



**Cardiostim 2006**

**15th World Congress in Cardiac Electrophysiology and Cardiac Techniques**

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# Predicting the Sudden Death in the Young Athletes

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# La morte improvvisa nello sportivo

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# ATLETI COMPETITIVI CON ARITMIE

Popolazione studiata dal 1974 all'aprile 2004

Atleti	N	Maschi	Femmine	Età media (anni)	Follow-up (mesi) min-max	N con SD	N con CA
Atleti totali	2640	2286	354	21.5	3-190	24 (0.9%)	38 (1.4%)
Atleti di elite	345	298	47	24.4	3-180	6 (1.7%)	7 (2.0%)

# ATLETI COMPETITIVI CON ARITMIE

Popolazione studiata dal 1974 all'aprile 2004

**Atleti con arresto cardiaco resuscitato e fatale**  
**Condizioni nelle quali si è verificato l'evento**

Attività	CA	SD	Totale
A riposo	2	4	6 (9.6%)
Durante esercizio fisico	36	20	56 (90.4%)
- allenamento	19	10	26 (51.8%)

# ATLETI COMPETITIVI CON ARITMIE

Popolazione studiata dal 1974 all'aprile 2004

## Atleti con arresto cardiaco resuscitato e fatale Evento aritmico determinante

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TV / FV	44	} - 57 (91.9%) FV
T.d.P.	2	
Fibrillazione atriale preeccitata	11	
Blocco AV parossistico	4	} - 5 (8.1%) Asistolia
Commotio cordis asistolica	1	

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# ATLETI COMPETITIVI CON ARITMIE

Popolazione studiata dal 1974 all'aprile 2004

## PATOLOGIA ARITMOGENA DI ATLETI CON ARRESTO CARDIACO (CA) RESUSCITATO E FATALE

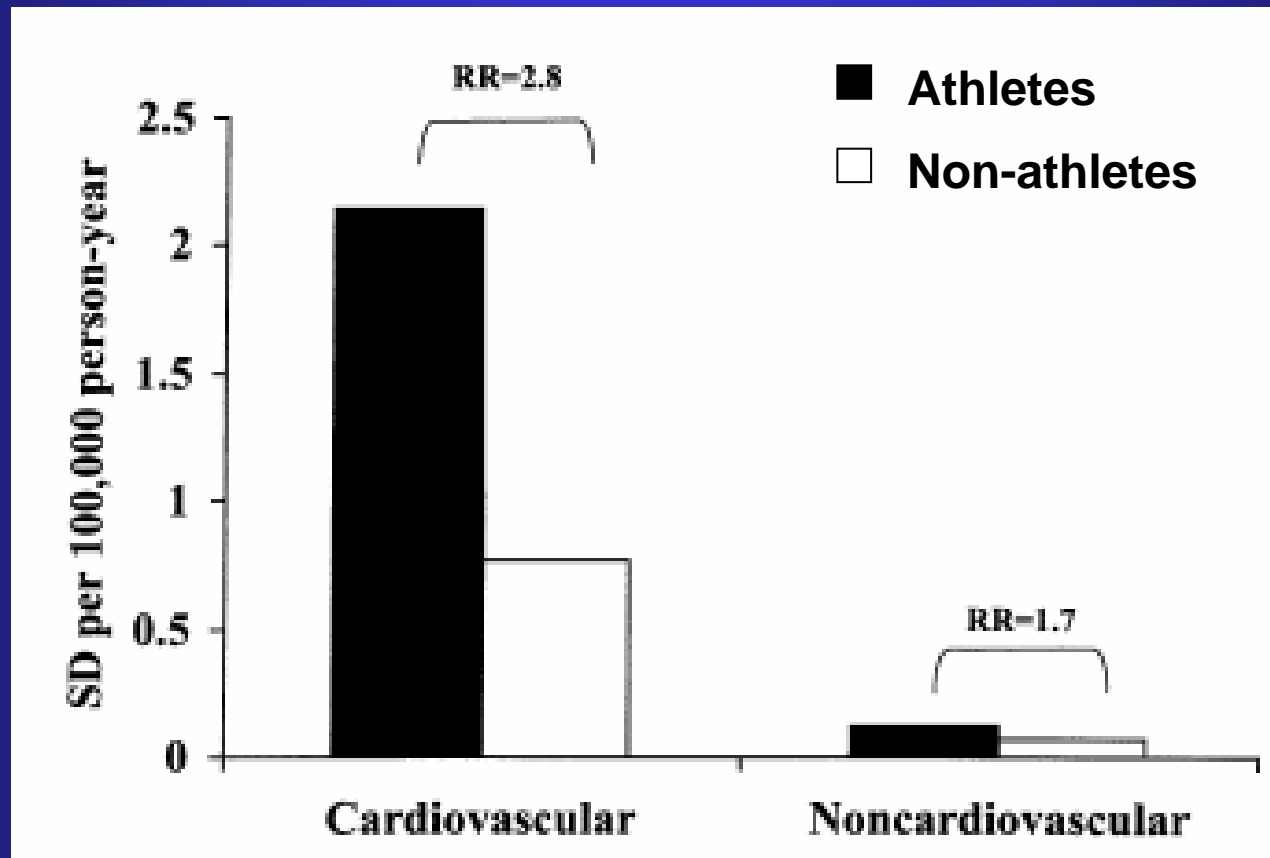
	Fatale CA		Resuscitato CA		Totale CA	
	Atleti	%	Atleti	%	Atleti	%
ARVD/C	6	25	9	23.6	15	24.2
WPW	2	8.3	7	18.4	9	14.5
Miocardite	6	25	3	7.9	9	14.5
Coronaropatia	4	16.6	3 (*)	7.9	7	11.3
Cardiomiopatia dilatativa	3	12.5	4	10.6	7	11.3
Malattia di Lev-Lenegre	--	--	4	10.6	4	6.4
Cardiomiopatia ipertrofica	--	--	3	7.9	3	4.8
Commotio cordis	1	4.16	1	2.6	2	3.2
"Non-compact myocardium" con TVPC(=)	--	--	1	2.6	1	1.6
Prolasso valvolare mitralico	2	8.3	1	2.6	3	4.8
Sindrome del QT lungo	-	--	1	2.6	1	1.6
Cardiopatía aritmogena primaria	--	--	1	2.6	1	1.6
TOTALE	24**		38		62	

(\*) uno congenito

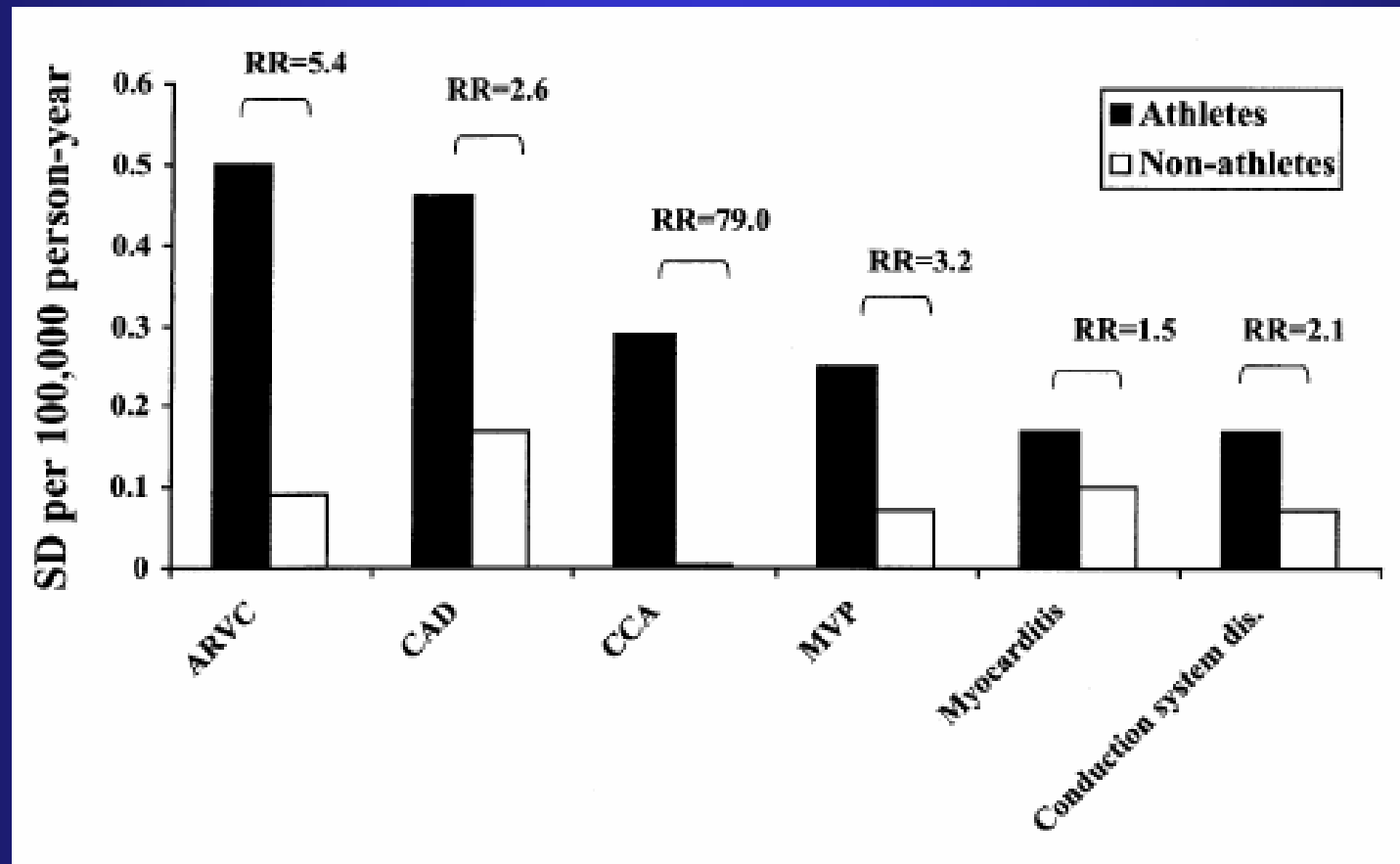
(\*\*) 4 con precedente CA

(=)TVPC: tachicardia ventricolare polimorfa catecolaminergica

# Incidence and relative risk of sudden death among athletes and non-athletes from cardiovascular and non-cardiovascular causes



# Incidence and relative risk of sudden death for specific cardiovascular causes among athletes and non-athletes



## Does sports activity enhance the risk of sudden death in adolescents and young adults?

Sports, per se, is not a cause of increased mortality; rather, it acts as a trigger for cardiac arrest in the presence of underlying cardiovascular diseases predisposing to life-threatening ventricular arrhythmias during physical exercise - namely, ARVC, premature CAD, and congenital coronary artery anomaly.

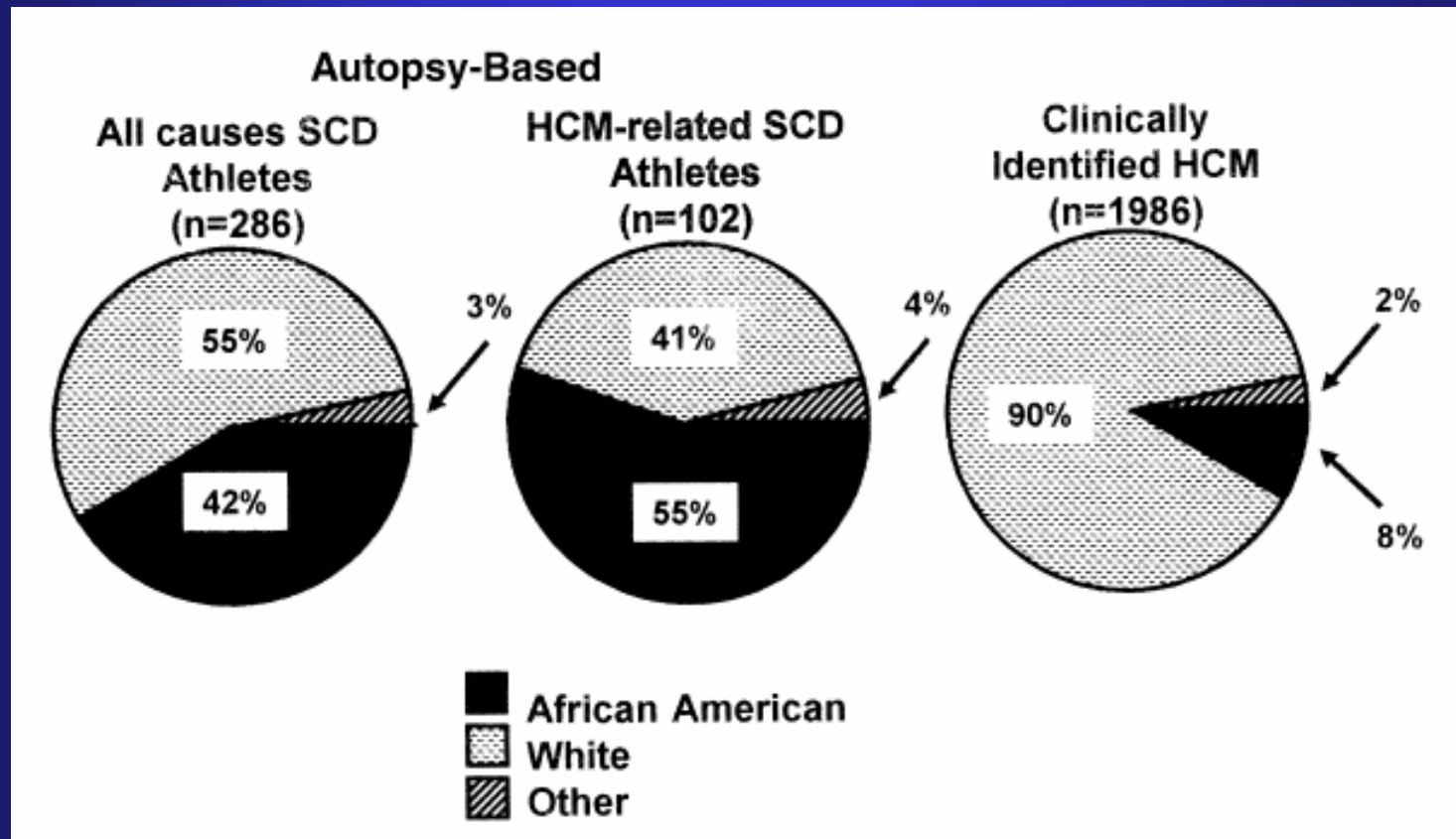
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## Hypertrophic Cardiomyopathy

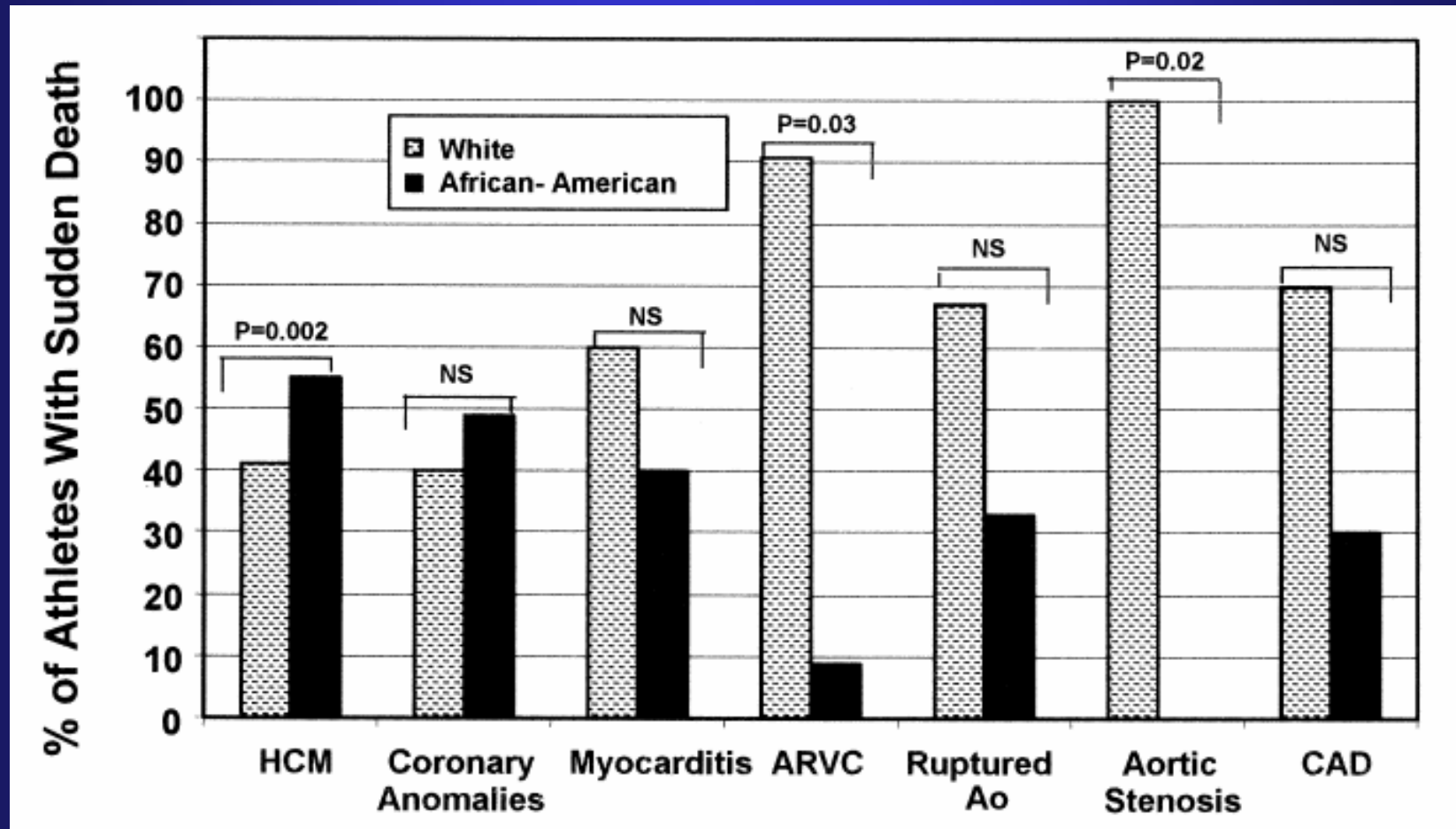
# Relationship of Race to Sudden Cardiac Death in Competitive Athletes With Hypertrophic Cardiomyopathy

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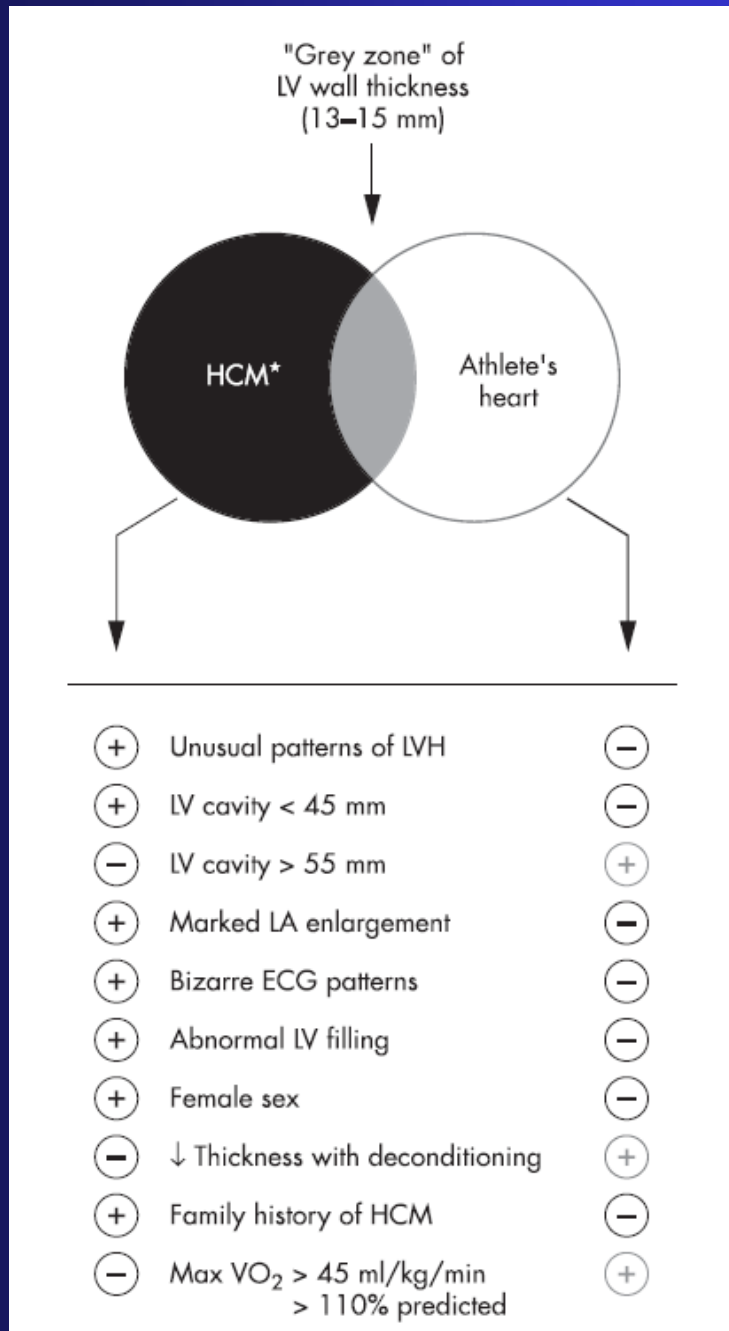
# Relationship of race to sudden cardiac death in competitive athletes with hypertrophic cardiomyopathy



# Impact of race on cardiovascular causes of sudden death in competitive athletes

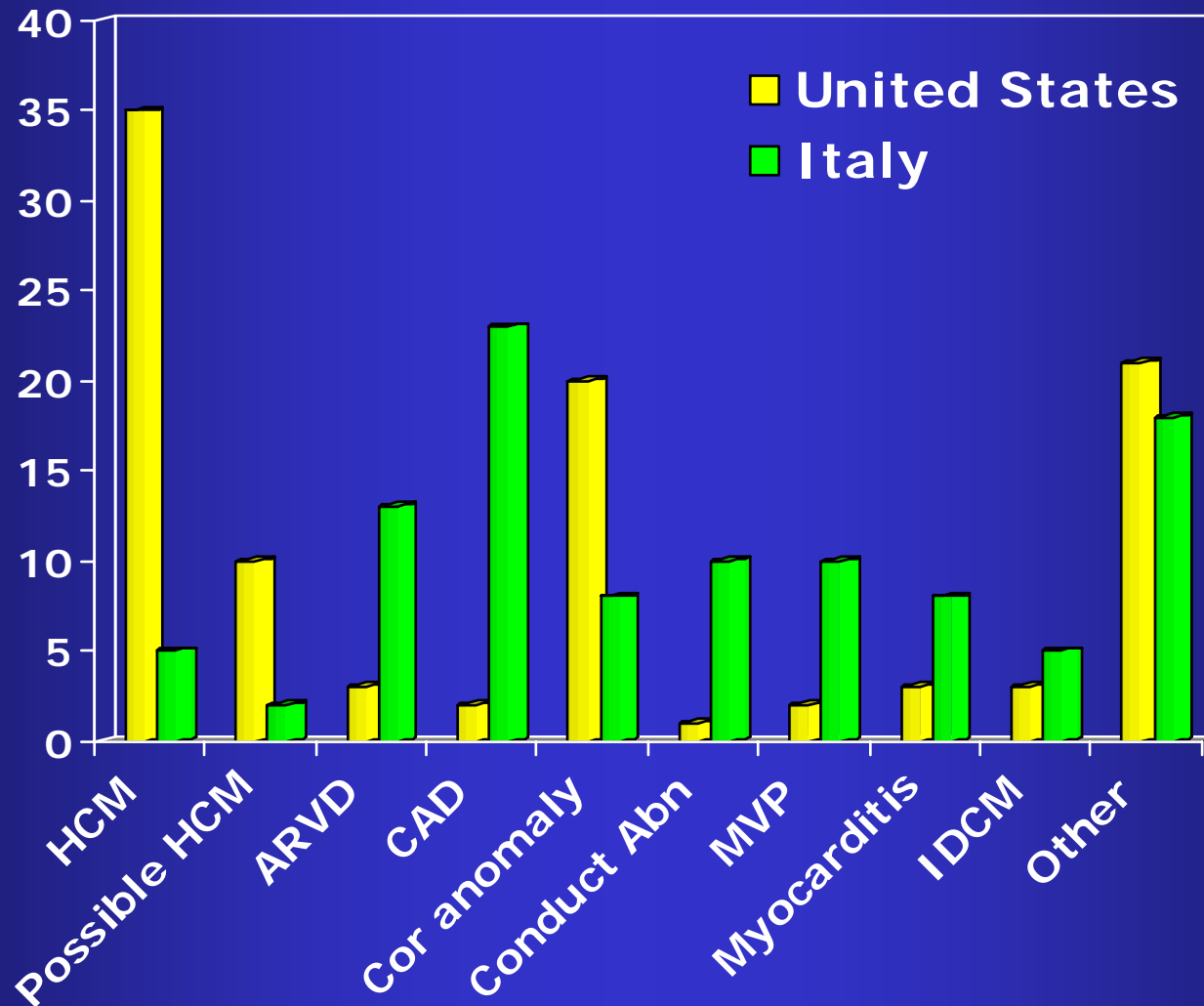


# Distinguishing hypertrophic cardiomyopathy from athlete's heart: a clinical problem of increasing magnitude and significance



Criteria used to distinguish hypertrophic cardiomyopathy (HCM) from athlete's heart when the left ventricular (LV) wall thickness is within the shaded "grey zone" of overlap, consistent with both diagnoses. Q indicates decreased; LA, left atrial; LVH, left ventricular hypertrophy.

# Comparison of incidence of underlying heart disease in young athletes dying suddenly in the United States and Italy



## Normal ECG or ECG with Minor Alteration

- Increased PR interval duration ( $> 0.20$  sec)
- Mild increased R or S wave voltage (25 to 29 mm)
- Early repolarization (ST elevation  $> 2$  mm in  $> 2$  leads)
- Incomplete right bundle branch block (RSR' pattern in V1 e V2 of  $< 0.12$  sec in duration)
- Sinus bradycardia  $< 60$  bpm

# Mildly Abnormal ECG

Mildly abnormal ECGs were those that were compatible with the presence of cardiovascular disease:

- Increased R or S wave voltage (30 to 34 mm) in any lead
- Q waves 2 to 3 mm in depth and present in >2 leads
- Repolarization pattern with either flat, minimally inverted, or particularly (ie, > 15 mm) T waves in >2 leads
- Abnormal R wave progression in the anterior precordial leads
- Right bundle branch block ( RSR pattern >0.12 in V1 and V2)
- Right atrial enlargement ( peaked P waves >2.5 mm in leads II, III, or V1)
- Left atrial enlargement ( prolonged positive P wave in lead II and/or deep, prolonged negative P wave in V1)
- Short PR interval ( <0.12 s)

# Distinctly Abnormal ECG

Distinctly abnormal ECGs were those that were strongly suggestive of cardiovascular disease:

- striking increase in R or S wave voltage ( $> 35$  mm) in any lead
- Q waves  $> 4$  mm in depth and present in  $> 2$  leads
- ripolarization pattern with inverted T wave  $> 2$  mm in  $> 2$  leads
- left bundle branch block
- marked left ( $< -30^\circ$ ) or right ( $> 110^\circ$ ) QRS axis deviation
- WPW pattern

# Distribution of ECG Abnormalities in 1005 Trained Athletes

Distinctly Abnormal ECG (n=145)		Mildly Abnormal ECG (n=257)		ECG Normal or With Minor Alterations (n=603)	
Parameter	n (%)	Parameter	n (%)	Parameter	n (%)
R or S wave ≥35 mm	92 (63)	R or S wave 30–34 mm	141 (55)	R or S wave 25–29 mm	170 (28)
Negative T wave	27 (19)	Flat/tall T wave	59 (23)	J-junction elevation	144 (24)
Q wave ≥4 mm	17 (12)	Q wave 2–3 mm	69 (27)	Incomplete RBBB	122 (20)
LAD	11 (8)	LAE	9 (4)	PR interval >0.20 s	75 (12)
RAD	8 (6)	RAE	2 (0.8)	Sinus bradycardia <60 bpm	369 (61)
WPW	3 (2)	Incomplete R wave progression V <sub>1</sub> to V <sub>3</sub>	40 (16)		
LBBB	2 (1)	PR interval ≤0.12 s	9 (4)		
		RBBB	2 (0.8)		

J-junction indicates early repolarization pattern; LAD, left axis deviation ( $\leq -30^\circ$ ); LBBB, left bundle branch block; LAE, left atrial enlargement; Incomplete R wave progression V<sub>1</sub> to V<sub>3</sub>, abnormal pattern of R wave progression in the anterior precordial leads V<sub>1</sub> to V<sub>3</sub>; RAD, right axis deviation ( $\geq 110^\circ$ ); RAE, right atrial enlargement; RBBB, right bundle branch block; and WPW, Wolff-Parkinson-White pattern.

# Abnormal ECGs in Athletes

53 athletes with cardiovascular abnormalities

Abnormal ECGs  
n 27

Normal ECGs  
n 26

## Cardiac abnormalities in ECG subgroup

Distinctly Abnormal ECGs	Mildly Abnormal ECGs	Normal ECGs
70%	5% (p<0.001)	4% (p<0.001)

# ECG abnormalities in various disease states

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## Diagnosis of heart disease

## ECG abnormalities

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**Arrhythmogenic right ventricular dysplasia**

T-wave inversions anteriorly  
Epsilon wave  
RBBB (complete or incomplete)  
Rarely normal

**Hypertrophic cardiomyopathy**

Left ventricular hypertrophy  
Pseudoinfarct with Q-waves anteriorly  
Rarely normal

**Idiopathic dilated cardiomyopathy**

LBBB  
Prolonged QT  
Can be normal

**Long QT syndrome**

Prolonged QT  
Abnormal appearance of ST segment

**Brugada syndrome**

RBBB (complete or incomplete)  
ST elevation anteriorly  
Changes can vary with time

**Anomalous coronary artery  
Coronary artery disease**

Typically no abnormalities  
Typically no abnormalities  
Q-waves  
ST segment abnormalities

**Wolff-Parkinson-White**

Short PR  
Delta Waves  
Pseudoinfarct patterns

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## Cardiovascular Conditions Associated with Arrhythmias and Sudden Death in the Athlete

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Hypertrophic cardiomyopathy  
Atherosclerotic coronary artery disease  
Arrhythmogenic right ventricular dysplasia  
Anomalous origin of coronary artery  
Long QT syndrome  
Wolff-Parkinson-White syndrome  
Idiopathic ventricular fibrillation  
Brugada syndrome  
Myocardial bridge  
Aortic valve stenosis  
Subvalvular aortic stenosis  
Pulmonary hypertension  
Congenital heart disease  
Myocarditis  
Dilated cardiomyopathy  
Marfan syndrome  
Commotio cordis

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# Causes of sudden cardiac death in young athletes

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Hypertrophic cardiomyopathy (responsible for up to 50% of cases)

Arrhythmogenic right ventricular cardiomyopathy

Congenital coronary artery anomalies

Premature coronary artery disease

Wolff-Parkinson-White syndrome

Long QT and Brugada syndromes

Idiopathic dilated cardiomyopathy

Myocarditis

Marfan's syndrome

Congenital aortic stenosis

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# Marked Ventricular Repolarization Abnormalities in Highly Trained Athletes' Electrocardiograms: Clinical and Prognostic Implications

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Lluís Bernà, MD,† Teresa Prat, MD\*

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In conclusion, despite the significant alterations found in ECG at rest, the performance in the exercise test and the results of echocardiography, myocardial perfusion and antimyosin studies, together with the lack of events during the long-term follow-up, indicate that **marked repolarization abnormalities (MRA) have no pathological implications and should not, therefore, preclude physical training and competitive availability when a cardiac pathology can be excluded.** Thus, the presence of similar ECG alterations must lead to a careful examination of the athlete. The cause of MRA is unknown, and training is likely the trigger. Further research is needed to determine the origin.

# The significance of QT dispersion in endurance athletes

	Group I (n=60)	Group II (n=45)	Group III (n=42)	Group IV (n=30)	P
Maximum QT (ms)	459±4	460±5	414±4	388±8 <sup>a</sup>	<0.001
Maximum QTc (ms)	463±4	467±6	409±3	395±9 <sup>b</sup>	<0.001
QRS duration (ms)	94±12	92±10	82±15	72±10	<0.001
Heart rate (beats/min)	73±3	70±4	56±5	72±5	<0.001
QT dispersion (ms)	78±14	72±15	53±5	43±5	<0.001
QTc dispersion (ms)	81±13	74±13	51±8	45±5	<0.001

<sup>a</sup> P<0.05 versus athletes.

<sup>b</sup> P>0.05 versus athletes.

- Group I** essential hypertension
- Group II** isolated valvular aortic stenosis
- Group III** endurance athletes
- Group IV** healthy subjects

# **QT Interval and QT Dispersion in Endurance Athletes and in Power Athletes Using Large Doses of Anabolic Steroids**

Anu Stolt, MD, Tuomo Karila, MD, Matti Viitasalo, MD, Matti Mäntysaari, MD, Urho M. Kujala, MD, and Jouko Karjalainen, MD

In conclusion, physiologic adaptive LV hypertrophy does not increase QT dispersion in endurance athletes, although the QT interval is prolonged due to increased vagal tone. However, **power training combined with use of large doses of anabolic steroids is associated with increased QT dispersion despite short QT intervals.** This possibly reflects altered myocardial structure in the hypertrophied heart and **increased risk of malignant arrhythmias.**

# Differences between athletes with early repolarization and patients with the Brugada syndrome

	Athletes with early repolarization	Patients with Brugada syndrome
Subjects	139	23
Heart rate (beats . min <sup>-1</sup> )	50.8 ± 6.9	76.9 ± 19.3*
Sokolov index (mm)	46.5 ± 11.6	23.3 ± 8.2*
QRS duration (s)	0.095 ± 0.011	0.116 ± 0.019*
ST elevation (mm)	2.3 ± 0.6	4.4 ± 1.9*
QT (s)	0.427 ± 0.038	0.378 ± 0.043*
QTc (s)	0.39 ± 0.031	0.424 ± 0.049*

\* $P \leq 0.001$ .

ST elevation does not usually extend beyond lead V4 and is rarely associated with giant positive T waves.

# Athlete's heart

## Rhythm disturbances

- ▶ Sinus bradycardia
- ▶ Sinus arrhythmia, mostly related to respiration
- ▶ Sinus arrest, with ectopic escape beat or rhythm, or resumption of sinus rhythm
- ▶ Wandering atrial pacemaker
- ▶ Other rhythms such as junctional rhythm, coronary sinus rhythm.

## Atrioventricular block

- ▶ First degree atrioventricular block
- ▶ Second degree atrioventricular (AV) block, Möbitz type I, or Wenckebach-type
- ▶ Atrioventricular dissociation.

Higher grade AV blocks have rarely been observed in athletes; they may be indicative of underlying heart disease and are an indication for further evaluation.

**Table 3** Heart rate and frequency of cardiac events on ambulatory monitoring of athletes and non-athletes

	Controls	Athletes	p Values
Number	35	35	
Heart rate (beats/min)			
Lowest nocturnal	45 (33–63)	38 (24–48)	<0.001
Sinus pause			
PP >2.0 s	5.7%	37.1%	<0.01
Longest PP (s)	2.60	2.76	–
Atrioventricular block			
First degree (PR >0.22 s)	14.3 %	37.1%	<0.05
Longest PR interval (s)	0.40	0.54	–
Second degree			
Möbitz type I (Wenckebach type)	5.7%	22.9%	<0.05
Möbitz type II	0%	8.6%	
Atrioventricular dissociation	0%	20%	

# Long-Term Clinical Significance of Frequent and Complex Ventricular Tachyarrhythmias in Trained Athletes

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*Rome, Italy, and Minneapolis, Minnesota*

**Table 1.** Demographic and Clinical Data in 355 Competitive Athletes With Ventricular Tachyarrhythmias

	Group A (≥2,000 PVDs and ≥1 NSVT)	Group B* (≥100 to <2,000 PVDs)	Group C* (<100 PVDs)	p Value
No. athletes	71	153	131	
Age	24 ± 10	24 ± 10	25 ± 11	NS
Male:Female	51:20	120:33	102:29	NS
Palpitations†	8 (11%)	10 (6%)	0	0.0013
12-lead ECG abnormalities‡	15 (21%)	5 (3%)	2 (1.5%)	< 0.001¶
Echo abnormalities§	21 (30%)	8 (5%)	0	< 0.001¶

\*NSVT was absent in these subgroups. †Defined as a frequent sensation of irregular heart beat (also during exercise), unassociated with dizziness. ‡Increased R and/or S wave ≥30 min, inverted T waves (≥2 leads), deep Q waves (≥2 mm), LBBB or RBBB, left axis deviation. §Mitral valve leaflet redundancy and prolapse (n = 11); dilated cardiomyopathy (end-diastolic dimension ≥60 mm) associated with systolic left ventricular dysfunction (ejection fraction ≤45% and/or segmental wall motion abnormalities) (n = 4); segmental wall motion abnormalities consistent with either ARVC and myocarditis (n = 11); and bicuspid aortic valve without aortic regurgitation (n = 3); ||Group A versus Group C and Group B versus Group C (p < 0.05); ¶Group A versus Group B and Group A versus Group C (p < 0.05).

ECG = electrocardiogram; Echo = two-dimensional echocardiography; NSVT = nonsustained ventricular tachycardia; PVDs = premature ventricular depolarizations.

## Prevalence of structural cardiovascular abnormalities in 355 competitive athletes with ventricular tachyarrhythmias

	Group A ( $\geq 2,000$ PVDs and $\geq 1$ NSVT)	Group B* ( $\geq 100$ to $< 2,000$ PVDs)	Group C* ( $< 100$ PVDs)	p Value
No. of athletes	71	153	131	
ARVC	7 (10%)	0	0	$< 0.001$ †
MVP	6 (9%)	5 (3%)	0	0.0042‡
Myocarditis	4 (5.5%)	0	0	0.0003†
DCM	4 (5.5%)	0	0	0.0003†
Totals	21 (30%)	5 (3%)	0	$< 0.001$ †

\*NSVT was absent in these subgroups; †Group A versus Group B and Group A versus Group C ( $p < 0.05$ ); and ‡Group A versus Group C ( $p < 0.05$ ).

ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy; MVP = mitral valve prolapse; Other abbreviations as in Table 1.

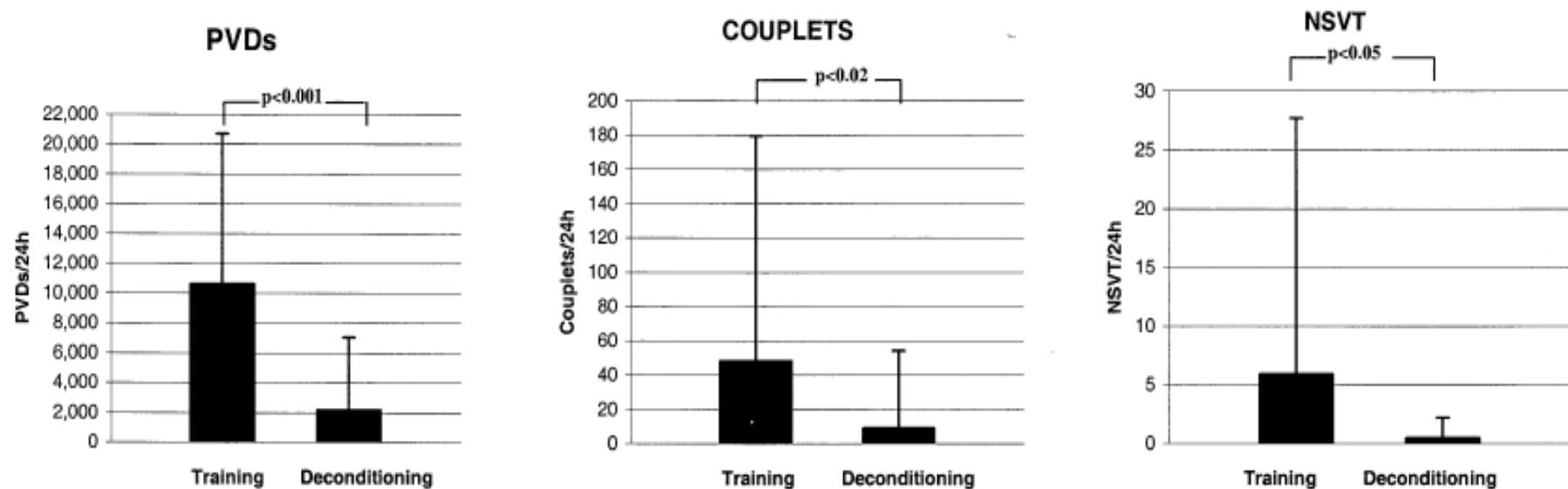
## Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes

These findings underscore the view that ventricular tachyarrhythmias in athletes subjected to intense physical exertion during training and competition do not usually develop unfavorable consequences and that, in athletes without cardiovascular abnormalities, they would appear to represent another previously unappreciated expression of the "athlete's heart syndrome," probably insufficient per se to definitively dictate withdrawal from competitive sports.

# Impact of Physical Deconditioning on Ventricular Tachyarrhythmias in Trained Athletes

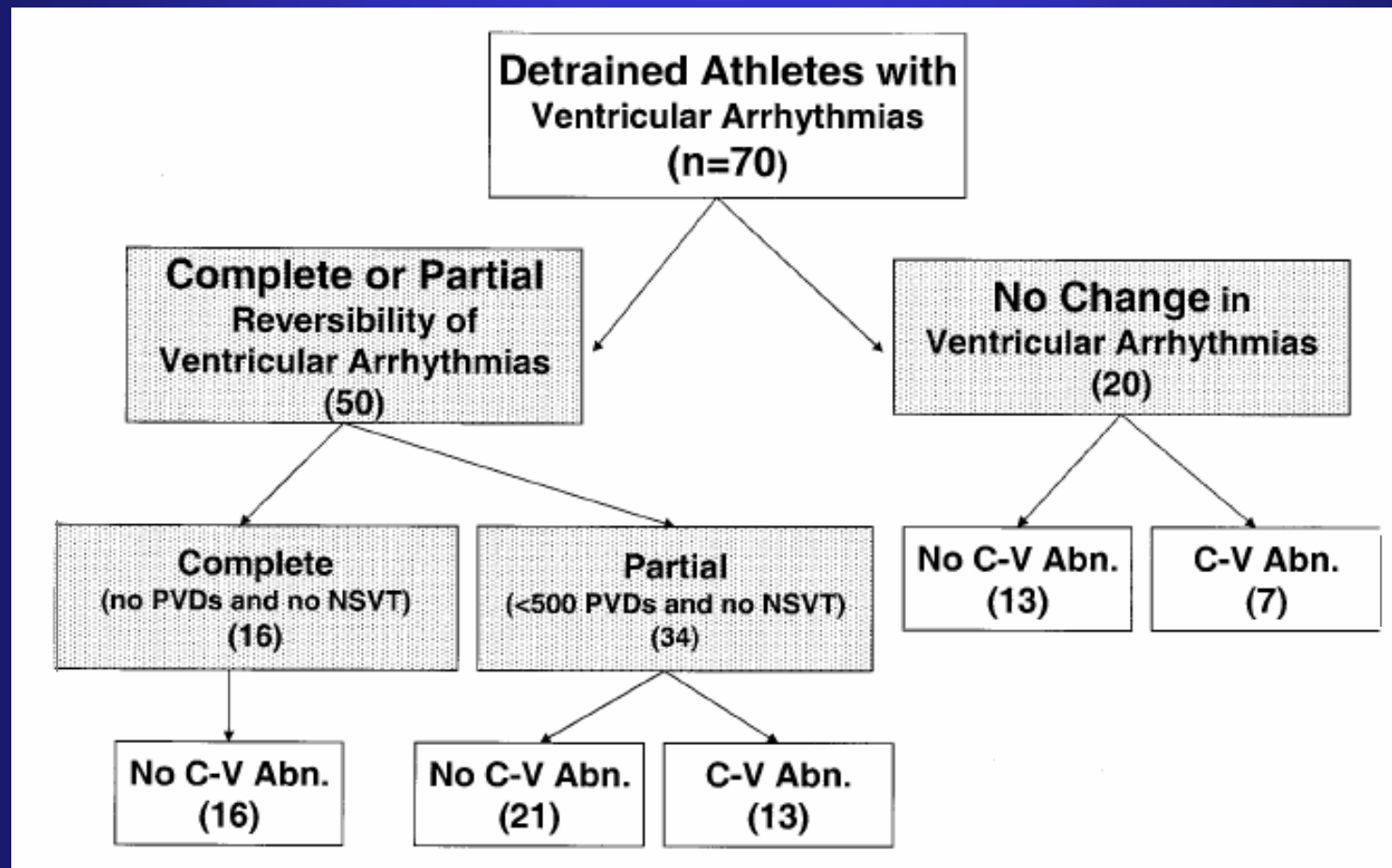
Alessandro Biffi, MD,\* Barry J. Maron, MD, FACC,‡ Luisa Verdile, MD,\* Fredrick Fernando, MD,\* Antonio Spataro, MD,\* Giuseppe Marcello, MD,\* Roberto Ciardo, MD,\* Fabrizio Ammirati, MD,† Furio Colivicchi, MD,† Antonio Pelliccia, MD\*

Rome, Italy; and Minneapolis, Minnesota



**Figure 1.** Number of premature ventricular depolarizations (PVD), ventricular couplets, and bursts of non-sustained ventricular tachycardia (NSVT) during 24-h Holter electrocardiogram recording at peak training and after the period of deconditioning in 70 trained athletes.

# Effect of deconditioning on frequent and/or complex ventricular tachyarrhythmias in trained athletes



## Impact of physical deconditioning on ventricular tachyarrhythmias in trained athletes

Frequent and/or complex ventricular tachyarrhythmias in trained athletes (with and without cardiovascular abnormalities) are sensitive to brief periods of deconditioning. **In athletes with heart disease**, the resolution of such arrhythmias with detraining may represent a mechanism by which risk for sudden death is reduced. Conversely, **in athletes without cardiovascular abnormalities**, reduction in frequency of ventricular tachyarrhythmias and the absence of cardiac events in the follow-up support the benign clinical nature of these rhythm disturbances as another expression of athlete's heart.

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

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

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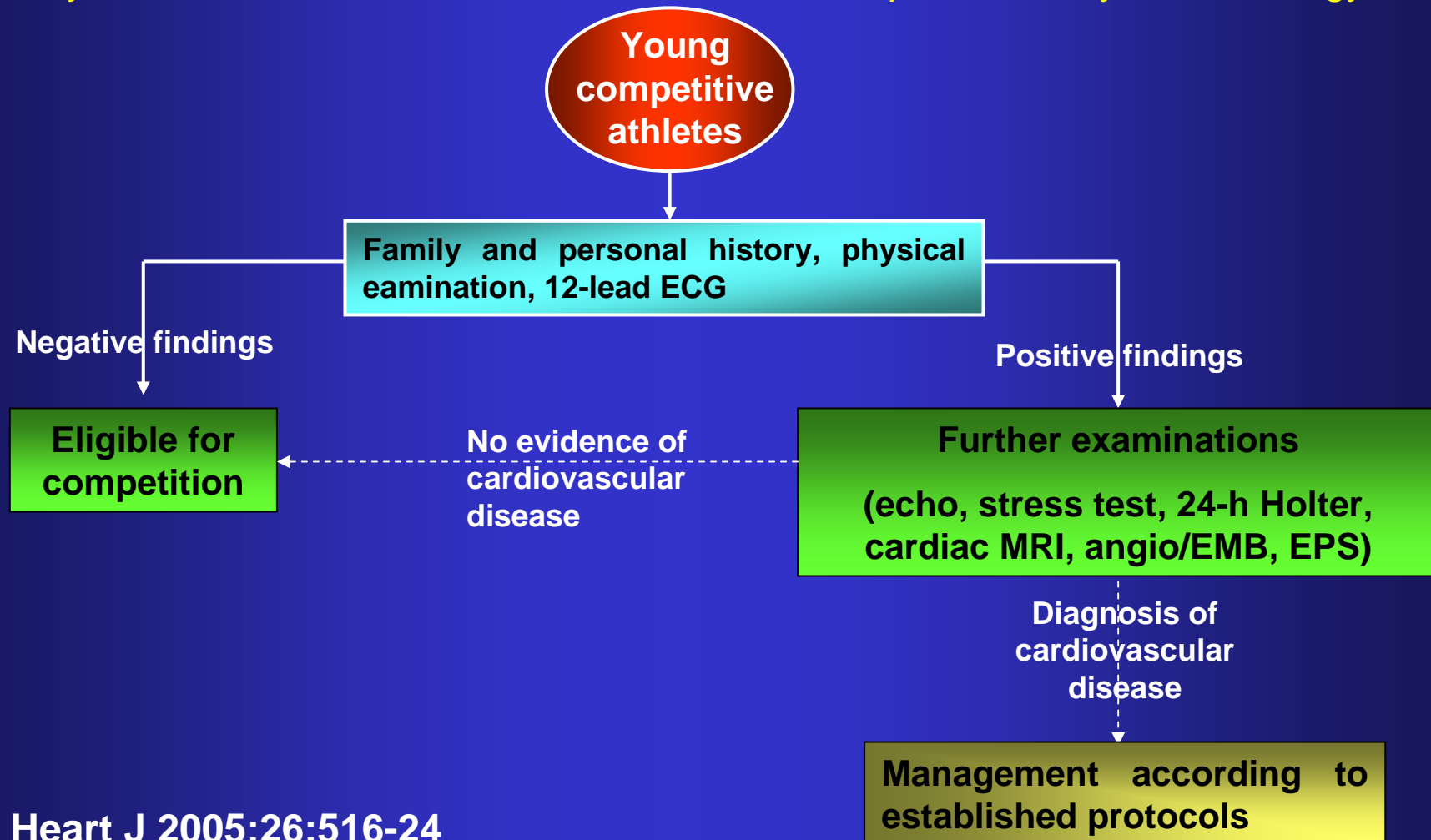
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<sup>19</sup> Pathological Anatomy, University of Padova, Italy

# Proposed screening protocol for young competitive athletes

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



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

The family history is considered positive when close relative(s) had experienced a premature heart attack or sudden death (<55 years of age in males and <65 years in females), or in the presence of a family history of cardiomyopathy, Marfan syndrome, long QT syndrome, Brugada syndrome, severe arrhythmias, coronary artery disease, or other disabling cardiovascular diseases.

The personal history is considered positive in the case of exertional chest pain or discomfort, syncope or near-syncope, irregular heartbeat or palpitations, and in the presence of shortness of breath, or fatigue out of proportion to the degree of exertion.

## Current recommendations regarding athletic participation for athletes with cardiac conditions causing sudden death in young athletes

Diagnosis	Recommendation
HCM	<ol style="list-style-type: none"> <li>1 Should not participate in most competitive sports with the possible exception of those of low intensity</li> <li>2 Older athletes may participate depending on risk factor stratification</li> </ol>
ARVC	Should not participate in competitive sports
Coronary artery anomalies	<ol style="list-style-type: none"> <li>1 Should be excluded from competitive sports</li> <li>2 Athletes without ischaemia on exercise stress testing may participate in sports &gt;6 months after surgical treatment</li> </ol>
WPW	<ol style="list-style-type: none"> <li>1 Athletes without structural heart disease, palpitations or tachycardia can participate in all competitive sports</li> <li>2 Athletes with re-entrant tachycardia should be treated with radiofrequency ablation</li> <li>3 Athletes with atrial flutter/fibrillation with slow accessory pathway conduction and no syncope can participate freely. Those with syncope or fast accessory pathway conduction should be treated with radiofrequency ablation</li> <li>4 Athletes with successful ablation of accessory pathway who are asymptomatic, have normal atrioventricular conduction on electrophysiological study, and have no recurrence of tachycardia for 3–6 months can participate in all sports</li> </ol>
Ion channelopathies*	Should not participate in competitive sports
IDCM	Should not participate in competitive sports

ARVC, arrhythmogenic right ventricular cardiomyopathy; HCM, hypertrophic cardiomyopathy; IDCM, idiopathic dilated cardiomyopathy; WPW, Wolff-Parkinson-White syndrome.  
 \*Long QT and Brugada syndromes.  
 †Low risk defined by normal systolic function, normal exercise tolerance for age, no ischaemia on exercise stress testing, no exercise induced complex ventricular arrhythmia, and no haemodynamically significant coronary artery stenosis.

## Current recommendations regarding athletic participation for athletes with cardiac conditions causing sudden death in young athletes

Diagnosis	Recommendation
Premature coronary artery disease	<ol style="list-style-type: none"> <li>1 If considered low risk†, can participate in low and moderate intensity sports. Should be re-evaluated annually</li> <li>2 If considered to be at high risk†, may only participate in low intensity sports. Should be re-evaluated every 6 months</li> </ol>
Marfan's syndrome	<ol style="list-style-type: none"> <li>1 Athletes without a family history of premature sudden cardiac death and without aortic root dilatation can participate in low and moderate intensity competitive sports. Serial 6 monthly monitoring of aortic root should be repeated</li> <li>2 Athletes with aortic root dilatation can participate in low intensity sports only</li> </ol>
Myocarditis	<ol style="list-style-type: none"> <li>1 Should be withdrawn from competitive sports for about 6 months after onset of symptoms for convalescence</li> <li>2 May return to competitive sports after normalisation of ventricular function and absence of clinically relevant arrhythmias on ambulatory ECG monitoring</li> </ol>
Aortic stenosis	<ol style="list-style-type: none"> <li>1 Athletes with mild aortic stenosis (&lt;20 mm Hg) can participate in all competitive sports</li> <li>2 Athletes with mild to moderate aortic stenosis (21 to 40 mm Hg) can participate in all low intensity sports. Some, depending on exercise stress testing, can participate in low and moderate intensity sports</li> <li>3 Athletes with severe aortic stenosis (&gt;40 mm Hg) or symptoms should not engage in any competitive sports</li> <li>4 Athletes with bicuspid aortic valve, even without stenosis but with aortic dilatation, can participate in low intensity sports only. Serial 6 monthly echocardiographic monitoring of aortic root and ascending aorta is recommended</li> </ol>

ARVC, arrhythmogenic right ventricular cardiomyopathy; HCM, hypertrophic cardiomyopathy; IDCM, idiopathic dilated cardiomyopathy; WPW, Wolff-Parkinson-White syndrome.

\*Long QT and Brugada syndromes.

†Low risk defined by normal systolic function, normal exercise tolerance for age, no ischaemia on exercise stress testing, no exercise induced complex ventricular arrhythmia, and no haemodynamically significant coronary artery stenosis.

# Aritmie sopraventricolari

**TABLE 2**  
Supraventricular Arrhythmias in the Athlete\*

Arrhythmia	Baseline ECG	Symptoms	Diagnosis	Treatment Options	Guidelines for Athletic Participation <sup>17</sup>
APCs	Often WNL	Palpitations	Monitor	Reassurance	No restrictions; beta-blocker if highly symptomatic
Atrial fibrillation	Often WNL	Palpitations	Monitor	Antiarrhythmics, anticoagulation, and rate control	Bodily contact prohibited with warfarin
Atrial flutter	Often WNL	Palpitations	Monitor	RFA, antiarrhythmics, rate control, and anticoagulation	Bodily contact prohibited with warfarin
Ventricular preexcitation (WPW)	Short PR, delta waves	Asymptomatic	Monitor, ECG, EPS	No therapy, RFA if high risk	Consider EPS to risk stratify
Ventricular preexcitation (WPW)	Short PR, delta waves	Palpitations	Monitor, ECG	RFA, antiarrhythmias	No restrictions after 3–6 months without symptoms
AVNRT	Normal	Palpitations	Monitor, EPS	RFA, antiarrhythmics	No restrictions after 3–6 months without symptoms

\*Modified from reference 3.

APC = atrial premature contractions; AVNRT = AV nodal reentrant tachycardia within normal limits; EPS = electrophysiologic study; RFA = radiofrequency ablation; WNL = within normal limits; WPW = Wolff-Parkinson-White syndrome.

# Aritmie ventricolari

**TABLE 3**  
Ventricular Arrhythmias In the Athlete\*

Arrhythmia	Baseline ECG	Symptoms	Diagnosis	Treatment Options	Guidelines for Athletic Participation <sup>17</sup>
PVCs	WNL	Palpitations	Monitor	Reassurance, beta-blockers	No restrictions if no SHD
NSVT	WNL	Palpitations	Monitor	Assess for SHD; if no SHD, reassurance	No restriction if no SHD
Sustained VT/VF	WNL or SHD	Palpitations, syncope, cardiac arrest	Monitor, EPS	If SHD, further evaluation; RFA if no SHD; ICD or AAD if SHD	No restrictions if no SHD and successful RFA; low-intensity sports otherwise

\*Modified from reference 3.

EPS = electrophysiologic study; NSVT = nonsustained ventricular tachycardia; PVCs = premature ventricular contractions; RFA = radiofrequency ablation; SHD = structural heart disease; VF = ventricular fibrillation; VT = ventricular tachycardia; WNL = within normal limits.

## Some considerations . . .

Competitive sport can put athletes with mild cardiac disorders in extreme, insoluble and not predictable environmental conditions, (associated with blood volume, electrolytic and hydration modifications).

## Some considerations . . .

The use of antiarrhythmic drugs can not be considered a mean to offer safety and specific protection against life-threatening arrhythmias, or a primary tool to obtain the qualification to strenuous competitive sport practice.

## Some considerations . . .

The availability of external defibrillators in the place of competition must not be considered an absolute protection against sudden death and a warranty for participating to otherwise prohibited competitive sports.

## Some considerations . . .

The presence of an ICD can not be considered a protective therapy and therefore a justification to allow the participation to otherwise avoided competitive sports.