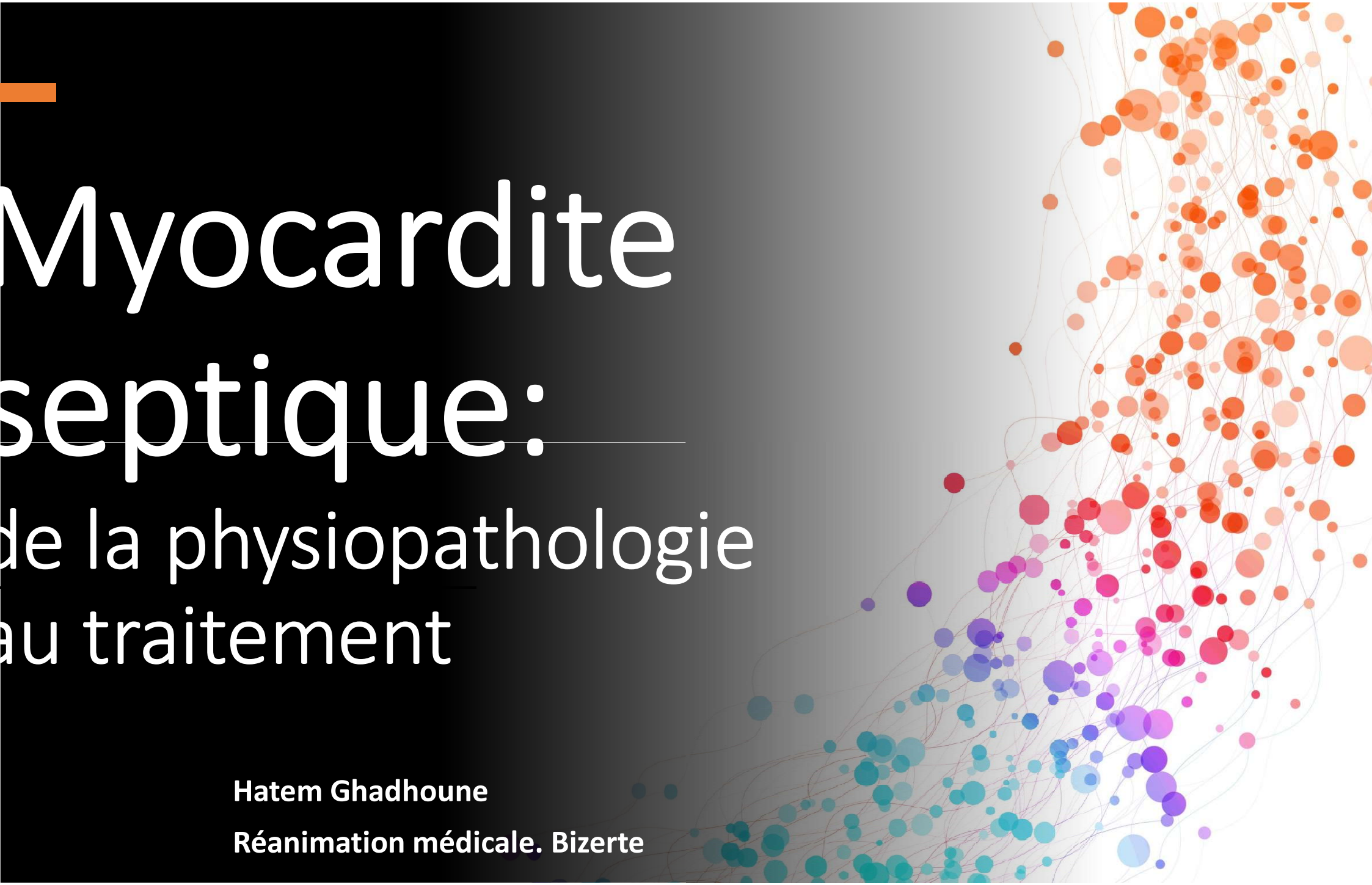
Myocardite septique:

de la physiopathologie au traitement

Hatem Ghadhoune

Réanimation médicale. Bizerte



Pas De Conflit D'intérêt

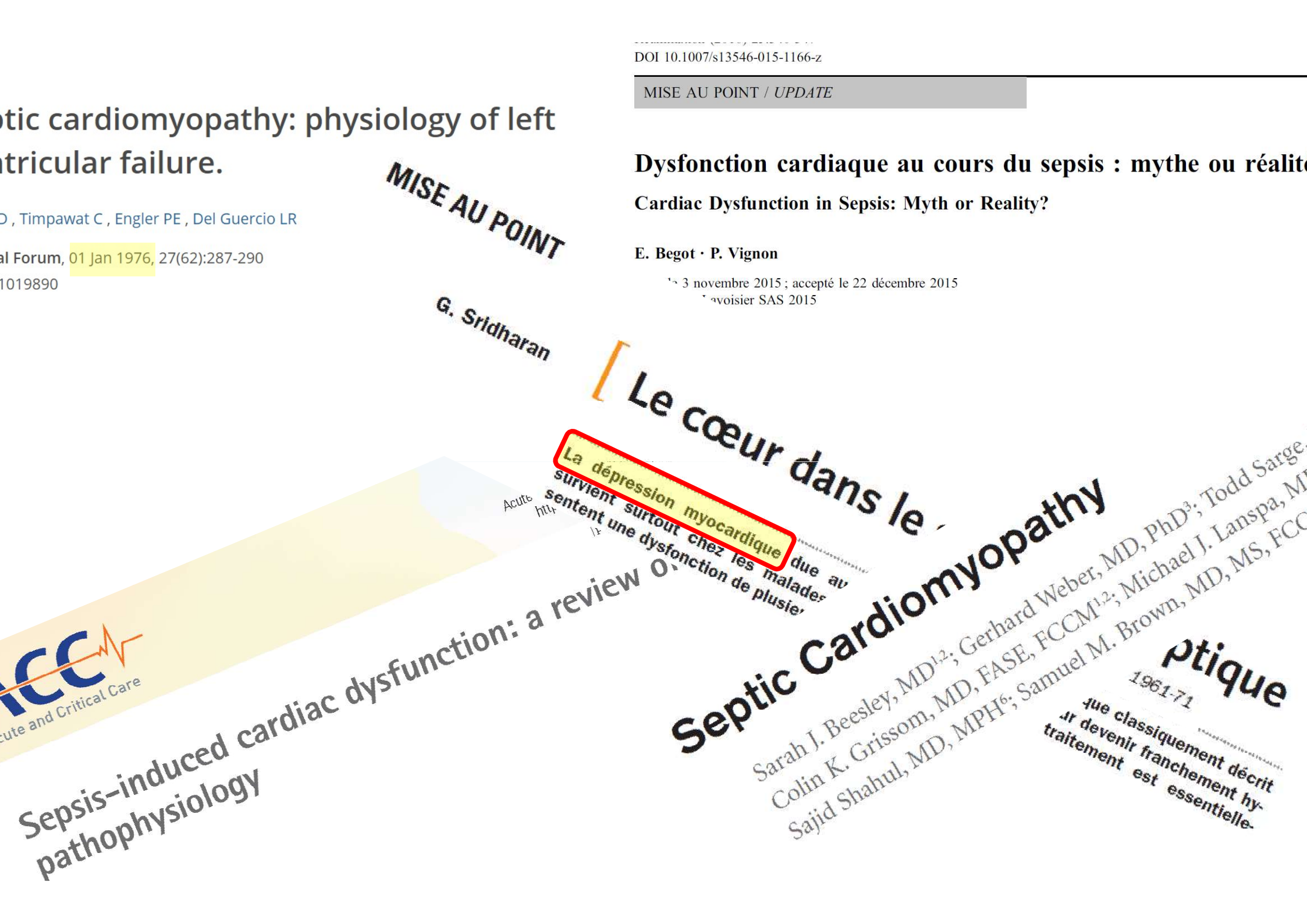
Appellation et définitions ?

Mécanismes physiopathologiques ?

Difficultés diagnostiques?

Thérapeutiques ?

Pronostic ?



otic cardiomyopathy: physiology of left
ntricular failure.

D , Timpawat C , Engler PE , Del Guercio LR

al Forum, 01 Jan 1976, 27(62):287-290
1019890

MISE AU POINT

G. Sridharan

DOI 10.1007/s13546-015-1166-z

MISE AU POINT / *UPDATE*

Dysfonction cardiaque au cours du sepsis : mythe ou réalité

Cardiac Dysfunction in Sepsis: Myth or Reality?

E. Begot · P. Vignon

3 novembre 2015 ; accepté le 22 décembre 2015
avoisier SAS 2015

Le cœur dans le

La dépression myocardique due au
survient surtout chez les malade
sentent une dysfonction de plusie

Acute
http

Septic Cardiomyopathy

Sarah J. Beesley, MD[1,2]; Gerhard Weber, MD, PhD[3]; Todd Sarge
Colin K. Grissom, MD, FASE, FCCM[1,2]; Michael J. Lanspa, M
Sajid Shahul, MD, MPH[6]; Samuel M. Brown, MD, MS, FC

ptique

1961-71

que classiquement décrit
ır devenir franchement hy-
traitement est essentielle

ACC
cute and Critical Care

Sepsis-induced cardiac dysfunction: a review o
pathophysiology

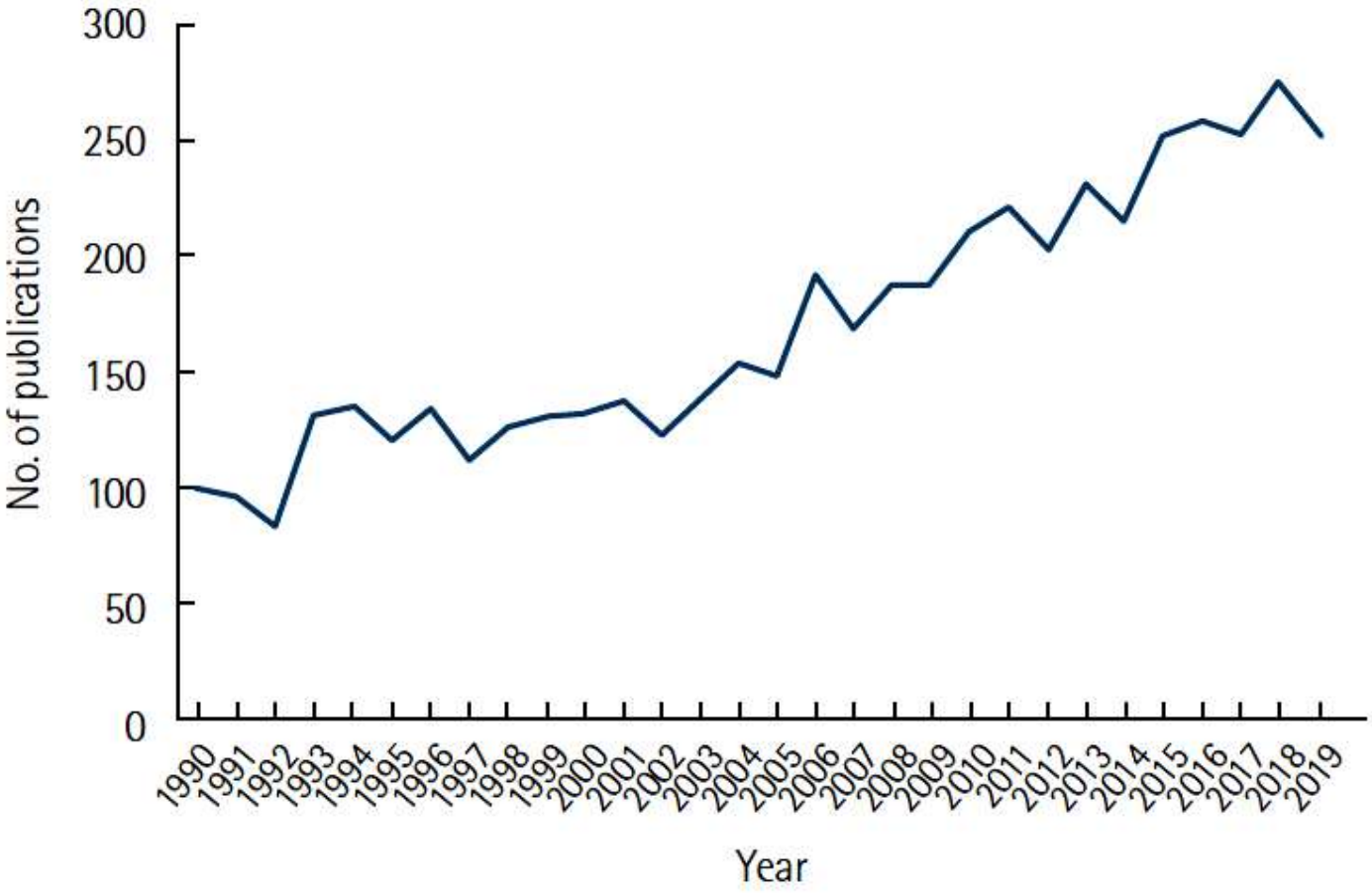


Figure 1. The number of publications per year on sepsis-induced cardiac dysfunction, obtained by searching for "sepsis," "cardiac dysfunction," "myocardial dysfunction," and "cardiomyopathy" in PubMed. The number was calculated from January 1, 1990 to December 31, 2019.

Cardiomyopathie septique

Appellation et définitions ?

sepsis

life-threatening **organ dysfunction** caused by a dysregulated host response to infection

EDCS

a vasopressor requirement to maintain a mean arterial pressure (MAP) ≥65 mmHg and serum lactate level ≥2 mmol/L (≥18 mg/dL), after adequate fluid resuscitation

Patterns of Septic Shock in Man—A Detailed Study of 56 Patients

LLOYD D. MACLEAN, M.D., F.R.C.S.(C), WILLIAM G. MULLIGAN, M.D., A. P. H. MCLEAN, M.D., F.R.C.S.(C), JOHN H. DUFF, M.D., F.R.C.S.(C)

1967

Index cardiaque bas

rofound but Reversible Myocardial Depression in Patients with Septic Shoc

MARGARET M. PARKER, M.D.; JAMES H. SHELHAMER, M.D.; STEPHEN L. BACHARACH, Ph.D.; MICHAEL V. GREEN, M.S.; CHARLES NATANSON, M.D.; TERRI M. FREDERICK, B.S.N.; BARBARA A. DAMSKE, R.N.; and JOSEPH E. PARRILLO, M.D.; Bethesda, Maryland

1984

FEVG < 40% et Volumes télé-systolique et télé-diastoliques augmentés

Apparition J2-3 et amélioration J7-10

The Septic Heart

Current Understanding of Molecular Mechanisms and Clinical Implications

Lukas Martin, MD; Matthias Derwall, MD; Sura Al Zoubi, PhD; Elisabeth Zechendorf, MSc; Daniel A. Reuter, MD; Chris Thiemermann, PhD; and Tobias Schuerholz, MD

CHEST **2019**; 155(2):427-437

acute syndrome of cardiac dysfunction unrelated to ischemia with one or more of the main characteristics:
left ventricular dilatation with normal- or low-filling pressure
reduced ventricular contractility
right ventricular dysfunction or left ventricular (systolic or diastolic) dysfunction with a reduced response to volume infusion

ptic Cardiomyopathy

arah J. Beesley, MD[1,2]; Gerhard Weber, MD
Colin K. Grissom, MD, FASE, FCCM[1,2]; Mic
ajid Shahul, MD, MPH[6]; Samuel M. Brown

Revista Pc

Portuguese Society of CARDIOLOGY

Revista Po
Cardi
Portuguese Journal o
www.revportcardio

REVIEW ARTICLE

Septic cardiomyopathy: A narrative review

Doroteia Silva[b,c]

Journal of Intensive Medicine 2 (2022) 8–16

Contents lists available at ScienceDirect

Journal of Intensive Medicine

journal homepage: www.elsevier.com/locate/jointm

opathy: Diagnosis and management☆

adia Aissaoui[3,4,*]

athophysiology of sepsis-induced
ardiomyopathy

Department of Pharmacy, Changchun University of Chinese Medicine, Changchun, Jil

JIAYU SONG, XIAOLEI FANG, KAIXUAN ZHOU, HUIWEI BA

MOLECULAR MEDICI

Sepsis-induced cardiac dy
pathogenetic mechanisms

ACC
Acute and Critical Care

Sepsis–induced cardiac dysfunction: a re
pathophysiology

Reverien Habimana[1,*], Insu Choi[2,*], Hu

Appellation et définitions ?

Dysfonction systolique du VG induite par le sepsis

Appellation et définitions ?

Mécanismes physiopathologiques ?

Difficultés diagnostiques?

Thérapeutiques ?

Pronostic ?

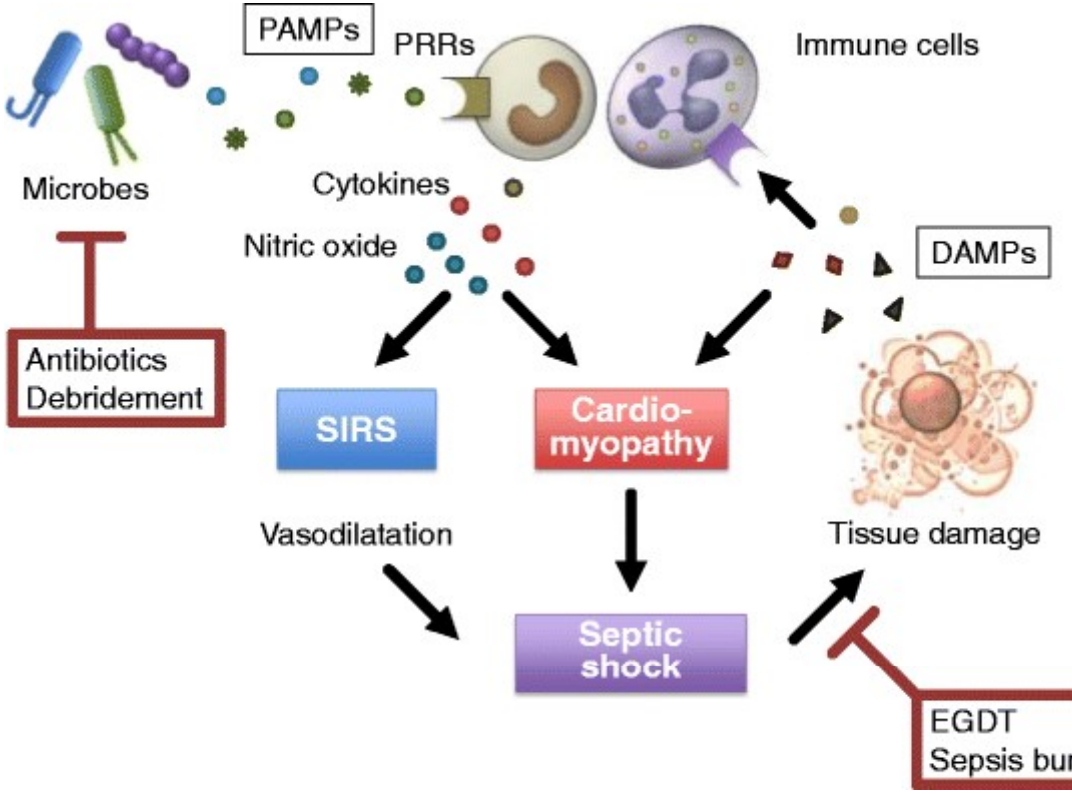
Dérégulation des médiateurs inflammatoires

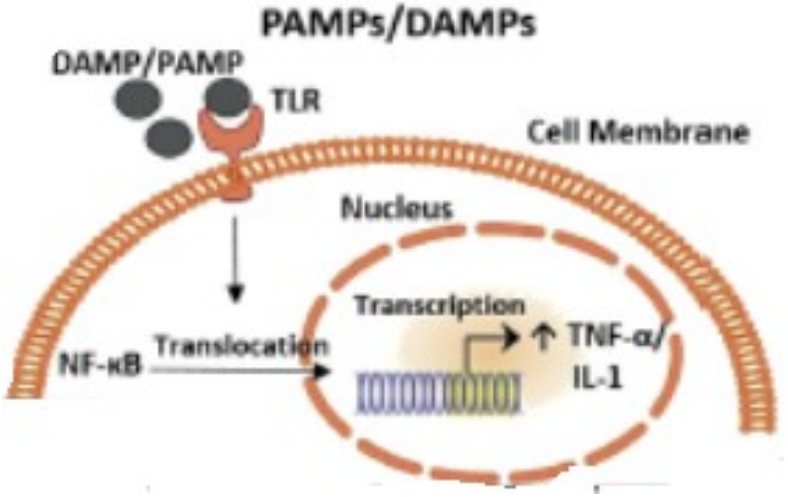
transcription accrue des médiateurs inflammatoires

→Apoptose des myocytes

→Action directe sur le système vasculaire périphérique

→Bloquer les effets adrénergiques sur la contractilité





fonctionnement mitochondrial

Sepsis

↓

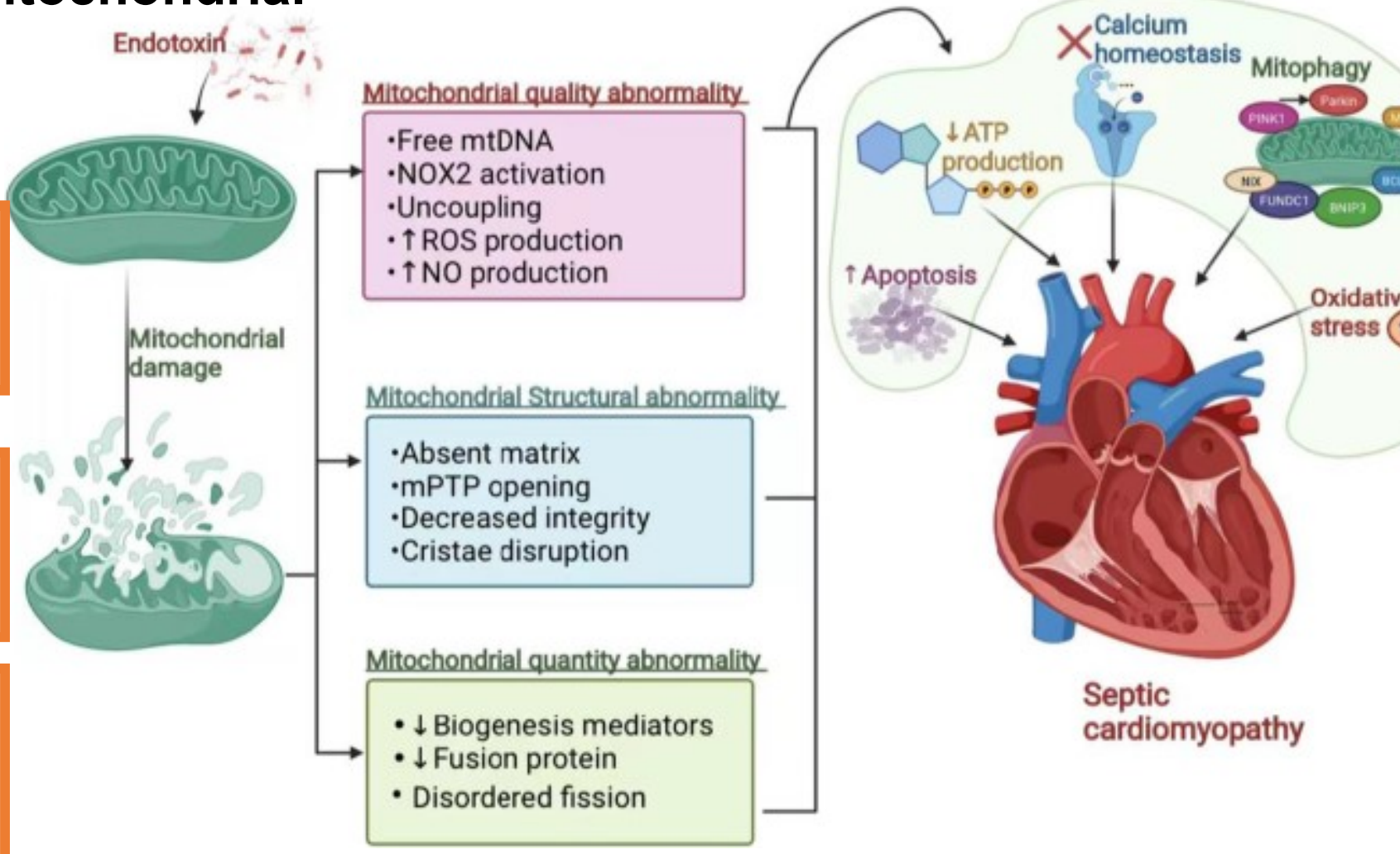
ibition non compétitive
de la cytochrome C
oxydase

↓

errompre l'inhibition de
la phosphorylation
oxydative

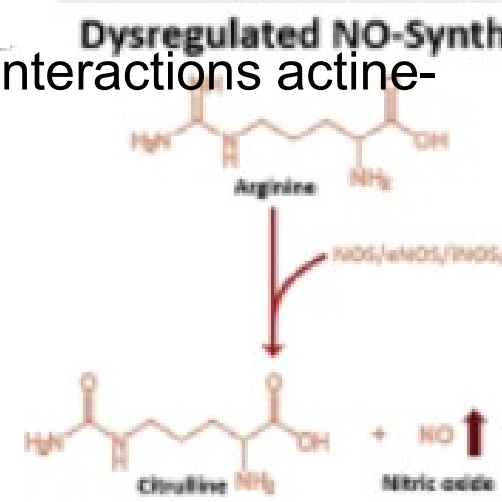
↓

pression myocardique

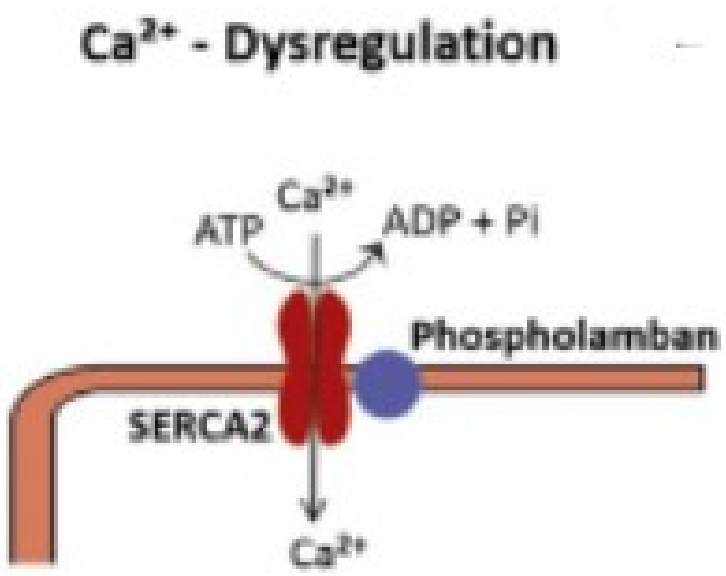


Stress oxydatif

- production déséquilibrée d'espèces réactives de l'oxygène (ROS) et d'espèces réactives de l'azote:
  - altère la phosphorylation oxydative
  - aboutit à l'apoptose mitochondriale
  - provoque des lésions des tissus adjacents et une réaction inflammatoire amplifiée
  - diminue la réponse des myofibrilles au calcium (rupture focale des interactions actine-myosine, entraînant une dilatation ventriculaire)
  - régulation négative des récepteurs adrénergiques



trouble de la régulation du calcium



- Diminution de la densité des canaux calciques
- la séquestration du calcium est perturbée (problème de phosphorylation du phospholamban)
- Diminution de la sensibilité des myofilaments au Ca
- Participe au dysfonctionnement mitochondrial

ysfonctionnement du système nerveux utonome

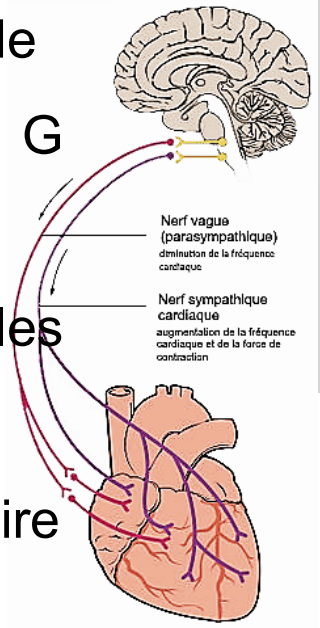
Résistance aux catécholamines et la perte le variabilité de la fréquence cardiaque en état septique

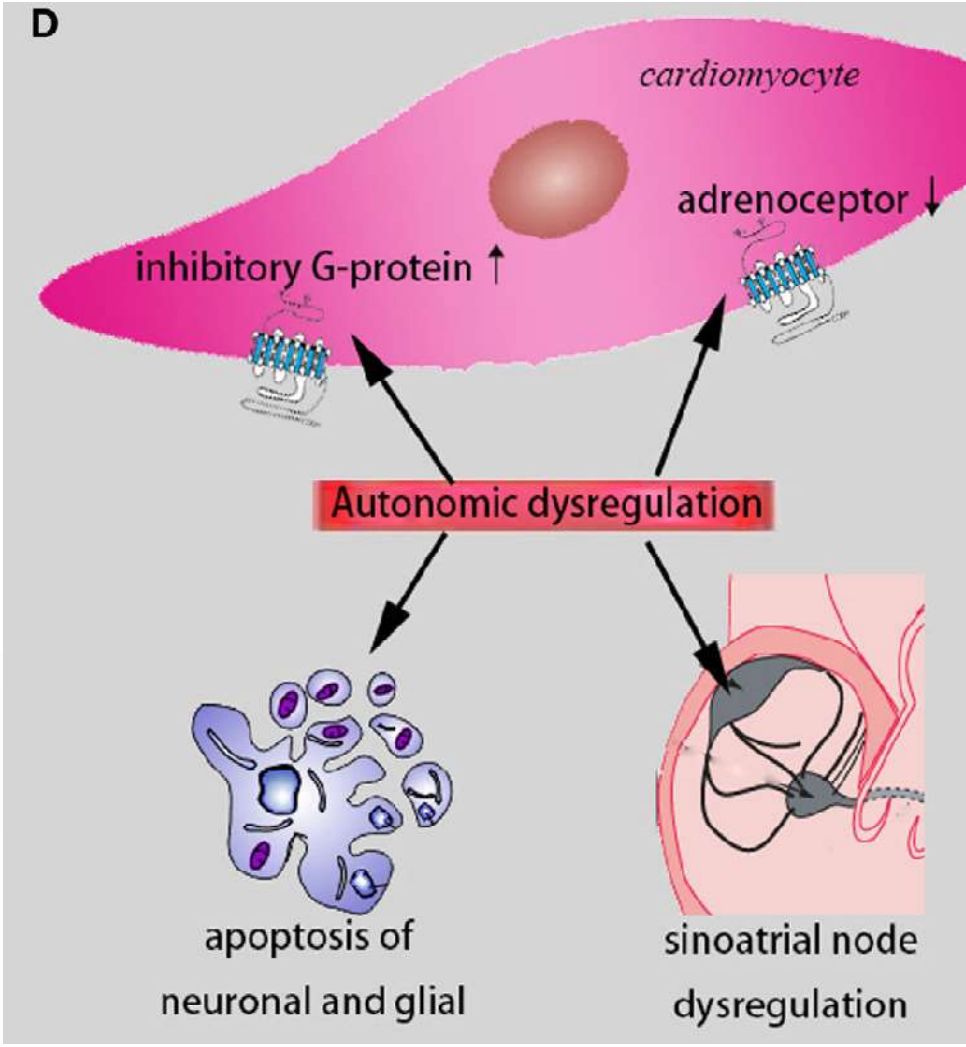
Médié par:

- la diminution de la densité des récepteurs adrénergiques du myocarde
- L'expression accrue de la protéine G inhibitrice

apoptose des neurones et des cellules les

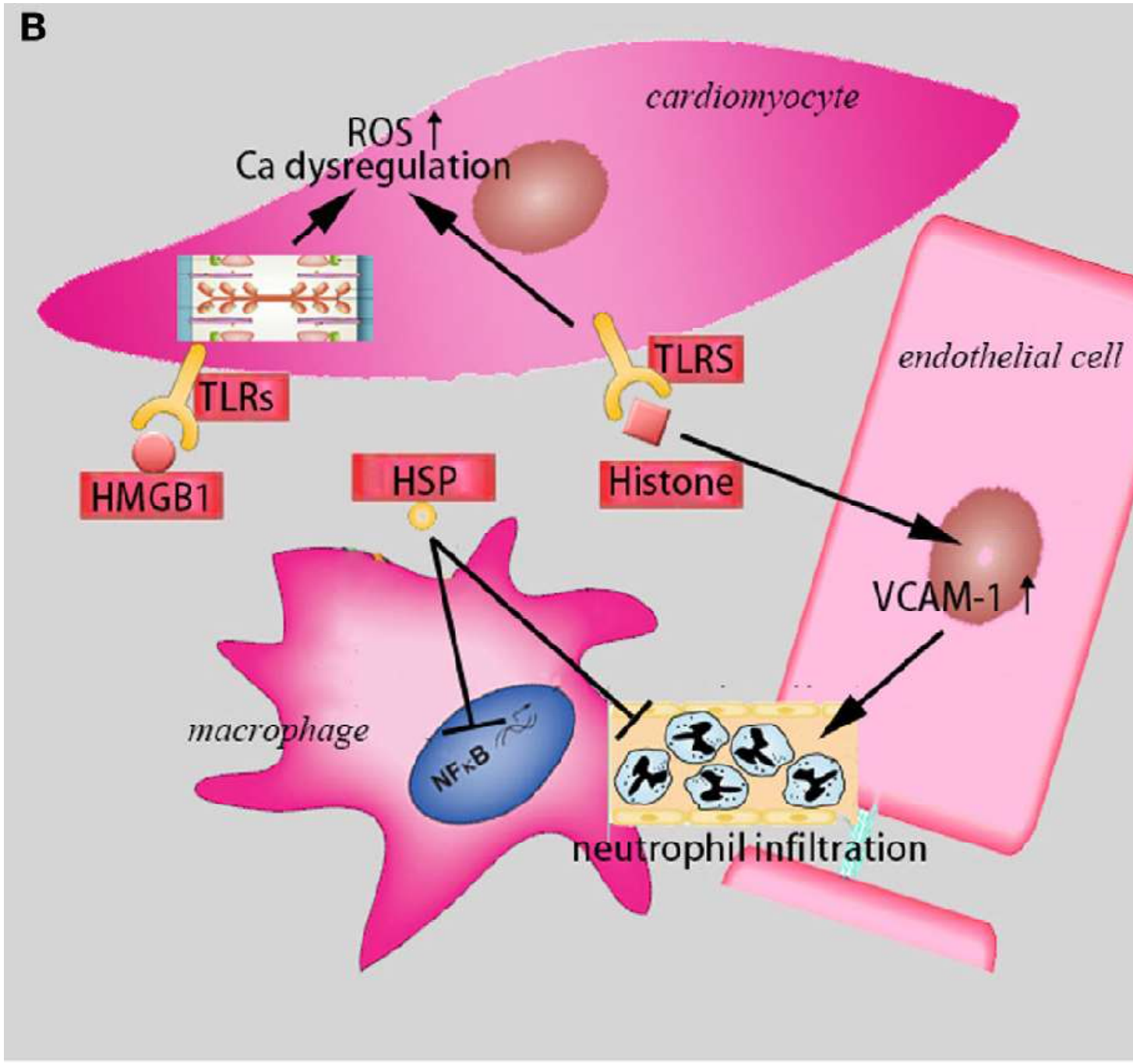
ysfonctionnement du nœud sino-auriculaire

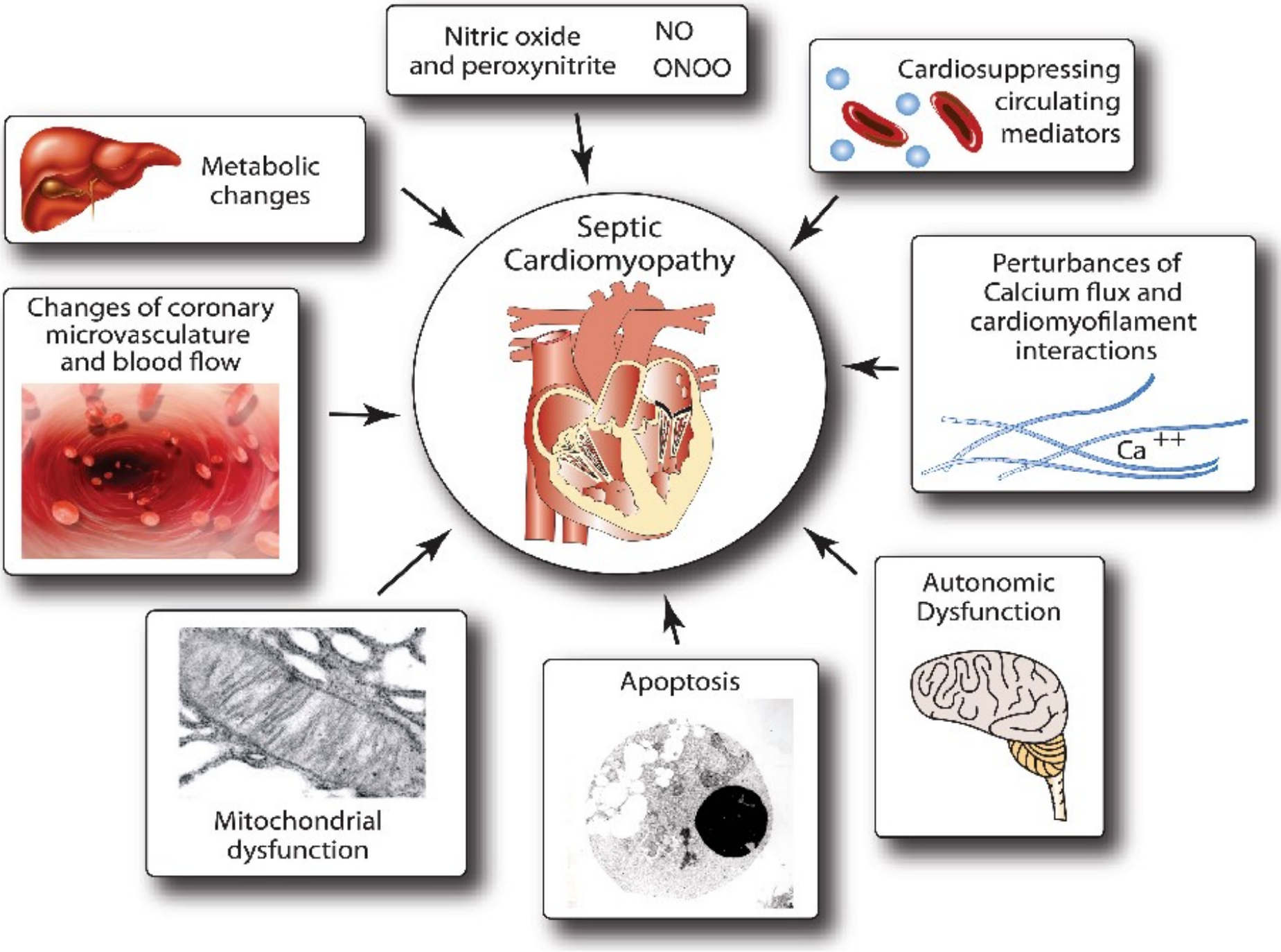




dysfonctionnement endothélial

Les cellules endothéliales sécrètent davantage de molécules d'adhésion, qui facilitent **l'infiltration nocive des neutrophiles** dans les cardiomyocytes et augmentent l'interaction leucocyte endothélium en réponse aux cytokines inflammatoires lors du sepsis




Nitric oxide and peroxynitrite
NO
ONOO
Cardiosuppressing circulating mediators
Metabolic changes
Septic Cardiomyopathy
Perturbances of Calcium flux and cardiomyofilament interactions
Ca ++
Changes of coronary microvasculature and blood flow
Autonomic Dysfunction
Apoptosis
Mitochondrial dysfunction

Appellation et définitions ?

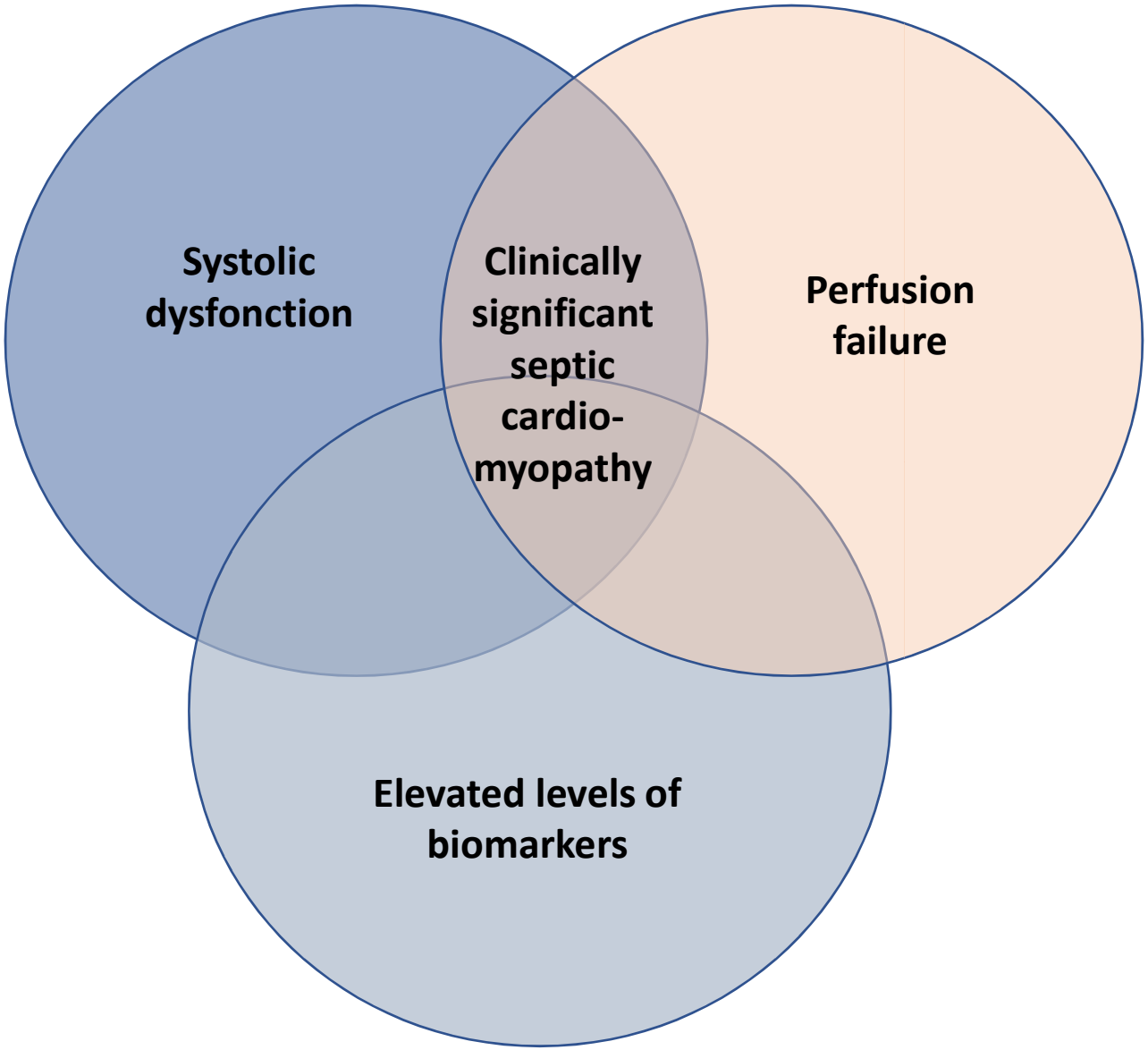
Mécanismes physiopathologiques ?

Difficultés diagnostiques?

Thérapeutiques ?

Pronostic ?

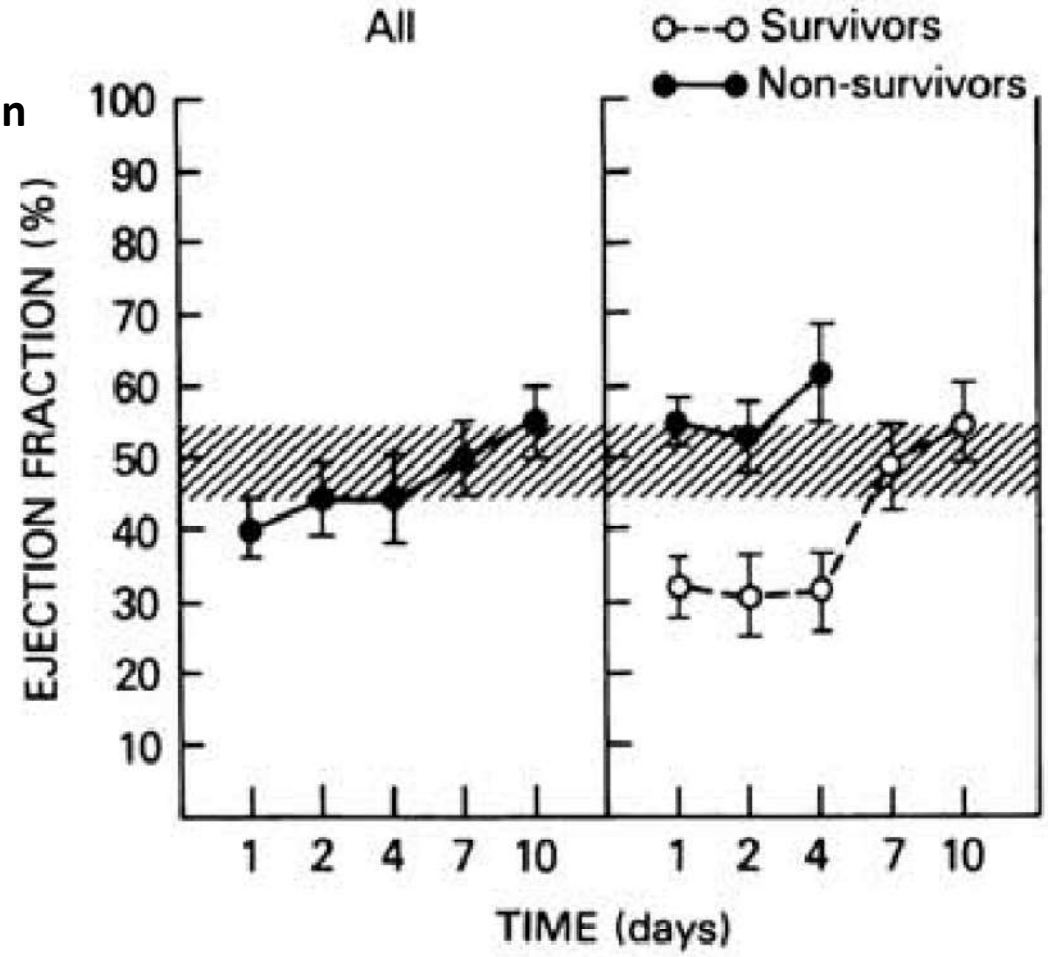
Difficultés diagnostiques cliniques



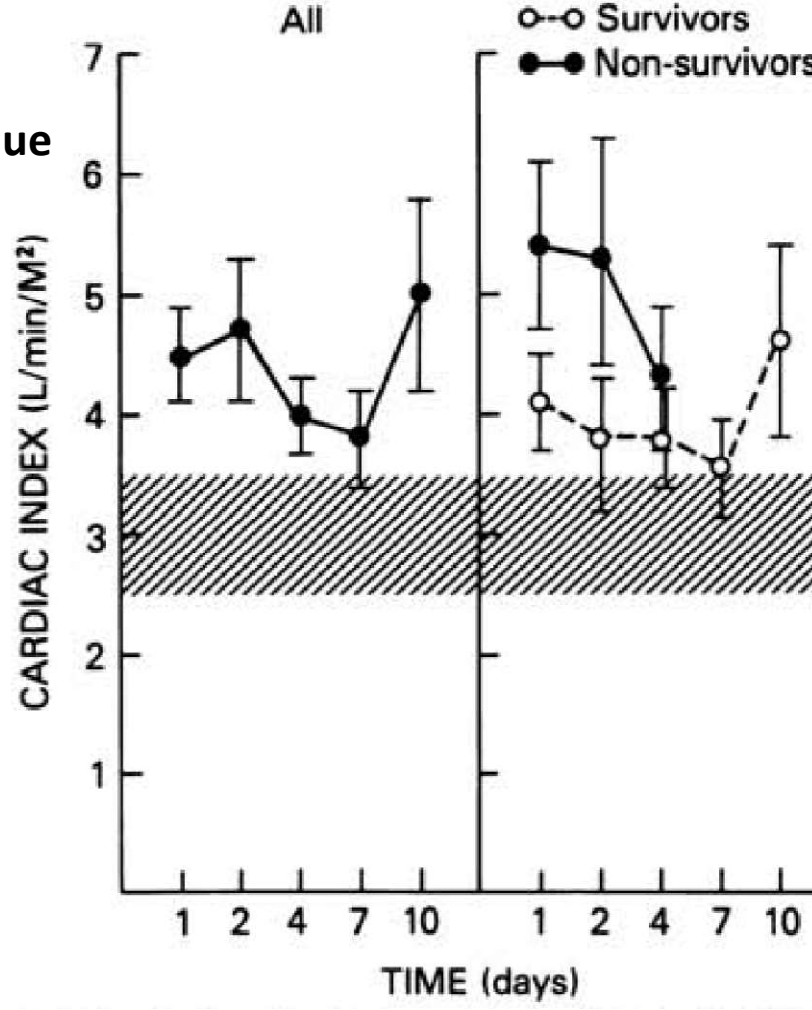
- Le tableau hémodynamique au cours du choc septique après expansion volémique :
  - état hyperdynamique
  - effondrement des résistances vasculaires périphériques
  - un débit cardiaque normal ou élevé
  - une pression artérielle abaissée

dysfonctionnement myocardique intrinsèque dès la phas aiguë

n d’éjection



Index cardiaque



Parker MM, *et al.* Profound but reversible myocardial depression in patients with se shock. *Ann Intern Med* 1984 ; 100 : 483-

cal Commentary

odynamic Instability in Sepsis
e Assessment by Doppler Echocardiography

eillard-Baron, Sebastien Prin, Karim Chergui, Olivier Dubourg, and François Jardin

sive Care Unit and the Department of Cardiology, University Hospital Ambroise Paré, Assistance Publique Hôpitaux de Paris, lex, France

n J Respir Crit Care Med 2003; 68 :1270-6

64/183 pts: 35%
FEVG: **38±17%**

Intensive Care Med (2008) 34:250–256
DOI 10.1007/s00134-007-0929-z

ORIGINAL

Caroline Etchecopar-Chevreuil
Bruno François
Marc Clavel
Nicolas Pichon
Hervé Gastinne
Philippe Vignon

Cardiac morphological and functional chan during early septic shock: a transesophage echocardiographic study

Intensive Care Med 2008; 34 :250-6

16/35 pts: 46%
FEVG: **36±14%**

Prévalence entre 30 et 60%

al incidence of global left ventricular kinesia in adult septic shock

Baron, Antoine MD; Caille, Vincent MD; Charron, Cyril MD; Belliard, Guillaume MD; Page, MD; Jardin, François MD

Crit Care Med 2008; 36 :1701-6

40/67 pts: 60%
FEVG: **31±8%**

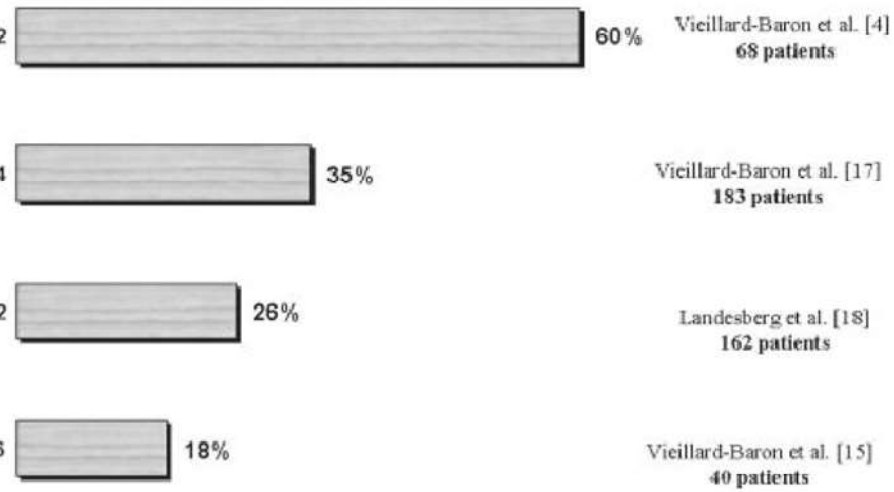
Initial resuscitation guided by the Surviving Sepsis Campaign recommendations and early echocardiographic assessment of hemodynamics in intensive care unit septic patients: A pilot s

Koceila Bouferrache, MD; Jean-Bernard Amiel, MD; Loïc Chimot, MD; Vincent Caille, MD; Cyril Charr Philippe Vignon, MD, PhD: Antoine Vieillard-Baron, MD, PhD

Crit Care Med 2012; 40 :2821-7

14/46 pts: 30%
FEVG: **27±8%**

FE < 40%



gure 1. Incidence of left ventricular systolic dysfunction cording to the time of the evaluation. H6, H12, H24, H72, the ne (hours) between admission and echocardiographic evaluation 15,17,18].

ssé X, et al.: Evaluation of left ventricular systolic function ed in septic shock. Critical Care 2013, 17:164.

Actual incidence of global left ventricular hypokinesia in adult septic shock

Antoine Vieillard-Baron, MD; Vincent Caille, MD; Cyril Charron, MD; Guillaume Belliard, MD; Bernard Page, MD; François Jardin, MD

Table 2. Hemodynamic data obtained in nonhypokinetic, primary, and secondary hypokinetic pat and at the time of vasopressors weaning

n = 67	1 Non-hypo. (n = 27)	2 Primary Hypo. (n = 26)	3 Secondary Hypo. (n = 14)	4 Wea (n =
SAP (mm Hg)	93 ± 23	97 ± 22	110 ± 21	124 ±
HR (beats/min)	106 ± 21	108 ± 26	112 ± 24	88 ±
CI (L/min/m²)	3.6 ± 1.5	2.6 ± 0.9[a]	2.1 ± 0.8[a]	3.3 ±
LVEF (%)	65 ± 9	31 ± 9[a]	31 ± 8[a]	57 ±
LVEDV (mL/m²)	68 ± 24	76 ± 24	61 ± 15	75 ±
SAP/LVESV	4.8 ± 2	2.1 ± 0.8[a]	2.9 ± 1.1[a]	4.4 ±

Primary 39% (J1)

Secondary 21% (J2-J3)

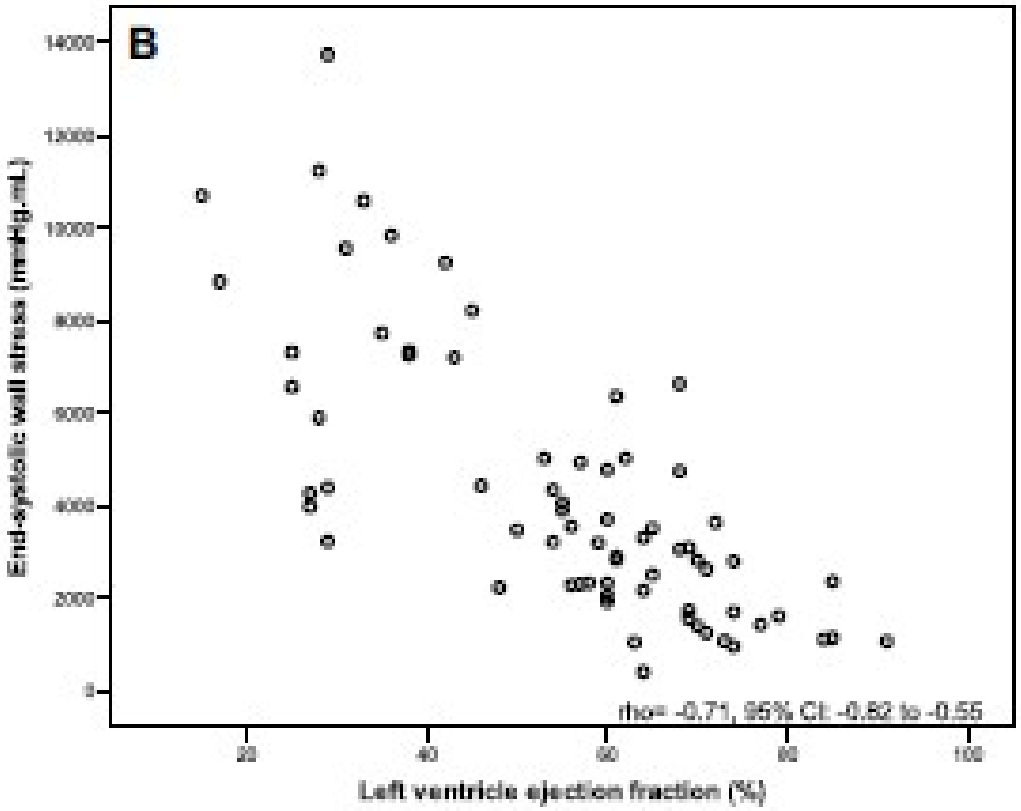
Diagnostic entre J1 et J3 du début du CS

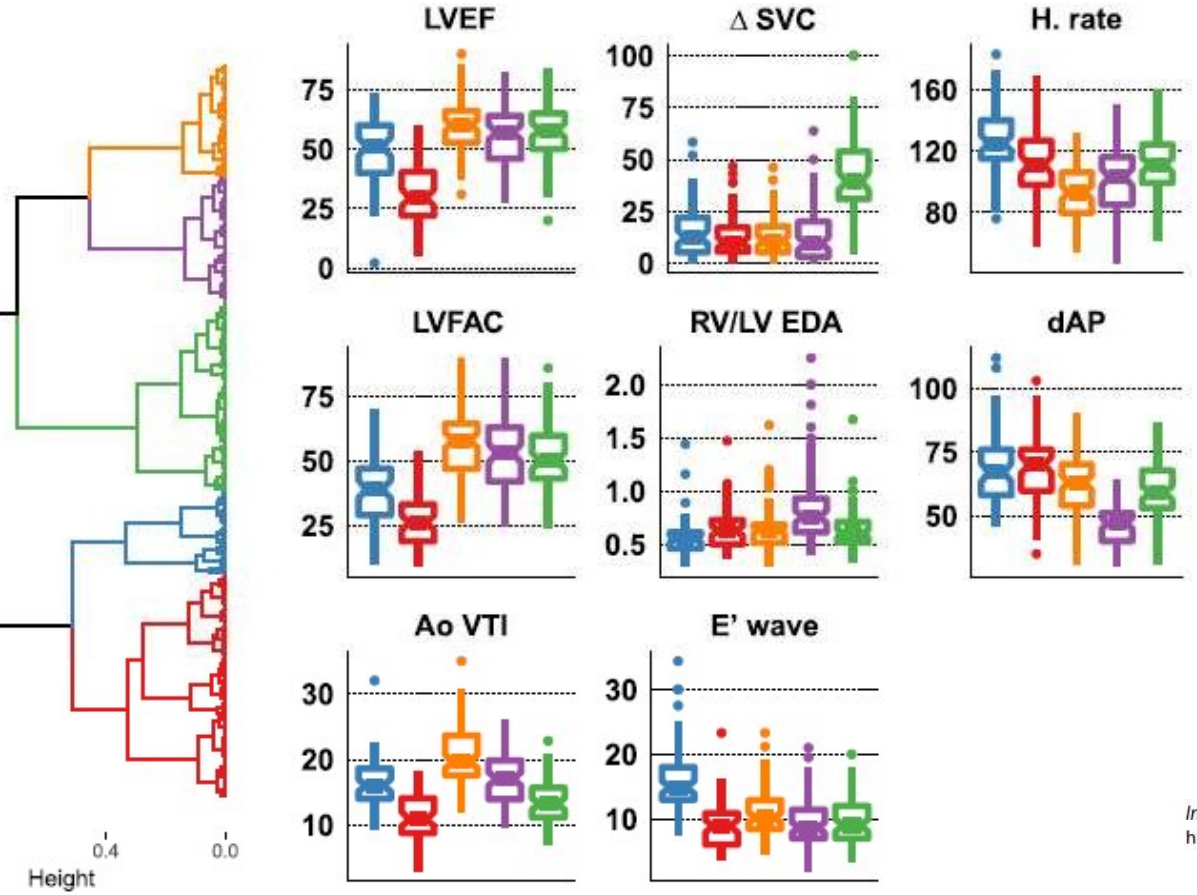
sive Care Med (2017) 43:633–642
10.1007/s00134-017-4698-z

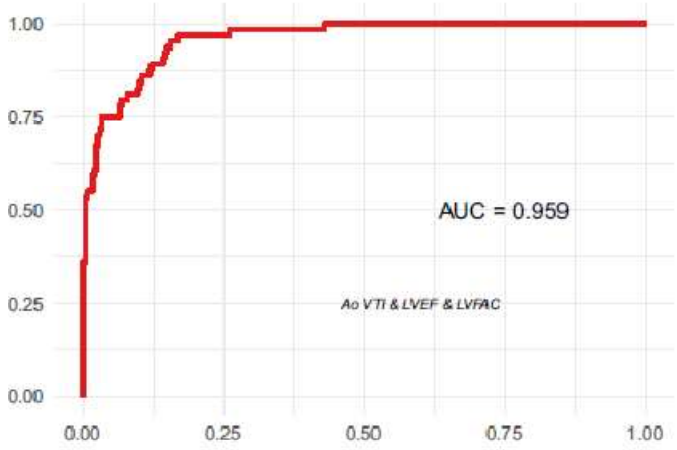
RIGINAL

eft ventricular systolic dysfunction uring septic shock: the role of loading onditions

ence Boissier[1,2,4,5], Keyvan Razazi[1,2], Aurélien Seemann[1,3], Alexandre Bedet[1,2], Arnaud W. Thille[1,4,5], olas de Prost[1,2], Pascal Lim[3], Christian Brun-Buisson[1,2] and Armand Mekontso Dessap[1,2,6*]







LVEF <40% & Ao VTI <14cm

LV failure	
n	35
Sensibility	54.
Specificity	97.
Pos. pred. value	83.
Neg. pred. value	90.

Cluster
LV failure
Still hypovolemic
Hyperkinetic
Well–resus.
RV failure

FE<40% et ITV sous-aortique < 14cm

(Et pas de précharge-dépendance)

Intensive Care Med (2019) 45:657–667
https://doi.org/10.1007/s00134-019-05596-z

ORIGINAL

Cardiovascular clusters in septic shock combining clinical and echocardiographic parameters: a post hoc analysis

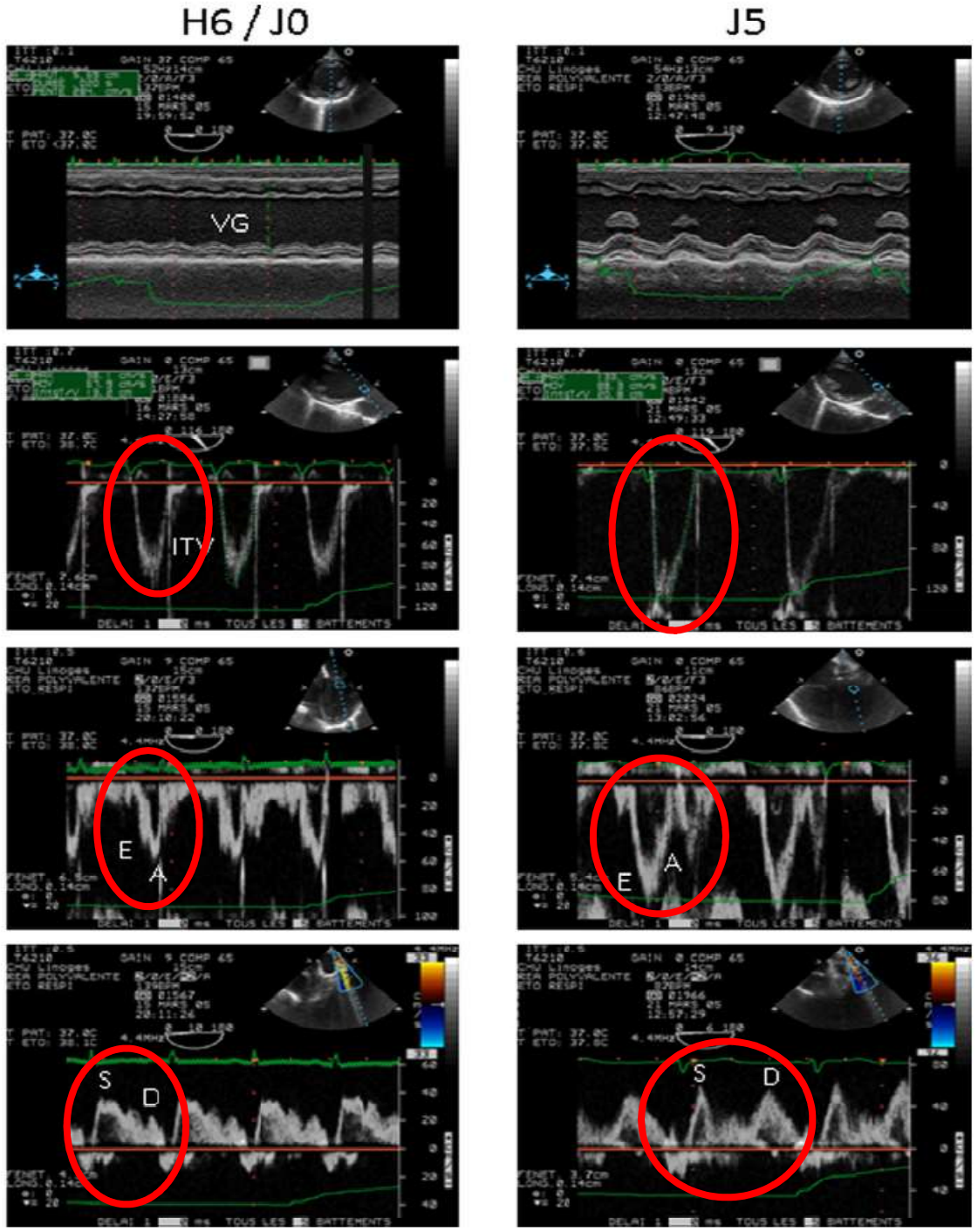
Guillaume Geri[1,2,3], Philippe Vignon[4,5,6], Alix Aubry[1,2], Anne-Laure Fedou[4], Cyril Charron[1], Stein Silva, Xavier Repessé[1] and Antoine Vieillard-Baron[1,2,3*]

a la limite de la
tation

VG diminué
<14cm)

ssions de
plissage ne sont
élevées:
E/A inversé

S prédominante
sur l'onde D



Réver

bal longitudinal strain

Technique basée sur la déformation myocardique régionale

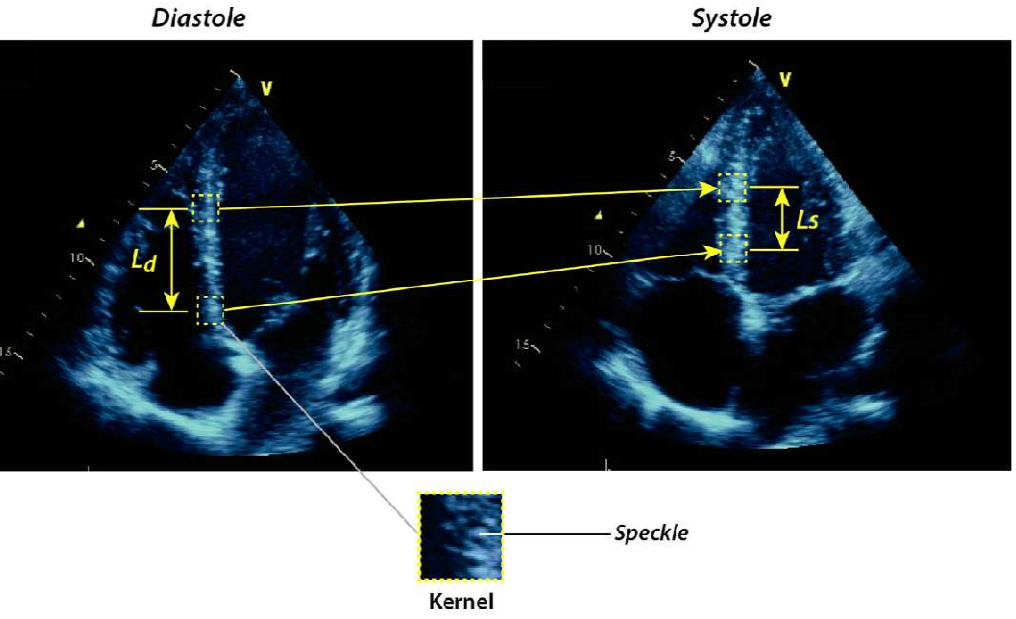
ensive Care Med (2015) 41:1851–1853
DI 10.1007/s00134-015-3962-3

EDITORIAL

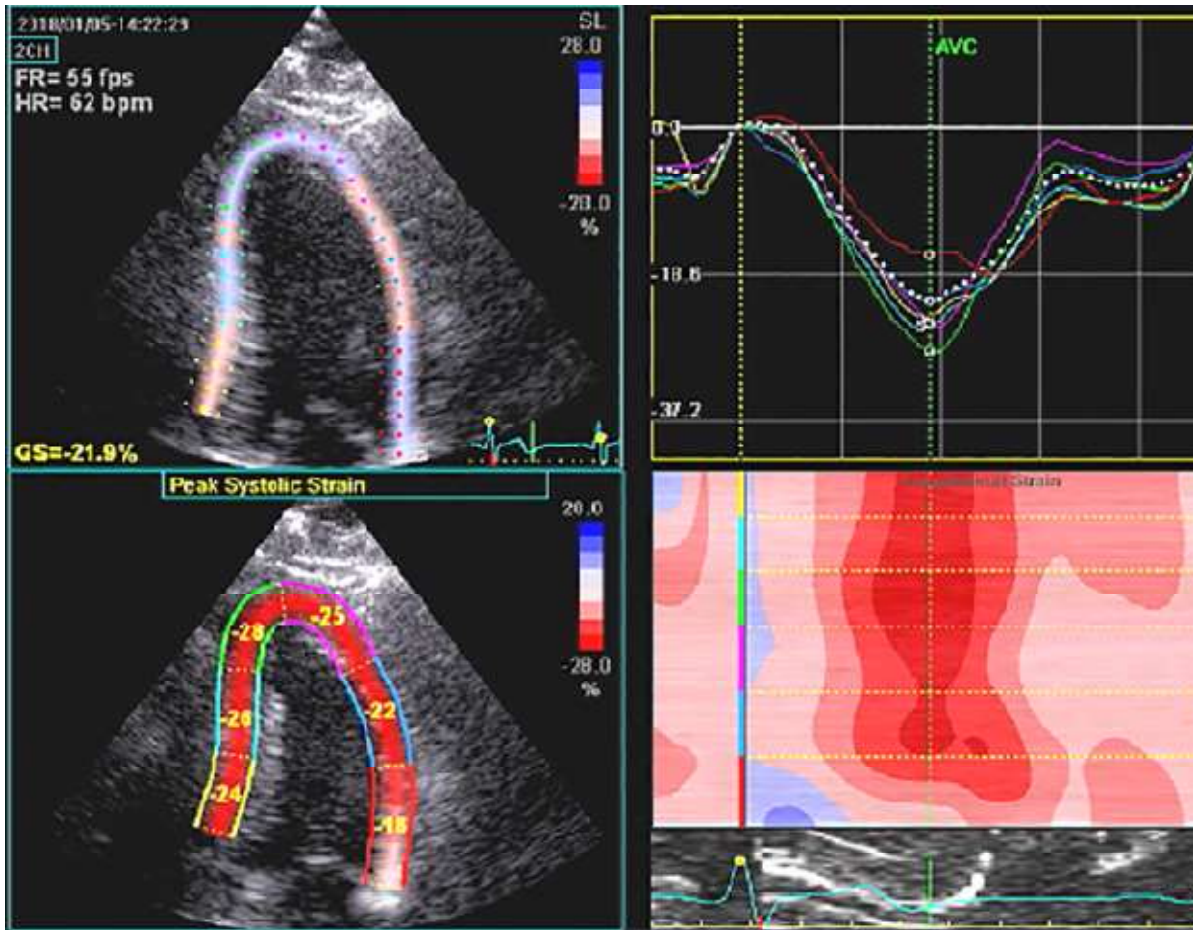
CrossMark

ilippe Vignon
ephen J. Huang

Global longitudinal strain in septic cardiomyopathy: the hidden part of the iceberg?



$$\text{Systolic strain} = {}^{(L_S - L_d)}/_{L_d} X\ 100\%$$



Plus la valeur est négative plus il est normal

bal longitudinal strain

Technique basée sur la déformation myocardique régionale

ensive Care Med (2015) 41:1851–1853
DI 10.1007/s00134-015-3962-3

EDITORIAL

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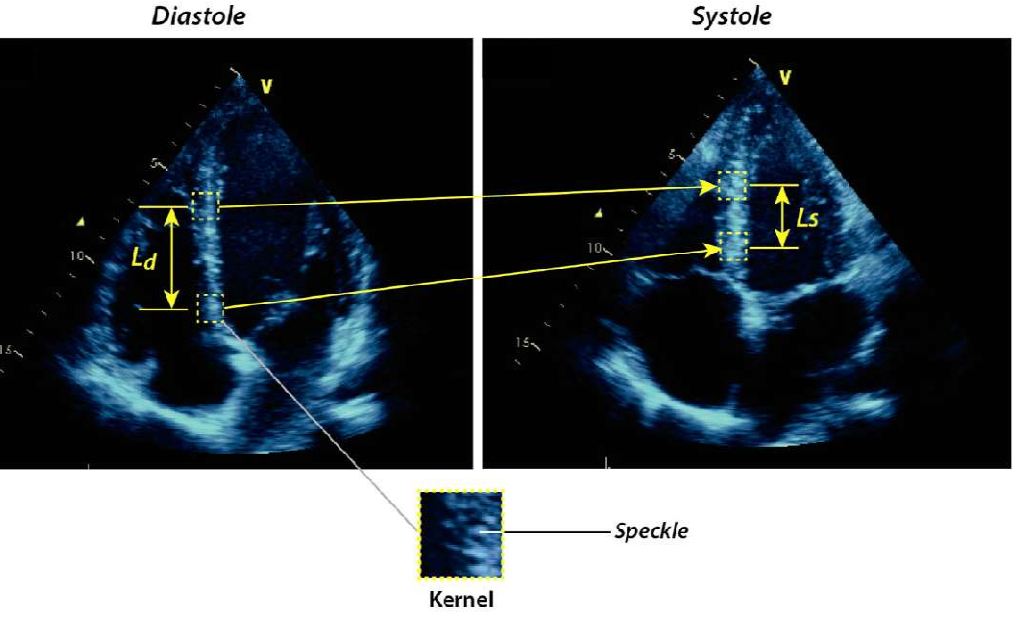
Global longitudinal strain in septic cardiomyopathy: the hidden part of the iceberg?



$$\text{Systolic strain} = (L_s - L_d)/L_d \times 100\%$$

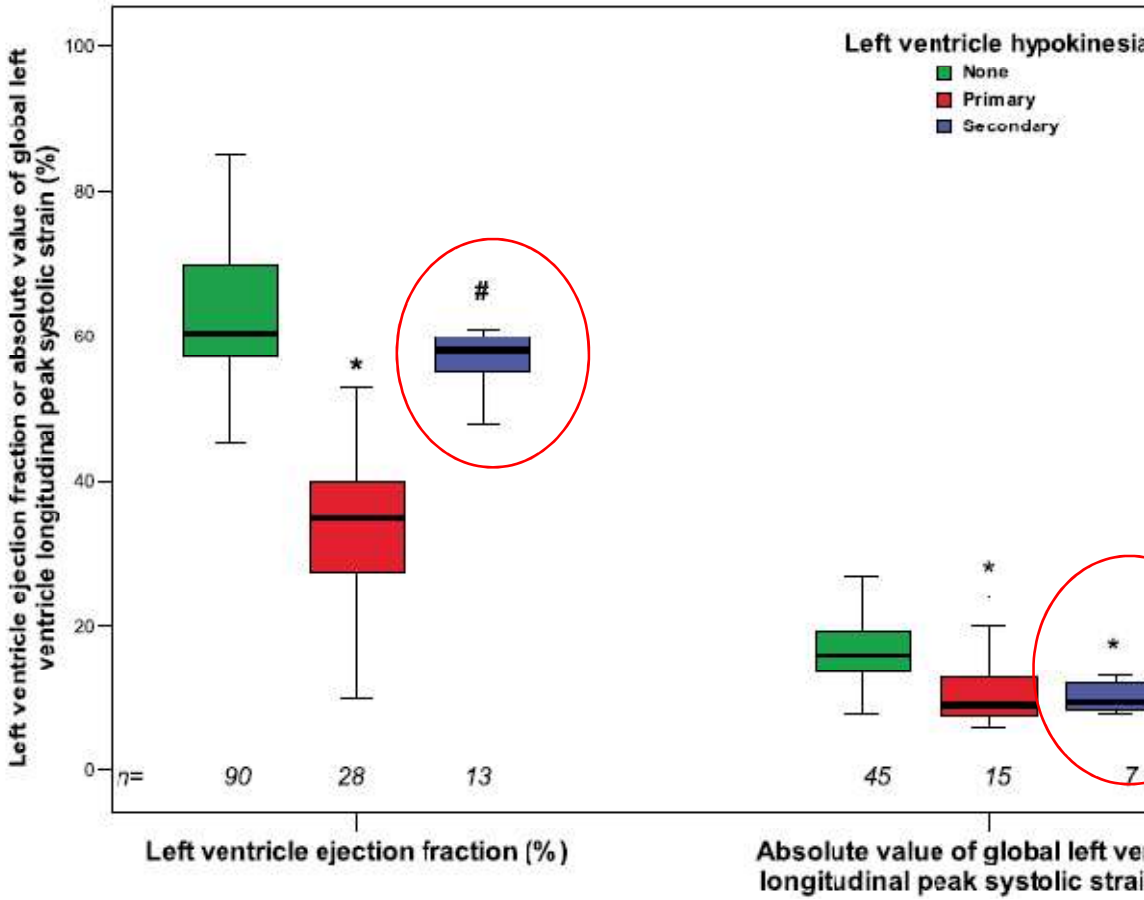
ORIGINAL

CrossMark

Left ventricular systolic dysfunction during septic shock: the role of loading conditions

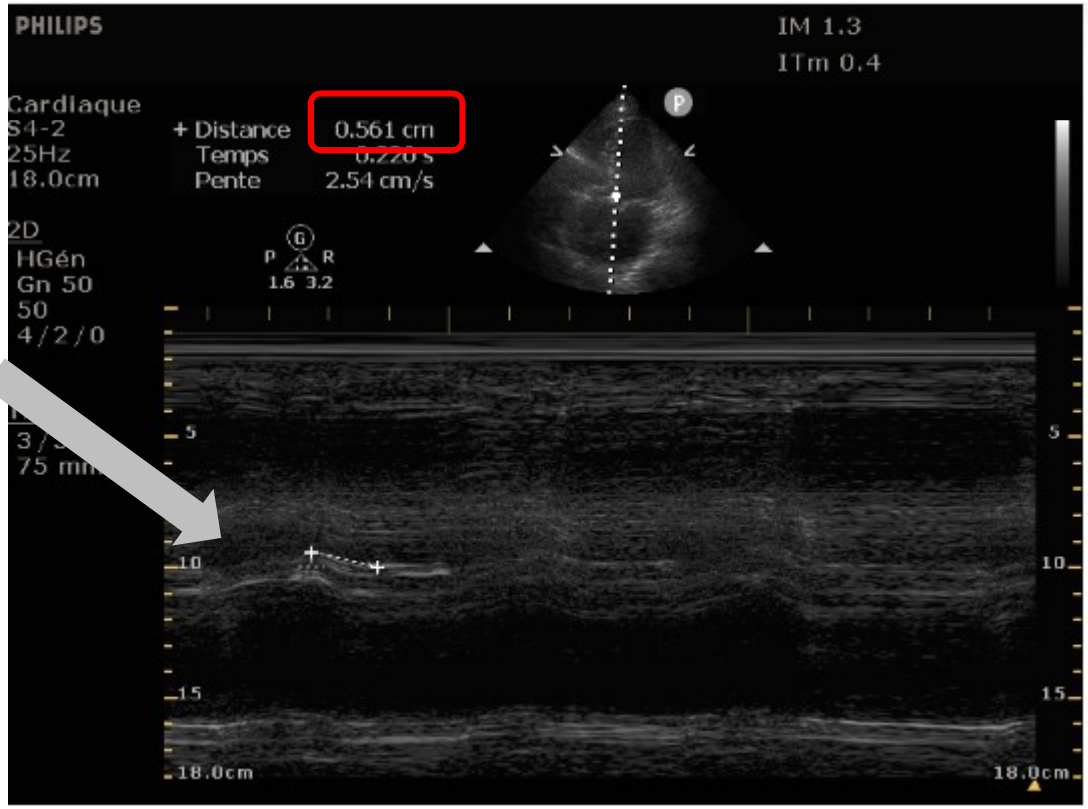
Florence Boissier[1,2,4,5], Keyvan Razazi[1,2], Aurélien Seemann[1,3], Alexandre Bedet[1,2], Arnaud W. Thille[1,4,5], Nicolas de Prost[1,2], Pascal Lim[3], Christian Brun-Buisson[1,2] and Armand Mekontso Dessap[1,2,6*]

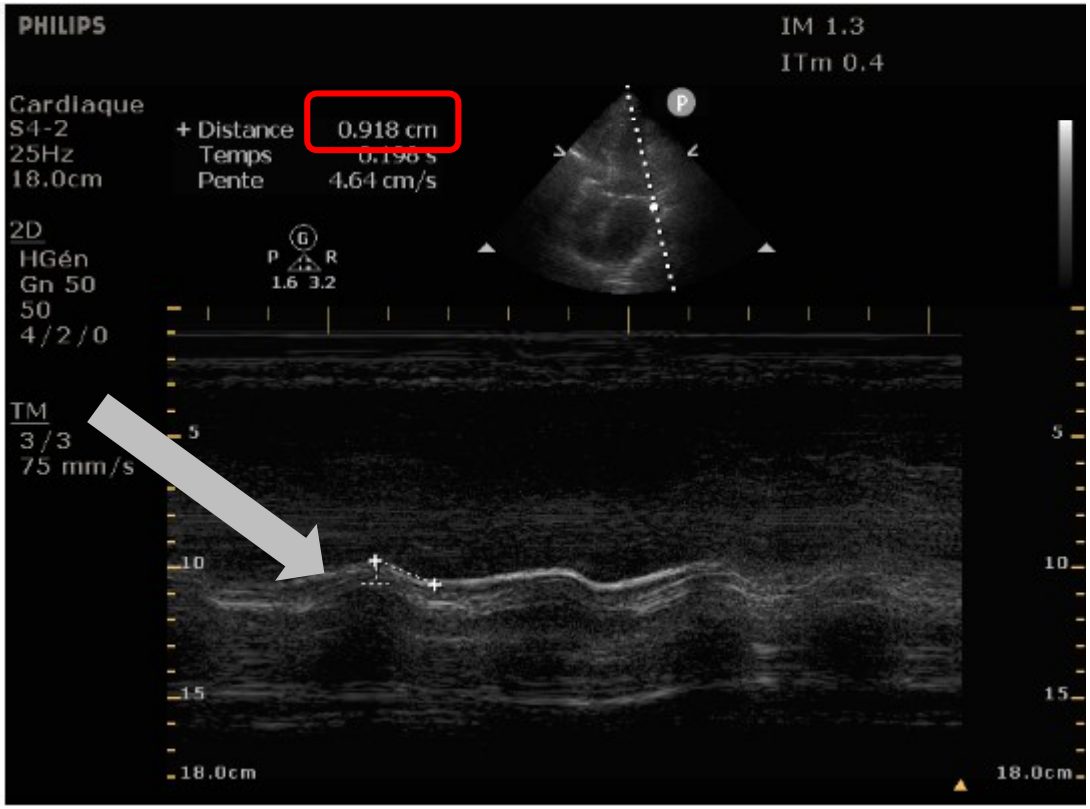
Intensive Care Med 2017 May;43(5):633-642



Mitral annular plane systolic excursion MAPSE

La MAPSE mesure la contraction longitudinale du VG. Elle se prend en coupe 4 cavités mode TM visant l'anneau mitral, la première mesure sur le versant septal, la seconde sur le versant latéral, comme montré sur les deux clichés ci-dessous.





Mitral annular plane systolic excursion MAPSE

BJA Open

BJA Open, 7 (C): 100220 (2023)

doi: 10.1016/j.bjao.2023.100220
Original Research Article

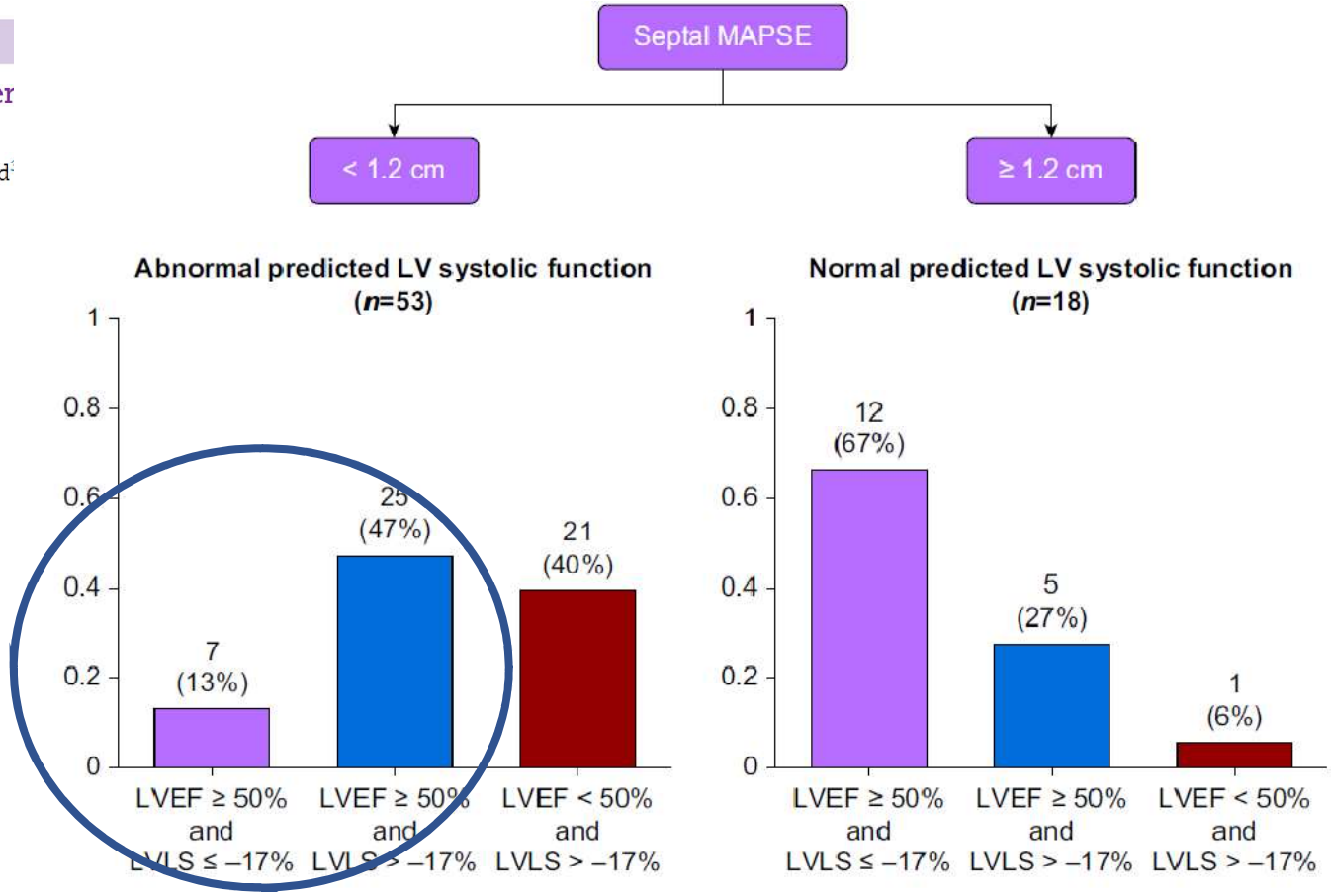
ORIGINAL RESEARCH ARTICLE

Mitral annular plane systolic excursion for assessing left ver systolic dysfunction in patients with septic shock

Clément Brault[1,*], Yoann Zerbib[1], Pablo Mercado[1,2], Momar Diouf[3], Audrey Michaud[1] Christophe Tribouilloy[1], Julien Maizel[1] and Michel Slama[1]

BJA Open, 7 (C): 100220 (2023)

60% des patients avec FE nle



néré le 2 oct. 2023 19:11

édecine sciences

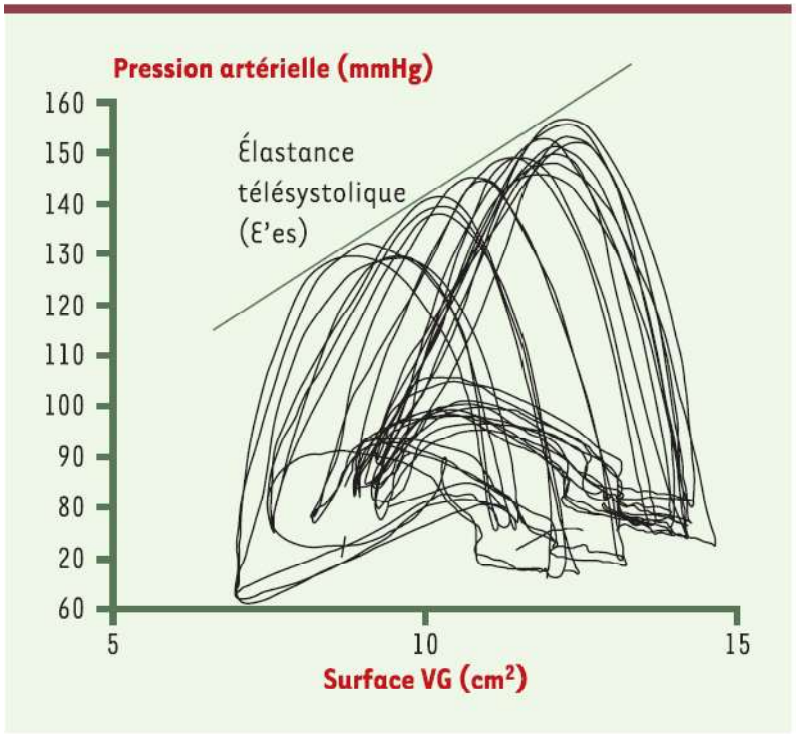
nctionnement myocardique et choc septique
rdial depression in sepsis

en Gibot, Bruno Lévy, Rémi Nevière, Alain Cariou et Olivier Lesur

Indices indépendants des conditions de charge

- Élastance maximale (Emax)

le volume télésystolique ventriculaire gauche peut être apprécié par la surface télésystolique en échographie et la pression ventriculaire gauche par la pression aortique centrale ou fémorale



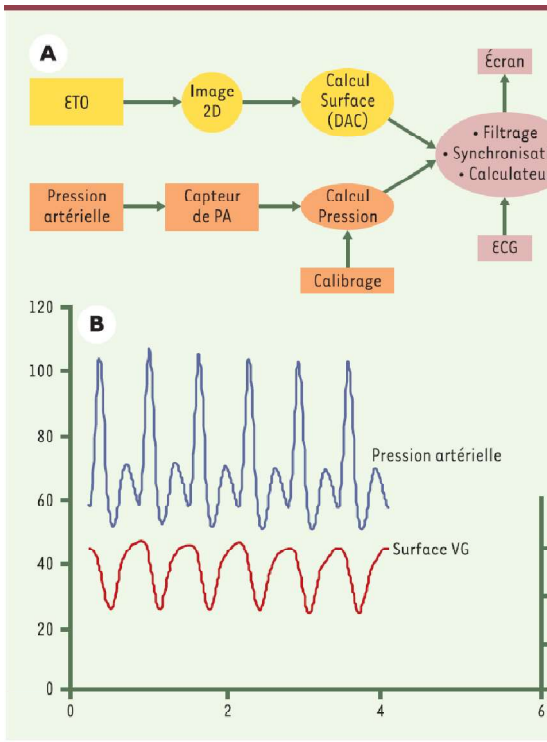
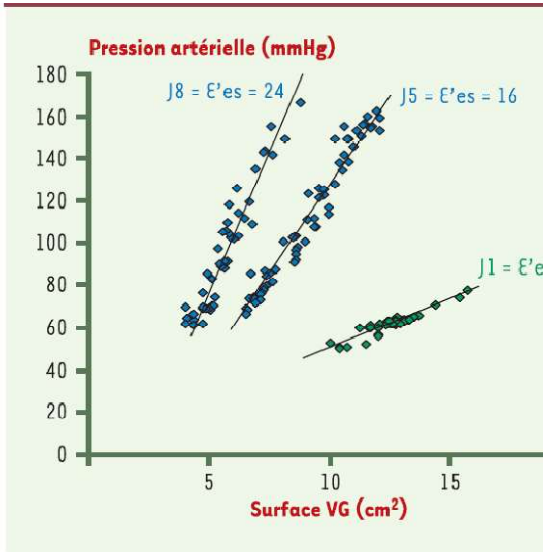


Figure 1. Reconstruction *off-line* des boucles pression-surface du ven
gauche. Deux signaux doivent être enregistrés simultanément *(A)* : la pr



Appellation et définitions ?

Mécanismes physiopathologiques ?

Difficultés diagnostiques?

Thérapeutiques ?

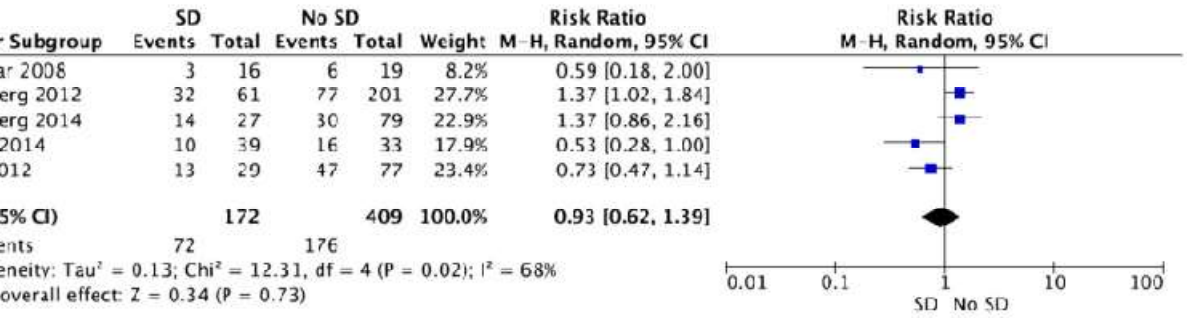
Pronostic ?

Intensive Care Med (2015) 41:1004–1013
DOI 10.1007/s00134-015-3748-7

SYSTEMATIC REVIEW

Filippo Sanfilippo
Carlos Corredor
Nick Fletcher
Giora Landesberg
Umberto Benedetto
Pierre Foex
Maurizio Cecconi

Diastolic dysfunction and mortality in septic patients: a systematic review and meta-analysis



fect of systolic dysfunction vs no systolic dysfunction
n mortality at longest follow-up in septic patients

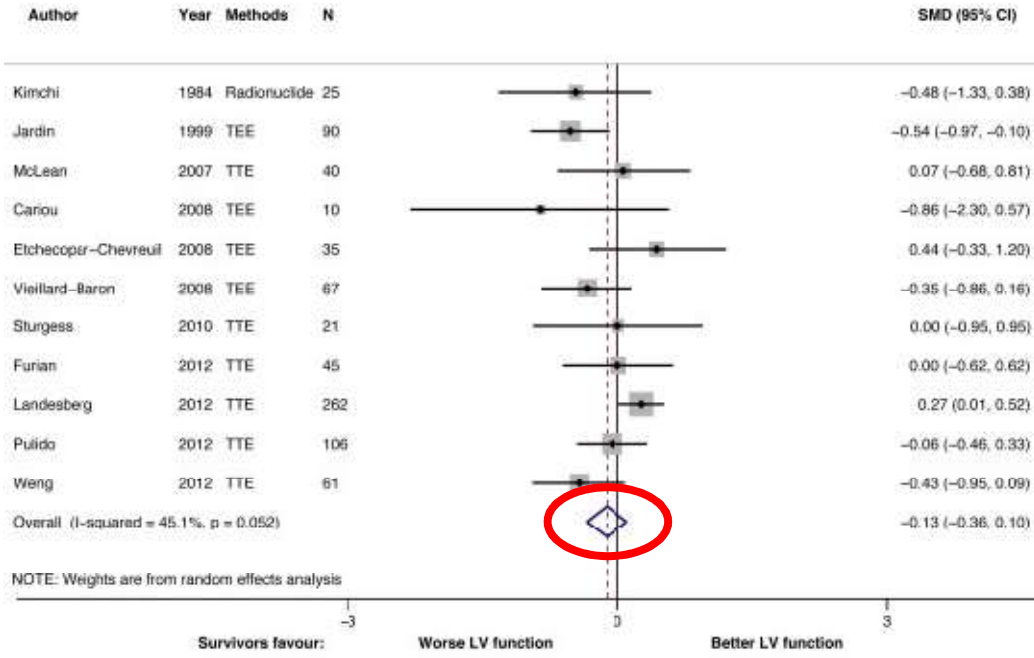
Huang et al. Critical Care 2013, 17:R96
http://ccforum.com/content/17/3/R96

CRITICAL CARE

RESEARCH Open Access

Is early ventricular dysfunction or dilatation associated with lower mortality rate in adult severe sepsis and septic shock? A meta-analysis

Stephen J Huang*, Marek Nalos and Anthony S McLean



nals of Intensive Care (2022) 12:112
org/10.1186/s13613-022-01089-3

Annals of Intensive Care

ARCH Open Access

ociation of sepsis-induced diomyopathy and mortality: a systematic ew and meta-analysis

Lin[1†], Mei-Chuan Lee[2,3†], Han Siong Toh[4,5,6], Wei-Ting Chang[1,5,7], Sih-Yao Chen[1], Fang-Hsiu Kuo[1], Tang[8], Yi-Ming Hua[2], Dongmei Wei[9], Jesus Melgarejo[9], Zhen-Yu Zhang[9] and Chia-Te Liao[1,3,5*]

Study or Subgroup	With SIC Events	With SIC Total	Without SIC Events	Without SIC Total	Weight	Risk Ratio M-H, Random, 95% CI
Chayakul 2020	9	24	6	51	6.5%	3.19 [1.28, 7.93]
Jeong 2018	10	25	62	273	11.6%	1.76 [1.04, 2.99]
Lahham 2020	0	8	3	16	1.0%	0.27 [0.02, 4.67]
Narváez 2017	4	13	5	44	4.7%	2.71 [0.85, 8.64]
Sato 2016	9	29	23	181	9.5%	2.44 [1.26, 4.74]
Shin 2020	13	36	90	330	12.7%	1.32 [0.83, 2.12]
Song 2020	18	49	95	259	13.9%	1.00 [0.67, 1.50]
Vallabhajosyula 2016	8	17	21	41	10.7%	0.92 [0.51, 1.65]
Vallabhajosyula 2017	49	214	39	174	14.5%	1.02 [0.71, 1.48]
Vallabhajosyula 2018	42	206	61	228	15.0%	0.76 [0.54, 1.08]
Total (95% CI)		**621**		**1597**	**100.0%**	**1.28 [0.96, 1.71]**
Total events	162		405			

Heterogeneity: $Tau^2 = 0.11$; $Chi^2 = 22.72$, $df = 9$ ($P = 0.007$); $I^2 = 60\%$
Test for overall effect: $Z = 1.70$ ($P = 0.09$)

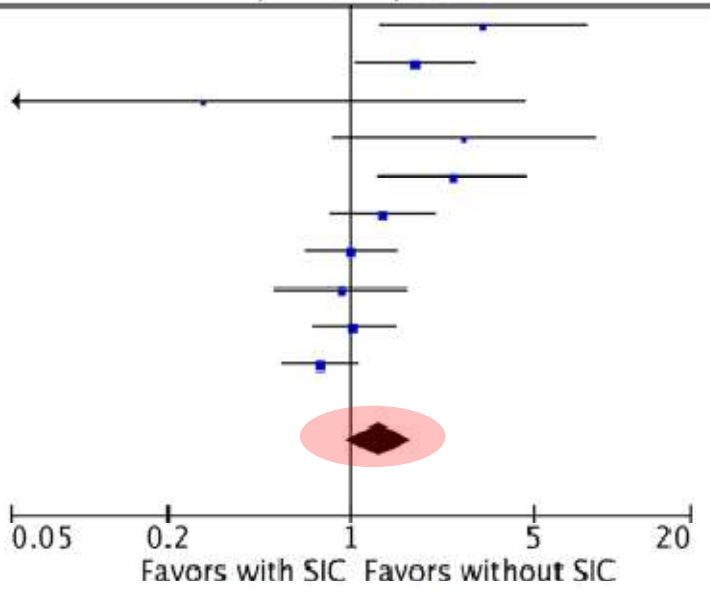


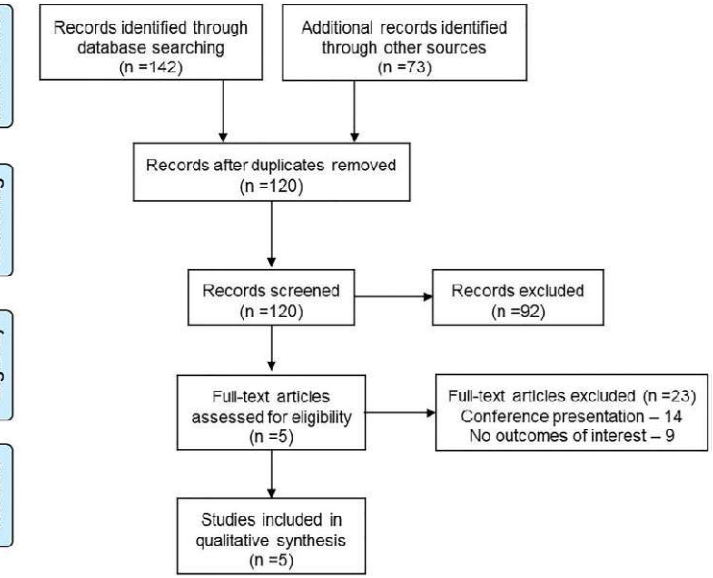
Fig. 2 The forest plot shows in-hospital mortality between septic patients with and without sepsis-induced cardiomyopathy (SIC). SIC is non-statistically associated with higher risk of in-hospital mortality among septic patients

al Longitudinal Strain Using kle-Tracking Echocardiography Mortality Predictor in Sepsis: stematic Review

Journal of Intensive Care Medicine
2019, Vol. 34(2) 87-93

DOI: 10.1177/0885066618761750
journals.sagepub.com/home/jic
SAGE

andra Vallabhajosyula, MBBS, FACP[1,2], Hamza A. Rayes, MBBCh[2],
akhuja, MBBS, FACP, FASN[2], Mohammad Hassan Murad, MD, MPH[3,4],
B. Geske, MD, FACC[1], and Jacob C. Jentzer, MD, FACC[1,2]



Conclusions

This systematic review examined the role of STE in predicting mortality in patients with sepsis. Overall, the quality of evidence and strength of the conclusion are low due to heterogeneity in study populations, GLS technologies, cutoffs, and timing of STE. Optimal STE GLS cutoffs and the clinical

Sanfilippo et al. Critical Care (2018) 22:183
https://doi.org/10.1186/s13054-018-2113-y

Critical Care

RESEARCH Open Access

Left ventricular systolic function evaluated by strain echocardiography and relationship with mortality in patients with severe sepsis or septic shock: a systematic review and meta-analysis

F. Sanfilippo[1*], C. Corredor[2], N. Fletcher[3], L. Tritapepe[4], F. L. Lorini[5], A. Arcadipane[1], A. Vieillard-Baron[6,7] and M. Cecconi[8,9]

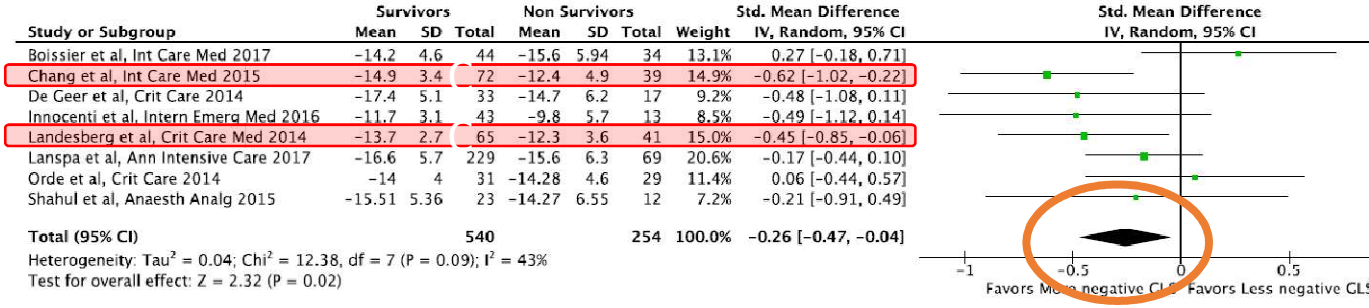


Fig. 3 Comparison of global longitudinal strain (GLS) values between survivors and non-survivors among patients with severe sepsis and septic shock

Conclusions

Worse values of global longitudinal strain are associated with higher mortality in patients with severe sepsis or septic shock, while such an association is not valid for left ventricular ejection fraction. More research is warranted to elucidate such an association, which could be related to the ability of speckle-tracking echocardiography in demonstrating underlying intrinsic myocardial disease as opposed to left ventricular ejection fraction.

Appellation et définitions ?

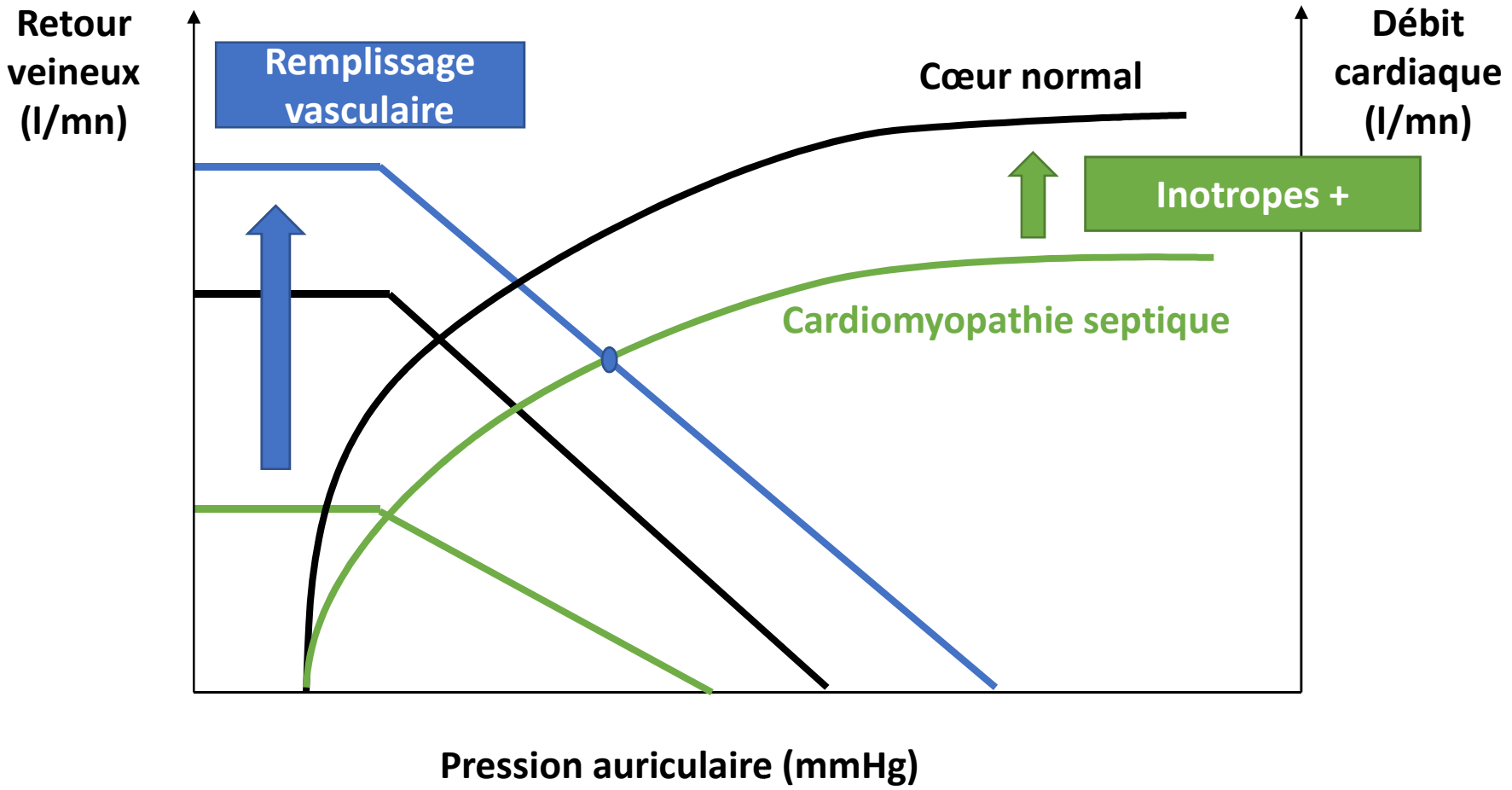
Mécanismes physiopathologiques ?

Difficultés diagnostiques?

Thérapeutiques ?

Pronostic ?

Thérapeutiques



esuscitation guided by the Surviving Sepsis Campaign
endations and early echocardiographic assessment of
namics in intensive care unit septic patients: A pilot study*

errache, MD; Jean-Bernard Amiel, MD; Loïc Chimot, MD; Vincent Caille, MD; Cyril Charron, MD;
on, MD, PhD: Antoine Vieillard-Baron, MD, PhD

Crit Care Med 2012; 40:2821–2827

	LVFAC <45% (n = 14)	
	T3	T4
rate (bpm)	119 (110–135)*	102 (94–113)*
ic blood pressure (mm Hg)	100 (86–113)	128 (118–141)*
olic blood pressure (mm Hg)	58 (48–69)	58 (51–73)
arterial pressure (mm Hg)	66 (57–76)	85 (70–91)*
al venous pressure (mm Hg)	15 (11–19)*	9 (6–12)*
al venous oxygen saturation (%)	75 (73–80)	76 (70–79)
C (%)	24 (18–37)*	48 (39–61)*
ior vena cava collapsibility index (%)	10 (0–16)a	12 (6–16)
ac index (L/min/m²)	1.7 (1.4–2.1)*	2.4 (2.1–2.9)*
e (mmol/L)	4.8 (3.6–5.8)	5.0 (3.1–6.5)
deficit (mmol/L)	8 (4.3–11.9)	7.0 (4.5–9.0)
ulative volume expansion (mL)	1921 (1563–2239)	2125 (1750–2645)
inephrine, n	11	10
/min	0.543 (0.40–0.876)*	0.387 (0.28–0.54)
amine, n	1	8
/min	5	6.3 (5–7.5)
phrine, n	2	6
/min	0.340 (0.33–0.34)	0.475 (0.32–0.55)

Actual incidence of global left ventricular hypokinesia in adult septic shock

Antoine Vieillard-Baron, MD; Vincent Caille, MD; Cyril Charron, MD; Guillaume Belliard, MD;
Bernard Page, MD; François Jardin, MD

	Before Dobutamine (n = 30)	After 24 hrs Dobutamine (n = 30)
SAP (mmHg)	96 ± 22	108 ± 15
HR (beats/min)	107 ± 24	100 ± 22
CI (L/min/m²)	2.3 ± 1	2.9 ± .9
LVEF (%)	32 ± 9	49 ± 13[a]
LVEDV (mL/m²)	69 ± 23	72 ± 19
SAP/LVESV	2.5 ± 1.1	4.4 ± 2.5[a]

re Med (2021) 47:1181–1247
org/10.1007/s00134-021-06506-y

ELINES

viving sepsis campaign: international
delines for management of sepsis and septic
ck 2021

Check for updates

pes

mendations

adults with septic shock and cardiac dysfunction with persistent
perfusion despite adequate volume status and arterial blood
ure, we **suggest** either adding dobutamine to norepinephrine or
epinephrine alone
ecommendation, low quality of evidence

Adjunctive DobutAmine in sePtic Cardiomyopathy With Tissue Hypoperfusion (ADAPT

Essai clinique NCT04166331

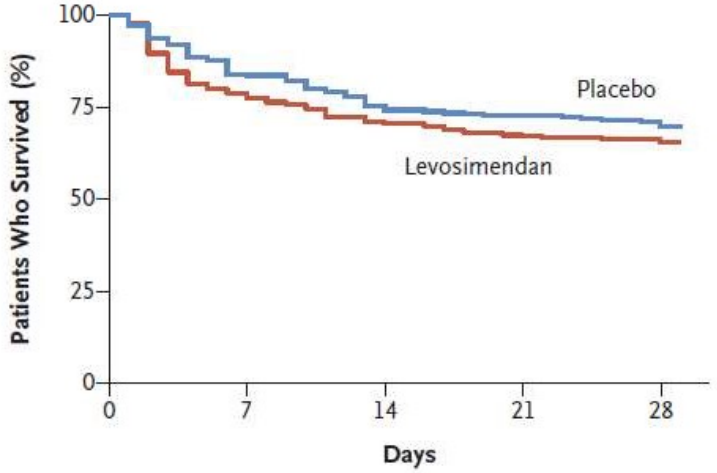
N Engl J Med 2016;375:1638-48.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

evosimendan for the Prevention of Acute Organ Dysfunction in Sepsis

A.C. Gordon, G.D. Perkins, M. Singer, D.F. McAuley, R.M.L. Orme, Santhakumaran, A.J. Mason, M. Cross, F. Al-Beidh, J. Best-Lane, D. Brealey, C.L. Nutt, J.J. McNamee, H. Reschreiter, A. Breen, K.D. Liu, and D. Ashby



o. at Risk					
evosimendan	258	203	183	174	171
acebo	257	216	194	186	182

gure 2. Kaplan–Meier Estimates of the Probability of Survival to Day 28.

e adjusted hazard ratio for death in the levosimendan group, as com-red with the placebo group, was 1.24 (95% CI, 0.91 to 1.67; P=0.17).

Journal of Clinical Anesthesia 39 (2017) 67–72

Contents lists available at ScienceDirect

Journal of Clinical Anesthesia

Original Contribution

Levosimendan does not provide mortality benefit over dobutamine in adult patients with septic shock: A meta-analysis of randomized controlled trials☆

Sulagna Bhattacharjee [a], Kapil D. Soni [b], Souvik Maitra [a,*], Dalim K. Baidya [a]

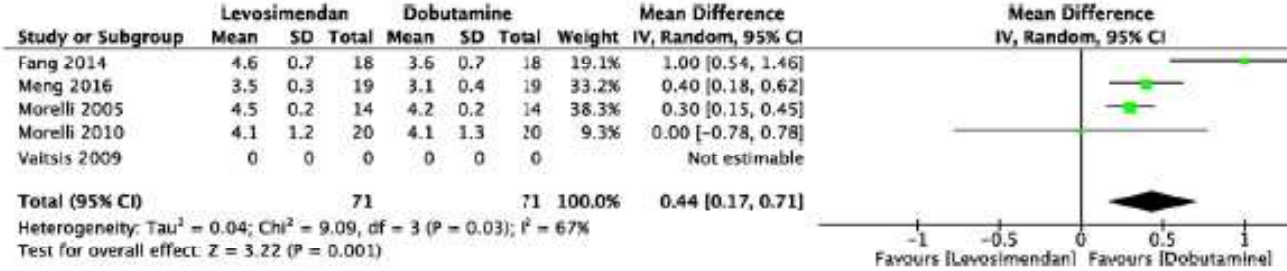


Fig. 5. Cardiac index at 24 h of randomization at individual study level and pooled analysis level.

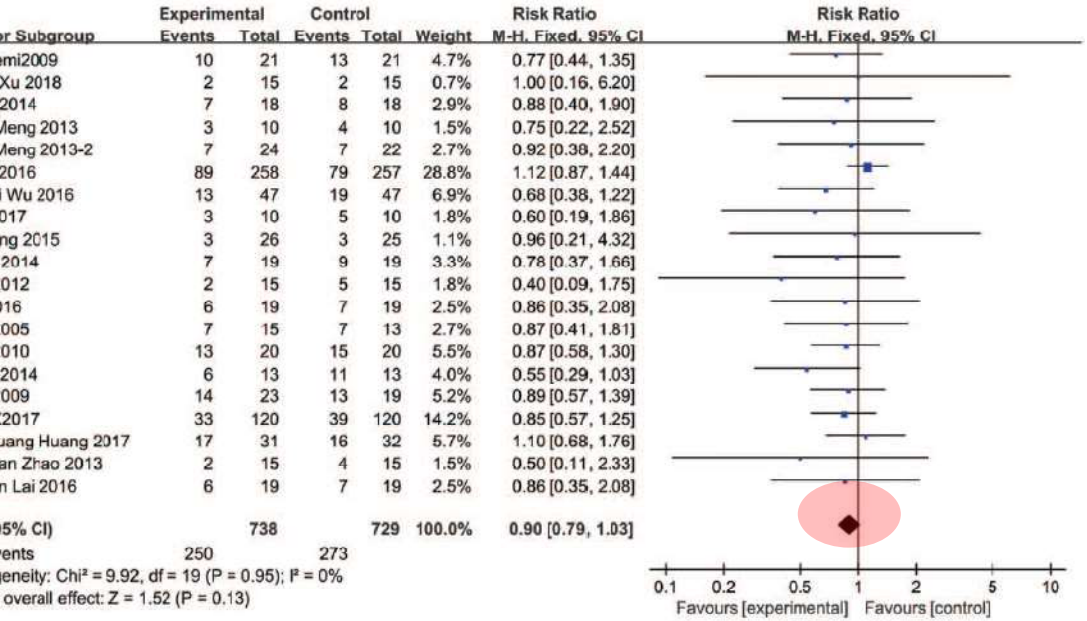
Chinese Medical Journal®

osimendan does not reduce the mortality of critically ill adult ents with sepsis and septic shock: a meta-analysis

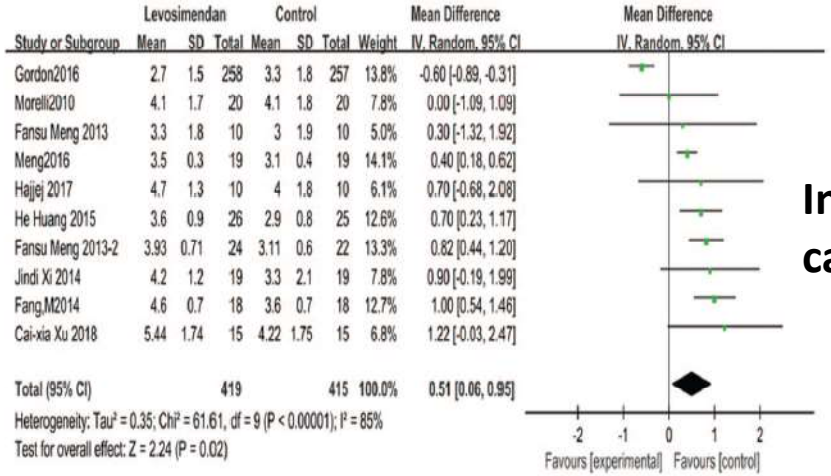
eng, Yu Chen, Min Li, Jiao-Jiao Yuan, Xue-Ni Chang, Chen-Ming Dong

nt of Intensive Care Unit, Lanzhou University Second Hospital, Lanzhou University, Lanzhou, Gansu 730030, China.

Chinese Medical Journal 2019;132(10)

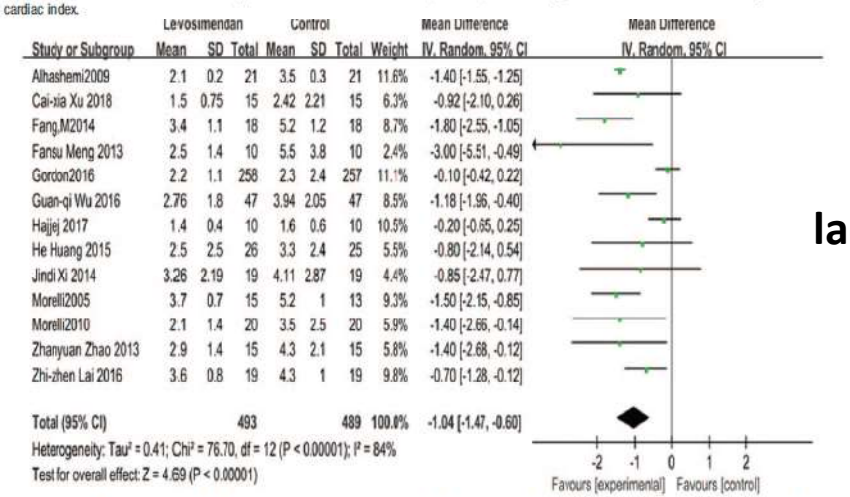


st of mortality. Seven studies reported 28-day mortality, two studies reported 30-day mortality, and others were ICU mortality. The analysis showed that there was no rence in the mortality between the levosimendan and control groups. ICU: Intensive care unit.



Index↗ cardiaq

Figure 4: Forest of cardiac index. The cardiac index was reported in ten of the included studies; however, the measuring methods were varied. Our meta-analysis showed that levosimendan improved the cardiac index.



lactate↘

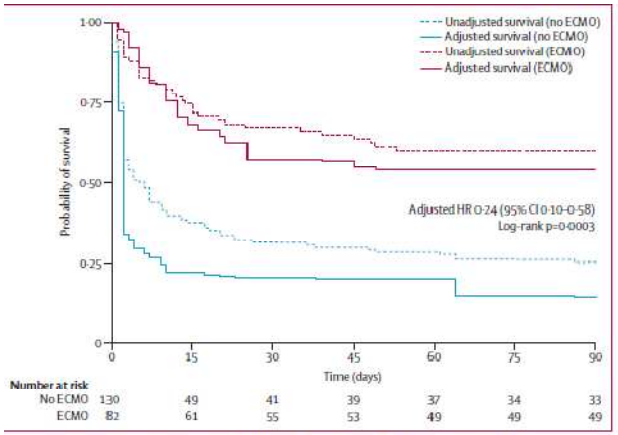
Figure 5: Forest of lactic acid. Thirteen of all the included studies reported the serum lactate, the mean serum lactate level was lower in the levosimendan group, the synthetic analysis showed that the serum lactate level was significantly lower.

MO

Nicolas Bréchot et al Lancet 2020

arterial extracorporeal membrane oxygenation to e sepsis-induced cardiogenic shock: a retrospective, icentre, international cohort study

chot, David Hajage, Antoine Kimmoun, Julien Demiselle, Cara Agerstrand, Santiago Montero, Matthieu Schmidt, uard Luyt, Guillaume Lebreton, Guillaume Hékimian, Erwan Flecher, Elie Zogheib, Bruno Levy, Arthur S Slutsky, Daniel Brodie*, Alain Combes*, for the International ECMO Network



igure 2: Kaplan-Meier survival estimates of patients receiving ECMO and not receiving ECMO
Inadjusted and propensity-weighted for covariables associated with the severity of myocardial dysfunction (cardiac ndex); covariables associated with survival during septic shock (inotrope score, lactataemia, Sequential Organ ailure Assessment (SOFA) score, age, immunocompromised status, and cumulative fluid therapy before inclusion); nd delay between shock onset and inclusion. ECMO=extracorporeal membrane oxygenation. HR=hazard ratio.

	Baseline population			Propensity-weighted patients		
	ECMO (n=82)	Non-ECMO (n=130)	p value	ECMO (n=82)	Non-ECMO (n=130)	ASD
Demographic data						
Age	48 (15)	66 (16)	<0·0001	56 (15)	56 (20)	0·00
Male sex	36 (44%)	93 (72%)	0·0001	39%	57%	0·04
Charlson score	1·1 (1·3)	1·6 (1·2)	0·0013	1·4 (1·3)	1·2 (1·1)	0·14
Immunodeficiency	13 (16%)	34 (26%)	0·0787	16%	16%	0·00
Community-acquired infection	71 (87%)	90 (69%)	0·0022	77%	76%	0·04
Source of infection: lung	64 (78%)	50 (38%)	<0·0001	87%	22%	1·66
Postoperative	7 (9%)	20 (15%)	0·1452	21%	14%	0·15
Delay after shock onset, days‡	1·1 (0·9)	0·7 (1·0)	<0·0001	1·1 (0·9)	1·1 (1·4)	0·00
Haemodynamic condition						
Mean arterial pressure, mm Hg	70 (15)	72 (14)	0·1204	68 (16)	68 (15)	0·04
Heart rate	123 (24)	114 (27)	0·0011	119 (28)	111 (25)	0·32
Arterial pH	7·13 (0·15)	7·23 (0·16)	<0·0001	7·14 (0·14)	7·22 (0·16)	0·51
Serum lactate level, mmol/L	8·9 (4·4)	6·5 (4·5)	0·0001	7·9 (4·0)	7·9 (5·1)	0·00
Inotrope score, µg/kg per min	279 (247)	145 (128)	<0·0001	169 (157)	169 (148)	0·00
Cardiac index, L/min per m²	1·54 (0·54)	2·21 (0·59)	<0·0001	1·77 (0·56)	1·77 (0·67)	0·00
LVEF, %	17·1% (7·3)	27·5% (6·2)§	<0·0001	15·3% (6·3)	26·7% (7·8)	1·63
Fluid therapy before inclusion, mL	4925 (2886)	1654 (1952)	<0·0001	2764 (2113)	2764 (2779)	0·00
General clinical status						
Mechanical ventilation	82 (100%)	124 (95%)	0·0840	100%	100%	0·09
PaO_2:FiO_2	105 (77)	177 (106)	<0·0001	97 (60)	161 (92)	0·08
Acute kidney injury¶	66 (80%)	90 (69%)	0·0810	75%	80%	0·11
SOFA score	16·6 (2·9)	12·7 (3·5)	<0·0001	15·0 (3·1)	15·0 (3·7)	0·00
SAPS-II score	78·3 (16·1)	68·4 (18·8)	0·0001	76·8 (20·7)	75·6 (19·5)	0·06

Interpretation Patients with severe sepsis-induced cardiogenic shock treated with VA-ECMO had a large and significant improvement in survival compared with controls not receiving ECMO. However, despite the careful propensity-weighted analysis, we cannot rule out unmeasured confounders.

Journal of Intensive Medicine 2 (2022) 8–16

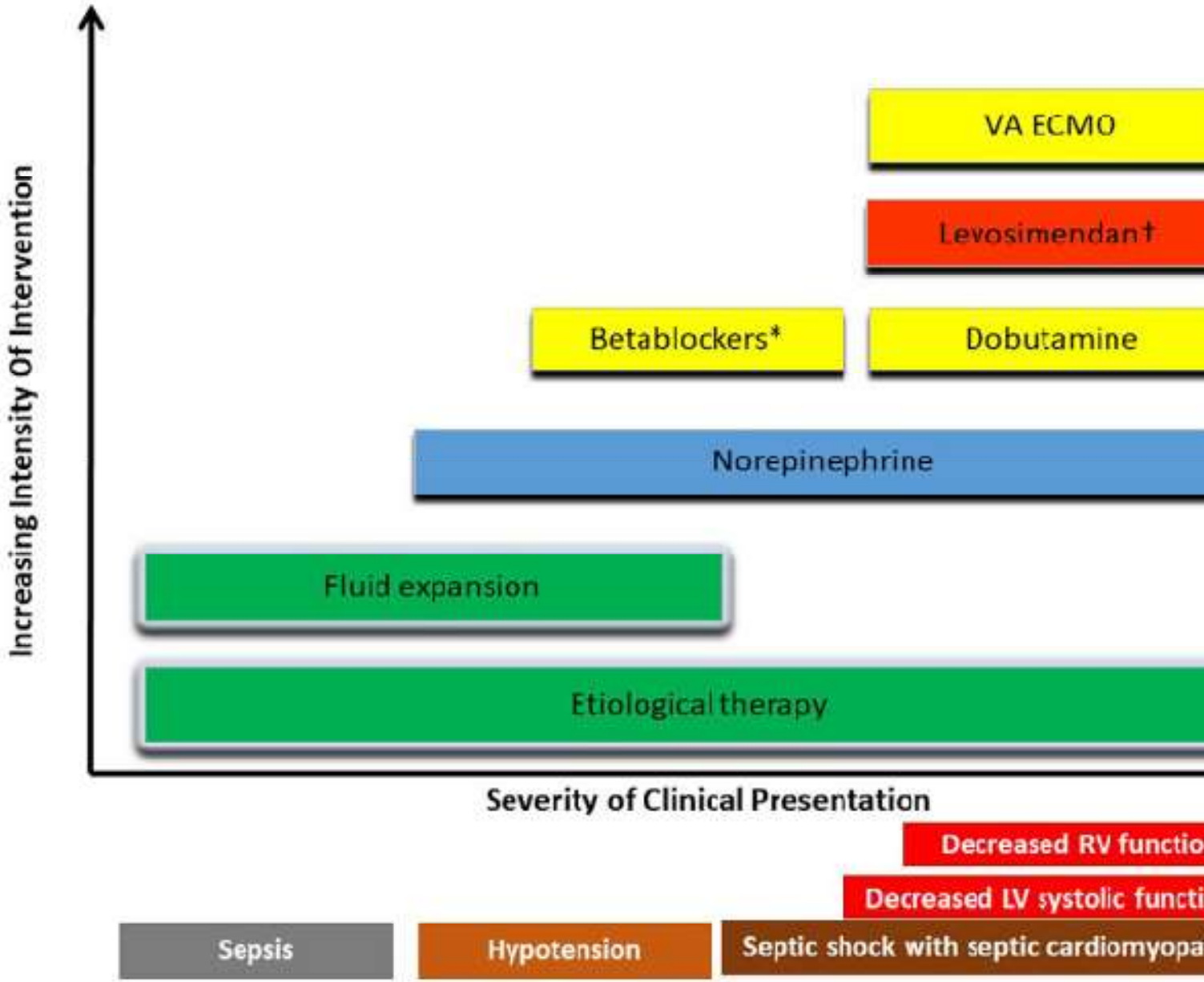
Contents lists available at ScienceDirect

Journal of Intensive Medicine

journal homepage: www.elsevier.com/locate/jointm

omyopathy: Diagnosis and management☆

er[1,2], Nadia Aissaoui[3,4,*]



Conclusion

- Dysfonction systolique du VG induite par le sepsis
- Fréquente, parfois secondaire (J2-J3)
- Réversible même si sévère
- Échocardiographie: Gold standard
- La FE est largement influencée par la postcharge, elle reflète aussi la vasoplégie
- Pronostic non clair!
- Pas de traitement spécifique, le traitement éthologique est la pierre angulaire de l’arsenal thérapeutique