

Recomendaciones para la práctica deportiva en miocardiopatías

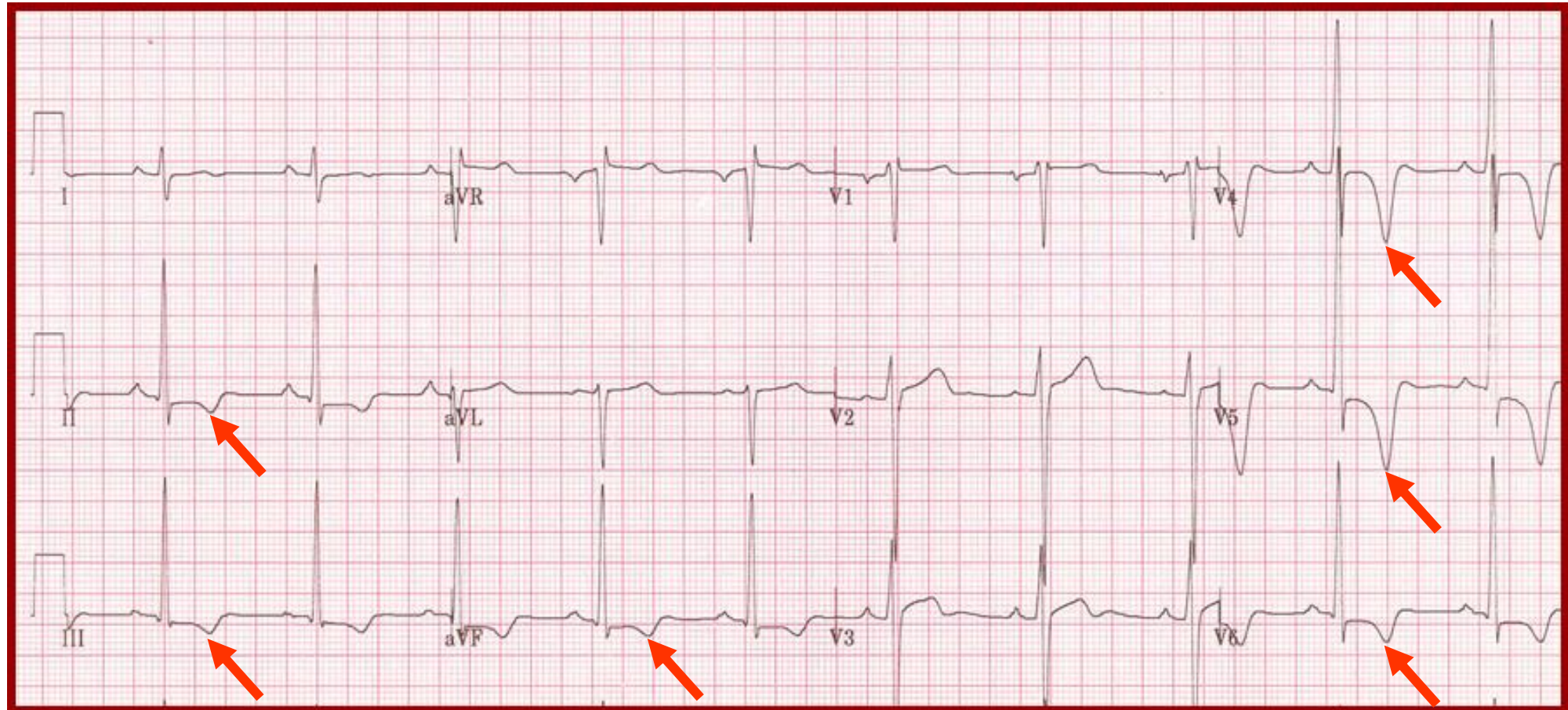
ESC-EAPC 2019

Luis Serratos, MD, PhD



Baloncesto profesional, 24 a

Asintomático, historia personal y familiar normal, caucásico



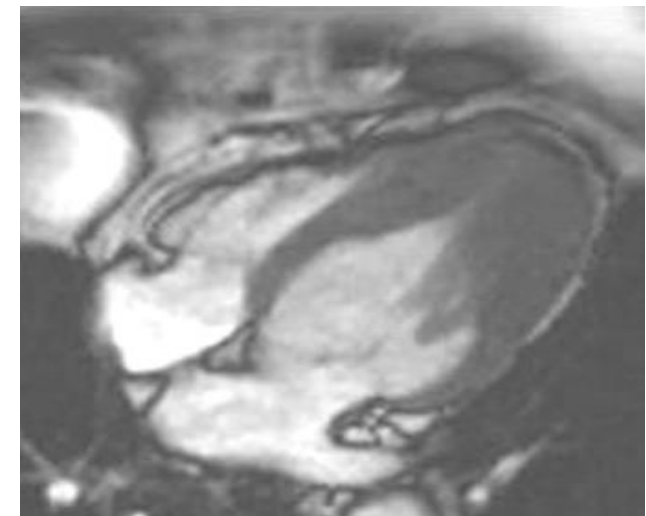
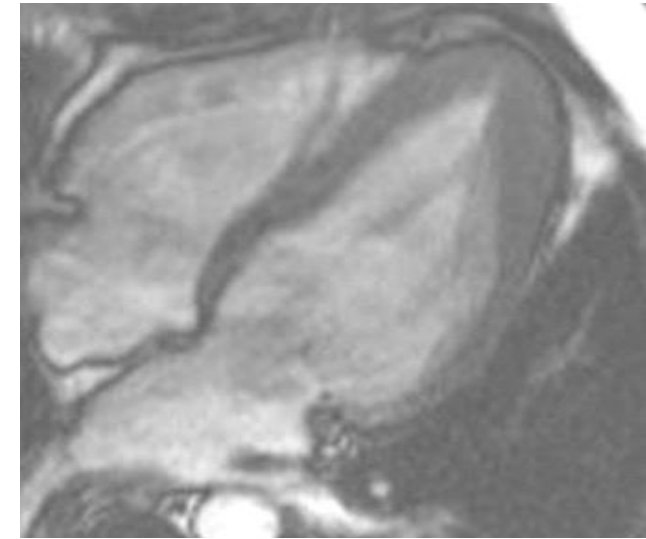
ECHO:

- *Left & right apical hypertrophy up to 18 mm*
- *LVDd 52 mm*
- *Normal systolic and diastolic function*

CPX & ECG Holter monitoring:

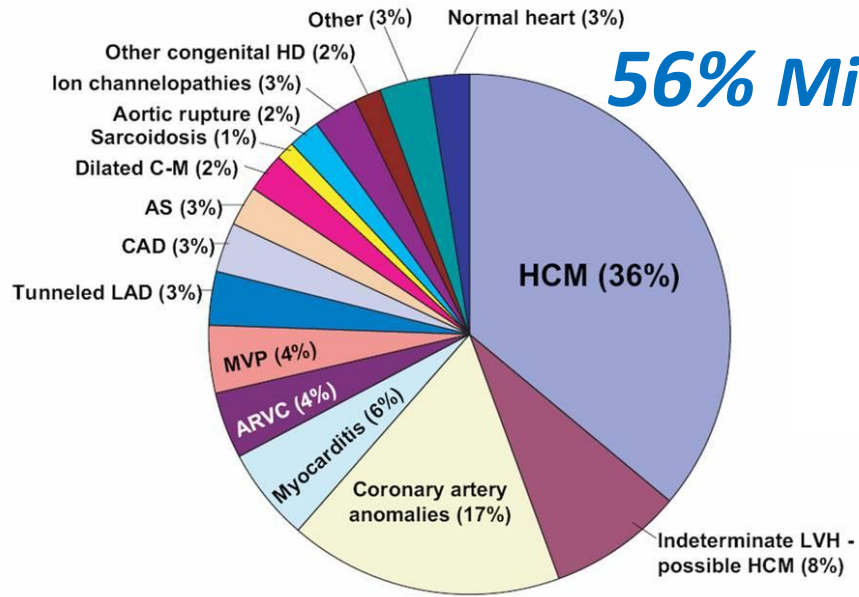
- *NO arrhythmias / Normal exercise BP response*
- *$VO_2max < 50$ ml/kg/min*

MRI: NO LGE



Apical Hypertrophic Cardiomyopathy

MUERTE SÚBITA CARDIACA EN DEPORTISTAS: CAUSAS

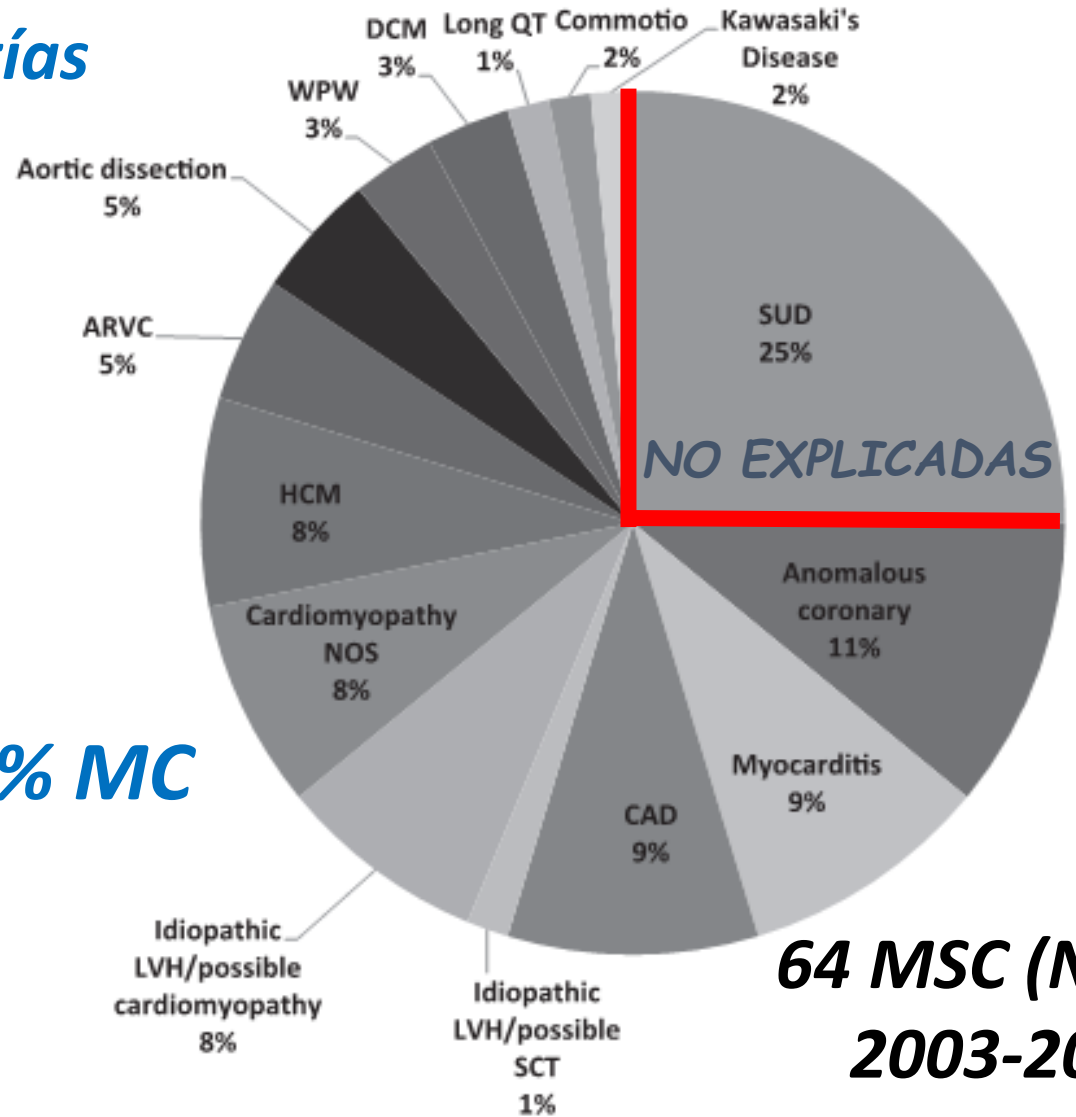


1435 MSC (jóvenes)

1980-2005

Maron et al. Circulation 2007

42 % MC



64 MSC (NCAA)

2003-2013

Harmon et al. Circulation 2015

1994-2014

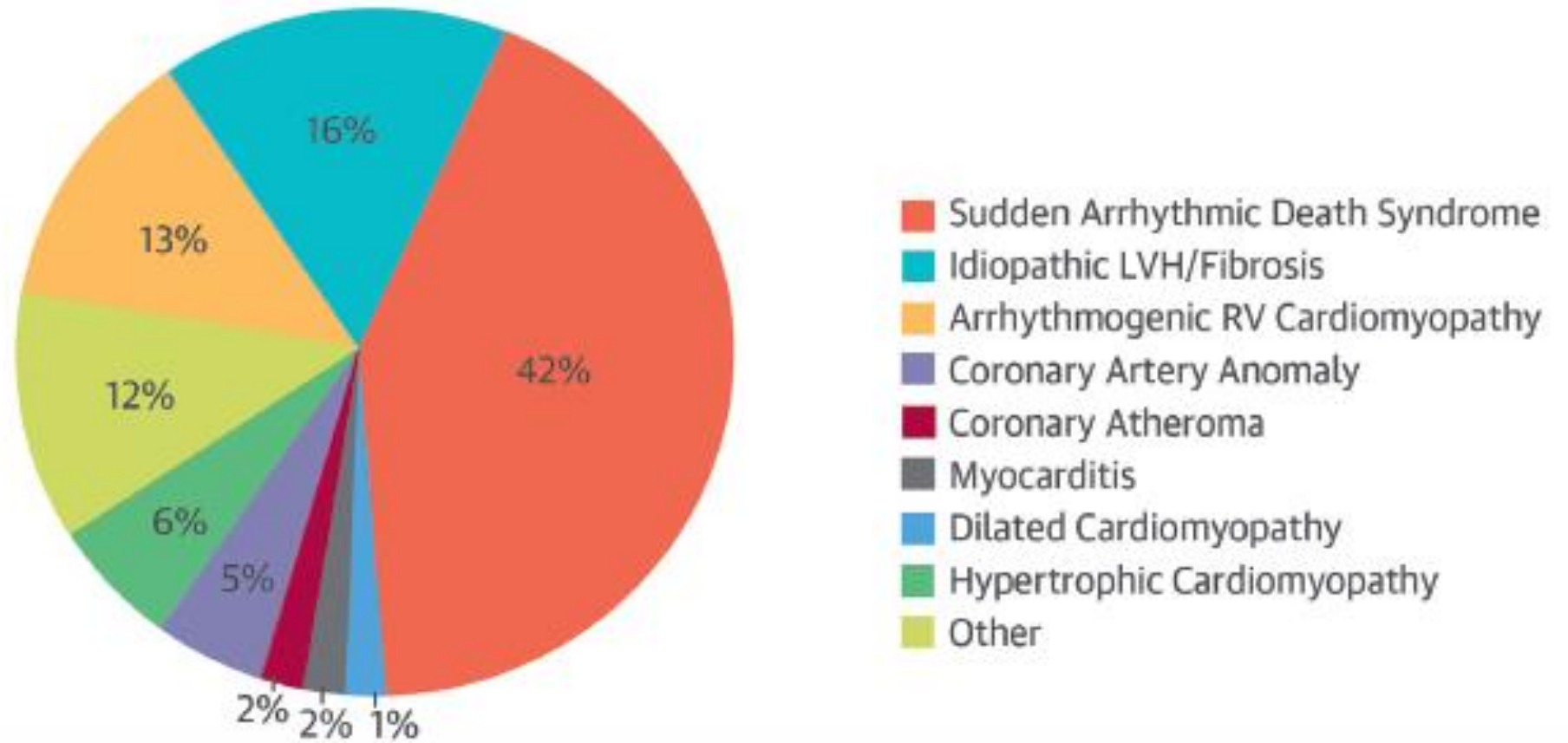
n: 357 MSC

29 ± 11 a (7-67 a)

69 % Competición

25 % Fútbol

38 % Miocardiopatías



ORIGINAL ARTICLE

Outcomes of Cardiac Screening
in Adolescent Soccer Players

Malhotra et al. NEJM 2018

- *1996-2016*
- *11168 jugadores fútbol (15-17 a, FA)*
- *0,38% con cardiopatías riesgo MSC*
- ***8 casos de MSC: 7 miocardiopatías (3 MCH, 6 recon normal)***
- *MSC: 1/14794/año (6,8/100000/año)*

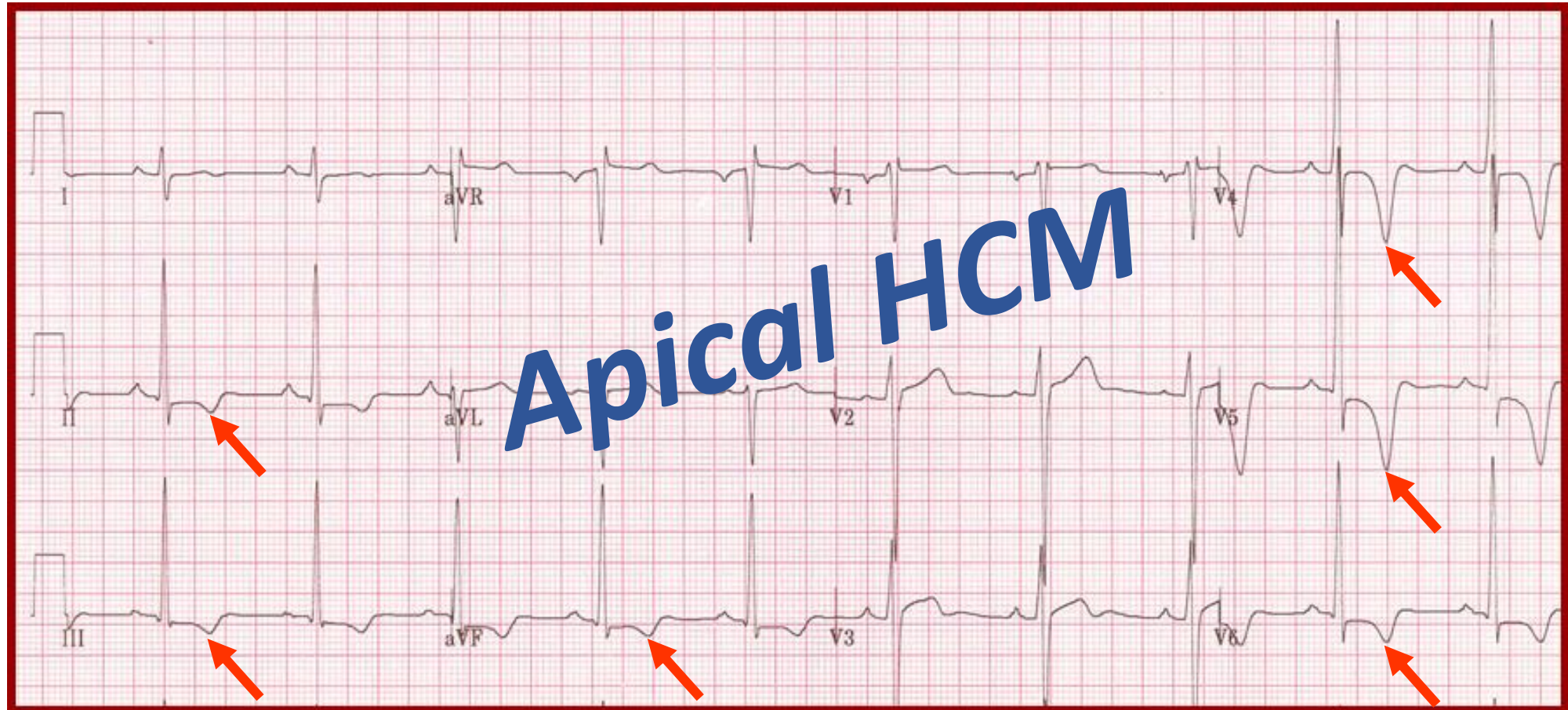
Valoración riesgo

- *MSC/PC*
- *Arritmias*
- *Acelerar progresión de la cardiopatía*



Baloncesto profesional, 24 a

Asintomático, historia personal y familiar normal, caucásico



Recommendations for competitive sports participation in athletes with cardiovascular disease **2005**

A consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology

AHA/ACC SCIENTIFIC STATEMENT

2015

Eligibility and Disqualification
Recommendations for Competitive
Athletes With Cardiovascular Abnormalities:
Task Force 3: Hypertrophic Cardiomyopathy,
Arrhythmogenic Right Ventricular
Cardiomyopathy and Other
Cardiomyopathies, and Myocarditis

A Scientific Statement From the American Heart Association and American College of Cardiology

*“Athletes with a probable or unequivocal clinical expression and diagnosis of HCM (ie, with the disease phenotype of LV hypertrophy) **SHOULD NOT PARTICIPATE** in most competitive sports, with the exception of those of low intensity (class IA sports)”*

Sport Disciplines



Skill



Power



Mixed



Endurance

Isometric	+/-	Isometric	+++/+	Isometric	++/+++	Isometric	++/+++
Isotonic	+/-	Isotonic	+/++	Isotonic	++/+++	Isotonic	+++/+
Cardiac remodeling	+/-	Cardiac remodeling	+/++	Cardiac remodeling	++/+++	Cardiac remodeling	+++

- Golf
- Archery
- Sailing
- Table Tennis
- Equestrian
- Karate
- Shooting/Rifle
- Curling
- Sled disciplines
- Ski Jumping

- Weightlifting
- Wrestling / Judo
- Boxing
- Short distance running
- Shot-putting
- Discus / Javelin
- Artistic gymnastics
- Bobsleigh
- Short-track skating
- Alpine skiing
- Snowboarding

- Soccer
- Basketball
- Volleyball
- Water polo
- Badminton
- Tennis
- Fencing
- Handball
- Rugby
- Hockey / Ice-hockey

- Cycling
- Rowing
- Mid/long distance swimming
- Mid/long distance running
- Canoeing
- Triathlon
- Pentathlon
- X-country skiing
- Biathlon
- Long distance skating

Surviving Competitive Athletics with Hypertrophic Cardiomyopathy

Barry J. Maron, MD, and Heinrich G. Klues, MD *AJC 1994*

n: 14 (13 males)

43 yrs (30-66)

HCM identified at 34 (24-57)

LVWTh 18-28 mm

Sports (distance running, swimming, triathlon, basketball, football)

Training 15 yrs (6-22)

12/14 performed either at the national, collegiate or professional level

*“It is possible for **some patients with HCM to tolerate particularly intense athletic training and competition for many years, and even maintain high levels of achievement without incurring symptoms and disease progression or dying suddenly. The explanation for such a favourable clinical course in these particular athletes with HCM is uncertain, and provides an **incentive for further efforts at risk stratification in such persons**”***

ICD Multinational Registry (Lampert et al. Circulation 2013)

n= 372 (60+72 competitive)

5 hrs/wk (13 hrs/wk)

33 yrs (10-60)

n= 89 <20 yrs

33% Female

62% on β -blockers

42% ICDs 2ndary prevention

31 months follow-up

Shocks due to VT/VF during training/competition (n= 21), n (%)

Cardiac diagnosis		
Idiopathic VT/VF (normal heart)	33	4 (12)
CAD	39	3 (8)
ARVD	53	8 (15)
HCM	65	1 (2)
LQTS	73	2 (3)
Others	109	3 (3)

HCM with ICD: same frequency of shocks exercise vs rest

- **88 athletes with HCM**
- *71% asymptomatic (11% syncope/presyncope)*
- *88% low risk, 9% intermediate risk, 3% high risk*
- *Football, athletics, rowing, swimming, basket, cycling, triathlon.....*
- **7 yr follow-up**
- **61 stopped or leisure-time sport**
 - *1 SCD, 1 SCA, 23% symptoms (3 syncope, 7 palpitations, 2 dyspnea, 2 chest pain)*
- **27 continued training & competition**
 - *No SCA/SCD, 15% symptoms (1 syncope, 3 palpitations)*

*“In the absence of robust evidence, these guidelines largely reflect **EXPERT OPINION** and **canNOT be considered as LEGALLY BINDING**”*

*“the present document encourages **SHARED DECISION MAKING** with the athlete and respects the autonomy of the athlete **after careful information** about the potential risks of an adverse event”*

High Risk HCM: absolute contraindication

- *History of aborted SCD/CA*
- *Symptoms, particularly unheralded syncope*
- *Exercise-induced ventricular tachycardia*
- *High ESC 5-year risk score**
- *Significant increase in LV outflow gradient (>50 mmHg)*
- *Abnormal blood pressure response to exercise (<20 mmHg increase in SBP, or exercise-induced hypotension)*

Class IIb/Level C



HCM Risk-SCD Calculator

Age	<input type="text"/>	Years	Age at evaluation
Maximum LV wall thickness	<input type="text"/>	mm	Transthoracic Echocardiographic measurement
Left atrial size	<input type="text"/>	mm	Left atrial diameter determined by M-Mode or 2D echocardiography in the parasternal long axis plane at time of evaluation
Max LVOT gradient	<input type="text"/>	mmHg	The maximum LV outflow gradient determined at rest and with Valsalva provocation (irrespective of concurrent medical treatment) using pulsed and continuous wave Doppler from the apical three and five chamber views. Peak outflow tract gradients should be determined using the modified Bernouilli equation: $\text{Gradient} = 4V^2$, where V is the peak aortic outflow velocity
Family History of SCD	<input type="radio"/> No <input type="radio"/> Yes		History of sudden cardiac death in 1 or more first degree relatives under 40 years of age or SCD in a first degree relative with confirmed HCM at any age (post or ante-mortem diagnosis).
Non-sustained VT	<input type="radio"/> No <input type="radio"/> Yes		3 consecutive ventricular beats at a rate of 120 beats per minute and <30s in duration on Holter monitoring (minimum duration 24 hours) at or prior to evaluation.
Unexplained syncope	<input type="radio"/> No <input type="radio"/> Yes		History of unexplained syncope at or prior to evaluation.

Risk of SCD at 5 years (%):

ESC recommendation:

Should not be used in elite/competitive athletes

2014 ESC Guidelines on Diagnosis and Management of Hypertrophic Cardiomyopathy (Eur Heart J 2014 – doi:10.1093/eurheartj/ehu284)

O'Mahony C et al Eur Heart J (2014) 35 (30): 2010-2020

HCM Risk-SCD should not be used in:

- Paediatric patients (<16 years)
- Elite/competitive athletes
- HCM associated with metabolic diseases (e.g. Anderson-Fabry disease), and syndromes (e.g. Noonan syndrome).
- Patients with a previous history of aborted SCD or sustained ventricular arrhythmia who should be treated with an ICD for secondary prevention.

Caution should be exercised when assessing the SCD in patients following invasive reduction in left ventricular outflow tract obstruction with myectomy or alcohol septal ablation.

Pending further studies, HCM-RISK should be used cautiously in patients with a maximum left ventricular wall thickness ≥ 35 mm.

HCM = hypertrophic cardiomyopathy; LV = left ventricular; LVOT = left ventricular outflow tract; NSVT = non-sustained ventricular tachycardia; SCD = sudden cardiac death; VT = ventricular tachycardia

HCM

Following comprehensive evaluation and **explanation** of the disease characteristics, **risk factors** and **potential outcomes** and assuring that a reasonable understanding and **AGREEMENT** has been reached between the athlete and the physician, it seems reasonable that adult athletes with:

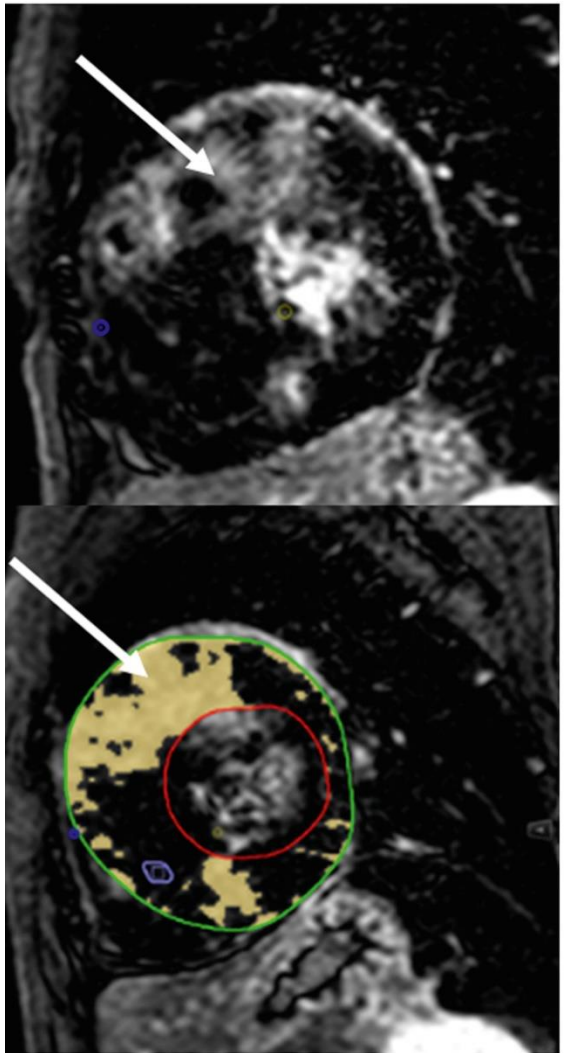
- **Mild clinical expressions of HCM**
- **Low ESC risk score ***
- **Adult age**

Class IIb/Level C

MAY **selectively** BE **ALLOWED** to participate in **all competitive sports**, with **exception of those where occurrence of syncope may be associated with harm or death.**

Such athletes should be **reviewed annually** to assess symptoms and changes in risk profile.

MRI & HCM Risk



1423 low-/intermediate-risk patients with HCM
≥18 yrs (66 ± 14 yrs), 60% men
Preserved LVEF
5-year SCD risk score 2.3 ± 2.0
Primary endpoint: SCD and ICD appropriate shocks

Extensive LGE ($\geq 15\%$ of LV myocardium)
> Risk Ventricular Arrhythmias
> SCD

Mentias et al. JACC 2018

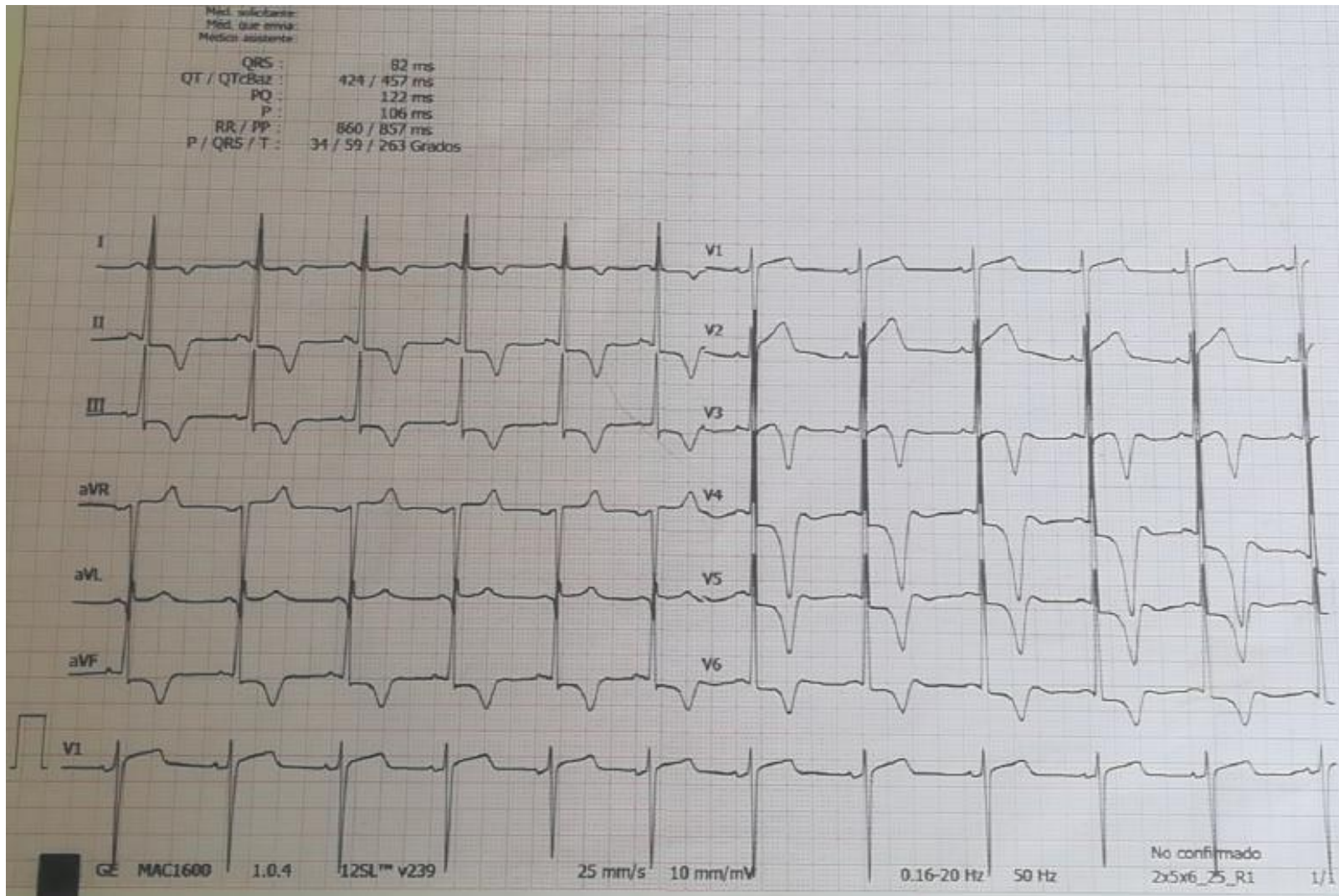
24 yr female, west african, asymptomatic pro football player

Apical HCM

MRI:13.5 mm
NO LGE

No LVOT gradient
No arrhythmias
Normal BP at Exercise
Low ESC Risk Score

Family history?



Hypertrophic cardiomyopathy (G+ P-)

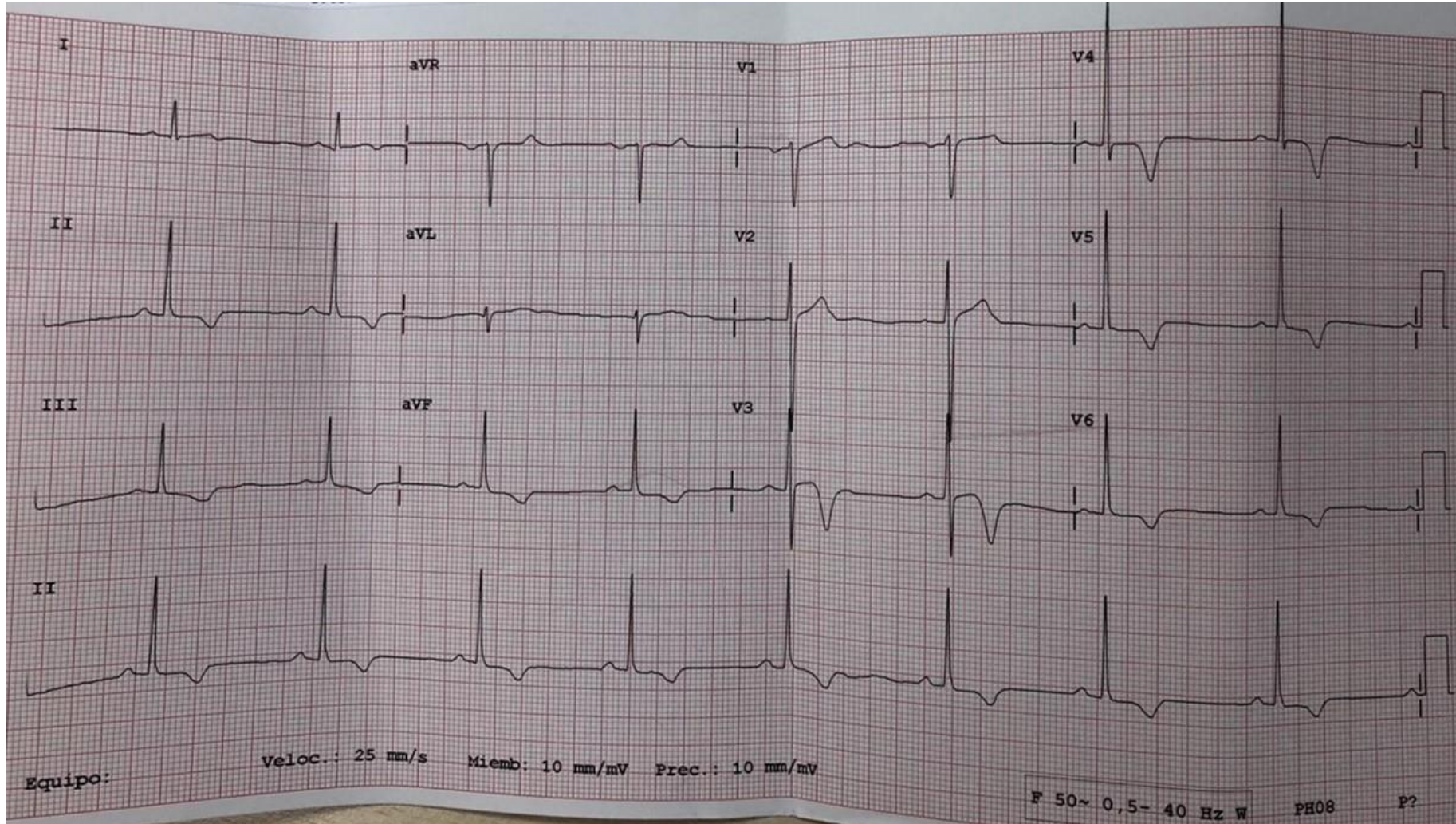
- 1. *G+P-*** individuals should be assessed to exclude the broader phenotypic and clinical features of HCM (with ECG, CMR, exercise test, and 24-h ECG monitoring). *In the absence of phenotypic features of HCM, these athletes **may be allowed to engage in all competitive sports.***

Class IIa/Level C
2. There is *limited data* in individuals with **positive genotype, negative phenotype, and an abnormal ECG in isolation**; therefore, until further data is available it is recommended that such individuals are managed similarly to those without LV hypertrophy.

Class III/Level C

Fútbol 3ª div, 25 a, raza negra

RM: SIV-PP 12 mm predominio apical



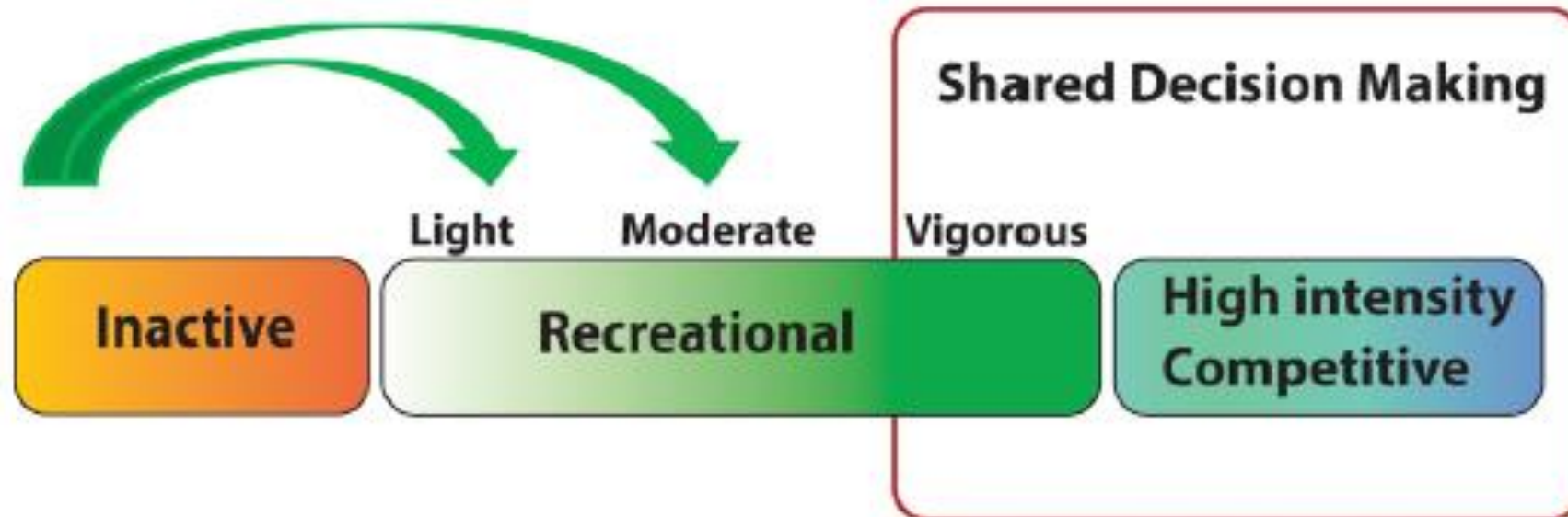
Miocardopatía Hipertrófica

PERSPECTIVE

Exercise and Hypertrophic Cardiomyopathy

Time for a Change of Heart

Saberi, Circulation 2018



Effect of Moderate-Intensity Exercise Training on Peak Oxygen Consumption in Patients With Hypertrophic Cardiomyopathy A Randomized Clinical Trial

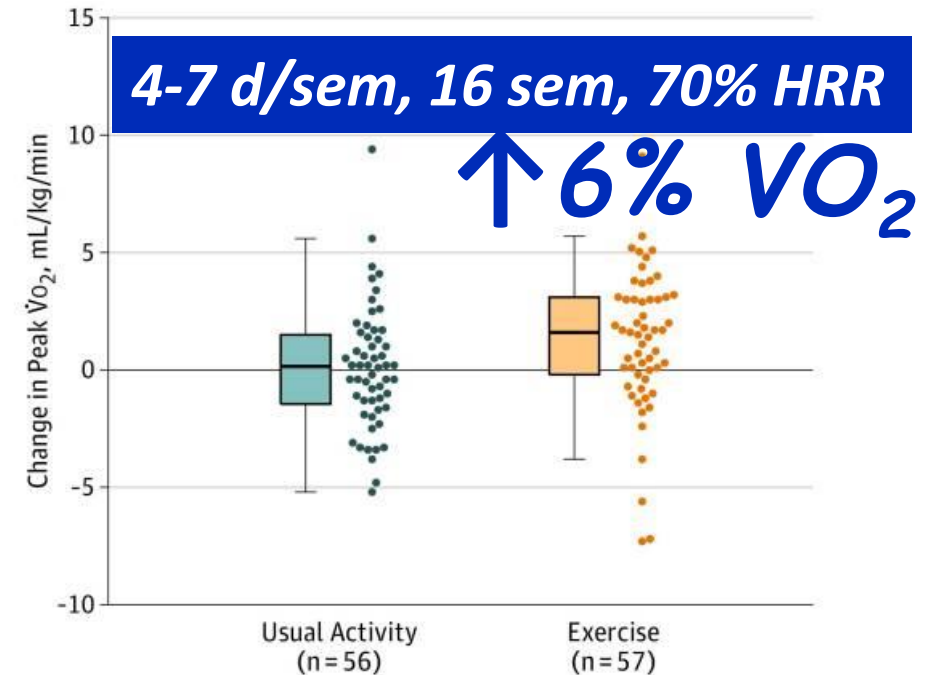
Saberri, JAMA 2017

Estudio RESET HCM

136 pacientes

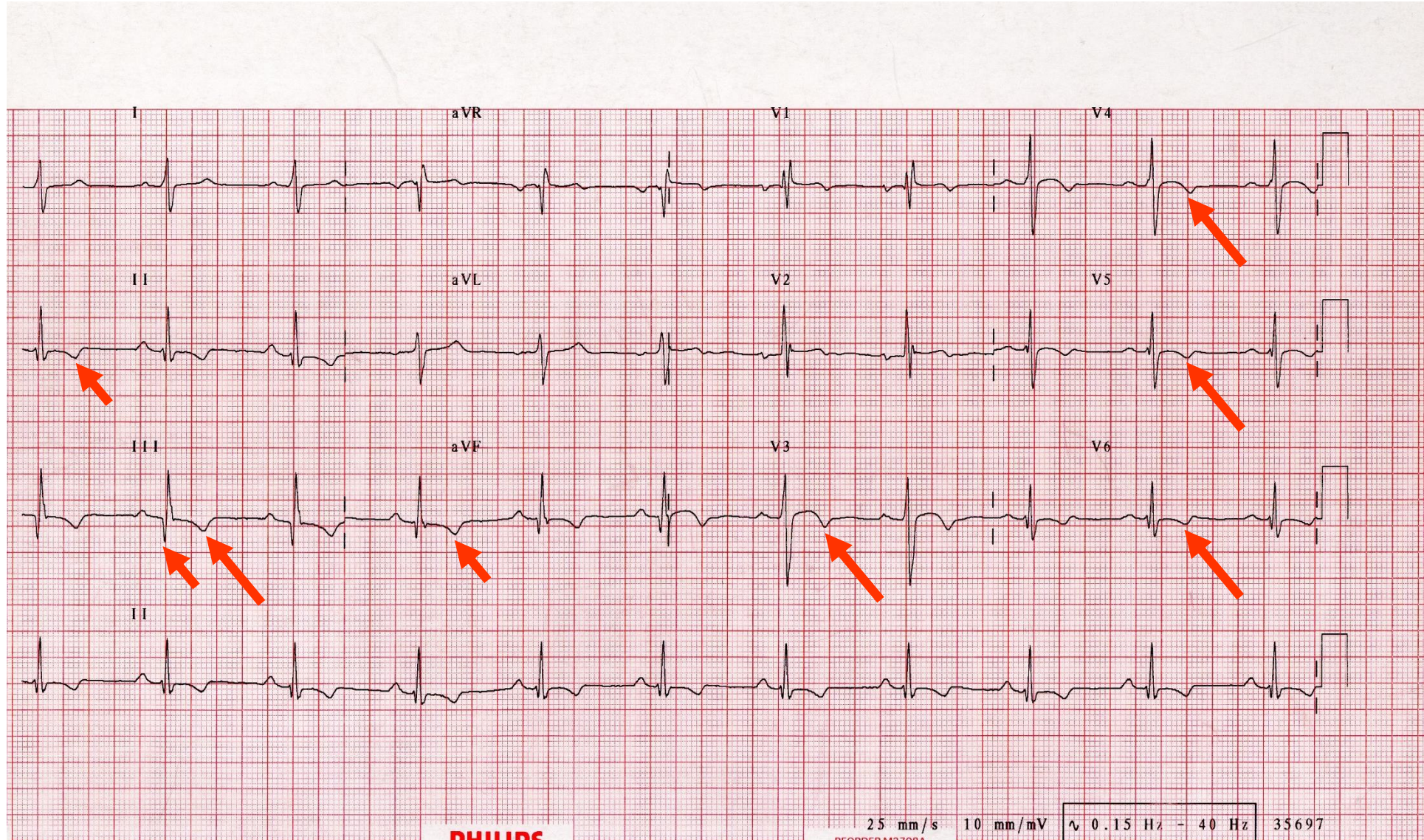
50 años

SIN eventos (AV, MS, PC, desfib)



“menor riesgo de mortalidad cardiovascular/hospitalizaciones”

Fútbolista profesional, 23 a asintomático, historia familiar y exploración física normales



- *EKG changes & 2nd-degree (type 1) AV block*
- *ECHO: low normal EF*
- *X test: sporadic isolated PVCs*
- *24 h Holter: infrequent PVCs (2 morphol), 1 triplet, 2nd degree type 1 AV block*

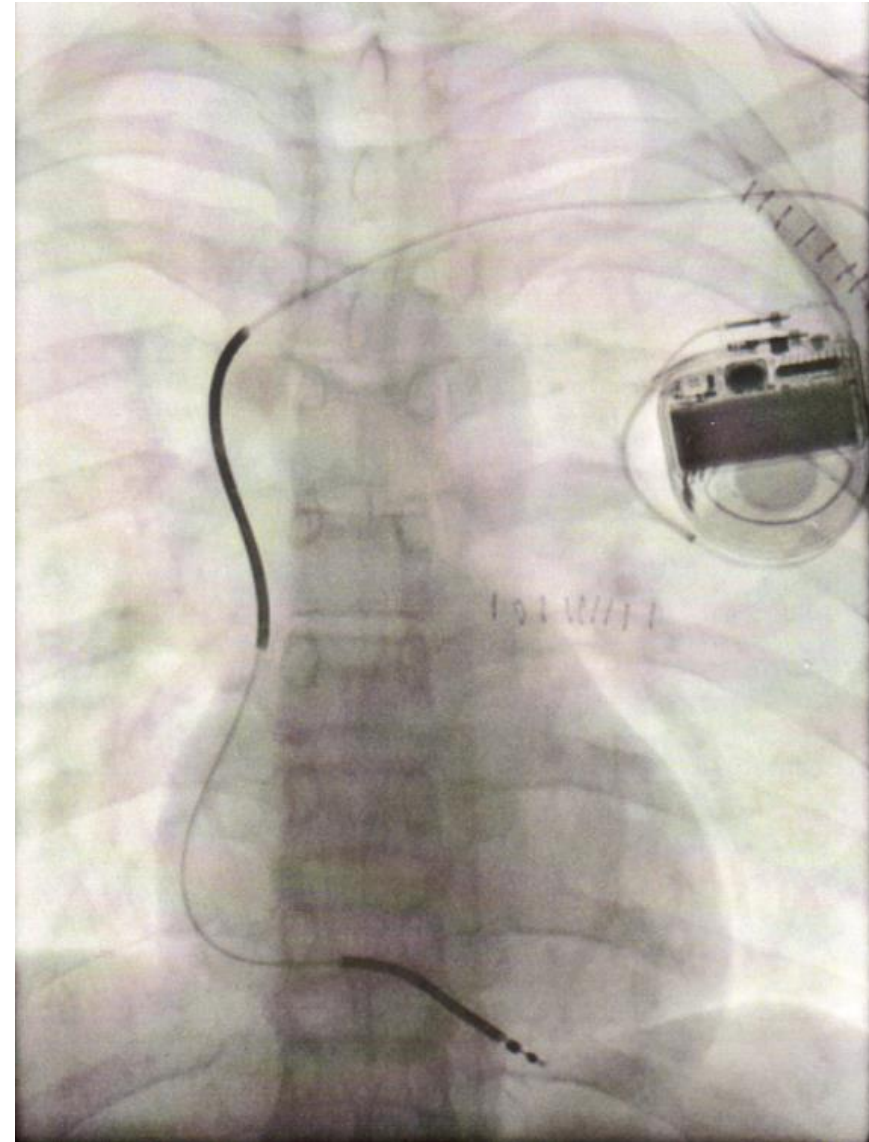
8 days later: EFFORT SYNCOPE



***Mid to apical inferolateral
LV late gadolinium enhancement***

***Left dominant
arrhythmogenic
cardiomyopathy***

*18 months post syncope
ICD*



Arrhythmogenic cardiomyopathy

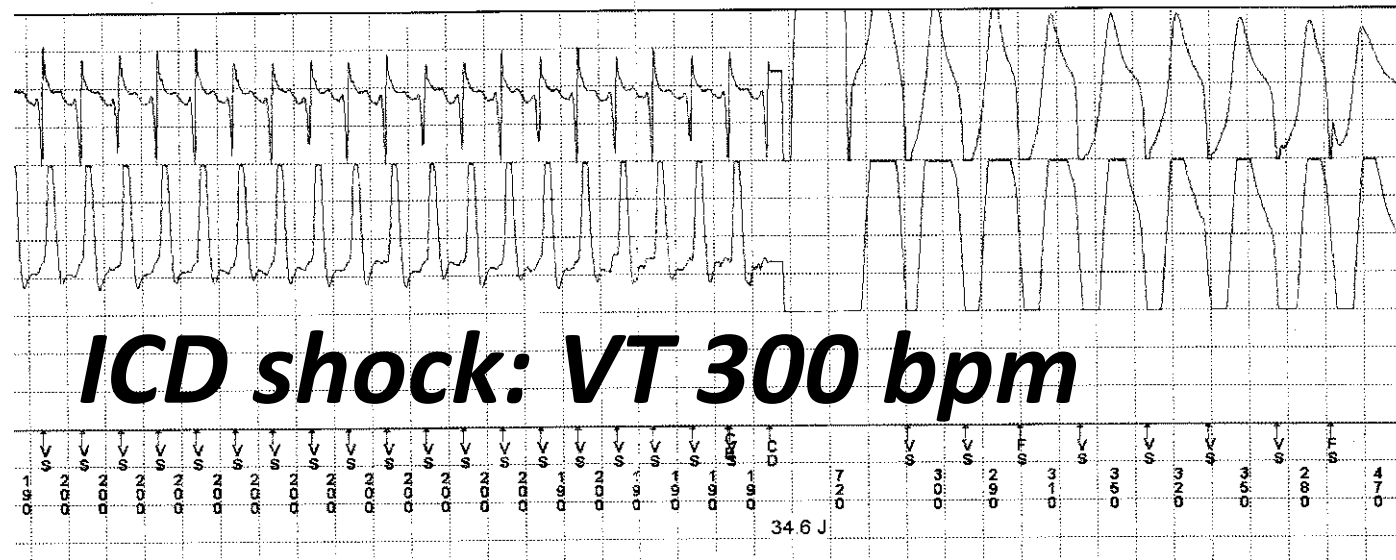
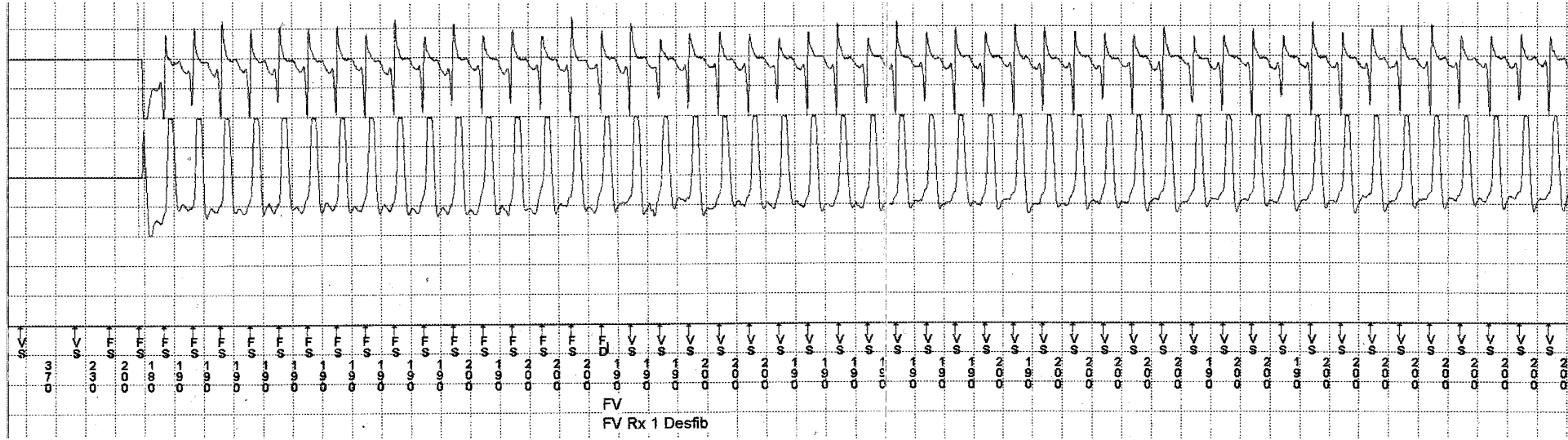
Athletes with unequivocal or probable diagnosis of AC **should not participate in competitive sports.**

These patients should be advised to limit their exercise programmes to **leisure-time activities**, and remain under clinical surveillance.

Class IIa/Level C

Athletes who are **genetic carriers** of pathogenic AC-associated desmosomal mutations (even in the **absence of phenotypic expression** of the disease) **should not participate in competitive sports.** These athletes should be advised to limit their exercise programmes to **leisure-time activities** and remain under clinical surveillance.

3 yrs post síncope



Myocarditis

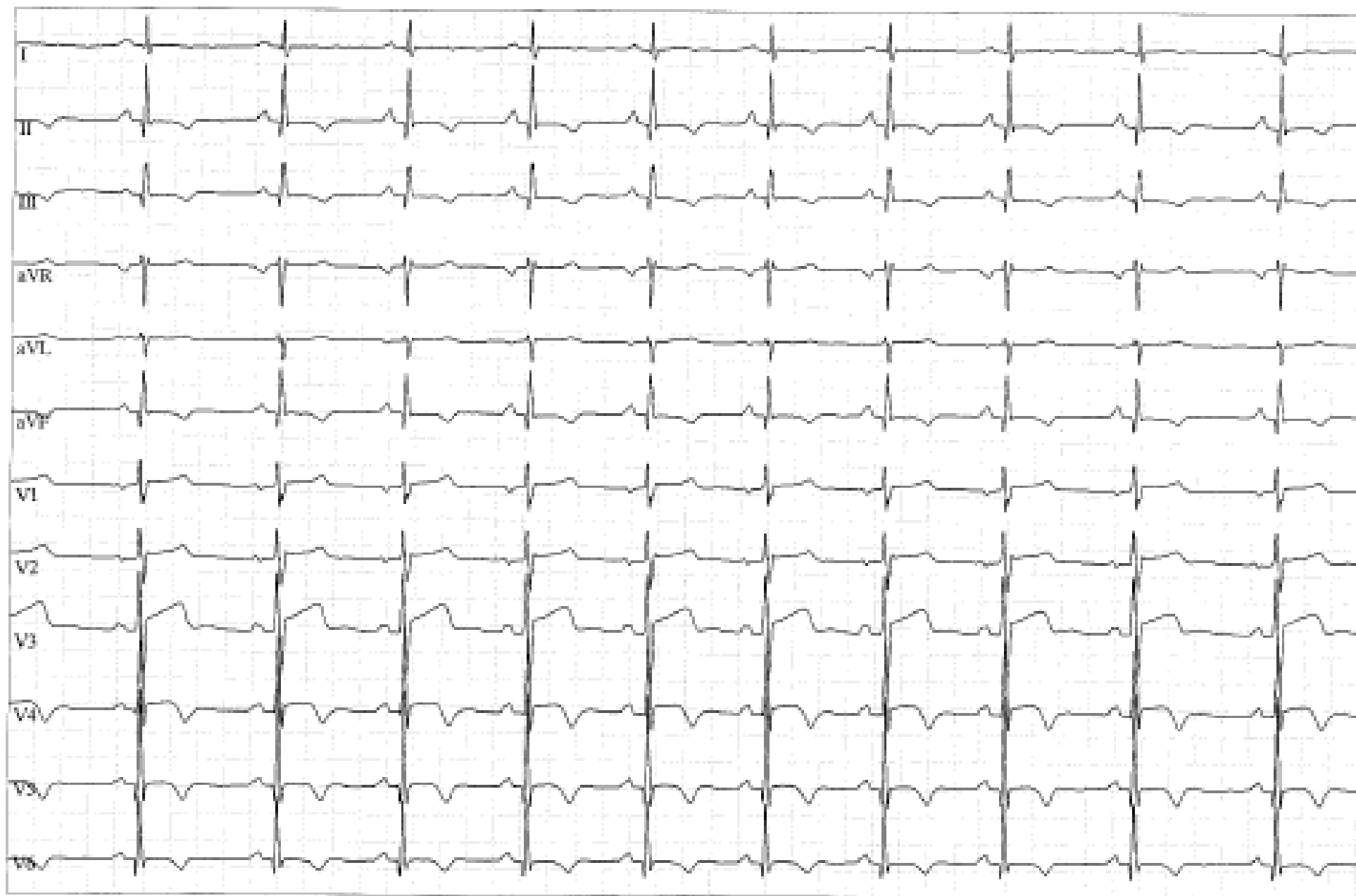
*General consensus exists that athletes with diagnosis of myocarditis should be restricted from exercise programmes for a period of **3–6 months**, according to the clinical severity and duration of the illness, LV function at onset, and extent of inflammation on the CMR. This time period is considered appropriate to ensure clinical and biological resolution of the disease.* **Class IIb/Level C**

*It is reasonable for athletes to resume training and competition after a myocarditis if **ALL** of the following criteria are met:*

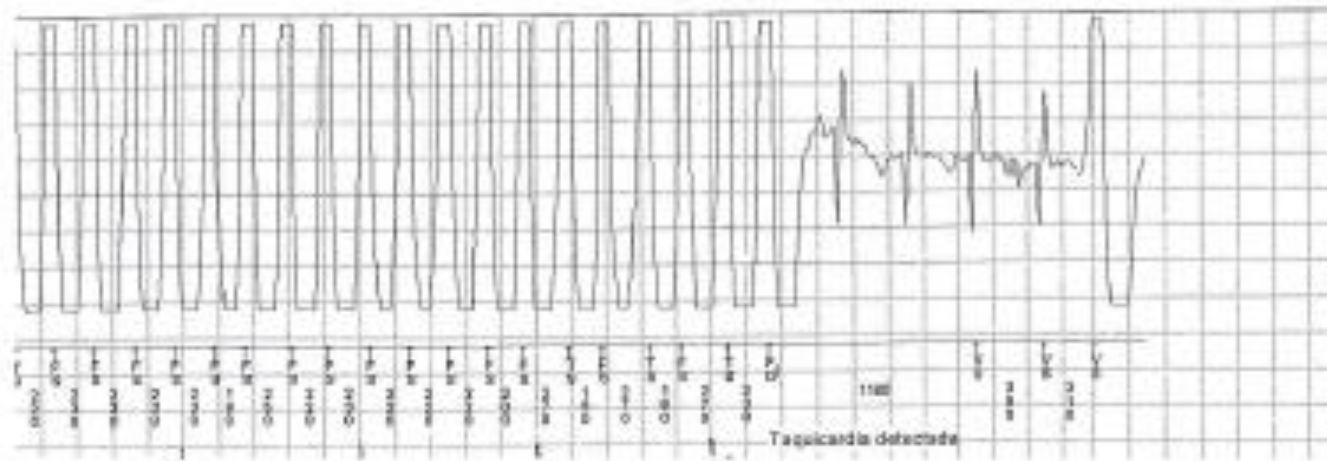
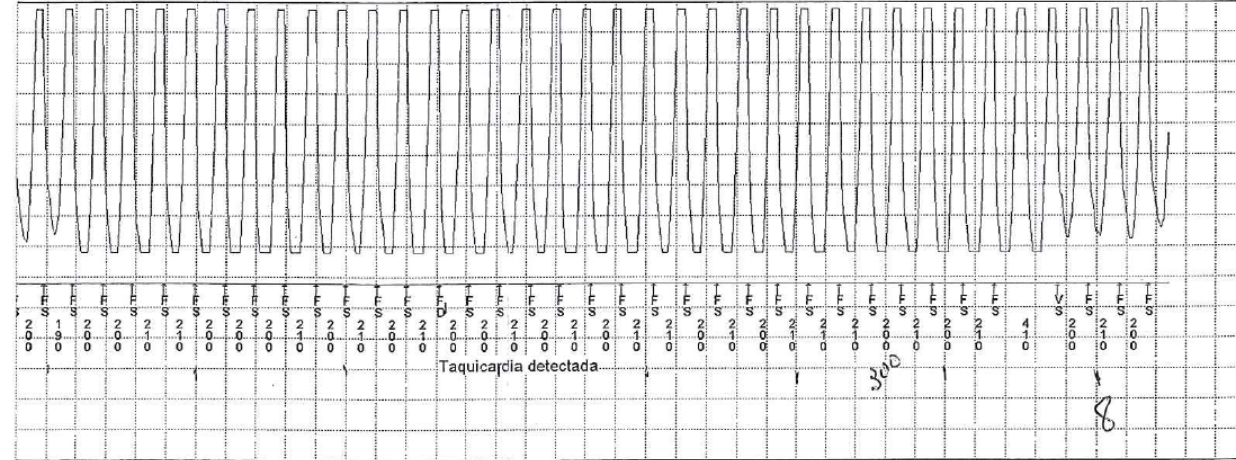
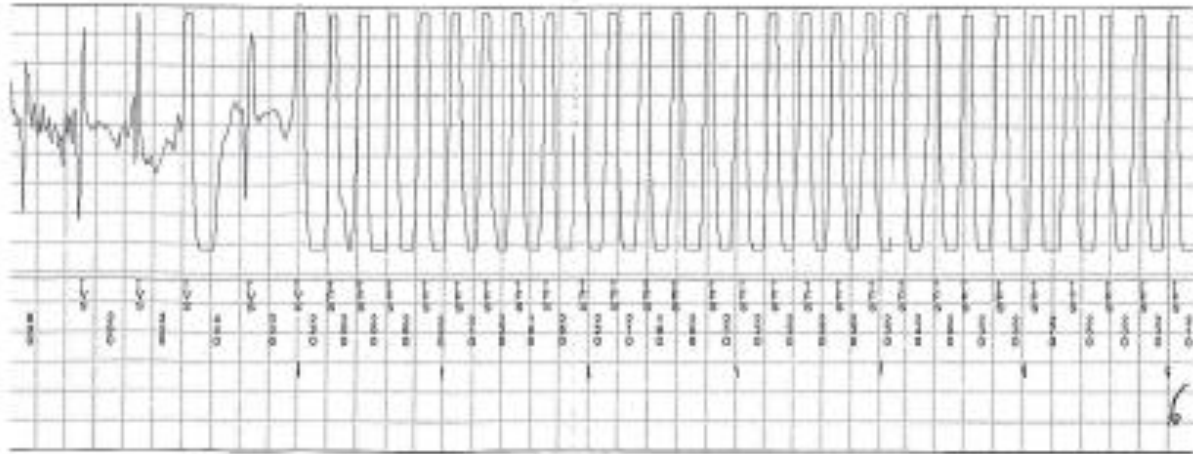
- (1) LV systolic function has returned to the normal range.*
- (2) Serum biomarkers of myocardial injury have normalized.*
- (3) Clinically relevant arrhythmias, such as frequent or complex repetitive forms of ventricular or supraventricular arrhythmias are absent on 24-h ECG monitoring and exercise test.* **Class IIa/Level C**

Fútbol profesional, 24 a, raza negra

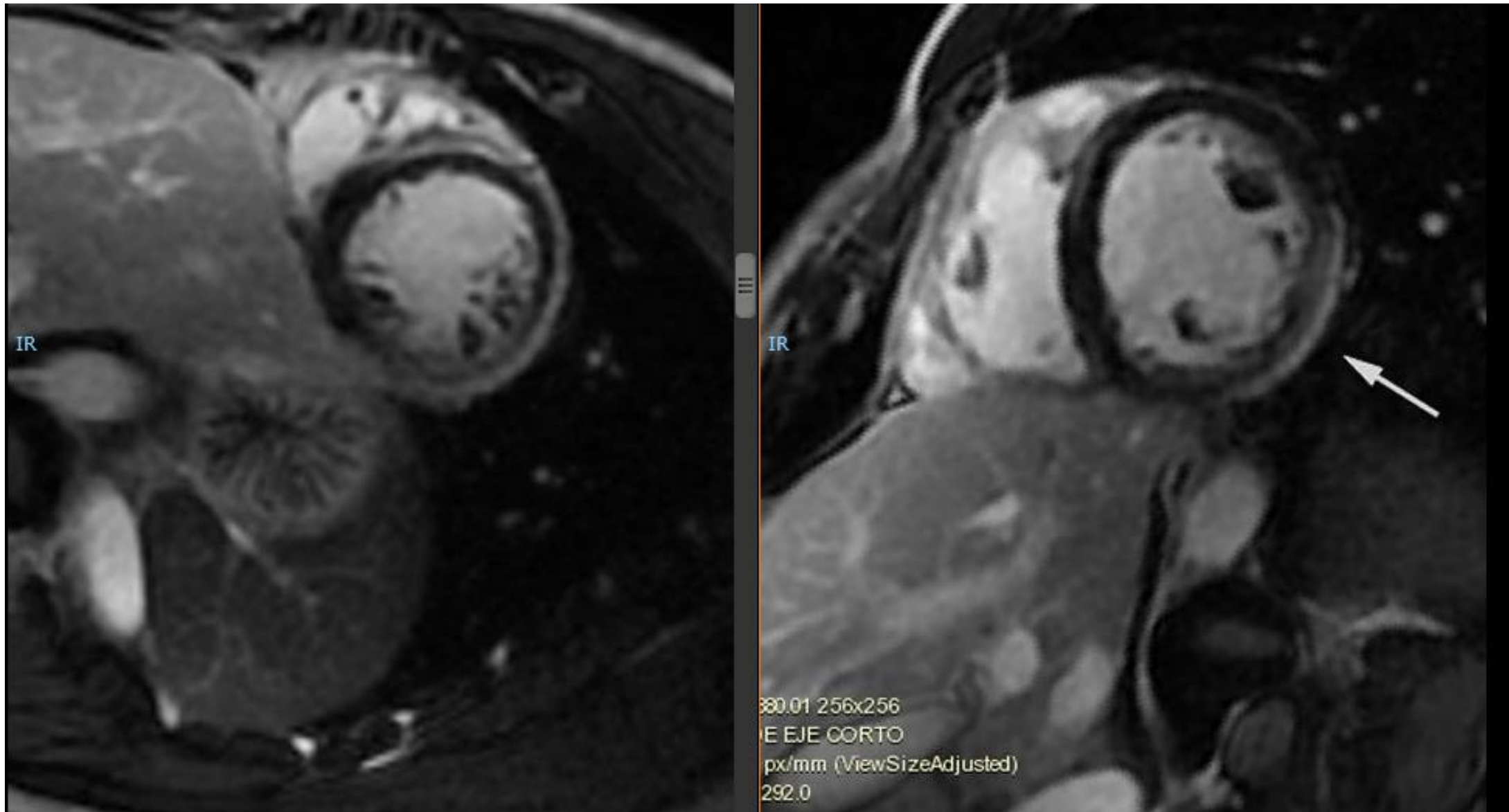
- 2ª opinión tras habersele recomendado abandonar deporte competición*
- Seguimiento al menos de 2 años: alteraciones repolarización en ECG reposo + fibrosis subepicárdica infero lateral apical extensa en RM + FEVI 44%*
- Asintomático, FEVI normal, EEF -, juega 36 partidos con Reveal (sin arritmias)*
- Siguiente temporada, juega 15 partidos, asintomático, sin arritmias*



Inicio siguiente temporada: mareas coincidiendo con rachas de TV (8-35 sg hasta 300 lpm)



Extenso RT subepicárdico ínfero-lateral medio-basal y apical



Myocarditis

*The clinical significance of **persistent LGE** in an asymptomatic athlete with clinically healed myocarditis is **unknown**, however, myocardial scar is a potential source of ventricular tachyarrhythmias.*

At present, it seems reasonable for these athletes to resume training and participate in competitive sport if LV function is preserved and in the absence of frequent or complex repetitive forms of ventricular or supraventricular arrhythmias during maximal exercise and on 24-h ECG monitoring (including session of training/competition).

*Asymptomatic athletes with LGE, however, should remain under **annual clinical surveillance**.*

Class III/Level C



EAPC
European Association
of Preventive Cardiology



Section of Sports
Cardiology



SOCIEDAD
ESPAÑOLA DE
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Grupo de
Cardiología del Deporte

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 quirónsalud

