



# Dysfonction myocardique du sepsis

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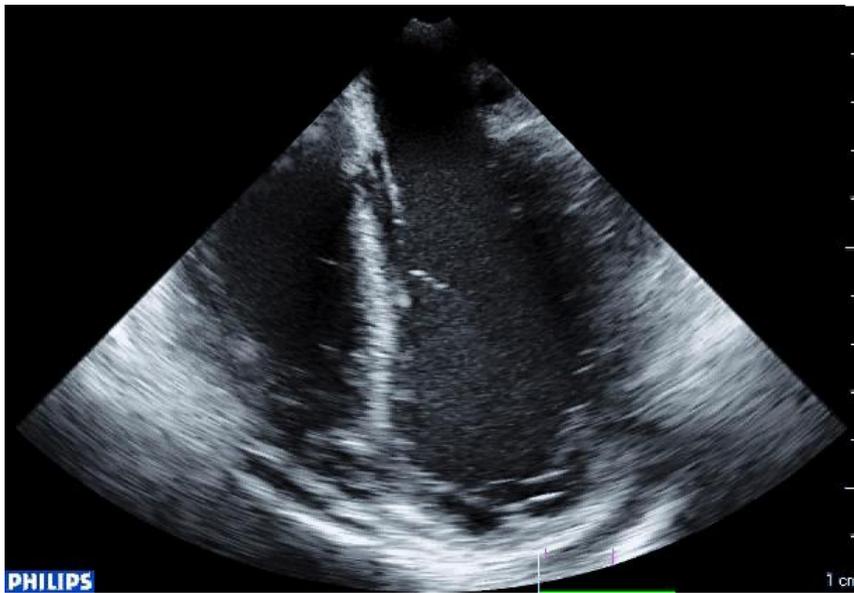
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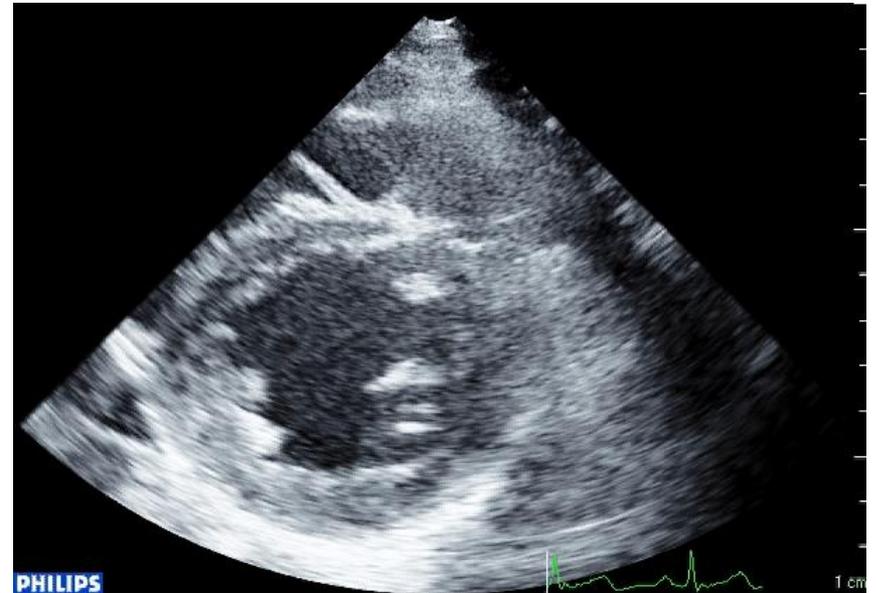
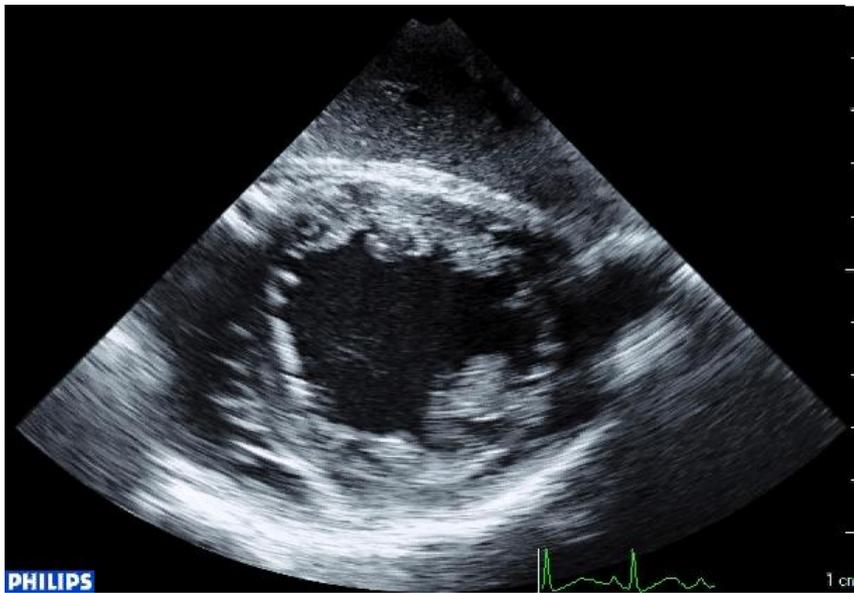
# **PRESENTATION CLINIQUE**

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# Dysfonction VG systolique



# Dysfonction VG systolique

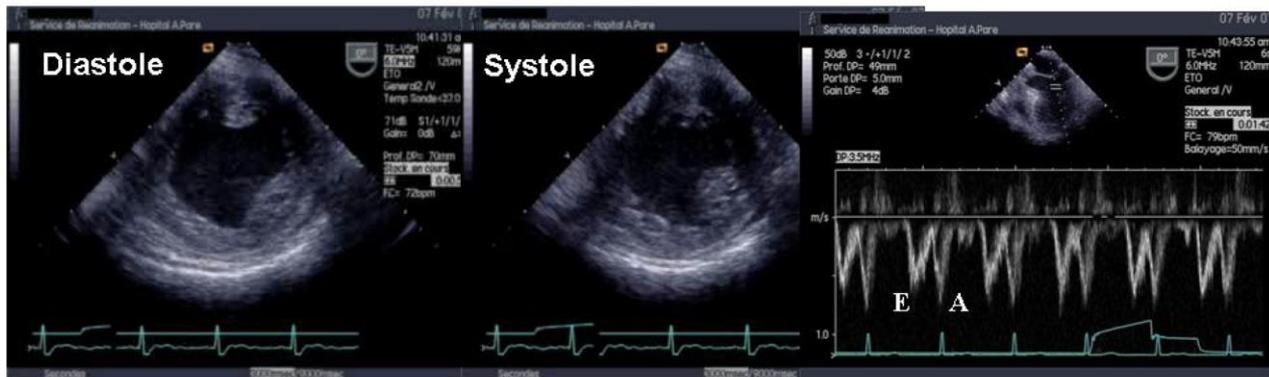


# Pressions de remplissage typiquement basses

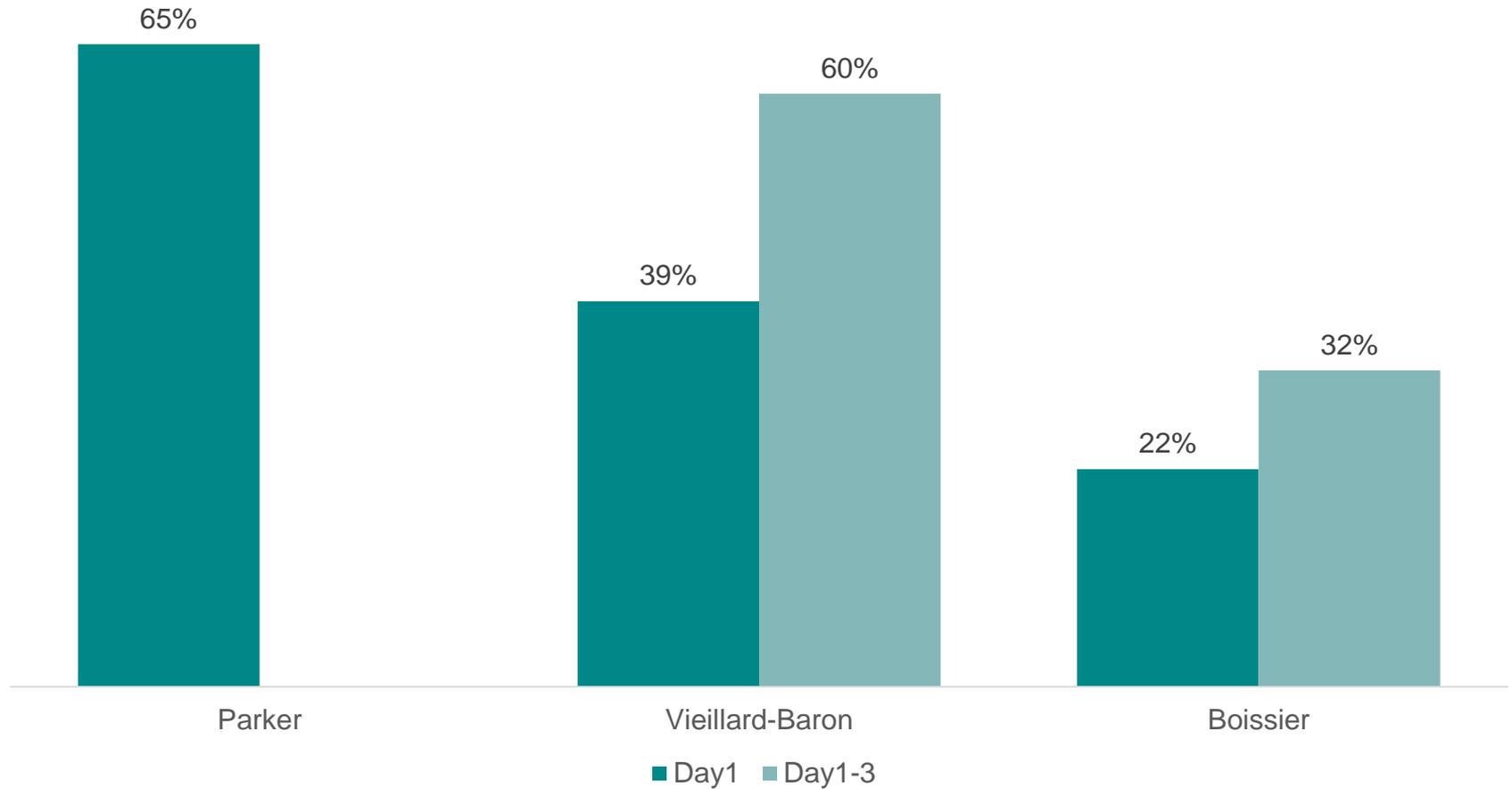
## CARDIOGENIC SHOCK



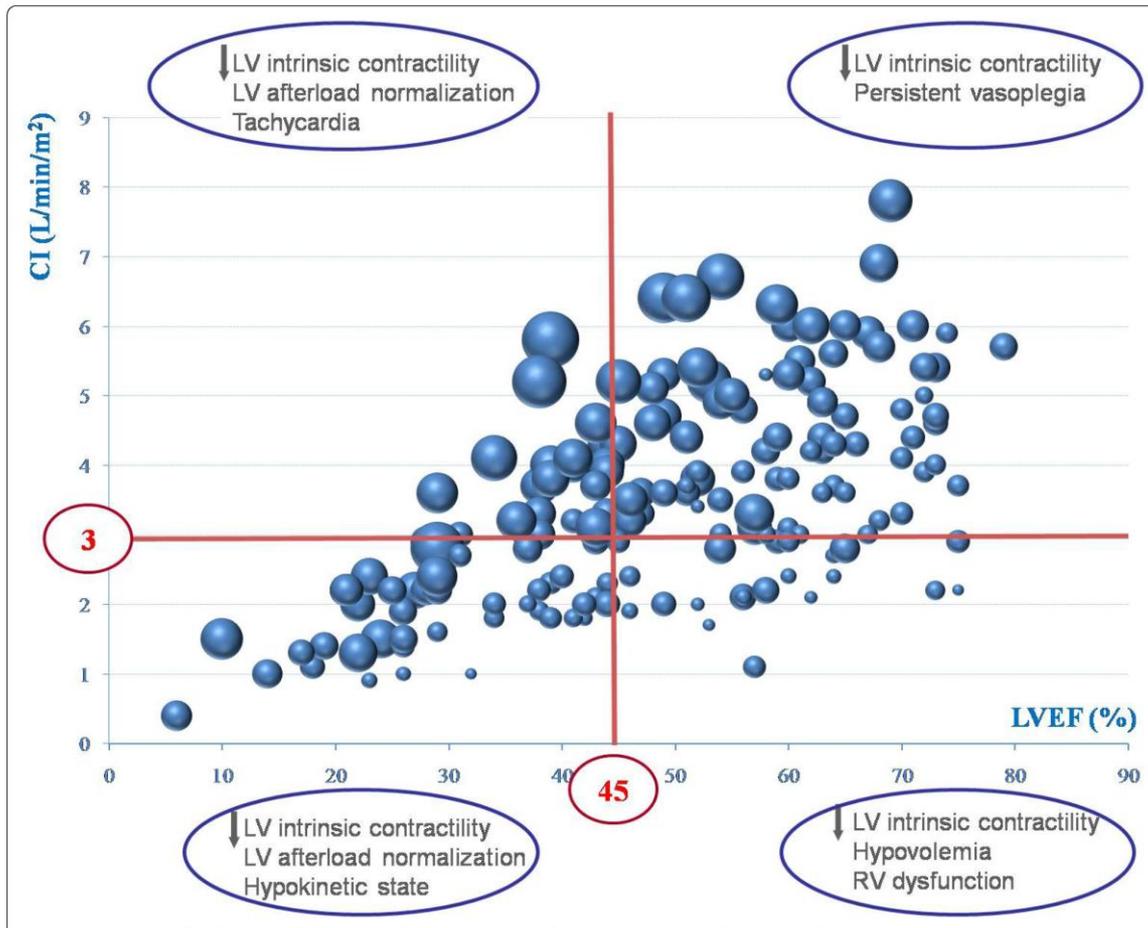
## SEPTIC SHOCK SHOCK



# Incidence de la dysfonction systolique



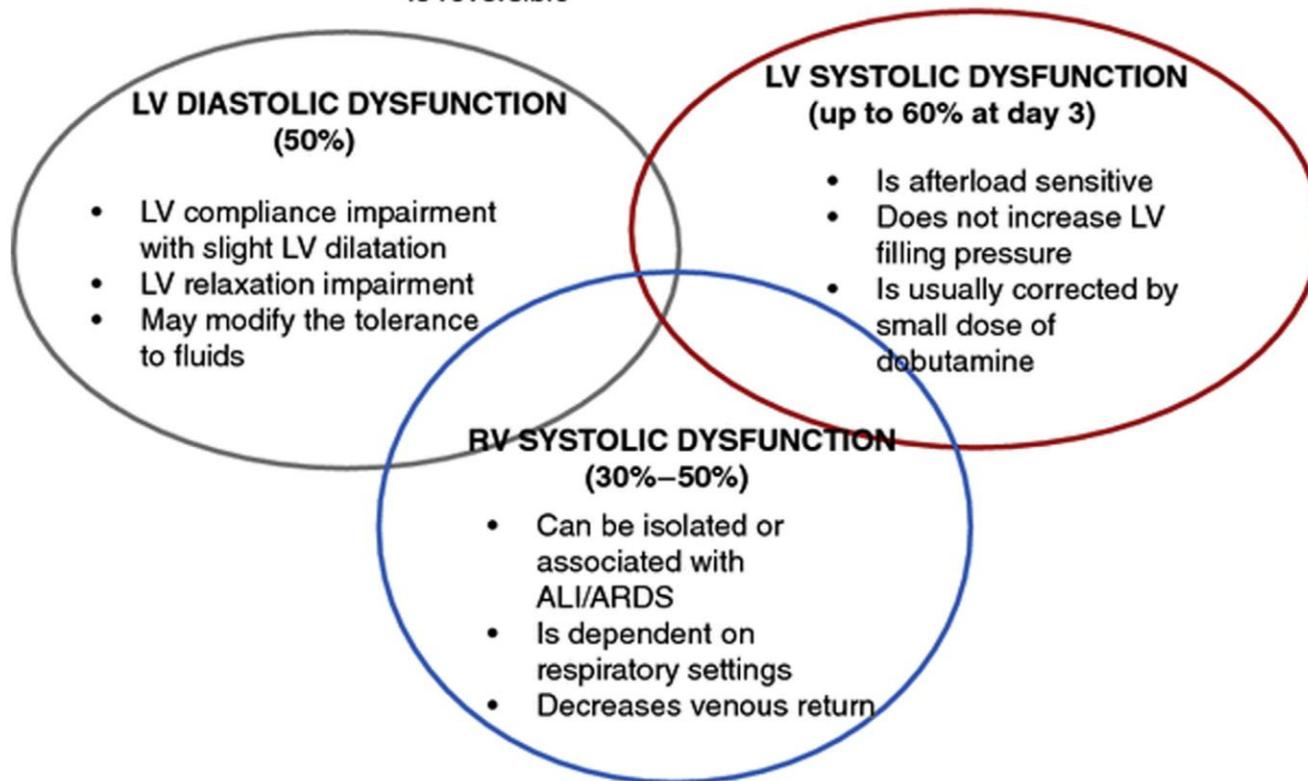
# Fonction cardiaque globale très variable



# Multiple alterations hémodynamiques

## DEPRESSED INTRINSIC MYOCARDIAL PERFORMANCE (100%)

- May induce cardiac dysfunction very early
- May be unmasked according to preload and afterload conditions
- May lead to cardiac failure
- Is reversible

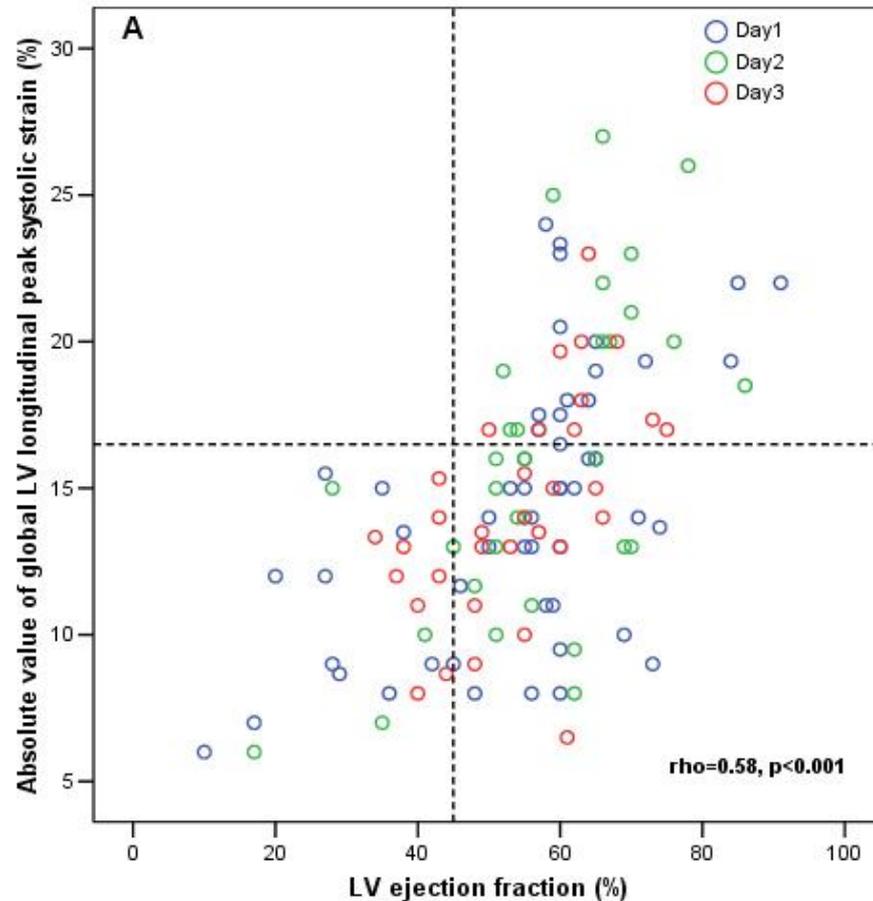


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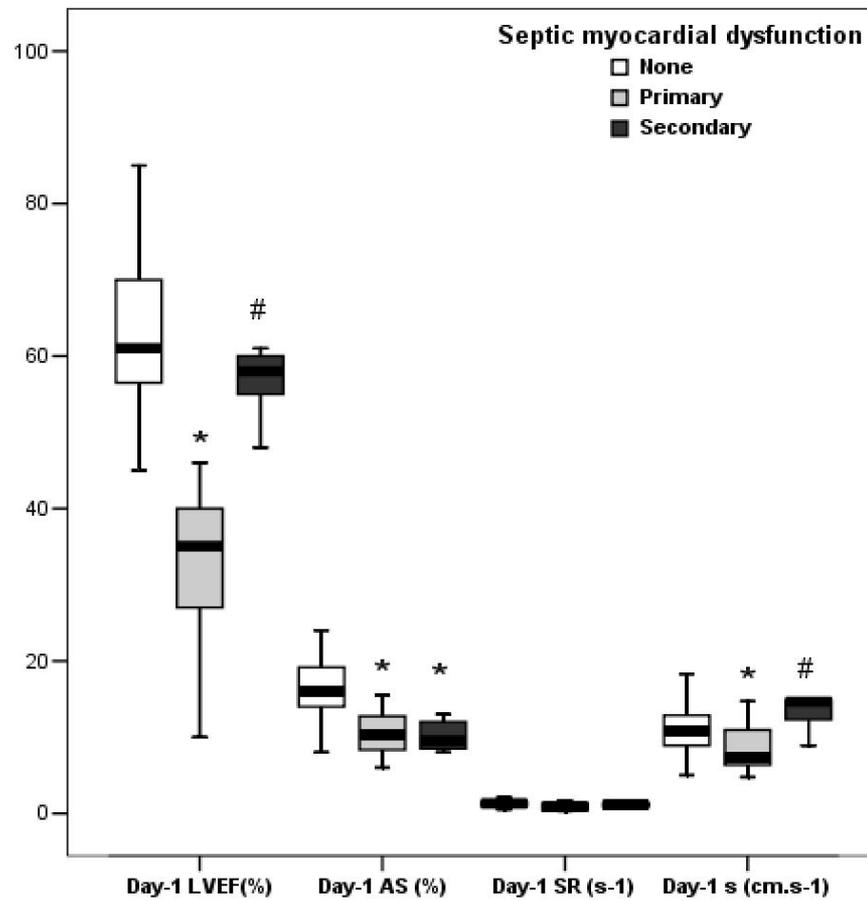
# L'écho est l'outil le plus adapté pour l'évaluation détaillée :

- Fonction globale
  - Systole vs. Diastole
  - VG vs. VD
  - Influence de l'hypovolémie
  - Influence de la vasoplégie
  - Classification pronostique
  - Optimisation thérapeutique ?
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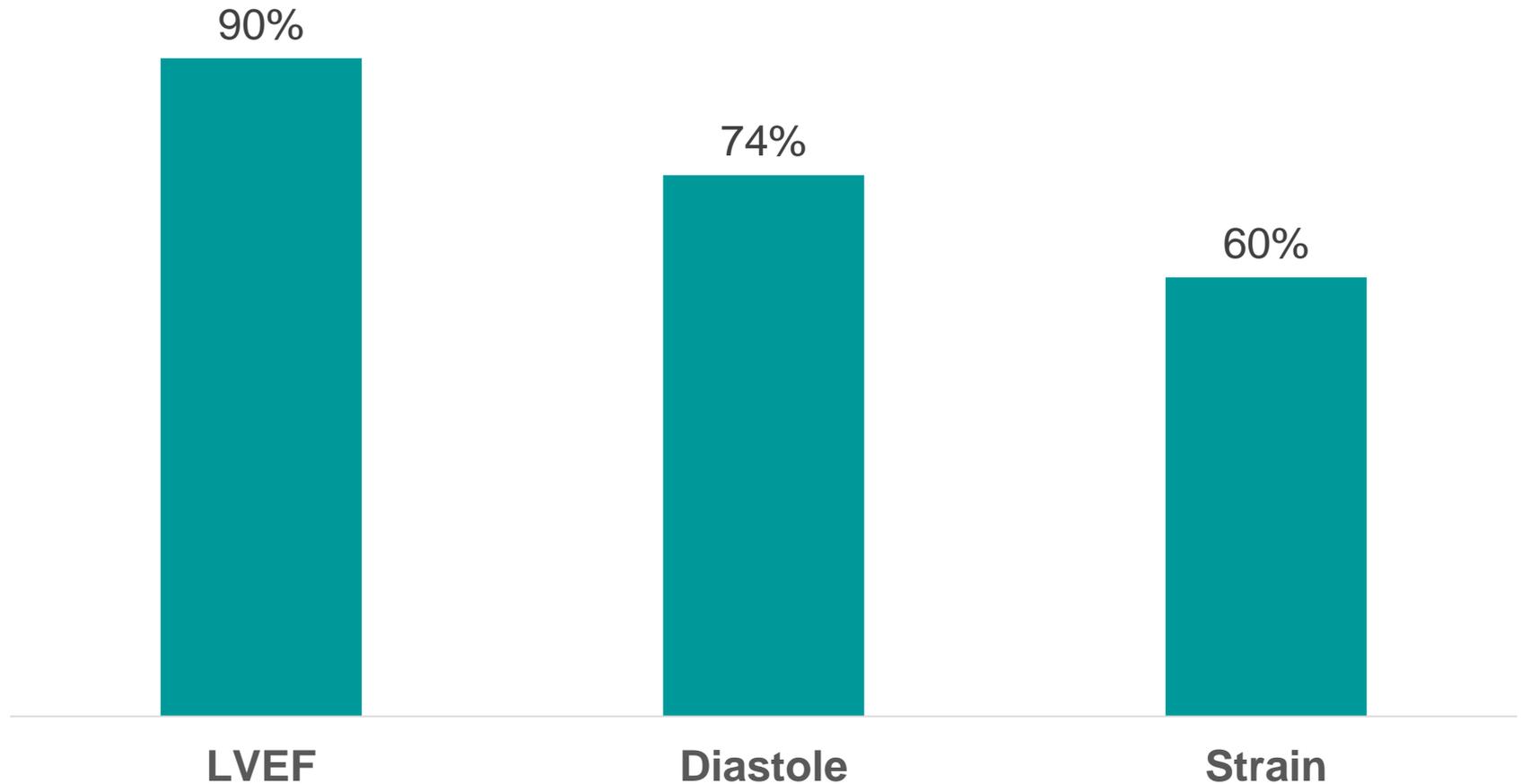
# Dépression myocardique intrinsèque chez la majorité des malades



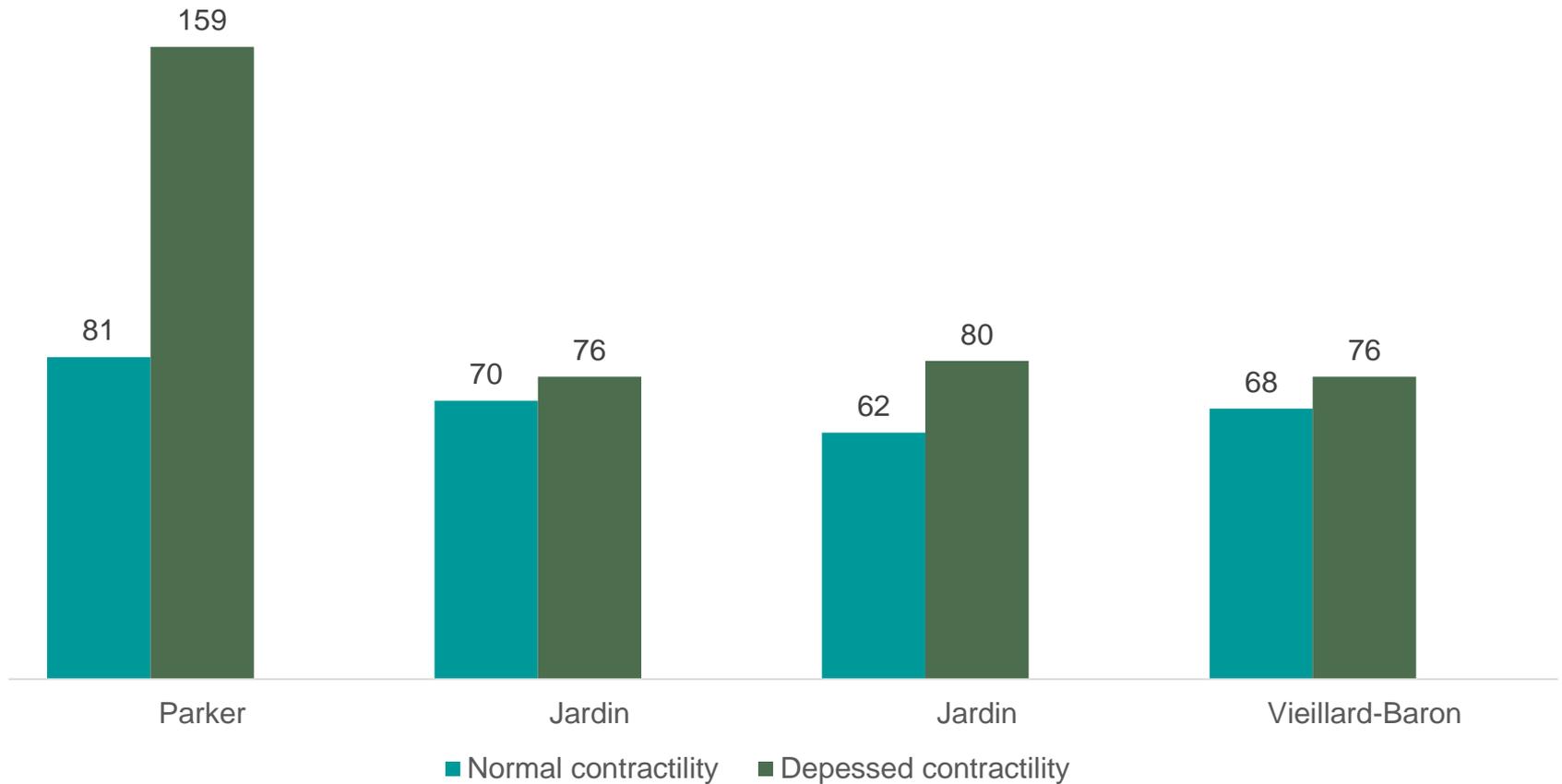
# Le strain pour la détection précoce de la dysfonction systolique



# Faisabilité de l'écho au cours du choc septique



# Dilatation (seulement) modérée du VG



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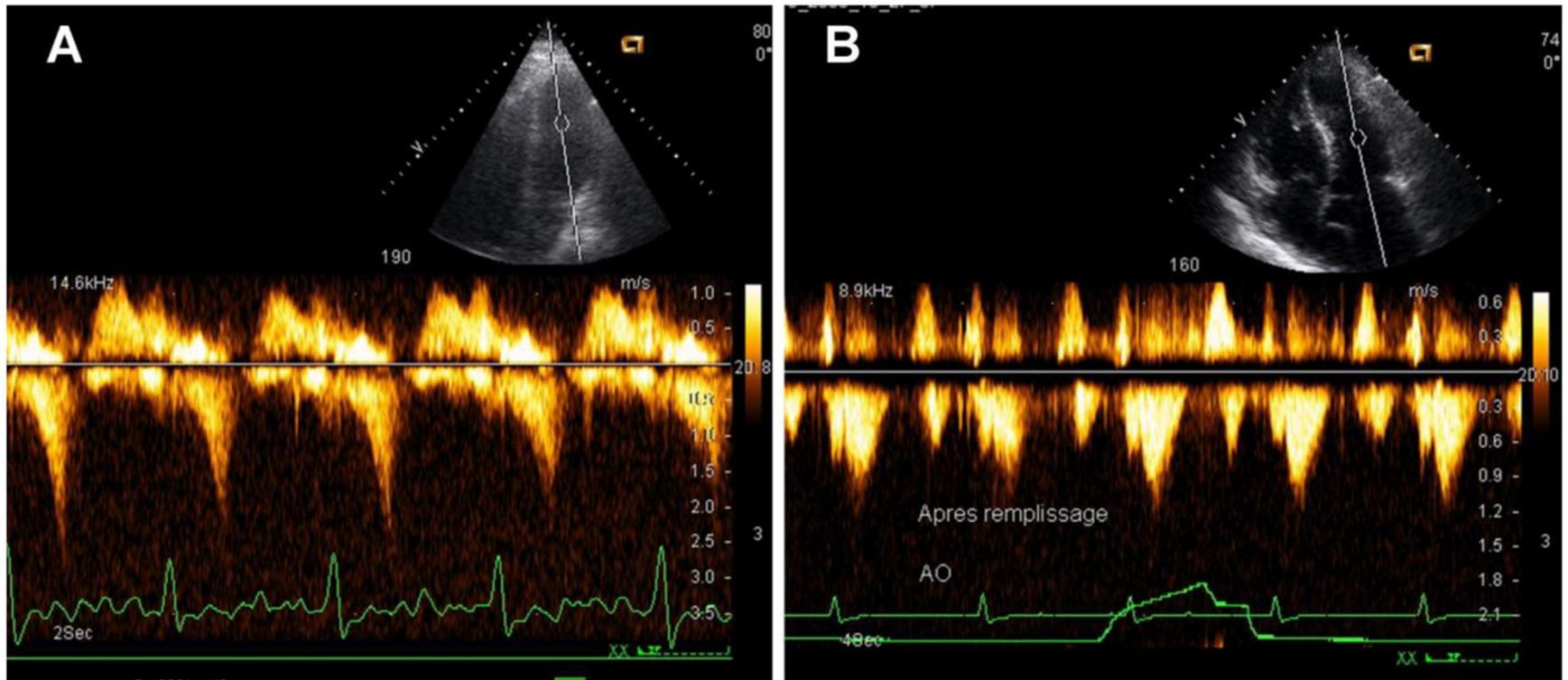
**PATHOPHYSIOLOGIE**  
**ROLE DES CONDITIONS DE CHARGE**

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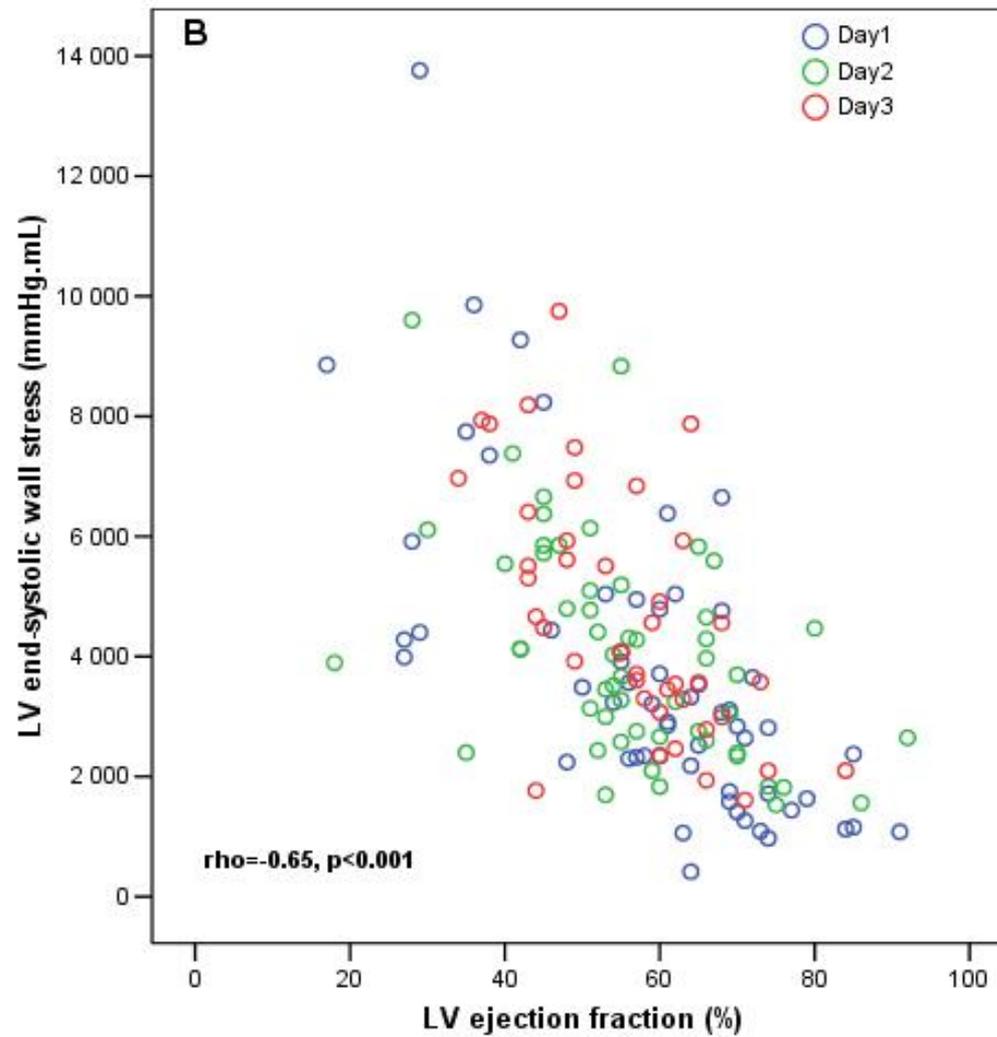
# Hypovolémie: pseudohypertrophie et exclusion systolique



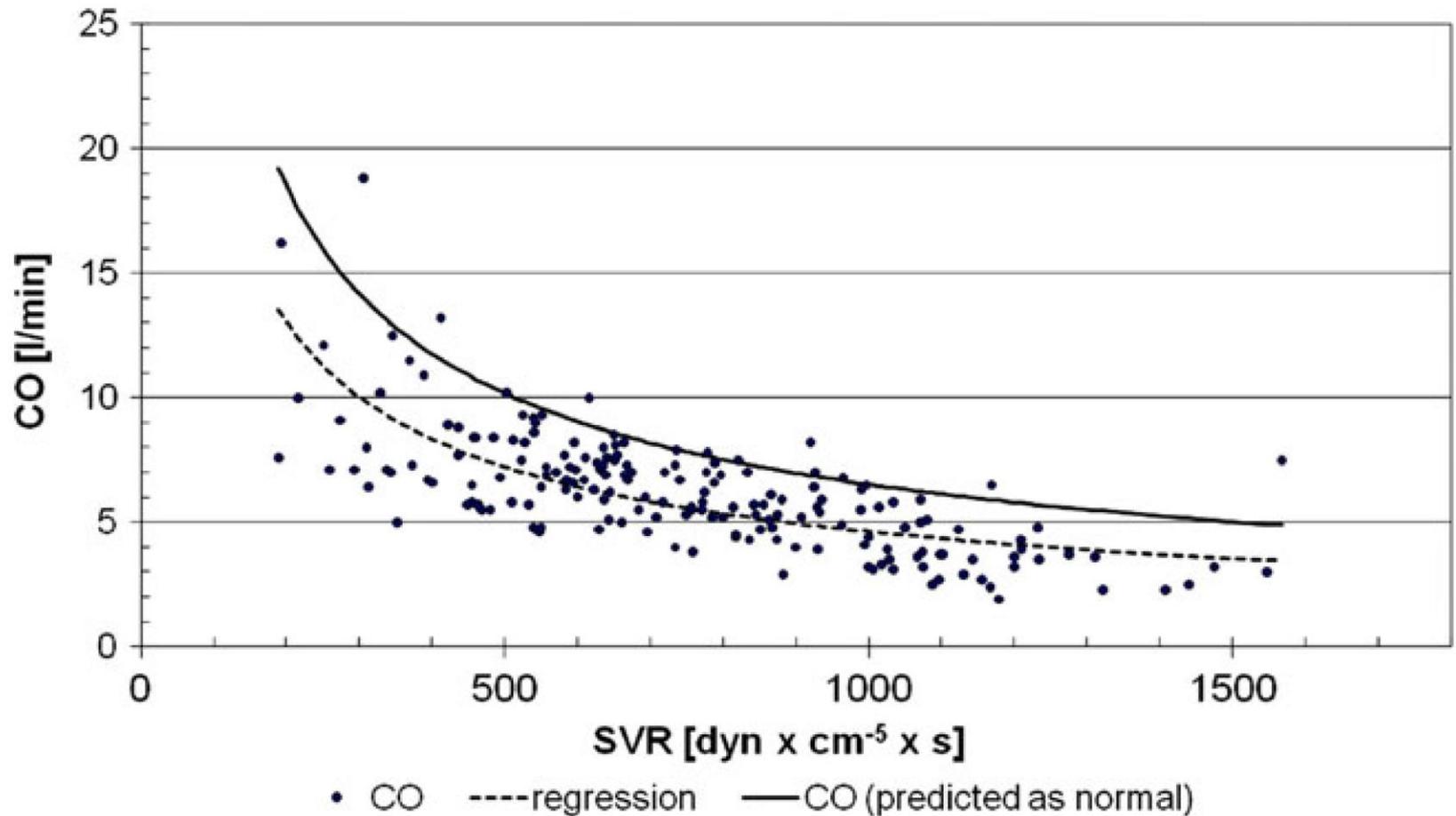
# Hypovolémie: obstruction intraventriculaire



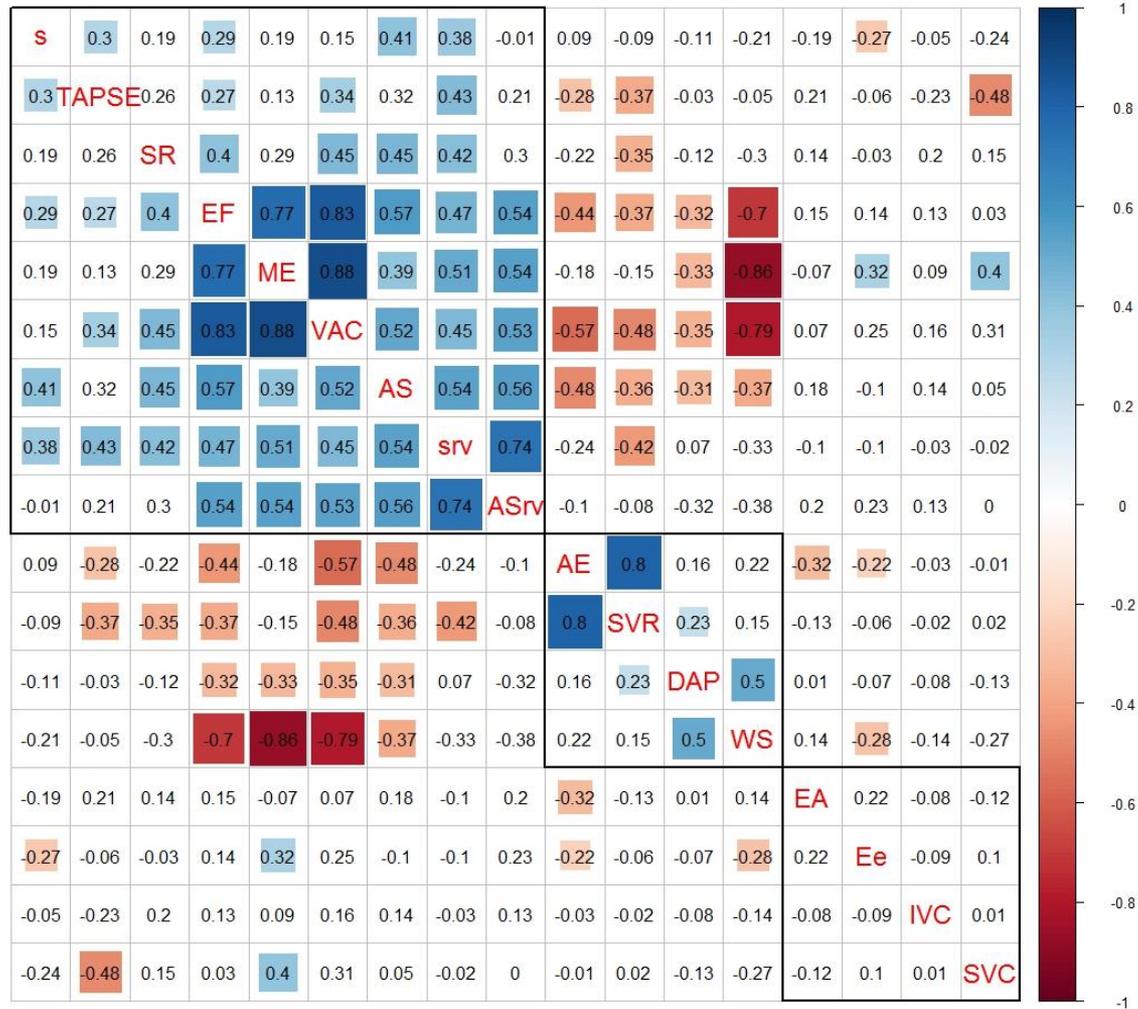
# Vasoplégie



# Vasoplégie



# Cohérence physiologique

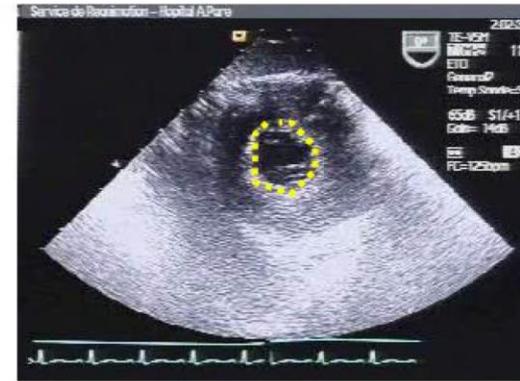
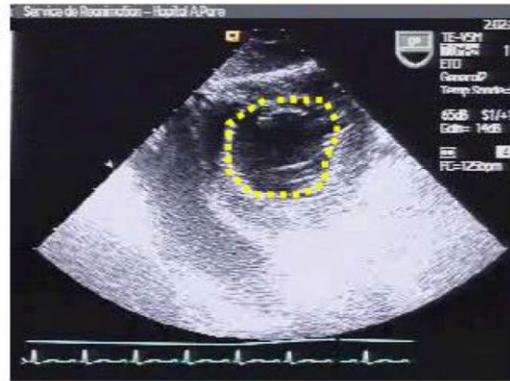


# Manipulation de la postcharge

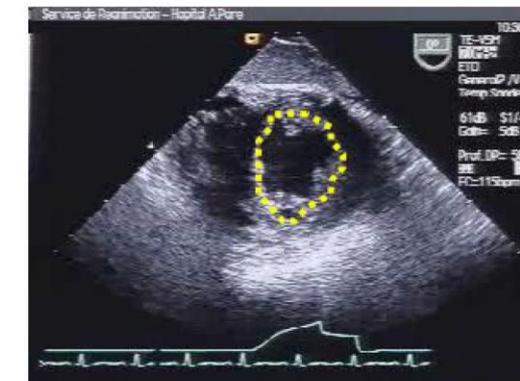
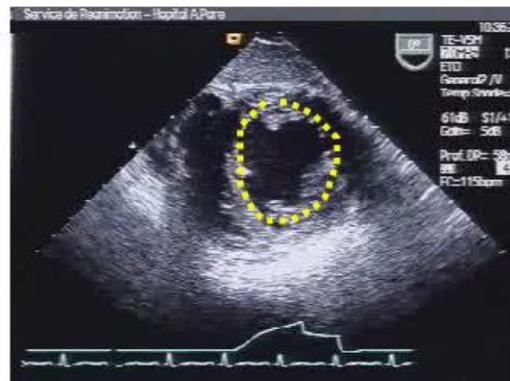
Diastole

Systole

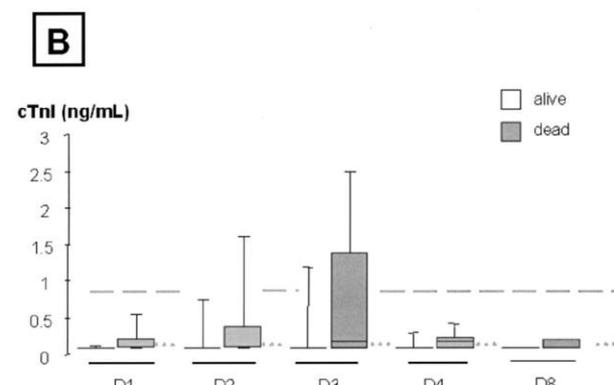
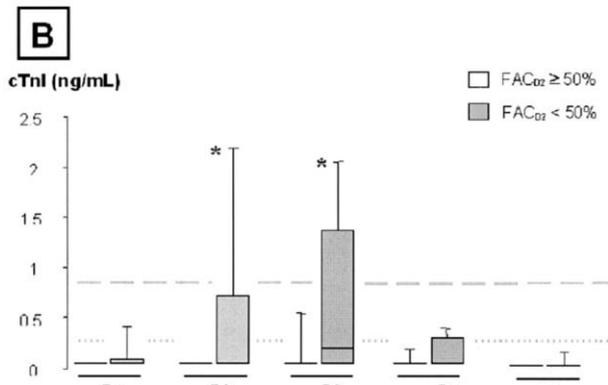
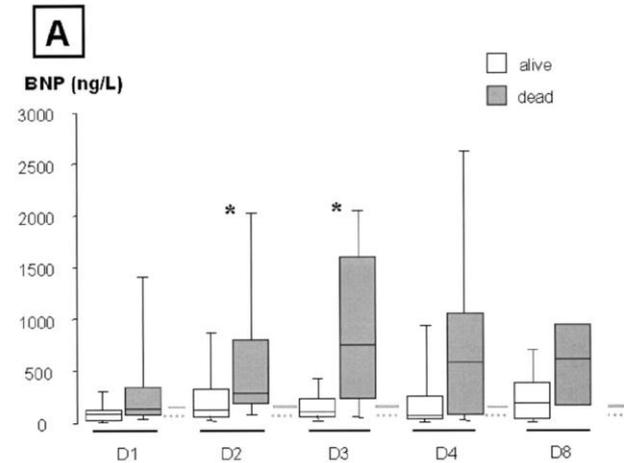
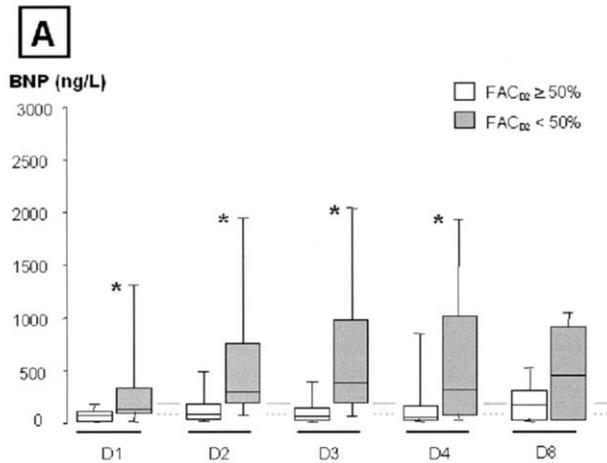
**Baseline**  
LVEF 70%



**Norepinephrine**  
LVEF 40%



# Place des biomarqueurs ?

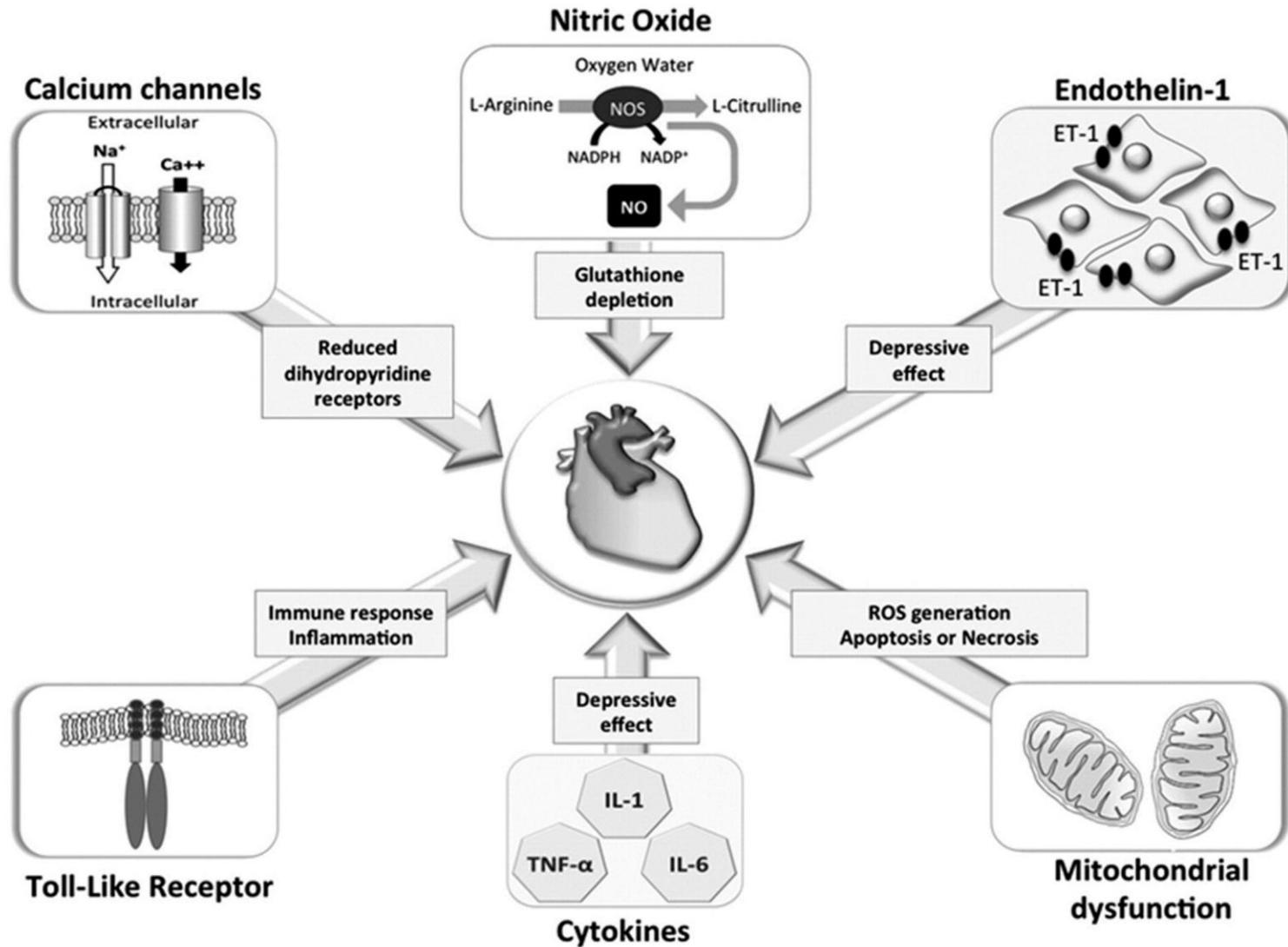


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# **PATHOBIOLOGIE**

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# Mécanismes complexes



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# **IMPLICATIONS CLINIQUES**

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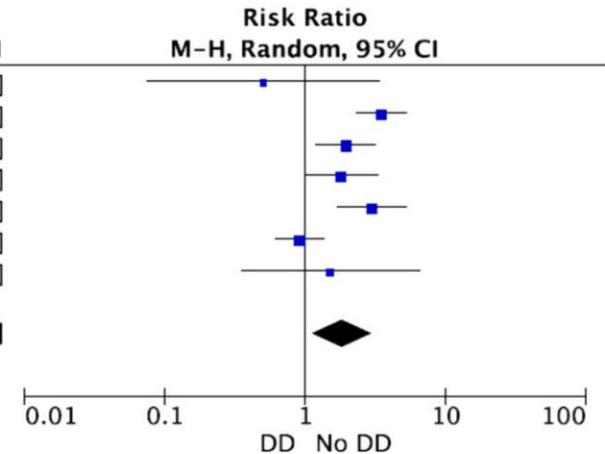
# Implications cliniques complexes

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

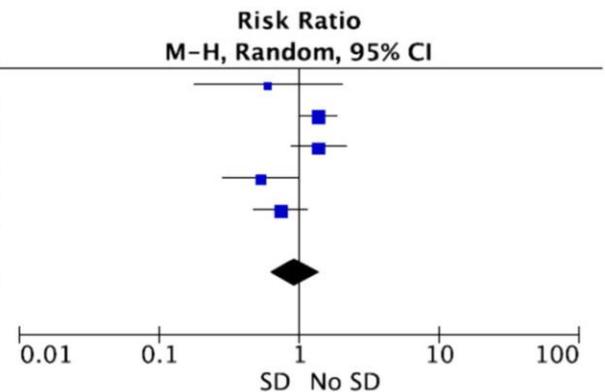
**a**

Study or Subgroup	DD		No DD		Weight	Risk Ratio M-H, Random, 95% CI
	Events	Total	Events	Total		
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]
<b>Total (95% CI)</b>		<b>305</b>		<b>331</b>	<b>100.0%</b>	<b>1.82 [1.12, 2.97]</b>
Total events	178		96			
Heterogeneity: Tau <sup>2</sup> = 0.29; Chi <sup>2</sup> = 26.42, df = 6 (P = 0.0002); I <sup>2</sup> = 77%						
Test for overall effect: Z = 2.40 (P = 0.02)						



**b**

Study or Subgroup	SD		No SD		Weight	Risk Ratio M-H, Random, 95% CI
	Events	Total	Events	Total		
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]
<b>Total (95% CI)</b>		<b>172</b>		<b>409</b>	<b>100.0%</b>	<b>0.93 [0.62, 1.39]</b>
Total events	72		176			
Heterogeneity: Tau <sup>2</sup> = 0.13; Chi <sup>2</sup> = 12.31, df = 4 (P = 0.02); I <sup>2</sup> = 68%						
Test for overall effect: Z = 0.34 (P = 0.73)						



# Optimisation du débit cardiaque chez des patients non sélectionnés

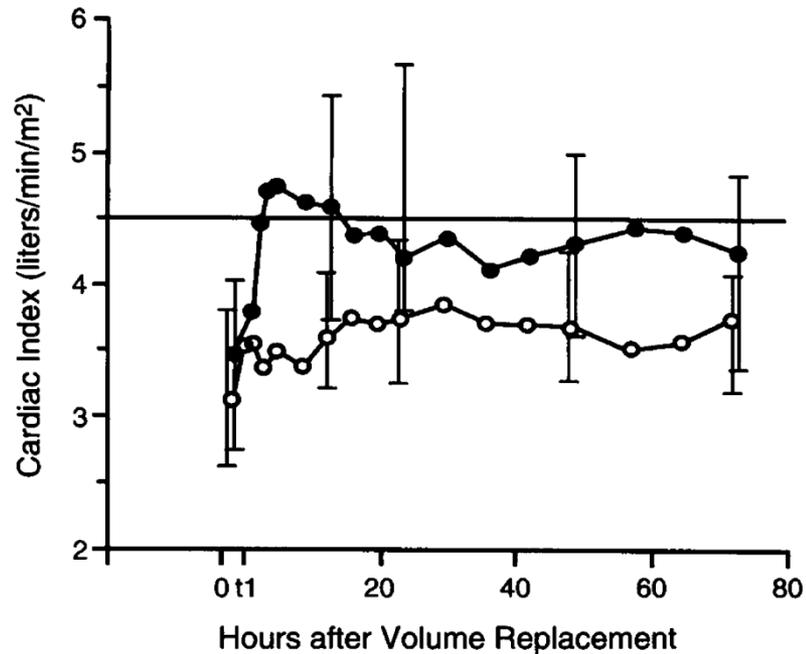


Figure 1. Median Cardiac Index in the Treatment and Control Groups.

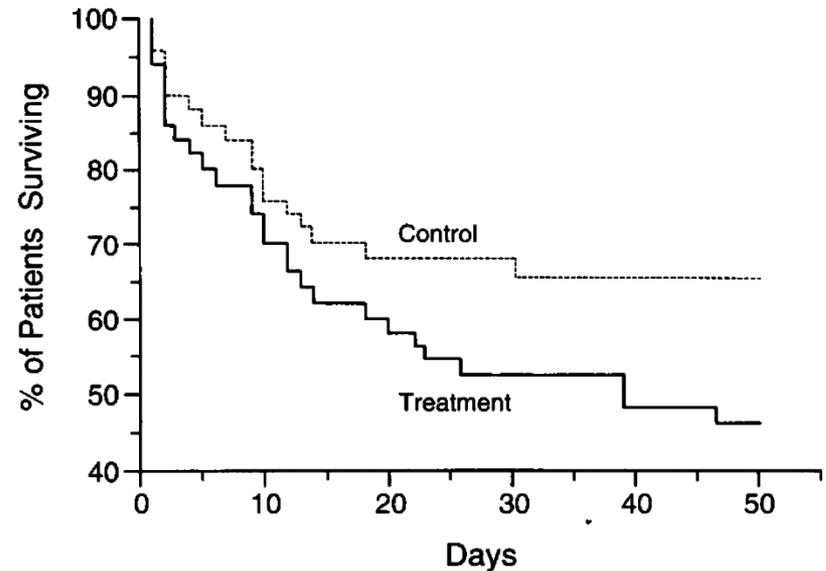
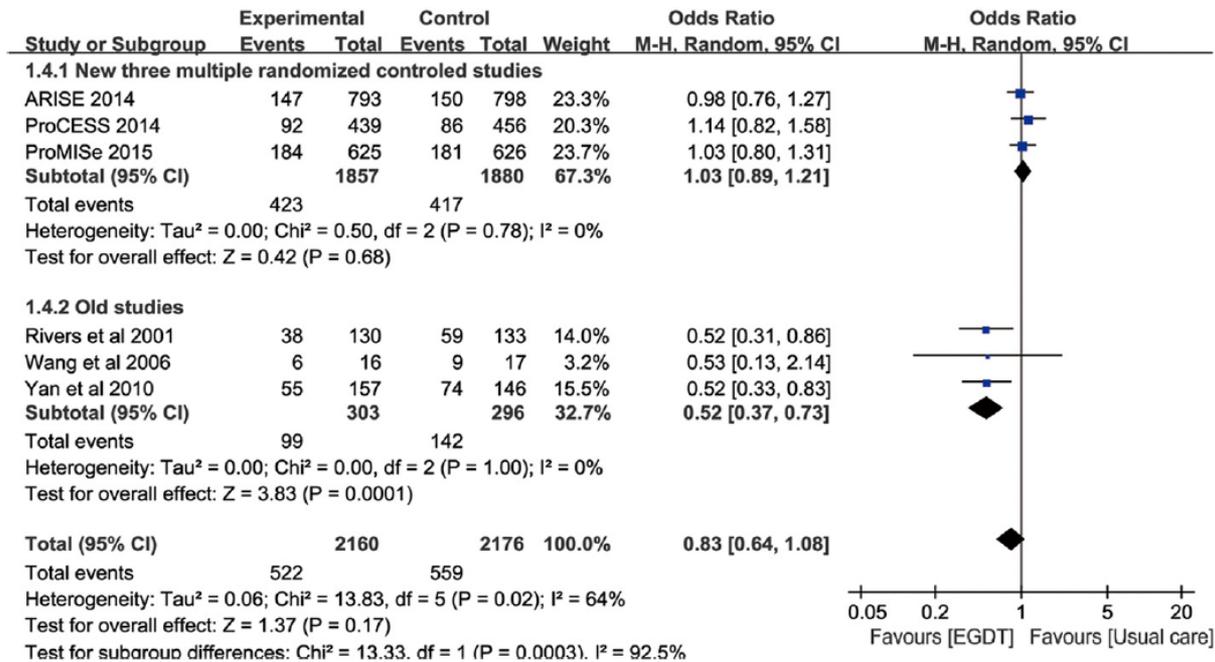


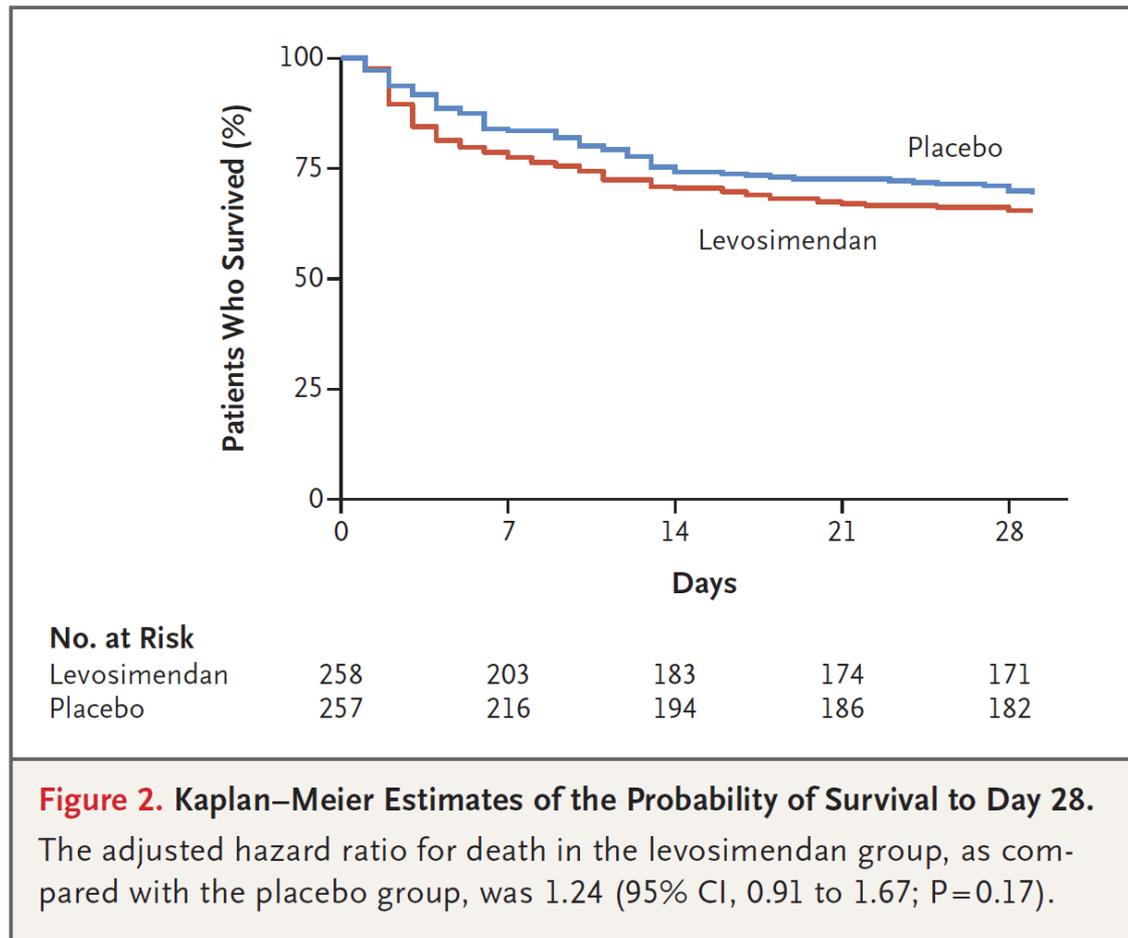
Figure 4. In-Hospital Survival of Patients in the Treatment and Control Groups.

# EGDT

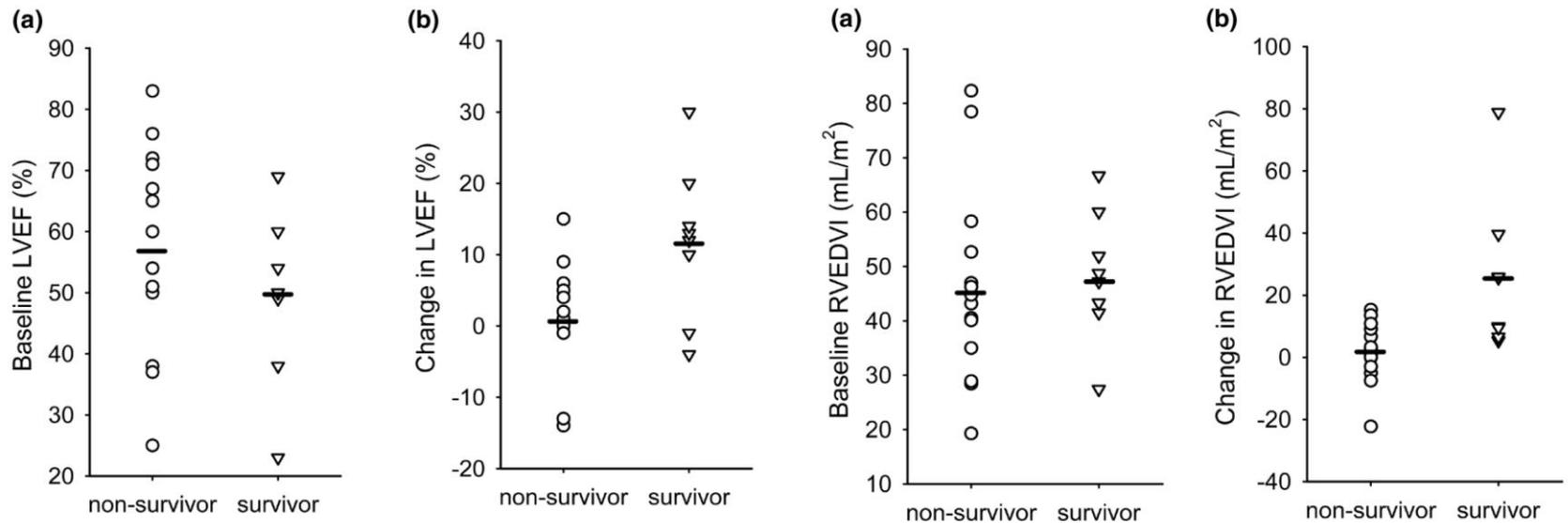


**Fig. 3** Forest plot showing the effects of early goal-directed therapy on all-cause mortality in patients with severe sepsis and septic shock

# Levosimendan chez des patients non sélectionnés



# Dobutamine stress test



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# 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 nécessaire pour la stratégie thérapeutique; role majeur de l'échocardiographie
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