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4-5 DÉCEMBRE 202(

HOTEL VILLA M.
MARSEILLE I FRANCE

1Çèmes journées françaises pratiques de rythmologie & de stimulation cardiaque

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Patient asymptomatique et ECG pathologique au cabinet : quelle prise en charge en 2025 ?

Brugada et QT long

Fabrice Extramiana

AP-HP. Nord Université Paris Cité

Hôpital Bichat Claude-Bernard AP-HP

Université Paris Cité

Centre de Référence des Maladies Cardiaques Héréditaires ou Rares

cardiogen

filière nationale de santé maladies cardiaques héréditaires

Conflict of Interest

- CHIESI: adjudication committee
- J&J: scientific committee
- Patent processing for AI based algorithm in LQTS

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Patient asymptomatique et ECG pathologique : Brugada et QT long

- Penser et faire le diagnostic : différents aspects ECG
- Eliminer les diagnostics différentiels
- Test de provocation : Quand ? Comment ?
- Prises en charge initiale et ultérieure

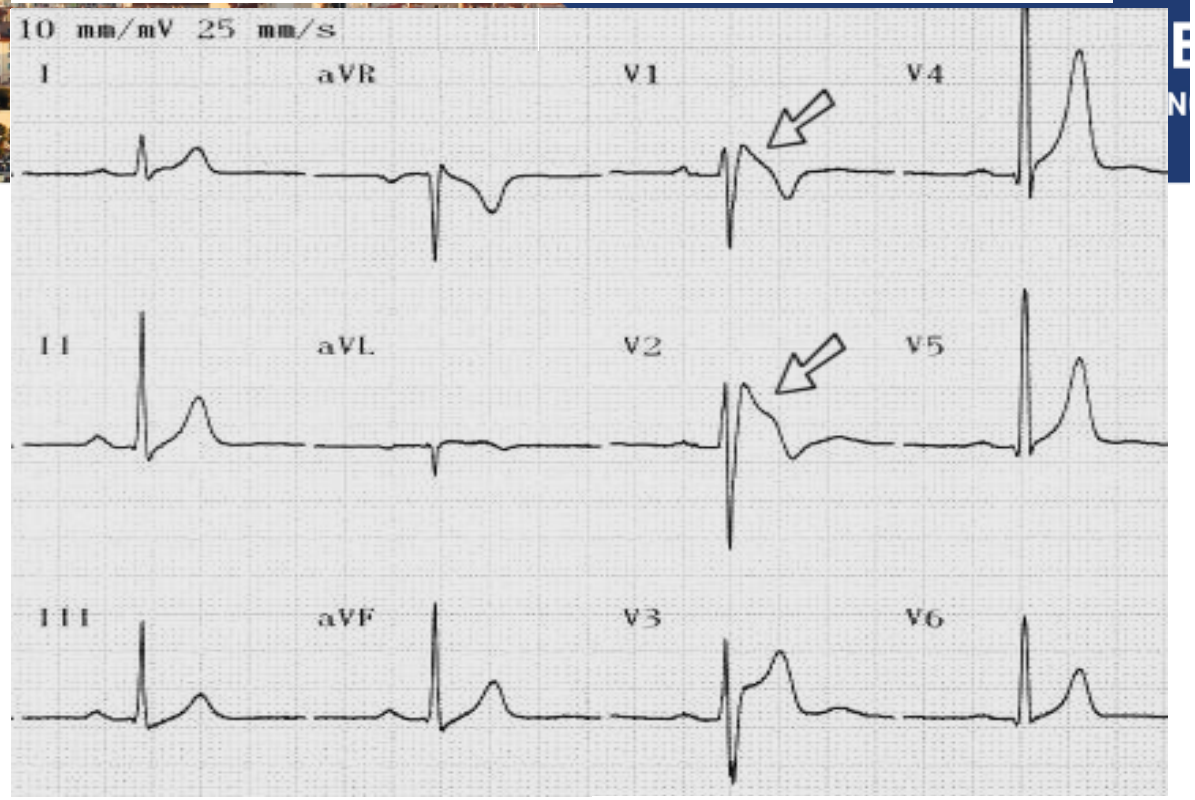
Syndrome de Brugada

Maladie rare (5/10 000 chez l'adulte)
Prédominance masculine (9/10)
Age moyen 41 ±15 ans

Substrat d'arythmies ventriculaires

- Syncopes
- Arrêts cardiaques récupérés / Morts subites
  - <4% de l'ensemble de mort subite
  - Mais jusqu'à 20 des MS sur cœur « sain »

2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

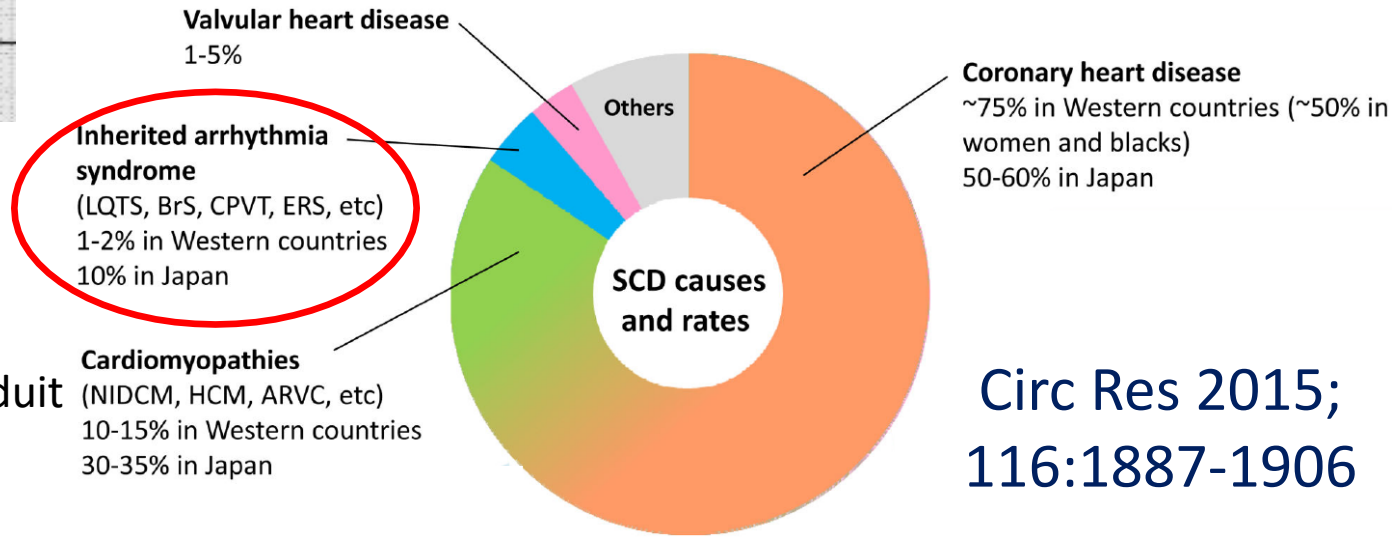


Recommendations	Class[a]	Level[b]
Brugada syndrome is diagnosed in patients with ST-segment elevation with type 1 morphology ≥2 mm in one or more leads among the right precordial leads V1 and/or V2 positioned in the second, third, or fourth intercostal space, occurring either spontaneously or after provocative drug test with intravenous administration of sodium channel blockers (such as ajmaline, flecainide, procainamide or pilsicainide).	I	C

Sus-décalage ST

- type 1
- ≥ 2 mm
- V1V2V3 ou V1V2 Hautes
- spontané ou pharmaco-induit

Eliminer phénocopie



Circ Res 2015; 116:1887-1906

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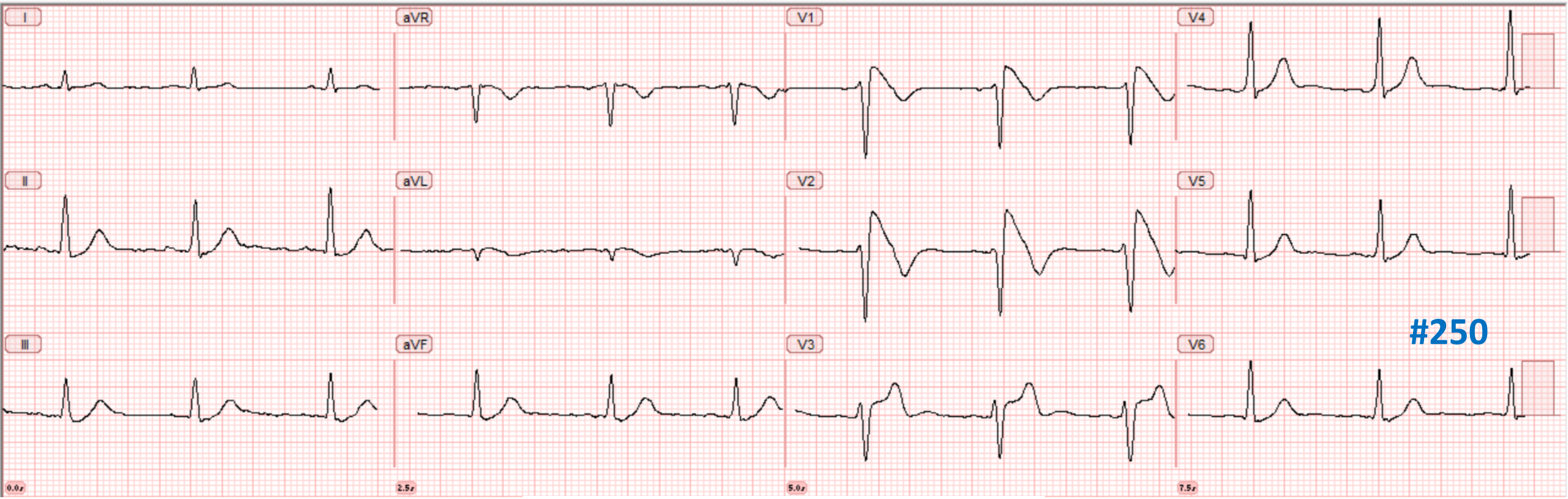

Aspect ECG de Brugada
Type 1 spontané

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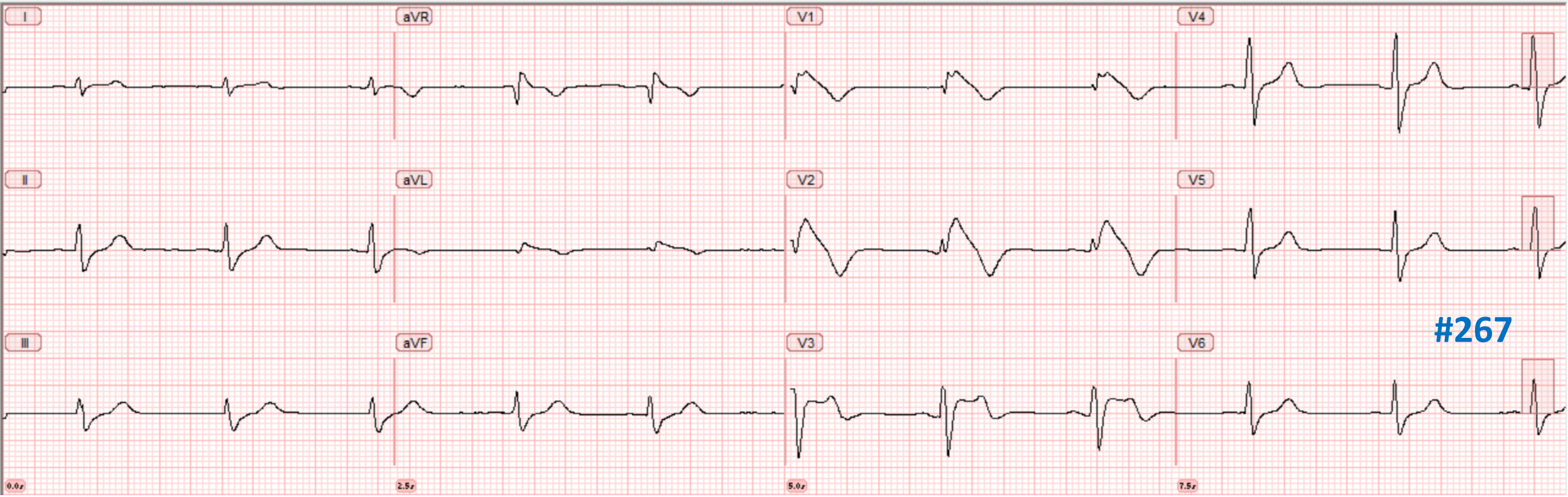


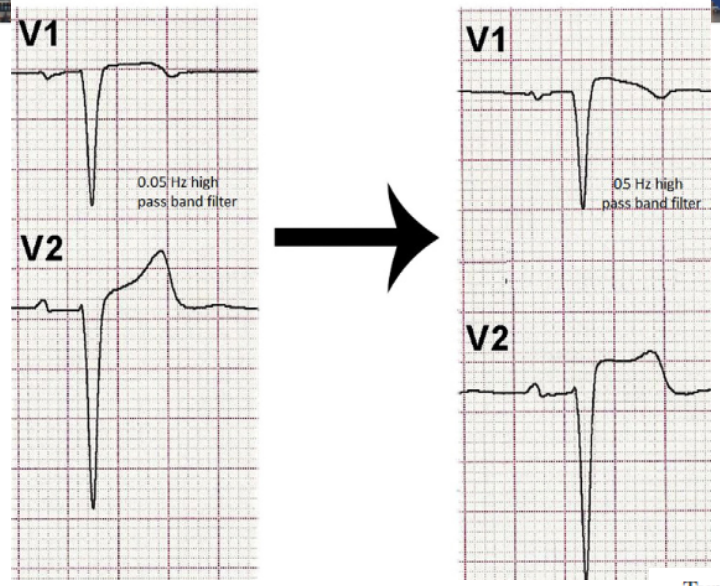
Aspect ECG de Brugada
Type 1 spontané

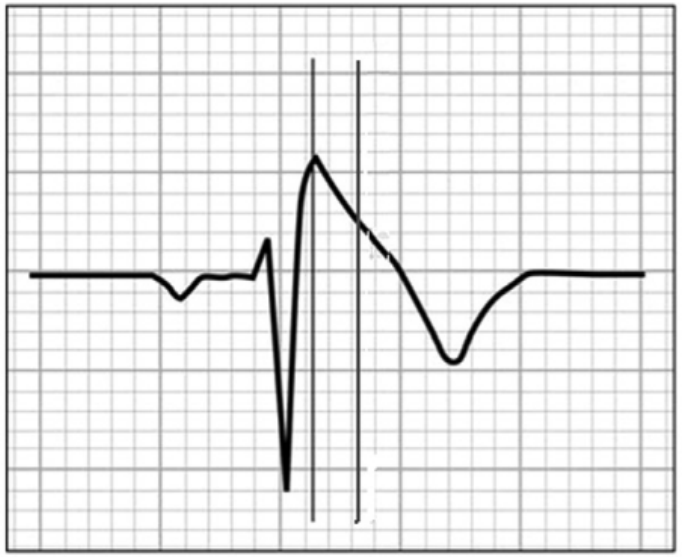
Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report☆

Antonio Bayés de Luna, MD, PhD,[a,*] Josep Brugada, MD, PhD,[b] Adrian Baranchuk, MD,[c] Martin Borggrefe, MD,[d] Guenter Breithardt, MD,[e] Diego Goldwasser, MD,[a] Pier Lambiase, MD,[f] Andrés Pérez Riera, MD, PhD,[g] Javier Garcia-Niebla, RN,[h] Carlos Pastore, MD, PhD,[i] Giuseppe Oreto, MD,[j] William McKenna, MD,[f] Wojciech Zareba, MD, PhD,[k] Ramon Brugada, MD, PhD,[l] Pedro Brugada, MD, PhD[m]

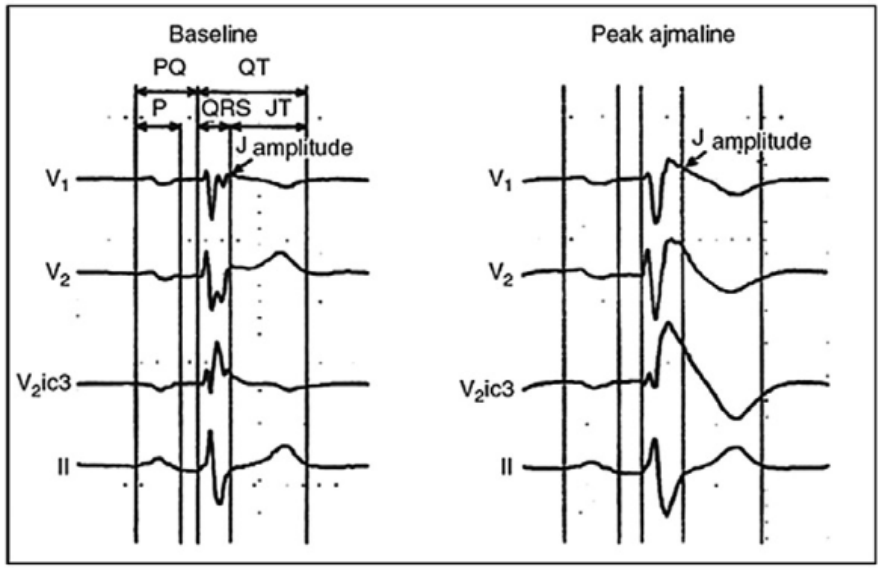
J Electrocardiol. 2012;45:433-442

55 years old female with hypertension

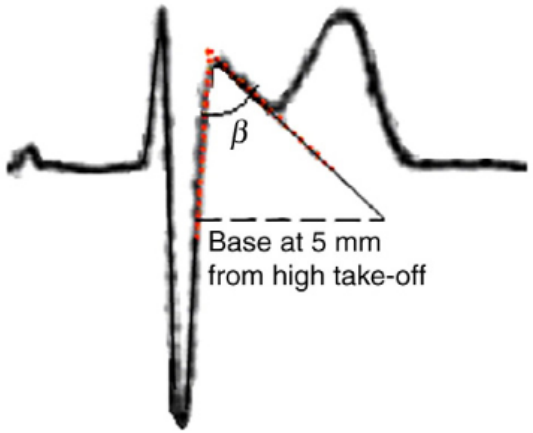




Corrado et al. Eu Heart J 2010: 31;243



Type 2: saddle-back pattern



This typical saddle-back pattern present in V1-V2 the following:

a. High take-off of r' (that often does not coincide with J point) ≥2 mm.

b. Descending arm of r' coincides with beginning of ST (often is not well seen).

c. Minimum ST ascent ≥0.5 mm

d. ST is followed by positive T wave in V2 (T peak > ST minimum > 0) and of variable morphology in V1.

e. The characteristics of triangle formed by r' allow to define different criteria useful for diagnosis (see above and text).

- β angle.[26]
- Duration of the base of the triangle of r' at 5 mm from the high take-off greater than 3.5mm[31]

f. The duration of QRS is longer in BrP type 2 than in other cases with r' in V1, and there is a mismatch between V1 and V6 (see text).

J-Wave syndromes expert consensus conference report: Emerging concepts and gaps in knowledge

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Table 3 **Differential diagnosis and modulating factors in Brugada syndrome**

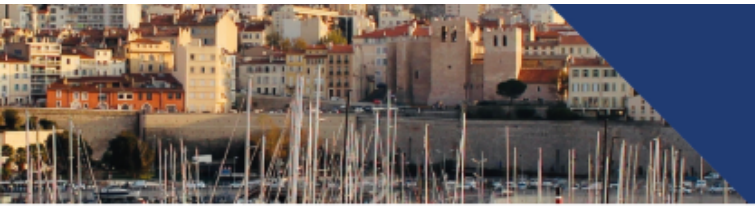
B. Modulating factors

- Electrolyte abnormalities:
  - Hyperkalemia
  - Hypokalemia
  - Hypercalcemia
  - Hyponatremia
- Temperature: hyperthermia (fever), hypothermia
- Hypertestosteronemia
- Treatment with:
  - Antiarrhythmic drugs: sodium channel blockers (Class IC, Class IA), calcium antagonists, beta-blockers
  - Antianginal drugs: calcium antagonists, nitrates, potassium channel openers
  - Psychotropic drugs: tricyclic/tetracyclic antidepressants, phenothiazines, selective serotonin reuptake inhibitor, lithium, benzodiazepines
  - Anesthetics/analgesics: propofol, bupivacaine, procaine
  - Others: histamine H_1 antagonist, alcohol intoxication, cocaine, cannabis, ergonovine

A. Differential diagnosis

- Atypical right bundle branch block
- Ventricular hypertrophy
- Early repolarization (especially in athletes)
- Acute pericarditis/myocarditis
- Acute myocardial ischemia or infarction (especially of the right ventricle)
- Pulmonary thromboembolism
- Prinzmetal angina
- Dissecting aortic aneurysm
- Central and autonomic nervous system abnormalities
- Duchenne muscular dystrophy
- Friedreich ataxia
- Spinobulbar muscular atrophy
- Myotonic dystrophy
- Arrhythmogenic right ventricular dysplasia
- Mechanical compression of the right ventricular outflow tract (e.g., pectus excavatum, mediastinal tumor, hemopericardium,
- Hypothermia
- Postdefibrillation ECG

J-Wave syndromes expert consensus conference report: Emerging concepts and gaps in knowledge



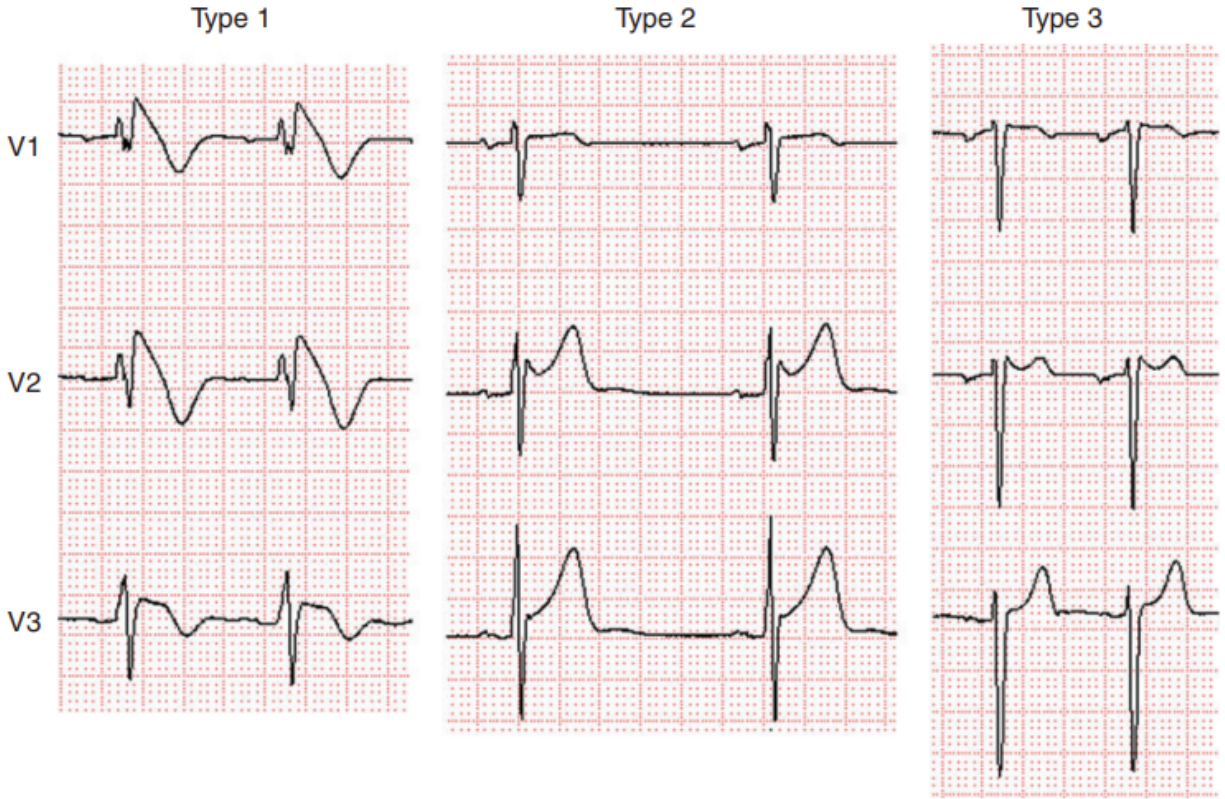
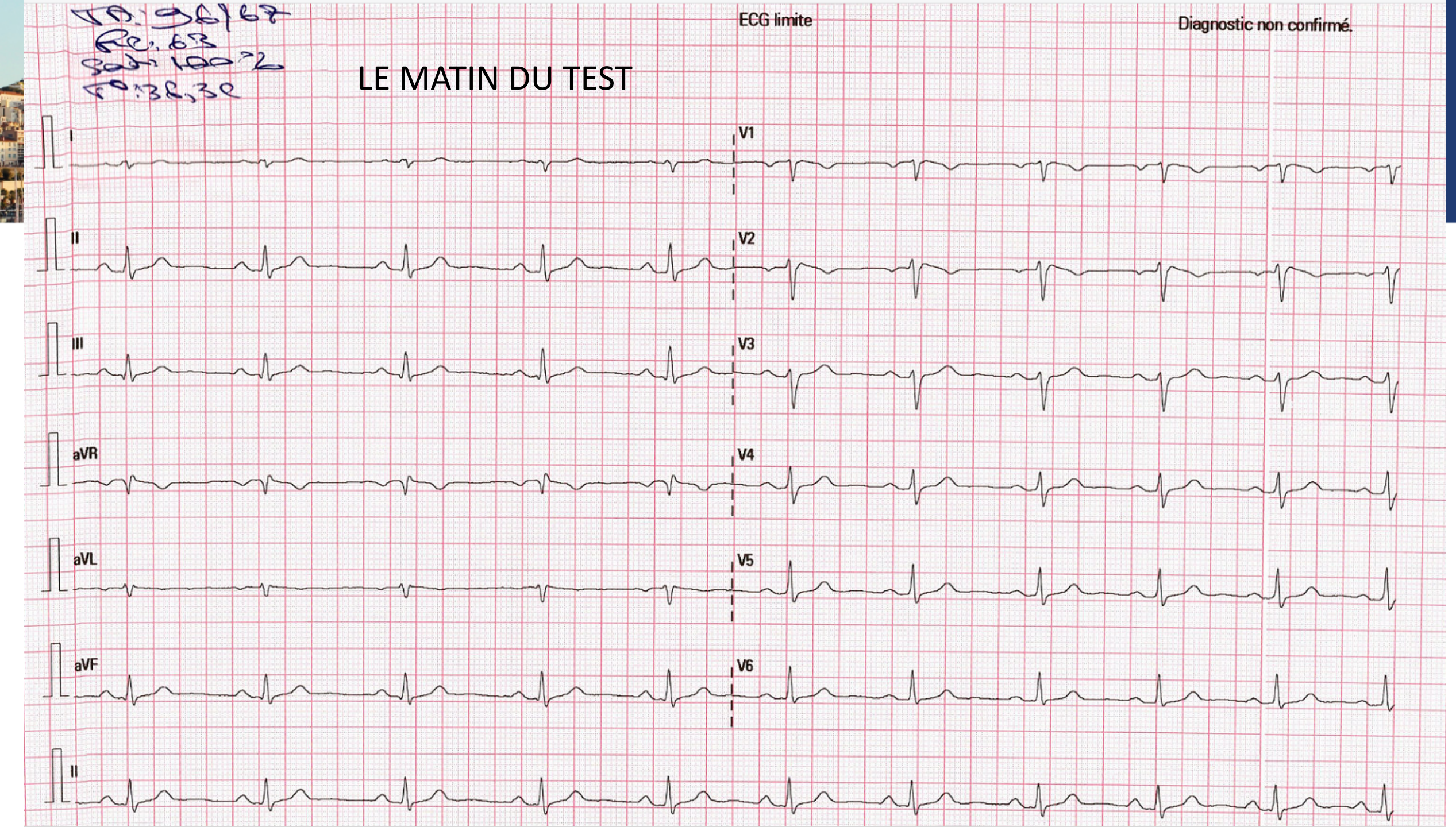
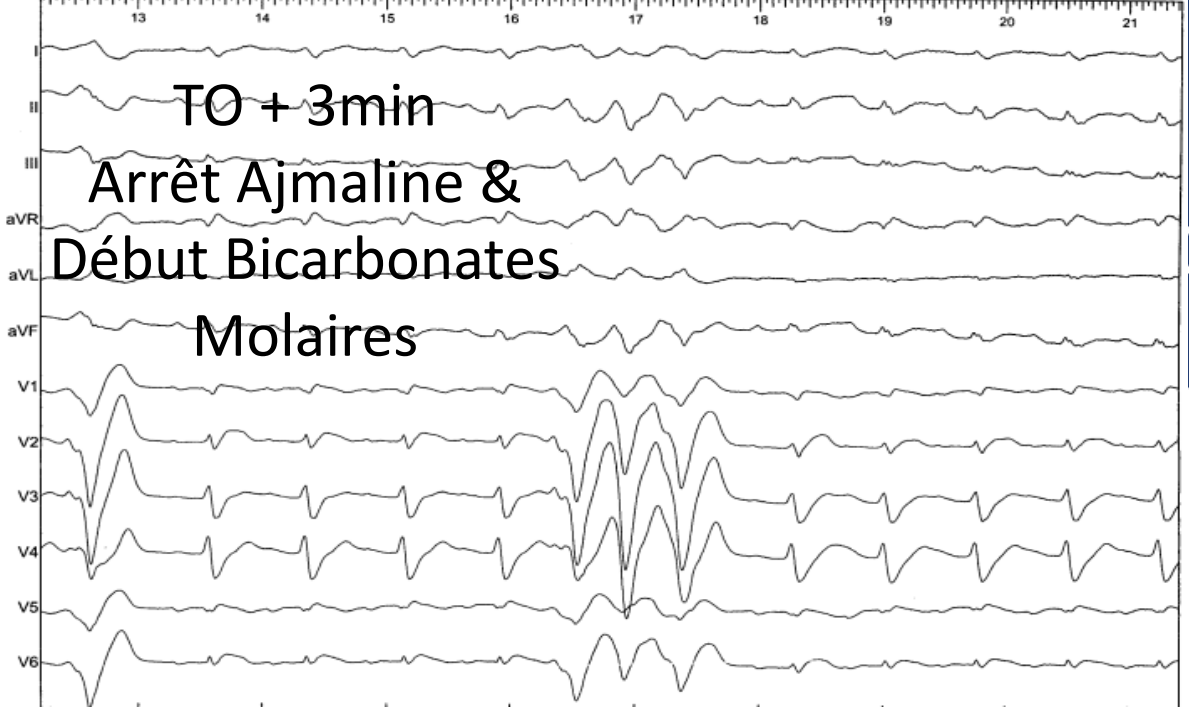


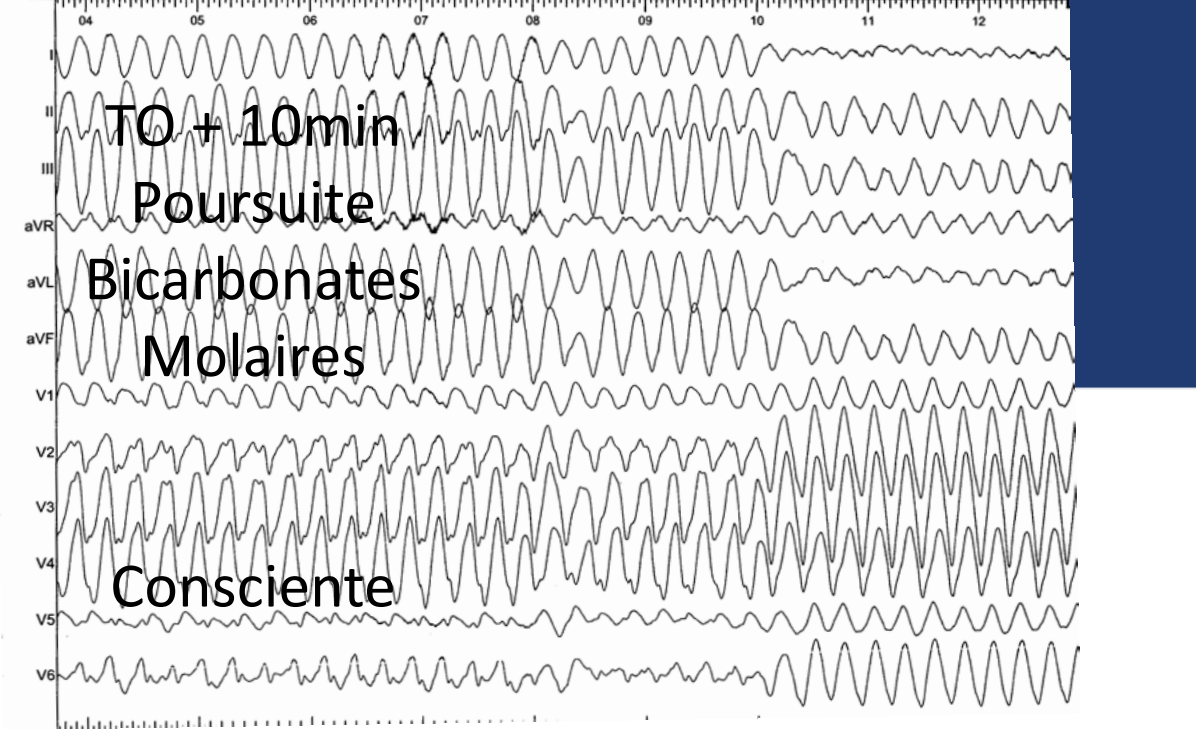
Table I Drugs used to unmask the Brugada ECG

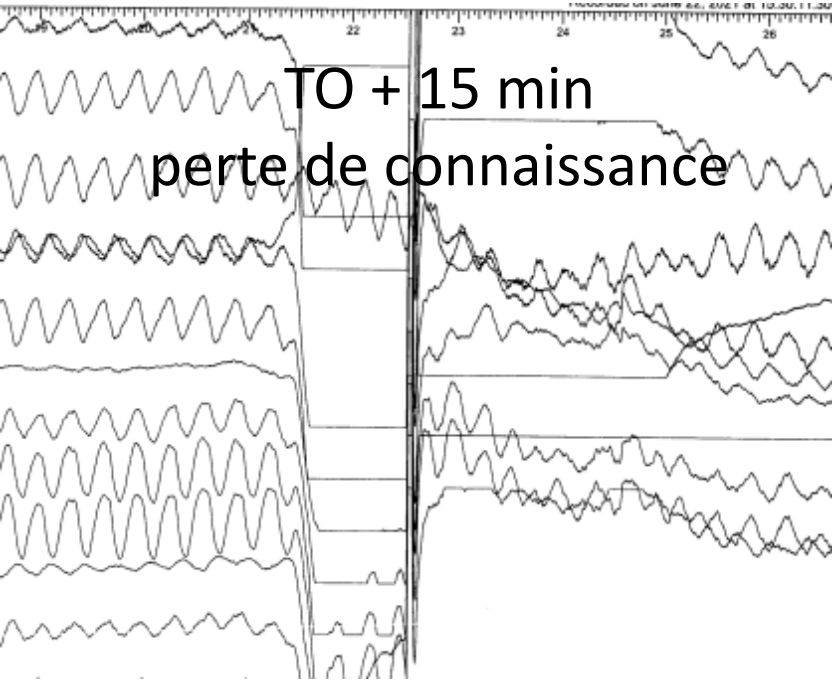
Drug	Dose	Administration
Ajmaline	1 mg/kg over 10 minutes	Intravenous
Flecainide	2 mg/kg over 10 minutes 200–300 mg	Intravenous Oral (41 hour)
Procainamide	10 mg/kg over 10 minutes	Intravenous
Pilsicainide	1 mg/kg over 10 minutes	Intravenous

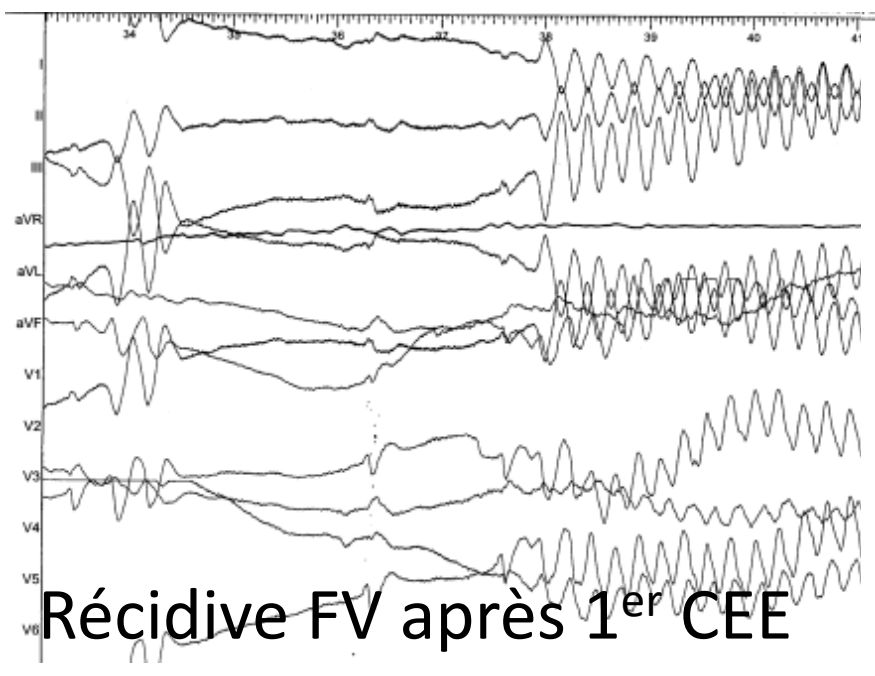
LE MATIN DU TEST

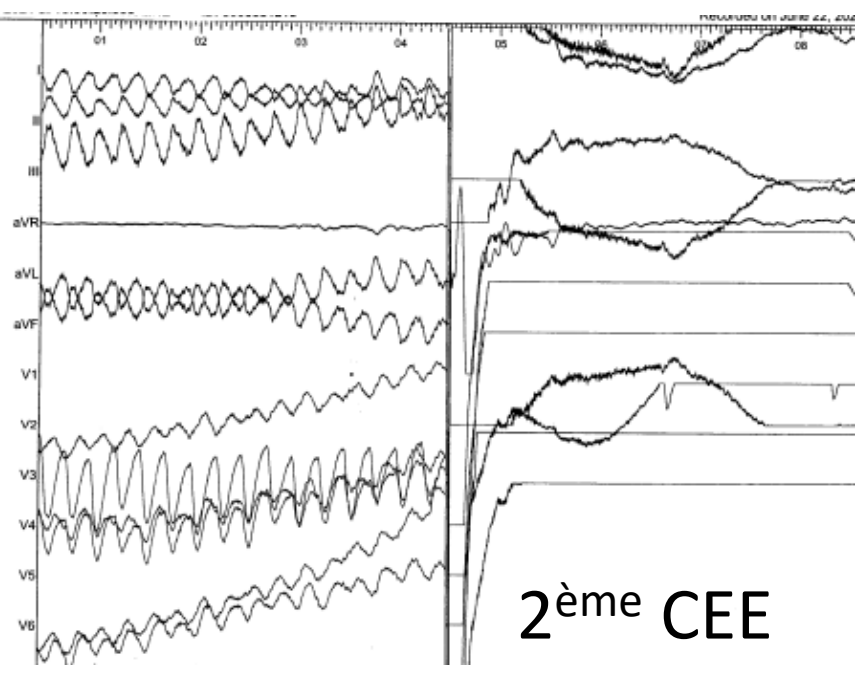



TO + 3min
Arrêt Ajmaline &
Début Bicarbonates
Molaires


TO + 10min
Poursuite
Bicarbonates
Molaires
Consciente


TO + 15 min
perte de connaissance


Récidive FV après 1er CEE


2ème CEE

The diagnostic role of pharmacological provocation testing in cardiac electrophysiology: a clinical consensus statement of the European Heart Rhythm Association and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC, the ESC Working Grou Pharmacother Paediatric and the Paediatric Society (PACE (HRS), the Asi (APHRS), and Society (LAHF

Elijah R. Behr (Chair)[1,2,3]*, Bo Gregers Winkel[4,5]†, Bode Ensam[1,6]†, Alberto Alfie (LAHRS)[7], Elena Arbelo[5,8,9,10], Colin Berry (EAPCI)[11], Marina Cerrone (HRS)[12], Giulio Conte[13], Lia Crotti[14,15], Cecilia M. Gonzalez Corcia (PACES)[16], Juan Carlos Kaski (Cardio Pharma WG)[1], Koonlawee Nademanee (APHRS)[17], Pieter G. Postema[5,18], Silvia Priori[5,19,20], Vincent Probst[5,21], Georgia Sarquella-Brugada (AEPC)[5,22], Eric Schulze-Bahr[5,23], Rafik Tadros[24], Arthur Wilde[5,21], and Jacob Tfelt-Hansen (Co-Chair)[4,5,25]

Europace 2025; 27:euaf067

Suspected Brugada syndrome (BrS) considered for SCB testing in the context of at least one of the following

- Cardiac arrest or syncope
- Family history of BrS
- Family history of sudden unexplained death
- Type 2/3 Brugada ECG pattern with other ECG features and/or one of the above

No → **Do not perform SCB test**

Yes ↓

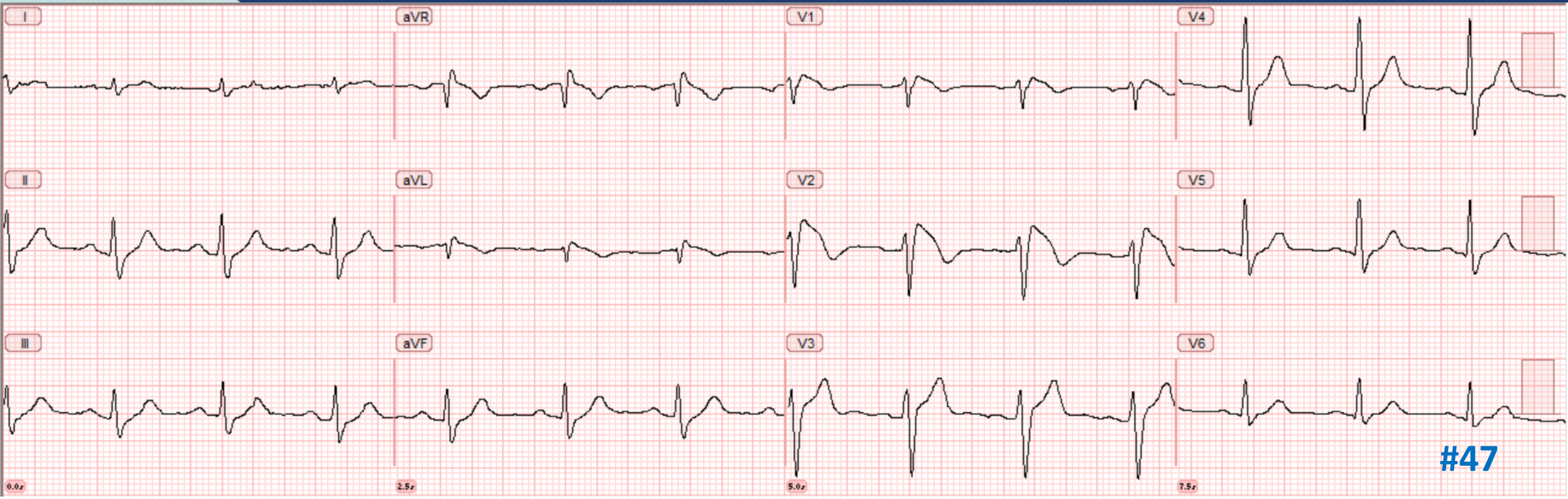
Patient counselling of pros and cons before taking consent for SCB test

Advantages of performing SCB testing

- Excludes BrS in presence of a negative test, especially when using ajmaline
- Avoids diagnostic ambiguity
- Guides extended family screening
- Informs on safety of sodium channel blocker use in patients who require such drugs
- Informs of need for suppressing fever

Disadvantages of performing SCB testing

- Limited specificity (e.g. ajmaline) and sensitivity (e.g. procainamide)
- A positive test can generate anxiety and unnecessary interventions despite favourable prognosis in asymptomatic patients
- Potential negative impact on insurability
- Procedural risk especially for patients with a pathogenic *SCN5A* variant



Aspect ECG de Brugada
Type 1 spontané

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When not to perform SCB provocation	Strength of evidence
Do not perform a diagnostic SCB test when a type 1 Brugada pattern has already been documented in the absence of suspected phenocopy.	

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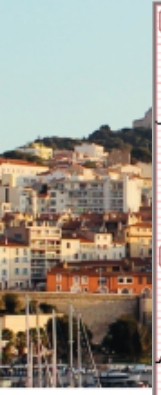
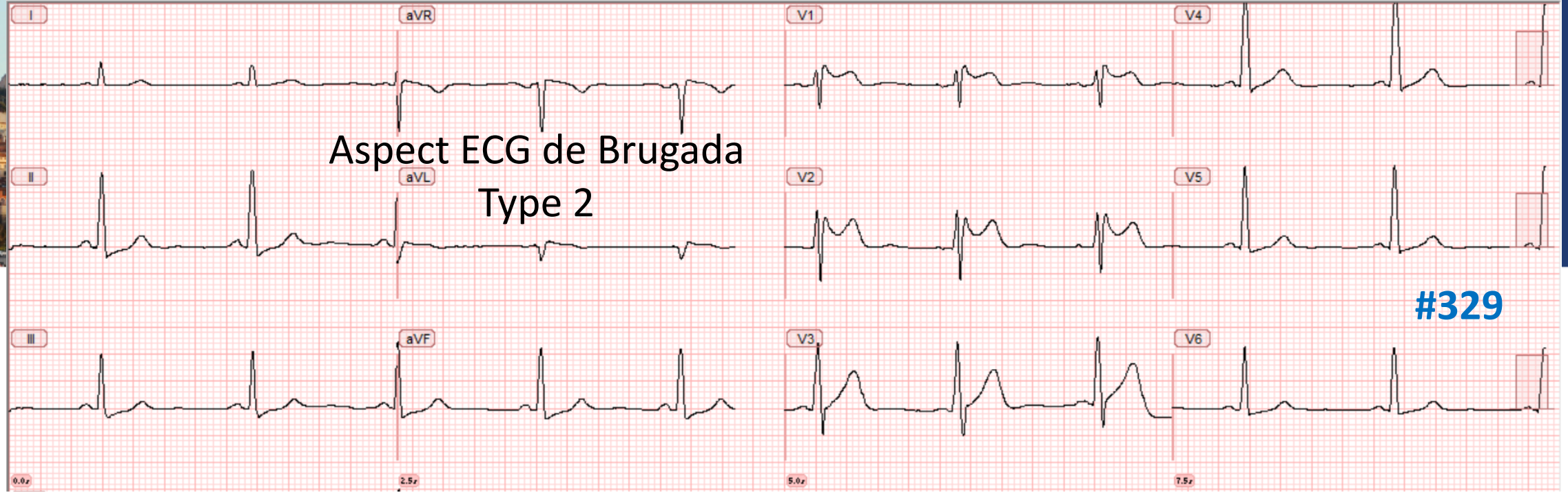
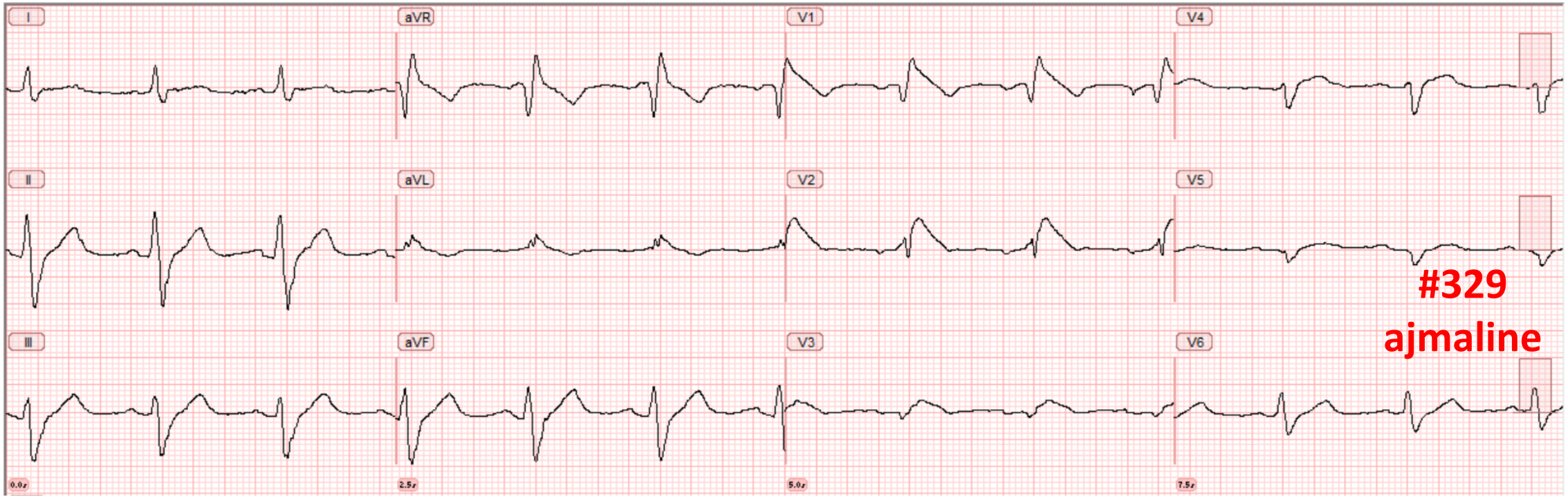
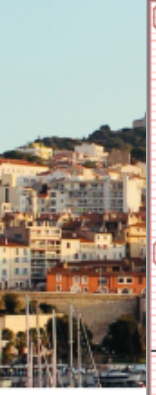


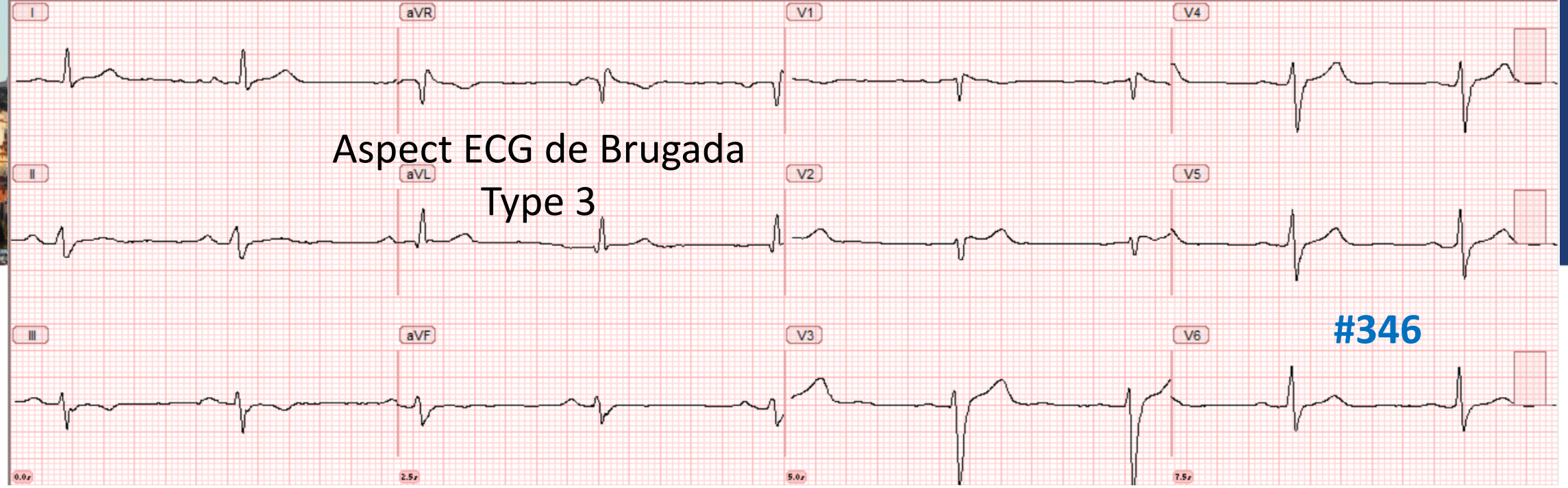
Aspect ECG de Brugada Type 2







Aspect ECG de Brugada Type 3



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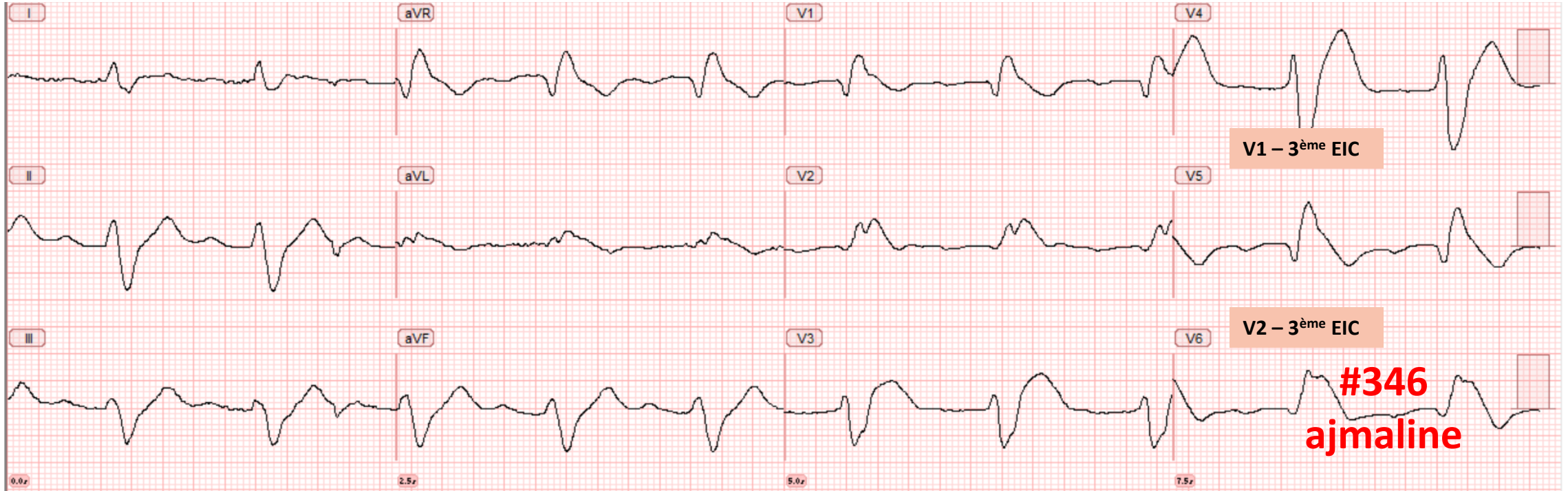

V1 – 3ème EIC

V2 – 3ème EIC

#346

ajmaline

The diagnostic role of pharmacological provocation testing in cardiac electrophysiology: a clinical consensus statement of the European Heart Rhythm Association and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC, the ESC Working Group on Cardiovascular Pharmacotherapy, the Association of European Paediatric and Congenital Cardiology (AEPC), the Paediatric & Congenital Electrophysiology Society (PACES), the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), and the Latin American Heart Rhythm Society (LAHRS)

Europace 2025; 27: euaf067

Continued

When to perform SCB provocation	Strength of evidence
An SCB test is advised for a patient with a type 2/3 Brugada ECG pattern and a history of cardiac or suspected cardiac syncope in the absence of significant structural heart disease.	>90% agree
An SCB test is advised in a first-degree relative of a SADS[a] decedent whose circumstances of death are suggestive of BrS-related death (i.e. in sleep, during	

Areas of uncertainty	Strength of evidence
It is uncertain whether it is appropriate to offer an SCB test to genotype-negative subjects from *SCN5A* families.	>70% agree

Continued

Areas of uncertainty	Strength of evidence
It is uncertain whether it is appropriate to form an SCB test in an asymptomatic degree relative of an index patient only has a drug-induced or -induced type 1 Brugada ECG ern and no other ECG features, al or family history supportive of	>90% agree
certain whether it is appropriate to orm an SCB test on a person aged r 30 presenting with atrial lation for no other reason.	>70% agree

When to perform SCB provocation	Strength of
It is advised that all patients undergoing an SCB test are counselled about the advantages and disadvantages of testing, including the generally low lifetime risk of life-threatening arrhythmia if asymptomatic, and the possibility of a false positive or false negative result.	>90%
An SCB test is advised for a patient with VF or polymorphic VT that remains unexplained following comprehensive clinical testing.	
An SCB provocation test is advised in an asymptomatic first-degree relative of an index patient with definite *SCN5A*-negative BrS.	>90%
An SCB provocation test may be appropriate to aid segregation analysis in relatives with a rare variant of uncertain significance in *SCN5A* and symptoms and/or a family history of BrS ± sudden death.	>90%

When not to perform SCB provocation	Strength of evidence
Do not perform a diagnostic SCB test when a type 1 Brugada pattern has already been documented in the absence of suspected phenocopy.	>90% agree
Do not routinely perform an SCB test in asymptomatic subjects with an incidental finding of type 2/3 pattern and no other ECG features, clinical or family history supportive of BrS.	>70% agree

include SCB provocation (preferably ajmaline) to enable determination of the size of the substrate.

not to perform SCB ation	Strength of evidence
perform a diagnostic SCB test a type 1 Brugada pattern has y been documented in the ce of suspected phenocopy. routinely perform an SCB test in ptomatic subjects with an ntal finding of type 2/3 pattern and no other ECG features, clinical or family history supportive of BrS.	>90% agree >70% agree

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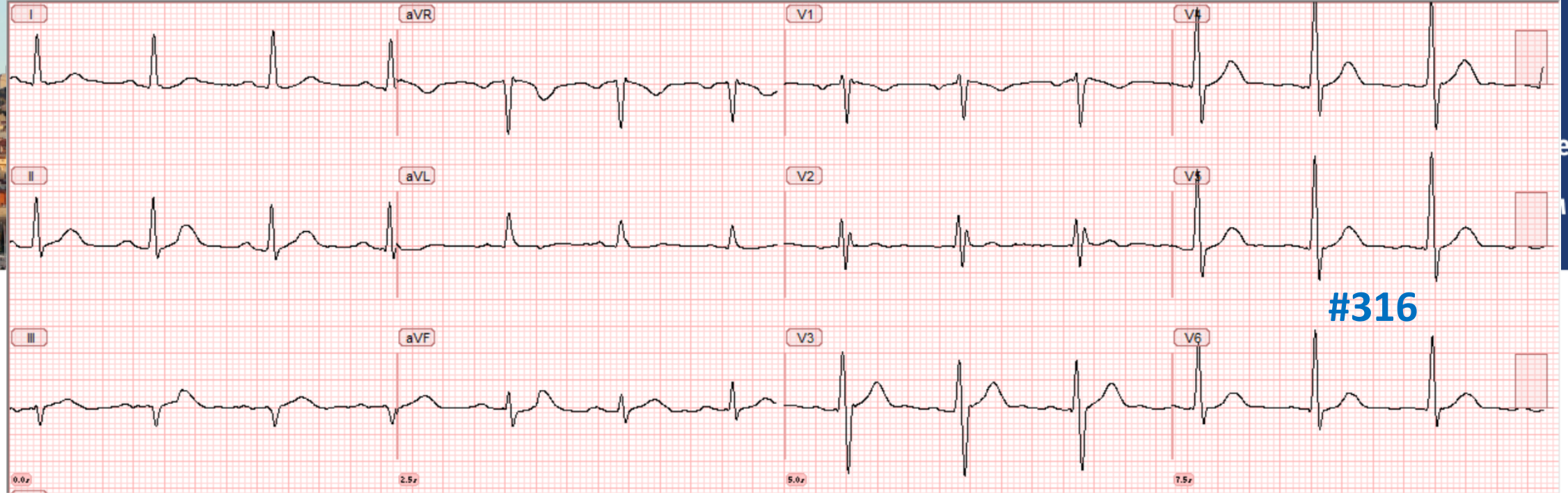
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V2
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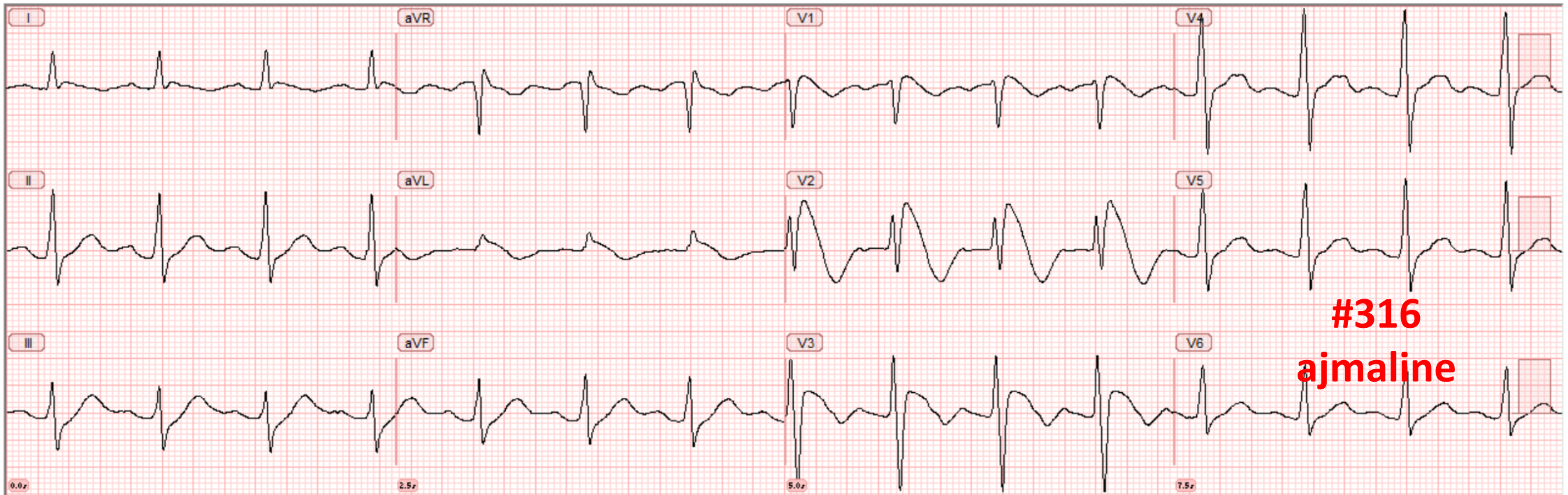
III
aVF
V3
V6
#316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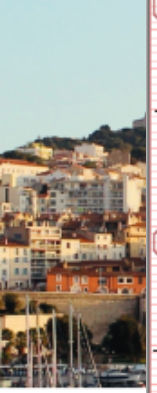
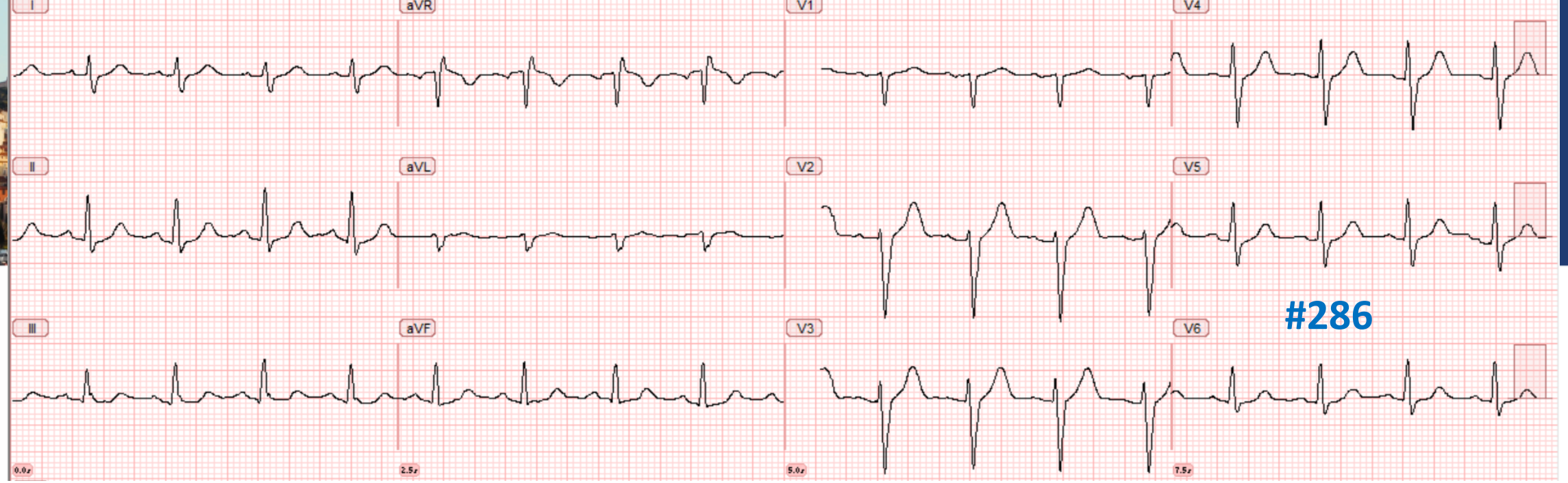
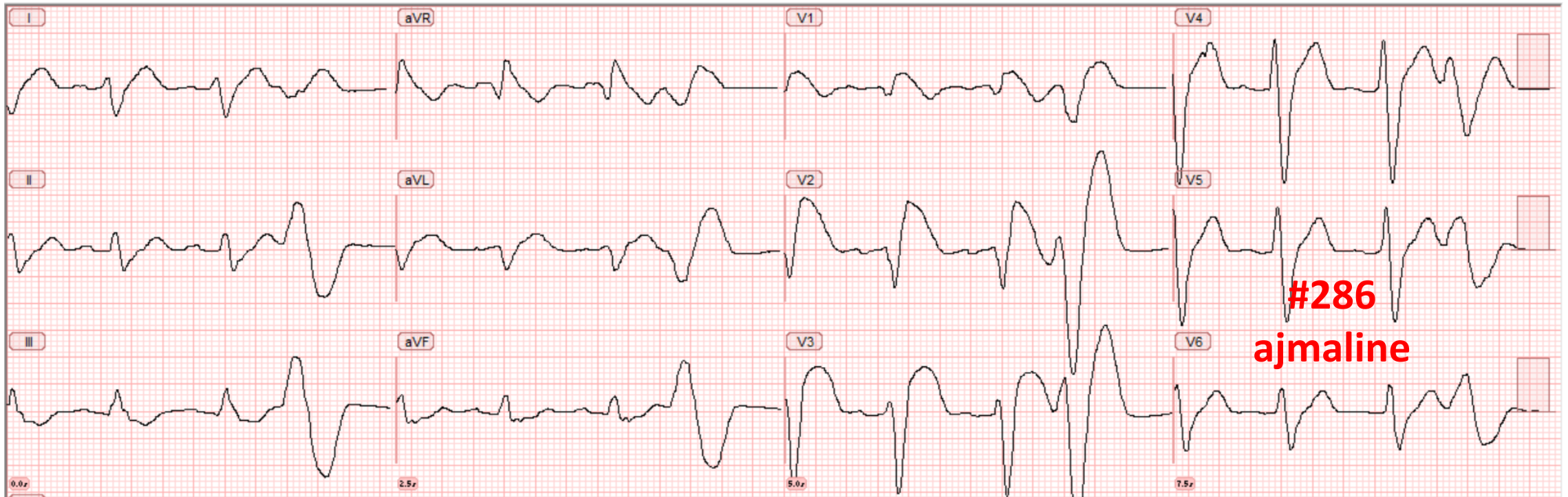
ajmaline




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
#286


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
#286
ajmaline

The diagnostic role of pharmacological provocation testing in cardiac electrophysiology: a clinical consensus statement of the European Heart Rhythm Association and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC, the ESC Working Group on Cardiovascular Pharmacotherapy, the Association of European Paediatric and Congenital Cardiology (AEPC), the Paediatric & Congenital Electrophysiology Society (PACES), the Heart Rhythm Society (HRS), the Asia Pacific Heart Rhythm Society (APHRS), and the Latin American Heart Rhythm Society (LAHRS)

Europace 2025; 27: euaf067

When to perform SCB provocation	Strength of evidence
It is advised that all patients undergoing an SCB test are counselled about the advantages and disadvantages of testing, including the generally low lifetime risk of life-threatening arrhythmia if asymptomatic, and the possibility of a false positive or false negative result.	>90% agree
An SCB test is advised for a patient with VF or polymorphic VT that remains unexplained following comprehensive clinical testing.	
An SCB provocation test is advised in an asymptomatic first-degree relative of an index patient with definite *SCN5A*-negative BrS.	>90%
An SCB provocation test may be appropriate to aid segregation analysis in relatives with a rare variant of uncertain significance in *SCN5A* and symptoms and/or a family history of BrS ± sudden death.	>90%

Continued

When to perform SCB provocation	Strength of evidence
An SCB test is advised for a patient with a type 2/3 Brugada ECG pattern and a history of cardiac or suspected cardiac syncope in the absence of significant structural heart disease.	>90% agree
An SCB test is advised in a first-degree relative of a SADS[a] decedent whose circumstances of death are suggestive of BrS-related death (i.e. in sleep, during fever, and/or a suspicious ECG in the decedent). Comprehensive assessment and exclusion of alternative causes in the relative is required.	
An SCB test may be appropriate in a first-degree relative of a SADS[a] decedent where comprehensive	
include SCB provocation (preferably ajmaline) to enable determination of the size of the substrate.	

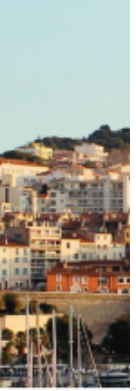
Areas of uncertainty	Strength of evidence
It is uncertain whether it is appropriate to offer an SCB test to genotype-negative subjects from *SCN5A* families.	>70% agree

Continued

Areas of uncertainty	Strength of evidence
It is uncertain whether it is appropriate to perform an SCB test in an asymptomatic first-degree relative of an index patient who only has a drug-induced or fever-induced type 1 Brugada ECG pattern and no other ECG features, clinical or family history supportive of BrS.	>90% agree
It is uncertain whether it is appropriate to perform an SCB test on a person aged under 30 presenting with atrial fibrillation for no other reason.	>70% agree

When not to perform SCB provocation	Strength of evidence
Do not perform a diagnostic SCB test when a type 1 Brugada pattern has already been documented in the absence of suspected phenocopy.	>90% agree
Do not routinely perform an SCB test in asymptomatic subjects with an incidental finding of type 2/3 pattern and no other ECG features, clinical or family history supportive of BrS.	>70% agree

An SCB provocation test is advised in an asymptomatic first-degree relative of an index patient with definite *SCN5A*-negative BrS.	>90% agree
An SCB provocation test may be appropriate to aid segregation analysis in relatives with a rare variant of uncertain significance in *SCN5A* and symptoms and/or a family history of BrS ± sudden death.	>90% agree



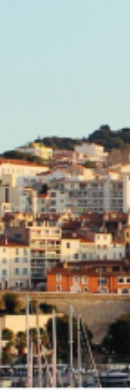
2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

General recommendations		
The following is recommended in all patients with BrS: (a) Avoidance of drugs that may induce ST-segment elevation in right precordial leads (http://www.brugadadrugs.org). (b) Avoidance of cocaine, cannabis, and excessive alcohol intake. (c) Treatment of fever with antipyretic drugs.	I	C
Risk stratification, prevention of SCD and treatment of VA		

2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

Table 7 Genetic tests and suggested work-up of probands and relatives with primary electrical diseases

			LQTS	BrS	CPVT	Idiopathic VF	ERS
	Genetic test		Class I[a]	Class I	Class I[a]	Class IIb	Class IIb
Proband	**Initial clinical test**	**Cornerstone for diagnosis**	ECG Exercise test	ECG and high precordial lead ECG Sodium channel blockers provocative test[c]	Exercise test	See *Section 5.2.3*, scenario 3	ECG
		Other tests/processes	Exclude acquired LQTS	Exclude phenocopy[b]	Exclude phenocopy[b]/SHD		Holter Echocardiography
	Follow-up		1–3 years dependent on level of risk				
Relatives	**Clinical screening**		ECG Exercise test (when feasible) From birth	ECG and high precordial lead ECGs: start at 10 years Sodium channel blockers provocative test[c]: start >16 years unless clinically indicated[180,181]	ECG Exercise test From birth	ECG and high precordial lead ECGs Exercise test Echocardiogram[182]	ECG Echocardiogram
	Follow-up	Positive phenotype and/or Class IV/V variant	1–3 years dependent on level of risk				
		Negative phenotype and no Class IV/V variant	Discharge				



2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

When a putative causative variant is first identified, evaluation for pathogenicity is recommended using an internationally accepted framework.[176]	I	C
When a Class IV or Class V variant has been identified in a living or deceased individual with a condition that carries a risk of VA and SCD, genetic testing of first-degree and symptomatic relatives and obligate carriers is recommended.	I	C
It is recommended that genetic testing and counselling on its potential consequences should be undertaken by an expert multidisciplinary team.[179]	I	C
It is recommended that Class III (variants of uncertain significance) and Class IV variants should be evaluated for segregation in families where possible, and the variant re-evaluated periodically.	I	C

Centre de Référence des Maladies Cardiaques Héréditaires ou Rares

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maladies cardiaques héréditaires

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VILLA M. - MARSEILLE I FRANCE
19èmes journées françaises pratiques de rythmologie & de stimulation cardiaque
WWW.CONGRES-ELECTRA.COM

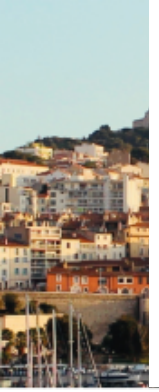
Patient asymptomatique et ECG pathologique : **Brugada** et QT long

- Penser et faire le diagnostic : type 1 – dérivations hautes
- Eliminer les diagnostics différentiels
- Test à l'ajmaline : réfléchi & sécurisé
- Prises en charge initiale et ultérieure : génétique / famille : médicaments & drogues / hyperthermie / stratification MS

ELECTRA
4-5 DÉCEMBRE 2025
VILLA M. - MARSEILLE I FRANCE
19èmes journées françaises pratiques de rythmologie & de stimulation cardiaque
WWW.CONGRES-ELECTRA.COM

Patient asymptomatique et ECG pathologique : Brugada et **QT long**

- Penser et faire le diagnostic : différents aspects ECG
- Eliminer les diagnostics différentiels
- Test de provocation : Quand ? Comment ?
- Prises en charge initiale et ultérieure



2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

7.2.2. Long QT syndrome (including acquired long QT syndrome)

(1) Autosomal-dominant LQTS (prevalence: 1 in 2500) without extra-cardiac manifestation.

(2) Autosomal-dominant LQTS with extra-cardiac manifestation, comprising:

  (a) Andersen–Tawil Syndrome (LQT7), increasingly considered its own entity.[933,934]

  (b) Timothy Syndrome (LQT8), characterized by prolonged QT, syndactyly, cardiac malformations, autism spectrum disorder and dysmorphism.[935]

(3) Autosomal-recessive LQTS (Jervell and Lange–Nielsen Syndrome), combining extreme QT prolongation with congenital deafness.[936]

2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

çaises
ologie
rdiaque

RA.COM

Score >3

=

LQTS diagnosis

Findings			Points
ECG	QTc	≥480 ms	3.5
		=460–479 ms	2
		=450–459 ms (in males)	1
		≥480 ms during 4th minute of recovery from exercise stress test	1
	Torsade de pointes		2
	T wave alternans		1
	Notched T wave in 3 leads		1
	Low heart rate for age		0.5
Clinical history	Syncope	With stress	2
		Without stress	1
Family history	Family member(s) with definite LQTS		1
	Unexplained SCD at age <30 years in first-degree family		0.5
Genetic finding	Pathogenic mutation		3.5

Transient causes excluded

rançaises
nmologie
cardiaque

CTRA.COM

File Tools Display Annotations Demographics Views Window Algorithm Help

Leads 12 Seconds 6 mVolts 2 Reports 6x2

I V1

II V2

III V3

aVR V4

aVL V5

aVF V6

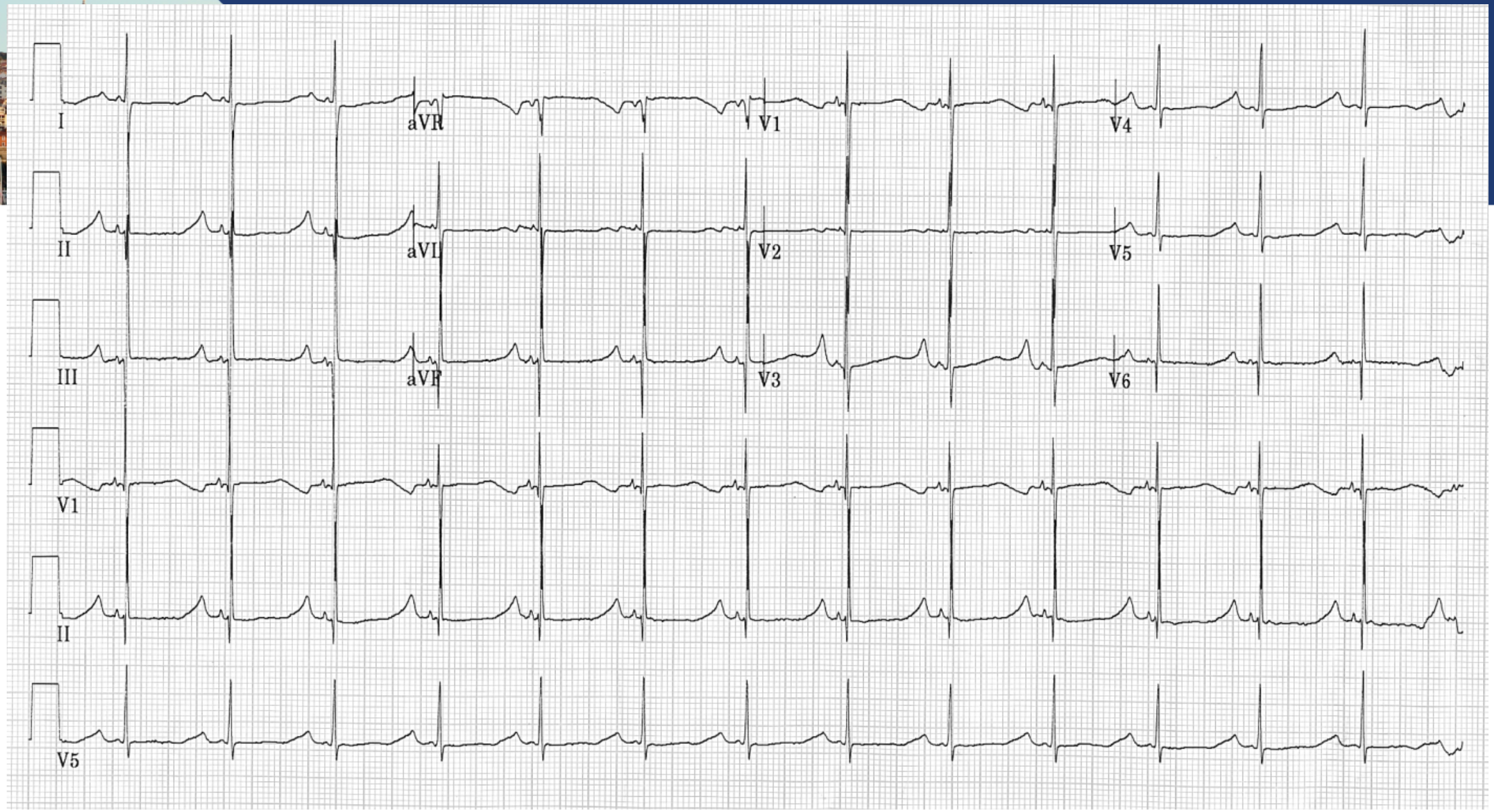
HR: 47 bpm | ECG Length: 10.0 s | SR: 250 Hz | Start: 0.0 s | SRes: 7.84 ms | PSratio: 0.51

File Tools Display Annotations Demographics Views Window Algorithm Help

Leads 12 Seconds 6 mVolts 2 Reports 6x2

I II III aVR aVL aVF V1 V2 V3 V4 V5 V6

HR: 51 bpm | ECG Length: 10.0 s | SR: 250 Hz | Start: 0.0 s | SRes: 7.84 ms | PSratio: 0.51


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Determination and Interpretation of the QT Interval

Comprehensive Analysis of a Large Cohort of Long QT Syndrome Patients and Controls

Circulation 2018; 138:2345-58

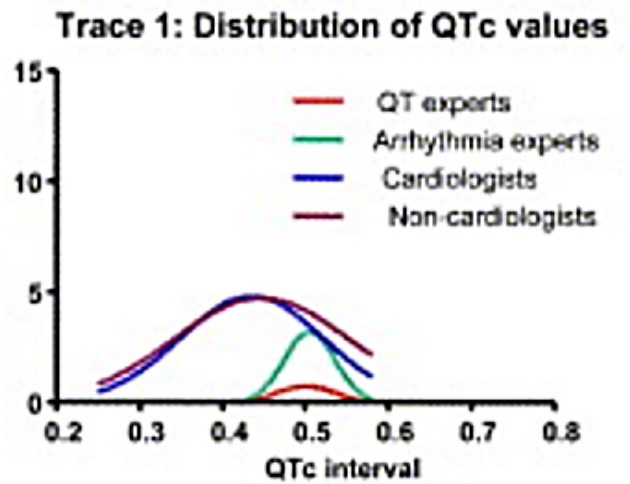
Tangent method

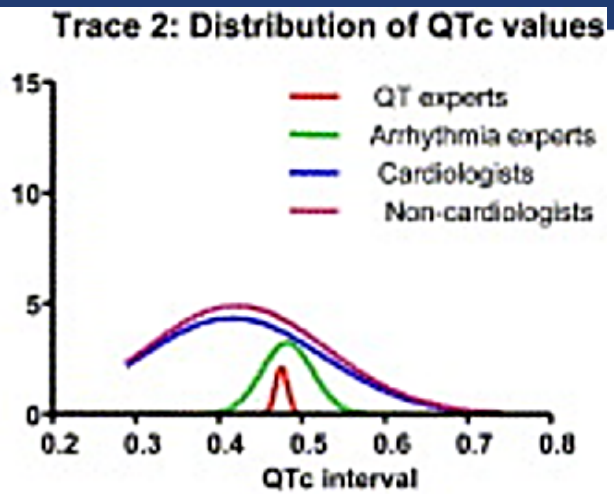
Threshold method

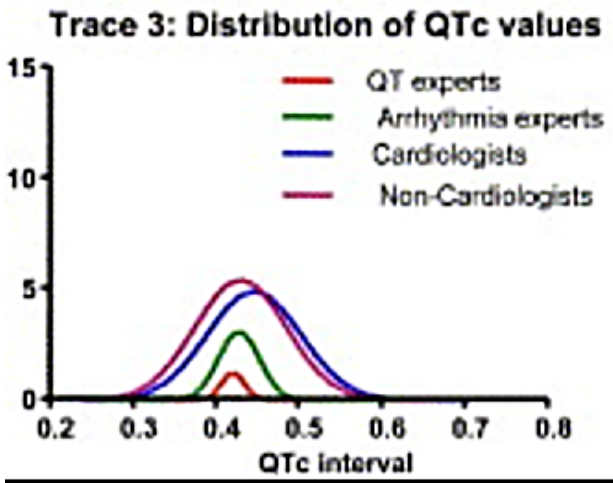
Inaccurate electrocardiographic interpretation of long QT: the majority of physicians cannot recognize a long QT when they see one

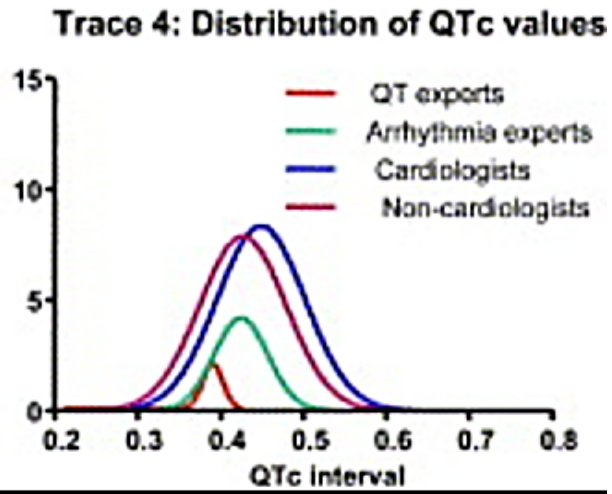
Sami Viskin [1], Uri Rosovski, Andrew J Sands, Edmond Chen, Peter M Kistler, Jonathan M Kalman, Laura Rodriguez Chavez, Pedro Iturralde Torres, Fernando E S Cruz F, Osmar A Centurión, Akira Fujiki, Philippe Maury, Xiaomin Chen, Andrew D Krahn, Franz Roithinger, Li Zhang, G Michael Vincent, David Zeltser

Heart Rhythm
2005;2:569-74









Determination and Interpretation of the QT Interval

Comprehensive Analysis of a Large Cohort of Long QT Syndrome Patients and Controls

Circulation 2018; 138:2345-58

Amsterdam UMC
Universitair Medische Centra

Department of Cardiology
Academic Medical Center
University of Amsterdam
The Netherlands

QT Calculator

for LQTS probability calculation

- Home
- How to measure the QT-interval
- How to use this calculator
- References

Please follow Step 1 to Step 6 for an indication of LQTS probability in your patient

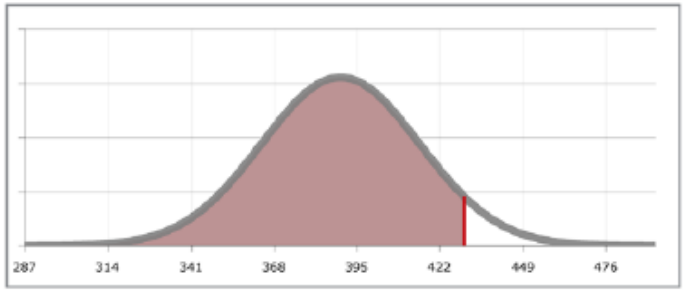
Step 1:	Gender	◉ Male ○ Female	
Step 2:	Age	35	Years
Step 3:	QT interval	430	milliseconds
Step 4:	Heart rate	60	beats / minute
	RR Interval	1000	milliseconds
Step 5:	QT method	◉ Tangent ○ Threshold	
Step 6:	QT correction	◉ Bazett : 430	
		○ Fridericia : 430	
		○ Framingham : 430	
		○ Hodges : 430	
		○ Rautaharju : 424	

Calculate

LQTS probability calculation

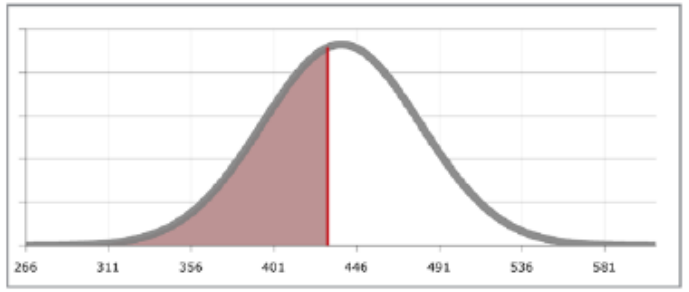
Occurrence in control subjects

In reference control subjects 94.26% of QTc values are below this level while 5.74% are above this QTc



Occurrence in LQTS patients

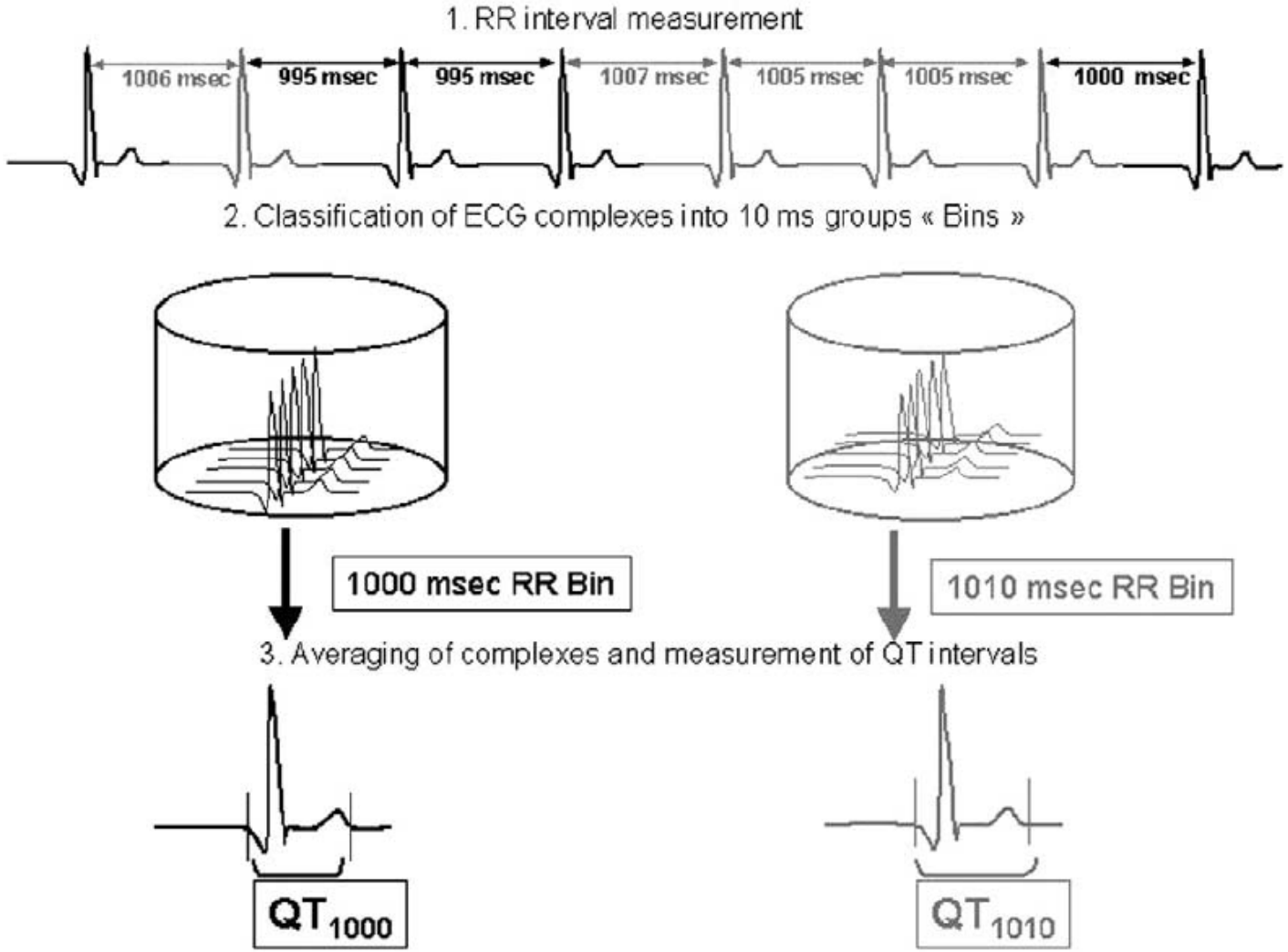
In reference LQTS patients 42.99% of QTc values are below this level while 57.01% are above this QTc



Please use these values to assess the probability of LQTS in your patient.

Clinical assessment of drug-induced QT prolongation in association with heart rate changes

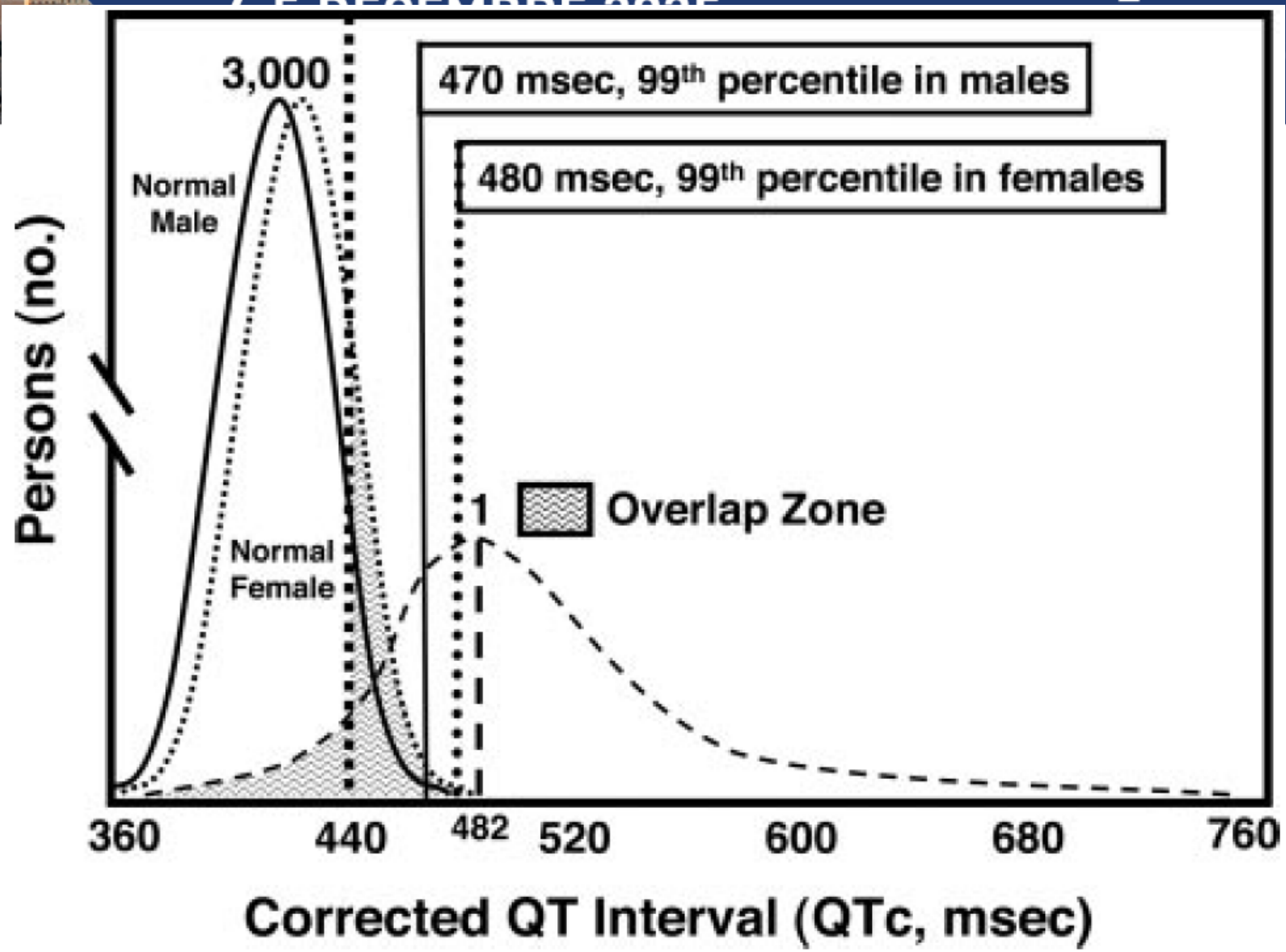
Fabrice Extramiana, MD, Pierre Maison-Blanche, MD, Marie-José Cabanis, PharmD, Catherine Ortemann-Renon, PharmD, PhD, Philippe Beaufils, MD, and Antoine Leenhardt, MD *Paris, France*



Diagnostic Miscues in Congenital Long-QT Syndrome

Nathaniel W. Taggart, MD; Carla M. Haglund; David J. Tester, BS; Michael J. Ackerman, MD, PhD

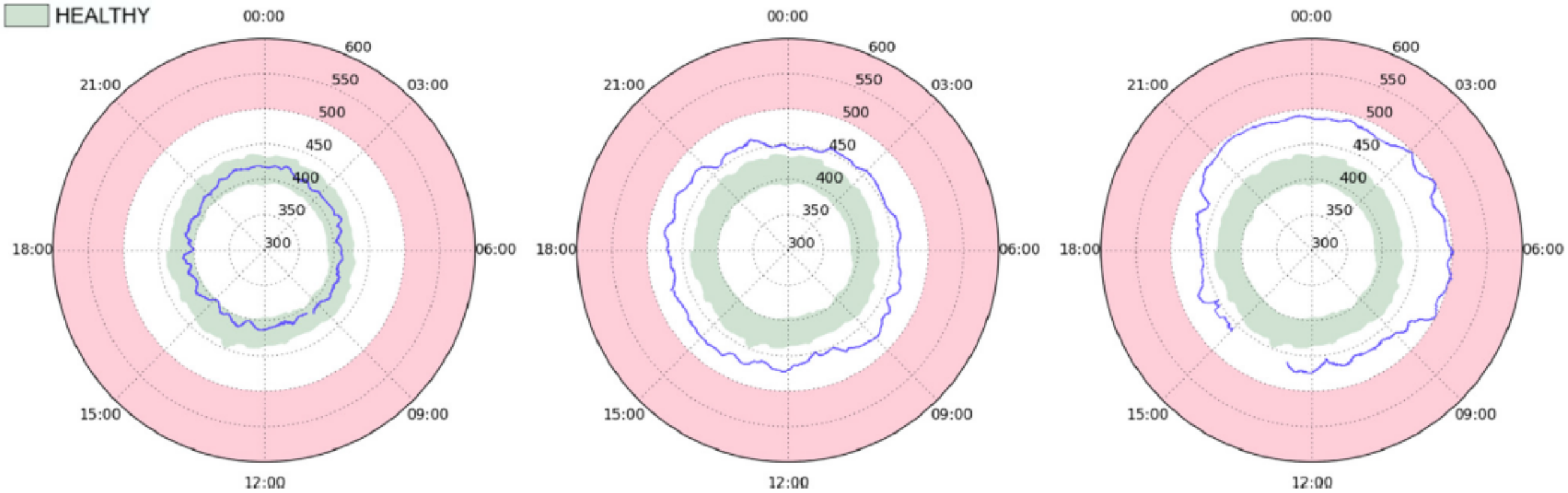
Circulation. 2007; 115:2613-2620



"QT clock" to improve detection of QT prolongation in long QT syndrome patients

Alex Page, MS,* Mehmet K. Aktas, MD, FHRS,† Tolga Soyata, PhD,*
Wojciech Zareba, MD, PhD,‡ Jean-Philippe Couderc, PhD*‡

Heart Rhythm. 2016;
13:190–198



24-hour Holter recordings from
202 LQT1, 89 LQT2, and 14 LQT3
200 healthy individuals

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Lying to Standing Mental stress test

Findings			Points
ECG	QTc	≥480 ms	3.5
		=460–479 ms	2
		=450–459 ms (in males)	1
		≥480 ms during 4th minute of recovery from exercise stress test	1
	Torsade de pointes		2
	T wave alternans		1
	Notched T wave in 3 leads		1
	Low heart rate for age		0.5
Clinical history	Syncope	With stress	2
		Without stress	1
Family history	Family member(s) with definite LQTS		1
	Unexplained SCD at age <30 years in first-degree family		0.5
Genetic finding	Pathogenic mutation		3.5

Table 4 Protocols for epinephrine testing

Progressive protocol ('Mayo')

Baseline ECG—resting supine for 10 min in a quiet room

Intravenous epinephrine infusion:

- Commence at 0.025 μg/(kg/min) for 10 min
- Increased to 0.05, then 0.10, and finally 0.20 μg/(kg/min) at 5-min intervals
- Cease infusion after 5 min of 0.20 μg/(kg/min) or earlier if SBP >200 mmHg, or occurrence of VT, 10 PVCs/min, T wave alternans or patient intolerance

Bolus protocol ('Shimizu')

Intravenous epinephrine infusion:

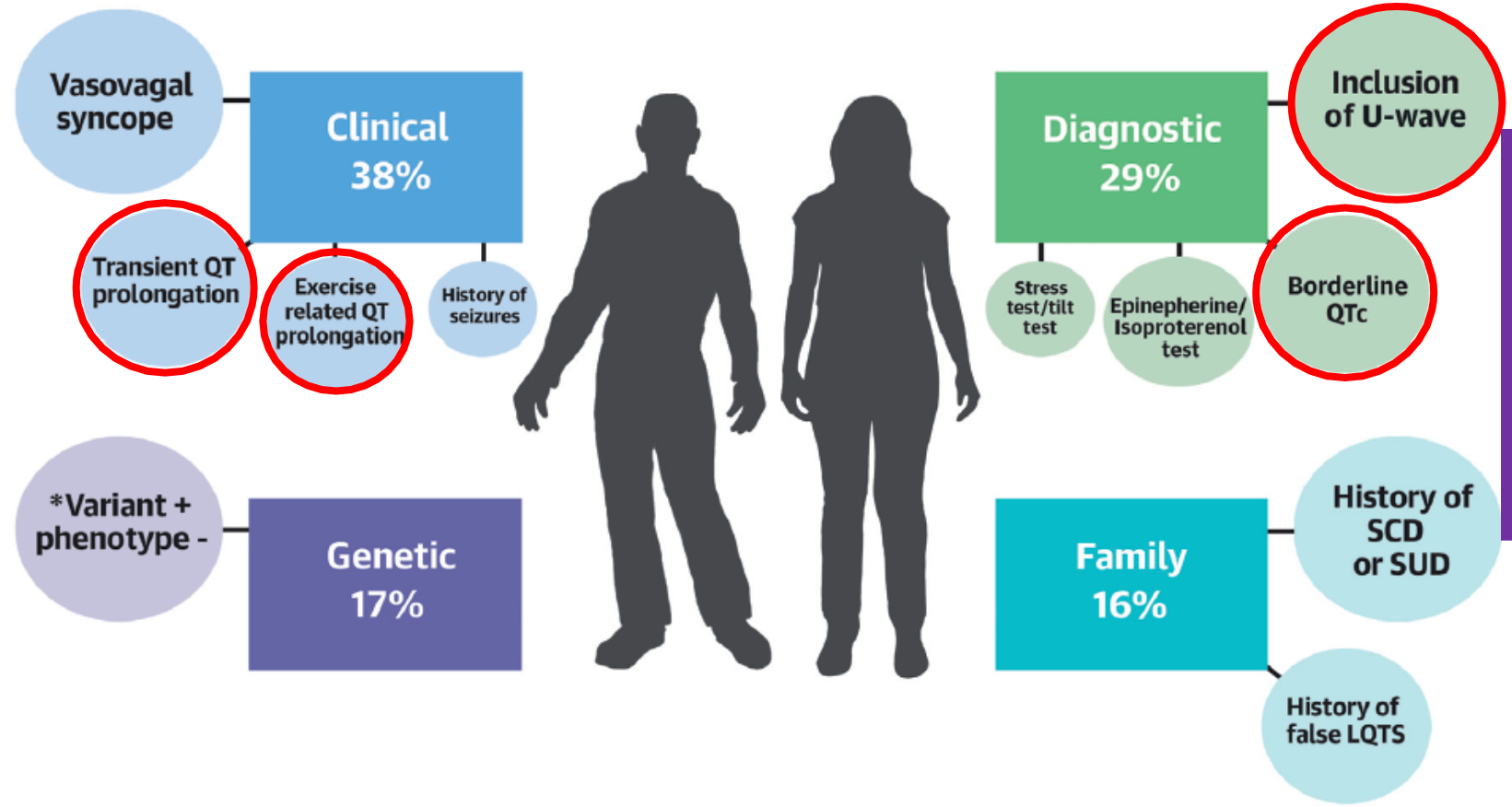
- Bolus of 0.10 μg/kg intravenous epinephrine
- Followed by 0.10 μg/(kg/min) infusion for 5 min

Guidelines did not recommend epinephrine testing in LQTS. The expert group agreed with the recommendation and so no further advice was given.[3] Nonetheless, epinephrine testing is still being performed in cases suspected of having LQTS, especially in Japan.

Phenotypes of Overdiagnosed Long QT Syndrome

Sahej Bains, BS,[a,b,*] Raquel Neves, MD,[b,*] J. Martijn Bos, MD, PhD,[b,c,d] John R. Giudicessi, MD, PhD,[d] Ciorsti MacIntyre, MD,[d] Michael J. Ackerman, MD, PhD[b,c,d]

Phenotypes of Long QT Syndrome (LQTS) Overdiagnosis



EDITORIAL COMMENT

Long QT Syndrome, a Diagnosis That Warrants Expert Opinion and Expert Centers*

Arthur A.M. Wilde, MD, PhD,[a,b,c] Peter J. Schwartz, MD[c,d]

J Am Coll Cardiol 2023;81:477–486

Eliminer un diagnostic différentiel

- Female gender
- Hypokalemia
- Bradycardia
- Recent conversion from atrial fibrillation
- Congestive heart failure
- Digitalis therapy
- High drug concentrations (*exception: quinidine*), often due to drug interactions
- Rapid rate of intravenous drug administration
- Baseline QT prolongation
- Ventricular arrhythmia
- Left ventricular hypertrophy
- Congenital long QT syndrome
- Certain DNA polymorphisms
- Severe hypomagnesemia
- Concomitant use of 2 or more drugs that prolong the QT interval
- Combination of QT-prolonging drug with its metabolic inhibitor

Roden DM. N Engl J Med 2004; 350:1013–22

www.QTdrugs.org www.crediblemeds.org

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- Guide for Safe Medication Use
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Info for Patients with Congenital Long QT (click here)

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News

www.QTdrugs.org www.crediblemeds.org

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Generic Name	Brand Name
Alfuzosin (PR)	Uroxatral
Amantadine (CR)	Symmetrel and others
Amiodarone (KR)	Cordarone and others
Amisulpride (CR)	Solian and others
Amitriptyline (CR)	Elavil (Discontinued 6/13) and others
Amphotericin B (CR)	Fungilin and others
Anagrelide (KR)	Agrylin and others
Apomorphine (PR)	Apokyn and others
Aripiprazole (PR)	Abilify and others
Arsenic trioxide (KR)	Trisenox
Artenimol+piperaquine (PR)	Eurartesim
Asenapine (PR)	Saphris and others
Astemizole (KR)	Hismanal
Atazanavir (CR)	Reyataz and others
Atomoxetine (PR)	Strattera
Azithromycin (KR)	Zithromax and others
Bedaquiline (PR)	Sirturo
Bendamustine (PR)	Treanda and others
Bendroflumethiazide or bendrofluazide (CR)	Aprinox
Bepridil (KR)	Vascor
Bortezomib (PR)	Velcade and others
Bosutinib (PR)	Bosulif
Buprenorphine (PR)	Butrans and others
Cabozantinib (PR)	Cometriq
Capecitabine (PR)	Xeloda
Ceritinib (PR)	Zykadia
Chloral hydrate (CR)	Aquachloral and others
Chloroquine (KR)	Aralen
Chlorpromazine (KR)	Thorazine and others

Generic Name	Brand Name
Cilostazol (KR)	Pletal
Ciprofloxacin (KR)	Cipro and others
Cisapride (KR)	Propulsid
Citalopram (KR)	Celexa and others
Clarithromycin (KR)	Biaxin and others
Clomipramine (PR)	Anafranil
Clozapine (PR)	Clozaril and others
Cocaine (KR)	Cocaine
Crizotinib (PR)	Xalkori
Cyamemazine (cyamepromazine) (PR)	Tercian
Dabrafenib (PR)	Tafinlar
Dasatinib (PR)	Sprycel
Degarelix (PR)	Firmagon
Delamanid (PR)	Deltyba
Desipramine (PR)	Pertofrane and others
Deutetrabenazine (PR)	Austedo
Dexmedetomidine (PR)	Precedex and others
Diphenhydramine (CR)	Benadryl and others
Disopyramide (KR)	Norpace
Dofetilide (KR)	Tikosyn
Dolasetron (PR)	Anzemet
Domperidone (KR)	Motilium and others
Donepezil (KR)	Aricept
Doxepin (CR)	Sinequan and others
Dronedarone (KR)	Multaq
Droperidol (KR)	Inapsine and others
Efavirenz (PR)	Sustiva and others
Eliglustat (PR)	Cerdelga
Epirubicin (PR)	Ellence and others

Generic Name	Brand Name
Eribulin mesylate (PR)	Halaven
Erythromycin (KR)	E.E.S. and others
Escitalopram (KR)	Cipralex and others
Esomeprazole (CR)	Nexium and others
Ezogabine (Retigabine) (PR)	Potiga and others
Famotidine (PR)	Pepcid and others
Felbamate (PR)	Felbatol
Fingolimod (PR)	Gilenya
Flecainide (KR)	Tambocor and others
Fluconazole (KR)	Diflucan and others
Fluoxetine (CR)	Prozac and others
Flupentixol (PR)	Depixol and others
Fluvoxamine (CR)	Faverin and others
Furosemide (frusemide) (CR)	Lasix and others
Galantamine (CR)	Reminyl and others
Garenoxacin (CR)	Geninax
Gatifloxacin (KR)	Tequin
Gemifloxacin (PR)	Factive
Granisetron (PR)	Kytril and others
Grepafloxacin (KR)	Raxar
Halofantrine (KR)	Halfan
Haloperidol (KR)	Haldol (US & UK) and others
Hydrochlorothiazide (CR)	Apo-Hydro and others
Hydrocodone - ER (PR)	Hysinglaå,¢ ER and others
Hydroxychloroquine (CR)	Plaquenil and others
Hydroxyzine (CR)	Atarax and others
Ibogaine (KR)	None
Ibutilide (KR)	Corvert
Iloperidone (PR)	Fanapt and others

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Imipramine (melipramine) (PR)	Tofranil
Indapamide (CR)	Lozol and others
Isradipine (PR)	Dynacirc
Itraconazole (CR)	Sporanox and others
Ivabradine (CR)	Procoralan and others
Ketanserin (PR)	Sufrexal
Ketoconazole (CR)	Nizoral and others
Lansoprazole (CR)	Prevacid
Lapatinib (PR)	Tykerb and others
Lenvatinib (PR)	Lenvima
Leuprolide (PR)	Lupron and others
Levofloxacin (KR)	Levaquin and others
Levomepromazine (KR)	Nosinan and others
Levomethadyl acetate (KR)	Orlaam
Levosulpiride (KR)	Lesuride and others
Lithium (PR)	Eskalith and others
Loperamide (CR)	Imodium and many other OTC and Rx brands
Melperone (PR)	Bunil and others
Mesoridazine (KR)	Serentil
Methadone (KR)	Dolophine and others
Metoclopramide (CR)	Reglan and others
Metronidazole (CR)	Flagyl and many others
Midostaurin (PR)	Rydapt
Mifepristone (PR)	Korlym and others
Mirabegron (PR)	Myrbetriq
Mirtazapine (PR)	Remeron
Moexipril/HCTZ (PR)	Uniretic and others
Moxifloxacin (KR)	Avelox and others
Necitumumab (PR)	Portrazza
Nelfinavir (CR)	Viracept
Nicardipine (PR)	Cardene
Nilotinib (PR)	Tasigna
Norfloxacin (PR)	Noroxin and others
Nortriptyline (PR)	Pamelor and others
Nusinersen (PR)	Spinraza
Ofloxacin (PR)	Floxin
Olanzapine (CR)	Zyprexa and others
Omeprazole (CR)	Losec and others

Ondansetron (KR)	Zofran and others
Osimertinib (PR)	Tagrisso
Oxaliplatin (KR)	Eloxatin
Oxytocin (PR)	Pitocin and others
Paliperidone (PR)	Invega and others
Palonosetron (PR)	Aloxi
Panobinostat (PR)	Farydak
Pantoprazole (CR)	Protonix and others
Papaverine HCl (Intra-coronary) (KR)	none
Paroxetine (CR)	Paxil and others
Pasireotide (PR)	Signifor
Pazopanib (PR)	Votrient
Pentamidine (KR)	Pentam
Perflutren lipid microspheres (PR)	Definity and others
Perphenazine (PR)	Trilafon and others
Pilsicainide (PR)	Sunrythm
Pimavanserin (PR)	Nuplazid
Pimozide (KR)	Orap
Pipamperone (PR)	Dipiperon (E.U) and others
Piperacillin/Tazobactam (CR)	Tazosyn and Zosyn
Posaconazole (CR)	Noxafil and others
Probucol (KR)	Lorelco
Procainamide (KR)	Pronestyl and others
Promethazine (PR)	Phenergan
Propofol (KR)	Diprivan and others
Prothipendyl (PR)	Dominal and others
Quetiapine (CR)	Seroquel
Quinidine (KR)	Quinaglute and others
Quinine sulfate (CR)	Qualaquin
Ranolazine (CR)	Ranexa and others
Ribociclib (PR)	Kisqali
Rilpivirine (PR)	Edurant and others
Risperidone (PR)	Risperdal
Ritonavir (CR)	Norvir
Romidepsin (PR)	Istodax
Roxithromycin (KR)	Rulide and others
Saquinavir (PR)	Invirase(combo)
Sertindole (PR)	Serdolect and others

Sertraline (CR)	Zoloft and others
Sevoflurane (KR)	Ultane and others
Solifenacin (CR)	Vesicare
Sorafenib (PR)	Nexavar
Sotalol (KR)	Betapace and others
Sparfloxacin (KR)	Zagam
Sulpiride (KR)	Dogmatil and others
Sultopride (KR)	Barnetil and others
Sunitinib (PR)	Sutent
Tacrolimus (PR)	Prograf and others
Tamoxifen (PR)	Nolvadex(discontinued 6/13) and others
Telaprevir (CR)	Incivo and others
Telavancin (PR)	Vibativ
Telithromycin (PR)	Ketek
Terfenadine (KR)	Seldane
Terlipressin (KR)	Teripress and others
Terodiline (KR)	Micturin and others
Tetrabenazine (PR)	Nitoman and others
Thioridazine (KR)	Mellaril and others
Tiapride (PR)	Tiapridal and others
Tipiracil and Trifluridine (PR)	Lonsurf
Tizanidine (PR)	Zanaflex and others
Tolterodine (PR)	Detrol and others
Toremifene (PR)	Fareston
torsemide (CR)	Demadex and others
Trazodone (CR)	Desyrel (discontinued 6/13) and others
Trimipramine (PR)	Surmontil and others
Tropisetron (PR)	Navoban and others
Valbenazine (PR)	Ingrezza
Vandetanib (KR)	Caprelsa
Vardenafil (PR)	Levitra
Vemurafenib (PR)	Zelboraf
Venlafaxine (PR)	Effexor and others
Voriconazole (CR)	VFend
Vorinostat (PR)	Zolinza
Ziprasidone (CR)	Geodon and others
Zotepine (PR)	Losizopilon and others

...ng basis to ass... ...ued placemen...

...ecommend always checking the website at crediblemeds.org for the... ...multiple brand names...

...more common...

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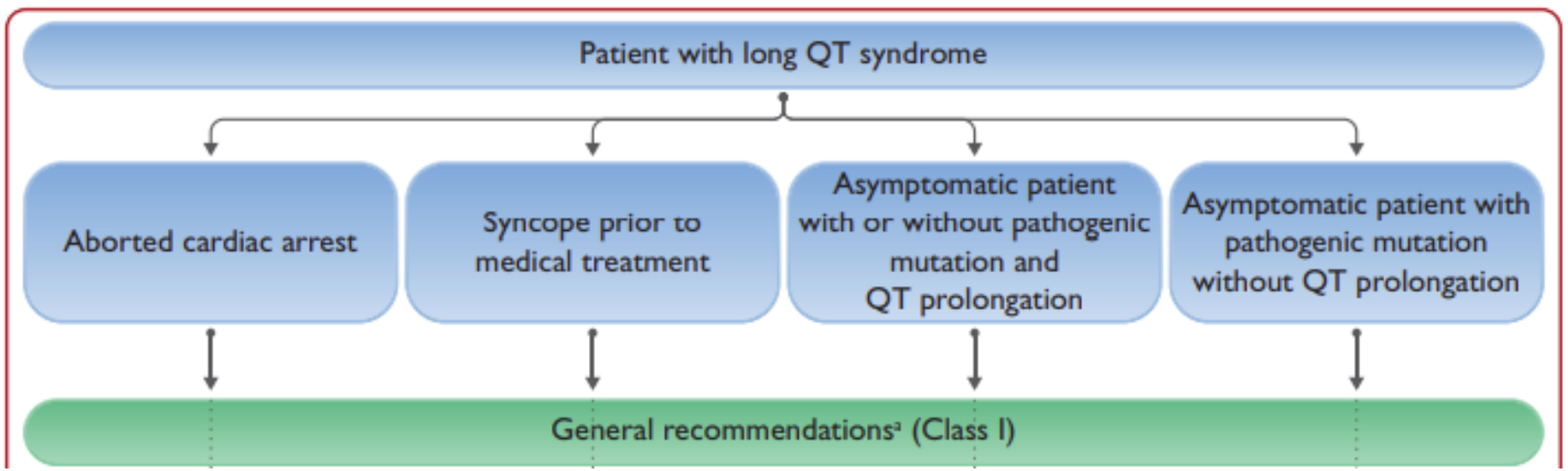
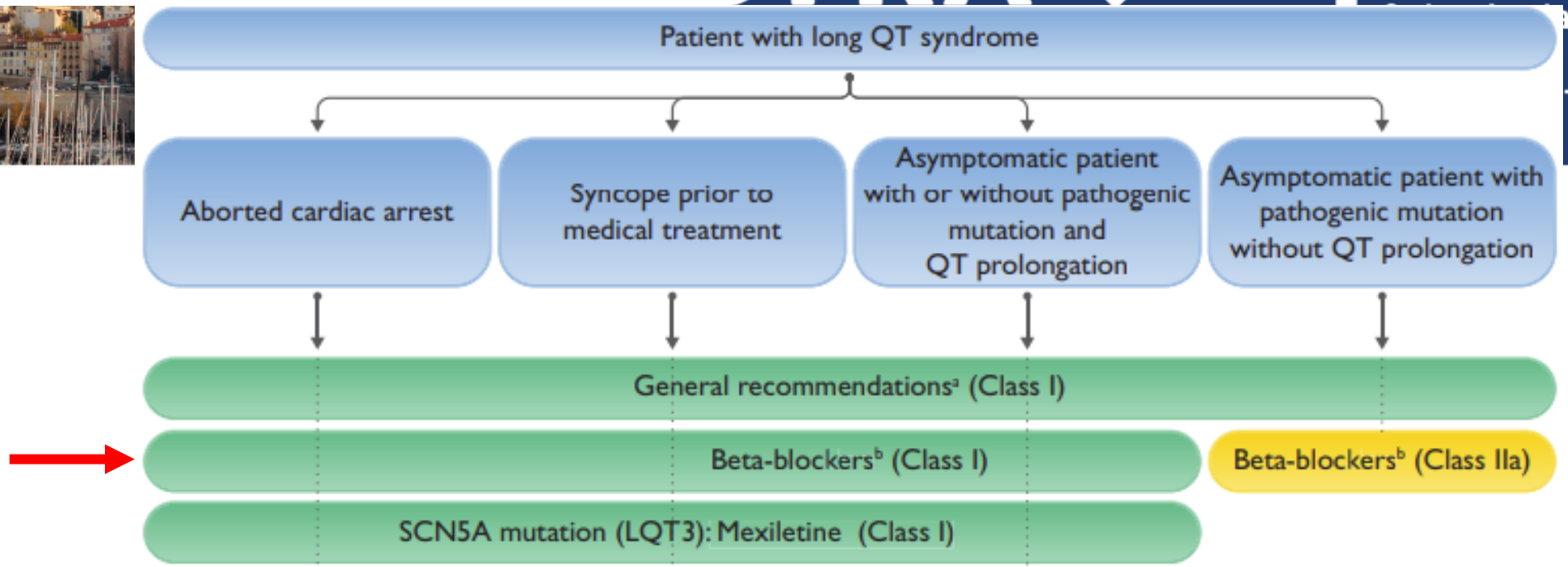


General recommendations to prevent SCD		
The following is recommended in LQTS: • Avoid QT-prolonging drugs.[c] • Avoid and correct electrolyte abnormalities. • Avoid genotype-specific triggers for arrhythmias.[943]	I	C

2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death



Beta-blockers, ideally non-selective beta-blockers (nadolol or propranolol), are recommended in LQTS patients with documented QT interval prolongation, to reduce risk of arrhythmic events.[940,945,946]	I	B
Mexiletine is indicated in LQT3 patients with a prolonged QT interval.[948]	I	C
Beta-blockers should be considered in patients with a pathogenic mutation and a normal QTc interval.[82]	IIa	B

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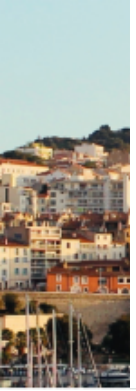
Table 7 Genetic tests and suggested work-up of probands and relatives with primary electrical diseases

			LQTS	BrS	CPVT	Idiopathic VF	ERS
	Genetic test		Class I[a]	Class I	Class I[a]	Class IIb	Class IIb
Proband	**Initial clinical test**	**Cornerstone for diagnosis**	ECG Exercise test	ECG and high precordial lead ECG Sodium channel blockers provocative test[c]	Exercise test	See *Section 5.2.3*, scenario 3	ECG
		Other tests/processes	Exclude acquired LQTS	Exclude phenocopy[b]	Exclude phenocopy[b]/SHD		Holter Echocardiography
	Follow-up		1–3 years dependent on level of risk				
Relatives	**Clinical screening**		ECG Exercise test (when feasible) From birth	ECG and high precordial lead ECGs: start at 10 years Sodium channel blockers provocative test[c]: start >16 years unless clinically indicated[180,181]	ECG Exercise test From birth	ECG and high precordial lead ECGs Exercise test Echocardiogram[182]	ECG Echocardiogram
	Follow-up	Positive phenotype and/or Class IV/V variant	1–3 years dependent on level of risk				
		Negative phenotype and no Class IV/V variant	Discharge				



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When a putative causative variant is first identified, evaluation for pathogenicity is recommended using an internationally accepted framework.[176]	I	C
When a Class IV or Class V variant has been identified in a living or deceased individual with a condition that carries a risk of VA and SCD, genetic testing of first-degree and symptomatic relatives and obligate carriers is recommended.	I	C
It is recommended that genetic testing and counselling on its potential consequences should be undertaken by an expert multidisciplinary team.[179]	I	C
It is recommended that Class III (variants of uncertain significance) and Class IV variants should be evaluated for segregation in families where possible, and the variant re-evaluated periodically.	I	C

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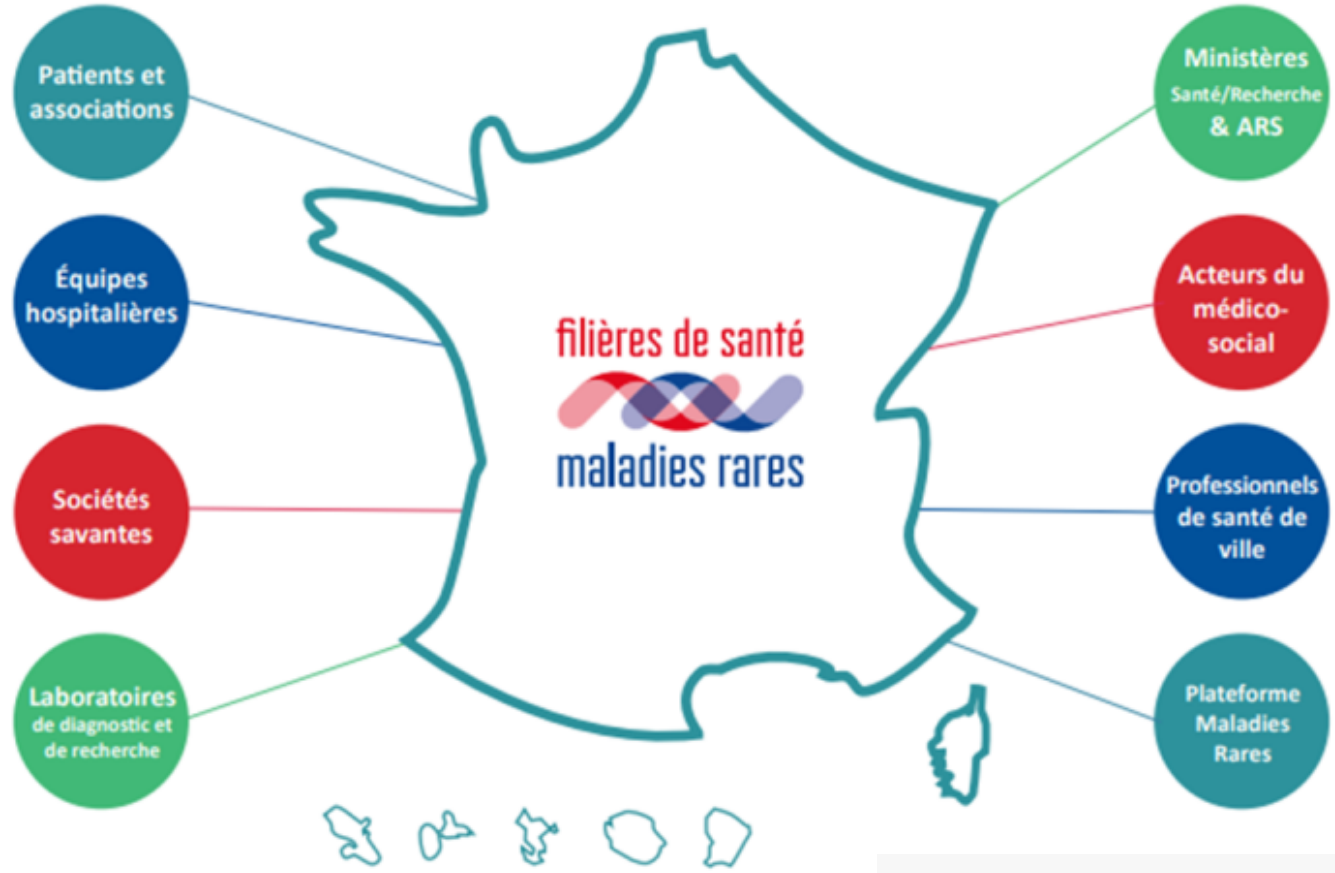

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