Original Research

Eighteen Years' Experience Applying Old and Current Strategies in the Pre-Participation Cardiovascular Screening of Athletes

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Key words: **Athletes, electrocardiography, echocardiography, cardiovascular problems.**

Introduction: Cardiovascular pre-participation screening (PPS) is recommended for the identification of athletes at risk for sudden cardiac death. However, there is currently no universally accepted screening protocol.

Methods: Two distinct PPS strategies were studied in a large cohort of Greek athletes (5 to 39 years old): PPS I, with routine 12-lead ECG and echo, in addition to personal and family history, and physical examination; and PPS II, without routine echo. PPS I (12,353 athletes) was performed from 1992 to 2002, and PPS II (9852 athletes) from 2003 to 2010.

Results: "Abnormal" findings were observed in 49.3% of the athletes (49.6% in PPS I and 48.9% in PPS II, p=0.299). Most of them were age- or exercise-related. Further evaluation was recommended for 8.3% of the athletes. Finally, 39 athletes (22 from PPS I) were excluded from competitive sports. Hypertrophic cardiomyopathy was found in 7 athletes. Other abnormalities were: dilated cardiomyopathy; complete heart block; coronary artery disease; Wolf-Parkinson-White syndrome; and severe hypertension. The ECG played a critical role in the exclusion of 13 athletes, compared to only one for echo. Both PPS methods revealed an almost equal incidence of findings.

Conclusions: We suggest that the routine use of ECG alone is sufficient for the successful screening of athletes.

Manuscript received: June 11, 2012;
Accepted: December 10, 2012.

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Cardiovascular disorders are the leading cause of sudden cardiac death (SCD) in young competitive athletes.[1,2] In high school and college athletes, the rate of SCD ranged from 1:3500 to 1:300,000 in the USA, while in Italy 0.4 per 100,000 athletes annually died suddenly.[3,4] The mortality rate of the Italian athletic population was reduced by 89% following the implementation of a national pre-participation screening (PPS) program.[3] Some of these deaths are not predictable or preventable in asymptomatic athletes by the use of common screening tests. In many cases of SCD there are prodromal symptoms, clinical, ECG or echocardiographic abnormalities, as well as a family history of SCD at a young age.[5] Thus, accurate cardiovascular non-invasive assessments before competition may be the most important step towards improving the outcome for athletes at risk.[1,6] So far, specific efficient and cost-effective PPS strategies, which have been accepted for implementation in all European countries, have not yet been assessed. Moreover, it has not been determined whether genetic and/or environmental factors may affect the distribution of causes of SCD in the different European countries.

The Study Group of Sports Cardiology of the European Society of Cardiology (ESC) recommended a common European screening protocol in competitive athletes, essentially based on the 12-lead ECG in addition to history and physical examination.[1,7] These recommendations were derived from the over 30-year Italian experience of systemic PPS of competitive athletes.[4] In the US, the American Heart Association expert consensus panel recommended a PPS program limited to the personal and family history and physical examination, while the ECG was excluded, largely based on cost-efficacy considerations.[8] Additional testing, such as echocardiography, requires enormous financial support and raises a number of criticisms, since it is considered to have limited diagnostic accuracy and efficiency in many cardiac disorders and is also time-consuming.[8] Similarly, Magalski et al recently suggested that the incremental value of echocardiography was negligible in the PPS of competitive collegiate athletes.[9] Therefore, data from large-scale studies performed in different ethnicities with distinct protocols are interesting.

The aim of this study was to report the "abnormal" cardiovascular findings from two distinct PPS strategies applied to a large cohort of competitive Greek athletes during an 18-year period. In addition, an attempt was made to evaluate the performance of PPS that included ECG and echo, as well as PPS without routine echo screening. The hypothesis that an additional echocardiographic study would add value and efficiency to athletes' cardiovascular pre-participation screening was tested.

Methods

Study population

From 1992 to 2010, 22,205 competitive athletes (13,706 males), aged 5 to 39 years, from Northern Greece, were examined for the mandatory confirmation of their yearly sports-related health identity card in the Laboratory of Sports Medicine of the Aristotle University of Thessaloniki, which is an authorised sports medicine centre. All athletes competed at the regional and national level, had more than 6 months of training experience, and exercised at least 3 times per week with ≥6 h of weekly training workload. Most of the participants (96%) were amateur athletes and participated in 39 different sports (all summer Olympic sport disciplines were included), while the rest were professional athletes, mainly soccer players. The athletes were divided into two groups according to the cardiovascular PPS period. Specifically, 12,353 athletes (7563 males) were examined over 10 consecutive years (1992 to 2002) receiving routine echocardiography as part of the screenings (PPS I). From 2003 (according to the new relative ESC guidelines at that time) to 2010, 9852 athletes (5983 males) were examined and underwent echocardiography only electively, when indicated (PPS II). To examine the impact of age on the presence of abnormal findings, we classified the athletes of both PPS studies into 3 subgroups: a: 5-11 years old, b: 12-35 years and c: 36-39 years, as indicated in other studies.[10,11] No exclusion criteria were applied in any athlete.

The study complied with the ethical standards of the Aristotle University ethics committee and with the Helsinki Declaration of 1975, as revised in 2000. All subjects gave informed consent.

Procedures

The screenings were performed by cardiologists who had medical skills and a scientific background in sports cardiology. All individuals were asked to fill in the same medical history questionnaire under the supervision of an experienced clinician, so that information about the medical records of the athletes and their families could be collected. For children under the age of ten, the parents were asked to fill in the questionnaires. Additionally, all athletes underwent a standardised physical examination, including precordial auscultation in both the supine and standing positions. Murmurs were further evaluated with the Valsalva manoeuvre. Blood pressure was measured according to the guidelines using the standard mercury cuff sphygmomanometer. Severe hypertension was defined as a sustained systolic blood pressure ≥180 mmHg and/or diastolic ≥110 mmHg.[12]

Electrocardiography

A resting ECG was recorded from all athletes using standard 12-lead placement and equipment (Cardiette Excel 106, Trendo, Italy). For each PPS strategy we adopted the recognised ECG abnormalities for "athlete's heart" at that time.[10,13] However, all ECG data are reported after re-classification according to the 2010 ESC criteria.[7] Based on these criteria, ECG findings were classified into common/training-related and uncommon/training-unrelated ECG abnormalities. The cardiologists who re-evaluated the ECGs were blinded to the previous reports.

Echocardiography

Routine M-mode and 2-D echo studies were performed in all athletes of PPS I, while in PPS II echo was only used in athletes who had abnormal findings from the clinical examination and ECG. Specifically, the indication criteria for the echo Doppler study in PPS II were: a. exertional symptoms or history of syncope or near-syncope; b. positive family history (i.e. SCD <35 y old); c. systolic murmur ≥2/6 or diastolic murmur; d. arterial hypertension (≥140/90 mmHg on >1 reading); e. signs of Marfan syndrome (i.e. arachnodactylia); f. uncommon/training-unrelated ECG abnormalities.

The echocardiographic study was carried out using a Sigma 44HVCD echocardiograph (Kontron Instruments). All standard echocardiographic views were recorded, according to recommended methods.[14] The echocardiographic study was performed with the athlete in the left lateral semi-erect position at 30° from the horizontal plane. During the measurements the chest was kept in a mid-expiratory phase in order to minimise the effects of breathing. The transducer (2.5/3.5 MHz) was placed in the left parasternal intercostal space in such a position as to give the best quality images. Echocardiographic images were recorded on videotape for later analysis by cardiologists who were blinded to the clinical and ECG results. The echo findings were classified into three categories: (i) normal findings; (ii) mildly abnormal findings, mainly consistent with exercise training adaptations; (iii) marked abnormalities that were suggestive of or diagnostic for cardiac problems relevant to sports participation risk.[15] Additional testing, including 24-h ECG or blood pressure Holter monitoring, exercise testing, magnetic resonance imaging, or other examinations such as genetic analysis, was performed individually when indicated. Until the end of the evaluation period the referred athletes were asked to refrain from exercise training. Athletes with a diagnosis of cardiovascular disease were managed according to the AHA and ESC recommendations.[1,6,8]

Statistical analysis

Continuous variables are expressed as mean ± SD. Categorical variables are presented as frequencies or percentages, and their analysis was based on the theory of log-linear models, aiming to discover how the findings for each measured factor were related to PPS studies and subgroups.[16] In the case of two-way contingency tables, either Fisher's Exact test ($pn \leq 5$) or the z test for proportions was used.[16] The analysis was performed using SPSS 16.0 software. P-values <0.05 (two-sided) were considered significant.

Results

Overall, 49.3% of the athletes (64.1% of all the males, 23.1% of the females, $p<0.001$) presented some "abnormal" cardiovascular findings; 49.6% (66.3% of the males, 23.2% of the females, $p<0.001$) in PPS I and 48.9% (65.6% of the males, 23% of the females, $p<0.001$) in PPS II (PPS I vs. PPS II $p=0.299$).

The clinical data of the studied population are presented in Table 1. Five athletes (3 in PPS I) were found to have severe arterial hypertension and were disqualified from competitive sports. Interestingly, 4.3% of the athletes with elevated blood pressure were considered as "white-coat" hypertensives, based on the recommendations.[17]

The abnormalities in the athletes' ECGs are presented in Table 2. There was no statistically significant difference in the ECG abnormalities between PPS I and PPS II. Common and training-related ECG findings were found in 31.6% of athletes in PPS I and 30.9% in PPS II (p:NS). Moreover, in the whole sample there was a statistically significant 2-way interaction for sinus bradycardia ($\chi^2(2)=170.718$, $p<0.001$), first-degree atrioventricular block ($\chi^2(2)=66.708$, $p<0.001$), incomplete right bundle branch block ($\chi^2(2)=6.989$, $p=0.030$), early repolarisation alterations ($\chi^2(2)=326.422$, $p<0.001$), and increased QRS voltage ($\chi^2(2)=55.955$, $p<0.001$), showing that findings were dependent only on subgroup totals. Thus, more athletes from subgroups b and c had these common ECG changes compared to younger athletes (subgroup a). In addition, 8.3% of the athletes had abnormal or training-unrelated ECG findings (8.0% in PPS I vs. 8.6% in PPS II, p:NS) and were referred for further evaluation and treatment.

The incidence of abnormal echo findings in the two PPS strategies is shown in Figure 1. In particular, only 84 athletes (0.68%) in PPS I had marked echocardiographic findings diagnostic of a cardiac disease relevant to sports participation risk, such as distinct myocardial hypertrophy, left ventricular dilatation, valve regurgitation, or atrial and ventricular septal defects. Similarly, in PPS II, 68 of the 9852 athletes (0.69%) had such findings.

From the routine echo study in PPS I, "grey

Table 1. Clinical data and summary results of the two studies. Data are expressed as number (n) of athletes unless otherwise indicated.

		PPS Study I				PPS Study II				Results
		Total (n=12353)	Subgroup a (n=3855)	Subgroup b (n=7833)	Subgroup c (n=665)	Total (n=9852)	Subgroup a (n=2950)	Subgroup b (n=6410)	Subgroup c (n=492)	
Sex, M/F		7563/4790	2324/1531	4753/3081	486/179	5983/3869	1770/1180	3850/2560	351/141	-
Age, mean ± SD		21.8 ± 10.3	8.3± 2.4	20.4 ± 8.1	37.5 ± 1.3	22.6 ± 11.1	8.9 ± 2.6	21.7 ± 7.3	36.2 ± 2.4	-
Years of training, mean ± SD		16.3 ± 9.6	2.1 ± 0.8	12.2 ± 4.3	27.9 ± 9.4	16.9 ± 8.7	2.4 ± 1.1	12.9 ± 5.1	26.8 ± 8.6	a*
Positive family history		2	-	2		-	-	-	-	-
Palpitations, n (%)	at rest	754 ± 6.1	201 ± 5.2	533 ± 6.8	20 ± 3.0	650 ± 6.6	174 ± 5.9	429 ± 6.7	47 ± 9.6	b*
	on exertion	105 ± 0.8	34 ± 0.9	64 ± 0.8	7 ± 1.1	89 ± 0.9	27 ± 0.9	56 ± 0.9	6 ± 1.2	NS
Dizziness, n (%)	at rest	161 ± 1.3	30 ± 0.8	118 ± 1.5	13 ± 2.0	108 ± 1.1	18 ± 0.6	83 ± 1.3	7 ± 1.4	a*
	on exertion	32 ± 0.3	9 ± 0.2	22 ± 0.3	1 ± 0.2	18 ± 0.2	4 ± 0.1	13 ± 0.2	1 ± 0.2	NS
Thoracic pain, n (%)	at rest	482 ± 3.9	127 ± 3.3	337 ± 4.3	18 ± 2.7	335 ± 3.4	106 ± 3.6	224 ± 3.5	25 ± 5.1	b*
	on exertion	18 ± 0.1	5 ± 0.1	12 ± 0.2	1 ± 0.2	11 ± 0.1	4 ± 0.1	6 ± 0.1	1 ± 0.2	NS
Dyspnoea, n (%)	at rest	100 ± 0.8	15 ± 0.4	74 ± 0.9	11 ± 1.7	73 ± 0.7	16 ± 0.5	50 ± 0.8	7 ± 1.4	a*
	on exertion	28 ± 0.2	8 ± 0.2	19 ± 0.2	1 ± 0.2	16 ± 0.2	4 ± 0.1	12 ± 0.2	-	NS
Soft/mild systolic murmur, n (%)		1717 ± 13.9	613 ± 15.9	1046 ± 13.4	58 ± 8.7	1429 ± 14.5	532 ± 18.0	850 ± 13.3	47 ± 9.6	a*
Prominent systolic/ diastolic murmur, n (%)		98 ± 0.8	58 ± 1.5	38 ± 0.5	2 ± 0.3	73 ± 0.7	44 ± 1.5	28 ± 0.4	1 ± 0.2	a*
Syncope or near-syncope		3	-	3		2	-	1	1	-
Elevated blood pressure (≥140/90 mmHg), n (%)		101 ± 0.8	19 ± 0.5	69 ± 0.9	13 ± 2.0	72 ± 0.7	13 ± 0.4	50 ± 0.8	9 ± 1.8	a*

a: Statistically significant two way interaction between Subgroup and findings, showing difference in total findings between the 3 subgroups.
b: Statistically significant three way interaction (PPS×Subgroup×findings), showing that findings were depended on Subgroup and PPS study ($p<0.001$).
NS: non-significant k-way interactions, for k=2,3; *$p<0.001$.

Table 2. "Abnormal" ECG findings. Data are expressed as number (n) of athletes.

	PPS Study I (n=12353)								PPS Study II (n=9852)								Results
	Total		Subgroups a		b		c		Total		Subgroups a		b		c		
Common and training-related ECG findings:																	
Incomplete RBBB, n (%)	1248	(10.1)	386	(10.0)	783	(10.0)	79	(11.9)	1074	(10.9)	295	(10.0)	712	(11.1)	67	(13.6)	c†
Right axis deviation, n (%)	2248	(18.2)	671	(17.4)	1504	(19.2)	73	(11.0)	1823	(18.5)	531	(18)	1237	(19.3)	55	(11.2)	c*
Premature arrhythmias, n (%) atrial	692	(5.6)	150	(3.9)	501	(6.4)	41	(6.2)	601	(6.1)	144	(4.9)	429	(6.7)	28	(5.7)	c*
ventricular	383	(3.1)	58	(1.5)	305	(3.9)	20	(3.0)	296	(3.0)	38	(1.3)	231	(3.6)	27	(5.5)	c*
First degree AV block, n (%)	1519	(12.3)	127	(3.3)	1261	(16.1)	131	(19.7)	1123	(11.4)	16	(0.5)	1019	(15.9)	88	(17.9)	c*, b*
Mobitz I AV block, n (%)	202	(1.6)	39	(1.0)	149	(1.9)	14	(2.1)	158	(1.6)	27	(0.9)	109	(1.7)	22	(4.5)	a†
Sinus bradycardia (>30 bpm and/or pauses <2s), n (%)	3496	(28.3)	645	(16.7)	2624	(33.5)	227	(34.1)	2877	(29.2)	519	(17.6)	2186	(34.1)	172	(35.0)	c*
Increased QRS voltage, n (%)	2026	(16.4)	68	(1.8)	1810	(23.1)	148	(22.3)	1665	(16.9)	44	(1.5)	1513	(23.6)	108	(22.0)	c*
Early repolarisation alterations, n (%)	1314	(10.6)	204	(5.3)	1010	(12.9)	100	(15.0)	1096	(11.1)	172	(5.8)	846	(13.2)	78	(15.9)	c*
Uncommon and training-unrelated ECG findings:																	
RBBB, n (%)	778	(6.3)	231	(6.0)	501	(6.4)	46	(6.9)	678	(6.9)	201	(6.8)	449	(7.0)	28	(5.7)	NS
LBBB	2		-		-		2		-		-		-		-		
Tachyarrhythmias (VT, AF)	2		-		1		1		2		-		1		1		
Sinus bradycardia (≤30 bpm and/or pauses ≥2s), n (%)	408	(3.3)	42	(1.1)	329	(4.2)	37	(5.6)	355	(3.6)	44	(1.5)	282	(4.4)	29	(5.9)	c*
Mobitz II AV block	26		6		16		4		15		3		9		3		
Third degree AV block	5		1		3		1		2		-		2		-		
Short QT interval	10		3		6		1		7		2		4		1		
Long QT interval	1		-		1		-		-		-		-		-		
Q waves	2		-		1		1		-		-		-		-		
T-wave inversion (≥2 leads), n (%)	1594	(12.9)	204	(5.3)	1144	(14.63)	246	(37.0)	1192	(12.1)	145	(4.9)	968	(15.1)	79	(16.1)	a*, b†
WPW	8		3		5		-		5		2		3		-		

AF – atrial fibrillation; AV – atrioventricular; LBBB – left bundle branch block; RBBB – right bundle branch block; VT – ventricular tachycardia; WPW – Wolff-Parkinson-White syndrome.
P-values are from z test or Fisher's Exact test (p n≤5). *p<0.001, †p<0.05, NS: non-significant k-way interactions, for k=2,3
a: statistically significant three-way interaction (PPS Subgroup findings), showing that findings were depended on Subgroup and PPS study.
b: statistically significant two-way interaction between PPS and findings, showing difference in total findings between PPS I and PPS II.
c: statistically significant two way interaction between Subgroup and findings, showing difference in total findings between the 3 Subgroups.

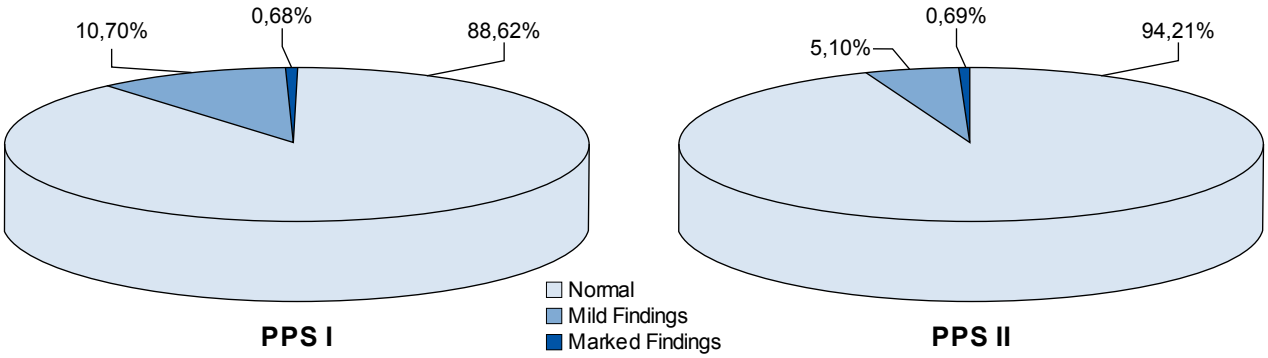

Figure 1. Incidence of abnormal echo findings in PPS I (echo as routine testing) and PPS II (echo as referred testing).

zone" hypertrophy (septal thickness between 13 to 16 mm) was found in 8 (0.07%) athletes (4 from subgroup b and 4 from subgroup c). In one of them, with deep T-wave inversion in the precordial leads and a positive family history, hypertrophic cardiomyopathy (HCM) was detected. HCM was also diagnosed in another 3 athletes, whose septal thickness was estimated at 24, 26 and 24 mm. Additionally, in 14 athletes (0.12%) of PPS I, the left ventricular end-diastolic diameter was detected to be ≥60 mm. Nine of these cases were in subgroup b and 5 in subgroup c. Three of these athletes, with a low ejection fraction and deep

Table 3. Clinical, electrocardiographic and echocardiographic data of the 39 athletes who were excluded from competitive sports.

PPS I	Screening method(s)					PPS II	Screening method(s)				
	PH	FH	PE	ECG	Echo		PH	FH	PE	ECG	Echo
1. HCM					+	1. HCM				+	+
2. HCM	+	+		+	+	2. HCM	+	+	+	+	+
3. HCM	+		+	+	+	3. HCM			+	+	+
4. HCM			+	+	+	4. DCM	+		+	+	+
5. DCM	+			+	+	5. DCM	+			+	+
6. DCM	+		+	+	+	6. CAD	+			+	
7. CAD	+					7. CAD	+				
8. CAD	+					8. CAD	+				
9. AR			+	+	+	9. CPVT	+			+	
10. AR	+		+	+	+	10. AR			+	+	+
11. MR			+		+	11. AR			+		+
12. MR	+		+		+	12. MR			+		+
13. Complete heart block	+			+		13. Complete heart block	+			+	
14. Complete heart block				+		14. VSD			+		+
15. LQTS	+			+		15. WPW	+		+	+	
16. MVP with arrhythmias			+	+	+	16. Severe AH			+		
17. WPW with arrhythmias	+			+		17. Severe AH			+		+
18. WPW with arrhythmias				+							
19. Severe AH			+	+							
20. Severe AH			+								
21. Severe AH			+								
22. Non cardiac reason											

AH – arterial hypertension; AR – aortic regurgitation; CAD – coronary artery disease; CPVT – catecholaminergic polymorphic ventricular tachycardia; DCM – dilated cardiomyopathy; FH – family history; HCM – hypertrophic cardiomyopathy; LQTS – long-QT syndrome; MVP – mitral valve prolapse; PE – physical examination; PH – personal history; VSD – ventricular septal defect; WPW – Wolff-Parkinson-White syndrome.

T-wave inversion in the precordial leads, had a history of myocarditis and were excluded from competitive sports.

From PPS II, 1045 athletes (10.6%) were referred for an echo study according to the indication criteria. "Grey zone" hypertrophy was found in 7 (0.08%) athletes. Two of them had a loud apical systolic murmur, deep negative T waves and markedly increased voltage in leads V_5 and V_6. They were both diagnosed with HCM. Additionally, one more athlete with a positive family history, abnormal ECG, and septal thickness >28 mm, was diagnosed with HCM. In another 10 cases (0.10%) the left ventricular end-diastolic diameter was found to be ≥60 mm. Two of these, who had a low ejection fraction, severe arrhythmias and a history of myocarditis, were disqualified from competitive sports.

Finally, 39 athletes (26 males) were disqualified from any competitive sport activity; 22 (0.18%) from PPS I and 17 (0.17%) from PPS II (p:NS; Table 3). In particular, 2 athletes from PPS I subgroup a, 10 from subgroup b, and 10 from subgroup c were excluded; for PPS II, 1 athlete in subgroup a, 10 in subgroup b, and 6 athletes in subgroup c were judged to be at high risk. One of them was excluded because of a non-cardiac reason. Training-unrelated ECG abnormalities helped the screening process for the detection of an underlying heart disease with high risk for SCD in 8 athletes from PPS I and 5 from PPS II (NS). In contrast, abnormal echocardiographic findings were only a diagnostic criterion in 1 athlete with HCM from PPS I.

Discussion

The present study demonstrated that almost half of 22,205 Greek competitive athletes had "abnormal" cardiovascular findings during an 18-year period of pre-participation screening. However, the incidence of uncommon and training-unrelated ECG findings, which potentially reflect a cardiac disease with a risk for SCD, was low. The efficiency of the two PPS programs to detect or exclude cardiovascular findings with relevance to sports participation was equal. Importantly, the routine ECG was an effective tool in detecting high-risk cardiac disorders in 13 athletes. In contrast, the routine use of an echo study was the critical tool for sports disqualification in only one case.

In our study, 0.76% of the athletes were found to have elevated blood pressure, while 4.3% of them were considered to present "white coat" syndrome.[12,17] Five athletes with severe uncontrolled hypertension were excluded from competitive sports. The abuse of performance-enhancing substances may in part be responsible for the significant increase in blood pressure in some athletes;[18] however, this was not tested in the present study.

The high incidence of "abnormal" ECG findings in our study was similar to those of earlier reports in young athletes.[10,11,19] Importantly, most "abnormal" ECGs in both PPS studies (I and II) reflected only common and training-related ECG changes. Specifically, the most common electrocardiographic findings were sinus bradycardia, early repolarisation abnormalities, right frontal plane axis deviation, first degree atrioventricular block, incomplete right bundle branch block, increased QRS voltage, and premature atrial and ventricular contractions. These alterations have been proved to be indicative of exercise training-induced "physiological" cardiac adaptations.[19,20] The higher prevalence of training-related ECGs in our older athletes supports the suggestion that ECG alterations in athletes are likely to be an innocent consequence of long-term, intense physical training.[7,21,22] Pelliccia et al reported that athletes engaged in certain endurance sports, such as cycling, rowing/canoeing, and cross-country skiing, demonstrated the largest left ventricular dimensions on echocardiographic study, and that these athletes most often had "abnormal" ECG patterns.[23] No athlete from our study with common and training-related electrocardiographic findings was excluded from competitive sports, except one athlete with a congenital artery anomaly, who experienced thoracic pain on exertion and minor repolarisation abnormalities on the ECG. Uncommon training-unrelated ECG findings were observed in 8.3% of our athletes, leading them to further evaluation. Similar rates of uncommon ECG findings, possibly associated with cardiac problems, that required further testing were also reported in previous studies.[19,24] Regarding the clinical outcome of the ECG repolarisation pattern, it has been suggested that T-wave inversions in leads V_1-V_4 represent an ethnic variant of "athlete's heart", while T-wave inversions in the later leads require further additional testing.[25] Furthermore, most athletes who were disqualified from any competitive sports activity reflected distinctly abnormal ECGs that helped the screening process for the detection of an underlying cardiac disease, such as a long QT interval, polymorphic ventricular tachycardia, abnormal Q waves, distinctly increased R voltage, delta waves, and deep precordial T-wave inversion. As mentioned above, of the

7 athletes with HCM, 6 exhibited pathological ECG findings in addition to the positive clinical findings.

It has been reported that, among elite competitive athletes, the resting ECG has only 51% sensitivity and 61% specificity for detecting cardiac abnormalities.[26] Recently, a consensus panel developed ECG criteria for athletes, which are expected to increase the ECG specificity (to ~70%) and to restrict secondary testing (to ~9%) of the screened population.[7] Baggish et al claimed that adding the ECG to PPS improved the detection of cardiac abnormalities, but also increased the false-positive result rate.[15] However, it was recently reported that the 12-lead ECG is the most cost-effective pre-participation cardiovascular modality in comparison to medical history and physical examination, as well as to 2D echocardiography.[7,26,27]

It is noteworthy that in PPS I all athletes were examined using echocardiography. However, the echo study confirmed the clinical diagnosis of marked abnormalities in only a limited number of athletes (0.68%). In PPS II, 10.6% of the athletes required further evaluation by echocardiography according to the indication criteria. However, the prevalence of the marked echo findings in both studies was similar. In both studies, most of these abnormalities did not place the athletes in jeopardy when engaging in competitive sports. Eight athletes in PPS I and 7 in PPS II had "grey zone" hypertrophy in the echocardiographic study. Three of them had HCM (one from PPS I). In total, 7 athletes (4 in PPS I) were diagnosed with HCM, of whom 2 had a positive family history, 4 a loud systolic murmur, and 6 uncommon ECG abnormalities. Echocardiography proved to confirm the diagnosis in 6 athletes and was the main diagnostic tool in only one. Thus, our findings support the suggestion that an echocardiographic study is not necessary in the routine pre-participation screening of athletes. Furthermore, the addition of this test was judged to be unworkable, due to prohibitive costs and a number of practical issues.[1,8] Some authors supported the use of limited echocardiography for the screening of athletes because it was easily performed at low cost.[28] In contrast, others reported that echocardiography cannot itself guarantee identification of all important lesions, even in relation to HCM, and some cardiac diseases may not be detectable with any screening method.[7] Furthermore, there is concern that the widespread use of echocardiography in PPS of athletes could result in many false-positive or false-negative results. For example, false-positive results may arise from the assessment of athletes with "grey zone" cardiac hypertrophy,[21] while in athletes younger than 15 years false-negative results may be reported.[29]

Some authors suggested that cardiovascular screening should be performed only for elite athletes, some believe it should be generalised for the vast majority of the young population, while others maintain that there are still insufficient research data available concerning a cost-effective PPS method.[28,30-32] However, Maron et al compared the US and Italian experiences regarding PPS strategies in competitive athletes and claimed that routine electrocardiography and examinations by specially trained personnel are unlikely to influence the mortality rate.[33] Therefore, it remains debatable whether the addition of a routine ECG to a history and physical examination in the pre-participation cardiovascular screening is of value, and further evaluation based on a large-scale study is needed.[32,34,35]

A limitation of our study was the inability to compare the cost-efficiency issue between the two PPS studies. It was mentioned that the two screening methods were not assessed during the same time period. In addition, the balance between the usefulness of ECG and echo testing and the rate of false-positive results was not studied. Another limitation was that this was not the initial PPS for most of the athletes. Thus, our sample could be considered as selected. Moreover, there was no follow-up report of these athletes. This may explain the lower number of finally disqualified athletes compared to those reported in the Italian studies.[4,25] Additionally, it was not possible to confirm the diagnosis as HCM or coronary artery disease with additional testing such as DNA analysis and coronary angiography, although they were suggested by our centre. Finally, we cannot draw conclusions regarding the outcomes of the PPS strategies related to SCD rates.

In spite of these limitations and critical points, we conclude that almost half of the Greek athletes examined presented "abnormal" findings in both PPS programs. Furthermore, the PPS test that includes only routine electrocardiographic study, which is in agreement with the "European" recommendations, is suggested as a successful strategy to identify young athletes at cardiovascular risk.

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