Síndromes da onda J

Síndromes de Repolarização Precoce

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J wave syndromes (JWS) – BrS e ERS

ERS: Doença elétrica primária do coração caracterizada por um padrão de repolarização precoce no ECG e risco aumentado de FV

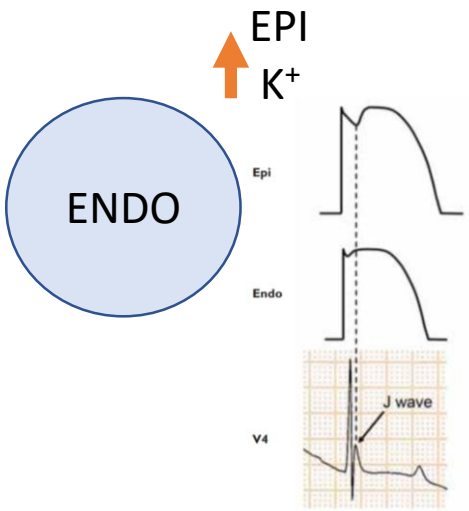
- BrS (Pedro e Josep Brugada, 1992)
- BrS Consensus Conference – 2000 e 2004
- **ERS – 2008**
- Consensus Conference – 2013
- Task Force- 2015
- Consensus Conference - 2017

Ondas J = early repolarization pattern (ERP)

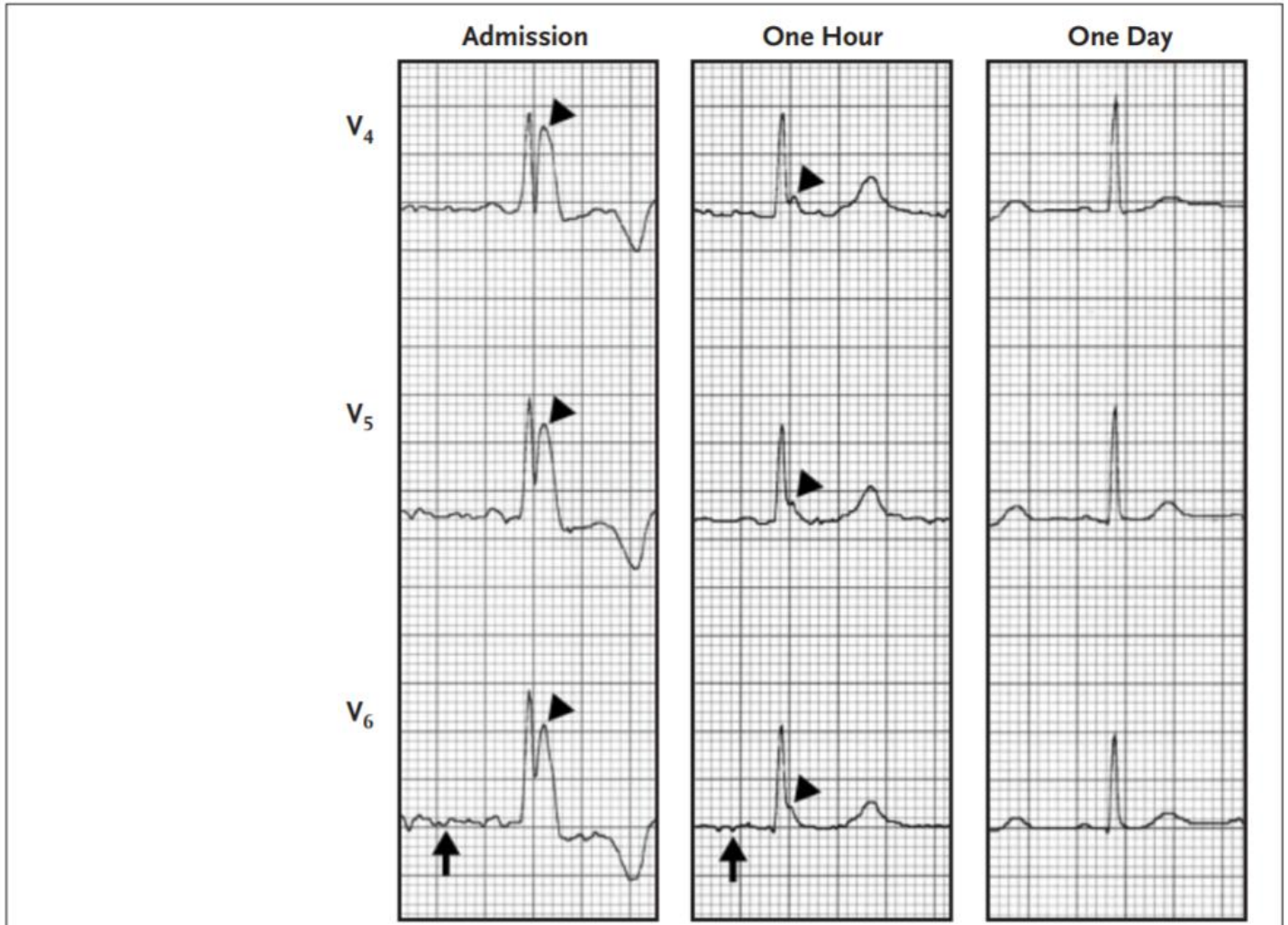
- Padrão ST juvenil
- Hipotermia profunda
- Hipertermia
- Tumor miocárdico
- Coração de atleta
- Hipocalcemia
- Doença pericárdica
- Isquemia miocárdica
- QRS fragmentado
- Hiperpotassemia
- Timoma
- ARVD
- Miocardite
- Tako-Tsubo
- Cocaína
- Hemorragia intracraniana

Giant Osborn Waves in Hypothermia

John Osborn, 1953
"Injury current"



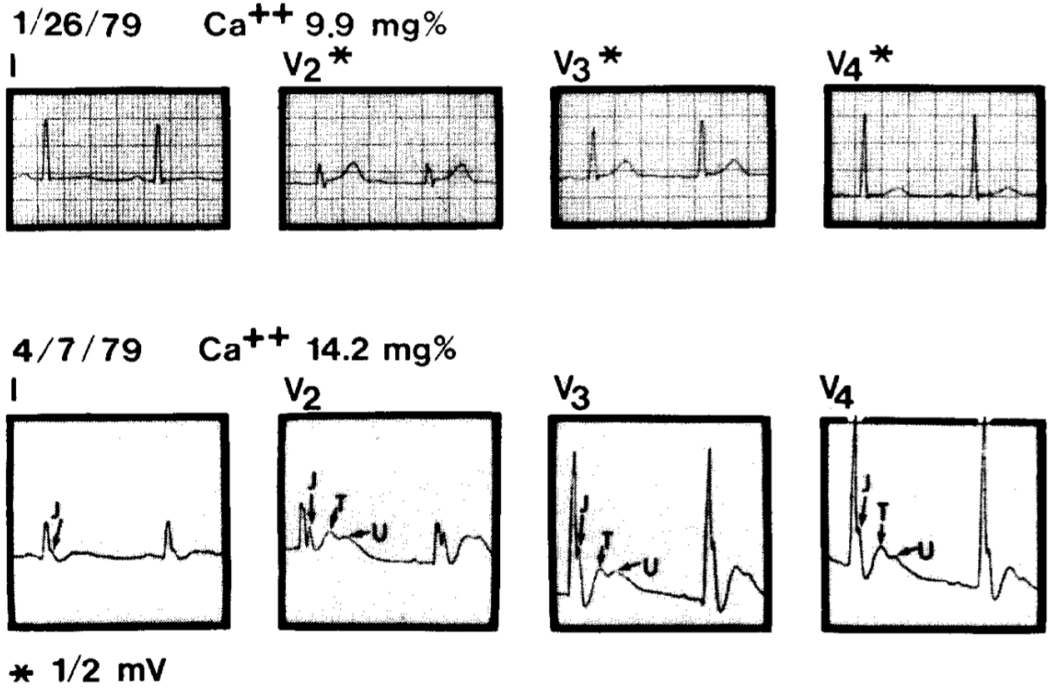
1938 Tomaszewski notes changes to the electrocardiogram in a man who died of hypothermia. Tomaszewski W. Changements electrocardiographiques observes chez un homme mort de froid. Arch Mal Coeur 1938;31:525.



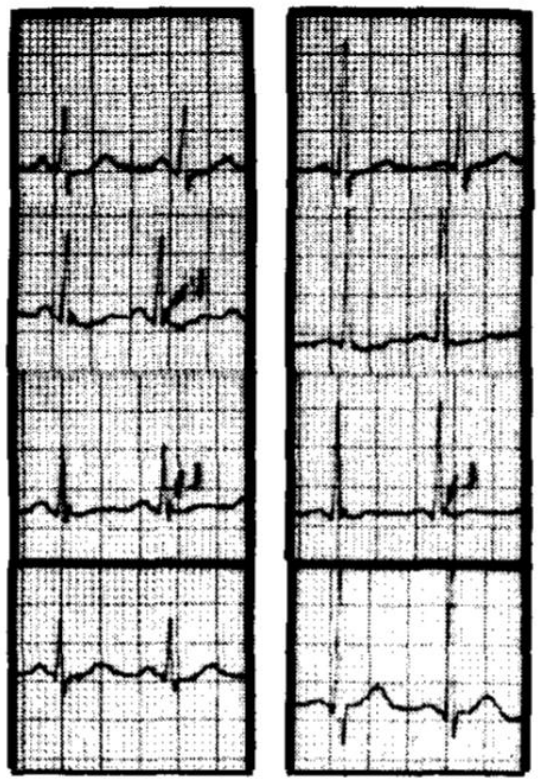
	Admission	One Hour	One Day
Temperature (C°)	24.1	29.4	36.6
Heart rate (beats/min)	50	70	98
QRS interval (msec)	184	119	71
QTc interval (msec)	516	502	403

NEJM 2005;352:2

Electrocardiographic J wave of hypercalcemia



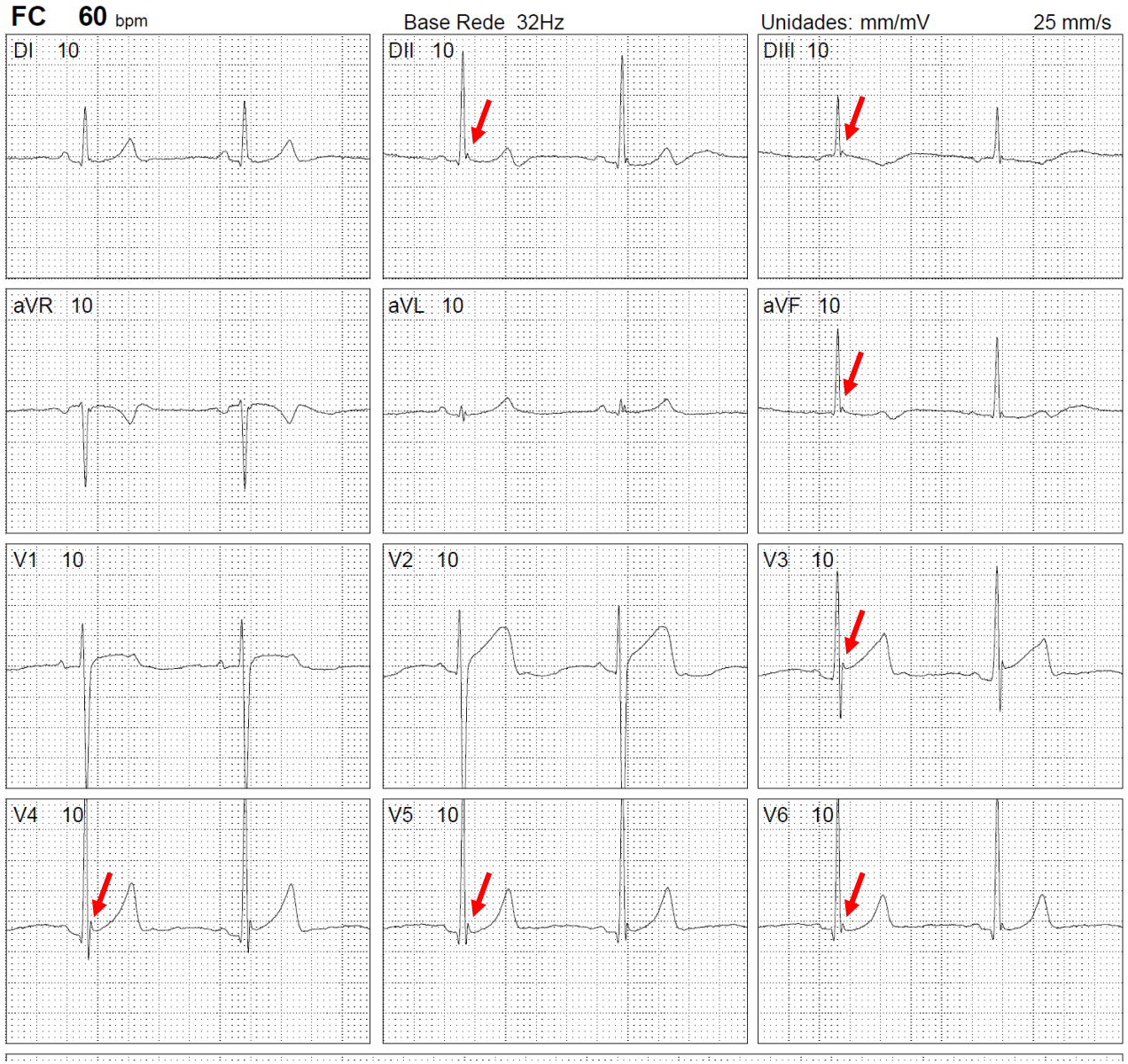
Date	Ca^{++} [mg%]
3/18/83	9.6
4/ 6/83	15.6
4/ 7/83	16.2
4/11/83	10.4

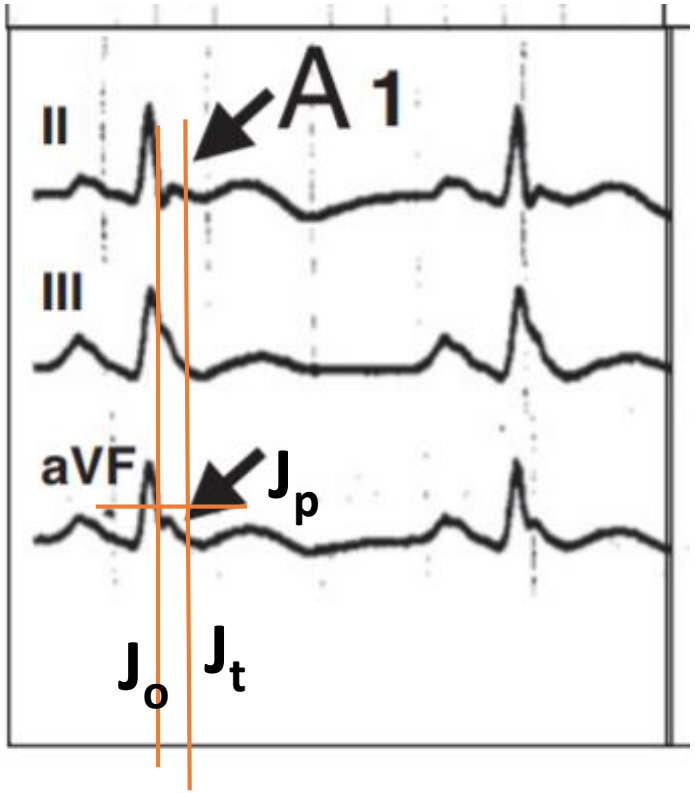


Am J Cardiol 1984;54:672

Ondas J normais

- Onda J normal é uma elevação do ponto-J, com parte da onda J dentro do QRS.
- Padrão de ER consiste:
- onda J ou ponto J distintos
- empastamento/entalhe na porção terminal do QRS, c/ ou sem supradesnível ST-T





O pico de um entalhe no final do QRS → J_p

J_p 0.1mv ≥ 2 derivações contíguas

O início do entalhe do QRS: J_o e o final J_t

SHANGAI SCORE PARA ERS

I. Clinical History	
A. Unexplained cardiac arrest, documented VF or polymorphic VT	3
B. Suspected arrhythmic syncope	2
C. Syncope of unclear mechanism/unclear etiology	1
**Only award points once for highest score within this category*	

Score (requires at least 1 ECG finding)

≥5 points: Probable/definite ERS

3–4.5 points: Possible ERS

<3 points: Nondiagnostic

SHANGAI SCORE PARA ERS

I. Clinical History	
A. Unexplained cardiac arrest, documented VF or polymorphic VT	3
B. Suspected arrhythmic syncope	2
C. Syncope of unclear mechanism/unclear etiology	1
II. Twelve-Lead ECG	
A. ER ≥0.2 mV in ≥2 inferior and/or lateral ECG leads with horizontal/descending ST segment	2
B. Dynamic changes in J-point elevation (≥0.1 mV) in ≥2 inferior and/or lateral ECG leads	1.5
C. ≥0.1 mV J-point elevation in at least 2 inferior and/or lateral ECG leads	1

Score (requires at least 1 ECG finding)

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III. Ambulatory ECG Monitoring	
A. Short-coupled PVCs with R on ascending limb or peak of T wave	2

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A. Short-coupled PVCs with R on ascending limb or peak of T wave	2
IV. Family History	
A. Relative with definite ERS	2
B. ≥2 first-degree relatives with a II.A. ECG pattern	2
C. First-degree relative with a II.A. ECG pattern	1
D. Unexplained sudden cardiac death <45 years in a first- or second-degree relative	0.5

Score (requires at least 1 ECG finding)

≥5 points: Probable/definite ERS

3–4.5 points: Possible ERS

<3 points: Nondiagnostic

SHANGAI SCORE ERS

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C. First-degree relative with a II.A. ECG pattern	1
D. Unexplained sudden cardiac death <45 years in a first- or second-degree relative	0.5
V. Genetic Test Result	
A. Probable pathogenic ERS susceptibility mutation	0.5

Score (requires at least 1 ECG finding)

≥5 points: Probable/definite ERS

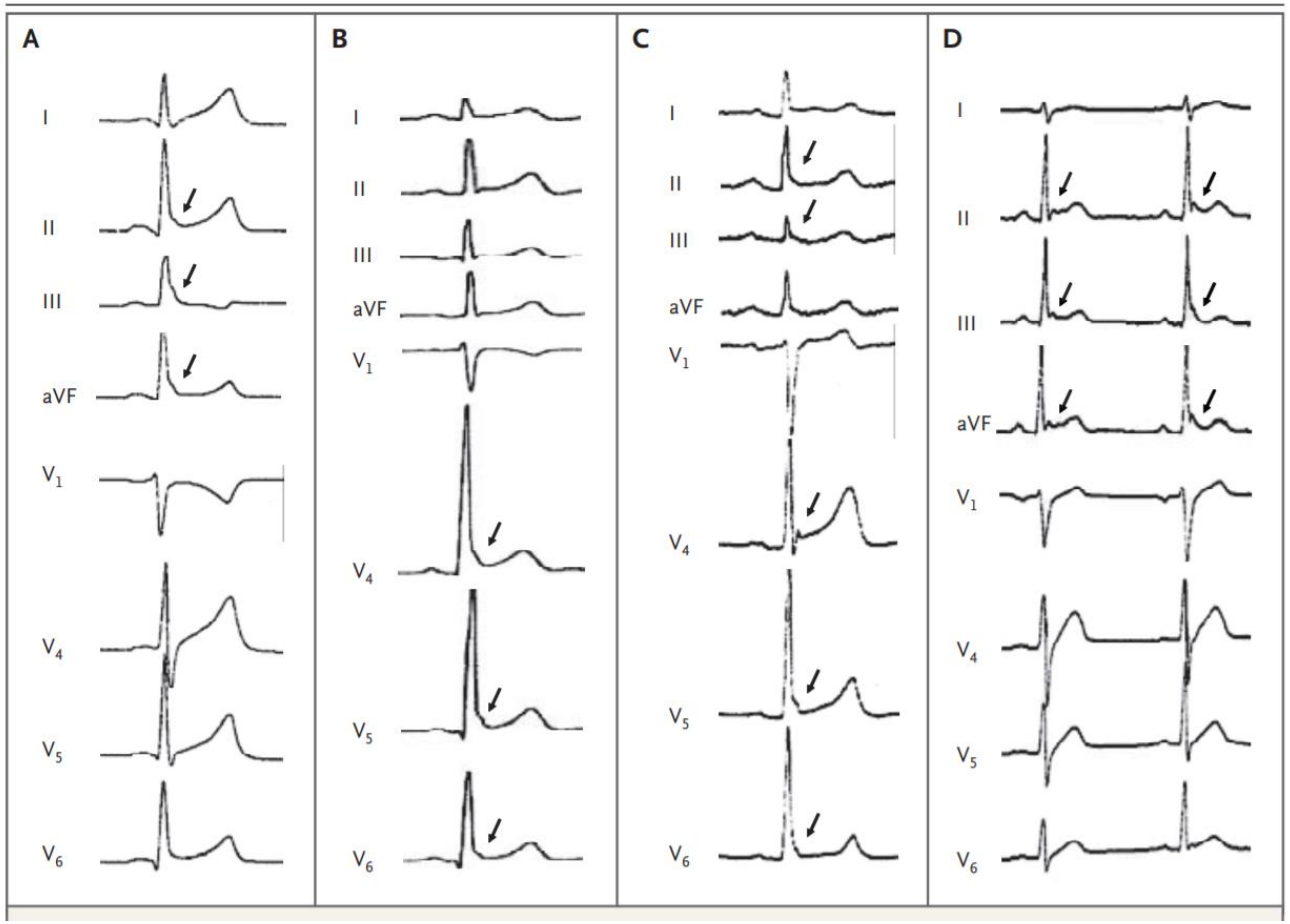
3–4.5 points: Possible ERS

<3 points: Nondiagnostic

Beingna ou maligna? – ERP or ERS?

- 2000- Experimentos sugerem predisposição para TVP-FV
- 2008- Validação em humanos*

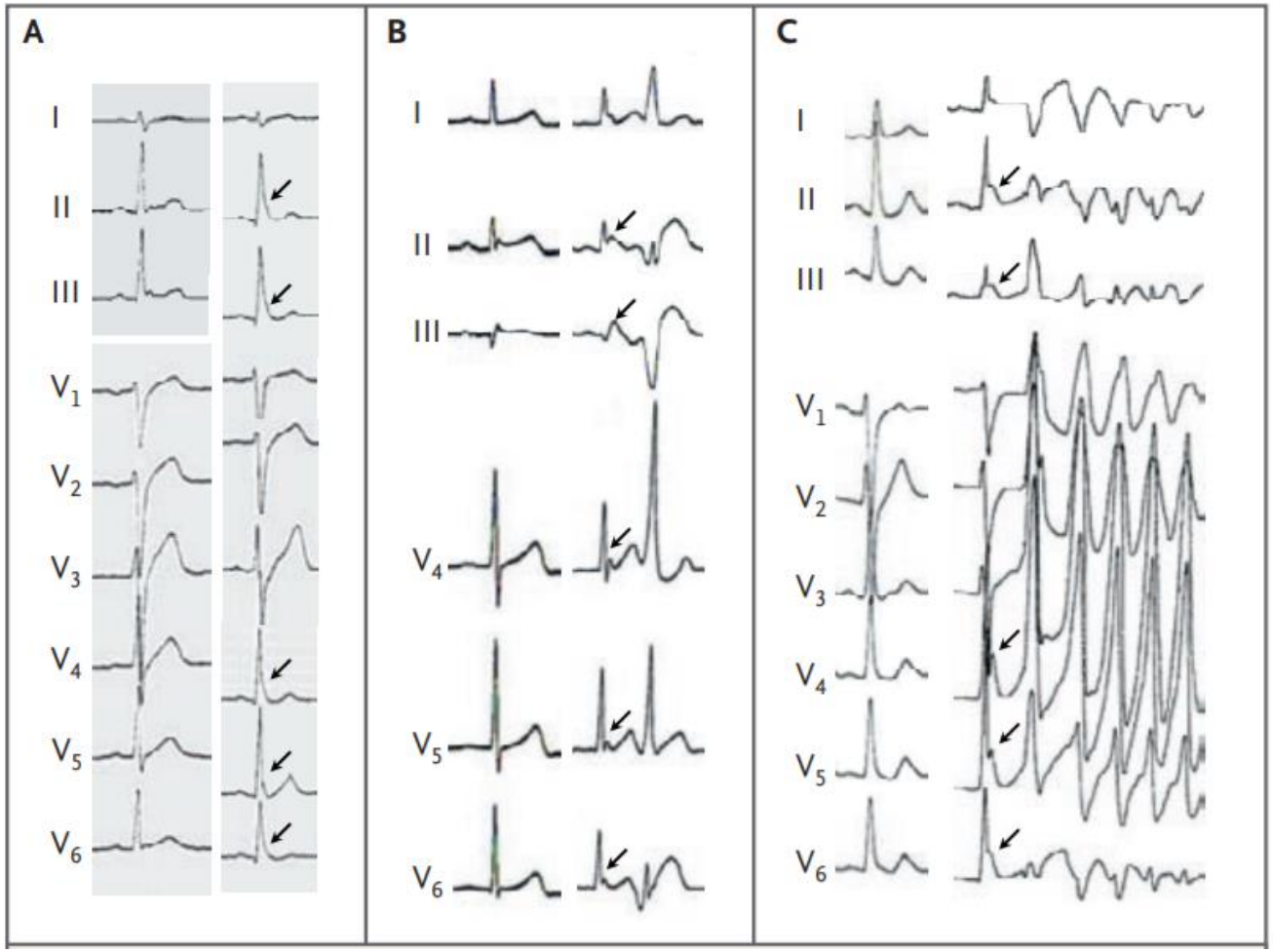
*Haissaguerre et al. NEJM 2008;358:2016
Nan et al. NEJM 2008;358:2078
Rosso et al. JACC 2008;12:1231



- N=206 idio FV (22 centros)
- <60 anos. Coronarias nls
- elevação *QRS-ST* 0.1 mV
- Parede inferior e-ou lateral
- N=412 controles
- **31 vs 5**% com ERP

N= 15 idio VF (excluiu BrS, TVPC, SQTL, SQTC)
N= 1395 controles
9/15 – **60%** com ER vs **3.3%** dos controles com padrão de ER

Haissaguerre et al, NEJM 2008,
Nan et al, NEJM 2008



- 3 casos com ECG basal e antecedendo evento maligno
- Todos mostram acentuação da ER antecedendo TV-FV de segundos a horas

Haissaguerre et al, NEJM 2008

Table 1. Characteristics of the Case Subjects.*

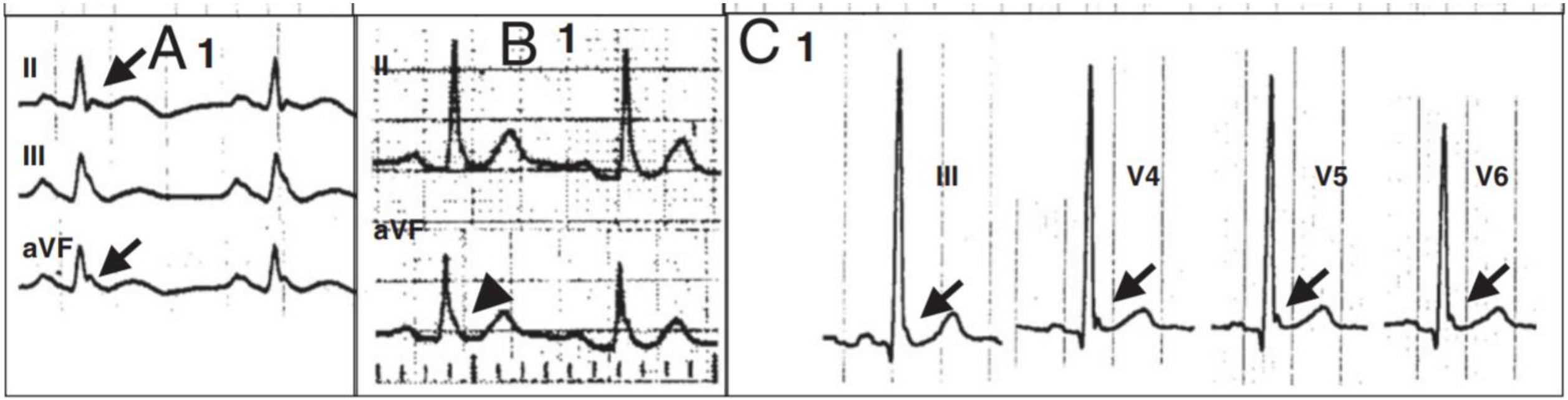
Characteristic	Early Repolarization (N=64)	No Early Repolarization (N=142)	P Value
Demographic and clinical			
Male sex — no. (%)	46 (72)	76 (54)	0.007
Age — yr	35±13	37±13	0.49
Race or ethnic group — no.†			0.69
Prolonged PR interval (>200 msec) — no. (%)	3 (5)	7 (5)	0.93
Duration of QRS complex — msec	91±10	92±15	0.39
Duration of QTc interval — msec	392±22	401±23	0.01
Presence of late potentials — no./total no. (%)§	5/44 (11)	8/63 (13)	0.84
Electrophysiological			
His bundle–ventricular interval — msec	45±7	46±10	0.77
Inducibility of ventricular fibrillation — no./total no. (%)	16/47 (34)	17/85 (20)	0.07
Inducible with 3 extrastimuli — %	81	85	0.96
Shortest coupling interval — msec	209±30	209±23	0.84
Confirmation of idiopathic ventricular fibrillation			
Coronary angiogram — no. (%)¶	62 (97)	128 (90)	0.09
Right ventricular angiogram or MRI — no. (%)	50 (78)	107 (75)	0.66
Sodium-blocker infusion — no. (%)	54 (84)	81 (57)	<0.001
Exercise test or catecholamine infusion — no. (%)	54 (84)	69 (49)	<0.001

Uma prevalência maior que a esperada de repolarização precoce (inferolateral) em menores de 60 anos, com síncope arrítmica, ou fibrilação ventricular idiopática

Table 1 Incidence of J-Point Elevation Among 45 Patients With Idiopathic VF and 124 Healthy Control Subjects Matched for Age and Gender

	Idiopathic VF		Control Subjects				
	n	%	n	%	p Value*	OR	95% CI
Any lead							
Any J-point elevation	19	42.2%	16	13.0%	0.001	3.2	1.7–6.3
J-point >1.0 mm	14	31.1%	11	8.9%	0.002	3.4	1.5–7.5
Inferior leads							
Any J-point elevation	12	26.7%	10	8.1%	0.006	3.2	1.4–7.5
J-point >1.0 mm	8	17.8%	8	6.5%	0.052	2.6	1.0–7.1
Leads I and aVL							
Any J-point elevation	6	13.3%	1	0.8%	0.009	16.9	2.0–140.3
J-point >1.0 mm	5	11.1%	0	0			
Leads V_4 to V_6							
Any J-point elevation	3	6.7%	9	7.3%	0.860	0.9	0.2–3.3
J-point >1.0 mm	3	6.7%	6	4.9%	0.686	1.3	0.3–5.3

All electrocardiograms were recorded at standard gain (10 mm = 1 mV). *p value calculated with conditional logistic regression.
CI = confidence interval; OR = odds ratio; VF = ventricular fibrillation.



N= 45 pacientes (71% masculino), 14-69 anos

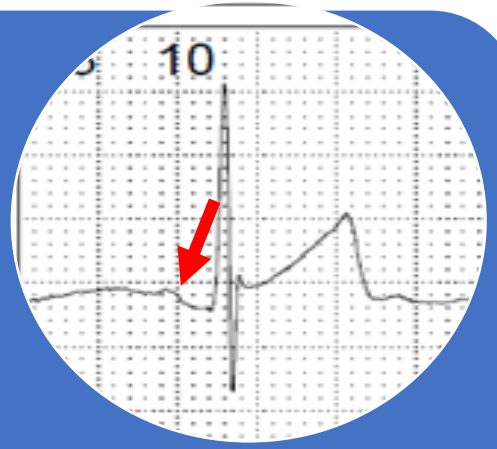
N= 124 controles

N= 121 atletas

Elevação do ponto-J:

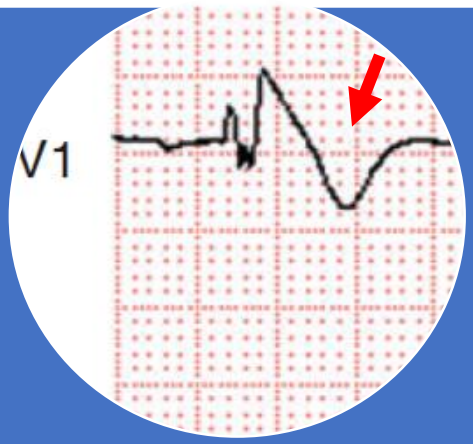
42% vs 13%	**45**	**Rosso**
31% vs 5%	**206**	**Haissaguerre**
60% vs 3.3%	**15**	**Nan**

Espectro clínico das síndromes de ER



Síndrome de repolarização precoce

Homens 70-80%
MSC 1ª manifestação
3ª década de vida
Influência vagal
QTi curto
Resposta a "pacing"
e a fármacos:
Beta-agonistas
Quinidina
Cilostazol

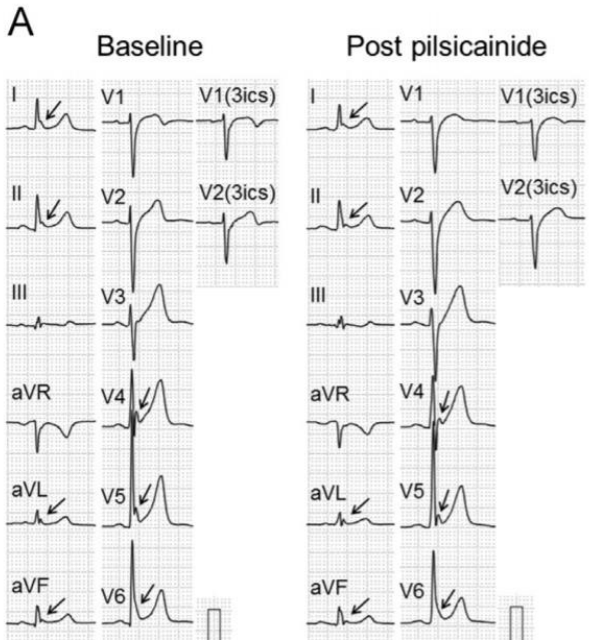


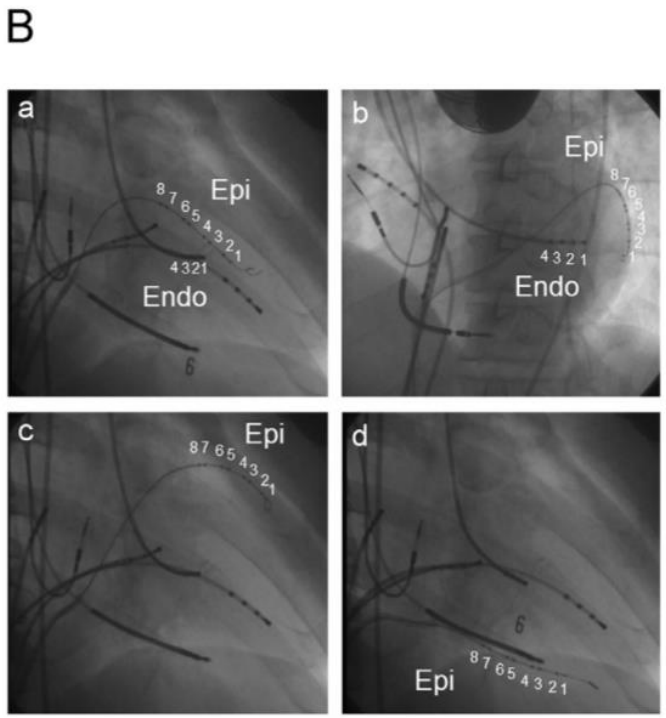
Síndrome de Brugada

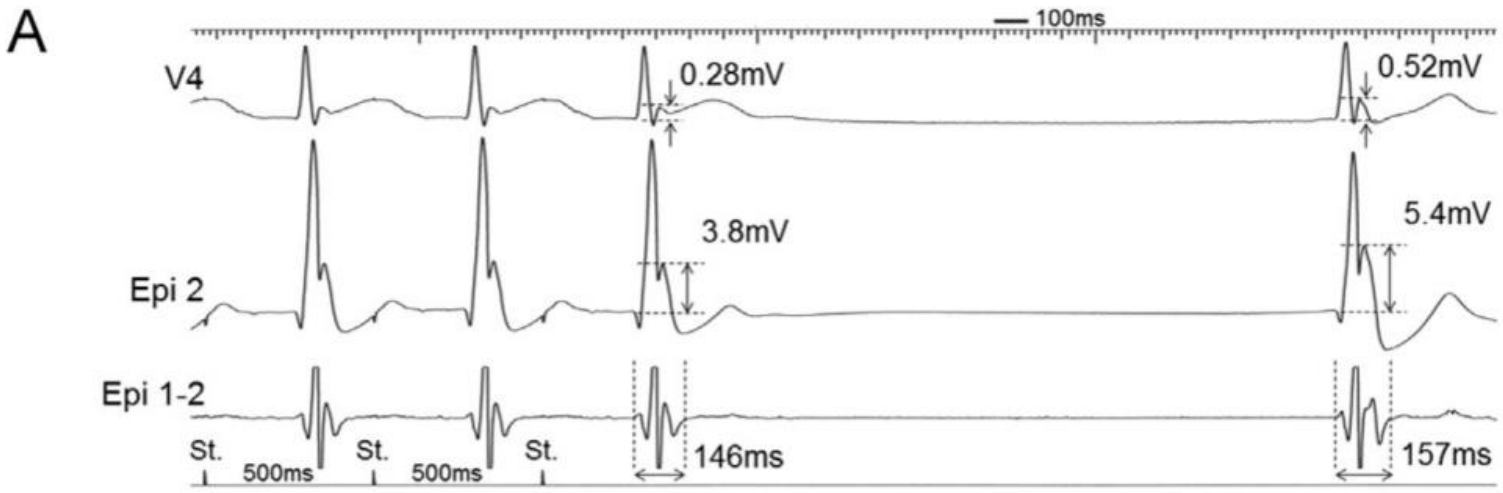
Sem diferencias regionais (negros>caucasianos) → asiáticos

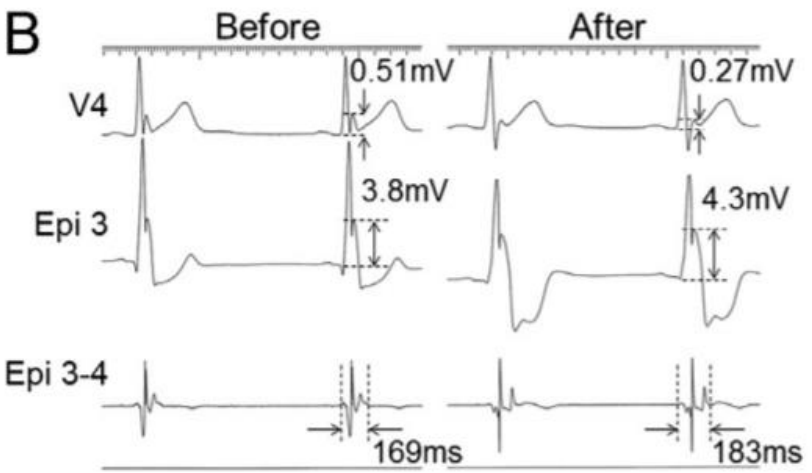
Bradicardia mediada pelo vago ↑I_{to} no VE lateral causando ↑ponto-J e ↑segmento ST V_{4-6} (ER) não arritmogênico. O aumento de J em parede inferior do VE pode representar substrato arritmogênico com EVs

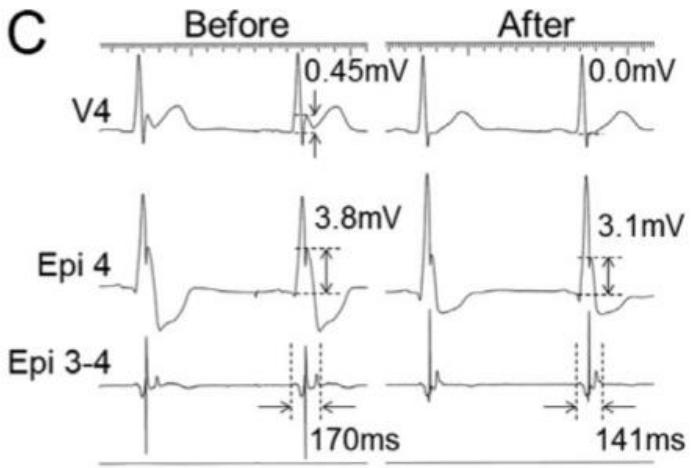
↑Influxo Na^+ por canal disfuncionante causa perda do domo do PA no EPI VSVD onde densidade de I_{to} é máxima
↑Ponto-J e ↑segmento ST de V_{1-3}





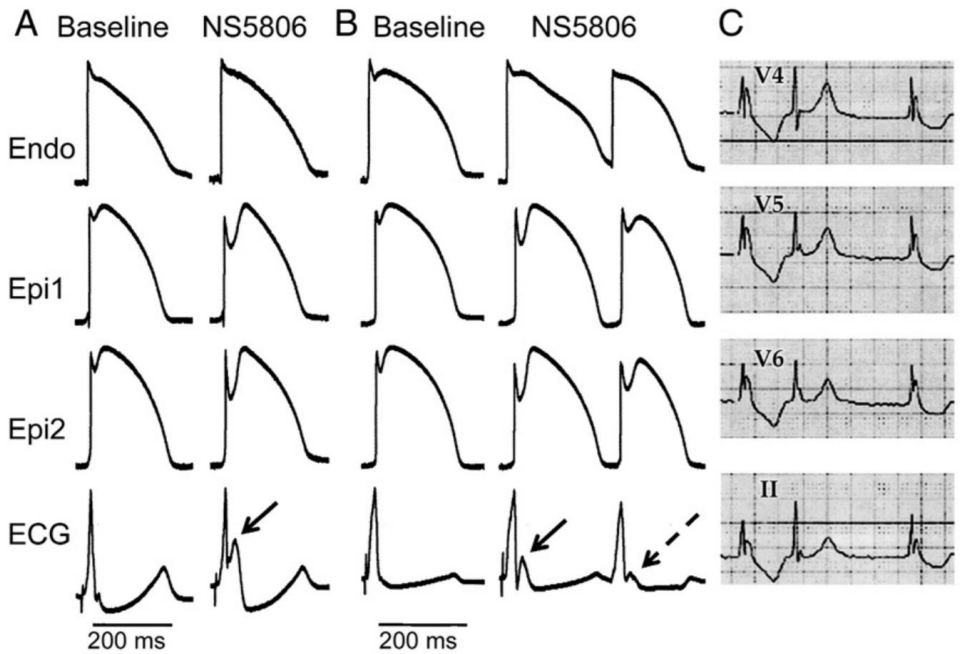


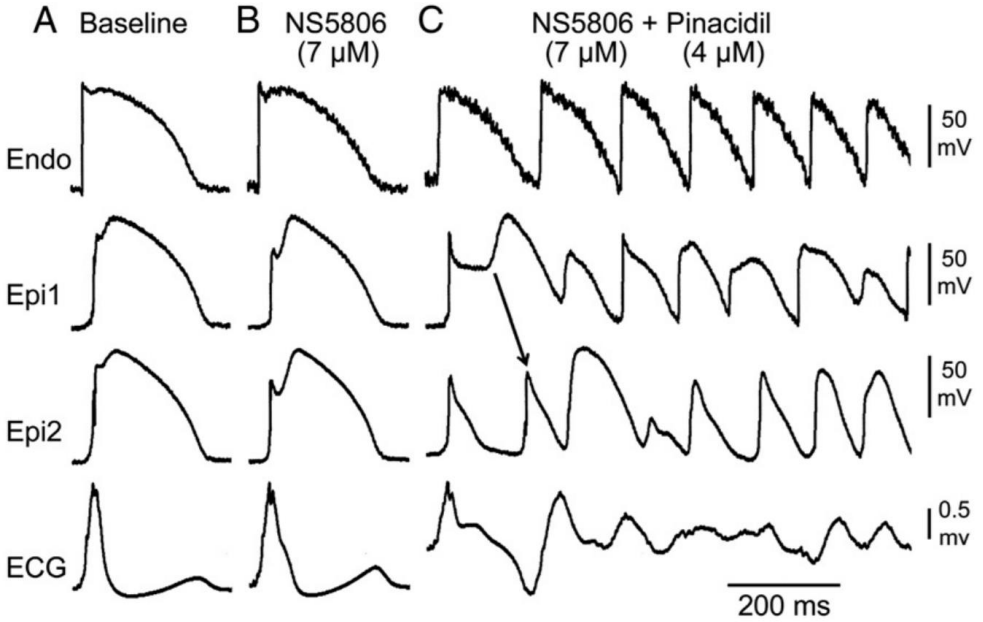


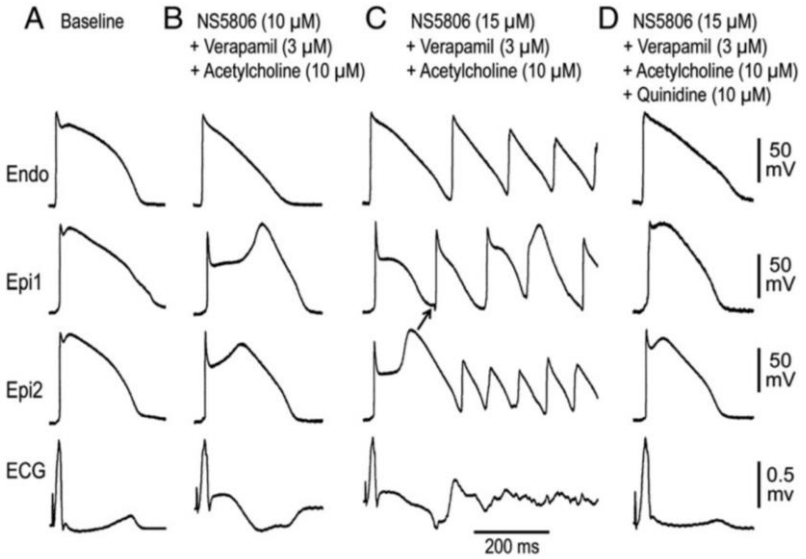


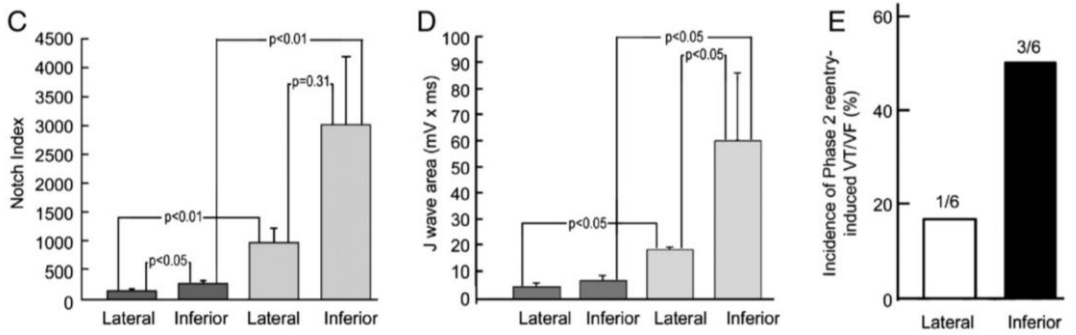
Nakagawa et al. Heart Rhythm 2013;11:314

↑ I_{To}









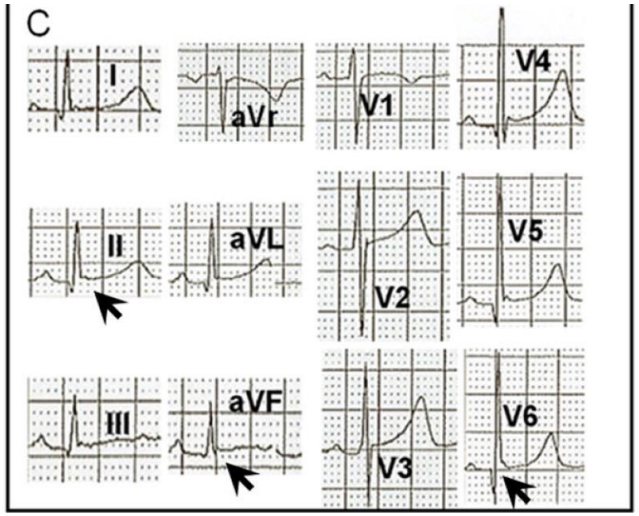
Koncz et al. J Moll Cell Cardiol 2014

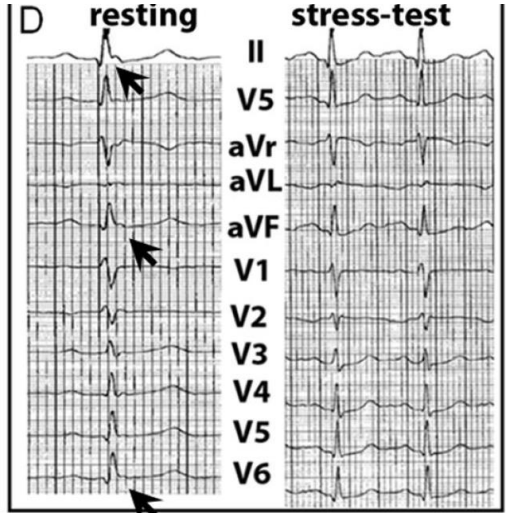
Onda J: repolarização precoce ou despolarização tardia?

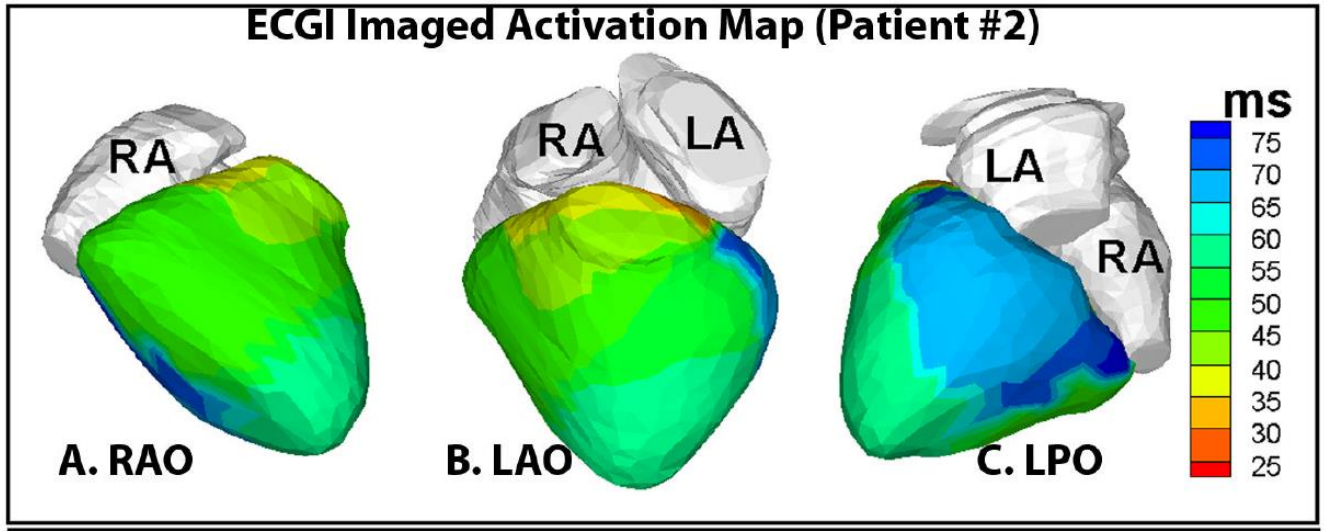
Ausência de potenciais tardios (ECG-AR) nos casos com ER sugere que a elevação do ponto J representa anormalidade de “repolarização precoce” e não “despolarização tardia”

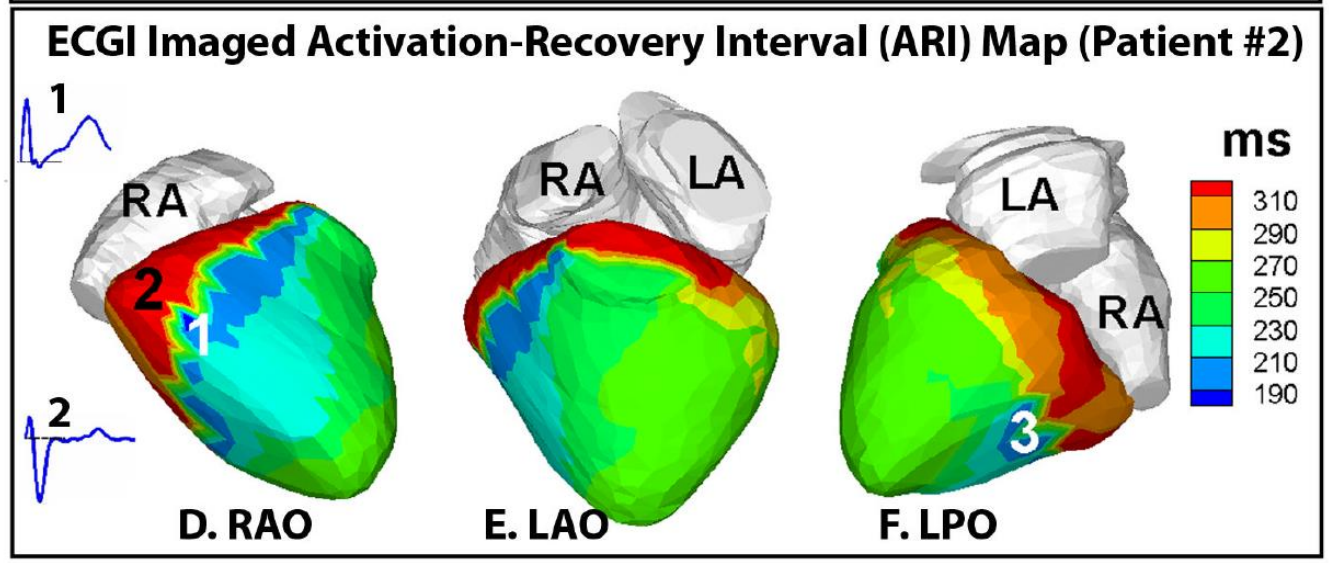
	J wave	IVCD-induced end QRS notch
Male predominance	Yes	No
Average age at initial presentation	Young adults	Older adults
Most common morphology	Dome-like smooth appearance	Relatively sharp appearance
Response to change in heart rate	Bradycardia- and pause-dependent augmentation of J wave, which may be accompanied by T-wave inversion	Tachycardia and prematurity-dependent augmentation of the notch
Structural heart diseases	Rare	Common History of myocardial infarction and/or cardiomyopathy

55 a, caucasiano, masc, HP síncope, encontrado em PCR por FV. Exames clinico e complementares normais (sem BrS, SQTL, Coronarias nls, ECO nl, RNM nl: FV Idiop → CDI.









Ghosh et al, Heart Rhythm 2010;7:534

Genetic Defects Associated with ERS

	Locus	Gene/protein	Ion channel	Percent of Probands
ERS1	12p11.23	*KCNJ8, Kir6.1*	↑$I_{K\text{-}ATP}$	Rare
ERS2	12p13.3	*CACNA1C, $Ca_v1.2$*	↓ I_{Ca}	4.1%
ERS3	10p12.33	*CACNB2b, $Ca_vß2b$*	↓ I_{Ca}	8.3%
ERS4	7q21.11	*CACNA2D1, $Ca_vα2δ1$*	↓ I_{Ca}	4.1%
ERS5	12p12.1	*ABCC9, SUR2A*	↑ $I_{K\text{-}ATP}$	Rare
ERS6	3p21	*SCN5A, $Na_v1.5$*	↓ I_{Na}	Rare
ERS7	3p22.2	*SCN10A, $Na_v1.8$*	↓ I_{Na}	Rare

Genetic Defects Associated with BrS

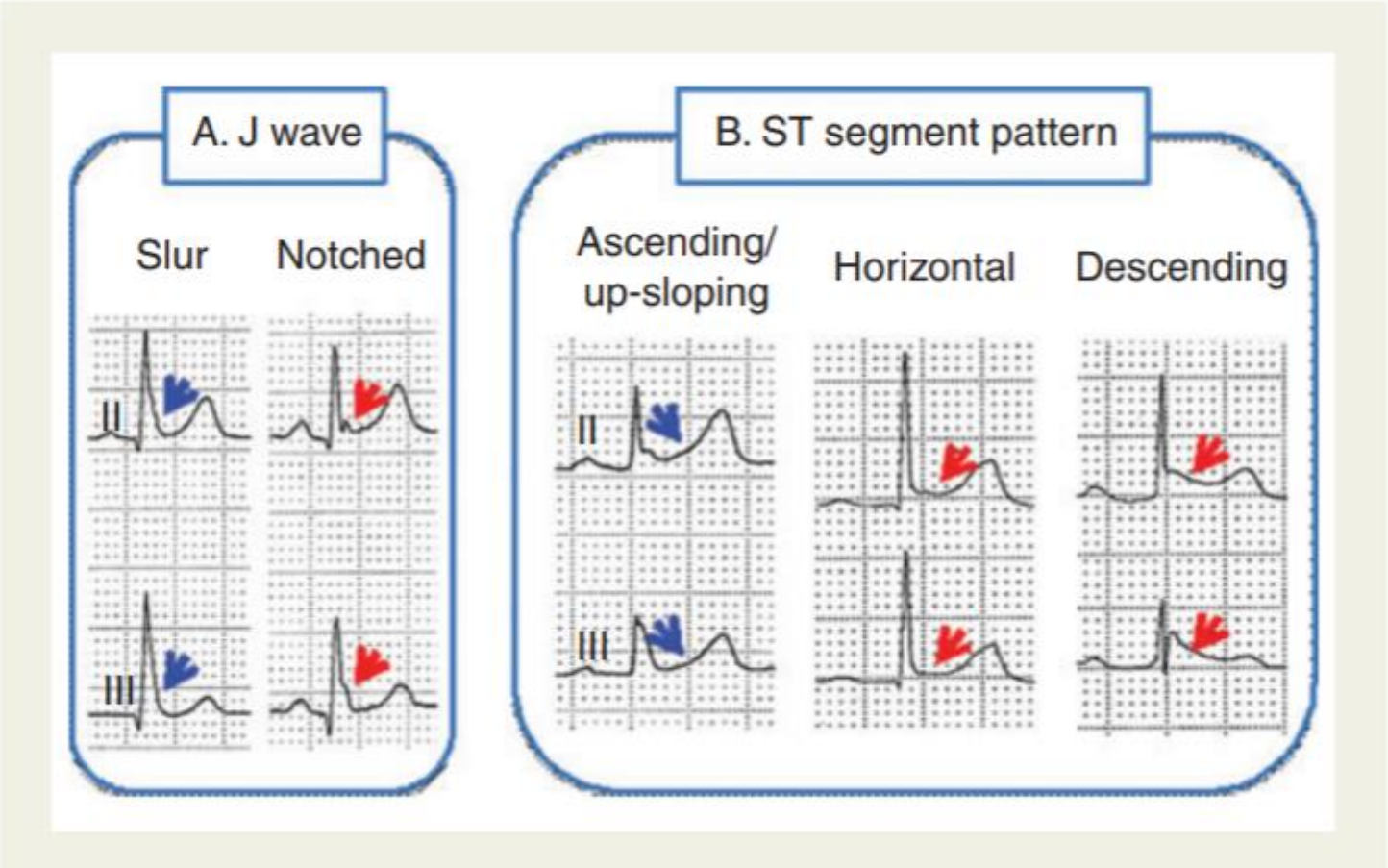
BrS1	3p21	*SCN5A, $Na_v1.5$*	↓ I_{Na}	11%–28%
BrS2	3p24	*GPD1L*	↓ I_{Na}	Rare
BrS3	12p13.3	*CACNA1C, $Ca_v1.2$*	↓ I_{Ca}	6.6%
BrS4	10p12.33	*CACNB2b, $Ca_vß2b$*	↓ I_{Ca}	4.8%
BrS5	19q13.1	*SCN1B, $Na_vß1$*	↓ I_{Na}	1.1%
BrS6	11q13-14	*KCNE3, MiRP2*	↑ I_{to}	Rare
BrS7	11q23.3	*SCN3B, $Na_vß3$*	↓ I_{Na}	Rare
BrS8	12p11.23	*KCNJ8, Kir6.1*	↑ $I_{K\text{-}ATP}$	2%
BrS9	7q21.11	*CACNA2D1, $Ca_vα2δ1$*	↓ I_{Ca}	1.8%
BrS10	1p13.2	*KCND3, $K_v4.3$*	↑ I_{to}	Rare
BrS11	17p13.1	*RANGRF, MOG1*	↓ I_{Na}	Rare
BrS12	3p21.2-p14.3	*SLMAP*	↓ I_{Na}	Rare
BrS13	12p12.1	*ABCC9, SUR2A*	↑ $I_{K\text{-}ATP}$	Rare
BrS14	11q23	*SCN2B, $Na_vß2$*	↓ I_{Na}	Rare
BrS15	12p11	*PKP2, Plakophillin-2*	↓ I_{Na}	Rare
BrS16	3q28	*FGF12, FHAF1*	↓ I_{Na}	Rare
BrS17	3p22.2	*SCN10A, $Na_v1.8$*	↓ I_{Na}	5%–16.7%
BrS18	6q	*HEY2 (transcriptional factor)*	↑ I_{Na}	Rare

ERP enquanto fator de Risco de MSC

- Diagnóstico fortuito de onda J no ECG aumenta risco de FV idiopática de:

3,4:100.000 → 11:100.000

Finlandia



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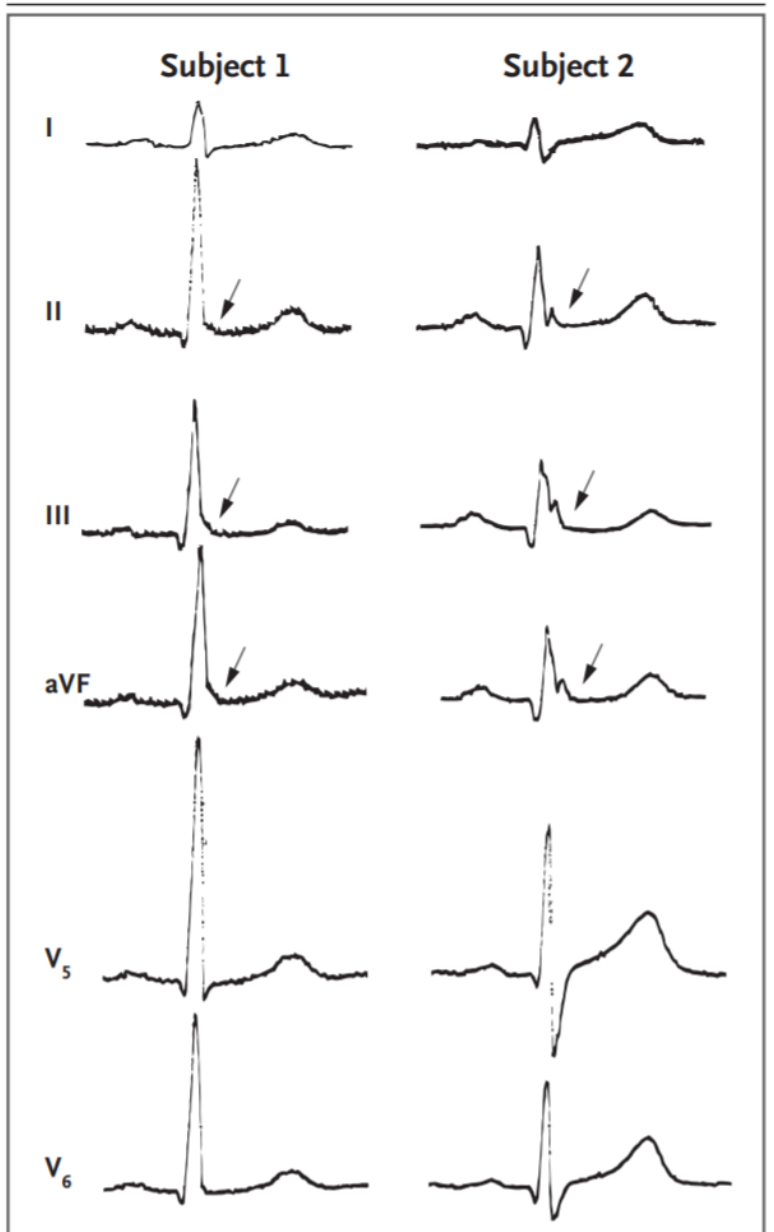
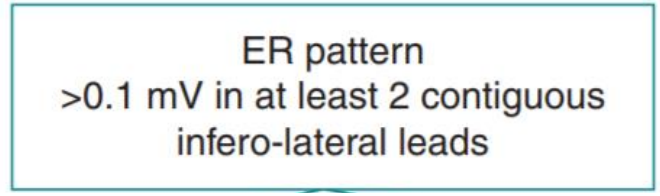
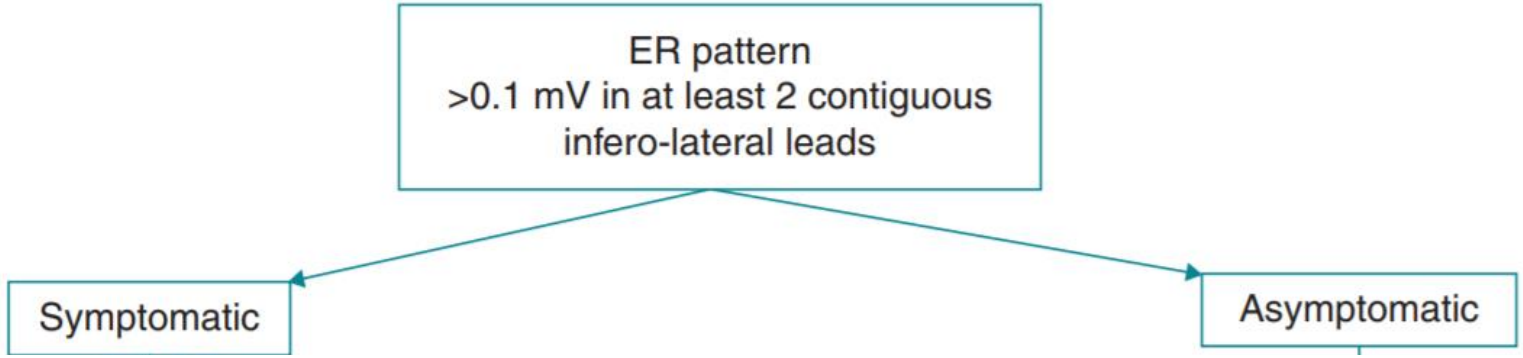
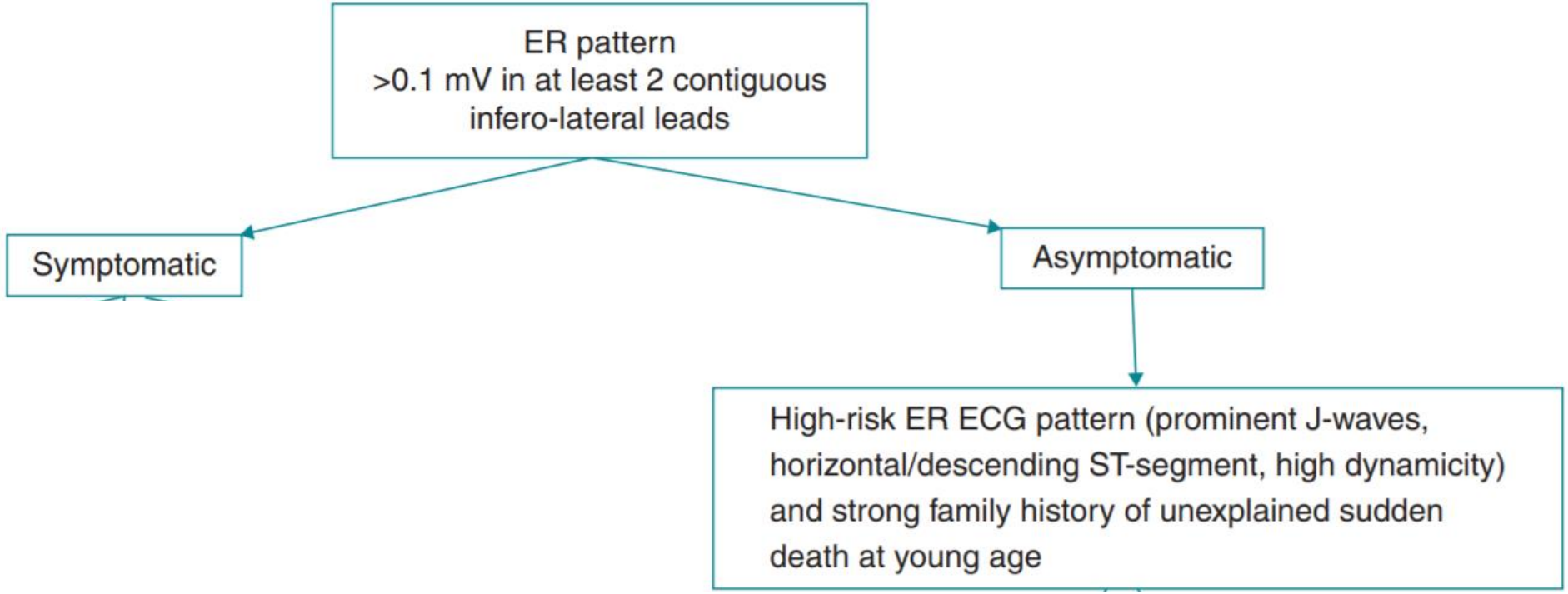
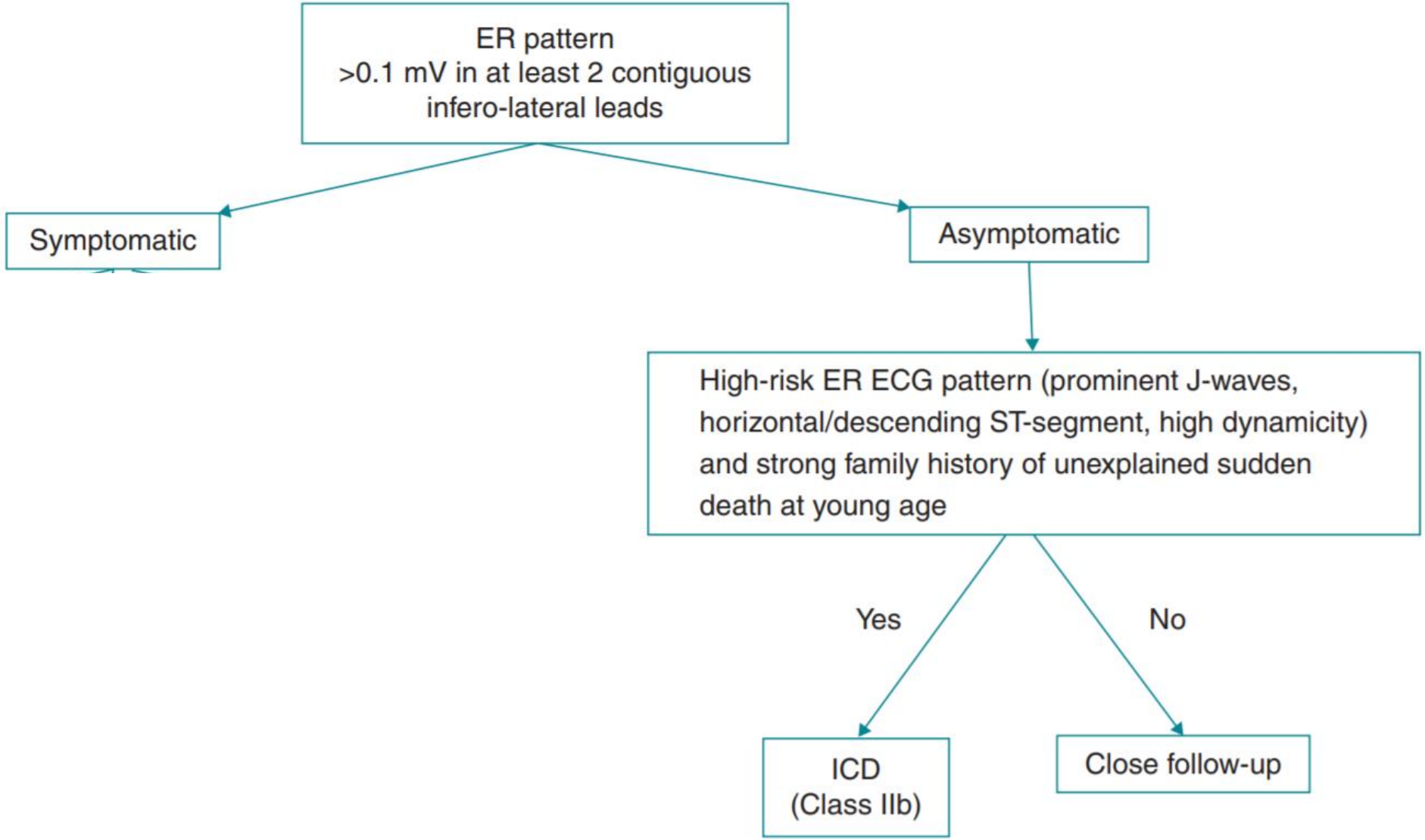


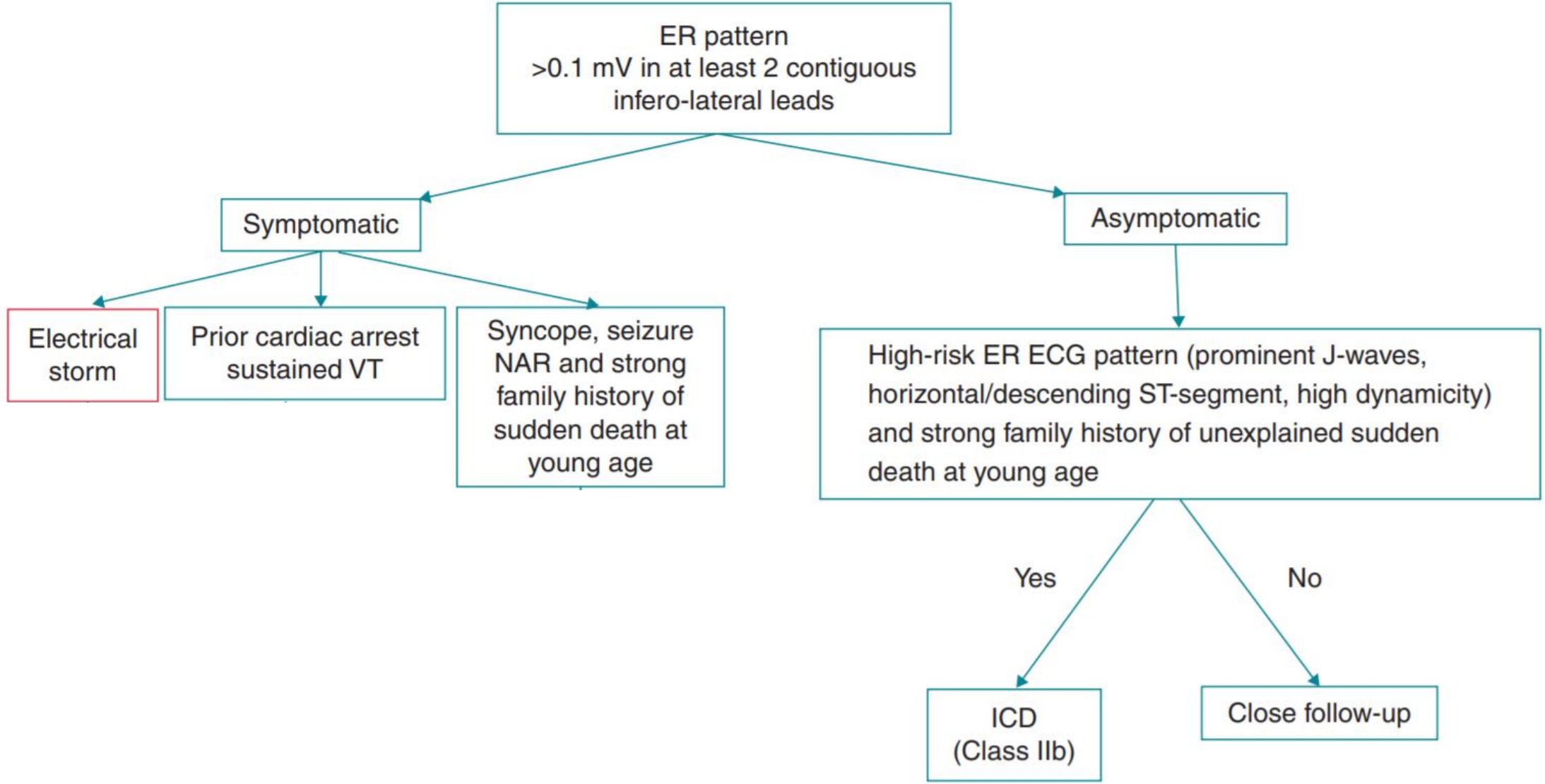
Figure 2. Baseline Electrocardiograms of Two Male Subjects with J-Point Elevation of More Than 0.2 mV in the Inferior Leads.

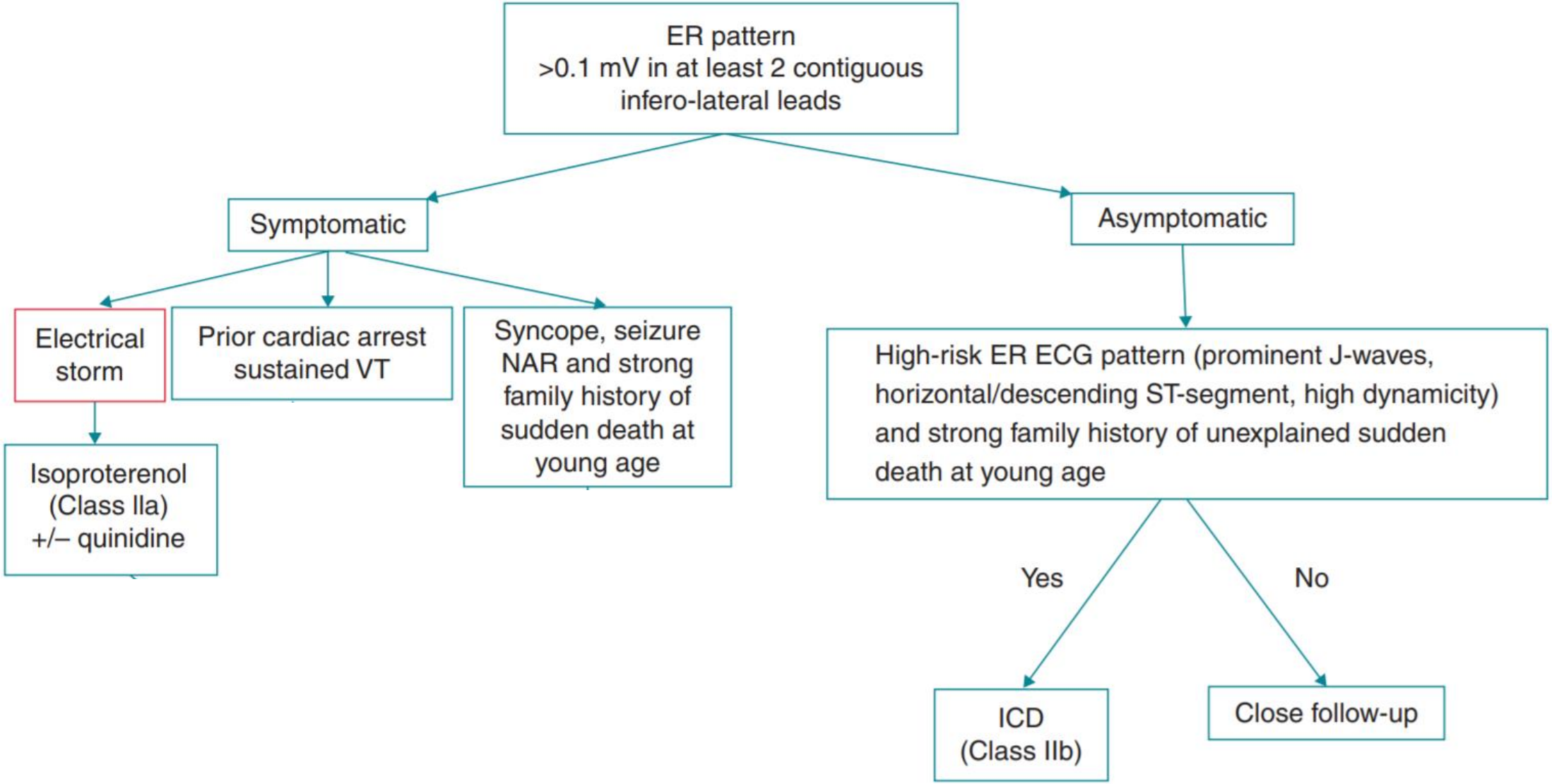

ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads

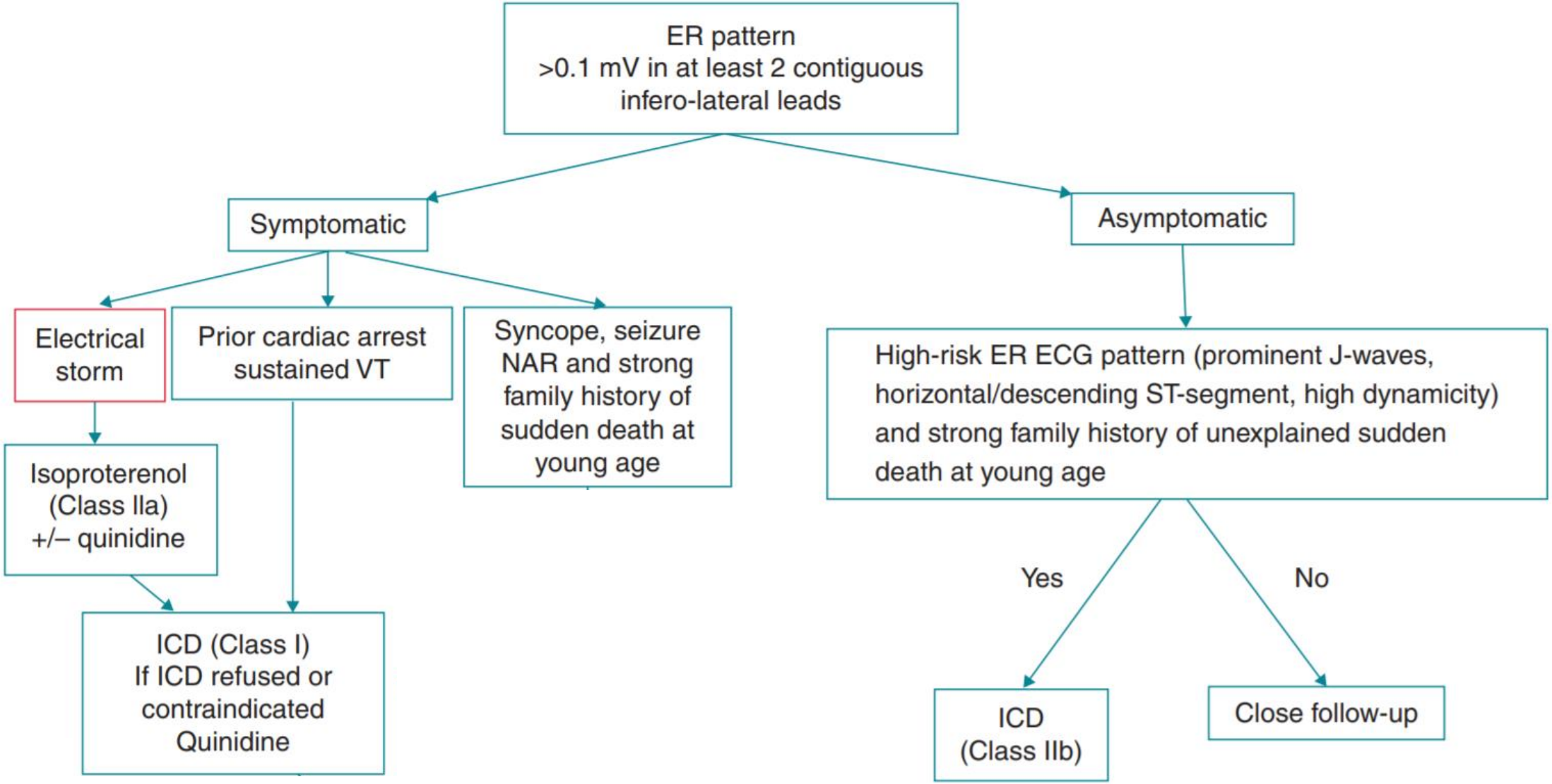

ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads
Symptomatic
Asymptomatic

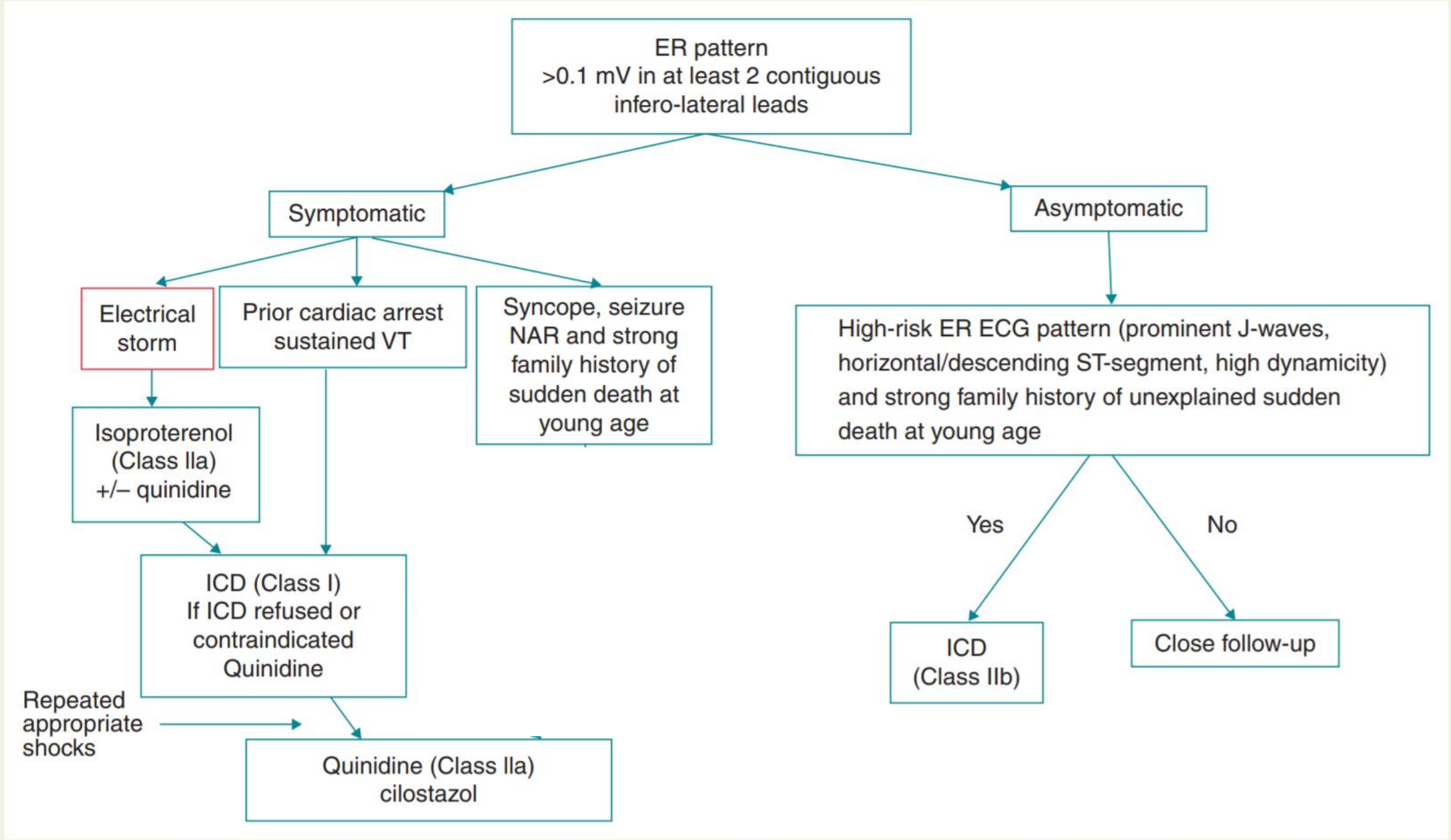

ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads
Symptomatic
Asymptomatic
High-risk ER ECG pattern (prominent J-waves,
horizontal/descending ST-segment, high dynamicity)
and strong family history of unexplained sudden
death at young age

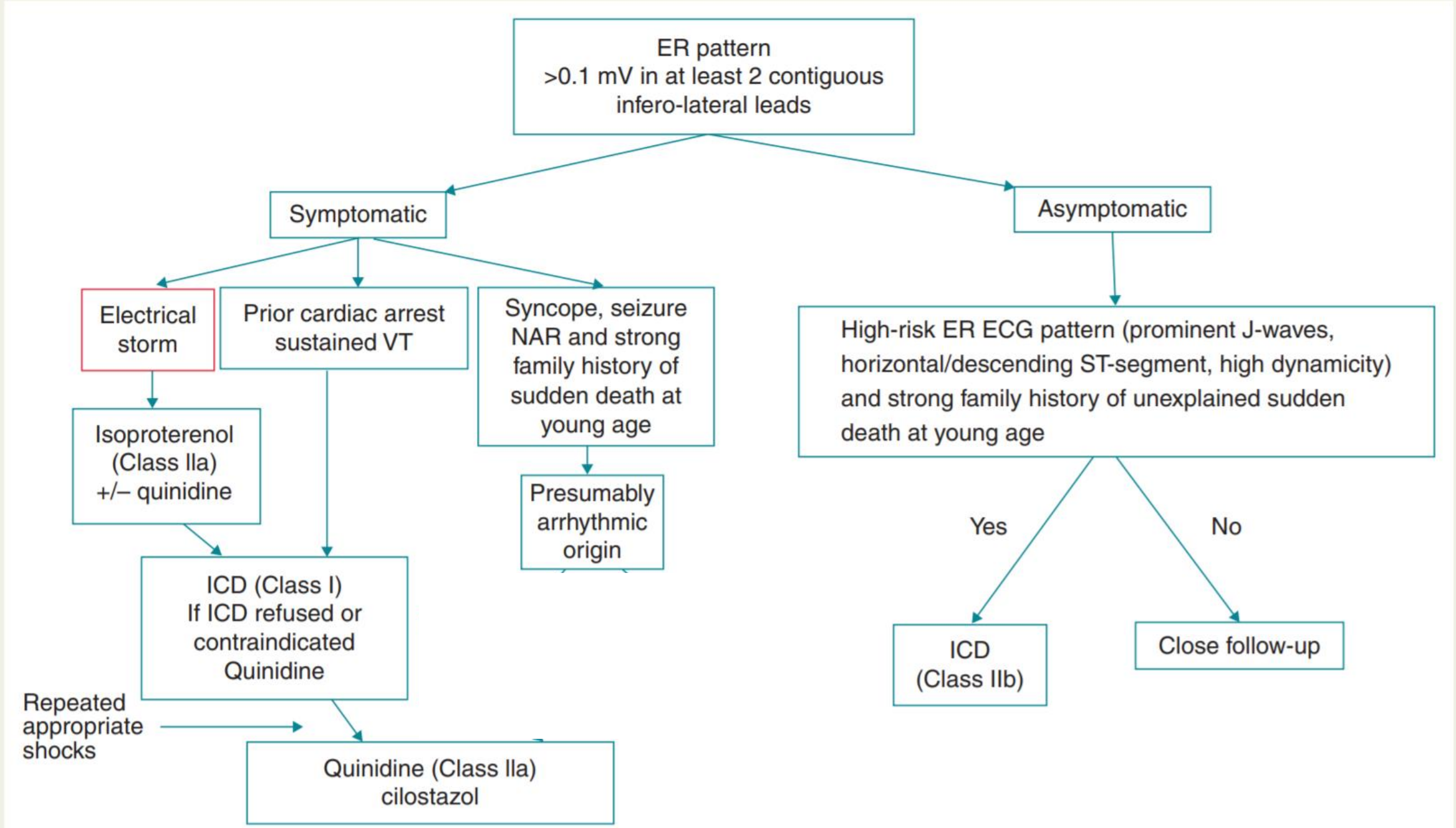

ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads
Symptomatic
Asymptomatic
High-risk ER ECG pattern (prominent J-waves,
horizontal/descending ST-segment, high dynamicity)
and strong family history of unexplained sudden
death at young age
Yes
No
ICD
(Class IIb)
Close follow-up

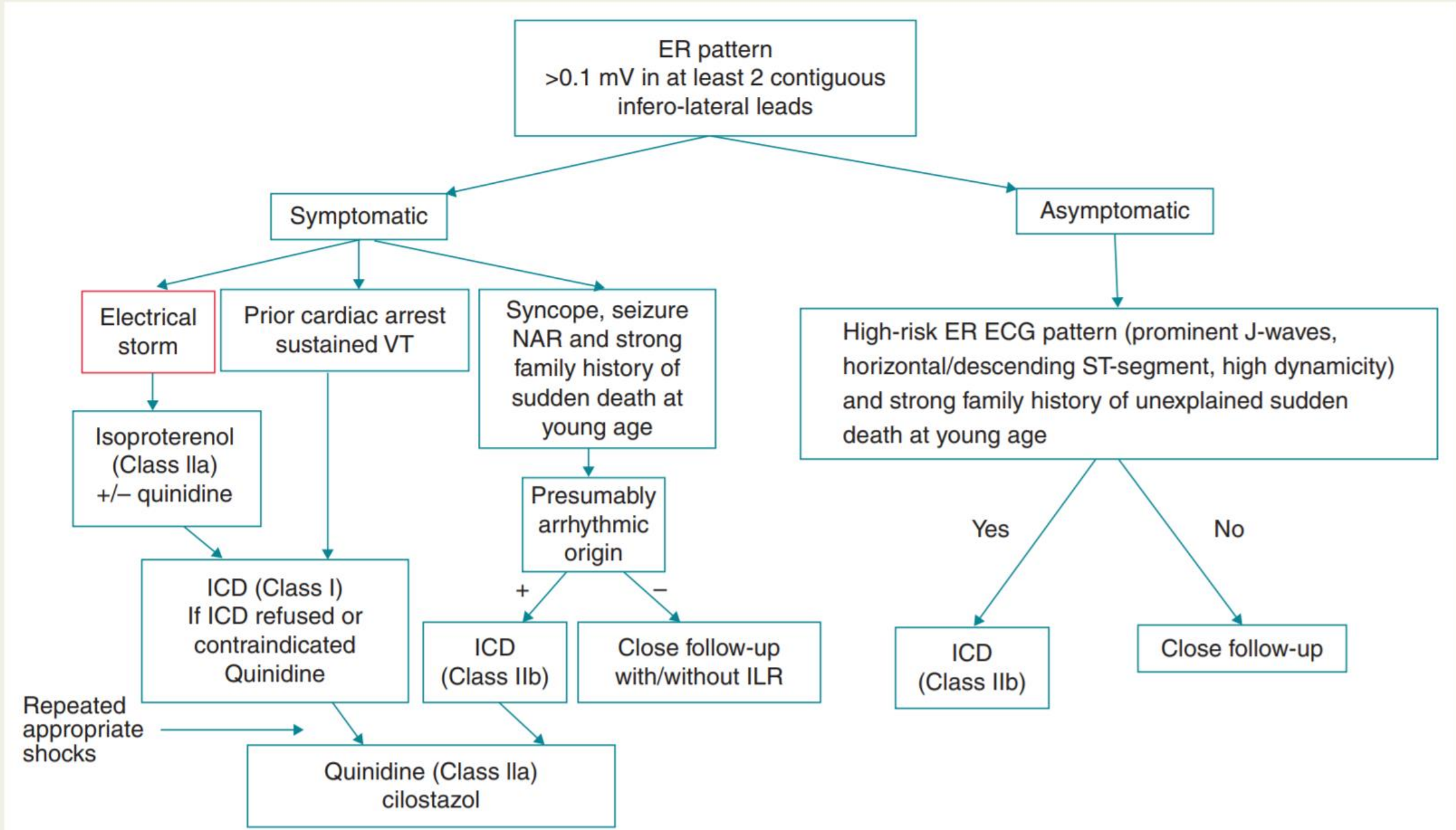

ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads
Symptomatic
Asymptomatic
Electrical
storm
Prior cardiac arrest
sustained VT
Syncope, seizure
NAR and strong
family history of
sudden death at
young age
High-risk ER ECG pattern (prominent J-waves,
horizontal/descending ST-segment, high dynamicity)
and strong family history of unexplained sudden
death at young age
Yes
No
ICD
(Class IIb)
Close follow-up


ER pattern
>0.1 mV in at least 2 contiguous
infero-lateral leads
Symptomatic
Asymptomatic
Electrical
storm
Prior cardiac arrest
sustained VT
Syncope, seizure
NAR and strong
family history of
sudden death at
young age
Isoproterenol
(Class IIa)
+/– quinidine
High-risk ER ECG pattern (prominent J-waves,
horizontal/descending ST-segment, high dynamicity)
and strong family history of unexplained sudden
death at young age
Yes
No
ICD
(Class IIb)
Close follow-up


ER pattern
>0.1 mV in at least 2 contiguous infero-lateral leads
Symptomatic
Asymptomatic
Electrical storm
Prior cardiac arrest sustained VT
Syncope, seizure NAR and strong family history of sudden death at young age
High-risk ER ECG pattern (prominent J-waves, horizontal/descending ST-segment, high dynamicity) and strong family history of unexplained sudden death at young age
Isoproterenol (Class IIa) +/– quinidine
ICD (Class I) If ICD refused or contraindicated Quinidine
Yes
No
ICD (Class IIb)
Close follow-up


ER pattern
>0.1 mV in at least 2 contiguous infero-lateral leads
Symptomatic
Asymptomatic
Electrical storm
Prior cardiac arrest sustained VT
Syncope, seizure NAR and strong family history of sudden death at young age
High-risk ER ECG pattern (prominent J-waves, horizontal/descending ST-segment, high dynamicity) and strong family history of unexplained sudden death at young age
Isoproterenol (Class IIa) +/– quinidine
ICD (Class I) If ICD refused or contraindicated Quinidine
Repeated appropriate shocks
Quinidine (Class IIa) cilostazol
Yes
No
ICD (Class IIb)
Close follow-up


ER pattern
>0.1 mV in at least 2 contiguous infero-lateral leads
Symptomatic
Asymptomatic
Electrical storm
Prior cardiac arrest sustained VT
Syncope, seizure NAR and strong family history of sudden death at young age
High-risk ER ECG pattern (prominent J-waves, horizontal/descending ST-segment, high dynamicity) and strong family history of unexplained sudden death at young age
Isoproterenol (Class IIa) +/– quinidine
Presumably arrhythmic origin
Yes
No
ICD (Class I) If ICD refused or contraindicated Quinidine
ICD (Class IIb)
Close follow-up
Repeated appropriate shocks
Quinidine (Class IIa) cilostazol


ER pattern
>0.1 mV in at least 2 contiguous infero-lateral leads
Symptomatic
Asymptomatic
Electrical storm
Prior cardiac arrest sustained VT
Syncope, seizure NAR and strong family history of sudden death at young age
High-risk ER ECG pattern (prominent J-waves, horizontal/descending ST-segment, high dynamicity) and strong family history of unexplained sudden death at young age
Isoproterenol (Class IIa) +/– quinidine
Presumably arrhythmic origin
Yes
No
ICD (Class I) If ICD refused or contraindicated Quinidine
+
–
ICD (Class IIb)
Close follow-up with/without ILR
ICD (Class IIb)
Close follow-up
Repeated appropriate shocks
Quinidine (Class IIa) cilostazol

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Heart Rhythm Disorders

Characteristics of Recurrent Ventricular Fibrillation Associated With Inferolateral Early Repolarization

Role of Drug Therapy

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Objectives Our purpose was to evaluate the efficacy of antiarrhythmic drugs (AADs) in recurrent ventricular fibrillation (VF) associated with inferolateral early repolarization pattern on the electrocardiogram.

Background Although an implantable cardioverter-defibrillator is the treatment of choice, additional AADs may be necessary to prevent frequent episodes of VF and reduce implantable cardioverter-defibrillator shock burden or as a lifesaving therapy in electrical storms.

Methods From a multicenter cohort of 122 patients (90 male subjects, age 37 ± 12 years) with idiopathic VF and early repolarization abnormality in the inferolateral leads, we selected all patients with more than 3 episodes of VF (multiple) including those with electrical storms (≥3 VF in 24 h). The choice of AAD was decided by individual physicians. Follow-up data were obtained for all patients using monitoring with implantable defibrillator. Successful oral AAD was defined as elimination of all recurrences of VF with a minimal follow-up period of 12 months.

Results Multiple episodes of VF were observed in 33 (27%) patients. Electrical storms (34 ± 47 episodes) occurred in 16 and were unresponsive to beta-blockers (11 of 11), lidocaine/mexiletine (9 of 9), and verapamil (3 of 3), while amiodarone was partially effective (3 of 10). In contrast, isoproterenol infusion immediately suppressed electrical storms in 7 of 7 patients. Over a follow-up of 69 ± 58 months, oral AADs were poorly effective in preventing recurrent VF: beta-blockers (2 of 16), verapamil (0 of 4), mexiletine (0 of 4), amiodarone (1 of 7), and class 1C AADs (2 of 9). Quinidine was successful in 9 of 9 patients, decreasing recurrent VF from 33 ± 35 episodes to nil for 25 ± 18 months. In addition, quinidine restored a normal electrocardiogram.

Conclusions Multiple recurrences of VF occurred in 27% of patients with early repolarization abnormality and may be life threatening. Isoproterenol in acute cases and quinidine in chronic cases are effective AADs. (J Am Coll Cardiol 2009;53:612–9) © 2009 by the American College of Cardiology Foundation

Nos pacientes com ERS:
33/122pts com >3 FV

EFETIVIDADE AGUDA:

1. Bbloqueador: 0/11
2. Lidocaina: 0/9
3. Verapamil: 0/3
4. Amiodarona: 3/10
5. **Isoproterenol: 7/7**

EFETIVIDADE DE DAA via oral
69±58 meses:

1. BBloqueador- 2/16
2. Verapamil- 0/4
3. Mexiletina- 0/4
4. Amiodarona- 1/7
5. Propafenona- 2/9
6. **Quinidina- 9/9 – aboliu ERP**