Erken Repolarizasyon

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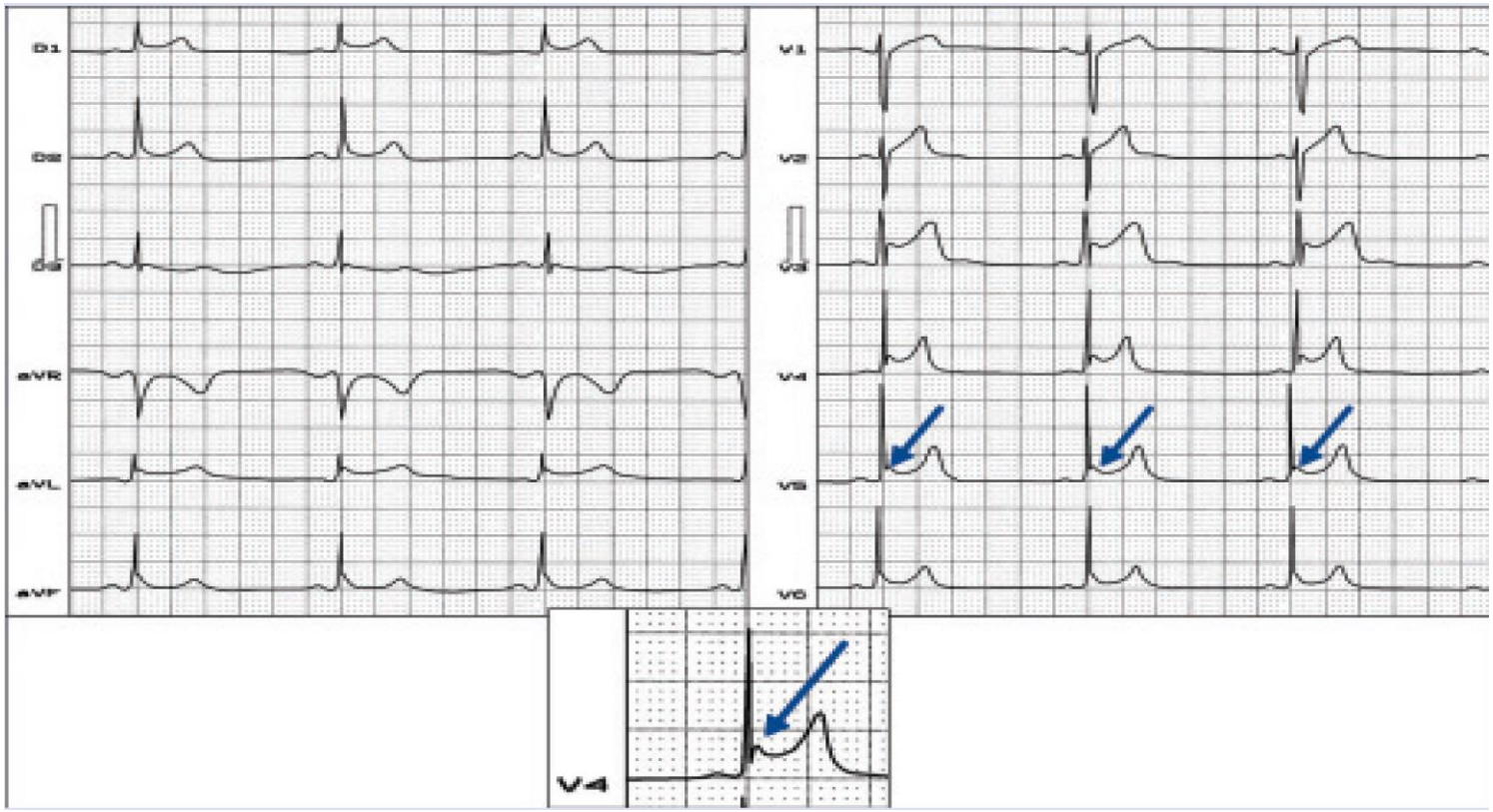
AFD atriyal fibrilasyon derneği
4. Atriyal Fibrilasyon Zirvesi 2015
10 - 11 Nisan 2015
Cornelia Hotel, Antalya

Erken Repolarizasyon (ER) ?

Inferiyor (D2, D3, aVF)- lateral (D1, aVL, V4-6)



ER: Patofizyoloji

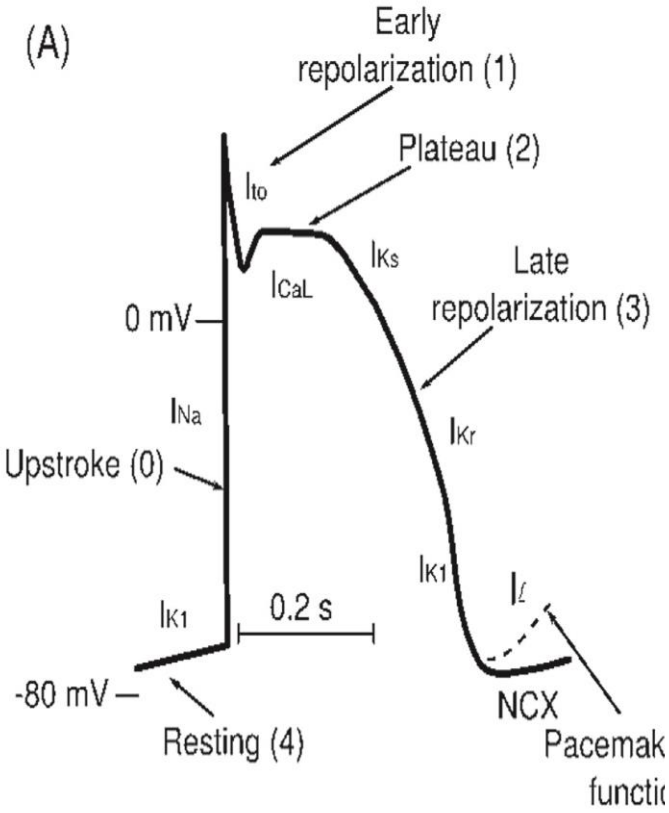


Fig. 2 – Ito responsible for ER.

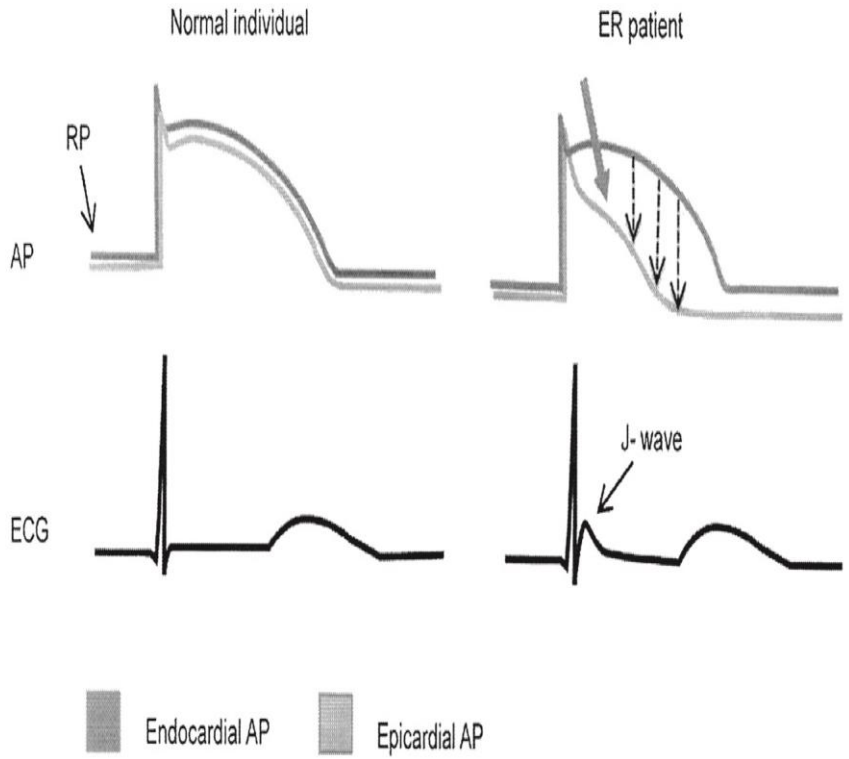


Fig. 1 – Possible mechanism of J wave genesis. Action potential in normal and J wave syndrome.

Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. Circulation. 1996;93:372-79.

Tarihçe: ER ilk tanımlayanlar (1936-1953)

Shipley, R. A., & Hallaran, W. R. (1936). The four lead electrocardiogram in 200 normal men and women. American Heart Journal, 11, 325–345.

→ **Benign**

Osborn, J. J. (1953). Experimental hypothermia; respiratory and blood pH changes in relation to cardiac function. American Journal of Physiology, 175(3), 389–398.

→ **Malign**

ER benigndir (1940-2000'ler)

Table 1 Summary of studies on the prognosis of the ER pattern

Year	Study design/cohort	Result	ER definition	Leads	Ref
1944	Retrospective 52 ER patients	**Benign prognosis** ER in 6%–25% of leads V_1–V_6; normal hearts on autopsy	RS-T junction ↑	V_1–V_6	51
1953	Retrospective 23 ER patients	**Benign prognosis** No events during 2-year follow-up	RS-T junction ↑	V_3–V_6	7
1961	Retrospective 48 ER patients	**Benign prognosis** No events over many months	RS-T junction ↑	II/III/aVF, V_3–V_6	8
1976	Retrospective 65 ER patients	**Benign prognosis** No events during 26-year follow-up	RS-T junction ↑	II/III/aVF , V_1–V_6	3
2003	Retrospective 680 ER patients 1081 controls	**Benign prognosis** Risk of death; ER vs no ER; HR 0.8 (95% CI 0.6–1.2)	RS-T junction ↑	II/III/aVF, V_1–V_6	1

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ER malignd ir (>2008)

2008	Case-control 206 idiopathic VF patients 412 controls	**Increased risk of SCD** ER prevalence; idiopathic VF patients vs controls; 31% vs 5% ($P < .001$)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	16
2008	Case-control 45 idiopathic VF patients 121 controls	**Increased risk of SCD** ER prevalence; idiopathic VF patients vs controls; 42% vs 13% ($P = .001$)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	17
2008	Case-control 15 idiopathic VF patients 1395 controls	**Increased risk of SCD** ER prevalence; idiopathic VF patients vs controls; 60% vs 3.3% ($P = .001$)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	18
2009	Retrospective community-based 10,864 subjects (630 with ER)	**Increased risk of SCD** Inferior ER (≥0.1 mV) (RR of SCD 1.43, 95% CI 1.06–1.94, $P = .03$). Inferior ER (≥0.2 mV) (RR of SCD 2.92, 95% CI 1.45–5.89, $P = .01$)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	2
2010	Retrospective case-cohort 6213 subjects (812 with ER)	**Increased risk of SCD** HR 1.96 (95% CI 1.05–3.68, $P = .035$); age 35–54 years Inferior ER: HR 3.15 (95% CI 1.58–6.28, $P = .001$)	0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	38
2011	Retrospective community-based 10,864 subjects (576 with ER)	**Increased risk of SCD** ER ≥0.1 mV and horizontal/descending ST; RR 1.43 (95% CI 1.05-1.94) ER ≥0.2 mV and horizontal/descending ST; RR 3.14 (95% CI 1.56-6.30)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	21
2011	Retrospective community-based 29,281 subjects (873 with ER)	**Benign prognosis** (RR of SCD 0.89, 95% CI 0.34–2.39)	≥0.1-mV ST ↑ 1 or 2 leads	II/III/aVF, V_3–V_6	25
2010	Retrospective community-based 5976 subjects (1429 with ER)	**Increased risk of SCD** HR 1.83 (95% CI 1.12–2.97, $P = .02$)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	52
2011	Case-control 45 idiopathic VF patients 124 controls	**Increased risk of SCD** J waves associated with idiopathic VF; OR 4.0 (95% CI 2.0-7.9) J waves with horizontal/descending ST; OR 13.8 (95% CI 5.1-37.2)	≥0.1-mV J-point ↑ ≥2 contiguous leads	II/III/aVF, V_3–V_6	40
2011	Retrospective community-based 15,141 subjects (1429 with ER)	**Increased risk of SCD** Whites; HR 2.03 (95% CI 1.28–3.21) Females; HR 2.54 (95% CI 1.34–4.82)	≥0.1-mV J-point ↑ in any 1 lead	II/III/aVF, aVF/aVR/aVL V_1–V_6	53
2011	Prospective 100 SCD patients	**Increased risk of SCD** ER prevalence; idiopathic VF vs explained SCD; 23% vs 14% Idiopathic VF group; higher amplitude and wider distribution of J waves	≥0.1-mV ST ↑ 1 or 2 leads	II/III/aVF, V_3–V_6	19

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ORIGINAL ARTICLE

Sudden Cardiac Arrest Associated with Early Repolarization

Michel Haïssaguerre, M.D., Nicolas Derval, M.D., Frederic Sacher, M.D.,

ABSTRACT

BACKGROUND

Early repolarization is a common electrocardiographic finding that is generally considered to be benign. Its potential to cause cardiac arrhythmias has been hypothesized from experimental studies, but it is not known whether there is a clinical association with sudden cardiac arrest.

METHODS

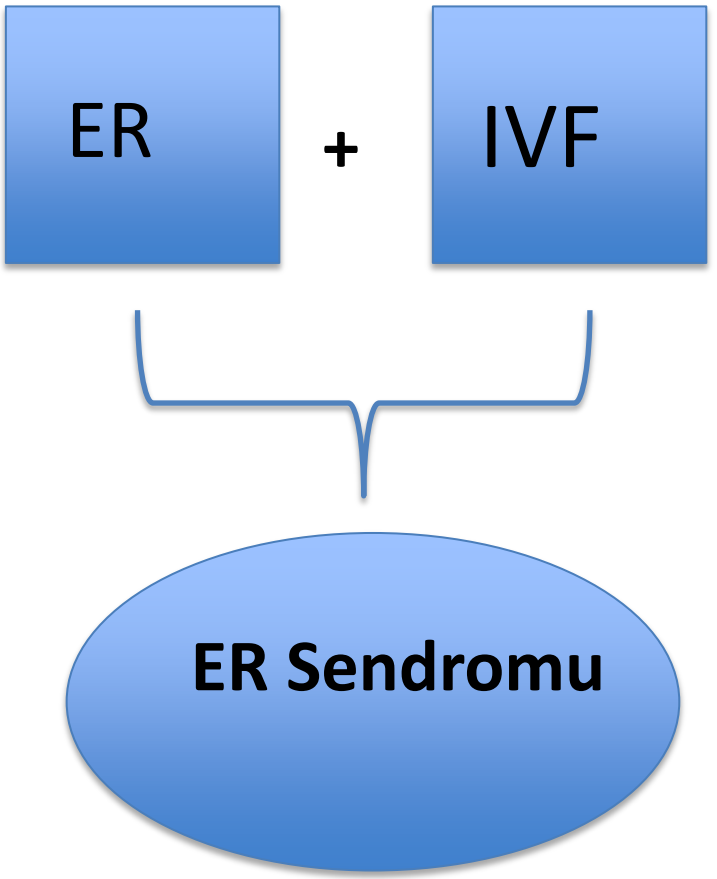
We reviewed data from 206 case subjects at 22 centers who were resuscitated after cardiac arrest due to idiopathic ventricular fibrillation and assessed the prevalence of electrocardiographic early repolarization. The latter was defined as an elevation of the QRS–ST junction of at least 0.1 mV from baseline in the inferior or lateral lead, manifested as QRS slurring or notching. The control group comprised 412 subjects without heart disease who were matched for age, sex, race, and level of physical activity. Follow-up data that included the results of monitoring with an implantable defibrillator were obtained for all case subjects.

RESULTS

Early repolarization was more frequent in case subjects with idiopathic ventricular fibrillation than in control subjects (31% vs. 5%, P<0.001). Among case subjects, those with early repolarization were more likely to be male and to have a history of syncope or sudden cardiac arrest during sleep than those without early repolarization. In eight subjects, the origin of ectopy that initiated ventricular arrhythmias was mapped to sites concordant with the localization of repolarization abnormalities. During a mean (±SD) follow-up of 61±50 months, defibrillator monitoring showed a higher incidence of recurrent ventricular fibrillation in case subjects with a repolarization abnormality than in those without such an abnormality (hazard ratio, 2.1; 95% confidence interval, 1.2 to 3.5; P=0.008).

CONCLUSIONS

Among patients with a history of idiopathic ventricular fibrillation, there is an increased prevalence of early repolarization.



Long-Term Outcome Associated with Early Repolarization on Electrocardiography

Jani T. Tikkanen, B.S., Olli Anttonen, M.D., M. Juhani Junttila, M.D., Aapo L. Aro, M.D., Tuomas Kerola, M.D., Harri A. Rissanen, M.Sc., Antti Reunanen, M.D., and Heikki V. Huikuri, M.D.

ABSTRACT

BACKGROUND

Early repolarization, which is characterized by an elevation of the QRS–ST junction (J point) in leads other than V_1 through V_3 on 12-lead electrocardiography, has been associated with vulnerability to ventricular fibrillation, but little is known about the prognostic significance of this pattern in the general population.

METHODS

We assessed the prevalence and prognostic significance of early repolarization on 12-lead electrocardiography in a community-based general population of 10,864 middle-aged subjects (mean [±SD] age, 44±8 years). The primary end point was death from cardiac causes, and secondary end points were death from any cause and death from arrhythmia during a mean follow-up of 30±11 years. Early repolarization was stratified according to the degree of J-point elevation (≥0.1 mV or >0.2 mV) in either inferior or lateral leads.

RESULTS

The early-repolarization pattern of 0.1 mV or more was present in 630 subjects (5.8%): 384 (3.5%) in inferior leads and 262 (2.4%) in lateral leads, with elevations in both leads in 16 subjects (0.1%). J-point elevation of at least 0.1 mV in inferior leads was associated with an increased risk of death from cardiac causes (adjusted relative risk, 1.28; 95% confidence interval [CI], 1.04 to 1.59; P=0.03); 36 subjects (0.3%) with J-point elevation of more than 0.2 mV in inferior leads had a markedly elevated risk of death from cardiac causes (adjusted relative risk, 2.98; 95% CI, 1.85 to 4.92; P<0.001) and from arrhythmia (adjusted relative risk, 2.92; 95% CI, 1.45 to 5.89; P=0.01). Other electrocardiographic risk markers, such as a prolonged QT interval corrected for heart rate (P=0.03) and left ventricular hypertrophy (P=0.004), were weaker predictors of the primary end point.

CONCLUSIONS

An early-repolarization pattern in the inferior leads of a standard electrocardiogram is associated with an increased risk of death from cardiac causes in middle-aged subjects.

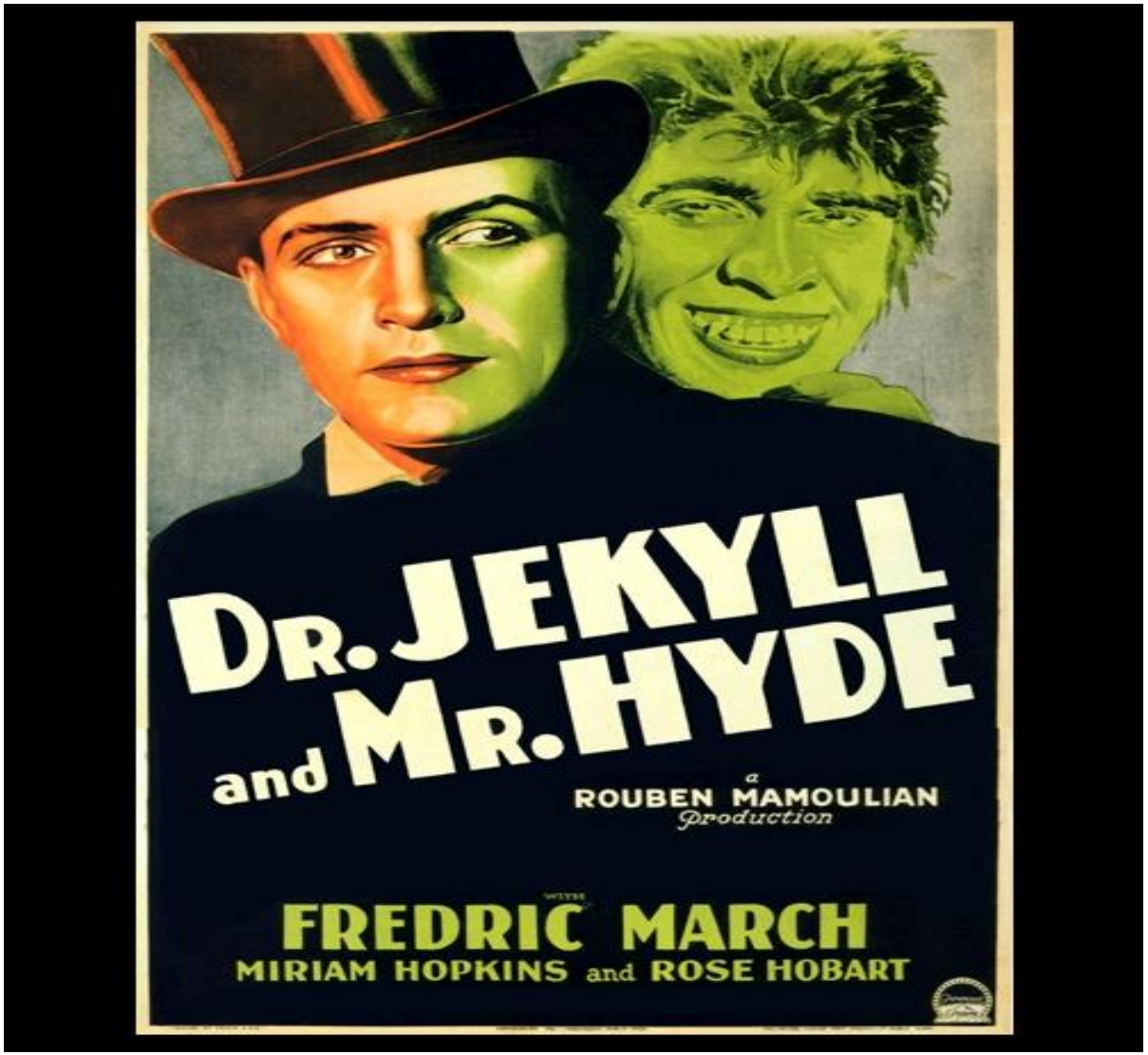
FİN TOPLUMUNDA ER PATERNİ %9 ‘DUR.

İNFERİYOR ER, ORTA YAŞ GRUBUNDA KARDİYAK ÖLÜMLE İLİŞKİLİDİR.

ER: İnsidans

- Genel popülasyonda %4-13
- Atletlerde %20-40
- AMİ ile ilişkili VF %30-45
- İdiyopatik VF %15-70

Benign ER? Malign ER?



ER risk değerlendirmesi

- Klinik
  - Aile öyküsü ve senkop
  - Ani ölümden kurtulma ve/veya rekürren VF
  - Diğer aritmilerle birliktelik
- Laboratuvar
  - 12 derivasyon EKG ve Holter EKG
  - Provakatif testler
  - Konvansiyonel EPS
  - Yeni elektroanatomik yöntemler

Aile öyküsü ve ER

Prevalence of J-Point Elevation in Sudden Arrhythmic Death Syndrome Families

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Peter W. Macfarlane, MD, DSC,† Pauline Rogers, MSC,‡ William J. McKenna, MD,*
Perry M. Elliott, MD,* Pier D. Lambiase, MD, PHD*

London, England; and Glasgow, Scotland

Objectives The purpose of this study was to assess the prevalence of J-point elevation among the relatives of sudden arrhythmic death syndrome (SADS) probands.

Background J-point elevation is now known to be associated with idiopathic ventricular fibrillation. We hypothesized that this early repolarization phenomenon is an inherited trait responsible for a proportion of otherwise unexplained SADS cases.

Methods Families of SADS probands were evaluated in an inherited arrhythmia clinic. Twelve-lead electrocardiograms were analyzed for J-point elevation defined as >0.1 mV from baseline present in 2 or more of the inferior (II, III, and aVF) or lateral (I, aVL, V_4 to V_6) leads. Electrocardiographic data were compared with those of 359 controls of a similar age, sex, and ethnic distribution.

Results A total of 363 first-degree relatives from 144 families were evaluated. J-point elevation in the inferolateral leads was present in 23% of relatives and 11% of control subjects (odds ratio: 2.54, 95% confidence interval: 1.66 to 3.90; $p < 0.001$).

Conclusions J-point elevation is more prevalent in the relatives of SADS probands than in controls. This indicates that early repolarization is an important potentially inheritable pro-arrhythmic trait or marker of pro-arrhythmia in SADS.
(J Am Coll Cardiol 2011;58:286–90)

ER PATERNİ AKÖ GEÇİRENLERİN YAKINLARINDA SAĞLIKLI KONTROLLERE GÖRE ANLAMLI YÜKSEK BULUNMUŞTUR (%23'e karşı %11)

Aile öyküsü-senkop ve ER

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Sudden Cardiac Arrest Associated with Early Repolarization

Michel Haïssaguerre, M.D., Nicolas Derval, M.D., Frederic Sacher, M.D.,

Table 1. Characteristics of the Case Subjects.*

Characteristic	Early Repolarization (N=64)	No Early Repolarization (N=142)	P Value
Demographic and clinical			
Male sex — no. (%)	46 (72)	76 (54)	0.007
Age — yr	35±13	37±13	0.49
Race or ethnic group — no.†			0.69
White	58	132	
Asian	5	9	
Black	1	1	
History of unexplained syncope — no. (%)	24 (38)	35 (25)	0.06
Family history of unexplained sudden death — no. (%)	10 (16)	13 (9)	0.17
Physical activity — no. (%)‡	4 (6)	18 (13)	0.11
Activity at the time of initial sudden cardiac arrest — no. (%)			
Sleeping	12 (19)	6 (4)	0.03
Physical effort	6 (9)	19 (13)	
Other activity	46 (72)	117 (82)	

Rekürren VF

Table 2. Outcome after Initial Aborted Sudden Cardiac Arrest.*

Variable	Early Repolarization (N=64)	No Early Repolarization (N=142)	P Value
Duration of follow-up (mo)			0.81
Mean	60±45	62±52	
Median	49	54	
Interquartile range	24–90	17–92	
No. of recurrent episodes of ventricular fibrillation per patient			0.001
Median	8	2	
Interquartile range	2–35	1–6	
Successful treatment (no./total no.)†			
Beta-blockers	2/13	9/17	
Amiodarone	0/7	3/7	
Flecainide, cifenline, or pilsicainide	0/10	2/4	
Quinidine or disopyramide	4/4	1/3	
Verapamil	0/5	3/8	
Mexiletine	0/5	0/2	
Catheter ablation	5/8	6/7	
Current outcome			
No. of subjects alive	63	142	
No. of subjects with recurrence in the past 12 mo	5‡	5	

* Plus–minus values are means ±SD.
† Successful treatment was defined as no ventricular fibrillation for at least 12 months, as documented by an implantable defibrillator.
‡ Quinidine was recently prescribed for three subjects.

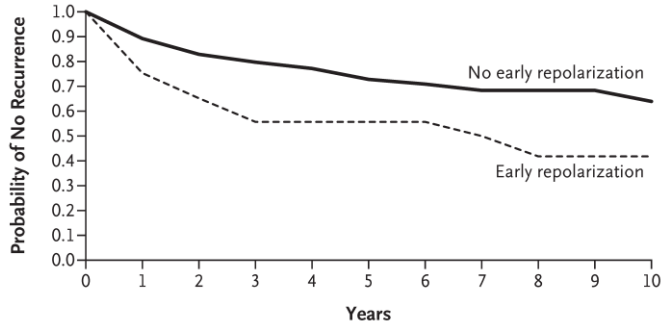


Figure 3. Actuarial Curves for Case Subjects, According to the Presence or Absence of Early Repolarization.

Case subjects with a repolarization abnormality were at increased risk for recurrent ventricular fibrillation, as compared with those without such an abnormality (hazard ratio, 2.1; 95% CI, 1.2 to 3.5; P=0.008).

TEKRARLAYAN VF ATAKLARI ERS'DA SIK GÖRÜLMEKTEDİR

Brugada Sendromu +ER: kötü prognoz

Electrocardiographic Parameters and Fatal Arrhythmic Events in Patients With Brugada Syndrome

Combination of Depolarization and Repolarization Abnormalities

Koji Tokioka, MD,* Kengo F. Kusano, MD, PHD,† Hiroshi Morita, MD, PHD,* Daiji Miura, PHD,* Nobuhiro Nishii, MD, PHD,* Satoshi Nagase, MD, PHD,* Kazufumi Nakamura, MD, PHD,* Kunihisa Kohno, MD, PHD,* Hiroshi Ito, MD, PHD,* Tohru Ohe, MD, PHD‡

Okayama and Osaka, Japan

Objectives This study aimed to determine the usefulness of the combination of several electrocardiographic markers on risk assessment of ventricular fibrillation (VF) in patients with Brugada syndrome (BrS).

Background Detection of high-/low-risk BrS patients using a noninvasive method is an important issue in the clinical setting. Several electrocardiographic markers related to depolarization and repolarization abnormalities have been reported, but the relationship and usefulness of these parameters in VF events are unclear.

Methods Baseline characteristics of 246 consecutive patients (236 men; mean age, 47.6 ± 13.6 years) with a Brugada-type electrocardiogram, including 13 patients with a history of VF and 40 patients with a history of syncope episodes, were retrospectively analyzed. During the mean follow-up period of 45.1 months, VF in 23 patients and sudden cardiac death (SCD) in 1 patient were observed. Clinical/genetic and electrocardiographic parameters were compared with VF/SCD events.

Results On univariate analysis, a history of VF and syncope episodes, paroxysmal atrial fibrillation, spontaneous type 1 pattern in the precordial leads, and electrocardiographic markers of depolarization abnormalities (QRS duration ≥120 ms, and fragmented QRS [f-QRS]) and those of repolarization abnormalities (inferolateral early repolarization [ER] pattern and QT prolongation) were associated with later cardiac events. On multivariable analysis, a history of VF and syncope episodes, inferolateral ER pattern, and f-QRS were independent predictors of documented VF and SCD (odds ratios: 19.61, 28.57, 2.87, and 5.21, respectively; $p < 0.05$). Kaplan-Meier curves showed that the presence/absence of inferolateral ER and f-QRS predicted a worse/better prognosis (log-rank test, $p < 0.01$).

Conclusions The combination of depolarization and repolarization abnormalities in BrS is associated with later VF events. The combination of these abnormalities is useful for detecting high- and low-risk BrS patients. (J Am Coll Cardiol 2014;63:2131–8)

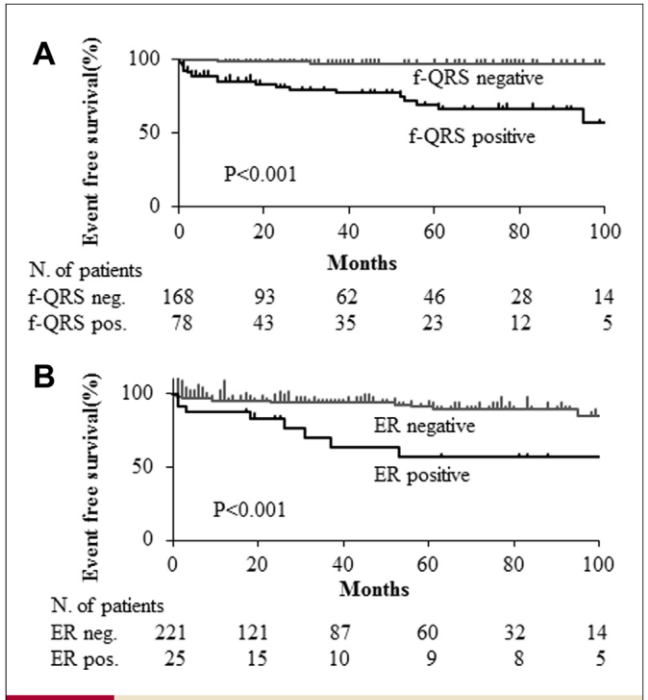


Figure 2 Kaplan-Meier Analysis of VF/SCD Events

Ventricular fibrillation (VF)/sudden cardiac death (SCD) were observed often in the presence of fragmented QRS (f-QRS) **(A)**, and early repolarization (ER) **(B)**.

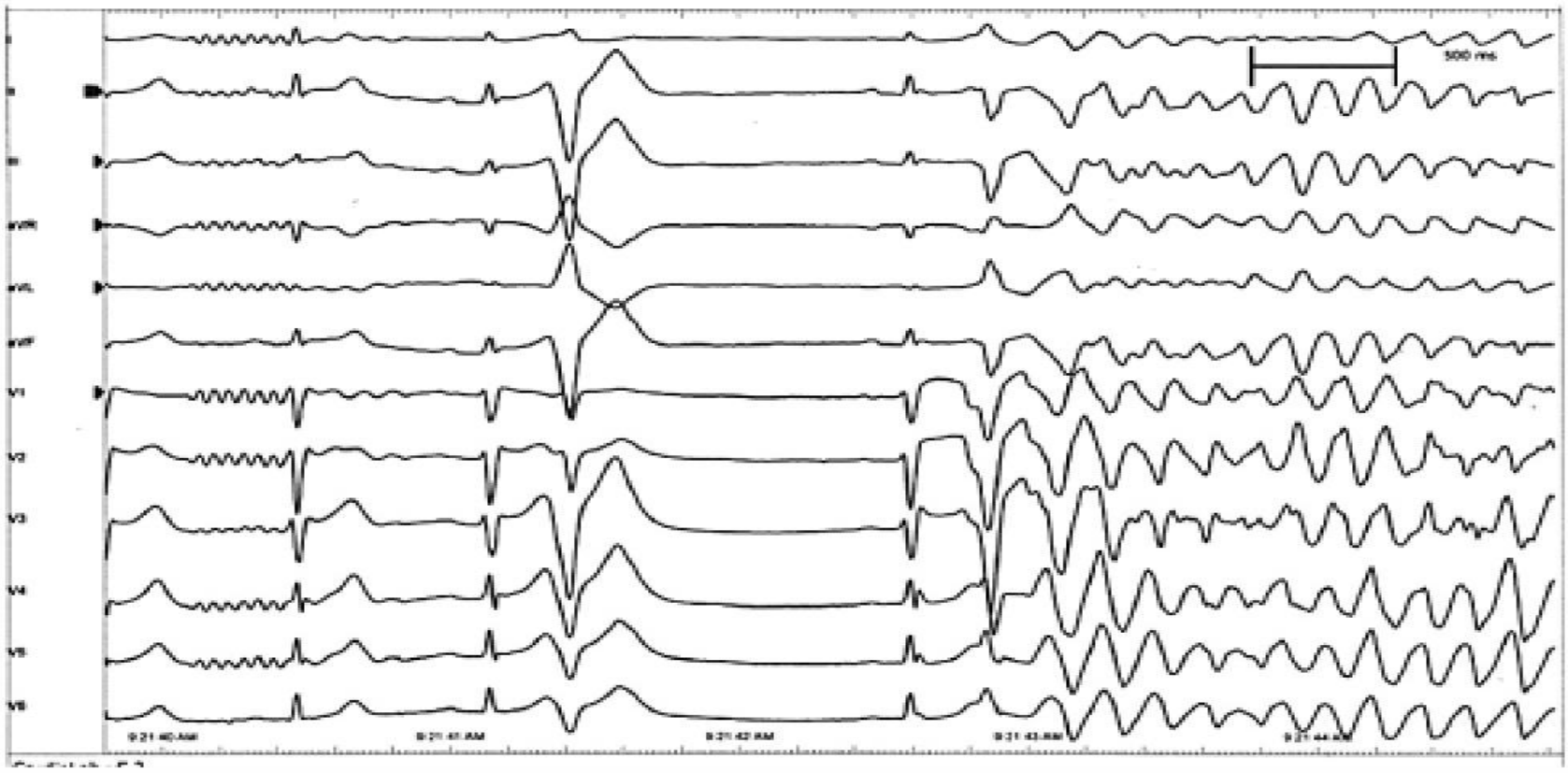
EKG (ER: J dalgası lokalizasyonu)

Table 3 Incidence Rates and Risks for Arrhythmia Death, Cardiac Death, and All-Cause Death in Subjects With ERP During Follow-Up

	Events per Person-Years			
	ERP Positive	ERP Negative	RR (95% CI)	p Value
Primary end point				
All-cause death	1,899/111,331	20,503/1,771,583	1.06 (0.85–1.31)	0.62
Cardiac death	387/80,388	9,570/1,747,749	0.78 (0.27–2.21)	0.63
Arrhythmia death	142/84,346	966/640,004	1.70 (1.19–2.42)	0.003
Subgroup study: J-point elevation				
≥0.1 mV in inferior leads	90/22,170	—	1.72 (1.39–2.13)	<0.00001
≥0.2 mV in inferior leads	16/2,196	—	3.06 (1.87–4.99)	<0.00001
≥0.1 mV in lateral leads	31/16,106	—	0.8 (0.56–1.14)	0.22
Configuration				
Notching	38/25,852	—	1.54 (1.11–2.15)	0.01
Slurring	31/12,483	—	1.36 (0.95–1.94)	0.09

CI = confidence interval; ERP = early repolarization pattern; RR = risk ratio.

Kısa coupled intervalli VES ve Dinamik J dalgası VF'e neden oluyor



J dalgası ve ST yükselmesi mortaliteyi artırıyor

Distinguishing "benign" from "malignant early repolarization": The value of the ST-segment morphology

Raphael Rosso, MD,*‡ Eran Glikson,*‡ Bernard Belhassen, MD,* Amos Katz, MD,† Amir Halkin, MD,* Arie Steinvil, MD,* Sami Viskin, MD*

*From the *Department of Cardiology, Tel Aviv Sourasky Medical Center and, Sackler School of Medicine, Tel Aviv University, Tel Aviv, †Department of Cardiology, Barzilai Hospital, Ashkelon, and Ben Gurion University of the Negev, Beer-Sheva, Israel.*

BACKGROUND Means for distinguishing the very common "benign early repolarization" from the very rare but malignant form are needed. Recently, the presence of early repolarization with "horizontal ST segment" was found to predict arrhythmic death during long-term follow-up in a large population study. We therefore speculated that the combination of "J waves with horizontal ST segment" would correlate with a history of idiopathic ventricular fibrillation (VF) better than the mere presence of J waves.

OBJECTIVES To determine whether the morphology of the ST segment adds diagnostic value to the mere presence of J waves in a case–control series of idiopathic VF.

as either "horizontal" or "ascending" according to predefined criteria.

RESULTS The presence of J waves was associated with a history of idiopathic VF with an odds ratio of 4.0 (95% confidence intervals = 2.0–7.9), but having both J waves and horizontal ST segment yielded an odds ratio of 13.8 (95% confidence intervals = 5.1–37.2) for having idiopathic VF.

CONCLUSIONS We report, for the first time, that the combination of J waves with horizontal/descending ST segment improved our ability to distinguish patients with idiopathic VF from controls matched by gender and age.

Table 1 Clinical and electrocardiographic characteristics of patients with idiopathic ventricular fibrillation and controls matched for age and gender

	No J waves (n = 133)	J wave (n = 35)	*P* value	Ascending ST (n = 18)	Horizontal ST (n = 17)	*P* value
Age (y)	39 ± 13	33 ± 15	**.024**	31 ± 17	36 ± 14	.359
RR (ms)	866 ± 140	888 ± 149	.423	890 ± 132	885 ± 170	.928
QT (ms)	355 ± 28	355 ± 26	.904	355 ± 24	355 ± 29	.993
Corrected QT interval (ms)	384 ± 25	379 ± 26	.297	378 ± 31	380 ± 20	.887
Male gender	90 (68%)	31 (89%)	**.019**	17 (94)	14 (82)	.338
Idiopathic ventricular fibrillation	26 (20%)	19 (54%)	**<.001**	6 (33)	13 (77)	**.018**

Horizontal ST, hızlı yükselen ST elevasyonuna göre daha mortaldir

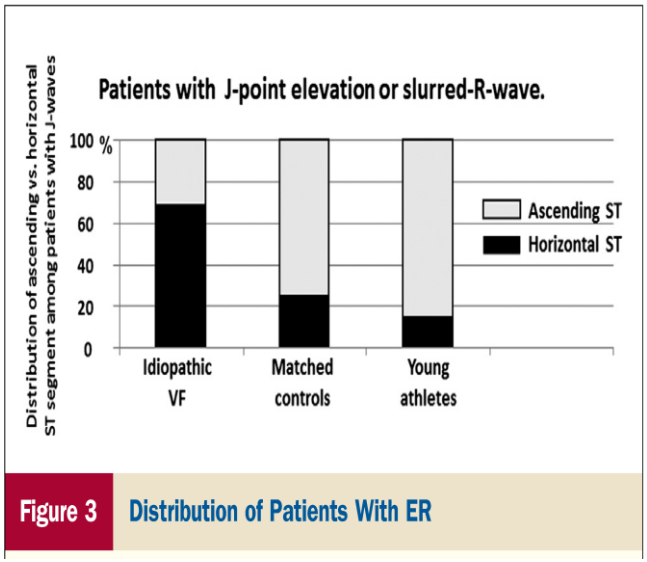


Figure 3 Distribution of Patients With ER

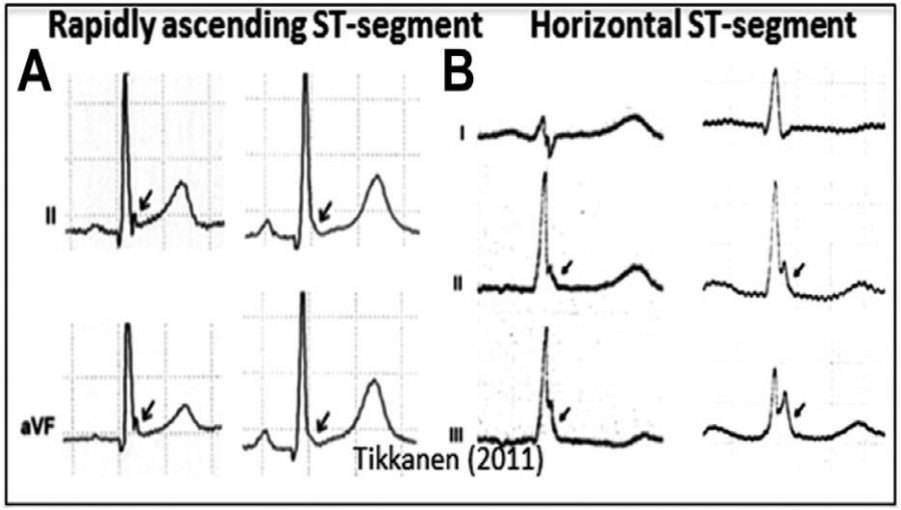


Figure 2 Electrocardiogram of a male patient with idiopathic ventricular fibrillation with J waves in the inferior leads. Leads II, III, and aVF are also enlarged to show the horizontal/descending ST segment.

Horizontal ST yükselmesi VF ile ilişkilidir

JACC Vol. 62, No. 10, 2013
September 3, 2013:863–8

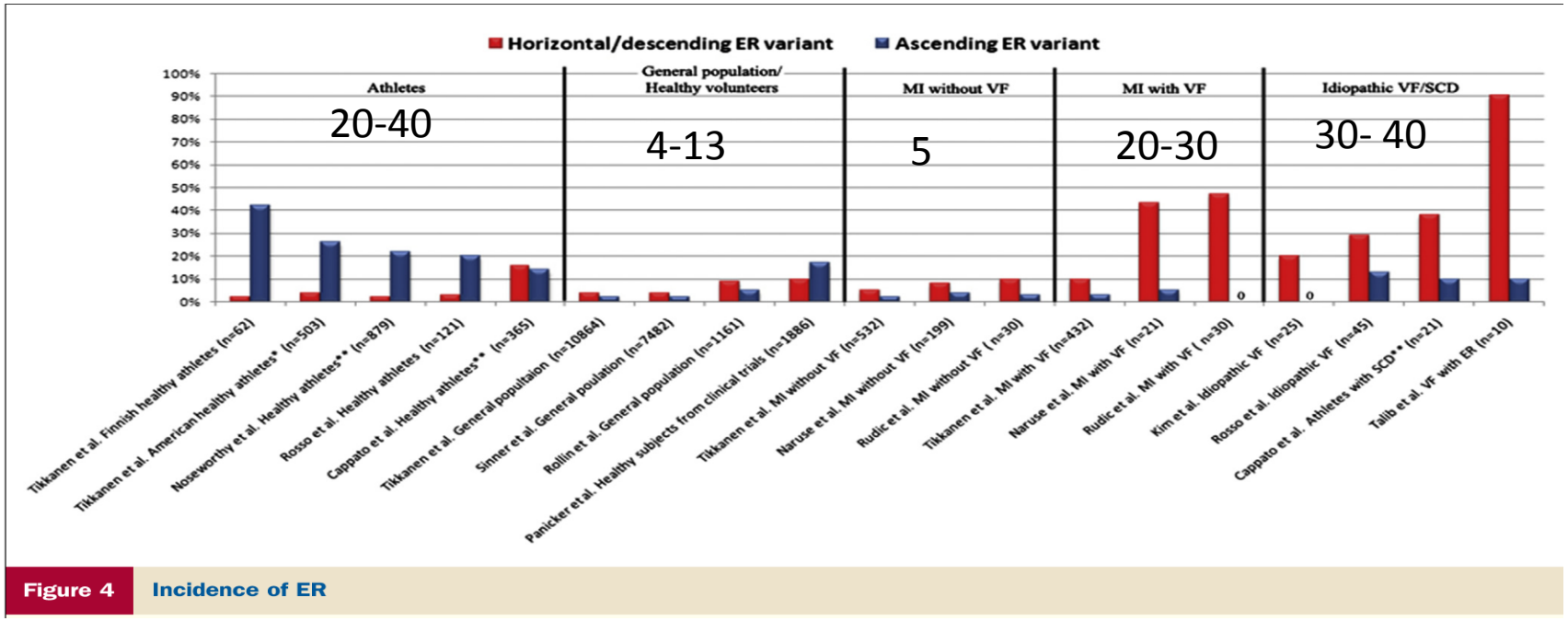


Figure 4 Incidence of ER

Adler et al. J Am Coll Cardiol 2013;62:863–8

Provakatif testler bir ipucu vermiyor

History and clinical significance of early repolarization syndrome

Saagar Mahida, MBChB, Nicolas Derval, MD, Frederic Sacher, MD, Benjamin Berte, MD, Seigo Yamashita, MD, PhD, Darren A. Hooks, MD, PhD, Arnaud Denis, MD, Han Lim, MBBS, PhD, Sana Amraoui, MD, Nora Aljefairi, MD, Meleze Hocini, MD, Pierre Jais, MD, Michel Haissaguerre, MD

Provocative testing currently has no role in risk stratification of ER syndrome. In contrast to Brugada syndrome, which as discussed previously displays significant overlap with ER syndrome, sodium channel blockade results in a paradoxical attenuation of J-point elevation in ER syndrome patients.[43,44] A number of additional drugs also have been tested as potential provocative agents, including verapamil, epinephrine, ATP, cibenzoline, and pilsicainide, and have been reported to have a minimal effect on the degree of J-point elevation.[45] Finally, although the role of programmed

ER Sendromu risk değerlendirmesinde PES

Role of Electrophysiological Studies in Predicting Risk of Ventricular Arrhythmia in Early Repolarization Syndrome

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ABSTRACT

BACKGROUND The early repolarization (ER) pattern is associated with an increased risk of arrhythmogenic sudden death. However, strategies for risk stratification of patients with the ER pattern are not fully defined.

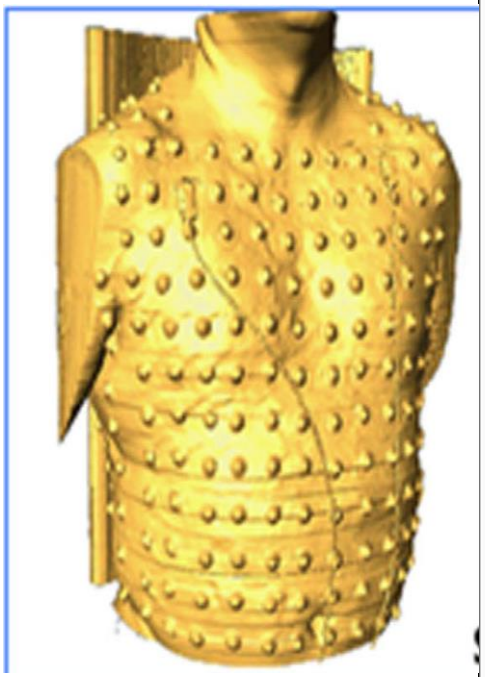
OBJECTIVES This study sought to determine the role of electrophysiology studies (EPS) in risk stratification of patients with ER syndrome.

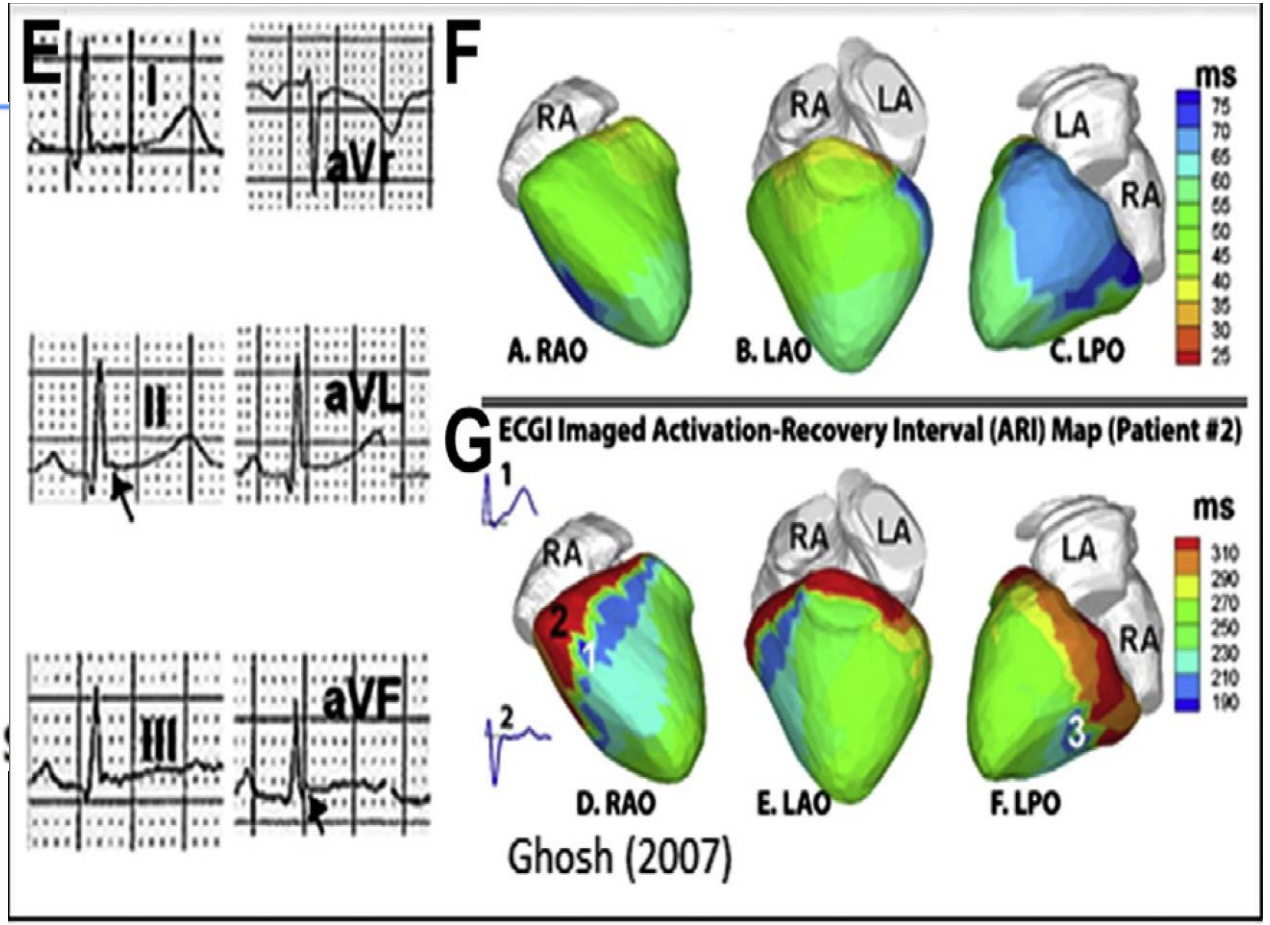
METHODS In a multicenter study, 81 patients with ER syndrome (age 36 ± 13 years, 60 males) and aborted sudden death due to ventricular fibrillation (VF) were included. EPS were performed following the index VF episode using a standard protocol. Inducibility was defined by the provocation of sustained VF. Patients were followed up by serial implantable cardioverter-defibrillator interrogations.

RESULTS Despite a recent history of aborted sudden death, VF was inducible in only 18 of 81 (22%) patients. During follow-up of 7.0 ± 4.9 years, 6 of 18 (33%) patients with inducible VF during EPS experienced VF recurrences, whereas 21 of 63 (33%) patients who were noninducible experienced recurrent VF ($p = 0.93$). VF storm occurred in 3 patients from the inducible VF group and in 4 patients in the noninducible group. VF inducibility was not associated with maximum J-wave amplitude (VF inducible vs. VF noninducible; 0.23 ± 0.11 mV vs. 0.21 ± 0.11 mV; $p = 0.42$) or J-wave distribution (inferior, odds ratio [OR]: 0.96 [95% confidence interval (CI): 0.33 to 2.81]; $p = 0.95$; lateral, OR: 1.57 [95% CI: 0.35 to 7.04]; $p = 0.56$; inferior and lateral, OR: 0.83 [95% CI: 0.27 to 2.55]; $p = 0.74$), which have previously been demonstrated to predict outcome in patients with an ER pattern.

CONCLUSIONS Our findings indicate that current programmed stimulation protocols do not enhance risk stratification in ER syndrome. (J Am Coll Cardiol 2015;65:151–9) © 2015 by the American College of Cardiology Foundation.

ER risk değerlendirmesinde yeni yöntemler (ECGI)





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ER risk değerlendirmesinde yeni yöntemler (MAP)

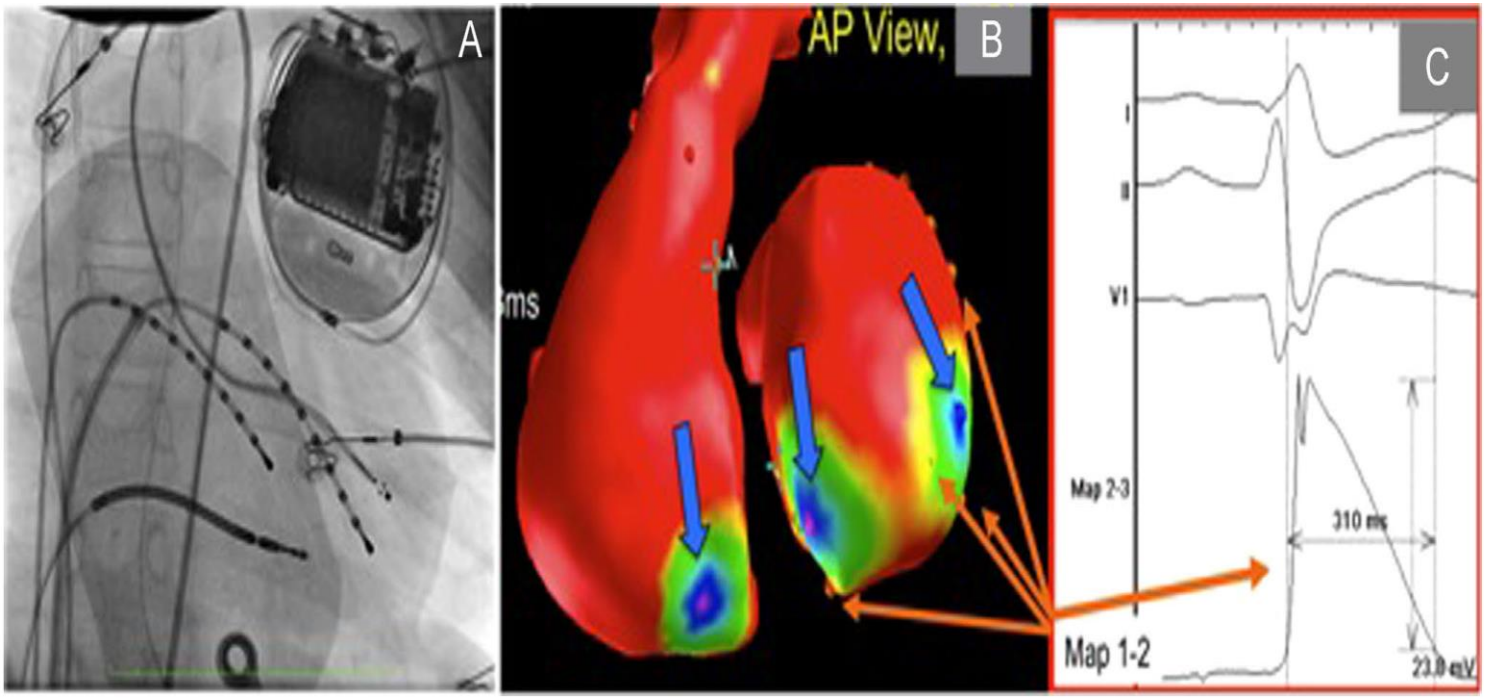

Figure 4. Phenotyping using catheters equipped with multiple monophasic action potential (MAP) electrodes. **A:** Radiographic image of a MAP catheter. **B:** Action potential map of a patient with early repolarization syndrome. The blue arrows point to areas of pronounced action potential notching. AP = anteroposterior. **C:** Example of a MAP recorded from the epicardium of a patient with early repolarization syndrome. The action potential demonstrates a prominent notch. (Courtesy of M. Hocini, F. Sacher, and K. Nademanee.)

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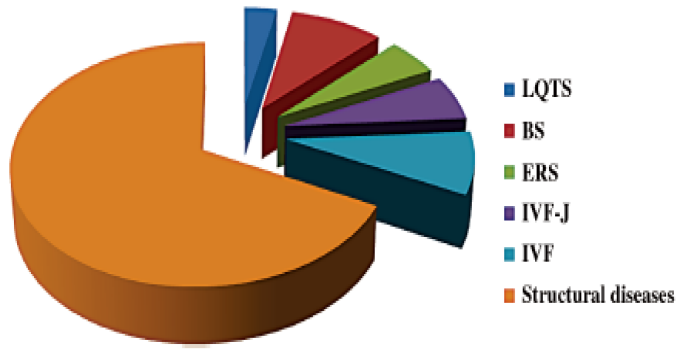
Ani ölüm ve ER

Underlying diseases	Number	Percent
Long QT syndrome (LQTS)	10	3.2%
Brugada syndrome (BS)	29	9.4%
Early repolarization syndrome (ERS)	16	5.2%
J wave-related VF in the absence of structural heart diseases (IVF-J)	19	6.1%
Idiopathic ventricular fibrillation (IVF)	26	8.4%
Myocardial infarction or coronary artery disease	79	25.6%
Variant angina	10	3.2%
Dilated cardiomyopathy	33	10.7%
Hypertrophic cardiomyopathy	45	14.6%
Arrhythmogenic right ventricular cardiomyopathy	13	4.2%
Others (Sarcoidosis, valve, congenital diseases, etc)	29	9.4%
Total	309	100.0%



NAM GB Circ J 2012; 76: 2723 – 2731

ER toplumda % 4-13

IVF'lerin %15-70 nedeni ER

IVF Tüm AKÖ'lerin %3.5

AKÖ içinde ER %0.3-2.0

Adler et al. J Am Coll Cardiol 2013;62:863–8

ER: Risk piramidi

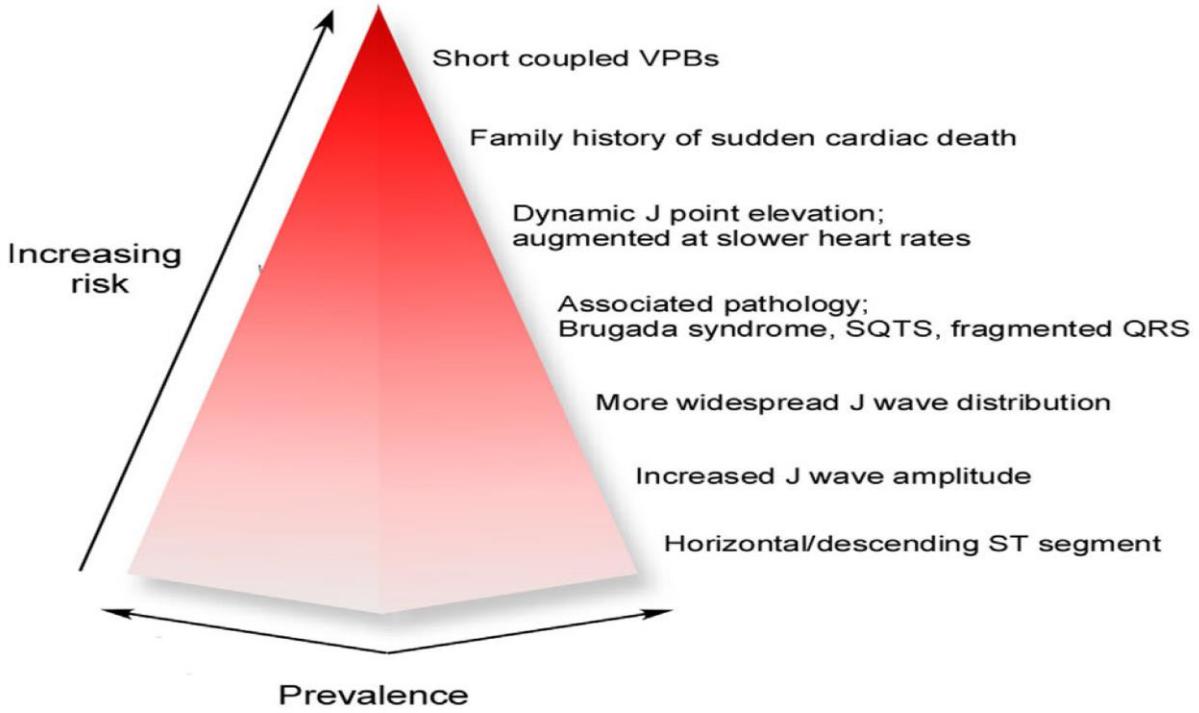


Figure 1 Risk stratification of patients with early repolarization. The highest risk corresponds to the *top* of the pyramid whereas the lowest risk is at the *bottom*. The estimated prevalence of the risk factor corresponds to the width of the pyramid. SQTS = short QT syndrome; VPBs = ventricular premature beats. (Modified with permission from Junttila et al. Eur Heart J 2012; Nov;33 (21):2639-43. doi: 10.1093/eurheartj/ehs110. Epub 2012 May 29.)

ER: Tedavi

11.1. *Treatment*

Following are recommendations from latest Consensus document of HRS/ACC/ESC.[22]

11.2. *Class I*

1. ICD implantation is recommended in patients with a diagnosis of ER syndrome who have survived a cardiac arrest.

11.3. *Class II a*

1. Isoproterenol infusion can be useful in suppressing electrical/VT storms in patients with diagnosis of ER syndrome.
2. Quinidine in addition to an ICD can be useful for secondary prevention and suppression of VT/VF in patients with a diagnosis of ER syndrome.

11.4. *Class II b*

1. ICD implantation may be considered in symptomatic family members of ER syndrome, with history of syncope in the presence of ST segment elevation >1 mm in 2 or more inferior or lateral leads.
2. ICD implantation may be considered in asymptomatic individuals who demonstrate a high-risk ER ECG pattern (high J-wave amplitude, horizontal/descending ST) in infero-posterior leads the presence of a strong family history of juvenile unexplained sudden death with or without a pathogenic mutation.

11.5. *Class III*

ICD implantation is not recommended in asymptomatic patients with an isolated ER pattern on ECG.

HRS/EHRA/APHRS Expert Consensus Statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. Heart Rhythm. 2013;Vol 10:No.10.

Teşekkür ederim