

ARRHYTHMIC DANGER IN SENIOR ATHLETES



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DR. DAN HADAS

THE ISRAELI SPORTS CARDIOLOGY CENTER

2 CASES SAME STORY DIFFERENT ENDING



THIS IS HOW IT COULD HAVE ENDED

**IMMAGINI
SCONSIGLIATE
A UN PUBBLICO
SENSIBILE**

R.I.P Piermario Morosini (5 July 1986 – 14 April 2012)

MISTER B

40 YEARS OLD MALE

UNTIL 2012 SUFFERED FROM OBESITY WITH BMI OF 45

STARTED BIKING AND LOST 50 KG

PREFERENCE.....UP HILL.....300-400 KM/W

NO CONSUMPTION OF SUPPLEMENTS (APART FROM VIT. C)

NOT SMOKING

NO HISTORY OF ISCHEMIC HEART DISEASE

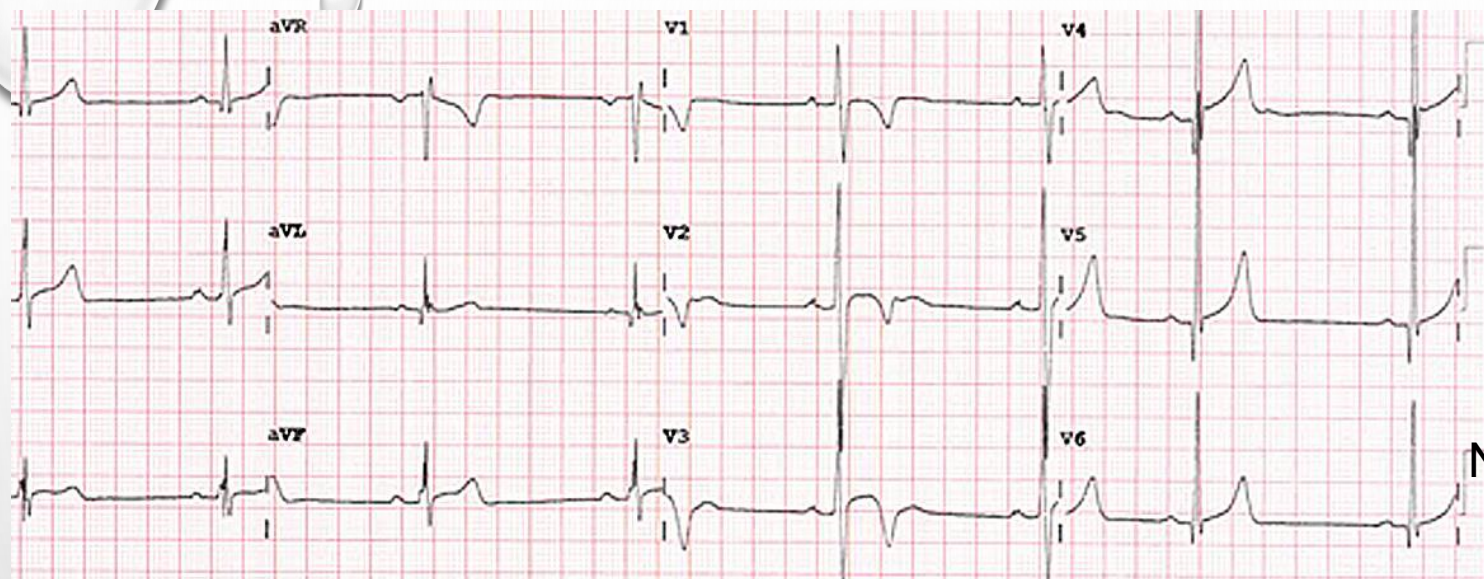
NO FAMILY HISTORY OF SUDDEN DEATH



THE D DAY

- THAT DAY WAS A RESTING DAY
- SINCE THE MORNING STARTED COMPLAINING OF CHEST PAIN THAT IS NOT DESCRIBED AS TIGHTNESS, PRESSURE, OR SQUEEZING BUT MORE OF A SHARP NEEDLE AND REPRODUCED ON PALPATION OR INHALING DEEPLY
- NO FEVER OR URTI SYMPTOMES
- SENT BY HIS WIFE TO THE DOCTOR





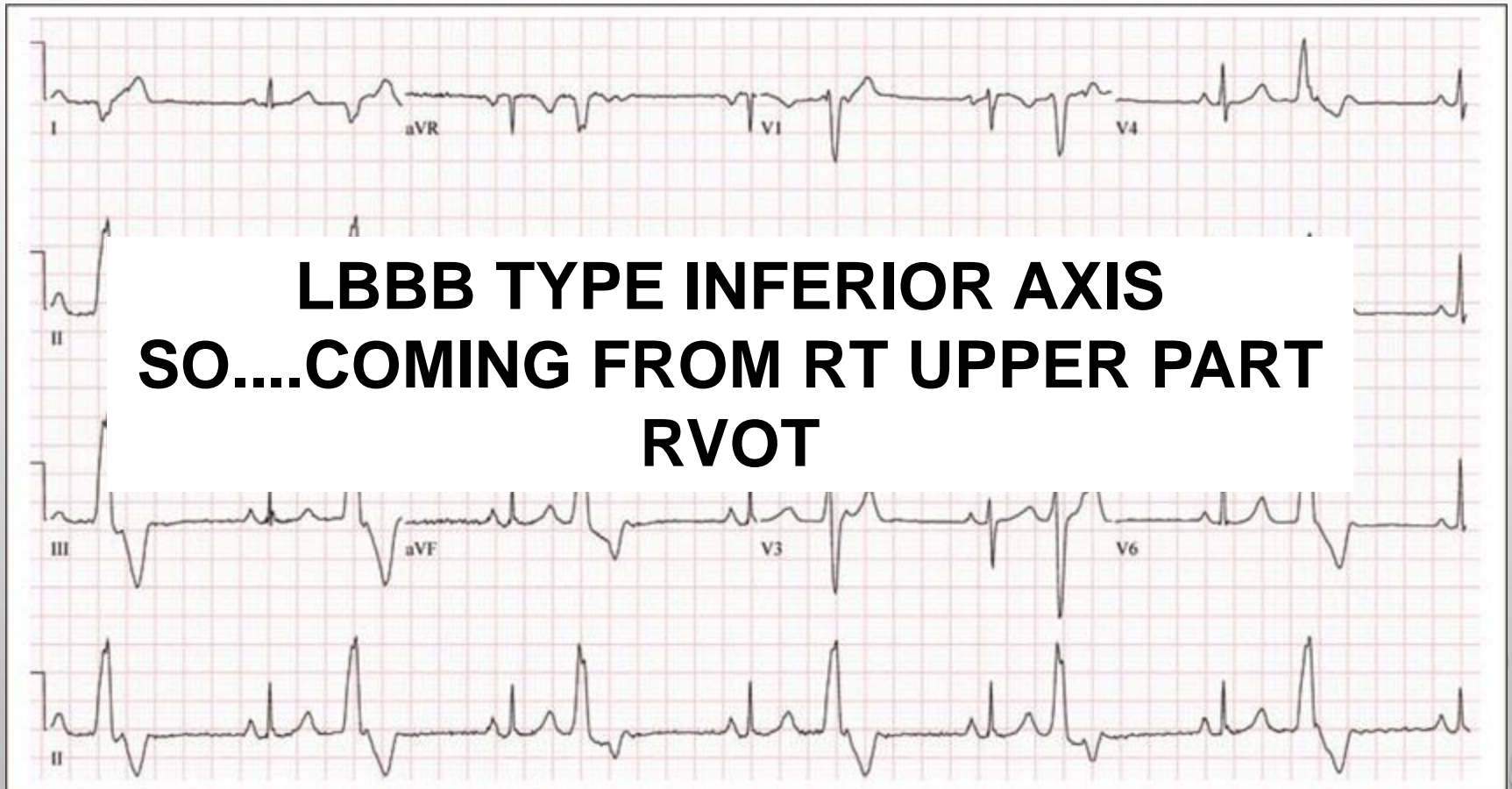
Sinus bradycardia

Neg t waves v1-v3

ST elevations

- **TREATED WITH ASPIRIN 300 MG**
- **SENT BY AMBULANCE TO THE ER**

IN THE AMBULANCE....BIGEMINY



ECG IN THE ER....THE SAME

ECHOCARDIOGRAPHY BED SIDE-MILD LV DYSFUNCTION

TROPONIN-0.02 (TWICE THE DETECTABLE VALUES)

CXR- MILD HEART DILATION

ASYMPTOMATIC

ADMITTED FOR OBSERVATION...

Physical activity, exercise and cardiac troponins: Clinical implications

Kristin M. Aakre, Torbjørn Omland

Next day Troponin below 0.013

Full research paper

Cardiovascular stress biomarker assessment of middle-aged non-athlete marathon runners

Michał Kosowski^{1,2}, Katarzyna Młynarska², Jan Chmura³, Dorota Kustrzycka-Kratochwil², Małgorzata Sukiennik-Kujawa², John A Todd⁴, Ewa A Jankowska^{1,2}, Waldemar Banasiak², Krzysztof Reczuch^{1,2} and Piotr Ponikowski^{1,2}

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SAGE

Sex differences in heart rate recovery after endurance exercise

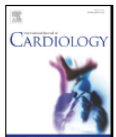
Zhaowei Kong, Jinlei Nie, Hua Lin, Keith George, Gang Zhao, Ming Zhang, Tomas K. Tong & Qingde Shi

International Journal of Cardiology 283 (2019) 1–8

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Race duration and blood pressure are major predictors of exercise-induced cardiac troponin elevation

Øyunn Kleiven^{a,*,1}, Torbjørn Omland^{b,1}, Øyvind Skadberg^{c,1}, Tor Harald Melberg^{a,1}, Magnus Friestad Bjørkavoll-Bergseth^{a,1}, Bjørn Auestad^{d,e,1}, Rolf Bergseth^{f,1}, Ole Jakob Greve^{g,1}, Kristin Moberg Aakre^{h,i,j,1}, Stein Ørn^{a,k,1}



WHAT WOULD YOU DO....



REPEAT ECHO

Table 2 Athlete's left heart morphologic and functional parameters including upper or lower limits

First author	Year	No. of athletes	Type of sport	Parameter	Gender	Mean value	Cut-off value
Pelliccia	1999	1309	S P M E	LV End diastolic diameter (mm)	♂	55	70
Whyte	2004	442	P E	LV End diastolic diameter (mm)	♀	49	65
Pelliccia	1996	600	S P M E	LV End diastolic diameter (mm)	♀	49	66
Makan	2005	900	E	LV End diastolic diameter (mm)	♂ and ♀ Adolescent	51	60
Spirito	1994	947	S P M E	LV wall thickness (mm)	♂	10	16
Rawlins	2010	440	P E	LV wall thickness (mm)	♀ Black	9.5	13
Sharma	2002	720	P E	LV wall thickness (adolescent) (mm)	♂ and ♀ Adolescent	9.5	12
Basavarajaiah	2008	300	P E	LV wall thickness (black athletes) (mm)	♂ Black	11.5	16
Caselli	2015	1145	S P M E	LV mass/BSA (g/m ²)	♂ and ♀	103	146
Finocchiaro	2016	1083	P M E	LV mass/BSA (g/m ²)	♂	83	117
					♀	101	143

Echocardiogram:

NORMAL CARDIAC FUNCTION AND ANATOMY

LA-4.3 LVD-54 LVS-35 IVSD-11 PW-10

Caselli	2015	1145	S P M E	LV ejection fraction (%)	♂ and ♀	64	55
				E/A		1.93	1.3
				TDI e' septal (cm/s)		13.8	10.3
				TDI e'/a' septal (cm/s)		2.04	1.23
				E/e' septal		6.4	8.5
D'Andrea	2010	650	P E	TDI s' septal (cm/s)	♂ and ♀	13	8
				TDI e' septal (cm/s)		24	10
				TDI s' lateral (cm/s)		15	9
				TDI e' lateral (cm/s)		16	11
				TDI e'/a' lateral		1.45	1.2
D'Andrea	2006	155	P	LV Intra-ventricular delay (ms)	♂ and ♀	9.5	45

BSA, body surface area; LA, left atrium; LV, left ventricle; TDI, tissue Doppler imaging. Type of sport: S, skill; P, power; M, mixed; E, endurance. ♀, female; ♂, male.

24 HRS HOLTER ECG

- HEART RATE 32-62 AVERAGE 40
- 2070 VPC'S RECORDED (4.1%)
- 1 EPISODE OF TRIPLET HR 139
- WENCKEBACH EPISODES AND 19 PAUSES UP TO 2.8 SEC.
- 220 APC'S
- **VPC'S ARE OF RVOT AND MIDDLE RV ORIGIN**

DISCHARGE?



- JUST AFTER A CMRI WAS DONE , WITHOUT LGE OBSERVED....

AFTER A WEEK OF REST... EXERCISE TEST

- BRUCE PROTOCOL
- 19.09 MINUTES UP TO 23.6 METS
- HR 52-176
- BP 120/80-186/74
- **ACHIEVED MAXIMAL EFFORT**

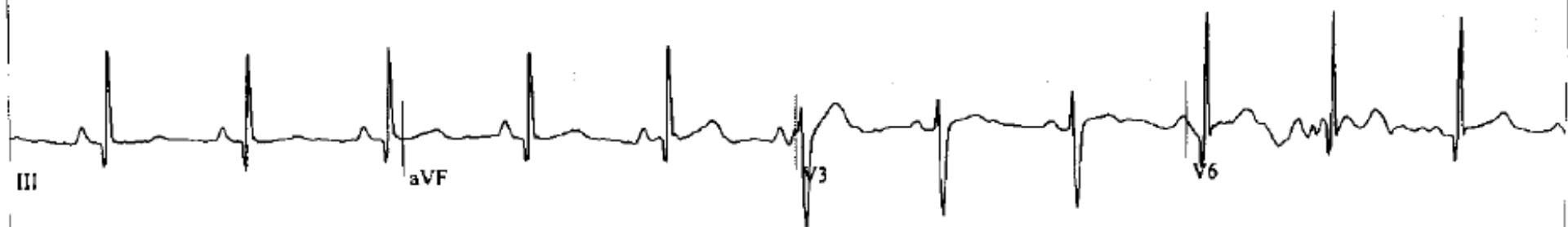
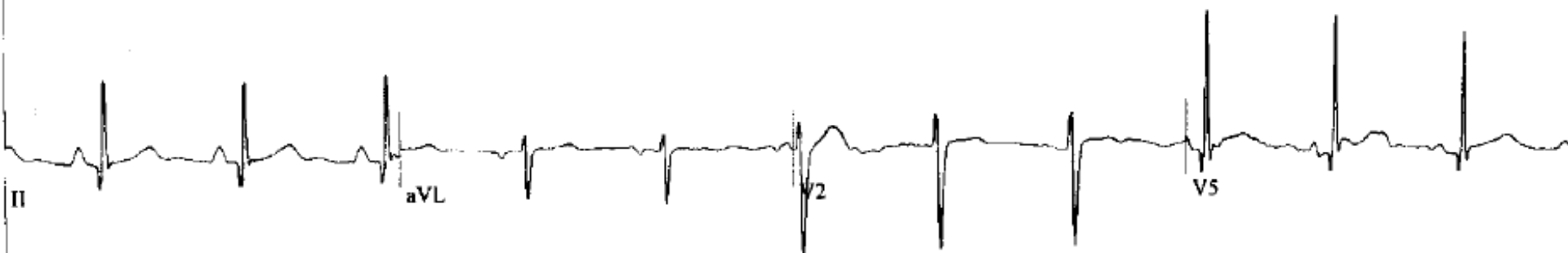
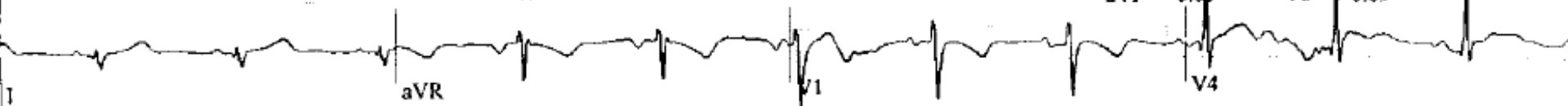


12-Lead Report

PRETEST
WARM-UP

01:04

II	0.02	V1	
III	-0.03	V3	0.16
aVR	-0.04	V4	0.12
aVL	0.04	V5	0.11
aVF	-0.01	V6	0.09



Recall Report

EXERCISE

STAGE 1

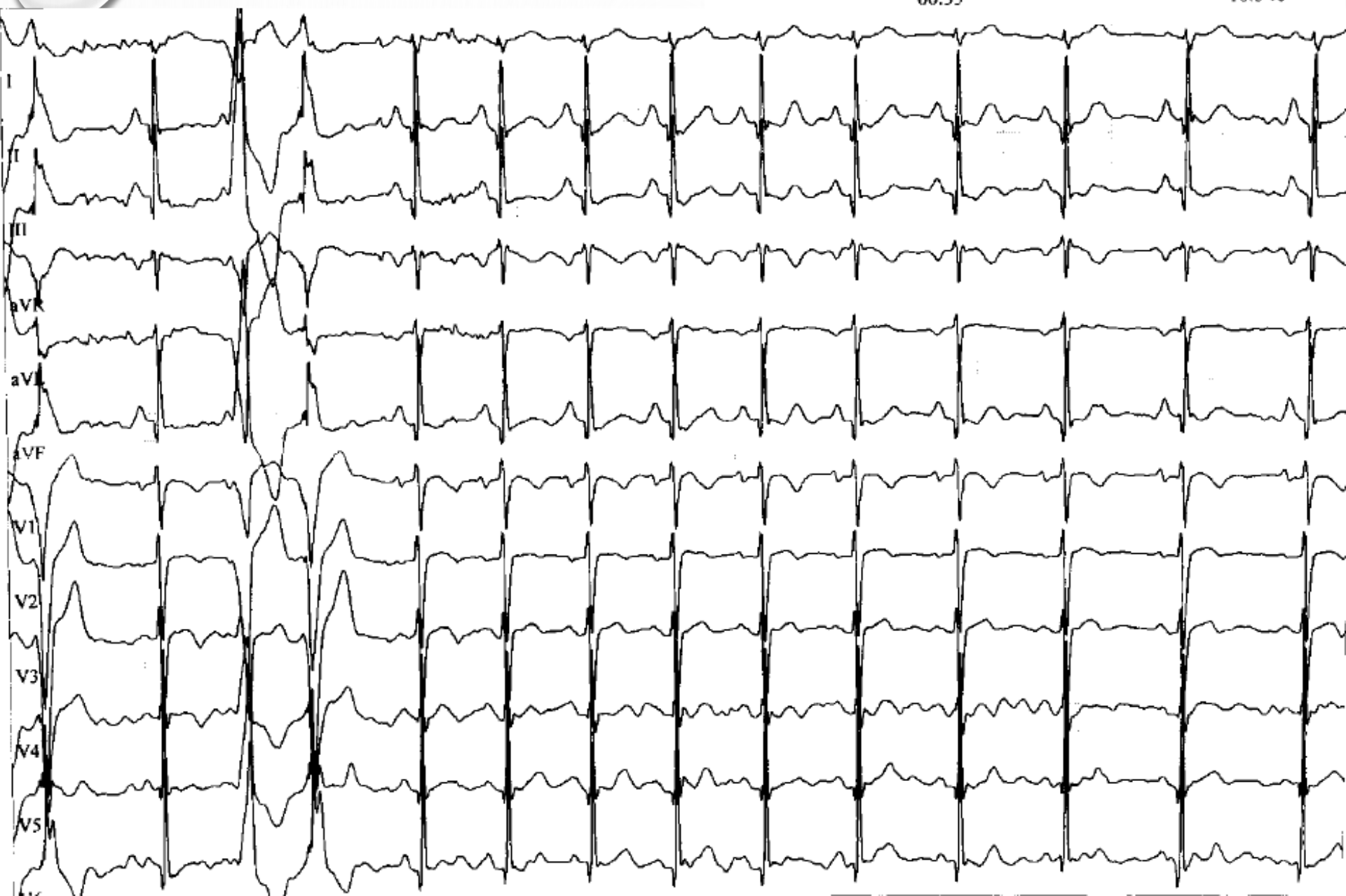
00:55

BRUCE

2.7 km/h

10.0 %

91 bpm



91 bpm
136/73 mmHg

EXERCISE
STAGE 2
03:09

BRUCE
4.0 km/h
12.0 %



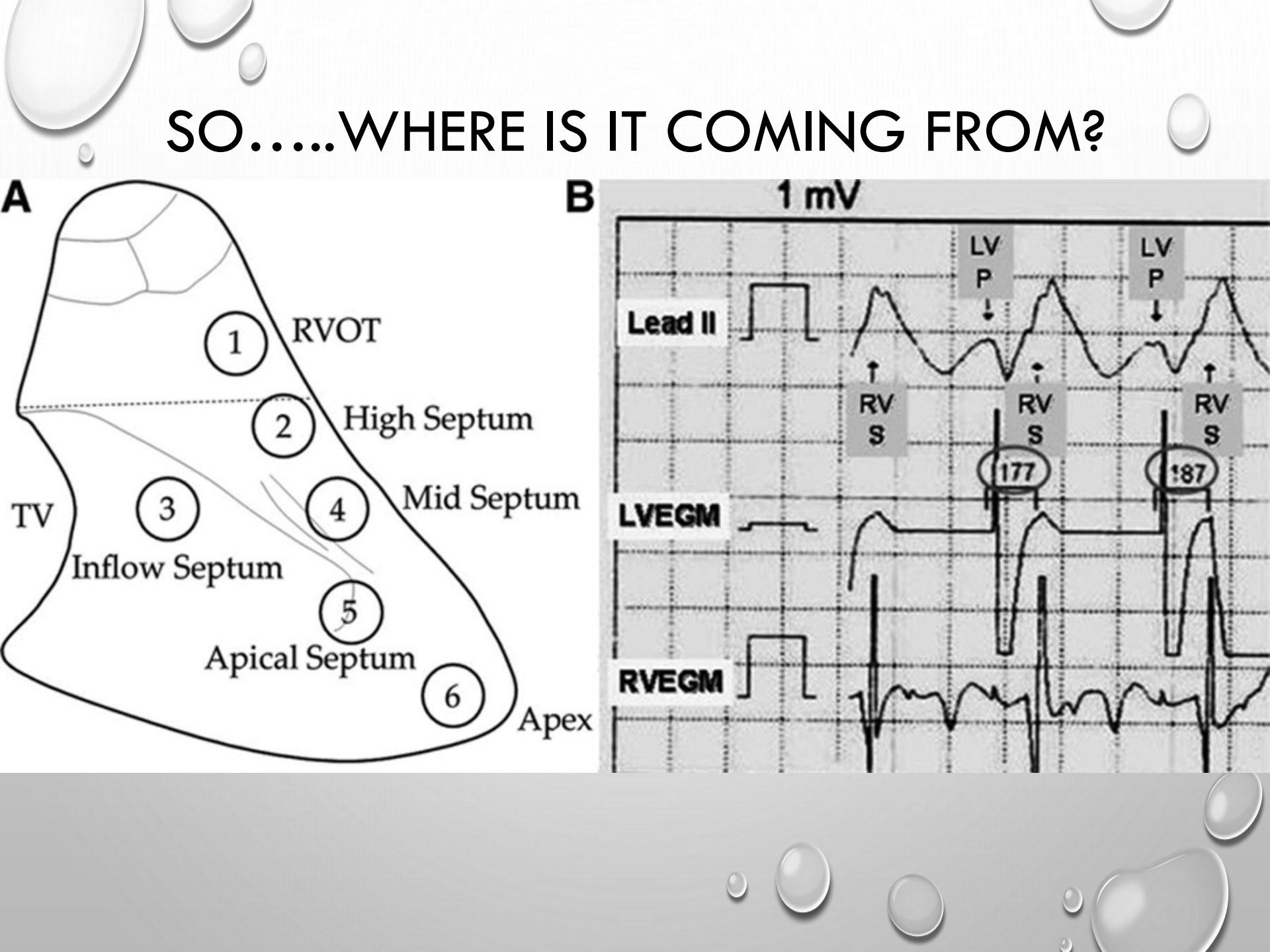
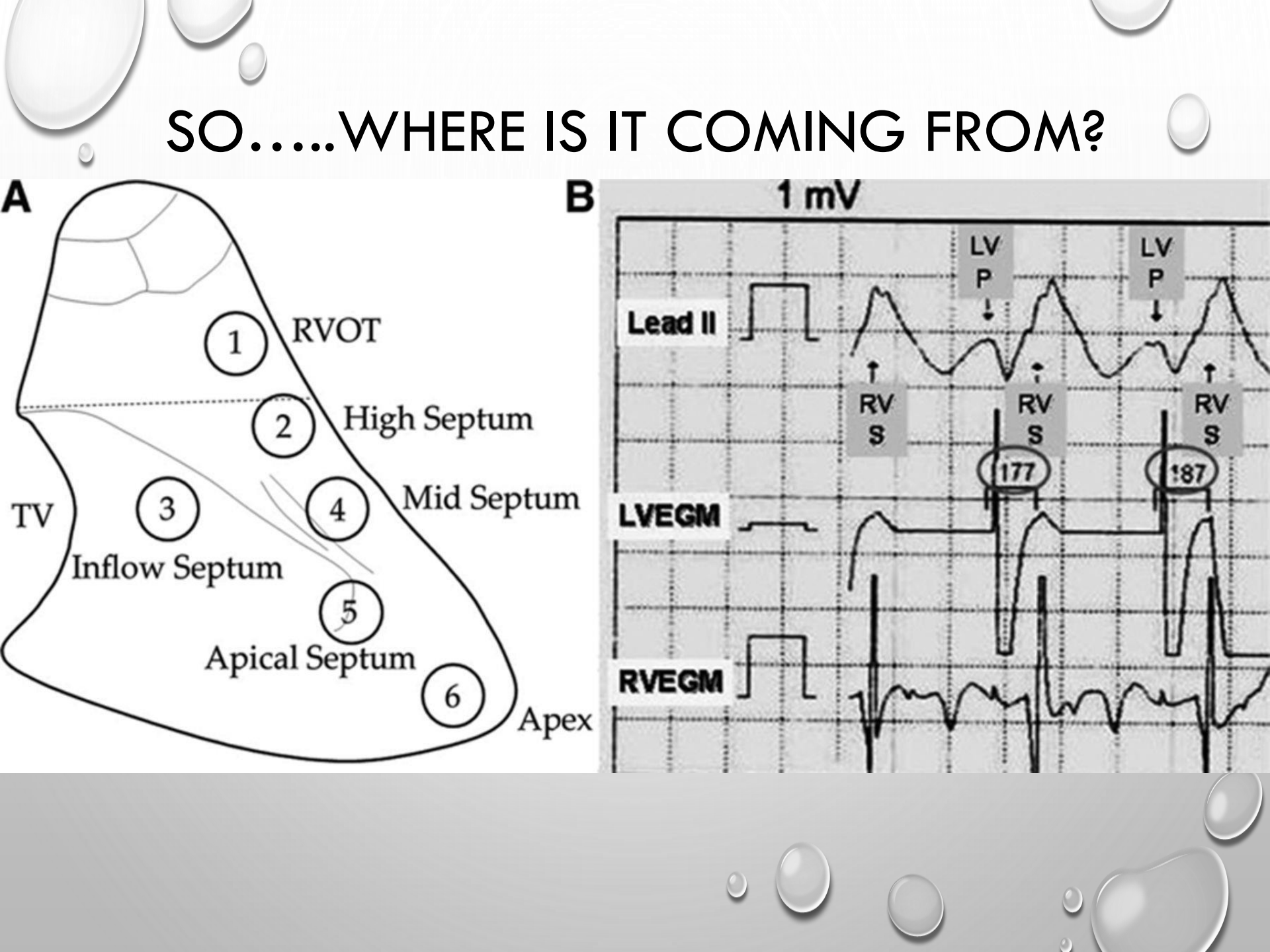
SO.....WHERE IS IT COMING FROM?

A

RVOT
High Septum
Mid Septum
Inflow Septum
Apical Septum
Apex
TV

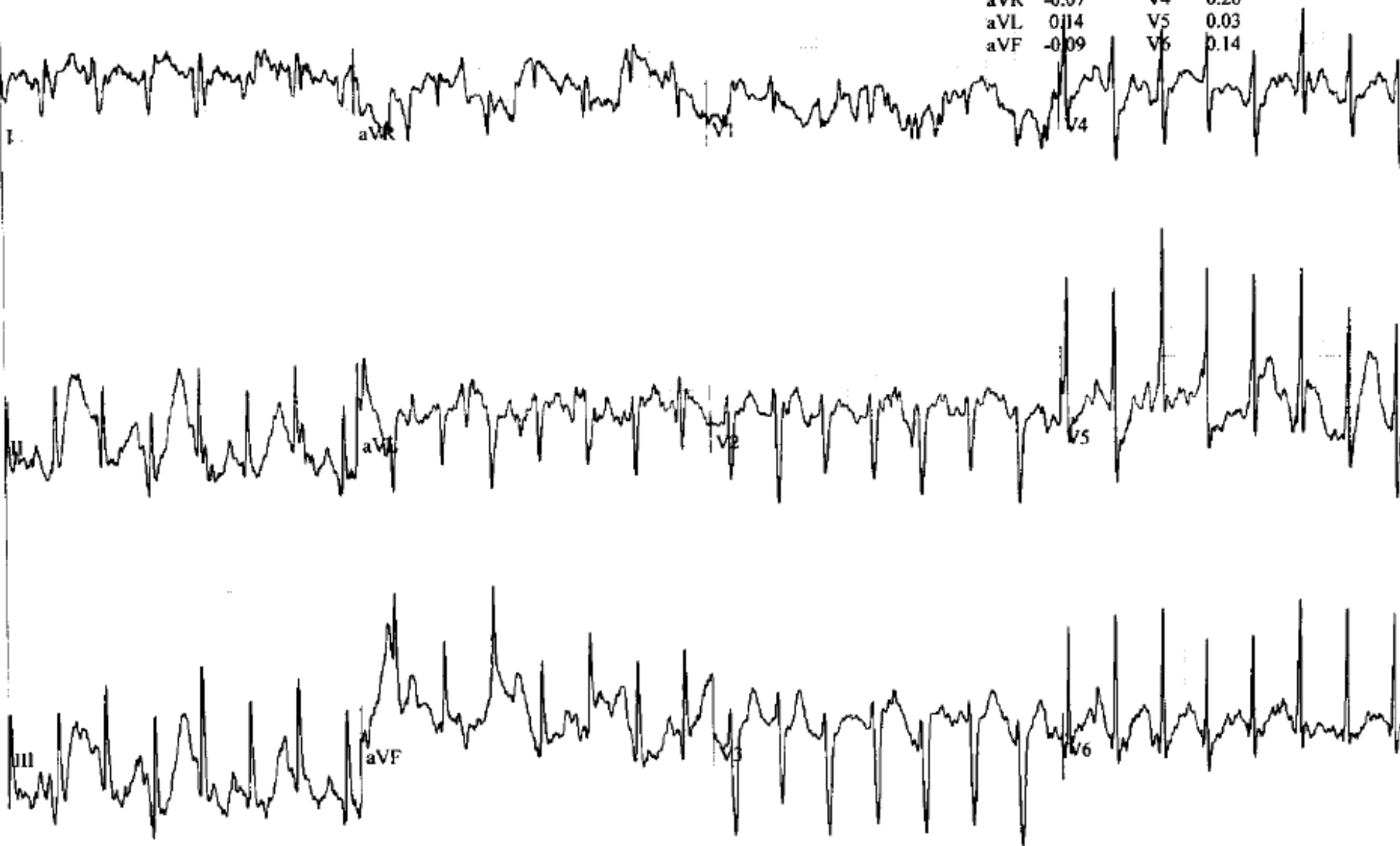
B

1 mV
Lead II
LVEGM
RVEGM
LV P
RV S
177
187



176 bpm

II	-0.01	V2	0.23
III	-0.16	V3	0.41
aVR	-0.07	V4	0.20
aVL	0.14	V5	0.03
aVF	-0.09	V6	0.14



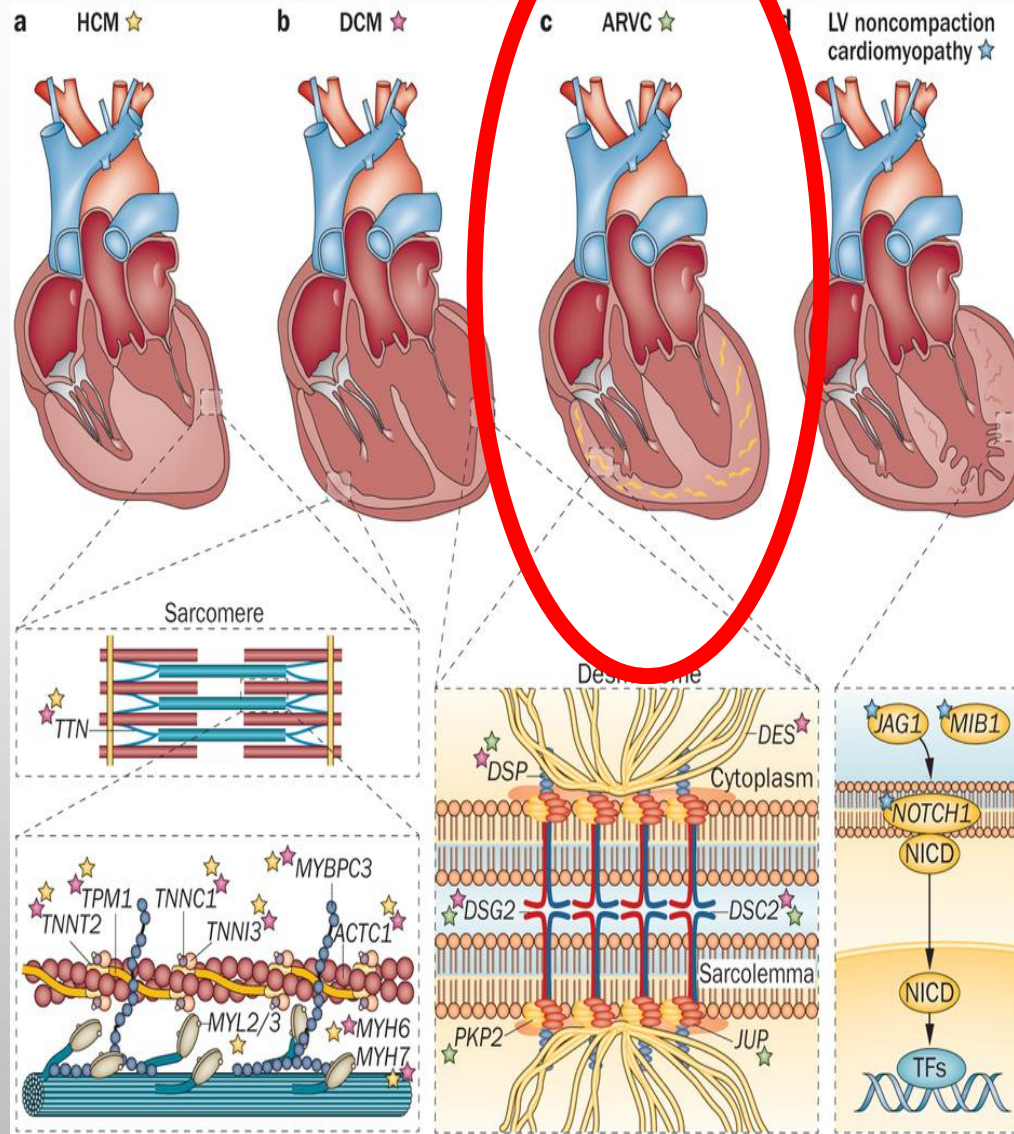
RECOVERY

- **DURING RECOVERY WHEN HEART RATE WENT BELOW 130 THE VPC'S STARTED APPEARING AGAIN....**
- **RVOT ORIGIN...**
- **THEN A COUPLET FROM THE RIGHT SIDE....**
- **THENTHIS**

LBBBTYPE



ARVC

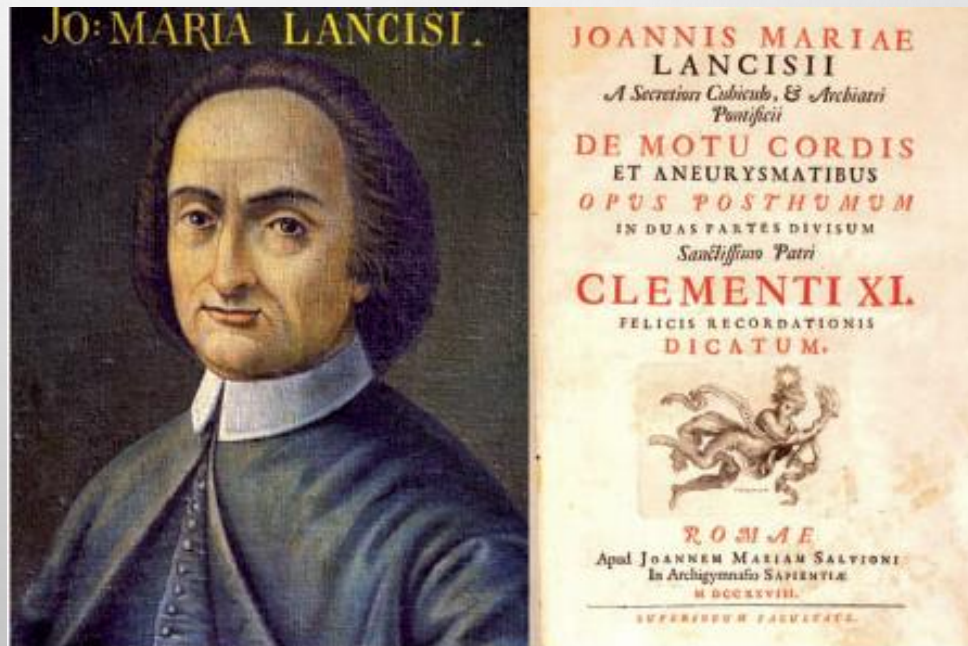


History of the discovery of Arrhythmogenic Cardiomyopathy

The history of arrhythmogenic cardiomyopathy (AC) is a paradigm in the progress of Cardiovascular Medicine knowledge, from nosology to diagnosis, treatment, and prevention. In this review, we focus on the discovery of this heart muscle disease at the beginning of Modern Medicine, something you cannot find on the Internet or PubMed

European Heart Journal, Volume 40, Issue 14, 07 April 2019

- THE DISCOVERY OF ARRHYTHMOGENIC CARDIOMYOPATHY (AC), DATES BACK TO THE XVIII CENTURY WHEN IN 1728, LANCISI REPORTED A FOUR-GENERATION FAMILY AFFECTED BY PALPITATIONS, HEART FAILURE, AND SUDDEN DEATH, IN WHICH AUTOPSY REVEALED DILATATION AND ANEURYSMS OF THE RIGHT VENTRICLE (RV).



WHAT DOES THE 2019

2019 HRS expert consensus
risk stratification,
arrhythmogenic

European Heart Journal (2019) 0, 1–16
doi:10.1093/eurheartj/ehz669



Arrhythmogenic right ventricular
cardiomyopathy: evaluation of the current
diagnostic criteria and differential diagnosis

CURRENT OPINION
Heart failure/cardiomyopathy



Table 1. International Task Force Criteria for the Diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy.*		
Category	Major Criteria	Minor Criteria
Global or regional dysfunction and structural alteration†		
On two-dimensional echocardiography	Regional RV akinesia, dyskinesia, or aneurysm and one of the following (end diastole): PLAX RVOT ≥ 32 mm (≥ 19 mm per square meter when corrected for body-surface area), PSAX RVOT ≥ 36 mm (≥ 21 mm per square meter when corrected for body-surface area), or fractional area change of $\leq 33\%$	Regional RV akinesia or dyskinesia and one of the following (end diastole): PLAX RVOT 29 to <32 mm (16 to <19 mm per square meter when corrected for body-surface area), PSAX RVOT 32 to <36 mm (18 to <21 mm per square meter when corrected for body-surface area), or fractional area change of 34 to 40%
On MRI	Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and one of the following: ratio of RV end-diastolic volume to body-surface area ≥ 110 ml per square meter (male patients) or ≥ 100 ml per square meter (female patients), or RV ejection fraction $\leq 40\%$	Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and one of the following: ratio of RV end-diastolic volume to body-surface area 100 to <110 ml per square meter (male patients) or 90 to <100 ml per square meter (female patients), or RV ejection fraction 41 to 45%
On RV angiography	Regional RV akinesia, dyskinesia, or aneurysm	
Tissue characterization	$<60\%$ residual myocytes on morphometric analysis (or $<50\%$, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample	60 to 75% residual myocytes, on morphometric analysis (or 50 to 65%, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample
Repolarization abnormalities	Inverted T waves in right precordial leads (V ₁ , V ₂ , and V ₃) or beyond in patients older than 14 yr of age (in the absence of complete right bundle-branch block, QRS ≥ 120 msec)	Inverted T waves in leads V ₁ and V ₂ in patients older than 14 yr of age (in the absence of complete right bundle-branch block) or in V ₄ , V ₅ , or V ₆ ; inverted T waves in leads V ₁ , V ₂ , V ₃ , and V ₄ in patients older than 14 yr of age (in the presence of complete right bundle-branch block)
Depolarization and conduction abnormalities	Epsilon wave (reproducible low-amplitude signals from end of QRS complex to onset of the T wave) in the right precordial leads (V ₁ , V ₂ , and V ₃)	Late potentials on signal-averaged ECG in at least one of three parameters in the absence of a QRS complex duration of ≥ 110 msec on the standard ECG; filtered QRS complex duration, ≥ 114 msec; duration of terminal QRS complex <40 μ V (low-amplitude signal duration), ≥ 38 msec; root-mean-square voltage of terminal 40 msec, ≤ 20 μ V; terminal activation duration of QRS complex, ≥ 55 msec, measured from the nadir of the S wave to the end of the QRS complex, including R', in V ₁ , V ₂ , or V ₃ , in the absence of complete right bundle-branch block
Arrhythmias	Nonsustained or sustained ventricular tachycardia with a left bundle-branch block and superior axis pattern (negative or indeterminate QRS complex in leads II, III, and aVF and positive QRS complex in lead aVL)	Nonsustained or sustained ventricular tachycardia of RV outflow configuration with a left bundle-branch block and inferior axis pattern (positive QRS complex in leads II, III, and aVF and negative QRS complex in lead aVL) or unknown axis, or >500 ventricular extrasystoles per 24 hr (on Holter monitoring)
Family history	ARVC confirmed in a first-degree relative who meets current task-force criteria, ARVC confirmed pathologically at autopsy or surgery in a first-degree relative, or identification of a pathogenic mutation categorized as associated or probably associated with ARVC in the patient under evaluation‡	History of ARVC in a first-degree relative in whom it is not possible or practical to determine whether current task-force criteria are met, premature sudden death (at <35 yr of age) due to suspected ARVC in a first-degree relative, or ARVC confirmed pathologically or by current task-force criteria in a second-degree relative

* The table is adapted from Marcus et al.* The diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) is considered to be definite if the patient meets two major criteria, one major and two minor criteria, or four minor criteria from different categories; the diagnosis is considered to be borderline if the patient meets one major and one minor criteria or three minor criteria from different categories, and the diagnosis is classified as possible if the patient meets one major or two minor criteria from different categories. ECG denotes electrocardiogram, PLAX parasternal long-axis view, PSAX parasternal short-axis view, RV right ventricular, and RVOT RV outflow tract.

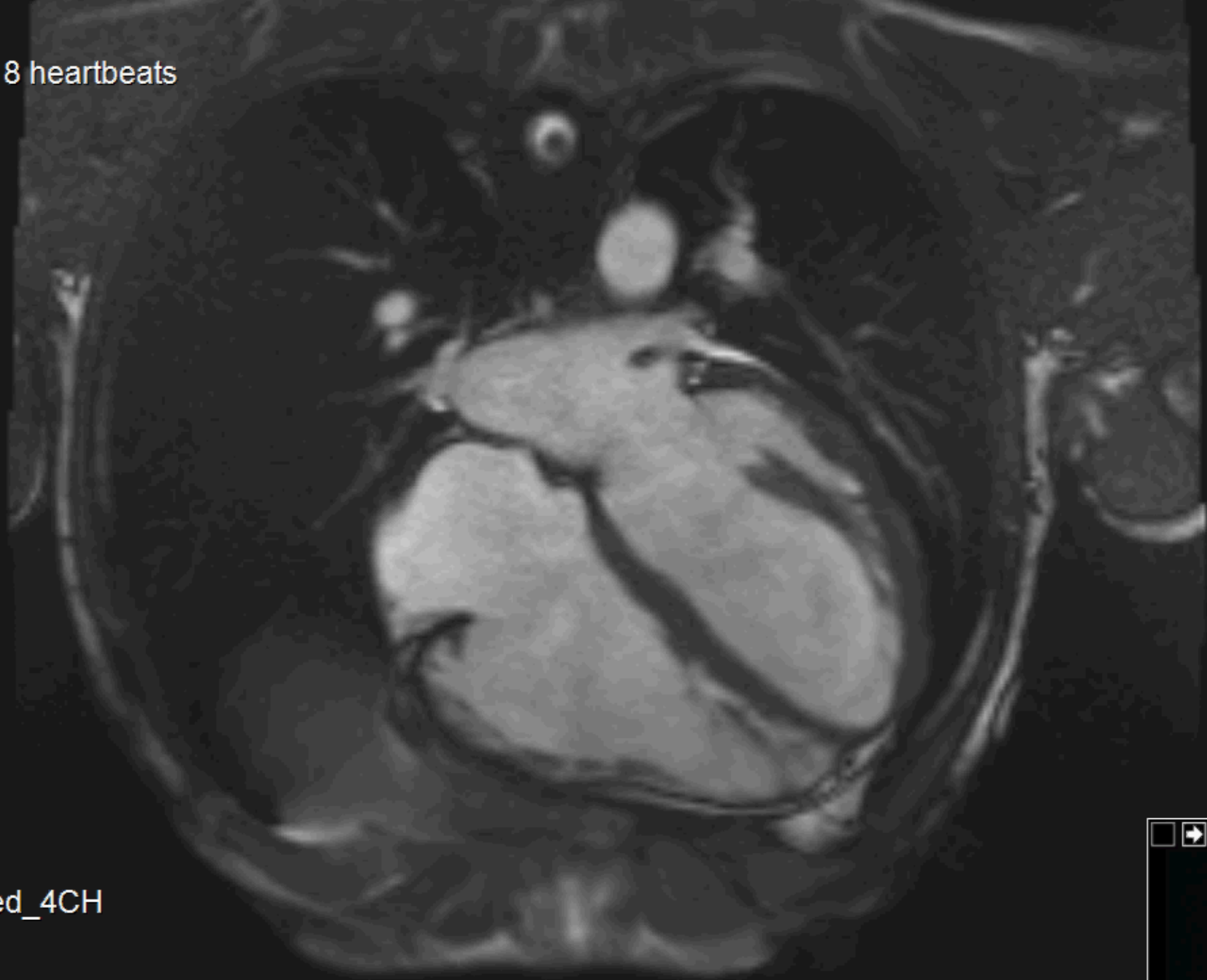
† Hypokinesia is not included in this or subsequent definitions of RV regional wall-motion abnormalities for the proposed modified criteria.

‡ A pathogenic mutation is a DNA alteration associated with ARVC that alters or is expected to alter the encoded protein, is unobserved or rare in a large, non-ARVC control population, and either alters or is predicted to alter the structure or function of the protein or has shown linkage to the disease phenotype in a conclusive pedigree (i.e., a pedigree providing conclusive evidence of a mendelian inheritance of the disease phenotype).

The background of the slide is a light gray gradient. It is decorated with several realistic water droplets of various sizes. In the top-left corner, there are three droplets of different sizes. In the top-right corner, there are two droplets. In the bottom-right corner, there is a cluster of several droplets, including a large one and several smaller ones. In the bottom-center area, there are two more droplets.

RESULTS OF CMRI CAME BACK

+/- 54; 8 heartbeats



gmented_4CH

19 09:17:03

1/2

Sequence: *tfi2d1_18

Slice: 8 mm

TR: 48.8

TE: 1.13

RR 1605 +/- 54; 8 heartbeats

C: 154.0, W: 392.0

Algo1 1/4

- **LV NORMAL ANATOMY AND FUNCTION EF-58%**
- **RV MID-LATERAL DYSKINESIS**
- **DILATED RV 5.5CM**
- **EDV/BSA-123**
- **RV EF-39%**

CINE_segmented_4CH

Pos: HFS

Series: 9

02/06/2019, 09:17:03

Image 1 of 25

02/06/2019

FA

Image no: 1

9

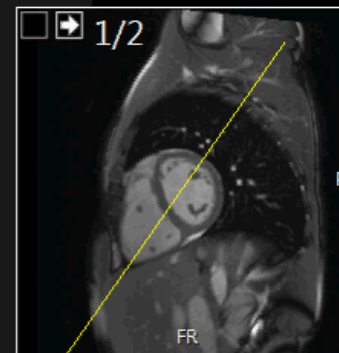


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SENT FOR GENETICS....

PKP POSITIVE MUTATION

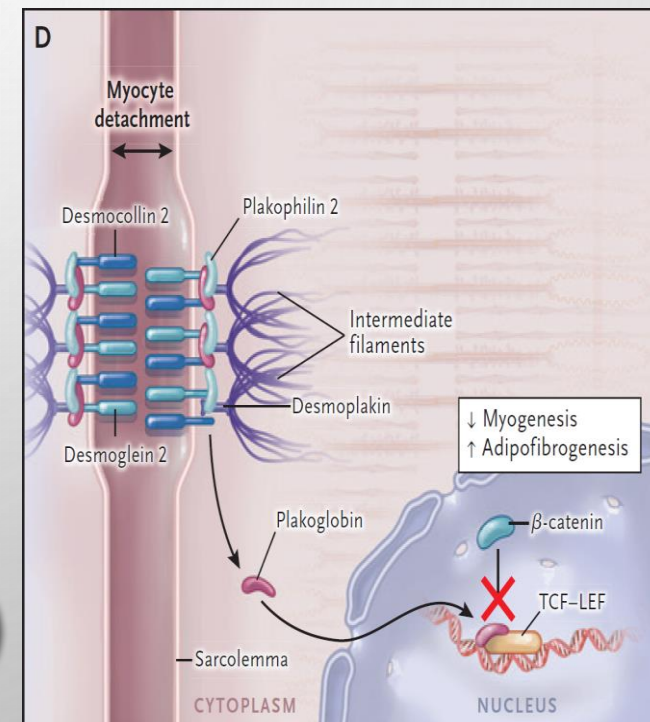


A BIT OF MOLECULAR BIOLOGY...

- ABNORMAL DESMOSOMES CONFER A PREDISPOSITION OVER TIME TO DISRUPTION OF THE INTERCELLULAR JUNCTION ,MOSTLY UNDER CONDITIONS OF INCREASED MECHANICAL STRESS, SUCH AS SPORTS ACTIVITY

Impairment of desmosomal assembly by genetically defective proteins causes the translocation of **plakoglobin** from the sarcolemma to the nucleus), where it may antagonize the effects of β -catenin. By competing with β -catenin, intranuclear plakoglobin suppresses Wnt- β -catenin signaling and **induces a gene transcriptional** switch from myogenesis **to adipogenesis** and fibrogenesis

Loss of expression of desmosomal proteins may cause or contribute to potentially fatal arrhythmias by inducing gap junction remodeling



GENETICS

- THE MOST COMMONLY AFFECTED GENE IS *PKP2* (UP TO 45% OF PATIENTS), FOLLOWED BY *DSP* (10 TO 15%), *DSG2* (7 TO 10%), AND *DSC2* (2%).
- IT HAS BEEN REPORTED THAT 16% OF HEALTHY PERSONS HAVE MISSENSE MUTATIONS IN ONE OF THE MAJOR ARVC SUSCEPTIBILITY GENES
- GENOTYPING TO CONFIRM THE DIAGNOSIS IN AN ISOLATED PATIENT WITH A BORDERLINE OR QUESTIONABLE PHENOTYPE IS NOT INDICATED ON A ROUTINE BASIS.

ARVC	PKP2 (12p11)	Plakophilin 2	25-40	60%
	DSG2 (18q12.1)	Desmoglein 2	5-10	
	DSP (6p24)	Desmoplakin	2-12	
	DSC2 (18q12.1)	Desmocollin 2	2-7	

Table 1. International Task Force Criteria for the Diagnosis of Arrhythmogenic Right Ventricular Cardiomyopathy.[‡]

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Tissue characterization	$<60\%$ residual myocytes on morphometric analysis (or $<50\%$, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample	60 to 75% residual myocytes, on morphometric analysis (or 50 to 65%, if estimated) and fibrous replacement of the RV free-wall myocardium, with or without fatty replacement of tissue, in at least one endomyocardial-biopsy sample
Repolarization abnormalities	Inverted T waves in right precordial leads (V ₁ , V ₂ , and V ₃) or beyond in patients older than 14 yr of age (in the absence of complete right bundle-branch block, QRS ≥ 120 msec)	Inverted T waves in leads V ₁ and V ₂ in patients older than 14 yr of age (in the absence of complete right bundle-branch block) or in V ₄ , V ₅ , or V ₆ ; inverted T waves in leads V ₁ , V ₂ , V ₃ , and V ₄ in patients older than 14 yr of age (in the presence of complete right bundle-branch block)
Depolarization and conduction abnormalities	Epsilon wave (reproducible low-amplitude signals from end of QRS complex to onset of the T wave) in the right precordial leads (V ₁ , V ₂ , and V ₃)	Late potentials on signal-averaged ECG in at least one of three parameters in the absence of a QRS complex duration of ≥ 110 msec on the standard ECG; filtered QRS complex duration, ≥ 114 msec; duration of terminal QRS complex <40 μ V (low-amplitude signal duration), ≥ 38 msec; root-mean-square voltage of terminal 40 msec, ≤ 20 μ V; terminal activation duration of QRS complex, ≥ 55 msec, measured from the nadir of the S wave to the end of the QRS complex, including R', in V ₁ , V ₂ , or V ₃ , in the absence of complete right bundle-branch block
Arrhythmias	Nonsustained or sustained ventricular tachycardia with a left bundle-branch block and superior axis pattern (negative or indeterminate QRS complex in leads II, III, and aVF and positive QRS complex in lead aVL)	Nonsustained or sustained ventricular tachycardia of RV outflow configuration with a left bundle-branch block and inferior axis pattern (positive QRS complex in leads II, III, and aVF and negative QRS complex in lead aVL) or unknown axis, or >500 ventricular extrasystoles per 24 hr (on Holter monitoring)
Family history	ARVC confirmed in a first-degree relative who meets current task-force criteria, ARVC confirmed pathologically at autopsy or surgery in a first-degree relative, or identification of a pathogenic mutation categorized as associated or probably associated with ARVC in the patient under evaluation‡	History of ARVC in a first-degree relative in whom it is not possible or practical to determine whether current task-force criteria are met, premature sudden death (at <35 yr of age) due to suspected ARVC in a first-degree relative, or ARVC confirmed pathologically or by current task-force criteria in a second-degree relative

[‡] The table is adapted from Marcus et al.⁴⁹ The diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) is considered to be definite if the patient meets two major criteria, one major and two minor criteria, or four minor criteria from different categories; the diagnosis is considered to be borderline if the patient meets one major and one minor criteria or three minor criteria from different categories, and the diagnosis is classified as possible if the patient meets one major or two minor criteria from different categories. ECG denotes electrocardiogram, PLAX parasternal long-axis view, PSAX parasternal short-axis view, RV right ventricular, and RVOT RV outflow tract.

[†] Hypokinesia is not included in this or subsequent definitions of RV regional wall motion abnormalities for the proposed modified criteria.

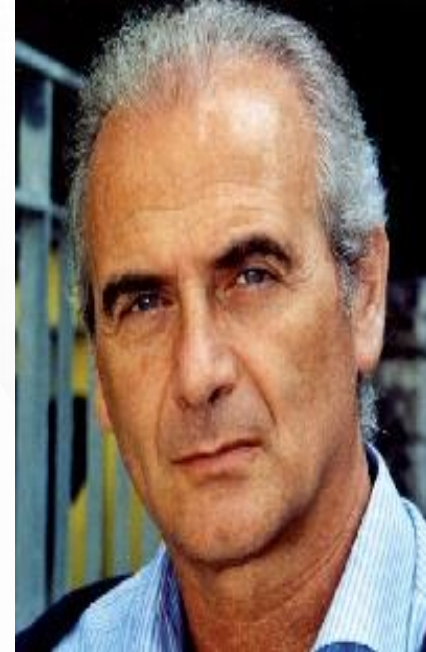
[‡] A pathogenic mutation is a DNA alteration associated with ARVC that alters or is expected to alter the encoded protein, is unobserved or rare in a large, non-ARVC control population, and either alters or is predicted to alter the structure or function of the protein or has shown linkage to the disease phenotype in a conclusive pedigree (i.e., a pedigree providing conclusive evidence of a mendelian inheritance of the disease phenotype).

REVIEW ARTICLE

John A. Jarcho, M.D., *Editor*

Arrhythmogenic Right Ventricular Cardiomyopathy

Domenico Corrado, M.D., Ph.D., Mark S. Link, M.D., and Hugh Calkins, M.D.



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Arrhythmogenic right ventricular
cardiomyopathy: evaluation of
diagnostic criteria and differential diagnosis
of
on evaluation,
anatomic differential
ventricular outflow tract ectopy from
arrhythmogenic right ventricular cardiomyopathy

SECTION EDITOR)

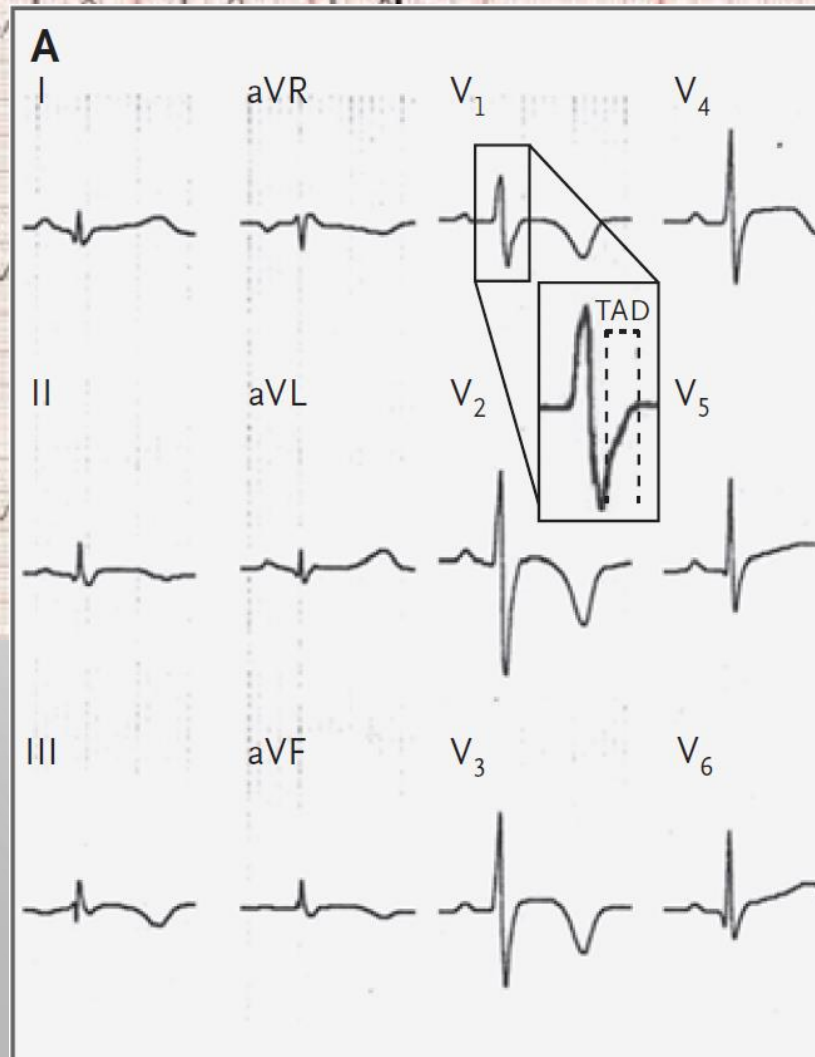
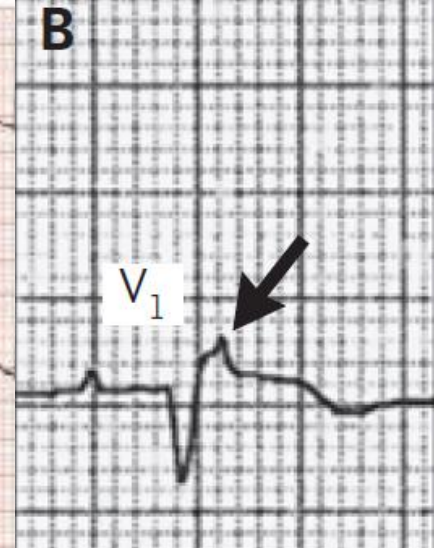
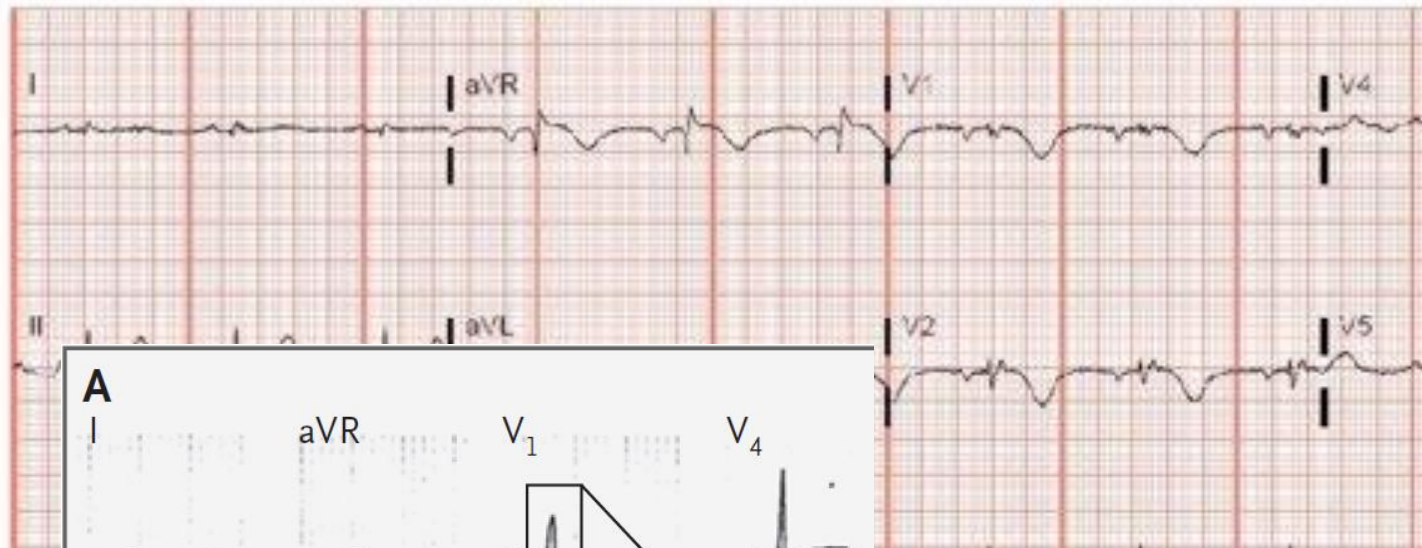


WHAT IS ARVC

- ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC), ALSO KNOWN AS ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA, IS A HERITABLE HEART MUSCLE DISORDER THAT PREDOMINANTLY AFFECTS THE RIGHT VENTRICLE.
- PROGRESSIVE LOSS OF RIGHT VENTRICULAR MYOCARDIUM AND ITS REPLACEMENT BY FIBROFATTY TISSUE IS THE PATHOLOGICAL HALLMARK OF THE DISEASE
- THE FIBROFATTY TISSUE THAT REPLACES MYOCARDIUM IN ARVC IS THOUGHT TO CONTRIBUTE TO THE DEVELOPMENT OF VENTRICULAR ARRHYTHMIAS BY SLOWING INTRAVENTRICULAR CONDUCTION AND ACTING AS A SUBSTRATE FOR ARRHYTHMIAS THROUGH A SCAR-RELATED MACROREENTRY MECHANISM, SIMILAR TO THAT OBSERVED AFTER MYOCARDIAL INFARCTION

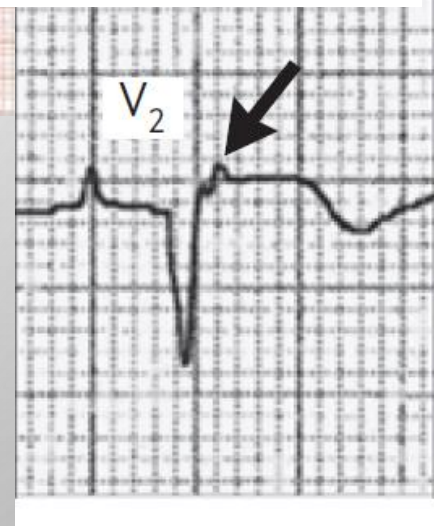
CARDIOMYOPATHY

- THE SUBSEQUENT DISCOVERY THAT THE DISEASE IS CAUSED BY A GENETIC DEFECT IN THE CARDIAC DESMOSOMES HAS LED TO ITS RECOGNITION AS A CARDIOMYOPATHY AND ITS INCLUSION IN THE CLASSIFICATION OF CARDIOMYOPATHIES BY THE AMERICAN HEART ASSOCIATION.
- THE PREVALENCE OF ARVC IS ESTIMATED TO RANGE FROM 1 CASE IN 5000 PERSONS IN THE GENERAL POPULATION TO 1 IN 2000 IN SOME EUROPEAN COUNTRIES SUCH AS ITALY AND GERMANY



The terminal activation duration (TAD), which is the interval between the nadir of the S wave and the end of all depolarization deflections, is prolonged, at 80 msec, in lead V1.....but not here

e inversion in V1-V3 fulfilling classic for ARVC



MORTALITY

THE NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

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Arrhythmogenic Right Ventricular Cardiomyopathy

Domenico Corrado, M.D., Ph.D., Mark S. Link, M.D., and Hugh Calkins, M.D.

Major Arrhythmic Events

Cardiac arrest due to ventricular
fibrillation

Sustained ventricular tachycardia

High Risk
>10%/yr

Major Risk Factors

Unexplained syncope

Nonsustained ventricular tachycardia

Severe right or left ventricular
dysfunction

Intermediate
Risk
1–10%/yr

Minor Risk Factors

Proband status, male sex

Frequent PVBs ($\geq 1000/24$ hr)

Inducibility on electrophysiological
study

Extent of negative T waves

Amount of right ventricular fibrofatty
scarring

Multiple desmosomal gene mutations

No Events or Risk Factors

Healthy gene carriers

Patients with definite ARVC

Low Risk
<1%/yr

WHAT NEXT

- IN GENERAL, PATIENTS ARE ENCOURAGED TO REFRAIN FROM VIGOROUS HIGH INTENSITY EXERCISE, BUT UP TO MODEST EXERCISE IS PROBABLY REASONABLE.

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<https://doi.org/10.1016/j.hlc.2018.10.013>

REVIEW

Arrhythmogenic Cardiomyopathy in 2018–2019: ARVC/ALVC or Both?



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LET THEM COME BACK TO THEIR FAMILY



THANK YOU FOR LISTENING



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