The Athlete's Heart and Echocardiographic Screening for Competitive Athletes

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Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

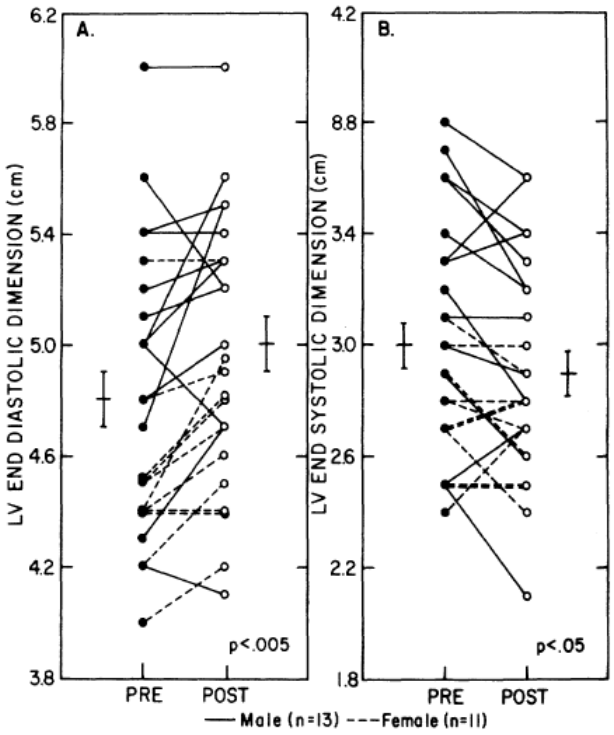
American Heart Association®

Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography.

A N DeMaria, A Neumann, G Lee, W Fowler and D T Mason

Circulation. 1978;57:237-244
doi: 10.1161/01.CIR.57.2.237
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539



European Association of Preventive Cardiology (EAPC) and European Association of Cardiovascular Imaging (EACVI) joint position statement: recommendations for the indication and interpretation of cardiovascular imaging in the evaluation of the athlete's heart

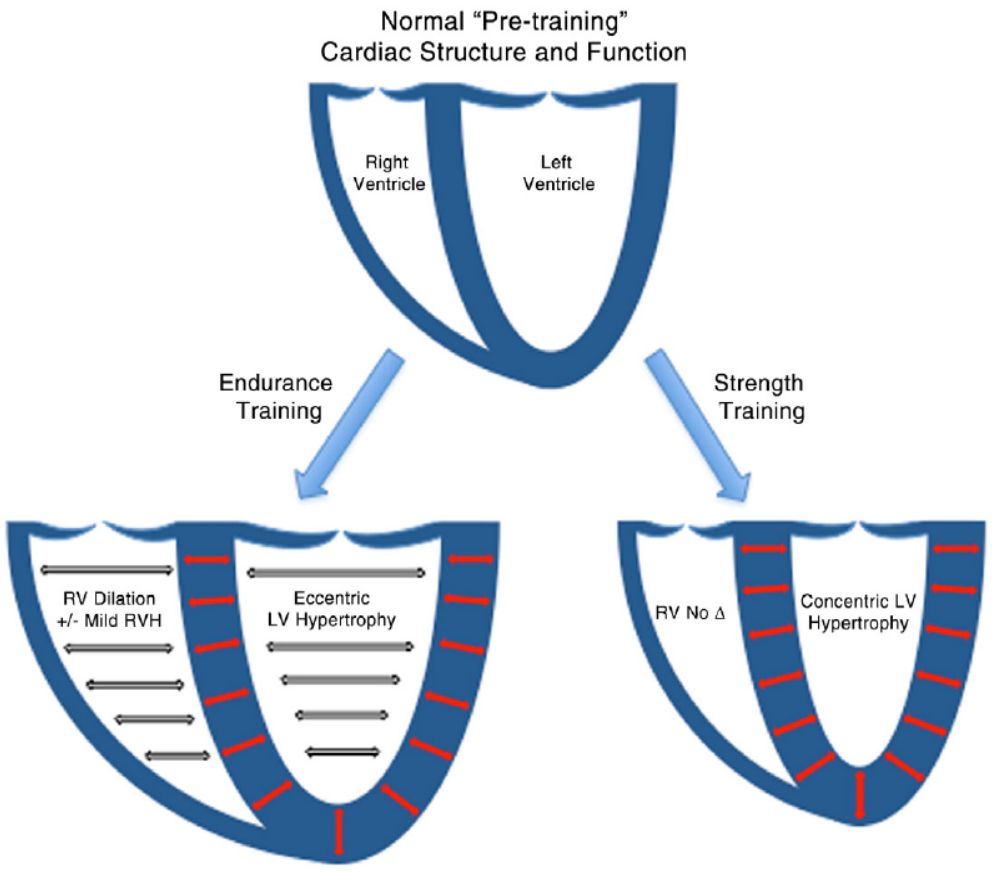
Antonio Pelliccia (Chairperson)[1], Stefano Caselli (Co-chairperson)[1]*, Sanjay Sharma[2], Cristina Basso[3], Jeroen J. Bax[4], Domenico Corrado[3], Antonello D'Andrea[5], Flavio D'Ascenzi[6], Fernando M. Di Paolo[1], Thor Edvardsen[7], Sabiha Gati[8], Maurizio Galderisi[9], Hein Heidbuchel[10], Alain Nchimi[11], Koen Nieman[12], Michael Papadakis[2], Cataldo Pisicchio[1], Christian Schmied[13], Bogdan A. Popescu[14], Gilbert Habib[15], Diederick Grobbee[16], and Patrizio Lancellotti (Chairperson)[17]

Athlete Heart: Definition

- Athlete's heart is generally regarded as a benign **increase in cardiac mass**, with specific circulatory and **cardiac morphological alterations**, that represents a **physiological adaptation** to systematic training.
- There is increasing recognition of the impact that prolonged conditioning has on cardiac remodeling, **which may eventually mimic certain pathological conditions** with the potential for sudden death or disease progression.

Beneficial Adaptation versus Overuse Pathology

Cardiac Remodeling with Exercise



Characteristic Adaptations

- Mild to Moderate Eccentric LVH and RV dilation
- Biatrial enlargement
- Normal to slightly reduced resting LVEF
- Normal or enhanced Early LV Diastolic Function
- Normal or enhanced LV twisting / untwisting

Characteristic Adaptations

- Mild concentric LVH but No RV remodeling
- Normal to mildly enlarged left atrial size
- Normal to hyperdynamic resting LVEF
- Normal to slightly reduced early LV diastolic function
- Compensatory increase in late LV diastolic function

Classes of Athletic Activity

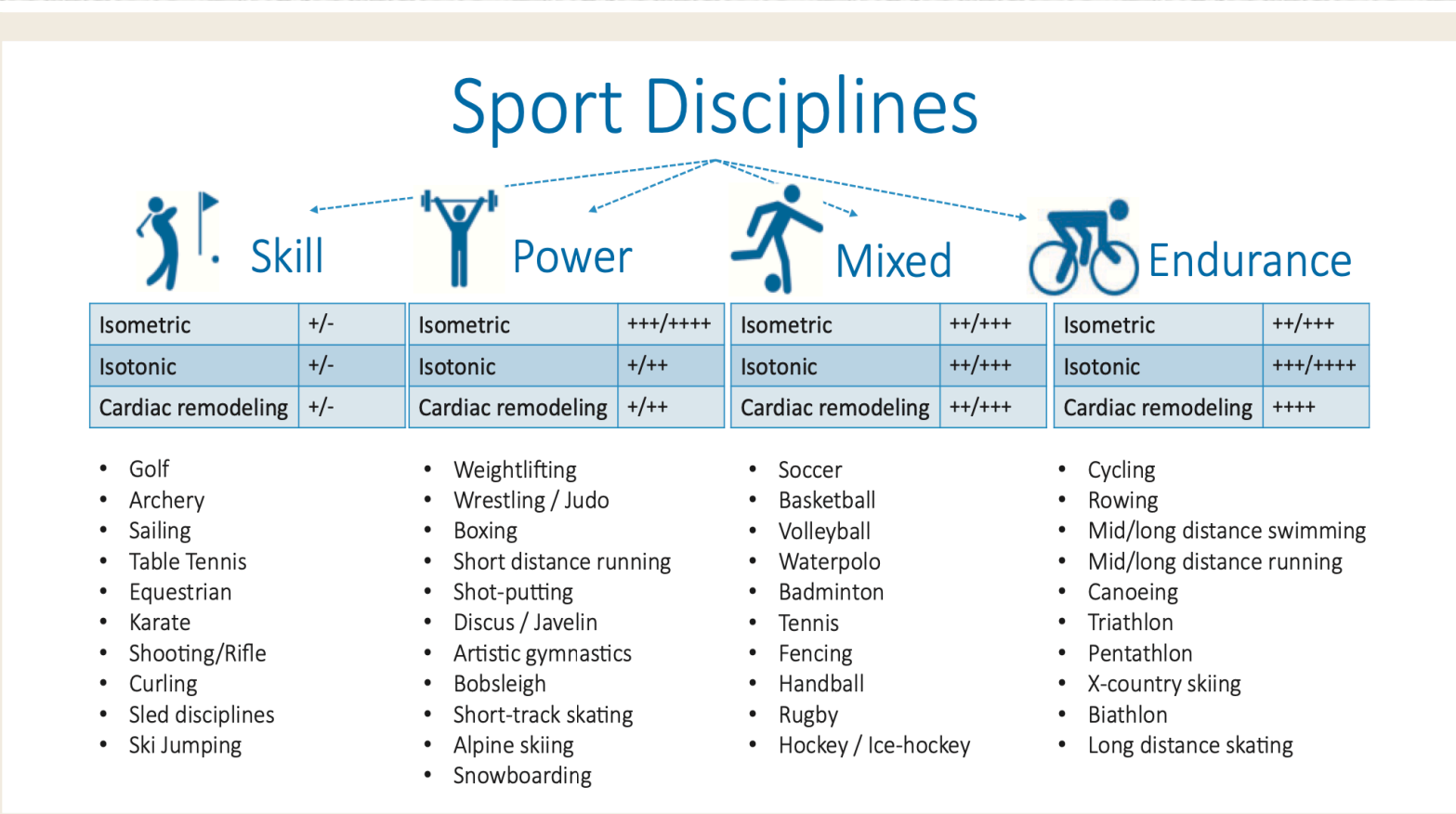


Figure 3 Simplified classification of the most common Olympic sport disciplines, according to the relative isometric and isotonic components of exercise and resulting cardiovascular adaptation.

Athletic Heart: General Concepts

- Athletes generally show relatively small (but statistically significant) increases of 10% to 20% for wall thickness or cavity size, and values in most individual athletes remain within accepted normal limits.

- Strength training is associated with only mildly increased wall thicknesses (often disproportionate relative to cavity size), whereas absolute values uncorrected for body surface area usually remain within the accepted normal range (12 mm)

Environment vs Genetics in Athletic Remodeling

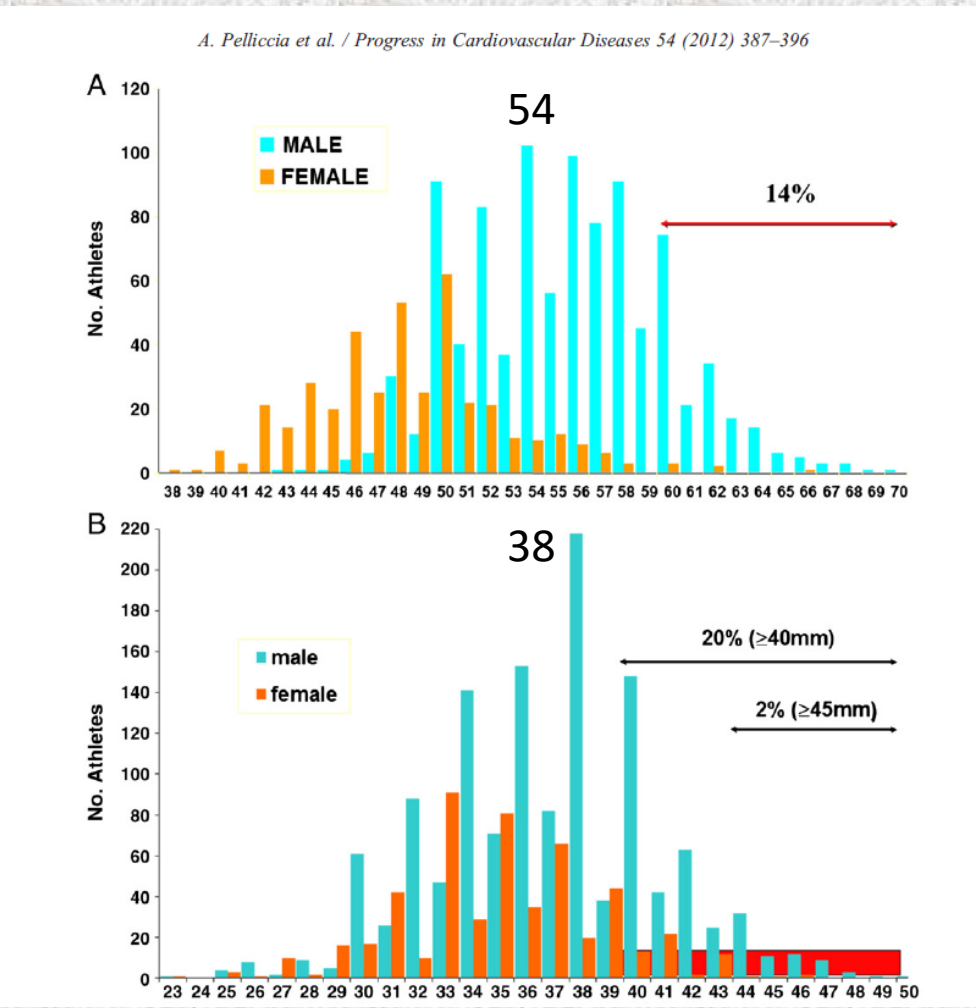
- The relative contributions of demographic and environmental or genetic determinants to LV remodeling in trained athletes is controversial

- 75% of variability in LV cavity size is attributable to nongenetic factors, such as ***body size, type of sport, gender***, and ***age, ethnicity***, with ***body surface area*** the largest of these components. The remaining 25% of cavity size variability is otherwise unexplained and possibly caused in small part by genetic factors

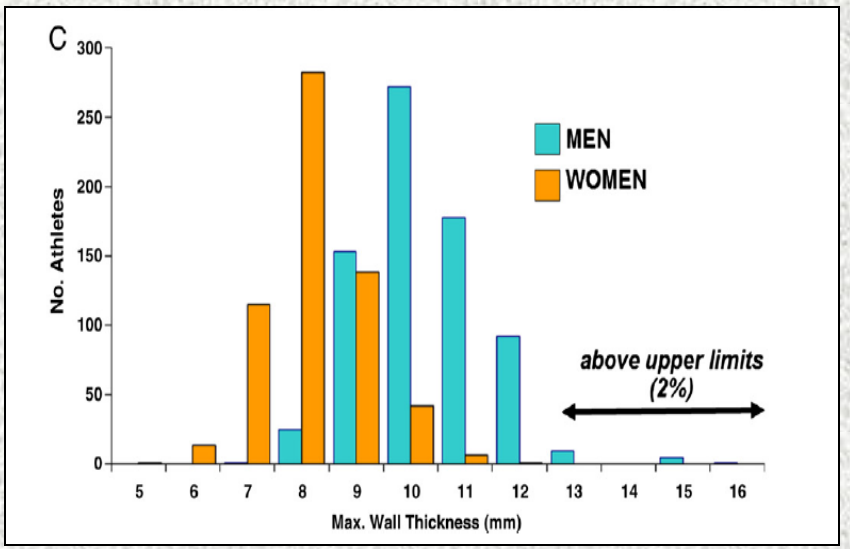
LV and LA Dimensions in Athletes

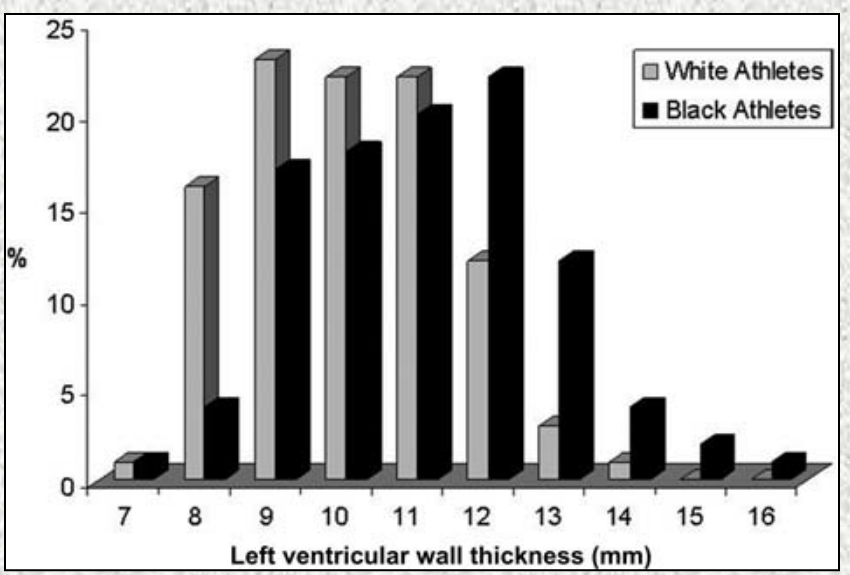
LVDd

LAd

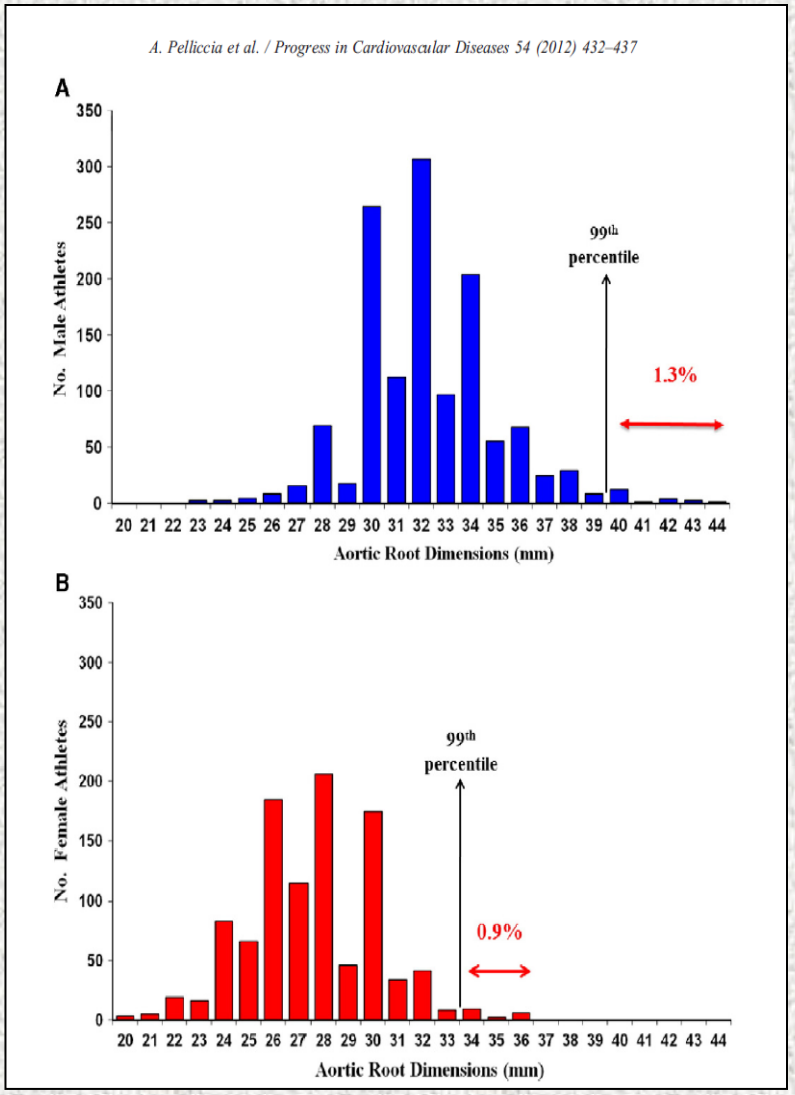


LV Wall Thickness in Athletes

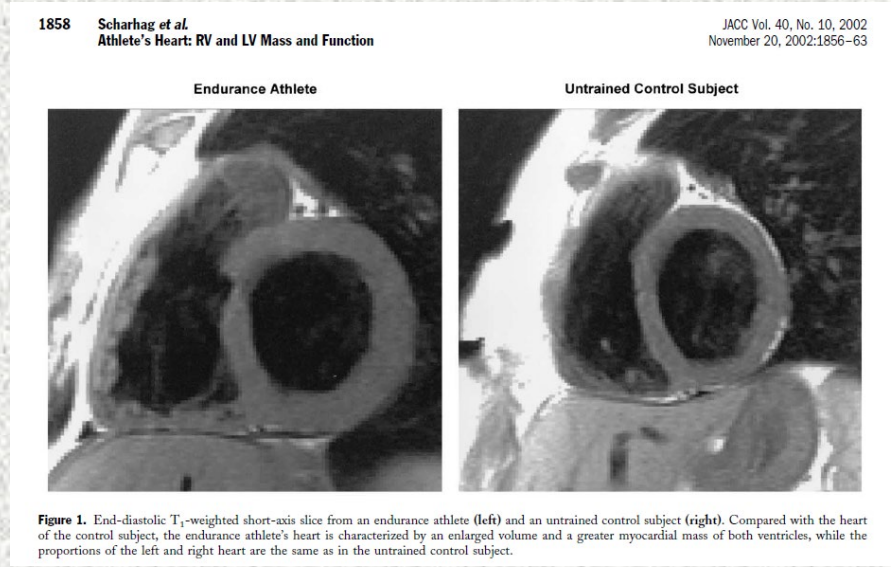


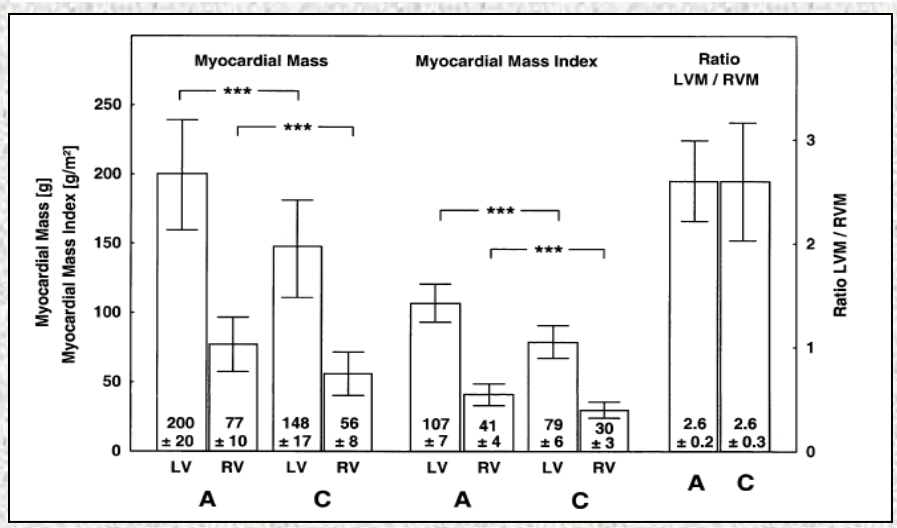


Aortic Root Size in Athletes

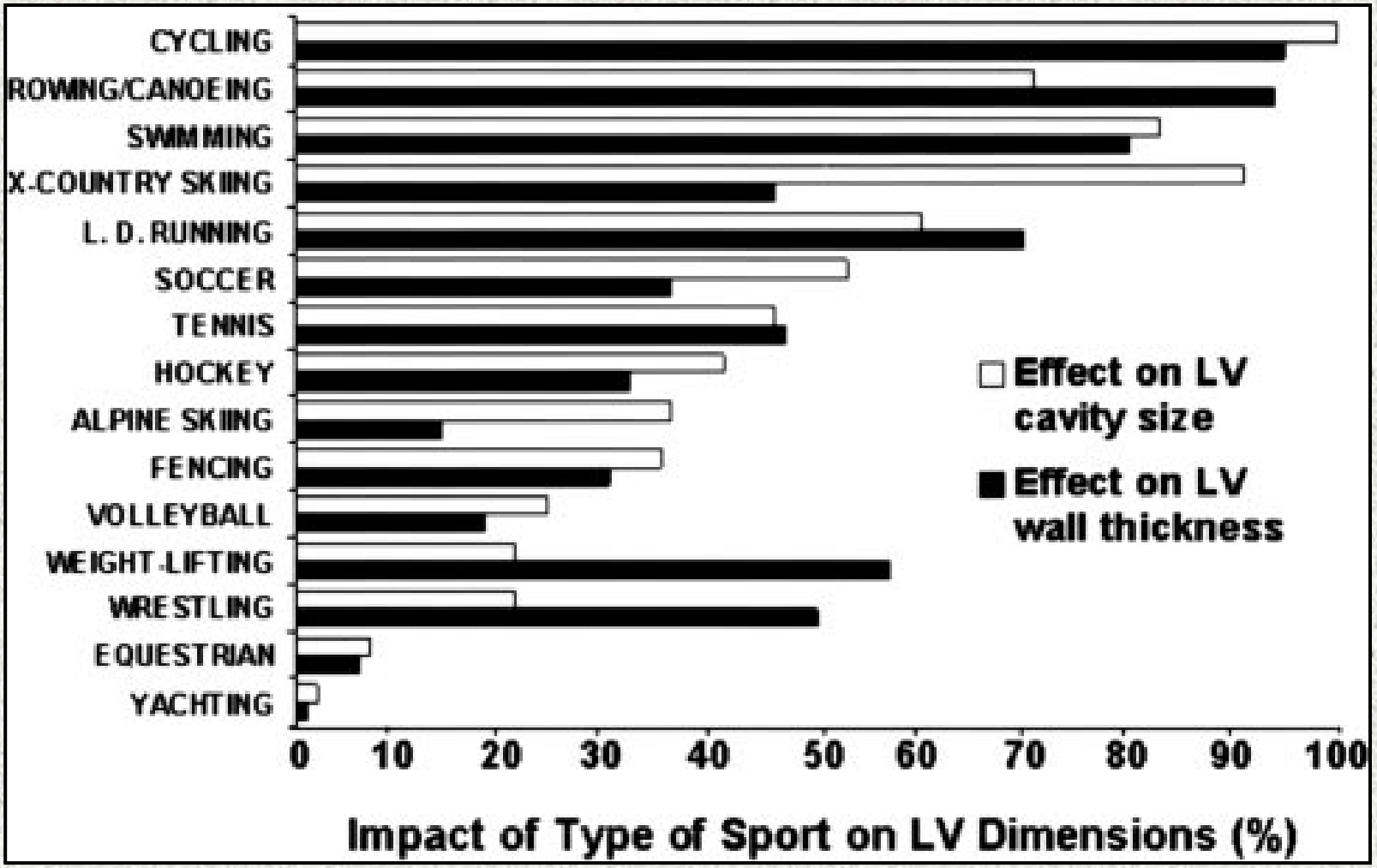


Right Ventricle in Athletes





Impact of Sport on Athletic Heart



Diastole and Deformation

Endurance Athletes

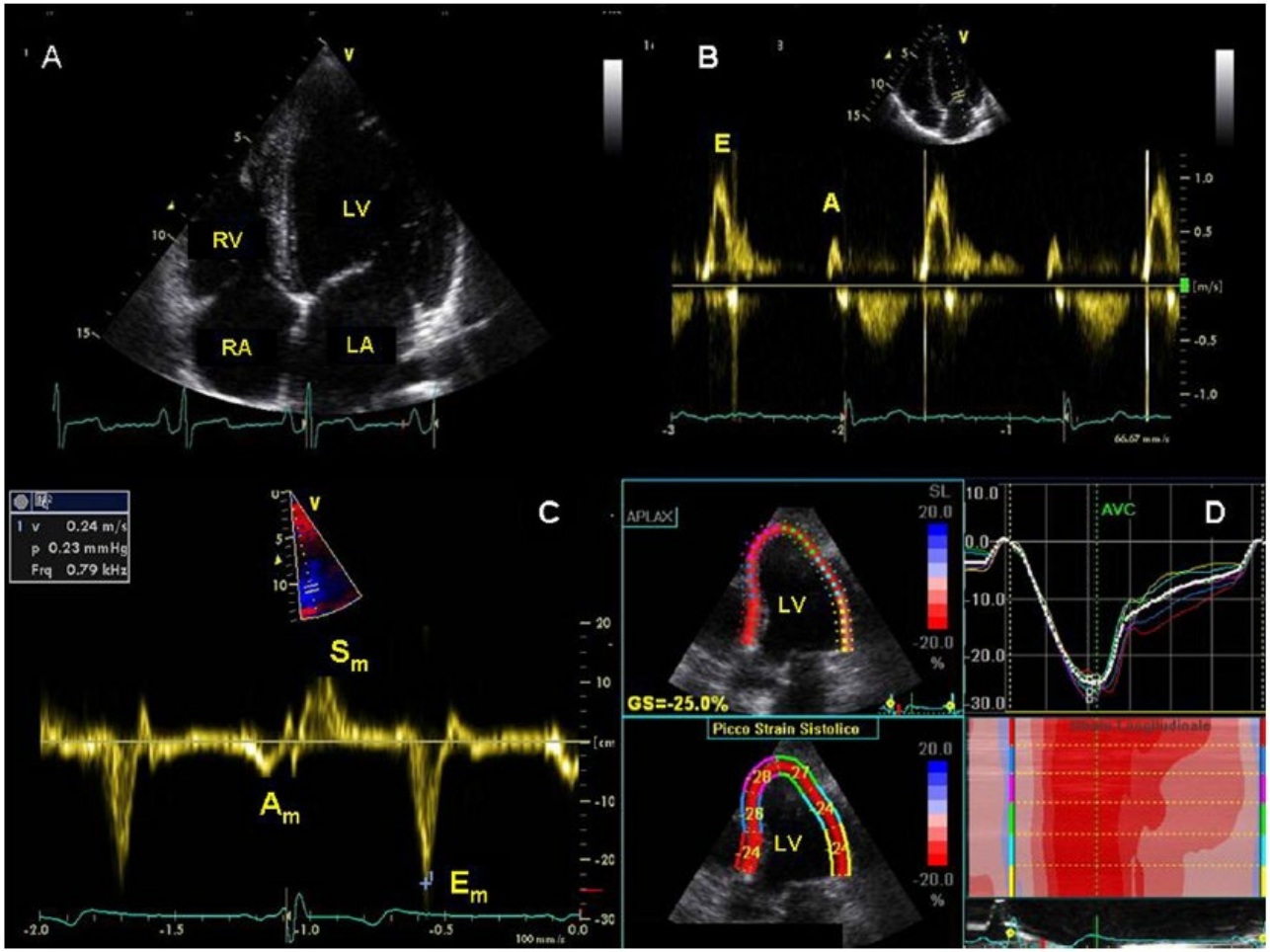


Figure 1 Echocardiographic evaluation of athlete's heart in an ATE. **(A)** Standard echocardiography, apical four-chamber view: left ventricular hypertrophy. **(B)** Transmitral flow pattern: high early diastolic wave. **(C)** Pulsed tissue Doppler pattern: increased early diastolic myocardial velocity. **(D)** Two-dimensional strain: normal myocardial longitudinal deformation.

D'Andrea et al. JASE, 2010

Diastole and Deformation

Strength Athletes

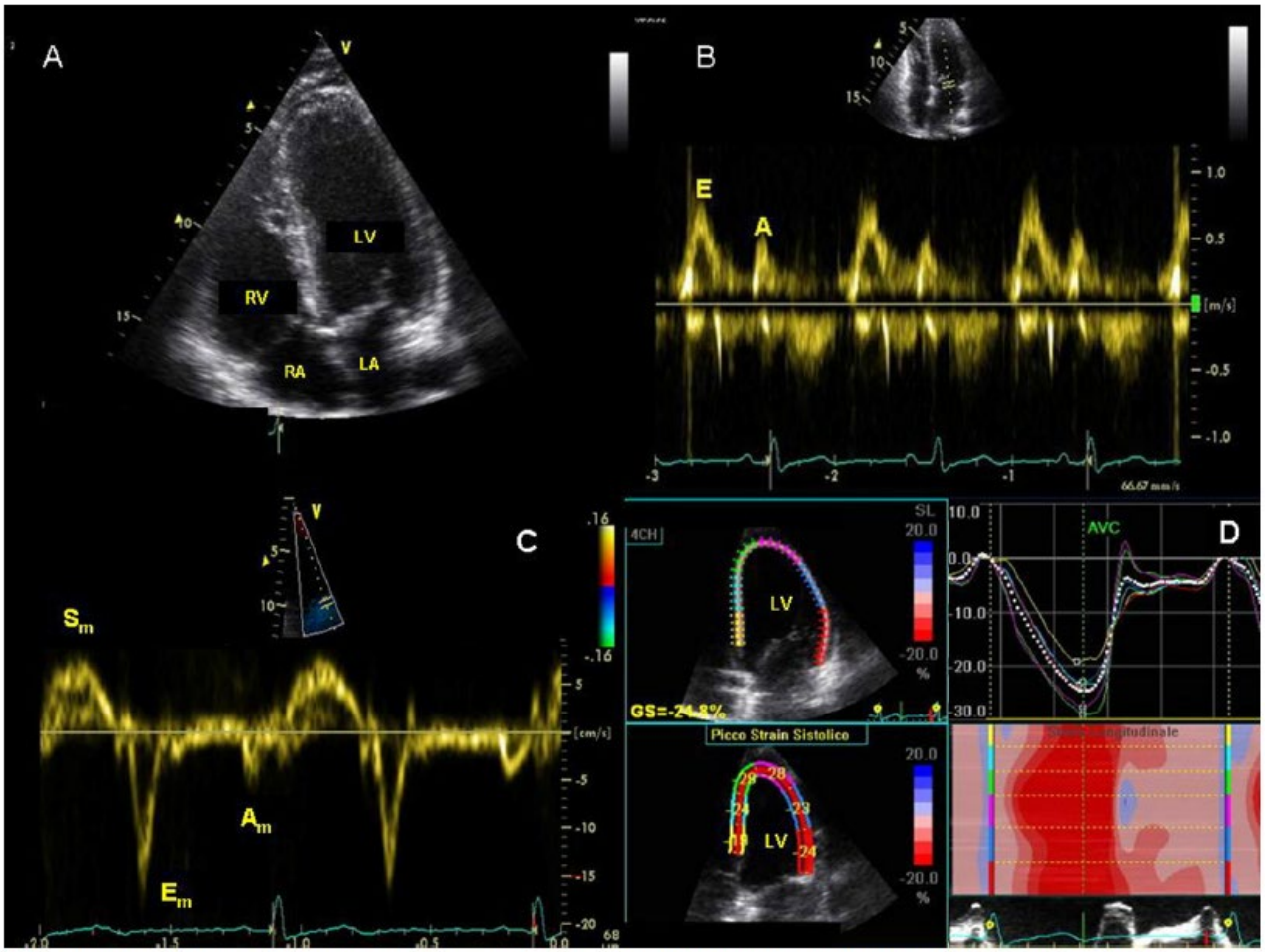
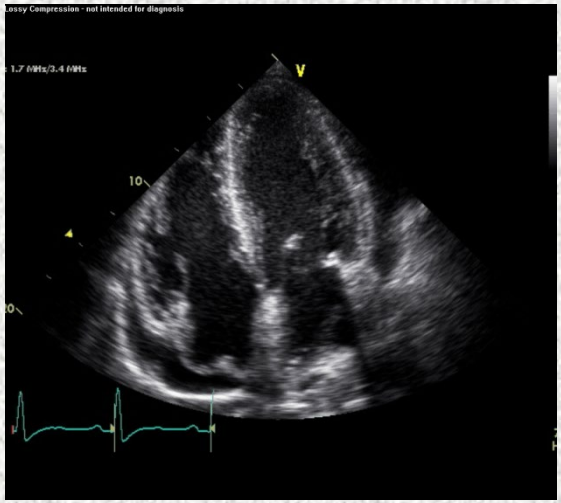
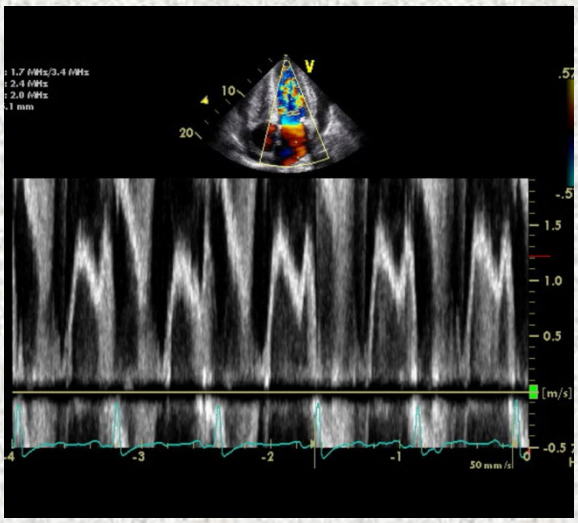


Figure 2 Echocardiographic evaluation of athlete's heart in an ATP. **(A)** Standard echocardiography, apical four-chamber view. **(B)** Transmitral flow pattern. **(C)** Pulsed tissue Doppler pattern. **(D)** Two-dimensional strain.

D'Andrea et al. JASE, 2010

Diastole and Deformation
HCM





LV and RV Strain in Athletes

Table 5 **Most relevant studies assessing left (upper panel) and right (lower panel) ventricular strain by speckle-tracking echocardiography in athletes**

Author	Year	Sport discipline	Nr	Longitudinal strain
				Left ventricle
Caselli *et al.*	2014	Olympic athletes	200	-18.1 ± 2.2%
Nottin *et al.*	2008	Elite cyclists	16	19.2 ± 1.9%
Cappelli *et al.*	2010	Endurance athletes	50	-18.4 ± 3.0%
Galderisi *et al.*	2010	Top level rowers	22	-22.2 ± 2.7%
Simsek *et al.*	2913	Marathon runners	22	-22.3 ± 2.2% (global)
Simsek *et al.*	2013	Wrestlers	24	-21.8 ± 1.7% (global)
Weiner *et al.*	2013	University Rowers	15	-16.8 ± 2.1% (pre-training)
				-18.3 ± 2.8% (post-training)
				Right ventricle
Teske *et al.*	2009	Endurance athletes/Olympic endurance athletes	58/63	-28.5 ± 2.9%/
				-27.6 ± 3.1%
Oxborough *et al.*	2012	Endurance athletes	102	-27.0 ± 6.0%
Pagourelias *et al.*	2013	Endurance/Power athletes	80/28	-23.1 ± 3.7%/
				-25.1 ± 3.2%
Esposito *et al.*	2014	Top level rowers	40	-26.3 ± 3.6% (global)
				-29.1 ± 4.1% (free wall)
D'Ascenzi *et al.*	2015	Mixed sport disciplines	29	-28.7 ± 4.9% (Pre-season)
				-29.2 ± 4.1% (Mid-season)
				-30.0 ± 3.7% (End-season)

Prognostic Value of Global Longitudinal Strain in Hypertrophic Cardiomyopathy

A Systematic Review of Existing Literature

Albree Tower-Rader, MD,[a,*] Divyanshu Mohananey, MD,[a,*] Andrew To, MD,[a,b] Harry M. Lever, MD,[a] Zoran B. Popovic, MD, PhD,[a] Milind Y. Desai, MD[a]

ABSTRACT

OBJECTIVES The association of left ventricular global longitudinal strain (LV-GLS) with clinical outcomes in patients with hypertrophic cardiomyopathy (HCM) has been examined in multiple studies. The authors conducted a systematic review aimed at summarizing and critically appraising the current evidence.

BACKGROUND HCM is a common genetic cardiovascular disease with an estimated prevalence of 1 in 500 patients. LV-GLS derived from speckle tracking echocardiography is a sensitive noninvasive method of assessing regional left ventricular function. Several studies have suggested association of abnormal LV-GLS with outcomes in HCM patients.

METHODS A computerized literature search of all English language publications in the PubMed and EMBASE databases was made looking at all randomized and nonrandomized studies conducted on patients with HCM where association of

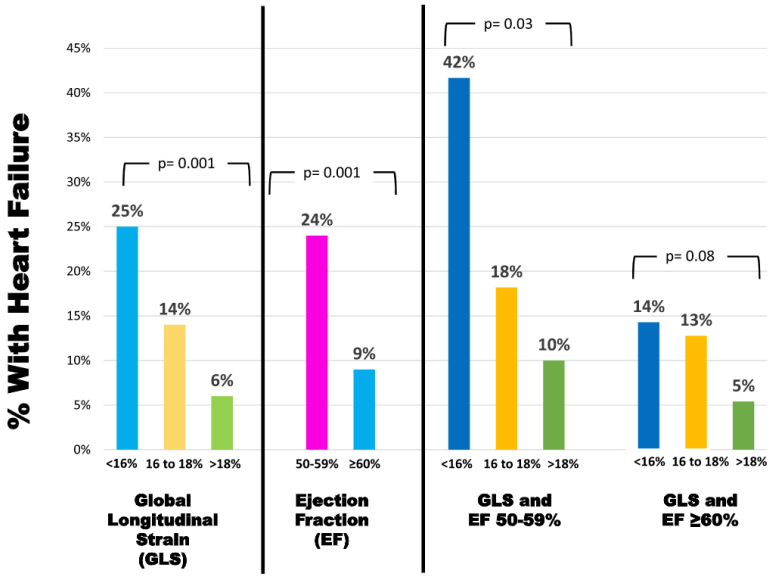
We noted wide variability in inclusion, methodology, follow-up, and consequently effect estimates, which was not conducive to performing a meta-analysis. However, despite the variation, all studies revealed a degree of association of abnormal LV-GLS with poor cardiac outcomes

implantable cardiac defibrillator discharge. We noted wide variability in inclusion, methodology, follow-up, and consequently effect estimates, which was not conducive to performing a meta-analysis. However, despite the variation, all studies revealed a degree of association of abnormal LV-GLS with poor cardiac outcomes.

CONCLUSIONS Our systematic review of more than 3000 HCM patients suggests an association of abnormal LV-GLS with adverse composite cardiac outcomes and ventricular arrhythmias. (J Am Coll Cardiol Img 2019;12:1930–42)

Usefulness of Global Longitudinal Strain to Predict Heart Failure Progression in Patients With Nonobstructive Hypertrophic Cardiomyopathy

Ethan J. Rowin, MD[a,*], Barry J. Maron, MD[a], Sophie Wells, MD[a], Austin Burrows, BA[a], Christopher Firely, MD[a], Benjamin Koethe, MPH[b], Ayan R. Patel, MD[a], and Martin S. Maron, MD[a]

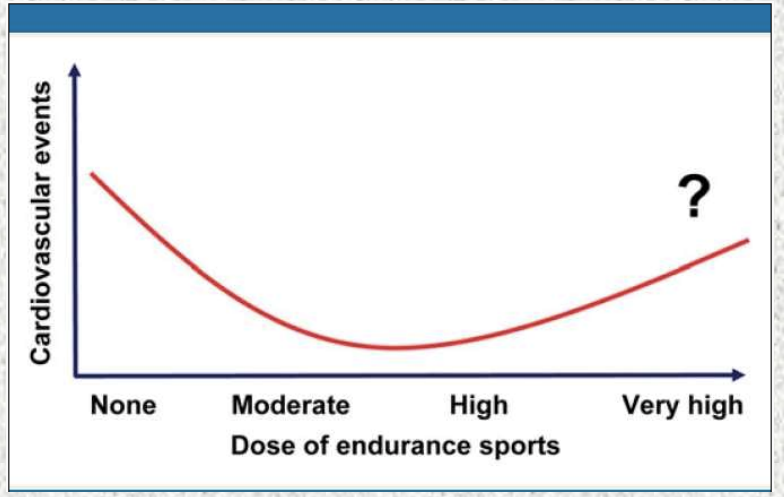


Luc-Olivier Merson's painting depicting Pheidippides giving word of victory at the Battle of Marathon to the people of Athens

Adverse Effects of Endurance Exercise

- LV dilation and reduced EF
- Elevated troponins
- Elevated BNP
- Increased frequency of atrial fibrillation
- Increased coronary calcification

- Cardiac fatigue
- Exercise overdosing?



Serial Left Ventricular Adaptations in World-Class Professional Cyclists

Implications for Disease Screening and Follow-Up

Eric Abergel, MD,* Gilles Chatellier, MD,† Albert A. Hagege, MD, PHD,* Agnes Oblak, MD,* Ales Linhart, MD,* Alain Ducardonnet, MD,‡ Joël Menard, MD, PHD§

JACC Vol. 44, No. 1, 2004
July 7, 2004:144–9

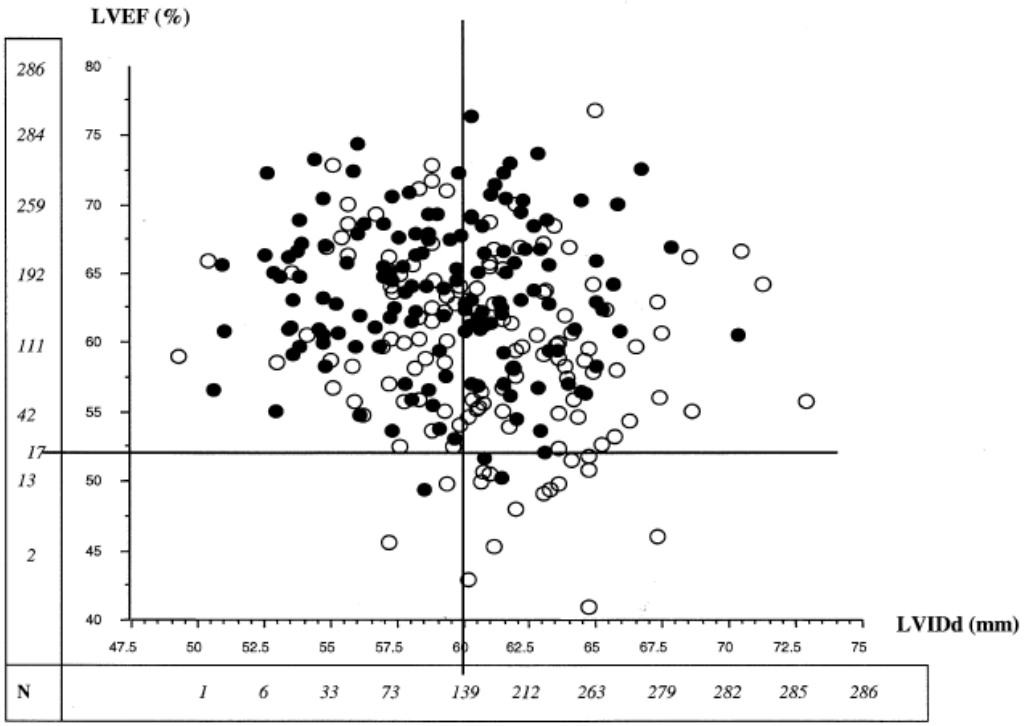


Figure 1. Plot of left ventricular ejection fraction (LVEF) against left ventricular internal diameter at end diastole (LVIDd) in all cyclists (**solid circles** = 1995; **open circles** = 1998). The **solid vertical bar** represents the normality threshold (60 mm) for LVIDd, and the **solid horizontal bar** represents the normality threshold (52%) for LVEF. Numbers in **italics** in front of each axis value are cumulative numbers of cyclists with a value below the corresponding axis value.

Serial LV Changes in Elite Cyclists

Table 2. Echocardiographic Characteristics of the Subgroup of 37 Cyclists That Participated in Both the 1995 and 1998 Races

	1995	1998	p Value
LVIDd (mm)	58.3 ± 4.8	60.3 ± 4.2	0.001
BSA (m^2)	1.89 ± 0.11	1.89 ± 0.11	0.16
LVIDdi (mm/m^2)	30.9 ± 2.4	32.0 ± 1.8	0.0011
LVIDs (mm)	37.8 ± 4.2	40.3 ± 3.9	<0.0001
IVSTd (mm)	11.8 ± 1.2	10.8 ± 1.2	0.0002
PWTd (mm)	10.6 ± 1.0	9.9 ± 0.8	0.0014
LVMi (g/m^2)	144 ± 23	138 ± 21	0.05
RWT	0.39 ± 0.05	0.35 ± 0.03	<0.0001
LVEF (%)	63.5 ± 6.3	60.8 ± 5.7	0.01
eFS	35.2 ± 4.7	33.3 ± 4.1	0.019
mFS	17.0 ± 2.0	17.4 ± 1.9	0.32

The question of drugs?

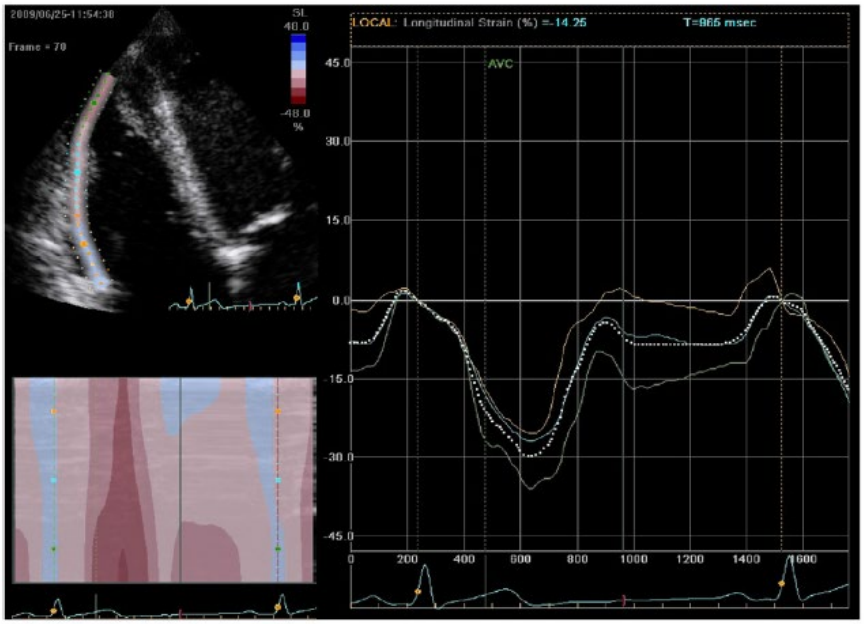
Exercise-Induced Cardiac Injury: Evidence From Novel Imaging Techniques and Highly Sensitive Cardiac Troponin Assays

Rob Shave[a,*], David Oxborough[b]

[a]*Cardiff School of Sport, Cardiff Metropolitan University, Cyncoed Campus, Cardiff, UK*
[b]*The Leeds Institute for Diagnostics and Therapeutics, Faculty of Medicine and Health, University of Leeds, Leeds, UK*

Right Ventricular Strain

PRE



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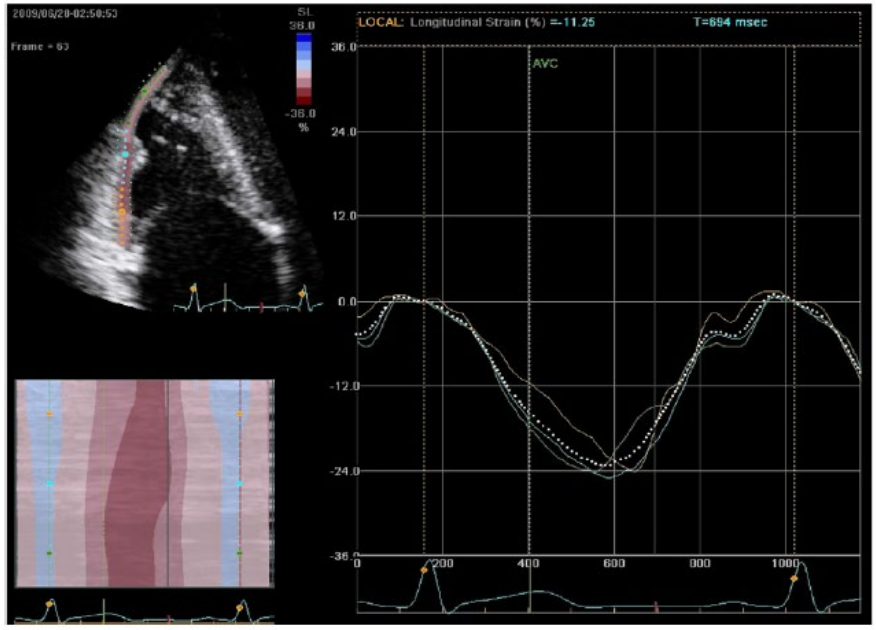


Long-Term Clinical Consequences of Intense, Uninterrupted Endurance Training in Olympic Athletes

Antonio Pelliccia, MD,* Norimitsu Kinoshita, MD,† Cataldo Pisicchio, MD,* Filippo Quattrini, MD,* Fernando M. DiPaolo, MD,* Roberto Ciardo, MD,* Barbara Di Giacinto, MD,* Emanuele Guerra, MD,* Elvira De Blasiis, MD,* Maurizio Casasco, MD,* Franco Culasso, PHD,‡ Barry J. Maron, MD§

(J Am Coll Cardiol 2010;55:1619–25)

114 Olympic Athletes competing in 2-4 Games (average intense training 8.3 yrs)

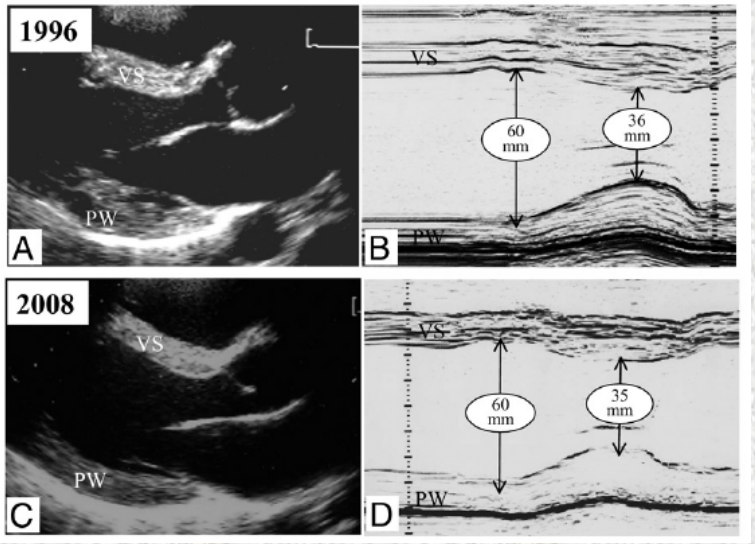


Table 3 Demographic, Dimensional, and Cardiac Functional Characteristics of the 97 Athletes Comprising the Comparison Group Who Participated in Only 1 Olympic Games

	Initial Evaluation	Most Recent Evaluation	p Value
Age (yrs)	23.1 ± 4.2 (15–34)	27.1 ± 4.4 (19–38)	0.001
BSA (m^2)	1.86 ± 0.20 (1.48–2.26)	1.86 ± 0.21 (1.48–2.27)	0.84
AVS (mm)	9.9 ± 1.2 (7–13)	9.9 ± 1.2 (7–13)	1.0
PFW (mm)	9.7 ± 1.1 (7–12)	9.7 ± 1.2 (7–13)	0.94
LVDd (mm)	53.9 ± 4.4 (45–64)	54.3 ± 4.1 (46–65)	0.47
LV volume (ml)	142.1 ± 26.3 (92–208)	144.6 ± 25.2 (97–216)	0.51
LVM index (g/m^2)	108.8 ± 20.6 (57.4–157.1)	109.9 ± 22.4 (60.6–166.9)	0.73
LA (mm)	36.1 ± 4.0 (25–38)	36.8 ± 3.7 (29–48)	0.18
EF (%)	63.4 ± 5.8 (50–77)	64.2 ± 6.0 (51–81)	0.39
E wave (mm/s)	79.7 ± 16.4 (46–131)	80.3 ± 15.7 (47–127)	0.79
A wave (mm/s)	39.9 ± 8.8 (24–82)	42.2 ± 8.5 (23–66)	0.08
E/A ratio	2.1 ± 0.6 (1.1–3.6)	1.9 ± 0.5 (1.0–3.3)	0.12
SBP/ESV	4.4 ± 1.0 (1.9–7.6)	4.7 ± 1.3 (2.5–8.6)	0.21

Values are reported as mean ± SD (range).

A wave = peak late (atrial) diastolic filling velocity; AVS = anterior ventricular septum; BSA = body surface area; E wave = peak early diastolic filling velocity; EF = ejection fraction; LA = left atrium; LV = left ventricular; LVDd = left ventricular end-diastolic diameter; LVM index = left ventricular mass normalized to body surface area; PFW = posterior free wall; SBP/ESV = ratio of systolic blood pressure to normalized end-systolic volume.

Athletic Heart vs Cardiomyopathy

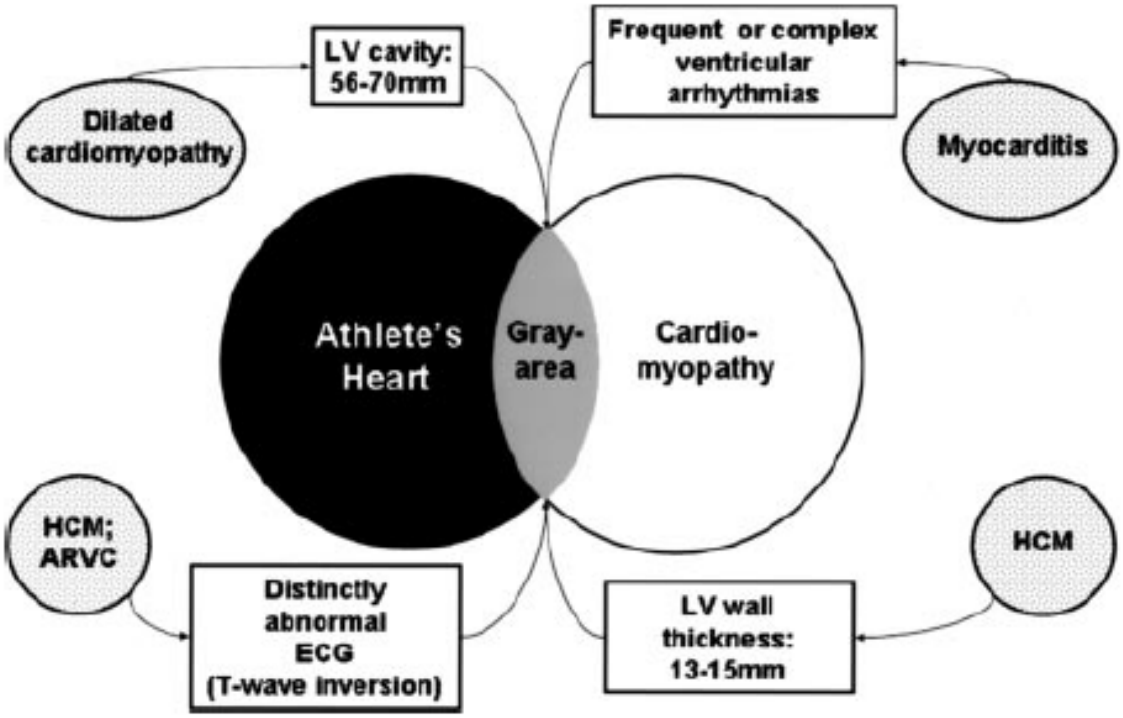
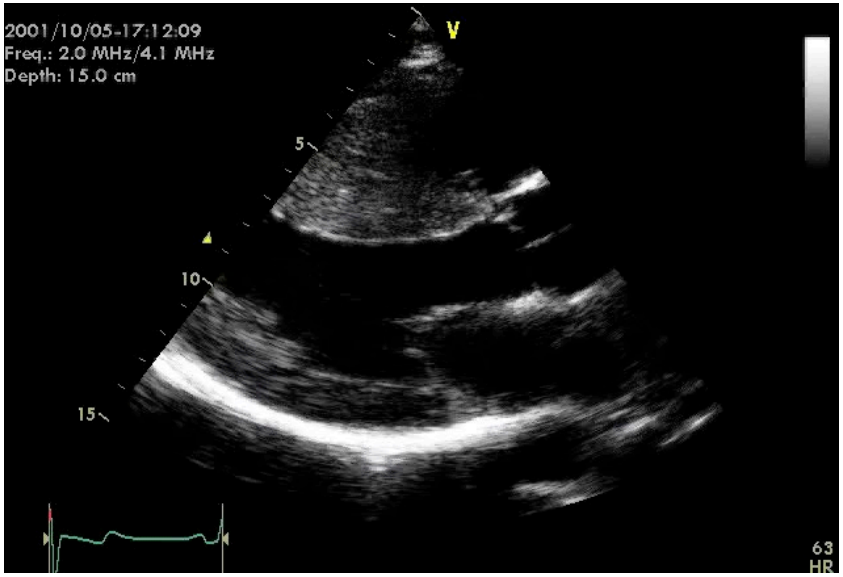


Figure 10. Differential diagnosis between athlete's heart and cardiac disease. Gray zone of overlap between physiological hypertrophy and pathological cardiomyopathies (including myocarditis, HCM, and ARVC). Adapted from Maron[1] with permission of the Massachusetts Medical Society. Copyright 2003.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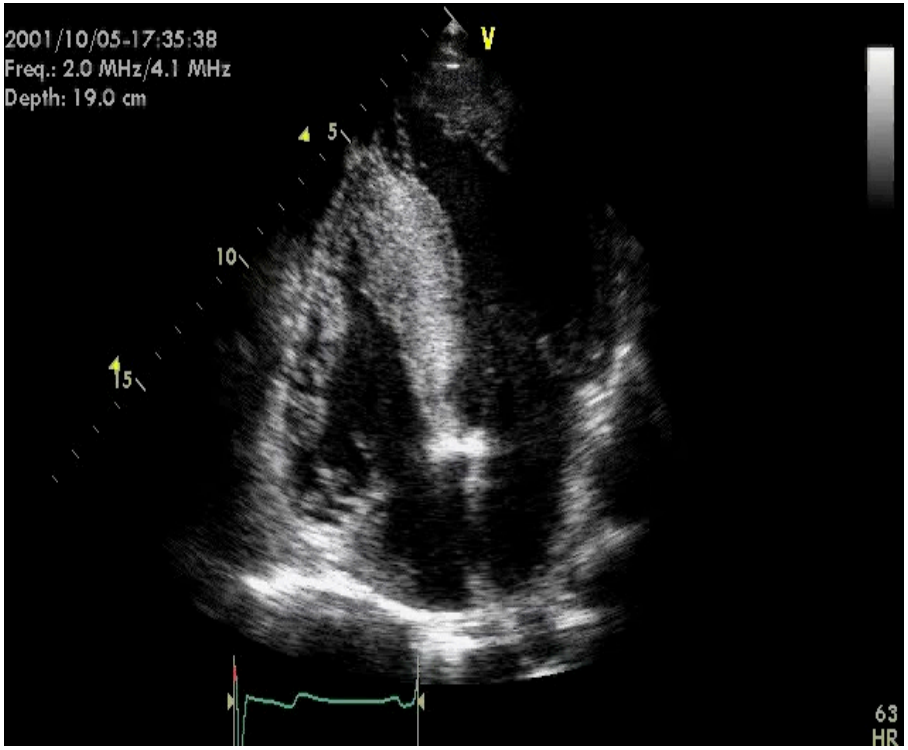
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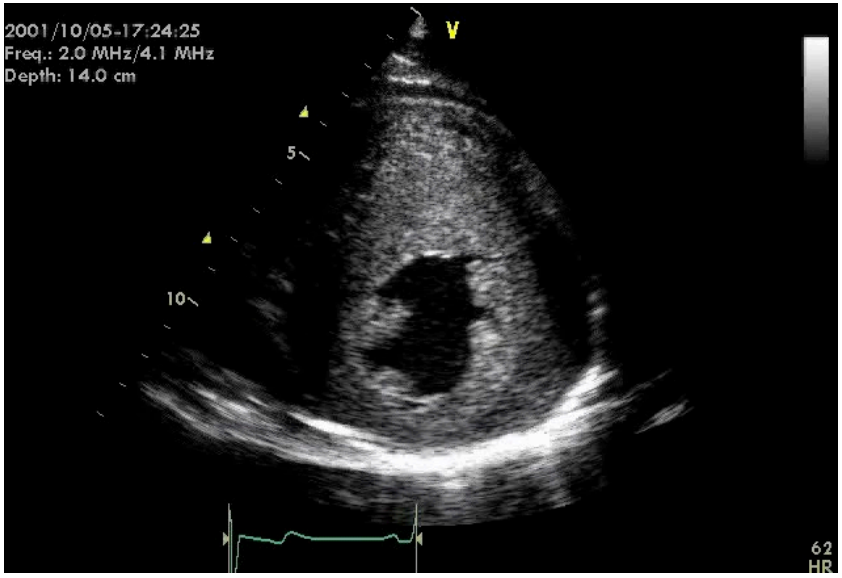
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HR

HCM vs Athletic Heart: Distinction

Parameter	HCM	Athletic heart
LV wall thickness and morphology	Can be >12 mm; can be concentric or asymmetric across segments	Typically <12 mm, especially in women; concentric
Diastolic LV cavity	<45 mm (except in late, dilated phase)	>55 mm
LA size	Enlarged	Normal
LV diastolic filling pattern	Impaired relaxation (E:A ratio <1, prolonged diastolic deceleration time)	Normal
Response to deconditioning	None	LV wall thickness decreases
Family history of HCM	Present (except de novo mutations)	Absent
ECG findings	Very high QRS voltages; Q waves; deep negative T waves	Criteria for LVH but without unusual features

LA, left atrium; LV, left ventricular; LVH, left ventricularhypertrophy.

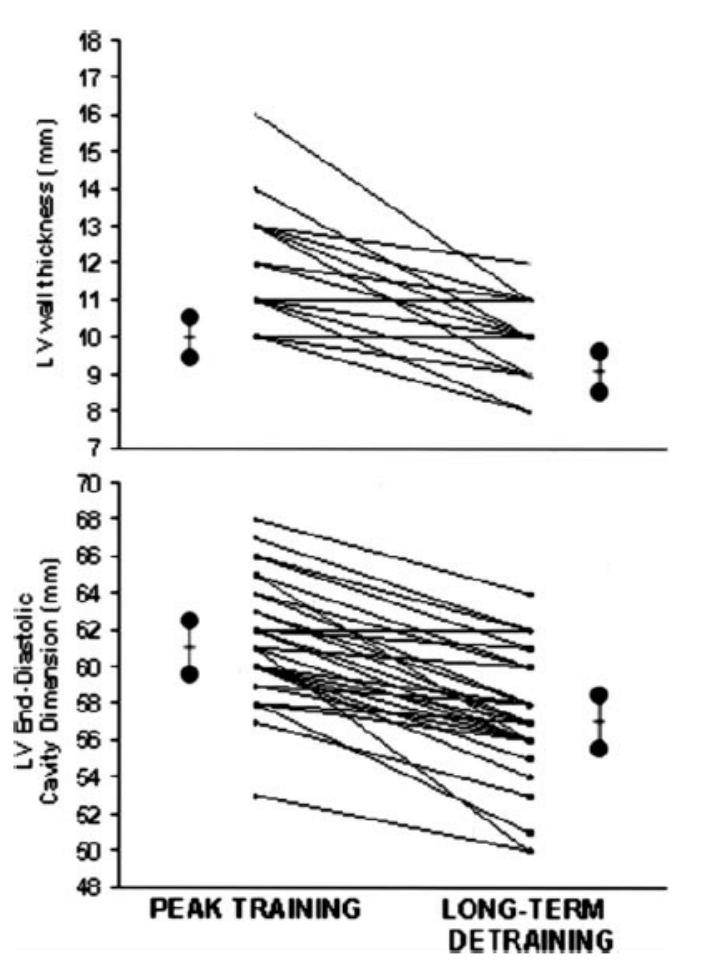
Athletic Heart vs HCM

Table 7 **Cardiac imaging findings consistent with diagnosis of athlete's heart when left ventricular wall thickness is mildly increased, ranging from 13 mm to 16 mm**

Diagnosis of athlete's heart in the gray-zone of left ventricular hypertrophy(13–16 mm)		
HCM	**Findings**	**Athlete's Heart**
+	Family history of sudden cardiac death/HCM	–
+	Major ECG abnormalities (ST segment/T wave inversion, wide, and deep Q waves)	–
+	Normal or reduced LV cavity size (<54 mm)	–
+	Abnormal LV cavity geometry and/or segmental LV hypertrophy	–
+	LV outflow tract obstruction	–
+	Abnormal LV diastolic relaxation/filling(septal e′ velocity < 8.0 cm/s and/or *E*/*A* ratio < 1.0)	–
+	Left atrial remodelling disproportionate to LV remodelling	–
+	Positive LGE on CMR	–
+	Unchanged LV wall thickness after detraining	–

CMR, cardiac magnetic resonance; HCM, hypertrophic cardiomyopathy; ECG, electrocardiogram; LGE, late gadolinium enhancement; LV, left ventricular.

Detraining Effects on Athletic Heart

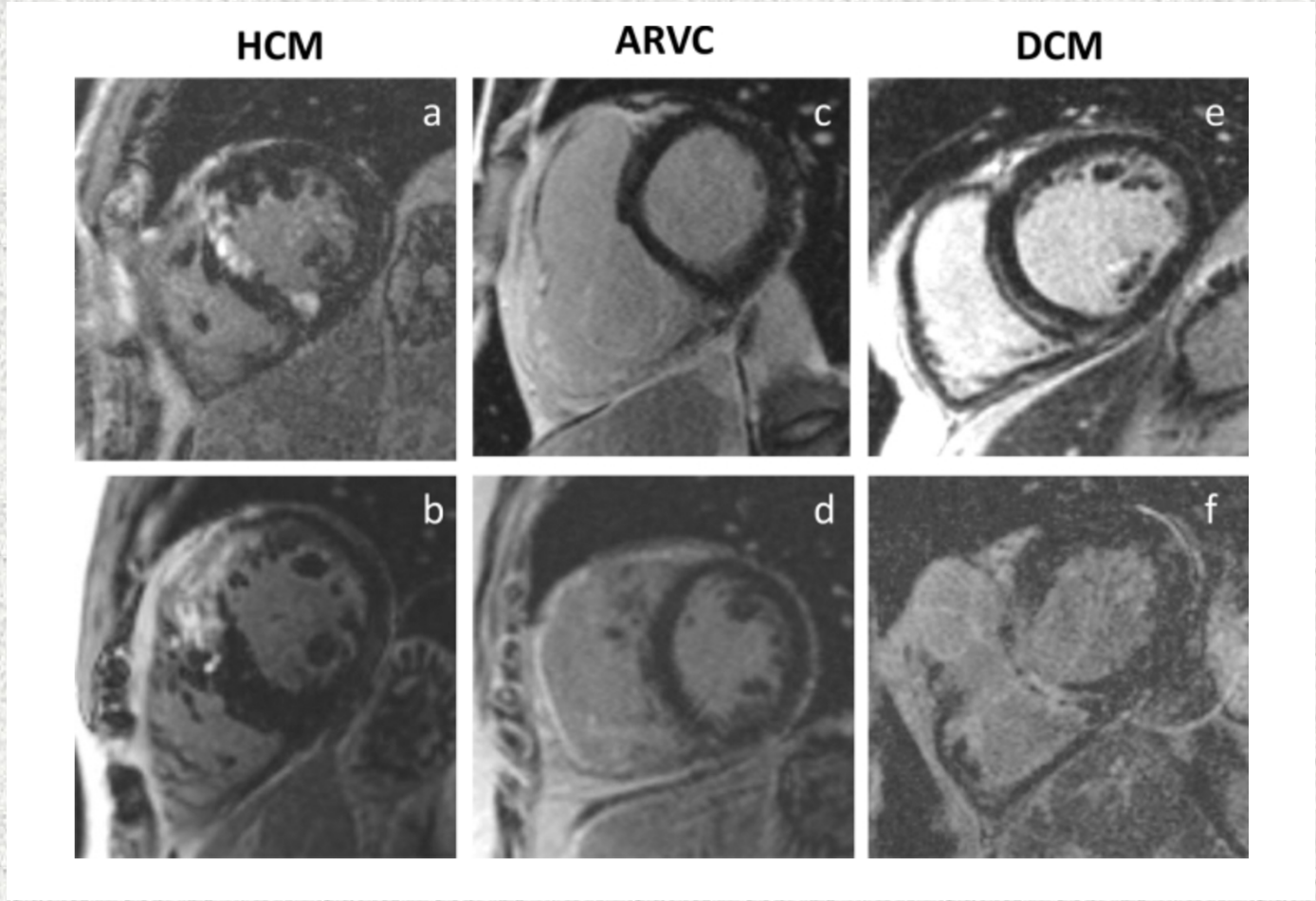


CMR in Cardiomyopathy

Cardiomyopathy	Typical pattern of fibrosis seen on CMR which allows differentiation from Athletes Heart
HCM	Classically, fibrosis at the junction of the right ventricle and interventricular septum
Ischaemic DCM	Subendocardial extending to transmural fibrosis, generally restricted to the perfusion territory of one coronary artery
Non-ischaemic DCM	Patchy, mid-wall distribution in 28%.
	Sub-endocardial pattern indistinguishable from ischaemic cardiomyopathy in 13%
ARVC	Differentiated from Athlete's Heart as RV and LV show disproportionate changes.
LVNC	Non-compacted myocardium
	Differentiated from Athlete's Heart as significant fibrosis in 55% of patients, which may occupy up to 5% of LV myocardium
Myocarditis	Most commonly fibrosis has been shown to involve the epicardium of the inferior lateral wall.
	Differentiated from Athlete's Heart due to lack of overt arrhythmias or classical symptoms (palpitations, presyncope or syncope)

CMR, cardiovascular magnetic resonance; HCM,hypertrophic cardiomyopathy; DCM, dilated cardiomyopathy; ARVC,arrhythmogenic right ventricular cardiomyopathy; LVNC,left ventricular non-compaction

CMR in Cardiomyopathy: LGE



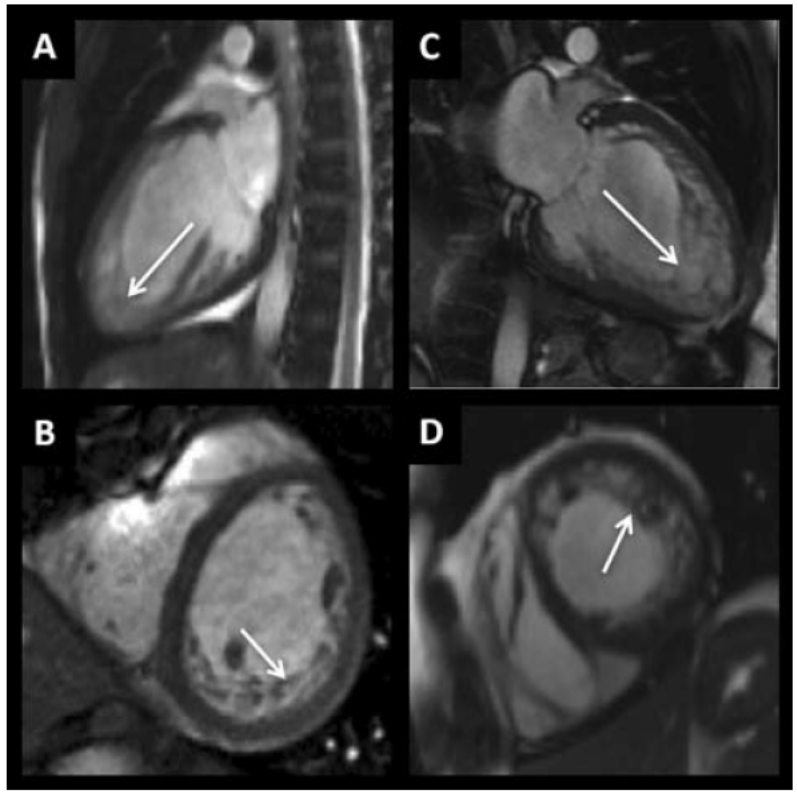
Athletic Heart vs ARVC

Table 8 **Clinical and multi-modality imaging findings that may help in differential diagnosis between physiologic RV adaptation as opposed to AC**

Differential diagnosis between athlete's heart and AC		
AC	**Findings**	**Athlete**
+	Family history of sudden cardiac death/AC	-
+	Anterior T wave inversion on ECG	-
+	Ventricular arrhythmias with LBBB morphology	-
+	Exercise induced VT	-
+	RV size exceeding major Task Force Criteria for echocardiography or CMR (consider only indexed values)	-
+	Regional wall motion abnormalities (akinesia or dyskinesia) on cardiac imaging.	-
+	Global RV dysfunction on echocardiography (RVFAC < 33%) or CMR (RVEF ≤ 40)	-
+	Abnormal RV function on exercise echocardiography/CMR	-

AC, arrhythmogenic cardiomyopathy; CMR, cardiac magnetic resonance; ECG, electrocardiogram; LGE, late gadolinium enhancement; LBBB, left bundle branch block; RV, right ventricle; RVEF, right ventricular ejection fraction; RVFAC, right ventricular fractional area change; VT, ventricular tachycardia.

Athletic Trabeculations vs Non-compaction



Athletic Heart vs Non-compaction

Table 9 **Findings consistent with diagnosis of athlete's heart or left ventricular non-compaction when increased trabeculations are occasionally found on echocardiography in athletes**

Athletes with increased trabeculations		
LVNC	**Findings**	**Athlete**
+	Family history of sudden cardiac death/LVNC	-
+	Symptoms	-
+	Reduced LV systolic function (EF < 50%)	-
+	Reduced thickness of compact layer	-
+	Late gadolinium enhancement on CMR	-
+	T-wave inversion on ECG	-
+	Left bundle branch block on ECG	-
+	Exercise induced VT/AF	-
+	Abnormal diastolic function	-

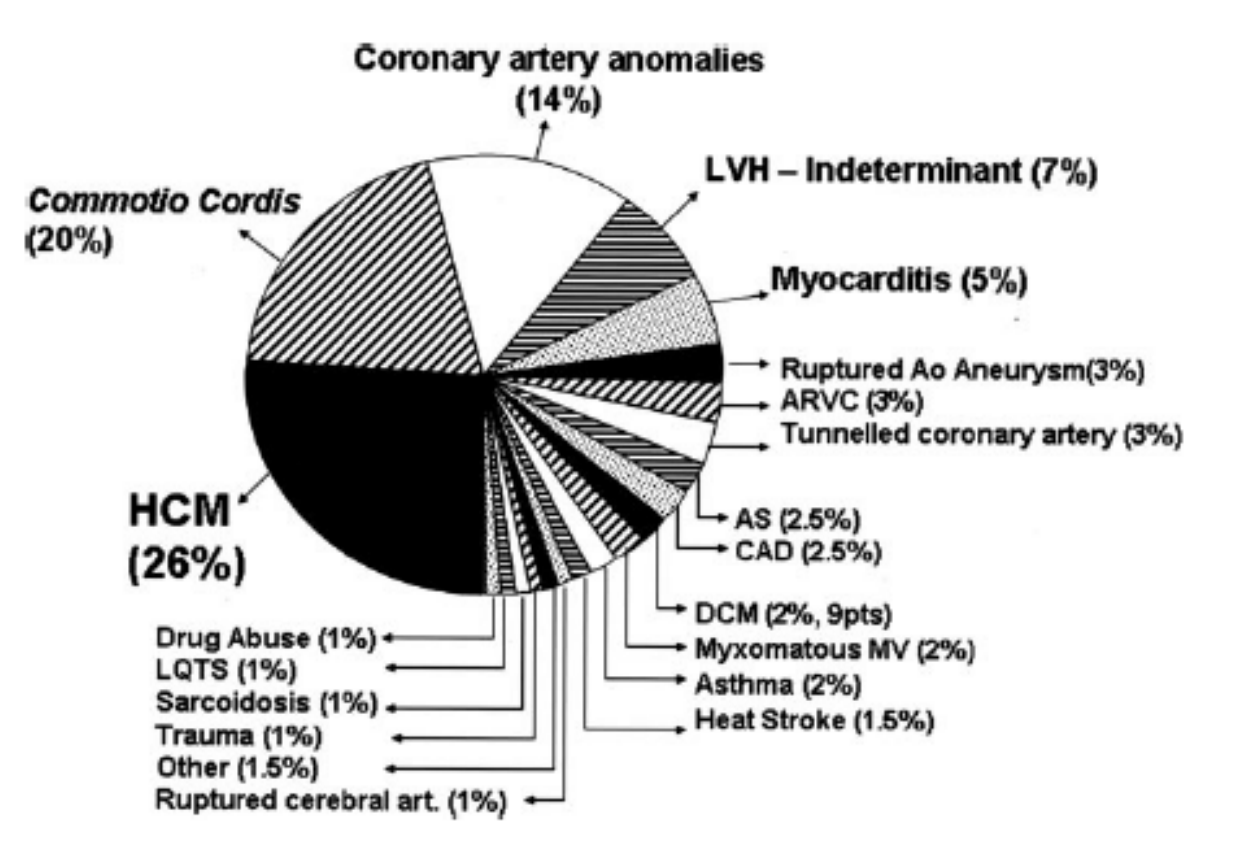
AF, atrial fibrillation; CMR, cardiac magnetic resonance; ECG, electrocardiogram; LV, left ventricle; LVNC, left ventricular non-compaction; VT, ventricular tachycardia.

Athletic Heart: General Concepts

- Athletes generally show relatively small (but statistically significant) increases of 10% to 20% for wall thickness or cavity size, and values in most individual athletes remain within accepted normal limits.

- Strength training is associated with only mildly increased wall thicknesses (often disproportionate relative to cavity size), whereas absolute values
- uncorrected for body surface area usually remain within the accepted normal range (12 mm)

Sudden Death in Athletes: Causes



Preparticipation Athletic ECG Screening: Rationale For

- Sudden death in young healthy athletes is particularly devastating
- Preparticipation ECG can detect risk for SCD
- Restriction from athletic participation may prevent sudden death
- One study yielded data of reduced sudden death after preparticipation ECG screening

Trends in Sudden Cardiovascular Death in Young Competitive Athletes After Implementation of a Preparticipation Screening Program

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Context A nationwide systematic preparticipation athletic screening was introduced in Italy in 1982. The impact of such a program on prevention of sudden cardiovascular death in the athlete remains to be determined.

Objective To analyze trends in incidence rates and cardiovascular causes of sudden death in young competitive athletes in relation to preparticipation screening.

Design, Setting, and Participants A population-based study of trends in sudden cardiovascular death in athletic and nonathletic populations aged 12 to 35 years in the Veneto region of Italy between 1979 and 2004. A parallel study examined trends in cardiovascular causes of disqualification from competitive sports in 42 386 athletes undergoing preparticipation screening at the Center for Sports Medicine in Padua (22 312 in the early screening period [1982-1992] and 20 074 in the late screening period [1993-2004]).

Main Outcome Measures Incidence trends of total cardiovascular and cause-specific sudden death in screened athletes and unscreened nonathletes of the same age range over a 26-year period.

Results During the study period, 55 sudden cardiovascular deaths occurred in screened athletes (1.9 deaths/100 000 person-years) and 265 sudden deaths in unscreened nonathletes (0.79 deaths/100 000 person-years). The annual incidence of sudden cardiovascular death in athletes decreased by 89% (from 3.6/100 000 person-years in 1979-1980 to 0.4/100 000 person-years in 2003-2004; P for trend $<.001$), whereas the incidence of sudden death among the unscreened nonathletic population did not change significantly. The mortality decline started after mandatory screening was implemented and persisted to the late screening period. Compared with the prescreening period (1979-1981), the relative risk of sudden cardiovascular death in athletes was 0.56 in the early screening period (95% CI, 0.29-1.15; $P=.04$) and 0.21 in the late screening period (95% CI, 0.09-0.48; $P=.001$). Most of the reduced mortality was due to fewer cases of sudden death from cardiomyopathies (from 1.50/100 000 person-years in the prescreening period to 0.15/100 000 person-years in the late screening period; P for trend $=.002$). During the study period, 879 athletes (2.0%) were disqualified from competition due to cardiovascular causes at the Center for Sports Medicine: 455 (2.0%) in the early screening period and 424 (2.1%) in the late screening period. The proportion of athletes who were disqualified for cardiomyopathies increased from 20 (4.4%) of 455 in the early screening period to 40 (9.4%) of 424 in the late screening period ($P=.005$).

Conclusions The incidence of sudden cardiovascular death in young competitive athletes has substantially declined in the Veneto region of Italy since the introduction of a nationwide systematic screening. Mortality reduction was predominantly due to a lower incidence of sudden death from cardiomyopathies that paralleled the increasing identification of athletes with cardiomyopathies at preparticipation screening.

JAMA. 2006;296:1593-1601 www.jama.com

THE MAJORITY OF YOUNG ATHletes who die suddenly have previously unsuspected structural heart disease.[1-8] Cardiomyopathies have been consistently implicated as the leading cause of cardiac arrest in young competitive athletes, with hypertrophic cardiomyopathy accounting for more than one third of fatal cases in the United States[1,4-6] and arrhythmogenic right ventricular cardiomyopathy for approximately one fourth of fatal cases in Italy.[2,3,7,8]

Medical evaluation of athletic populations before competition offers the potential to identify asymptomatic athletes with potentially lethal cardiovascular abnormalities and to prevent sudden death through disqualification from competitive sports.[9-13] Italian law mandates that every participant engaged in competitive sports activity must undergo a clinical evaluation and obtain eligibility.[14] Accordingly, a nationwide systematic screening program was launched in Italy in 1982.[15,16] This preparticipation screening essentially based on 12-lead electrocardiogram (ECG) has been shown to be effective in identifying athletes with hypertrophic cardiomyopathy and in

For editorial comment see p 1648.

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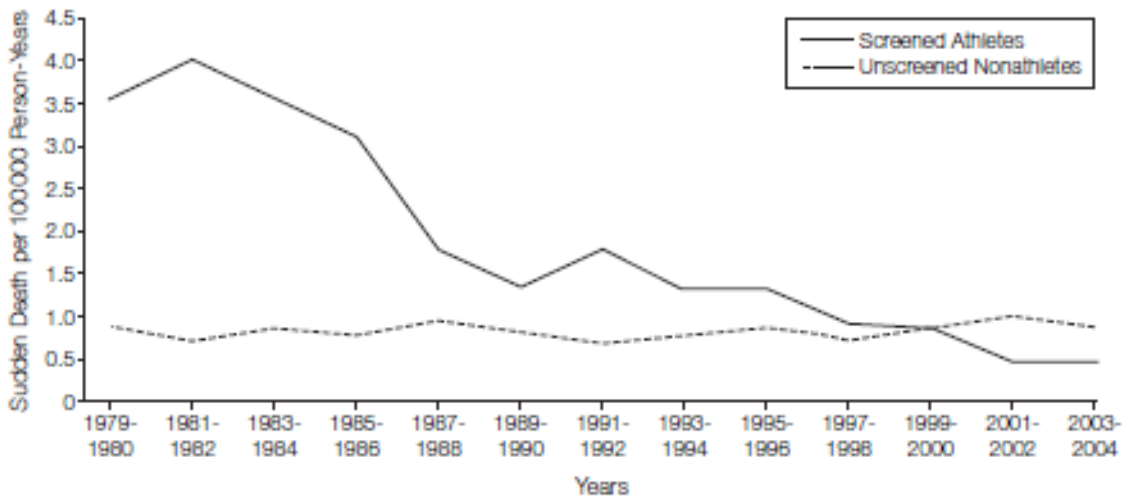
Reduced Sudden Deaths with Preparticipation ECG Screening in Italy

Figure. Annual Incidence Rates of Sudden Cardiovascular Death in Screened Competitive Athletes and Unscreened Nonathletes Aged 12 to 35 Years in the Veneto Region of Italy (1979-2004)



During the study period, the annual incidence of sudden cardiovascular death decreased by 89% in screened athletes (P for trend<.001). In contrast, the incidence rate of sudden cardiovascular death did not demonstrate consistent changes over time in unscreened nonathletes.

Corrado et al; JAMA, 2006

Preparticipation Athletic ECG Screening: Rationale Against

- Sudden death in athletes is extremely rare
  - Less than in non-athletic population
- Minimal data exist that screening identifies risk or that it can be reduced with restriction
- False positives far outnumber true positives
- Elite athletes may be erroneously restricted
- Screening would be very expensive

The Feasibility, Diagnostic Yield, and Learning Curve of Portable Echocardiography for Out-of-Hospital Cardiovascular Disease Screening

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Background: The reduction in the size of full-capability echocardiographic machines facilitates "out-of-hospital" transthoracic echocardiography (TTE). Data documenting the feasibility, yield, and logistical considerations of out-of-hospital TTE for preparticipation evaluation of athletes are sparse.

Methods: A multiyear study was conducted to examine the role of 12-lead electrocardiography for athlete screening in which TTE was used to document or exclude underlying structural heart disease. Using a commercially available portable transthoracic echocardiographic system, the rate of technically adequate imaging, diagnostic yield, and the time required for the completion of TTE (including setup, performance, and interpretation) were examined. TTE was performed in university medical offices and at "out-of-office" athletic facilities. Measurements were recorded during each year of the study to determine the impact of targeted attempts to improve efficiency.

Results: Four hundred sixty-seven of 510 participants had transthoracic echocardiographic images that were technically adequate for complete interpretation (imaging success rate, 92%). Echocardiographic evidence of physiologic, exercise-induced cardiac remodeling was observed in 110 of 510 (22%). Cardiac abnormalities with relevance to sports participation risk were detected in 11 of 508 participants (2.2%). Over 3 years, the average time for the completion of TTE (including setup, imaging, and interpretation) decreased (year 1, 17.4 ± 3 min; year 2, 14.0 ± 2.1 min; year 3, 11.0 ± 1.8 min; $P < .001$). This was driven by a significant decrease in the time required for TTE at out-of-office athletic facilities.

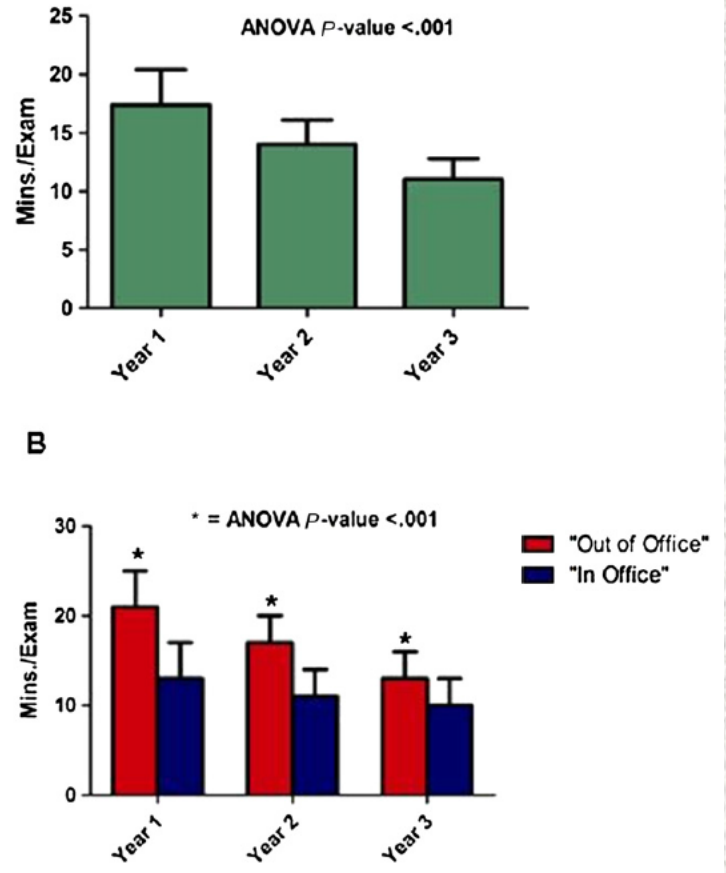
Conclusions: Community-based TTE in athletes is feasible and is associated with a high rate of technically adequate imaging. Importantly, there appears to be a significant learning curve associated with out-of-hospital TTE. (J Am Soc Echocardiogr 2012;25:568-75.)

Preparticipation TTE Screening

Table 2 Focused transthoracic echocardiographic athlete screening protocol

Transducer position/view	Images	Number of Acquisitions
Parasternal long axis	2D image CF Doppler of mitral and aortic valves RV inflow CW Doppler of tricuspid regurgitation	3
Parasternal short axis	2D image of aortic valve CF Doppler of aortic valve 2D image of pulmonic valve CW Doppler of pulmonic valve 2D image at papillary muscle level 2D image at apex	6
Apical four chamber	2D image maximizing both left and right ventricles PW Doppler of transmitral flow PW DTI of lateral mitral annulus PW DTI of septal mitral annulus PW DTI of RV base	5
Apical five chamber	2D image CW Doppler of aortic valve	2
Apical two chamber	2D image	1

Time to Perform TEE (Comprehensive)



Weiner et al; JASE, 2012

Preparticipation TTE Screening

Table 4 Echocardiographic findings from the study population of university athletes

Parameter	Male (n = 300)		Female (n = 197)	
	Normal (n = 209)	Physiologic remodeling (n = 91)	Normal (n = 178)	Physiologic remodeling (n = 19)
Structural parameters				
Interventricular septal thickness (mm)	9.8 ± 0.9	11.6 ± 0.5	8.3 ± 0.7*	10.6 ± 0.5†
LV posterior wall thickness (mm)	10.0 ± 1.2	11.8 ± 1.4	8.6 ± 1.1*	10.7 ± 0.7†
LV inner dimension at end-diastole (mm)	51 ± 3	57 ± 5	42 ± 4*	54 ± 4†
LA diameter (mm)	36 ± 4	40 ± 4	32 ± 3*	38 ± 4
RV end-diastolic diameter (mm)	30 ± 5	36 ± 3	28 ± 4*	33 ± 3†
Functional parameters				
LV ejection fraction (%)	65 ± 7	58 ± 4	68 ± 6	64 ± 6†
Transmitral E wave (cm/sec)	86 ± 16	96 ± 13	81 ± 17	88 ± 12
Transmitral A wave (cm/sec)	40 ± 12	42 ± 14	44 ± 10	44 ± 18
E′ lateral PW (cm/sec)	14.2 ± 5.3	18.8 ± 4.6	13.2 ± 4.2	15.6 ± 3.3†
E′ septal PW (cm/sec)	12.1 ± 3.2	14.1 ± 5.3	12.7 ± 4.1	13.8 ± 4.2
A′ lateral PW (cm/sec)	3.3 ± 2.1	3.9 ± 1.8	4.4 ± 1.6*	4.8 ± 2.0†
A′ septal PW (cm/sec)	4.1 ± 2.0	3.9 ± 2.4	5.3 ± 2.2*	4.6 ± 3.4

LA, Left atrial; *PW*, pulsed-wave.
Data are expressed as mean ± SD.
*$P < .05$ for comparison with male athletes in the normal cardiac structure and function group.
†$P < .05$ for comparison with male athletes in the physiologic remodeling group.

Weiner et al; JASE, 2012

Imaging for Preparticipation Screening

- A screening strategy incorporating multimodality imaging for PPCS in athletes has not been rigorously tested to date and is therefore not recommended.
- Multimodality imaging as a component of PPCS is currently not advised by any major society, other than FIFA, who currently requires a TTE prior to all World Cup events.
- Multimodality imaging plays an important role as secondary or "downstream" testing following the identification of abnormal H&P and/or ECG findings.

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Vol. 54, No. 5
March/April 2012
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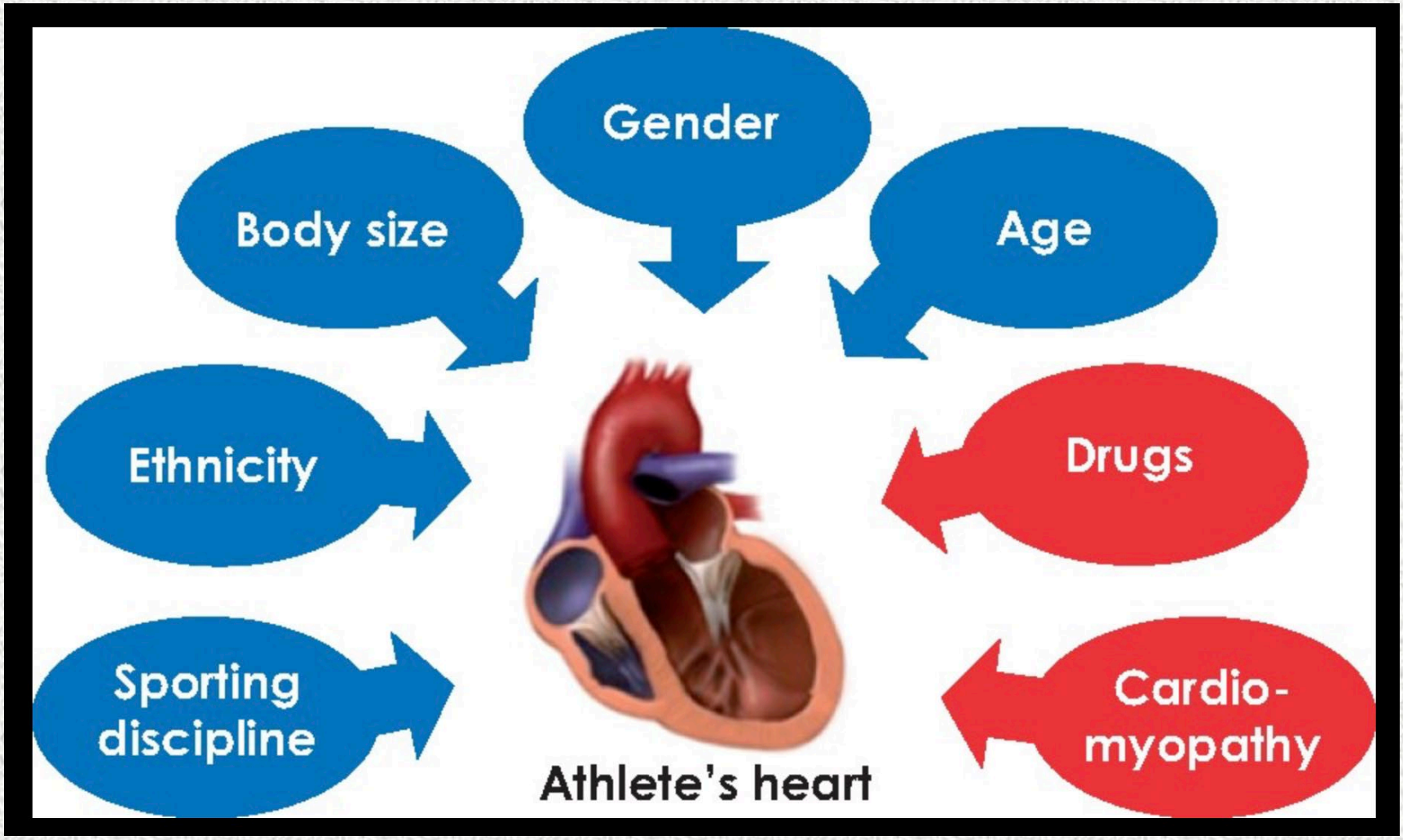
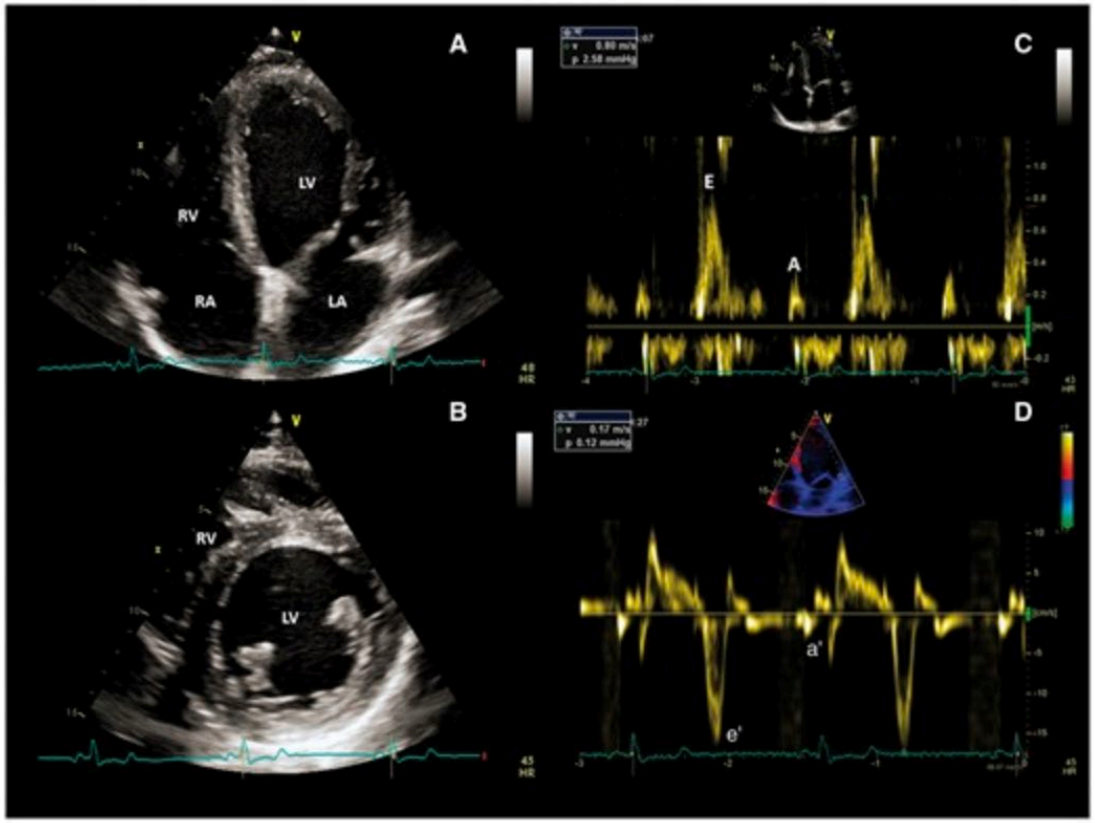
CARDIOVASCULAR CARE OF THE ATHLETIC PATIENT

- The athlete’s heart is commonly (up to 80%) associated with ECG changes such as sinus bradycardia, first-degree AV block, and early repolarization resulting from physiologic adaptation of the cardiac autonomic nervous system to training, i.e. increased vagal tone and/or reduced sympathetic activity. Moreover, the ECG of trained athletes often exhibits pure voltage criteria for LV hypertrophy that reflect the physiological LV remodelling, consisting of increased LV wall thickness and chamber size. Although these ECG changes (i.e. training related) may be considered ‘abnormal’, they do not imply the presence of cardiovascular disorders or an increased cardiovascular risk in the athlete. These ECG abnormalities should be clearly separated from training unrelated ECG patterns (present in <5%), such as ST-segment depression and T-wave inversion, pathologic Q waves, major intraventricular conduction defects, ventricular pre-excitation, long or short QT interval, and ventricular arrhythmias, which may be an expression of cardiovascular disorders, notably cardiomyopathies and cardiac ion channel diseases, with potential risk of SCD during sports.


Gender
Body size
Age
Ethnicity
Drugs
Sporting discipline
Cardio-myopathy
Athlete's heart



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Twenty-two years-old male competitive endurance athlete (swimmer). Panel A: Apical 4-chamber and (Panel B) parasternal short-axis views, showing left ventricular (LV) hypertrophy, with symmetric increase of both wall thickness and LV internal cavity diameters. Panel C: Standard Doppler transmitral inflow pattern, showing a 'supranormal' early-diastolic function, with increased E velocity and E/A ratio. Panel D: Pulsed Tissue Doppler pattern of LV lateral wall, highlighting the enhanced early-diastolic myocardial function, i.e. increased e' velocity. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.