Ventricular tachyarrhythmias induced by exercise: are they an unsafe clinical pattern?

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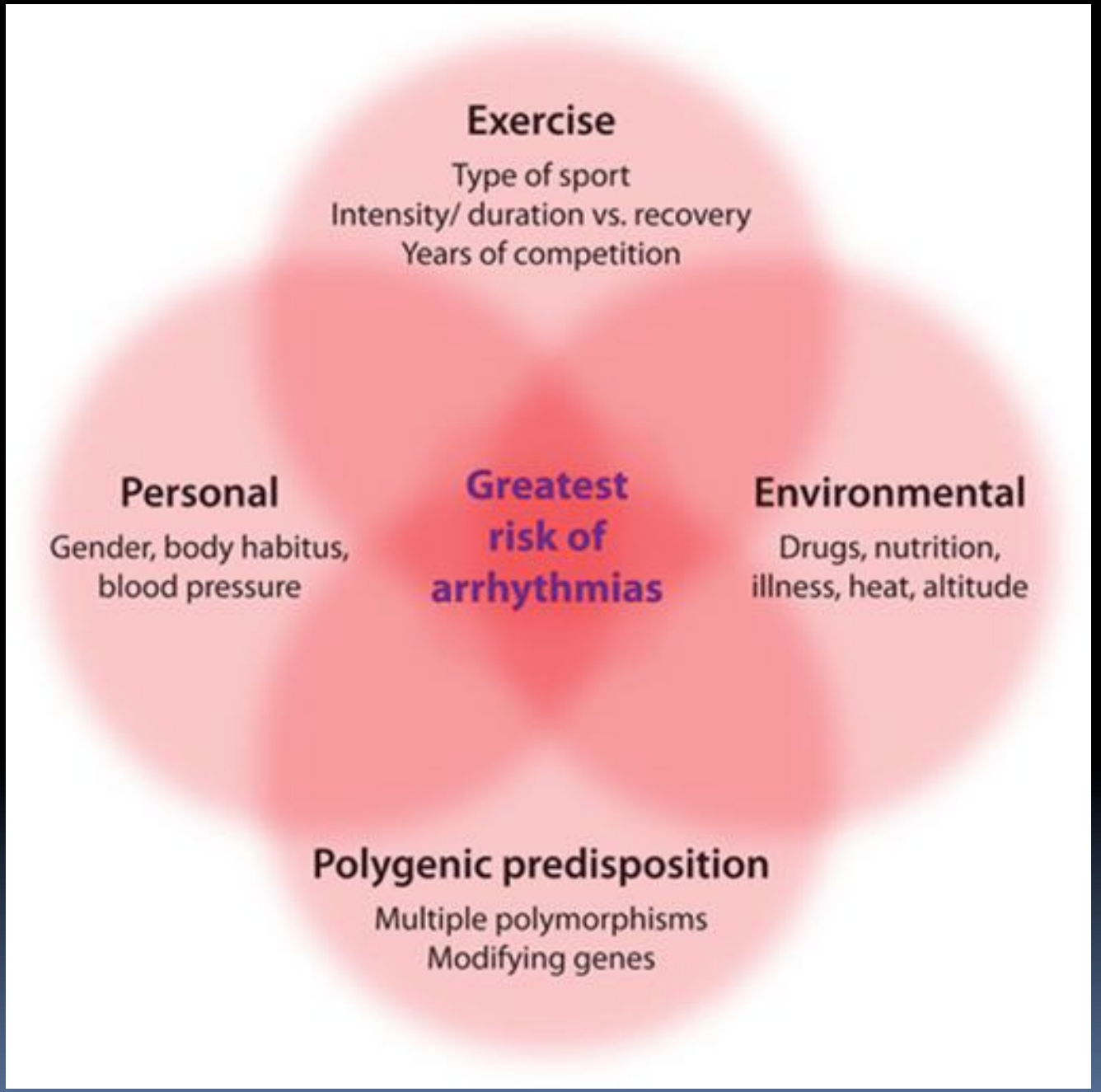
Italian Olympic Commitee – Rome, Italy


Exercise
Type of sport
Intensity/ duration vs. recovery
Years of competition
Personal
Gender, body habitus,
blood pressure
Greatest
risk of
arrhythmias
Environmental
Drugs, nutrition,
illness, heat, altitude
Polygenic predisposition
Multiple polymorphisms
Modifying genes

Prevalence of Ventricular Tachyarrhythmias in Healthy Athletes
(24-h Holter monitoring ECG)

Authors	Ref.	Year	Population (n=)	PVDs (%)	Complex PVDs(%)
H.Paparo	**11**	**1980**	**32**	**6.2**	**0**
Viitasalo	9	1982	35	28	**5.7**
Talan	10	1982	20	70	**20**
Palatini	12	1985	20	70	**25**
SIC SPORT	**13**	**1987**	**407**	**32**	**4.4**

Arrhythmic risk related to exercise

- Increased sympathetic activity
- Facility to induction of paroxysmal supraventricular tachycardias and VT
- Reduction of VF threshold in ischaemic patients
- Increased risk in some cardiomyopathies (HCM and ARVC) and myocarditis
- Facility to induction of arrhythmias in long-QT syndrome and catecolaminergic VT
- Facility to induction of bradycardia-dependent nocturnal tachyarrhytmias (Brugada syndrome).

Journal of the American College of Cardiology
© 2002 by the American College of Cardiology Foundation
Published by Elsevier Science Inc.

2002

Vol. 40, No. 3, 2002
ISSN 0735-1097/02/$22.00
PII S0735-1097(02)01977-0

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

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Rome, Italy, and Minneapolis, Minnesota

OBJECTIVES The aim of this study was to clarify the clinical relevance of ventricular tachyarrhythmias assessed by 24-h ambulatory electrocardiograms (ECG) in a large, unique, and prospectively evaluated athletic population.

BACKGROUND For athletes with ventricular tachyarrhythmias, the risk of sudden cardiac death associated with participation in competitive sports is unresolved.

METHODS We assessed 355 competitive athletes with ventricular arrhythmias (VAs) on a 24-h ambulatory (Holter) ECG that was obtained because of either palpitations, the presence of ≥3 premature ventricular depolarizations (PVDs) on resting 12-lead ECG, or both.

RESULTS Athletes were segregated into three groups: Group A with ≥2,000 PVDs/24 h (n = 71); Group B with ≥100 <2,000 PVDs/24 h (n = 153); and Group C with only <100 PVDs/24 h (n = 131). Cardiac abnormalities were detected in 26 of the 355 study subjects (7%) and were significantly more common in Group A (21/71, 30%) than in Group B (5/153, 3%) or Group C athletes (0/131, 0% p < 0.001). Only the 71 athletes in Group A were excluded from competition. During follow-up (mean, 8 years), 70 of 71 athletes in Group A and each of the 284 athletes in Groups B and C have survived without cardiovascular events. The remaining Group A athlete died suddenly of arrhythmogenic right ventricular cardiomyopathy while participating in a field hockey game against medical advice.

CONCLUSIONS Frequent and complex ventricular tachyarrhythmias are common in trained athletes and are usually unassociated with underlying cardiovascular abnormalities. Such VAs (when unassociated with cardiovascular abnormalities) do not convey adverse clinical significance, appear to be an expression of "athlete's heart syndrome," and probably do not per se justify a disqualification from competitive sports. (J Am Coll Cardiol 2002;40:446–52) © 2002 by the American College of Cardiology Foundation

"… ventricular tachyarrhytmias are common in trained athletes and are usually unassociated with underlying cardiovascular abnormalities … do not convey adverse clinical significance, appear to be an expression of athlete's heart syndrome".

European Heart Journal (2003) 24, 1473–1480

2003

ELSEVIER

EUROPEAN SOCIETY OF CARDIOLOGY

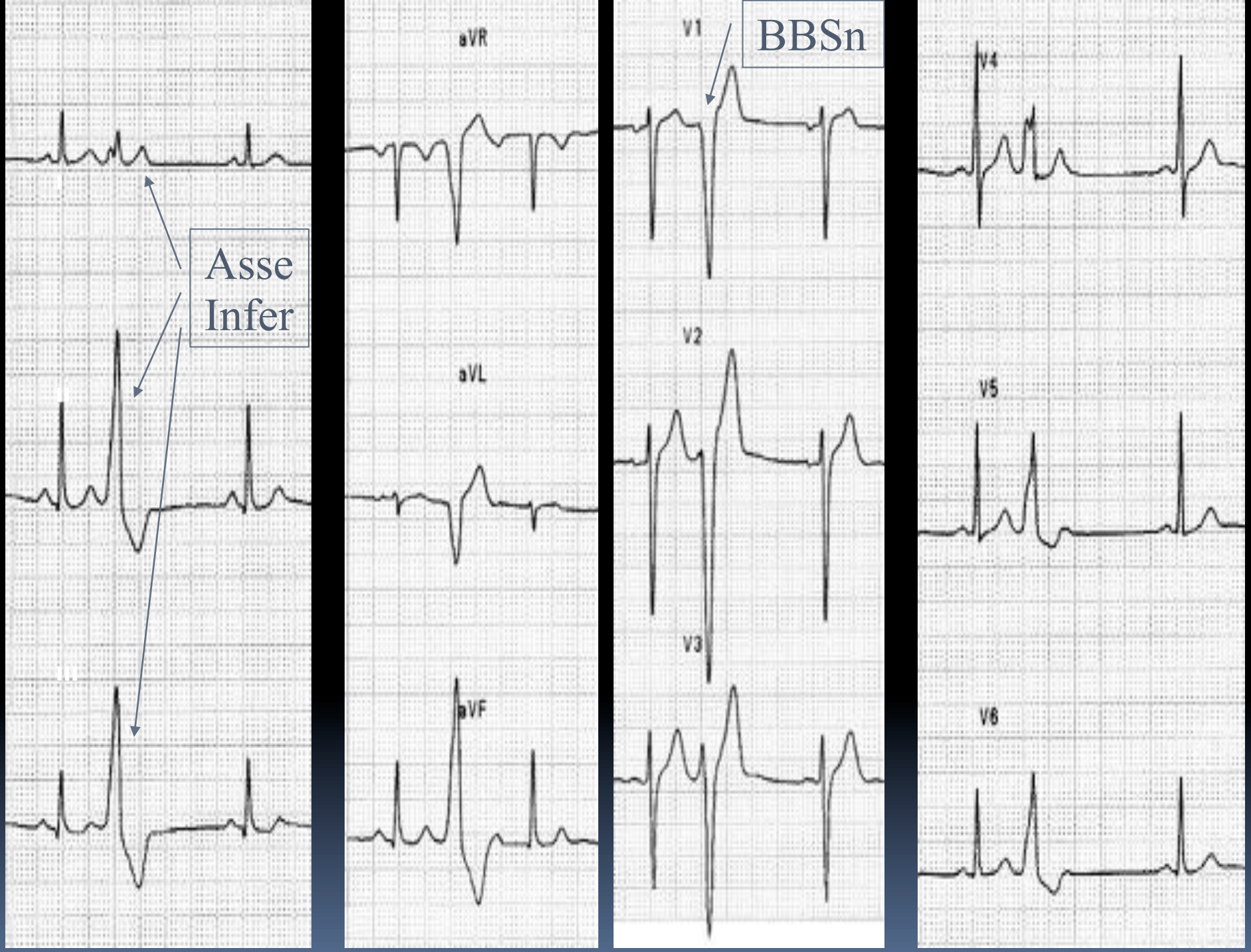
High prevalence of right ventricular involvement in endurance athletes with ventricular arrhythmias

Role of an electrophysiologic study in risk stratification

Hein Heidbüchel[a*], Jan Hoogsteen[b,d], Robert Fagard[a], L. Vanhees[a], Hugo Ector[a], Rik Willems[a], Johan Van Lierde[c,d]

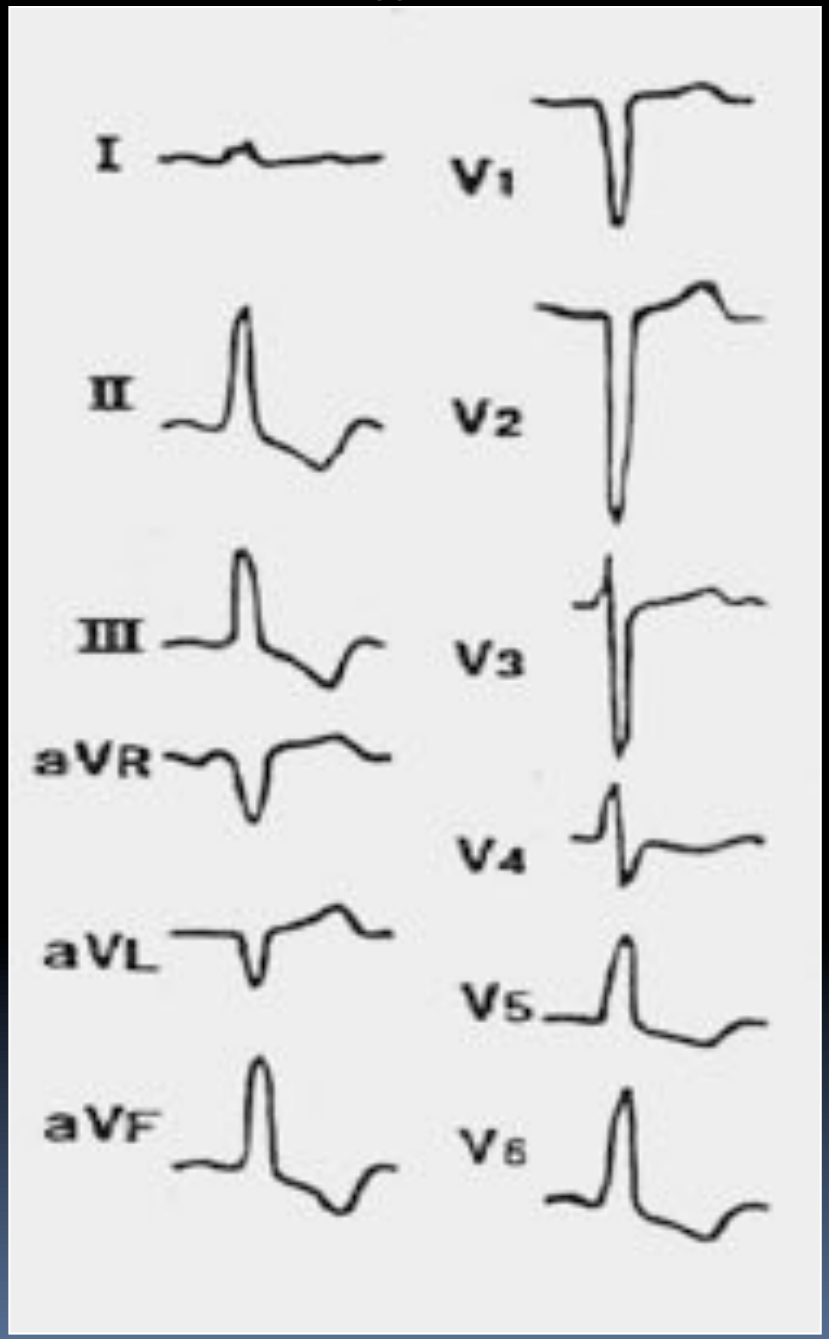
"… suggested a correlation between training and RV tachyarrhythmias, hypothesizing a form of acquired arrhythmogenic right ventricular cardiomyopathy".

ECG PATTERNS OF VENTRICULAR ARRHYTHMIAS

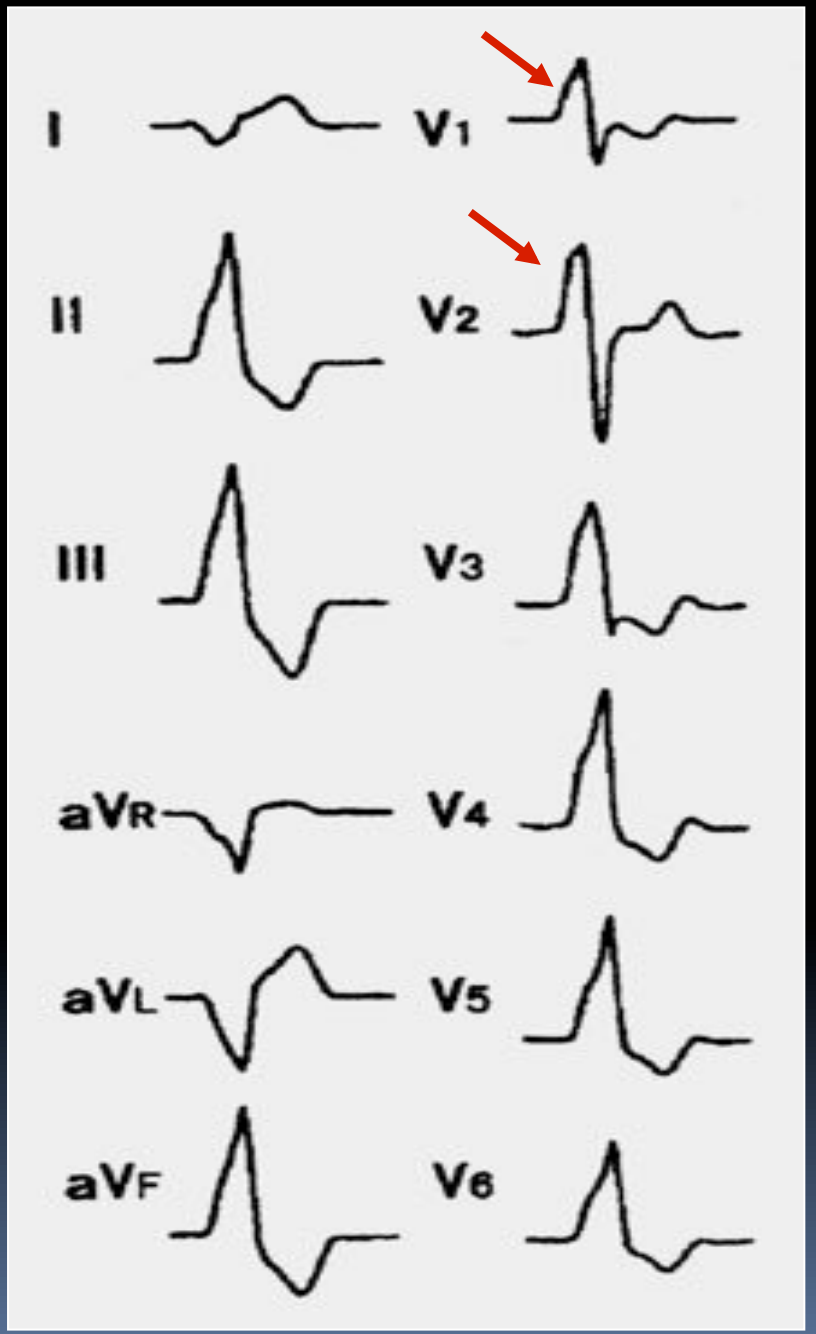


Tipica aritmia ventricolare idiopatica/benigna ad origine dal tratto d'efflusso del ventricolo destro (BBSn + Asse Inferiore)

BPV da efflusso destro



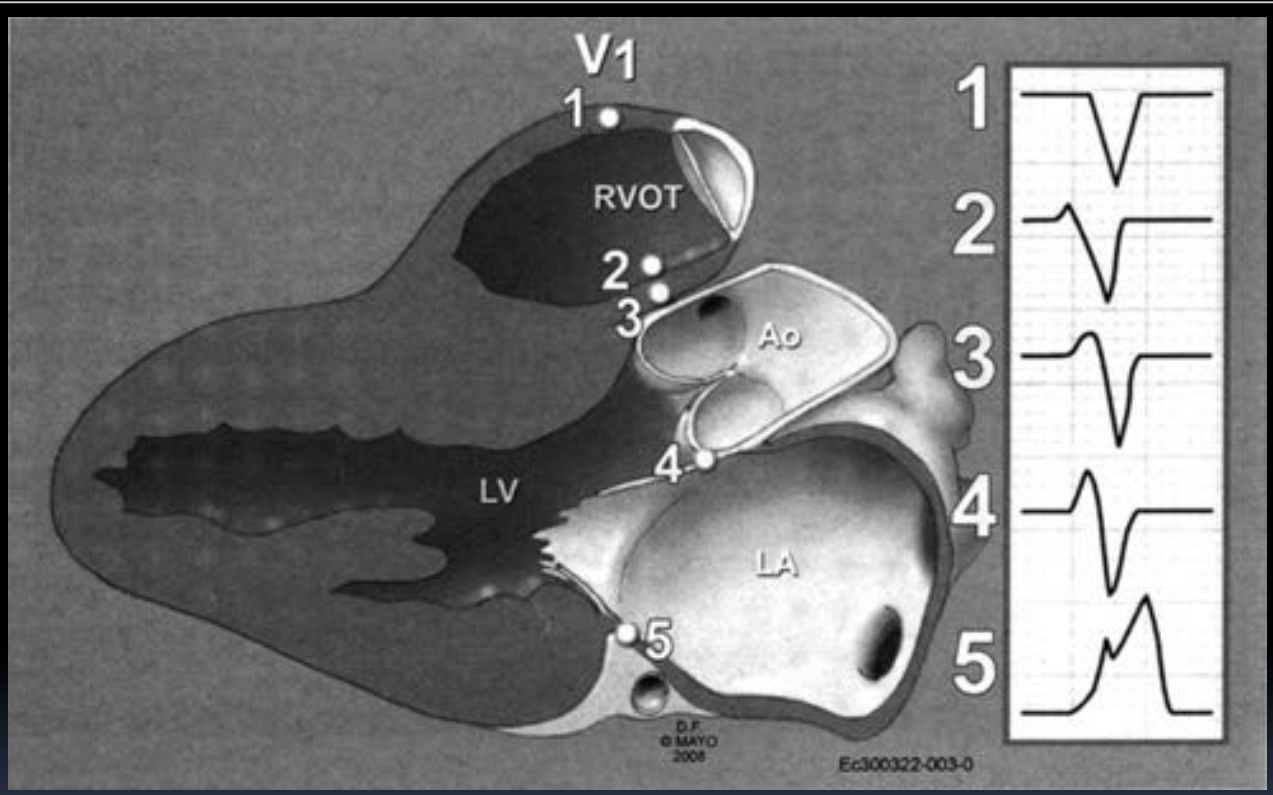
BPV da efflusso sinistro



JACC Journals

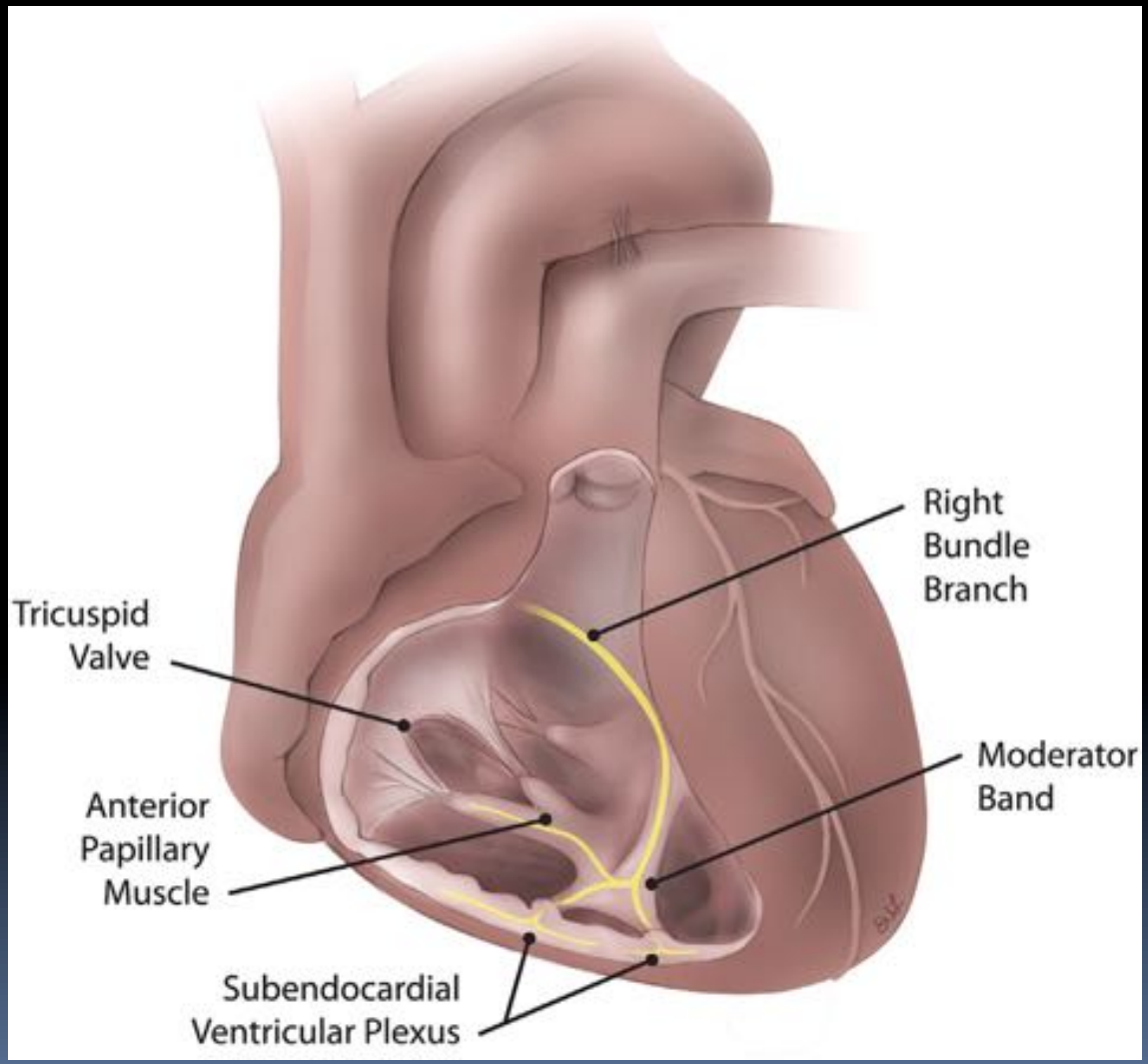
From: Ventricular Arrhythmias in the Absence of Structural Heart Disease

J Am Coll Cardiol. 2012;59(20):1733-1744. doi:10.1016/j.jacc.2012.01.036

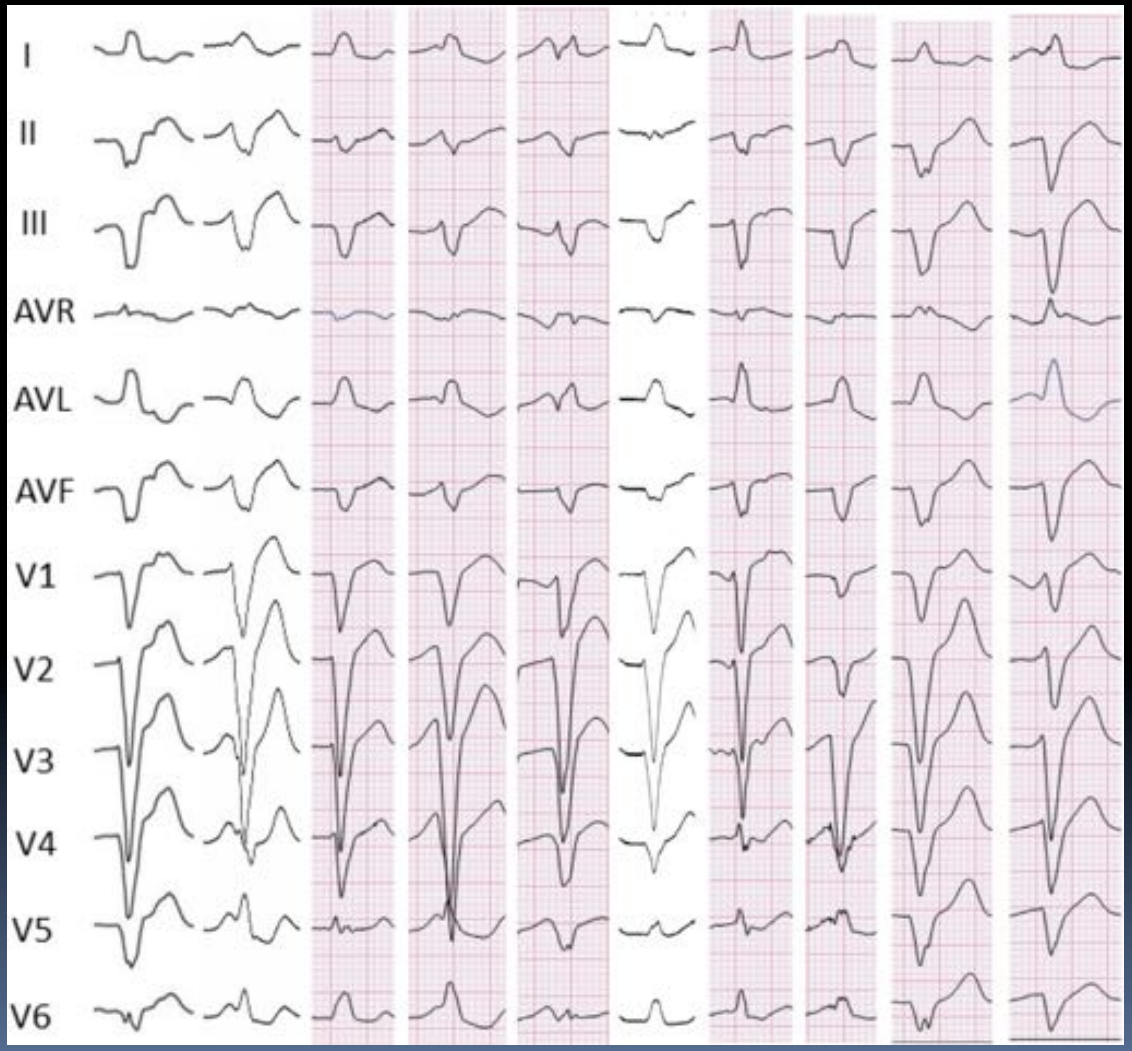


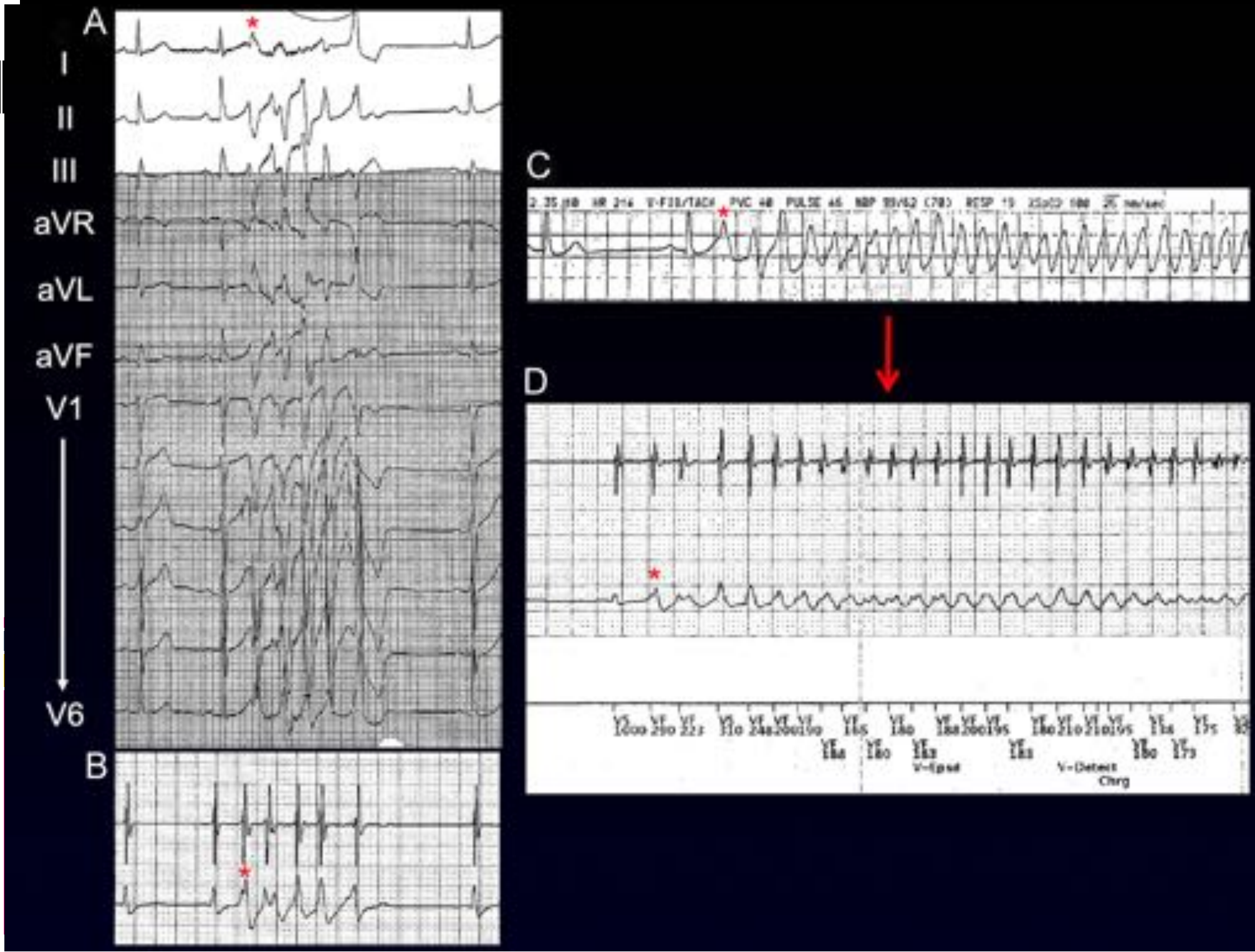
The anatomy of the outflow tract region is such that areas on the right and left sides of the heart can be in close proximity to each other. This can give similar ECG patterns in several leads. However, note that in V1, there is a gradual increase in the amplitude of the r-wave as the site of origin of the ventricular ectopy moves leftward.

Idiopathic ventricular arrhythmias originating from the moderator band:Electrocardiographic characteristics and treatment by catheter ablation



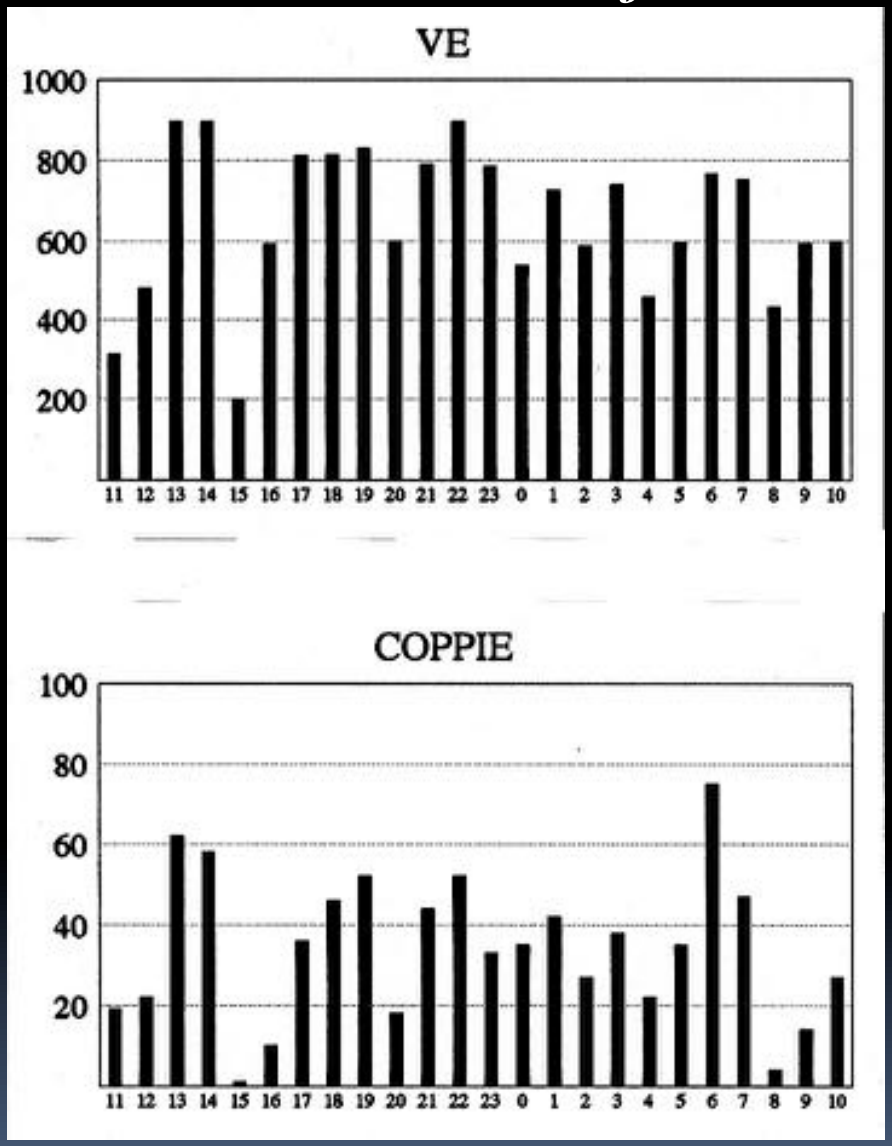
Left superior axis + late precordial transition > V4



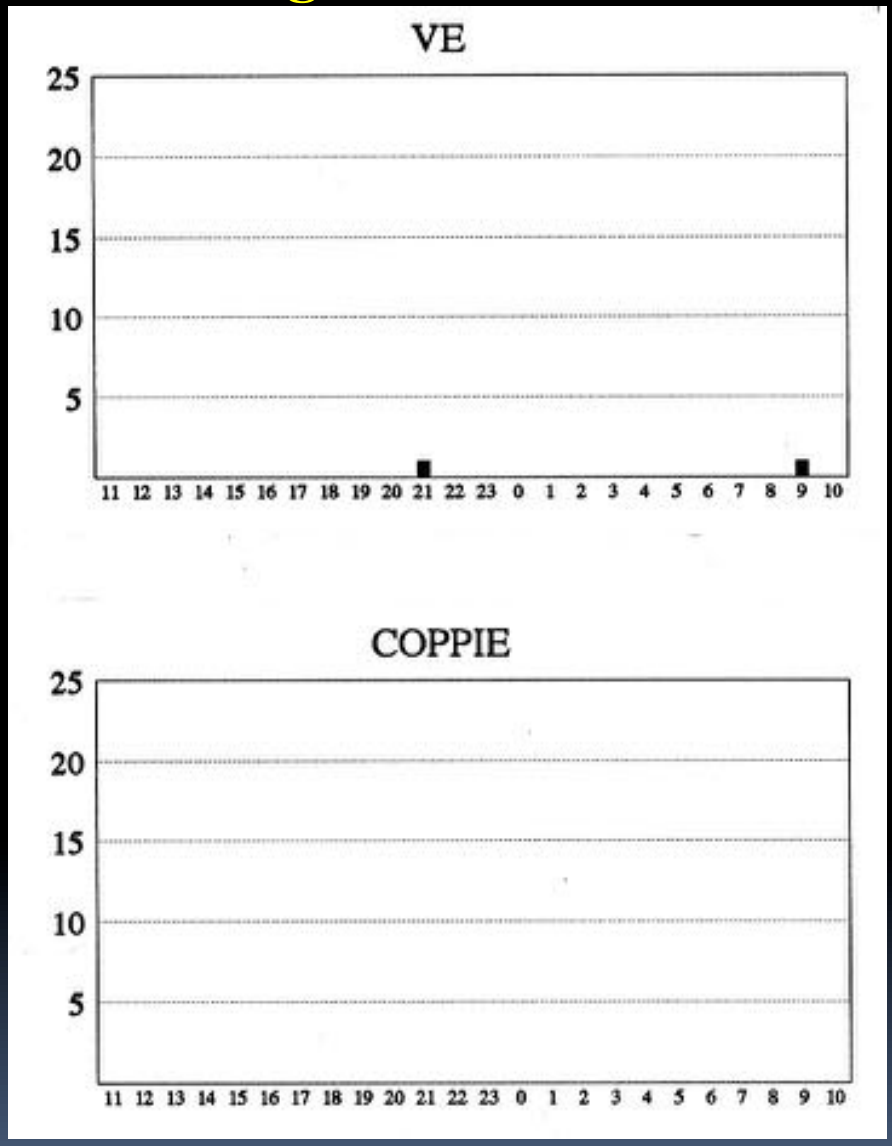

A
I
II
III
aVR
aVL
aVF
V1
V6
B
C
D
V-Epsd
V-Detect
Chrg

VENTRICULAR ARRHYTHMIAS AND DETRAINING

Reduction of VAs in athlete without CV disease after 3-month detraining

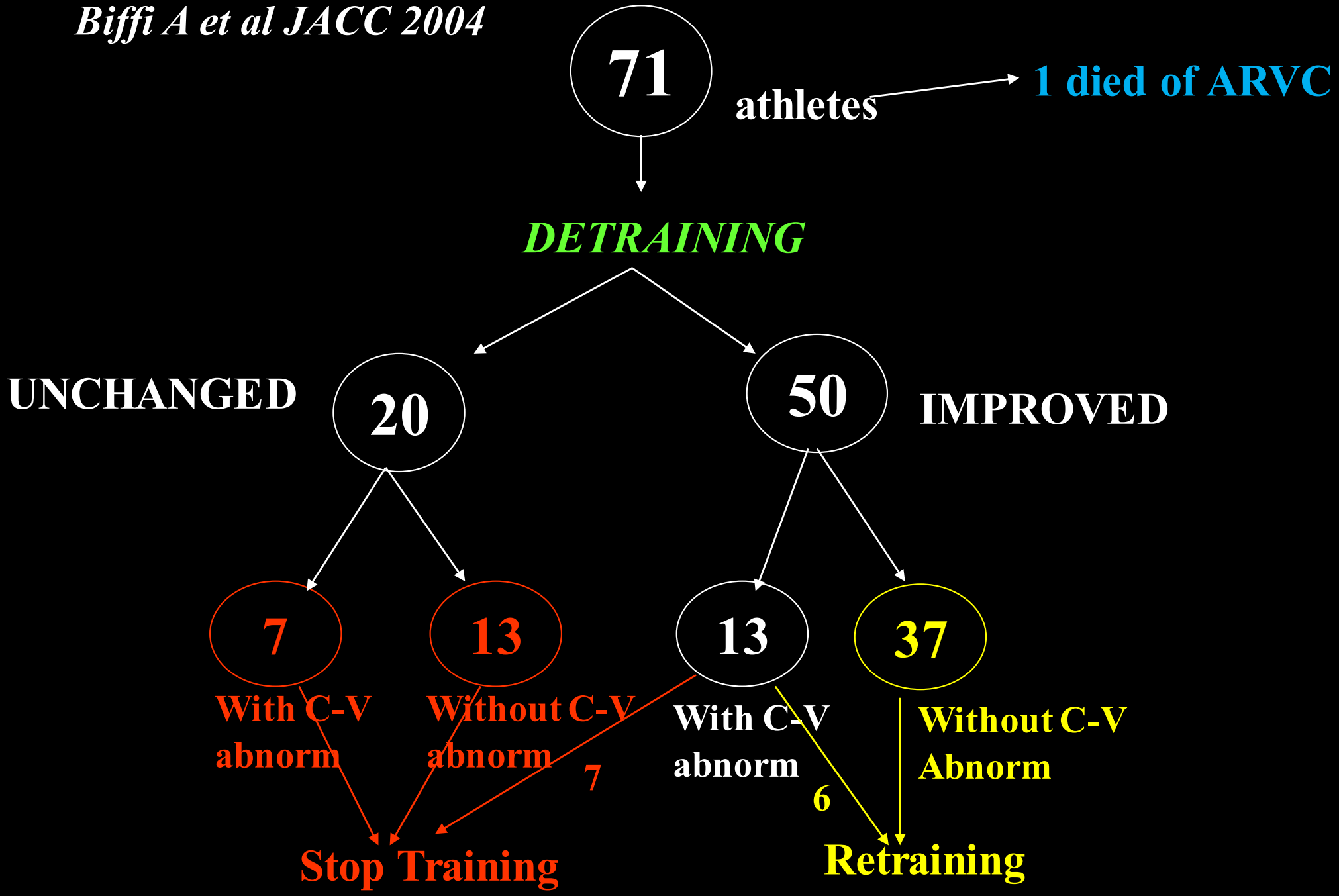


Peak training



After detraining

Biffi A et al JACC 2004



Long-Term Effect of Continuing Sports Activity in Competitive Athletes With Frequent Ventricular Premature Complexes and Apparently Normal Heart

Pietro Delise, MD[a,*], Nadir Sitta, MD[a], Emanuela Lanari, MD[a], Giuseppe Berton, MD[a], Monica Centa, MD[a], Giuseppe Allocca, MD[a], Arianna Cati, MD[a], and Alessandro Biffi, MD[b]

The long-term outcome of athletes with frequent ventricular premature complexes (VPCs) and apparently normal heart has not been fully clarified. To evaluate the clinical and prognostic significance of VPCs and the influence of continuing sports activity during follow-up, we studied 120 healthy athletes (96 men; median age 16 years) in whom frequent VPCs (>100 VPCs/24 hours) were discovered by chance during preparticipation screening. All athletes were followed up for a median of 84 months. During follow-up, 96 underwent serial 24-hour Holter recording and 62 underwent serial echocardiography. The median number of VPCs/24 hours on basal Holter was 3,760. During follow-up, 81 athletes continued sports activity, whereas 39 did not. No athlete died or developed overt heart disease. The median number of VPCs/24 hours decreased in both athletes who continued sports activity and those who did not (from 3,805 to 1,124, p <0.0001 and from 5,787 to 1,298, p <0.0001, respectively). During follow-up, left ventricular ejection fraction slightly decreased to <55% in 9 of 62 athletes who, in respect to the remaining 53, had more VPCs/24 hours both in the basal state (12,000 vs 3,880) and during follow-up (10,702 vs 1,368), and a longer follow-up (95 vs 36 months). In conclusion, (1) frequent VPCs in athletes without heart disease have a long-term benign prognostic significance, (2) sporting activity does not modify this benign outcome, (3) during follow-up, the burden of VPCs decreases whether or not subjects continue sports activity, and (4) in 14.5% of athletes, ejection fraction slightly decreases over time.

 (Am J Cardiol 2013;112:1396–1402)

Frequent ventricular premature complexes (VPCs) may be discovered by chance in otherwise healthy athletes during preparticipation screening.[1–4] As the long-term outcome of these subjects has not been fully clarified, in this study, we evaluated their clinical prognosis and the influence of continuing sports activity on the complexity of arrhythmias during a follow-up of several years.

Methods

We analyzed 205 competitive athletes, <35 years old, consecutively referred to the arrhythmologic center of our division of cardiology from several sports medicine centers in Italy, after the discovery of ventricular premature beats during screening for eligibility for sport. Cases were collected from 1979 to 2008.

In accordance with the Italian screening program, all athletes had undergone medical examination, standard 12-lead electrocardiography (ECG), and submaximal exercise testing. When enrolled in our center, all athletes underwent echocardiography, 24-hour Holter monitoring, and maximal exercise testing. Further instrumental evaluations were decided on a clinical basis.

Forty-five subjects were excluded from the study because they presented ≥1 of the following: family history of juvenile (<40 years) sudden death or hereditary cardiomyopathies, syncope, hypertension, or any kind of heart disease such as right ventricular cardiomyopathy, mitral valve prolapse with significant valvular insufficiency, hypertrophic cardiomyopathy, or dilated cardiomyopathy. Furthermore, subjects were excluded if they had sustained (>30 seconds) ventricular tachycardia (SVT), rapid (shortest RR <300 ms) nonsustained ventricular tachycardia (NSVT), or iterative right or left ventricular outflow tract tachycardia. The criteria used for the diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC), hypertrophic cardiomyopathy, mitral valve prolapse, ventricular outflow tract tachycardia, and so forth are those commonly recommended.[5–9] All these athletes were excluded from sport activity and entrusted to their respective clinical cardiologists.

Of the remaining 160 athletes, 20 were excluded because they had <100 VPCs/24 hours on Holter monitoring. Another 20 were excluded because they had been enrolled <1 year before the date of the last follow-up examination. Thus, 120

[a]Department of Cardiovascular, Neurologic and Kidney Diseases, Division of Cardiology, Hospital of Conegliano, Veneto, Italy and [b]Institute of Sports Medicine and Science, Italian Olympic Committee, Rome, Italy. Manuscript received April 1, 2013; revised manuscript received and accepted June 24, 2013.

See page 1402 for disclosure information.

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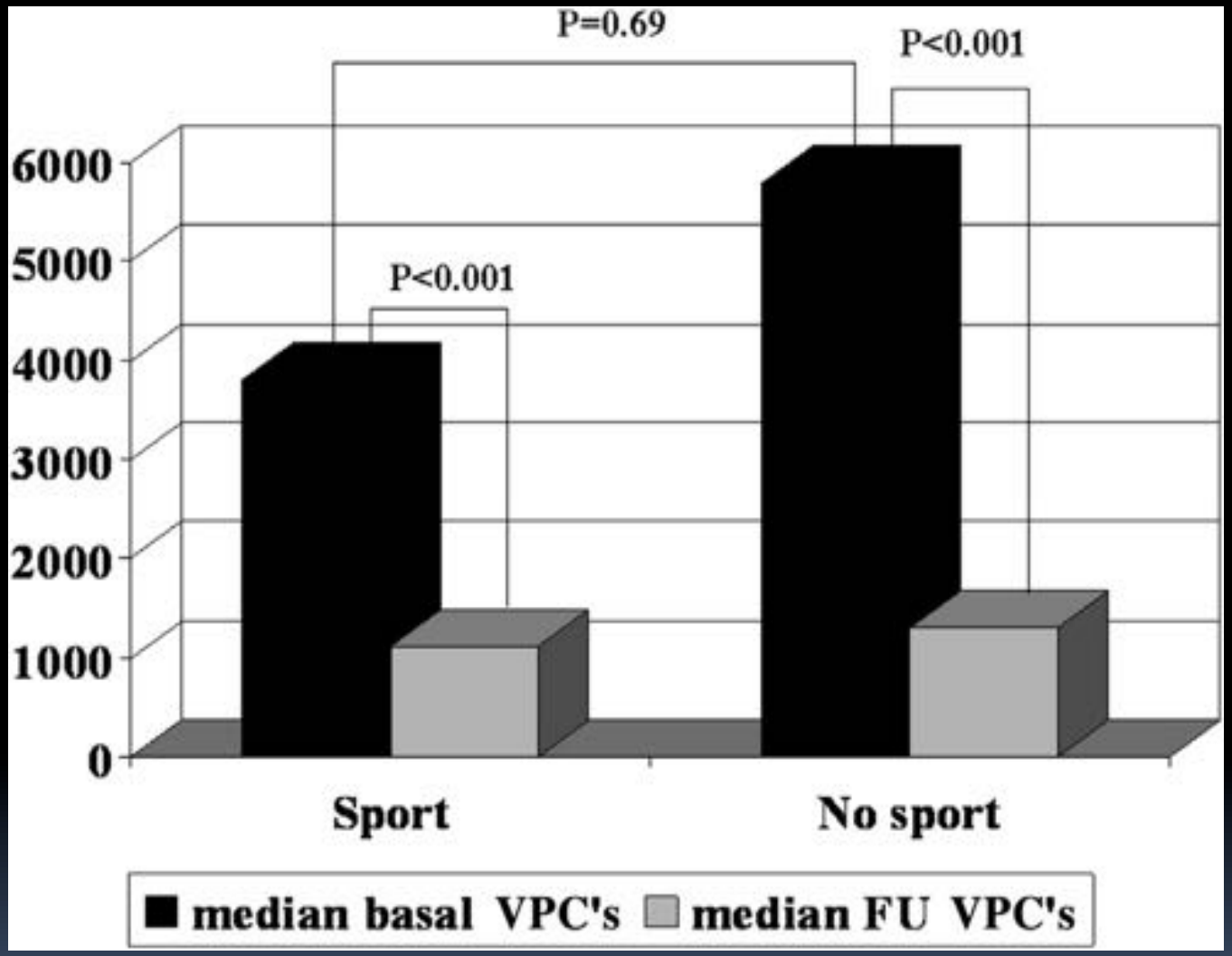
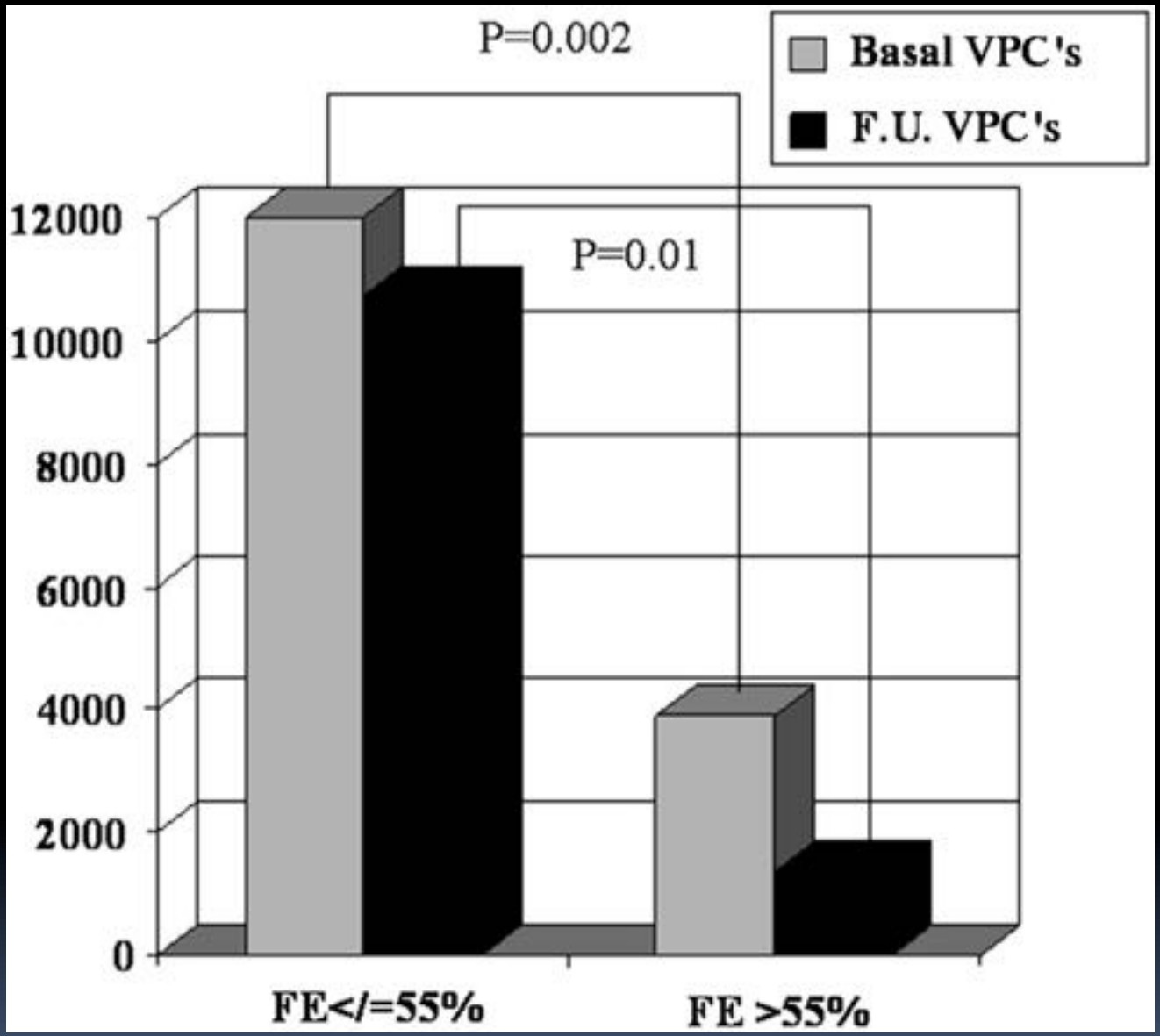
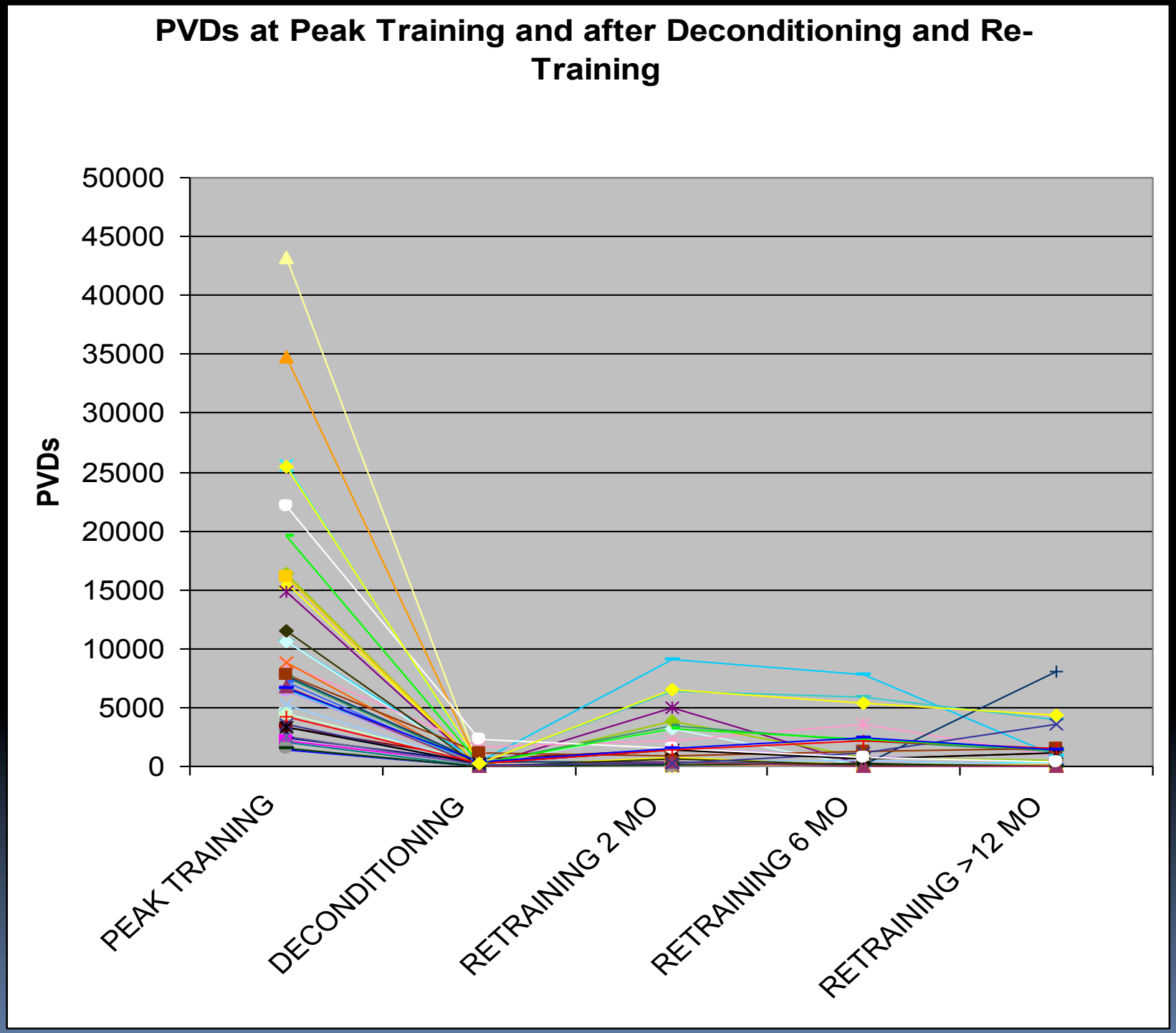
0002-9149/13
http://dx.doi.org/10.1016/j.amjcard.2013.06.032



Delise P et al. Am J Cardiol 2013



Delise P et al. Am J Cardiol 2013

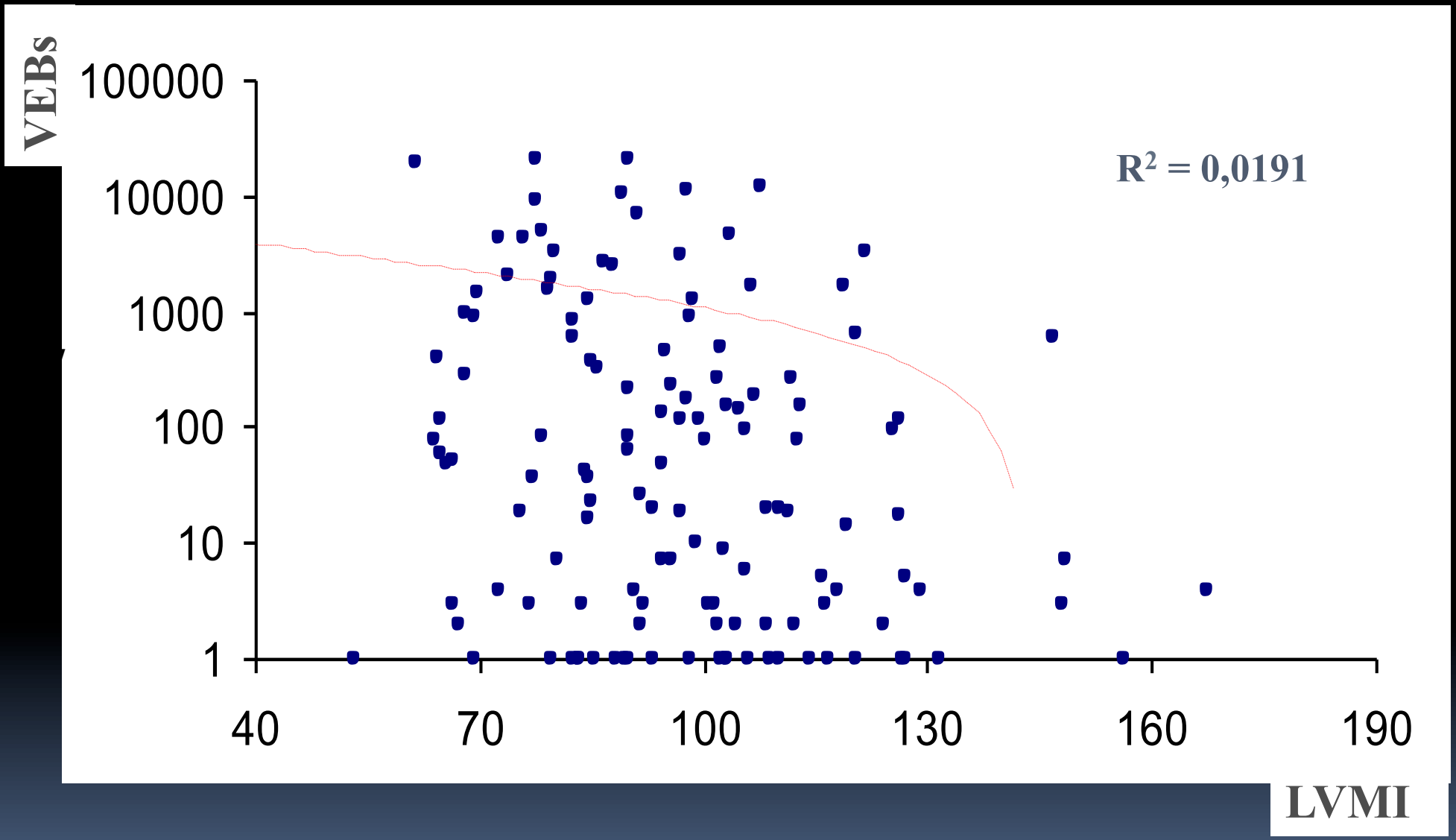


Biffi A et al Am J Cardiol 2011

Can physiologic cardiac remodeling induced by training facilitate the occurrence of VA in a normal heart?

- Our group *(Biffi et al. Am J Cardiol 2008)* shows that left ventricular remodeling is not related to the presence and frequency of VA in elite athletes free of CV abnormalities
- Paradoxically, trained athletes with the smallest extent of LV remodeling demonstrated a propensity to more frequent VA

LV Mass and Ventricular Arrhythmias



Frequency of VAs is not related to physiologic LV Hypertrophy ! *(A.Biffi et al. AJC 2011)*

Physical Training and Ventricular Arrhythmias

- Intensive training has been reported to shift CV autonomic modulation from parasympathetic toward sympathetic dominance

(*Iellamo F et al. Circulation 2002)*

- Such sympathetic predominance could be responsible for increased ventricular irritability in some athletes and explain why ventricular arrhythmias were diminished or abolished with deconditioning

EXERCISE-INDUCED VENTRICULAR TACHYARRHYTHMIAS

European Heart Journal Advance Access published June 2, 2015

European Heart Journal
doi:10.1093/eurheartj/ehv202

CLINICAL RESEARCH
Sports cardiology

Exercise-induced right ventricular dysfunction is associated with ventricular arrhythmias in endurance athletes

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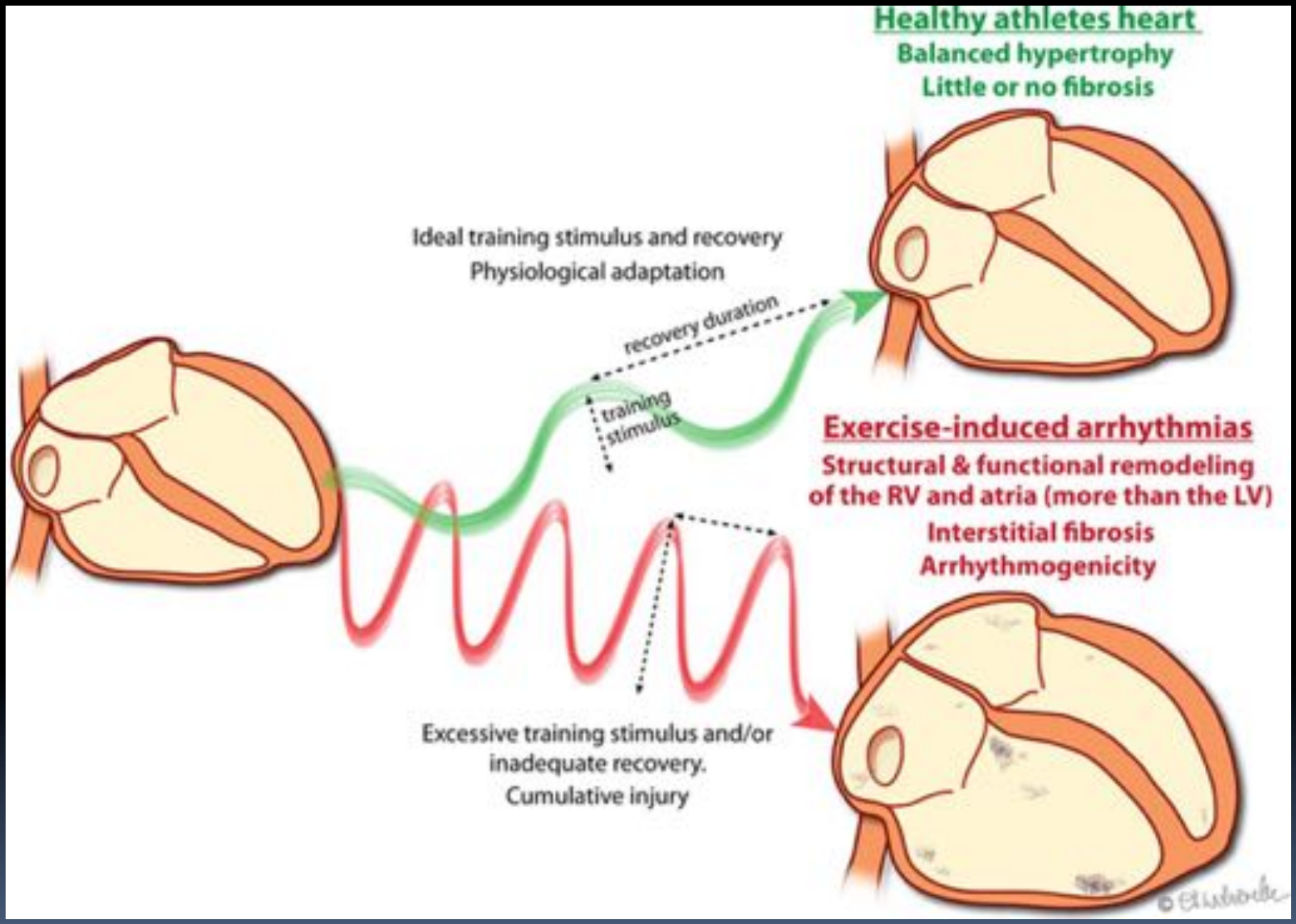
Received 9 October 2014; revised 22 April 2015; accepted 1 May 2015

Aims Intense exercise places disproportionate strain on the right ventricle (RV) which may promote pro-arrhythmic remodelling in some athletes. RV exercise imaging may enable early identification of athletes at risk of arrhythmias.

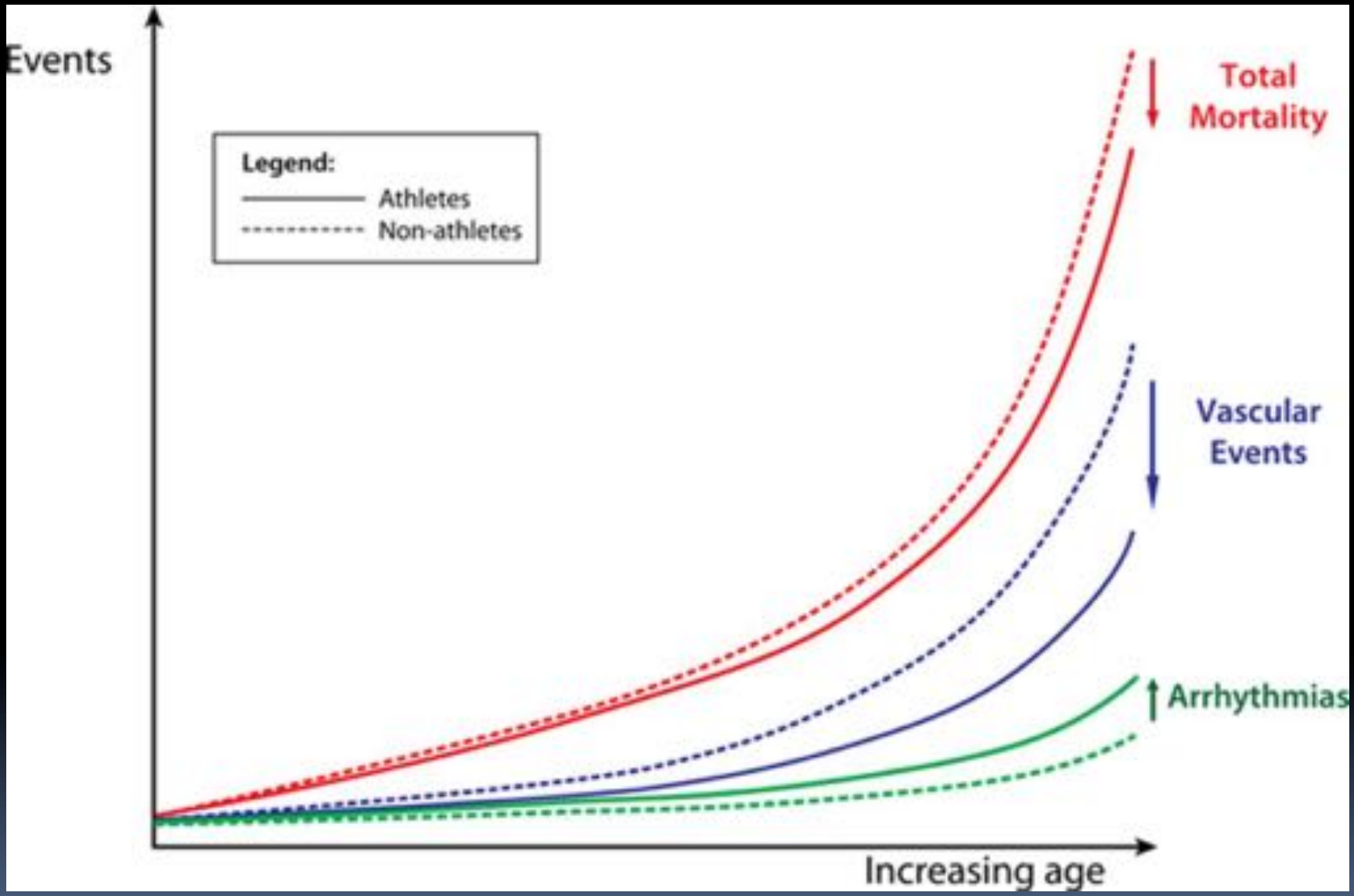
Methods and results Exercise imaging was performed in 17 athletes with RV ventricular arrhythmias (EA-VAs), of which eight (47%) had an implantable cardiac defibrillator (ICD), 10 healthy endurance athletes (EAs), and seven non-athletes (NAs). Echocardiographic measures included the RV end-systolic pressure–area ratio (ESPAR), RV fractional area change (RVFAC), and systolic tricuspid annular velocity (RV S'). Cardiac magnetic resonance (CMR) measures combined with invasive measurements of pulmonary and systemic artery pressures provided left-ventricular (LV) and RV end-systolic pressure–volume ratios (SPVESV), biventricular volumes, and ejection fraction (EF) at rest and during intense exercise. Resting measures of cardiac function were similar in all groups, as was LV function during exercise. In contrast, exercise-induced increases in RVFAC, RV S', and RVESPAR were attenuated in EA-VAs during exercise when compared with EAs and NAs ($P < 0.0001$ for interaction group × workload). During exercise-CMR, decreases in RVESV and augmentation of both RVEF and RV SPVESV were significantly less in EA-VAs relative to EAs and NAs ($P < 0.01$ for the respective interactions). Receiver–operator characteristic curves demonstrated that RV exercise measures could accurately differentiate EA-VAs from subjects without arrhythmias [AUC for ΔRVESPAR = 0.96 (0.89–1.00), $P < 0.0001$].

Conclusion Among athletes with normal cardiac function at rest, exercise testing reveals RV contractile dysfunction among athletes with RV arrhythmias. RV stress testing shows promise as a non-invasive means of risk-stratifying athletes.

Keywords Athletes • Right ventricle • Arrhythmias • Sports cardiology • Cardiac magnetic resonance imaging • Exercise • Arrhythmogenic Right ventricular cardiomyopathy • Echocardiography



André La Gerche, and Hein Heidbuchel Circulation. 2014;130:992-1002



André La Gerche, and Hein Heidbuchel Circulation. 2014;130:992-1002

American Heart Association.

Clinical significance of exercise-induced ventricular tachyarrhythmias in trained athletes without cardiovascular abnormalities

Luisa Verdile, MD,* Barry J. Maron, MD,† Antonio Pelliccia, MD,* Antonio Spataro, MD,* Massimo Santini, MD,* Alessandro Biffi, MD*

*From the *Institute of Sports Medicine and Science, Italian Olympic Committee, Rome, Italy, and †Minneapolis Heart Institute Foundation, Minneapolis, Minnesota.*

BACKGROUND Exercise-induced ventricular tachyarrhythmias raise clinical concern as a marker of increased risk in the presence of underlying cardiovascular disease.

OBJECTIVE The aim of this study was to clarify the clinical significance of exercise-induced ventricular tachyarrhythmias in competitive athletes without evident cardiac abnormalities.

METHODS Exercise electrocardiographic testing was performed in 5011 consecutive athletes without heart disease and analyzed for the occurrence of ventricular arrhythmias.

RESULTS Of the 5011 athletes, 367 (7.3%) showed ≥1 premature ventricular beat (PVB), including 331 (6.6%) with ≤10 PVBs and 36 (0.7%) with >10 PVBs and/or ≥1 ventricular couplets, and/or ≥1 bursts of nonsustained ventricular tachycardia. The 331 athletes with ≤10 PVBs had no restriction from competitive sports, and repeated exercise testing over 3–12 months showed spontaneous reduction of arrhythmia (from 5.2 ± 4 to 4 ± 6 PVBs; $P = .002$), including 83 of 331 (23%) with disappearance of PVBs. The remaining 36 athletes were disqualified from sports because of frequent and/or complex arrhythmias; 23 showed reduction of arrhythmia at 3–12 months (from 46 ± 42 to 28 ± 11 PVBs, from 8 ± 10 to 3 ± 3 couplets, and from 3.6 ± 6 to 1 ± 1 nonsustained ventricular tachycardia; $P = .05$) and were readmitted to competition. The other 13 athletes with persistent arrhythmias were considered for radiofrequency ablation, of whom 8 were successfully treated with abolition of arrhythmias and permitted to return to competitive sports. No events or cardiovascular disease occurred in the 367 athletes over a follow-up period of 7.4 ± 5 years.

CONCLUSION Exercise-induced ventricular tachyarrhythmias were present in a sizable minority of highly trained athletes without heart disease. These arrhythmias proved to be benign and not associated with adverse events or later development of cardiovascular disease.

KEYWORDS Ventricular arrhythmias; Athletes; Sudden death; Sports; Exercise; Exercise stress testing

ABBREVIATIONS **ARVC** = arrhythmogenic right ventricular cardiomyopathy; **ECG** = electrocardiogram/electrocardiographic; **NSVT** = nonsustained ventricular tachycardia; **PVB** = premature ventricular beat; **RVOT** = right ventricular outflow tract

(Heart Rhythm 2015;12:78–85)

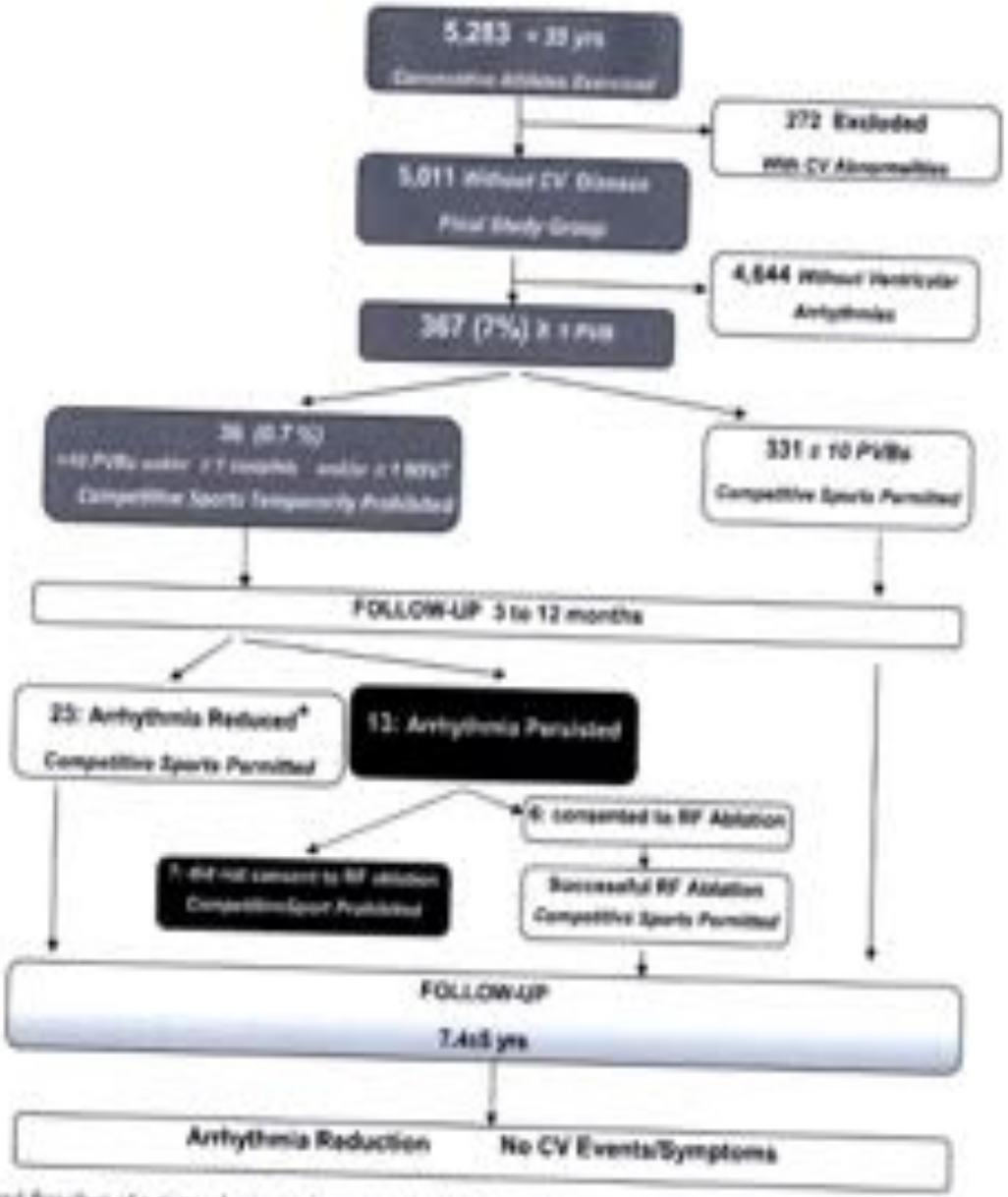


Figure 1 Protocol and flowchart of patient selection and outcome for 5011 competitive athletes studied with exercise testing. Asterisk indicates statistically significant reduction of PVBs and couplets and disappearance of NSVT between the initial evaluation and exercise testing after 3–12 months. CV = cardiovascular; NSVT = nonsustained ventricular tachycardia; PVB = premature ventricular beat; RF = radiofrequency.

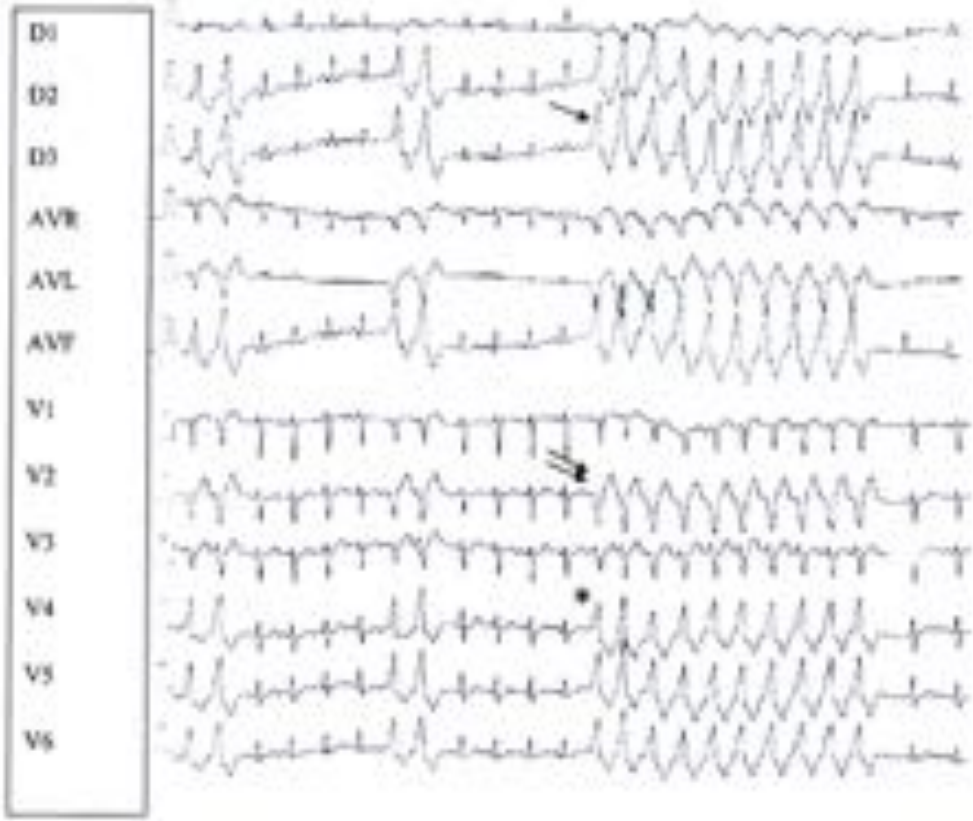
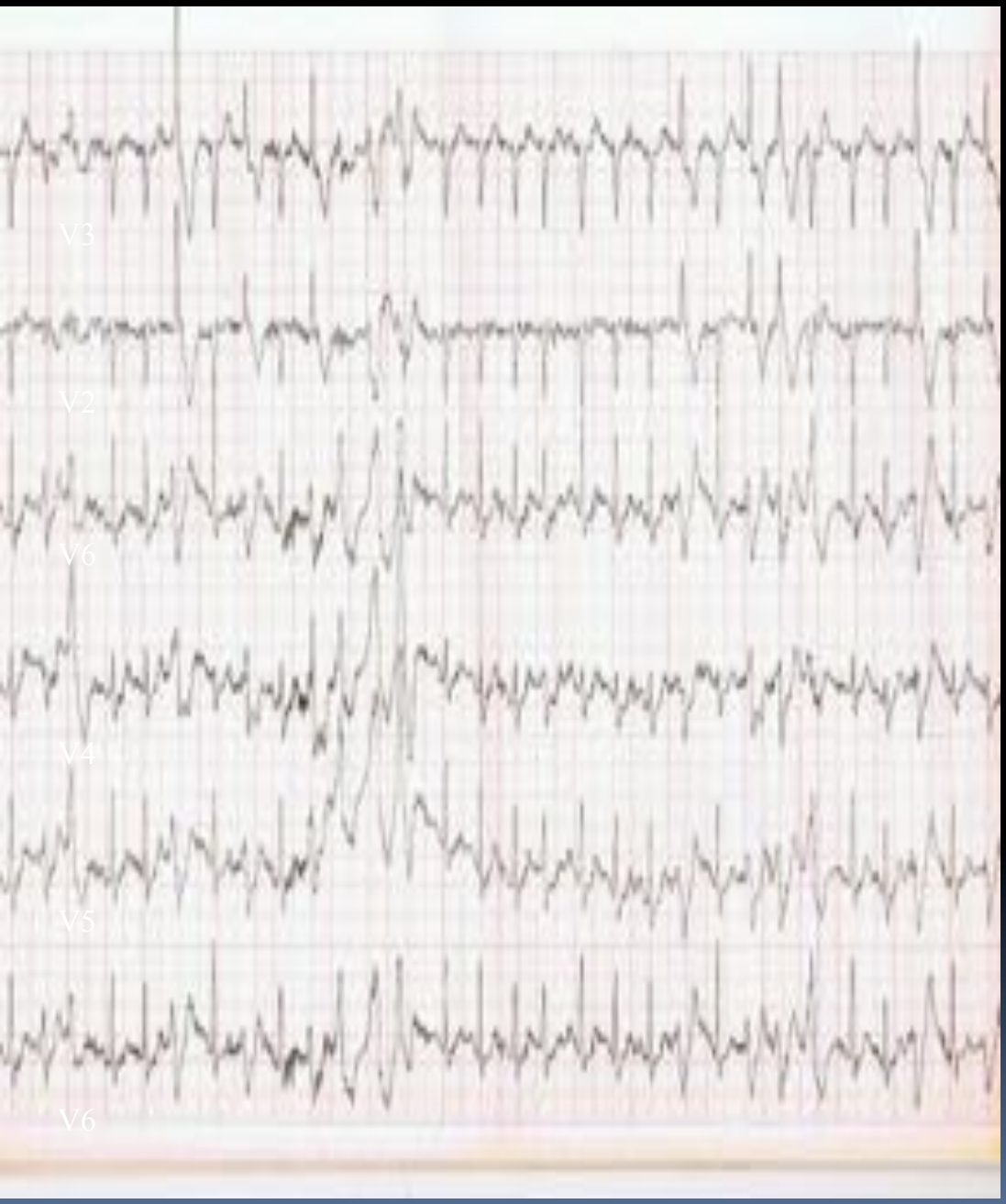
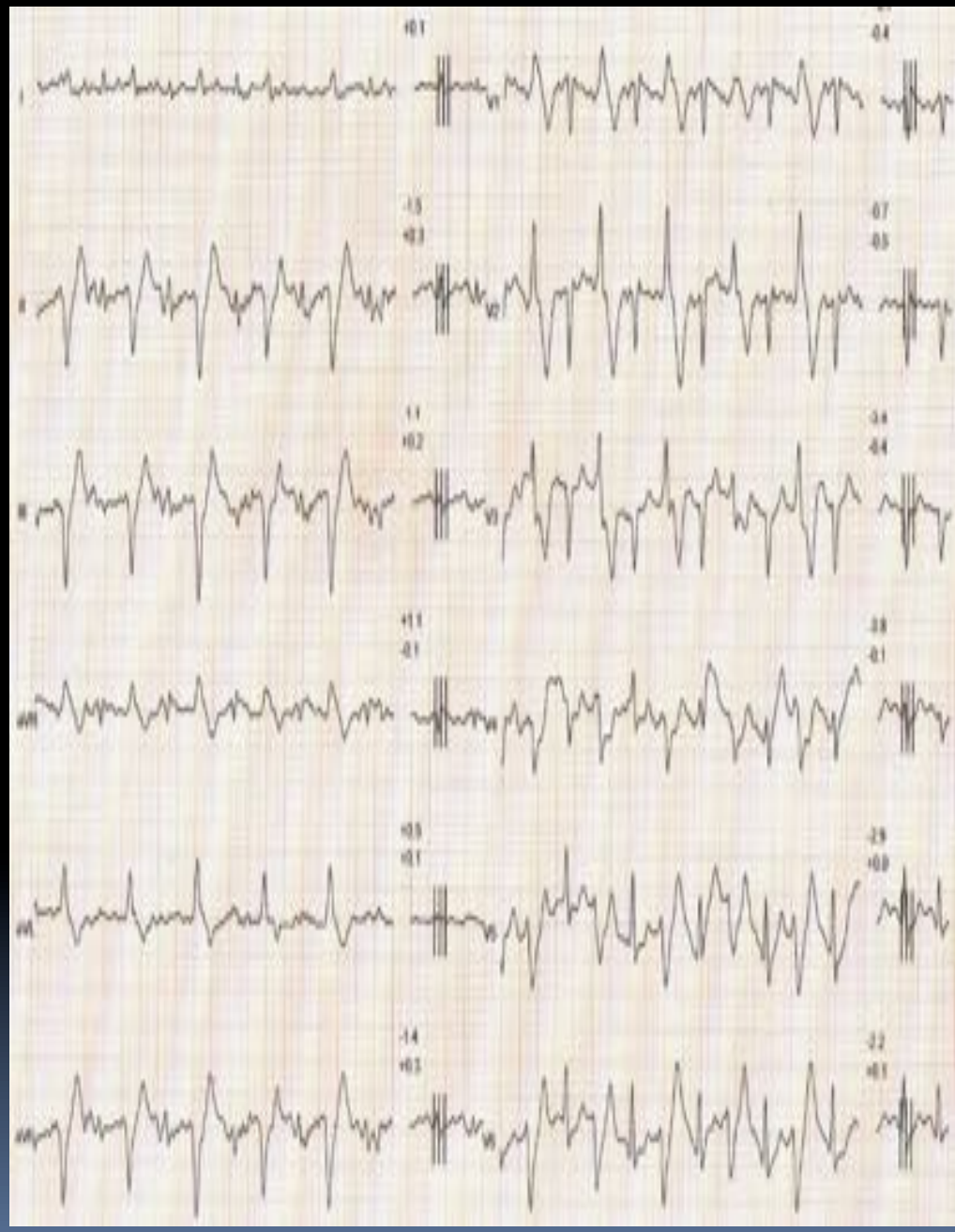


Figure 2 Exercise-induced right ventricular outflow tract ventricular tachycardia in a 16-year-old female volleyball player. The figure shows the 12-lead electrocardiographic tracing during exercise stress testing (workload of 150 W; sinus heart rate 130 beats/min). From the left, 2 ventricular couplets and a nonsustained episode of right ventricular outflow tract ventricular tachycardia (arrow) are induced by exercise. The episode of ventricular tachycardia lasts 10 beats, with a minimum R-R' interval of >400 ms, which is slightly symptomatic for palpitations. The premature ventricular beat morphology is like left bundle branch block in precordial leads (double arrow), with inferior axis in peripheral leads (one arrow) and with R/S transition in lead V_4 (asterisk). At 15-year follow-up, the athlete showed complete reversibility of the arrhythmia during exercise testing and the absence of cardiovascular events with continuation in competitive sports.

1994

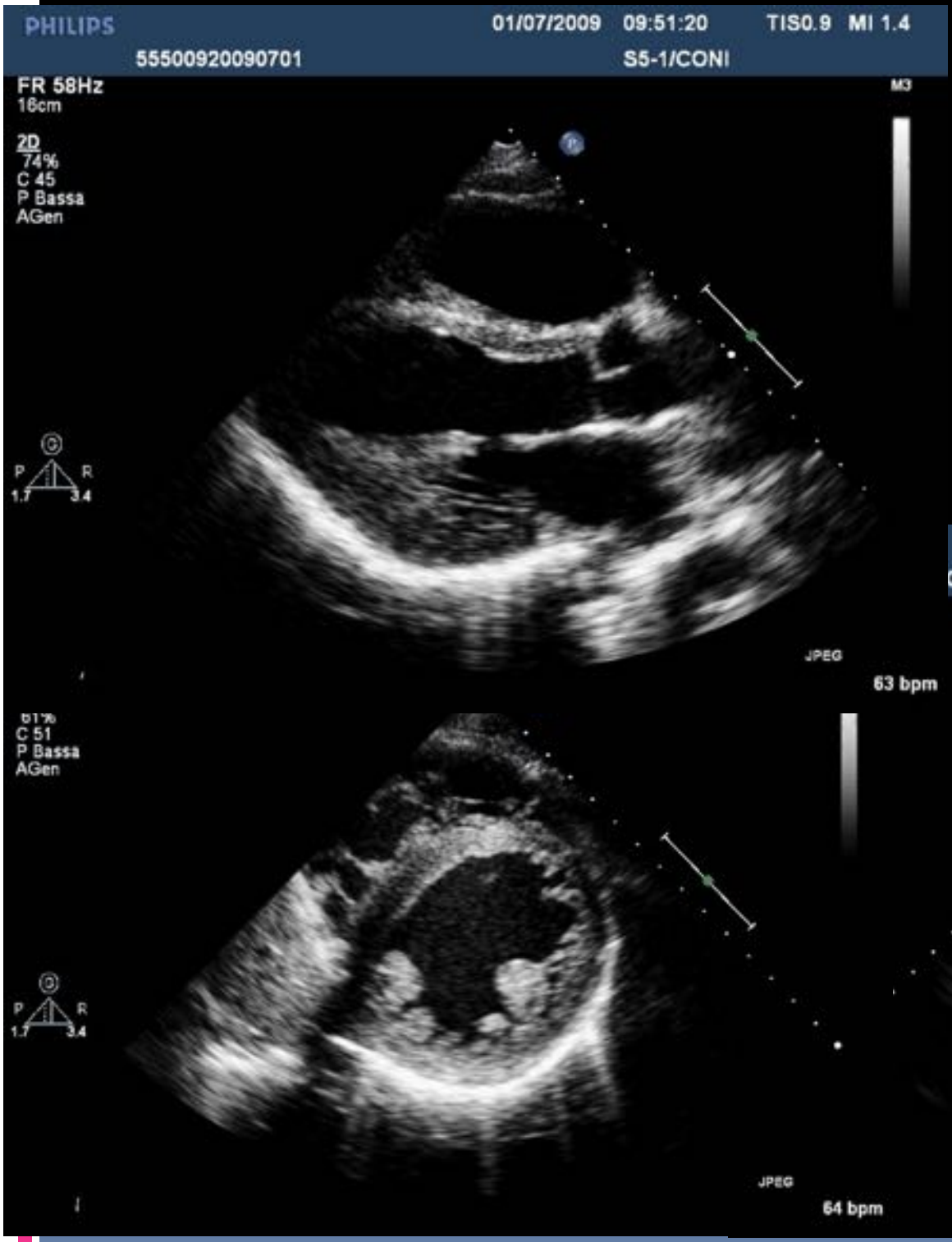


2010



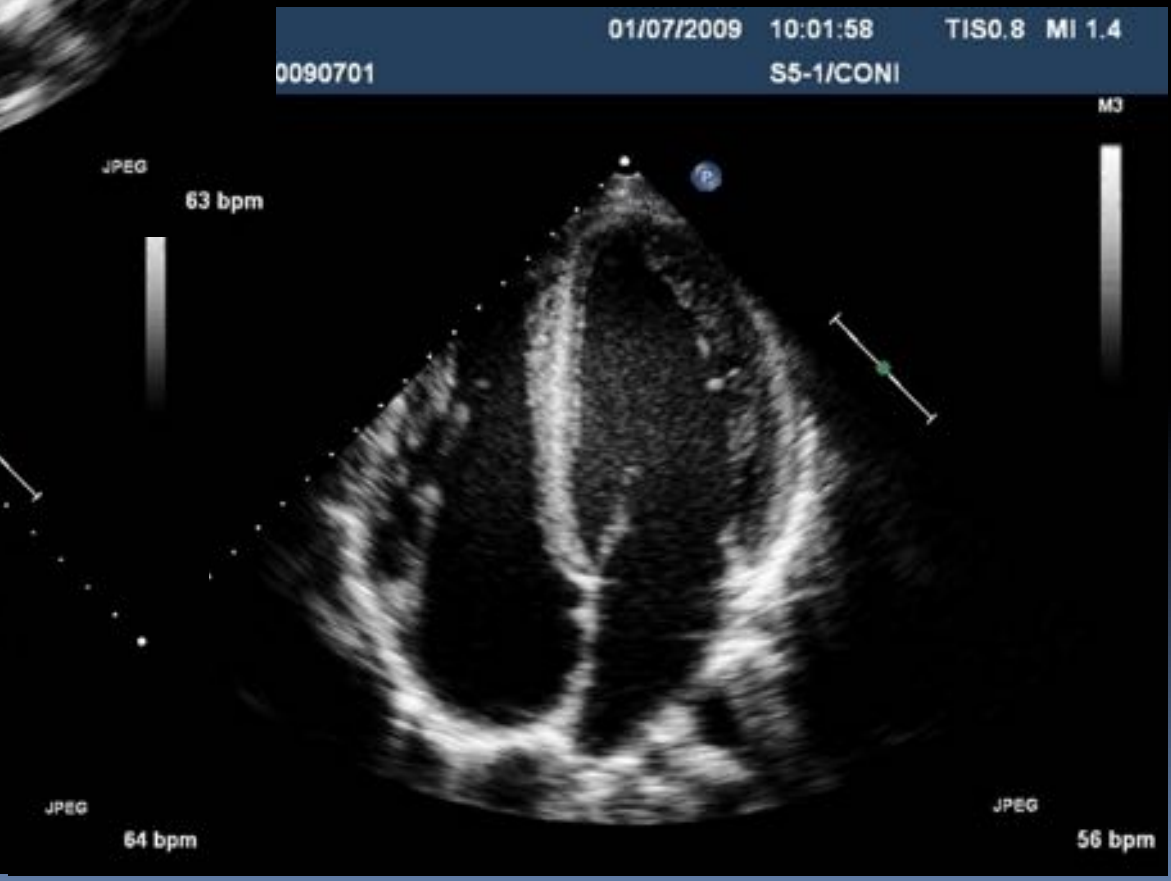
20-year, open-water male swimmer

- Undergoing PPS before the 2009 World Championships
- Height 187 cm; weight 76 Kg
- Family history negative for CMPs or SCD;
- Asymptomatic; no previous relevant events;
- PE unremarkable. BP 130/80 mmHg

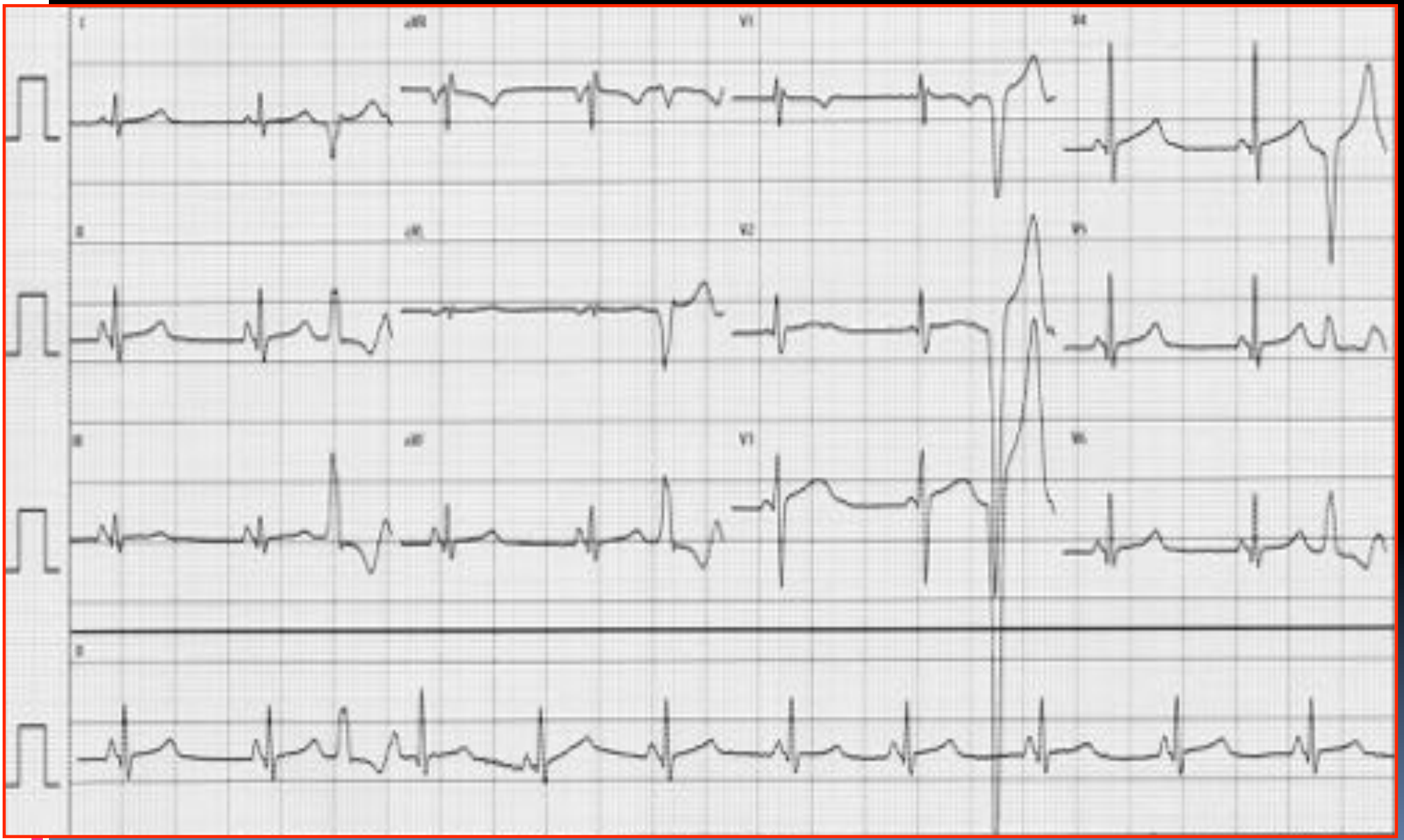


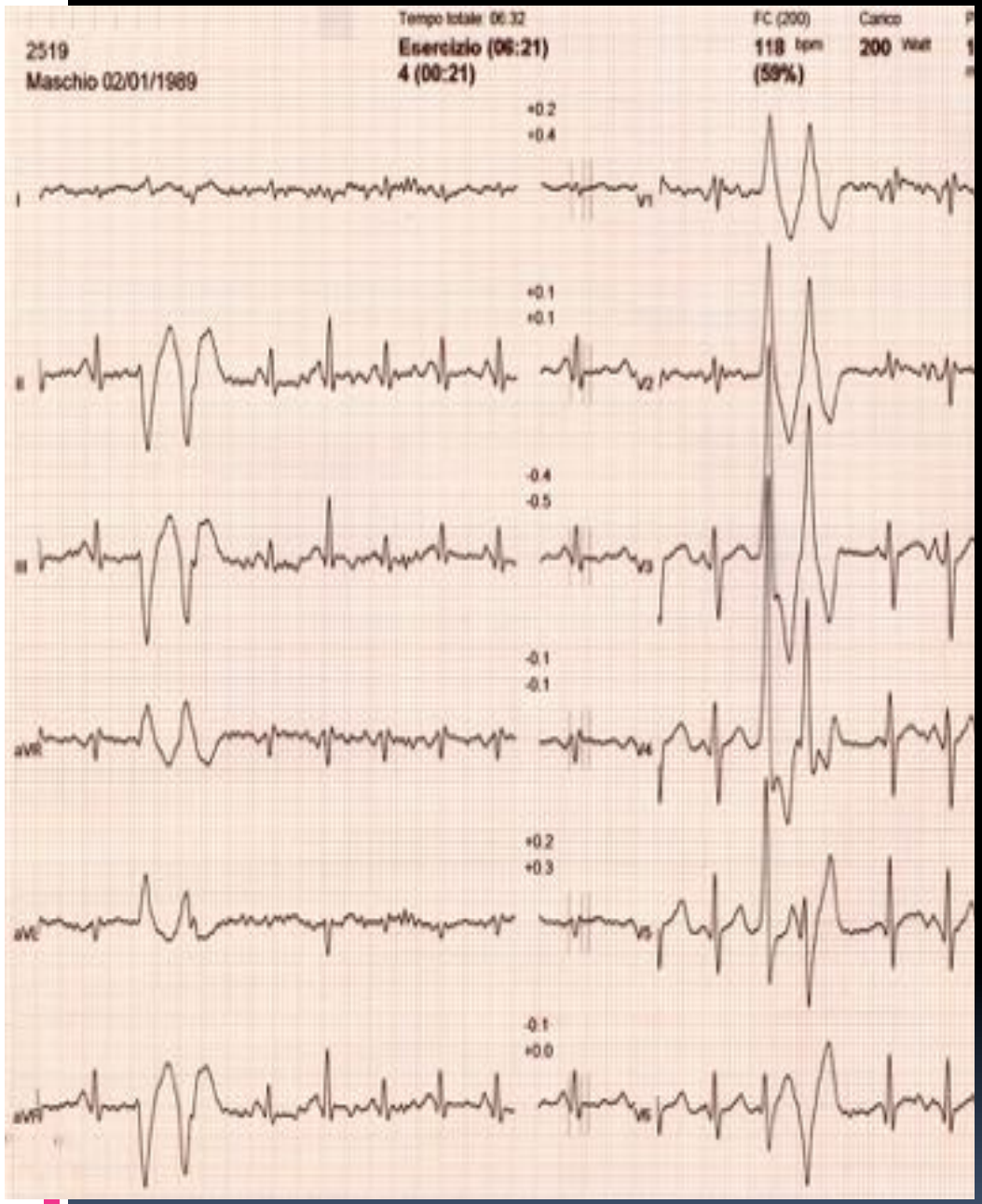
2D echo on July 2009,

at peak training



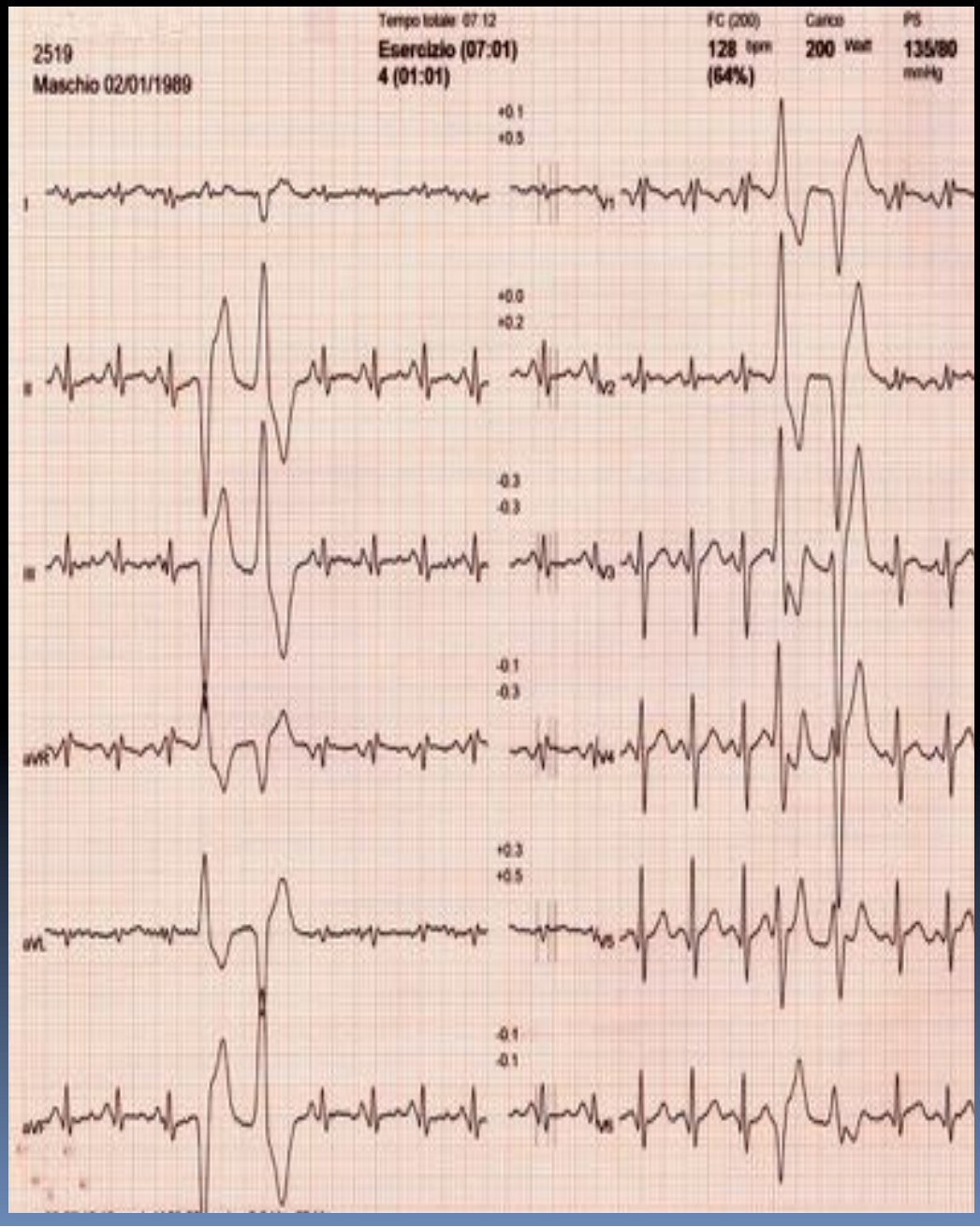
61 bpm, PQ 102 msec, QRS 98 msec QTc 427 msec





Max 300 watt

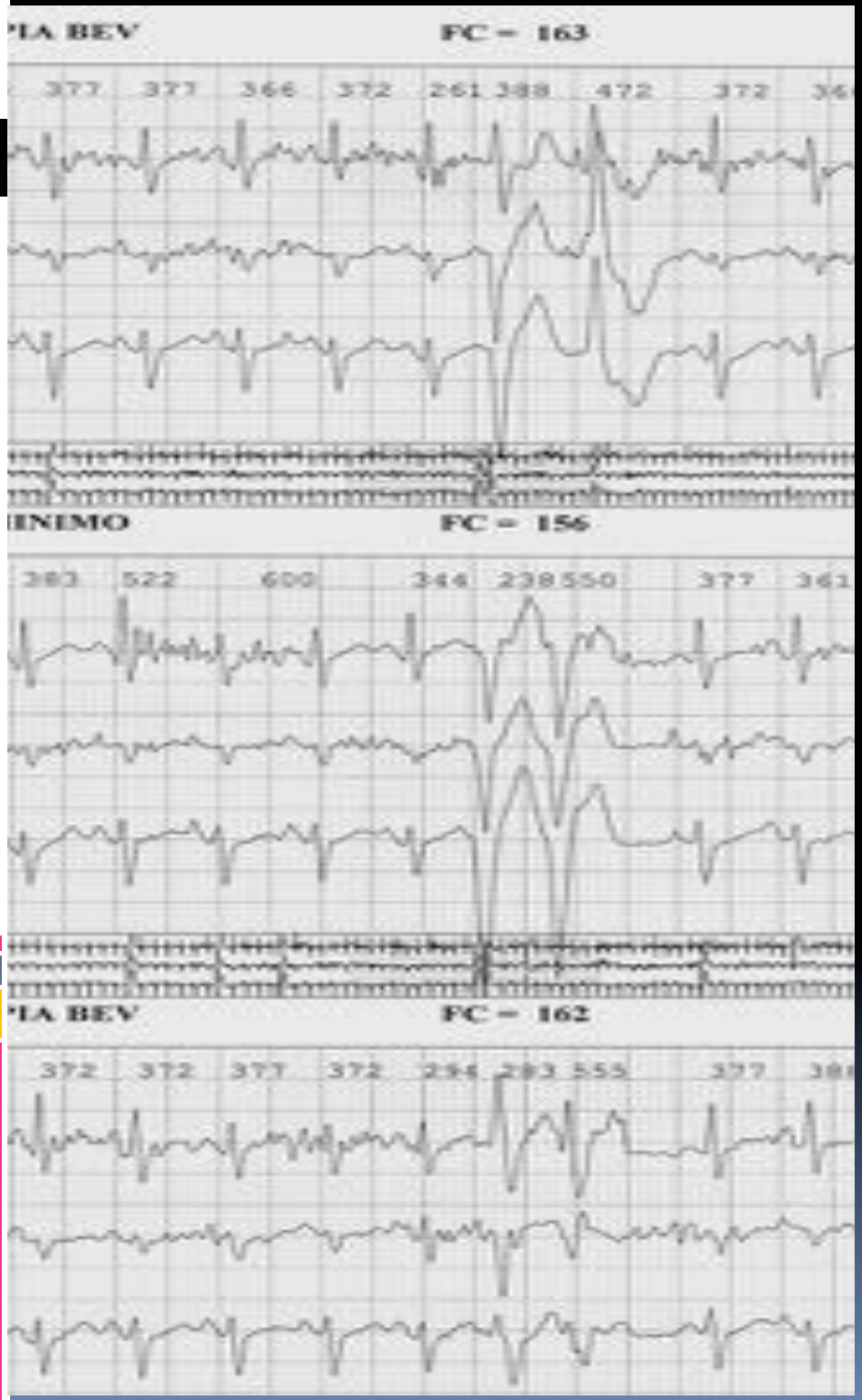
Hr:172 bpm; BP: 80/75 mmHg

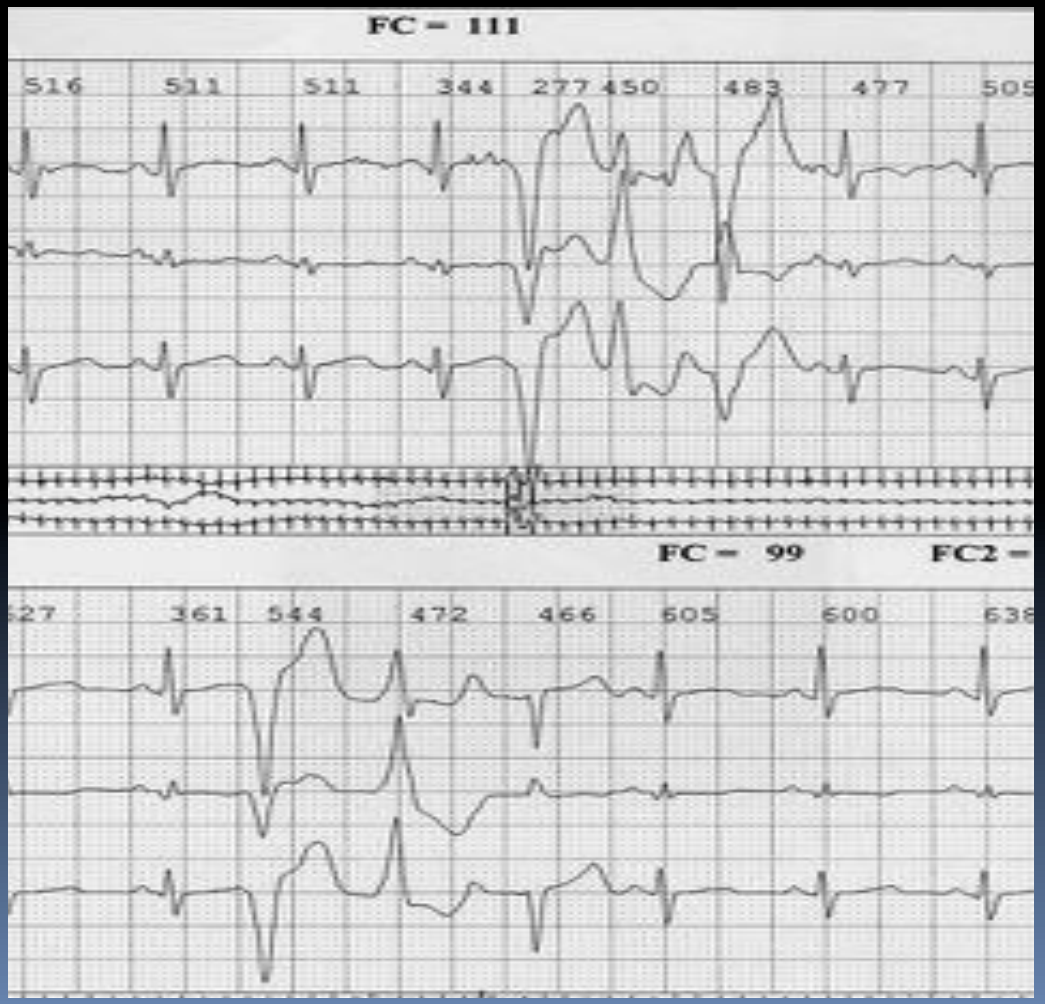


Polymorphic VEBs and couplets

24-hour ECG monitoring

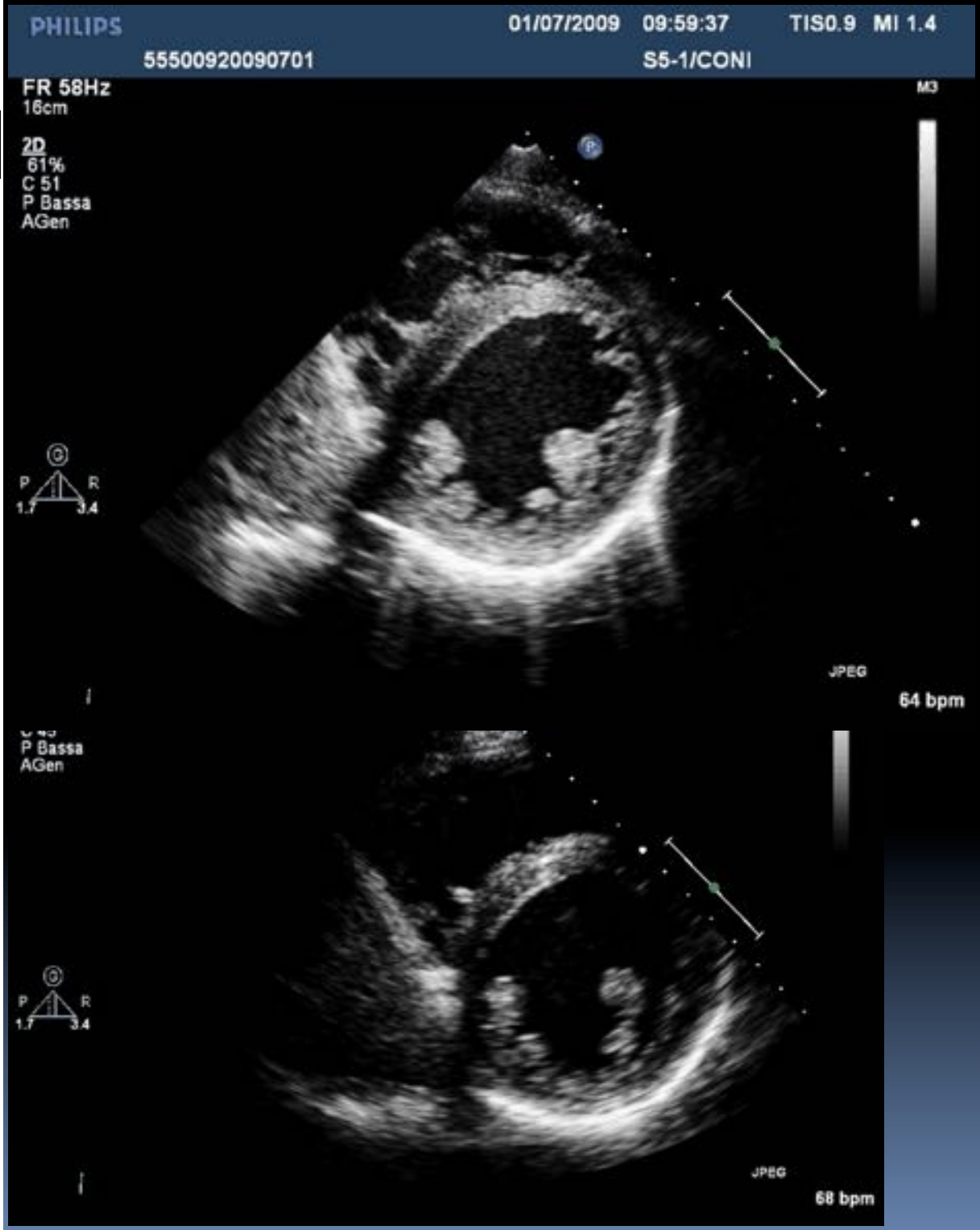
- 786 VEBs, polymorphic
- 24 couplets
- 5 NSVT





Benign arrhythmia in the athlete's heart

Arrhythmogenic cardiomyopathy



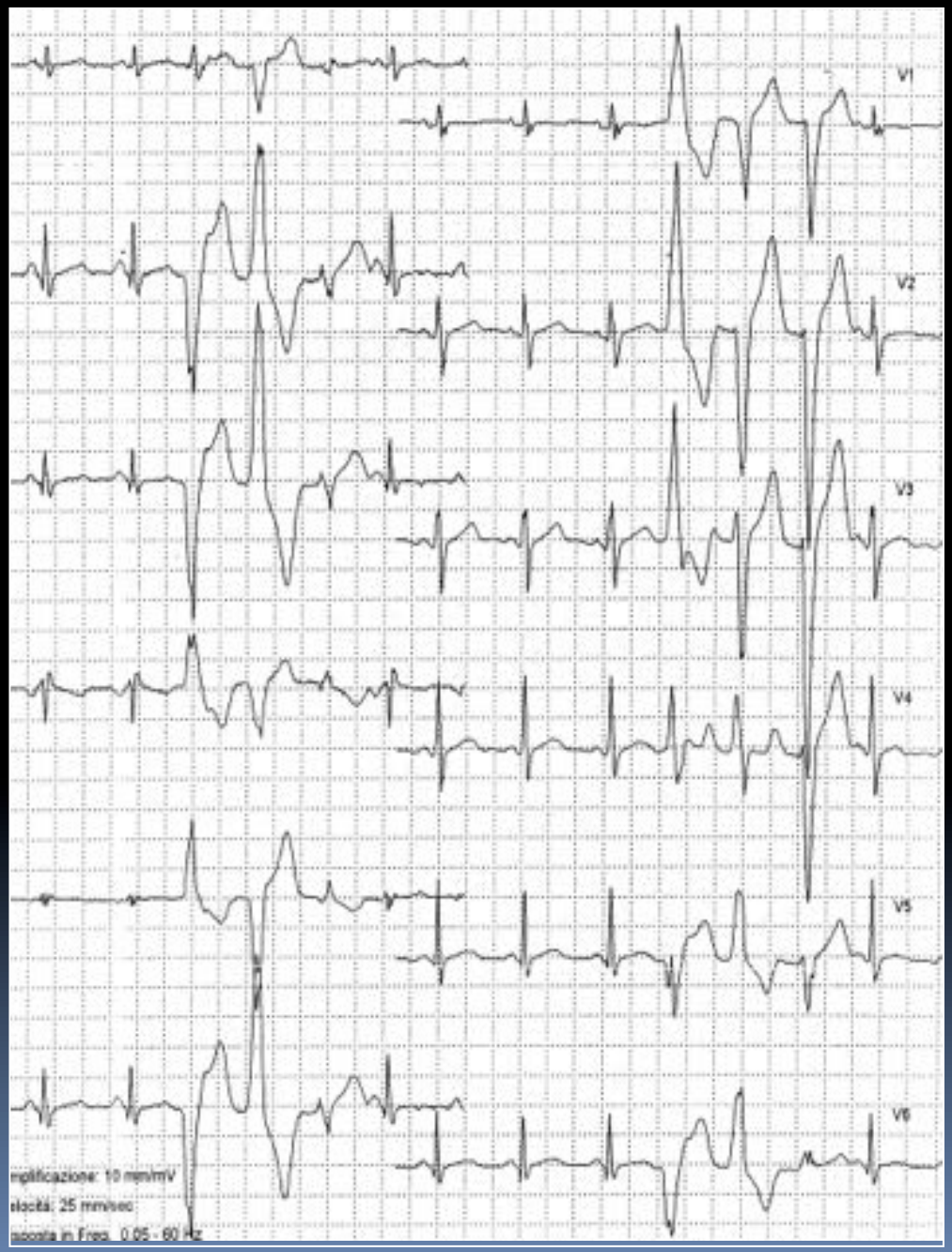
At peak-training:

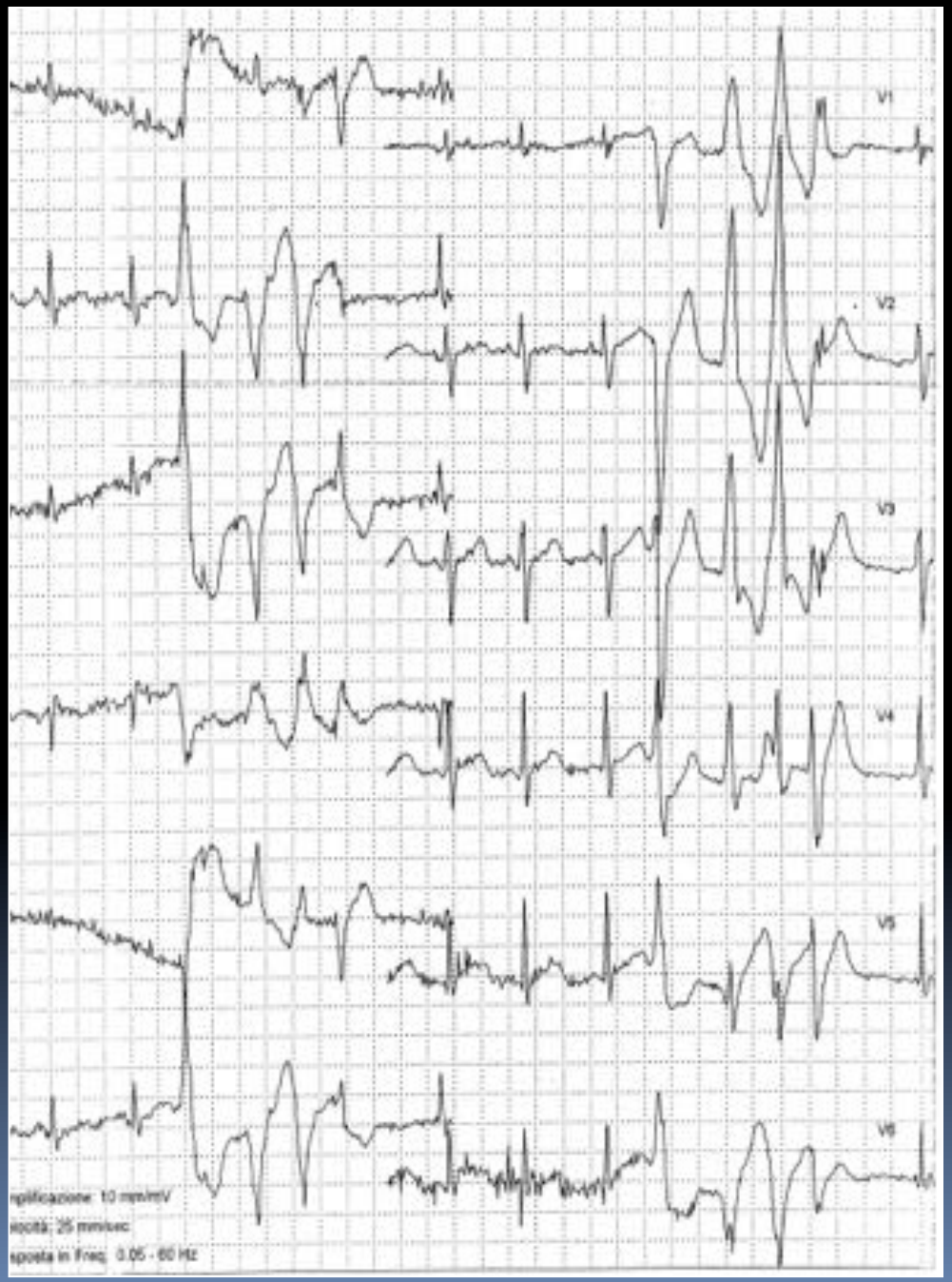
- LVDd 58 mm
- AVS 12 mm

After 2-month detraining:

- LVDd 56 mm
- AVS 11 mm

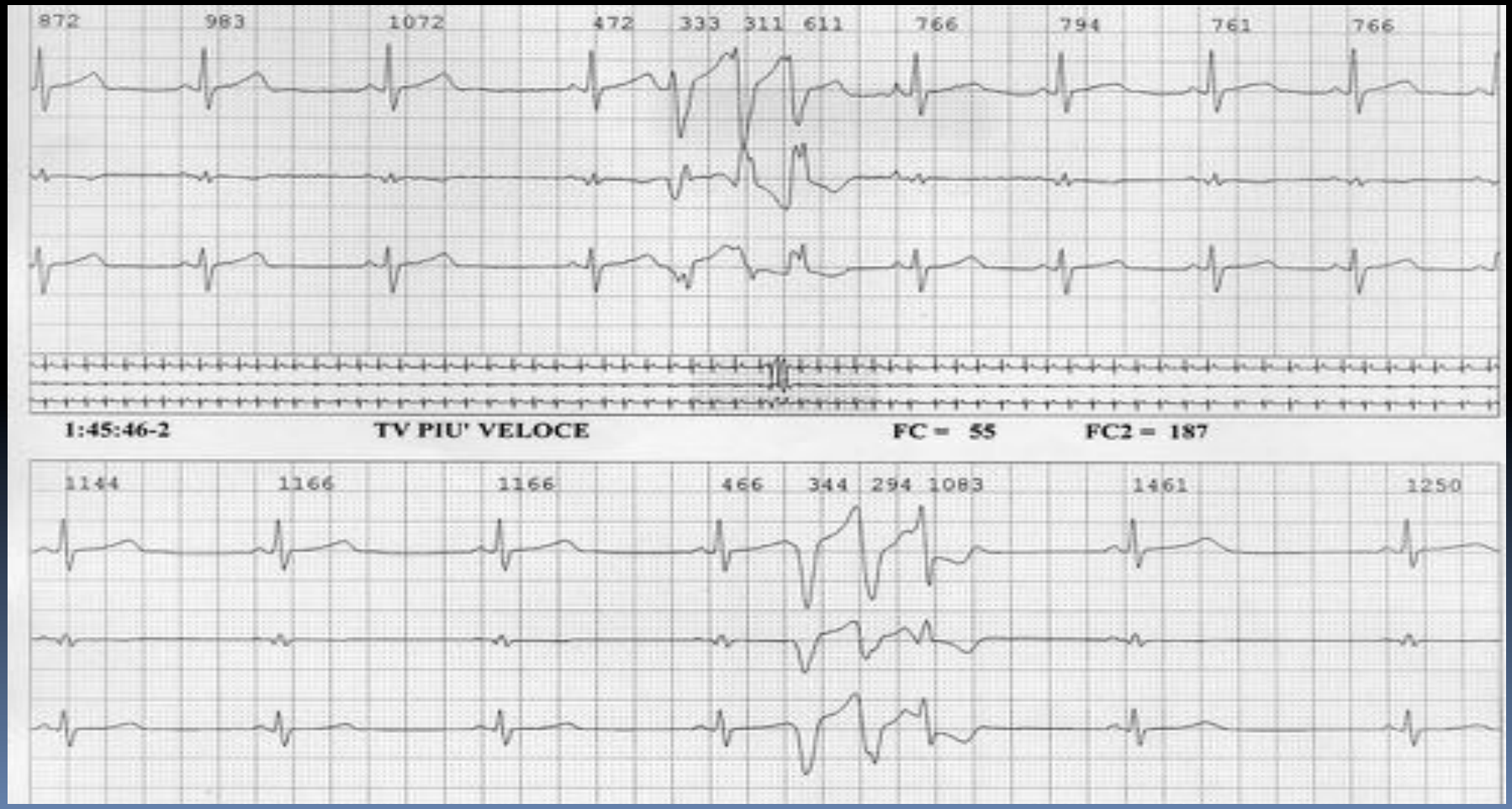
After 2-month detraining: hr:172 bpm; BP: 180/75 mmHg

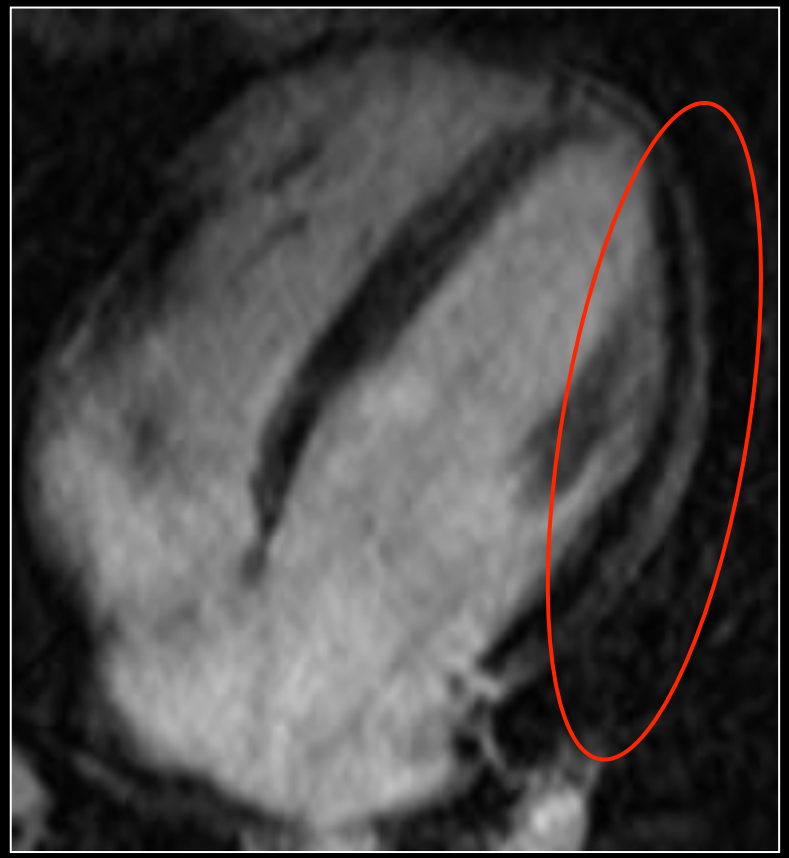


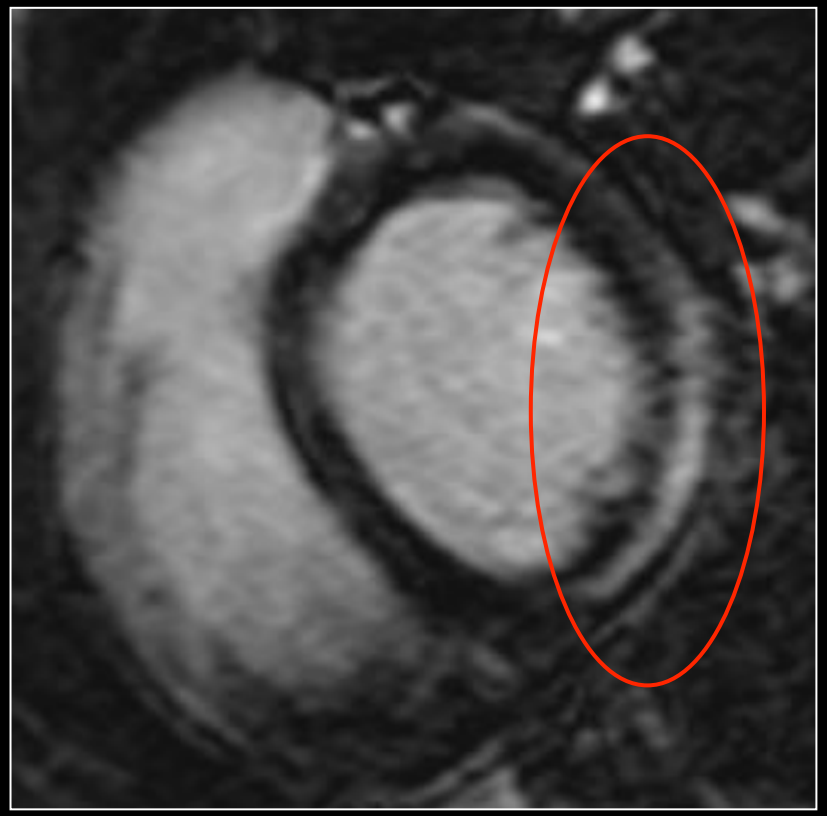


24-hour ECG monitoring after detraining

- 891 VEBs polymorphic
- 56 couplets
- 10 NSVT







… after gadolinium … evidence for late enhancement at subepicardial layer of the myocardial lateral wall

Conclusion: images compatible with degenerative/ inflammatory cardiomyopathy

Comments:

Not always standard echo is capable to detect, or raise suspicion, for subtle cardiac abnormalities.

When clinical evidence suggests the presence of cardiac abnormality, we **should search carefully and repeatedly for the pathologic substrate.**

Conclusions

- Competitive physical activity may have an arrhythmogenic effect both in subjects with cardiovascular abnormalities and in subjects without
- In athletes without heart disease, the variability and reduction of the arrhythmia over time, often increased after detraining, is a criterium "*ex adjuvantibus" of* benignity
- If the arrhythmia remains stable or worsens over time, an accurate research/diagnosis of cardiopathy, of an arrhythmogenic syndrome, of an assumption of illicit drugs, or dystiroidism becames mandatory and not delayed

THANK YOU

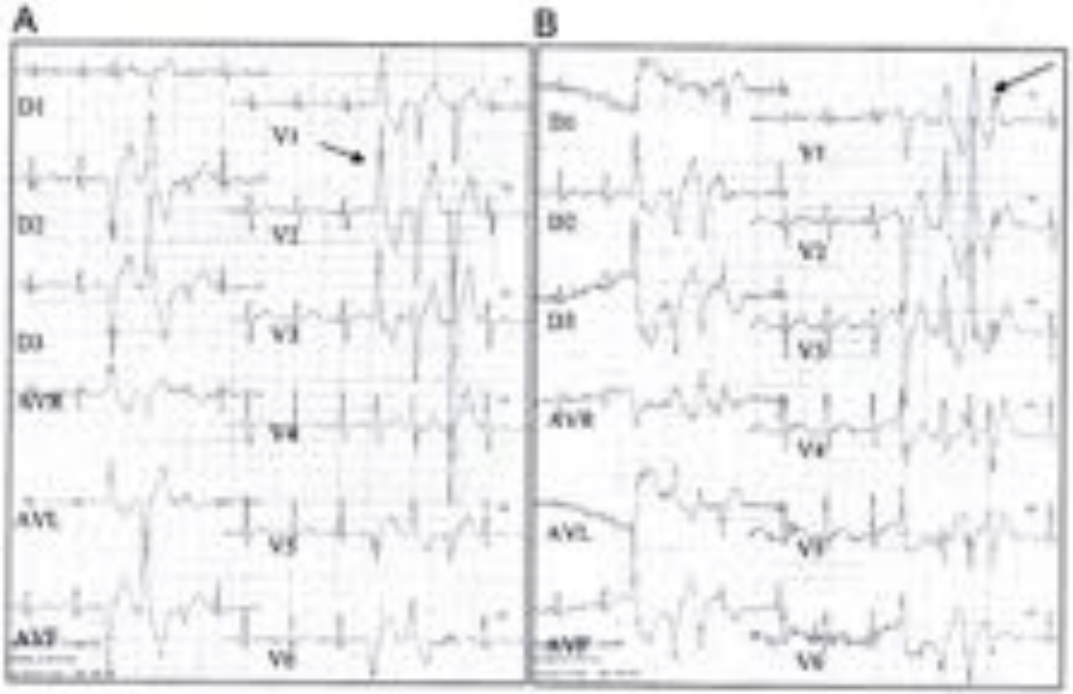
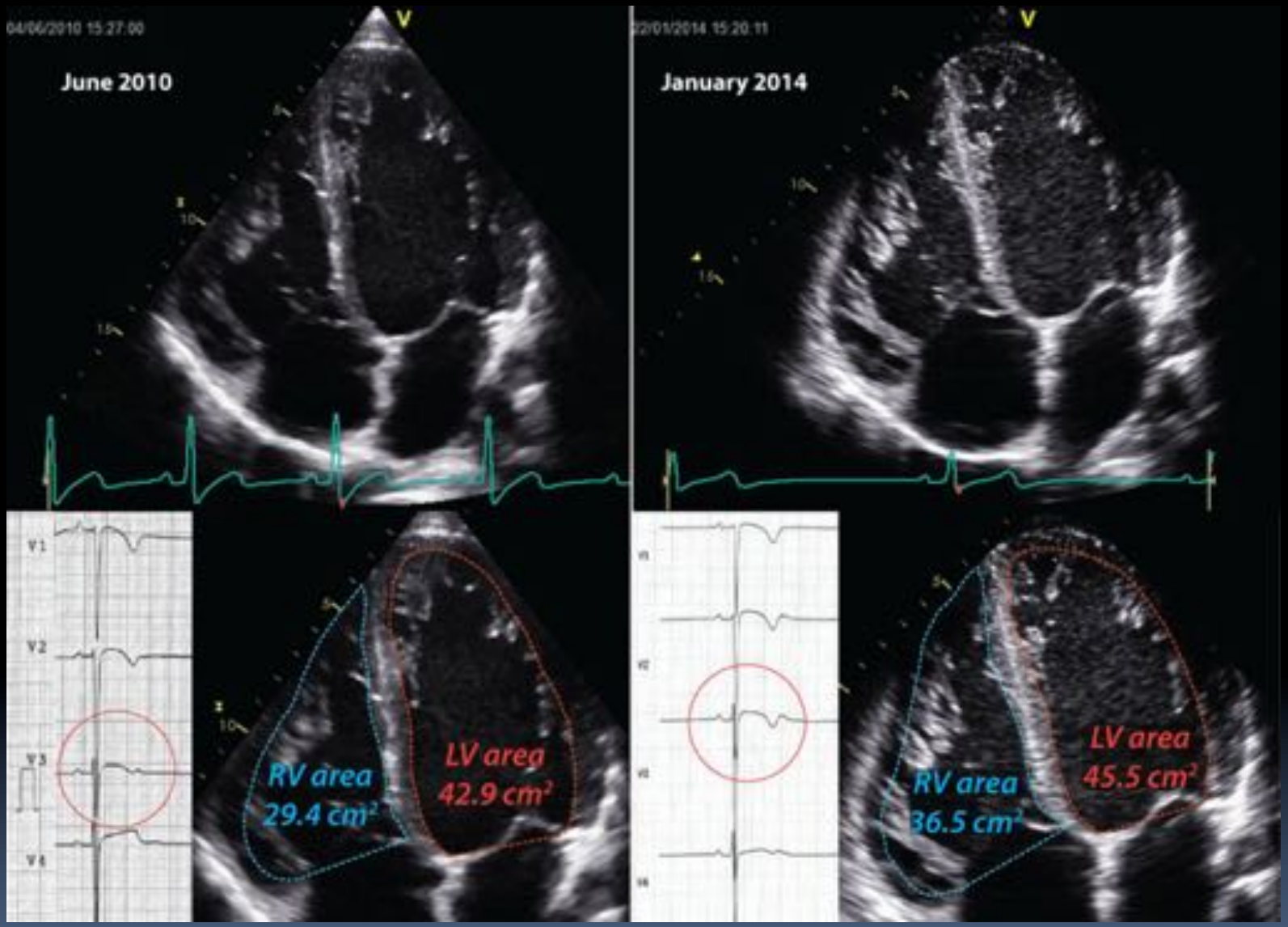


Figure 3 Exercise-induced polymorphic ventricular tachycardia in a 20-year-old elite male swimmer. **A:** At the early stage of testing, 12-lead exercise testing electrocardiographic tracing shows asymptomatic episodes of polymorphic, bidirectional nonsustained ventricular tachycardia (arrow). **B:** After 30 seconds, the electrocardiographic tracing shows the occurrence of another 4-beat episode of nonsustained polymorphic ventricular tachycardia, with the last R'R' interval of 250 ms (arrow). Further diagnostic investigations, such as contrast-enhanced cardiovascular magnetic resonance, excluded cardiovascular abnormalities. The athlete continued sports activity with spontaneous reduction of the arrhythmia over 10-year follow-up and without cardiovascular events or symptoms.



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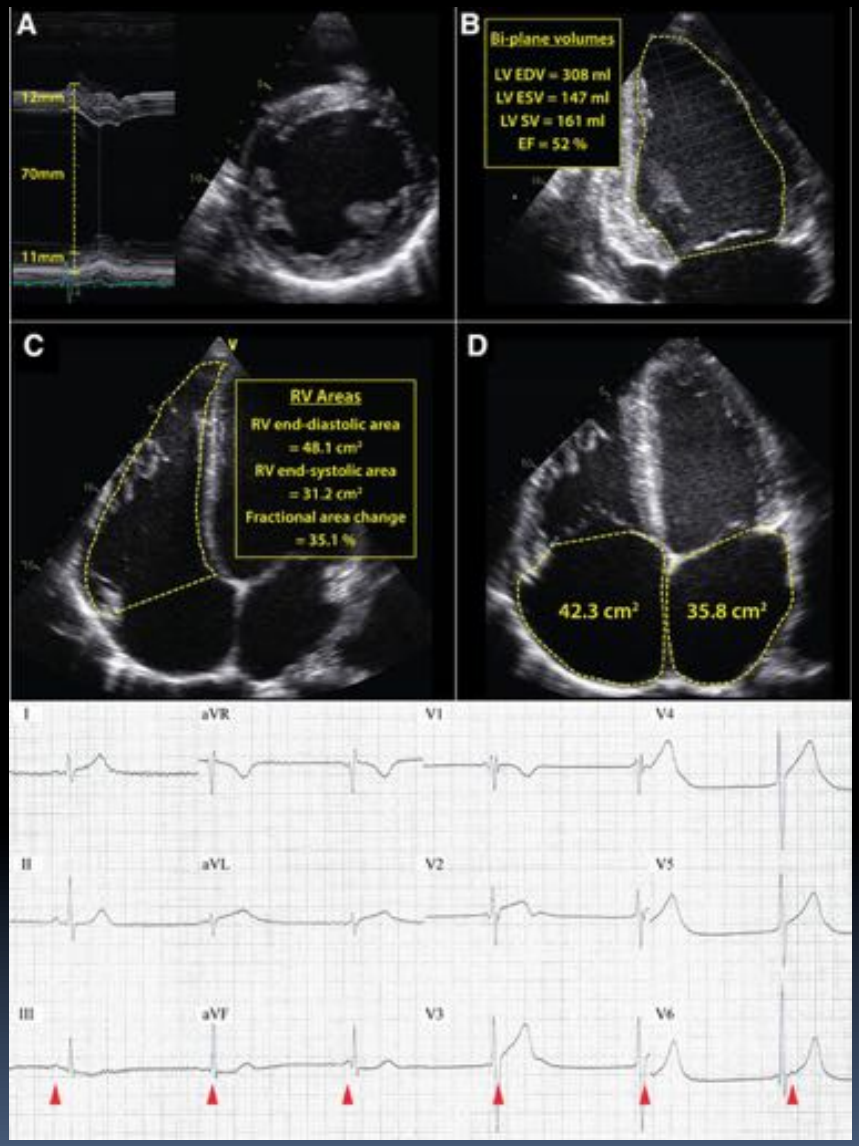

André La Gerche, and Hein Heidbuchel Circulation. 2014;130:992-1002