

Actualités sur la prise en charge du choc septique

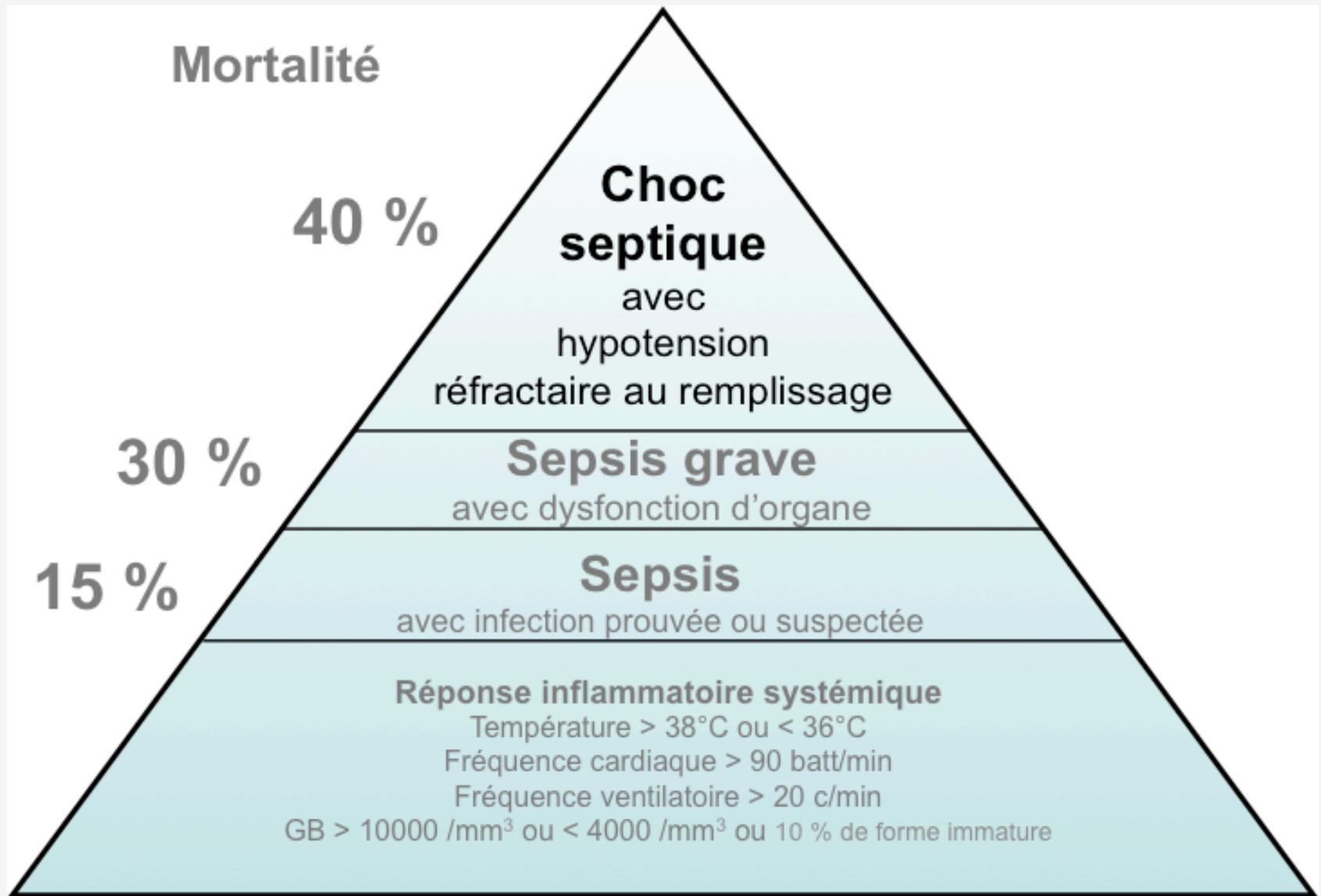
Antoine Roch

SAU- Réanimation DRIS

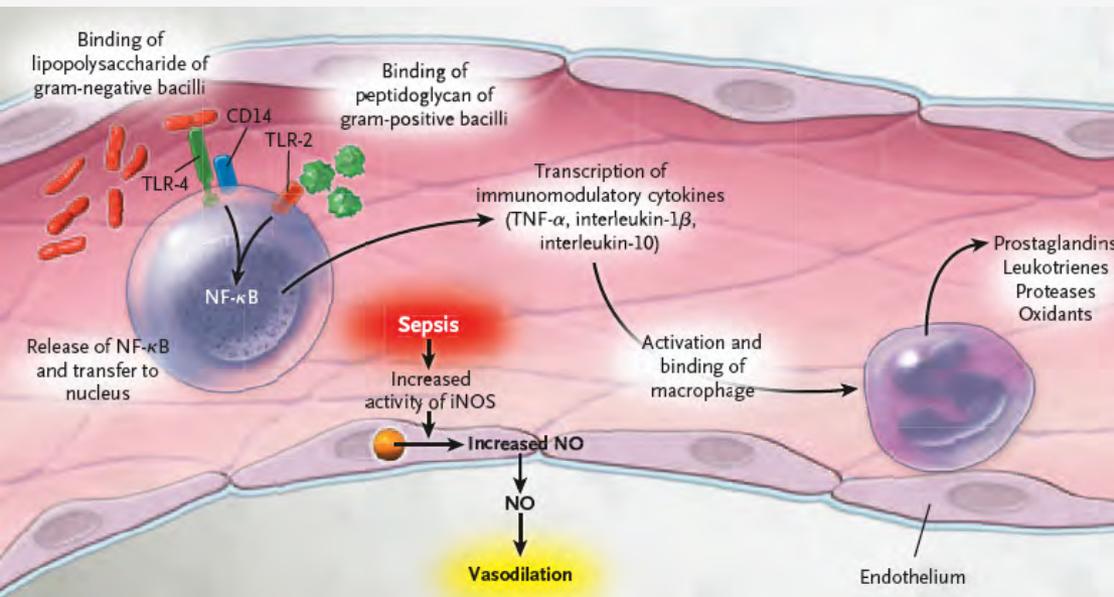
CHU Nord

Marseille

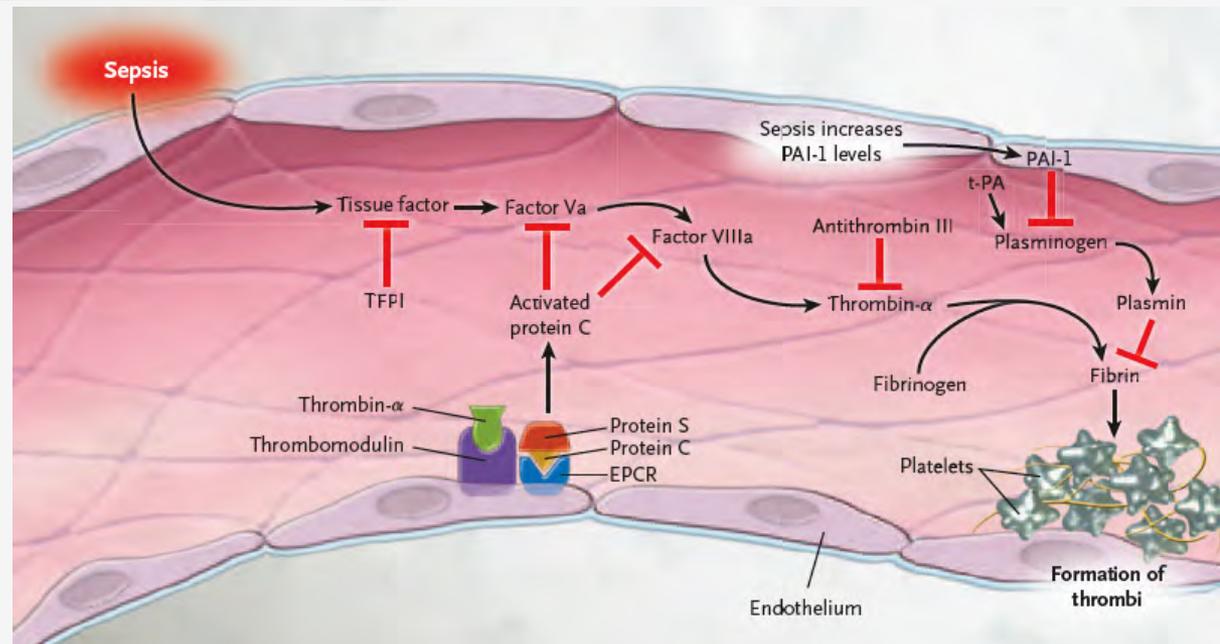




Physiopathologie



- Inflammation
- Atteinte endothéliale
- Tb coagulation



Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock, 2012

SURVIVING SEPSIS CAMPAIGN CARE BUNDLES

TO BE COMPLETED WITHIN 3 HOURS:

- 1) Measure lactate level
- 2) Obtain blood cultures prior to administration of antibiotics
- 3) Administer broad spectrum antibiotics
- 4) Administer 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

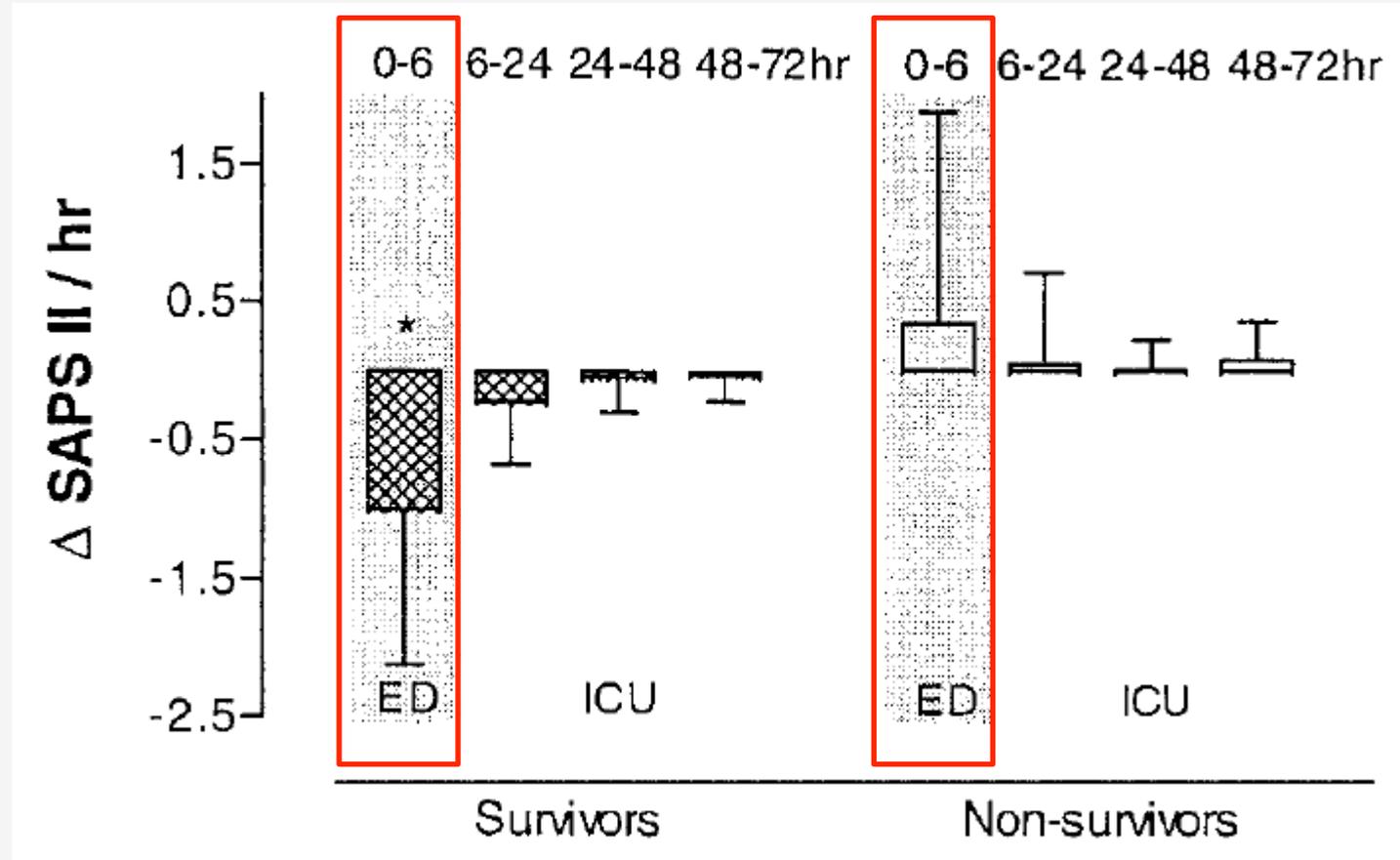
TO BE COMPLETED WITHIN 6 HOURS:

- 5) Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥ 65 mm Hg
- 6) In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate ≥ 4 mmol/L (36 mg/dL):
 - Measure central venous pressure (CVP)*
 - Measure central venous oxygen saturation (ScvO₂)*
- 7) Remeasure lactate if initial lactate was elevated*

*Targets for quantitative resuscitation included in the guidelines are CVP of ≥ 8 mm Hg, ScvO₂ of $\geq 70\%$, and normalization of lactate.

Critical Care in the Emergency Department: A Physiologic Assessment and Outcome Evaluation

ACADEMIC EMERGENCY MEDICINE • December 2000, Volume 7, Number 12



- Ne pas considérer que la rea va venir, donc on peut ne plus rien faire
- Ce n'est pas parce que qu'un patient s'améliore vite qu'il ne nécessite pas la rea par la suite



Prise en charge

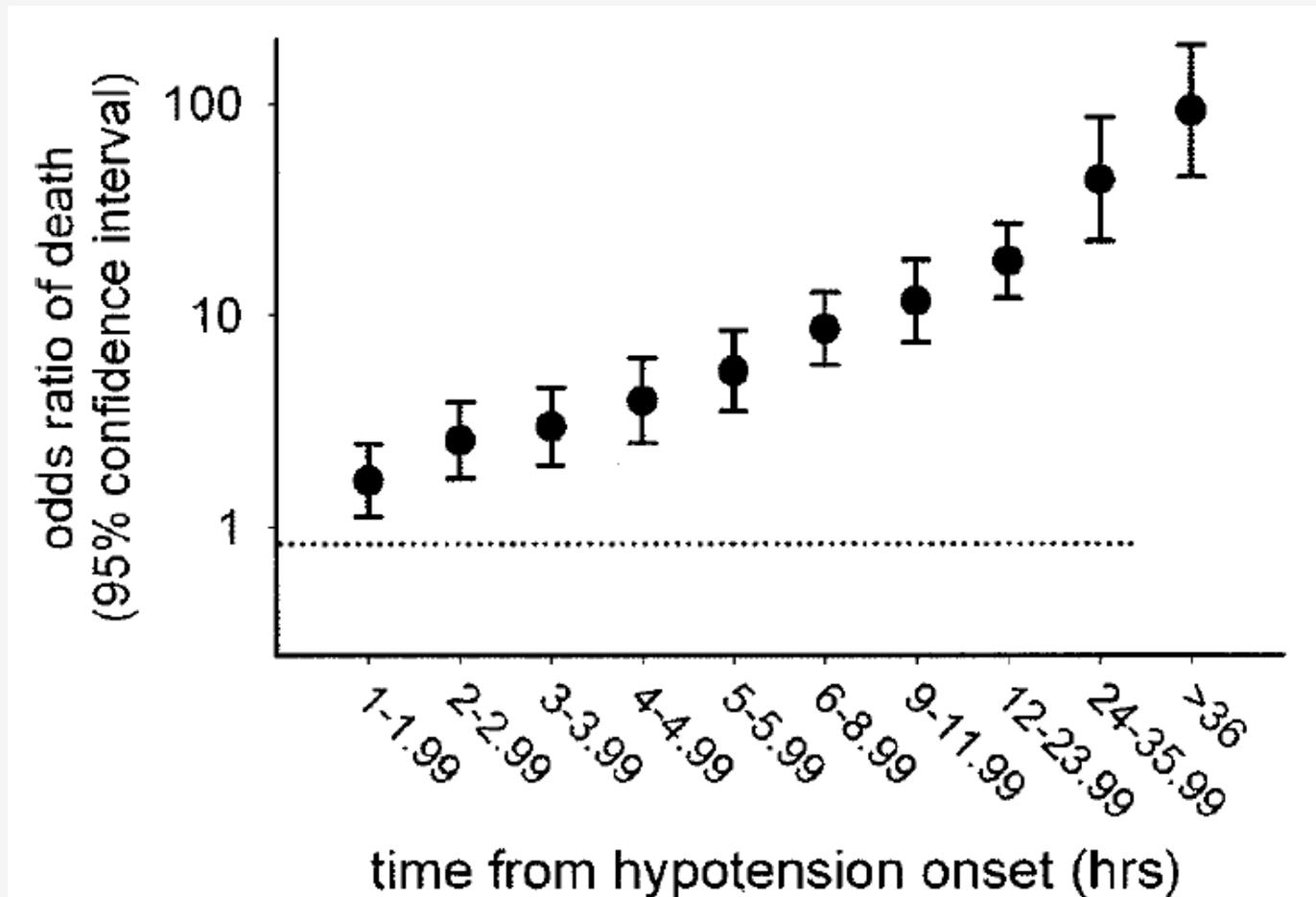
- **Arrêter le processus infectieux**
 - ▶ **Antibiothérapie (< 1 h du diagnostic)**
 - ▶ **Contrôle du foyer infectieux (6 h)**

Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock

Anand Kumar, MD; Daniel Roberts, MD; Kenneth E. Wood, DO; Bruce Light, MD; Joseph E. Parrillo, MD;

Crit Care Med 2006 Vol. 34, No. 6

- 2750 patients en choc septique, rétrospectif en réanimation





Antibiothérapie

Effect of Empirical Treatment With Moxifloxacin and Meropenem vs Meropenem on Sepsis-Related Organ Dysfunction in Patients With Severe Sepsis

No difference
SOFA score

**No effect of
combination**

Mortality D28

Mortality D90

No positive RCTs

Contrôle foyer infectieux

1C

- aussi rapidement que possible (6 h)
 - ▶ à rechercher chez tous les patients
 - ▶ imagerie rapide
 - ▶ retrait cathéter intravasculaire, PAC...





Prise en charge

- **Arrêter le processus infectieux**
 - ▶ Antibiothérapie (1 h)
 - ▶ Contrôle du foyer infectieux (6 h)

- **Conduire la réanimation hémodynamique**
 - ▶ Objectifs hémodynamiques (1 et 6 h)
 - ▶ Prise en charge (6 h)

Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock, 2012

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Objectifs hémodynamiques

■ « Survivre au sepsis »

▶ **PAM** \geq 65 mmHg (1 h)

▶ **Diurèse horaire** \geq 0,5 ml/kg/h (6 h)

▶ **PVC** : 8 - 12 mmHg ou **équivalent** (1 h)

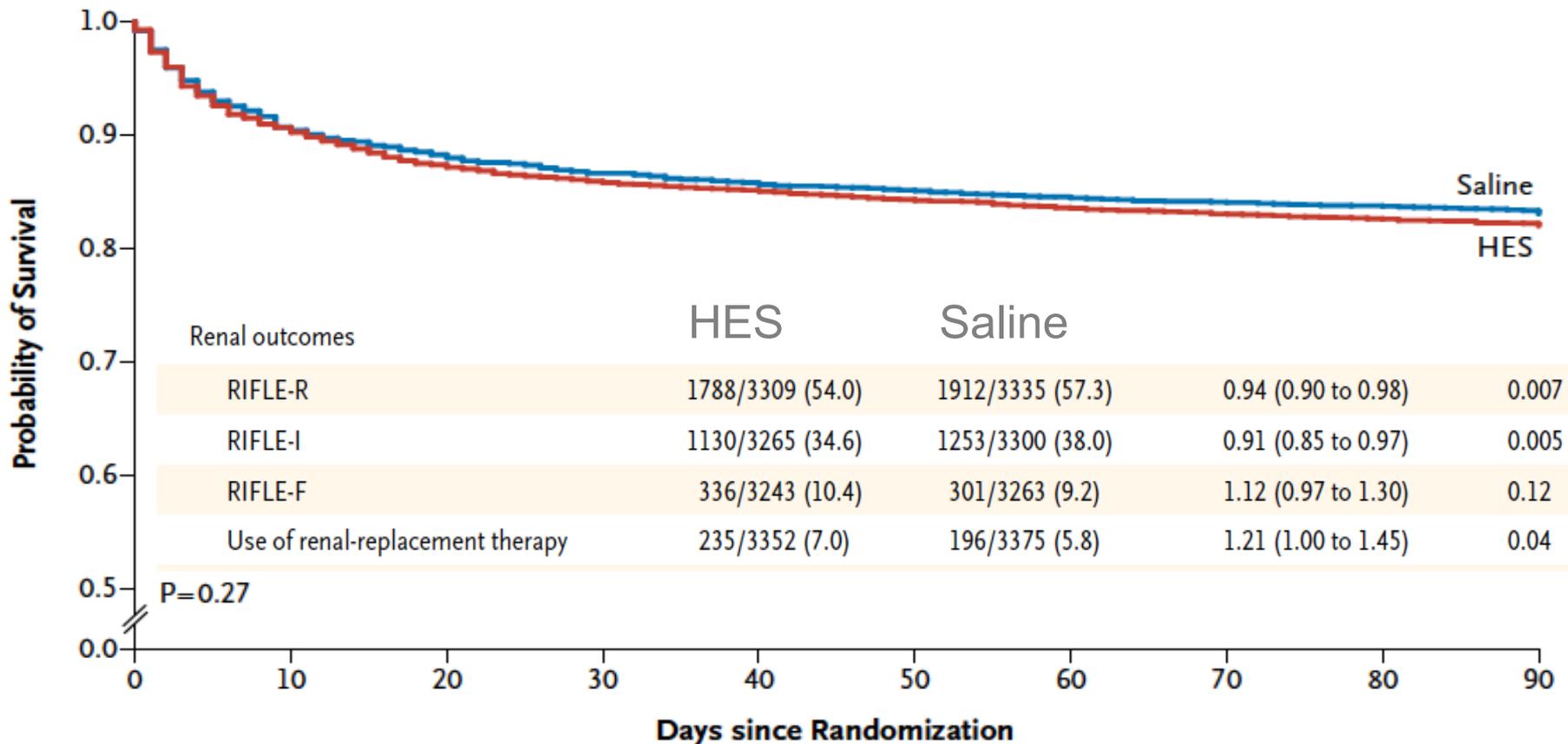


▶ **ScvO₂** \geq 70 % (6 h) **ET Lactate** < 20% (2h)

Remplissage vasculaire

■ Cristalloïde

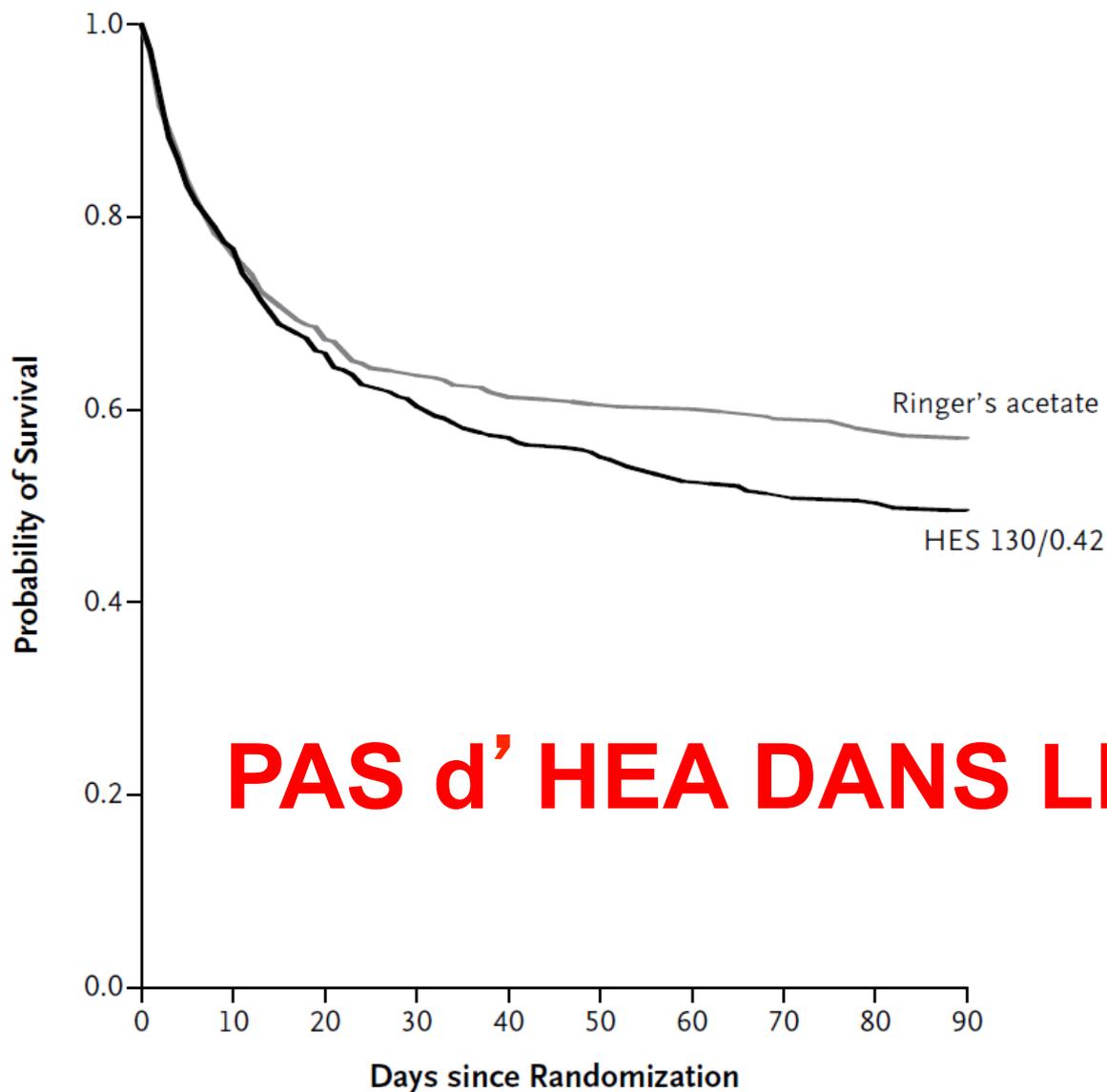
Hydroxyethyl Starch or Saline for Fluid Resuscitation in Intensive Care



Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis

N Engl J Med 2012;367:124-34

Anders Perner, M.D., Ph.D., Nicolai Haase, M.D.,

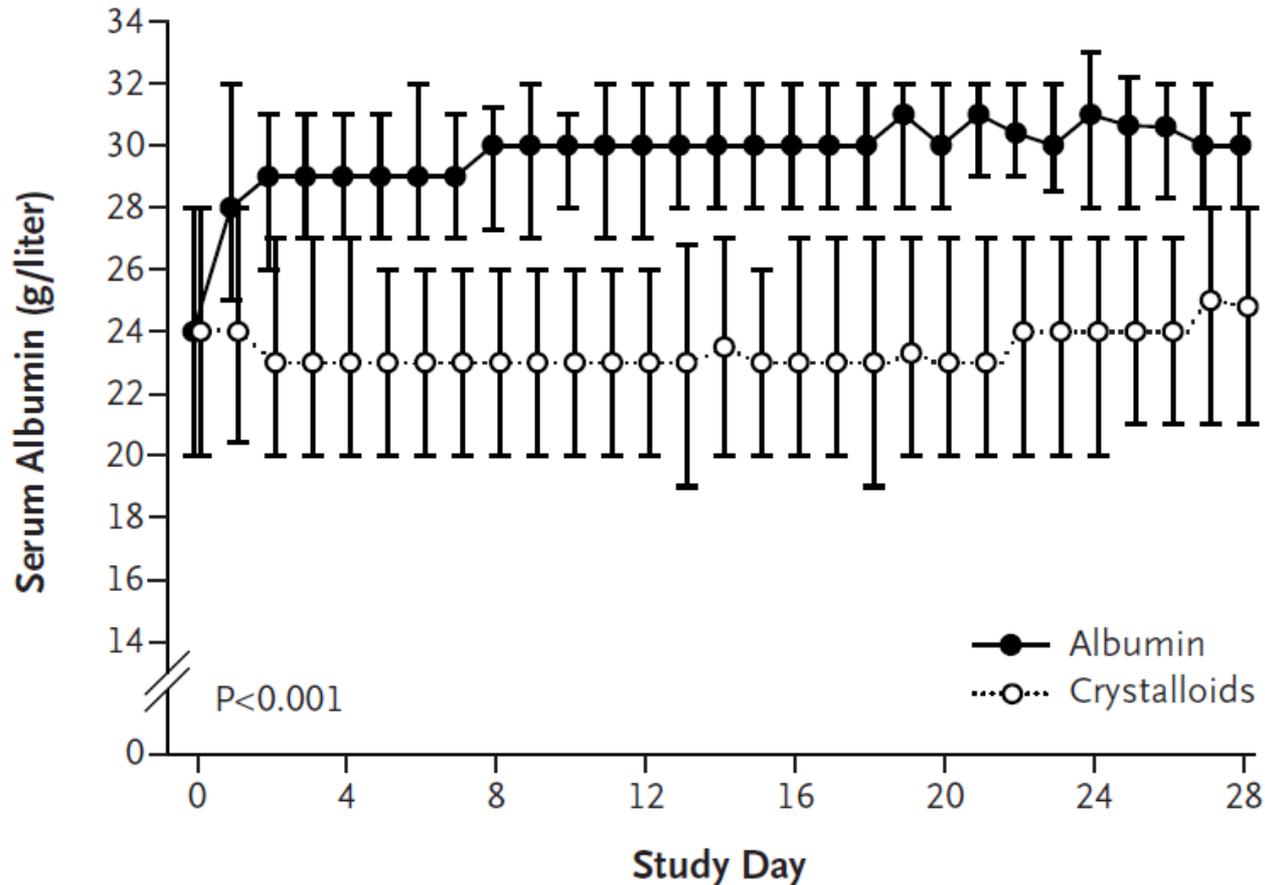


PAS d'HEA DANS LE SEPSIS

Albumin Replacement in Patients with Severe Sepsis or Septic Shock

NEJM march 18, 2014

- 1800 patients
- albumine si > 30 g/l vs si < 20 g/l



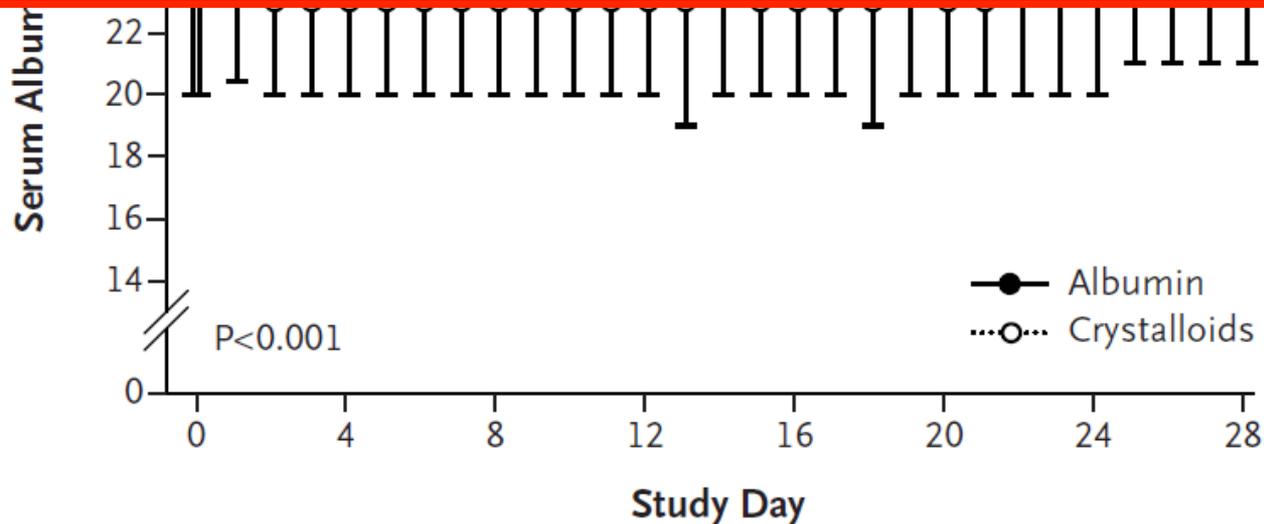
Albumin Replacement in Patients with Severe Sepsis or Septic Shock

NEJM march 18, 2014

- 1800 patients
- albumine si > 30 g/l vs si < 20 g/l

34

Outcome	Albumin Group	Crystalloid Group	Relative Risk (95% CI)	P Value
Primary outcome: death at 28 days — no./total no. (%)	285/895 (31.8)	288/900 (32.0)	1.00 (0.87–1.14)	0.94
Secondary outcomes				
Death at 90 days — no./total no. (%)	365/888 (41.1)	389/893 (43.6)	0.94 (0.85–1.05)	0.29
New organ failures — no./total no. (%)*				0.99

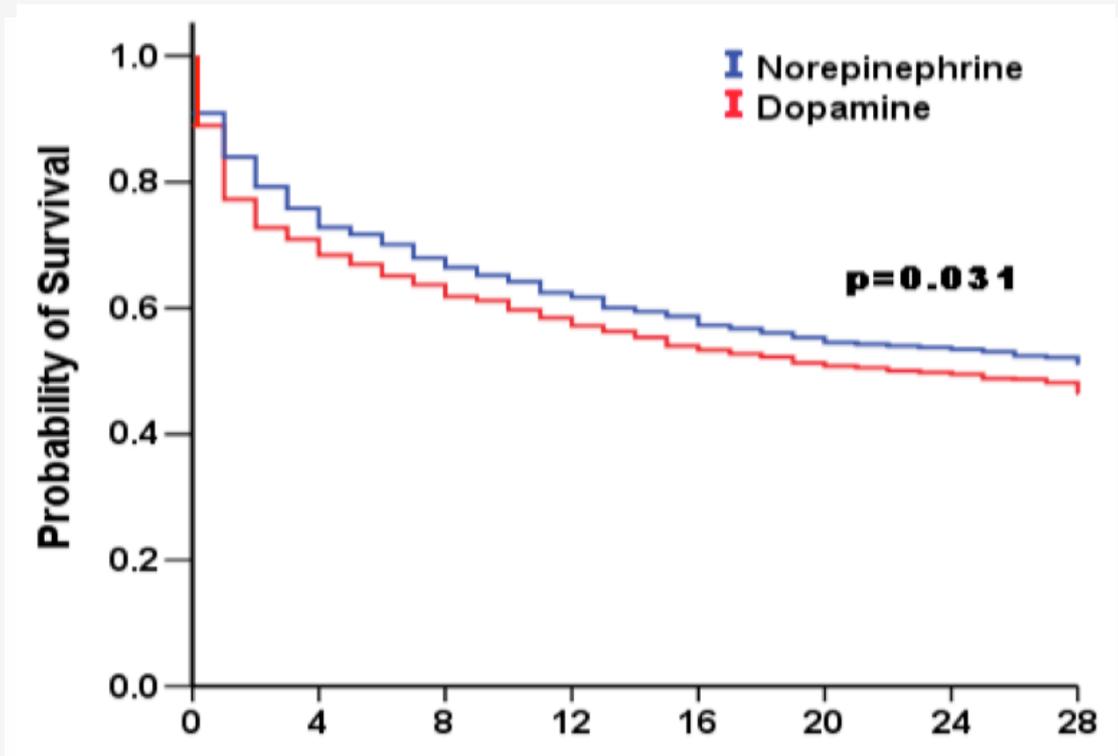


Vasopresseurs

■ Noradrénaline

Dellinger *et al.*, Intensive Care Med 2013

▶ Dopamine : ↑ échec clinique et effets secondaires



Arythmie

24 % vs. 12 %

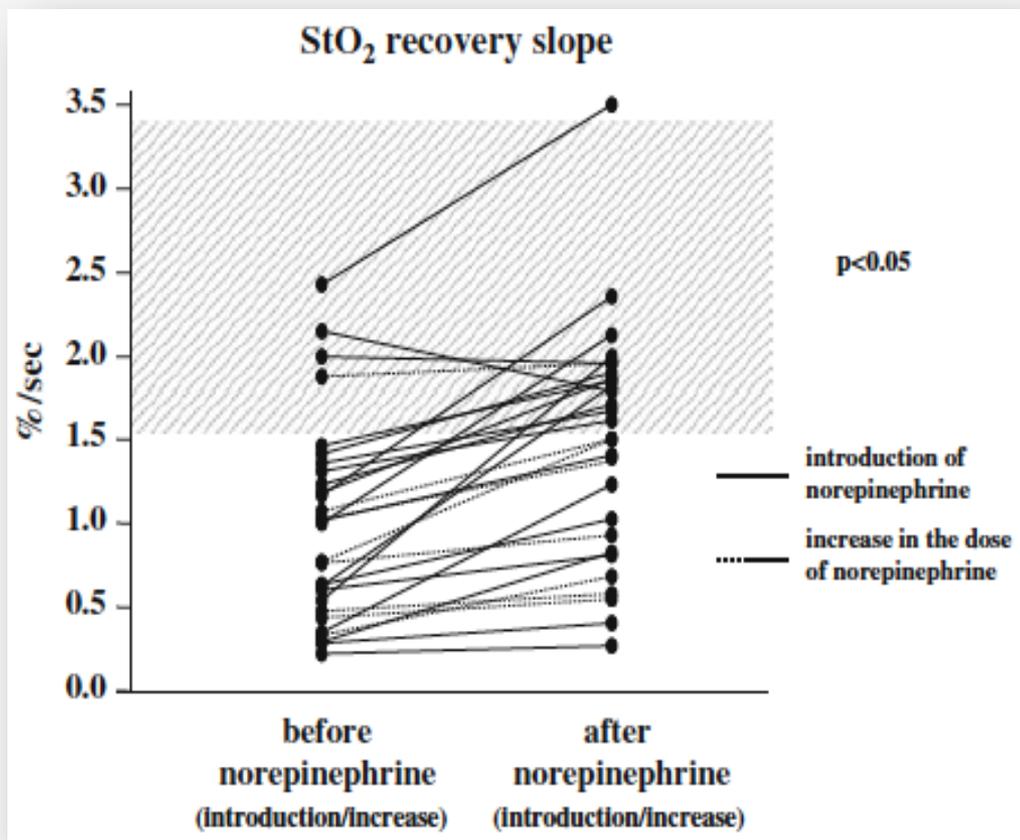
$p < 0,001$

Noradrénaline

Objectifs hémodynamiques

■ PAM ≥ 65 mmHg

Restoring arterial pressure with norepinephrine improves muscle tissue oxygenation assessed by near-infrared spectroscopy in severely hypotensive septic patients

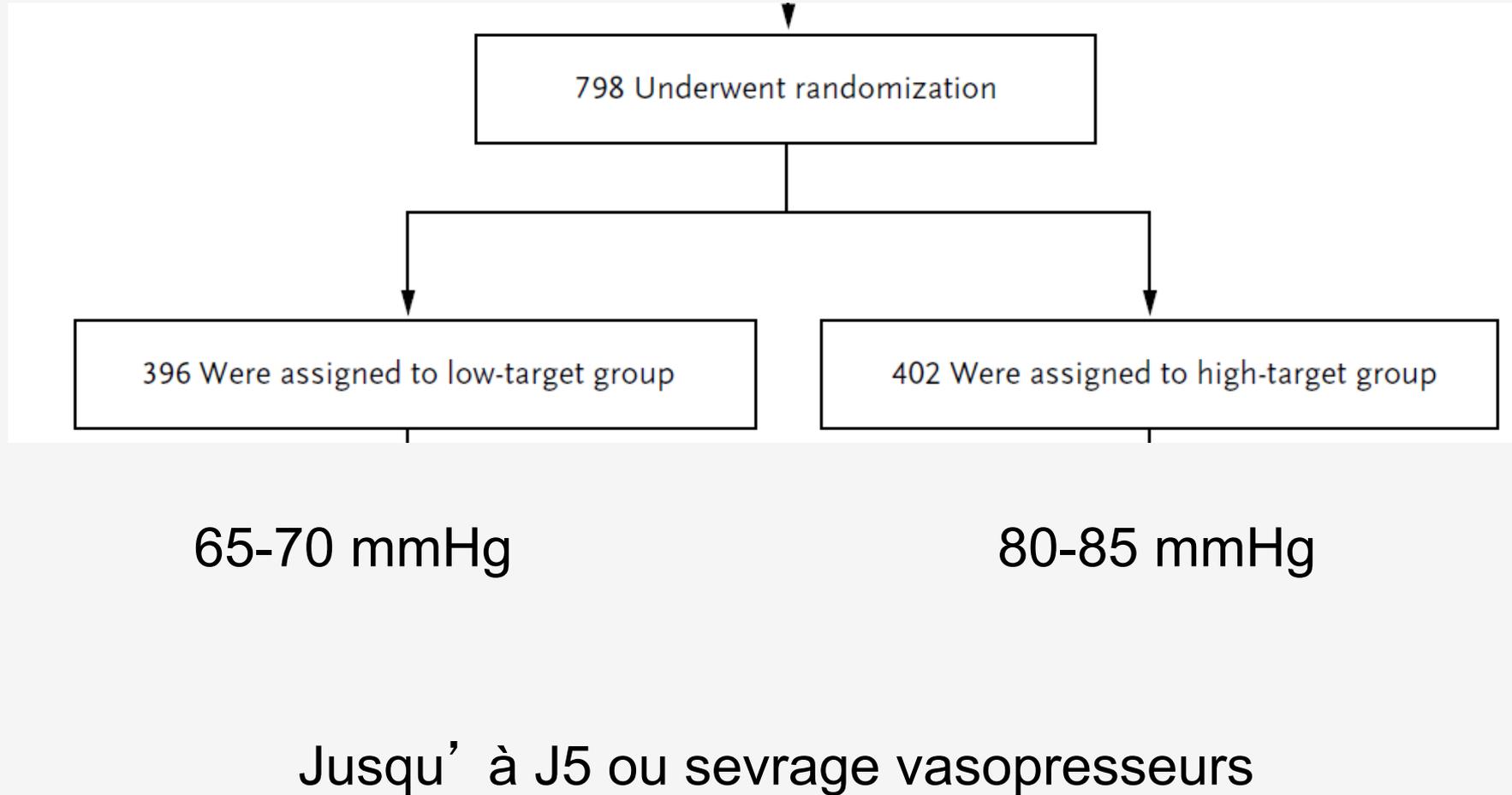


MAP
 54 ± 8 mmHg
vs.
 77 ± 9 mmHg

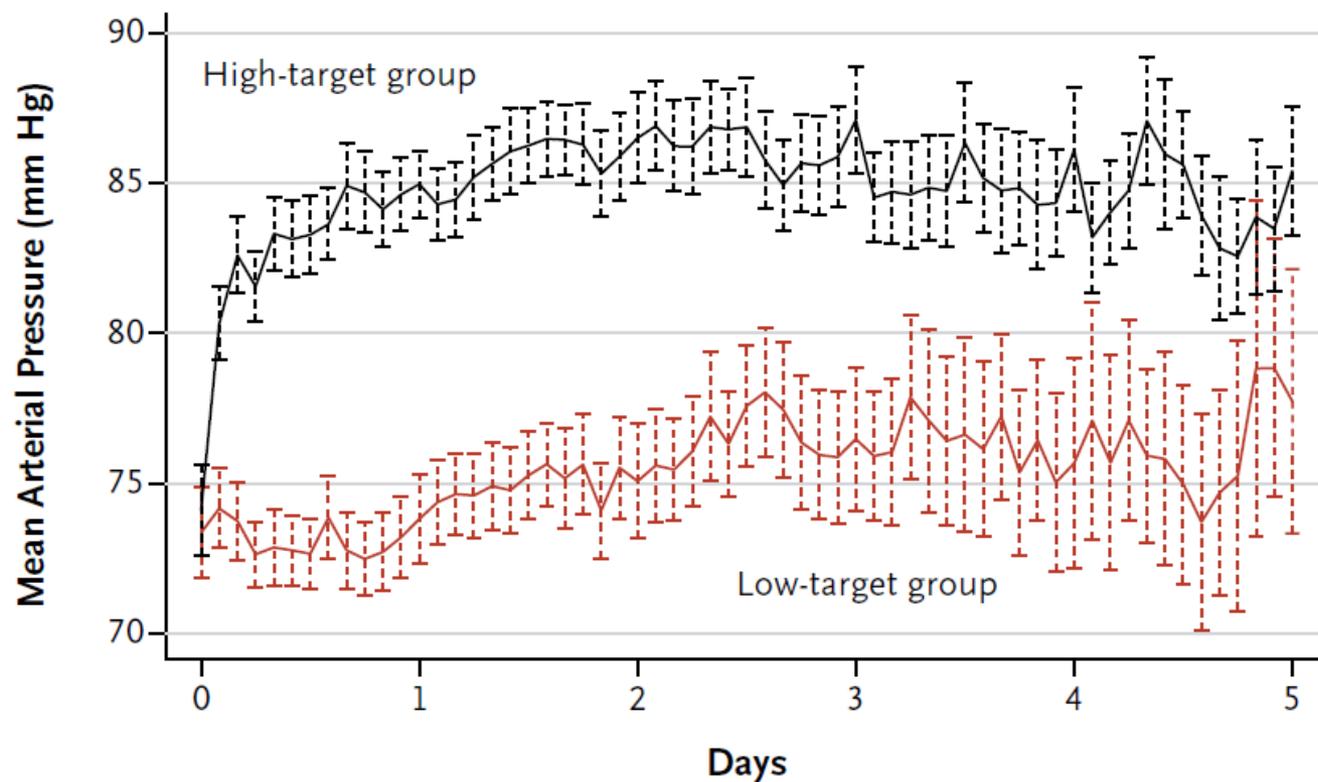
High versus Low Blood-Pressure Target in Patients with Septic Shock

NEJM march 18, 2014

Pierre Asfar, M.D., Ph.D., Ferhat Meziani, M.D., Ph.D., Jean-François Hamel, M.D.,



Characteristic	Low-Target Group (N=388)	High-Target Group (N=388)
Vasoactive drug infusions at randomization — no. (%)		
Norepinephrine	368 (94.8)	373 (96.1)
Epinephrine	20 (5.2)	15 (3.9)
Dobutamine	21 (5.4)	16 (4.1)
Median vasopressor dose at randomization — $\mu\text{g}/\text{kg}/\text{min}$ (IQR)		
Norepinephrine	0.35 (0.20–0.61)	0.40 (0.20–0.62)
Epinephrine	0.23 (0.17–0.32)	0.22 (0.13–0.64)
Mechanical ventilation — no. (%)	286 (73.7)	308 (79.4)



High versus Low Blood-Pressure Target in Patients with Septic Shock

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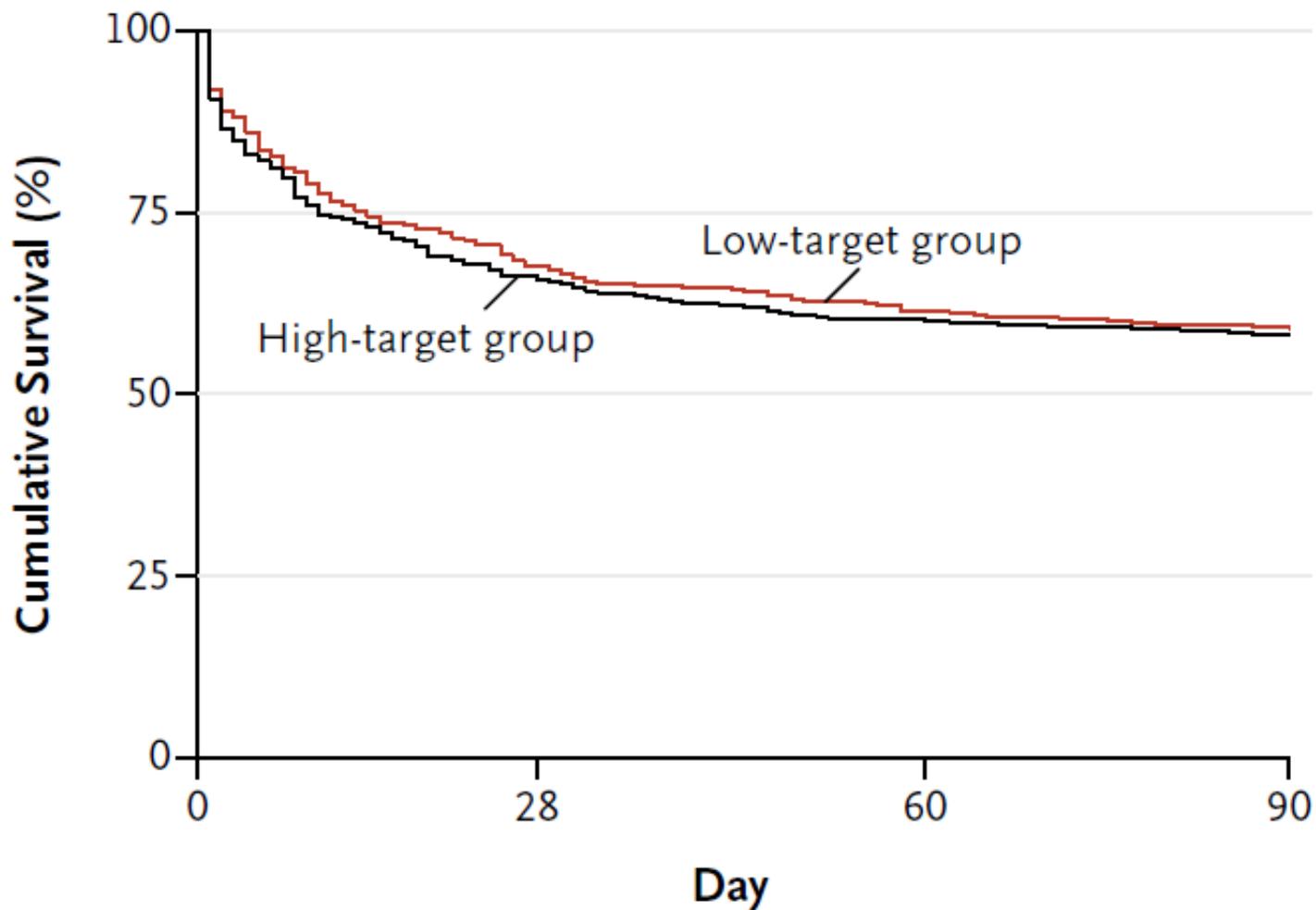


Table 2. Clinical Results, Primary and Secondary Outcomes, and Serious Adverse Events.

Variable	Low-Target Group (N = 388)	High-Target Group (N = 388)	P Value
Cumulative fluid intake from day 1 to day 5 — liters	10.0 (5.8–14.0)	10.5 (5.5–14.0)	0.89
Cumulative urine output from day 1 to day 5 — liters	6.7 (2.9–10.7)	6.9 (2.4–10.7)	0.87
Cumulative fluid balance from day 1 to day 5 — liters	2.8 (0.0–6.2)	2.4 (0.0–6.0)	0.74
Median dose of norepinephrine (IQR) — $\mu\text{g}/\text{kg}/\text{min}$			
Day 1	0.45 (0.17–1.21)	0.58 (0.26–1.80)	<0.001
Day 2	0.16 (0.03–0.48)	0.38 (0.14–0.90)	<0.001
Day 3	0.02 (0.00–0.16)	0.14 (0.01–0.50)	<0.001
Day 4	0.00 (0.00–0.05)	0.03 (0.00–0.22)	<0.001
Day 5	0.00 (0.00–0.03)	0.01 (0.00–0.15)	<0.001
Duration of catecholamine infusion — days	3.7 \pm 3.2	4.7 \pm 3.7	<0.001
Primary outcome: death at day 28 — no. (%) [*]	132 (34.0)	142 (36.6)	0.57
Secondary outcomes — no./total no. (%)			
Death at day 90 [†]	164 (42.3)	170 (43.8)	0.74
Survival at day 28 without organ support [‡]	241 (62.1)	235 (60.6)	0.66
Doubling of plasma creatinine	161 (41.5)	150 (38.7)	0.42
No chronic hypertension	71/215 (33.0)	85/221 (38.5)	0.32
Chronic hypertension	90/173 (52.0)	65/167 (38.9)	0.02
Renal-replacement therapy from day 1 to day 7	139 (35.8)	130 (33.5)	0.50
No chronic hypertension	66/215 (30.7)	77/221 (34.8)	0.36
Chronic hypertension	73/173 (42.2)	53/167 (31.7)	0.046
Serious adverse events — no. (%)			
Any	69 (17.8)	74 (19.1)	0.64
Acute myocardial infarction [§]	2 (0.5)	7 (1.8)	0.18
Atrial fibrillation	11 (2.8)	26 (6.7)	0.02
Ventricular fibrillation or tachycardia	15 (3.9)	22 (5.7)	0.24
Digital ischemia	9 (2.3)	10 (2.6)	0.82
Mesenteric ischemia	9 (2.3)	9 (2.3)	1.00
Bleeding	42 (10.8)	31 (8.0)	0.22

Objectifs hémodynamiques

■ « Survivre au sepsis »

▶ **PAM** \geq 65 mmHg (1 h)

▶ **Diurèse horaire** \geq 0,5 ml/kg/h (6 h)

▶ **PVC** : 8 - 12 mmHg ou **équivalent** (1 h)



▶ **ScvO₂** \geq 70 % (6 h) **ET Lactate** < 20% (2h)

Optimisation hémodynamique

Rivers, New Engl J Med, 2001

TREATMENT	HOURS AFTER THE START OF THERAPY		
	0-6	7-72	0-72
Total fluids (ml)			
Standard therapy	3499±2438	10,602±6,216	13,358±7,725
EGDT	4981±2984	8,625±5,162	13,443±6,390
P value	<0.001	0.01	0.73
Red-cell transfusion (%)			
Standard therapy	18.5	32.8	44.5
EGDT	64.1	11.1	68.4
P value	<0.001	<0.001	<0.001
Any vasopressor (%)†			
Standard therapy	30.3	42.9	51.3
EGDT	27.4	29.1	36.8
P value	0.62	0.03	0.02
Inotropic agent (dobutamine) (%)			
Standard therapy	0.8	8.4	9.2
EGDT	13.7	14.5	15.4
P value	<0.001	0.14	0.15
Mechanical ventilation (%)			
Standard therapy	53.8	16.8	70.6
EGDT	53.0	2.6	55.6
P value	0.90	<0.001	0.02

VARIABLE	STANDARD THERAPY (N=133)	EARLY GOAL-DIRECTED THERAPY (N=130)
	no. (%)	
In-hospital mortality† All patients	59 (46.5)	38 (30.5)

A Randomized Trial of Protocol-Based Care for Early Septic Shock

The ProCESS Investigators*

NEJM march 18, 2014

- 31 SAU aux USA
- Sepsis+ hypotension < 90 mmHg après remplissage ou nécessitant vasopresseurs ou avec lactate > 4 mmol/l
- Inclusion dans les 2 h suivant le début du sepsis

A Randomized Trial of Protocol-Based Care for Early Septic Shock

The ProCESS Investigators*

NEJM march 18, 2014

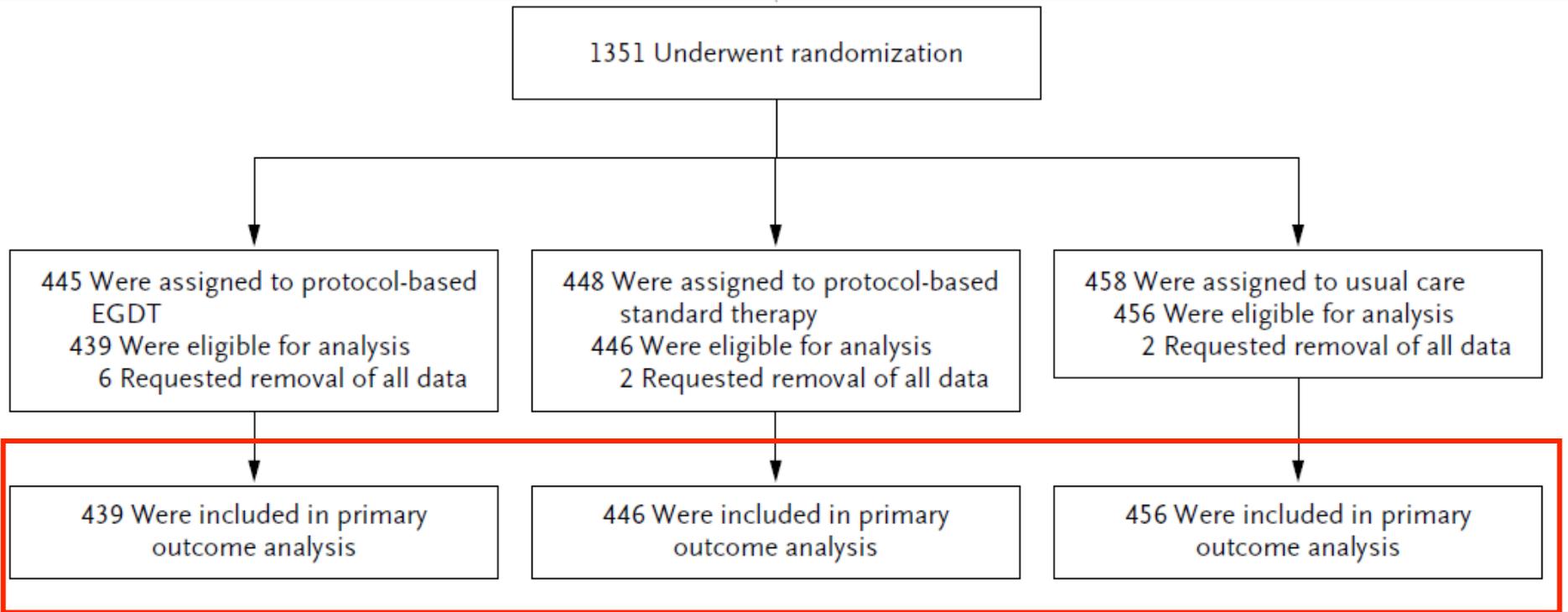


Figure S1. - Protocol for early goal-directed therapy (EGDT)

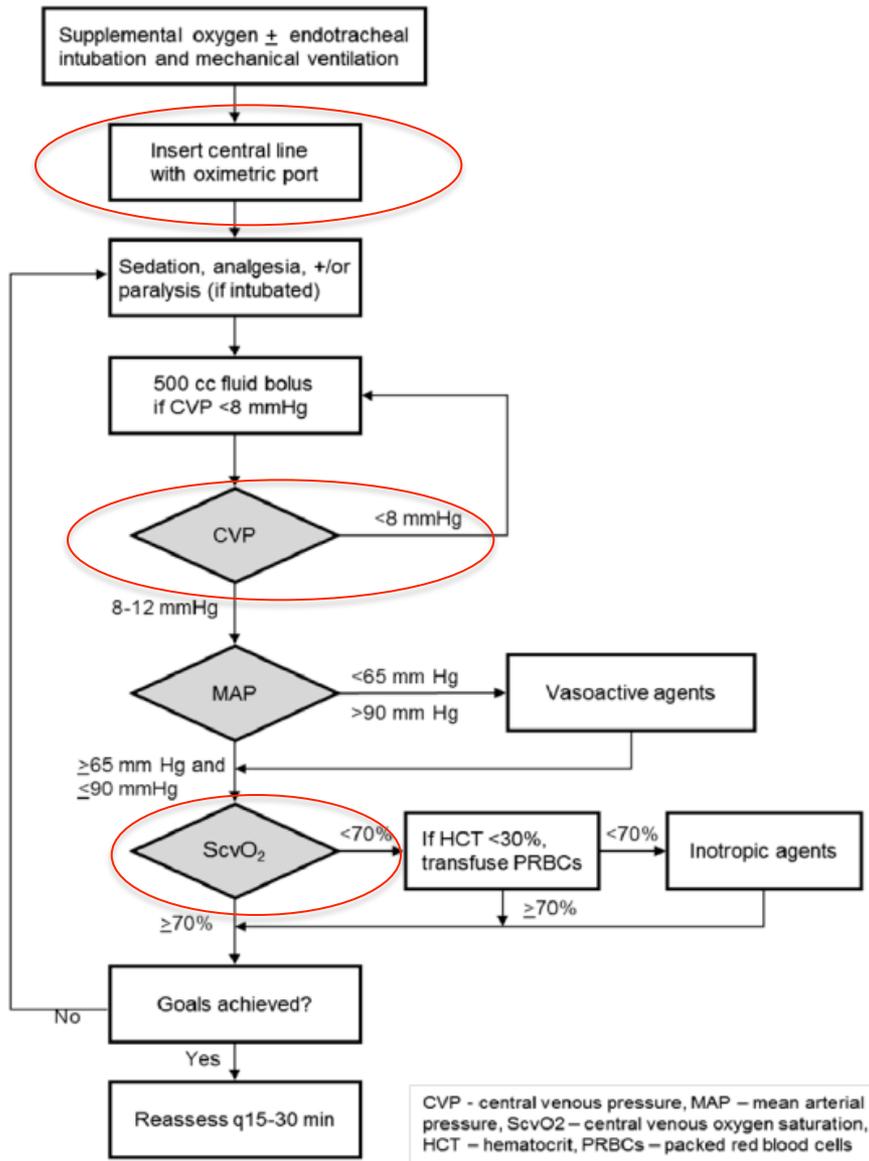


Figure S2. - Protocol for Standard Therapy.

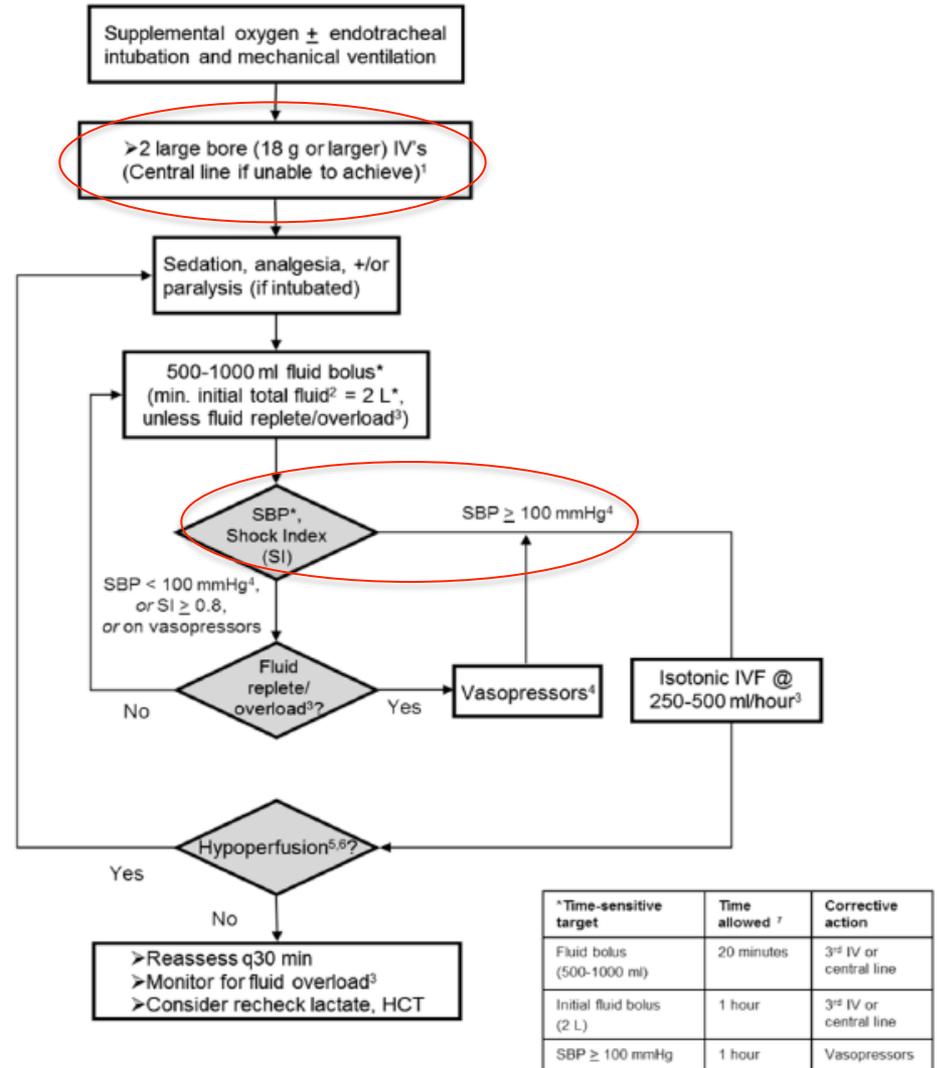
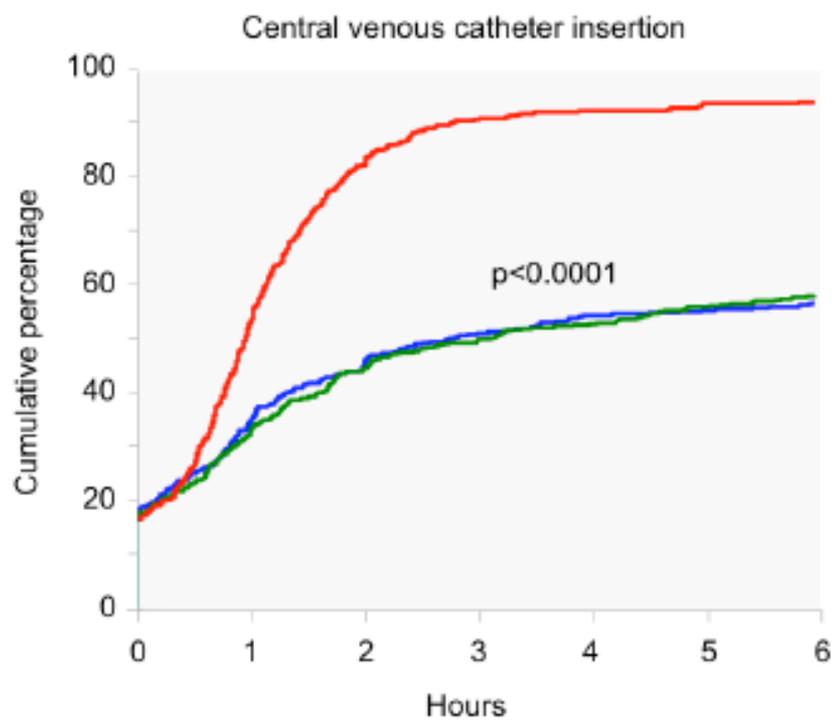
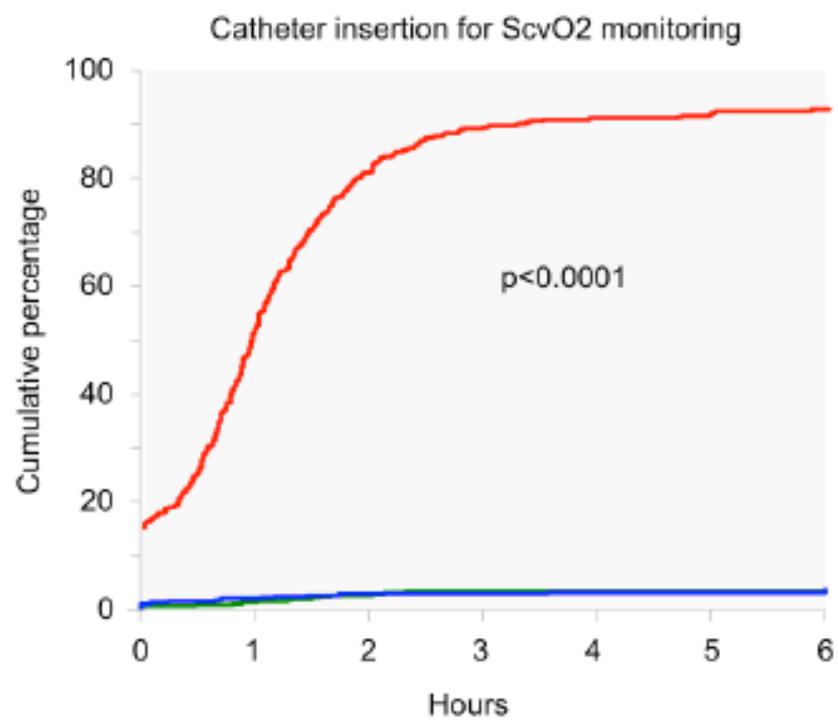


Table 1. Characteristics of the Patients at Baseline.*

Characteristic	Protocol-Based EGDT (N=439)	Protocol-Based Standard Therapy (N=446)	Usual Care (N=456)
Age — yr†	60±16.4	61±16.1	62±16.0
Male sex — no. (%)	232 (52.8)	252 (56.5)	264 (57.9)
Residence before admission — no. (%)‡			
Nursing home	64 (14.6)	72 (16.1)	73 (16.0)
Other	373 (85.0)	373 (83.6)	382 (83.8)
Charlson comorbidity score§	2.6±2.6	2.5±2.6	2.9±2.6
Source of sepsis — no. (%)			
Pneumonia	140 (31.9)	152 (34.1)	151 (33.1)
Urinary tract infection	100 (22.8)	90 (20.2)	94 (20.6)
Intraabdominal infection	69 (15.7)	57 (12.8)	51 (11.2)
Infection of unknown source	57 (13.0)	47 (10.5)	66 (14.5)
Skin or soft-tissue infection	25 (5.7)	33 (7.4)	38 (8.3)
Catheter-related infection	11 (2.5)	16 (3.6)	11 (2.4)
Central nervous system infection	3 (0.7)	3 (0.7)	4 (0.9)
Endocarditis	1 (0.2)	3 (0.7)	3 (0.7)
Other	28 (6.4)	31 (7.0)	26 (5.7)
Determined after review not to have infection	5 (1.1)	14 (3.1)	12 (2.6)
Positive blood culture — no. (%)	139 (31.7)	126 (28.3)	131 (28.7)
APACHE II score¶	20.8±8.1	20.6±7.4	20.7±7.5
Entry criterion — no. (%)			
Refractory hypotension	244 (55.6)	240 (53.8)	243 (53.3)
Hyperlactatemia	259 (59.0)	264 (59.2)	277 (60.7)
Physiological variables			
Systolic blood pressure — mm Hg	100.2±28.1	102.1±28.7	99.9±29.5
Serum lactate — mmol/liter**	4.8±3.1	5±3.6	4.9±3.1
Time to randomization — min			
From arrival in the emergency department††	197±116	185±112	181±97
From meeting entry criteria	72±77	66±38	69±45

Remplissage 2 I
avant
randomisation

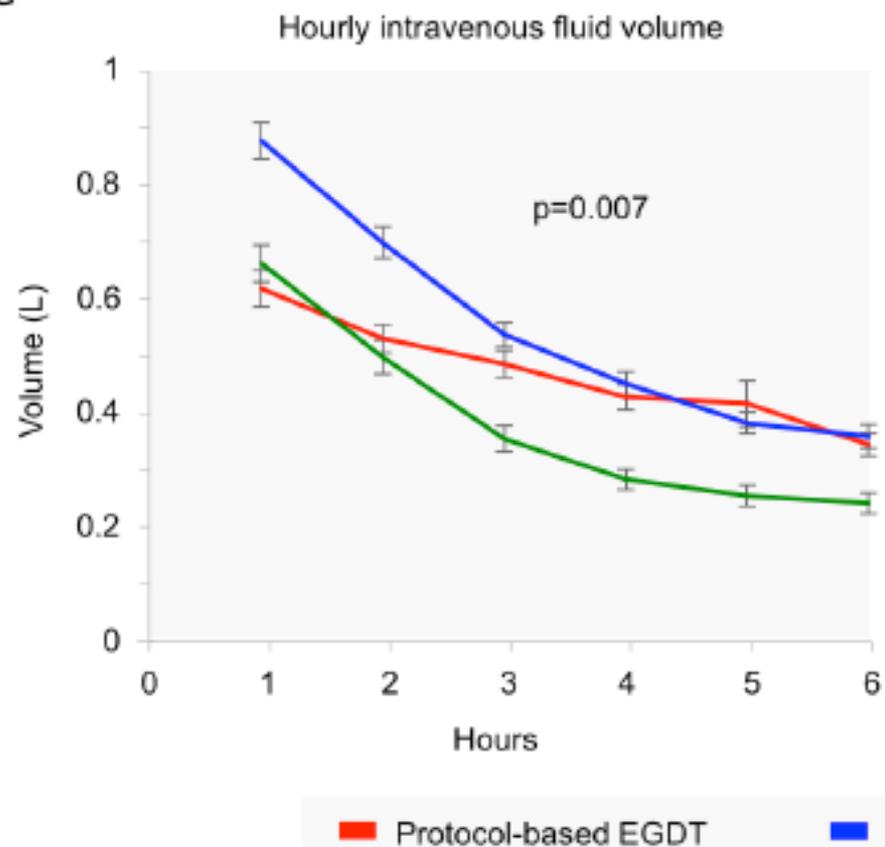
A**B**

Protocol-based EGDT

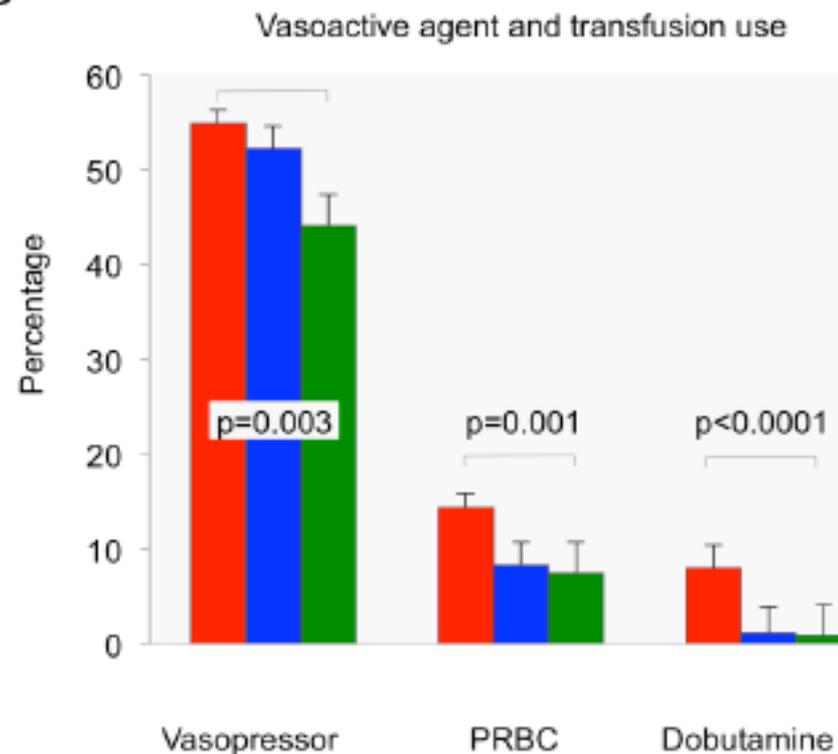
Protocol-based Standard Therapy

Usual care

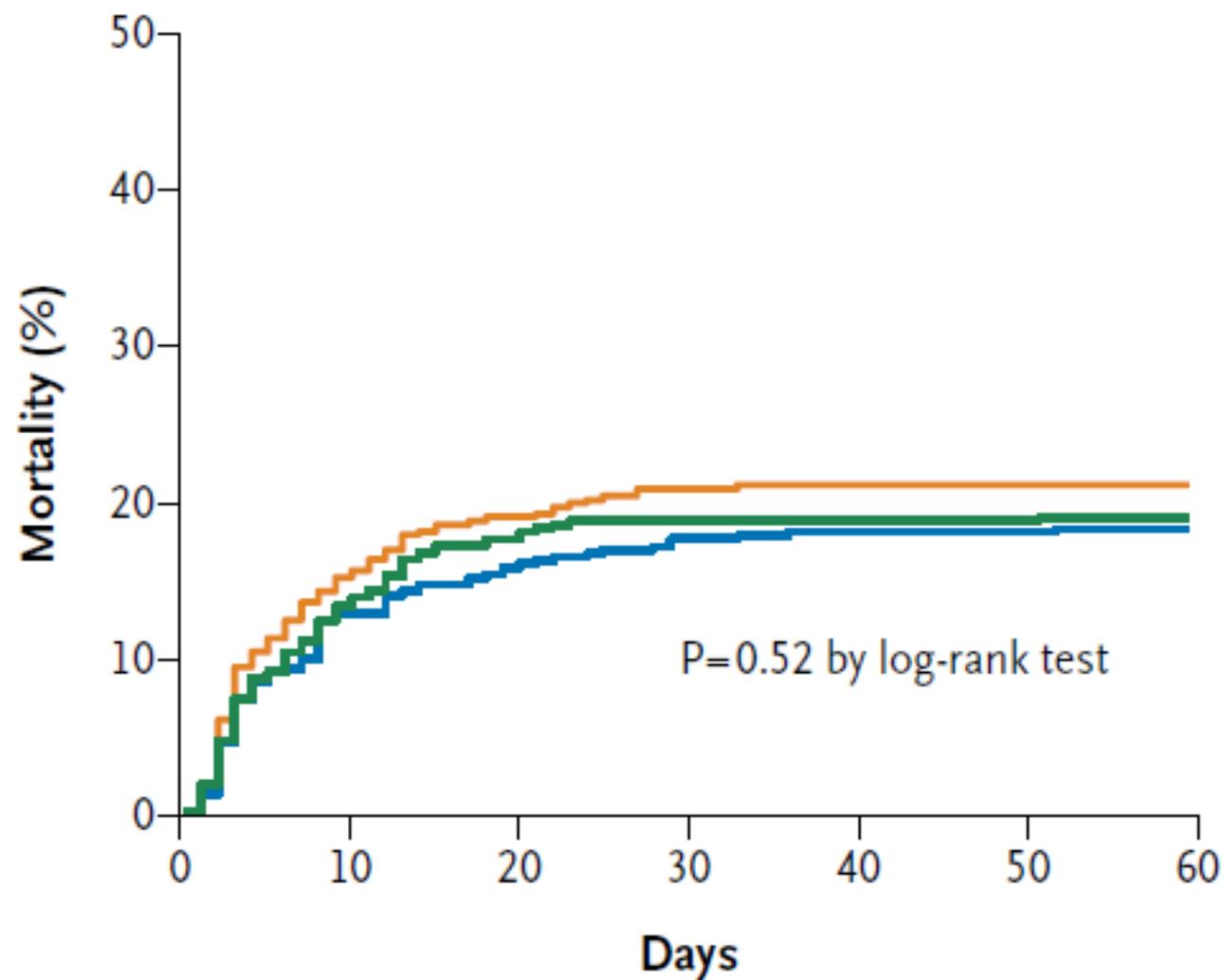
C



D



A Cumulative In-Hospital Mortality to 60 Days

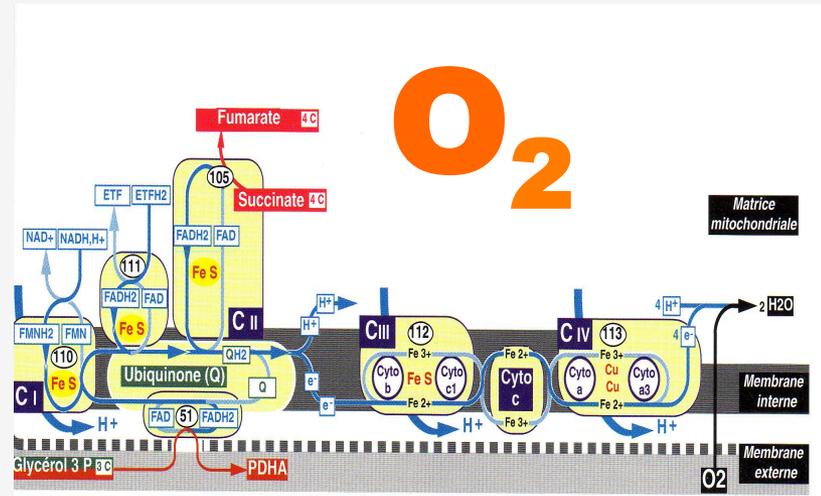


Inotropisme

$S_{cv}O_2 < 70\%$

**$S_{a}O_2 > 95\%$
 $Hb > 9\text{ g/dL}$
 VO_2 (ramsay 3)**

DC inadapté

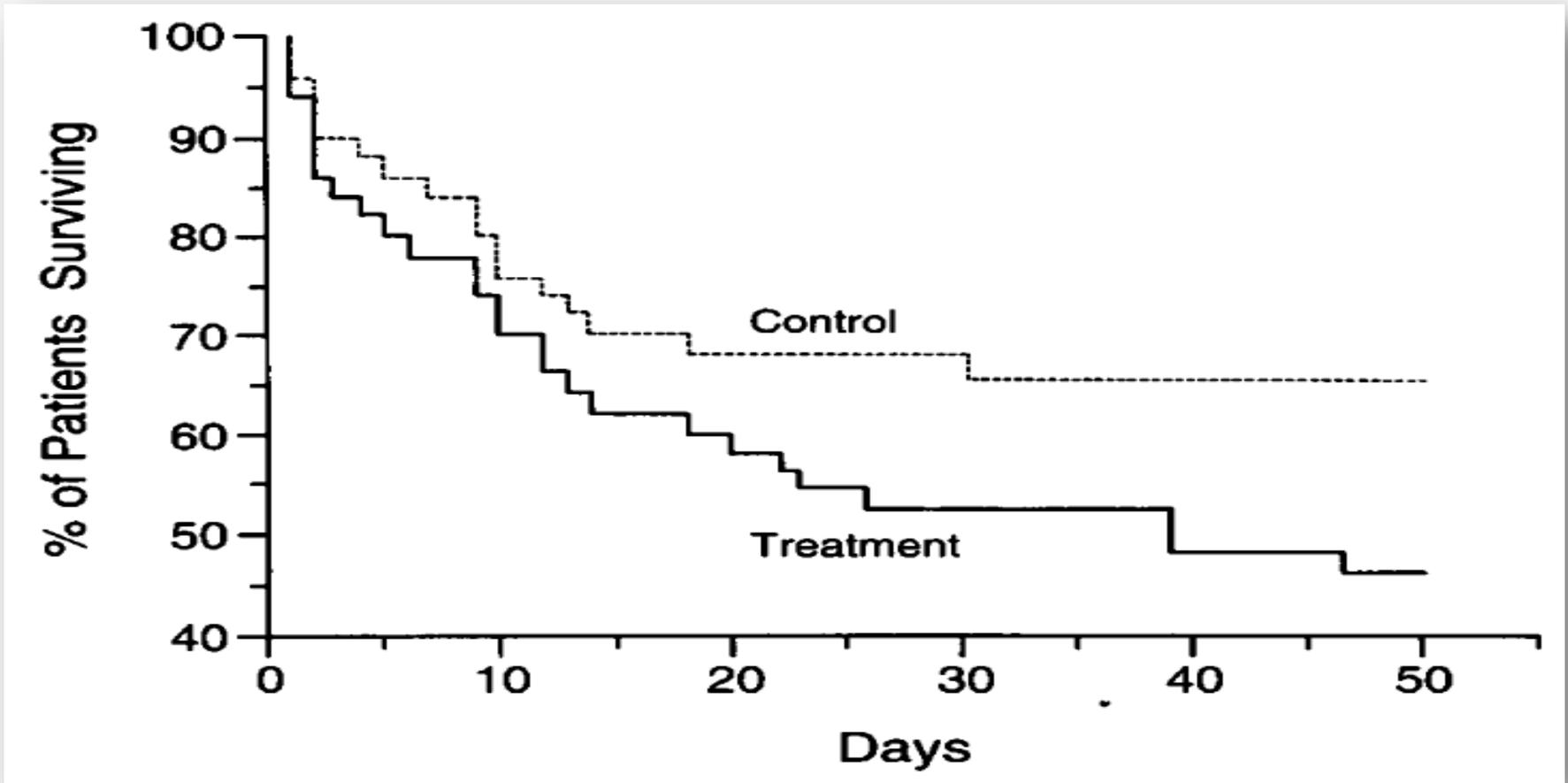


Inotrope positif

Dellinger *et al.*, Intensive Care Med 2013 ; Rivers *et al.*, N Engl J Med 2001

Inotropisme

■ Hyperdébit = Surmortalité



Surmortalité ?



Comment gérer l'hyperdébit ?



↓ **Demande énergétique**

Hypothermie

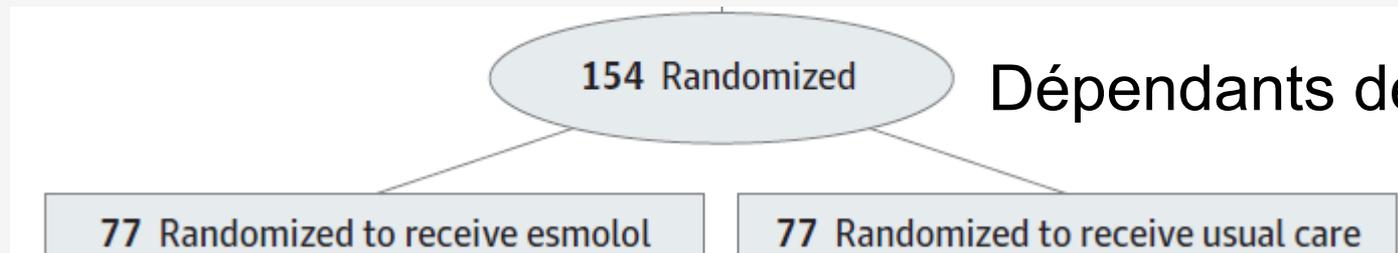
B-bloquants

Beta-bloquants

■ Contrôle FC

Preliminary Communication | CARING FOR THE CRITICALLY ILL PATIENT

Effect of Heart Rate Control With Esmolol on Hemodynamic and Clinical Outcomes in Patients With Septic Shock
A Randomized Clinical Trial



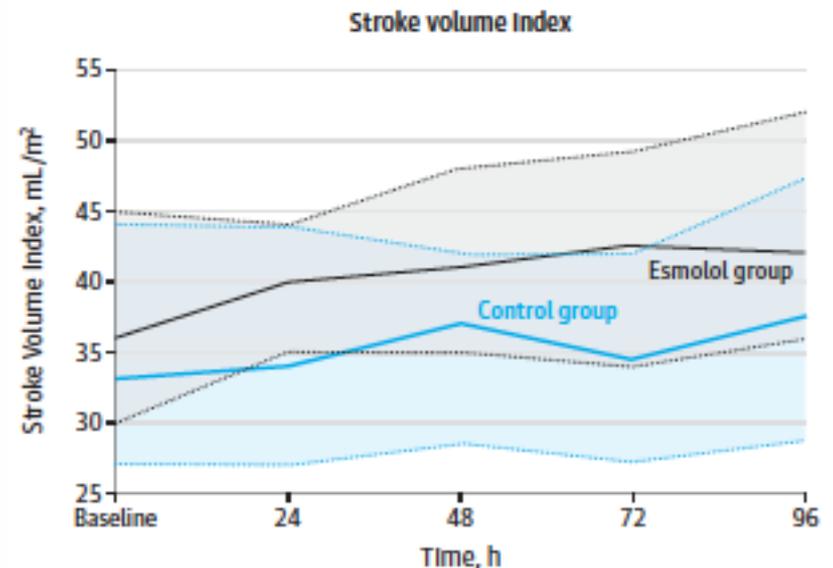
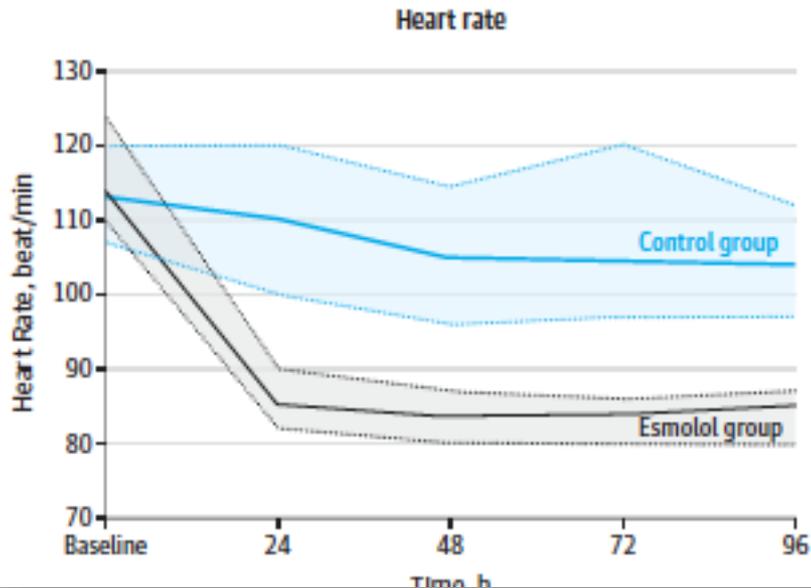
IVSE pour FC < 95 /min

Beta-bloquants

■ Contrôle FC

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Effect of Heart Rate Control With Esmolol on Hemodynamic and Clinical Outcomes in Patients With Septic Shock A Randomized Clinical Trial

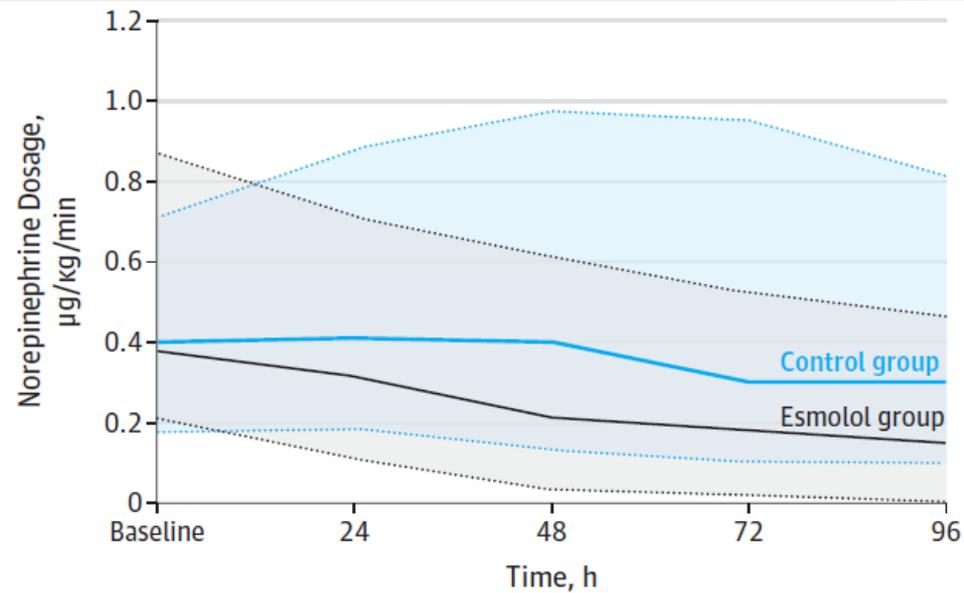
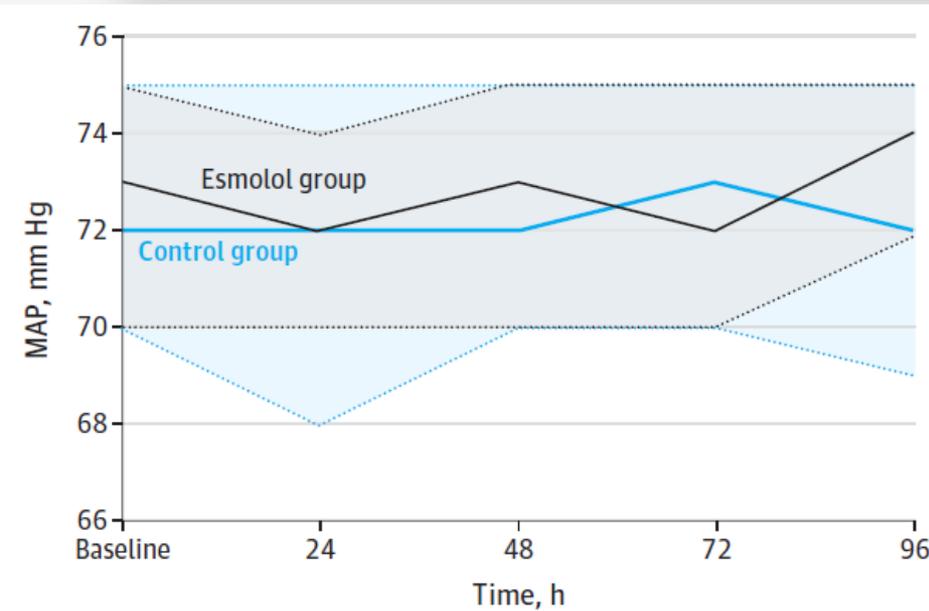


Beta-bloquants

■ Contrôle FC

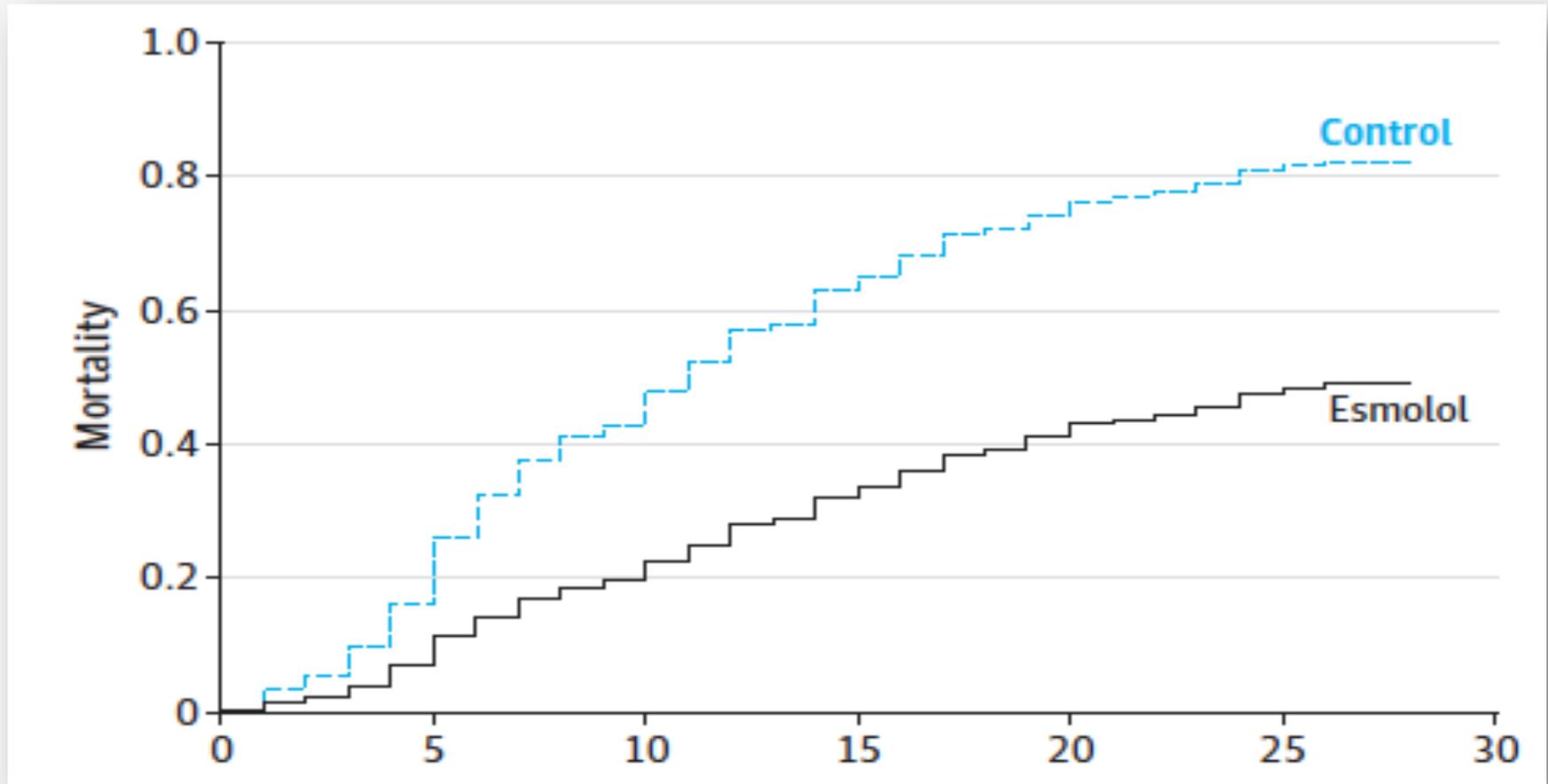
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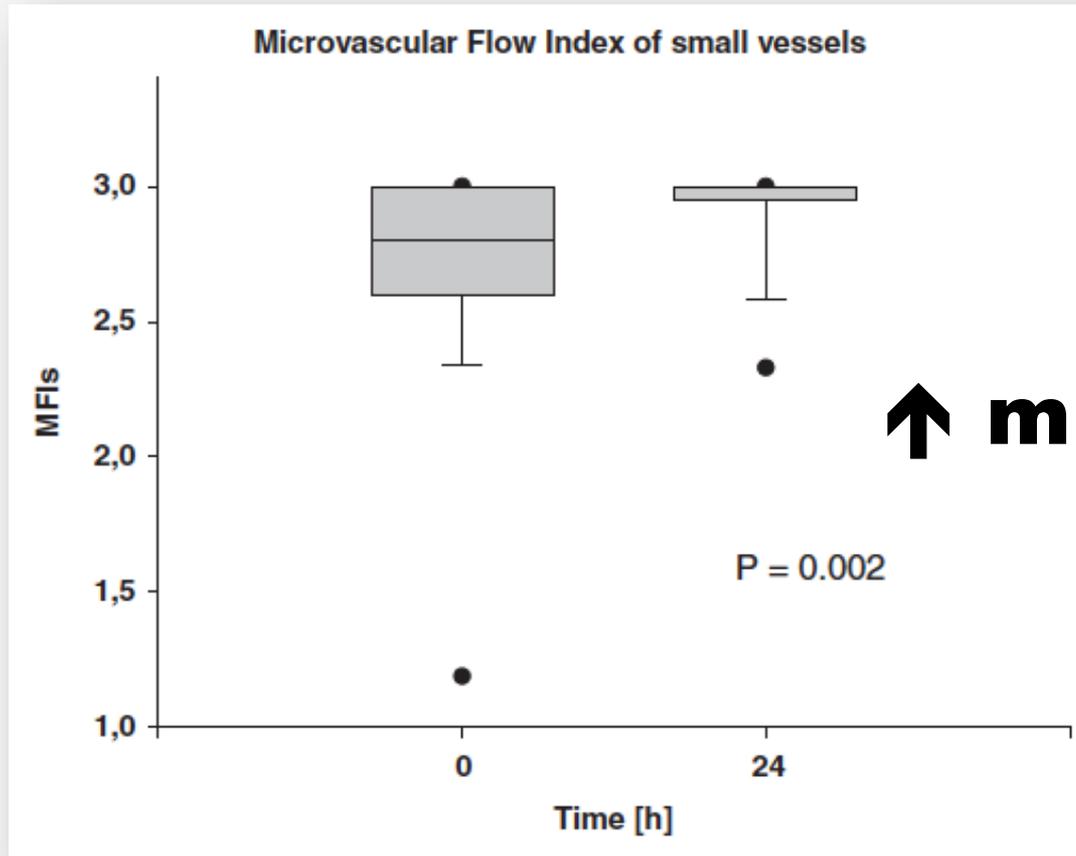
Beta-bloquants

■ Contrôle FC



Beta-bloquants

■ Contrôle FC



↑ microcirculation

Surmortalité ?



Comment gérer l'hyperdébit ?



↓ **Demande énergétique**

Hypothermie

B-bloquants

Contrôle température

Fever Control Using External Cooling in Septic Shock: a Randomized Controlled Trial

- Body temperature : 36.8 ± 0.7 vs. $38.4 \pm 1.1^{\circ}\text{C}$
- ↓ 50% vasopressor dose : 54% vs. 20%
- Shock reversal : 86% vs. 73%
- Day-14 mortality : 19% vs. 34% (similar at ICU discharge)
- Attention, surmortalité si sepsis + antipyrétique

Hydrocortisone ?

■ Hydrocortisone ? Immunomodulation

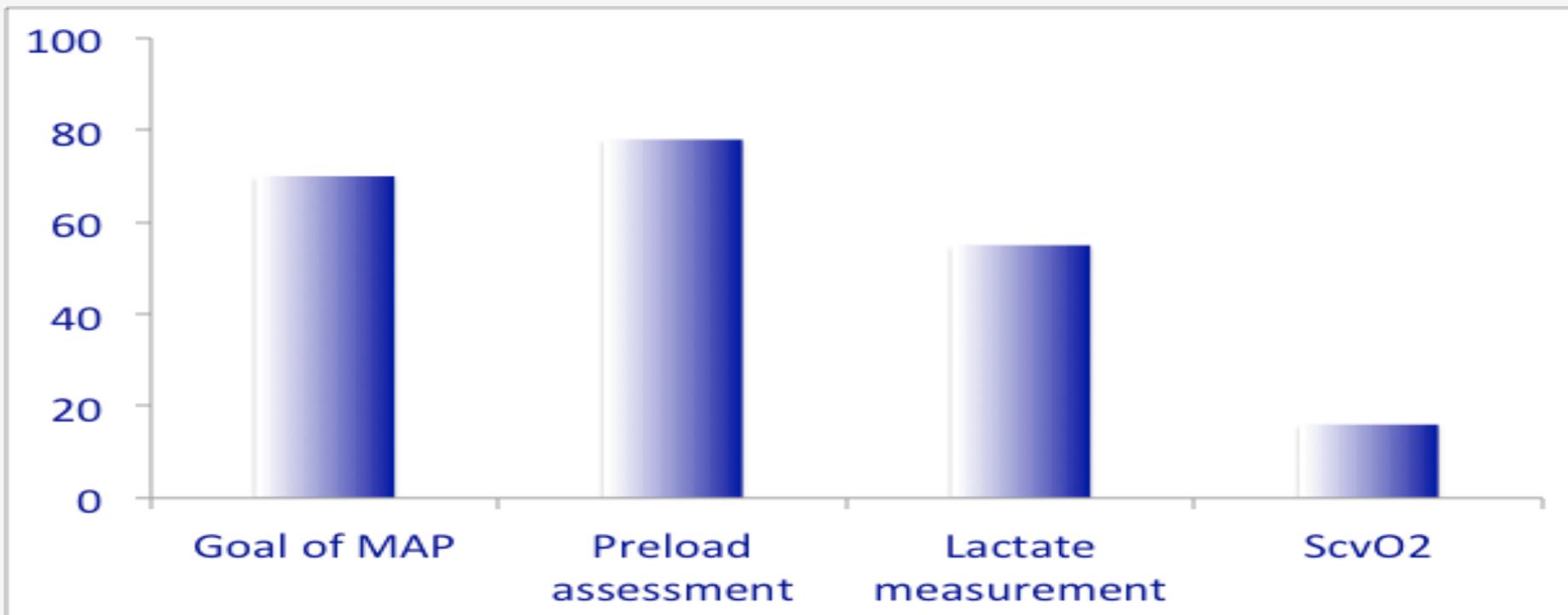
1. Ne pas utiliser si stabilisation
2. Si absence de stabilisation, 200 mg/j
3. Pas de test de stimulation
4. Décroissance progressive

2 études contradictoires : résultats APPROCHS ?

Améliorer nos pratiques

- 9. Glucose control: **45%**
- 10. **Ventilator setting: 37%**
- 11. **Sepsis bundle: 34%**
- 12. ET Tube cuff pressure: **26%**
- 13. **Analgesia monitoring: 24%**

Variable compliance with clinical practice guidelines identified in a 1-day audit at 66 French adult intensive care units



Conclusion



■ Basiques

- ▶ Cristalloïdes
- ▶ Antibiotiques
- ▶ Noradrénaline
- ▶ Eviction du foyer

Monitoring

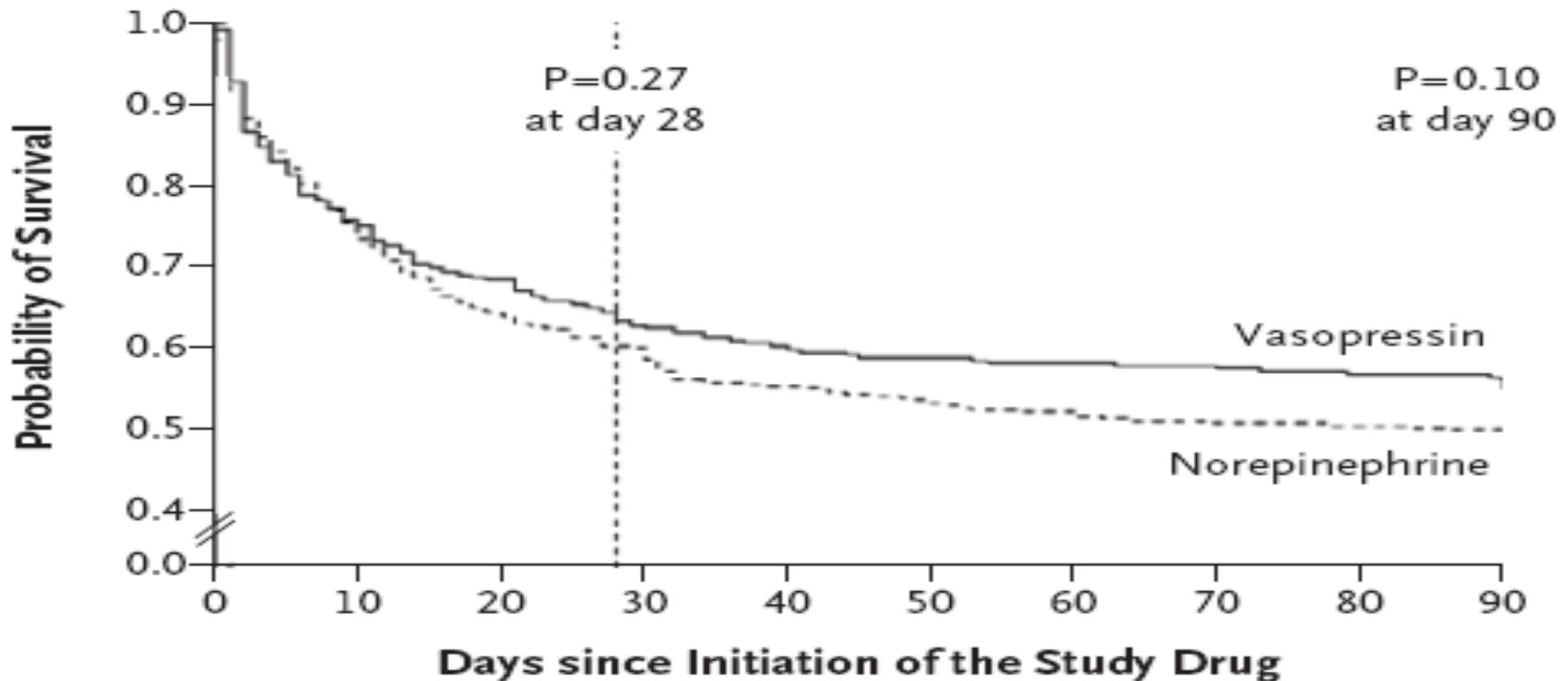
■ Vers le futur

- ▶ Immunostimulation
- ▶ Hibernation
- ▶ Microcirculation

Vasopresseurs

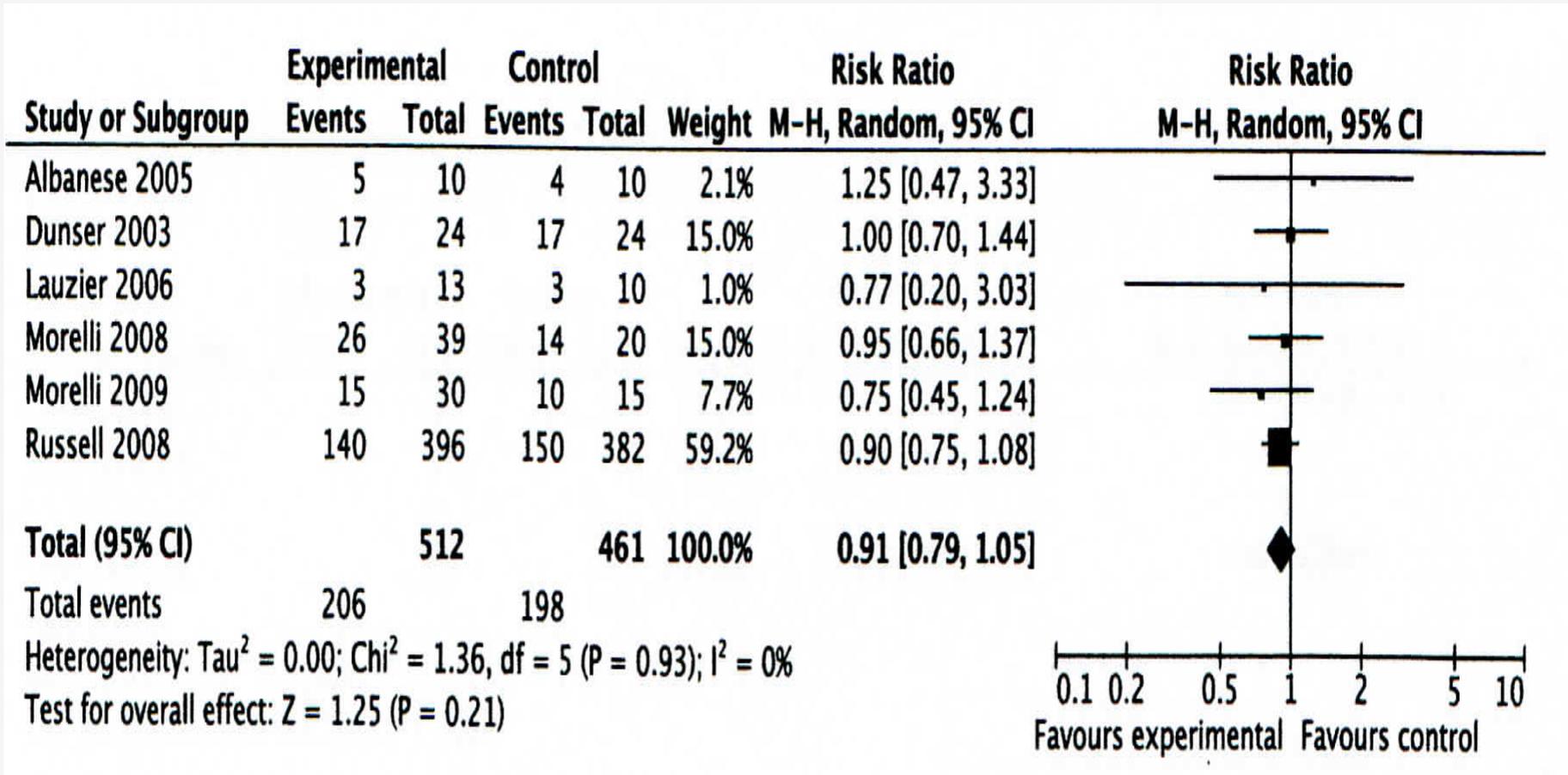
■ Vasopressine : traitement de recours

▶ VASST: AVP + NE = NE Dellinger *et al.*, Intensive Care Med 2013



Vasopresseurs

■ Vasopressine : traitement de recours



Vasopresseurs

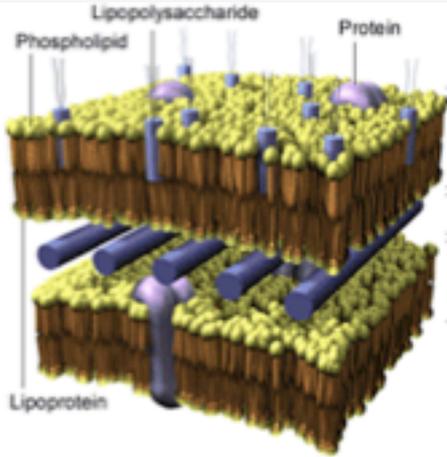
- **Vasopressine** : traitement de recours ?

Vasopressin Compared with Norepinephrine Augments the Decline of Plasma Cytokine Levels in Septic Shock

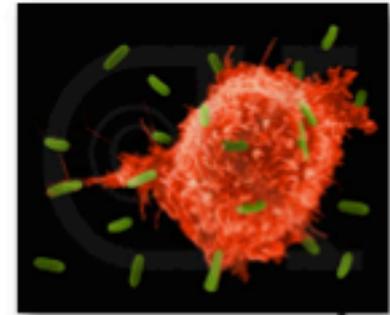
1. **Survivants** : ↑ clairance des cytokines
2. **AVP** : ↑ clairance / NE

Immunomodulation et AVP

Physiopathologie



Endotoxine (LPS)



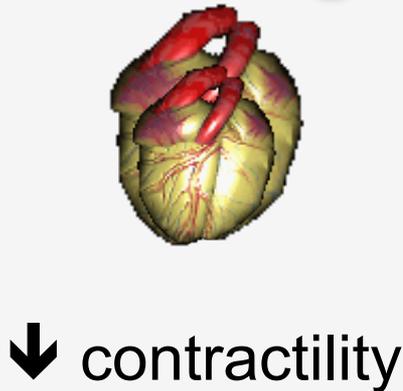
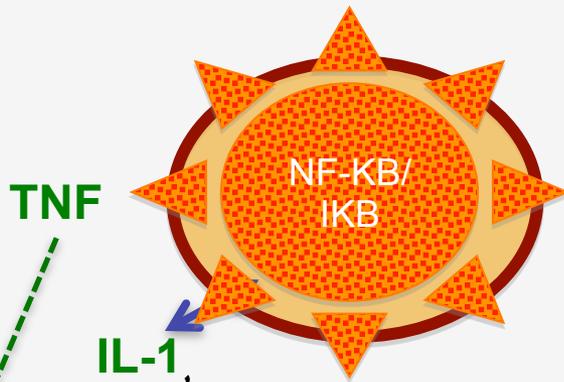
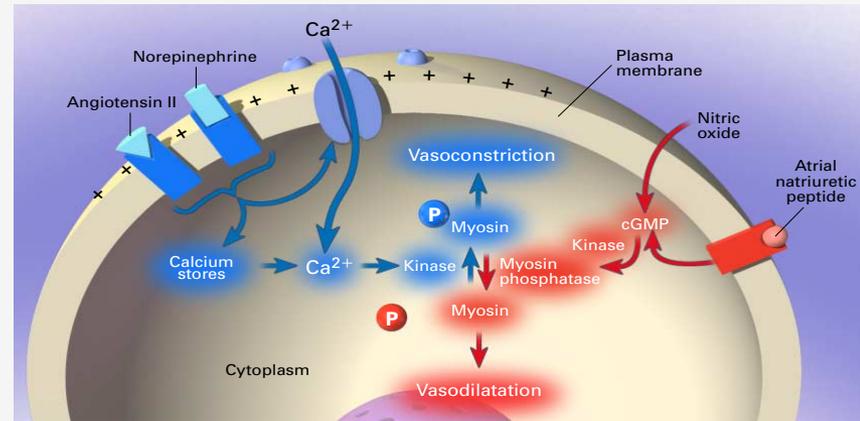
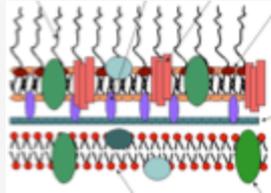
Médiateurs inflammatoires

Inflammation locale

décompartment

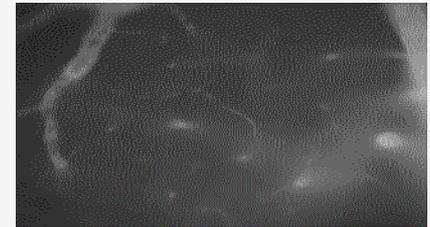
Choc septique

Physiopathologie



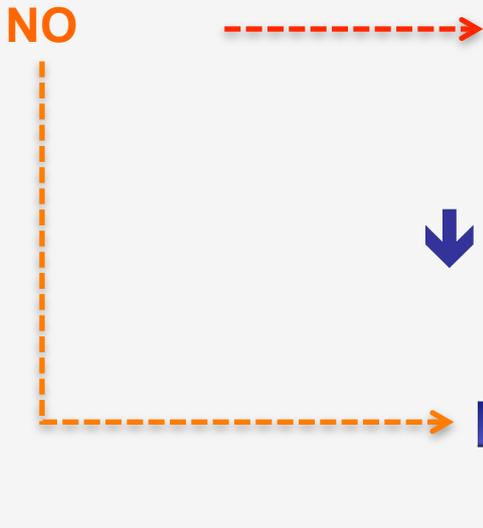
Vasodilation

↓ Perfusion pressure



DIC

Tissue hypoxia

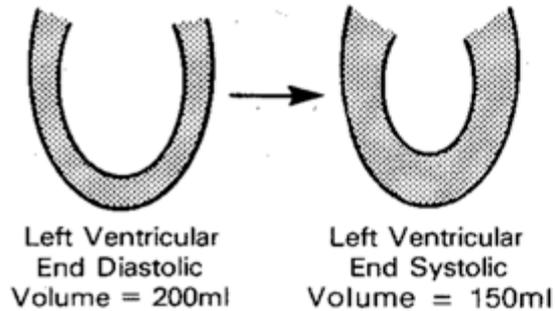


Physiopathologie

Défaillance cardiaque

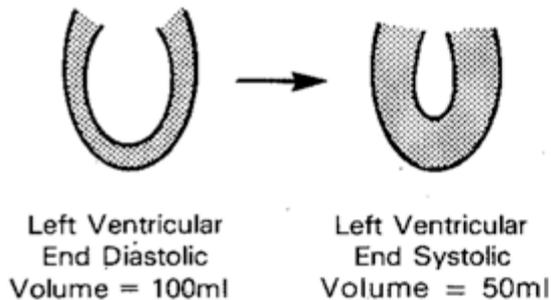
Profound but Reversible Myocardial Depression in Patients with Septic Shock

ACUTE PHASE OF SEPTIC SHOCK

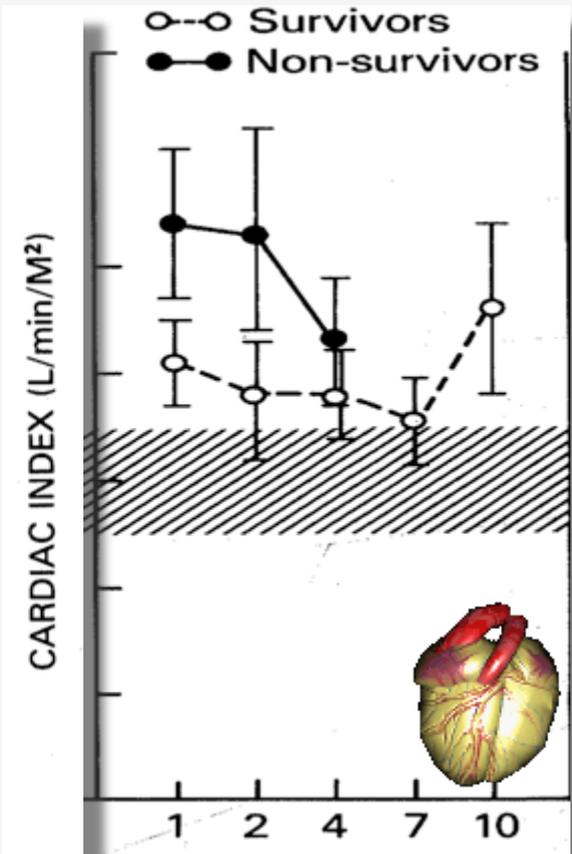


Stroke Volume = 50ml
Ejection Fraction = $\frac{200\text{ml} - 150\text{ml}}{200\text{ml}} = 25\%$

RECOVERY PHASE OF SEPTIC SHOCK



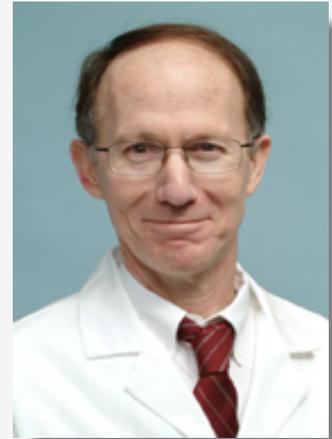
Stroke Volume = 50ml
Ejection Fraction = $\frac{100\text{ml} - 50\text{ml}}{50\text{ml}} = 50\%$



Physiopathologie

The sepsis seesaw

The immune response goes haywire during sepsis, a deadly condition triggered by infection. Richard S. Hotchkiss and his colleagues take the focus off of the prevailing view that the key aspect of this response is an exuberant inflammatory reaction. They assess recent human studies bolstering the notion that immunosuppression is also a major contributor to the disease. Many people with sepsis succumb to cardiac dysfunction, a process examined by Peter Ward. He showcases the factors that cause cardiomyocyte contractility to wane during the disease.



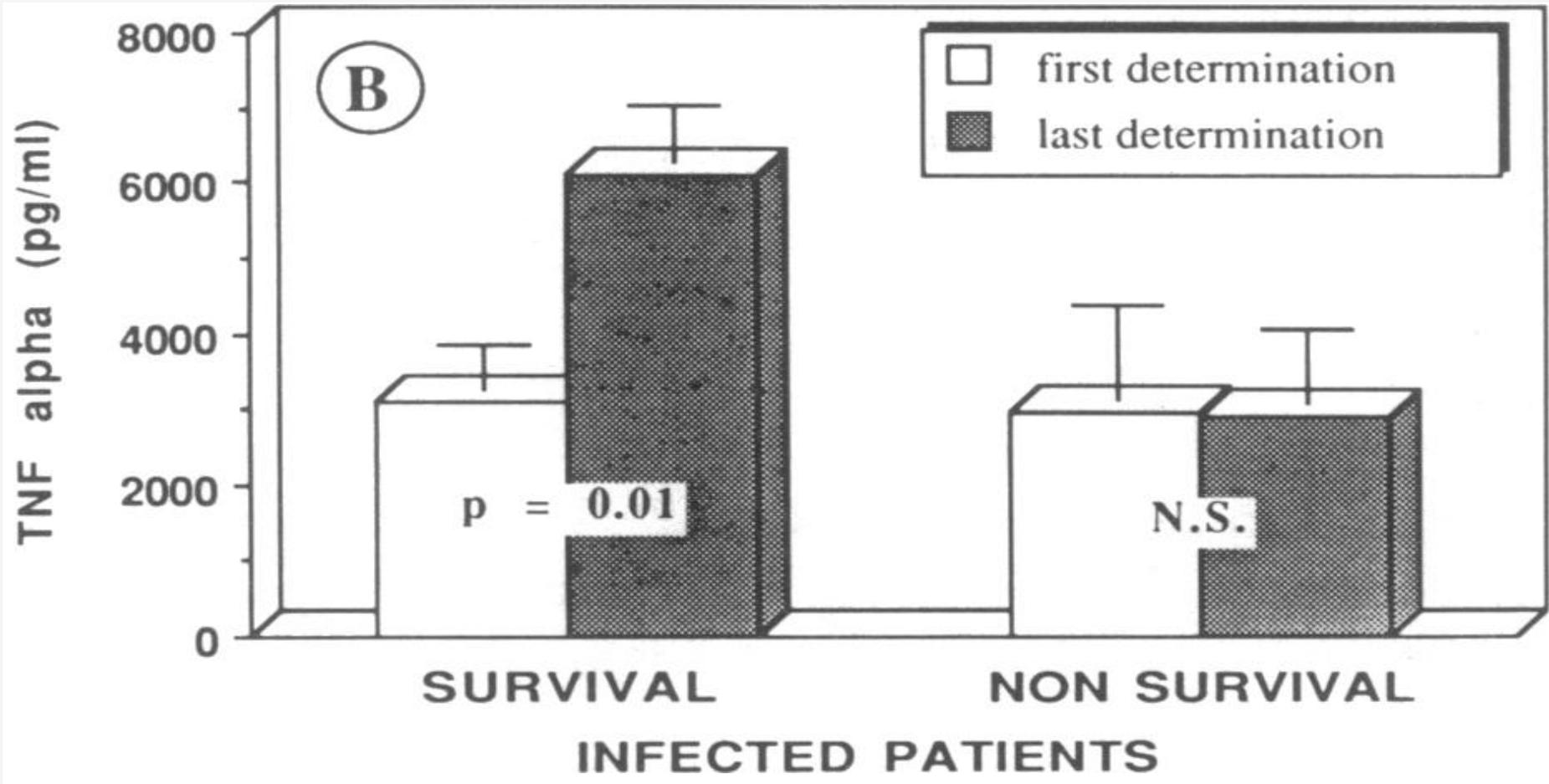
■ BEDSIDE TO BENCH

Tilting toward immunosuppression

Richard S Hotchkiss, Craig M Coopersmith, Jonathan E McDunn & Thomas A Ferguson

1. Anergie
2. Immunodépression
3. Temps

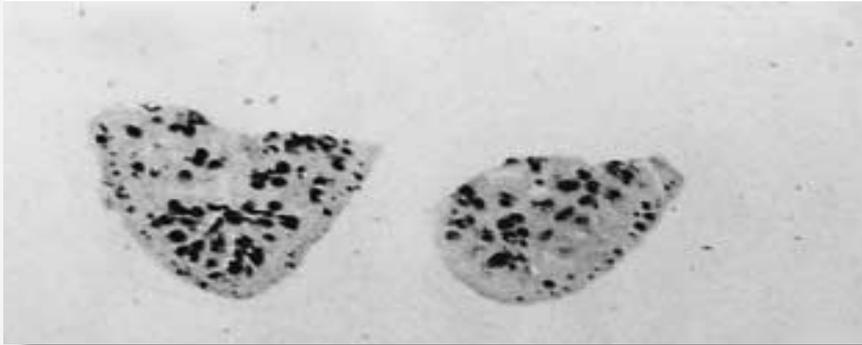
Anergie



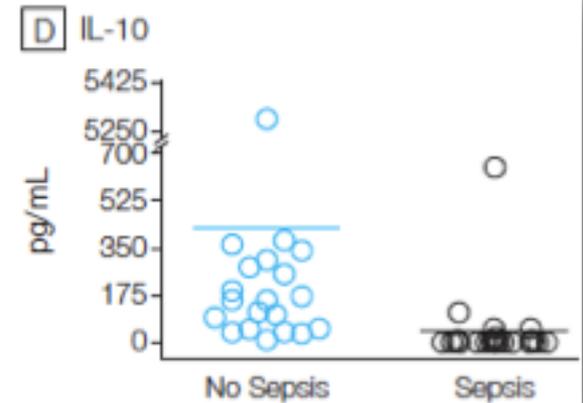
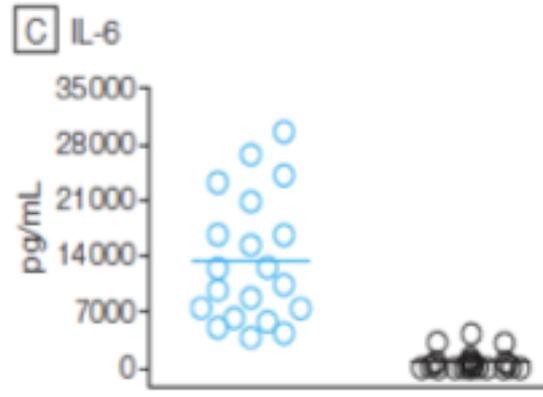
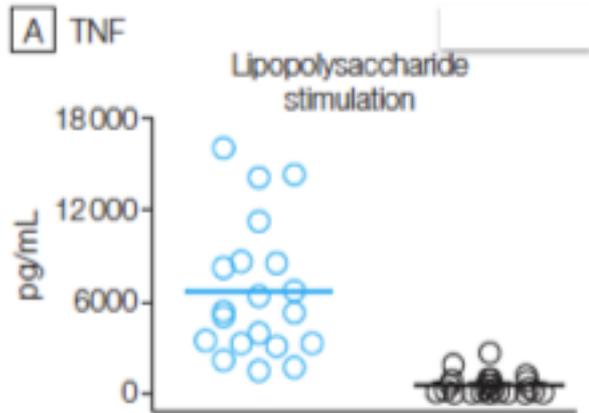
Seconde stimulation : anergie

Immunodépression

B Cells (CD20), Trauma



B Cells (CD20), Sepsis

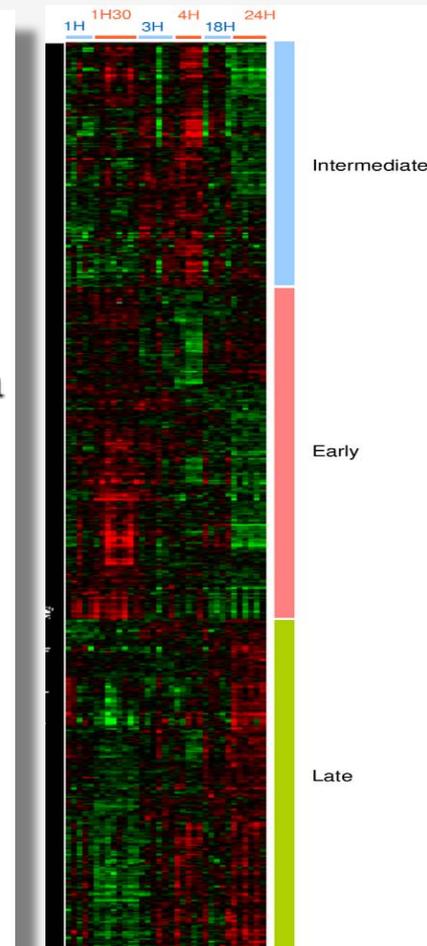
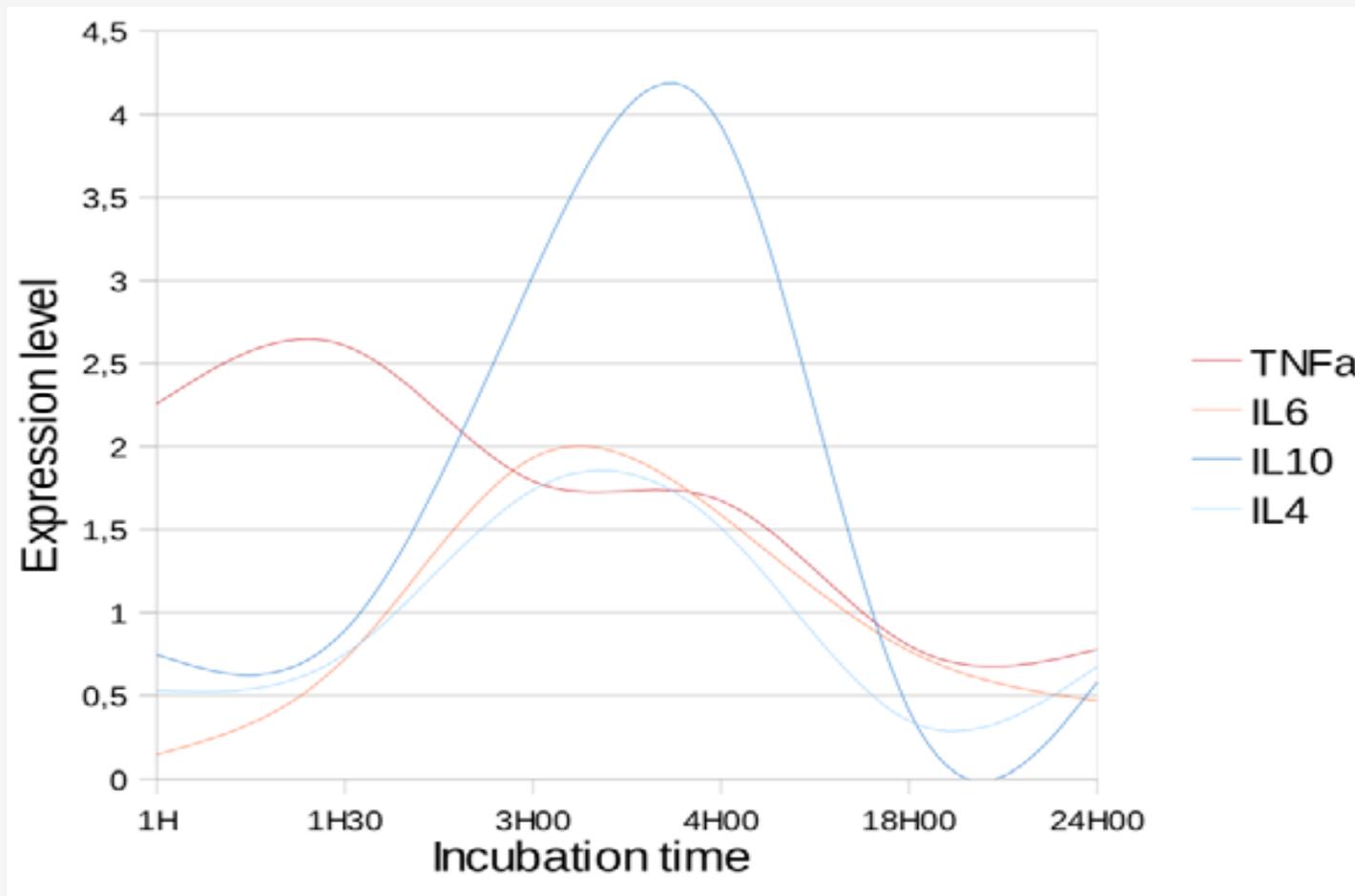


No Sepsis Sepsis

No Sepsis Sepsis

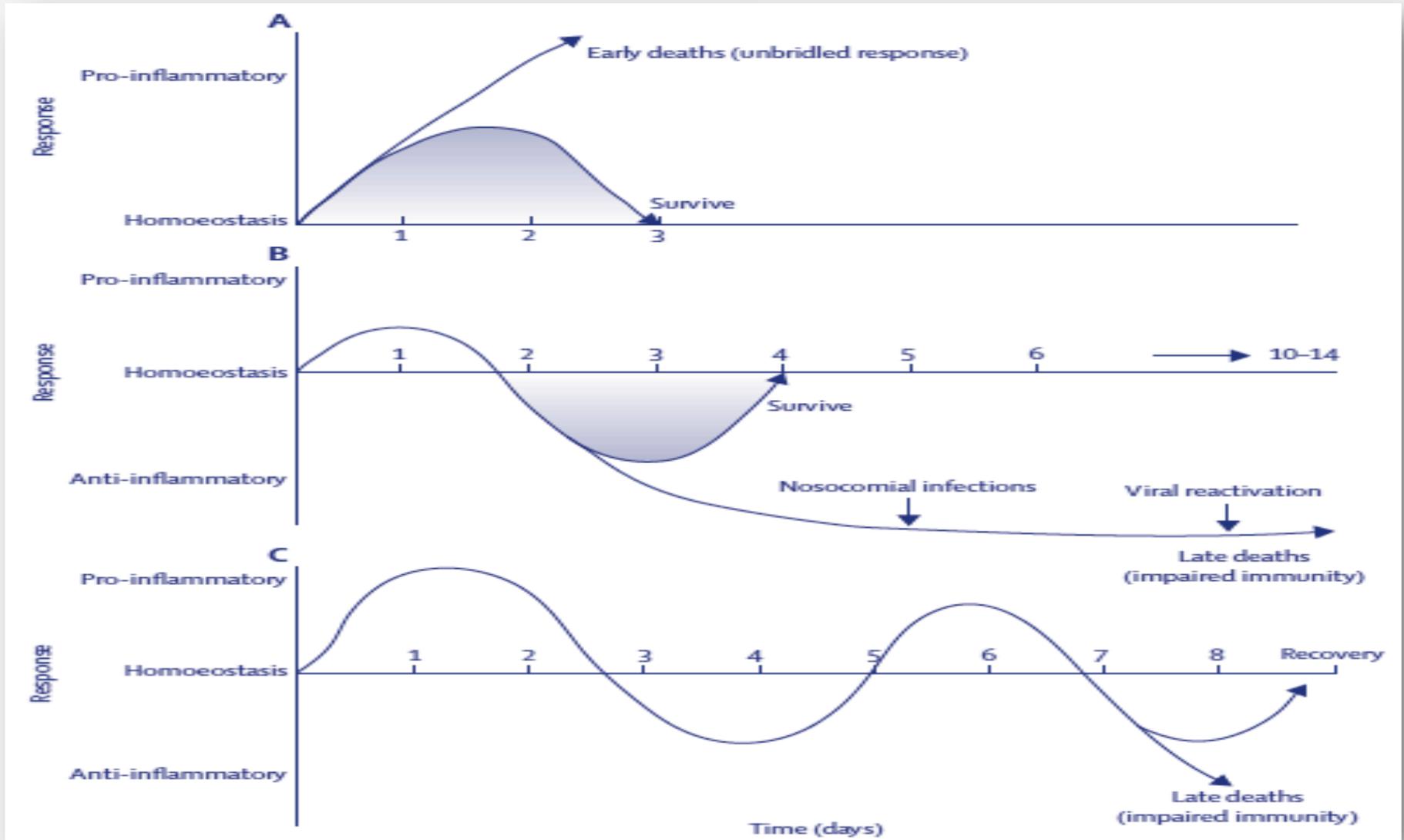
No Sepsis Sepsis

Temps



Temps : précoce vs. tardif

Temps

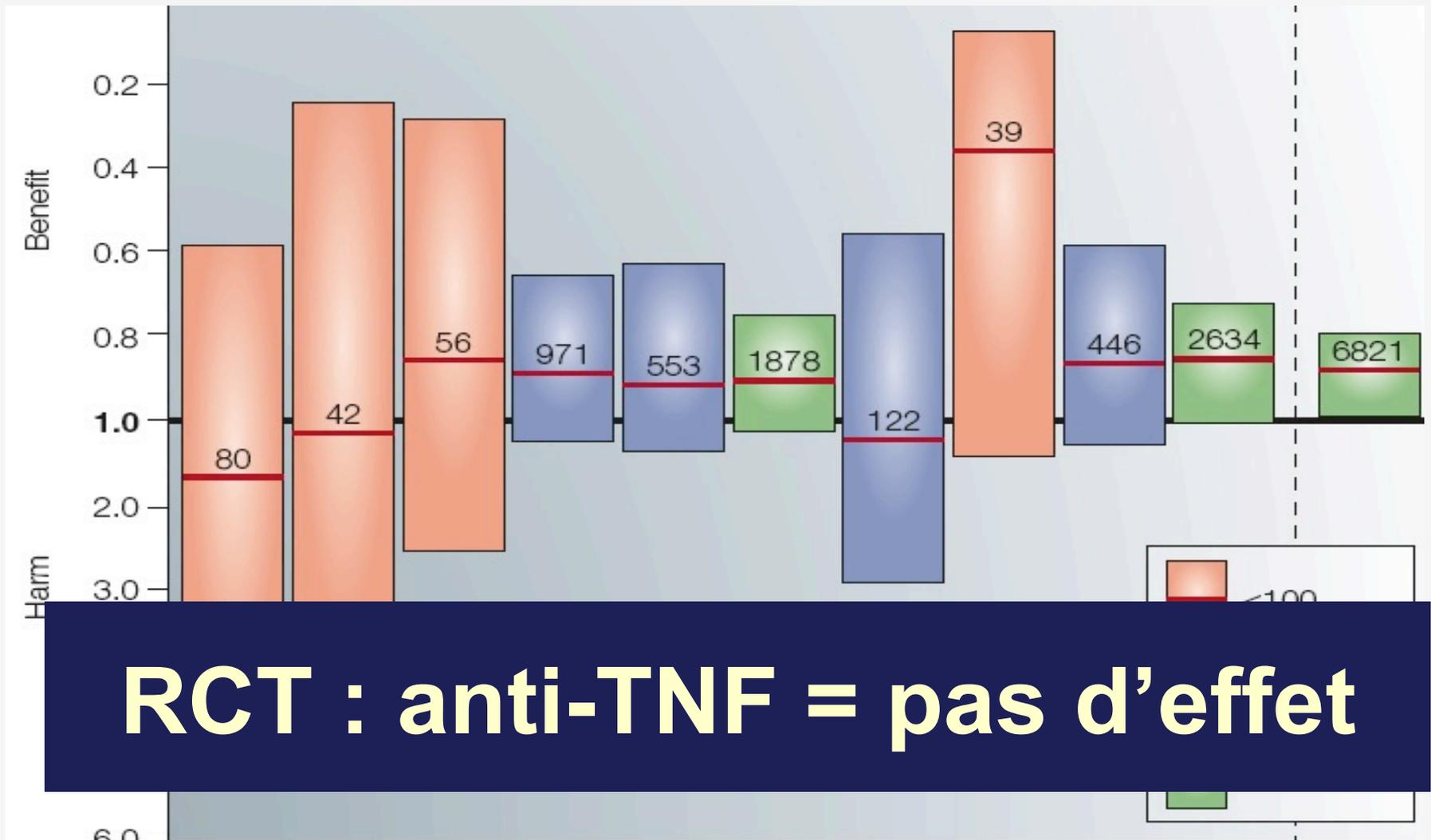


Physiopathologie

Réponse immune : dépression

Futur : stimuler l'immunité

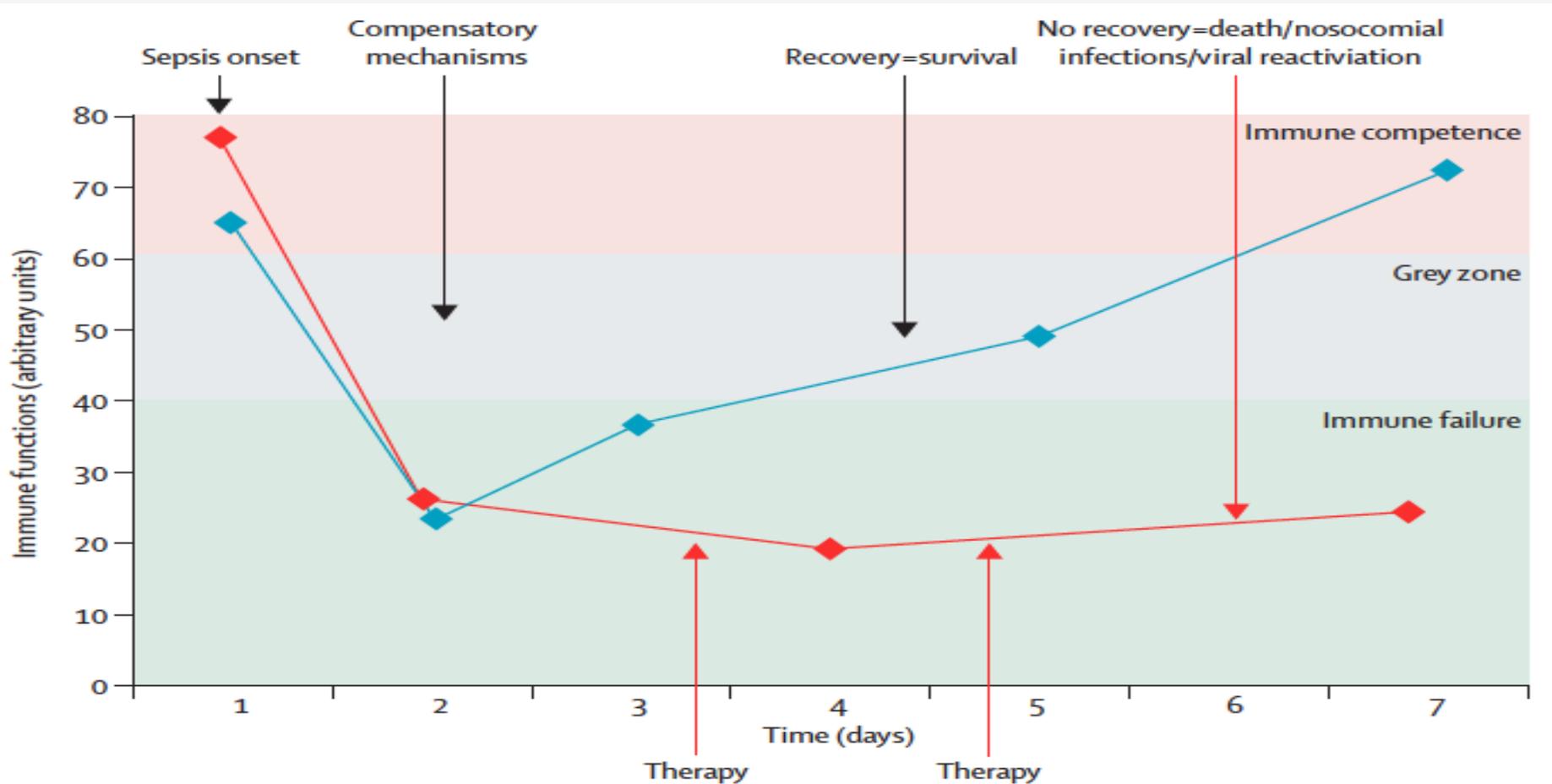
Preuve du concept



RCT : anti-TNF = pas d'effet

Biomarqueur : GPS

HLA-DR



GM-CSF : préliminaire

Granulocyte–Macrophage Colony-stimulating Factor to Reverse Sepsis-associated Immunosuppression

A Double-Blind, Randomized, Placebo-controlled Multicenter Trial

monocytic HLA-DR
required: < 8,000 mAB/ cell

dose escalation if
monocytic HLA-DR
<15,000 mAB/ cell



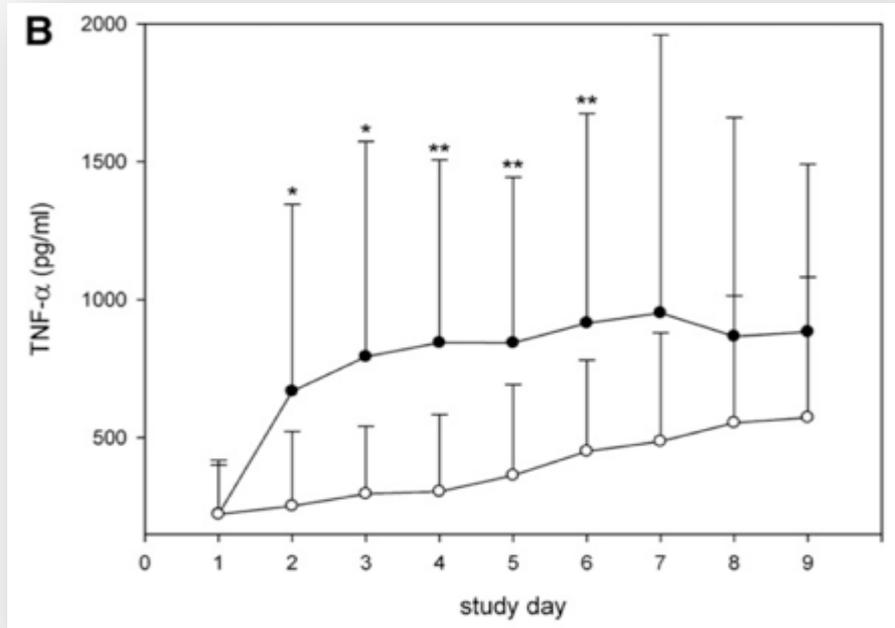
day -1 0 1 2 3 4 5 6 7 8 9 28

GMCSF-group: 4 µg/kg/day

4 or 8 µg/kg/day

Placebo-group: 0.9% NaCl

GM-CSF : préliminaire



Ventilation mécanique

GM-CSF: 148 ± 103 h

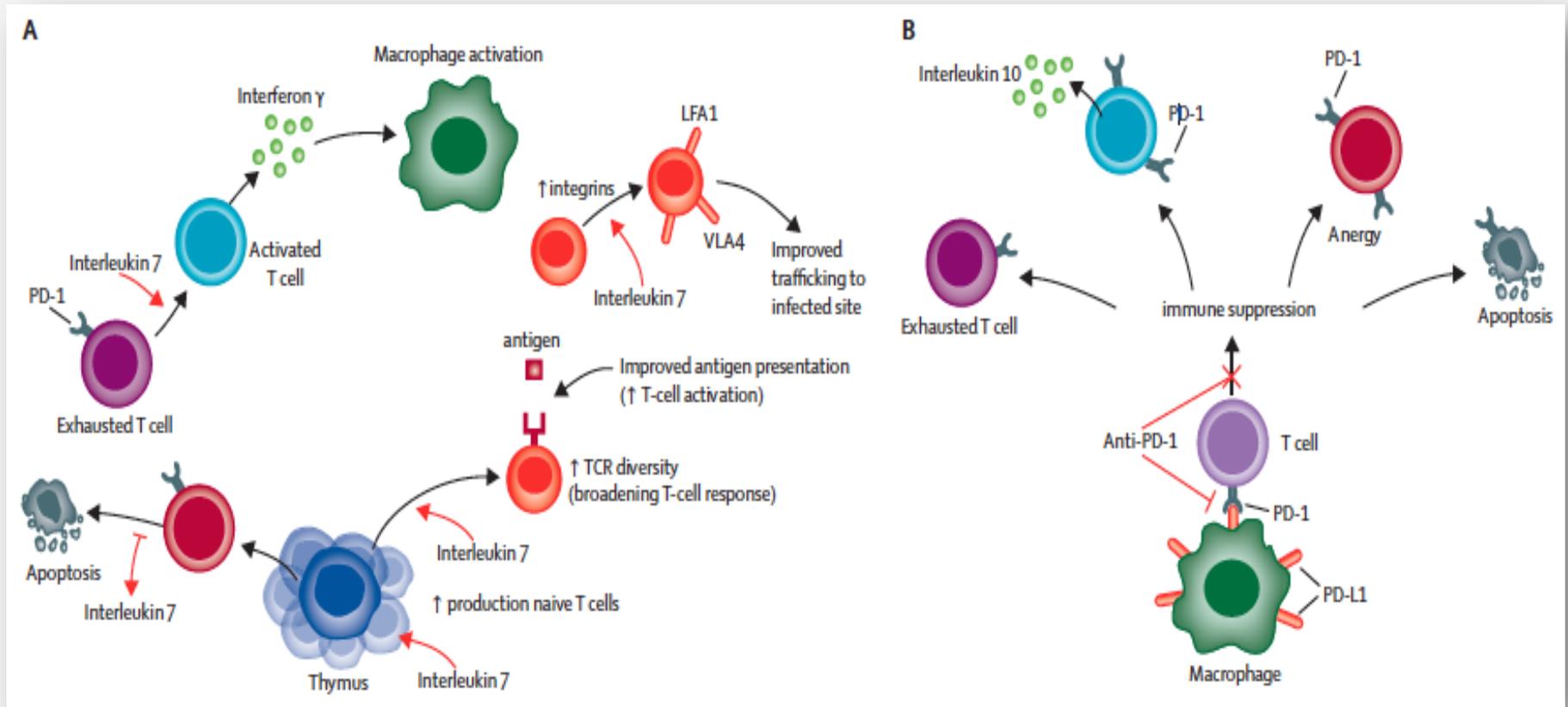
vs.

Placebo: 207 ± 57 h,

$p = 0,04$

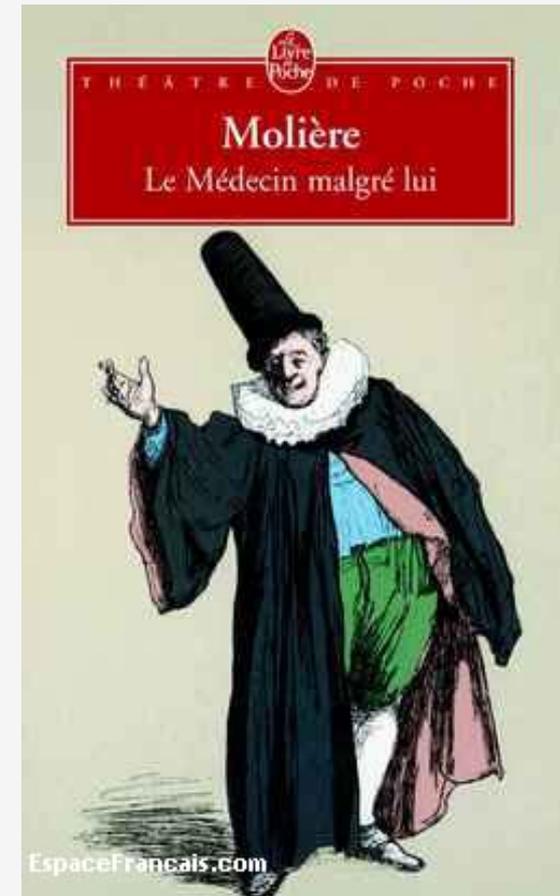
Etudes multicentriques en cours

Interleukine 7 ?



IL-7 Promotes T Cell Viability, Trafficking, and Functionality and Improves Survival in Sepsis

Croyance et business



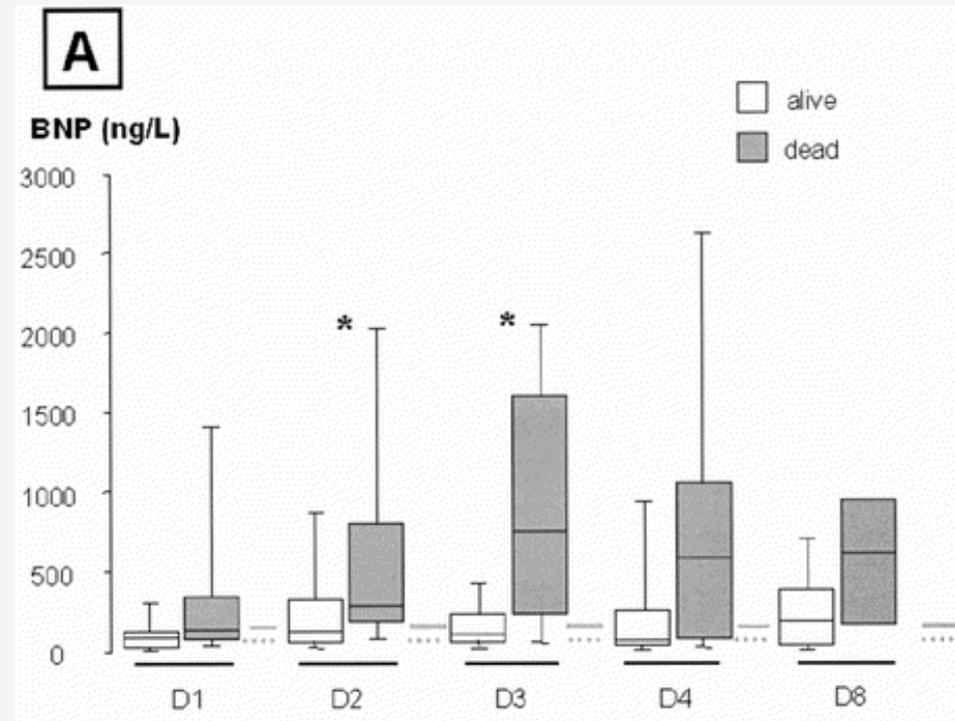
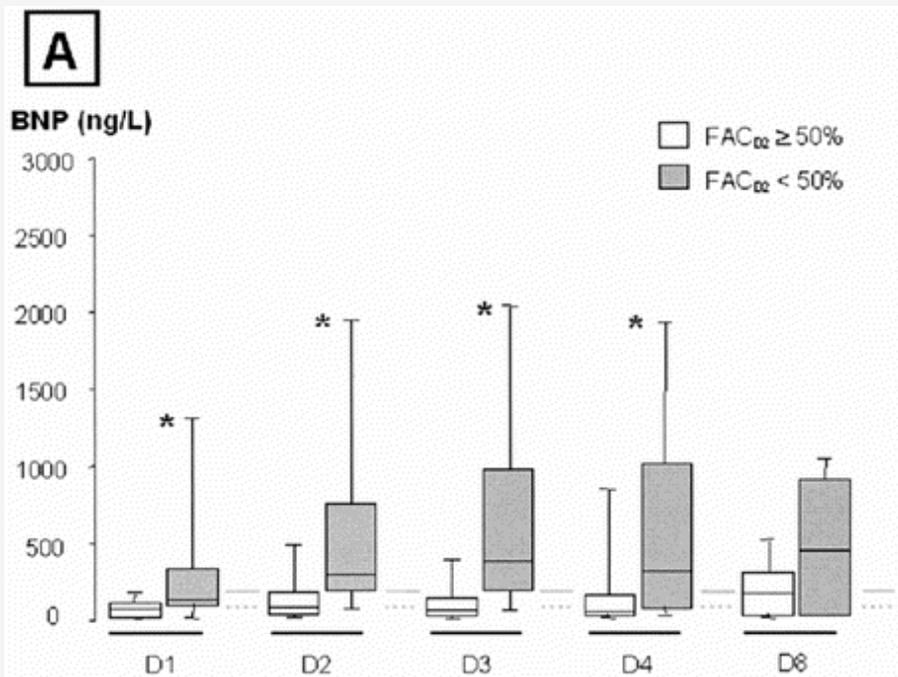
Sepsis

- Dépression myocardique

Ann Intern Med 1984; 100:483–490

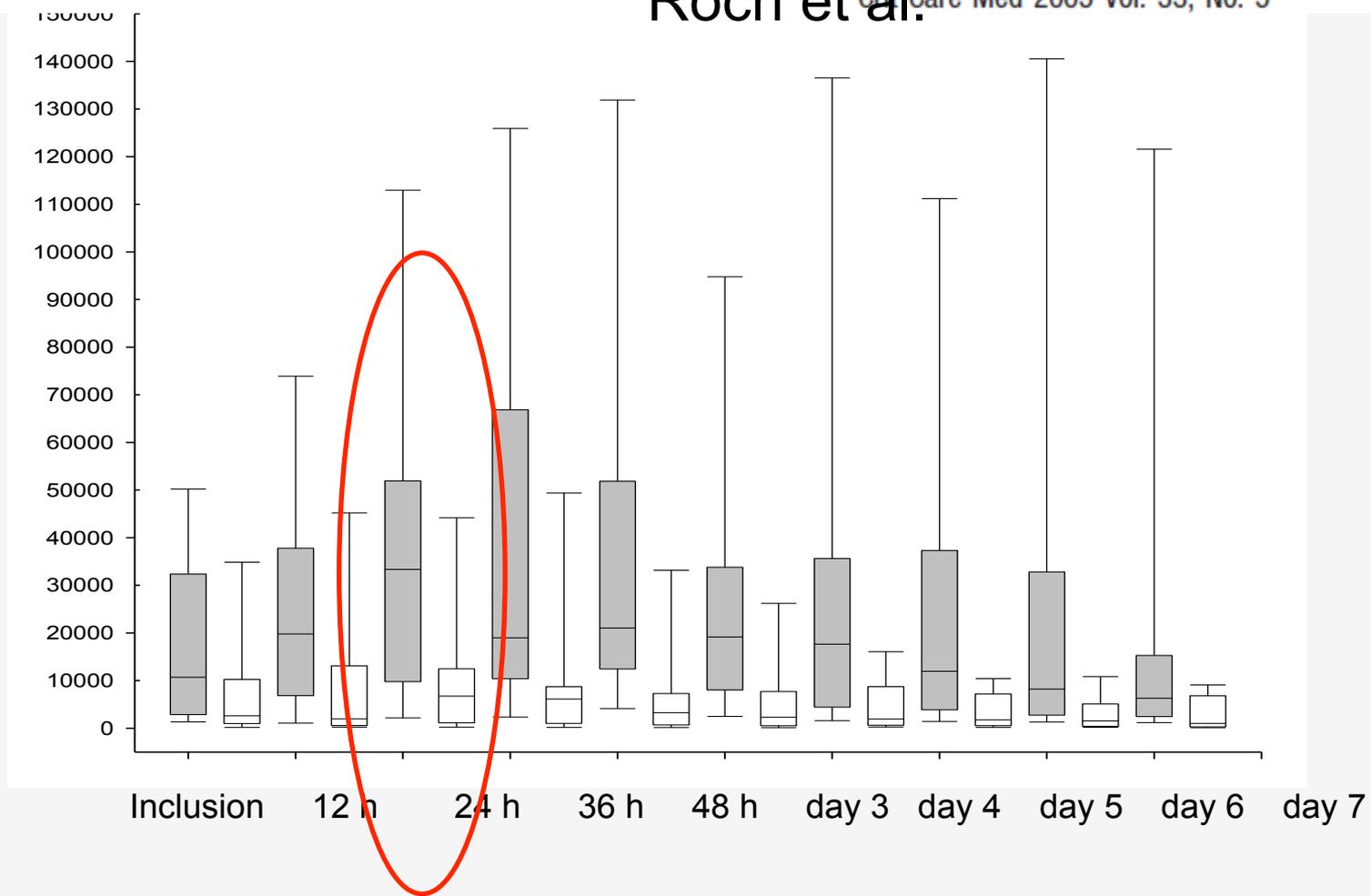
- Baisse contractilité VG
- Mais baisse de postcharge
- Donc le plus souvent pas de congestion

BNP- 34 patients en choc septique



NH₂ terminal pro-brain natriuretic peptide plasma level as an early marker of prognosis and cardiac dysfunction in septic shock patients*

Roch et al. Crit Care Med 2005 Vol. 33, No. 5



p < 0.05 entre non-survivants (n = 22) et survivants (n=17) à chaque temps

NH₂ terminal pro-brain natriuretic peptide plasma level as an early marker of prognosis and cardiac dysfunction in septic shock patients*

Roch et al. Crit Care Med 2005 Vol. 33, No. 5

Table 3. Factors that potentially influenced intensive care unit mortality by univariate analysis^a

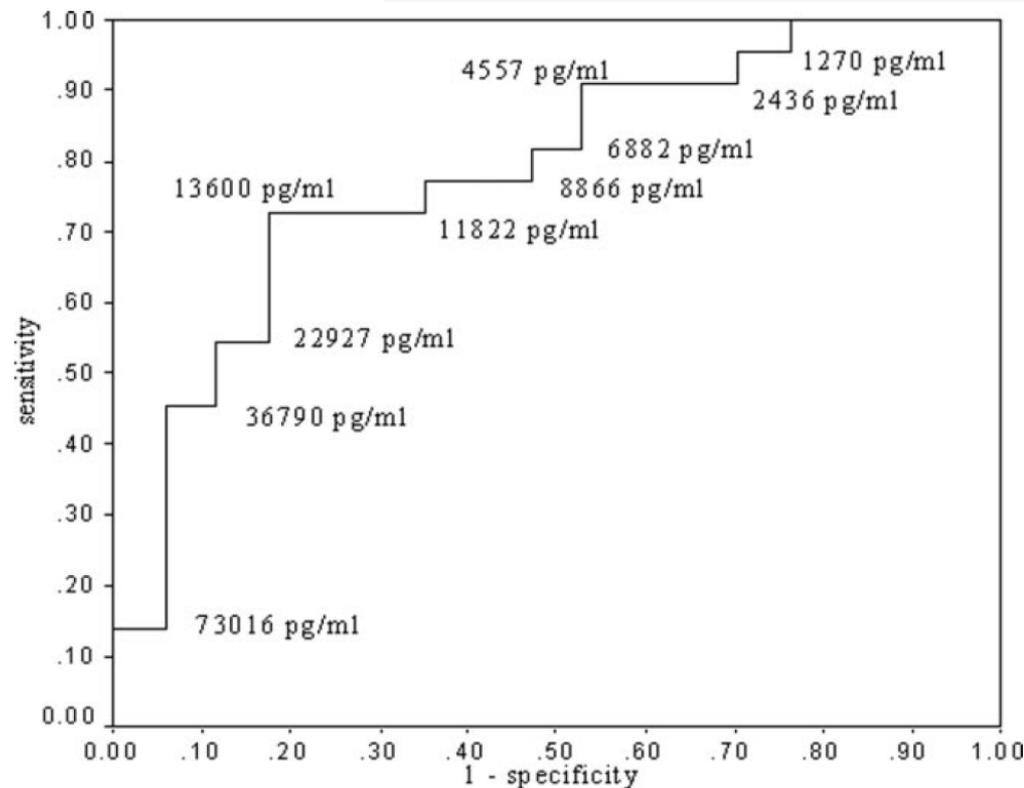
	Nonsurvivors	Survivors	<i>p</i> Value
Age, yrs	66 ± 10	60 ± 15	.15
Male sex	18/22	14/17	1.0
SOFA ^b	13 ± 3	9 ± 2	<.001
NT-proBNP, pg/mL ^b			
Median	34028	7856	.002
Interquartile range	11,735–49,320	1,291–12,972	
Lactate, ^b mmol/L	7.5 ± 6	4 ± 3	.034
Creatinine, ^b μmol/L	225 ± 77	161 ± 81	.016
LVSWI, ^c g · m ⁻¹ · m ⁻²	23 ± 10	36 ± 16	.005
cTnI, ^b μg/L			
Median	1.4	0.2	.002
Interquartile range	0.2–3.1	0.03–0.25	

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Table 4. Multivariate logistic regression of factors influencing intensive care unit mortality

Variable	<i>p</i> Value	OR	95% CI
Age, yrs	.143		
Sex	.136		
SOFA ^a	.031	4.71	1.15–19.3
NT-proBNP ^a per 1000 pg/mL increase	.041	1.13	1.005–1.26
Lactate, ^a mmol/L	.465		
LVSWI, ^b g · m ⁻¹ · m ⁻²	.536		
cTnI, ^a μg/L	.789		
Creatinine, ^a μmol/L	.371		



NH₂ terminal pro-brain natriuretic peptide plasma level as an early marker of prognosis and cardiac dysfunction in septic shock patients*

Roch et al. Crit Care Med 2005 Vol. 33, No. 5

