlete. Likewise, ECG abnormalities have also been documented in the majority of athletes who died from ARVC/D.^{3,11,12}

For more than 25 years, a systematic pre-participation screening predominantly based on 12-lead ECG in addition to history and physical examination, has been in practice in Italy.³⁻⁵ Italian law mandates that every subject engaged in competitive sports activity must

Table 1 Cardiovascular causes of sudden death in young competitive athletes in the United States

Most common
Hypertrophic cardiomyopathy
Congenital coronary artery anomaly
Less common
Myocarditis
Aortic rupture (Marfan syndrome)
Mitral valve prolapse
Uncommon
Arrhythmogenic RV cardiomyopathy
Atherosclerotic coronary artery disease
Conduction system abnormalities
Aortic valve stenosis

undergo a clinical evaluation and obtain eligibility before entering.⁴ The Italian pre-participation screening involves nearly 6 million athletes of all ages annually, representing about 10% of the overall Italian population.⁵

Corrado *et al.*³ reported a 17-year experience from the Center for Sports Medicine of Padova. During the interval 1979–1996, a consecutive series of 33 735 young athletes (<35 years) underwent pre-participation cardiovascular evaluation. Of these, 1058 were disqualified for medical reasons: 621 (1.8%) of them because of the recognition of clinically relevant cardiovascular abnormalities. The most frequent disqualifying conditions consisted of rhythm and conduction abnormalities (38.3%); hypertension (27%); valvular diseases including mitral valve prolapse complicated by significant ventricular arrhythmias, or mitral valve regurgitation, or both (21.4%); and HCM (3.6%). Less frequent reasons for non-eligibility included dilated cardiomyopathy, congenital and rheumatic heart diseases, and pericarditis.

Identification of young competitive athletes with HCM

HCM has been reported to be the leading cause of sudden death in young competitive athletes, accounting for up to 40% of athletic field deaths in the USA.^{2,10,13–16,21,22}

Although echocardiography is the main diagnostic tool for the recognition of HCM, it is expensive and impractical for screening large populations.² Twelve-lead ECG has been proposed as an alternative, cost-effective method for population-based screening. The Italian experience demonstrates that a protocol utilizing ECG in addition to history and physical examination successfully identifies HCM in the general population of young competitive athletes. Among 33 735 athletes undergoing pre-participation screening at the Center for Sport Medicine of Padova, 22 (0.07%) showed definitive evidence, both clinical and echocardiographic, of HCM.³ An absolute value of screening sensitivity for HCM in young competitive athletes cannot be derived from this study, because systematic echocardiographic data were not available. However, this 0.07% prevalence of HCM in the white athletic population of the Veneto Region of Italy, which was evaluated by history, physical examination, and ECG, is similar to that of 0.1% reported in young white individuals in the USA, assessed by echocardiography.²³ This indicates that Italian screening, essentially based on 12-lead ECG, may be as sensitive as screening by echocardiography in detecting HCM in the young athletic population.

Of the 33 735 athletes screened in Padova, 3016 (8.9%) were referred for echocardiographic evaluation and 22 ultimately showed definitive evidence of HCM.³ Therefore, the percentage of young athletes with a positive history, abnormal physical findings, or electrocardiographic abnormalities requiring further evaluation by echocardiography (or other testing) was 8.9%, with an estimated screening specificity of more than 90%.

Echocardiographic study in addition to the basal protocol does not seem to significantly improve efficacy of

the pre-participation screening in identifying HCM. Pelliccia *et al.*⁹ did not identify any HCM by routine echocardiographic examination in 4450 elite athletes previously cleared by ECG at pre-participation evaluation.

The Italian screening modality has proved more sensitive than the limited US protocol. Among the 22 Italian athletes (120 males and two females, aged 20 ± 4 years) who were identified and disqualified due to HCM,³ 18 (82%) had shown ECG changes at pre-participation evaluation, which included repolarization abnormalities in 14 (87.5%), elevated QRS precordial voltages in 11 (69%), and abnormal Q waves in five (31%). Moreover, premature ventricular beats were recorded in five (23%). It is noteworthy that only five of these 22 athletes (23%) had a positive family history, a cardiac murmur, or both, at pre-participation evaluation. These findings indicate that the Italian screening modality including 12-lead ECG has 77% greater power for detecting HCM and is expected to result in a corresponding additional number of lives saved, compared with the protocol limited to history and physical examination recommended by the American Heart Association. According to such a different diagnostic power, we recently estimated a three-fold greater cost-effectiveness of the Italian vs. US screening strategy for identification and prevention of sudden death of athletes with HCM.^{24,25}

Prevention of sport-related sudden death due to HCM

The Italian experience also shows that systematic pre-participation screening has efficacy in preventing sudden death in young athletes with HCM.³ None of the 22 young athletes with HCM who were identified in the Padova country area by pre-participation athletic screening and disqualified from training and competitions died during an average 8-year follow-up period.

The impact of screening on survival is confirmed by the results of systematic monitoring of sudden death in young people (<35 years of age), which has been carried out in the Veneto Region of Italy.³ From January 1979 to December 1996, 269 consecutive cases of juvenile sudden death were prospectively studied. Forty-nine (18%) young sudden death victims were competitive athletes (44 males, five females, aged 23.1 years), who had undergone pre-participation cardiovascular screening. The most common causes of death were ARVC/D (11 cases, 22.4%), atherosclerotic coronary artery disease (nine cases, 18.5%), and congenital anomalies of the coronary arteries (eight cases, 16.3%). HCM was responsible for only one sudden death in athletes (2%), whereas it accounted for 7.3% of sudden deaths in the general non-athletic young population (Table 2).

Comparison between these Italian findings and those reported by Burke in the USA¹³ shows a similar prevalence of HCM in non-sport-related sudden cardiac death, but a strong difference (2 vs. 24%) in sports-related cardiovascular events. This suggests a selective reduction of sports-related sudden death from HCM due

Table 2 Causes of sudden deaths in athletes and non-athletes (aged ≤ 35 years) in the Veneto region of Italy from 1979 to 1996

	Athletes (n = 49) n (%)	Non-athletes (n = 220) n (%)	Total (n = 269) n(%)
Arrhythmogenic RV cardiomyopathy	11 (22.4)	18 (8.2)*	29 (10.8)
Atherosclerotic coronary artery disease	9 (18.5)	36 (16.4)	45 (16.7)
Anomalous origin of coronary artery	6 (12.2)	1 (0.4)**	7 (2.6)
Conduction system pathology	4 (8.2)	20 (9)	24 (8.9)
Mitral valve prolapse	5 (10.2)	21 (9.5)	26 (9.7)
HCM	1 (2)	16 (7.3)	17 (6.3)
Myocarditis	3 (6.1)	19 (8.6)	22 (8.2)
Myocardial bridge	2 (4)	5 (2.3)	7 (2.6)
Pulmonary thrombo-embolism	1 (2)	3 (1.4)	4 (1.5)
Dissecting aortic aneurysm	1 (2)	11 (5)	12 (4.5)
Dilated cardiomyopathy	1 (2)	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.

Modified from Corrado *et al.*³

to identification and disqualification of affected athletes by Italian pre-participation screening.

Other ECG-detectable cardiovascular diseases

Twelve-lead ECG offers the potential to detect (or to raise clinical suspicion of) lethal conditions (other than HCM) manifesting with ECG abnormalities, such as ARVC/D, dilated cardiomyopathy, long QT syndrome, Lenègre disease, Brugada syndrome, short QT syndrome, and WPW syndrome (Tables 3 and 4). Overall, these conditions (including HCM) account for up to 60% of sudden deaths in young competitive athletes.^{3,15,17,21,26} It is noteworthy that a number of these conditions have been discovered only recently, so that diagnosis at pre-participation screening is being increased over time and its impact on mortality will be assessed in the near future. It is emblematic, in this regard, of the Italian screening for ARVC/D. We previously reported that 82% of athletes who died from such a disease had a history of syncope, ECG changes, and/or ventricular arrhythmias;³ however, they had not been identified at pre-participation screening. A plausible explanation is that ARVC/D is a condition that was discovered only recently (two decades ago) and for a long time has either been under-diagnosed or regarded with scepticism by cardiologists.²⁷⁻²⁹ Accordingly, we recently compared two decades of screening at the Center for Sport Medicine in Padova and found that the prevalence of athletes with ARVC/D identified and disqualified was significantly increased from 1992 to 2001 compared with the previous decade.^{25,30} This suggests that with the increased awareness of clinical findings of ARVC/D, more affected athletes are actually being identified by screening and protected from the risk of athletic competition.

The possibility of detecting either premature coronary atherosclerosis or anomalous coronary artery in young competitive athletes is limited by the scarcity of baseline ECG signs of myocardial ischaemia.^{20,31,32} However, we

reported that approximately a quarter of young athletes who died from coronary artery diseases had warning symptoms and/or ECG abnormalities at pre-participation screening that could raise the suspicion of a cardiac disease.³

Recommendations for pre-participation cardiovascular screening

With the present consensus document, the Sport Cardiology Study Group of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial diseases of the European Society of Cardiology recommends systematic pre-participation cardiovascular screening of young competitive athletes for the timely detection of cardiovascular abnormalities predisposing to sport-related cardiac death, thereby to reduce the cardiovascular risk associated with sport participation. This is in keeping with the previous American Heart Association consensus statement that pre-participation cardiovascular screening of young competitive athletes is justifiable and compelling on ethical, legal, and medical grounds.²

At present, in consideration of the heterogeneity (or lack) of recommendations and regulations existing in the European nations regarding pre-participation medical clearance of competitive athletes, this panel suggests a European standard for medical evaluation of competitive athletes. The recommended protocol includes 12-lead ECG in addition to history and physical examination, which is the only screening modality proved to be effective in identifying athletes with HCM, and preventing sudden death.

Protocol of pre-participation cardiovascular screening

A flow chart illustrating the proposed screening protocol is shown in Figure 1. The initial cardiovascular evaluation consists of complete personal and family history, physical

Table 3 Criteria for a positive 12-lead ECG**P wave**

left atrial enlargement: negative portion of the P wave in lead V1 ≥ 0.1 mV in depth and ≥ 0.04 s in duration;
right atrial enlargement: peaked P wave in leads II and III or V1 ≥ 0.25 mV in amplitude.

QRS complex

frontal plane axis deviation: right $\geq +120^\circ$ or left -30° to -90° ;
increased voltage: amplitude of R or S wave in a standard lead ≥ 2 mV, S wave in lead V1 or V2 ≥ 3 mV, or R wave in lead V5 or V6 ≥ 3 mV;
abnormal Q waves ≥ 0.04 s in duration or $\geq 25\%$ 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 s;
R or R' wave in lead V1 ≥ 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 heart rate corrected QT interval >0.44 s in males and >0.46 s in females.

Rhythm and conduction abnormalities

premature ventricular beats or more severe ventricular arrhythmias;
supraventricular tachycardias, atrial flutter, or atrial fibrillation;
short PR interval (<0.12 s) with or without 'delta' wave;
sinus bradycardia with resting heart rate ≤ 40 beats/min^a;
first (PR ≥ 0.21 s^b), second or third degree atrioventricular block.

^aIncreasing less than 100 beats/min during limited exercise test.

^bNot shortening with hyperventilation or limited exercise test.

Modified from Corrado *et al.*³

examination including blood pressure measurement, and 12-lead ECG. The athletic evaluation should be performed by a physician with the specific training, medical skill, and cultural background to identify reliably the clinical symptoms and signs associated with those cardiovascular diseases responsible for exercise-related sudden death. In Italy, physicians primarily responsible for pre-participation screening and eligibility for competitive sports attend postgraduate residency training programmes in sports medicine (and sports cardiology) full-time for four years. Such specialists work in sports medical centres specifically devoted to periodical evaluation of athletes.

The screening should start at the beginning of competitive athletic activity, which for the majority of sports disciplines corresponds to an age of 12–14 years. The screening should be repeated on a regular basis at least every 2 years for the timely identification of progression, over the period, of some diseases.

Medical history

The majority of conditions at risk of sudden death during sports are genetically determined diseases with an autosomal dominant pattern of inheritance, hence the importance of family history in identifying affected athletes. The family history is considered positive when close relative(s) had experienced a premature heart attack or sudden death (<55 years of age in males and <65 years in females), or in the presence of a family history of cardiomyopathy, Marfan syndrome, long QT syndrome, Brugada syndrome, severe arrhythmias, coronary artery disease, or other disabling cardiovascular diseases. The personal history is considered positive in the case of exertional chest pain or discomfort, syncope or near-syncope, irregular heartbeat or

palpitations, and in the presence of shortness of breath, or fatigue out of proportion to the degree of exertion.

Physical examination

Positive physical findings include musculoskeletal and ocular features suggestive of Marfan syndrome, diminished and delayed femoral artery pulses, mid- or end-systolic clicks, a second heart sound single or widely split and fixed with respiration, marked heart murmurs (any diastolic and systolic grade $\geq 2/6$), irregular heart rhythm, and brachial blood pressure $>140/90$ mmHg (on >1 reading).

ECG

Twelve-lead ECG is considered positive, according to accepted criteria,^{3,33} in the presence of one or more of the findings reported in Table 2.

Subjects who have positive findings at basal evaluation should be referred for additional testing, initially 'non-invasive' such as echocardiography, 24-h ambulatory Holter monitoring, and exercise testing. Alternatively, or in uncertain cases, 'invasive' tests such as contrast ventriculography (both right and left), coronary angiography, endomyocardial biopsy, and electrophysiological study may be necessary in order to confirm or rule out the suspicion of heart disease.^{25,26,29}

Finally, subjects recognized to be affected by cardiovascular conditions potentially responsible for sudden death in association with exercise and sport participation should be disqualified from competitive athletic activity, according to the recommendations stated by the Bethesda Conferences 16, 26 (and the forthcoming 36)^{34,35} and by the Italian Committee for Sports Eligibility (COCIS).³⁶ Updated European guidelines are being

Table 4 ECG Features of cardiac diseases detectable at pre-participation screening in young competitive athletes

Disease	QTc interval	P wave	PR interval	QRS complex	ST interval	T wave	Arrhythmias
HCM	Normal	(left atrial enlargement)	Normal	Increased voltages in mid-left precordial leads; abnormal Q waves in inferior and/or lateral leads; (LAD, LBBB); (delta wave)	Down-sloping (up-sloping)	Inverted in mid-left precordial leads; (giant and negative in the apical variant)	(Atrial fibrillation); (PVB); (VT)
Arrhythmogenic right ventricular cardiomyopathy/dysplasia	Normal	Normal	Normal	Prolonged >110 ms in right precordial leads; epsilon wave in right precordial leads; reduced voltages ≤ 0.5 mV in frontal leads; (RBBB)	(Up-sloping in right precordial leads)	Inverted in right precordial leads	PVB with a LBBB pattern; (VT with a LBBB pattern)
Dilated cardiomyopathy	Normal	(Left atrial enlargement)	(Prolonged ≥ 0.21 s)	LBBB	Down-sloping (up-sloping)	Inverted in inferior and/or lateral leads	PVB; (VT)
Long QT syndrome	Prolonged >440 ms in males >460 ms in females	Normal	Normal	Normal	Normal	Bifid or biphasic in all leads	(PVB); (torsade de pointes)
Brugada syndrome	Normal		Prolonged ≥ 0.21 s	S1S2S3 pattern; (RBBB/LAD)	Up-sloping coved-type in right precordial leads	Inverted in right precordial leads	(Polymorphic VT); (atrial fibrillation) (sinus bradycardia)
Lenègre disease	Normal	Normal	Prolonged ≥ 0.21 s	RBBB; RBBB/LAD; LBBB	Normal	Secondary changes	(2nd or 3rd degree AV block)
Short QT syndrome	Shortened <300 ms	Normal	Normal	Normal	Normal	Normal	Atrial fibrillation (polymorphic VT);
Pre-excitation syndrome (WPW)	Normal	Normal	Shortened <0.12 s	Delta wave	Secondary changes	Secondary changes	Supraventricular tachycardia; (atrial fibrillation)
Coronary artery diseases ^a	(Prolonged)	Normal	Normal	(Abnormal Q waves) ^b	(Down- or up-sloping)	Inverted in ≥ 2 leads	PVB; (VT);

Less common or uncommon ECG findings are reported in brackets.

QTc: QT interval corrected for heart rate by Bazett's formula. LBBB: left bundle branch block. RBBB: right bundle branch block. LAD: left axis deviation of -30° or more. PVB: either single or coupled premature ventricular beats. VT: either non-sustained or sustained ventricular tachycardia.

^aCoronary artery diseases: either premature coronary atherosclerosis or congenital coronary anomalies.

^bAbnormal Q waves (see Table 3).

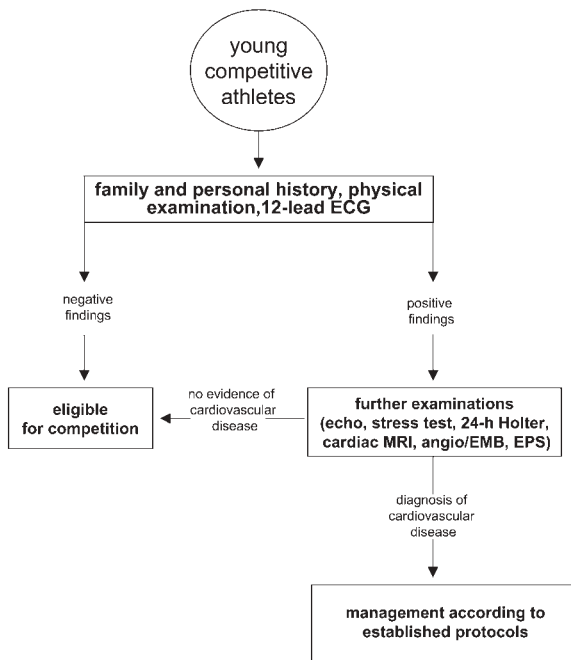


Figure 1 Flow diagram illustrating the proposed screening protocol for young competitive athletes. The initial cardiovascular protocol includes family and personal history, physical examination with determination of blood pressure, and basal 12-lead ECG. Additional tests, such as echocardiography, 24-h Holter monitoring, or stress testing, SAECG, and cardiac MRI are requested only for subjects who had positive findings at the initial evaluation. In uncertain cases, invasive tests such as contrast ventriculography (both right and left), coronary angiography, endomyocardial biopsy, and electrophysiological study may be necessary in order to confirm (or rule out) the diagnosis of cardiovascular disease. Athletes diagnosed with clinically relevant cardiovascular abnormalities are managed according to available guidelines for assessing athletic risk. EMB, endomyocardial biopsy; EPS, electrophysiological study; MRI, magnetic resonance imaging; SAECG, signal-averaged ECG.

elaborated by the present Study Group and are expected to be published soon.

Socio-economic impact

Screening of large athletic populations may have a significant socio-economic impact. Strategies for implementing the proposed screening programme across Europe depend on the particular socio-economic and cultural background as well as on the specific medical systems in place in different countries and go beyond the scope of the present consensus document. However, Italian experience indicates that the proposed screening design is made feasible because of the limited cost of 12-lead ECG in the setting of a mass screening. The cost of performing a pre-participation cardiac history/physical examination by qualified physicians, has been estimated to be 20 Euros per athlete and rises to 30 Euros per athlete if a 12-lead ECG is added. The screening cost is self-covered by the athlete or by his athletic team, except for athletes aged less than 18 years, for whom the expense is supported by the National Health System. Costs of infrastructure and training courses for pre-participation screening must also be taken into account.

Future directions

The future for prevention of sports-related fatalities lies in continuing efforts to better understand the substrates and mechanisms underlying sudden death in the athlete and to design more specific and efficient screening strategies. An international registry collecting all fatal events in young competitive athletes is warranted to evaluate whether genetic and/or environmental factors may influence the distribution of cardiovascular causes of sudden death in the different European countries. Echocardiography is a non-invasive and widely available tool with the potential to increase the screening accuracy for detection of diseases carrying a risk of sudden death in the athlete. Cost-effectiveness of screening modalities based on systematic echocardiographic examination, either complete or limited, remains to be prospectively assessed by studies on large athletic populations.

Conclusions

Pre-participation cardiovascular evaluation of young competitive athletes by 12-lead ECG (in addition to history and physical examination) is warranted on the basis of the available evidence, coming from the 25-year Italian experience, that athletes affected by HCM are successfully identified and athletic field fatalities reduced. In addition, such a pre-participation cardiovascular evaluation offers the potential for detecting other potentially lethal cardiovascular conditions mostly presenting with ECG abnormalities. Although the proposed screening protocol is at present difficult to implement in all European countries, the hope is that the successful Italian experience will lead progressively to its widespread adoption in the setting of European regulation of the health system.

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