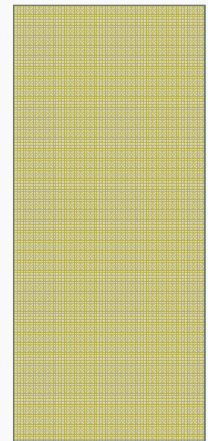


# CARDIOPATHIE DE STRESS

JÉRÉMY ROSMAN

DESC REANIMATION – OCTOBRE 2015



# HISTORIQUE

# HISTORIQUE

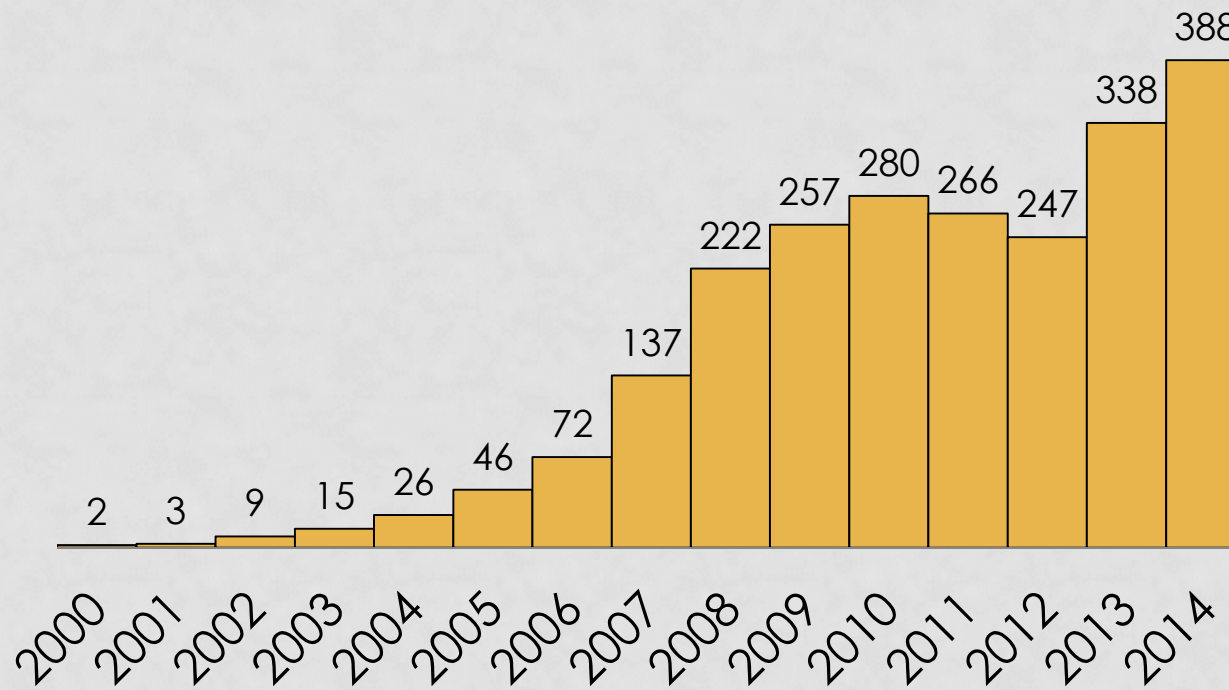
- 1<sup>er</sup> cas clinique : 1990



*Sato, Tokyo: Kagakuhyoronsha Publishing, 1990.*

# HISTORIQUE

## Référencement MedLine

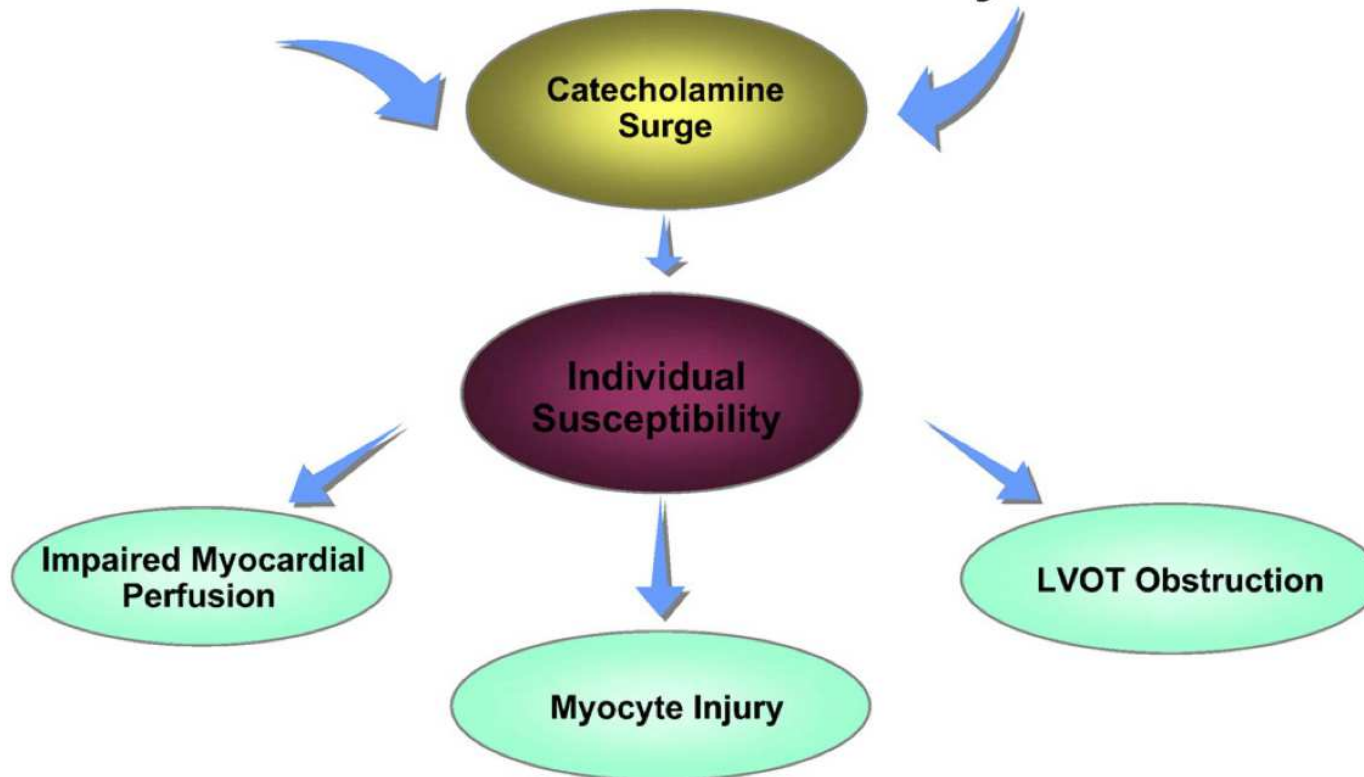


# PHYSIOPATHOLOGIE

# PHYSIOPATHOLOGIE

**Mental Stress**

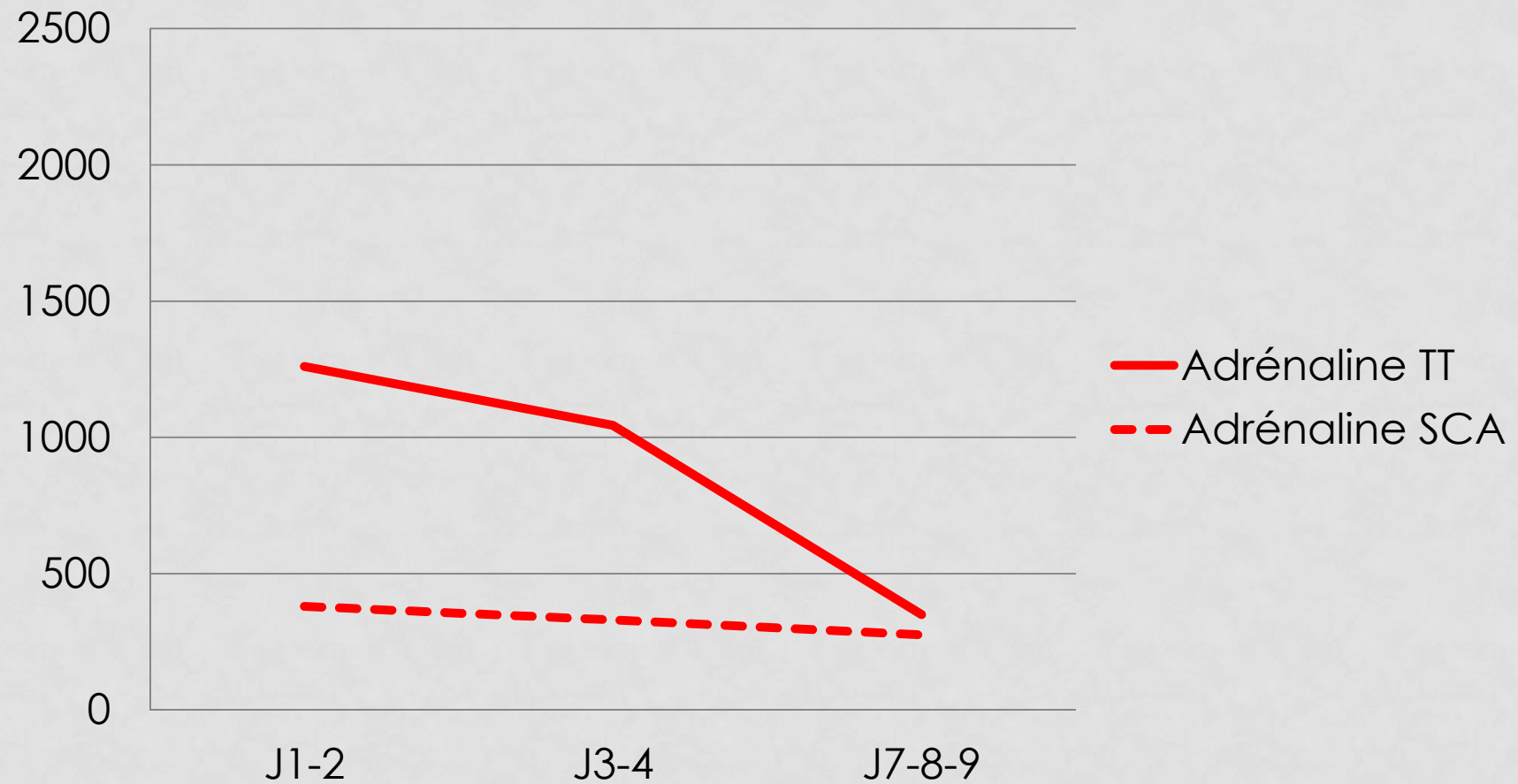
**Physical Stress**



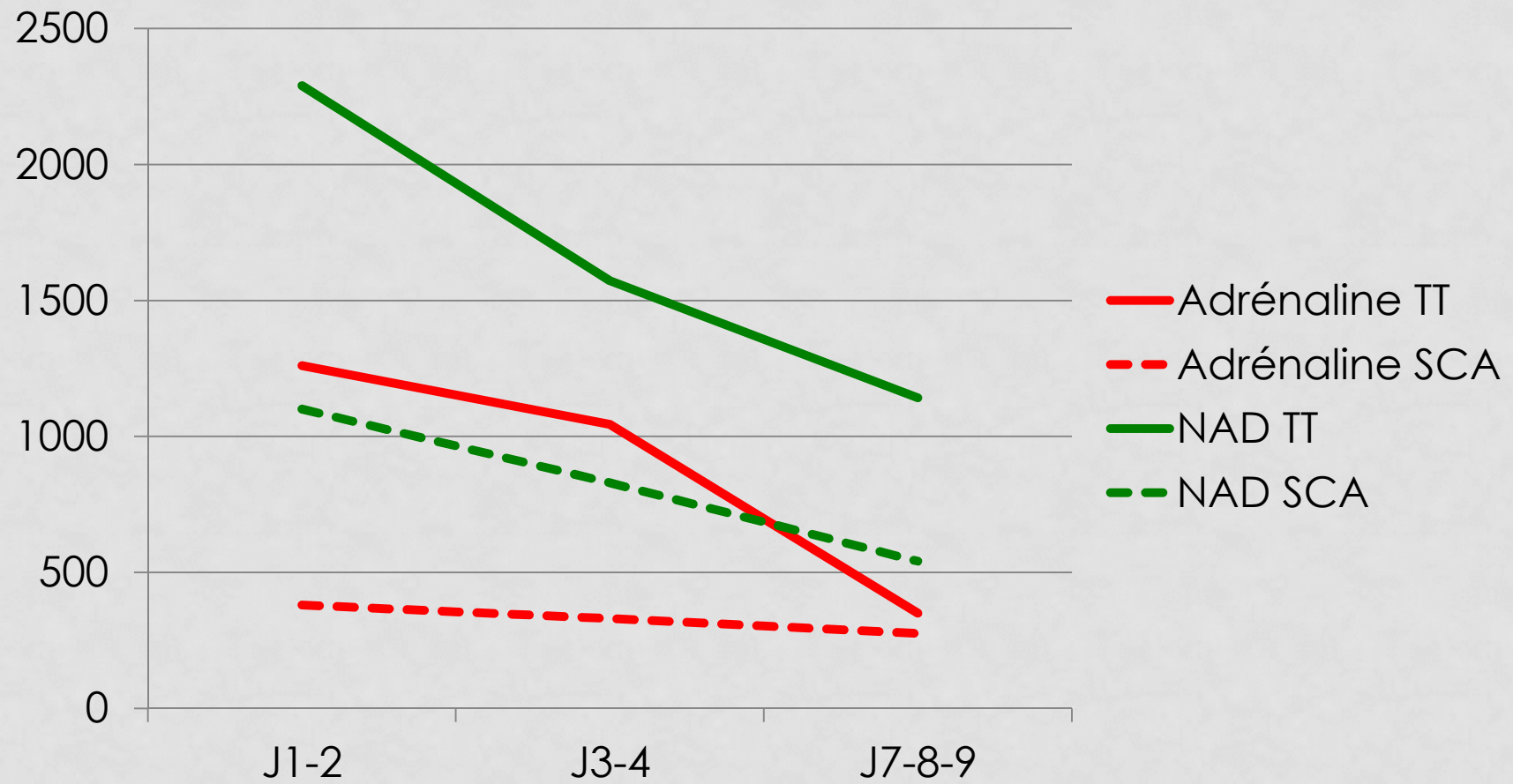
Proposed pathophysiology of ACS.



# PHYSIOPATHOLOGIE

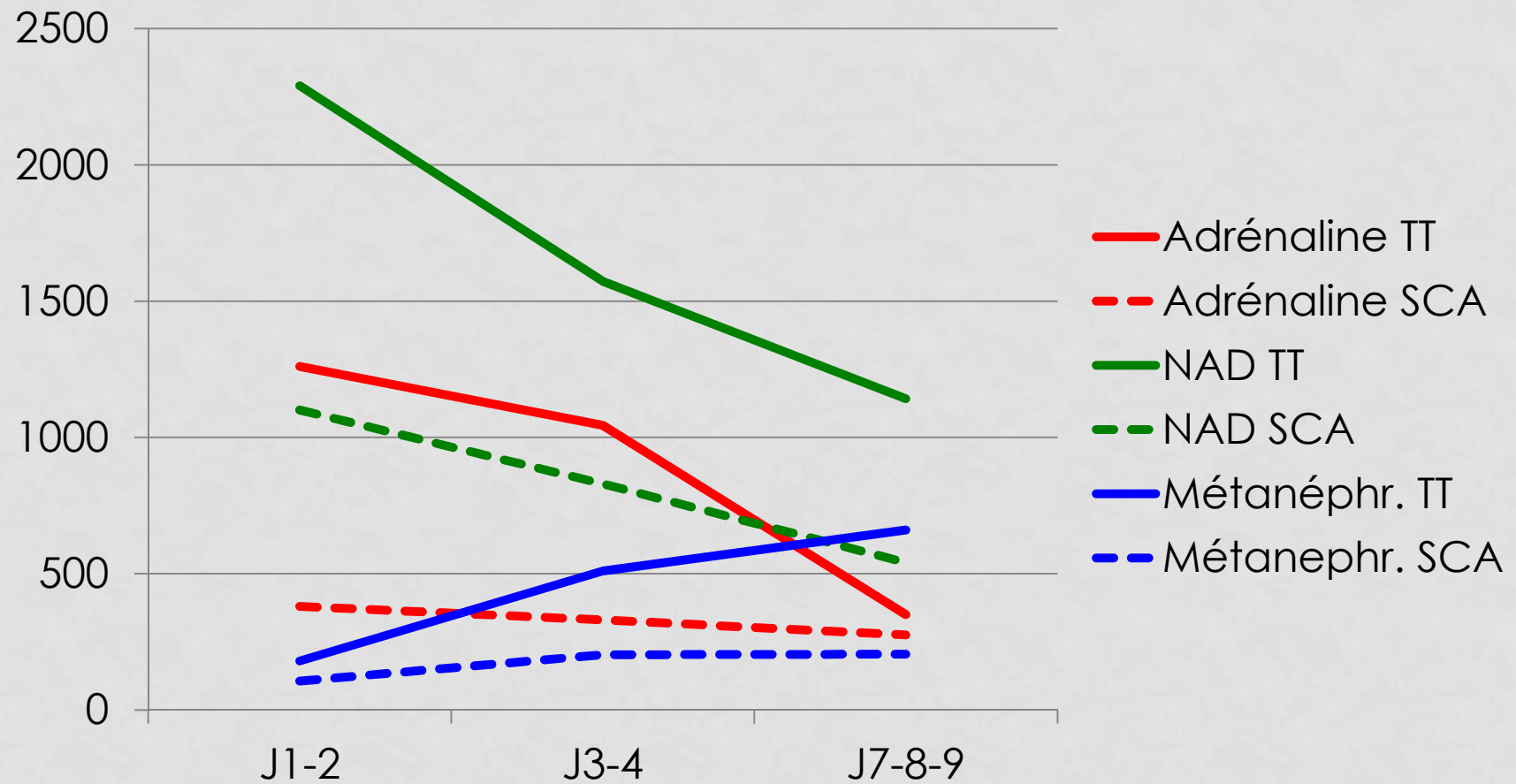


# PHYSIOPATHOLOGIE

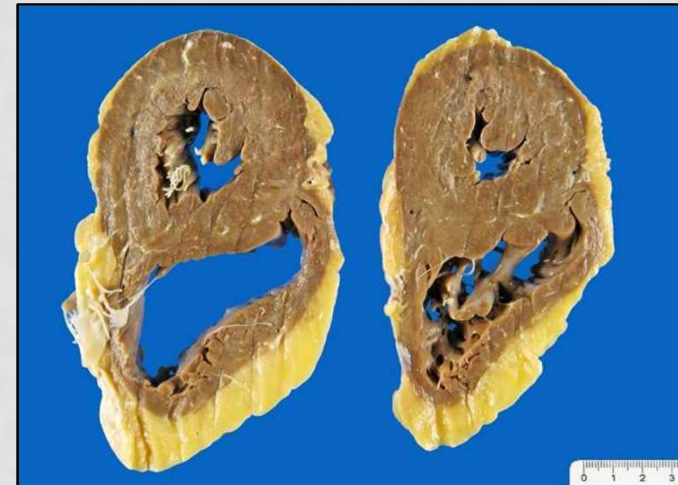




# PHYSIOPATHOLOGIE



# PHYSIOPATHOLOGIE



Josué, C R Soc Biol (Paris). 1907

DIAGNOSTIC

ORIGINAL ARTICLE

# Clinical Features and Outcomes of Takotsubo (Stress) Cardiomyopathy

C. Templin, J.R. Ghadri, J. Diekmann, L.C. Napp, D.R. Bataiosu, M. Jaguszewski, V.L. Cammann, A. Sarcon, V. Geyer, C.A. Neumann, B. Seifert, J. Hellermann, M. Schwyzer, K. Eisenhardt, J. Jenewein, J. Franke, H.A. Katus, C. Burgdorf, H. Schunkert, C. Moeller, H. Thiele, J. Bauersachs, C. Tschöpe, H.-P. Schultheiss, C.A. Laney, L. Rajan, G. Michels, R. Pfister, C. Ukena, M. Böhm, R. Erbel, A. Cuneo, K.-H. Kuck, C. Jacobshagen, G. Hasenfuss, M. Karakas, W. Koenig, W. Rottbauer, S.M. Said, R.C. Braun-Dullaeus, F. Cuculi, A. Banning, T.A. Fischer, T. Vasankari, K.E.J. Airaksinen, M. Fijalkowski, A. Rynkiewicz, M. Pawlak, G. Opolski, R. Dworakowski, P. MacCarthy, C. Kaiser, S. Osswald, L. Galiuto, F. Crea, W. Dichtl, W.M. Franz, K. Empen, S.B. Felix, C. Delmas, O. Lairez, P. Erne, J.J. Bax, I. Ford, F. Ruschitzka, A. Prasad, and T.F. Lüscher

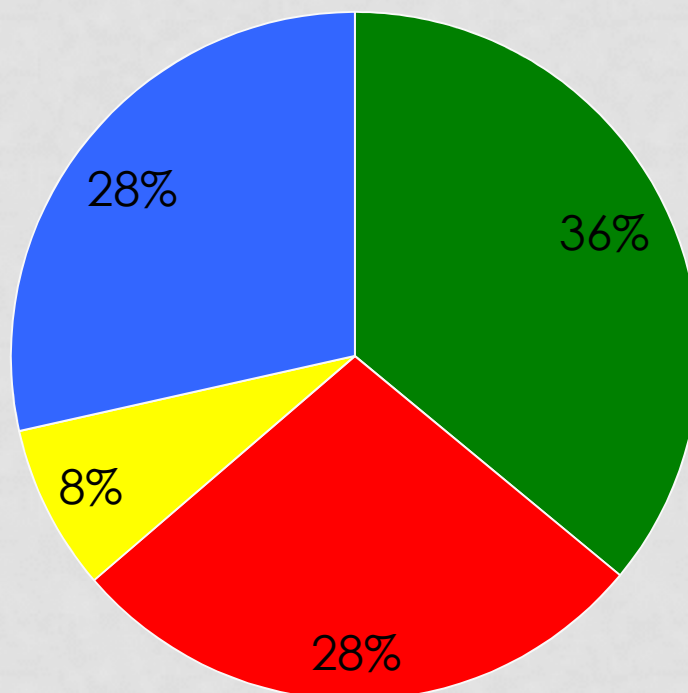


# DIAGNOSTIC

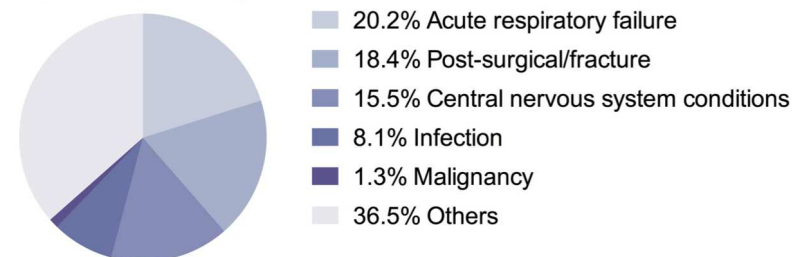
- Registre international du Takotsubo
- 9 pays, 26 centres
- Récupération rétrospective de 1750 cas entre 1998 et 2014 :
  - Clinique
  - Thérapeutique
  - Pronostic
- Comparaison avec cas de SCA (pairées âge/sexe)

**InterTAK** Registry

# DIAGNOSTIC FACTEURS DECLENCHANTS

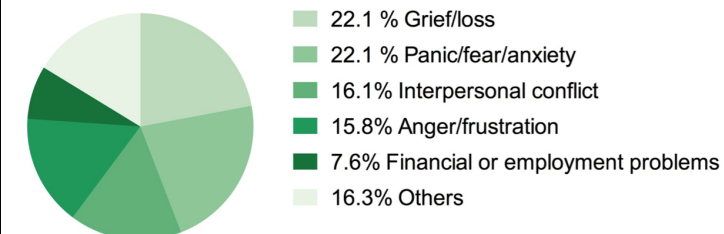


**Physical triggers  
(N=630, 36.0%)**



- Physique
- Emotionnel
- Les deux
- Non retrouvé

**Emotional triggers  
(N=485, 27.7%)**

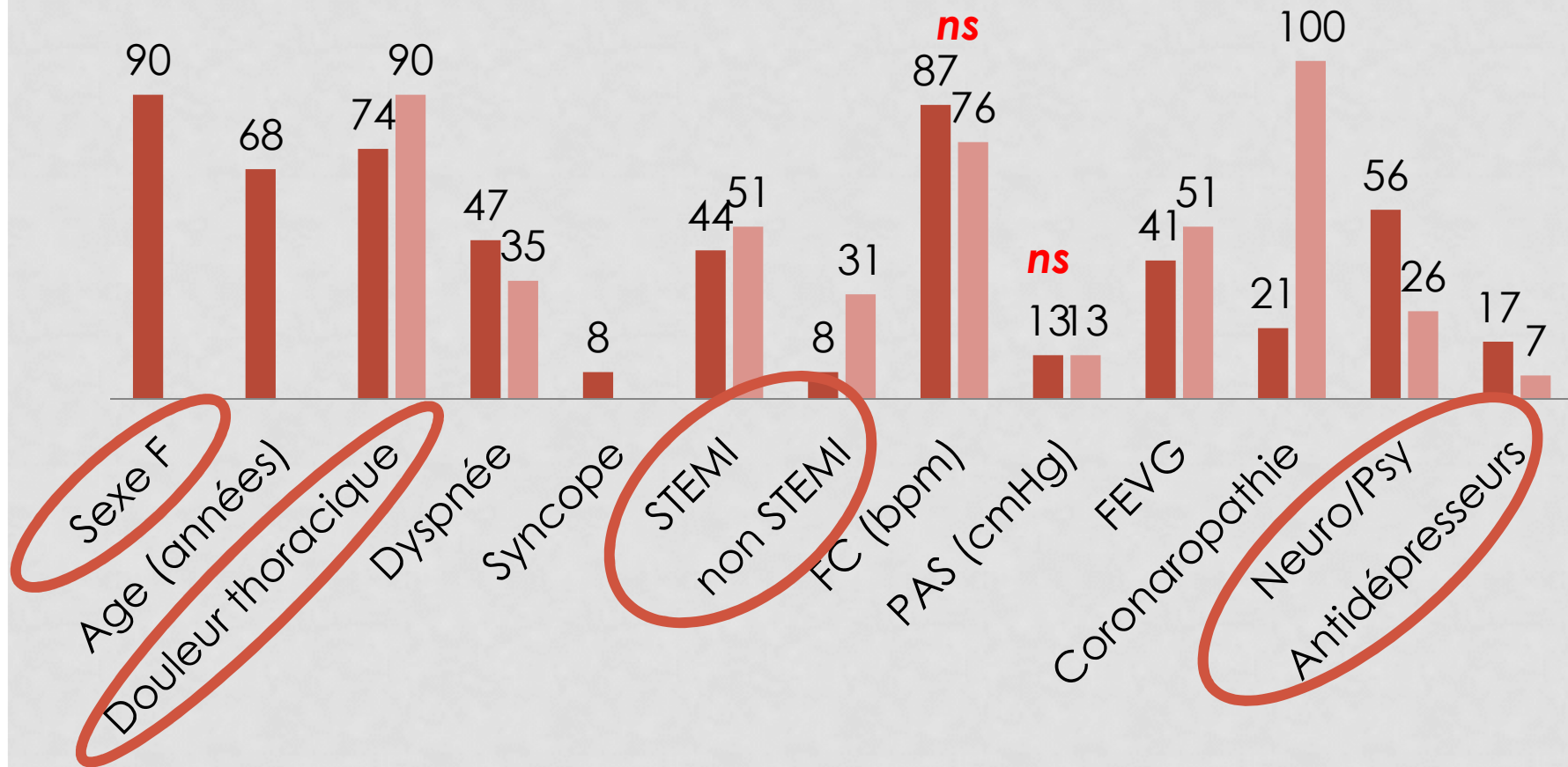


*Templin and al., NEJM 2015.*

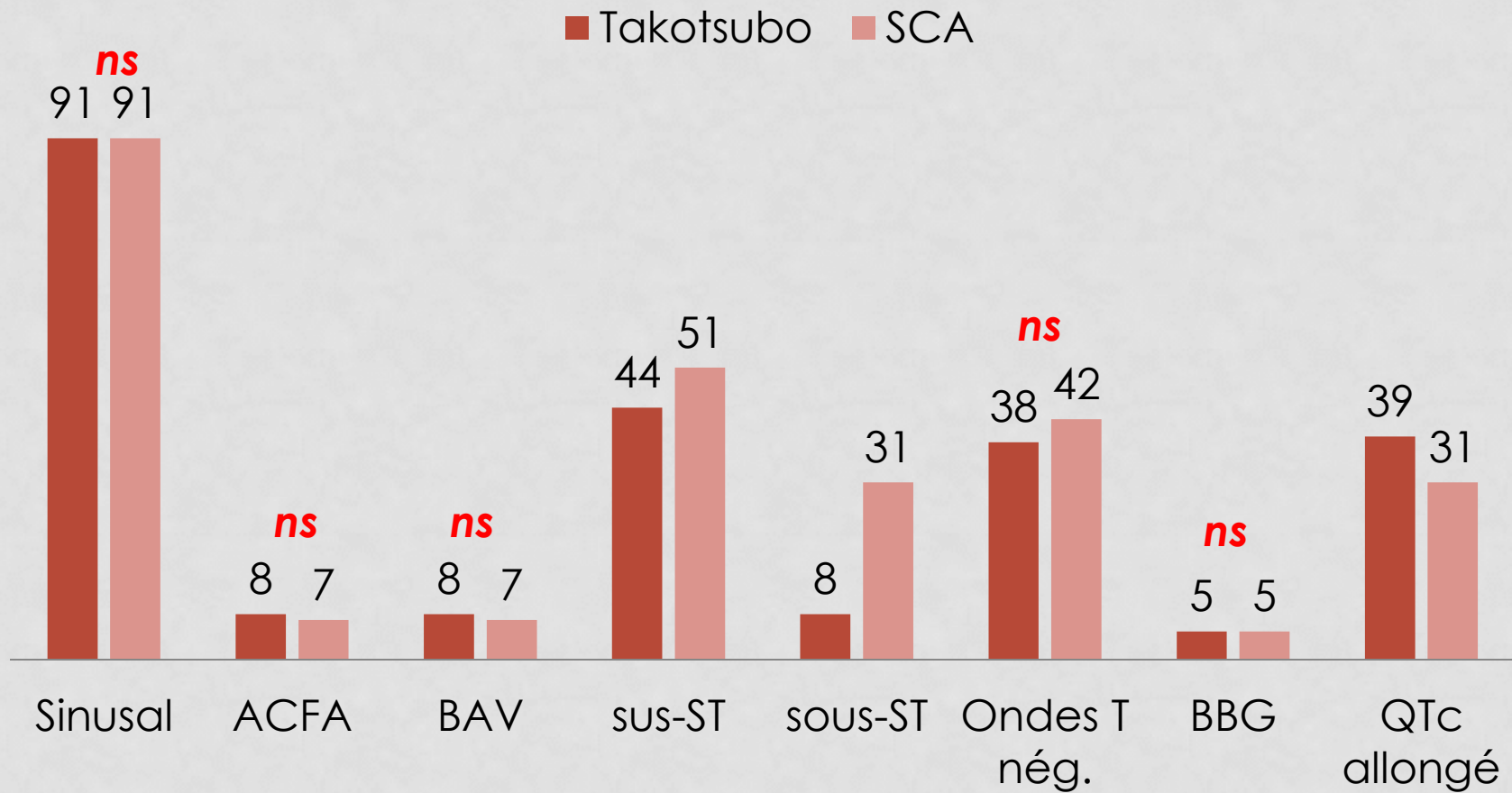


# DIAGNOSTIC CLINIQUE

■ Takotsubo ■ SCA

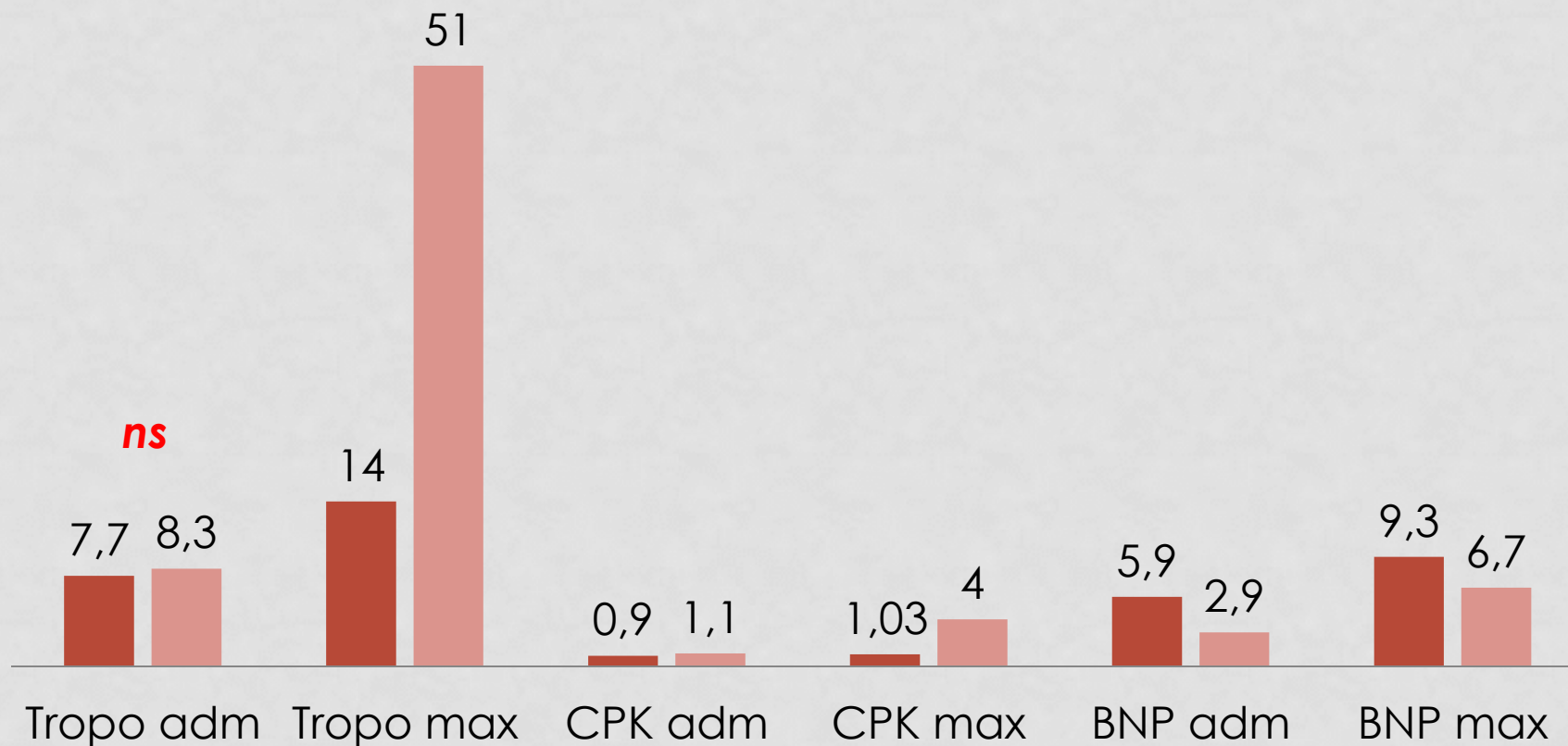


# DIAGNOSTIC MODIFICATIONS ECG (%)



# DIAGNOSTIC BIOLOGIE (% LIMITE SUP)

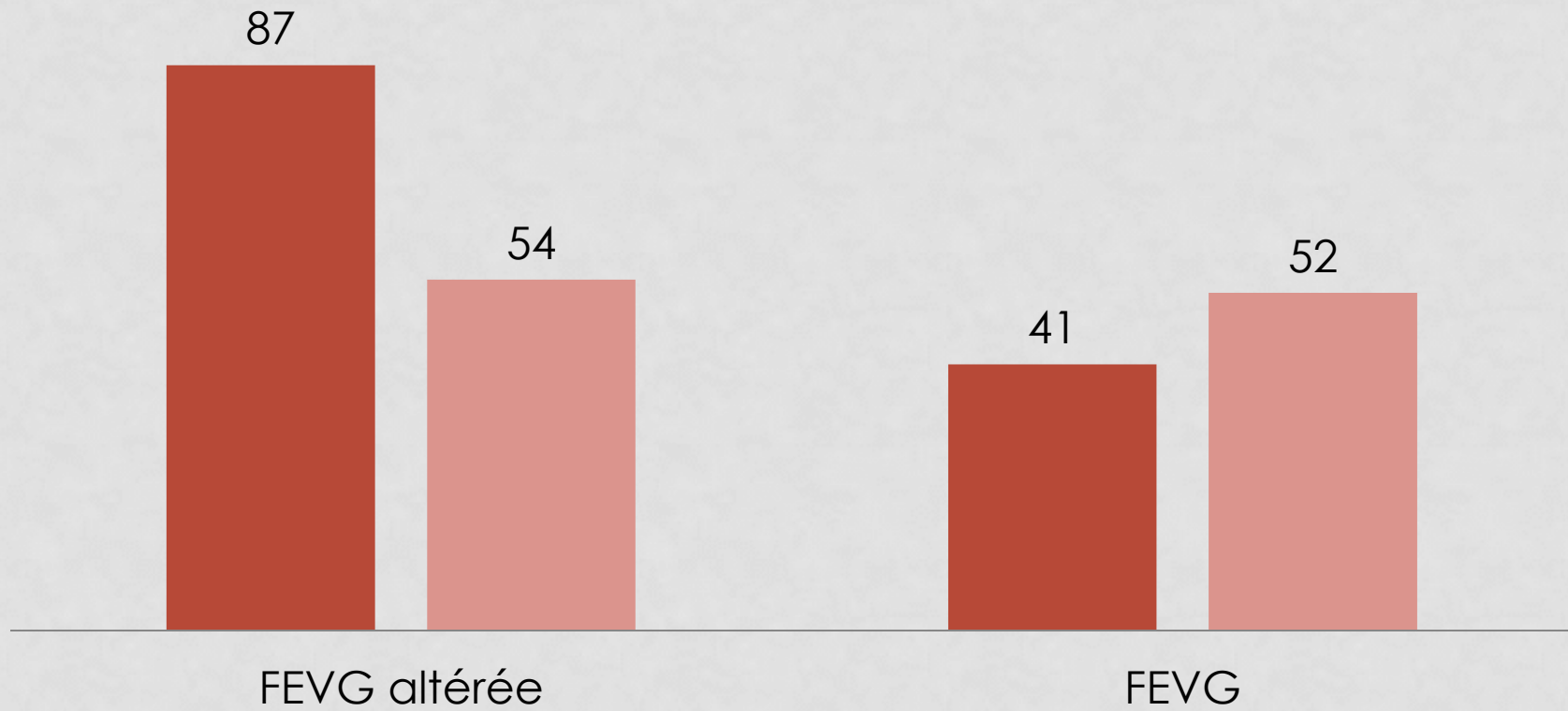
■ Takotsubo ■ SCA



*Templin and al., NEJM 2015.*

# DIAGNOSTIC MORPHOLOGIE

■ Takotsubo ■ SCA

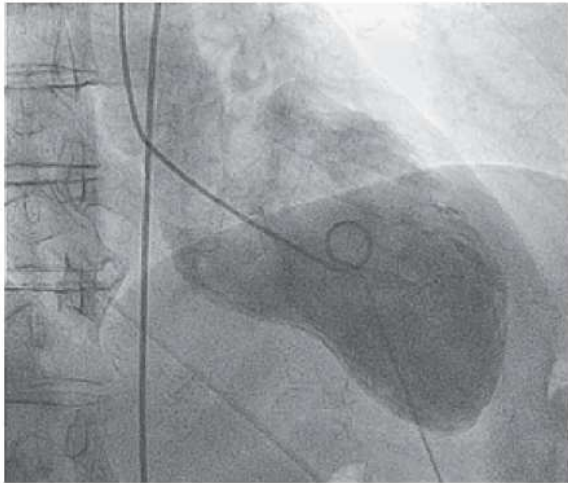


# DIAGNOSTIC MORPHOLOGIE

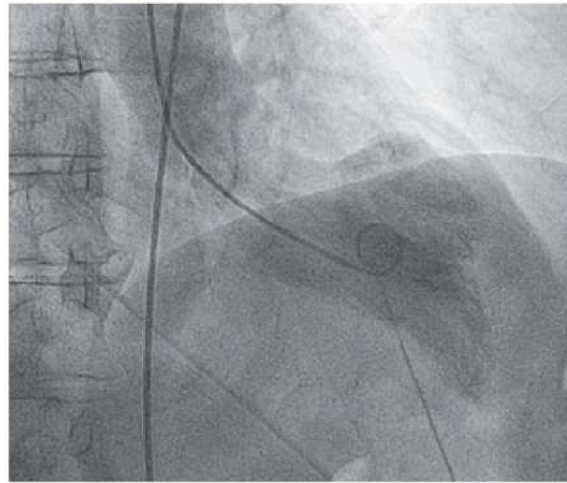


# DIAGNOSTIC MORPHOLOGIE

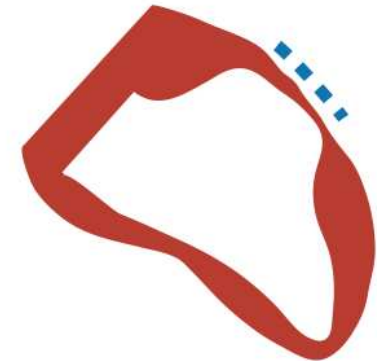
Focal Type  
A  
C  
E  
G



B  
D  
F  
H



N=26 (1.5%)





# DIAGNOSTIC MORPHOLOGIE

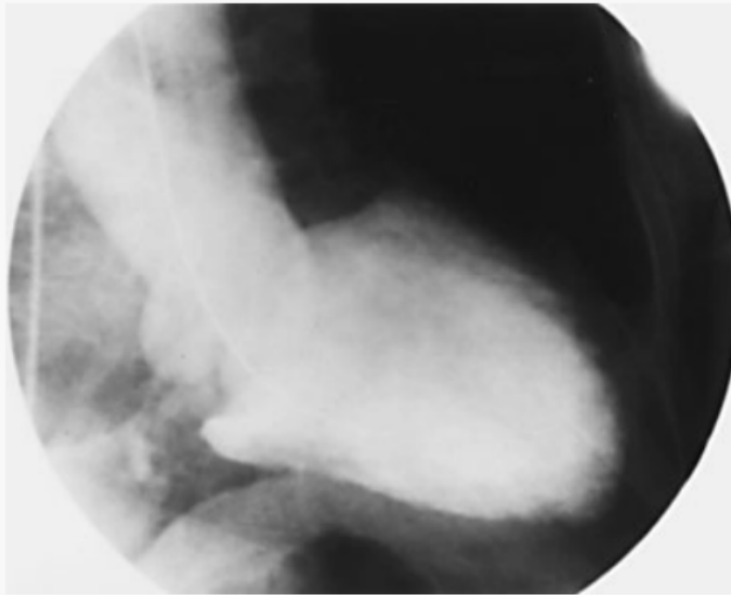


*Templin and al., NEJM 2015.*

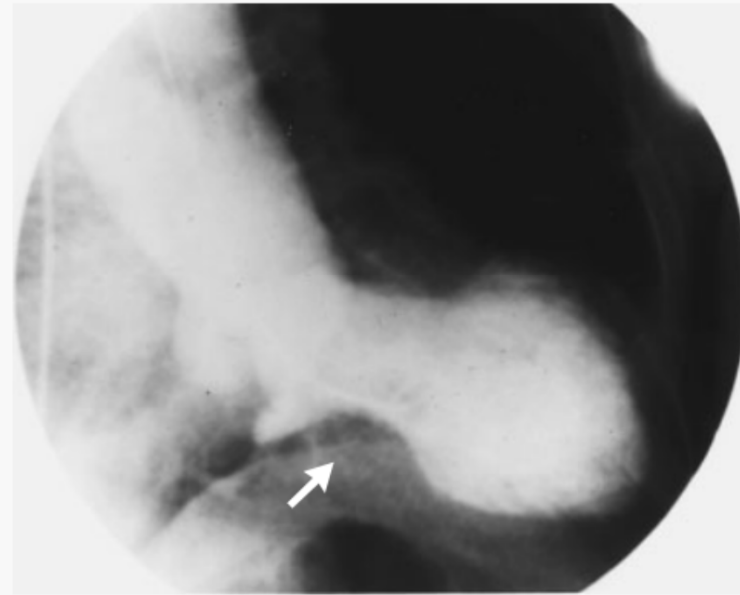
# DIAGNOSTIC CORONAROGRAPHIE

- Pas de lésion significative
- Ventriculographie

**A** Diastole



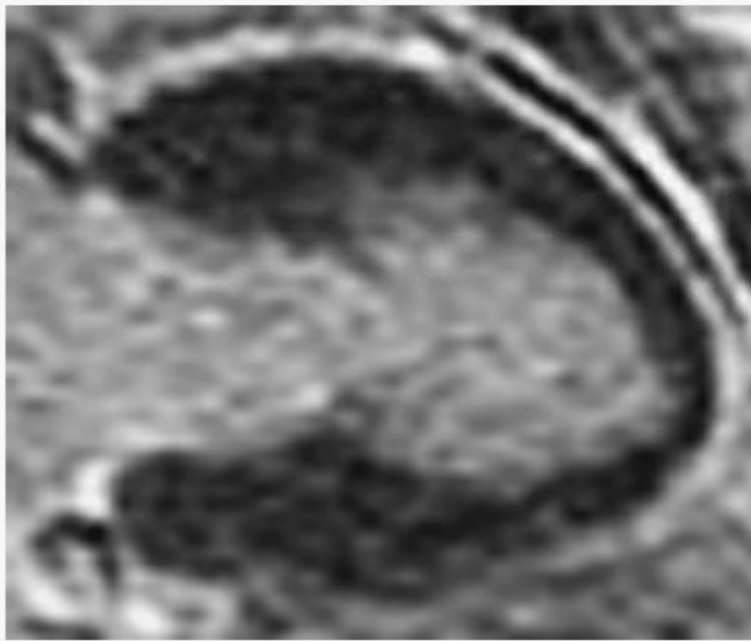
**B** Systole



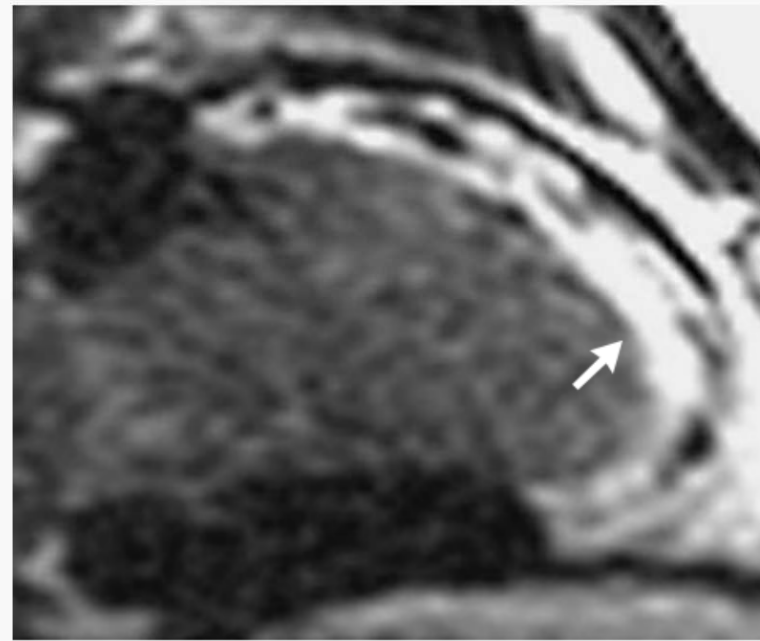
# DIAGNOSTIC IRM-GD

- Prédiction de la récupération de myocarde
- Elimination diagnostics différentiels

C



D



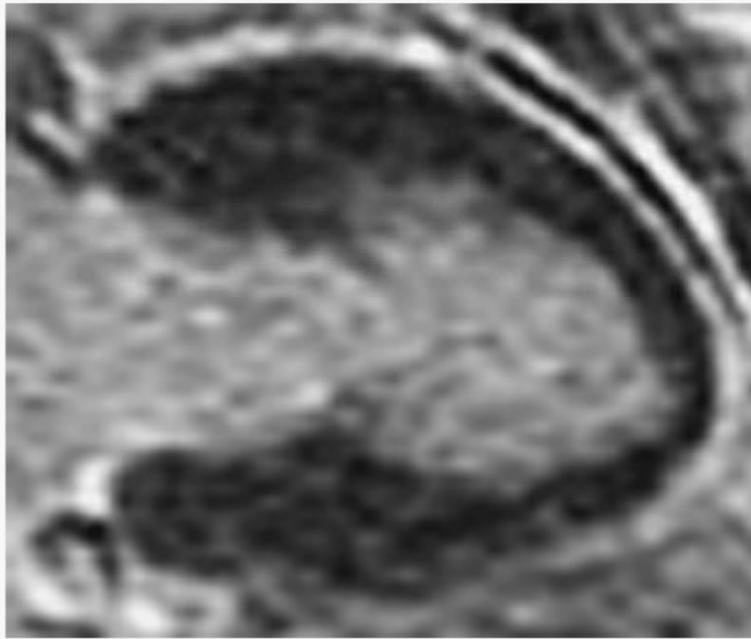
*Wittstein and al., NEJM 2005.*

*Thomson and al., J Magn Reson Imaging 2004*

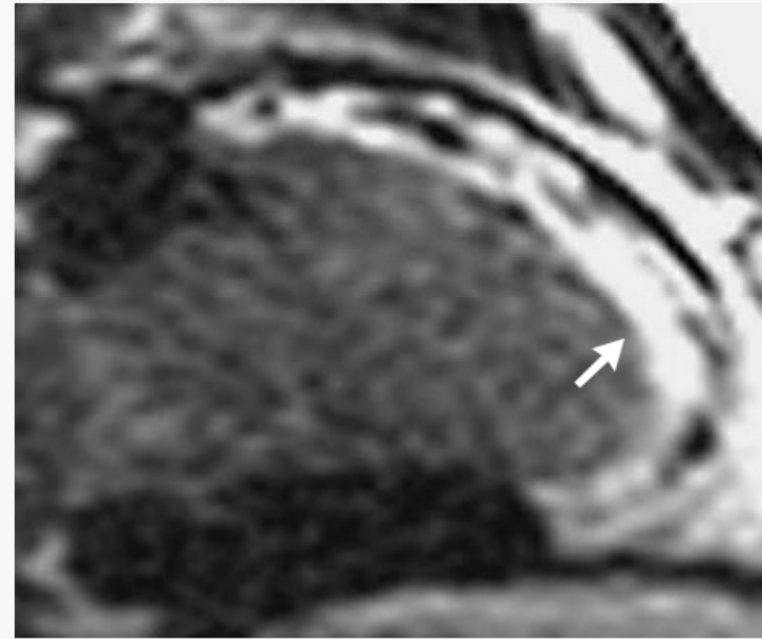
# DIAGNOSTIC IRM-GD

- Prédiction de la récupération de myocarde
- Elimination diagnostics différentiels

C



D



*Wittstein and al., NEJM 2005.*

*Thomson and al., J Magn Reson Imaging 2004*

# CRITERES DIAGNOSTIQUES

**Table II.** Proposed Mayo Clinic criteria for ABS

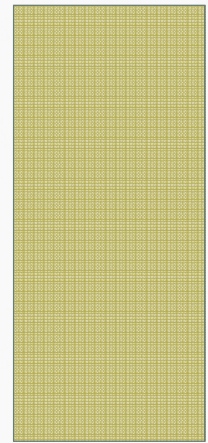
1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present. \*
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture. †
3. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin.
4. Absence of:  
Pheochromocytoma  
Myocarditis

In both of the above circumstances, the diagnosis of ABS should be made with caution, and a clear stressful precipitating trigger must be sought.

\* There are rare exceptions to these criteria such as those patients in whom the regional wall motion abnormality is limited to a single coronary territory.



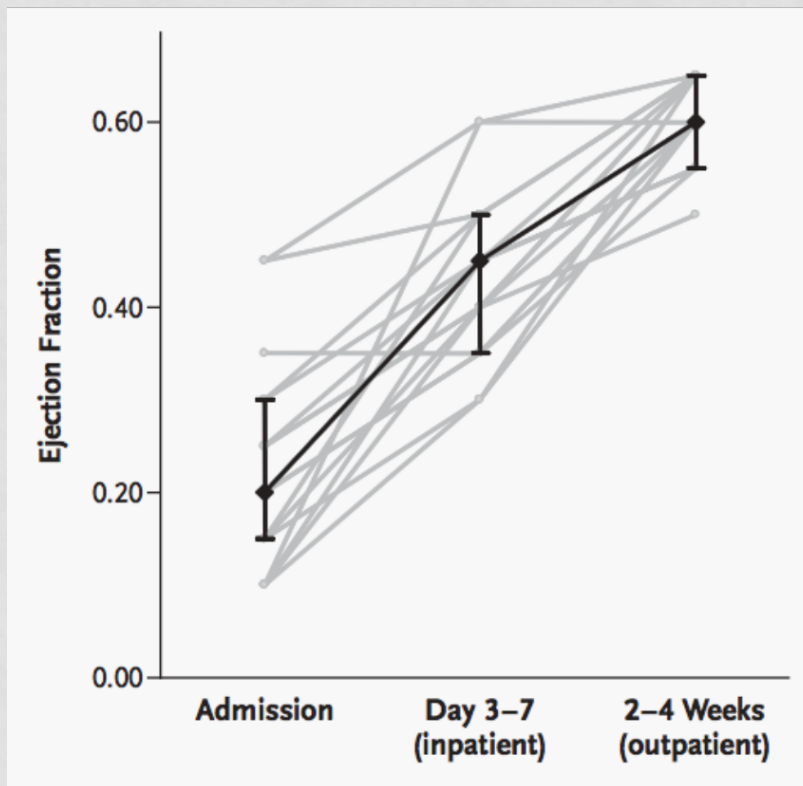
# EVOLUTION



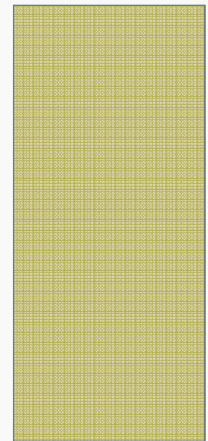


# EVOLUTION

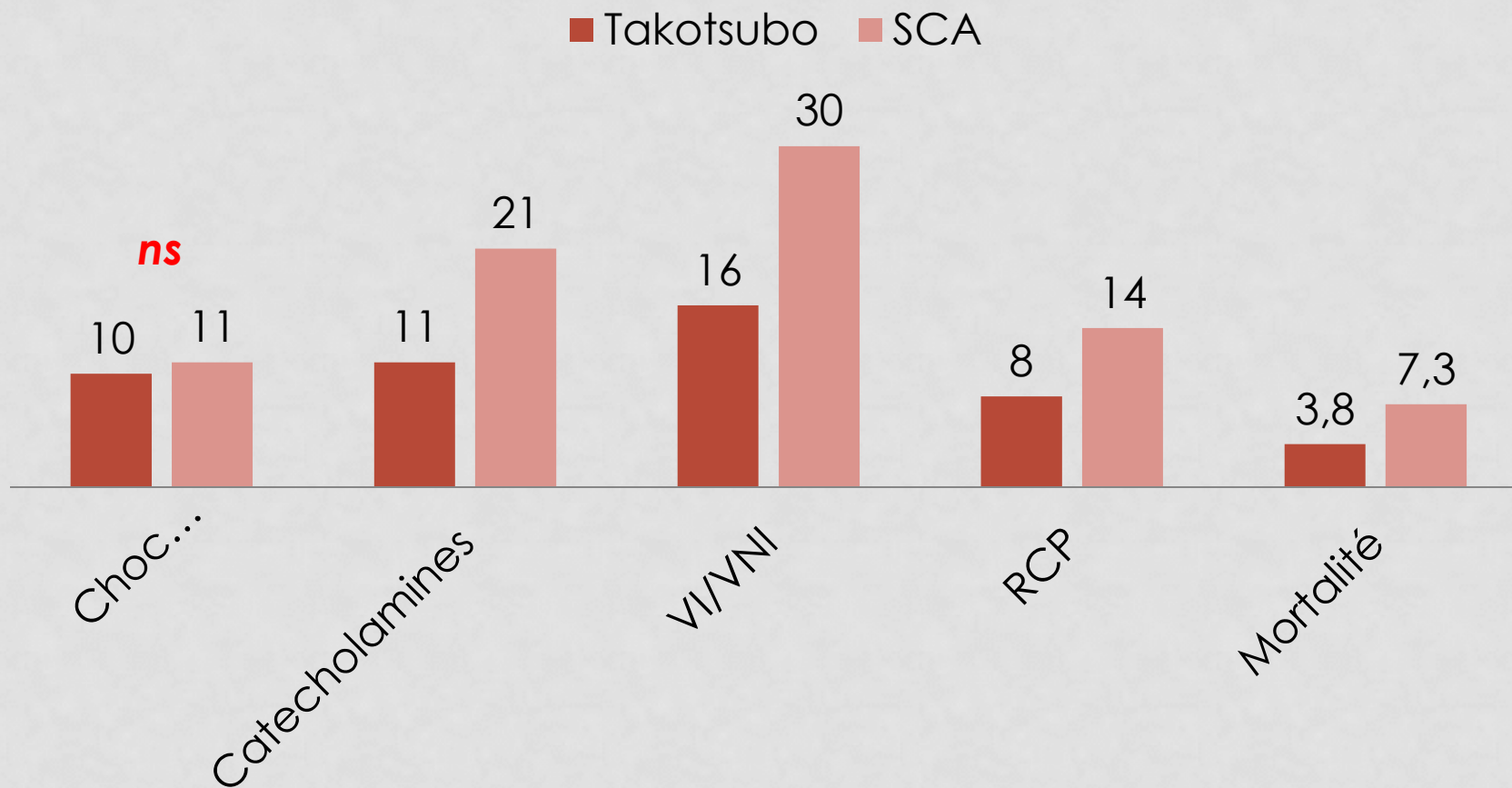
- Récupération *ad integrum* de la fonction cardiaque



# COMPLICATIONS

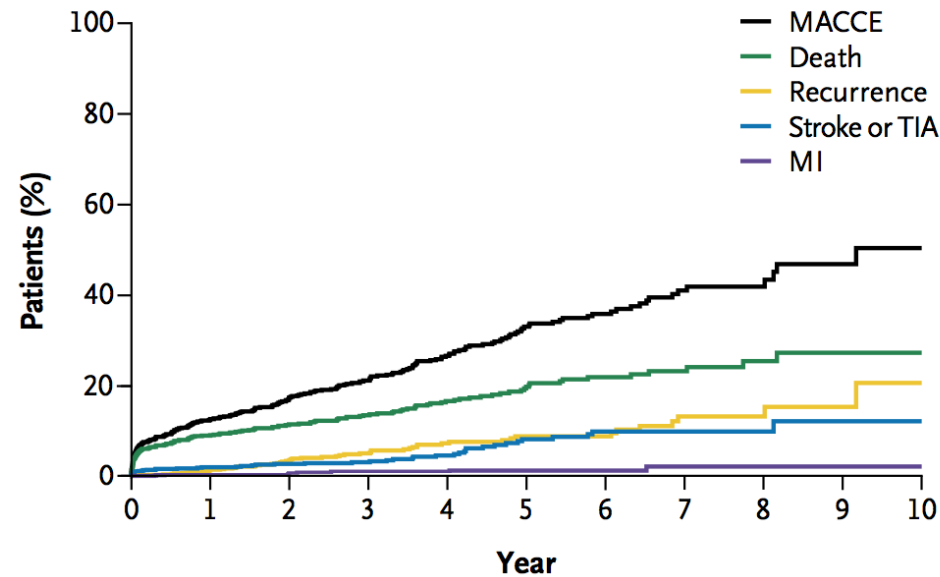


# COMPLICATIONS INTRA-HOSPITALIERES (%)



Templin and al., NEJM 2015.

# COMPLICATIONS LONG TERM

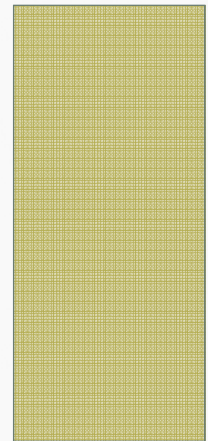


No. of Patients 1750 786 570 431 300 191 126 71 38 17 9

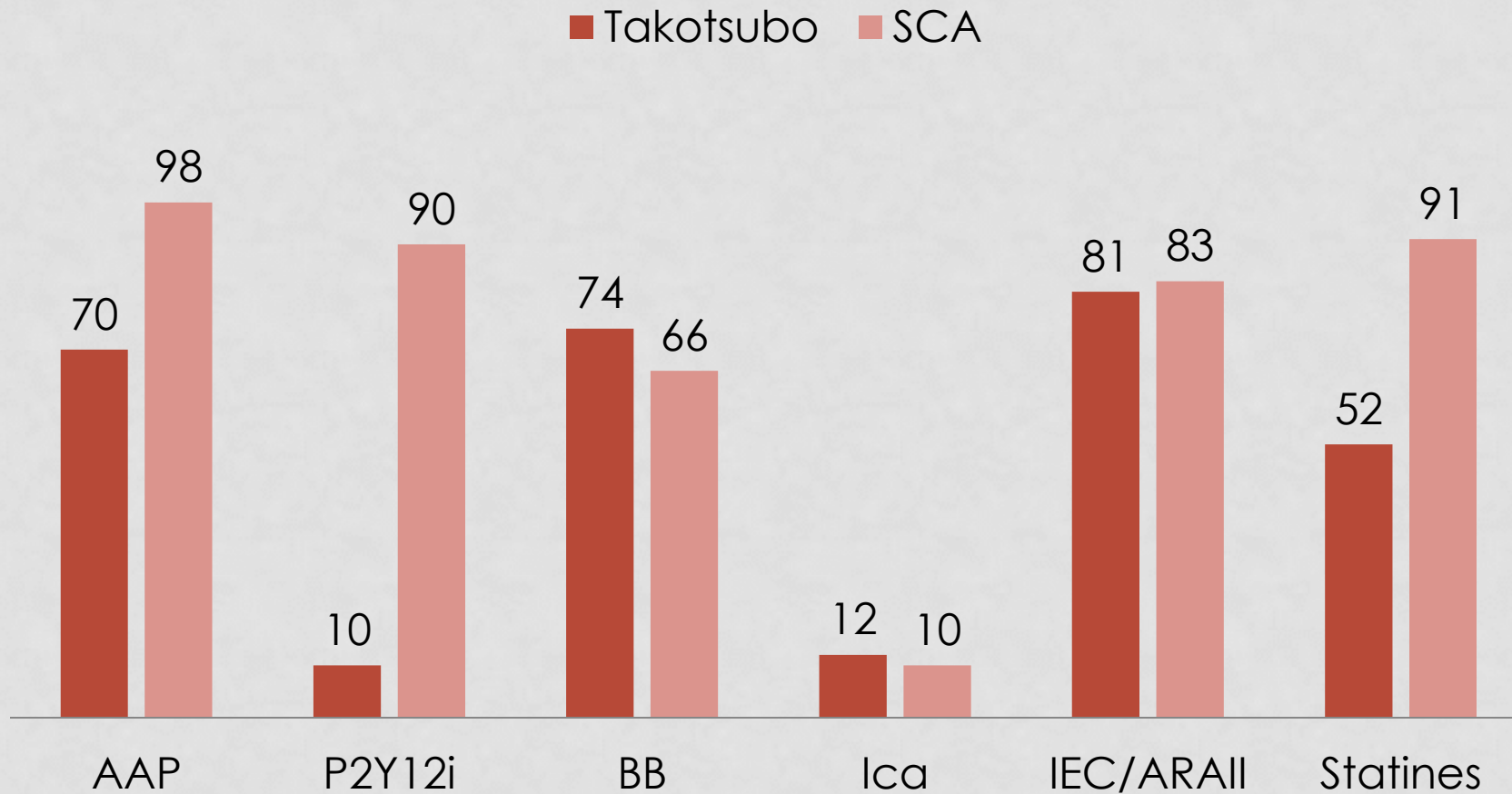
**Figure 3. Kaplan–Meier Estimates of 10-Year Outcome Events.**

Shown are the proportions of patients with any major adverse cardiac and cerebrovascular event (MACCE), which was a composite of death from any cause, recurrence of takotsubo cardiomyopathy, stroke or transient ischemic attack (TIA), or myocardial infarction (MI).

TRAITEMENT



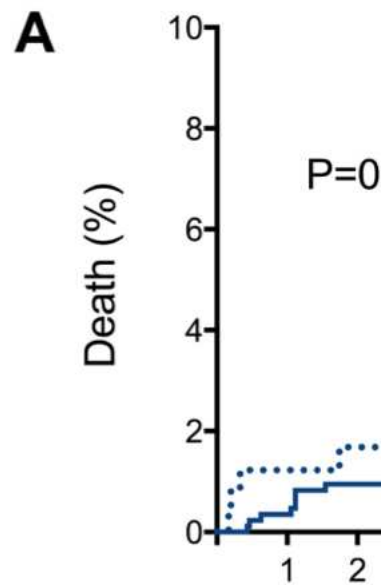
# TRAITEMENT



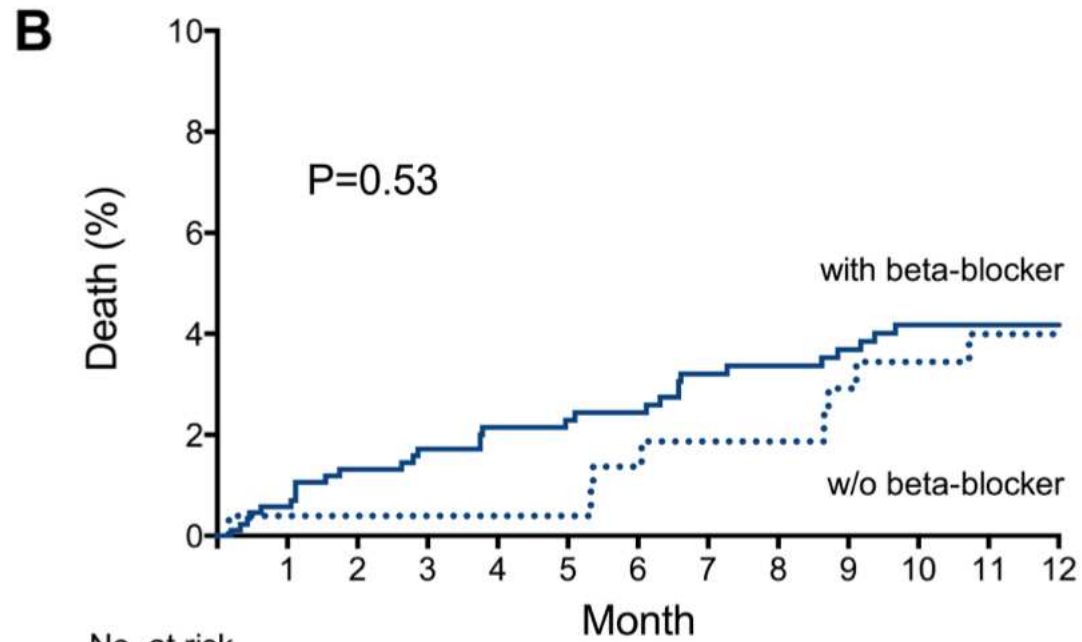
*Templin and al., NEJM 2015.*



# TRAITEMENT



No. at risk  
 — 872  
 ... 243



No. at risk

—	864	720	645	596	566
...	251	218	198	185	167

