Dysfonction myocardique du sepsis

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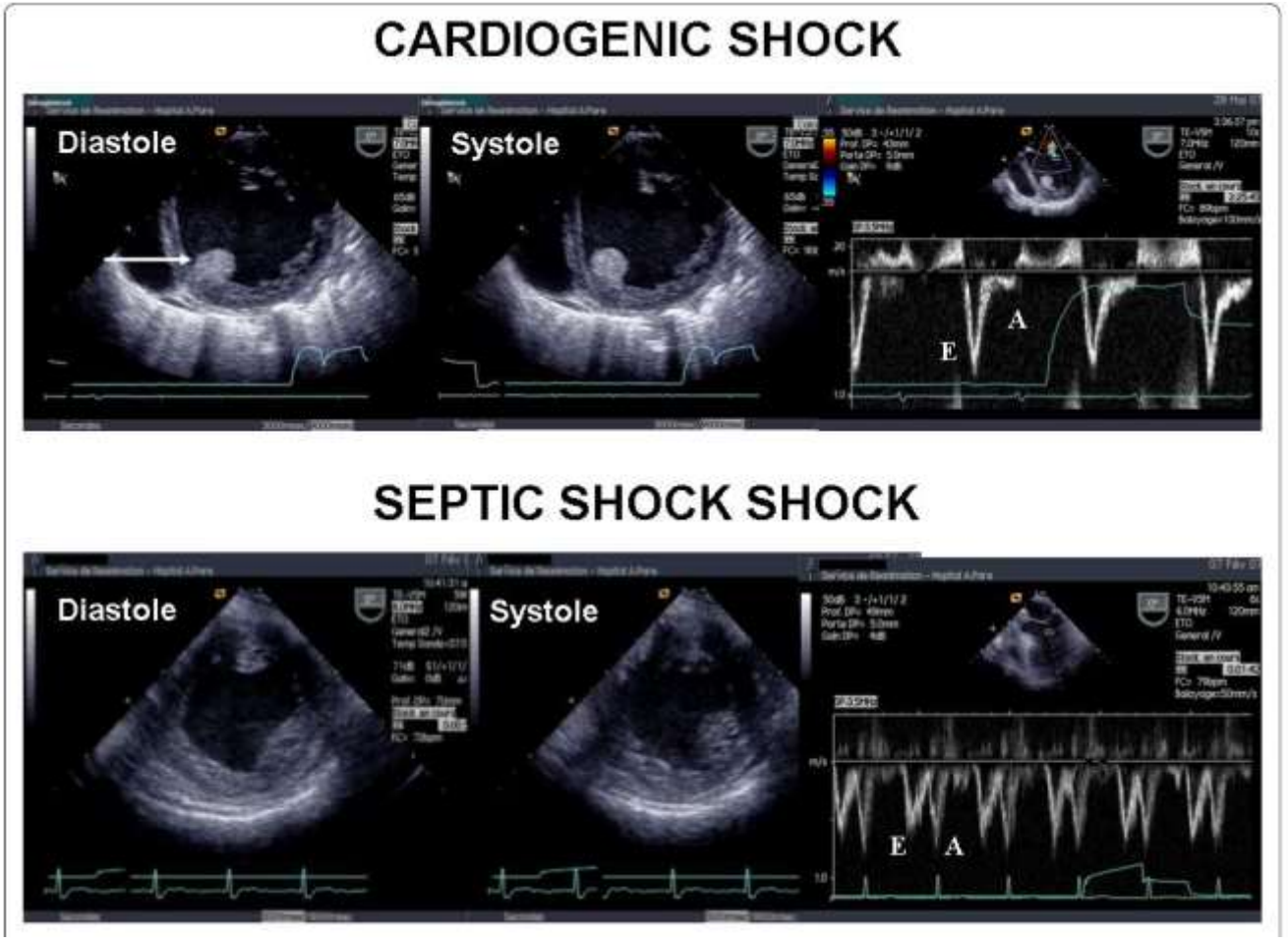
PRESENTATION CLINIQUE

Dysfonction VG systolique

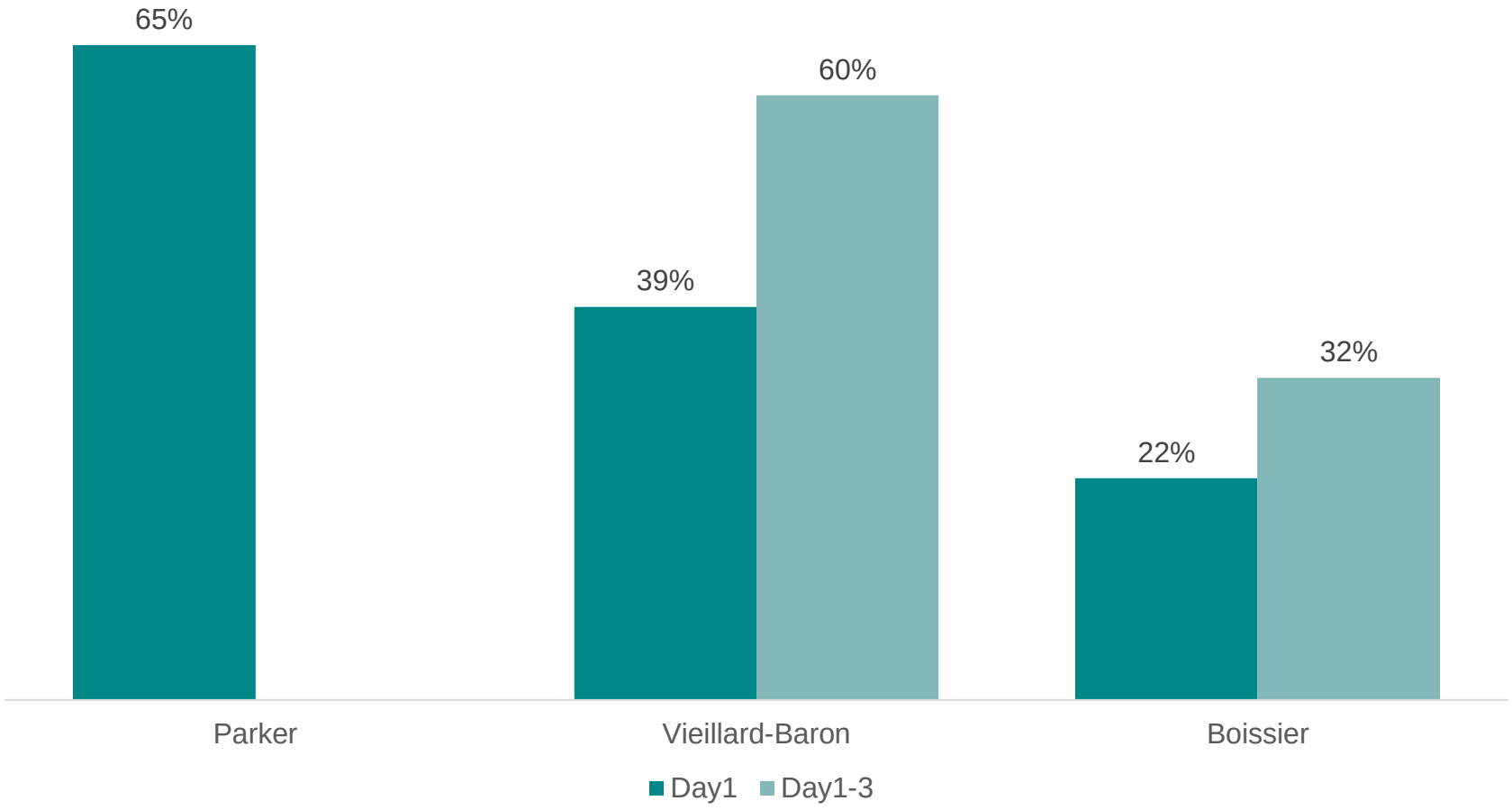
Dysfonction VG systolique

Pressions de remplissage typiquement basses

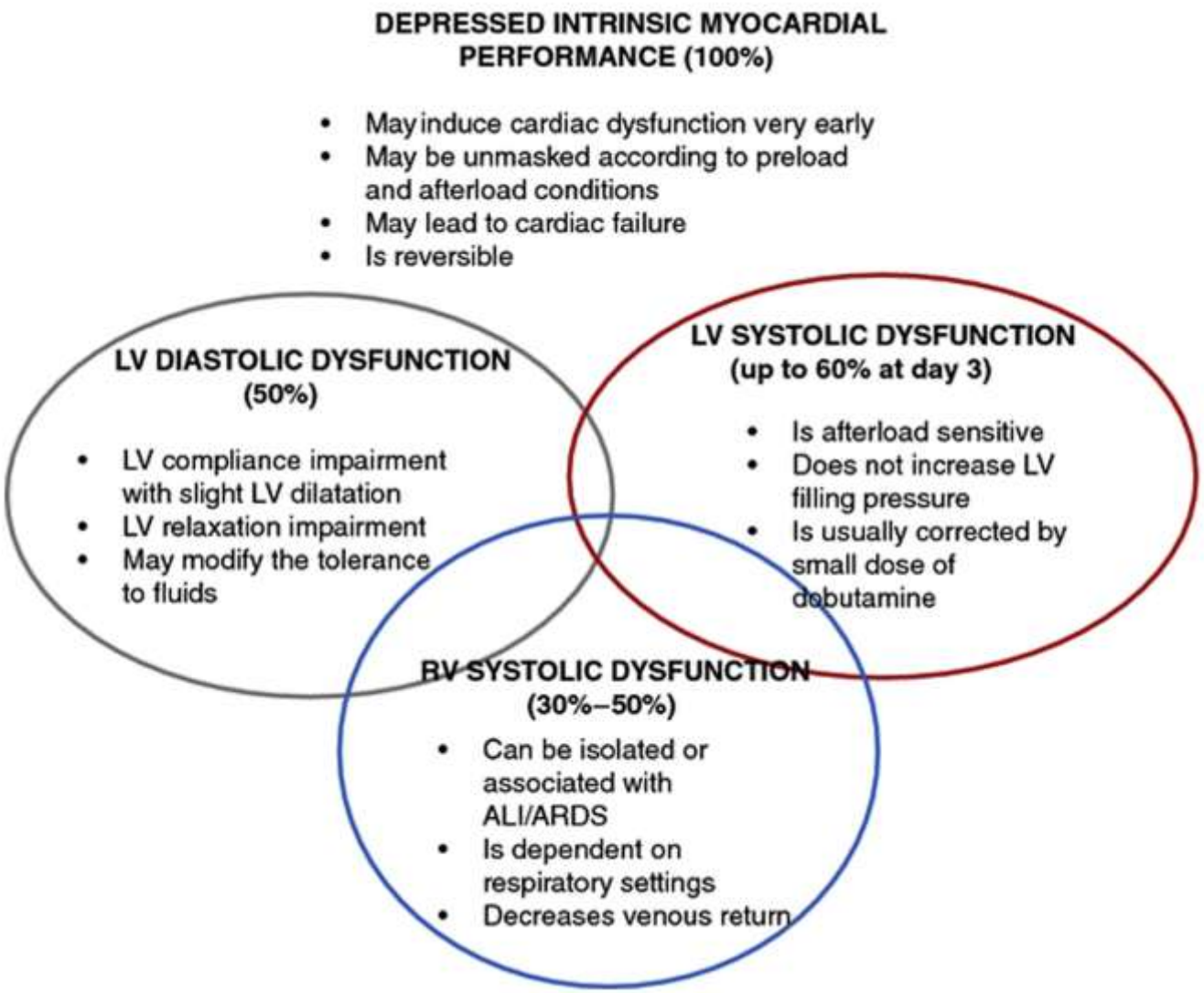


Vieillard-Baron AIC 2011

Incidence de la dysfonction systolique



Multiples altérations hémodynamiques

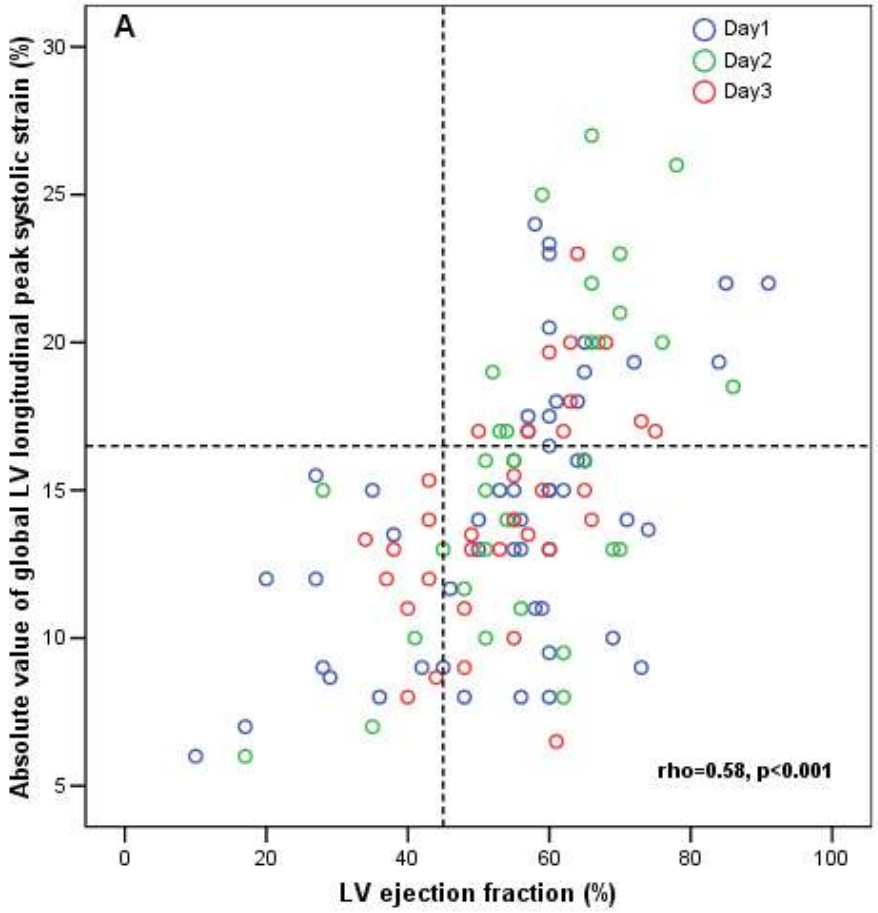


Fenton, Clin Chest Med 2016

L'écho est l'outil le plus adapté pour l'évaluation détaillée :

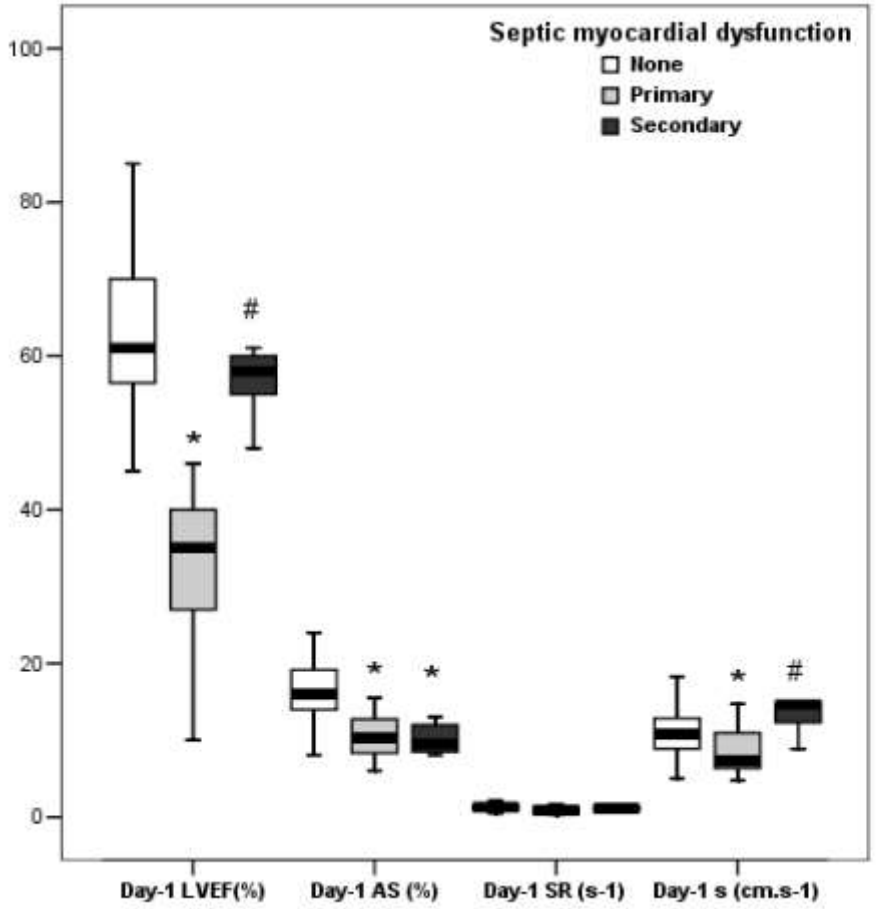
- Fonction globale
- Systole vs. Diastole
- VG vs. VD
- Influence de l'hypovolémie
- Influence de la vasoplégie
- Classification pronostique
- Optimisation thérapeutique ?

Dépression myocardique intrinsèque chez la majorité des malades



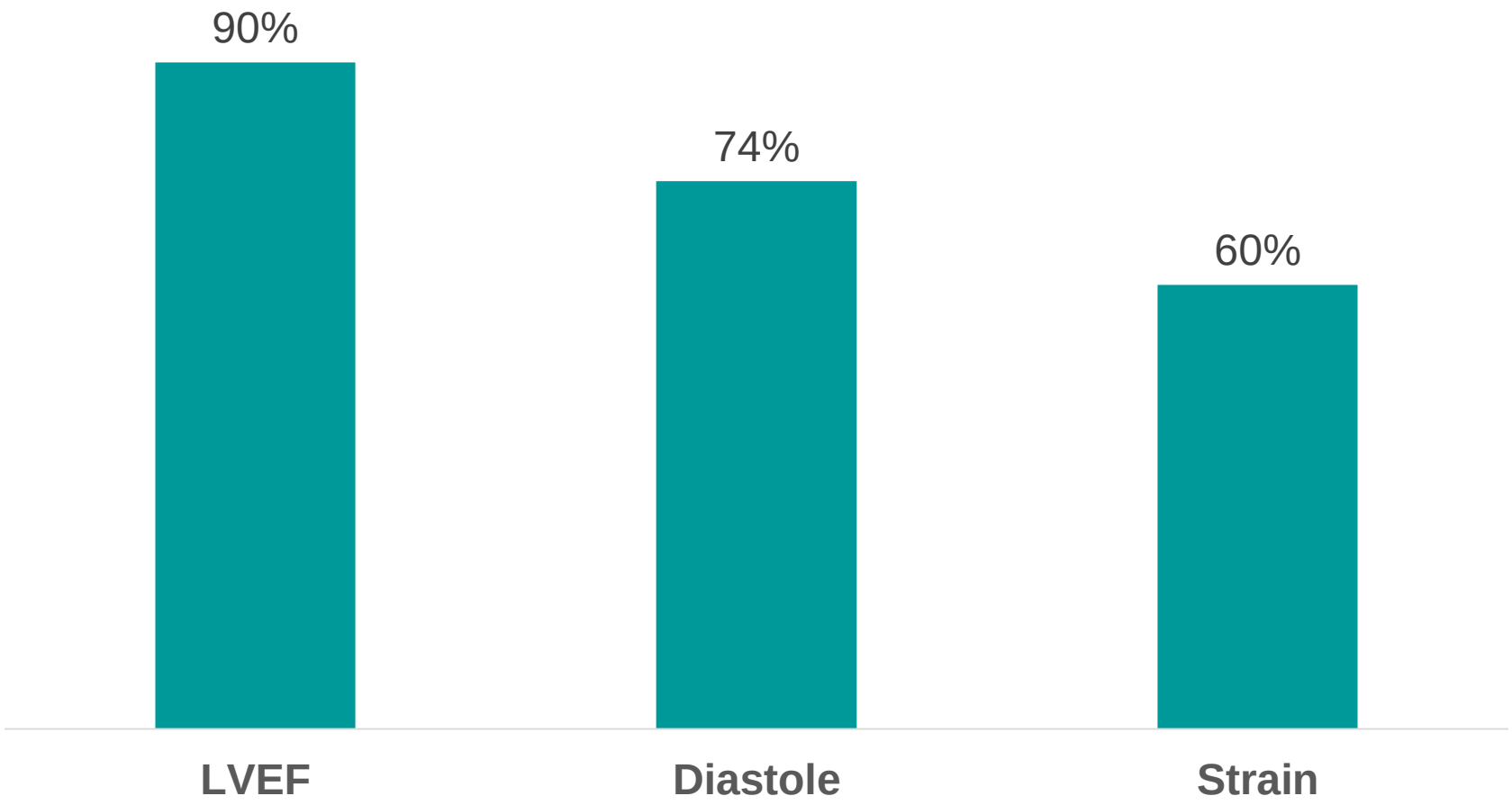
Boissier, ICM 2017

Le strain pour la détection précoce de la dysfonction systolique



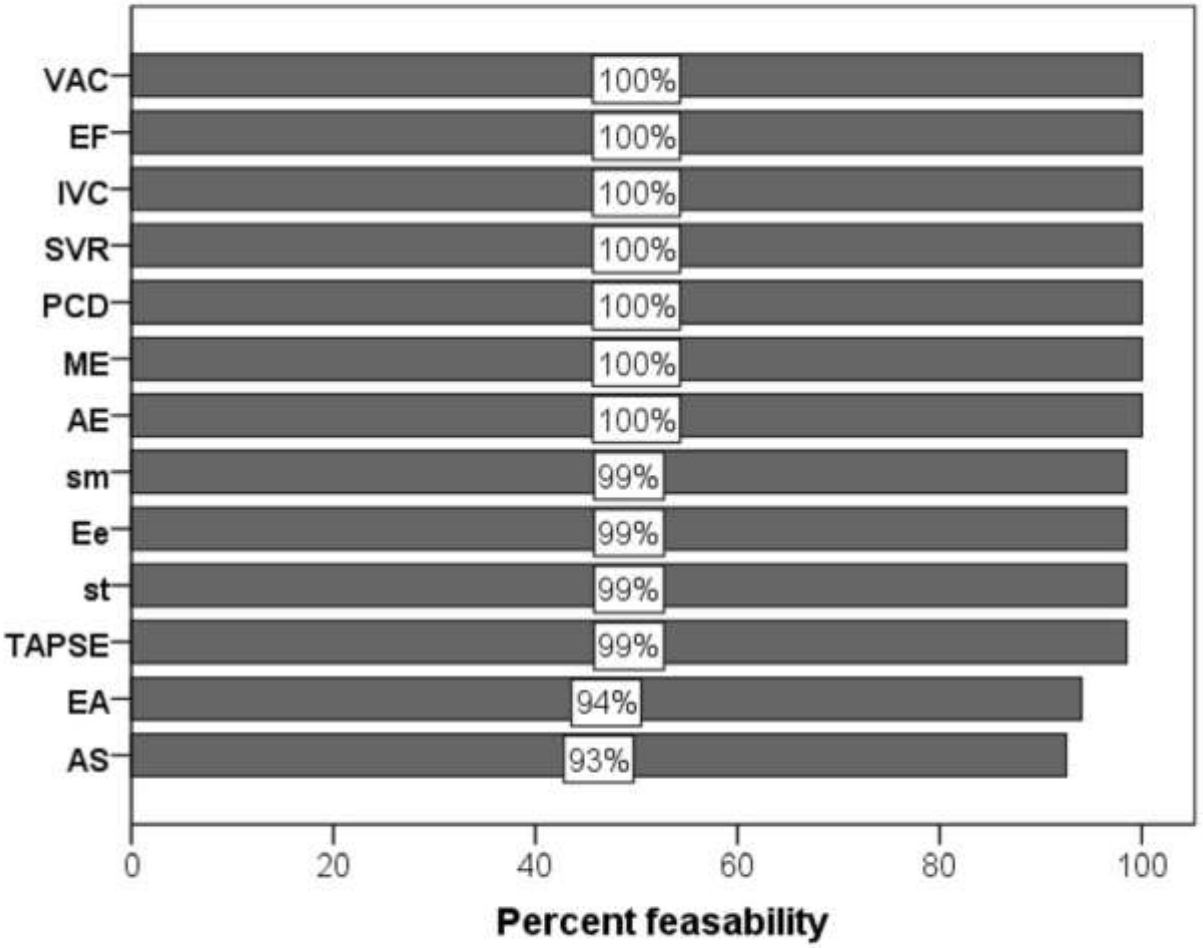
Boissier, ICM 2017

Faisabilité de l'echo au cours du choc septique



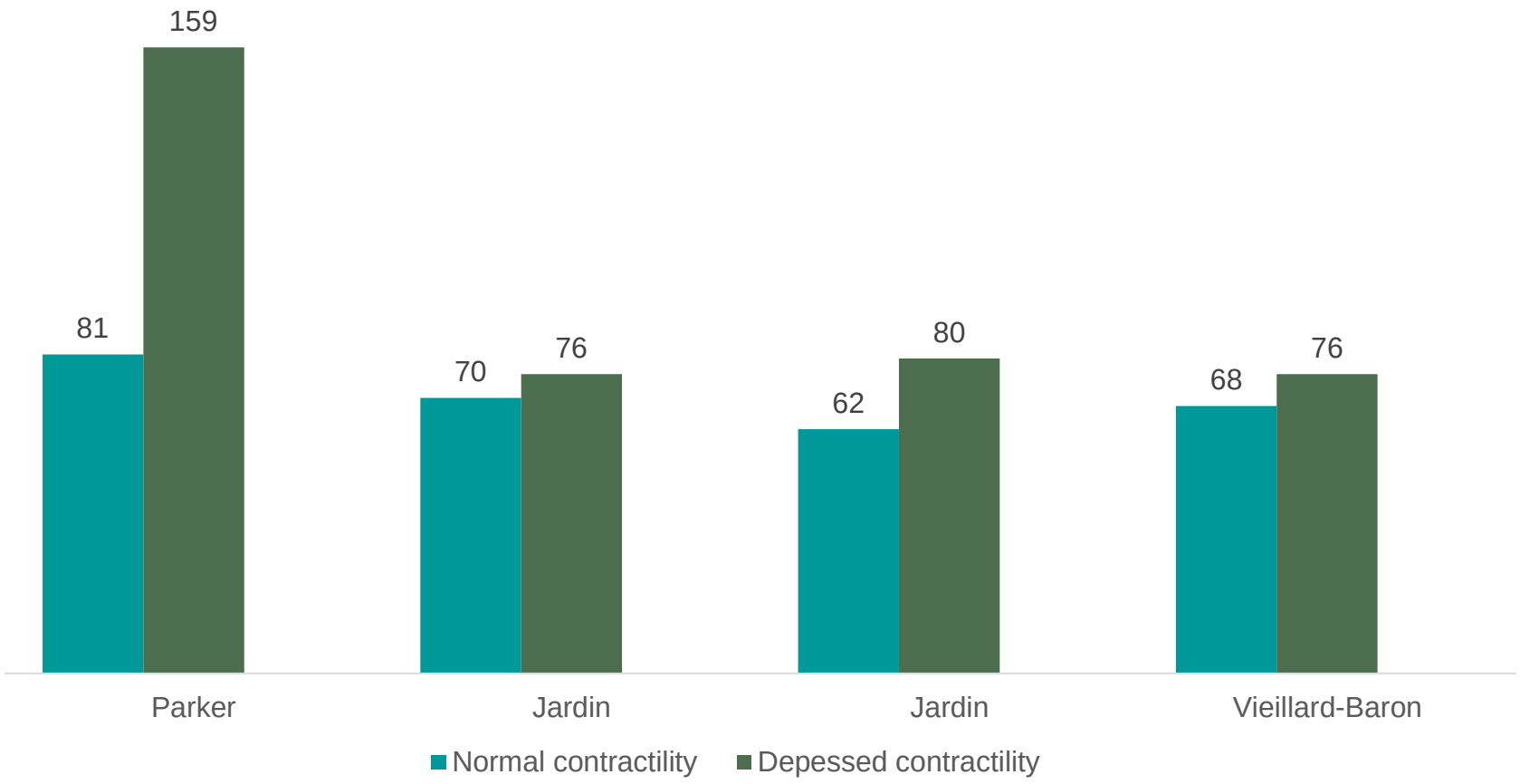
Boissier et al, ICM 2017

Faisabilité au cours du sepsis



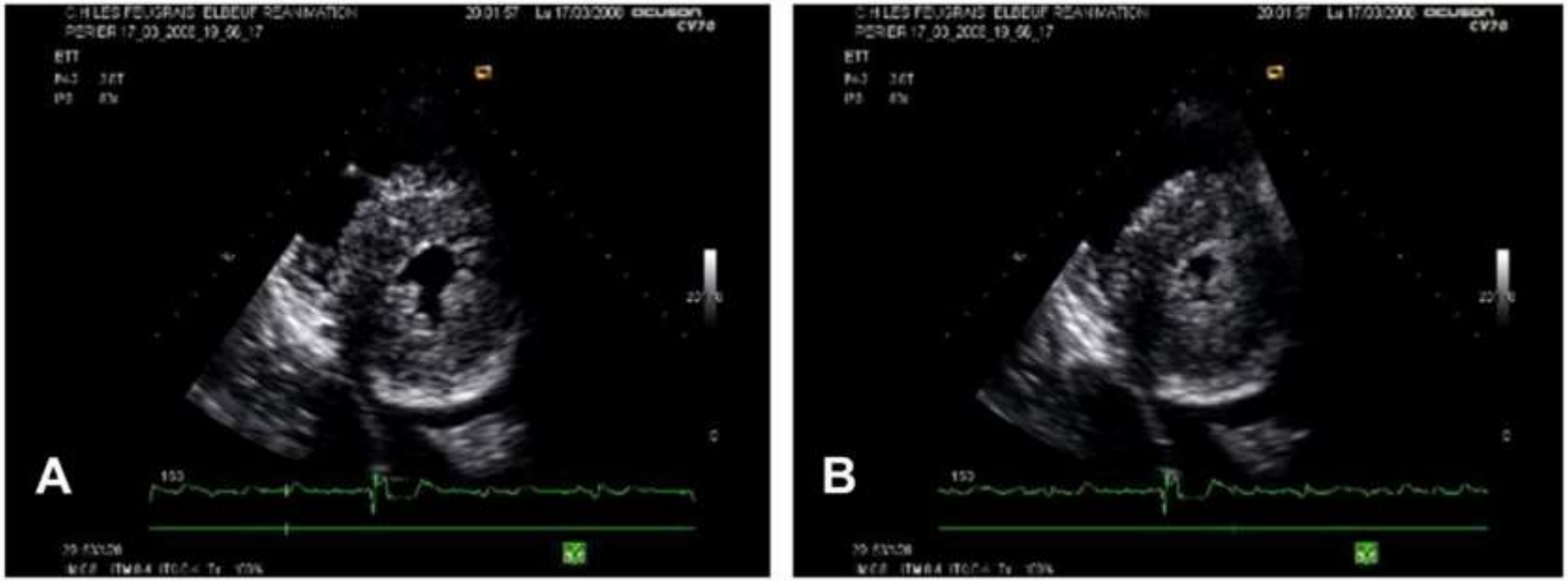
Bagate, JIC 2021

Dilatation modérée du VG



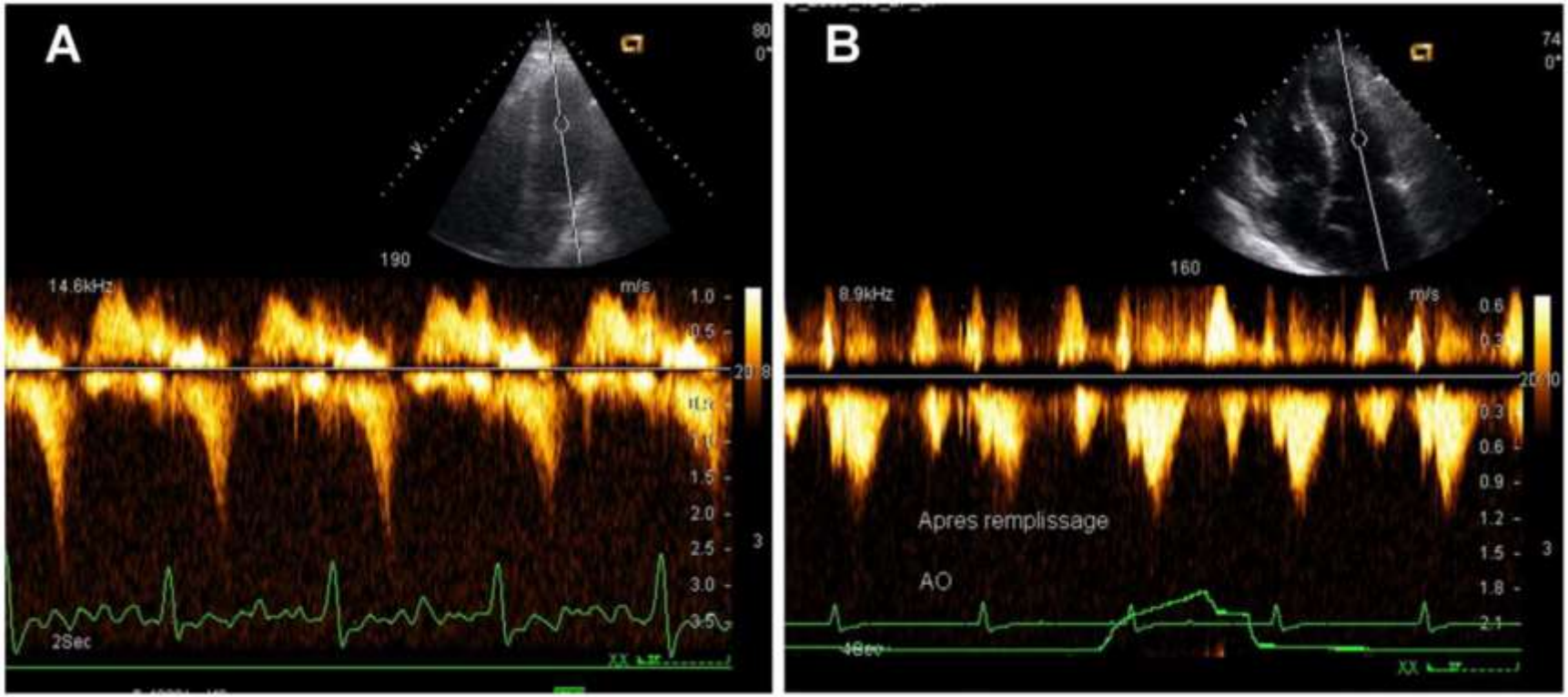
PHYSIOPATHOLOGIE
ROLE DES CONDITIONS DE CHARGE

Hypovolémie:
pseudohypertrophie et exclusion systolique



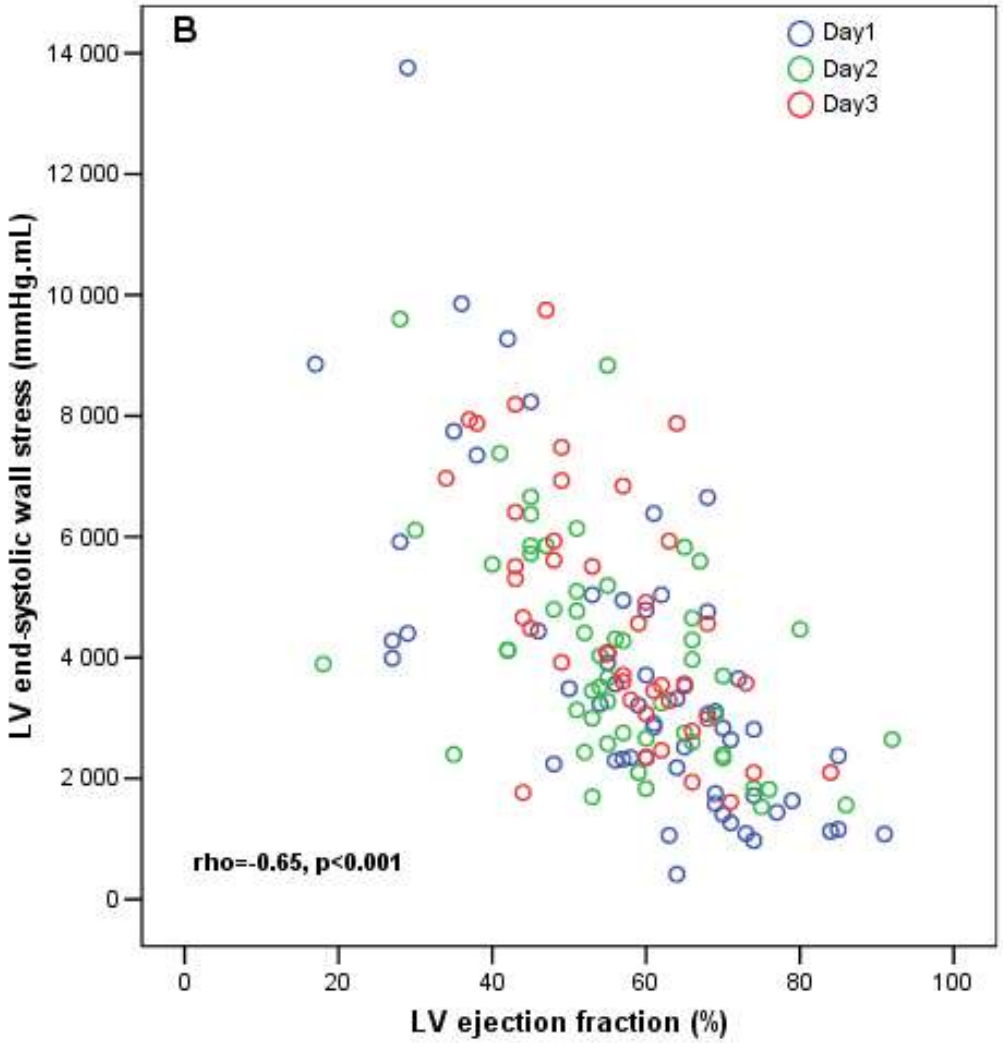
Chauvet, Crit Care 2015

Hypovolémie: obstruction intraventriculaire



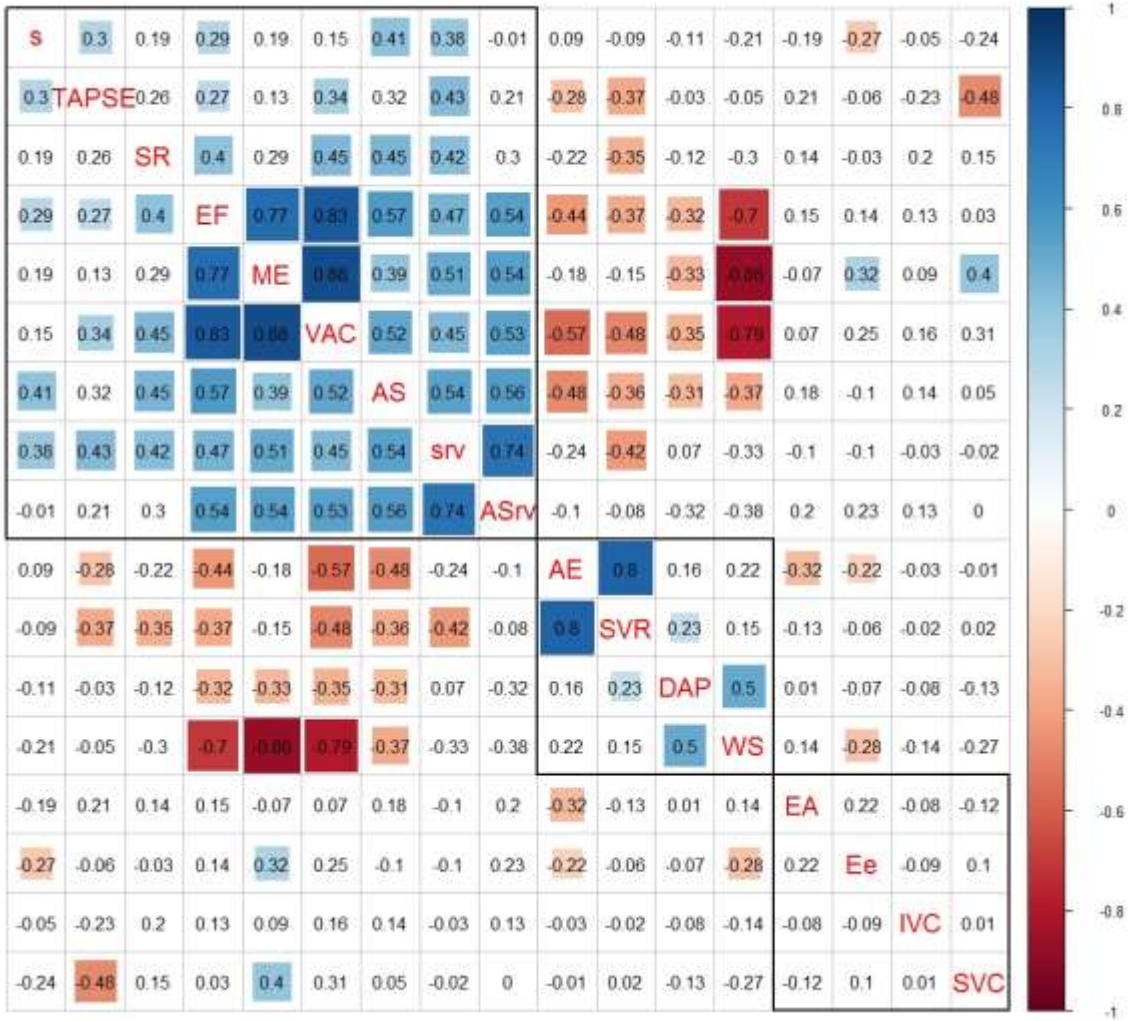
Chauvet, Crit Care 2015

Vasoplégie



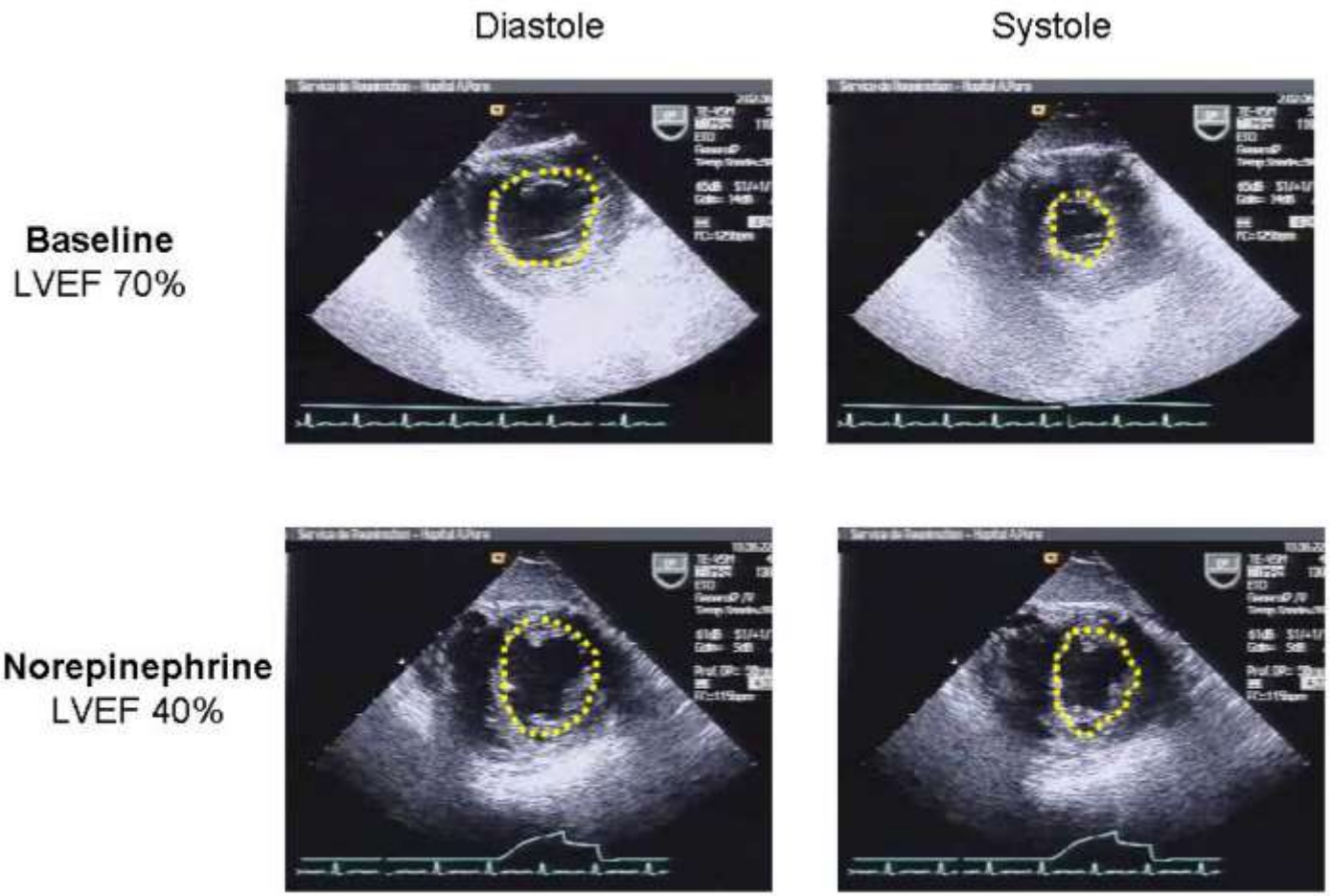
Boissier, ICM 2017

Cohérence physiologique



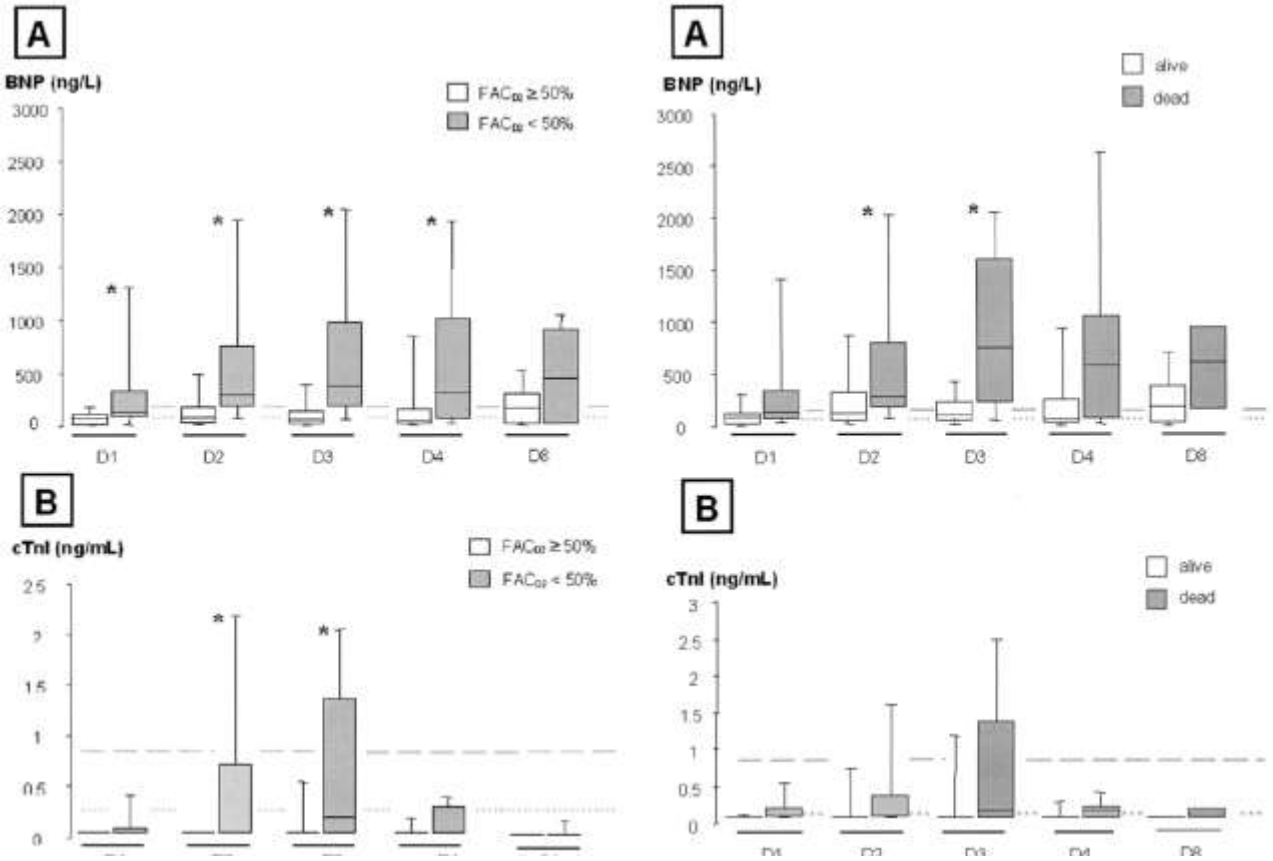
Boissier et al, ICM 2017

Manipulation de la postcharge



Vieillard-Baron AIC 2011

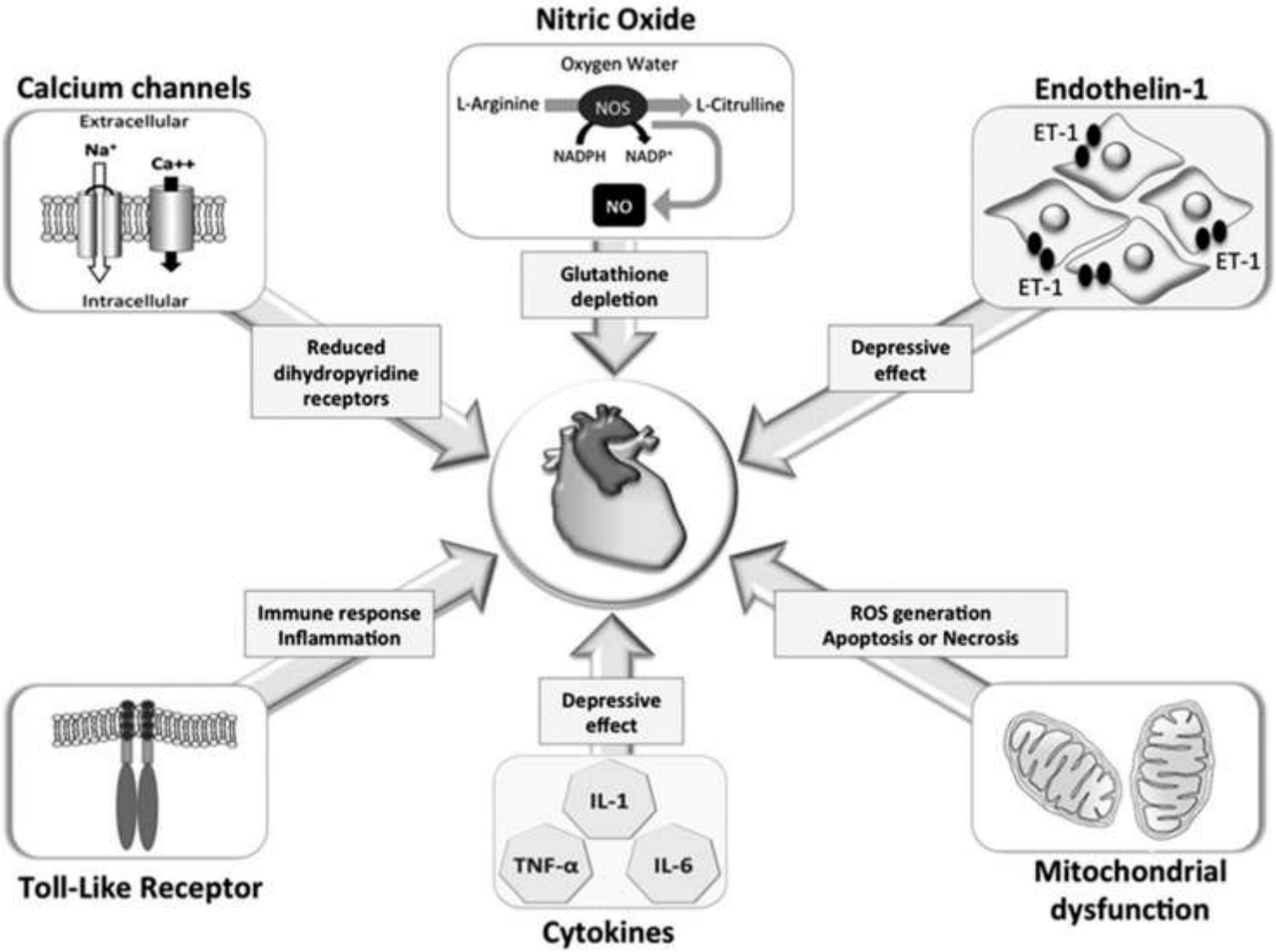
Place des biomarqueurs ?



Charpentier CCM 2004

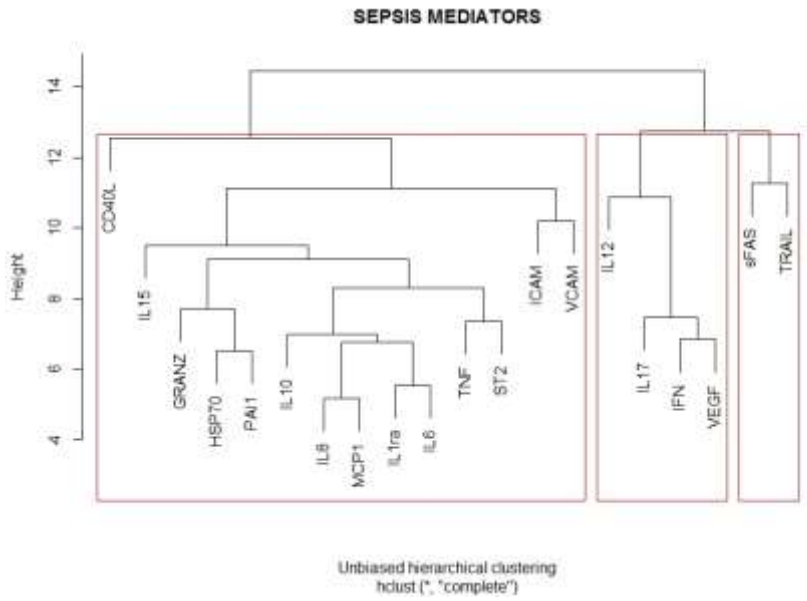
BIOPATHOLOGIE

Mécanismes complexes



Antonucci J Crit Care 2014

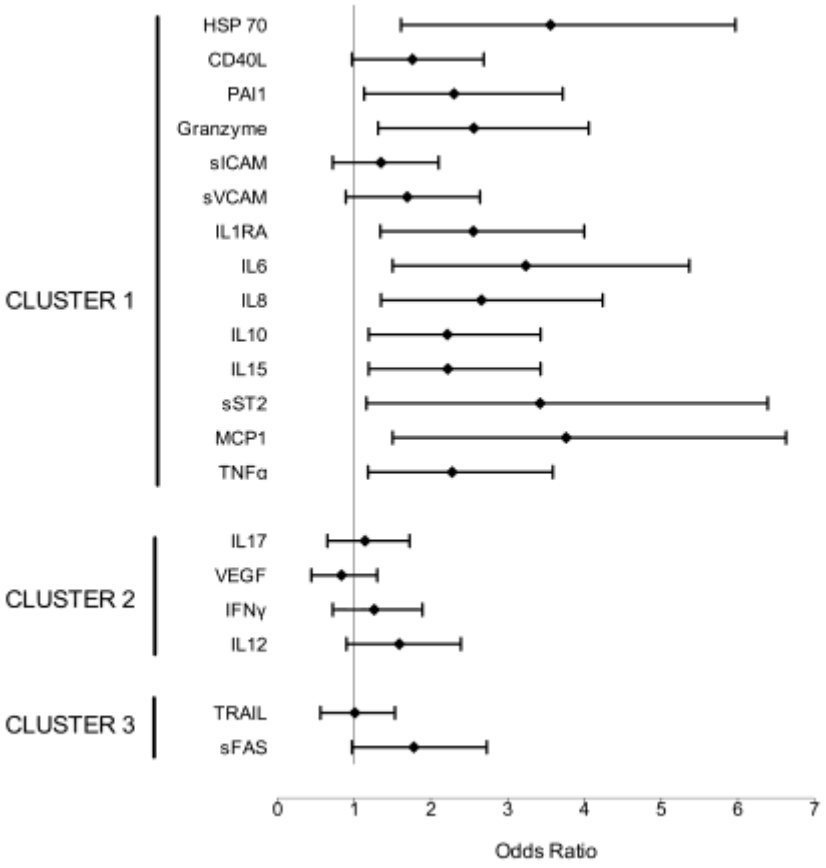
Médiateurs de la DMS



Cluster 1: Innate imm.

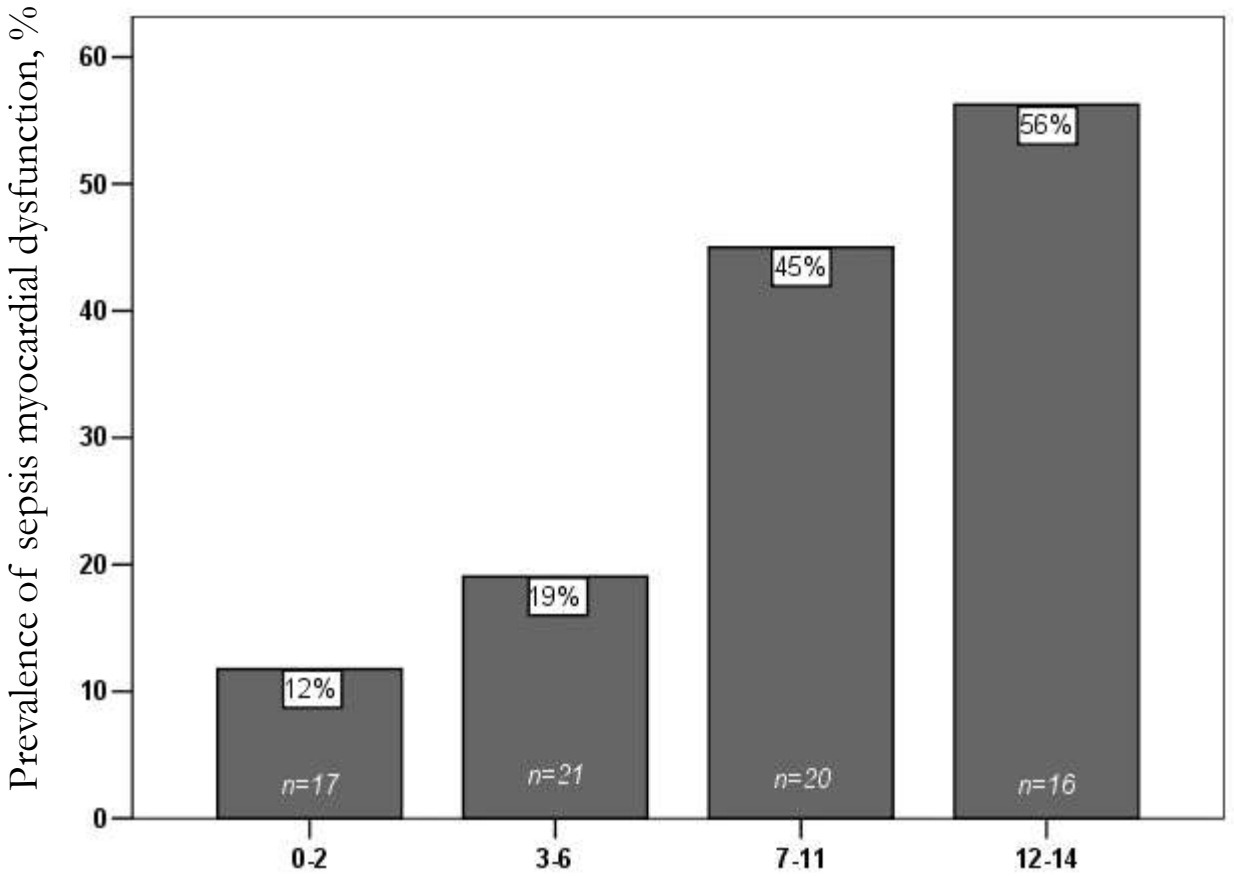
Cluster 2: Adaptive imm.

Cluster 3: Cell death and repair



Razazi, AIC 2019

Synergie



Number of sepsis mediators from the first cluster with increased plasma concentration (above the median value of fluorescence intensity)

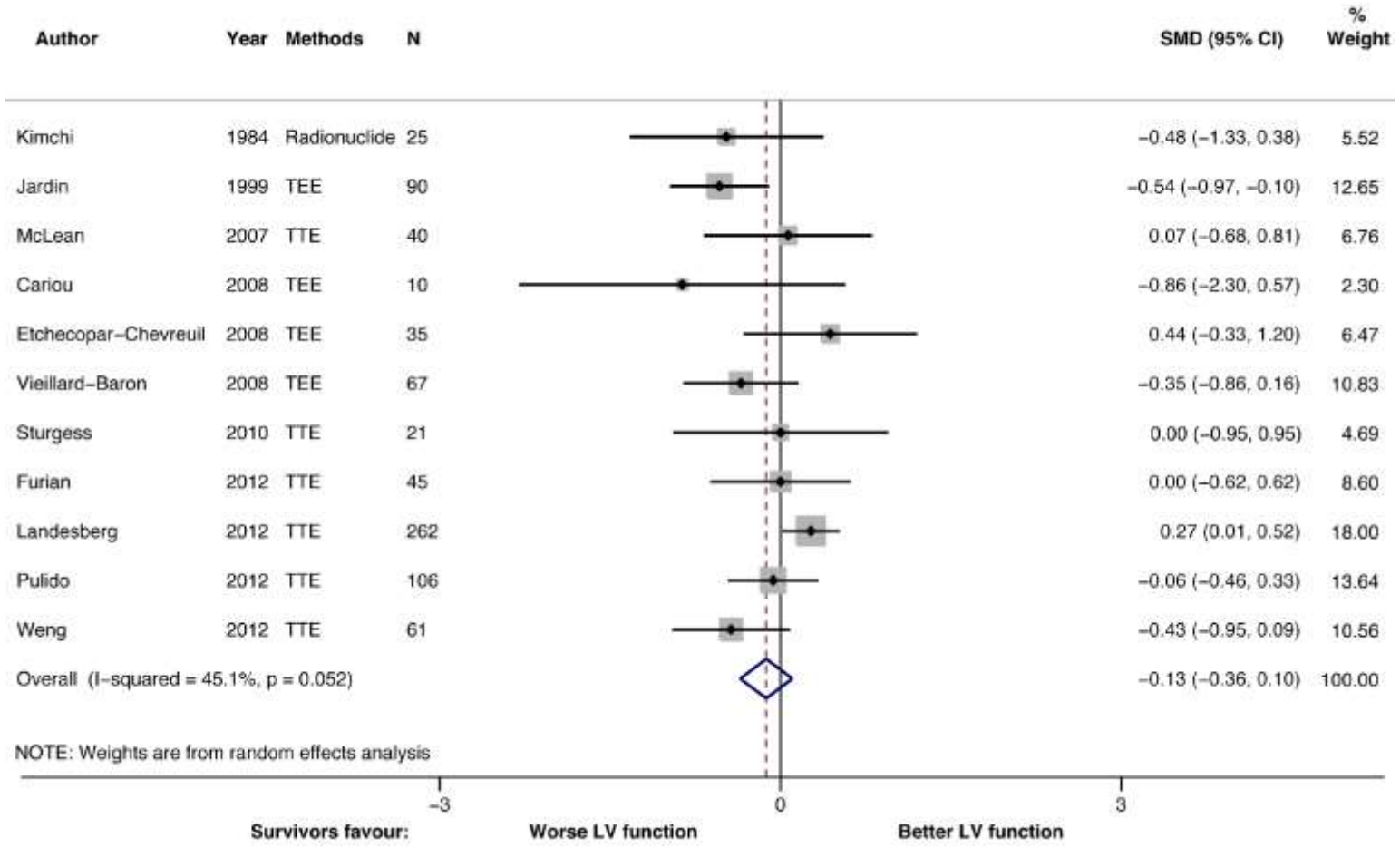
Razazi, AIC 2019

IMPLICATIONS CLINIQUES

Implications cliniques complexes

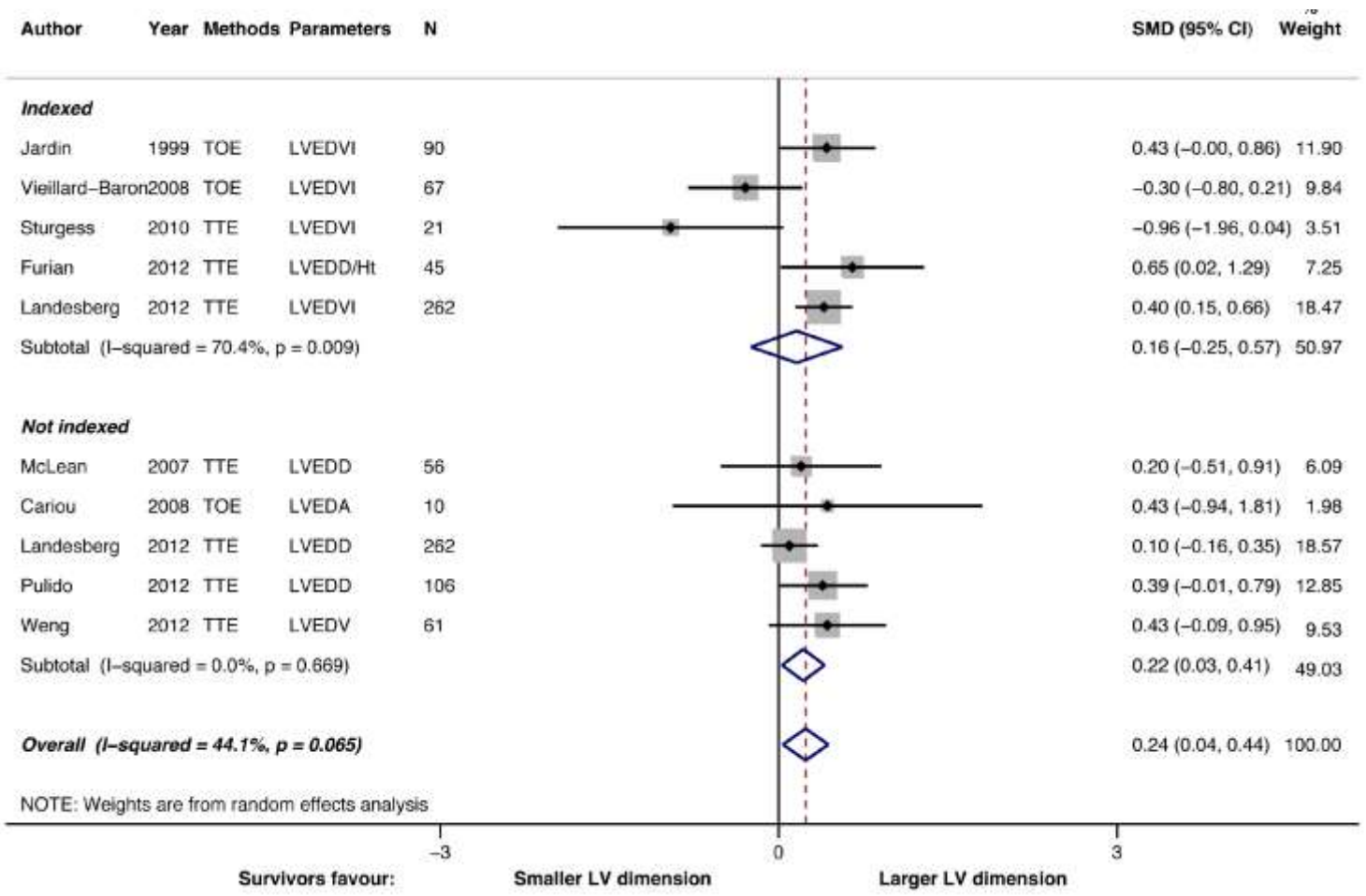
- Relation complexe à la mortalité
- Rôle controversé des inotropes et des vasopresseurs
- Utilité discutable de l'optimisation du débit cardiaque

Pronostic de la dysfonction VG systolique



Huang Crit Care 2013

Pronostic de la dilatation VG



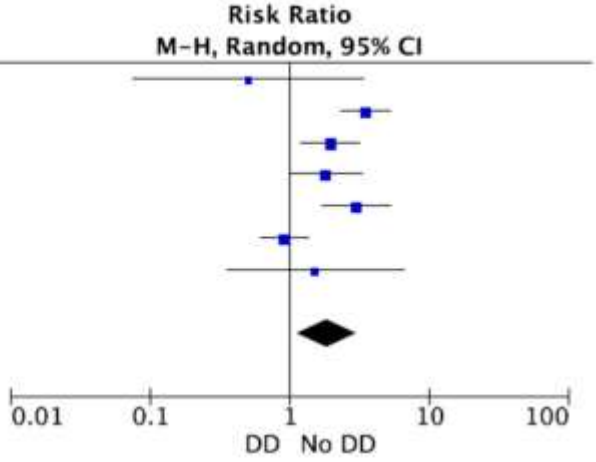
Huang Crit Care 2013

DMS et mortalité

a

Study or Subgroup	DD Events	DD Total	No DD Events	No DD Total	Weight	Risk Ratio M-H, Random, 95% CI
Ethecopar 2008	1	7	8	28	5.0%	0.50 [0.07, 3.37]
Landesberg 2012	88	143	21	119	18.6%	3.49 [2.32, 5.25]
Landesberg 2014	29	53	15	53	17.5%	1.93 [1.18, 3.17]
Mokart 2007	12	18	10	27	16.3%	1.80 [1.00, 3.25]
Moraud 2014	25	33	10	39	16.6%	2.95 [1.67, 5.22]
Pulido 2012	19	39	30	56	18.6%	0.91 [0.61, 1.36]
Sturgess 2010	4	12	2	9	7.4%	1.50 [0.35, 6.46]
Total (95% CI)		**305**		**331**	**100.0%**	**1.82 [1.12, 2.97]**
Total events	178		96			

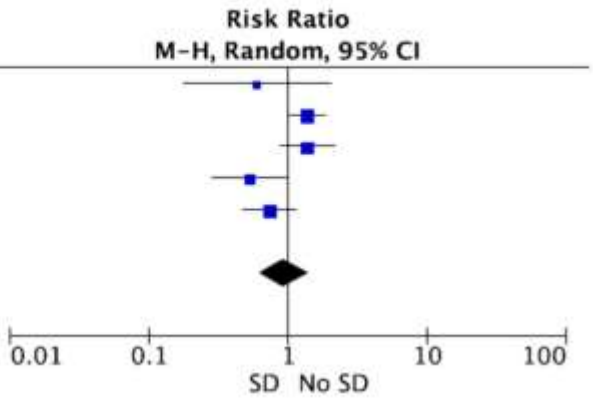
Heterogeneity: $Tau^2 = 0.29$; $Chi^2 = 26.42$, df = 6 (P = 0.0002); $I^2 = 77\%$
Test for overall effect: Z = 2.40 (P = 0.02)



b

Study or Subgroup	SD Events	SD Total	No SD Events	No SD Total	Weight	Risk Ratio M-H, Random, 95% CI
Ethecopar 2008	3	16	6	19	8.2%	0.59 [0.18, 2.00]
Landesberg 2012	32	61	77	201	27.7%	1.37 [1.02, 1.84]
Landesberg 2014	14	27	30	79	22.9%	1.37 [0.86, 2.16]
Moraud 2014	10	39	16	33	17.9%	0.53 [0.28, 1.00]
Pulido 2012	13	29	47	77	23.4%	0.73 [0.47, 1.14]
Total (95% CI)		**172**		**409**	**100.0%**	**0.93 [0.62, 1.39]**
Total events	72		176			

Heterogeneity: $Tau^2 = 0.13$; $Chi^2 = 12.31$, df = 4 (P = 0.02); $I^2 = 68\%$
Test for overall effect: Z = 0.34 (P = 0.73)



Sanfilipo, ICM 2015

Optimisation du débit cardiaque chez des patients non sélectionnés

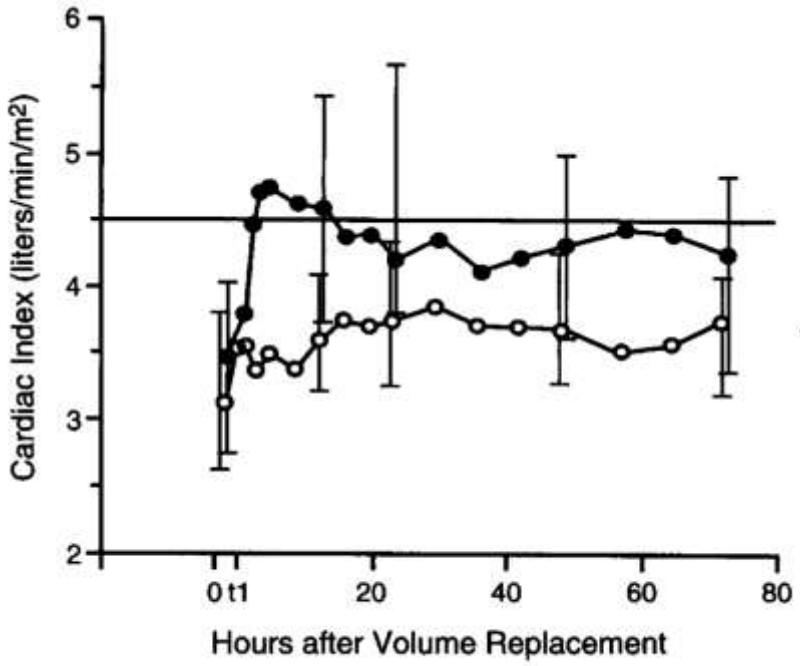


Figure 1. Median Cardiac Index in the Treatment and Control Groups.

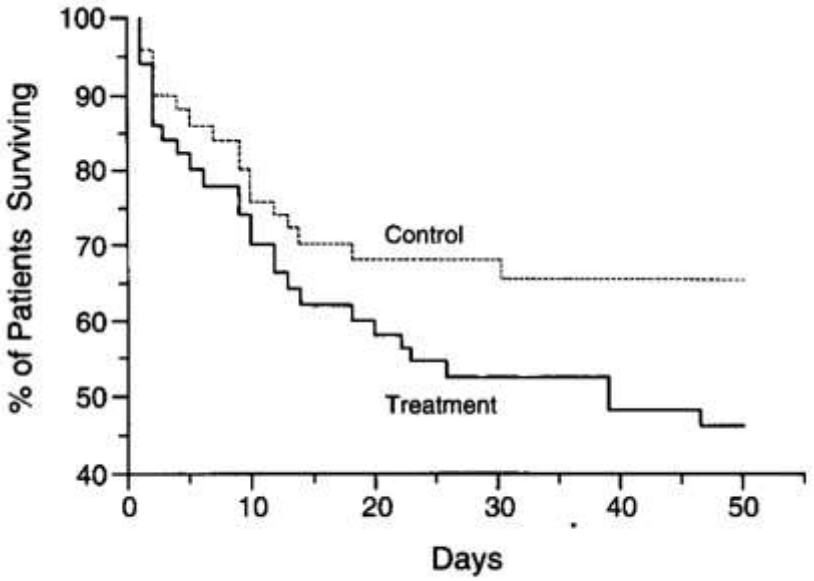


Figure 4. In-Hospital Survival of Patients in the Treatment and Control Groups.

Hayes, NEJM 1994

EGDT

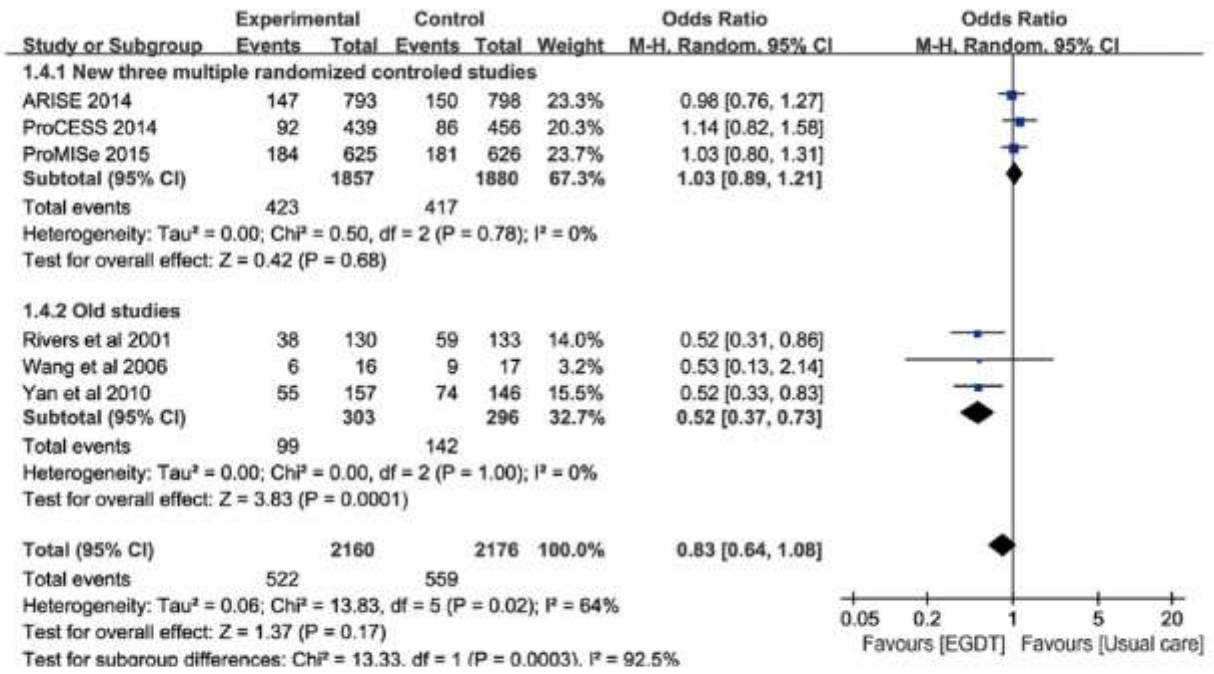


Fig. 3 Forest plot showing the effects of early goal-direced therapy on all-cause mortality in patients with severe sepsis and septic shock

Jiang, SJTR 2016

Inotropes chez des patients non selectionnés

Dobutamine

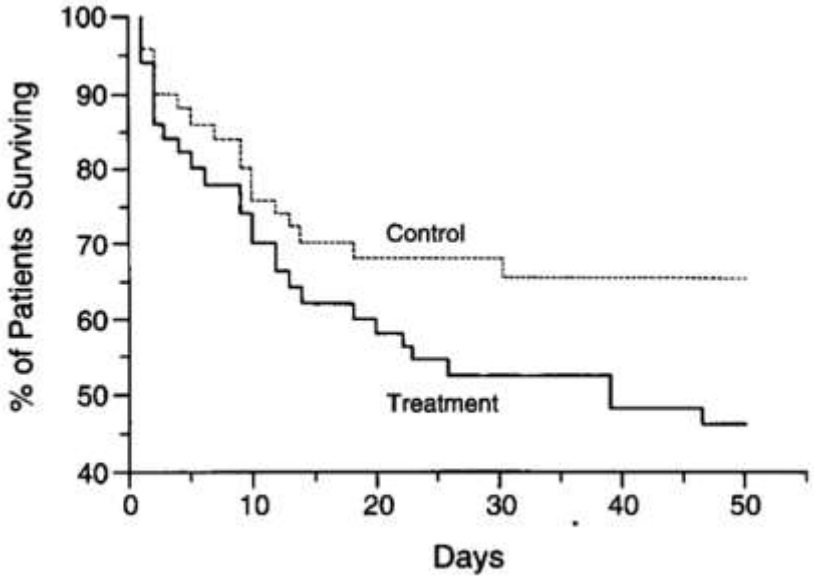


Figure 4. In-Hospital Survival of Patients in the Treatment and Control Groups.

Hayes, NEJM 1994

Levosimendan

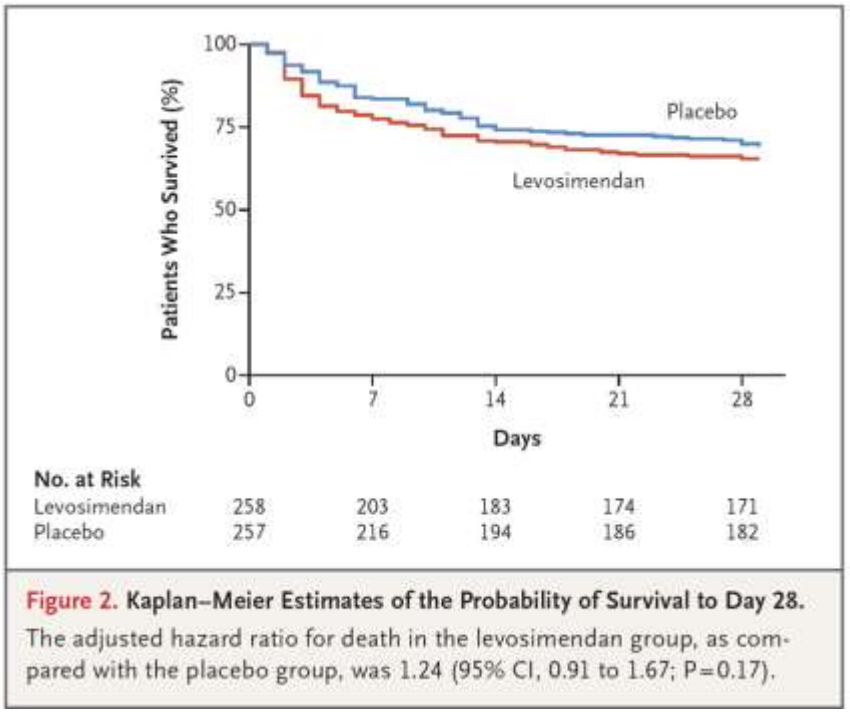
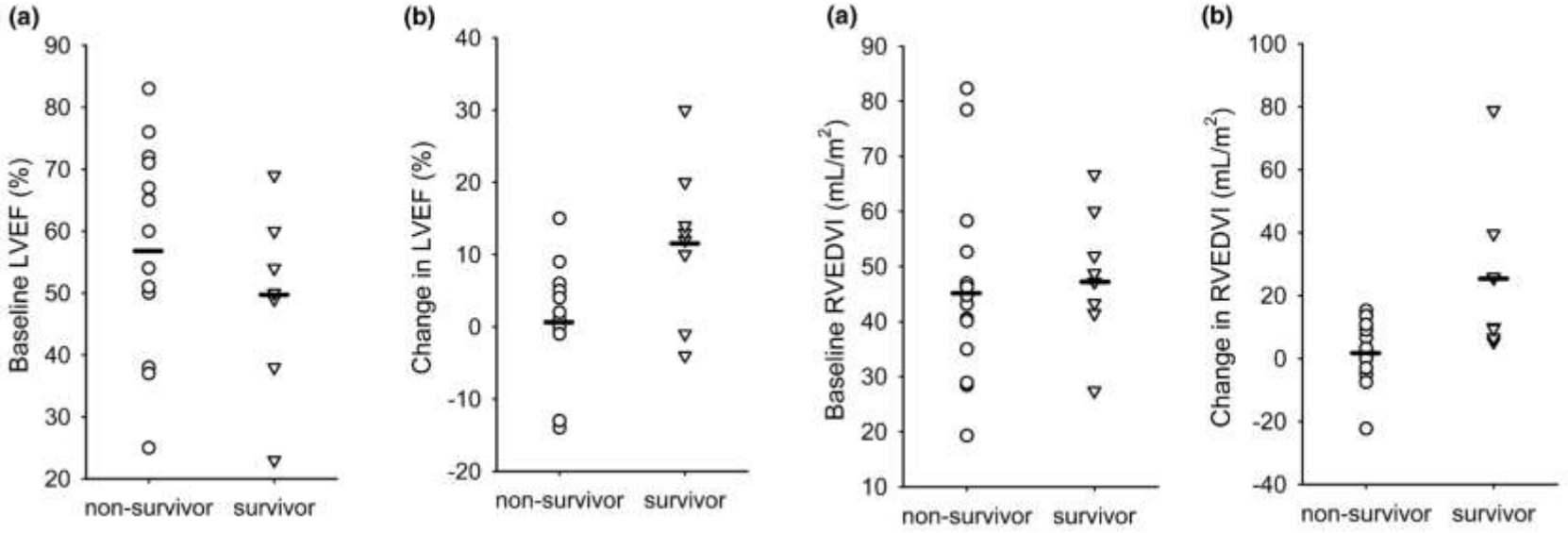


Figure 2. Kaplan–Meier Estimates of the Probability of Survival to Day 28.
The adjusted hazard ratio for death in the levosimendan group, as compared with the placebo group, was 1.24 (95% CI, 0.91 to 1.67; P=0.17).

Gordon, NEJM 2017

Dobutamine stress test



Kumar Crit Care 2008

Conclusions

- Mécanismes multiples, complexes et intriqués

- Depression de la contractilité intrinsèque chez une majorité de patients, mais la vasoplégie influence les paramètres de fonction systolique

- Un phénotypage cardiaque détaillé est necessaire pour la stratégie thérapeutique
  - Place majeure de l'échocardiographie