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Cardiovascular Screening in College Athletes With and Without Electrocardiography

A Cross-sectional Study

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Background: Although cardiovascular screening is recommended for athletes before participating in sports, the role of 12-lead electrocardiography (ECG) remains uncertain. To date, no prospective data that compare screening with and without ECG have been available.

Objective: To compare the performance of preparticipation screening limited to medical history and physical examination with a strategy that integrates these with ECG.

Design: Cross-sectional comparison of screening strategies.

Setting: University Health Services, Harvard University, Cambridge, Massachusetts.

Participants: 510 collegiate athletes who received cardiovascular screening before athletic participation.

Measurements: Each participant had routine history and examination–limited screening and ECG. They received transthoracic echocardiography (TTE) to detect or exclude cardiac findings with relevance to sports participation. The performance of screening with history and examination only was compared with that of screening that integrated history, examination, and ECG.

Results: Cardiac abnormalities with relevance to sports participation risk were observed on TTE in 11 of 510 participants (prevalence, 2.2%). Screening with history and examination alone detected abnormalities in 5 of these 11 athletes (sensitivity, 45.5% [95% CI, 16.8% to 76.2%]; specificity, 94.4% [CI, 92.0% to 96.2%]). Electrocardiography detected 5 additional participants with cardiac abnormalities (for a total of 10 of 11 participants), thereby improving the overall sensitivity of screening to 90.9% (CI, 58.7% to 99.8%). However, including ECG reduced the specificity of screening to 82.7% (CI, 79.1% to 86.0%) and was associated with a false-positive rate of 16.9% (vs. 5.5% for screening with history and examination only).

Limitation: Definitive conclusions regarding the effect of ECG inclusion on sudden death rates cannot be made.

Conclusion: Adding ECG to medical history and physical examination improves the overall sensitivity of preparticipation cardiovascular screening in athletes. However, this strategy is associated with an increased rate of false-positive results when current ECG interpretation criteria are used.

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Occult cardiovascular disease is the leading cause of sudden death in young athletes (1). Consequently, all major professional medical organizations recommend preparticipation screening of athletes for underlying cardiac abnormalities (2–4). Although the mandate to screen is universal, the guidelines that delineate screening recommendations are not uniform. The American College of Cardiology and American Heart Association recommend limiting screening to a focused medical history and physical examination, whereas the European Society of Cardiology and the International Olympic Committee advocate including resting 12-lead electrocardiography (ECG). This important difference has generated considerable debate (5–7).

Outcomes from a multidecade Italian national study (8) demonstrate the positive effect of preparticipation screening and suggest an important role for ECG. Although these observational data have important limitations (9), they underscore the need for further study of proposed screening strategies (10–12). Data that define the performance of screening practices in the United States are sparse, and no studies have compared athlete screening by medical history and physical examination only with a strategy that includes ECG. For this reason, we examined the performance of preparticipation screening with history and examination only and compared it with an ECG-inclusive strategy in a large cohort of U.S. university athletes.

METHODS

Study Design

We conducted this study over 3 consecutive years (2006 to 2008). Athletes were eligible to participate if they were 18 years or older and were newly matriculated Har-

See also:

Print
Editors' Notes 270
Editorial comment 324
Related article 276
Summary for Patients I-13

Web-Only
Appendix Tables
Conversion of graphics into slides
Audio summary

Context

Estimates suggest that about 1 in 220 000 young athletes experience sudden cardiac death each year. The European Society of Cardiology and the International Olympic Committee recommend that pre–sport participation screening include electrocardiography (ECG), but screening in the United States typically does not include ECG.

Contribution

In this study of 510 student athletes, the addition of routine ECG to history and physical examination improved the detection of echocardiographically documented cardiac abnormalities from 5 to 10 out of 11 but increased the false-positive result rate from 5.5% to 16.9%.

Implication

The decision to include ECG in pre–sport participation screening must balance improved sensitivity with increased false-positive results, which can lead to further testing or unnecessary exclusion of healthy persons from athletic participation.

—The Editors

vard University students. Before participating in organized athletic activity, each study participant received standard screening with history and examination. Each participant then immediately underwent resting 12-lead ECG and transthoracic echocardiography (TTE). The practitioners who took the history and performed the examination were blinded to the results of ECG and TTE; likewise, the study staff who performed ECG and TTE were blinded to the history and examination results. Each participant provided written consent at enrollment. The institutional review boards of Partners Human Research Committee and Harvard University approved our study protocol.

Screening Medical History and Physical Examination

All participants underwent a standardized, noninvestigational medical history and physical examination based on current American College of Cardiology, American Heart Association, and National Collegiate Athletic Association recommendations (2) during student-athlete group screening sessions. These consisted of 5 personal medical history elements, 3 family history elements, and 4 physical examination elements (**Appendix Table 1**, available at www.annals.org). The examinations were performed by Harvard University Health Services practitioners with previous practical experience in performing sports clearance examinations.

Twelve-Lead ECG

Immediately after history taking and examination, ECG was performed by using standard 12-lead placement and equipment (MAC 5500, GE Healthcare, Milwaukee, Wisconsin). We adopted current European Society of Cardiology criteria for ECG abnormalities (**Appendix Table 2**, available at www.annals.org) because these are the only published recommendations designed for preparticipation screening (3). We examined ECG tracings for abnormalities at acquisition, and an independent observer who was blinded to the initial interpretation later confirmed all findings. We recorded the duration of each ECG study, defined as the time required for athlete positioning, lead placement, recording, and initial interpretation.

Focused TTE

We used a commercially available, portable system (Vivid-I, GE Healthcare) for all TTE studies. We performed a 17-image protocol that used standard transducer orientations and 2-dimensional imaging techniques. We made basic measurements on site, in accordance with current guidelines, and used Echo Pac, version 6.3 (GE Healthcare), to confirm them in postacquisition analysis (13).

Classification of TTE Findings

We placed all participants into 1 of 3 categories on the basis of TTE findings (**Appendix Table 3**, available at www.annals.org): normal findings, on the basis of current American Society of Echocardiography practice guidelines (13); mildly abnormal findings, consistent with the benign physiologic cardiac remodeling common in trained athletes (14–18); and abnormalities that were suggestive of or diagnostic for cardiac disease relevant to sports participation risk (19). We set the criteria for the third category to maximize sensitivity for abnormalities and thus included athletes with indeterminate cardiac morphology (marked hypertrophy or dilation), for whom further diagnostic evaluation was necessary to confirm or exclude abnormality. We also included athletes with valvular heart disease, for whom participation eligibility depends on the presence or absence of associated high-risk features.

We referred all athletes with possible abnormalities for further diagnostic testing, including comprehensive ECG, exercise testing, cardiac magnetic resonance imaging, cardiac catheterization, and prescribed detraining, at the discretion of nonstudy clinical staff. We integrated screening findings with the results of this diagnostic testing to confirm or exclude abnormalities that required restricting sports participation.

Statistical Analysis

We present continuous data as means (SDs). We assessed differences between means by using a 2-tailed t test or the Mann–Whitney test, as appropriate for data distribution. We calculated screening test statistics, including sensitivity, specificity, and negative and positive predictive values, by using 2×2 contingency tables based on the ability of each screening technique to identify athletes with pathologic abnormalities. We calculated 95% CIs by using exact Clopper–Pearson methods. We used SPSS Statistics, version 16.0 (SPSS, Chicago, Illinois), for all analyses and considered a P value less than 0.05 to be significant.

Role of the Funding Source

This study was not supported by extramural funding.

RESULTS

Study Population

Table 1 shows demographic characteristics of the participants. Compared with female athletes, male athletes (61%) were taller and heavier and had engaged in more exercise training during the 8 weeks before enrollment. Nearly all participants (99.4% [507 of 510]) reported previous participation in organized high school–level athletic competition. Most were white, and ethnic representation did not differ between the men and women.

Prevalence of Cardiac Abnormalities on TTE

The **Figure** shows TTE results. We excluded 2 participants from subsequent analyses because they had inadequate images for complete measurements. Of the 508 participants screened, 387 (76%) had structurally normal hearts and 110 (22%) had mildly abnormal findings consistent with physiologic remodeling. These findings included isolated left ventricular (LV) hypertrophy (56 participants), LV hypertrophy with LV dilation (22 participants), LV dilation with right ventricular dilation (25 participants), and isolated LV dilation (7 participants). We observed left atrial enlargement in 29 participants with concomitant LV remodeling (hypertrophy or dilation), but not as an isolated finding in any participant. We definitively identified coronary artery origin in 467 (92%) participants, and it was normal in all cases.

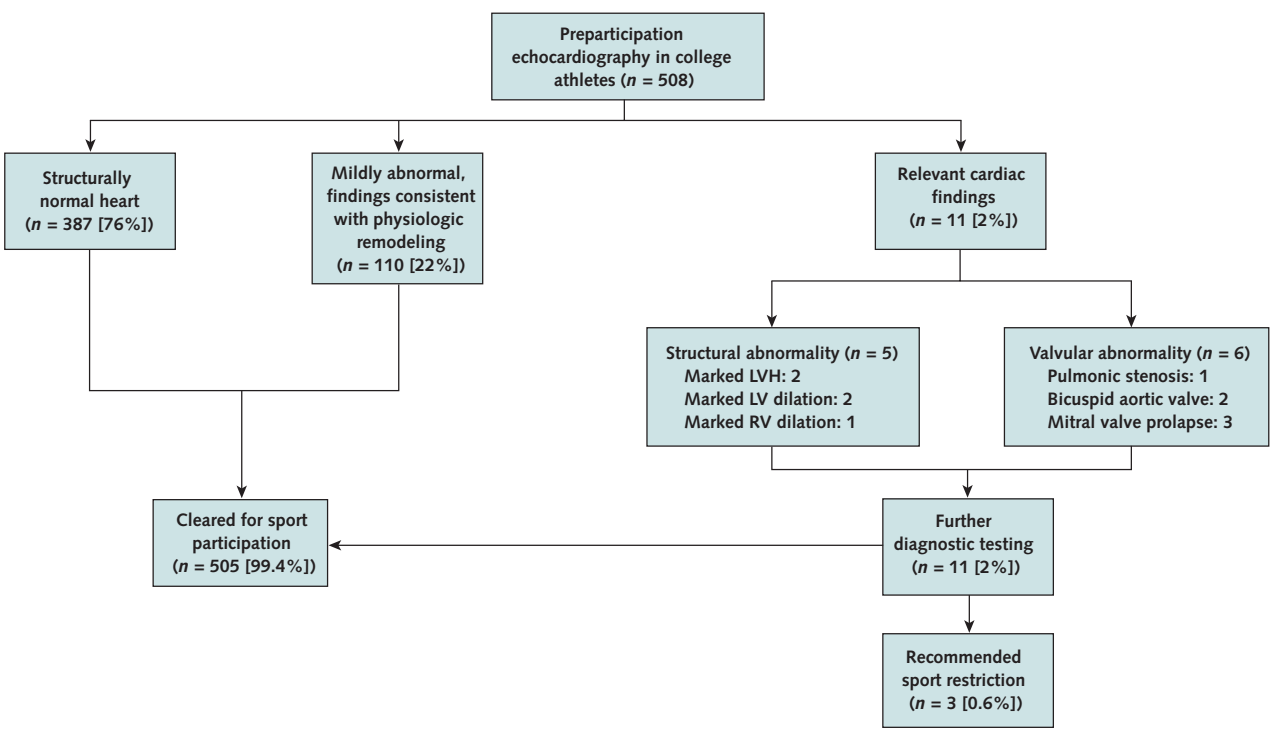
Table 1. **Participant Characteristics at Baseline**

Characteristic	Men (n = 311)	Women (n = 199)	Total
Mean age (SD), *y*	19.0 (0.6)	18.8 (0.4)	19.0 (0.3)
Height (SD), *m*	1.80 (0.15)	1.68 (0.09)*	1.76 (0.11)
Weight (SD), *kg*	82 (16)	59 (12)*	73 (18)
Ethnicity, %†			
White	66	72	68
Asian	10	14	12
Black	13	7	10
Hispanic or Latino	6	3	5
Other	5	4	5
Preseason training exposure			
Mean total training time (SD), *h/wk*	5.5 (2.4)	4.3 (2.6)*	5.1 (2.2)
Mean strength training time (SD), *h/wk*	3.1 (2.3)	1.1 (2.0)*	2.6 (2.3)
Mean aerobic training time (SD), *h/wk*	2.4 (3.1)	3.2 (4.1)	2.5 (3.0)

* $P < 0.050$ compared with men.
† Self-reported.

We found findings suggestive of or diagnostic for underlying abnormalities in 11 of 508 (2.2%) participants (**Figure**). Among these, 3 were ultimately found to have an abnormality that met current recommendations for permanent or temporary sports restriction, including pulmonic valve stenosis (peak gradient of 55 mm Hg

Figure. **Study flow diagram.**



LV = left ventricular; LVH = left ventricular hypertrophy; RV = right ventricular.

Table 2. **Findings During Preparticipation Medical History and Physical Examination Screening**

Finding	Positive Findings, *n* (%)
Personal medical history	
Exertional chest pain or discomfort	3 (0.6)
Unexplained syncope or near-syncope	6 (1.2)
Excessive and unexplained exertional dyspnea and fatigue	0
Previous recognition of a heart murmur	10 (2.0)
Elevated systemic blood pressure	2 (0.3)
Family medical history	
Premature (sudden and unexpected) death of ≥1 relative before age 50 y due to heart disease	0
Disability from heart disease in a close relative ≤50 y of age	0
Specific knowledge of hypertrophic or dilated cardiomyopathy, the long QT syndrome or other channelopathies, the Marfan syndrome, or clinically important arrhythmias in family members	0
Physical examination	
Heart murmur	10 (2.0)
Diminished or absent femoral pulse	0
Physical stigmata of the Marfan syndrome	0
Asymmetric or elevated brachial artery blood pressure*	2 (0.3)
Total	33 (6)

* Defined as absolute values >140 mm Hg (systolic) or >90 mm Hg (diastolic) or an upper-extremity difference in systolic blood pressure >10 mm Hg.

with right ventricular hypertrophy), hypertrophic cardiomyopathy (LV hypertrophy with septal and posterior wall thicknesses of 18 mm and no regression during detraining), and myocarditis (LV dilation, LV dysfunction with an ejection fraction of 0.35, and elevated serum troponin level after a recent viral illness). The overall prevalence of abnormality that required sports restriction was 0.6% (3 of 508). The remaining 8 athletes were all cleared for participation after noninvestigational diagnostic evaluation. Findings in this group included LV hypertrophy (wall thickness of 15 mm) in a highly trained rower, LV dilation (chamber diameter of 60 mm) in an elite-level rower, RV dilation (chamber diameter of 41 mm) in a long-distance runner, and valvular heart conditions (5 participants) with no high-risk features (such as clinically significant valvular incompetence or stenosis or aortic root dilation).

Performance of Current Standard of Care

Screening with medical history and physical examination took a mean of 8 minutes (SD, 6; range, 6 to 22 minutes) per athlete. Of the 510 participants screened, 33 (6%) fulfilled at least 1 criterion for abnormality (**Table 2**). History findings accounted for 21 (64%); previous recognition of a murmur (10 participants) and past unexplained syncope (6 participants) were the most commonly encountered issues. The remaining 12 participants with abnormal results were identified during physical examination; 10 participants had a murmur that was not clearly attributable to benign physiologic flow.

Table 3 shows findings detected by medical history or physical examination that suggested abnormality. History and examination correctly identified 5 of the 11 (45%) participants with such findings on TTE, all of whom had valvular heart disease. However, they failed to detect the other 6 (55%) such participants, including 1 with hypertrophic cardiomyopathy and 1 with myocarditis. Screening with history and examination only had a sensitivity of 45.5% (95% CI, 16.8% to 76.2%), a specificity of 94.4% (CI, 92.0% to 96.2%), a positive predictive value of 15.0% (CI, 5.1% to 31.9%), and a negative predictive value of 98.7% (CI, 97.3% to 99.5%).

Table 3. **Medical History, Physical Examination, and 12-Lead Electrocardiography Findings in Participants With Confirmed Abnormality**

Participant	TTE Abnormality	Findings		Final Diagnosis That Required Sport Participation Restriction
		Medical History or Physical Examination	Electrocardiography	
1	Bicuspid aortic valve	Murmur	None	None
2	Bicuspid aortic valve	Murmur and click	None	None
3	MVP	Murmur	None	None
4	MVP	Murmur	None	None
5	MVP	None	None	None
6	Pulmonic stenosis	Murmur	None	Moderate pulmonic stenosis
7	LV hypertrophy	None	QRS voltage, LAE	None
8	LV hypertrophy	None	QRS voltage, TWA*	Hypertrophic cardiomyopathy
9	LV dilation	None	LBBB	None
10	LV dilation	None	LBBB	Postviral myocarditis
11	RV dilation	None	RBBB	None

LAE = left atrial enlargement; LBBB = left bundle-branch block; LV= left ventricular; MVP = mitral valve prolapse; RBBB = right bundle-branch block; RV = right ventricular; TTE = transthoracic echocardiography; TWA = T-wave abnormality.
* Diffuse precordial lead T-wave inversions with ST-segment depression.

Performance of ECG-Integrated Screening

The mean duration of ECG acquisition was 3 minutes (SD, 2; range, 2 to 7 minutes). **Table 4** lists the ECG findings. Eighty-three of 510 (16%) participants fulfilled at least 1 criterion for ECG abnormality, with 48 (9%) having 1 abnormality and 35 (7%) having multiple abnormalities. Among the 83 participants with ECG abnormalities, 9 (11%) had structurally normal hearts, 72 (87%) had findings indicating physiologic remodeling, and 5 (6%) had findings that warranted further diagnostic evaluation.

Table 3 shows the ECG-integrated screening findings that suggested abnormality. This strategy detected abnormalities in 10 of the 11 athletes with TTE-detected abnormalities; the remaining participant had mitral valve prolapse and no associated high-risk features that necessitated sport restriction. Of note, the medical history, physical examination, and ECG-integrated screening strategy successfully identified all 3 participants with abnormalities that necessitated sport restrictions, whereas history and examination alone failed to detect 2 of these at-risk athletes. Integrated history, examination, and ECG screening had a sensitivity of 90.9% (CI, 58.7% to 99.8%), a specificity of 82.7% (CI, 79.1% to 86.0%), a positive predictive value of 10.4% (CI, 5.1% to 18.3%), and a negative predictive value of 99.8% (CI, 98.7% to 100.0%).

Table 4. **Findings on 12-Lead ECG During Preparticipation Screening**

Abnormality	Men (*n* = 311), *n* (%)	Women (*n* = 199), *n* (%)	Total Cohort (*n* = 510), *n* (%)
P wave			
Left atrial enlargement	18	8	26
Right atrial enlargement	3	2	5
QRS axis			
Right axis deviation >120°	1	0	1
Left axis deviation −30° to −90°	3	0	3
QRS voltage			
Precordial lead maximum >3 mV	29	8	37
Limb lead maximum >2 mV	22	5	27
QRS morphology			
Right bundle-branch block	10	3	13
Left bundle-branch block	2	0	2
Abnormal Q waves	0	0	0
R or R′ in V_1 >0.5 mV and R/S ≥1	1	0	1
ST segment and T wave			
T-wave inversions	3	0	3
ST-segment depression	0	0	0
Interval			
QTc interval prolongation	0	0	0
PR interval prolongation	0	0	0
PR interval shortening	0	0	0
Arrhythmia			
Premature ventricular complexes	0	0	0
Atrial fibrillation or flutter	0	0	0
Sinus bradycardia	0	0	0
Aggregate findings			
Total abnormalities	92	26	118
Participants with 1 ECG abnormality	32 (10)	16 (8)	48 (9)
Participants with >1 ECG abnormality	30 (10)	5 (3)	35 (7)
Total participants with abnormalities	62 (20)	21 (11)	83 (16)

ECG = electrocardiography; QTc = corrected QT interval.

DISCUSSION

To our knowledge, ours is the first prospective study to compare preparticipation screening by medical history and physical examination only with a strategy that integrates history, examination, and ECG in young competitive athletes. In a large, carefully phenotyped cohort of U.S. collegiate athletes, screening with history and examination alone correctly identified fewer than half of the athletes with potentially important cardiac findings, all of whom had valvular heart disease. Screening with history and examination alone also did not detect any of the athletes with structural abnormalities, including 2 athletes with cardiomyopathic conditions that necessitated sports restriction. Adding ECG to the history and examination improved the overall sensitivity and negative predictive value of athlete screening to 99.8% and led to the detection of all participants with abnormalities that required sport restriction. The ability of ECG to detect underlying myocardial abnormalities not found by history or physical examination largely explains this improved screening sensitivity. Our findings raise important concerns about the efficacy of screening with history and examination only, because cardiomyopathies account for most sports-related sudden cardiac death (1). Our data also suggest that 12-lead ECG and history and examination are complementary components of an overall screening program geared toward maximal sensitivity.

In 1982, the Italian government instituted and funded a national program that required mandatory preparticipation screening, including 12-lead ECG, for all athletes younger than 35 years (20). Recently published data from this ambitious program (8) suggest that this screening strategy has significantly reduced the incidence of sports-related sudden death. Although the Italian report has important limitations (9), most notably the uncontrolled, observational nature of the data, the report that an ECG-based screening program reduces sport-related sudden cardiac death justifies further study. Before this study, data that compared the performances of proposed screening practices were lacking. In another important study conducted before the development of current guidelines,

Maron and colleagues (21) performed personal and family medical histories, physical examinations, and 12-lead ECG on 501 university athletes and reported an overall false-positive result rate of 20%. Because only 18% of these participants were studied with definitive cardiac imaging (M-mode echocardiography), we could not quantify disease prevalence or comprehensively assess screening performance.

Current American College of Cardiology/American Heart Association guidelines do not endorse the use of 12-lead ECG during athlete preparticipation screening. This recommendation is based on speculative concerns about ECG performance (low specificity and high false-positive result rate) and the theoretical impracticalities of ECG-based screening (financial cost and technical logistics of interpretation). However, to our knowledge, no previous definitive study has applied a universal imaging standard, such as echocardiography, to compare the performance of an ECG-inclusive protocol with one limited to medical history and physical examination. Our study demonstrates that ECG inclusion leads to athlete screening with higher sensitivity and negative predictive value than history and examination alone.

Our study has limitations. First, we cannot draw definitive conclusions about the effect of the different screening strategies on the incidence of sudden death in athletes. Second, the relatively modest observed performance of screening with history and physical examination only may have been due to our screening practitioners not being cardiovascular specialists or dedicated sports medicine physicians. However, most athlete preparticipation screening in the United States is performed by generalists without subspecialty training, and thus our data represent the current standard of care. Third, some study participants had probably received screening before arriving at college, and our cohort may therefore underrepresent the true burden of occult cardiac disease. However, we still detected 11 participants with relevant cardiac abnormalities, of whom 3 were ultimately judged to require sport restriction. Fourth, because of inherent diagnostic limitations of echocardiography and our use of a practical but limited imaging protocol, we cannot exclude the possibility that we misclassified some participants. Finally, our study lacks the statistical power to address the performance of different screening approaches in important subgroups. Dedicated evaluation of screening technique performance with respect to sex and ethnicity is warranted.

Our results may not end the complicated debate about the role of 12-lead ECG in preparticipation screening. Although our data demonstrate that ECG improves the overall sensitivity and negative predictive value of screening, the associated decrease in positive predictive value and increase in false-positive results (16.9%) are formidable hurdles to the acceptance and implementation of this strategy. A screening program that falsely identifies approximately 1 in 6 participants as having cardiac disease has substantial and perhaps prohibitive financial, emotional, and logistical ramifications. However, screening strategies that maximize sensitivity have the greatest potential to minimize the incidence of sports-related sudden death. Our data suggest several important future directions for research to address this issue.

Most of the false-positive test results produced by the combination of medical history, physical examination, and ECG screening that we used were due to ECG findings. This is a direct function of the ECG abnormality criteria we used, which are accepted for widespread clinical use but were not derived from the study of athletes and therefore do not account for the numerous abnormal but benign ECG findings common in this population (22, 23). Our data demonstrate that certain ECG findings (such as increased QRS voltage in the absence of abnormal repolarization) are common among athletes who harbor no structural heart disease, whereas others (such as complete bundle-branch block) may be more specific markers of pathologic abnormalities. The current ECG abnormality criteria need to be revised or a novel algorithm developed with adequate accuracy to differentiate physiologic remodeling from pathologic heart disease in athletes. In addition, formal cost-effectiveness analyses and further longitudinal studies that document screening findings, sport restriction rates, and sudden death incidence are needed to determine the effect of ECG inclusion on health care costs and patient outcomes.

Electrocardiography has a controversial role in the screening of athletes for occult cardiovascular disease. Our results suggest that preparticipation screening limited to medical history and physical examination fails to identify a significant percentage of athletes with increased risk for adverse cardiac events. Adding ECG improves the overall sensitivity of screening and may be necessary to identify athletes who harbor the key diseases responsible for sports-related sudden death. However, using currently available ECG abnormality criteria is associated with high rates of false-positive screening results. Future efforts are needed to reduce the burden of false-positive results while maintaining the valuable sensitivity of ECG.

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References

1. **Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO.** Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009;119:1085-92. [PMID: 19221222]

2. **Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, et al; American Heart Association Council on Nutrition, Physical Activity, and Metabolism.** Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. Circulation. 2007;115:1643-455. [PMID: 17353433]

3. **Pelliccia A, Fagard R, Bjørnstad HH, Anastassakis A, Arbustini E, Assanelli D, et al; Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology.** Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports 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. Eur Heart J. 2005;26:1422-45. [PMID: 15923204]

4. **Bille K, Figueiras D, Schamasch P, Kappenberger L, Brenner JI, Meijboom FJ, et al.** Sudden cardiac death in athletes: the Lausanne Recommendations. Eur J Cardiovasc Prev Rehabil. 2006;13:859-75. [PMID: 17143117]

5. **Myerburg RJ, Vetter VL.** Electrocardiograms should be included in preparticipation screening of athletes. Circulation. 2007;116:2616-26; discussion 2626. [PMID: 18040041]

6. **Chaitman BR.** An electrocardiogram should not be included in routine preparticipation screening of young athletes. Circulation. 2007;116:2610-4; discussion 2615. [PMID: 18040040]

7. **Faber L, van Buuren F.** Athlete screening for occult cardiac disease: no risk, no fun? [Editorial]. J Am Coll Cardiol. 2008;51:1040-1. [PMID: 18325445]

8. **Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G.** Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. JAMA. 2006;296:1593-601. [PMID: 17018804]

9. **Thompson PD, Levine BD.** Protecting athletes from sudden cardiac death [Editorial]. JAMA. 2006;296:1648-50. [PMID: 17018808]

10. **Pelliccia A, Zipes DP, Maron BJ.** Bethesda Conference 36 and the European Society of Cardiology Consensus Recommendations revisited a comparison of U.S. and European criteria for eligibility and disqualification of competitive athletes with cardiovascular abnormalities. J Am Coll Cardiol. 2008;52:1990-6. [PMID: 19055990]

11. **Douglas PS.** Saving athletes' lives a reason to find common ground? J Am Coll Cardiol. 2008;52:1997-9. [PMID: 19055991]

12. **Thompson PD.** Preparticipation screening of competitive athletes: seeking simple solutions to a complex problem [Editorial]. Circulation. 2009;119:1072-4. [PMID: 19221215]

13. **Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al; Chamber Quantification Writing Group.** Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18:1440-63. [PMID: 16376782]

14. **Pelliccia A, Culasso F, Di Paolo FM, Maron BJ.** Physiologic left ventricular cavity dilatation in elite athletes. Ann Intern Med. 1999;130:23-31. [PMID: 9890846]

15. **Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P.** The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med. 1991;324:295-301. [PMID: 1824720]

16. **Pelliccia A, Maron BJ, Di Paolo FM, Biffi A, Quattrini FM, Pisicchio C, et al.** Prevalence and clinical significance of left atrial remodeling in competitive athletes. J Am Coll Cardiol. 2005;46:690-6. [PMID: 16098437]

17. **Scharhag J, Schneider G, Urhausen A, Rochette V, Kramann B, Kindermann W.** Athlete's heart: right and left ventricular mass and function in male endurance athletes and untrained individuals determined by magnetic resonance imaging. J Am Coll Cardiol. 2002;40:1856-63. [PMID: 12446071]

18. **Baggish AL, Wang F, Weiner RB, Elinoff JM, Tournoux F, Boland A, et al.** Training-specific changes in cardiac structure and function: a prospective and longitudinal assessment of competitive athletes. J Appl Physiol. 2008;104:1121-8. [PMID: 18096751]

19. **Maron BJ, Douglas PS, Graham TP, Nishimura RA, Thompson PD.** Task Force 1: preparticipation screening and diagnosis of cardiovascular disease in athletes. J Am Coll Cardiol. 2005;45:1322-6. [PMID: 15837281]

20. **Italian Ministry of Health.** Norme per la tutela sanitaria dell'ativita sportiva agonistica (rules concerning the medical protection of athletic activities). Gazetta Ufficiale. 1982;18 February:63.

21. **Maron BJ, Bodison SA, Wesley YE, Tucker E, Green KJ.** Results of screening a large group of intercollegiate competitive athletes for cardiovascular disease. J Am Coll Cardiol. 1987;10:1214-21. [PMID: 2960727]

22. **Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, et al.** Clinical significance of abnormal electrocardiographic patterns in trained athletes. Circulation. 2000;102:278-84. [PMID: 10899089]

23. **Choo JK, Abernethy WB 3rd, Hutter AM Jr.** Electrocardiographic observations in professional football players. Am J Cardiol. 2002;90:198-200. [PMID: 12106861]

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24. **Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, et al; 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.** 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. Eur Heart J. 2005; 26:516-24. [PMID: 15689345]

Appendix Table 1. Criteria for Abnormality on Preparticipation Medical History and Physical Examination Screening, as Proposed by the American Heart Association*

Medical history
- Personal history
  - Exertional chest pain or discomfort
  - Unexplained syncope or near-syncope not clearly attributable to neurocardiogenic or vasovagal mechanism
  - Excessive and unexplained dyspnea or fatigue associated with exercise
  - Previous recognition of a heart murmur
  - Elevated systemic blood pressure
- Family history
  - Premature (sudden and unexpected) death of ≥1 relative before age 50 y due to suspected or confirmed heart disease
  - Disability from heart disease in a close relative ≤50 y of age
  - Knowledge of hypertrophic or dilated cardiomyopathy, the long QT syndrome, the Marfan syndrome, or clinically important arrhythmias in any family member

Physical examination
- Heart murmur
- Diminished or asymmetric femoral pulses (to exclude aortic coarctation)
- Physical stigmata of the Marfan syndrome
- Asymmetric or elevated (>140/90 mm Hg) brachial artery blood pressure

* See reference 2.

Appendix Table 2. Criteria for Abnormality on Preparticipation 12-Lead Electrocardiography Screening, as Proposed by the European Society of Cardiology*

Atrial abnormalities
- Left atrial enlargement: negative portion of the P wave in lead V_1 ≥0.1 mV in depth and ≥0.04 s in duration
- Right atrial enlargement: peaked P wave in leads II and III or V_1 ≥0.25 mV in amplitude

QRS complex abnormalities
- Frontal plane axis deviation: right ≥120° or left between −30° and −90°
- Increased voltage: Amplitude of R or S wave in a limb 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 ≥25% of the height of the ensuing R wave, or QS pattern in ≥2 leads
- Morphology: right or left bundle-branch block configuration with QRS duration ≥0.12 s; R or R′ wave in lead V_1 ≥0.5 mV in amplitude and R/S ratio ≥1

Interval abnormalities
- QT interval: prolongation of heart rate–corrected QT interval ≥0.44 s in men and ≥0.46 s in women
- PR interval: first-degree (PR interval ≥0.21 s and no shortening with hyperventilation), second-degree, or third-degree atrioventricular block or short PR interval (≥0.12 s), with or without delta wave

Other abnormalities
- Repolarization: ST-segment depression or T-wave flattening or inversion in ≥2 leads
- Arrhythmia: Premature ventricular beats or more severe ventricular arrhythmia, supraventricular tachycardia, atrial flutter, atrial fibrillation, or profound sinus bradycardia not increasing to >100 beats/min with exertion

* See reference 24.

Appendix Table 3. Reference Values for Transthoracic Echocardiography Findings

Finding	Reference Values		Associated Abnormality
	Men	Women	
Normal echocardiography readings			
Normal LV wall thickness	LV wall thickness <11 mm	LV wall thickness <10 mm	–
Normal LV cavity size	LV internal diastolic diameter <60 mm	LV internal diastolic diameter <54 mm	–
Normal RV cavity size	RV internal diastolic diameter <34 mm	RV internal diastolic diameter <34 mm	–
Normal LA size	Maximal anterior–posterior diameter <41 mm	Maximal anterior–posterior diameter <39 mm	–
Mild abnormality consistent with physiologic remodeling			
Mild LV hypertrophy	LV wall thickness, 11–13 mm	LV wall thickness, 10–12 mm	–
Mild LV dilation	LV internal diastolic diameter, 60–63 mm	LV internal diastolic diameter, 54–57 mm	–
Mild RV dilation	RV internal diastolic diameter, 34–37 mm	RV internal diastolic diameter, 34–37 mm	–
LA enlargement	Maximal anterior–posterior diameter >41 mm	Maximal anterior–posterior diameter >39 mm	–
Abnormalities that suggest pathology			
Marked LV hypertrophy	LV wall thickness ≥14 mm	LV wall thickness ≥13 mm	Hypertrophic CMP
Marked LV dilation	LV internal diastolic diameter ≥64 mm	LV internal diastolic diameter ≥58 mm	Dilated CMP or myocarditis
Marked RV dilation	RV internal diastolic diameter ≥38 mm	RV internal diastolic diameter ≥38 mm	Arrhythmogenic RV CMP
Valvular pathology			
Bicuspid aortic valve	–	–	–
Mitral valve prolapse	–	–	–
Anomalous coronary origin	–	–	–

CMP = cardiomyopathy; LA = left atrial; LV = left ventricular; RV = right ventricular.