Chapter 2
Cardiovascular Screening of Athletes: Focused Exam, Electrocardiograms, and Limited Echocardiograms

Christine E. Lawless

Introduction

The incidence of sudden cardiac death (SCD) in athletes is estimated to occur at a frequency of 1/200,000 athletes per year [1]. It is postulated that intense exercise predisposes the competitive athlete with previously undetected cardiac disease to lethal rhythm disturbances, and that the risk can be diminished if the athlete is withheld from sports participation.

This chapter explores the rationale for preparticipation cardiovascular screening with and without cardiovascular testing such as electrocardiograms (ECGs) and echocardiograms (echoes); workup of athletes with past history of heart disease, symptoms, abnormal findings or family history; barriers to routine ECG-based screening in the USA; how to implement ECG-based screening programs; how to interpret ECGs in athletes; the role of echo in screening of athletes; and what questions need to be addressed before widespread screening can be implemented in the USA.

Rationale for Pre-participation Cardiovascular Screening with and Without ECG

Attempts to reduce or eliminate the risk of SCD have led to intense screening efforts to detect the underlying potentially lethal cardiac conditions, with the primary screening tool being the preparticipation history and physical examination (PPE). The true sensitivity and specificity of the PPE is not known, but early retrospective studies suggest that the sensitivity of the PPE to detect underlying cardiac disease appears to be quite low, in the range of 2.5–6% [2]. In a registry study published in

C.E. Lawless (✉)
Sports Cardiology Consultants, LLC, Clinical Associate Faculty, University of Chicago Consulting Cardiologist, Major League Soccer Team Physician, US Figure Skating World Teams, 360 West Illinois Street #7D, Chicago, IL 60654, USA
e-mail: christine.lawless@yahoo.com

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1996, 4 of 115 athletes who had undergone PPE were successfully diagnosed with underlying cardiovascular disease, suggesting a sensitivity of 2.5% for PPE alone in detecting previously unsuspected heart disease. However, in another 15 athletes who underwent PPE plus cardiac testing (ECG, echo, stress testing), the correct underlying diagnosis was made in 7 of 15 athletes, thus increasing the sensitivity of the PPE to correctly identify heart disease to about 50% [2]. Two recent studies of PPE in collegiate athletes suggest that careful history and physical examination contribute to a PPE sensitivity of approximately 40–50% [3, 4]. Reasons for increased sensitivity of PPE in recent years may include increased awareness of SCD in athletes, increasing requirements for and standardization of PPE forms at the state level, growing experience and skill of the US physician workforce in administration of the PPE, and increased use of the 2007 AHA guidelines describing a focused cardiovascular PPE [5]. However, despite improvements in the PPE over the last 15 years, the incidence and epidemiology of SCD in athletes, based on the latest report from the SCD registry, appears unchanged in the last decade [6].

Assuming that the PPE alone has a sensitivity between 2.5 and 40% in the athletic population, it has been suggested that routine cardiac testing such as ECG or echo be added to the standard PPE [7–9]. This approach appears to be effective, based on retrospective epidemiologic data obtained from Italy, where ECG-based screening has been in place long enough to determine long-term effects on mortality. The incidence of SCD in athletes appears to have decreased by 89% over the past 25 years. Italian researchers are convinced that this has been primarily due to ECG-based screening rather than administration of the PPE alone [10]. American critics of the Italian data have suggested that as multiple interventions were introduced in Italy over the past 25 years, and this is not randomized prospective data one cannot be certain that the reduction in SCD was solely due to the addition of ECG to the PPE.

The American Heart Association (AHA) consensus document on preparticipation screening of athletes published in March 2007 states that ECG-based screening protocols are not encouraged in the USA for several important reasons: (1) There is a lack of specialized practitioners carefully trained in screening and interpretation of ECGs in the athletic population; (2) The cost of conducting such screening in such a large number of eligible athletes may be prohibitive; (3) The mortality rate from SCD in athletes in the USA is already quite low, in fact, at the level achieved by the Italians after over 20 years of performing ECG-based screening; (4) Randomized trials with outcomes demonstrating clear superiority of the ECG-based screening over a standardized PPE without ECG are lacking; (5) There is a lack of standardization for interpretation of ECGs in athletes; and (6) There is a lack of normative data in certain demographic and ethnic groups [5]. At present, US athletic governing bodies such as the NCAA have not endorsed use of the ECG for routine screening of athletes. However, organized professional sports such as the NBA, NFL, and MLS have embraced the practice, with some including echocardiography and stress testing as well the ECG [11].

In lieu of widespread ECG-based screening for athletes, AHA consensus writers have recommended that the AHA 12 points or elements ought to be included in the focused cardiac portion of the PPE [5] (Table 2.1), and that cardiac signs and symptoms, and significant family history be promptly and thoroughly evaluated.

Table 2.1 The 12-element AHA recommendations for preparticipation cardiovascular screening of competitive athletes (reprinted with permission)

Medical history[a]
Personal history
1. Exertional chest pain/discomfort
2. Unexplained syncope/near-syncope[b]
3. Excessive exertional and unexplained dyspnea/fatigue, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
Family history
6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease, in ≥1 relative
7. Disability from heart disease in a close relative <50 years of age
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long-QT syndrome or other ion channelopathies, Marfan's syndrome, or clinically important arrhythmias
Physical examination
9. Heart murmur[c]
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan's syndrome
12. Brachial artery blood pressure (sitting position)[d]

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[a]Parental verification is recommended for high school and middle school athletes

[b]Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion

[c]Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction

[d]Preferably taken in both arms

Appropriate Workup of Athletes with Past History of Heart Disease, Symptoms, Positive Family History, or Abnormal Physical Examination

The main point of the cardiac portion of the PPE is to establish the risk of participation in those with prior history of heart disease, and to detect previously undetected lethal cardiac conditions. On occasion, the athlete may present with a prior history of heart disease or a prior workup for heart disease. The clinician is obliged to confirm the diagnosis and determine the risk of participation. Multiple sets of published guidelines, especially the 36th Bethesda Guidelines, provide a framework as to whether to allow such an athlete to participate [12]. Clinicians need to be aware that not all healthcare practitioners adhere to published guidelines, and that there may be differences of opinion as to whether play should be allowed [13, 14].

It is absolutely crucial that clinicians understand the common causes of SCD in athletes and know how to identify these conditions. For example, as hypertrophic cardiomyopathy (HCM) is the most common cause of SCD in athletes, the clinician must be aware that the cardinal features of HCM are syncope, chest pain, shortness

of breath, palpitations, fatigue, family history, and presence of a cardiac murmur. Anomalous coronary artery, the second most common underlying cardiac cause of SCD in athletes, may or may not be symptomatic. If symptoms are present, they will most likely consist of syncope, shortness of breath, chest pain with exertion, and palpitations.

Chest Pain

The differential diagnosis of chest pain is lengthy; cardiac causes probably account for less than 5%, with the majority of chest pain in athletes being caused by musculoskeletal conditions, gastrointestinal reflux, or pulmonary causes [15]. It is crucial that the clinician think of a cardiac cause before all others; once a significant cardiac condition is ruled out or deemed unlikely, other causes should be entertained.

Dyspnea

Shortness of breath is more likely to be pulmonary, but clinicians must carefully consider cardiac causes before assuming a pulmonary cause. Although not all physicians might agree with the practice, it is common for the physician to try empiric inhalers in a dyspneic athlete. However, if the athlete fails to improve, cardiac investigation is warranted.

Syncope, Near-Syncope, and Dizziness

Syncope *during exercise* necessitates a cardiac workup, while syncope occurring *at rest* may be of a benign nature [16]. Keep in mind that not all patient-athletes follow these widely held perceptions. Similarly, palpitations occurring with exercise are likely to be rhythm disturbances exacerbated or unmasked by catecholamines or enhanced adrenergic tone, while those occurring at rest are more likely to be of a benign nature. However, as with syncope, not all patient-athletes follow these common beliefs, and numerous examples to the contrary have been reported at sports medicine meetings and in small case studies [17]. This author strongly recommends that a recording of the underlying ECG rhythm *during the symptoms* be obtained in all athletes presenting with symptoms of palpitations, regardless of the symptoms occur at rest or with exercise [18]. If the rhythm is indeed benign, such as premature atrial beats, then no further workup is indicated. Some athletes present with either symptomatic or asymptomatic premature ventricular beats or contractions (PVCs). Biffi and colleagues have shown that the PVC burden correlates with the likelihood of underlying heart disease; the greater the PVC burden, the more likely the athlete is to have underlying disease [19]. Specifically, more than 2,000 PVCs/24 h indicates a 30% risk of underlying heart disease, but less than

2,000 PVCs/24 h correlates with a risk of less than 5%. More serious rhythms such as ventricular tachycardias or short runs require appropriate investigation [20].

Family History

Athletes with a family history of SCD prior to the age of 50 must be evaluated for inheritable forms of heart disease such as cardiomyopathy or channelopathy. It is more likely that athletes with a positive family history will have a relative who succumbed to coronary artery disease (CAD) rather than cardiomyopathy, as CAD is far more common in the general population. However, the PPE represents an opportunity to identify young people who are either carriers of cardiomyopathy genes or genes known to be associated with the development of premature CAD. Since there is an exhausting differential diagnosis for premature CAD, readers are referred to an excellent review of this subject from the AHA [21]. To illustrate that the concern for premature CAD is real among athletes, recall the case of Olympic figure skater Sergei Grinkov. He suffered fatal acute thrombosis of the left anterior descending artery during a practice session when he was 28 years old; postmortem analysis of his blood revealed a defect in a platelet proteoglycan, which is known to lead to premature coronary events [22].

Aside from history, physical examination may reveal clues to the presence of underlying heart disease. Blood pressure measurement, any delay in femoral pulses (indicative of coarctation of the aorta), presence of a cardiac murmur (consider HCM or valvular disease), or features of Marfan's syndrome may lead one to suspect an underlying cardiac condition (see chapters on murmurs 11, HCM 13, and Marfan's syndrome 16).

Once the PPE has been completed, any prior history of cardiac disease has been addressed, and any signs or symptoms have been satisfactorily explained, there are multiple sets of useful guidelines that suggest proper levels of participation and activity for the athlete. The most commonly used guideline is the 36th Bethesda Guidelines, updated in 2005 and summarized in Table 2.2 [12]. See chapter 21 for a comprehensive list and discussion of all existing guidelines.

Case Study of Positive Family History

On PPE, a 16–year-old high school basketball player had a significant family cardiac history. His father had a diagnosis of HCM since age 36 and suffered a cardiac arrest at the age of 42. After this happened, family members were evaluated for phenotypic and genotypic evidence of HCM. The player had no symptoms and no cardiac murmur. ECG, echo, and cardiac MRI were all normal, showing no hypertrophy or scar. Genetic blood testing revealed that the player carried the same HCM gene that his father carried. The player was allowed to participate in basketball, according to the recommendation of the 36th Bethesda Guidelines (genotype positive, phenotype negative HCM) [12]. However, the European guidelines would

Table 2.2 Summary of 36th Bethesda guidelines (adapted and reprinted with permission form reference [2])

	HCM	Anomalous coronary	ARVC	DCM	Long QT syndrome	Marfan's syndrome
Participation in all sports allowed	No	No	No	No	No	IA–IIA sports, with certain restrictions, depending on size of aorta (≤40 mm), absence of family history of dissection, and absence of significant valve disease)
Participation allowed if genotype +, phenotype –	Yes	N/A	Not specified in Bethesda guidelines	Not addressed	Yes, but no swimming allowed for Long QT1	Not specified
Participation allowed after corrective surgery	No	Yes	N/A	Yes, post heart transplant, provided no coronary luminal narrowing or ischemia	N/A	Low intensity (IA) sports only
Participation allowed with ICD	No	No	No	No	No	N/A
Participation allowed with beta blockers	No	Not specifically addressed	Not specifically addressed	Not addressed. If ejection fraction has normalized, no specific comment made	Not specifically addressed	Not specifically addressed

HCM = hypertrophic cardiomyopathy; ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy

not allow play in this instance [23]. Follow-up: The athlete has been playing without incidence for 4 years; annual ECG, echo, and MRI show no evidence of phenotypic expression of the disease.

ECG-Based Screening

Despite the limitations of the PPE alone, and the limitations of the ECG as a screening tool, some authors and professional organizations have recommended that ECG be added to routine PPE to enhance its ability to detect disease [8, 24]. In 2004, the International Olympic Committee recommended that an ECG be performed on all elite athletes prior to Olympic sports participation [23] and in 2005, the European Society of Cardiology recommended implementation of a common European ECG-based screening protocol [8]. Concurrently, the authors of the AHA/American College of Cardiology 36th Bethesda Guidelines for Sports Participation concluded that "ECG's are a practical and a cost effective strategic alternate to routine echocardiography for population based pre-participation screening," assuming that the ECG would be 75–95% sensitive in detecting HCM [12].

The true sensitivity and true specificity of the ECG in the athletic population is not known. However, Pelliccia has attempted to estimate such statistics by correlating surface ECG with underlying echocardiography in 1,005 consecutive elite athletes and categorizing the ECGs into three categories: normal, mildly abnormal, and distinctly abnormal, with the distinctly abnormal pattern being associated with a greater chance of underlying heart disease [24]. The combination of mildly abnormal and distinctly abnormal ECGs demonstrates a sensitivity of 51%, a specificity of 61%, a positive predictive accuracy of 7%, and a very high negative predictive accuracy of 96%. In the last decade, a number of investigators have evaluated the utility of ECG for screening of athletes, with varying results (Table 2.3).

Surface ECG patterns vary according to gender and sport, with distinctly abnormal patterns more likely to occur in males than in females and more likely to occur in endurance sports [24]. This may become clinically relevant when interpreting ECGs in specific athlete groups. For instance, if a distinctly abnormal pattern is seen in a female equestrian athlete, pathology is more likely than athletic adaptation.

In a study of 32,652 athletes, distinct ECG abnormalities included deeply inverted T waves (2.3%), significant LV hypertrophy (0.8%), right bundle branch block (1.0%), left anterior fascicular block (0.5%), left bundle branch block (0.1%), cardiac preexcitation pattern (WPW; 0.1%), and prolonged QTc interval (0.03%) [25]. Deeply inverted T waves may be particularly ominous, perhaps a precursor of cardiomyopathy, and may represent the earliest form of phenotypic expression [26]. The Italian authors note that the prevalence of markedly abnormal ECG patterns suggestive for structural cardiac disease is actually quite low (<5% in the general population) and should not represent an obstacle for implementation of 12-lead ECG-based screening program [25].

In 1999, Sharma and colleagues compared the ECGs of 1,000 junior elite British athletes (mean age 15 years) with the ECGs of 300 control, nonathletic,

Table 2.3 Studies of ECG as part of preparticipation examination for sports participation

Authors	Population	*N*	Incidence ECG abnormalities
Baggish et al. 2010	Collegiate athletes	510	2.2% had relevant cardiac abnormalities; ECG doubled sensitivity of screening over exam and history alone (45.5–90.9%)
Le et al. 2010	Collegiate athletes	658	10% distinctly abnormal ECG
Thunenkotter et al. 2010	Professional soccer players	605	4.8% pathological ECG
Crouse et al. 2009	NCAA football players	77	79% had ≥1 abnormal ECG finding
Sofi et al. 2008	General population seeking eligibility for sports participation	30,065	4.9% abnormal pattern on exercise ECG; 0.6% considered ineligible for sports
Basavarajaiah et al. 2008	Asymptomatic elite athletes in UK	3,500	0.08% LV hypertrophy consistent with HCM
Pelliccia et al. 2008	Trained athletes seeking eligibility for sports participation	From 12,550 tracings: $n = 81$ with ECG abnormalities; $n = 229$ matched controls	6% of athletes with abnormal ECG had cardiomyopathies
Wilson et al. 2008	National and international junior athletes	1,074 junior athletes; 1,646 active schoolchildren	0.3% had positive diagnosis of disease associated with SCD
Magalski et al. 2008	Elite American football players	1,959	25% had abnormal ECG; 30% among black players vs. 13% among white players; ECG abnormalities suggestive of cardiac disease: 6% vs. 2% in black and white athletes, respectively
Pelliccia et al. 2007	General population seeking eligibility for sports participation	32,652	11.8% had abnormal ECG; <5% had marked changes suggestive of cardiac disease
Pelliccia et al. 2006	Professional athletes	4,450	0.3% had cardiac abnormalities
Pelliccia et al. 2000	Competitive athletes	1,005	14% had distinctly abnormal ECG; 5% had structural cardiac deficits

See text for abbreviations cardiomyopathy

age-matched individuals [27]. Junior athletes were more likely to demonstrate sinus bradycardia (80%), sinus arrhythmia (52%), first degree A–V block (5%), incomplete right bundle branch blocks (29%), left atrial enlargement (14%), right atrial enlargement (16%), S–T segment elevation (45%), tall-peaked T waves (22%), and isolated Sokolow voltage criteria for LVH (45%). Corrected Q–T interval and the QRS duration were longer in the athletes than in the non-athletes. The authors concluded that the following might be indicative of underlying pathology in a highly trained junior athlete: ST depression or deep T inversion, minor T-wave inversions in any lead except V2–V3 when the athlete is less than 16 years old, Romhilt–Estes voltage criteria for LVH in female athletes, pathological Q wave, left axis deviation, and complete left bundle branch block.

American athletes have not been studied to the same degree as European athletes have been. However, isolated studies performed over the past 10 years illustrate the challenges faced in the USA. In 1997, Fuller et al. prospectively screened 5,615 high school athletes in northern Nevada with a PPE and ECG [7]. Five percent of subjects were found to have R or S waves greater than 30 mm; 6% had T-wave flattening or inversion in two or more leads; 2% had abnormal Q waves; and the axis was deviated greater than –30 or 120° in 1%. ECG abnormalities were present in 15.7% of the entire cohort. Fuller correlated findings on PPE and ECG with outcomes, defined as detection of any cardiovascular disease during the screening process that would preclude sports participation according to the 16th Bethesda guidelines. The sensitivity of the PPE to detect the abnormality was only 6%, whereas, ECG significantly increased the sensitivity to 70%.

Fuller's study was conducted primarily in Caucasian high school athletes in Reno, Nevada. However, based on early studies conducted in nonathletic African American subjects, there is reason to suspect that there are substantial ethnic differences in the appearance of the ECG in athletes [28, 29]. Two studies reported in 2008 shed light on this issue [30, 31]. Magalski et al. reported that the most abnormal ECG patterns (increased voltage, diffuse T inversion, deep Q waves) were found in 5.8% of blacks compared to only 1.8% of whites [30]. Concurrently, Basavarajaiah et al. showed that Sokolow–Lyon voltage criteria for LVH were more common in blacks than in whites with echocardiographic features of LVH, 68 vs. 40% [31]. Deep T-wave inversions in the precordial leads were also more prevalent (12 vs. 0%). Two recent studies of collegiate athletes suggest that ECG interpretation may lead to false-positive identification of athlete's cardiovascular risk [3, 4]. Baggish et al. screened 510 athletes at Harvard University [3]. The addition of ECG to physical examination and history improved the sensitivity of PPE to approximately 90%; however, inclusion of ECG reduced specificity and was associated with a false-positive rate of 16.9%. Le et al. evaluated the use of ECG in 658 athletes presenting for PPE at Stanford University [4]. Although 68% of female athletes had normal ECGs, only 38% of men had normal tracings. In all, 10% of athletes were considered to have distinctly abnormal ECG and were subjected to further testing. These studies are highly illustrative of the pitfalls of ECG interpretation in athletes in the USA; false positives are common, especially in the black population.

None of the above studies reflect the true impact of the PPE alone vs. the value added by the ECG in the prevention of SCD in athletes. Such an analysis would

require a prospective randomized study, comparing standardized PPE with the 12 AHA questions to the standardized PPE with AHA questions plus the 12-lead ECG. Such a study would also require a "gold standard" such as echocardiography, advanced cardiac imaging, or genetic testing on all athletes to determine whether or not underlying heart disease was present and would include a certain percentage of known abnormals such that sensitivity and specificity could be determined. Such a study would also demand that endpoints include the impact of diagnosing and treating possible underlying disease such that the true effect on overall mortality and SCD could be determined. Given the low incidence of SCD in athletes in the USA, and the potential morbidity of cardiac procedures it is entirely possible that cardiac screening and further testing might actually result in worse outcomes [32].

Barriers to Routine ECG-Based Screening in the USA

Large Numbers of Athletes and Size of Appropriate Physician Workforce to Conduct the Screenings

The population of Italy is about one-fifth that of the USA, and Italian athletes are seen in one of several screening clinics by a highly trained Italian screening physician. The training program for screening physicians consists of 4 years of specialized sports medicine training, including rotations like sports traumatology, sports dermatology, and sports cardiology. During the sports cardiology rotation, trainees spend a significant amount of time performing ECGs and echoes, evaluating symptomatic athletes, and making participation decisions for all athletes. Thus, upon completion of their 4-year training, Italian sports medicine physicians are very familiar with the discipline of sports cardiology. The Italian approach to screening athletes is illustrated in Fig. 2.1. It should be noted that in Europe there has been a shift from the three-category Pelliccia criteria to the two-category Corrado criteria, with or without a recommended cutoff for degree of LVH voltage [8, 33].

In contrast, in the USA, the number of athletes, the training programs for sports medicine physicians, and the types of clinicians clearing athletes are vastly different from the Italian model. An estimated 40 million athletes participate at the professional, collegiate, high school, middle school, club, and recreational levels in some type of organized sport. At the professional level, athletes typically undergo PPE by contracted team physicians who are granted the authority to make participation determinations. A recent survey indicated that 92% of professional athletes underwent ECG screening, with a smaller percentage undergoing additional cardiac testing such as echocardiography and stress testing [11]. This approach is possible because of the small numbers of athletes, the ability of the sports organization to pay for such testing, and the training of the specialized team physicians caring for these athletes. The current US approach to PPE for athletes is illustrated in Fig. 2.2.

According to the NCAA, 300,000 athletes participate at the collegiate level. The PPE is required and generally conducted by team physicians contracted by

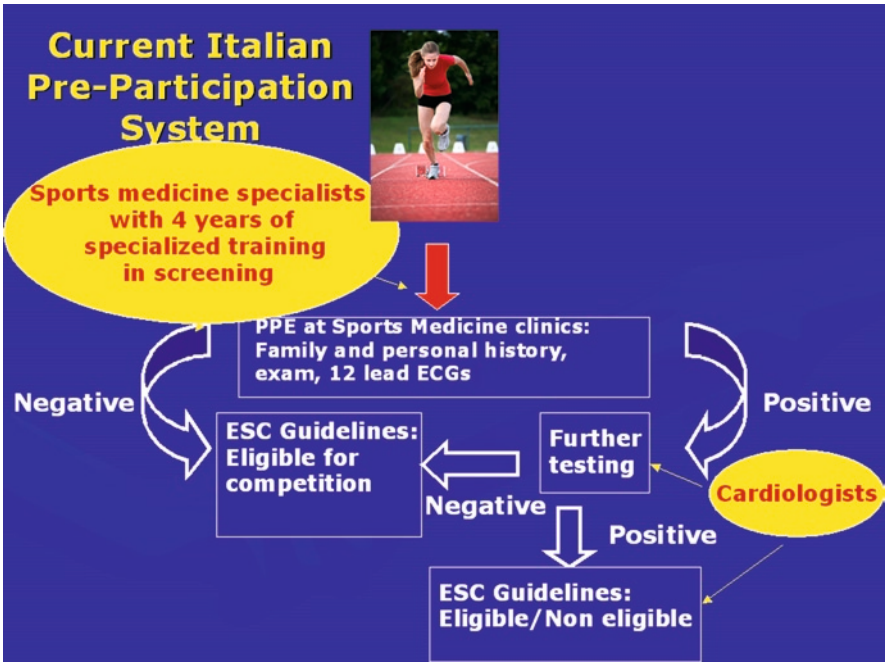

Fig. 2.1 The Italian approach to PPE screening for athletes, including the use of ECG

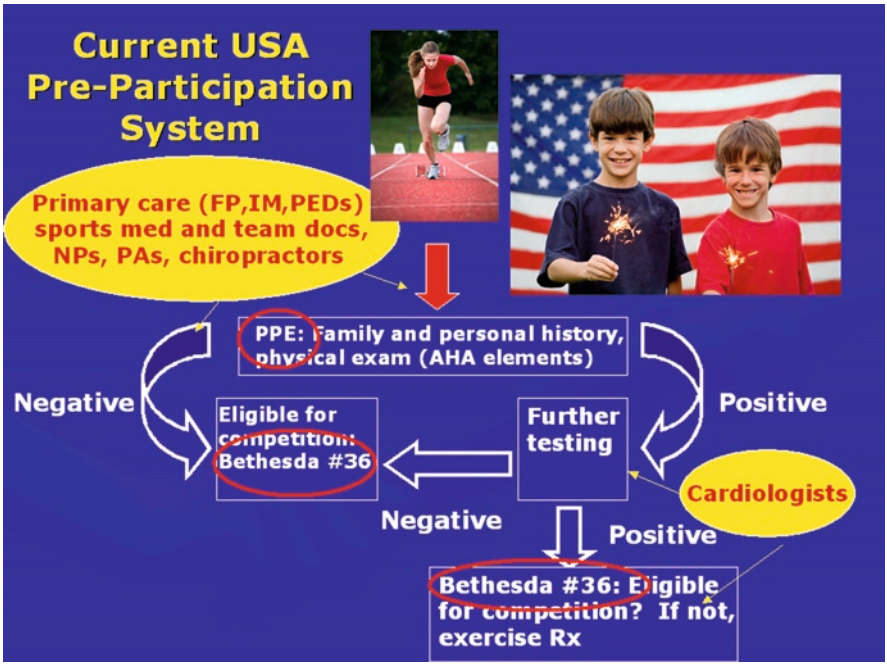

Fig. 2.2 The current US approach to PPE screening for athletes

the athletic departments. However, ECG in addition to the PPE is not mandated by the NCAA. Nonetheless, individual collegiate programs have chosen to include ECG based on the strength of the Italian data (personal communication, team physicians at University of South Florida, University of Georgia, University of Nevada, University of Florida, St. Louis University). In some instances, only athletes participating in high-risk sports such as basketball and football undergo cardiac testing (personal communication, Dr. Brolinson, Virginia Tech University).

The majority of professional and collegiate teams in the USA are served by sports medicine fellowship-trained team physicians. These physicians undergo a training program that is vastly different from that undergone by their European counterparts. In the USA, sports medicine physicians complete a 1-year sports medicine fellowship after 3 years of generalized family medicine, internal medicine, physiatry, pediatrics, emergency medicine, or orthopedics; the majority completing the fellowship are family medicine physicians. Typically, such physicians have had just 1 or 2 months of cardiology exposure during their family practice training and may have limited experience in ECG interpretation. During the sports medicine fellowship, trainees are not exposed to sports cardiology to any significant degree, as this does not exist as a specialty in the USA. Thus, sports medicine physicians have a substantial learning curve in ECG interpretation and if called upon to conduct a screening program, they often partner with the local cardiology consultant. Thus, it is crucial that the partnering cardiologist has a working knowledge of ECG interpretation in athletes.

At the high school level, the PPE may be conducted by a variety of clinicians, including sports medicine physicians, primary care physicians with no sports medicine background, nurse practitioners, and chiropractors. The quality of the PPE may be variable, exposure to cardiology may be extremely limited, and the ability to read an ECG may be highly variable. Given the large numbers of high school athletes, application of ECG-based screening would be a daunting task. Requiring club sport athletes to undergo PPE and ECG would be equally challenging.

Cost of Conducting Such Screening in Such a Large Number of Eligible Athletes

Although not the primary issue, the cost for conducting ECG-based screening is often mentioned in debates regarding such screening. For example, in a high school with 500 athletes and at a cost of $20 per ECG, theoretically it may cost up to $10,000 to conduct such a program. Despite the costs, some high schools have adopted ECG-based screening due to the efforts of volunteers, but the sustainability of such programs and quality may be questionable.

Mortality Rate from SCD in Athletes is Already Quite Low

One of the more compelling arguments against ECG-based screening in the USA is the unlikely possibility of improving upon the already low rates of SCD in US athletes. When the Italians embarked on their program in the early 1980s, the SCD rate in athletes was 3.5/100,000, but this declined to 1/200,000 approximately 25 years after ECG-based screening was initiated [10]. This figure is remarkably similar to what the USA already has achieved [6], opening to question whether ECG-based screening ought to be considered at all [32].

Lack of a Randomized Trial Demonstrating Clear Superiority of the ECG-Based Screening over a Standardized PPE Without ECG

Although the Italian data are compelling, the report published in 2007 was a retrospective analysis, and one cannot conclude with certainty that the results observed were due to the ECG alone. The program was implemented in 1981, but several interventions were implemented at once: enhanced history and physical by specially trained clinicians, ECG, and algorithms for work up and return to play. Over the years, protocols underwent gradual refinement. Thus, it cannot be concluded that the decline in SCD rate in athletes was due to the ECG alone; rather, one would have to conclude that the combination of practices allowed for the improvement.

Lack of Standardization for Interpretation of ECGs in Athletes

Computerized ECG interpretation algorithms have been derived from studies conducted in the general population many years ago. It has been well documented that athletes demonstrate distinctive 12-lead surface ECG patterns, which are more prevalent among endurance athletes and in male gender [24]. Interpretation criteria have been published by the Europeans, but these criteria have not yet been shown to have the ability to reliably identify underlying heart disease in a prospective, controlled trial. There are also the issues of false positives and overreading. As there is more hypertrophy, bradycardia, early repolarization, and ST–T alterations in athlete ECGs, there exists a false-positive rate of up to 40% among inexperienced readers [13, 30] to 1–2% among experienced readers [33]. This degree of variance would certainly justify aids such as algorithms or benchmarks for voltage or T waves that would trigger workups. At the moment, there are no such aids.

Lack of Normative Data in Certain Demographic and Ethnic Groups

It is important to note that the Italian ECG patterns are based on results obtained in elite Caucasian European athletes with a mean age of 23 years, and that rules of ECG interpretation derived from the Italian data may not necessarily be applied to younger athletes, to the recreational athlete, or to athletes of other ethnic groups. One such group is the African American athlete.

An early study in nonathletes of age 11–17 years indicated that black males ($n = 27$) demonstrated statistically higher voltage measurements in lead 1 (15 vs. 10 mm), V4 (50 vs. 36 mm), V5 (44 vs. 39 mm) and V6 (30 vs. 24 mm) than their white counterparts of the same gender ($n = 27$) [34]. Black males also had higher voltage than black females ($n = 34$) in lead V4 (50 vs. 47 mm), V5 (44 vs. 25 mm), and V6 (30 vs. 22 mm). In contrast, black females did not demonstrate significant differences from white females with the exception of the S wave in V1 (26 vs. 16 mm).

Recently, Magalski reported ECG findings of 1959 collegiate football players (67% black) [30]. The most abnormal patterns (distinctly abnormal, high voltage, diffuse deep T waves or Q waves) were found in 5.8% of blacks compared with 1.8% of whites. After adjustment for all other variables, black race was the only independent predictor of a distinctly abnormal ECG. Basavarajaiah reported in a group of British athletes that Sokolow–Lyon voltage criteria were more common in black athletes with echo evidence of LV hypertrophy than in white athletes (68 vs. 40%) [31]. Deep T-wave inversions in the precordial leads were seen in 12% of black athletes compared with none of the white athletes. These data suggest that screening large numbers of American athletes, especially if they are black, may not be practical due to the inability to distinguish normal athletic adaptation or normal ethnic variants from true pathology.

As there is currently no standard for ECG interpretation in the athlete, the ECG is subject to marked variation in interpretation by cardiologists and others involved in athlete care. As ECG is not 100% sensitive for the detection of underlying heart disease, the possibility of false negatives exists. Table 2.4 summarizes the sensitivity and specificity of the ECG for conditions known to predispose to SCD in athletes [35]. For some diagnoses, such as anomalous coronary artery, the sensitivity of the ECG will be extremely low, whereas for others, such as HCM, the ECG ought to be an excellent screening tool. False positives exist as well, as normal athletic adaptation to exercise can result in marked alterations of the surface ECG, resulting in delays due to the time it takes to follow-up and perform further cardiac evaluation.

How to Implement ECG-Based Screening Programs

Despite the critiques and limitations of the ECG, for those clinicians who choose to use an ECG-based approach in their preparticipation screening of athletes, the decision tree shown in Fig. 2.3 may be a useful guide [35]. This proposed approach

Table 2.4 Sensitivity and specificity of ECG for specific cardiovascular diagnoses (reproduced with permission)from reference #35

CV disease	Likelihood of being detected with ECG	ECG findings	Sensitivity	Specificity
Hypertrophic CM	High	LVH, abnormal axis, Q waves, ST-T changes	73–98%	Low
ARVD	Low to intermediate	T-wave inversions V leads	25–94%	Low, with exception of the epsilon wave
Dilated CM	High	May have infarct pattern, left anterior hemiblock, RBBB or LBBB, or LBBB, LVH, or ST-T abnormalities, LAE, or atrial fibrillation	Probably high ≤1% are normal	Low
Myocarditis	Low to intermediate	ST elevation	10–54%	Low
Long QT syndrome	High	Q–T c >440 ms males Q–T c >460 in females	83–100%	Intermediate
Brugada syndrome	Low	RBBB, and "coved" ST elevation in V1 and V2	At least 20%	High
Coronary anomalies	Low	None characteristic	Low	Low
Myocardial bridging	Low	None characteristic	Low	Low
Aortic stenosis	High	LVH, ST-T abnormalities	80%	Low
Mitral valve prolapse	Low	ST-T changes, PVCs	66%	Low
Marfan's syndrome	Low	None characteristic	Unknown	Unknown

CM cardiomyopathy, *LVH* left ventricular hypertrophy, *LBBB* left bundle branch block, *PVCs* premature ventricular contractions, *RBBB* right bundle branch block, *ARVD* arrhythmogenic right ventricular dysplasia/cardiomyopathy

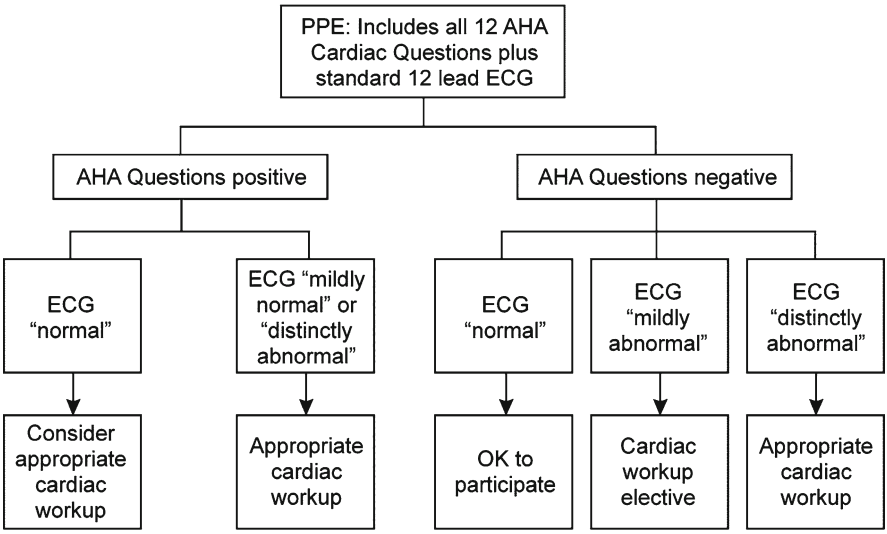

Fig. 2.3 Decision tree for ECG-based screening program (reproduced with permission from reference [35])

is consistent with what the Europeans have recommended [10]. The athlete with the distinctly abnormal ECG requires further investigation, regardless of symptoms. Cardiac workup is considered optional for the athlete without symptoms and mildly abnormal ECG or athletes with positive answers to the AHA questions but normal ECG. An alternative approach is to determine normative ECG data for the population one is screening and work up significant deviations from normal. This approach has been used by the NFL for years and has recently been adopted by MLS (personal communication, Dr. Christine Lawless, consulting cardiologist to MLS).

How to Interpret ECGs in Athletes

Although there is no uniform algorithm for interpretation of the ECG in asymptomatic athletes, the Europeans have proposed some simple rules that appear to reliably detect the majority of heart disease in young athletic populations (Table 2.5) [10, 30, 35]. It is important to note that the Italian ECG patterns are based on results obtained in elite, Caucasian, European athletes with a mean age of 24 years and that rules of ECG interpretation derived from the Italian data may not necessarily be applied to younger athletes, to the recreational athlete, or to athletes of other ethnic groups. One such group is likely the African American athlete. Based on published data in nonathletic African American populations, there is reason to suspect that ECGs in athletic African Americans may be markedly different from that in their white European counterparts [30, 31, 34]. Further study is necessary to determine normative data in this group of athletes. As there is currently no standard for ECG interpretation in the American

Table 2.5 Classification of abnormalities of the athlete's ECG (reproduced with permission from the European Society of Cardiology) [33]

Group 1: common and training-related ECG changes	Group 2: uncommon and training-unrelated ECG changes
Sinus bradycardia	T-wave inversion
First-degree AV block	ST-segment depression
Incomplete RBBB	Pathological Q waves
Early repolarization	Left atrial enlargement
Isolated QRS voltage criteria for left ventricular hypertrophy	Left-axis deviation/left posterior hemiblock
	Right ventricular hypertrophy
	Ventricular preexcitation
	Complete LBBB or RBBB
	Long- or short-QT interval
	Brugada-like early repolarization

RBBB right bundle branch block, *LBBB* left bundle branch block
Adapted from [33]

athlete, the ECG is subject to marked variation in interpretation by cardiologists and others involved in athlete care. It is not 100% sensitive for the detection of underlying heart disease; therefore, the possibility of false negatives exists. For some diagnoses, such as anomalous coronary artery, the sensitivity of the ECG will be extremely low, whereas for others, the ECG ought to be an excellent screening tool.

The Role of Echocardiography in Screening of Athletes

Because ECGs are not 100% sensitive, some authors advocate the addition of echo to the screening. However, there are many problematic issues inherent in this approach. Echo requires special equipment and training, is less portable, and more costly than ECG. Some would argue that there are quality concerns, since it is not likely that accredited echocardiographers would either perform and/or interpret all the screening tests. Given the number of athletes in the USA, widespread use of this technique does not seem practical. Nonetheless there are some who advocate this approach. Some collegiate programs have adopted an abbreviated echo for incoming athletes the first year they join their respective programs. Such abbreviated echoes tend not to be complete studies but are performed and interpreted by accredited laboratories and physicians and screen for the major causes of SCD in athletes. Wyman et al. reported results of a 5-min screening echo for collegiate athletes [9]. In 395 athletes studied, no athlete was found to have evidence of HCM. However, 19.5% were found to have trivial or mild mitral regurgitation, 13.4% trivial or mild tricuspid regurgitation, 3.9% trivial, mild, or moderate aortic insufficiency, 0.5% bicuspid aortic valve prolapse, and 1.3% mitral valve prolapse. Origin of the left coronary artery was identified in 99%, and origin of the right in 96%. This implies that anomalous coronary artery

can be detected in the majority of cases, but not all authorities agree that the echocardiogram is this sensitive for detecting this anomaly. Further study is warranted.

In a survey conducted in 2005 among 122 North American professional sports teams from the MLB, NHL, and NFL, 13% performed preparticipation echo [11]. There are a number of charitable organizations whose members are parents who have lost their teen-age athletes from sudden death during athletics. These groups promote echo screening of high school athletes and go as far as to advocate training of nonprofessionals in performance of inexpensive "screening" echo [36]. The quality of such screening programs, and the sensitivity and specificity of the echo in this model has not yet been validated. Momentum appears to have shifted in favor of the ECG because of ease of administration, cost, relatively low cost, and its ability to detect both HCM and long QT.

What Issues Still Need to be Addressed Before Wide-Spread Screening Can be Implemented in the USA

Efficacy of ECG Screening

Efficacy, cost, ability of the physician workforce to interpret the ECGs, and health-care disparities are cited as the main reasons that ECGs are not indicated in the USA [37]. In the USA, SCD rates in athletes may be as low as what Italy achieved after several decades of screening. At a rate of 1/200,000, the SCD rate amounts to 150–200 athletes per year. However, these figures are based on data gleaned from years of combing newspaper reports and internet reports of SCD in young athletes. Such data may be subject to selection bias. A well-designed epidemiologic study in Minnesota, which collected details of all deaths over time, showed that the rate of SCD was comparable to that in Padua Italy [38]. Thus, one is hard pressed to conclude that ECG screening would improve upon these rates. Some argue that the additional cardiac testing and use of cardiac procedures would actually add to the morbidity and mortality, resulting in higher rates of death [32]. Given the incidence of HCM in the general population of 1/500, of long QT 1/3,000, and of anomalous coronary 1/1,000, screening is unlikely to have impact.

If the question of efficacy were to be resolved by a large multicenter trial, huge numbers of participants would be necessary to power the study to detect differences. Supposing such a trial could be conducted, the next step is to determine who would pay for the cost of ECGs. If left to the individual, even a $20 ECG may prove cost prohibitive for certain demographics.

Lastly, the Italian program has been conducted by specially trained sports medicine physicians. These clinicians have learnt how to perform ECGs and echoes in athletes and are skilled in their interpretation. In contrast, the sports medicine physician workforce in the USA is small in number; the majority is trained for 3 years in general

family practice, receiving only 1–2 months of cardiology training in the hospital setting. Thus, the physicians trained to perform PPEs with ECGs are very small in number and inadequately trained in cardiology. As there are more than 40 million athletes in the USA, many athletes remain in the care of their primary doctor, who is unlikely to have significant experience in interpreting ECGs. Some authorities feel that the physician workforce is the greatest barrier to implementation of ECG-based screening in the USA. A recent abstract presented at the AHA meeting in November 2009 illustrates the challenge [13]. Surveys conducted in five physician specialties that screen athletes showed that up to 60% of physicians would overread the ECG (false positive) and up to 20–30% may miss pathology. Large-scale physician education would be required. Given these numbers and the low incidence of SCD in athletes in the USA, it is doubtful that a well-designed outcomes study would demonstrate the superiority of ECG-based screening over PPE alone.

Summary

In summary, the goal of PPE cardiovascular screening in athletes is to detect underlying, potentially lethal heart disease. With focused examination and increased awareness of the cardiovascular needs of the athlete, the sensitivity of the PPE alone appears to be increasing. The role of ECG is evolving as an adjunct to the standard PPE, and although well-controlled prospective trials are lacking, the ECG does appear to increase the sensitivity of the PPE alone to detect underlying cardiac disease. Because many of the conditions that cause SCD in athletes demonstrate ECG findings similar to what is seen in normal athletic adaptation, clinicians should follow some simple rules for ECG interpretation in athletes, and need to be prepared for the consequences of both over- and underinterpretation of the ECG in athletes. Although ECG-based screening has gained wide acceptance in Europe, ECG-based cardiovascular screening of young athletes is currently not recommended in the USA. However, future studies designed specifically for the American athlete at all levels may assist in evolving this field in the USA.

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