



PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

**ROMA**

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della Tecnica

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# Dilatazione ventricolare destra: Cuore d'atleta vs. cardiomiopatia aritmogena



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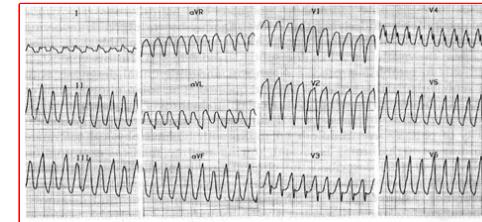
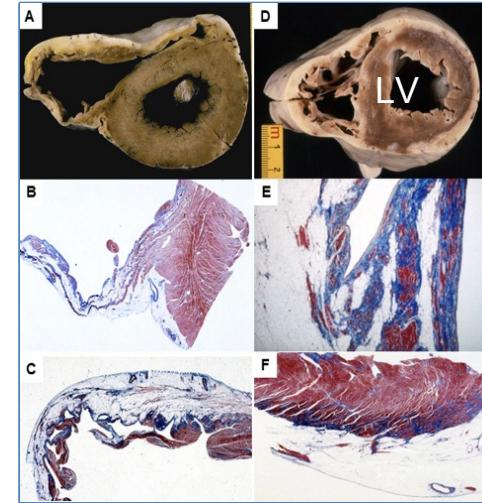


# Arrhythmogenic Cardiomyopathy:

Genetically-determined  
heart muscle disease,  
("cardiomyopathy")

Pathology: Fibrofatty replacement  
of the "right & left ventricular"  
myocardium

Clinical presentation: ventricular  
tachycardia and arrhythmic  
sudden death  
("arrhythmogenic")



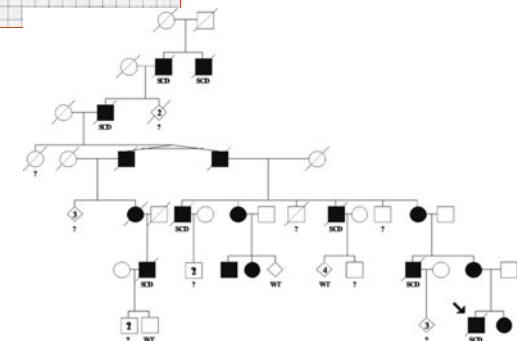
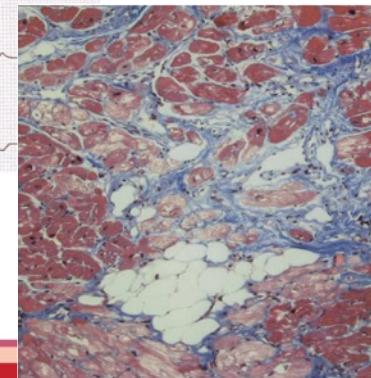
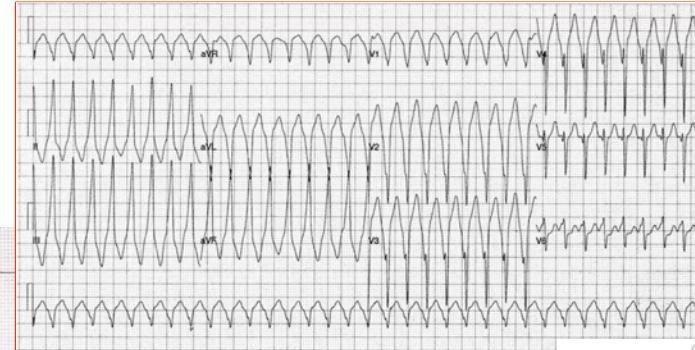
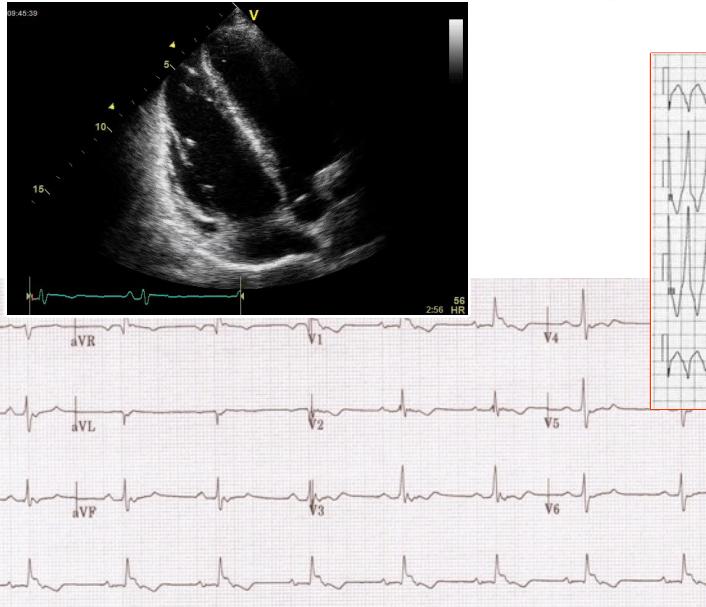
Corrado et al. Circ Res 2017; 121:784-802



# Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia

## Proposed Modification of the Task Force Criteria

Frank I. Marcus<sup>1\*</sup> Chair, William J. McKenna<sup>2</sup> Co-Chair, Duane Sherrill<sup>1</sup>,

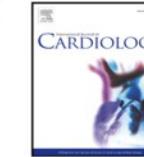




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### Diagnosis of arrhythmogenic cardiomyopathy: The Padua criteria

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**Table 1**  
 "Padua criteria" for diagnosis of Arrhythmogenic Cardiomyopathy.

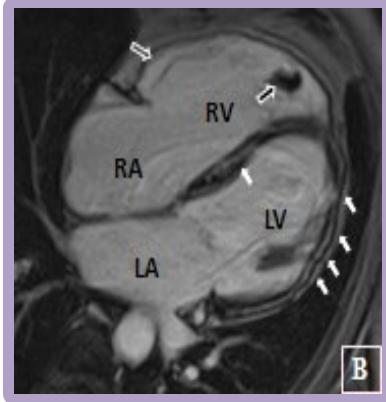
Category	Right ventricle (upgraded 2010 ITF diagnostic criteria)	Left ventricle (new diagnostic criteria)
I. Morpho-functional ventricular abnormalities	<p><i>By echocardiography, CMR or angiography:</i></p> <p><b>Major</b></p> <ul style="list-style-type: none"> <li>Regional RV akinesia, dyskinesia, or bulging plus one of the following:           <ul style="list-style-type: none"> <li>global RV dilatation (increase of RV EDV according to the imaging test specific nomograms)</li> <li>global RV systolic dysfunction (reduction of RV EF according to the imaging test specific nomograms)</li> </ul> </li> </ul> <p><b>Minor</b></p> <ul style="list-style-type: none"> <li>Regional RV hypokinesia or akinesia of LV free wall, septum, or both</li> </ul>	<p><i>By echocardiography, CMR or angiography:</i></p> <p><b>Minor</b></p> <ul style="list-style-type: none"> <li>Global LV systolic dysfunction (depression of LV EF or reduction of echocardiographic global longitudinal strain), with or without LV dilatation (increase of LV EDV according to the imaging test specific nomograms for age, sex, and BSA)</li> </ul> <p><b>Minor</b></p> <ul style="list-style-type: none"> <li>Regional LV hypokinesia or akinesia of LV free wall, septum, or both</li> </ul>
II. Structural myocardial abnormalities	<p><i>By CE-CMR:</i></p> <p><b>Major</b></p> <ul style="list-style-type: none"> <li>Transmural LGE (stria pattern) of <math>\geq 1</math> RV region(s) (inlet, outlet, and apex in 2 orthogonal views)</li> </ul> <p><i>By EMB (limited indications):</i></p> <p><b>Major</b></p> <ul style="list-style-type: none"> <li>Fibrous replacement of the myocardium in <math>\geq 1</math> sample, with or without fatty tissue</li> </ul>	<p><i>By CE-CMR:</i></p> <p><b>Major</b></p> <ul style="list-style-type: none"> <li>LV LGE (stria pattern) of <math>\geq 1</math> Bull's Eye segment(s) (in 2 orthogonal views) of the free wall (subepicardial or midmyocardial), septum, or both (excluding septal junctional LGE)</li> </ul>
III. Repolarization abnormalities	<p><i>Major</i></p> <ul style="list-style-type: none"> <li>Inverted T waves in right precordial leads (<math>V_1, V_2</math>, and <math>V_3</math>) or beyond in individuals with complete pubertal development (in the absence of complete RBBB)</li> </ul> <p><i>Minor</i></p> <ul style="list-style-type: none"> <li>Inverted T waves in leads V1 and V2 in individuals with completed pubertal development (in the absence of complete RBBB)</li> <li>Inverted T waves in <math>V_1, V_2, V_3</math> and V4 in individuals with completed pubertal development in the presence of complete RBBB.</li> </ul>	<p><i>Minor</i></p> <ul style="list-style-type: none"> <li>Inverted T waves in left precordial leads (<math>V_4-V_6</math>) (in the absence of complete LBBB)</li> </ul>
IV. Depolarization abnormalities	<p><i>Major</i></p> <ul style="list-style-type: none"> <li>Epsilon wave (reproducible low-amplitude signals between end of QRS complex to onset of the T wave) in the right precordial leads (V1 to V3)</li> <li>Terminal activation duration of QRS <math>\geq 55</math> ms measured from the nadir of the S wave to the end of the QRS, including R, in <math>V_1, V_2</math>, or V3 (in the absence of complete RBBB)</li> </ul>	<p><i>Minor</i></p> <ul style="list-style-type: none"> <li>Low QRS voltages (&lt;0.5 mV peak to peak) in limb leads (in the absence of obesity, emphysema, or pericardial effusion)</li> </ul>
V. Ventricular arrhythmias	<p><i>Major</i></p> <ul style="list-style-type: none"> <li>Frequent ventricular extrasystoles (&gt;500 per 24 h), non-sustained or sustained ventricular tachycardia of LBBB morphology</li> </ul> <p><i>Minor</i></p> <ul style="list-style-type: none"> <li>Frequent ventricular extrasystoles (&gt;500 per 24 h), non-sustained or sustained ventricular tachycardia of LBBB morphology with inferior axis ("RVOT pattern")</li> </ul>	<p><i>Minor</i></p> <ul style="list-style-type: none"> <li>Frequent ventricular extrasystoles (&gt;500 per 24 h), non-sustained or sustained ventricular tachycardia with a RBBB morphology (excluding the "fascicular pattern")</li> </ul>
VI. Family history/genetics	<p><i>Major</i></p> <ul style="list-style-type: none"> <li>ACM confirmed in a first-degree relative who meets diagnostic criteria</li> <li>ACM confirmed pathologically at autopsy or surgery in a first degree relative</li> <li>Identification of a pathogenic or likely pathogenetic ACM mutation in the patient under evaluation</li> </ul> <p><i>Minor</i></p> <ul style="list-style-type: none"> <li>History of ACM in a first-degree relative in whom it is not possible or practical to determine whether the family member meets diagnostic criteria</li> <li>Premature sudden death (&lt;35 years of age) due to suspected ACM in a first-degree relative</li> <li>ACM confirmed pathologically or by diagnostic criteria in a second-degree relative</li> </ul>	



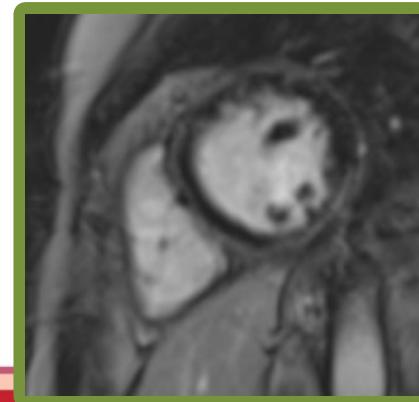
# ACM phenotypes



**RV phenotype:** either isolated or associated with some LV involvement



**Biventricular phenotype:** characterized by equal involvement of both ventricles



**Left dominant phenotype:** with early and prominent LV manifestations.



## ECHOCARDIOGRAPHIC MAJOR CRITERIA

**By 2D echo:**

- Regional RV akinesia, dyskinesia, or aneurysm
- and 1 of the following (end diastole):
  - PLAX RVOT  $\geq 32$  mm (corrected for body size [PLAX/BSA]  $\geq 19$  mm/m<sup>2</sup>)
  - PSAX RVOT  $\geq 36$  mm (corrected for body size [PSAX/BSA]  $\geq 21$  mm/m<sup>2</sup>)
  - or fractional area change  $\leq 33\%$

## ECHOCARDIOGRAPHIC MINOR CRITERIA

**By 2D echo:**

- Regional RV akinesia or dyskinesia
- and 1 of the following (end diastole):
  - PLAX RVOT  $\geq 29$  to  $<32$  mm (corrected for body size [PLAX/BSA]  $\geq 16$  to  $<19$  mm/m<sup>2</sup>)
  - PSAX RVOT  $\geq 32$  to  $<36$  mm (corrected for body size [PSAX/BSA]  $\geq 18$  to  $<21$  mm/m<sup>2</sup>)
  - or fractional area change  $>33\%$  to  $\leq 40\%$



**Table 1**  
"Padua criteria" for diagnosis of Arrhythmogenic Cardiomyopathy.

Category	Right ventricle (upgraded 2010 ITF diagnostic criteria)	Left ventricle (new diagnostic criteria)
I. Morpho-functional ventricular abnormalities	<p>By echocardiography, CMR or angiography:</p> <p><i>Major</i></p> <ul style="list-style-type: none"> <li>• Regional RV akinesia, dyskinesia, or bulging plus one of the following:           <ul style="list-style-type: none"> <li>- global RV dilatation (increase of RV EDV according to the imaging test specific nomograms)</li> <li>- global RV systolic dysfunction (reduction of RV EF according to the imaging test specific nomograms)</li> </ul> </li> </ul> <p><i>Minor</i></p>	<p>By echocardiography, CMR or angiography: <i>Minor</i></p> <ul style="list-style-type: none"> <li>• Global LV systolic dysfunction (depression of LV EF or reduction of echocardiographic global longitudinal strain), with or without LV dilatation (increase of LV EDV according to the imaging test specific nomograms for age, sex, and BSA)</li> <li>• Regional LV hypokinesia or akinesia of LV free wall, septum, or both</li> </ul>

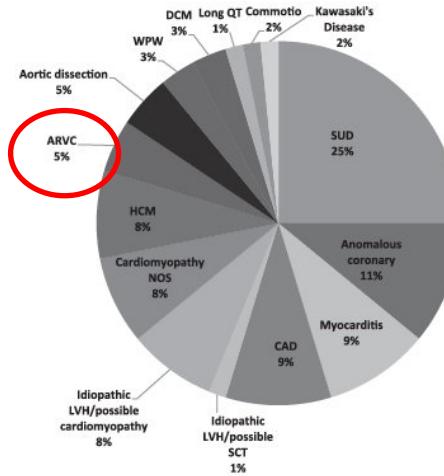




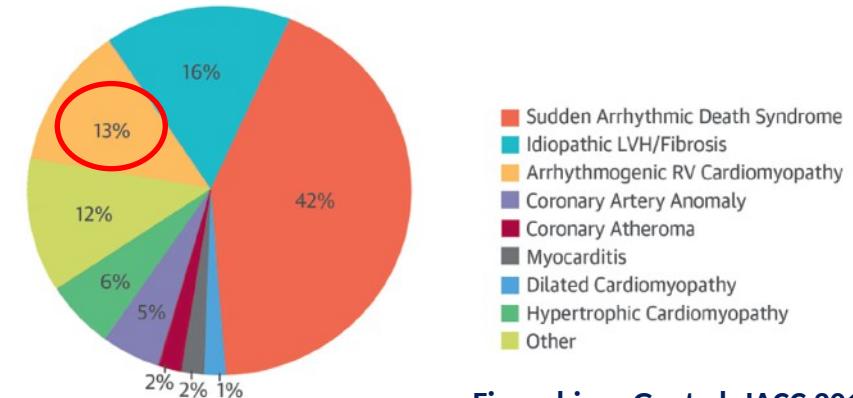
**TABLE 2.** CAUSES OF SUDDEN DEATH IN ATHLETES AND NONATHLETES 35 YEARS OF AGE OR LESS IN THE VENETO REGION OF ITALY, 1979 TO 1996.

CAUSE	ATHLETES (N=49)	NONATHLETES (N=220)	TOTAL (N=269)
	number (percent)		
Arrhythmogenic right ventricular cardiomyopathy	11 (22.4)	18 (8.2)*	29 (10.8)
Atherosclerotic coronary artery disease	9 (18.4)	36 (16.4)	45 (16.7)
Anomalous origin of coronary artery	6 (12.2)	1 (0.5)†	7 (2.6)
Disease of conduction system	4 (8.2)	20 (9.1)	24 (8.9)
Mitral-valve prolapse	5 (10.2)	21 (9.5)	26 (9.7)
Hypertrophic cardiomyopathy	1 (2.0)	16 (7.3)	17 (6.3)
Myocarditis	3 (6.1)	19 (8.6)	22 (8.2)
Myocardial bridge	2 (4.1)	5 (2.3)	7 (2.6)
Pulmonary thromboembolism	1 (2.0)	3 (1.4)	4 (1.5)
Dissecting aortic aneurysm	1 (2.0)	11 (5.0)	12 (4.5)
Dilated cardiomyopathy	1 (2.0)	9 (4.1)	10 (3.7)
Other	5 (10.2)	61 (27.7)	66 (24.5)

Corrado D, et al. NEJM 1998



Harmon KJ, et al. Circulation 2015



Finocchiaro G, et al. JACC 2016



## Diagnosis vs. Overdiagnosis



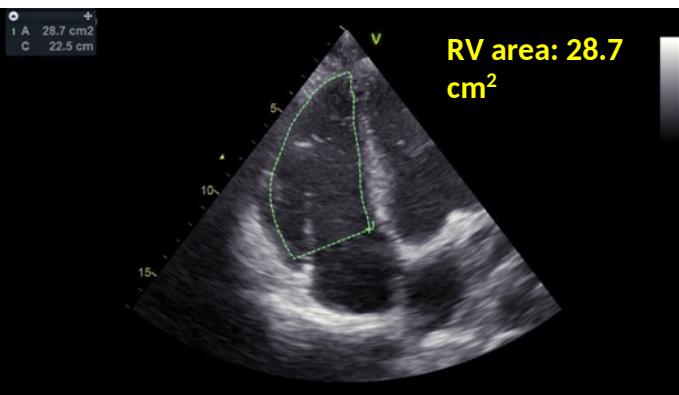
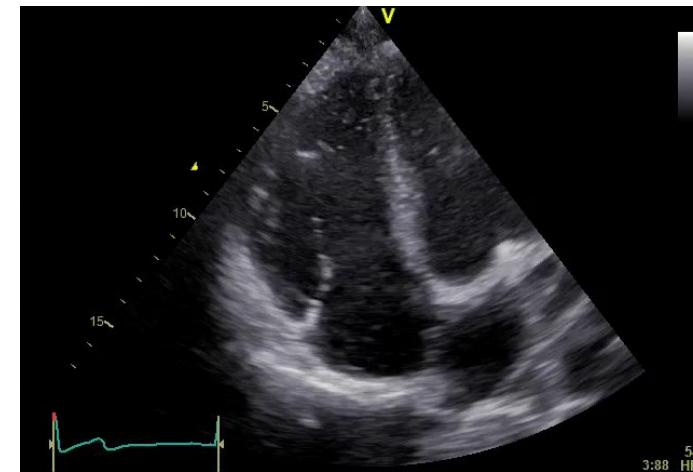
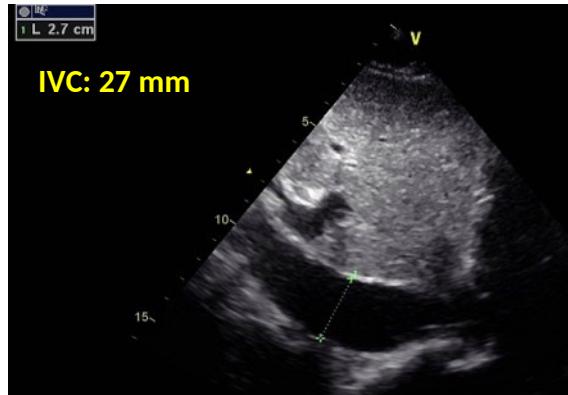
# RV physiological remodelling



PRELOAD

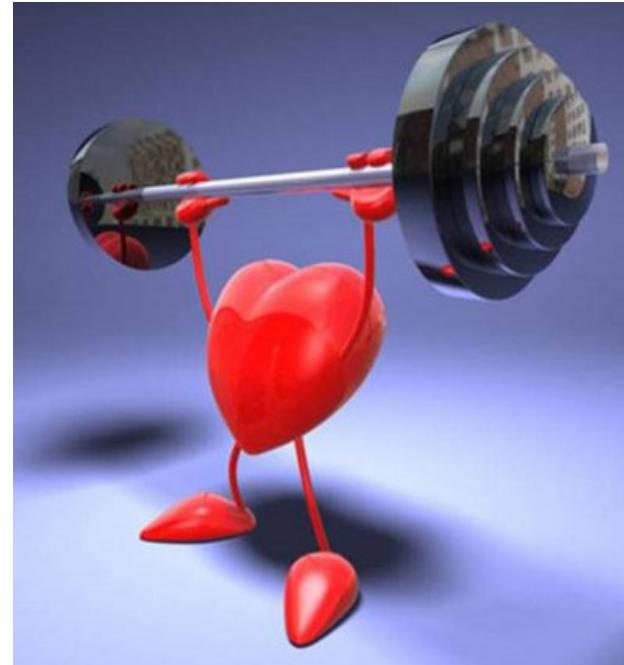


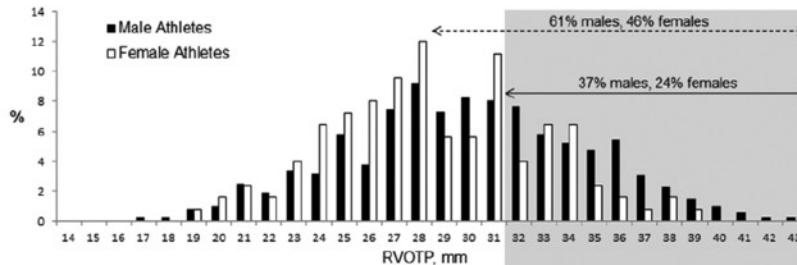
STROKE VOLUME



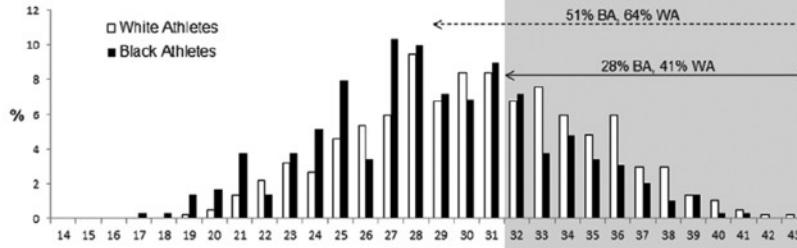
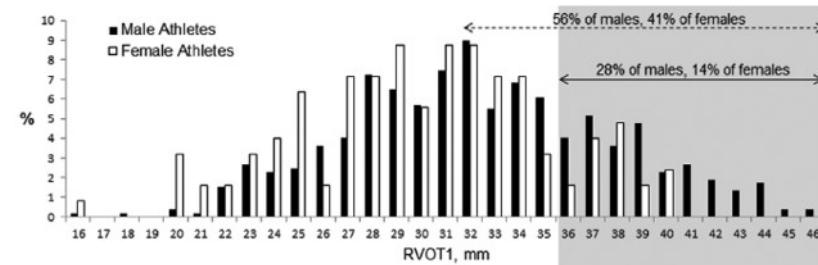


# Are the current diagnostic criteria for ARVC applicable to athlete's heart?

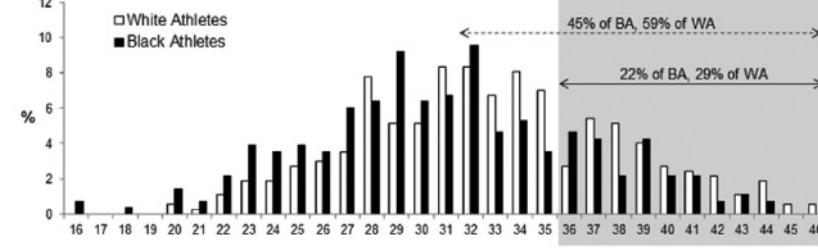




Normal	Minor	Major ARVC Criterion
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**RVOT PSAX**


Normal	Minor	Major ARVC Criterion
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Zaidi A, et al. Circulation 2013

**Authors' conclusions:** Physiological RV enlargement is commonly observed in both black and white athletes. The impact of ethnicity is minimal, which obviates the need for race-specific RV reference values. However, in the context of frequent ECG repolarization anomalies in BAs, the potential for erroneous diagnosis of arrhythmogenic RV cardiomyopathy is considerably greater in this ethnic group.



# RV Remodeling in Olympic Athletes

D'Ascenzi F, et al. JACC imaging 2016



**1009** highly-trained athletes screened for participation to 2012 London Summer or the 2014 Sochi Winter Olympic Games.



**SKILL**  
(n = 277)



**POWER**  
(n = 216)



**MIXED**  
(n = 254)



**ENDURANCE**  
(n = 262)

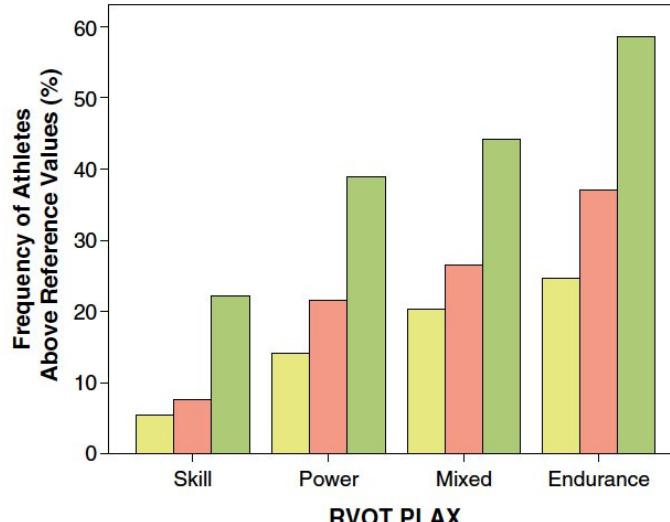
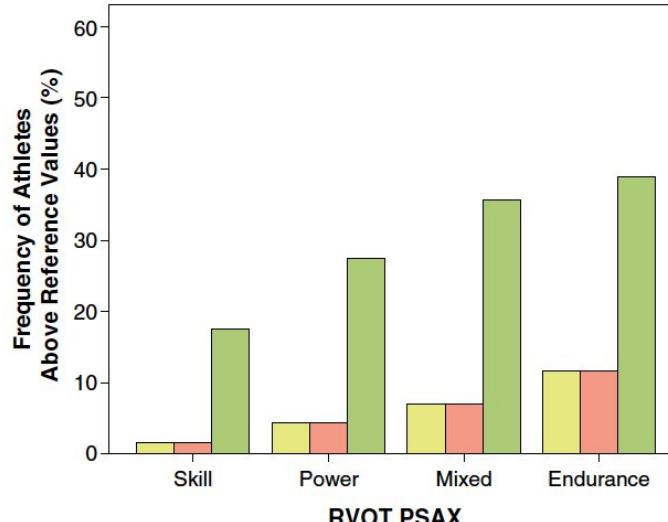




	Skill (n = 277)	Power (n = 216)	Mixed (n = 254)	Endurance (n = 262)	p Value
BSA, m <sup>2</sup>	1.8 ± 0.2	1.9 ± 0.2	2.0 ± 0.2	1.9 ± 0.2	<0.001
RVOT PLAX, mm	25.8 ± 3.5	27.4 ± 3.8	28.1 ± 3.6	29.2 ± 3.8	<0.001
RVOT PSAX, mm	27.4 ± 3.9	28.6 ± 4.2	29.5 ± 4.4	30.1 ± 4.2	<0.001
RVOT PLAX index, mm/m <sup>2</sup>	14.3 ± 2.1	14.7 ± 1.9	14.0 ± 1.8	15.7 ± 2.2	<0.001
RVOT PSAX index, mm/m <sup>2</sup>	15.2 ± 2.7	15.3 ± 2.4	14.8 ± 2.1	16.2 ± 2.5	<0.001
RV diastolic area, cm <sup>2</sup>	21.2 ± 5.3	22.6 ± 5.2	23.7 ± 4.6	24.7 ± 5.2	<0.001
RV diastolic area index, cm <sup>2</sup> /m <sup>2</sup>	11.6 ± 2.3	12.1 ± 2.2	11.8 ± 1.9	13.1 ± 2.3	<0.001
RV systolic area, cm <sup>2</sup>	10.1 ± 3.2	10.8 ± 3.4	11.4 ± 3.0	11.7 ± 3.3	<0.001
RV systolic area index, cm <sup>2</sup> /m <sup>2</sup>	5.5 ± 1.5	5.8 ± 1.5	5.7 ± 1.3	6.3 ± 1.6	<0.001
Fractional area change, %	53.1 ± 8.2	52.4 ± 8.9	51.9 ± 7.5	52.5 ± 7.6	0.34
TAPSE, mm	23.7 ± 3.3	24.1 ± 3.7	24.1 ± 3.5	25.1 ± 3.9	<0.001
s', cm/s	14.5 ± 2.1	14.4 ± 2.0	14.7 ± 2.3	14.7 ± 2.1	0.21



# Athlete's heart and diagnosis of ARVC

**A****B**

Frequency of Athletes Above Reference Values (%)

RVOT PLAX

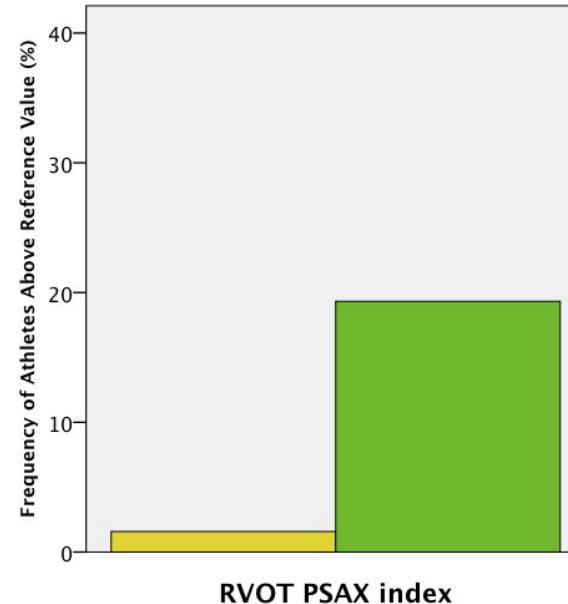
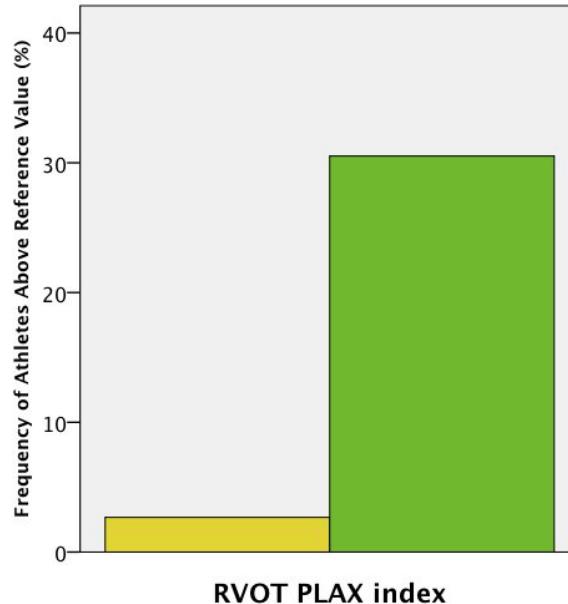
- TF major criterion
- ASE/EACVI reference
- TF minor criterion





# Athlete's heart and diagnosis of ARVC

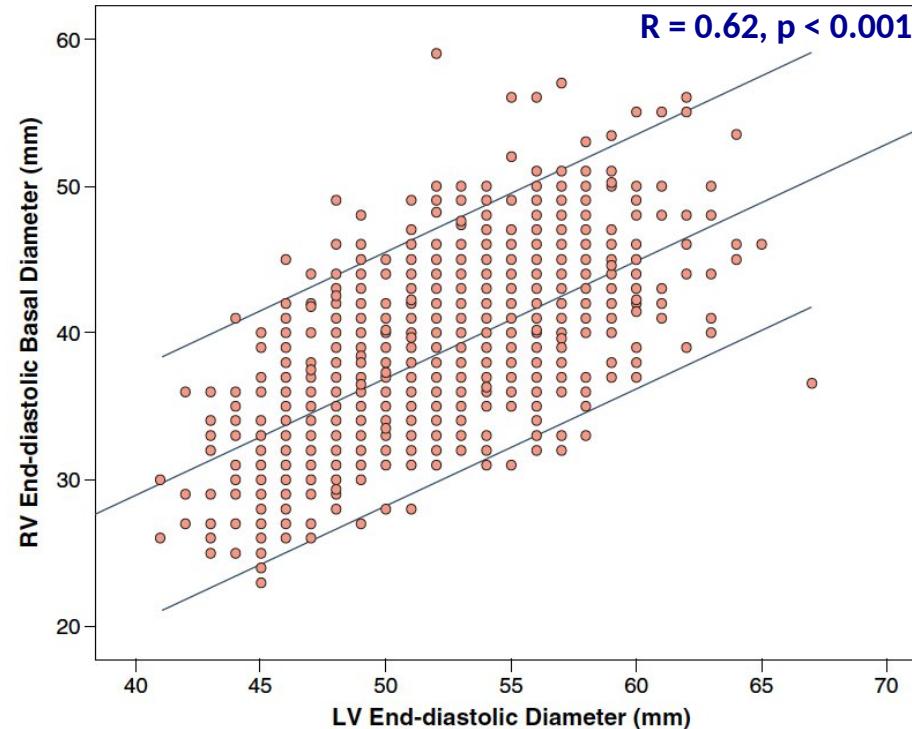
## Indexed RVOT, mm/m<sup>2</sup>



[Yellow square] TF major criterion [Red square] ASE/EACVI reference [Green square] TF minor criterion

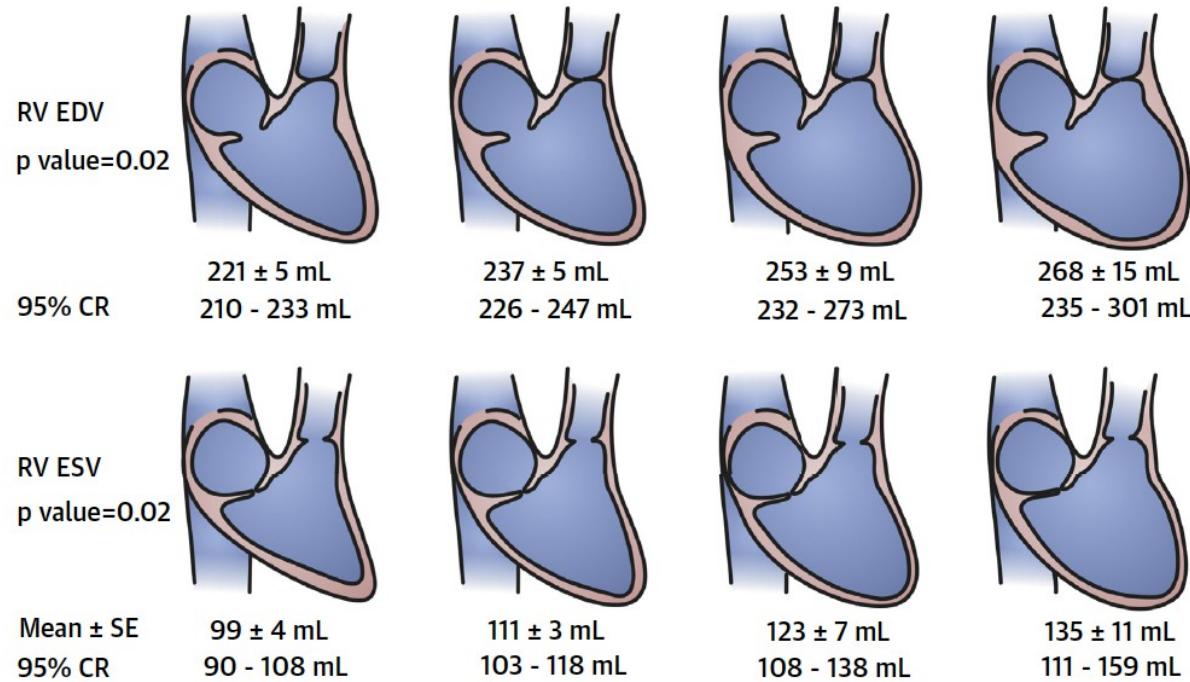


# Balanced remodelling in athletes





# Training volume and RV dimensions



D'Ascenzi F, et al. JACC IMG 2018



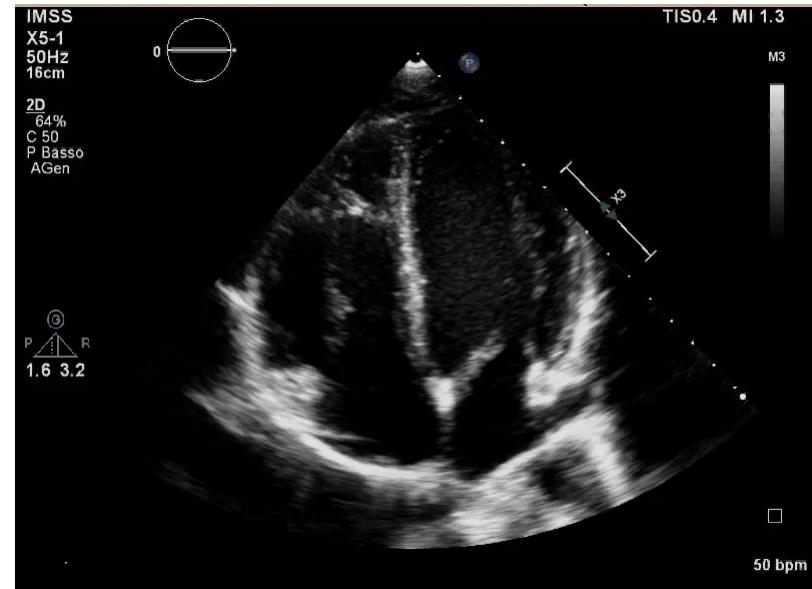
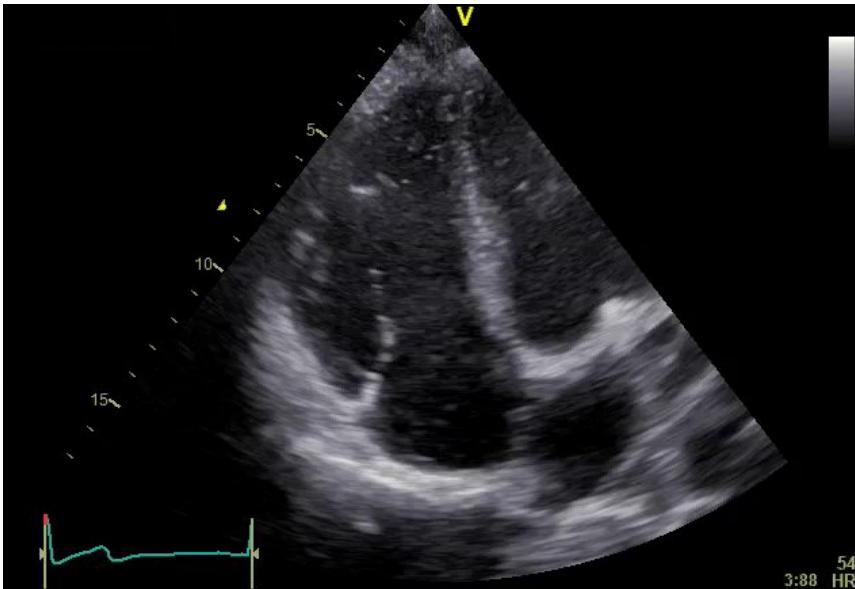
# Morphological peculiarities of the RV in highly-trained athletes

Qualitative parameters	N (%)
Rounded Apex of the RV	823 (81%)
Prominent RV Trabeculation	378 (37%)
Prominent/Hyper-reflective moderator band	5 (0.5%)
Akinesia, dyskinesia, aneurysm	0 (0%)



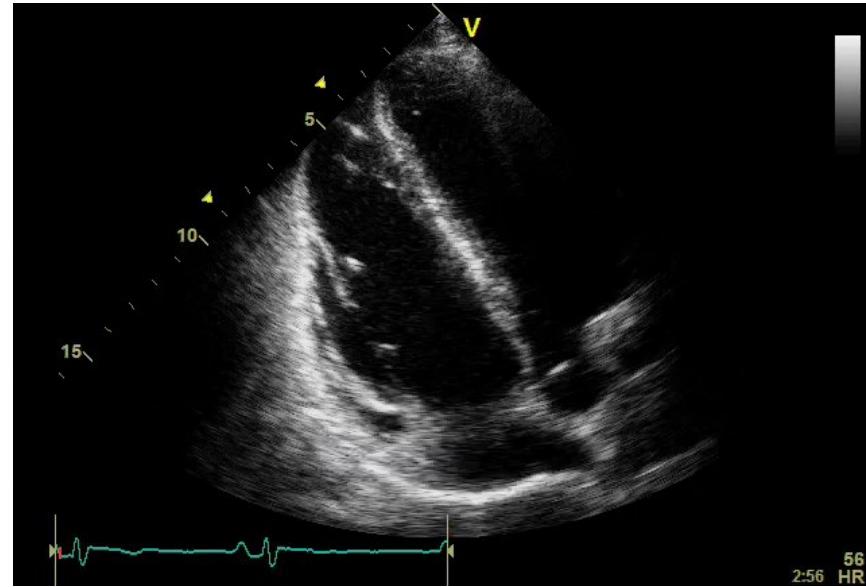
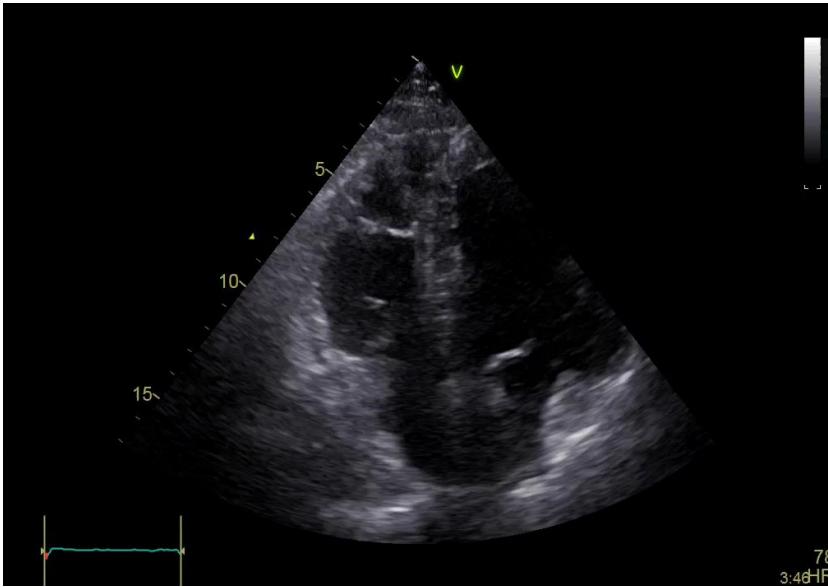


# The RV of a competitive athlete by echocardiography

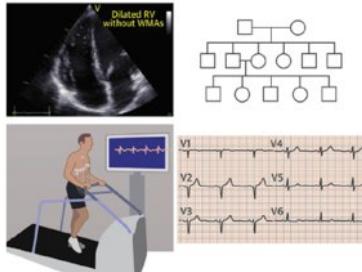




# The RV of a patient with ARVC by echocardiography



## ATHLETE'S HEART

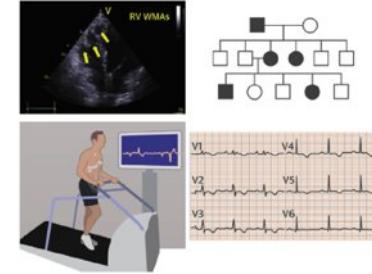


## Diagnostic Differentiation Between Arrhythmogenic Cardiomyopathy and Athlete's Heart by Using Imaging

Flavio D'Ascenzi, MD, PhD,<sup>a</sup> Marco Solari, MD,<sup>a</sup> Domenico Corrado, MD, PhD,<sup>b</sup> Alessandro Zorzi, MD, PhD,<sup>b</sup> Sergio Mondillo, MD,<sup>a</sup>



## ARRHYTHMOGENIC CARDIOMYOPATHY



Negative	Family history	Sudden death or ARVC
Absent	ECG abnormalities	QRS abnormalities, T-wave Inversion
Mainly main body <1	RV dilation	Mainly RVOT >1
Absent (or mild)	RV/LV Ratio	Present
Absent	RV dysfunction	Present
Absent (or only junctional)	RV regional WMA	Present
Absent	Late enhancement at CMR	RV and/or LV LGE with nonischemic distribution
Absent	Ventricular arrhythmias	Present



**TABLE 3 Dimensional and Functional Parameters Obtained by Echocardiography in Arrhythmogenic Right Ventricular Cardiomyopathy Versus Athlete's Heart**

**Echocardiography and Differential Diagnosis Between ARVC and Athlete's Heart**

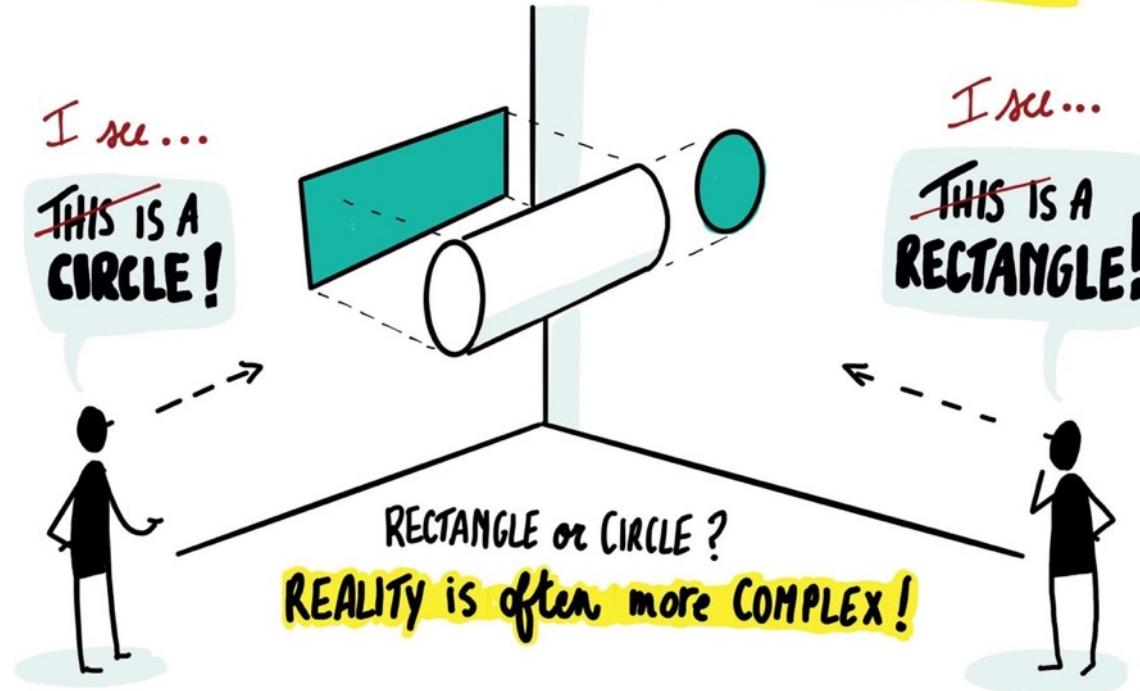
ARVC	Findings	Athlete's Heart
<b>Size</b>		
+	Marked dilation of RVOT	-
-	Moderate increase in RV main body with mild increase in RVOT	+
+	Disproportionate RV/LV ratio (<0.90)	-
-	Regression of RV dilation after detraining	+
<b>Function</b>		
+	RV wall motion abnormalities akinesia, dyskinesia, aneurysms, bulging	-
+	Reduced RV function (FAC <32%)	-
+	Reduced RV longitudinal strain (<20%)	-
+	Reduced RV s' velocity <0.10 m/s	-
+	Reduced RV function by CMR	-
+	RV and/or LV tissue abnormalities (fat infiltration and LGE) at CMR	-

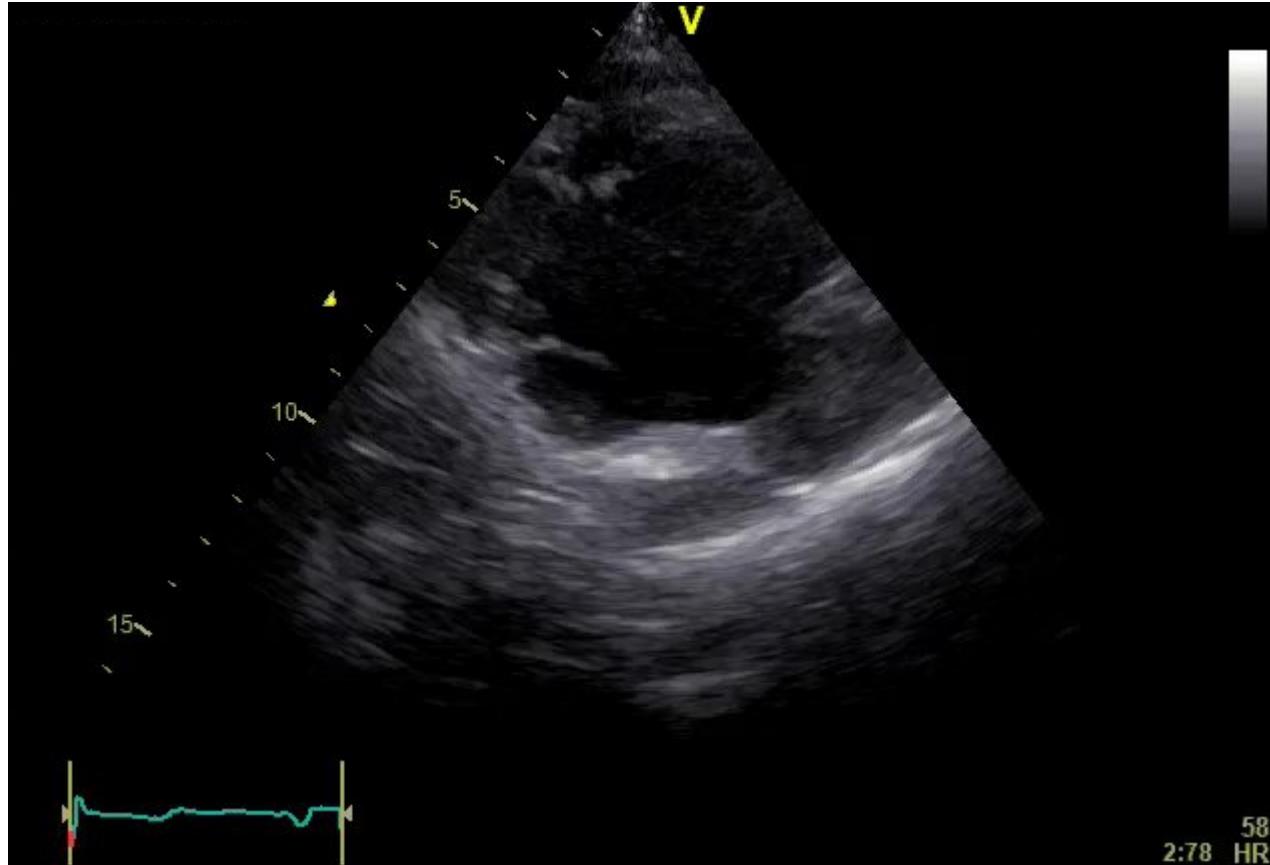
D'Ascenzi F, et al. JACC IMG 2018



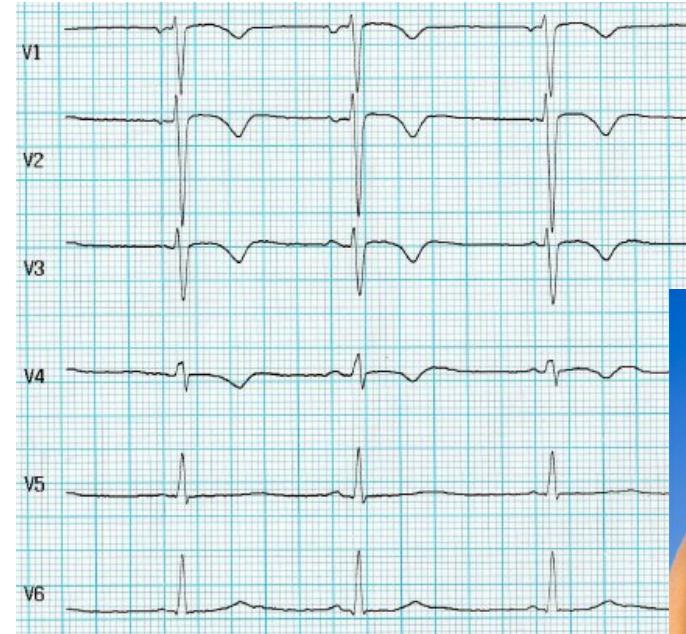
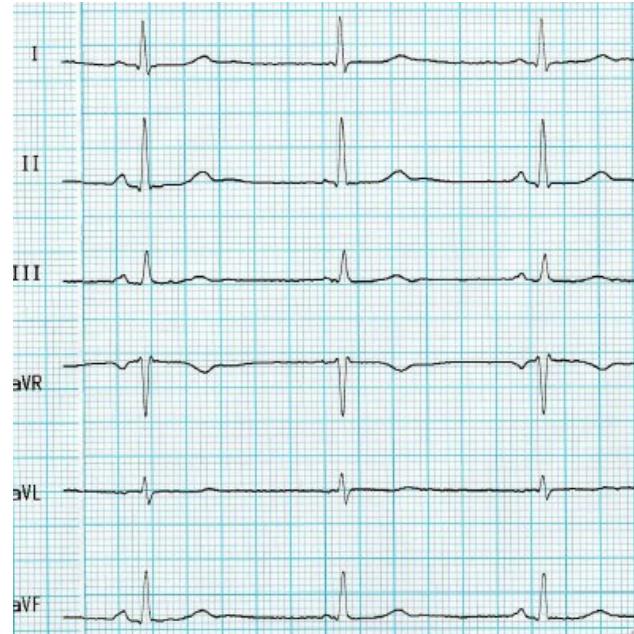


# DIFFERENT PERSPECTIVES ?





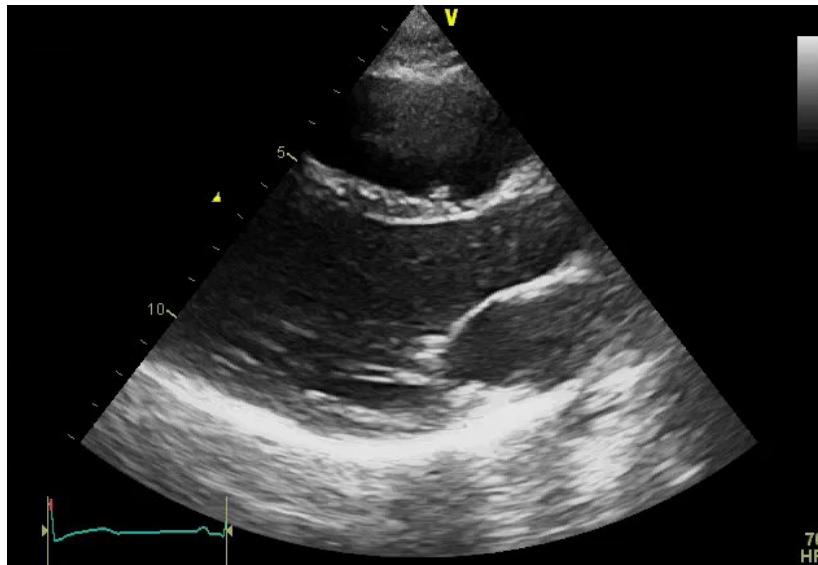
# Clinical case #1



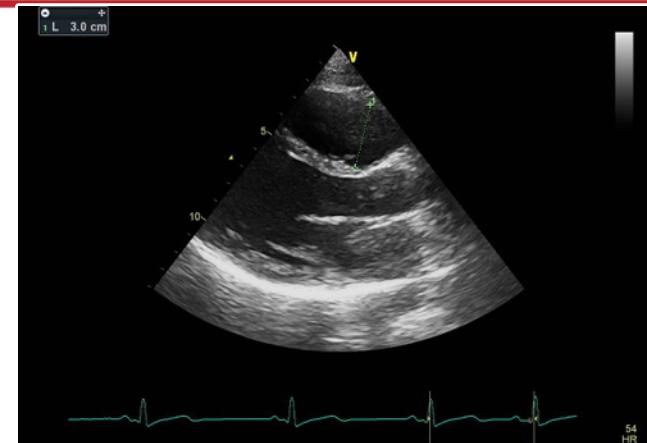
Siena Center for  
Sports Cardiology

## Echocardiographic examination

### Parasternal views

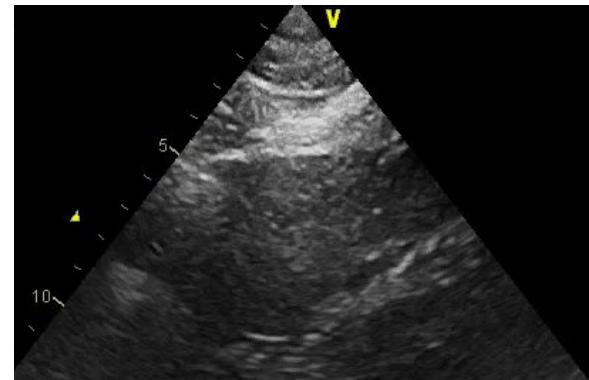
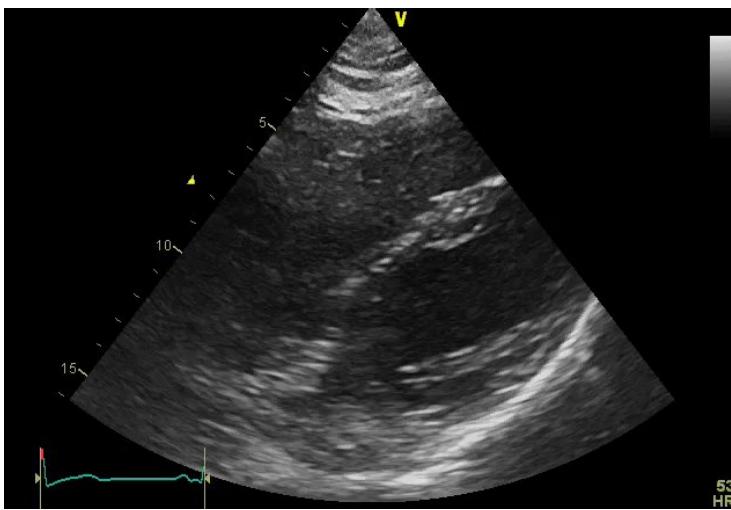
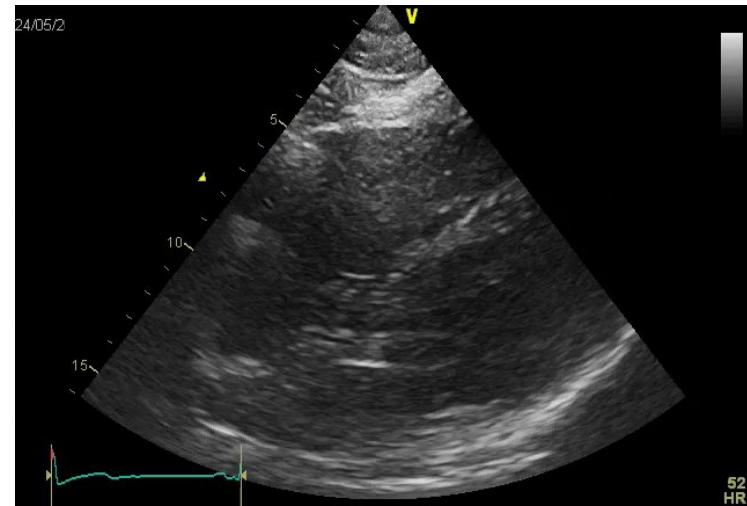
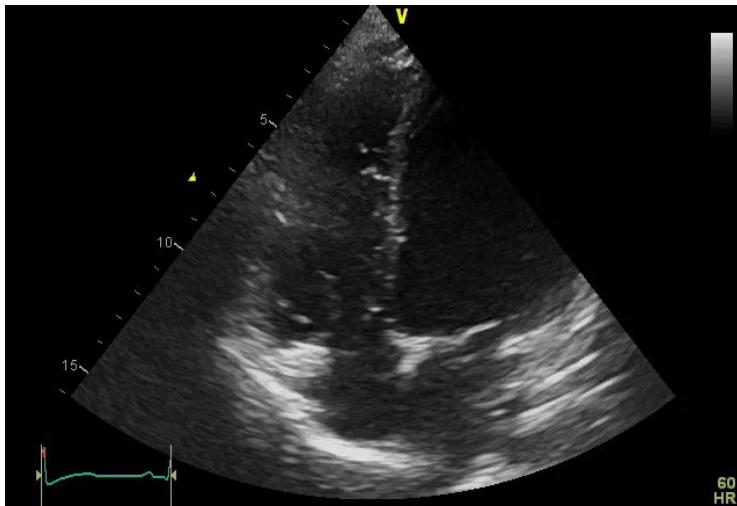


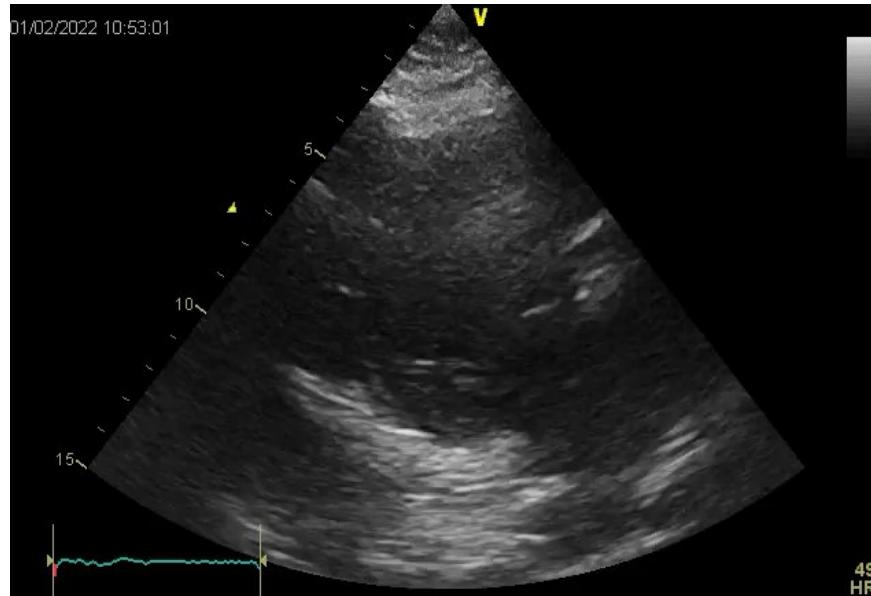
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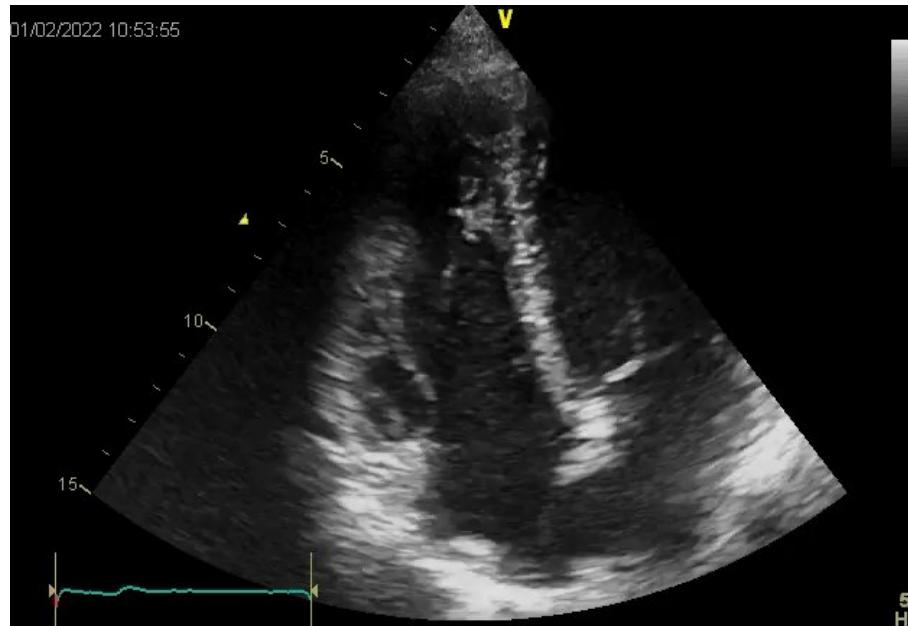




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Id:

Sconosciuto --- (--) Sconosciuto  
Altezza:-- cm Peso:-- kg PNI:0/0 mmHg

Med:

Tecnico:

Note:

01/02/2022 10:31:50

HR: 53 bpm  
PR: 124 ms  
QRS: 86 ms  
QT/QTC: 442/425 ms  
QTcB: 403 ms  
QTcF: 416 ms

Rv-wSv: 0.82/0.51 mV  
Sok-Lyon: 1.33 mV  
Asst: 9/72/41 °

REPORT NON CONFERMATO



Rep.Cardiologia Universitaria 25mm/s 10mm/mV LP:300Hz AC:50Hz Cardioline ECG200+ v. 2.15.11061



## Review

# The prevalence and clinical significance of premature ventricular beats in the athlete

F. D'Ascenzi<sup>1</sup>, A. Zorzi<sup>2</sup>, F. Alvino<sup>1</sup>, M. Bonifazi<sup>3</sup>, D. Corrado<sup>2</sup>, S. Mondillo<sup>1</sup>

<sup>1</sup>Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Siena, Italy, <sup>2</sup>Department of Cardiac, Thoracic, and Vascular Sciences, University of Padova, Padova, Italy, <sup>3</sup>Department of Medicine, Surgery, and NeuroScience, University of Siena, Siena, Italy

Corresponding author: Flavio D'Ascenzi, MD, Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Viale M. Bracci, 16 53100 Siena, Italy, Tel./Fax: 00390577585377, E-mail: flavio.dascenzi@unisi.it

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## Review



OPEN ACCESS

## How to evaluate premature ventricular beats in the athlete: critical review and proposal of a diagnostic algorithm

Domenico Corrado,<sup>1</sup> Jonathan A Drezner,<sup>2</sup> Flavio D'Ascenzi,<sup>1</sup> Alessandro Zorzi<sup>1</sup>

<sup>1</sup>Department of Cardiac, Thoracic, Vascular Sciences and Public Health, University of Padova, Padova, Italy

<sup>2</sup>Stadium Sports Medicine Center, University of Washington, Seattle, Washington, USA

<sup>3</sup>Department of Medical Biotechnologies, Division of Cardiology, University of Siena, Siena, Italy

### ABSTRACT

Although premature ventricular beats (PVBs) in young people and athletes are usually benign, they may rarely mark underlying heart disease and risk of sudden cardiac death during sport. This review addresses the prevalence, clinical meaning and diagnostic/prognostic assessment of PVBs in the athlete. The article focuses on the characteristics of PVBs, such as the morphological pattern of the ectopic QRS and the response to exercise, which accurately stratify risk. We propose an algorithm to

to competitive sports activity according to current guidelines are also addressed.

### IS THERE A GREATER PREVALENCE OF PVBs IN ATHLETES?

PVBs are a common ECG finding in the general population and are recorded in up to 75% of healthy individuals undergoing 24-hour ambulatory ECG monitoring, with a prevalence that increases with age.<sup>9–13</sup> While the PVBs of normal





# Characterisation of ventricular arrhythmias

	COMMON	UNCOMMON
Morphology	LBBB/inferior axis RBBB and narrow QRS (130 ms)	LBBB/int. or superior axis RBBB and wide QRS ( $\geq 130$ ms)
Response to exercise	Decrease/suppression	Persistence/increase
Complexity	Isolated, monomorphic	Repetitive, polymorphic
Short coupling interval (i.e. R on T)	No	Yes
<b>Clinical findings</b>		
Symptoms	No	Yes
Family history of premature SCD* or cardiomyopathy	No	Yes
ECG abnormalities	No	Yes
Imaging abnormalities	No	Yes

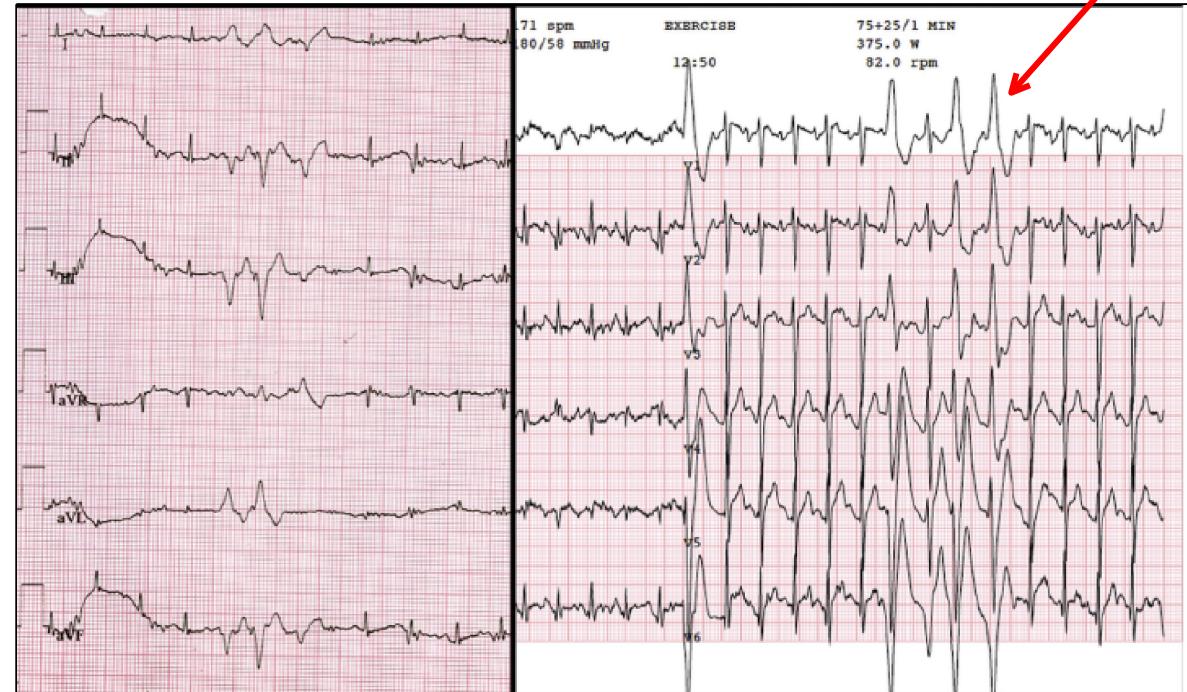
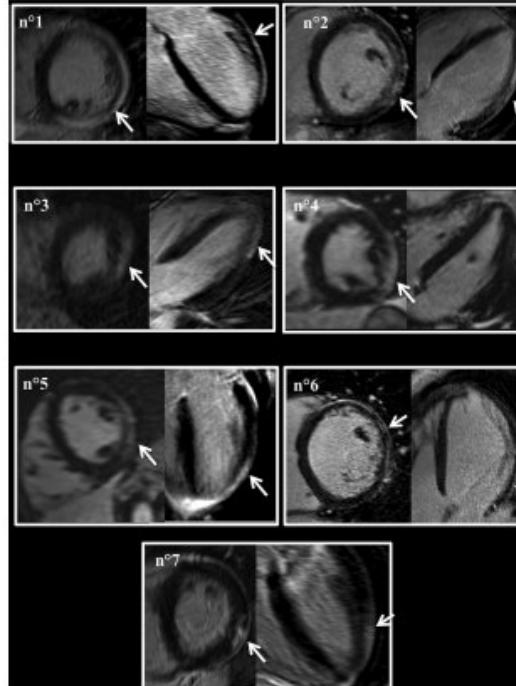


# RBBB & superior axis (wide QRS)

Subepicardial delayed gadolinium enhancement  
in asymptomatic athletes: let sleeping dogs lie?

Frédéric Schnell,<sup>1,2</sup> Guido Claessen,<sup>2</sup> André La Gerche,<sup>2,3</sup> Jan Bogaert,<sup>4</sup>  
Pierre-Axel Lentz,<sup>5</sup> Piet Claus,<sup>6</sup> Philippe Mabo,<sup>7</sup> François Carré,<sup>1</sup> Hein Heidbuchel<sup>8</sup>

Br J Sports Med 2016

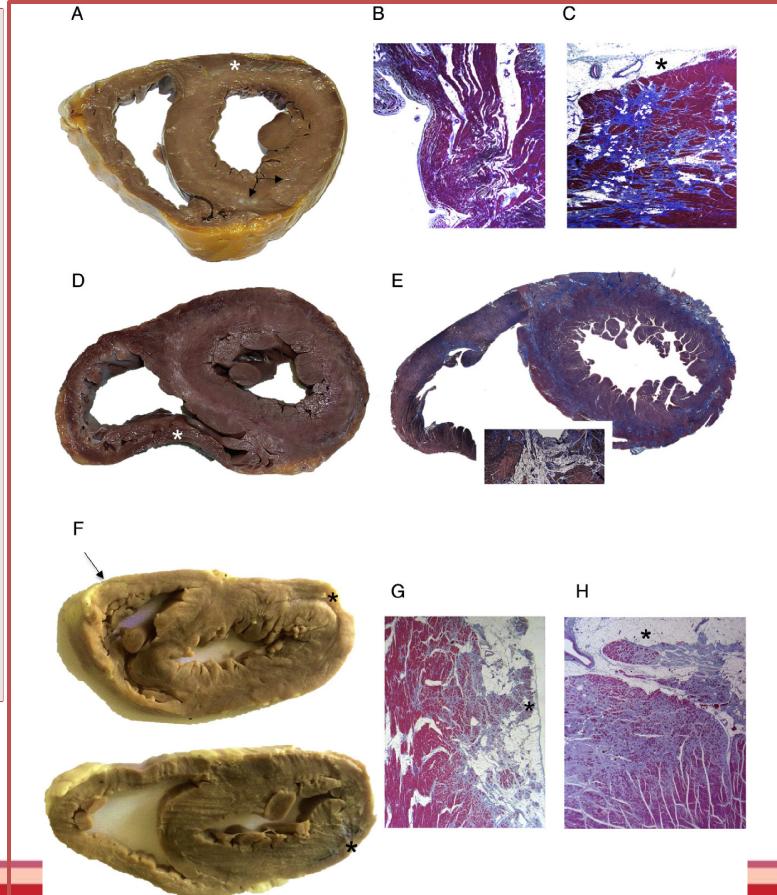




# Isolated non-ischemic LV scar and SCD

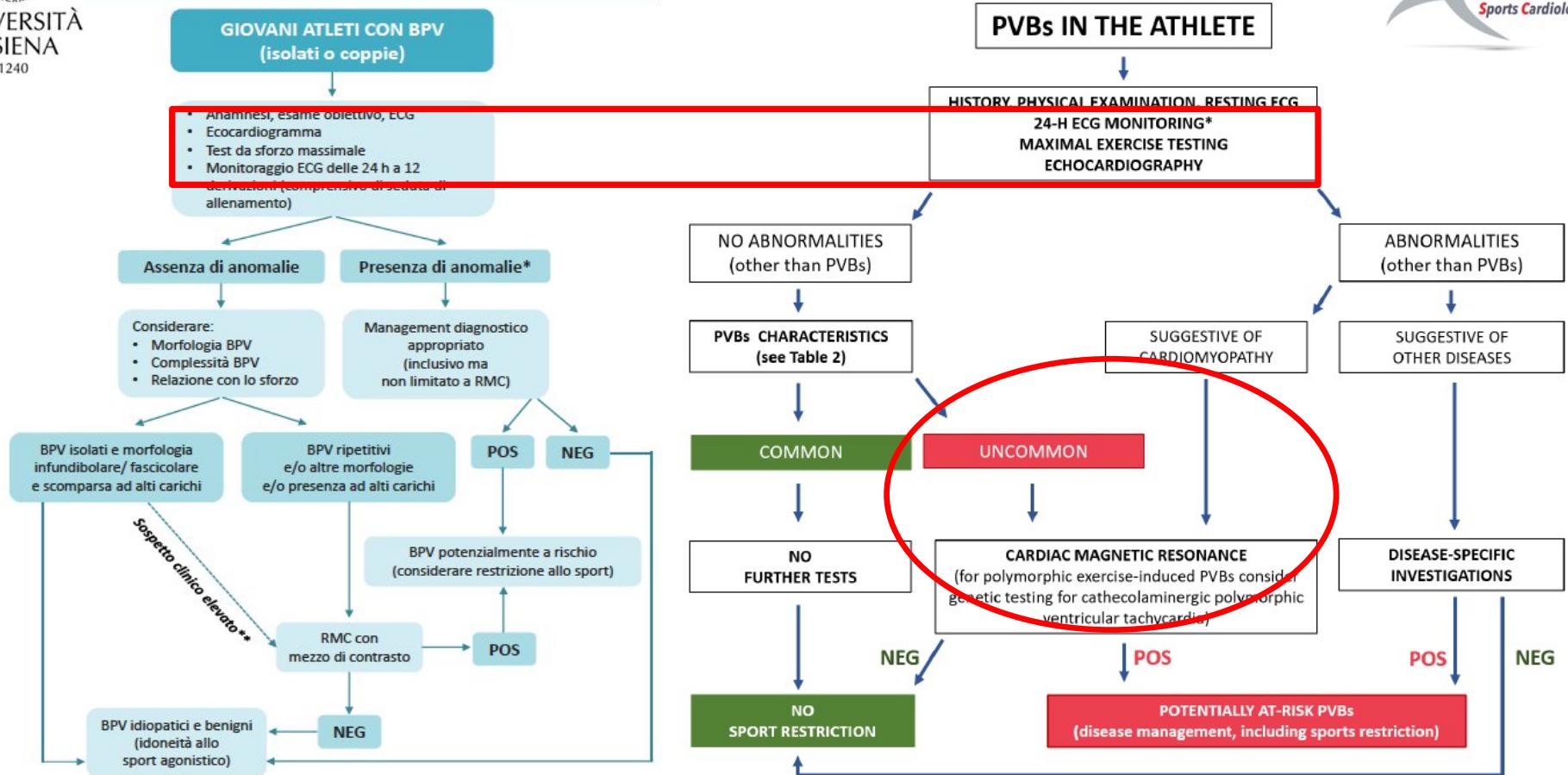
**Table 1** Anatomical substrates of juvenile SCD during sport and at rest

Morphologic findings	SCD during sports	SCD at rest
NLVS (a differential diagnosis between genetic ARVC/D and chronic acquired myocarditis is required)	25.0% (11/44)	1.3% (2/156)
HCM	16.0% (7/44)	3.8% (6/156)
Structurally normal heart	16.0% (7/44)	37.0% (58/156)
CHD	11.4% (5/44)	3.2% (5/156)
CCAA	6.8% (3/44)	1.3% (1/156)
Lymphocytic myocarditis	6.8% (3/44)	9.0% (14/156)
ATH CAD	4.5% (2/44)	18.0% (28/156)
Nonspecific LVH	2.3% (1/44)	16.0% (25/156)
Others (MVP, CSD, CV, DCM, etc)	11.4% (5/44)	11% (17/156)



Di Gioia et al. Hum Pathol 2016

# Proposed algorithm for evaluation of athletes with PVBs





G. M.

22-y old, female

Tennis player

Asymptomatic

Family history for SCD (father died suddenly at age of 35 y)



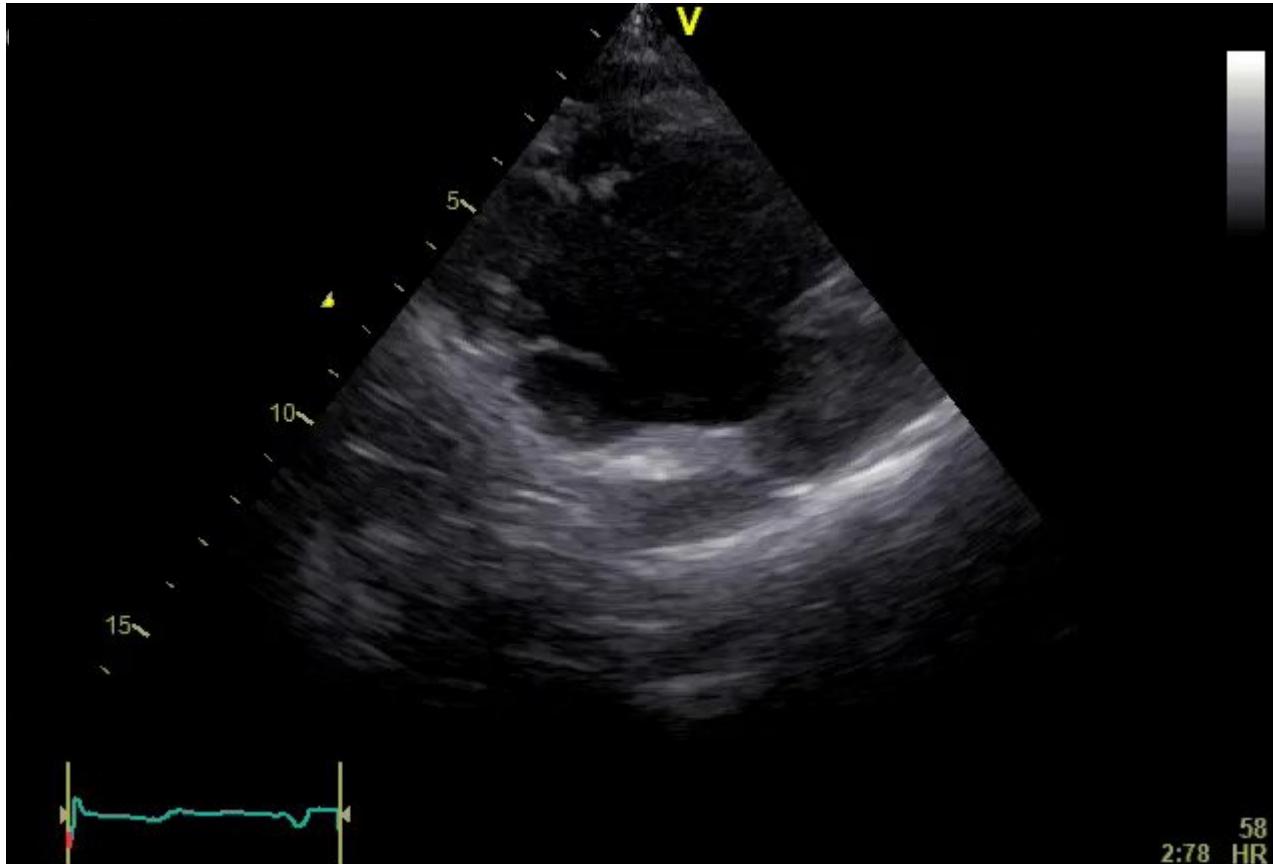
Resting ECG:  
T-wave inversion V1-V2



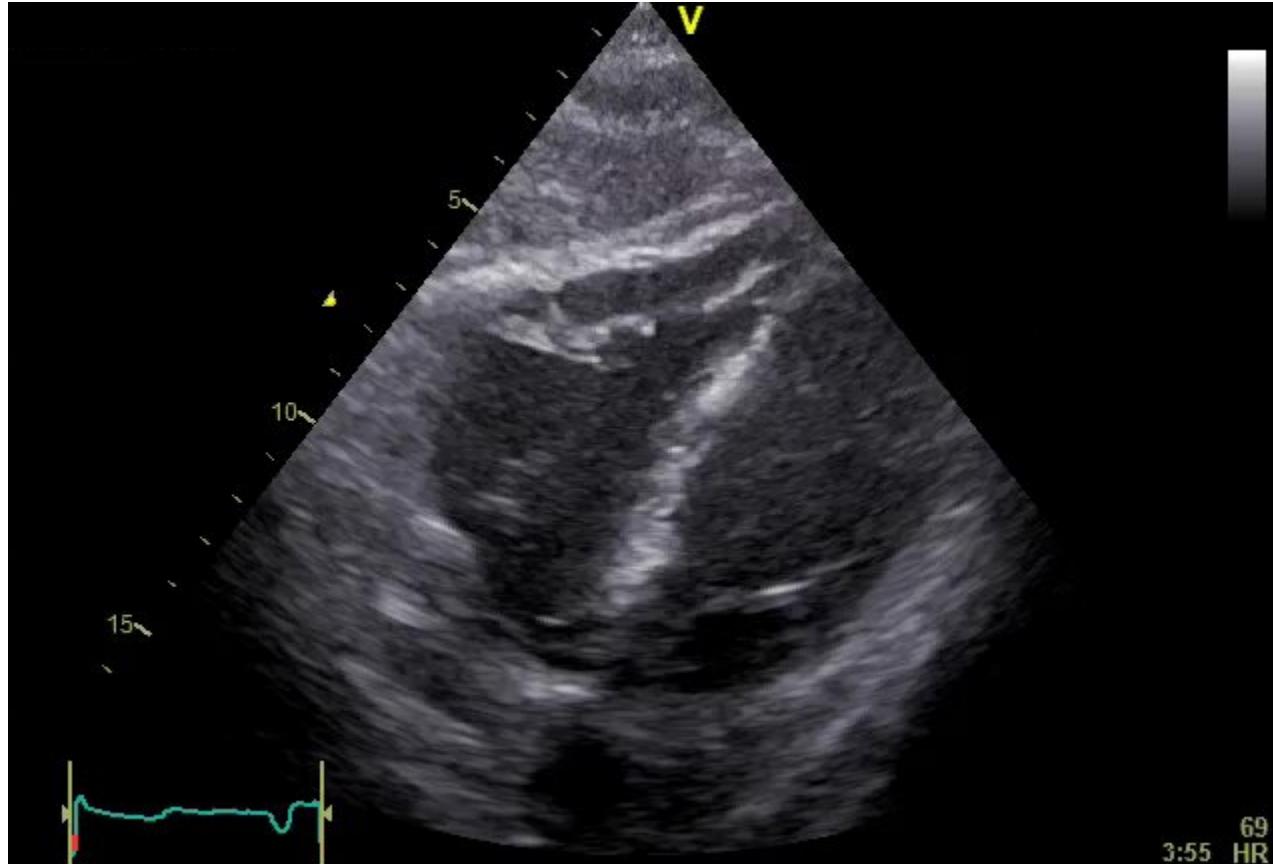
## ECHO: PARASTERNAL LONG-AXIS VIEW

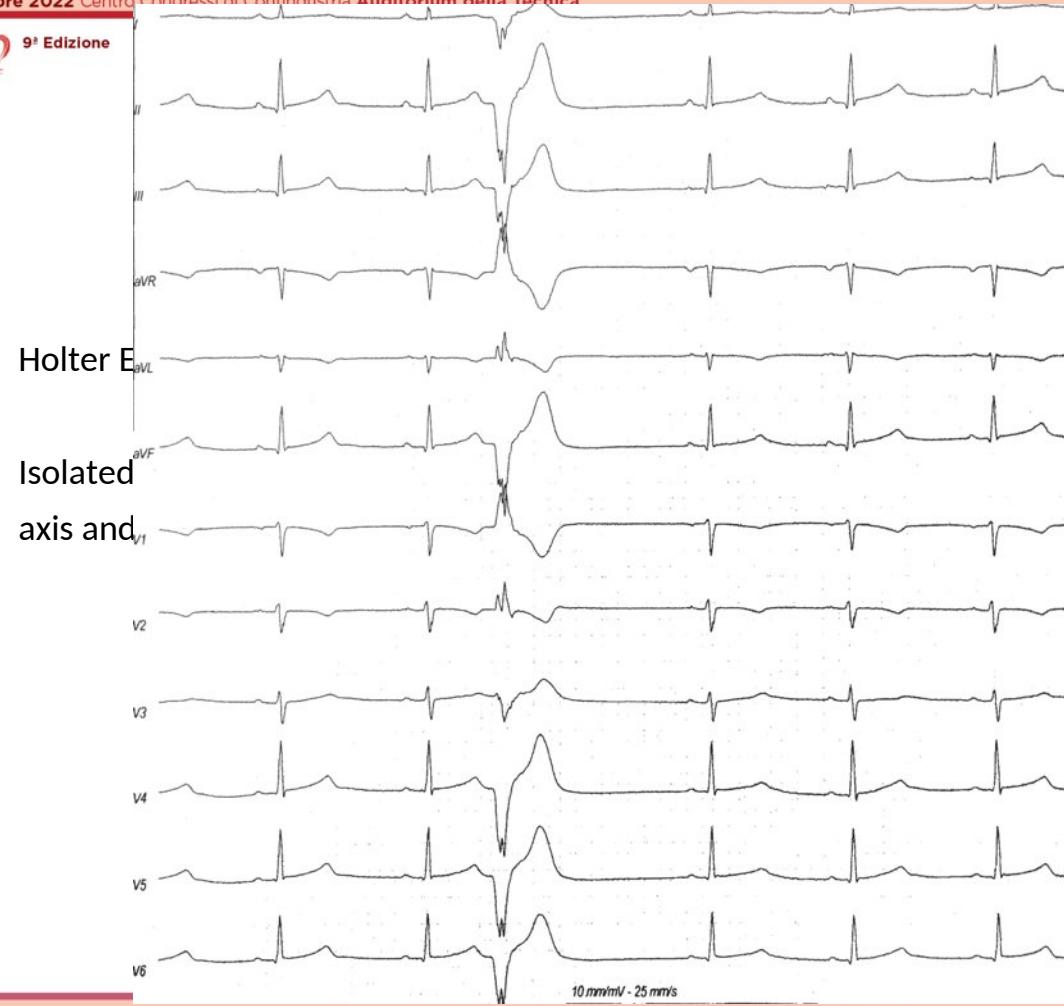


## ECHO: MODIFIED PARASTERNAL LONG-AXIS VIEW



## ECHO: SUBCOSTAL VIEW



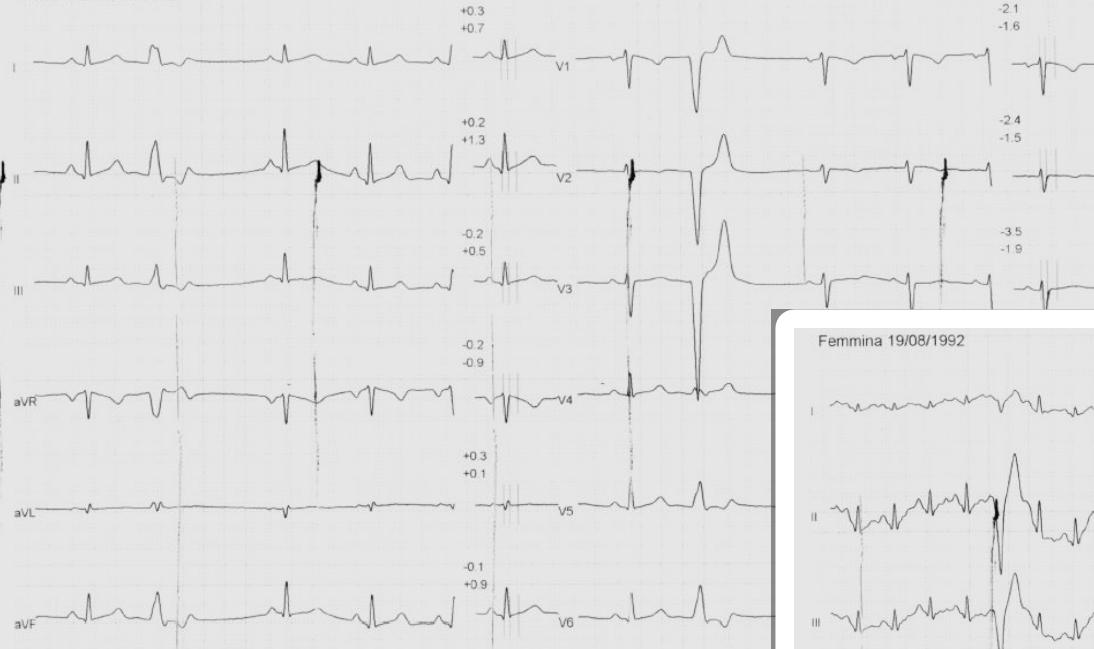


Femmina 19/08/1992

3 (01:36)

(33%)

mmHg



## Exercise testing



Femmina 19/08/1992

Picco (00:16)

(82%)

mmHg



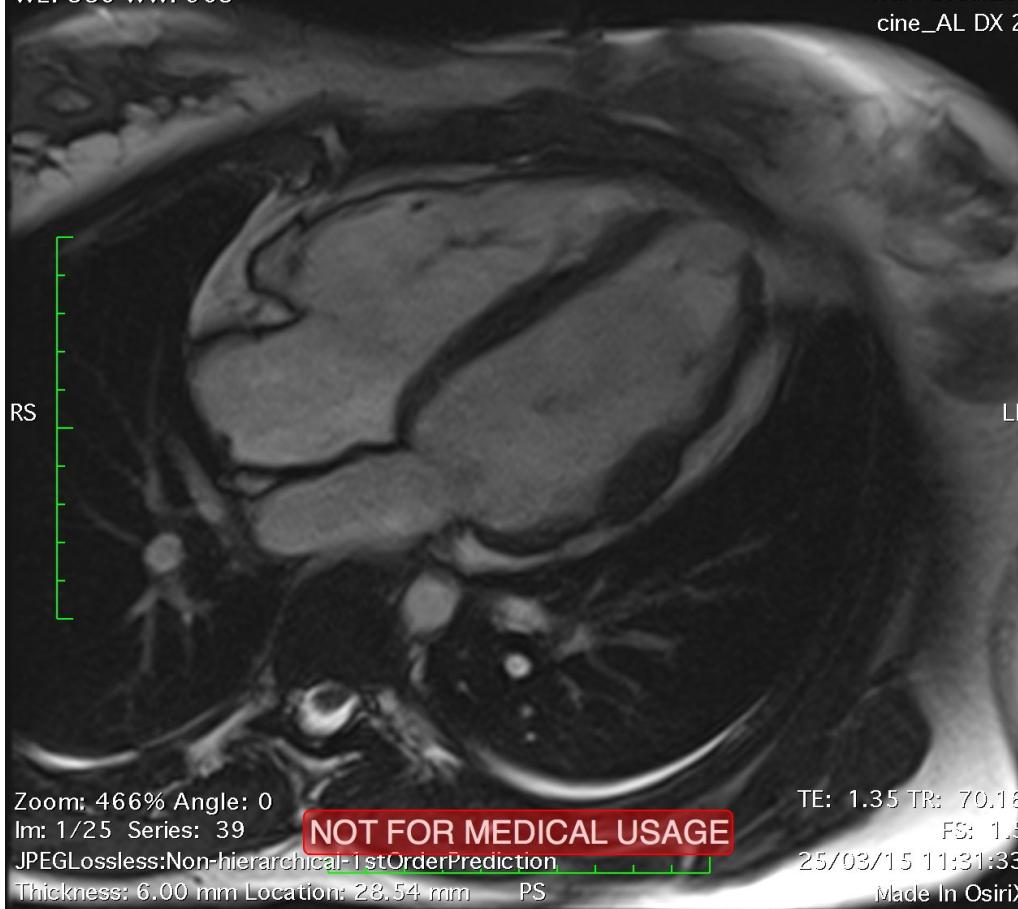




Image size: 216 x 272 SL 48507066 ( 22 y , 22 y )  
WL: 214 WW: 478 Heart Localizer  
cine\_volumi\_sak

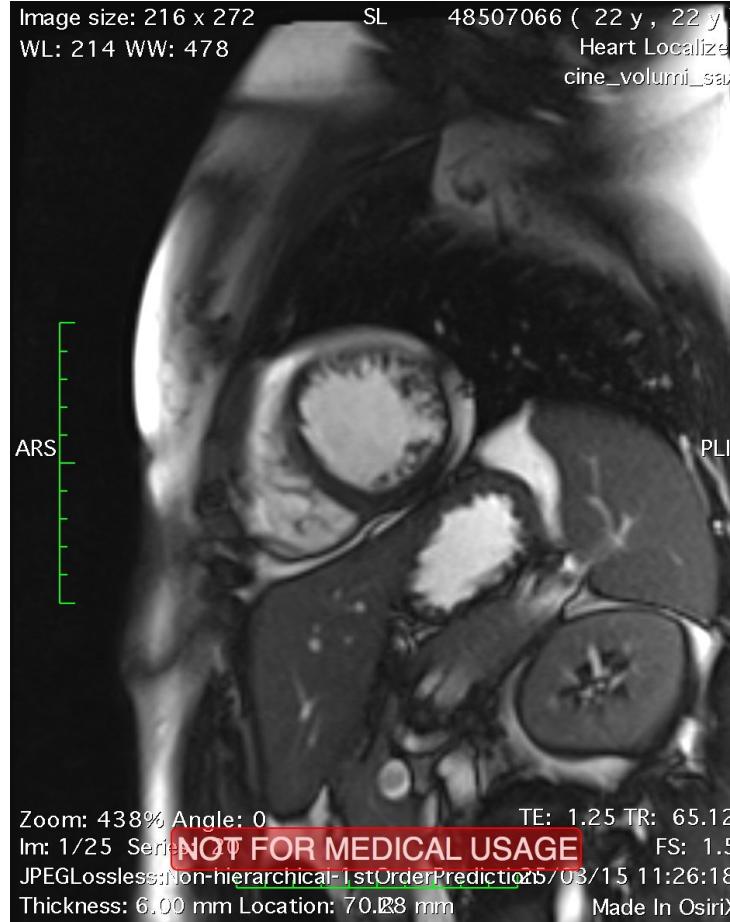
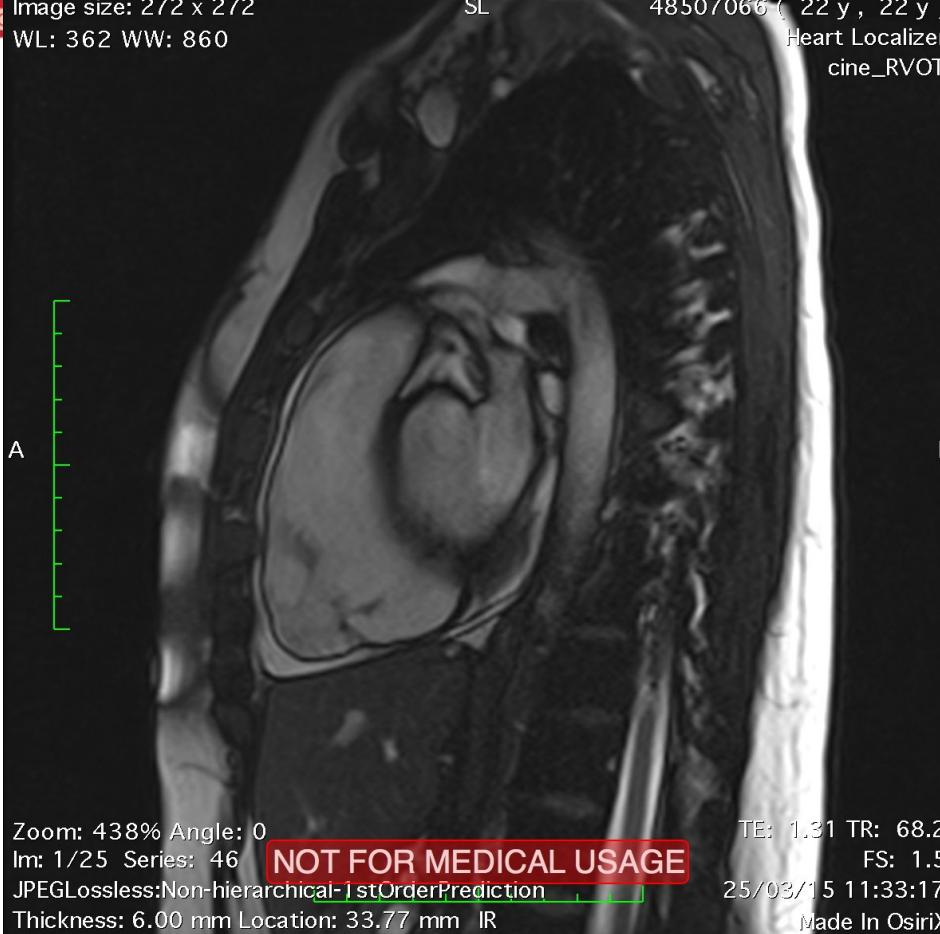


Image size: 272 x 272  
WL: 362 WW: 860

SL

48507066 ( 22 y , 22 y )  
Heart Localizer  
cine\_RVOT



Zoom: 438% Angle: 0

Im: 1/25 Series: 46

JPEGLossless:Non-hierarchical-1stOrderPrediction

Thickness: 6.00 mm Location: 33.77 mm IR

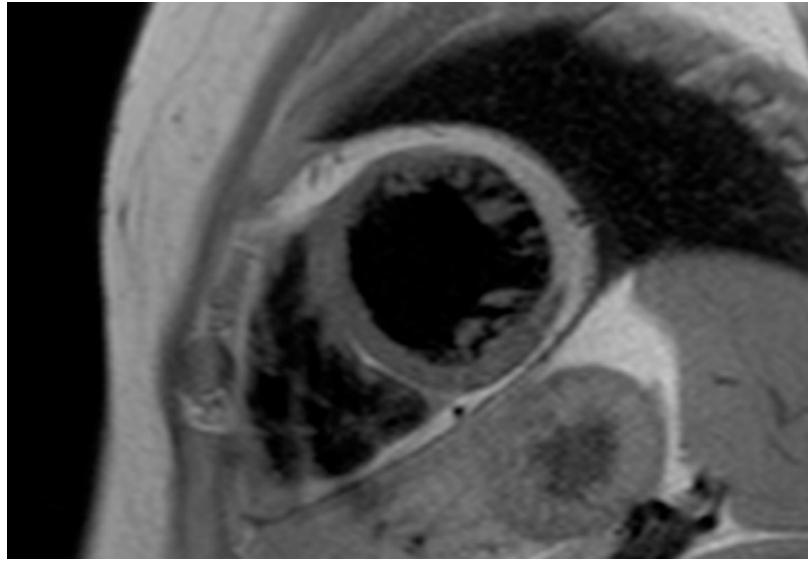
TE: 1.31 TR: 68.2

FS: 1.5

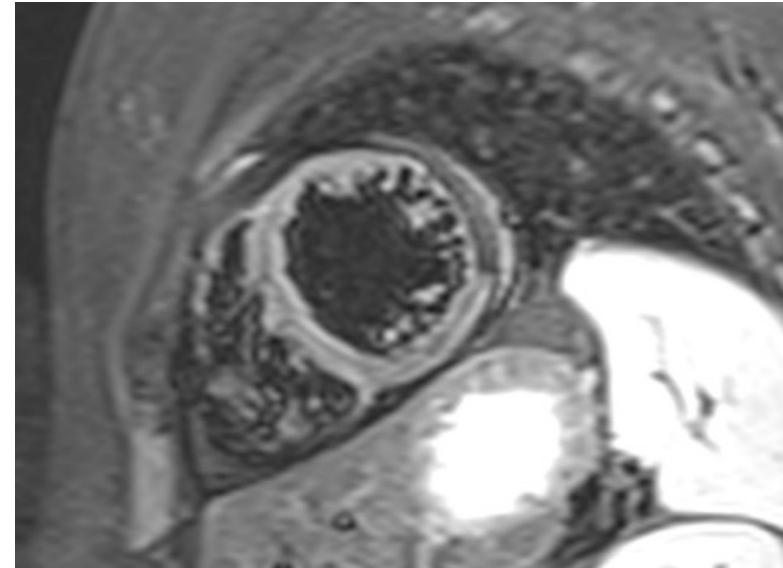
25/03/15 11:33:17

Made In OsiriX





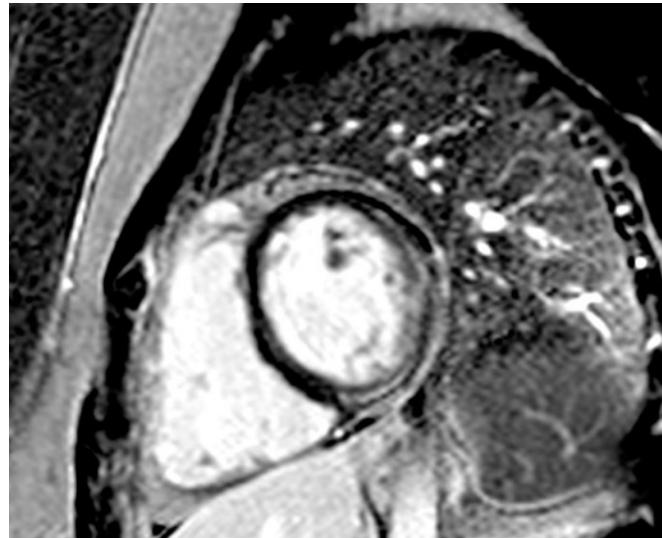
Black blood T2

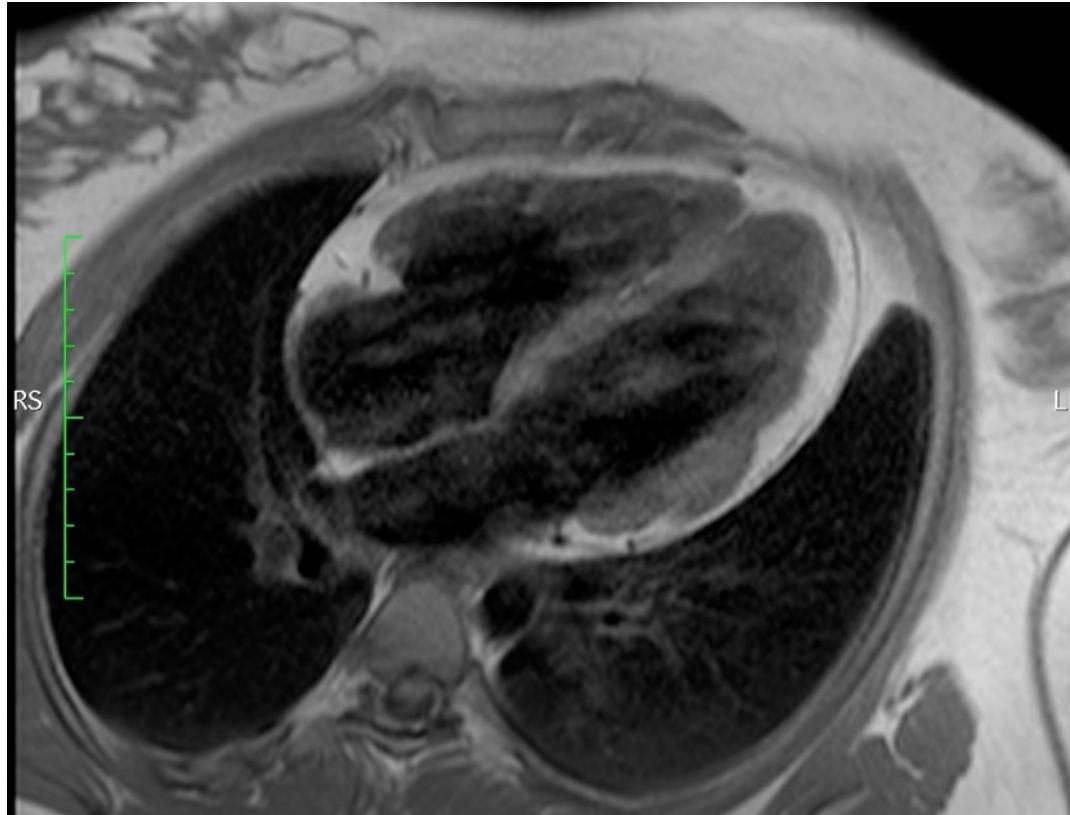


Black blood T1  
Fatty infiltration



## LGE: lateral, infero-lateral and inferior wall of the LV





Il giorno 31.1.1994 ore ..... ) è stato

eseguito il riscontro diagnostico ( da ore di

[REDACTED] a 35 deceduto

In codesto ospedale il 30.1.1994 (ore .....)

**DIAGNOSI ANATOMICA (reg. delle autopsie N. 11 1994 )**

Ipertrofia cardiaca biventricolare ed atriale con dilatazione delle cavità cardiache.

Sostituzione adiposa di ampi tratti della parete libera del ventricolo dx e delle porzioni anteriori del setto interventricolare.

Epatomegalia da stasi cronica riacutizzata. Edema polmonare acuto terminale.

C.M.: Sfiancamento delle cavità cardiache ed edema polmonare acuto in soggetto con cardio-

miopatia dilatativa idiopatica.

**Risultato esami istologici :**

Cuore - Ipertrofia miocellulare diffusa con fibrosi subendocardica, perivascolare e perimicocarditaria. infiltrazione adiposa di ampi tratti del ventricolo dx e del setto.

Fegato - Stasi cronica riacutizzata. Steatosi epatica diffusa a grandi gocce.

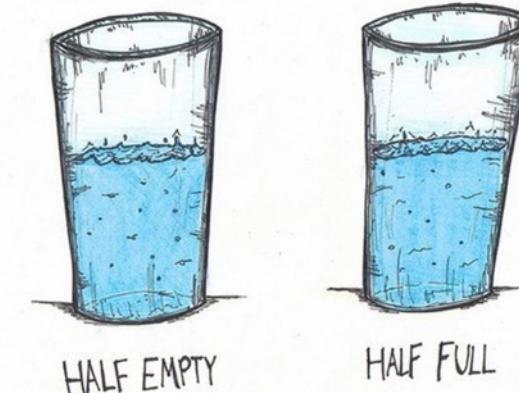
IL DIRETTORE





- Differential diagnosis
- Risk stratification for the treatment and indication to further investigations (e.g. estimation of LV ejection fraction)
- Crucial imaging technique for the follow up

*Is the glass half empty or half full for you?*





## CONCLUSIONI

- Il cuore d'atleta, soprattutto in alcune discipline, presenta frequentemente una dilatazione ventricolare destra indotta da esercizio, normalmente non associate a perdita di funzione e SENZA dimostrazione di anomalie della cinetica parietale segmentaria
- Al contrario della cardiomiopatia aritmogena, il cuore d'atleta è tipicamente caratterizzato da un rimodellamento armonico e simmetrico
- La diagnosi delle forme sinistre è particolarmente complessa. Tuttavia, un'attenta analisi dell'ECG ed un'analisi qualitativa delle aritmie ventricolari aiuta a porre il sospetto diagnostico



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## Master Teorico-pratico III edizione

### Ecocardiografia ed Imaging in Cardiologia dello Sport

Il Master si pone l'obiettivo di approfondire le conoscenze teoriche e pratiche nella diagnosi, stratificazione del rischio e gestione clinica degli atleti che presentano un rimodellamento fisiologico indotto dall'allenamento e in coloro che sono portatori di patologie potenzialmente a rischio di morte cardiaca improvvisa (v. cardiomiopatie).

Obiettivo del Master è imparare a condurre un esame ecocardiografico mirato alle specifiche richieste nel campo della Medicina dello Sport e della Cardiologia dello Sport e interpretare i dati di imaging ai fini di un inquadramento diagnostico appropriato. Verranno inoltre approfondite le indicazioni cliniche e l'interpretazione dell'imaging ecocardiografico avanzato, inclusi l'ecocardiogramma da sforzo e da stress, con particolare riferimento inoltre alle metodiche di *strain* ed ecocardiogramma tridimensionale. Infine, verranno inoltre trattate l'indicazione e l'interpretazione clinica degli esami di risonanza magnetica cardiaca, con particolare riferimento alle cardiomiopatie.



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Cardiologia dello Sport Siena

[flavio.dascerzi@unisi.it](mailto:flavio.dascerzi@unisi.it)

**DEADLINE DOMANDA ISCRIZIONE: 1 NOVEMBRE 2022!**



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