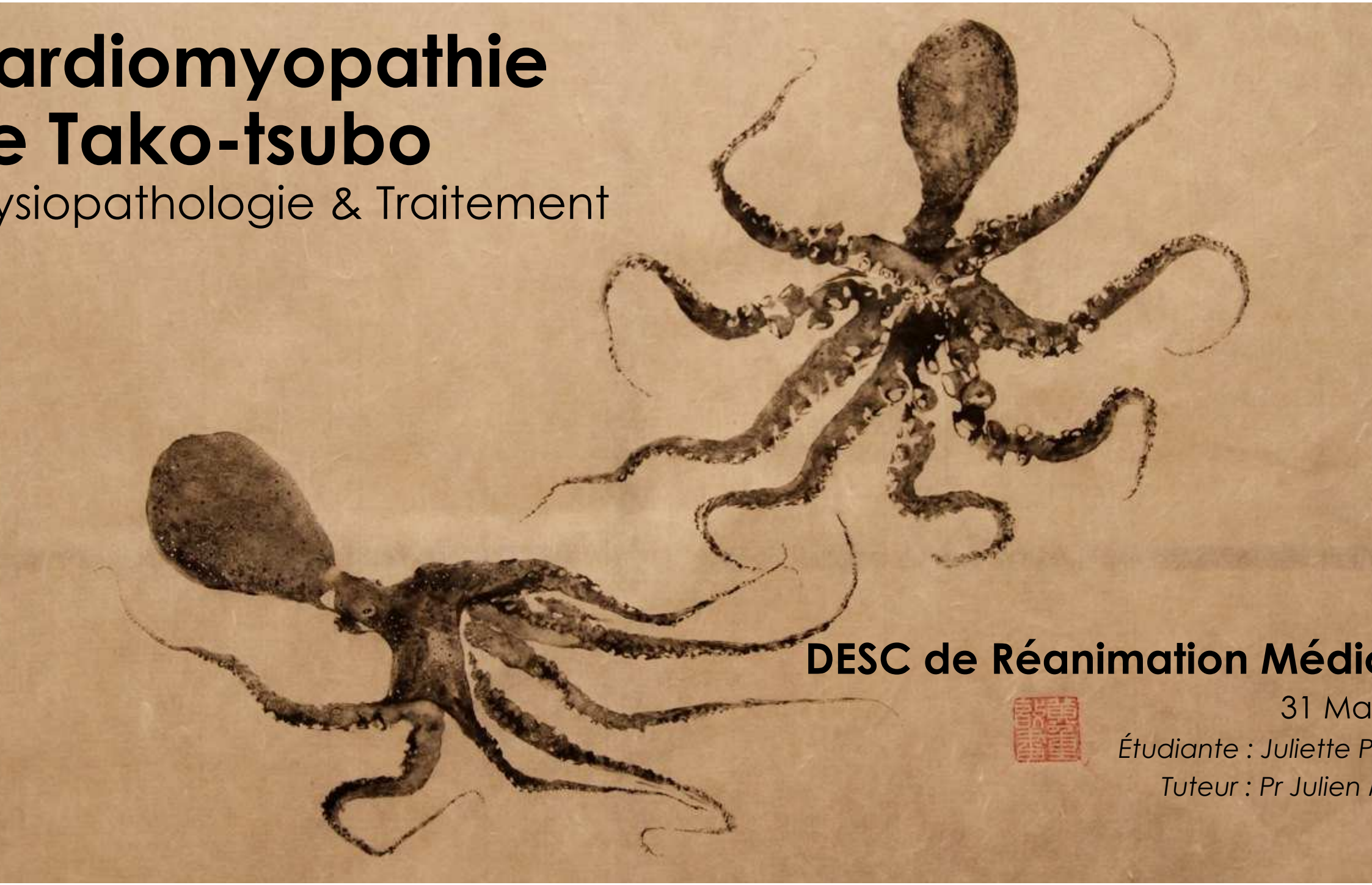
ardiomyopathie
e Tako-tsubo
ysiopathologie & Traitement



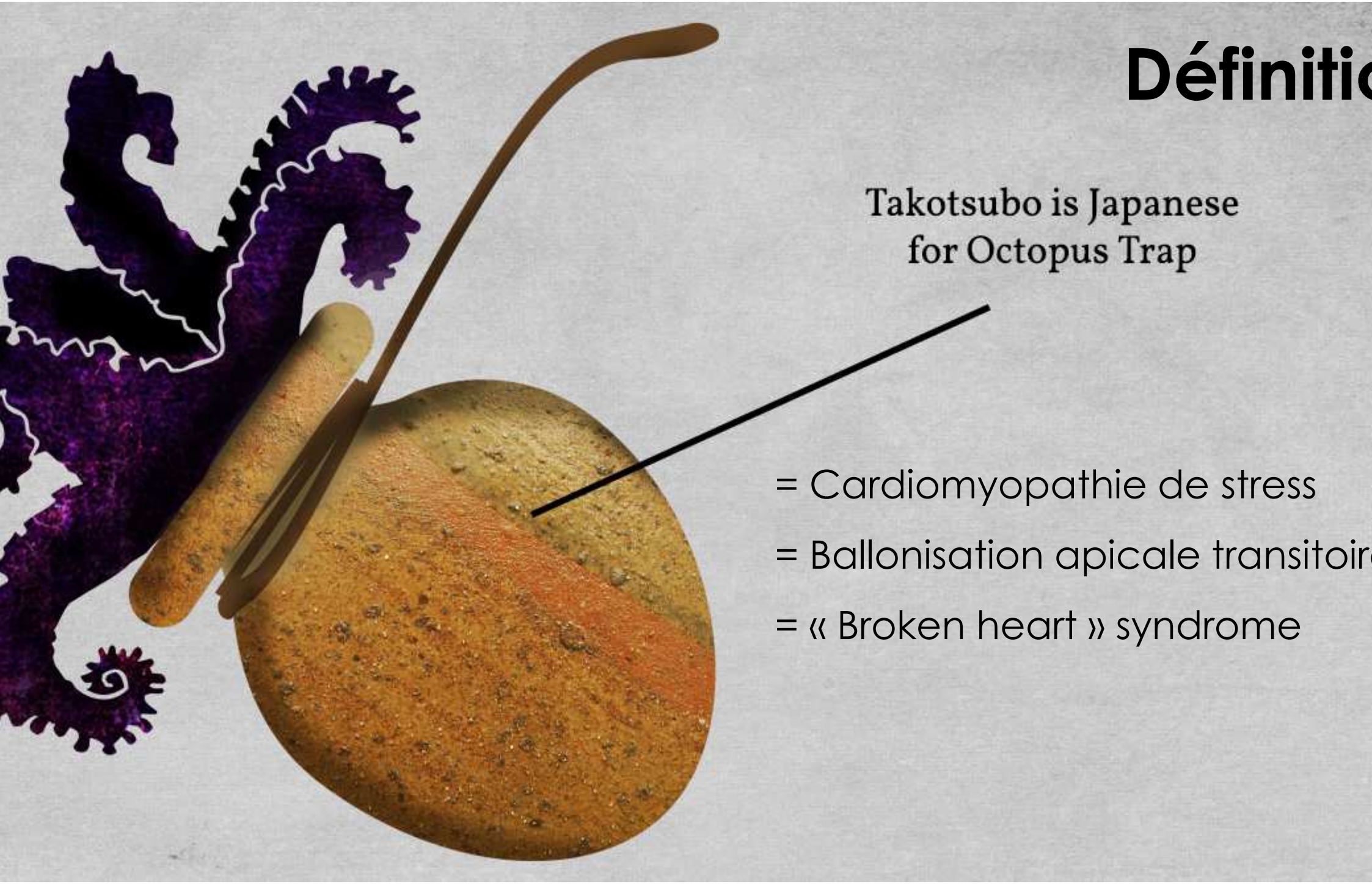
DESC de Réanimation Médi

31 Ma

Étudiante : Juliette P

Tuteur : Pr Julien

Définitio



= Cardiomyopathie de stress

= Ballonisation apicale transitoir

= « Broken heart » syndrome

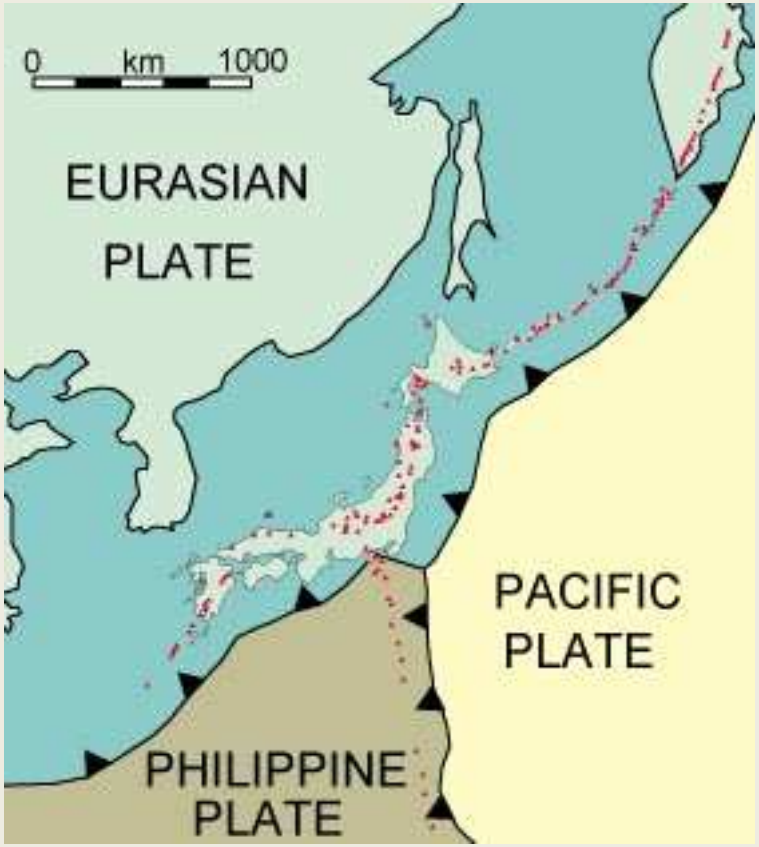
omment pèche-t-on la pieuvre ?

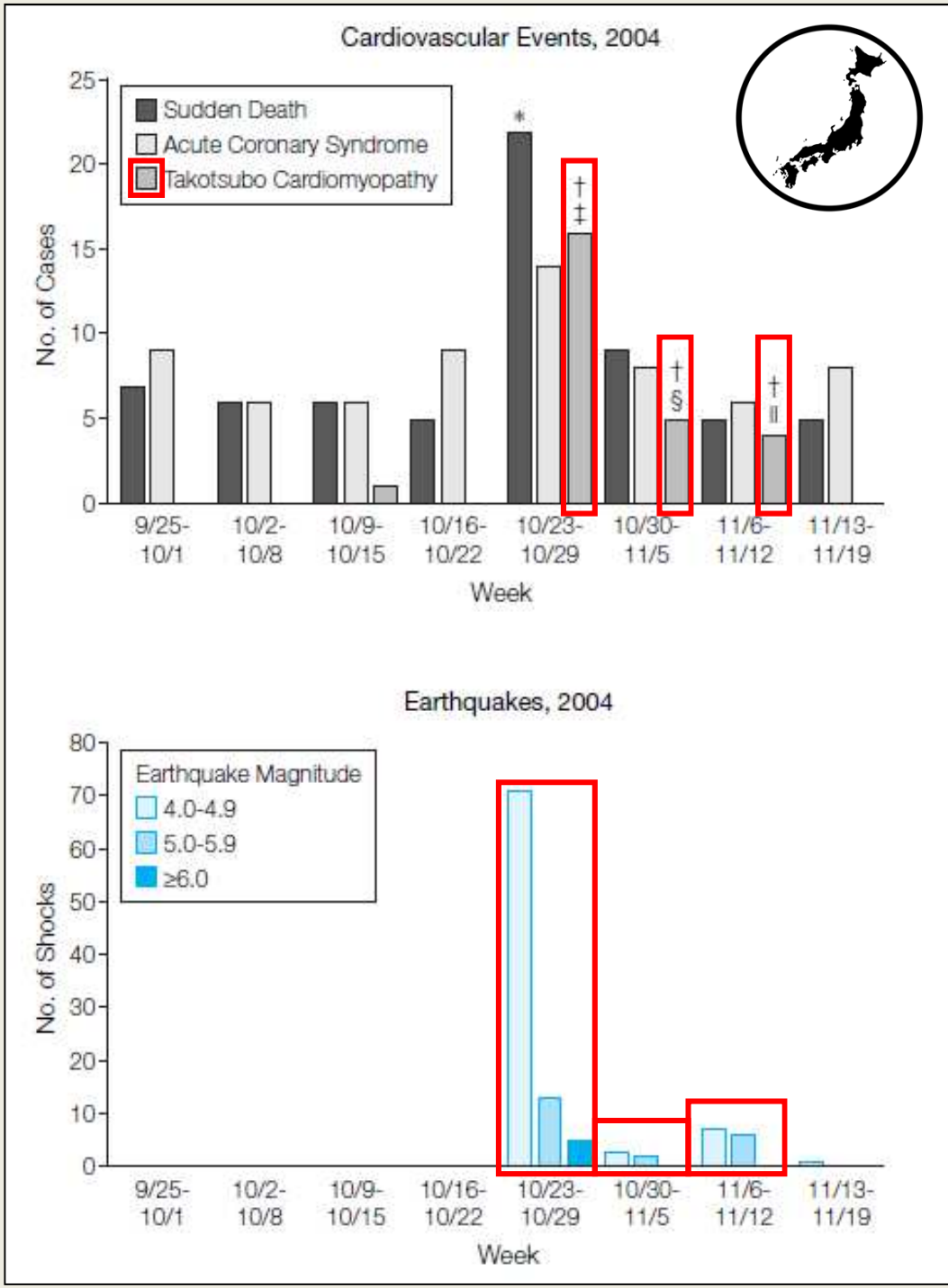
Pots en terre cuite lais
plusieurs jours dans
fond marin

Les pieuvres s'e
servent comme a

Quand les pots sont remontés à la surface, elles ne cherchent pas à s'e et se plaquent contre la paroi

Épidémiologie





nabe et al., JAMA, 2005

Épidémiologie

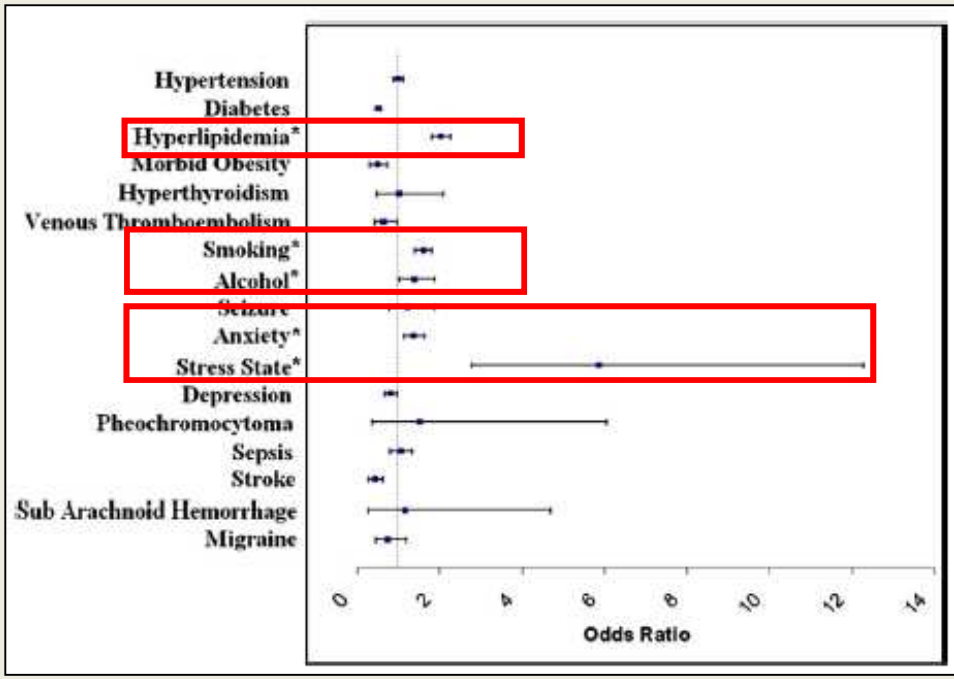
racteristics	General admissions, n (%)	TTC, n (%)
admissions	33 506 402	6837
y*		
-34	6 556 830 (19.6)	127 (1.9)
-49	5 588 174 (16.7)	581 (8.5)
-64	7 382 129 (22)	1975 (28.9)
-79	8 038 673 (24)	2952 (43.2)
80	5 963 187 (17.8)	1202 (17.6)
le	13 271 083 (39.7)	660 (9.7)
male	20 173 088 (60.3)	6178 (90.4)
*		
hite	18 957 769 (56.5)	4606 (67.4)
ck	3 445 928 (10.3)	300 (4.4)
panic	2 638 251 (7.9)	298 (4.3)
an	680 357 (2.0)	76 (1.1)
tive American	173 273 (0.5)	43 (0.6)
hers	882 716 (2.6)	103 (1.5)
own	6 750 699 (20.1)	1413 (20.7)
omes		
d	787 814 (2.4)	151 (2.2)

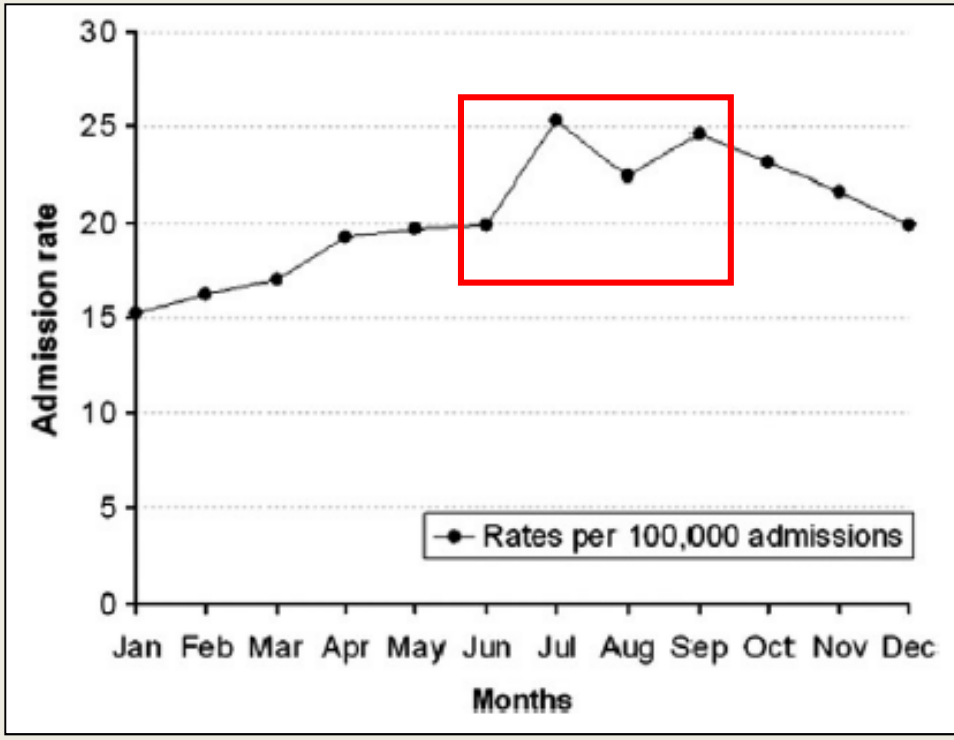
05 using χ^2 test. See text for details.

8,8

hmes + 55 ans **10,7** > Hommes + 55 ans
hmes + 55 ans **4,8** > Femmes - 55 ans

unk et al., Am Heart J, 2012

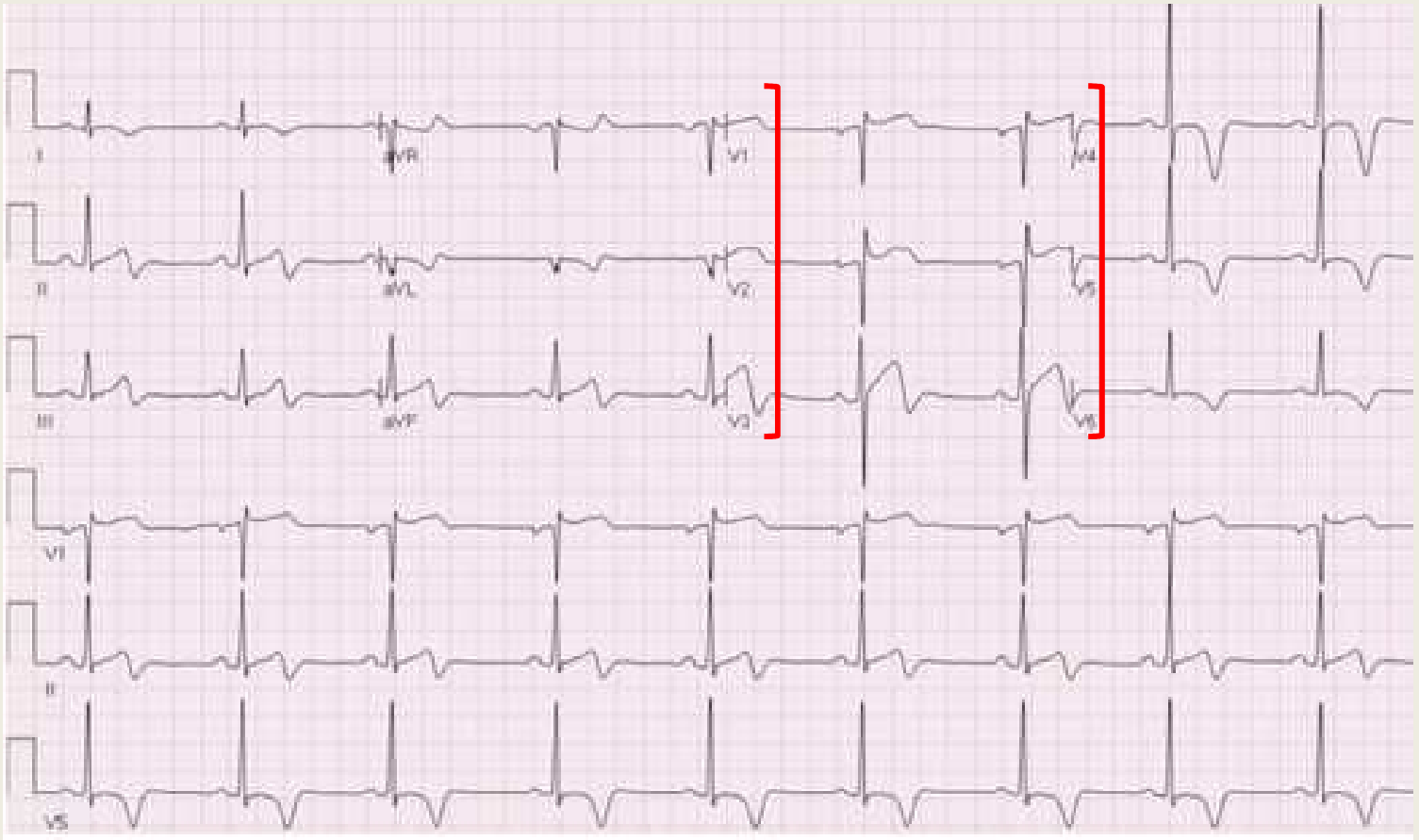




Diagnostic : principal diagnostic différentiel : SCA

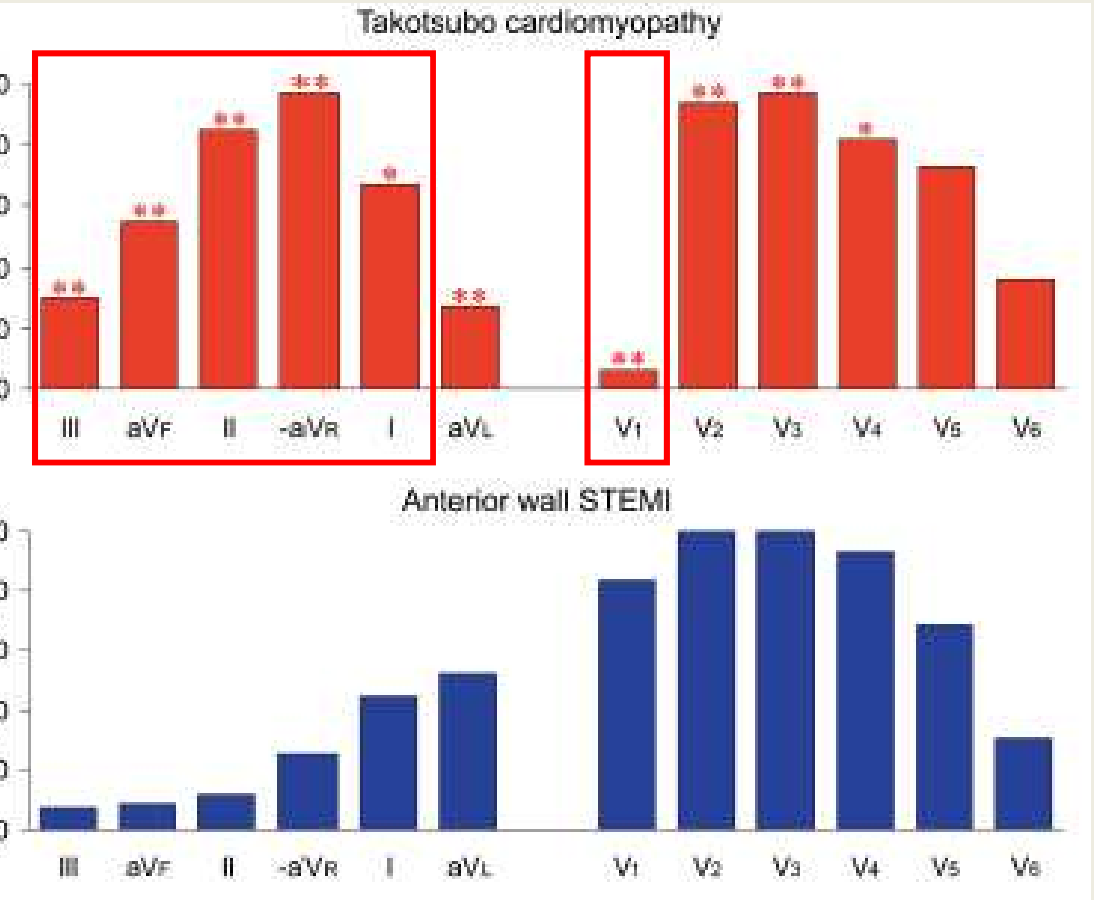
- Clinique :
  - Douleur thoracique +++ : mime un SCA

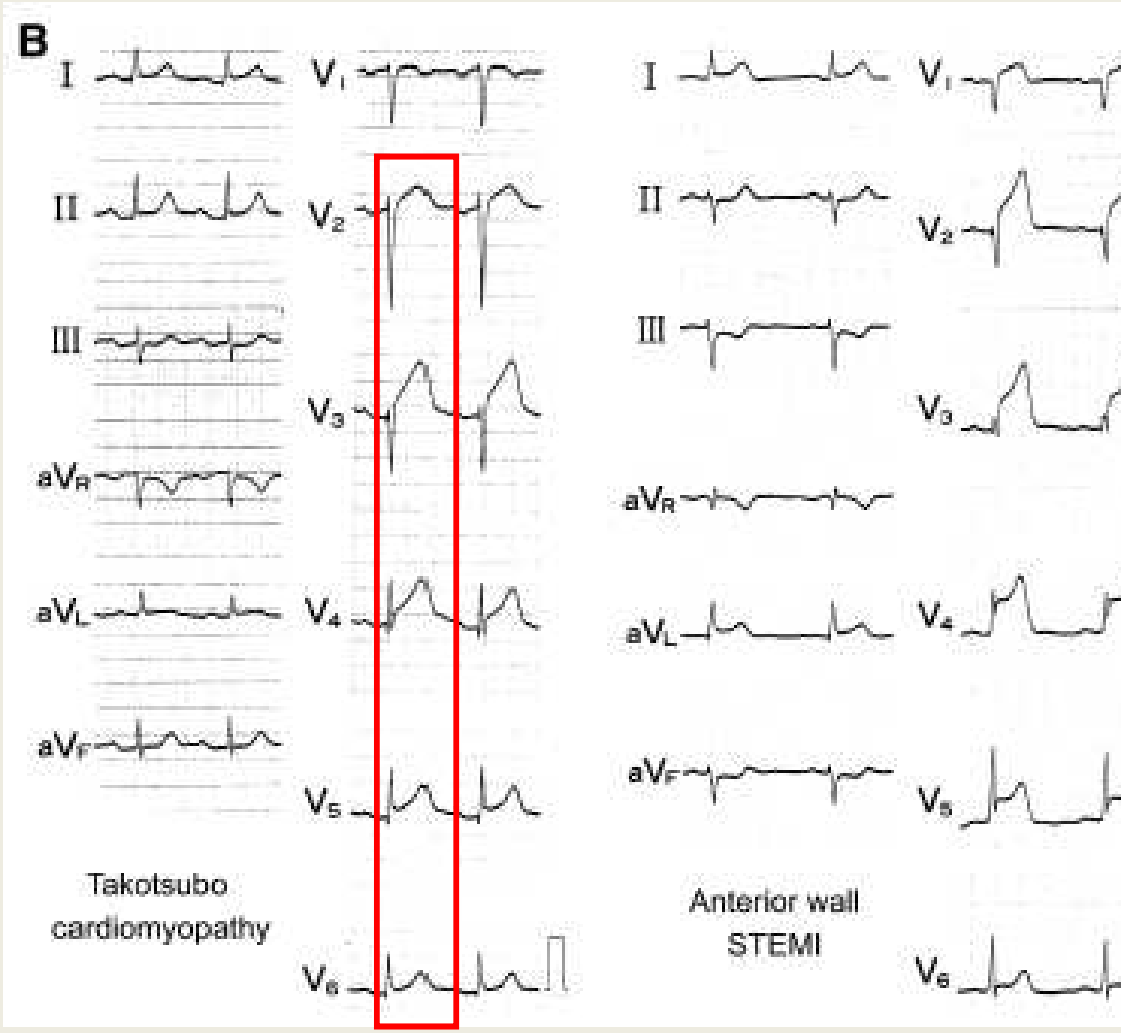
    Dyspnée, palpitations, asthénie
- ECG



Peters et al., Trends C Vasc Medicine

ECG : véritable aide au diagnostic ?





Kosuge et al., Circ Journa

Présentation clinique atypique : atteinte du VD

Table 1. Echocardiographic Data and Complications Incurred for Patients Without and With RV Involvement

	Patients With No RV Involvement	Patients With RV Involvement	p Value
n	17	8	
Age (±SD), yrs	72 (±11)	70 (±13)	0.76
Presentation LV ejection fraction (±SD)	42 (±14)	29 (±9)	0.021
Presentation TR velocity (±SD), cm/s	2.7 (±0.4)	3.2 (±0.5)	0.030
Presentation IVC diameter, cm	1.9 (±0.6)	2.1 (±0.3)	0.36
Repeat LV ejection fraction (±SD)	63 (±9)	58 (±9)	0.23
Repeat TR velocity (±SD), cm/s	2.5 (±0.6)	2.6 (±0.4)	0.80
Repeat IVC diameter, cm	1.7 (±0.6)	1.7 (±0.4)	0.92
Follow-up time for repeat echocardiography (±SD), days	61 (±55)	86 (±115)	0.60
Hospital stay (±SD), days	7 (±4)	11 (±5)	0.033
Severe CHF	2	6	0.0016
Intubation	0	5	0.0003
IABP	0	3	0.0071
CPR	0	1	0.14
Severe CHF, IABP, or CPR	2	7	0.0002
LV thrombus	1	0	0.48
LV outflow tract obstruction	2	0	0.31
Follow-up after event (months)	19.3 ± 13.9	7.0 ± 6.3	0.026
Recurrence	0	1	0.14
Death	1	2	NS

CHF = congestive heart failure; CPR = cardiopulmonary resuscitation; IABP = intra-aortic balloon pump; IVC = inferior vena cava; LV = left ventricular; NS = not significant; RV = right ventricular; TR = tricuspid valve regurgitation.

1/3 cas : atteinte du VD

FEVG **diminuée**

Plus de complication **isolée / associées**

Durée d'hospitalisation allongée

Si pas d'atteinte initiale

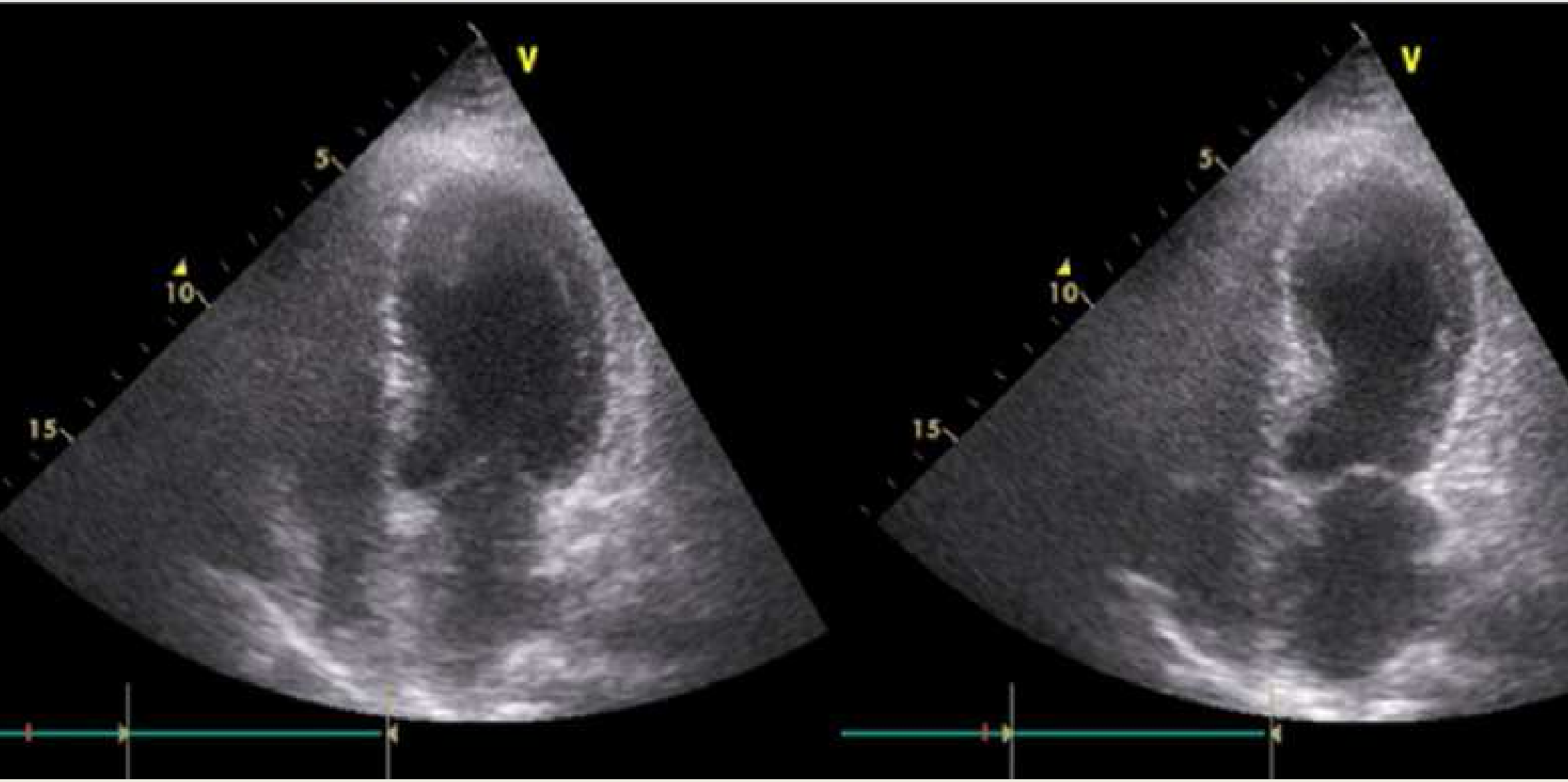
=> pas d'atteinte au cours de l'évolution

Elesber, J Am Coll Cardiology

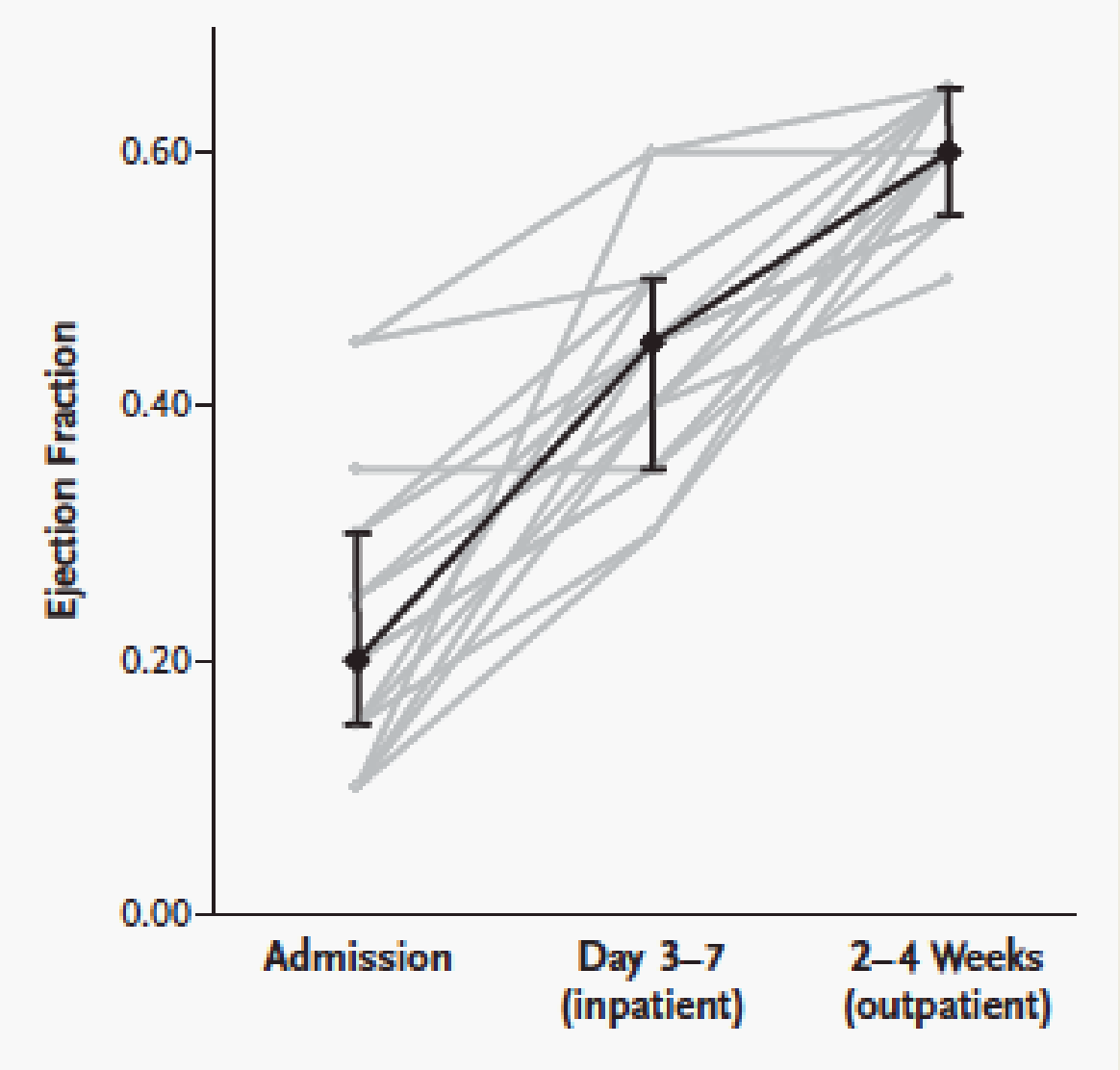
ETT

End diastole

End systole



Normalisation de la FEVG dans le temps

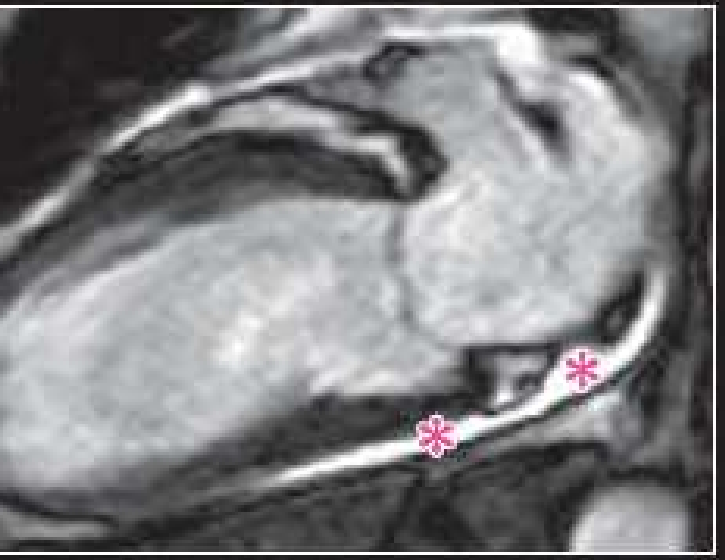


Wittstein et al., NEJM

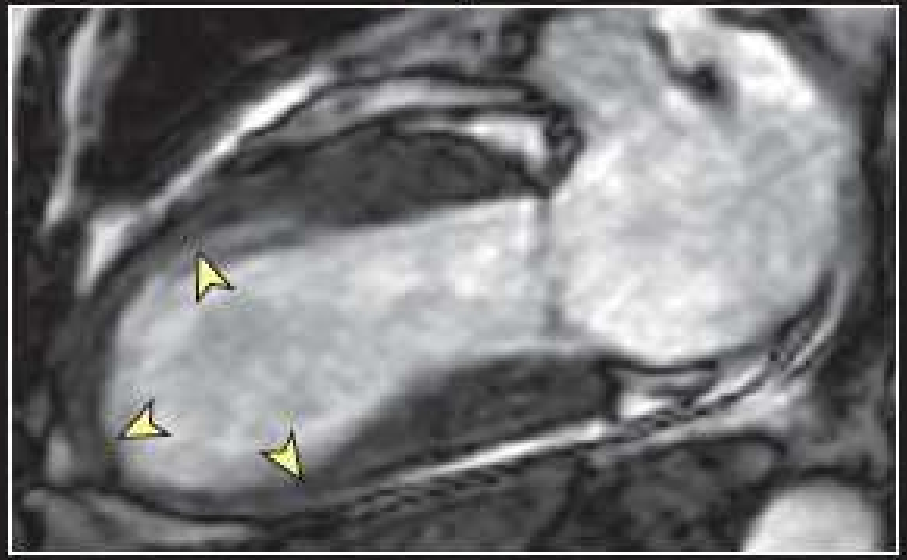
IRM cardiaqu

al ballooning

End diastole

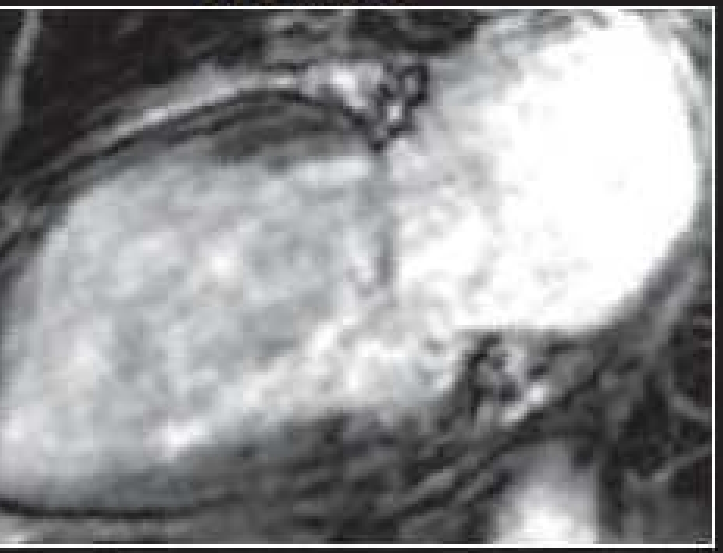


End systole

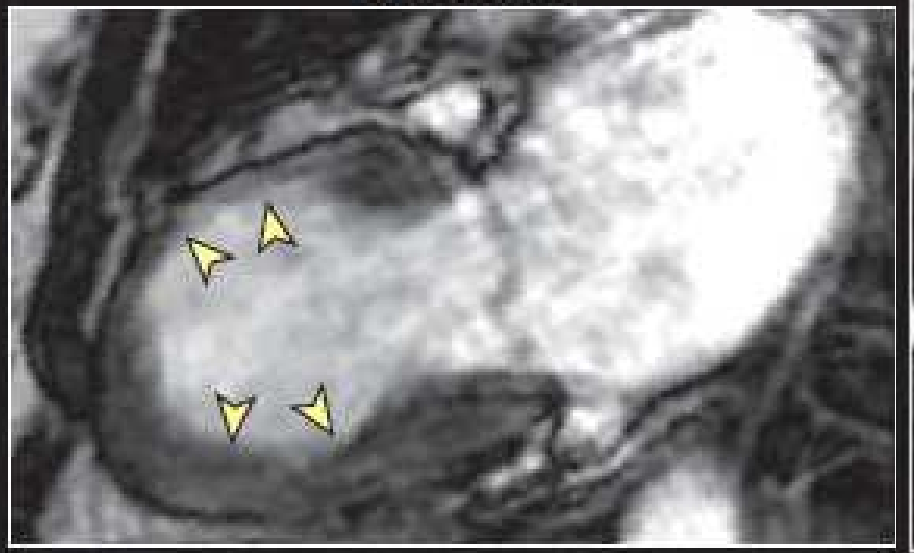


ventricular ballooning with sparing of apical and basal region

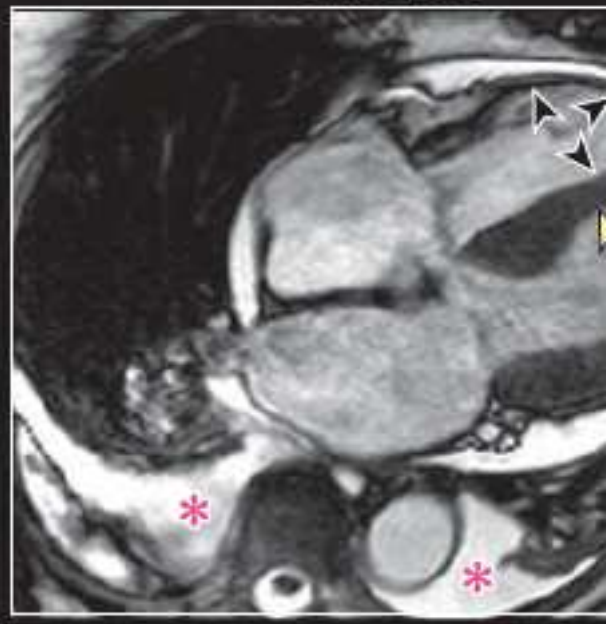
End diastole

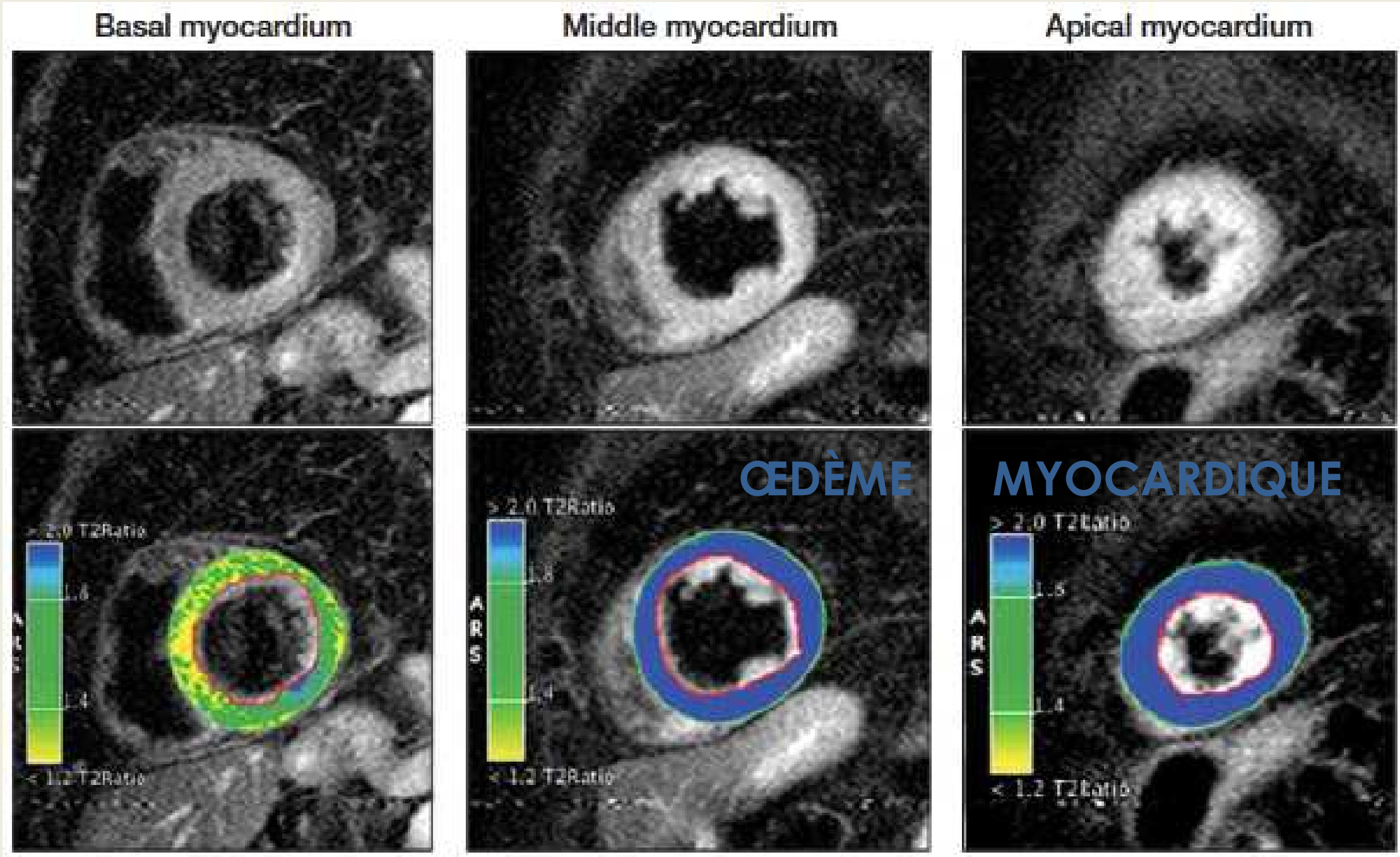


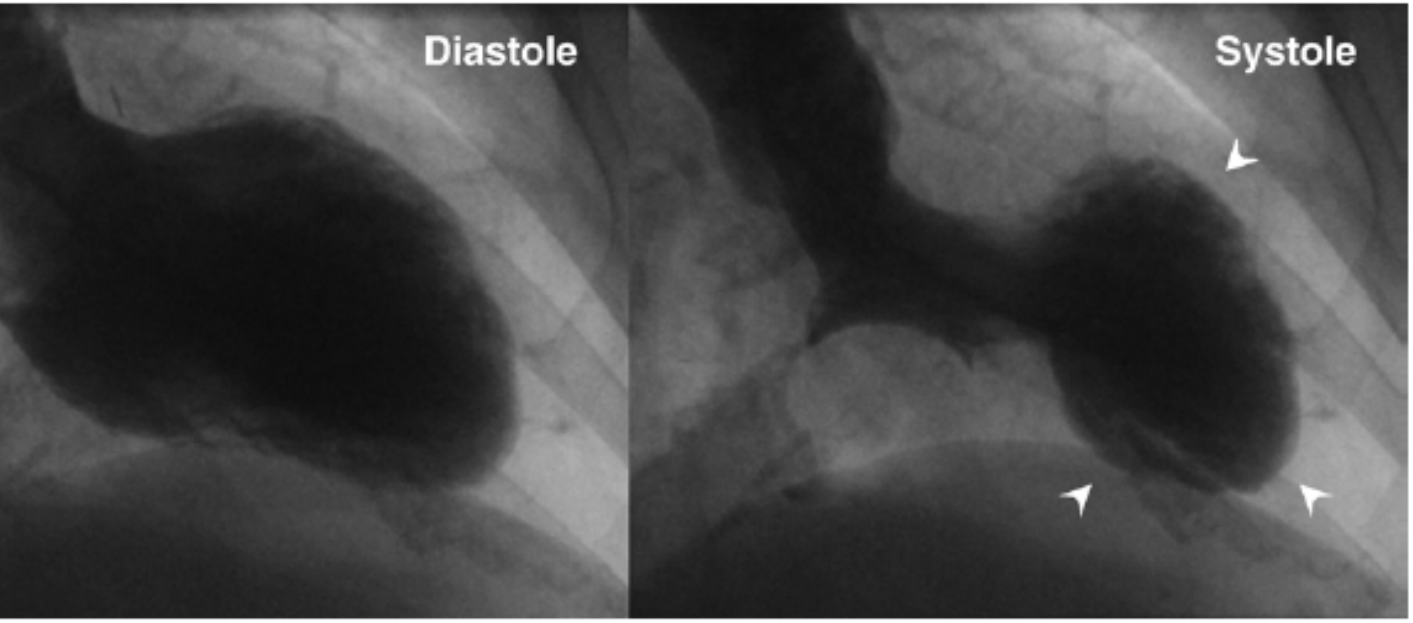
End systole

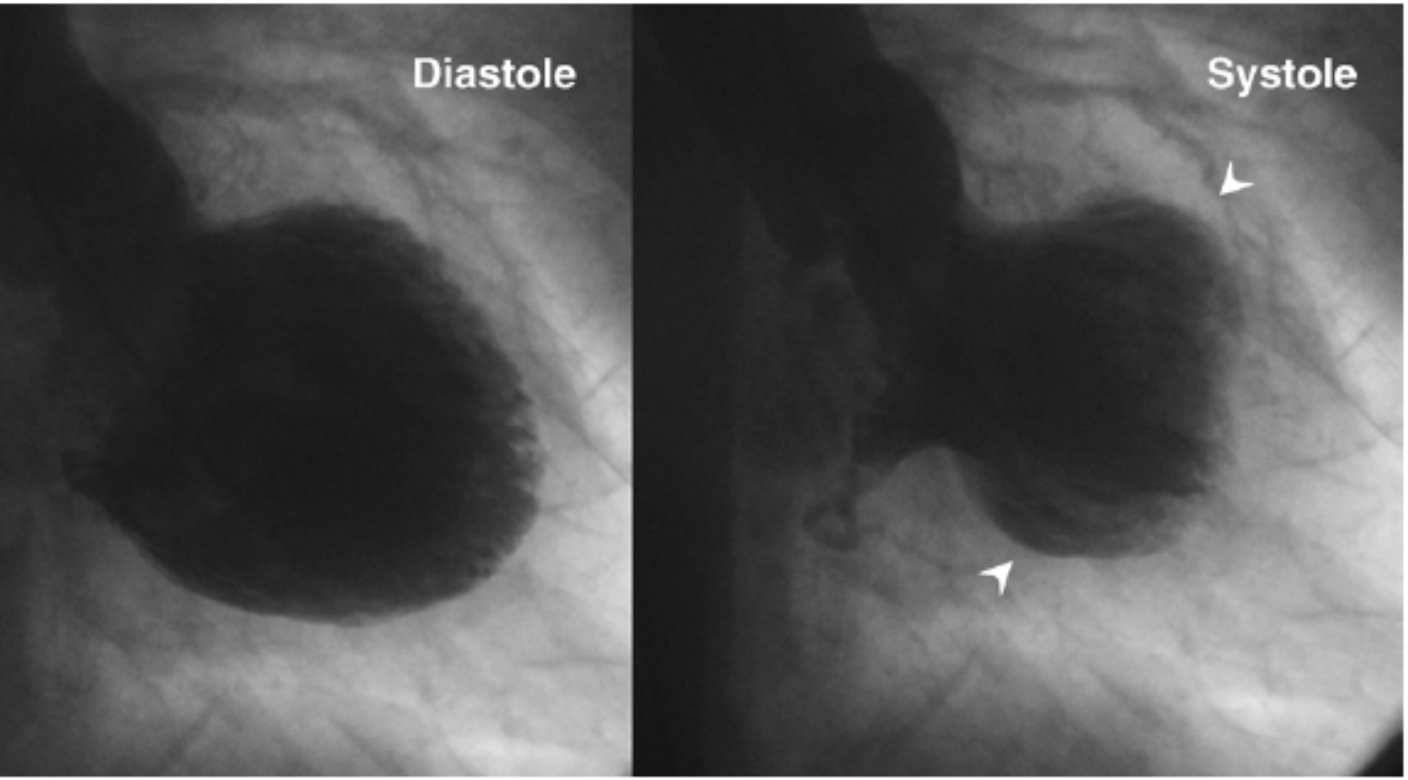


End systole




Basal myocardium
Middle myocardium
Apical myocardium
ŒDÈME
MYOCARDIQUE
> 2.0 T2Ratio
1.8
1.4
< 1.2 T2Ratio
ARS





KT cardiaque

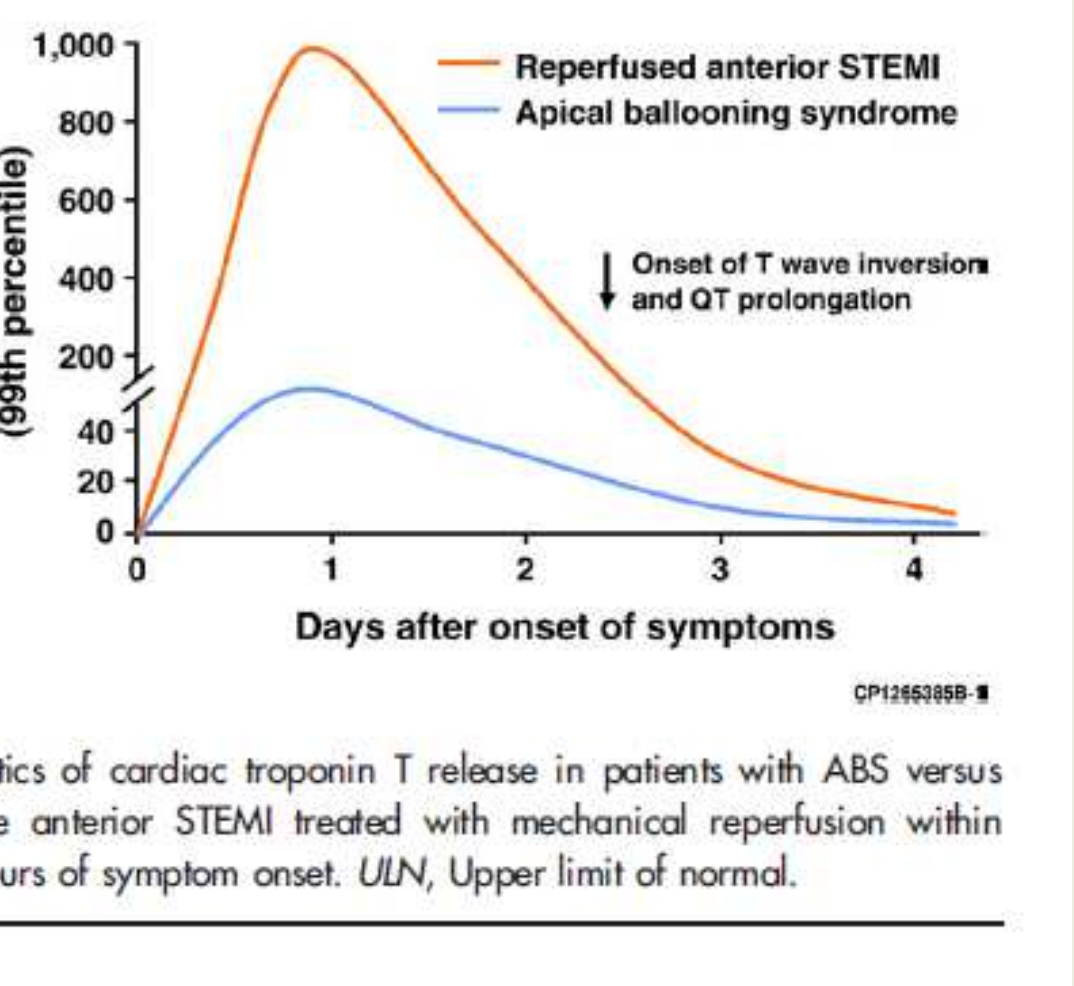
Contraction basale
hyperdynamique
Akinésie segment moyen et apical

Contraction basale et apicale
hyperdynamique
Akinésie segment moyen

Prasad et al., Am Heart

Biomarqueurs cardiaques

Troponine T



tics of cardiac troponin T release in patients with ABS versus e anterior STEMI treated with mechanical reperfusion within urs of symptom onset. *ULN*, Upper limit of normal.

d et al., Am Heart J, 2008

BNP/Troponine T : aide pour **SCA ST**

Table 2 Characteristics of apical ballooning syndrome and ST-elevation myocardial infarction controls

Variables	STEMI (*N*=50)	ABS (*N*=50)	
Age (years)	69.0±12.4	68.7±11.9	
Men	3 (6%)	3 (6%)	
Hypertension	31 (62%)	35 (70%)	
Diabetes mellitus	7 (14%)	5 (10%)	
Smoking	23 (47%)	16 (33%)	
Hypercholesterolemia	27 (63%)	22 (44%)	
Ejection fraction by echocardiogram (%)	52.0 (40.0, 63.5)	37.0 (30.0, 52.0)	<
BNP (pg/ml)	168.5 (63.0, 515.0)	554.5 (233.0, 937.0)	
Admission troponin T (ng/ml)	0.2 (0.1, 0.9)	0.2 (0.1, 0.6)	
Peak troponin T (ng/ml)	2.8 (1.0, 6.5)	0.5 (0.2, 0.8)	<
Peak CK-MB (ng/ml)	94.9 (18.4, 182.6)	12.3 (7.9, 25.3)	<
BNP/admission troponin T ratio	649.3 (146.5, 5405.4)	2069.1 (582.6, 7000.0)	
BNP/peak troponin T ratio	97.4 (17.9, 264.7)	1089.4 (446.7, 3334.8)	

Ahmed, Coronary Artery Disease

Biomarqueurs cardiaques

BNP/Troponine T : aide pour **SCA ST-** ?

3 Characteristics of apical ballooning syndrome and non-ST-elevation myocardial infarction controls

oles	NSTEMI (*N* = 25)	ABS (*N* = 25)	*P*
(years)	69.0 ± 11.4	69.3 ± 11.2	
	3 (12%)	3 (12%)	
rtension	18 (78%)	17 (68%)	
etes mellitus	3 (12%)	2 (8%)	
king	11 (48%)	11 (44%)	
rcholesterolemia	14 (64%)	11 (44%)	
ion fraction by echocardiogram (%)	55.0 (50.0, 60.0)	32.0 (28.0, 50.0)	<
(pg/ml)	198.0 (105.0, 283.0)	561.0 (140.0, 919.0)	
ssion troponin T (ng/ml)	0.3 (0.1, 0.6)	0.2 (0.1, 0.6)	
troponin T (ng/ml)	0.6 (0.3, 1.6)	0.5 (0.3, 0.6)	
CK-MB (ng/ml)	24.7 (10.1, 46.0)	11.9 (8.1, 25.5)	
admission troponin T ratio	1023.3 (243.8, 2358.3)	2114.3 (620.2, 7000.0)	
peak troponin T ratio	243.8 (123.5, 600.0)	1059.1 (500.0, 1938.2)	

Ahmed, Coronary Artery Disease

Catécholamines endogènes

e		Patients with Stress Cardiomyopathy (N=13)			Patients with Killip Class III Myocardial Infarction (		
		Day 1 or 2	Day 3, 4, or 5	Day 7, 8, or 9	Day 1 or 2	Day 3, 4, or 5	Day 7, 8
		median (interquartile range)					
olamine precursor (pg/ml)							
oxyphenylalanine		2859 (2721–2997)†	2495 (2386–2761)†	1656 (1065–2011)	1282 (1124–1656)	1203 (1193–1873)	907 (749-
olamines (pg/ml)							
hrine	37	1264 (916–1374)†	1044 (733–1118)†	348 (180–550)	376 (275–476)	330 (220–385)	275 (220-
nephrine	169	2284 (1709–2910)†	1573 (1235–2589)†	1142 (525–1252)	1100 (914–1320)	829 (727–914)	541 (516-
ine	15	111 (106–146)†	77 (63–110)	56 (47–77)	61 (46–77)	61 (61–77)	38 (30–(
euronal metabolites (pg/ml)							
ephrine		178 (140–187)	509 (385–789)	659 (590–738)§	106 (89–124)	203 (177–213)	205 (189-
tanephrine		216 (130–319)	456 (229–569)	661 (551–696)§	160 (145–170)	196 (181–209)	271 (225-
es (pg/ml)							
peptide Y		186 (162–236)§	185 (158–214)†	136 (90–182)§	77 (60–90)	69 (61–71)	60 (40–(
atriuretic peptide		1033 (805–1783)§	450 (205–684)	142 (72–236)	264 (192–483)	268 (249–574)	297 (142-

Wittstein et al., NEJM

Facteurs déclenchants

Stress physique
Stress psychologique

4)
on (n = 10)
ment with family members, friend, or landlord over rent
egarding work environment or personal property
th husband (afflicted by dementia)
elated to organizing a community event
ncial or employment problems (n = 7)
s including passing bad checks
iness failure with loss of life savings
of large business debt
meeting with management at work
inning to a new job
18)
f recent sudden death in a close relative or friend
rsary of son's death
t in husband (patient performed cardiopulmonary resuscitation)
post-operative cardiac arrest and anoxic encephalopathy
ths of family members in close proximity
n's death in military
after retiring from life-long occupation
eath of husband from cancer
ief at memorial service for public figure (U.S. senator)
et dogs
death of son-in-law during church meeting
onflict (n = 14)
ce culminating in sale of her home of 28 years
om husband
ssion with suicidal ideation
ep up with daughter during bicycle race
mily estrangement during psychological counseling
other's methamphetamine addiction and alcoholism
permanent residence
estic abuse by spouse
t from daughter
g emotion during 50th wedding anniversary
t best friend was moving a great distance
one call from friend
by relative
iety (n = 15)
ll outdoors in winter with hip fracture (feared freezing to death)
driving a remote road in Minnesota (without cell phone)
tion regarding motor vehicle accident
iving in unsafe neighborhood at night
d by new computer software at work
tire while driving mother to physician
burned down (fearing loss of chickens)
ding a public speaking event
s of husband
od during intense thunderstorm
t elevated blood pressure/paroxysmal atrial fibrillation
t elective cardioversion for atrial fibrillation
rying to load belongings into car during bitter cold
while alone with fear of not being found
with hip fracture; unable to call help
during lung biopsy for suspected malignancy

Physical (n = 57)
Acute respiratory failure (n = 15)
Exacerbation of chronic obstructive pulmonary disease*
Pulmonary embolism
Laryngeal obstruction from neoplasm
Respiratory distress from influenza
Acute epiglottitis requiring intubation
Central nervous system conditions (n = 10)
Subarachnoid hemorrhage
Brain contusion from accidental fall
Ruptured cerebral aneurysm
Vasculitis
Migraine headache
Seizure
Brain abscess
Malignancy† (n = 6)
Advanced or metastatic cancer
Chemotherapy for metastatic colon or esophageal cancer
Metastatic carcinoid tumor
Infection (n = 3)
Urosepsis
Spinal fusion wound infection
Peritonitis from ischemic bowel
Post-surgical/fracture (n = 8)
Hysterectomy and bilateral oophorectomy
Knee arthroplasty
Discectomy and T12-S1 fusion for scoliosis‡
Cholecystectomy
Decompression of spinal stenosis
Pericardiocentesis
Hip fracture
Other (n = 15)
Nosebleed treated with phenylephrine
Accidental home insulin overdose
Intentional Phenergan overdose
Dobutamine stress test
Gastrointestinal bleeding/gastroenteritis with dehydration
Acute rejection of renal transplant
Diabetic/nondiabetic gastroparesis
Anorexia with profound weight loss
Allergic drug reaction
Prolonged viral illness/dehydration
Hypertensive crisis
Withdrawal from alcohol

Sharkey et al., J Am Coll Cardiology

Critères diagnostiques

MAYO CLINIC

ansient hypokinesis, akinesis, or dyskinesis of the left ventricular mid
gments with or without apical involvement; the regional wall motion
normalities extend beyond a single epicardial vascular distribution; a
essful trigger is often, but not always present.*
sence of obstructive coronary disease or angiographic evidence of
ute plaque rupture. †
ew electrocardiographic abnormalities (either ST-segment elevation
d/or T-wave inversion) or modest elevation in cardiac troponin.
sence of:
eochromocytoma
ocarditis

h of the above circumstances, the diagnosis of ABS should be made with caution,
dear stressful precipitating trigger must be sought.
e are rare exceptions to these criteria such as those patients in whom the
al wall motion abnormality is limited to a single coronary territory.
possible that a patient with obstructive coronary atherosclerosis may also
p ABS. However, this is very rare in our experience and in the published
ure, perhaps because such cases are misdiagnosed as an acute coronary
ome.

SCA +++

et al., Am Heart J, 2008

JAPONESE CIRCULATION SOCIE

Table 1 Guidelines for Diagnosis of Takotsubo (Ampulla) Cardiomyopathy

I. Definition

Takotsubo (ampulla) cardiomyopathy is a disease exhibiting an acute left ventricular apical ballooning of unknow
In this disease, the left ventricle takes on the shape of a "takotsubo" (Japanese octopus trap). There is nearly com
solution of the apical akinesis in the majority of the patients within a month. The contraction abnormality occurs
the left ventricle, but involvement of the right ventricle is observed in some cases. A dynamic obstruction of the left
lar outflow tract (pressure gradient difference, acceleration of blood flow, or systolic cardiac murmurs) is also obse
Note: There are patients, such as cerebrovascular patients, who have an apical systolic ballooning similar to that
tsubo cardiomyopathy, but with a known cause. Such patients are diagnosed as "cerebrovascular disease with takot
myocardial dysfunction" and are differentiated from idiopathic cases.

II. Exclusion criteria

The following lesions and abnormalities from other diseases must be excluded in the diagnosis of takotsubo (ampu
diomyopathy.

A. Significant organic stenosis or spasm of a coronary artery. In particular, acute myocardial infarction due to a
the anterior descending branch of the left coronary artery, which perfuses an extensive territory including the left
lar apex (An urgent coronary angiogram is desirable for imaging during the acute stage, but coronary angiograph
necessary during the chronic stage to confirm the presence or absence of a significant stenotic lesion or a lesion
in the abnormal pattern of ventricular contraction).

B. Cerebrovascular disease
C. Pheochromocytoma
D. Viral or idiopathic myocarditis

Note: For the exclusion of coronary artery lesions, coronary angiography is required. Takotsubo-like myocardial dy
could occur with diseases such as cerebrovascular disease and pheochromocytoma.

III. References for diagnosis

A. Symptoms: Chest pain and dyspnea similar to those in acute coronary syndrome. Takotsubo cardiomyopathy c
without symptoms.
B. Triggers: Emotional or physical stress may trigger takotsubo cardiomyopathy, but it can also occur without a
rent trigger.
C. Age and gender difference: Known tendency to increase in the elderly, particularly females.
D. Ventricular morphology: Apical ballooning and its rapid improvement in the ventriculogram and echocardiogr
E. Electrocardiogram: ST segment elevations might be observed immediately after the onset. Thereafter, in a typi
the T-wave becomes progressively more negative in multiple leads, and the QT interval prolongs. These changes
gradually, but a negative T-wave may continue for several months. During the acute stage, abnormal Q-waves and
in the QRS voltage might be observed.
F. Cardiac biomarkers: In a typical case, there is only modest elevations of serum levels of cardiac enzymes and tr
G. Myocardial radionuclear study: Abnormal findings in myocardial scintigraphy are observed in some cases.
H. Prognosis: The majority of the cases rapidly recover, but some cases suffer pulmonary edema and other sequelae

Kawai et al, Circ Journa

Complications

Table 2
Clinical outcomes

Outcome	n (%)
Complication	
Heart failure	22 (26%)
Intra-aortic balloon pumping	4 (5%)
Mechanical ventilation	2 (2%)
Interventricular pressure gradient	11 (13%)
Severe acute mitral regurgitation	9 (11%)
Embolism	3 (4%)
Ventricular tachycardia/fibrillation	3 (4%)
Cardiogenic shock	9 (11%)
In-hospital death	2 (2%)
Number of above complications	
1	20 (24%)
≥2	14 (16%)

Takashio et al., Am J Cardiology

Pronostic

COURT TERME

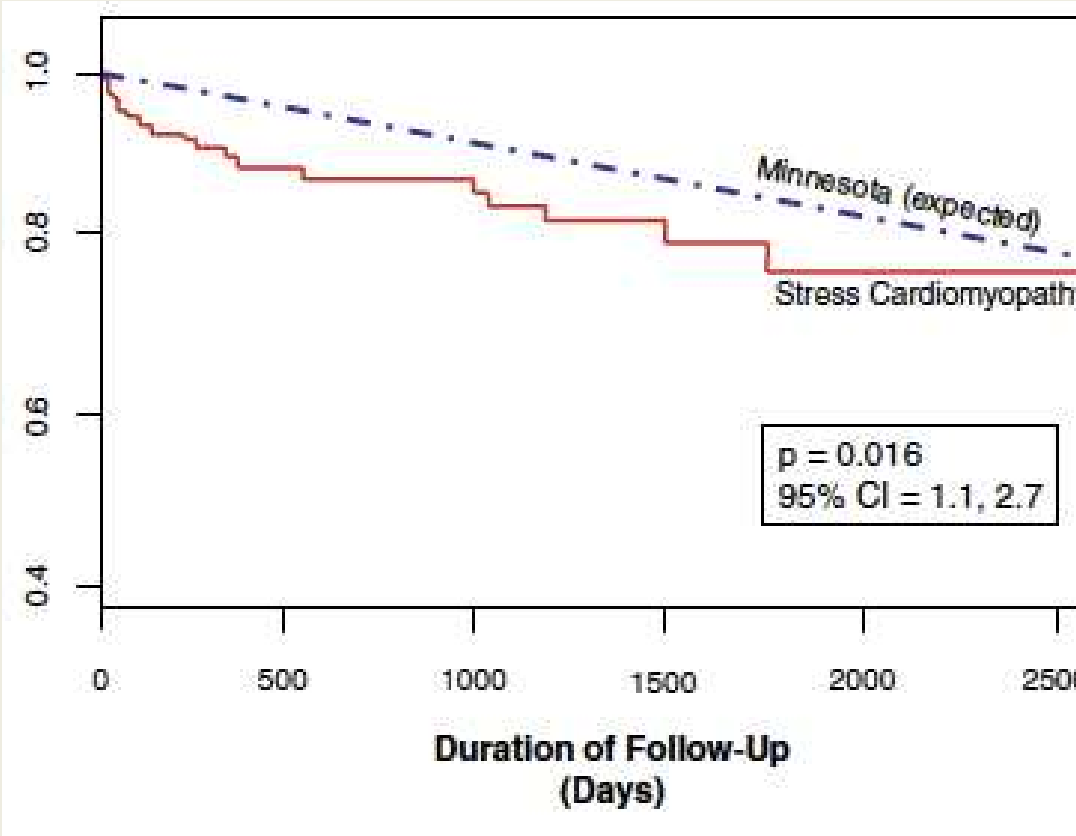
haracteristics	General admissions, n (%)	TTC, n (%)
tal admissions	33 506 402	6837
ge, y*		
18-34	6 556 830 (19.6)	127 (1.9)
35-49	5 588 174 (16.7)	581 (8.5)
50-64	7 382 129 (22)	1975 (28.9)
65-79	8 038 673 (24)	2952 (43.2)
≥ 80	5 963 187 (17.8)	1202 (17.6)
ex*		
Male	13 271 083 (39.7)	660 (9.7)
Female	20 173 088 (60.3)	6178 (90.4)
ace*		
White	18 957 769 (56.5)	4606 (67.4)
Black	3 445 928 (10.3)	300 (4.4)
Hispanic	2 638 251 (7.9)	298 (4.3)
Asian	680 357 (2.0)	76 (1.1)
Native American	173 273 (0.5)	43 (0.6)
Others	882 716 (2.6)	103 (1.5)
known	6 750 699 (20.1)	1413 (20.7)
utcomes		
Died	787 814 (2.4)	151 (2.2)

< .05 using χ^2 test. See text for details

81,4% causes non-cardiaques

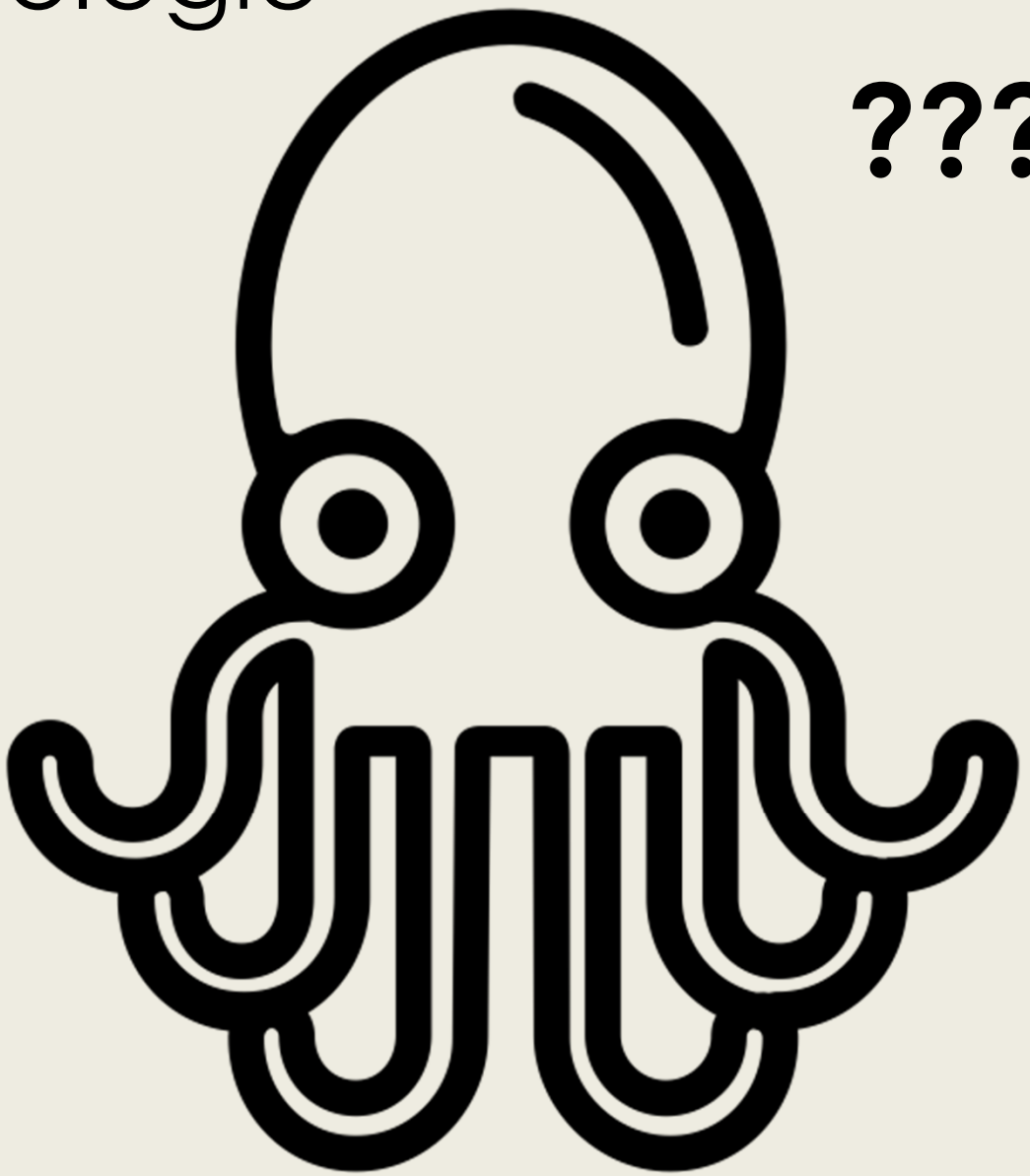
unk et al., Am Heart J, 2012

LONG TERME



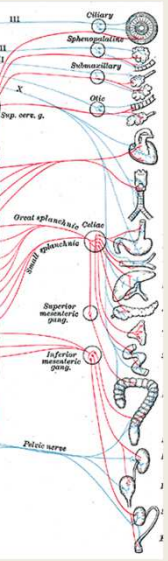
Sharkey et al., J Am Coll Cardiology

Physiopathologie



???

Rappel 1 : Synthèse catécholamines endogènes



Fibres post-ganglionnaires

Médullo Surrénale

Tyrosine → DOPA → Dopamine → NAD → AD

Tyrosine hydroxylase

DOPA décarboxylase

Dopamine Bhydroxylase

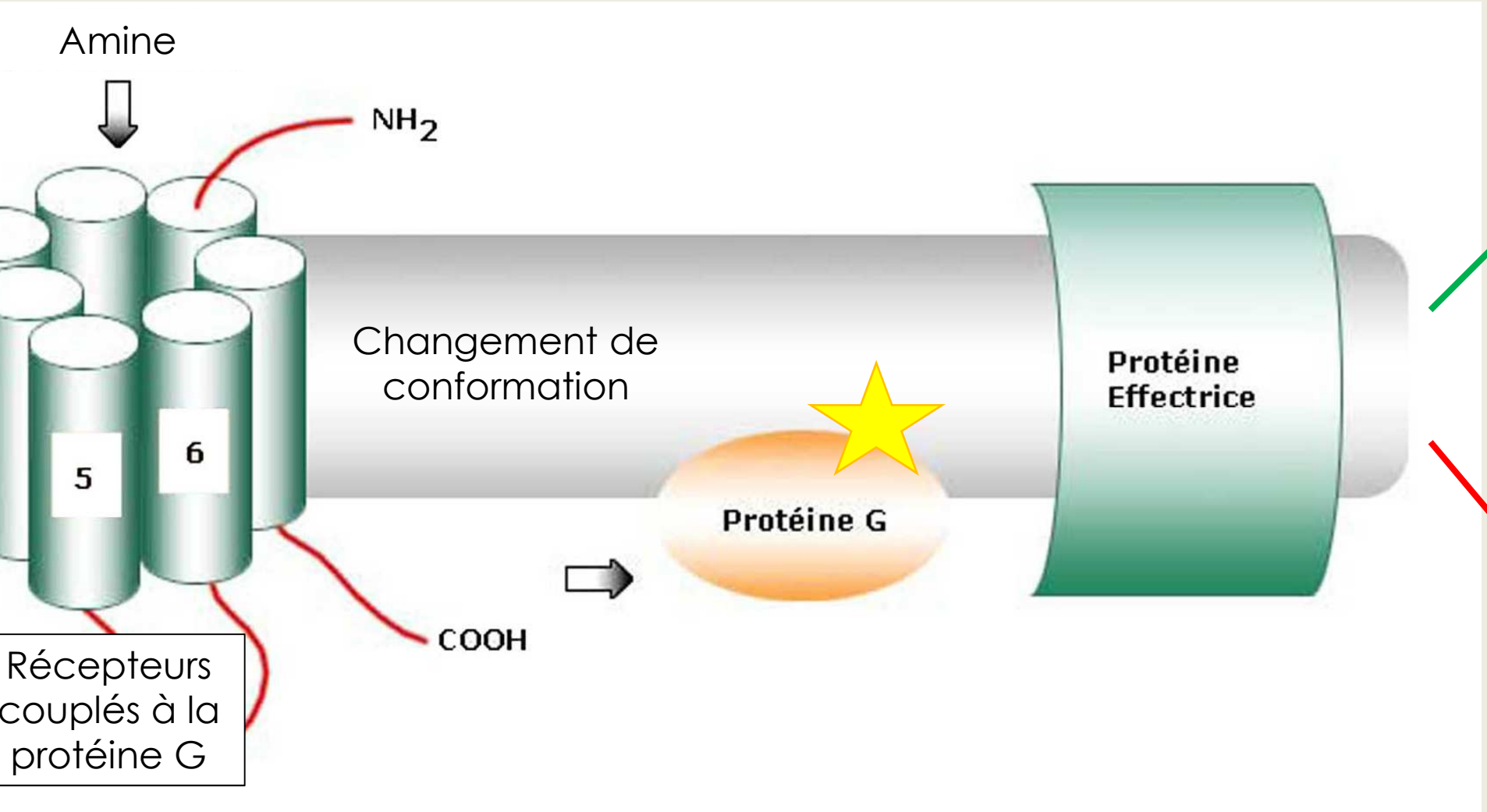
Méthylation

★ Régulation par la concentration intra cytoplasmique de NAD

Sympathomimétiques indirects : adrénaline, amphétamines

Induisent la libération de NAD par ses sites de stockage

Rappel 2 : Récepteurs adrénergiques



G protéin
activatric
Gs / Gq

Alpha 1
Béta 1
Béta 2

G protéin
inhibitric
Gi / Go

Alpha 2
Béta 2

cepteur béta 2 peut être couplé à une G protéine **activatrice OU inhibitr**

Rappel 2 : Récepteurs adrénergiques

RÉNALINE	ALPHA 1	+++	Fibres musculaires lisses	Vasculaires : vasoconstriction
	BÉTA 1	+++	Cardiaque	C+ I+ D+ B+
	BÉTA 2	++	Vaisseaux Bronches	Relaxation FML vasculaires broncho dilatation
RADRÉNALINE	ALPHA 1	+++	Fibres musculaires lisses	Vasculaires : vasoconstriction
	BÉTA 1	+	Cardiaque	C+ I+ D+ B+
BUTAMINE	BÉTA 1	+++	Cardiaque	C+ I+ D+ B+
	BÉTA 2	+	Vaisseaux Bronches	Relaxation FML vasculaires broncho dilatation

Concentration plasmatiques élevées en catécholamine

Catécholamines ENDOGÈNES

Catécholamines EXOGÈNES

Augmentation de la **synthèse**

Augmentation de la **libération**

Injection de catécholamines :
AD
NAD
Isoprénaline
Dobutamine

Pathologique :
Phéochromocytome

Physiologique :
Stress physique / psychique

Libération par les sites de stockage :
Cocaïne
Amphétamines…

CMP liée au
héochromocytome
e description 1989

Takotsubo

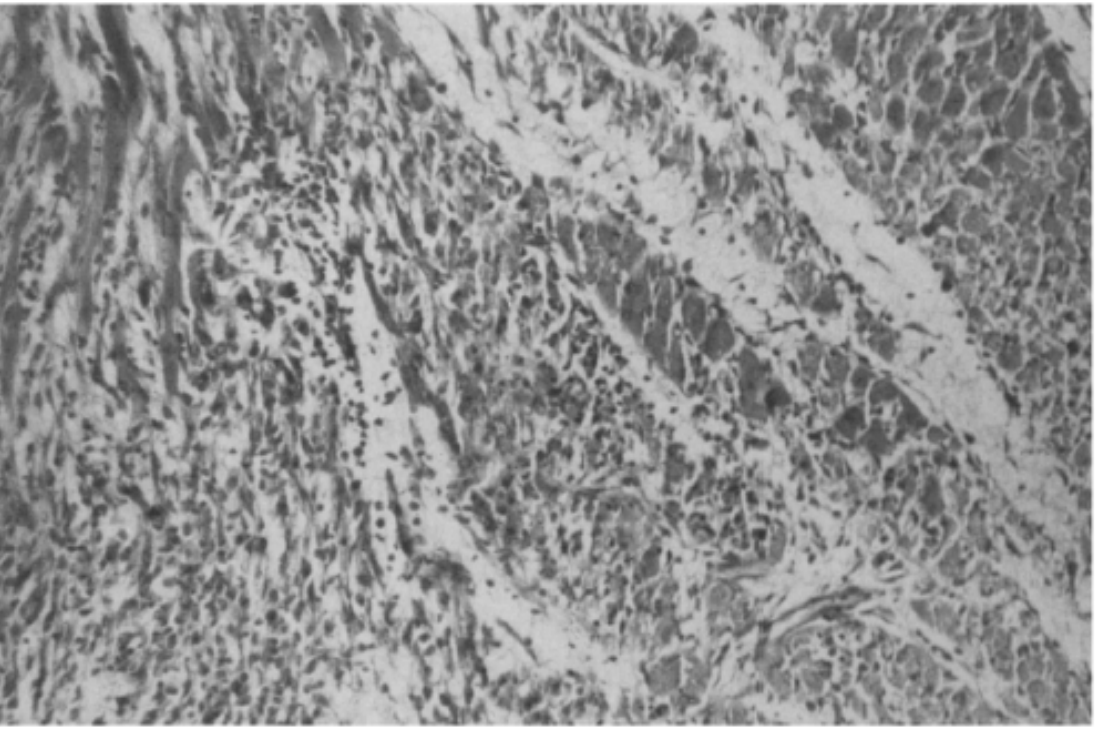
CMP liée aux catécholamine
1ère description 1950's

ll Cardiol **17**, 291–306 (1985)

EDITORIAL REVIEW

Catecholamine Cardiotoxicity

Cardiomyopathie associées aux catécholamines



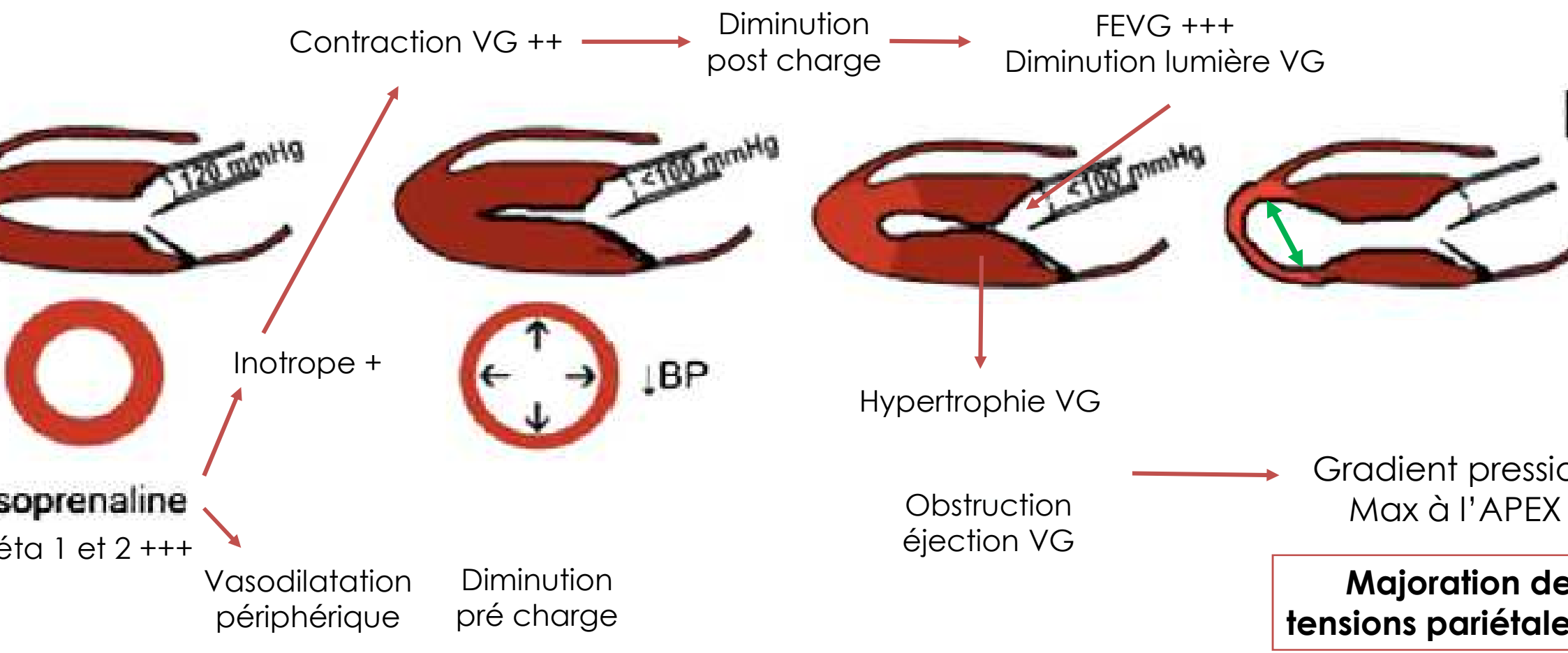
E 3. The necrotic cardiac muscle fibers are separated from normal fibers by mucoid edema. There is early
ction. PAS × 120.

Cardiotoxic effect of epinephrine and norepinephrine

Soon after the discovery of epinephr
Josué showed that this agent produces m
cardial hypertrophy [*53*]. The epinephr
induced myocardial alteration was designa
as myocardial necrosis [*80*] or myocard
[*38*]. In the late fifties, several publicat
appeared indicating that norepinephrin
also cardiotoxic [*62*, *72*, *112*, *121*]. Th
experimental works focused attention to
human myocardial changes following th
peutic norepinephrine administration [*122*

ypothèse 1 : « cardiac stunning »

'est à dire : mécanisme de protection du cardiomyocyte pour épargner les régions affectées pa
rose et les arythmies



Redfors et al., Int J Cardiology

pothèse 1 : « cardiac stunning »

Majoration des tensions pariétales VG

Augmentation des contraintes physiques locales Supra-physiologique

Hypertrophie VG
Mécanisme compensateur de la redistribution des contraintes

Augmentation de la demande contractile myocardique

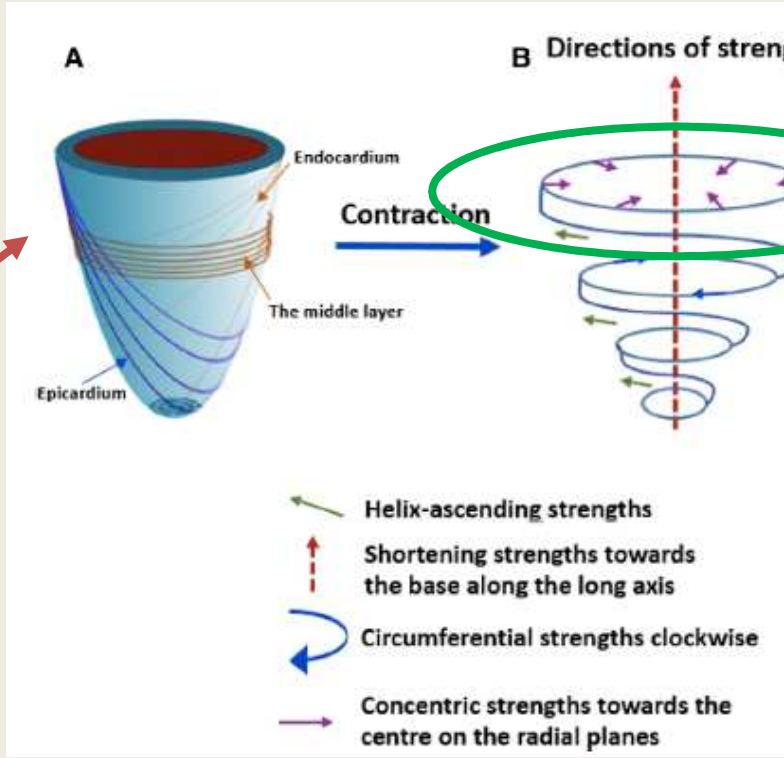
Augmentation de la demande d'NRJ

« cardiac stunning »

Déséquilibre entre demande et capacité

dèle de rat : pas les mêmes
ultats avec E/NE…

s et al., Int J Cardiology, 2014



Shao et al., Int J Cardiolog

ypothèse 2 : défaut de perfusion myocardique initial

Spasmes coronaires et défaut de la microvascularisation

Thrombus coronaire dissout

IRM : défauts de rehaussement

Spasmes pendant coro
Variant anatomique?

Défauts de perfusion : Conséquence

TOREGULATION

Pas de défaut de perfusion initial dans les modèles murins

Pas de corrélation fonctionnelle
Zone touchée trop étendue

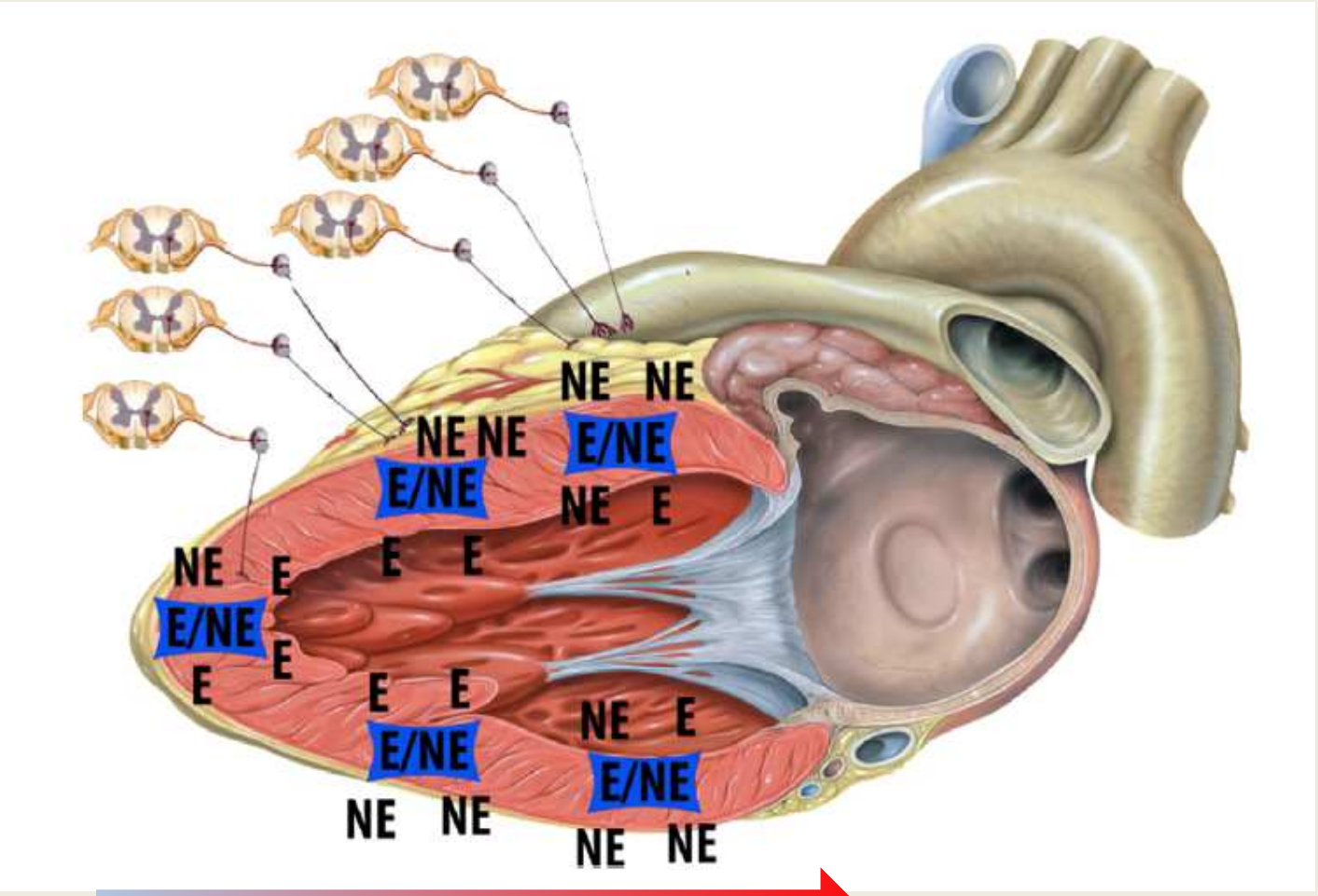
Spasme des coronaires principales

odèle de cardiopathie « ischémique »…? :
nadéquation entre demande et apports suffisants en O2 et nutriments
ones d'ischémies locales

- altération de la conduction / œdème
- redistribution des contraintes physiques sur les zones non- touchées

Redfors et al., Int J Cardiology

pothèse 3 : excès de relargage **local** de catécholamines



Gradient de répartition de NE

Pierpont et al., Am J Physiology, 1984

Apports des catécholamine
- Système sympathique
  - NE >> E
- Sang
- Zones de stockage cardiaques

Modèles murins : majoratio des catécholamines **LOCALEMENT** ne suffit pas

Redfors et al., Int J Cardiology

ypothèse 4 : « beta2 adrenoreceptor/Gi-pathway pothesis »

siologique
RÉNALINE

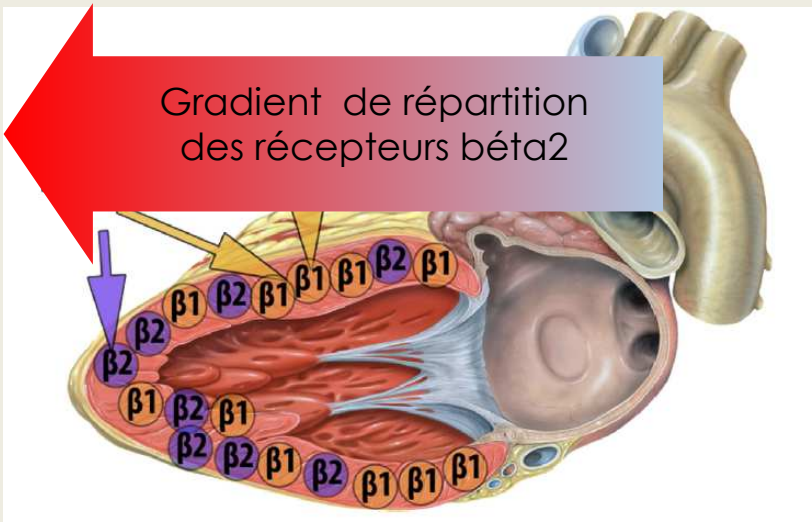
BÉTA 1 +++
BÉTA 2 ++
R Couplé à Gs
Activation PKA
INOTROPE +

SWITCH

hologique
centrations
aphysiologiques
RÉNALINE

BÉTA 2 ++
R Couplé à **Gi**
INOTROPE -

uctible AUSSI par NE
uctible MEME quand inhibition de la Gi (toxine tussique)



s et al., Int J Cardiology, 2014

ypothèse 5 : carence oestrogénique

STRESS, NEUROTRANSMITTERS, AND HORMONES

Catecholamines and Estrogen Are Involved in the Pathogenesis of Emotional Stress-induced Acute Heart Attack

Takashi Ueyama,[a] Ken Kasamatsu,[b] Takuzo Hano,[b] Yoshihiro Tsuruo,[a] and Fuminobu Ishikura[c]

[a]Department of Anatomy and Cell Biology and [b]Department of Cardiovascular Medicine, Wakayama Medical University School of Medicine, Wakayama, Japan

[c]Division of Functional Diagnostic Science, Osaka University Graduate School of Medicine, Suita, Japan

INCIDENCE MAXIMALE CHEZ LES FEMMES MÉNOPOSÉES

Majoration de la sensibilité des Béta récepteurs
Diminution de la concentration basale de catécholamine

raitement

ort risque…?

Box 2 Differences between Takotsubo syndrome and acute myocarditis

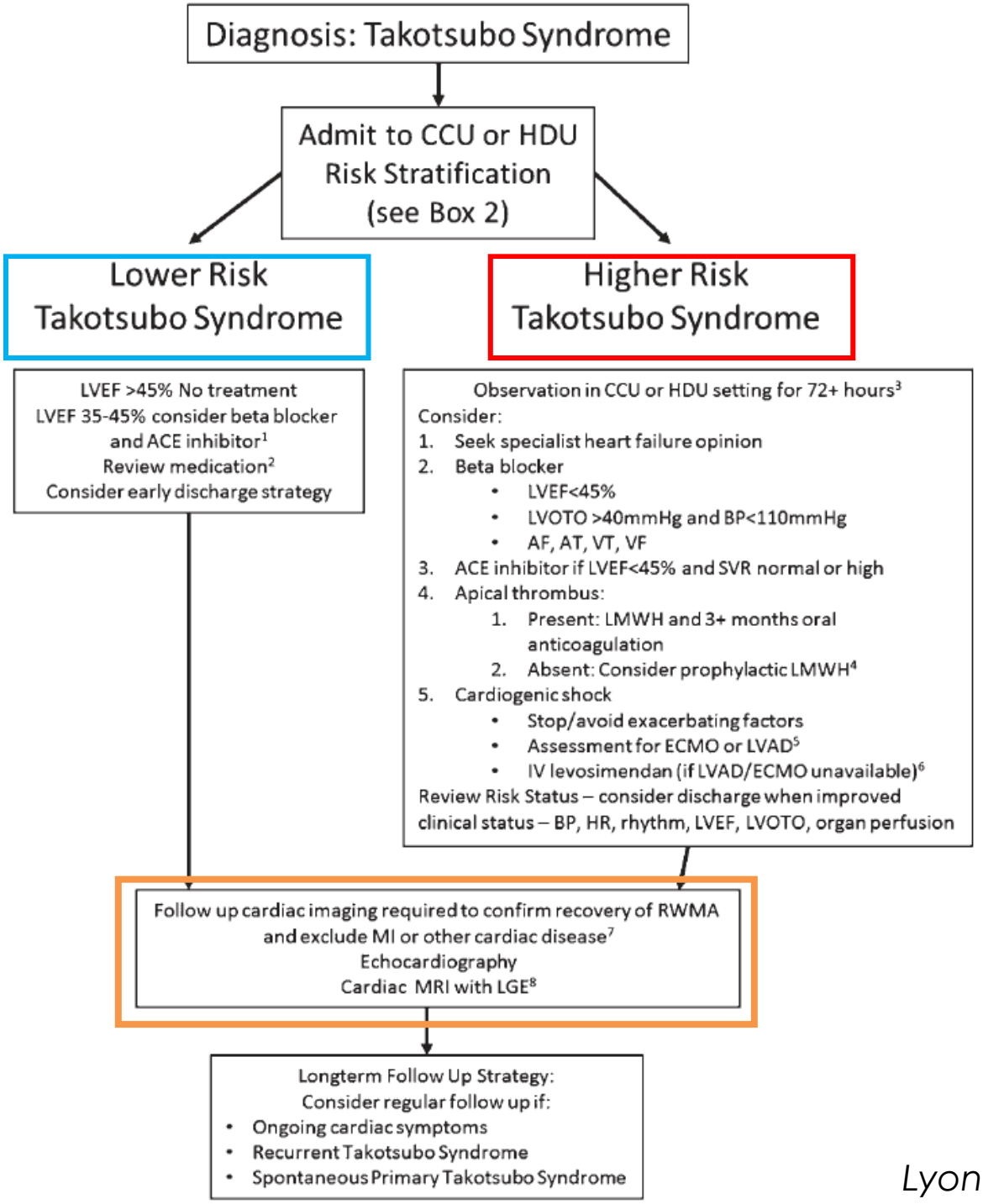
Category	Takotsubo syndrome	Acute myocarditis
Gender and age	90% female. Majority >50 years and post-menopausal.	No sex prevalence. More frequent in the young.
Preceding events	Stressor trigger identifiable in ~70% of cases.	Symptoms and signs of infection often present (fever, chills, headache, muscle aches, general malaise, cough, nausea, vomiting, diarrhoea).
Cardiac symptoms	Chest pain, dyspnoea, palpitations.	Chest pain, dyspnoea, peripheral oedema, fatigue, and palpitations.
Clinical signs	Pericardial rub rare.	Pericardial rub may be present.
ECG at admission	ST changes such as ST-segment elevation or non ST-segment elevation. Deep T wave inversion. QT prolongation. Rarely normal.	ST-segment elevation or depression, negative T-wave, bundle branch block, atrioventricular block, low voltage, and/or ventricular arrhythmias. Normal in several cases.
Cardiac enzymes	Low/moderate troponin rise. Discrepancy between the large amount of dysfunctional myocardium and peak troponin level.	Frequently significant troponin rise, proportional to the hypokinetic area. Normal in several cases.
Other biomarkers	C-reactive protein (CRP) mildly elevated unless infective trigger. BNP moderately or significantly elevated.	Erythrocyte sedimentation rate and CRP elevated. BNP basically elevated. Acute viral serology may be detected.
Echocardiography	Apical ballooning, anatomical variants, 'circumferential pattern', left ventricular outflow tract obstruction (LVOTO), right ventricular (RV) involvement, transient mitral regurgitation.	Localized or diffuse wall motion abnormalities of LV and/or RV dilatation, increased wall thickness, pericardial effusion.
Cardiac magnetic resonance imaging	High T2 signal intensity (oedema), late gadolinium enhancement (LGE) usually absent acutely. If present acutely patchy LGE which usually resolves at follow-up. Absence of typical infarct LGE pattern.	High T2 signal intensity (oedema), LGE with non-ischaemic distribution (often epicardial). Absence of typical infarct LGE pattern.
Histological findings	Contraction band necrosis.	Infiltration of many inflammatory cells. Interstitial oedema.
Viral genome, separation of virus, or identification of virus by antibody titre	Rare and usually absent where measured.	Often positive.
Prognosis	50% of cases have acute complications, 4–5% mortality.	Variable but majority full recovery. Highest mortality with fulminant myocarditis.
Therapy	Supportive.	Supportive. Immunosuppression in severe cases if giant cell myocarditis suspected.

aitement

EVG > 45%
rien

VG 35 – 45 %
B- +/- IEC

Surveillance
Vérifier
rmalisation FEVG



B-
FEVG < 45%
Arythmies A/
LVOTO+ / Ho

Thrombus
apical ?

Choc
cardiogéniqu
ECMO

Lyon et al, Eur J Heart Failure

Take Home Messages

Cardiomyopathie transitoire réversible

- Induite par stress physique / psychologique
- Akinésie / hypokinésie apicale et antéro-apicale du VG +/- VD

Incidence maximale chez les femmes ménopausées

Physiopathologie complexe – plusieurs phénomènes intriqués

- Hyperexcitabilité sympathique
- Cardiac stunning…

Traitement :

- Pas d'inotrope + / vasodilatateur
- Dépend de la sévérité