Prise en charge des patients avec ESV asymptomatiques

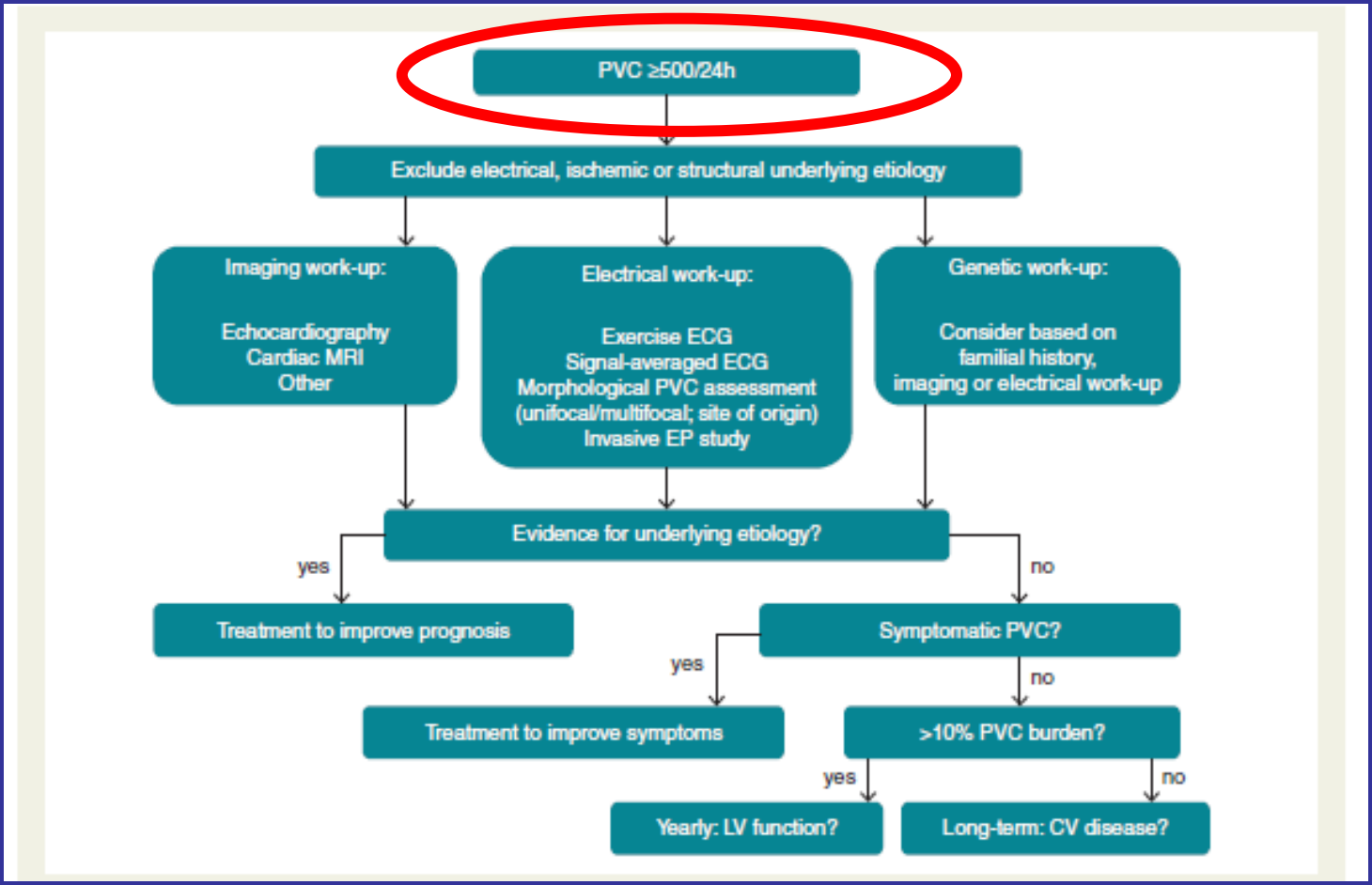
Nicolas Lellouche
Fédération de Cardiologie
Hopital Henri Mondor
Créteil

- ESV fréquente dans la population générale: 50% de la population sur Holter ECG systématique mais 2% avec au moins 50 ESV/24 heures

- Asymptomatique dans environ 30% des cas

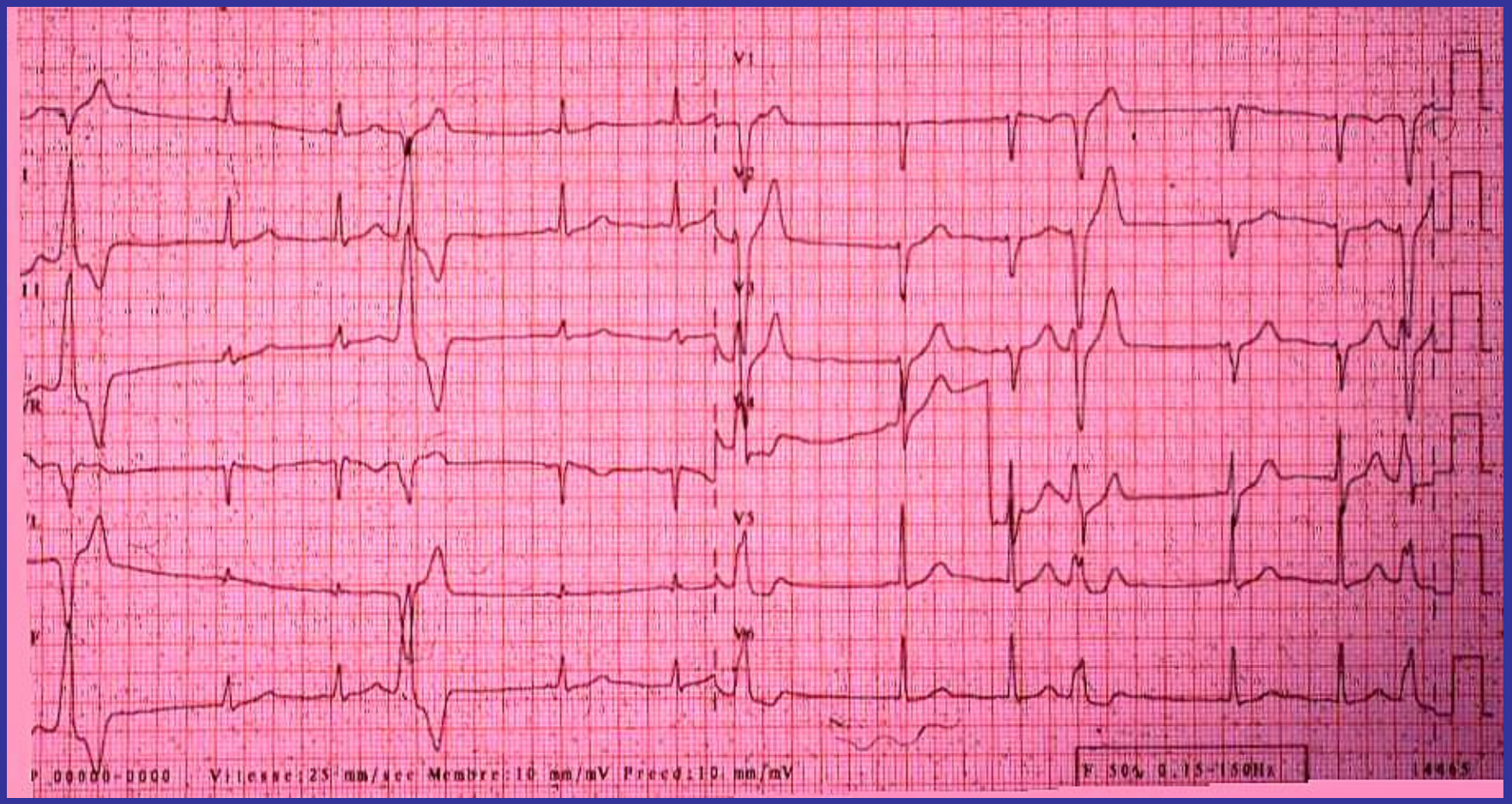
- Le pronostic des ESV est lié à la présence d'une cardiopathie sous jacente

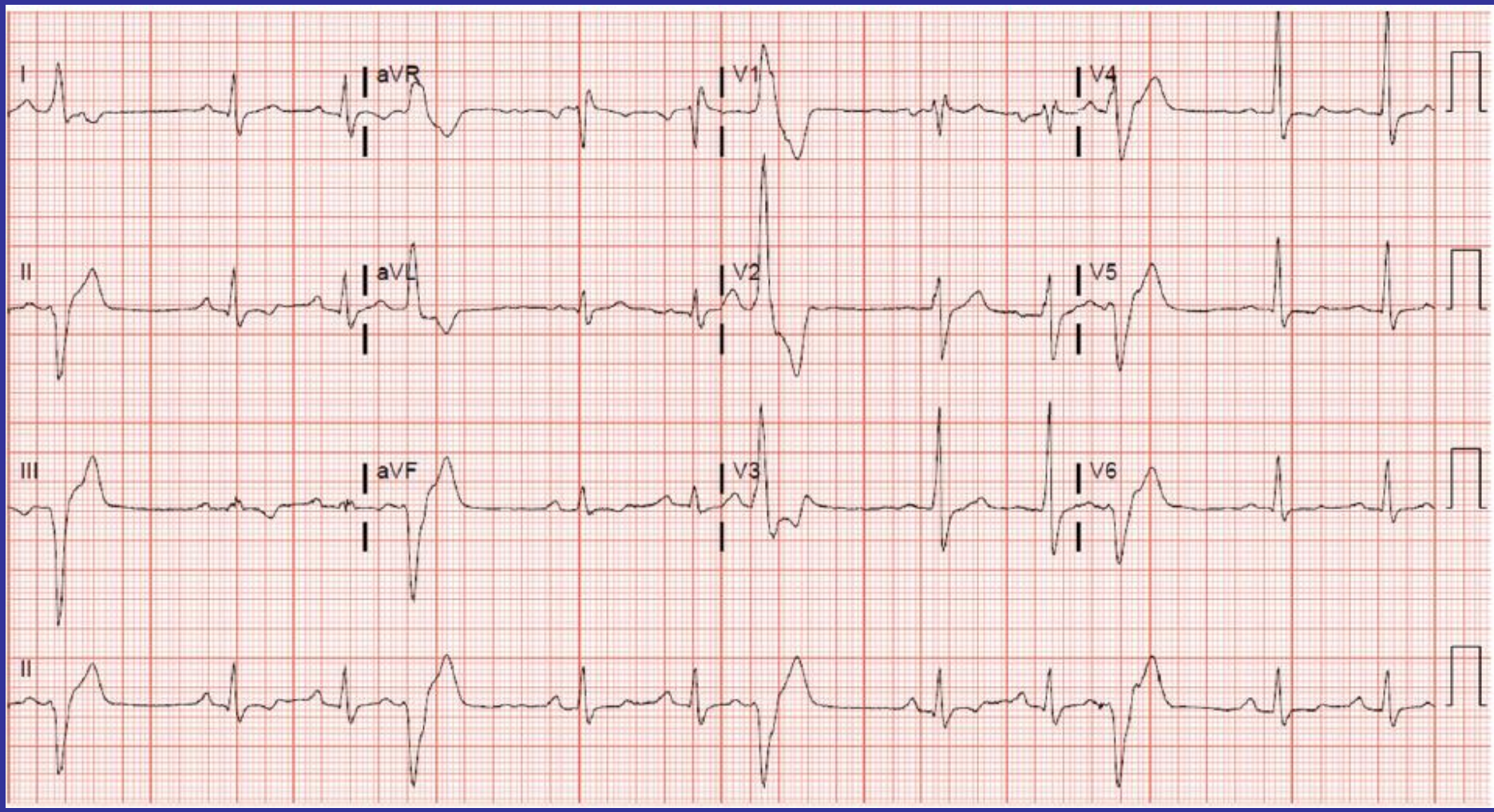
QUAND DEBUTER UN BILAN DIAGNOSTIC?



EHRA consensus, Europace 2019

Localisation de l'ESV



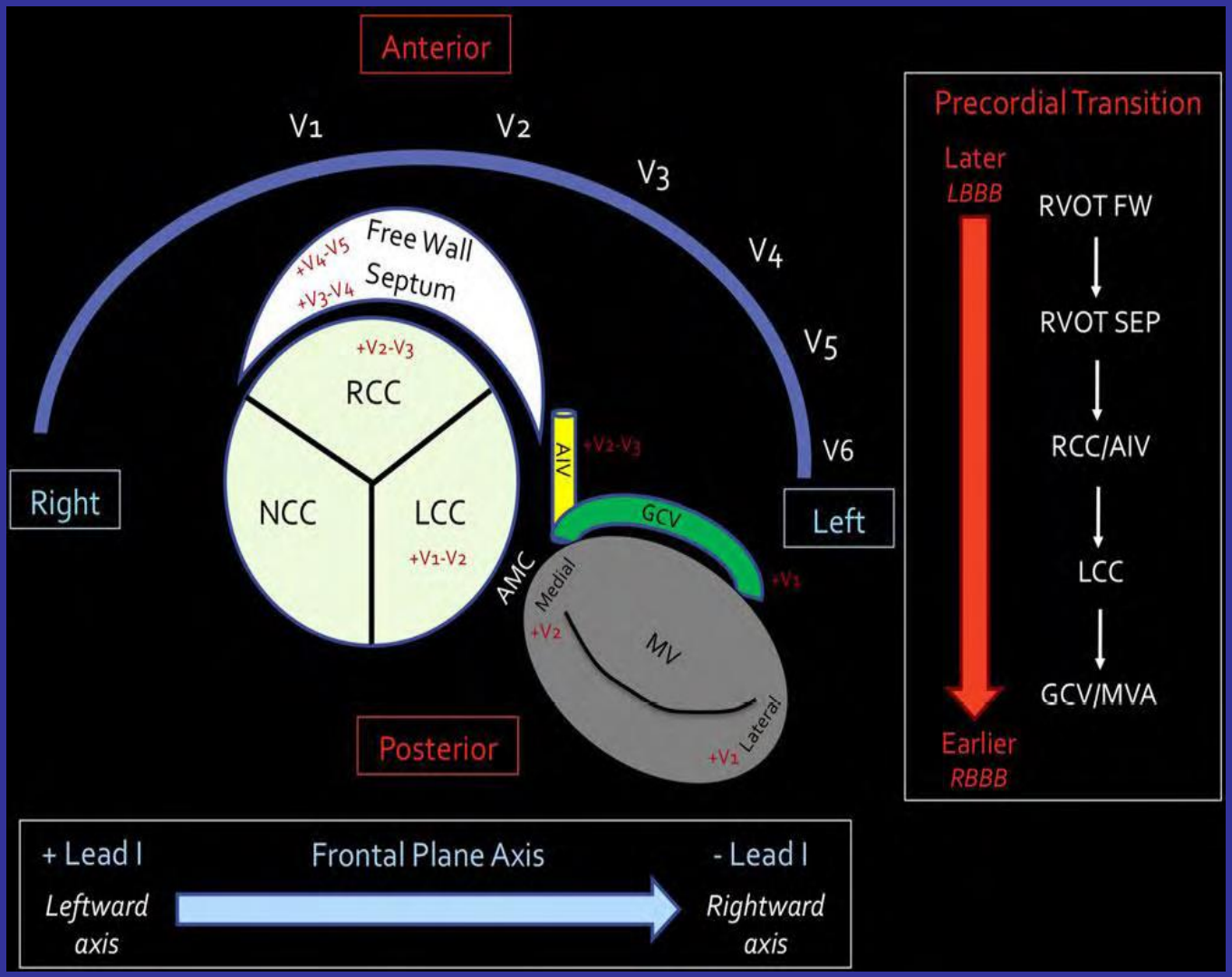

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

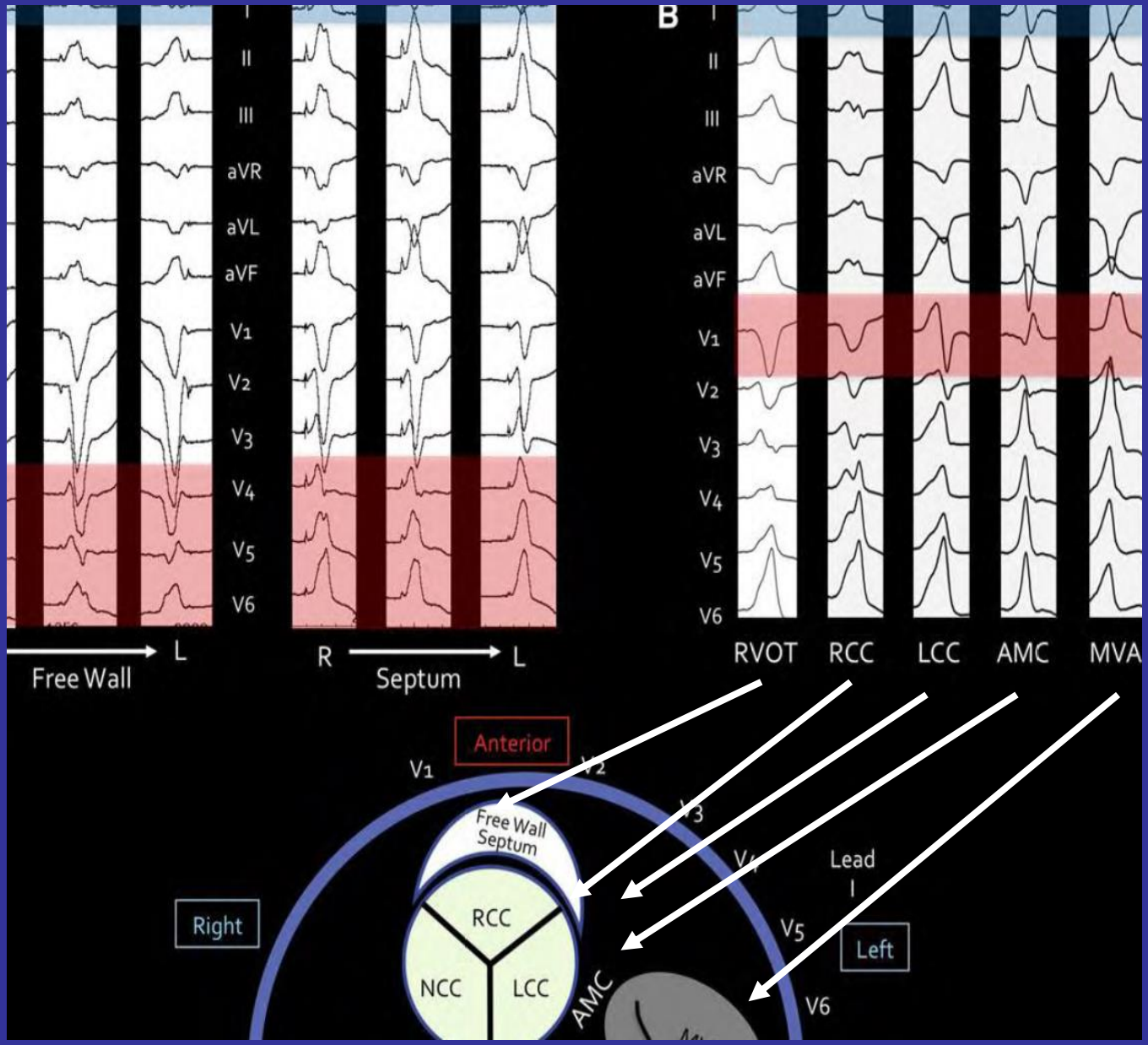
-Retard gauche = provient du VD

-Retard droit= provient du VG

-Si isoélectrique =plutôt septal droit ou gauche

-QRS fins = Septal


Anterior
V1
V2
V3
V4
V5
V6
Free Wall
Septum
+V4-V5
+V3-V4
+V2-V3
RCC
NCC
LCC
+V1-V2
AIV
+V2-V3
GCV
+V1
AMC
Medial
+V2
MV
+V1
Lateral
Right
Left
Posterior
Precordial Transition
Later
LBBB
RVOT FW
RVOT SEP
RCC/AIV
LCC
GCV/MVA
Earlier
RBBB
+ Lead I
Leftward axis
Frontal Plane Axis
- Lead I
Rightward axis


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
L
Free Wall
R
Septum
L
B
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RVOT
RCC
LCC
AMC
MVA
Anterior
V1
V2
V3
V4
V5
V6
Free Wall
Septum
RCC
NCC
LCC
AMC
Right
Lead
I
Left

Recherche de cardiopathie

- Si provenance du VD = rechercher une dysplasie arythmogène du VD= ETT, IRM, angiographie, scintigraphie de phase, signes ECG

- Si provenance du VG = cardiopathie hypertensive, ischémique, valvulaire, myocardite

- Facteurs favorisant: hypokaliémie, hyperthyroidie, anémie,….

Recherche de cardiopathie

- Faire épreuve d'effort : permet d'enregistrer les ESV sur un douze dérivations (problème de l'enregistrement Holter pour la morphologie). Plutôt critère de bénignité si disparition à l'effort
- Bilan de cardiopathie: ETT, IRM++, coronarographie: ESV provenant du VG se majorant à l'effort.

Morphologies ESV et TV

ESV idiopathiques VD : Monomorphes, durée QRS à 120ms, grand voltage, sans crochetages

Probable DVDA: Polymorphes (paroi inférieure du VD), Plusieurs axes, QRS larges, Peu voltées, crochetées

Patients with exercise-associated ventricular ectopy present evidence of myocarditis

Michael Jeserich[1,6*], Bela Merkely[2], Manfred Olschewski[3], Simone Kimmel[4], Gabor Pavlik[5] and Christoph Bode[1]

Abstract

Background: The origin and clinical relevance of exercise-induced premature ventricular beats (PVBs) in patients without coronary heart disease or cardiomyopathies is unknown. Cardiovascular magnetic resonance enables us to non-invasively assess myocardial scarring and oedema. The purpose of our study was to discover any evidence of myocardial anomalies in patients with exercise-induced ventricular premature beats.

Methods: We examined 162 consecutive patients presenting palpitations and documented exercise-induced premature ventricular beats (PVBs) but no history or evidence of structural heart disease. Results were compared with 70 controls matched for gender and age. ECG-triggered, T2-weighted, fast spin echo triple inversion recovery sequences and late gadolinium enhancement were obtained as well as LV function and dimensions.

Results: Structural anomalies in the myocardium and/or pericardium were present in 85 % of patients with exercise-induced PVBs. We observed a significant difference between patients with PVBs and controls in late gadolinium enhancement, that is 68 % presented subepicardial or midmyocardial lesions upon enhancement, whereas only 9 % of the controls did so ($p < 0.0001$). More patients presented pericardial enhancement (35 %) or pericardial thickening (27 %) compared to controls (21 % and 13 %, $p < 0.0001$). Myocardial oedema was present in 37 % of the patients and in only one control, $p < 0.0001$. Left ventricular ejection fraction did not differ between patients and controls (63.1 ± 7.9 vs. 64.7 ± 7.0, $p = 0.13$).

Conclusions: The majority of patients with exercise-associated premature ventricular beats present evidence of myocardial disease consistent with acute or previous myocarditis or myopericarditis.

Keywords: Cardiovascular magnetic resonance , Myocarditis, Pericarditis, Premature ventricular beats, Late gadolinium enhancement, STIR

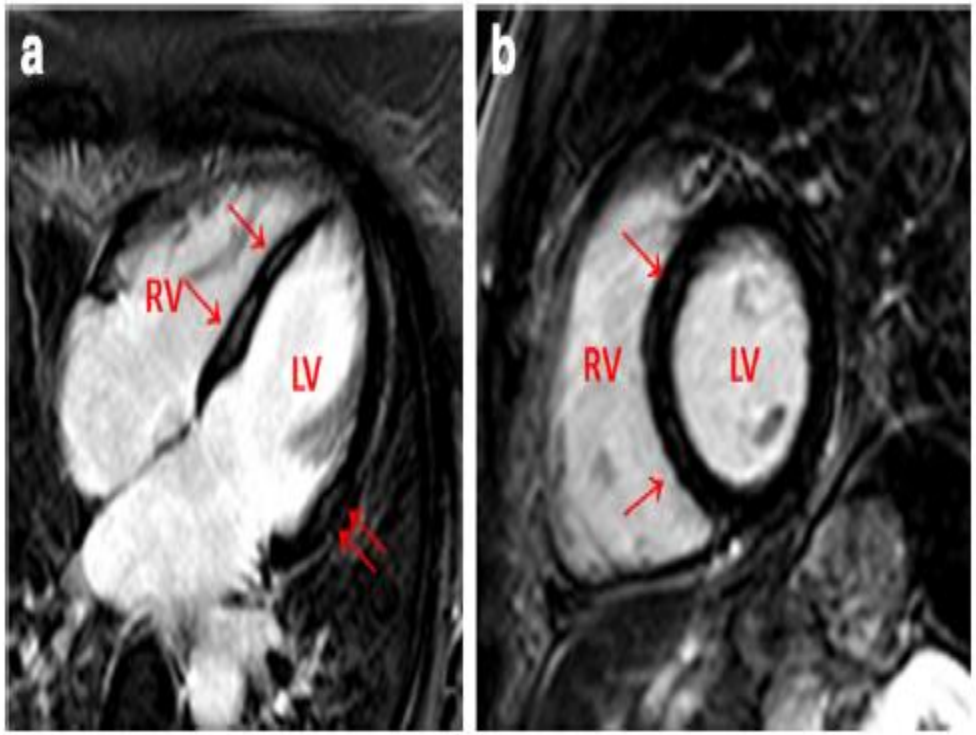
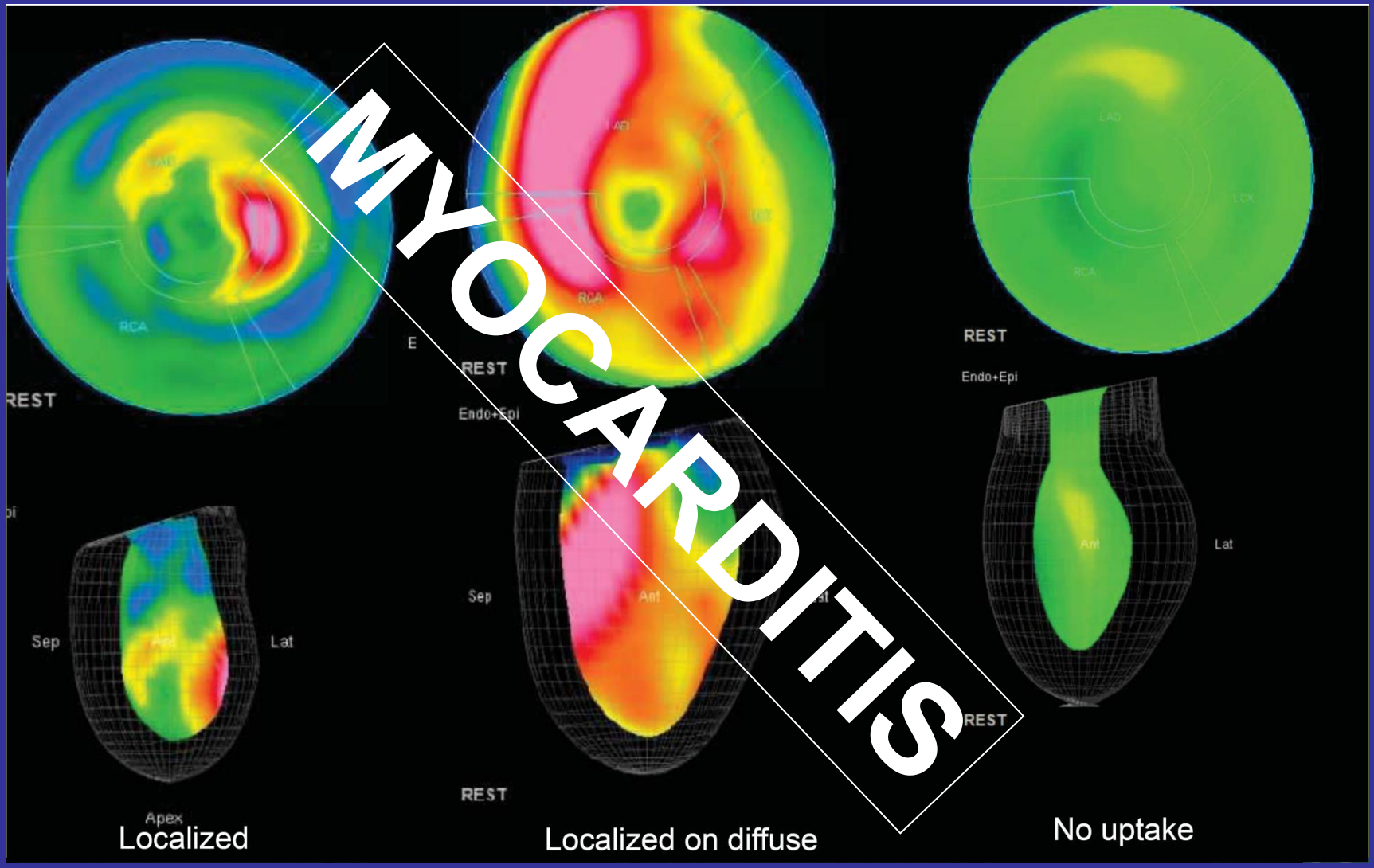
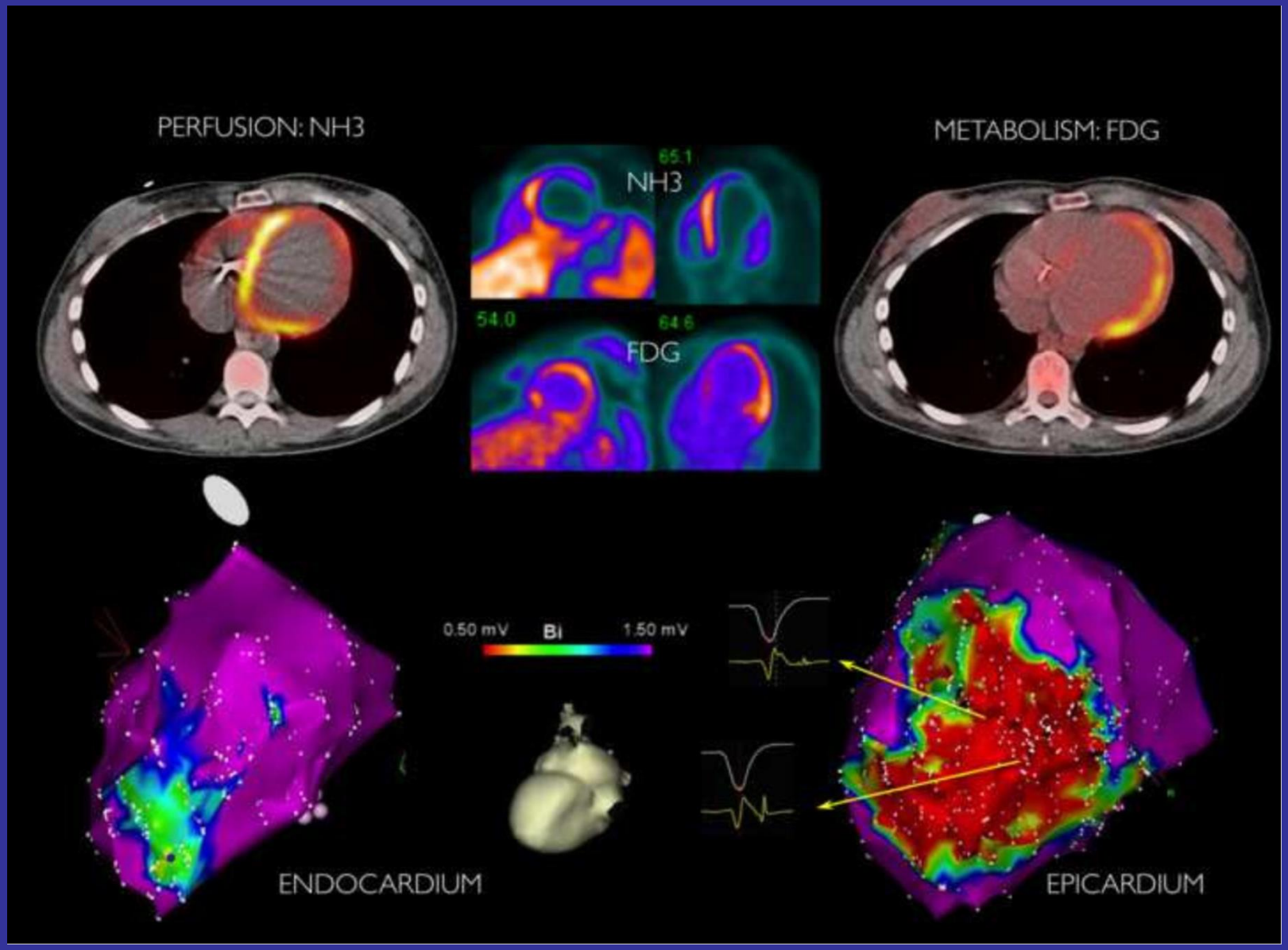


Fig. 2 a/b Late-enhancement image of one patient with exercise induced PVBs. Note the patchy enhancement of the midwall septal (↑) and lateral wall (↑↑). Four and two-chamber view. LV: Left ventricle. RV: Right ventricle

PET-Scan



MYOCARDIAL BIOPSY IF NECESSARY



Correlation FDG-PET SCAN and low voltage: 74%

ESV et cardiomyopathie

-Diagnostic certain de cardiopathie sous jacente = ischémique, hypertensive, valvulaire

-CMD= problème ESV est la cause ou la conséquence de la CMD=CMD causée par ESV dans 5-30% des cas suivant les ESV (manque de données++)

Concept de CMP induite par les ESV; seul moyen de savoir est de voir la régression de la CMD après traitement des ESV (amélioration de 5-10% de la FEVG)

Facteurs associés au risque de développement d'une CMD avec ESV

- Caractère asymptomatique
- Nombre d'ESV
- Largeur des QRS
- Caractère épicardique
- Présence d'une cardiopathie sous jacente
- Caractère intercalant

Premature ventricular contraction-induced cardiomyopathy: Related clinical and electrophysiologic parameters

Marie Sadron Blaye-Felice, MD,[*] David Hamon, MD,[†] Frédéric Sacher, MD,[‡] Patrizio Pascale, MD,[§] Anne Rollin, MD,[*] Alexandre Duparc, MD,[*] Pierre Mondoly, MD,[*] Nicolas Derval, MD,[‡] Arnaud Denis, MD,[‡] Christelle Cardin, MD,[*] Mélèze Hocini, MD,[‡] Pierre Jaïs, MD,[‡] Jürg Schlaepfer, MD,[§] Vanina Bongard, MD,[*] Didier Carrié, MD,[*] Michel Galinier, MD,[*] Etienne Pruvot, MD,[§] Nicolas Lellouche, MD,[†] Michel Haïssaguerre, MD,[‡] Philippe Maury, MD[*]

From the [*]University Hospital Rangueil, Toulouse, France, [†]University Hospital Mondor, Paris, France, [‡]University Hospital Haut-Leveque, Bordeaux-Pessac, France, and [§]CHUV, Lausanne, Switzerland.

BACKGROUND Factors associated with premature ventricular contraction-induced cardiomyopathy (PVCi-CMP) remain debated.

OBJECTIVE The purpose of this study was to test the correlation of various factors to the presence PVCi-CMP in a large multicenter population.

METHODS One hundred sixty-eight consecutive patients referred for ablation of frequent premature ventricular contractions (PVCs) were included. Patients were divided into 2 groups: group 1 with suspected PVCi-CMP (96 patients, ejection fraction 38% ± 10%, left ventricular end-diastolic diameter 62 ± 8 mm, with or without additional structural heart disease); and group 2 (control group, 72 patients with normal ejection fraction and left ventricular dimensions). Various clinical and electrophysiologic parameters were compared between groups.

RESULTS In univariate analysis, left ventricular origin of PVC, lack of palpitations, long PVC coupling interval, epicardial origin of the focus, long sinus beat QRS duration, male gender, high PVC burden, presence of polymorphic PVCs, high PVC QRS duration, and older age were significantly related to the presence of PVCi-CMP. In multivariate analysis, only lack of palpitations, PVC burden, and epicardial origin remained significantly and independently correlated with the presence of cardiomyopathy. Even if sinus QRS duration or PVC left ventricular origin were also found independently linked to PVCi-CMP in the whole population, they were no longer correlated when patients with additional heart disease were excluded.

CONCLUSION Lack of palpitations, PVC burden, and epicardial origin are independent factors that identify patients prone to developing PVCi-CMP.

KEYWORDS Premature ventricular contraction; Cardiomyopathy; Tachycardia-induced cardiomyopathy; Radiofrequency ablation

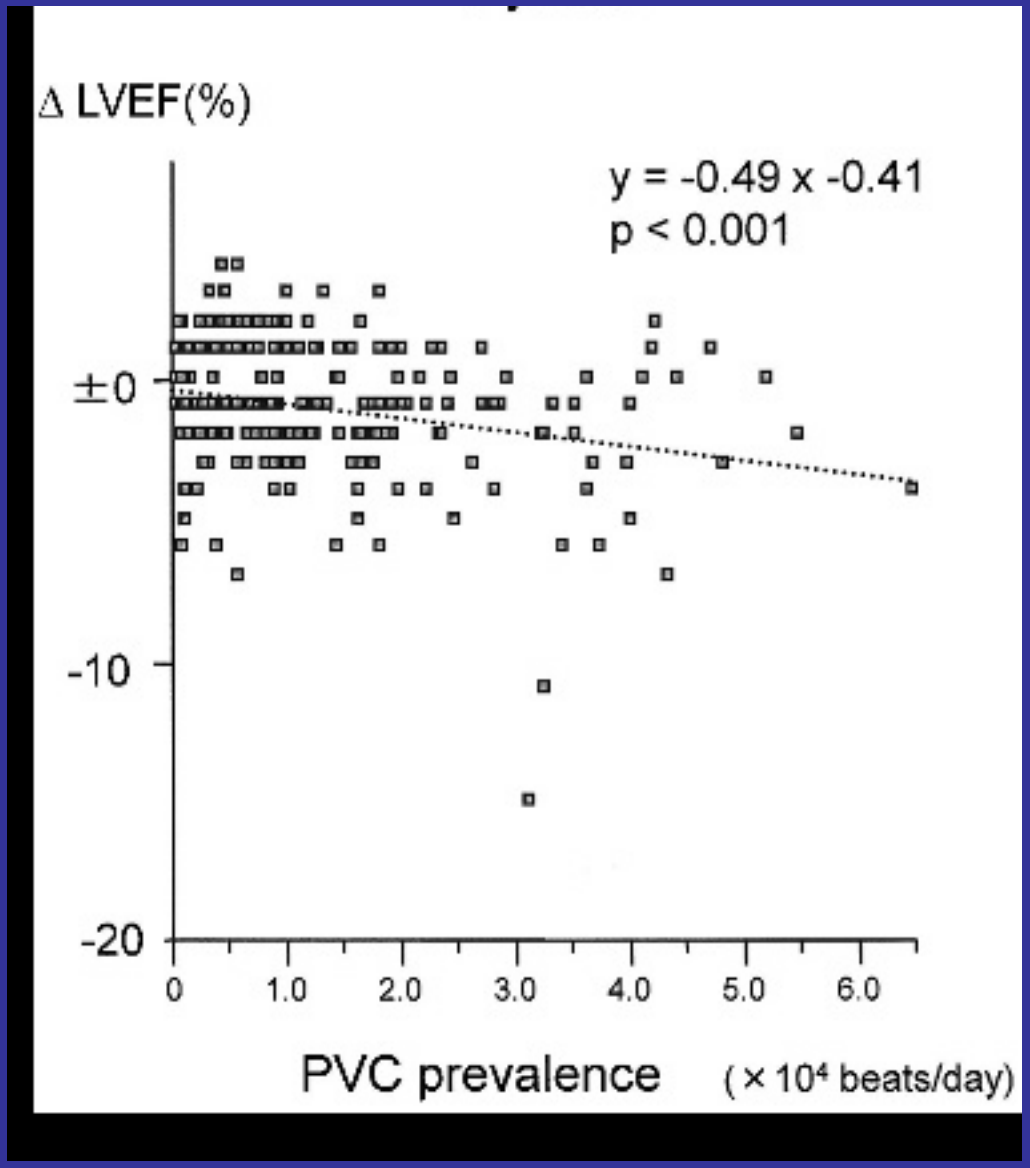
ABBREVIATIONS **CMP** = cardiomyopathy; **CS** = coronary sinus; **LV** = left ventricle; **LVEDD** = left ventricular end-diastolic diameter; **LVEF** = left ventricular ejection fraction; **PVC** = premature ventricular contraction; **PVCi-CMP** = premature ventricular contraction-induced cardiomyopathy; **RF** = radiofrequency; **RV** = right ventricle

(Heart Rhythm 2016;13:103–110)

Introduction

frequent PVCs, for example, after radiofrequency (RF)

Nombre d'ESV



Niwano et al, Heart 2009

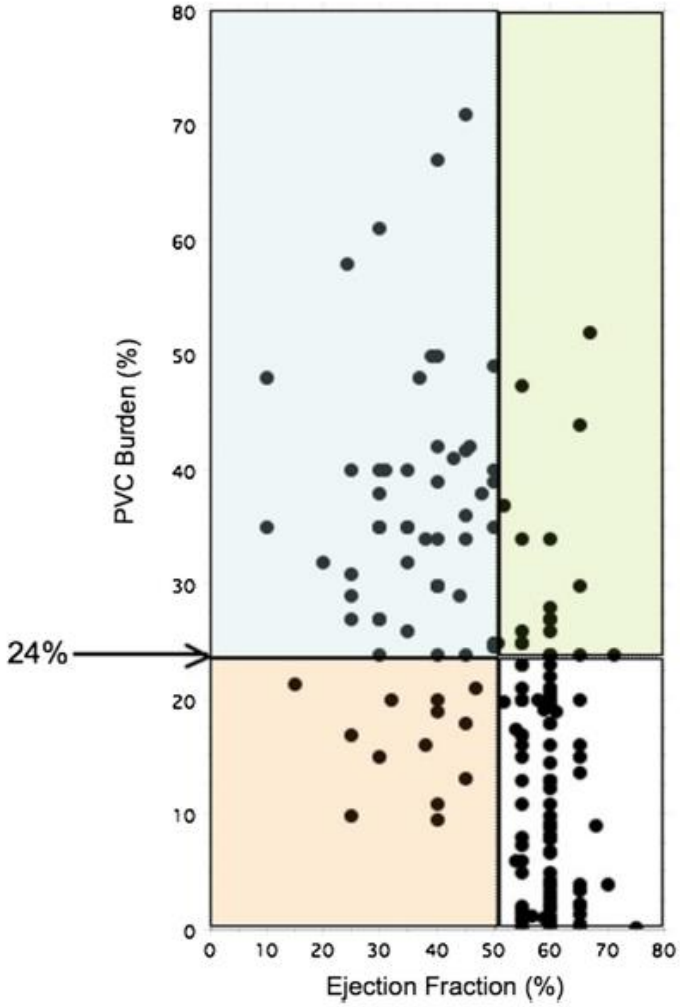


FIG 3. The relationship between PVC burden and ejection fraction. (Reproduced with permission from Baman et al.[63]) (Color version of figure is available online.)

10-25% selon les séries

ARSENAL THERAPEUTIQUE

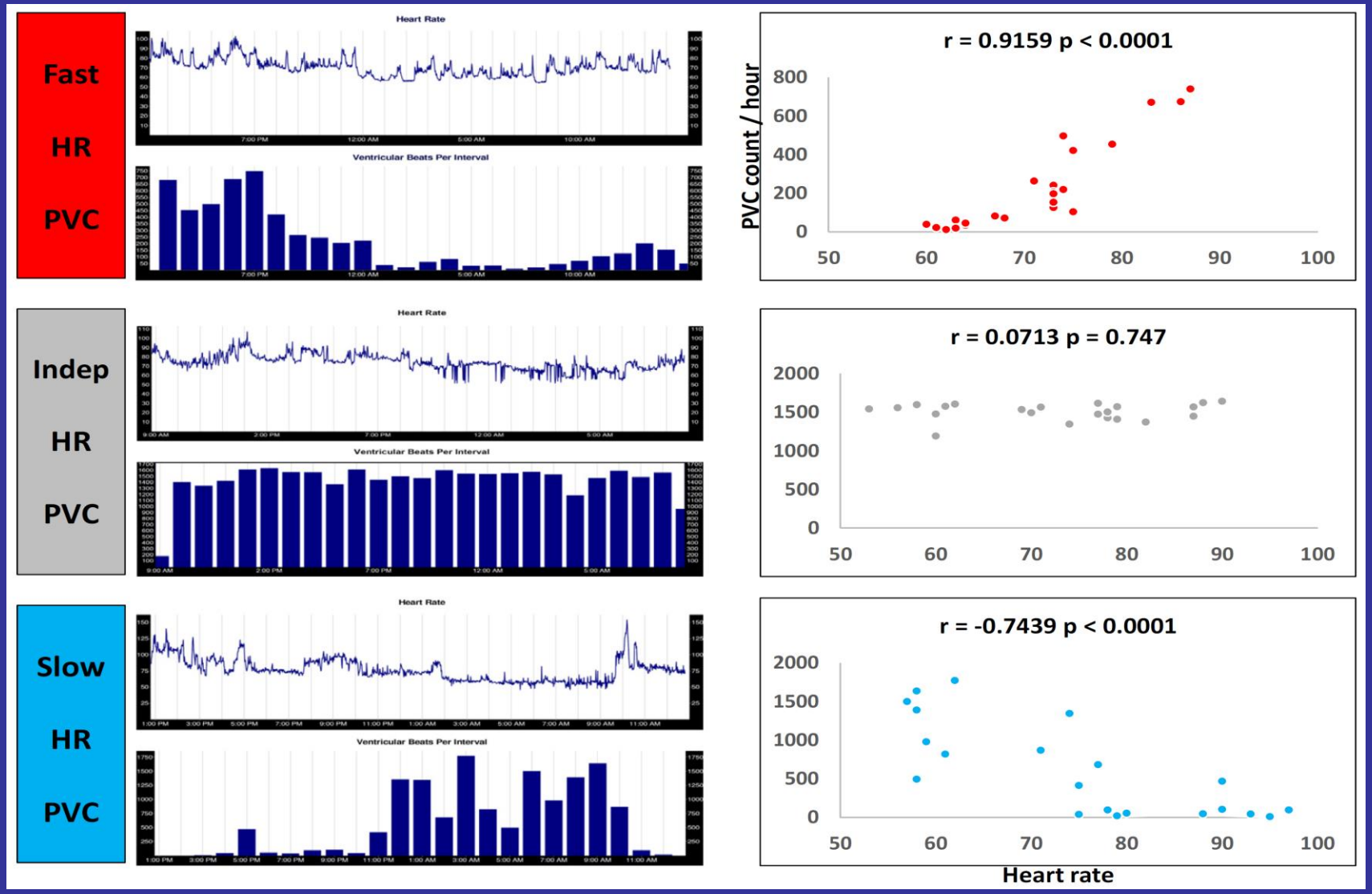
- **Règles hygiéno-diététiques (pour tout le monde)**:
  - Excès de boissons énergétiques et caféine
  - Nicotine, OH
  - Stress
  - Exercice physique
  - Relaxation, cohérence cardiaque, hypnose…

- **Traitement médicamenteux**
  - Bétabloquants/anticalciques
  - Ic (Flécaine*, Rythmol*)
  - Amiodarone: 2ème intention ou sujets âgés: effets secondaires au long cours++
  - Avantages: non invasif, « acceptation »
  - Inconvénients: efficacité intermédiaire (-8376 vs -21799/24h)
  - 330 patients rando: récurrence des ESV = 88.6% (vs 19.4%)
  - complications étalées dans le temps

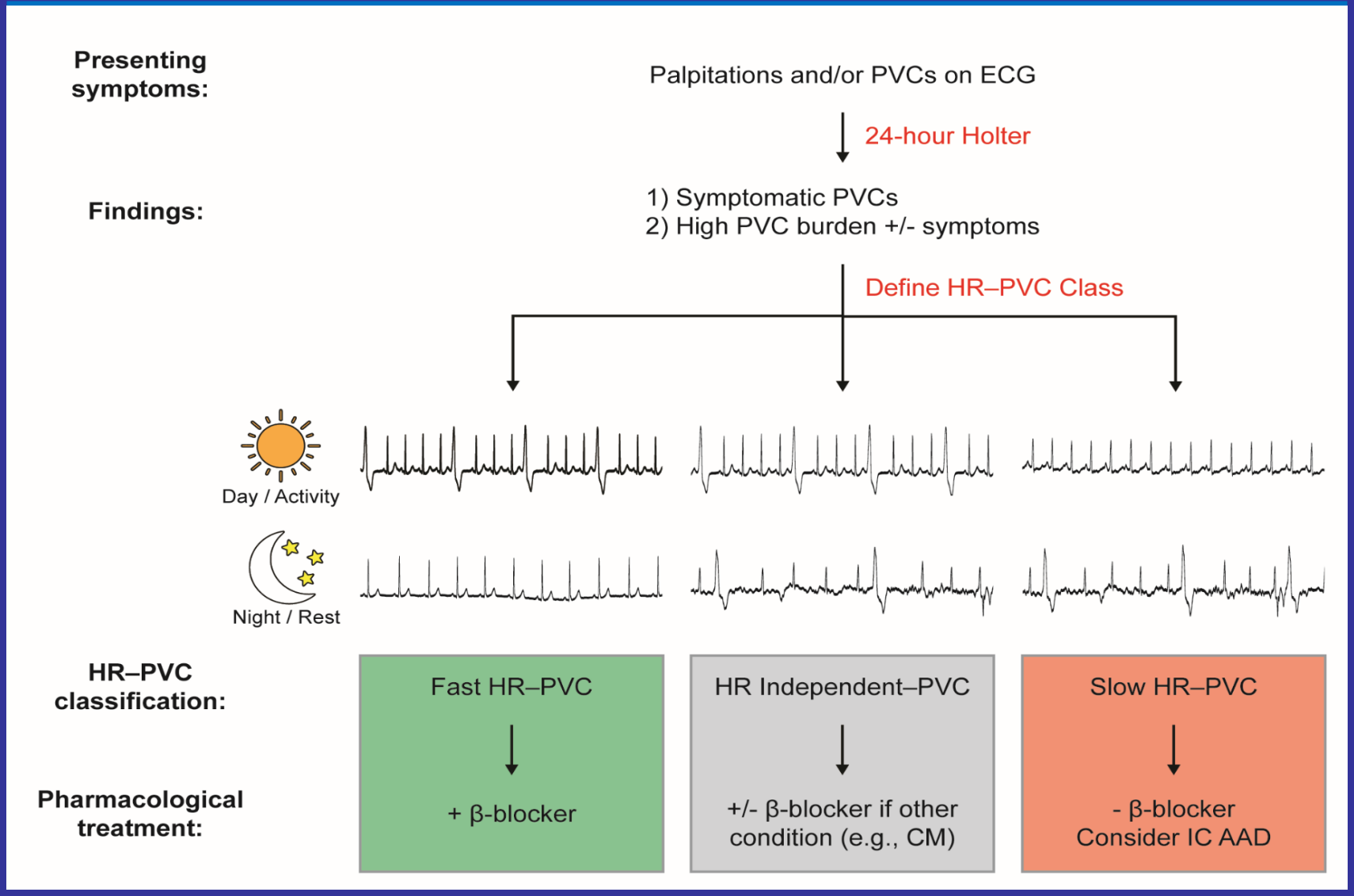
Zhong L et al. HR 2014
Ling Z et al. CircAE 2014

- **Ablation**
  - ≥70% succès long terme (85% avec AAD add-on)
  - Mais complications per-procédurales (5%/2% graves)

Comment choisir entre AA de classe Ic ou beta-bloquants?



Hamon D et al. Heart Rhythm 2017


Presenting symptoms:
Palpitations and/or PVCs on ECG
24-hour Holter
Findings:
1) Symptomatic PVCs
2) High PVC burden +/- symptoms
Define HR–PVC Class
Day / Activity
Night / Rest
HR–PVC classification:
Fast HR–PVC
HR Independent–PVC
Slow HR–PVC
Pharmacological treatment:
+ β-blocker
+/- β-blocker if other condition (e.g., CM)
- β-blocker
Consider IC AAD

INDICATIONS DU TRAITEMENT MEDICAMENTEUX

Patients asymptomatiques

NON **DYSFONCTION VG** **OUI**

TAUX D'ESV<ou > 10% au Holter ECG?

-Patient ESV < 10% =
Règles hygiéno-diététiques
Suivi Holter (stabilité)

-Patient ESV> 10%:?
PHRC

-Patient ESV < 10% =
Les ESV ne sont probablement pas responsables de la dysfonction VG= Tt de l'IC

-Patient ESV> 10%:possible cardiopathie rythmique
= amiodarone ou ablation

CONCLUSION

-Pour les patients asymptomatiques avec dysfonction VG et ESV> 10%: amiodarone ou plutôt ablation si sujet jeune (< 75 ans). Si ESV > 10% sans dysfonction VG ?. Si ESV < 10% et fonction VG normale pas de traitement

-Toujours traiter une cardiopathie sous-jacente qui peut améliorer les ESV

-Les traitements AA possibles: AA de classe Ic, béta-bloquants, Sotalol, Amiodarone

-Tenir compte des contre-indications et effets secondaires++

-Regarder le profil Holter des ESV: diurne vs nocturne pour adapter le traitement médical