7 & 8 FÉVRIER 2019

LES RENCONTRES LYONNAISES DE RYTHMOLOGIE

PRÉSIDENT DU CONGRÈS Philippe CHEVALIER, *Lyon*

Myocardite : différentes perspectives

BRUGADA et MYOCARDITES

Hospices Civils de Lyon

TROUBLES DU RYTHME CARDIAQUE D'ORIGINE HEREDITAIRE
CERA
CENTRE NATIONAL DE REFERENCE

Antoine DELINIERE, Lyon

Déclaration de conflits d’intérêts

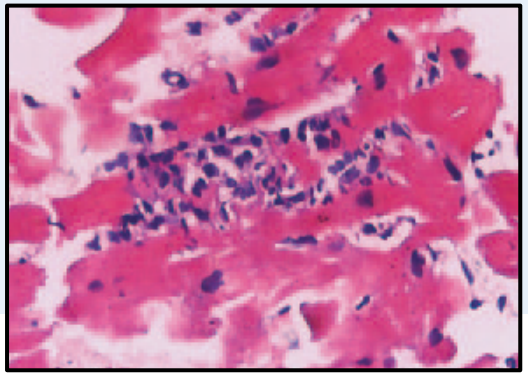
Aucun concernant le contenu de cette présentation

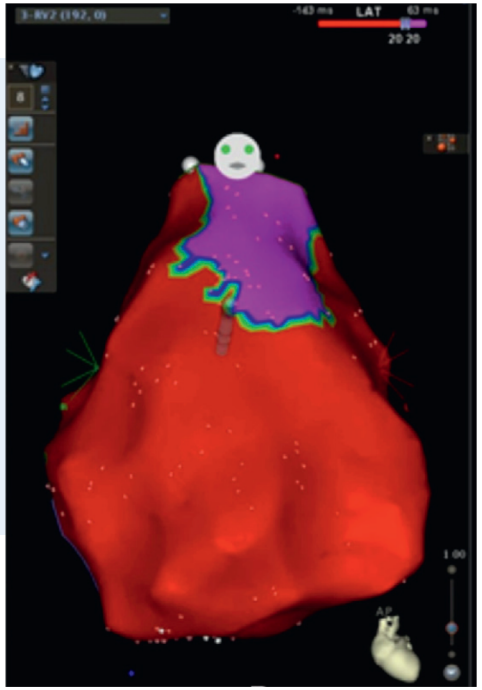
SYNDROME DE **BRUGADA**
BREFS RAPPELS

SYNDROME DE **BRUGADA**

- **Maladie cardiaque héréditaire**
- Actuellement classée comme **canalopathie**
- Prédisposant à un risque de **mort subite** par **fibrillation ventriculaire**
- Sujets jeunes **sans cardiomyopathie structurelle apparente**

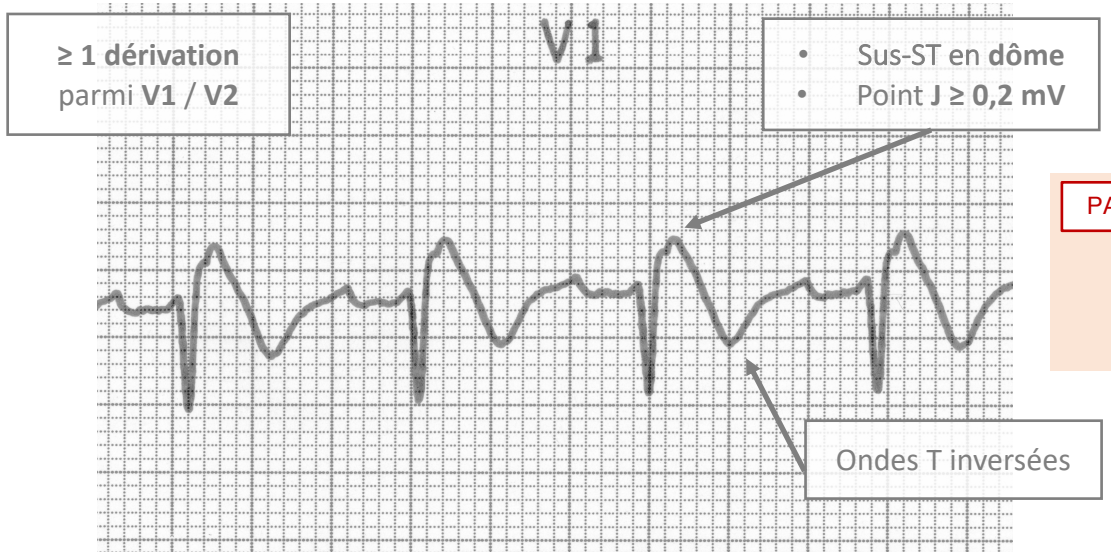
Mais…





Frustaci A et al. Circulation. 2005;112(24):3680-7.

Calò L et al. JACC. 2016;67(12):1427-40.

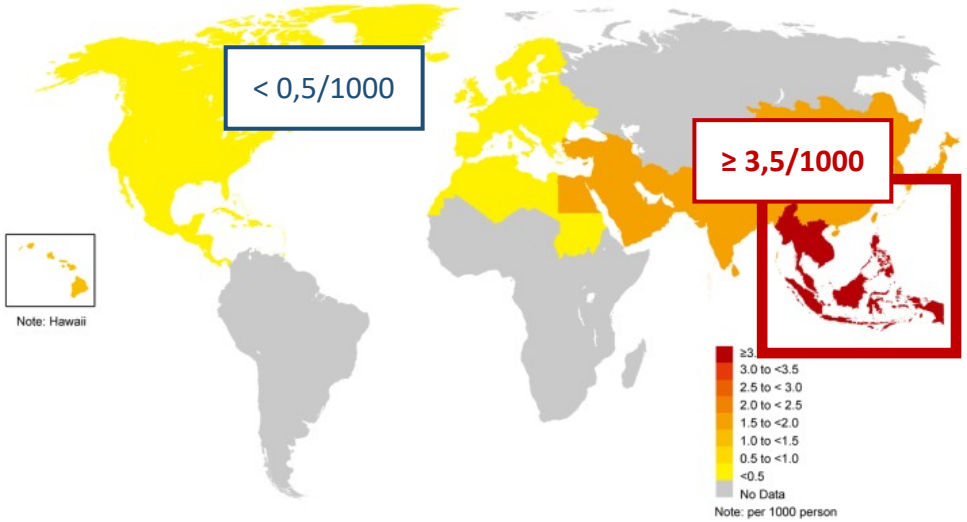


PATTERN DE BRUGADA DE **TYPE 1**

Obligatoire et **suffisant** actuellement pour poser le diagnostic

SYNDROME DE **BRUGADA**
BREFS RAPPELS

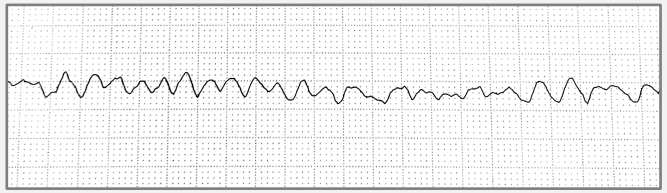
- **0,5 / 1000** individus à l'échelle mondiale
- 3 - 8 **hommes** / 1 femme
- **Héréditaire**, transmission autosomique dominante
    - Principal gène : **SCN5A**
    - Mais mutations identifiées dans seulement **1/4 à 1/3 des cas**



Vutthikraivit et al. Acta Cardiol Sin. 2018;34(3):267-77.

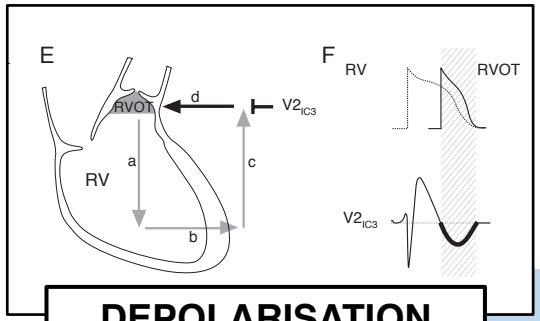
- **Fibrillation ventriculaire** en moyenne entre **38 et 48 ans**

- **4 à 12%** des morts subites, **20%** chez les sujets sans cardiomyopathie **apparente**



SYNDROME DE **BRUGADA**
BREFS RAPPELS

Une physiopathologie encore incertaine…



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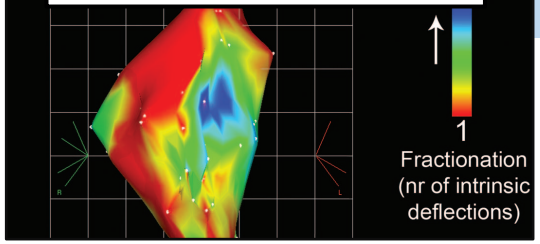

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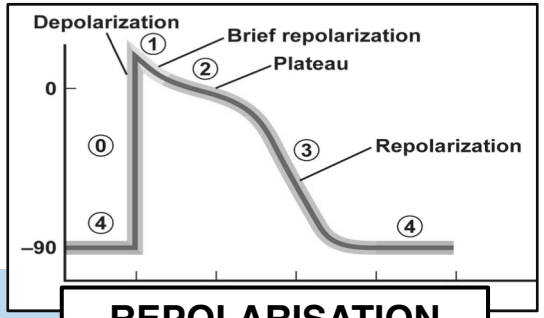
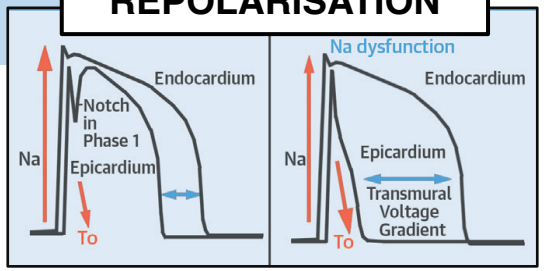


REPOLARISATION



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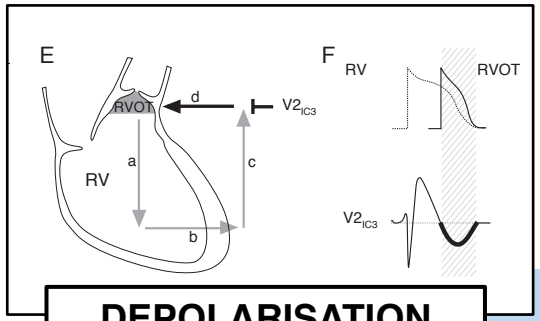

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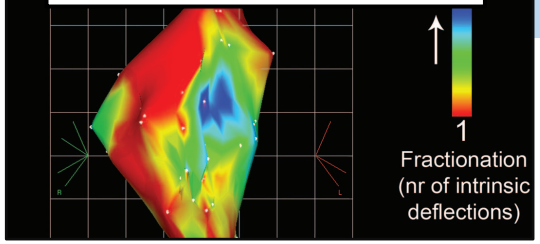

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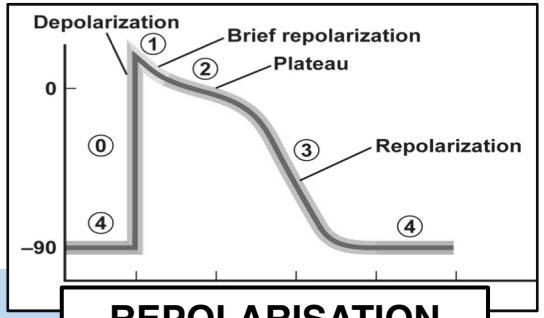

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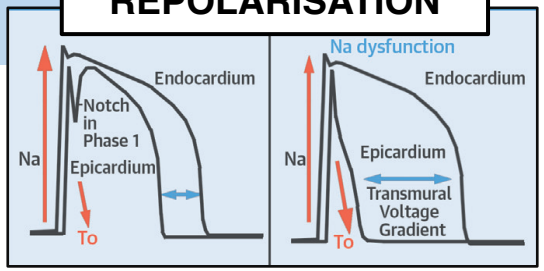

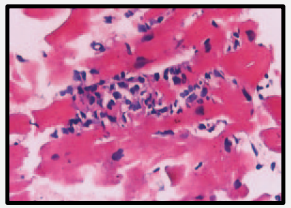

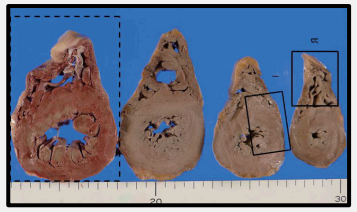

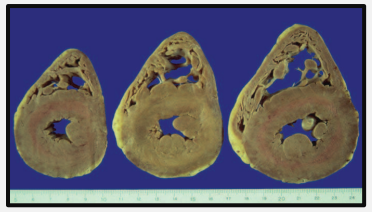

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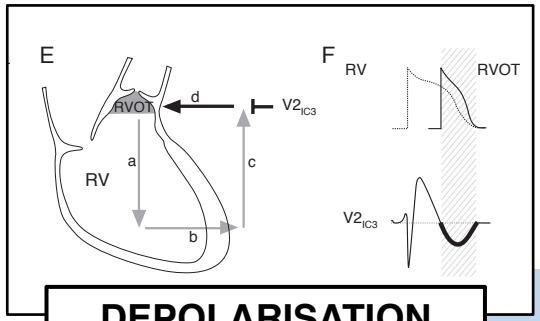

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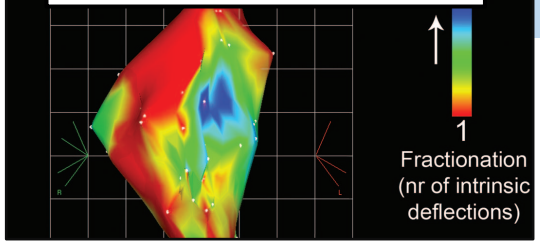

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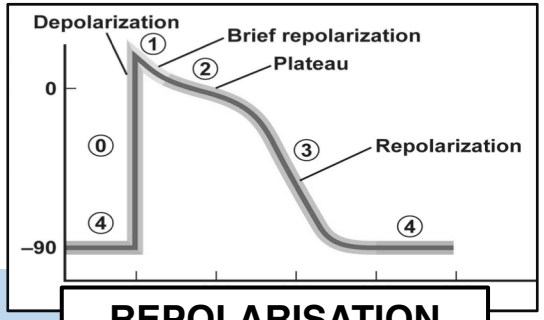

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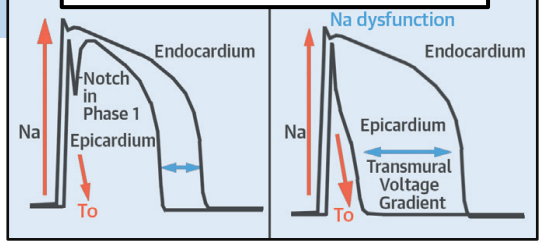

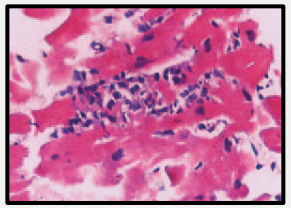

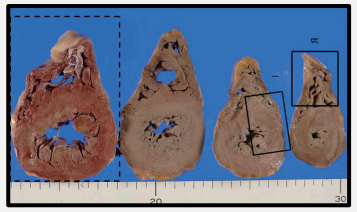

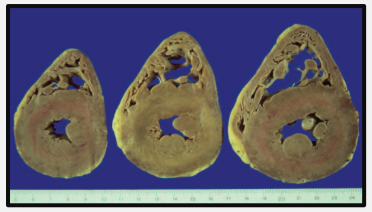

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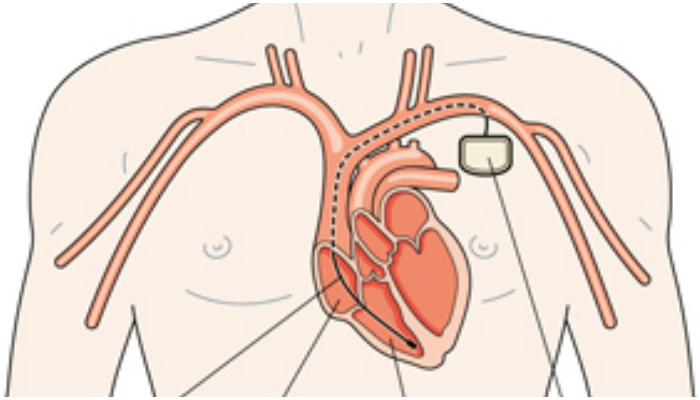
… et un arsenal thérapeutique limité

DAI



bhf.org.uk

The following lifestyle changes are recommended in all patients with a diagnosis of Brugada syndrome:

(a) Avoidance of drugs that may induce ST-segment elevation in right precordial leads (http://www.brugadadrugs.org)
(b) Avoidance of excessive alcohol intake and large meals
(c) Prompt treatment of any fever with antipyretic drugs.

QUINIDINE

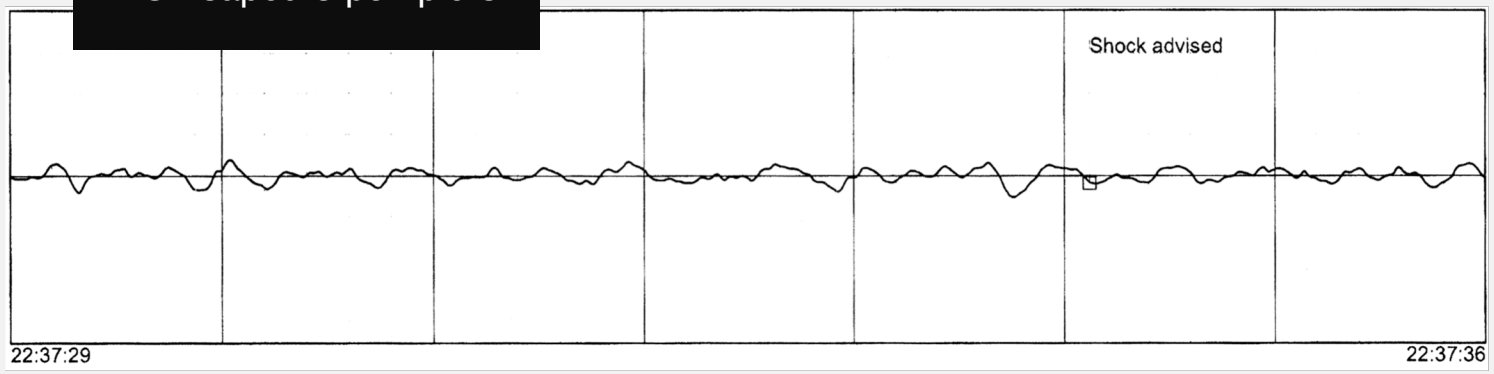
ABLATION

Priori et al. EHJ (2015) 36, 2793–2867

CASE REPORT
M. Y, 29 ans

- **Homme** de **29 ans**
- Pas d'antécédent personnel ni familial
- **ACR brutal**
- **Soir**
- Au cours d'un **repas riche**

DSA sapeurs-pompiers



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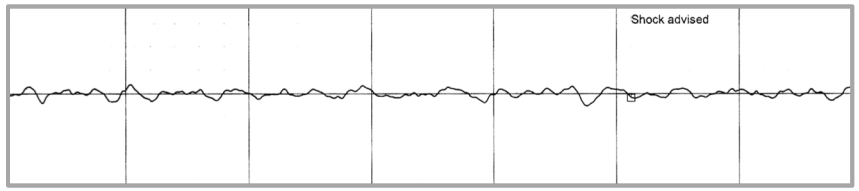

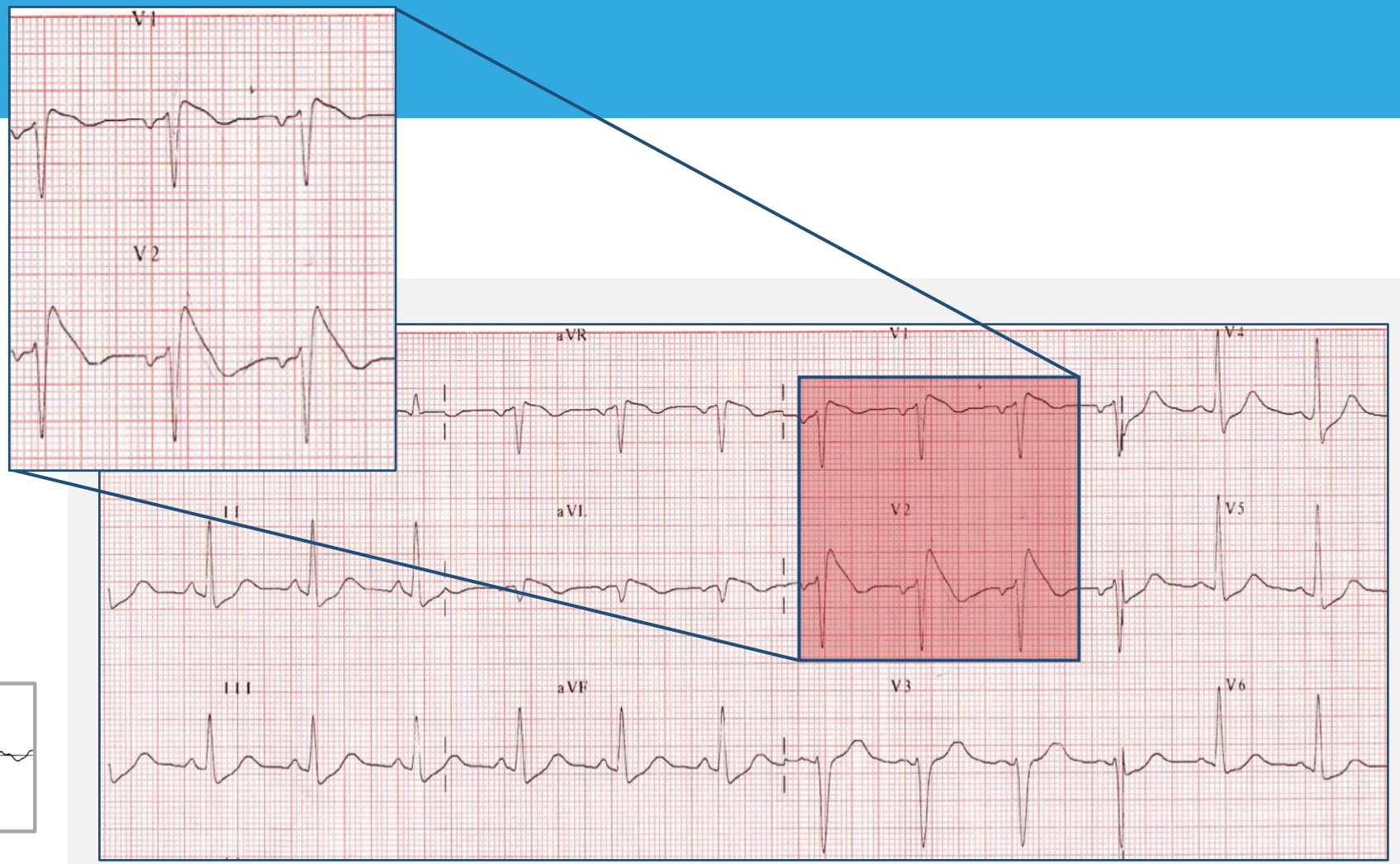

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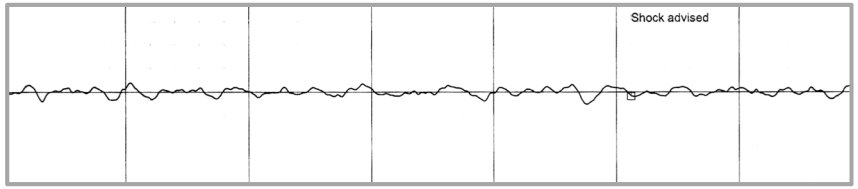
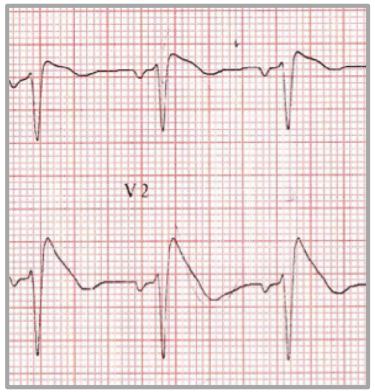
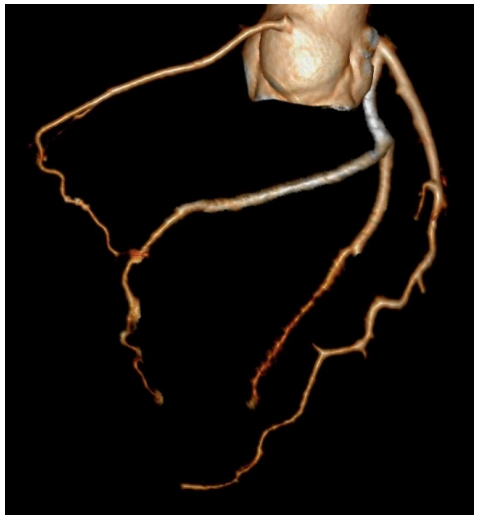
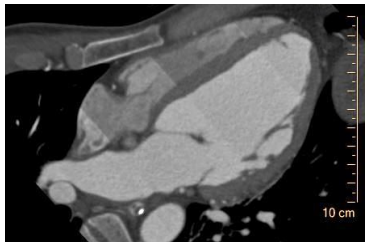




CRP Protéine C réactive .....: 0,3 mg/L
du
Immunoturbidimétrie
Troponine I .....: 1,61 µg/L





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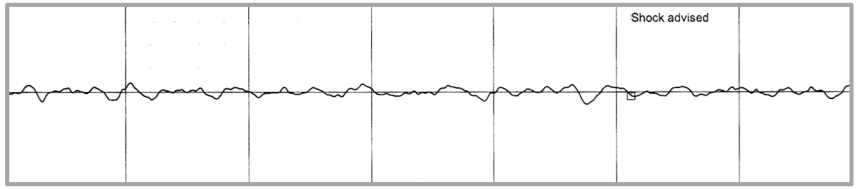

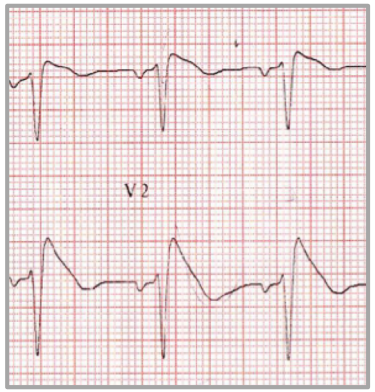

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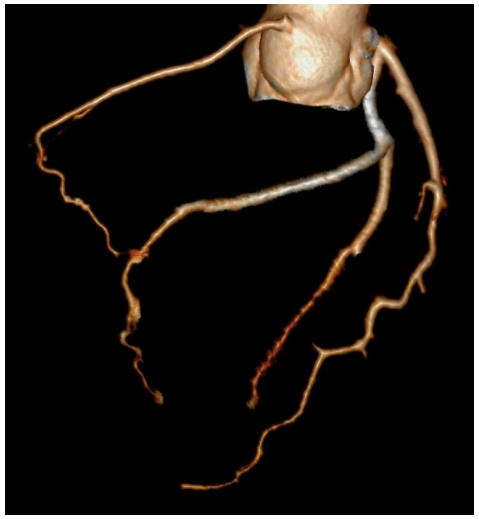

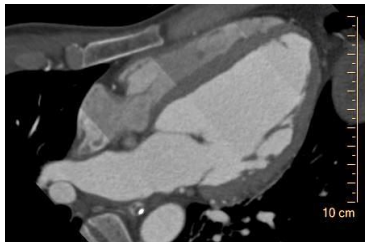

Pattern de type 1 fluctuant,
reproduit lors du test à la flecainide

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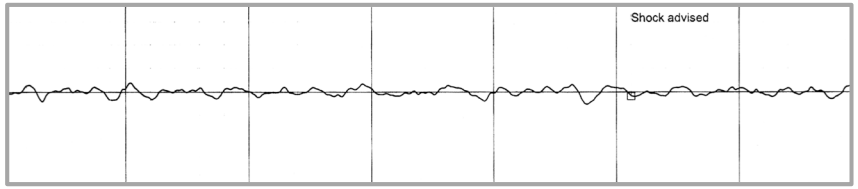

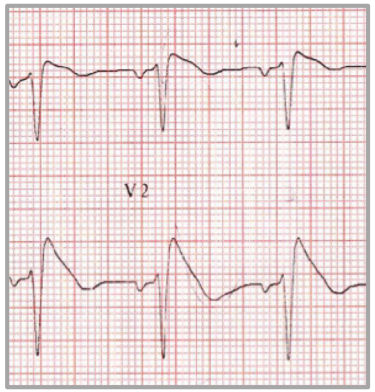

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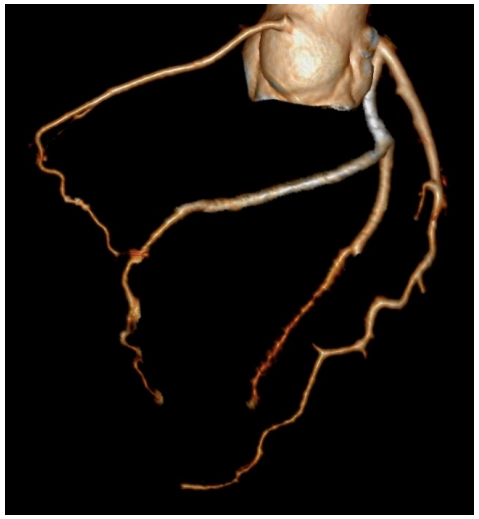

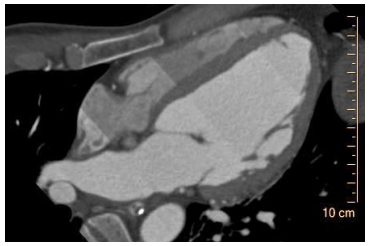

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CACNB2b (Ca_VB2b) = BrS4

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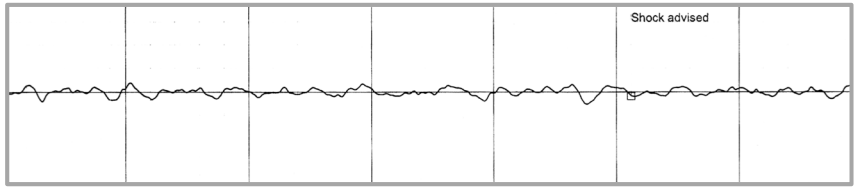

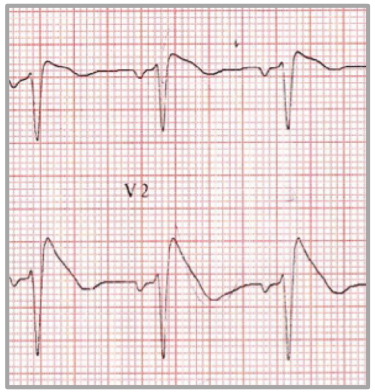

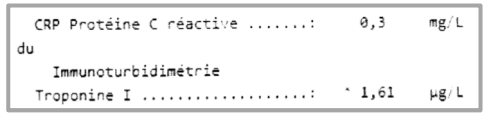

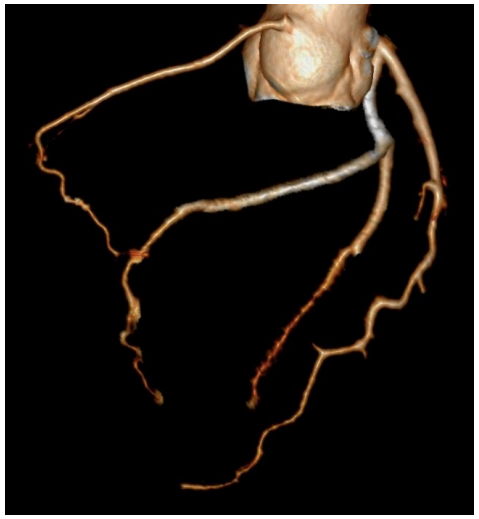

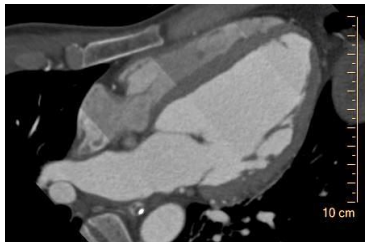

Pattern de type 1 fluctuant,
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CACNB2b ($Ca_V B2b$) = BrS4

Syndrome de
Brugada
a priori
sans ambiguïté

Recommendations	Class[a]	Level[b]	Ref.[c]
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	This panel of experts

ESC European Society of Cardiology

EHRA European Heart Rhythm Association

Priori et al. EHJ 2015

Table 1 Brugada phenocopy systematic diagnostic criteria

Brugada phenocopy diagnostic criteria (first five mandatory)
- (i) ECG pattern has type 1 or type 2 Brugada morphologic characteristics
- (ii) Patient has underlying condition that is identifiable and reversible
- (iii) ECG pattern resolves after resolution of the underlying condition
- (iv) There is low clinical pre-test probability of true BrS determined by lack of symptoms, medical history, and family history
- (v) Negative results on provocative testing with sodium channel blockers such as ajmaline, flecainide, or procainamide (unless clearly not clinically indicated)
- (vi) Provocative testing not mandatory if surgical right ventricular outflow tract manipulation has occurred within last 96 h
- (vii) Results of genetic testing are negative (desirable but not mandatory because the SCN5A mutation is identified in only 20–30% of probands affected by true BrS)

Features that suggest true congenital BrS
- (i) ECG pattern shows type 1 or type 2 Brugada morphologic characteristics
- (ii) There is a high clinical pre-test probability of true congenital BrS determined by presence of symptoms, medical history, and family history
- (iii) Positive results on provocative testing with sodium channel blockers such as ajmaline, flecainide, or procainamide; this indicates sodium channel dysfunction consistent with true BrS
- (iv) Genetic testing is positive in about 20–30% of probands

Gottschalk et aL. Europace 2016, adapted from Anselm et al. Can J Cardiol 2014

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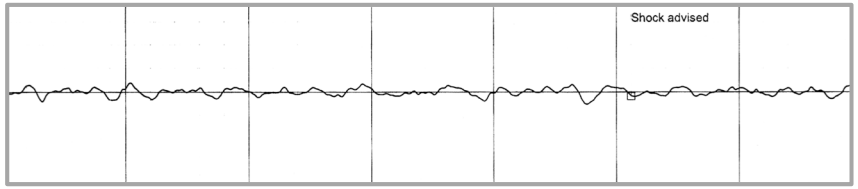
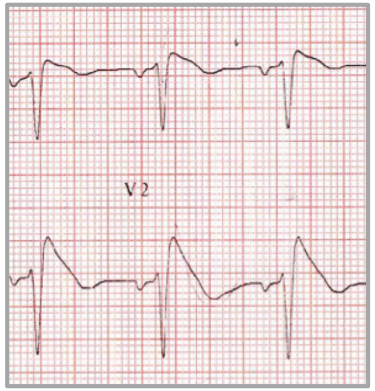
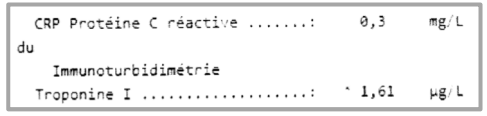
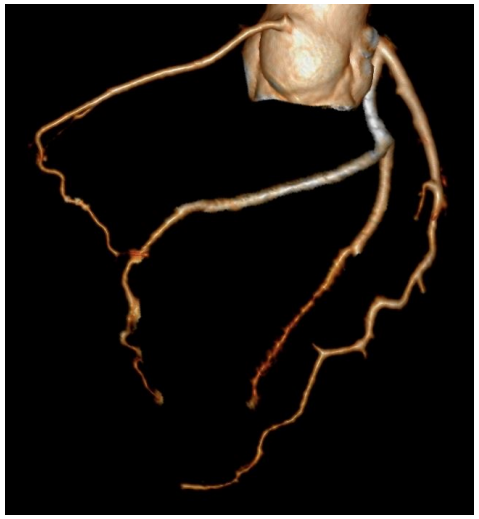
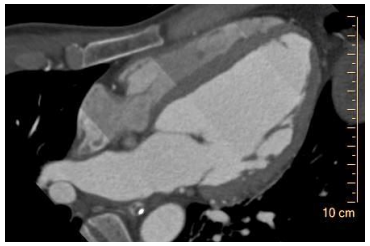










Pattern de type 1 fluctuant,
reproduit lors du test à la flecainide

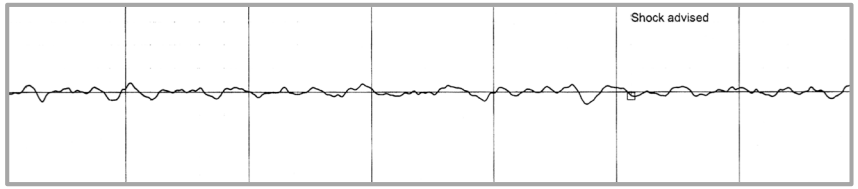
CACNB2b (Ca_VB2b) = BrS4

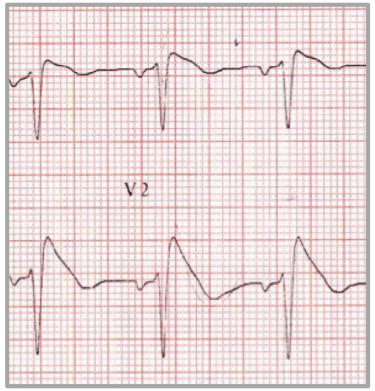
AUTOPSIE

MYOCARDITE LYMPHOCYTAIRE VIRALE AIGUE

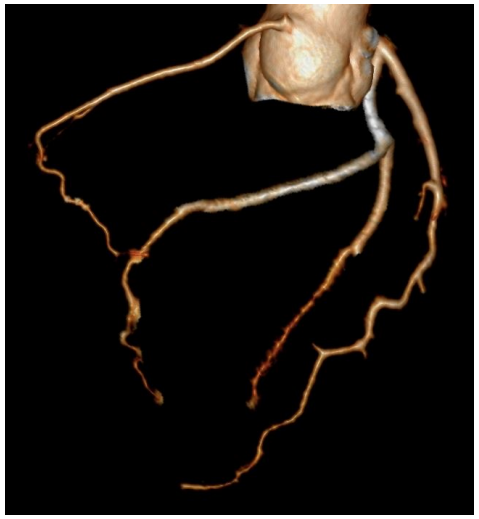
- Infiltrat inflammatoire composé de **lymphocytes T** (CD5+) et de quelques histiocytes, sans polynucléaires
- **Myolyse** locale des cardiomyocytes
  ↪ Paroi antérieure du VG, plages sous-endocardiques focales
- **PCR locale** : **PVB19** (700 copies)

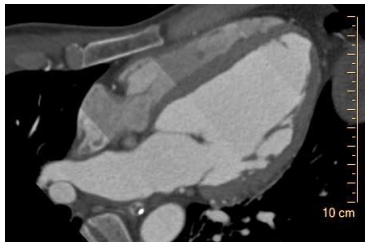
CASE REPORT
M. Y, 29 ans





CRP Protéine C réactive .....: 0,3 mg/L
du
Immunoturbidimétrie
Troponine I .....: 1,61 µg/L





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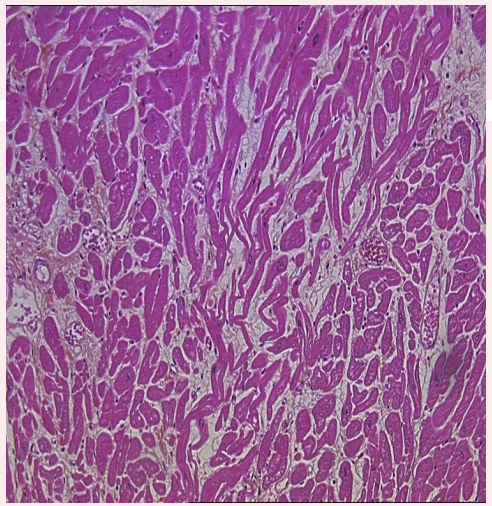


CMH

- Paroi postérieure **18 mm**
- Paroi antérieure 16 mm
- **Désorganisation des fibrilles myocardiques**

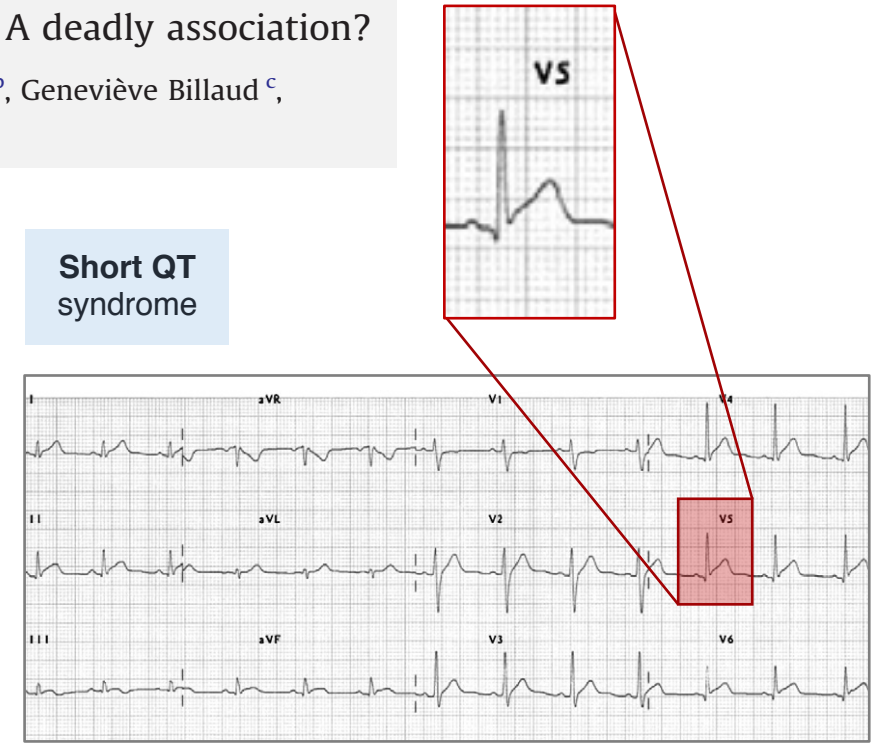
Un cas **isolé** ?

UN CAS ISOLÉ ?

Myocarditis and cardiac channelopathies: A deadly association?

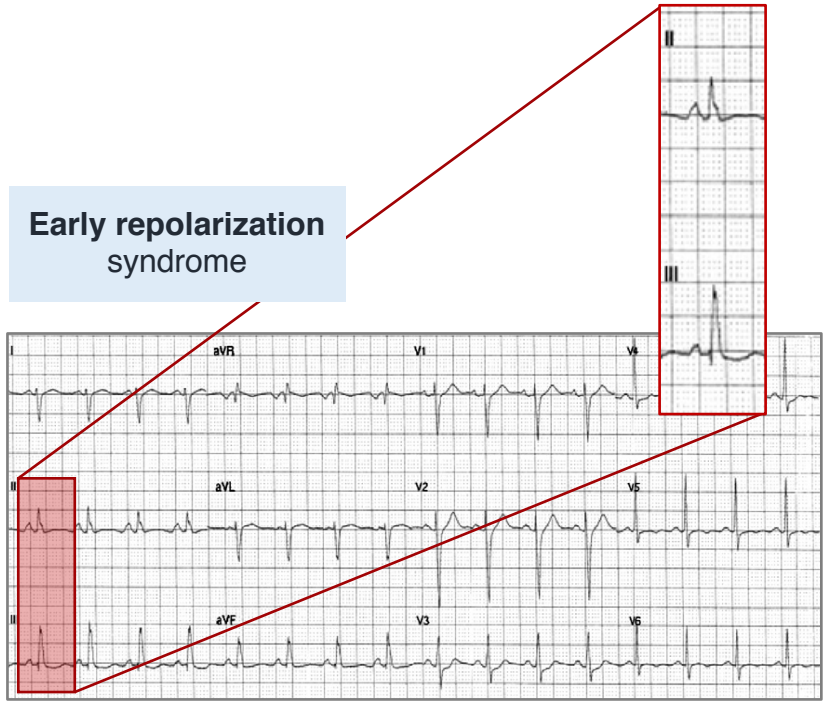
Fiorella Salerno [a], Nicolas Girerd [a], Lara Chalabreysse [b], Geneviève Billaud [c], Bruno Lina [c], Philippe Chevalier [a,d,*]

Short QT syndrome



- Normalisation FEVG durant FU
- **Persistance d'un QT court** à 1 an

Early repolarization syndrome



- Normalisation FEVG durant FU
- **Persistance d'un pattern de RP**

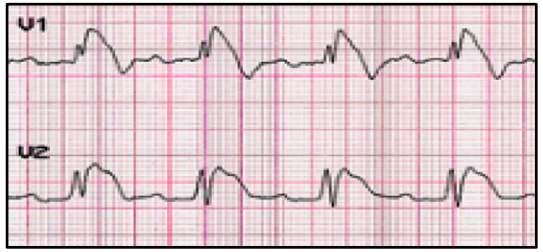
Salerno et al. Int J Cardiol. 2011 Mar 17;147(3):468-70

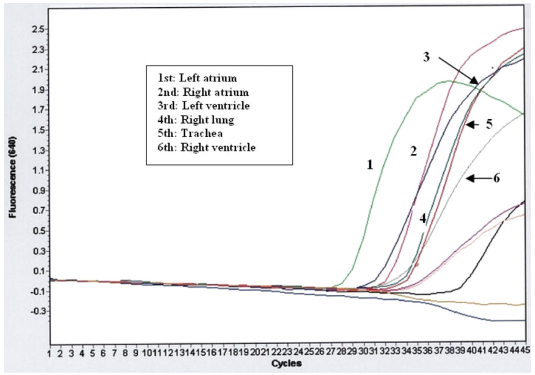
UN CAS ISOLÉ ?

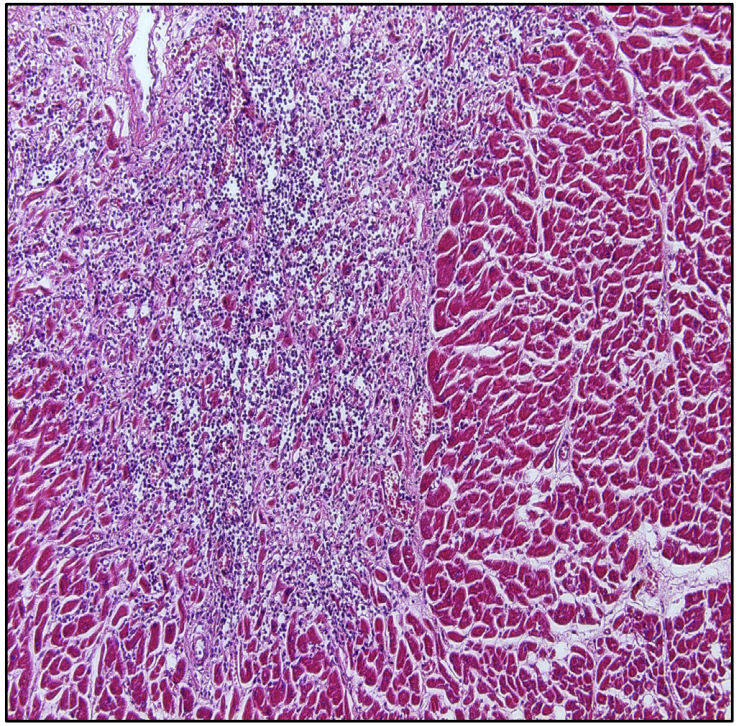
Case review

Sudden cardiac death from parvovirus B19 myocarditis in a young man with Brugada syndrome

Zoltan Juhasz [a,b,*], Laszlo Tiszlavicz [c], Beatrix Kele [d], Gabriella Terhes [d], Judit Deak [d], Laszlo Rudas [e], Eva Kereszty [b]







Juhasz Z et al. J Forensic Leg Medicine 25 (2014)

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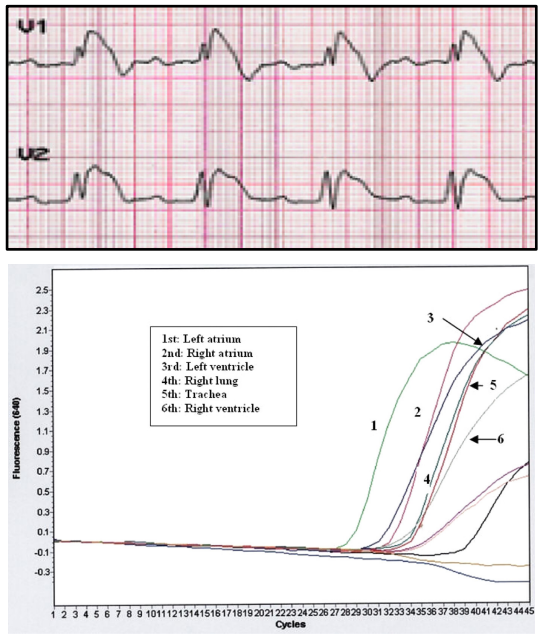
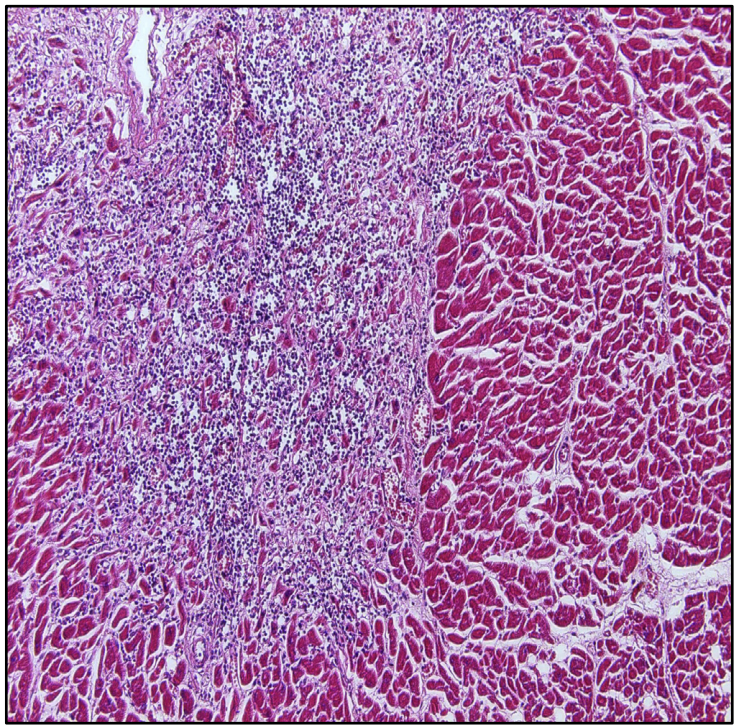
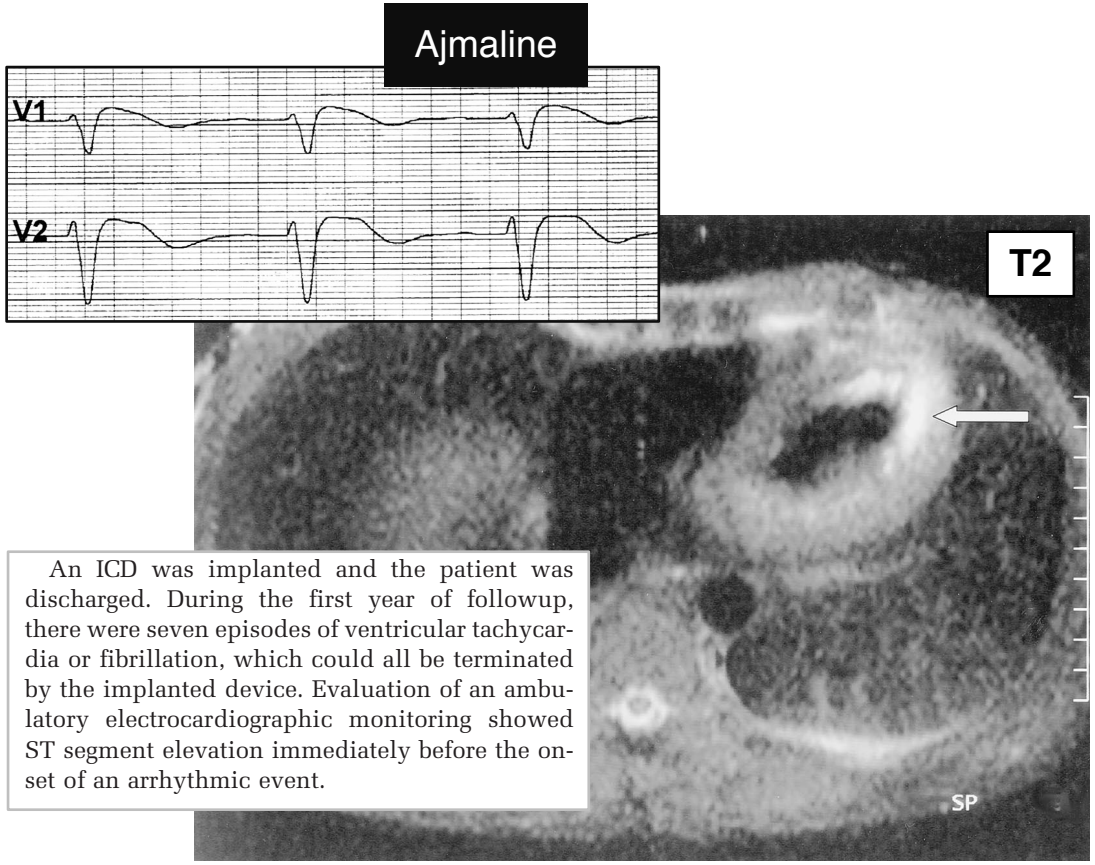




Juhasz Z et al. J Forensic Leg Medicine 25 (2014)

Focal Parvovirus B19 Myocarditis in a Patient with Brugada Syndrome

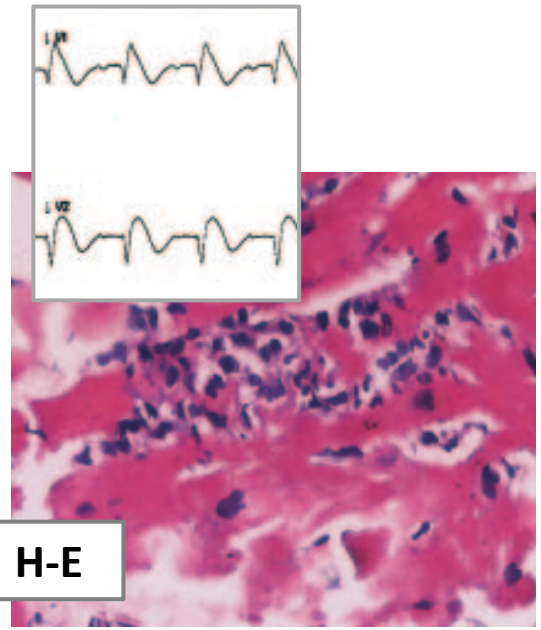
AXEL BUOB, MD*, STEPHANOS SIAPLAOURAS, MD*, INGRID JANZEN, MD*, BERNHARD SCHWAAB, MD*, BERND HAMMER, MD*, GÜNTHER SCHNEIDER, MD†, REINHARD KANDOLF, MD‡, MICHAEL BÖHM, MD*, and JENS JUNG, MD*



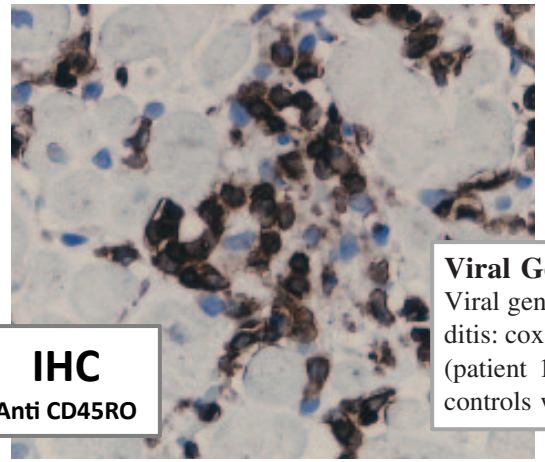
An ICD was implanted and the patient was discharged. During the first year of followup, there were seven episodes of ventricular tachycardia or fibrillation, which could all be terminated by the implanted device. Evaluation of an ambulatory electrocardiographic monitoring showed ST segment elevation immediately before the onset of an arrhythmic event.

Buob A et al. Cardiol Rev. 2003 Jan-Feb;11(1):45-9.

UN CAS ISOLÉ ?



H-E



IHC
Anti CD45RO

Pt	RV and LV Angiography	RV Histology†	LV Histology
1	Normal	Myocarditis	Normal
2	RVA, pile of dishes	Fatty infiltration	Normal
3	Normal	Myocarditis	Normal
4	Normal	Myocarditis	Normal
5	Normal	Myocarditis	Normal
6	Normal	Myocarditis	Normal
7	RVA, LVA	Myocarditis	Myocarditis
8	RVA, LVA	CM	CM
9	Normal	Myocarditis	Normal
10	RVA, LVA	Myocarditis	Myocarditis
11	RVA, LVA	Myocarditis	Myocarditis
12	Normal	CM	CM
13	RVA	Myocarditis	Normal
14	Normal	Myocarditis	Myocarditis
15	RVA	Myocarditis	Myocarditis
16	Normal	CM	CM
17	Normal	Myocarditis	Normal
18	Normal	Myocarditis	Normal

Viral Genome in Myocardial Specimens
Viral genome was detected in 4 patients (28%) with myocarditis: coxsackievirus B3 (patients 1 and 7), Epstein-Barr virus (patient 14), and parvovirus B19 (patient 18). None of the controls were positive for any viral genome.

Cardiac Histological Substrate in Patients With Clinical Phenotype of Brugada Syndrome

Andrea Frustaci, MD*; Silvia G. Priori, MD, PhD*; Maurizio Pieroni, MD, PhD; Cristina Chimenti, MD, PhD; Carlo Napolitano, MD, PhD; Ilaria Rivolta, PhD; Tommaso Sanna, MD; Fulvio Bellocci, MD; Matteo Antonio Russo, MD

- **18 pts consécutifs**
  - 13 types 1 spontanés, 5 pharmaco-induits
  - **Tous symptomatiques**: 8 VT, 7 VF, 3 syncopes
- ETT / IRM normales
- Biopsies biventriculaires multisites
- A **distance** (1-3 mois) d'un évènement rythmique

14 /18
myocardites

Frustaci et al. Circulation. 2005;112(24):3680-7.

« PIEGE » DES PHENOCOPIES ?

Expert cardiologists cannot distinguish between Brugada phenocopy and Brugada syndrome electrocardiogram patterns

Byron H. Gottschalk[1], Daniel D. Anselm[1], Josep Brugada[2], Pedro Brugada[3], Arthur A. Wilde[4,5], Pablo A. Chiale[6], Andres R. Pérez-Riera[7], Marcelo V. Elizari[6], Antoni Bayés De Luna[8], Andrew D. Krahn[9], Hanno L. Tan[4], Pieter G. Postema[4], and Adrian Baranchuk[1]*

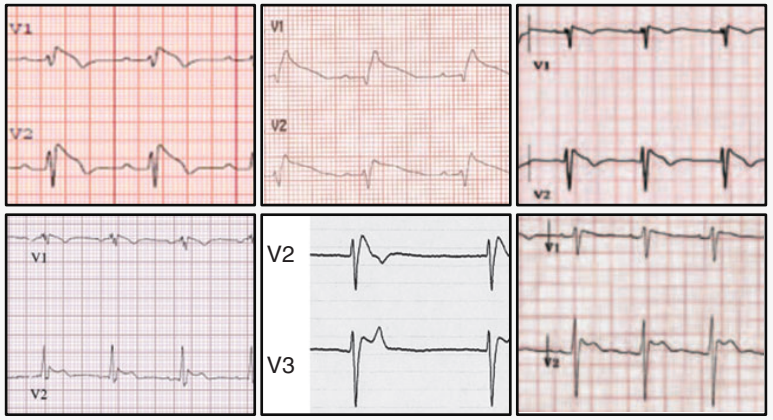


Table 2 Overall evaluator accuracy for each case

Case number	Correct diagnosis	Evaluator accuracy (%)
Case 1	BrP	0
Case 2	BrP	90
Case 3	BrS	40
Case 4	BrS	80
Case 5	BrP	20
Case 6	BrP	70
Case 7	BrS	90
Case 8	BrP	40
Case 9	BrS	60
Case 10	BrP	40
Case 11	BrS	10
Case 12	BrS	100

Gottschalk et al. Europace (2016) 18, 1095–1100

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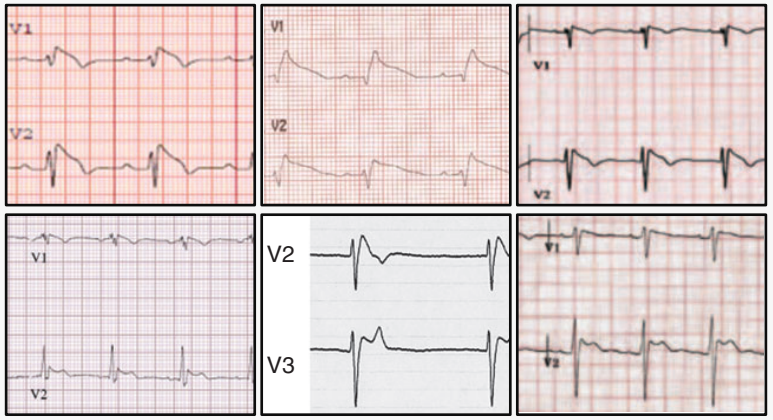

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Absence of Pathognomonic or Inflammatory Patterns in Cardiac Biopsies From Patients With Brugada Syndrome

Sven Zumhagen, MD; Tilmann Spieker, MD; Julia Rolinck, MSt; Hideo A. Baba, MD; Günter Breithardt, MD; Werner Böcker, MD; Lars Eckardt, MD; Matthias Paul, MD; Thomas Wichter, MD; Eric Schulze-Bahr, MD

Patient	*SCN5A* Mutation	Biopsy Sampling Location	Hypertrophy*	Fibrosis*	Fatty Replacement*	Inflammation*	Lymphocytes, n (HPF)†
1	+	Septal, RVOT	1	0	0	0	3
2	+	Apex, septal					2
3	+	span lang=PT-BR<Mid-RV, anterolateral, RVOT					1
4	+	Apex, septal, RVOT			1 (apex)		1
5	+	Apex, septal, RVOT	1		1 (septal)		1
6	+	Apex, septal, RVOT	1	1 (RVOT)			2
7	+	Apex, mid-RV, RVOT		1 (RVOT)			4
8	–	Apex, septal, mid-RV			1 (septal)		1
9	–	Apex, septal, RVOT	1				1
10	–	Septal	1				
11	–	Septal, RVOT			2 (RVOT)		1
12	–	Apex, septal, RVOT					1
13	–	Septal, RVOT		1 (RVOT)	1 (RVOT)		4
14	–	Septal	1				
15	–	Apex, septal, RVOT	1		1 (apex)		2
16	–	Mid-RV, apex, septal			2 (septal)		1
17	–	Apex, septal			2 (septal)		2
18	–	Septal	1	1 (septal)	1 (septal)		4
19	–	Apex, RVOT	1		2 (apex)		
20	–	Septal, RVOT	1				2
21	–	Mid-RV, septal, RVOT	1	1 (RVOT)			2
BrS (n=21)	*SCN5A*+: 33.3%	RVOT/mid-RV (76%), septum (86%), apex (57%)	Normal (47.6%)	Normal (76.2%)	Normal (52.4%)	Normal (100%)	1.5±1.2
Controls (n=12)	NP	Septum (100%)	Normal (50%)	Normal (50%)	Normal (75%)	Normal (100%) (ISHLT 0)	1.8±1.2

PAS d'inflammation

Seulement 5 VT/VF :

- Patients moins sévères ?
- Phase « calme » ?

Zumhagen et al. Circ AE. 2009 Feb;2(1):16-23.

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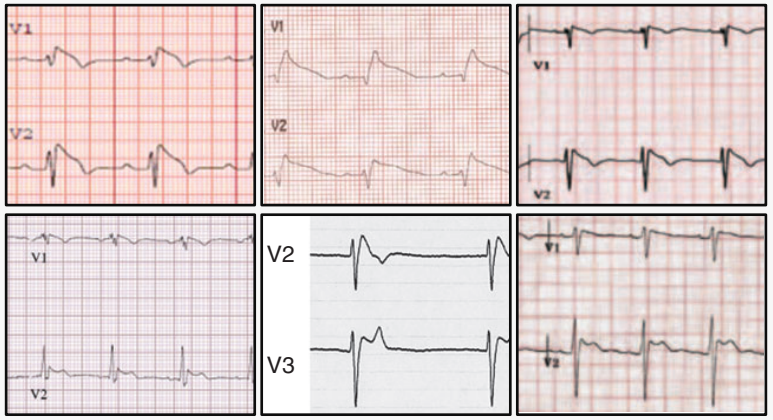

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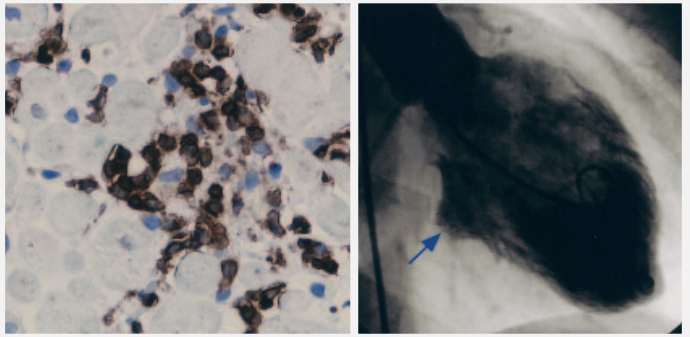
Parmi les 14 **myocardites**

- **0/14** mutation pathogène SCN5A
- **4/14** syncope ou TV/FV au cours d'un épisode fébrile
- **8/14** : normalisation de l'ECG en quelques semaines
- **5/15** : anévrysmes VD



Frustaci et al. Circulation. 2005;112(24):3680-7.

Avec des critères de sélection **rigoureux** ?

REVIEW

- Brugada de type 1 **spontané**
- **FV** documentée et/ou **mort subite**
- **Autopsie** et/ou **histologie biventriculaire**

EXCLUSION

- **Données individuelles non disponibles**
- Histologie **non détaillée** ; biopsie monoventriculaire

- Brugada uniquement de **type 2 & 3**
- Brugada uniquement **pharmaco-induit**
- ECG **atypique**
- **Pas d'ECG** réalisé
- ECG **non fournit** ou **non décrit**
- Onde **Epsilon**

- Argument pour une atteinte myocardique à l'**imagerie**

- Tachycardie ventriculaire
- Liens familiaux

Delinière A, Bencie N,…, Chevalier P. 2018

127 PATIENTS

16 ETUDES

ASPECT DE TYPE 1 SPONTANE

44 PATIENTS

PAS D'ANOMALIE MYOCARDIQUE APPARENTE

35 PATIENTS

Exclusion : 83 pts

ECG

- Type 1 pharmaco-induit : 19
- Type 2 ou 3 : 15
- ECG atypique : 2
- Pas d'ECG réalisé : 8
- Données d'ECG non disponibles : 38
- Ondes Epsilon : 1

Exclusion : 9 pts

IMAGERIE

- ETT, IRM, scanner et/ou angiographie anormal : 9

Exclusion : 9 pts

DONNÉES

- Données indiviudelles non disponibles : 5
- Données histologiques non détaillées : 4

26 PATIENTS

FV / MS	TV	Syncopes	Asymptomatiques
9 PATIENTS	3 PATIENTS	6 PATIENTS	8 PATIENTS

Delinière A, Bencie N,…, Chevalier P. 2018

9 patients : 35.3 ± 10.4 ans, 9 hommes (4 autopsies, 5 biopsies myocardiques biventriculaires)

5 génotypages : 1 mutation SCN5A (BrS1), 1 mutation CACNB2 (BrS4)

8/9 patients ont bénéficié d'examens d'imagerie, **tous normaux**

- **Aucun patient n'avait un cœur macroscopiquement normal à l'autopsie**

- 4/4 **Hypertrophies pariétales VG** à l'autopsie
- 6/9 **Myocardites. Inflammation** 7/9
- 1/9 critères **DAVD**

Delinière A, Bencie N,…, Chevalier P. 2018

HYPOTHESES

ROLE DE L'INFLAMMATION ?

FIEVRE TRIGGER ?

Point of View

May Fever Trigger Ventricular Fibrillation?

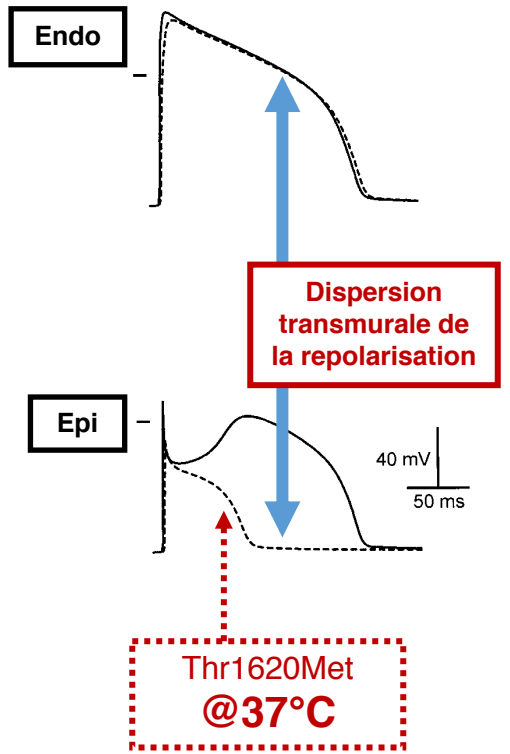
Jean Luc Pasquié, MD, PhD

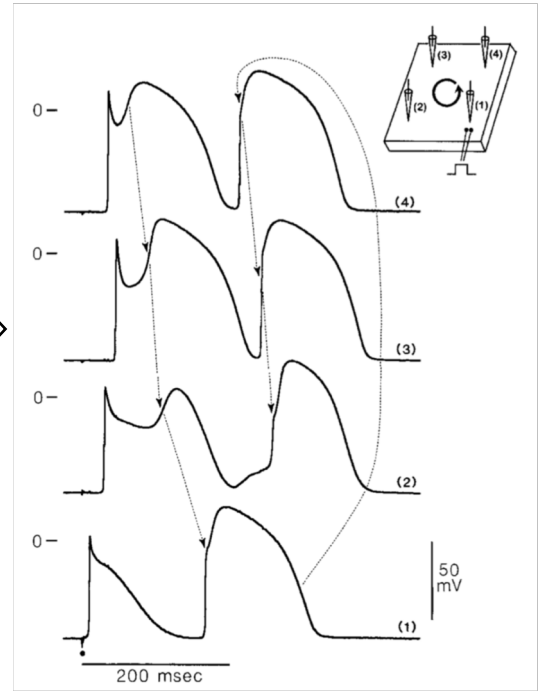
Author	Year	Gender	Age	Febrile illness	Clinical Symptoms	ECG	Flecainide challenge
Fever and Brugada syndrome							
Gonzalez Rebollo (15)	2000	M	30	upper respiratory tract	ICD shocks known Brugada	Unmasked Brugada VF storm	na
Porres (8)	2002	F	66	upper respiratory tract	Syncope cardiac arrest	Unmasked Brugada VF storm	+
Kum (9)	2002	M	39	pneumonia	none	Unmasked Brugada	+
Saura (11)	2002	M	69	pneumonia	none	Unmasked Brugada	-
Morita (10)	2002	M	69	upper respiratory tract	none	Unmasked Brugada T altemans/PVC	+
Patruno (13)	2003	M	53	influenza-like	none	Unmasked Brugada	+
Dinckal (14)	2003	M	55	pneumonia	cardiac arrest	VF storm Unmasked Brugada	na
Mok (12)	2003	M	54	cholangitis	none	Unmasked Brugada	-

Pasquié. Indian Pacing Electrophysiol J. 2005 Apr 1;5(2):139-45.

Ionic Mechanisms Responsible for the Electrocardiographic Phenotype of the Brugada Syndrome Are Temperature Dependent

Robert Dumaine, Jeffrey A. Towbin, Pedro Brugada, Matteo Vatta, Dmitri V. Nesterenko, Vladislav V. Nesterenko, Josep Brugada, Ramon Brugada, Charles Antzelevitch





Lukas A et al. Cardiovasc Res. 1996;32(3):593-603.

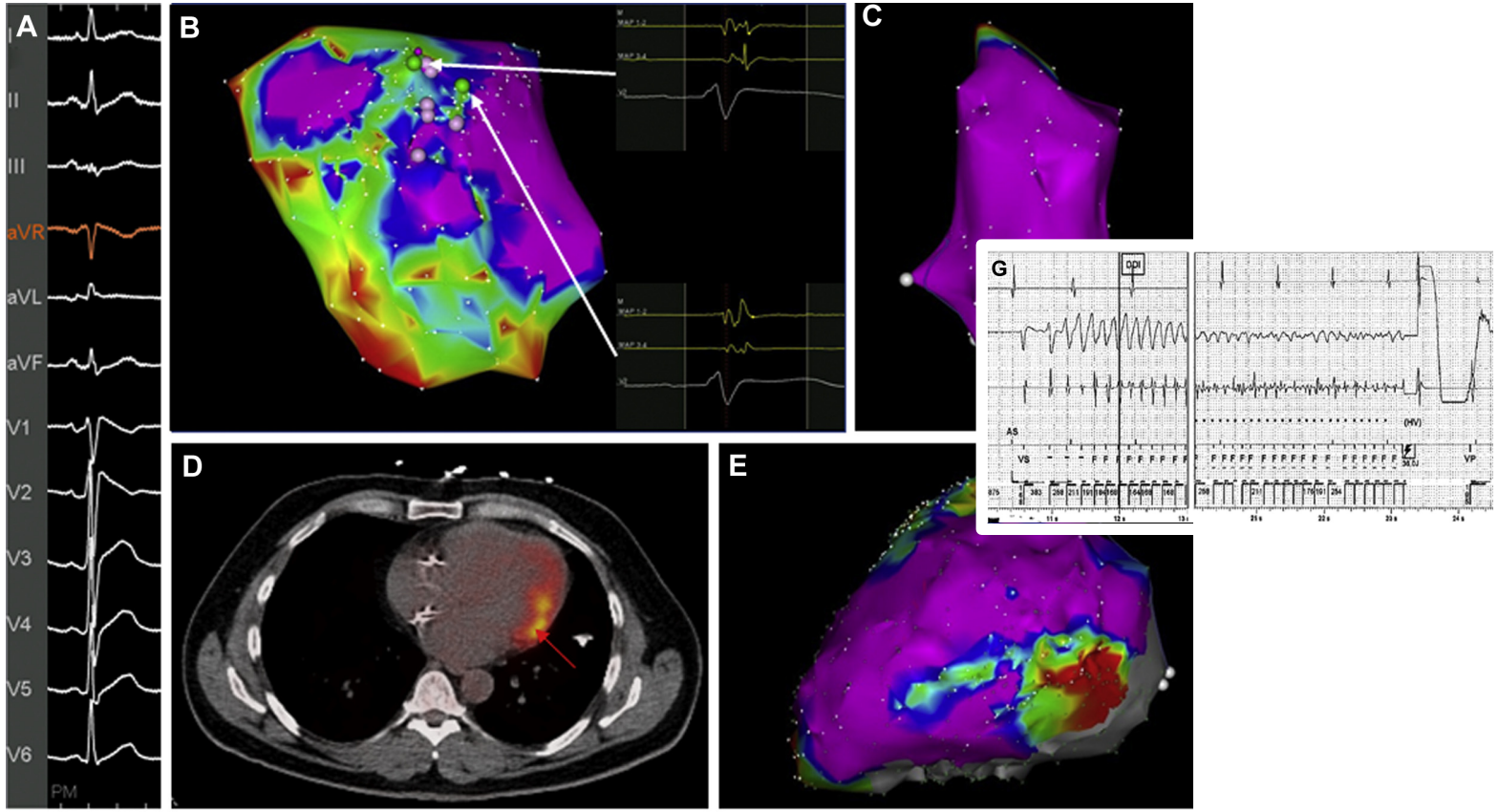
Dumaine et al. Circ Res. 1999 Oct 29;85(9):803-9.

ROLE DE L'INFLAMMATION ?

INFLAMMATION POUSSEES ?

Brugada syndrome—Malignant phenotype associated with acute cardiac inflammation?

Anthony Li, MBBS, MD, Roderick Tung, MD, FHRS, Kalyanam Shivkumar, MD, PhD, FHRS, Jason S. Bradfield, MD, FHRS

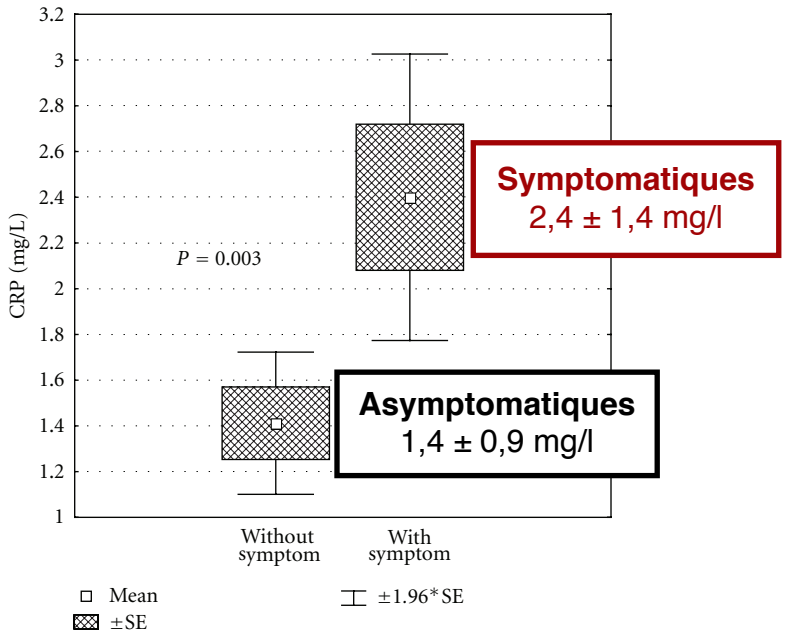


Li et al. HeartRhythm Case Rep. 2017 Jun 20;3(8):384-388.

Clinical Study

C-Reactive Protein Levels in the Brugada Syndrome

Aimé Bonny,[1,2] Joelci Tonet,[2] Manlio F. Márquez,[3] Antonio De Sisti,[2] Abdou Temfemo,[4] Caroline Himbert,[2] Fatima Gueffaf,[5] Fabrice Larrazet,[1] Ivo Ditah,[6] Robert Frank,[2] Françoise Hidden-Lucet,[2] and Guy Fontaine[2]



Symptomatiques
2,4 ± 1,4 mg/l

Asymptomatiques
1,4 ± 0,9 mg/l

CRP ≥ 2 mg/l

95%CI : **1.3 - 19.3**

Marqueur indépendant de risque de syncopes / MS avortée

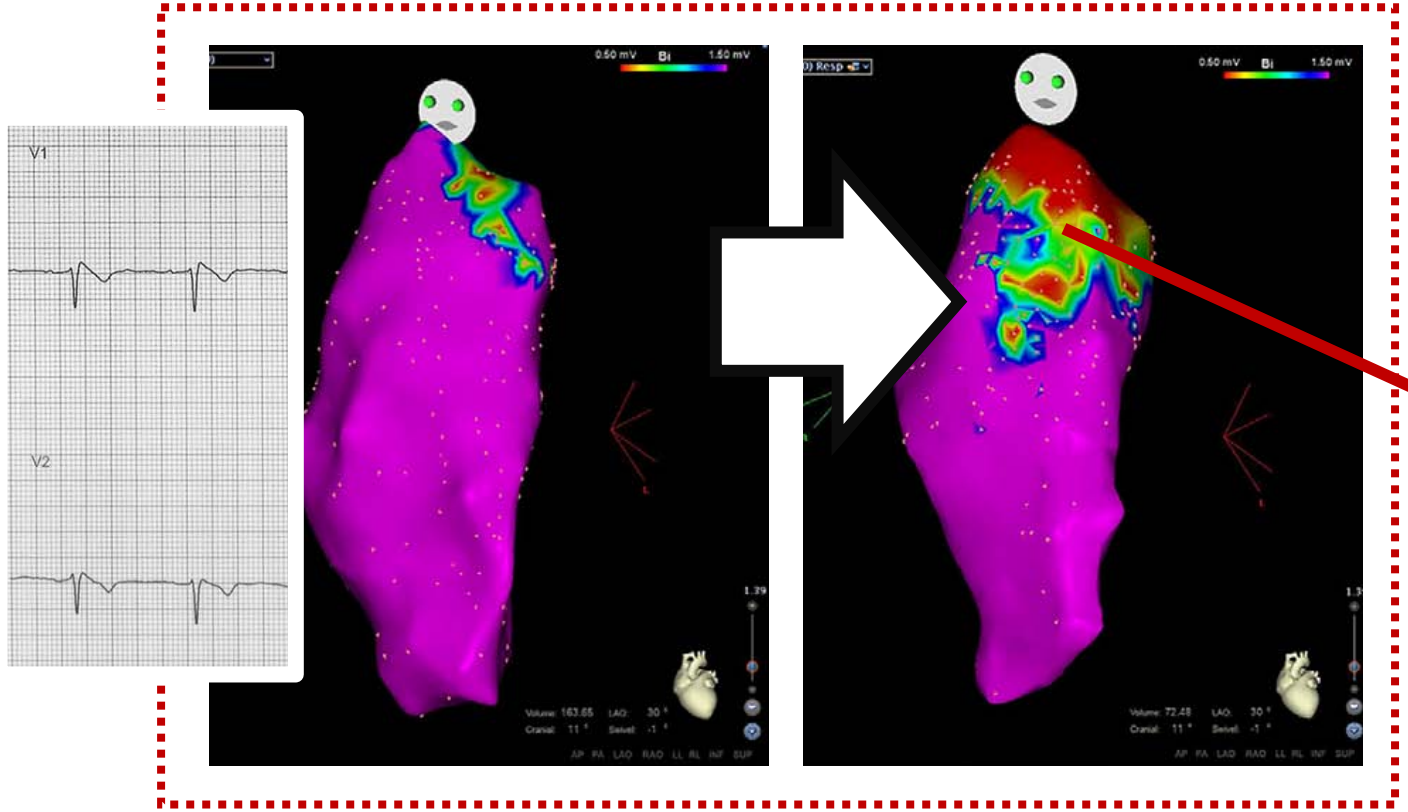
Bonny et al. Cardiol Res Pract. 2011;2011:341521.

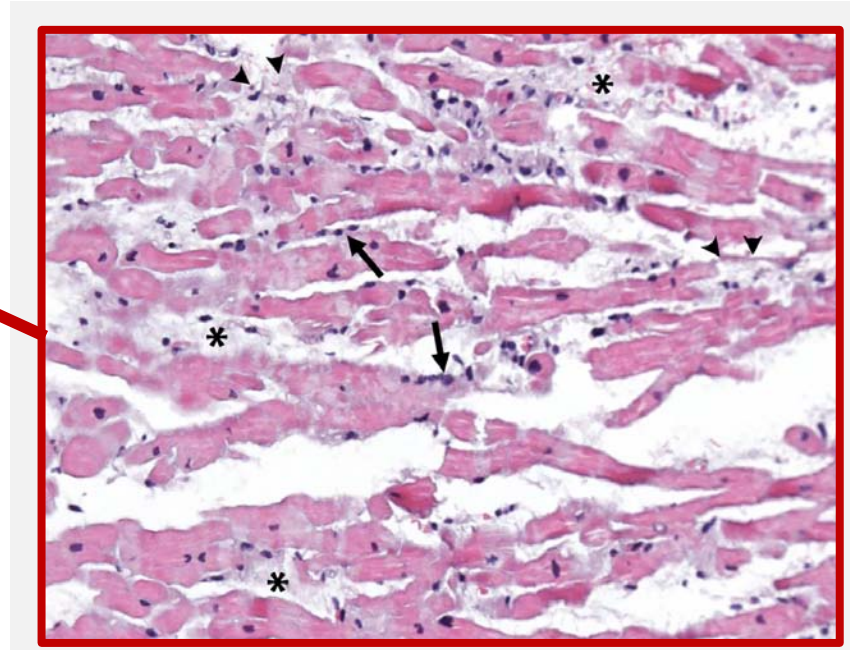
ROLE
DE L'INFLAMMATION ?

INFLAMMATION PROGRESSION DU SUBSTRAT ?

Progression of Electroanatomic Substrate and Electric Storm Recurrence in a Patient With Brugada Syndrome

Pasquale Notarstefano, MD; Maurizio Pieroni, MD, PhD; Raffaele Guida, MD; Teresa Rio, MD; Antonio Oliva, MD; Simone Grotti, MD; Aureliano Fraticelli, MD; Leonardo Bolognese, MD, FESC

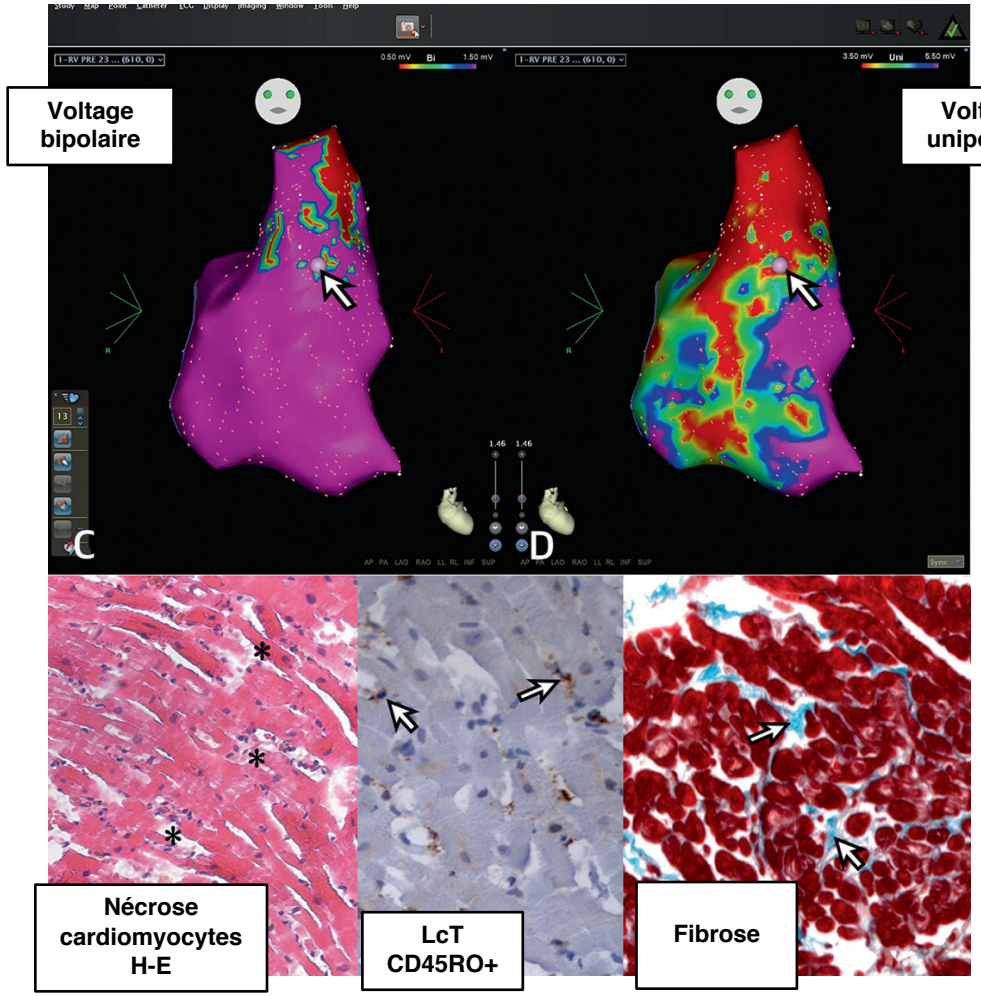




Notarstefano et al. Circulation. 2015 Mar 3;131(9):838-41.

ROLE DE L'INFLAMMATION ?

INFLAMMATION PROGRESSION DU SUBSTRAT ?



Electroanatomic and Pathologic Right Ventricular Outflow Tract Abnormalities in Patients With Brugada Syndrome

Maurizio Pieroni, MD, PhD,[a,*] Pasquale Notarstefano, MD,[a,*] Antonio Oliva, MD, PhD,[b,*] Oscar Campuzano, PhD,[c,d,e] Pasquale Santangeli, MD,[f] Monica Coll, PhD,[c] Martina Nesti, MD,[a] Andrea Carnevali, MD,[g] Aureliano Fraticelli, MD,[a] Anna Iglesias, MSc,[c] Simone Grassi, MD,[b] Ramon Brugada, MD, PhD,[c,d,e,h] Leonardo Bolognese, MD[a]

- 30 cartographies uni et bipolaires
- 20 biopsies RVOT guidées par cartographie

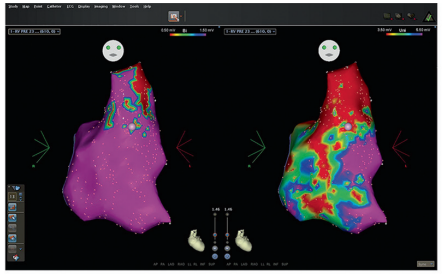
EPI → ENDO

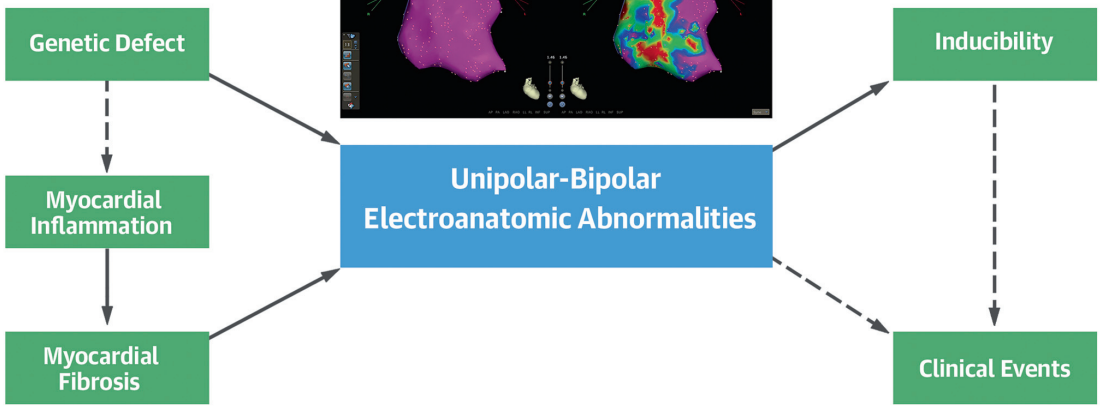
- **Bas voltage** RVOT **unipolaire 93%**, bipolaire 50%
- Inflammation 60%, 3 myocardites probables
- Fibrose subjectivement plus importante en cas d'inflammation
- Inductibilité plus marquée en cas d'inflammation 83% vs. 25%, p=0.032

Pieroni et al. JACC. 2018 Dec 4;72(22):2747-2757.

ROLE DE L'INFLAMMATION ?

INFLAMMATION — PROGRESSION DU SUBSTRAT ?





Pieroni et al. JACC. 2018 Dec 4;72(22):2747-2757.

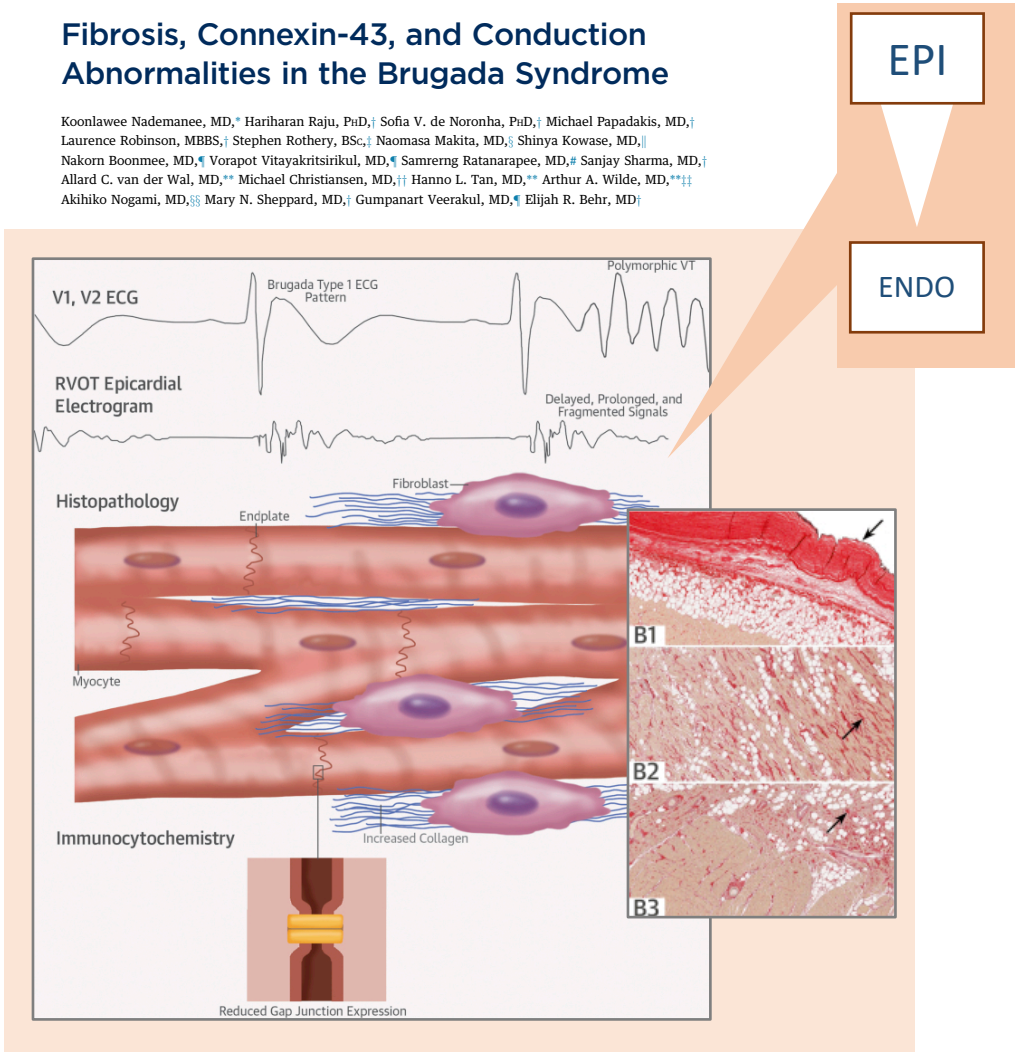
DOWNREGULATION OF CONNEXIN 43 GENE EXPRESSION IN RAT HEART DURING INFLAMMATION. THE ROLE OF TUMOUR NECROSIS FACTOR

Mariana Fernandez-Cobo,[1,3] Cynthia Gingalewski,[1] Doreen Drujan,[1] Antonio De Maio[1,2]

Fernandez-Cobo et al. Cytokine. 1999 Mar;11(3):216-24.

Fibrosis, Connexin-43, and Conduction Abnormalities in the Brugada Syndrome

Koonlawee Nademanee, MD,* Hariharan Raju, PhD,† Sofia V. de Noronha, PhD,† Michael Papadakis, MD,† Laurence Robinson, MBBS,† Stephen Rothery, BSc,‡ Naomasa Makita, MD,§ Shinya Kowase, MD,‖ Nakorn Boonmee, MD,¶ Vorapot Vitayakritsirikul, MD,¶ Samrerng Ratanarapee, MD,# Sanjay Sharma, MD,† Allard C. van der Wal, MD,** Michael Christiansen, MD,†† Hanno L. Tan, MD,** Arthur A. Wilde, MD,**‡‡ Akihiko Nogami, MD,§§ Mary N. Sheppard, MD,† Gumpanart Veerakul, MD,¶ Elijah R. Behr, MD†



Nademanee et al. J Am Coll Cardiol. 2015;66(18):1976-86.

CONCLUSION

- Rôle potentiel des épisodes de **myocardites** dans l'histoire naturelle du syndrome de Brugada
- **Gâchette initiatrice** via inflammation myocardique et la fièvre
- **Genèse / renforcement du substrat** via **fibrose** et réduction gap-junctions
- Conclusions prudentes (phénocopies ?)

MERCI DE VOTRE ATTENTION

Hospices Civils de Lyon

TROUBLES DU RYTHME CARDIAQUE D'ORIGINE HEREDITAIRE
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