Normandie Université

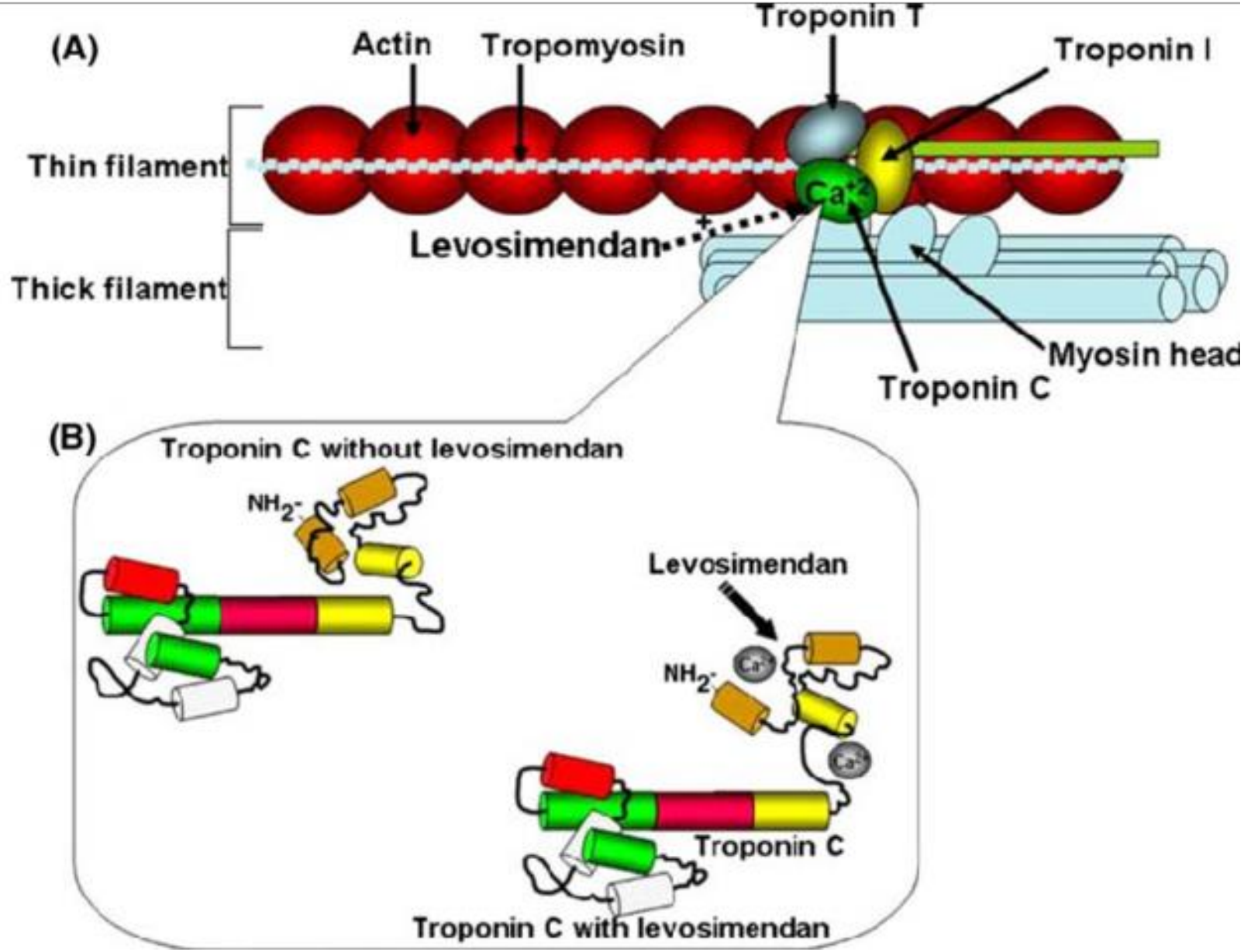
CHU Caen

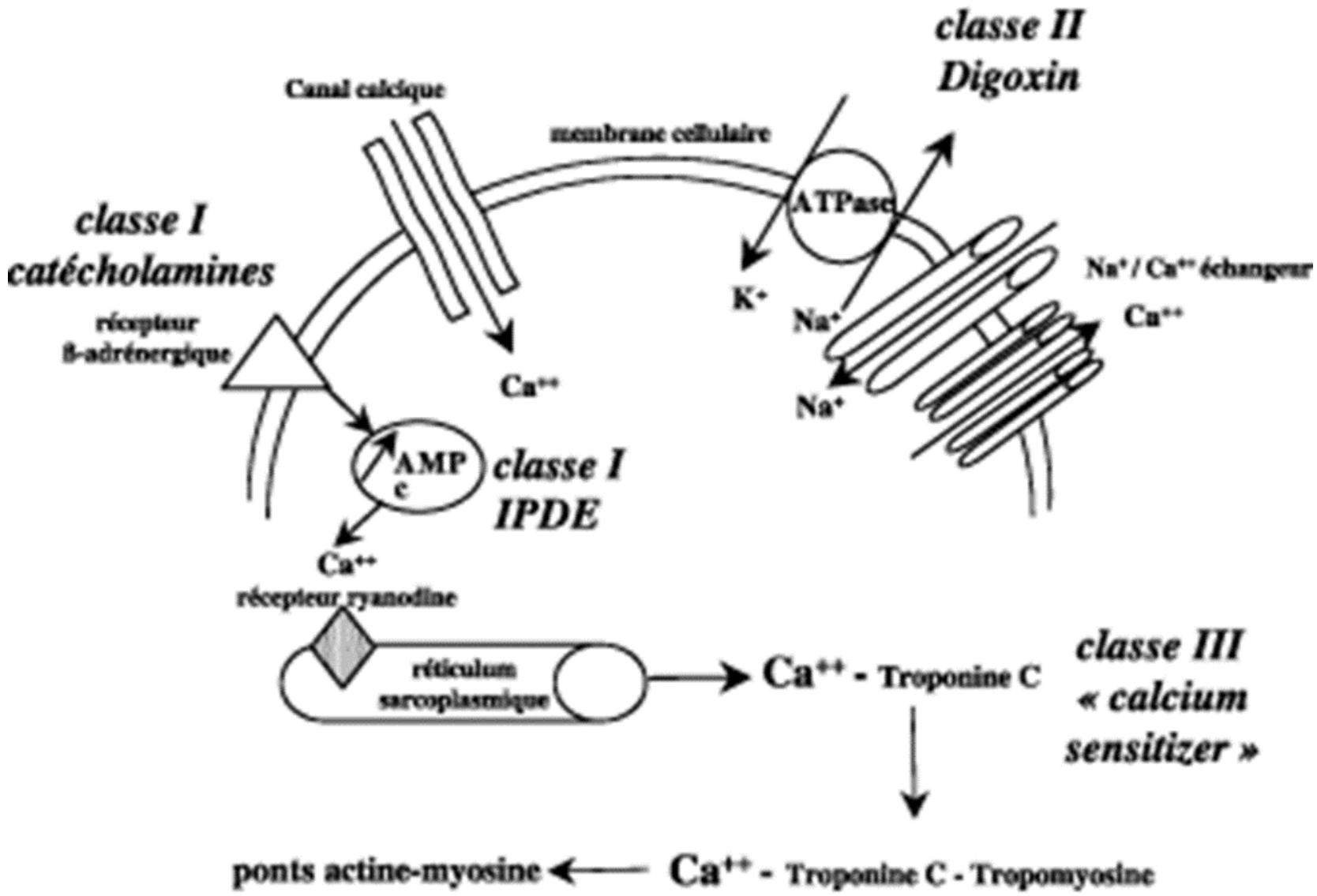
Place du levosimendan dans le choc septique

P.Ribstein DESC reanimation médicale

Tuteur: Pr Du Cheyron

Fig. 1 **a** Levosimendan as a cardiac enhancer. Levosimendan binds to troponin C during systole, increasing the sensitivity of myofilaments to Ca^{2+} levels. This process increases the contractility of myocardium during systole, without affecting diastolic function. **b** Levosimendan leads to an opening of the active sites of troponin C, increasing in this way its sensitivity to intracellular calcium (modified from reference [8])




classe II
Digoxin
Canal calcique
membrane cellulaire
ATPase
classe I
catécholamines
récepteur
ß-adrénergique
K+
Na+
Na+ / Ca++ échangeur
Ca++
Ca++
Na+
AMP
c
classe I
IPDE
Ca++
récepteur ryanodine
réticulum
sarcoplasmique
Ca++ - Troponine C
classe III
« calcium
sensitizer »
ponts actine-myosine
Ca++ - Troponine C - Tropomyosine

Mécanisme d’action

- **Fonction inotrope positive:**

Fixation sur la troponine C après qu’elle ait fixé le calcium. Prolonge le temps de contact entre l’actine et la myosine et donc augmente le nombre de ponts entre l’actine et la myosine.

Améliore la réponse des myofilaments au calcium.

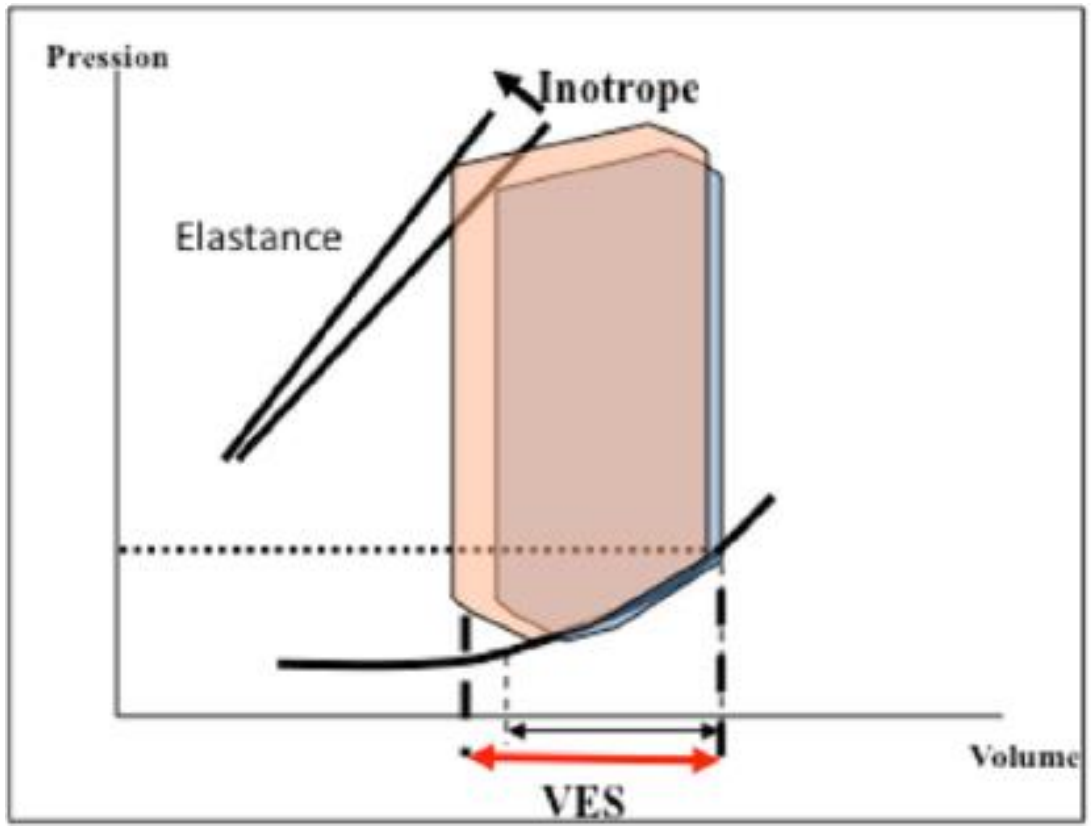
- **Fonction lusitrope positive:**

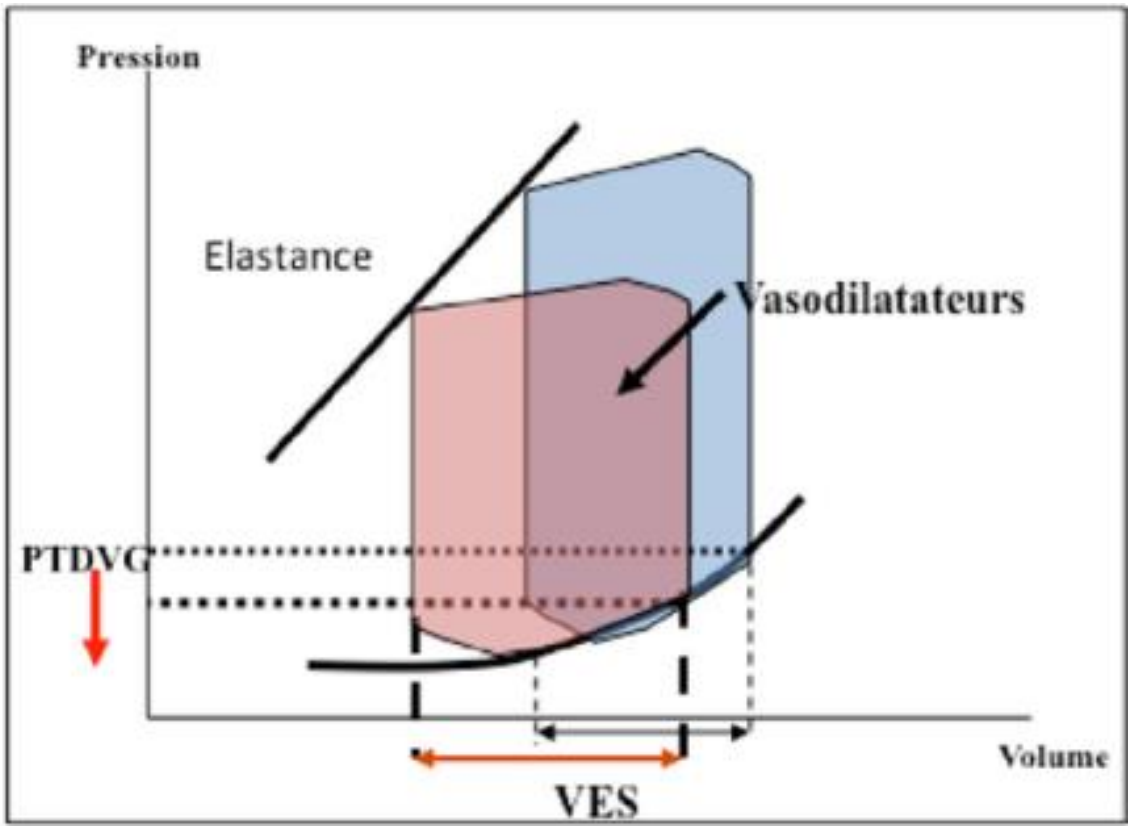
Seul « sensibilisateur calcique » qui se fixe à la troponine C de façon calcium-dépendante.

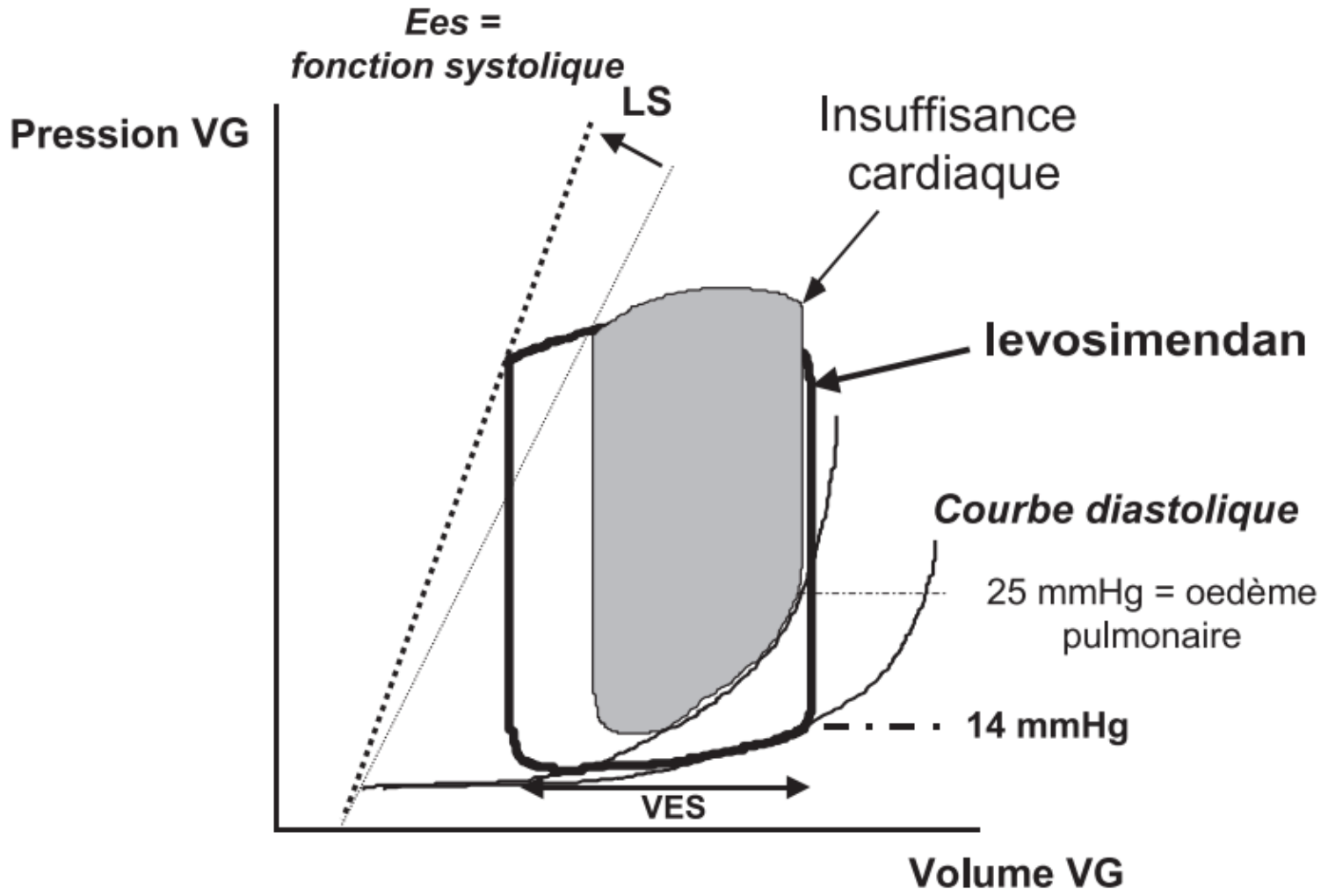
- **Effet vasodilateur**

Par l’ouverture des canaux potassiques ATP-dépendants. Vasodilatation artérielle, diminution de la postcharge du ventricule gauche.

Amélioration du débit coronaire et du débit sanguin rénal.


Pression
Inotrope
Elastance
Volume
VES

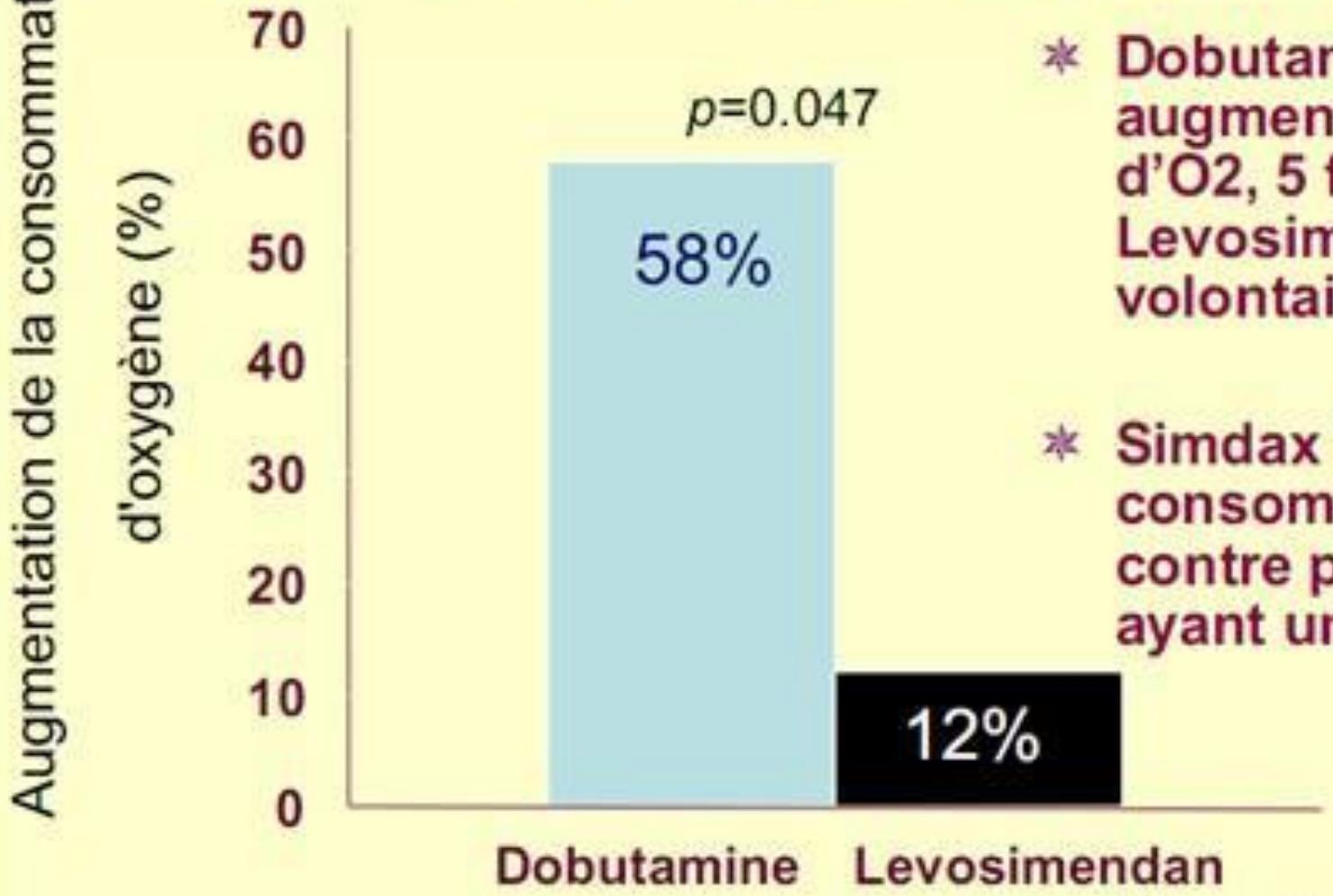

Pression
Elastance
Vasodilatateurs
PTDVG
Volume
VES



Le lévosimendan améliore les fonctions systolique et diastolique. La pente d'élastance (Ees) et la courbe de remplissage diastolique sont les deux améliorées.

VG ventricule gauche, VES volume d'éjection systolique, LS lévosimendan.

Simdax n'augmente pas la consommation d'oxygène.

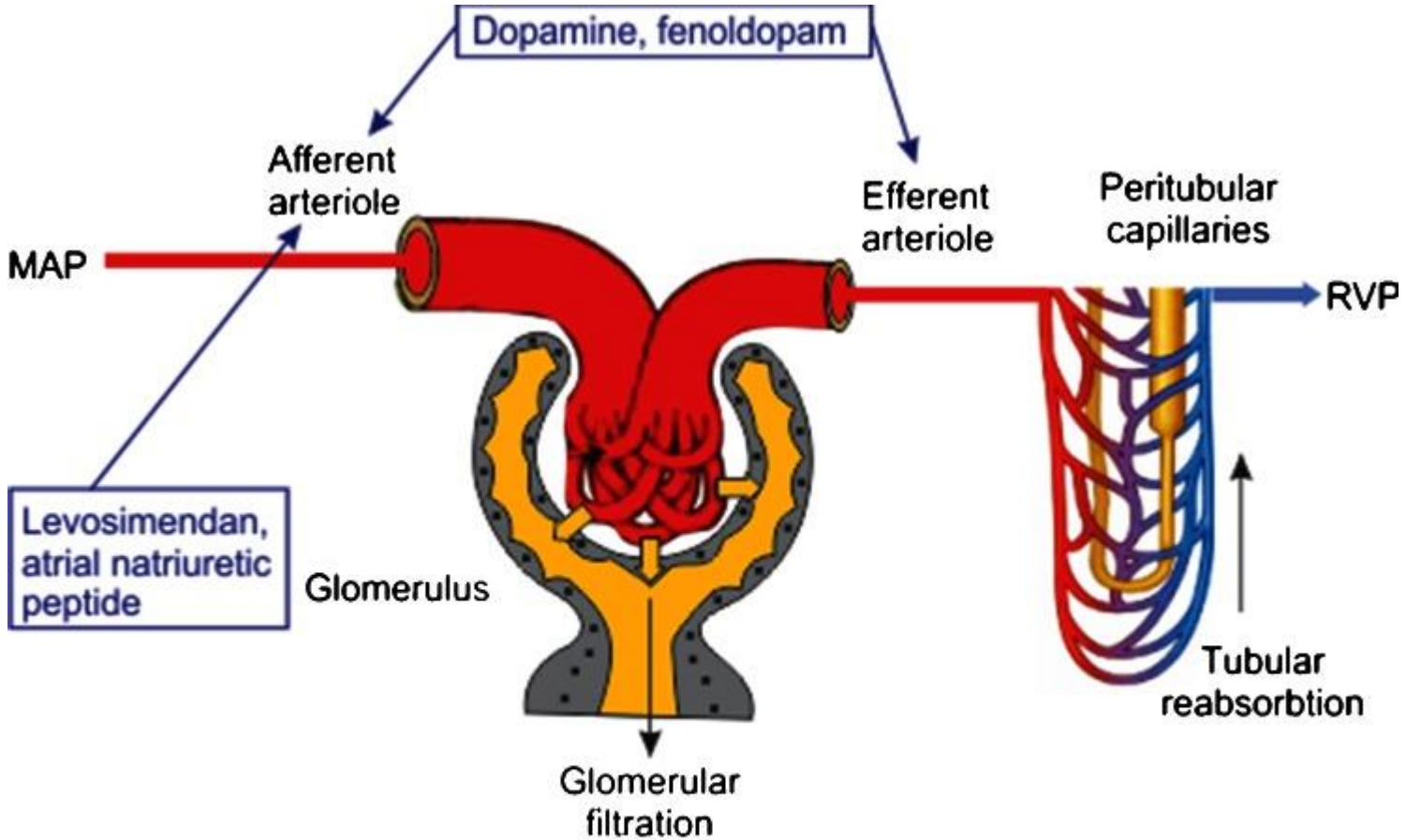


* Dobutamine a causé une augmentation de consommation d'O2, 5 fois plus importante que Levosimendan (chez des volontaires en bonne santé).

* Simdax était neutre pour la consommation d'O2 myocardique contre placebo (chez les patients ayant une IC décompensée).

Ukkonen *et al. Clin 1997 Pharmacol Ther* 61:596-607;
Ukkonen et al. *2000 Clin Pharmacol* Ther 68: 522-31.

Renal Effects of Levosimendan: A Consensus Report
Mehmet B.Yilmaz, 2013



Predominant afferent vasodilation: RBF ↑ ; GFR ↑
Predominant efferent vasodilation: RBF ↑ ; GFR ↓
Afferent + efferent vasodilation: RBF ↑ ↑ ; GFR ↑ ↓

Pharmacocinétique

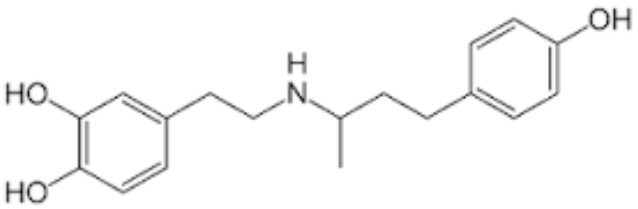
Demie - vie très courte.

Disparition rapide en moins de trois heures.

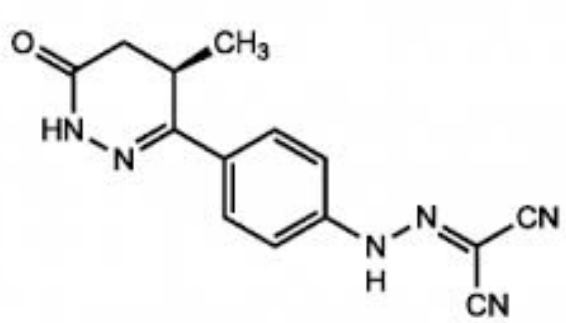
Transformation par le foie en un métabolite actif nommé OR-1855.

OR-1855: activité myocardique aussi importante que le lévosimendan mais une demi-vie de 80 heures.

Tarifs



1 flacon
250mg/20mL
0,69 euros HT

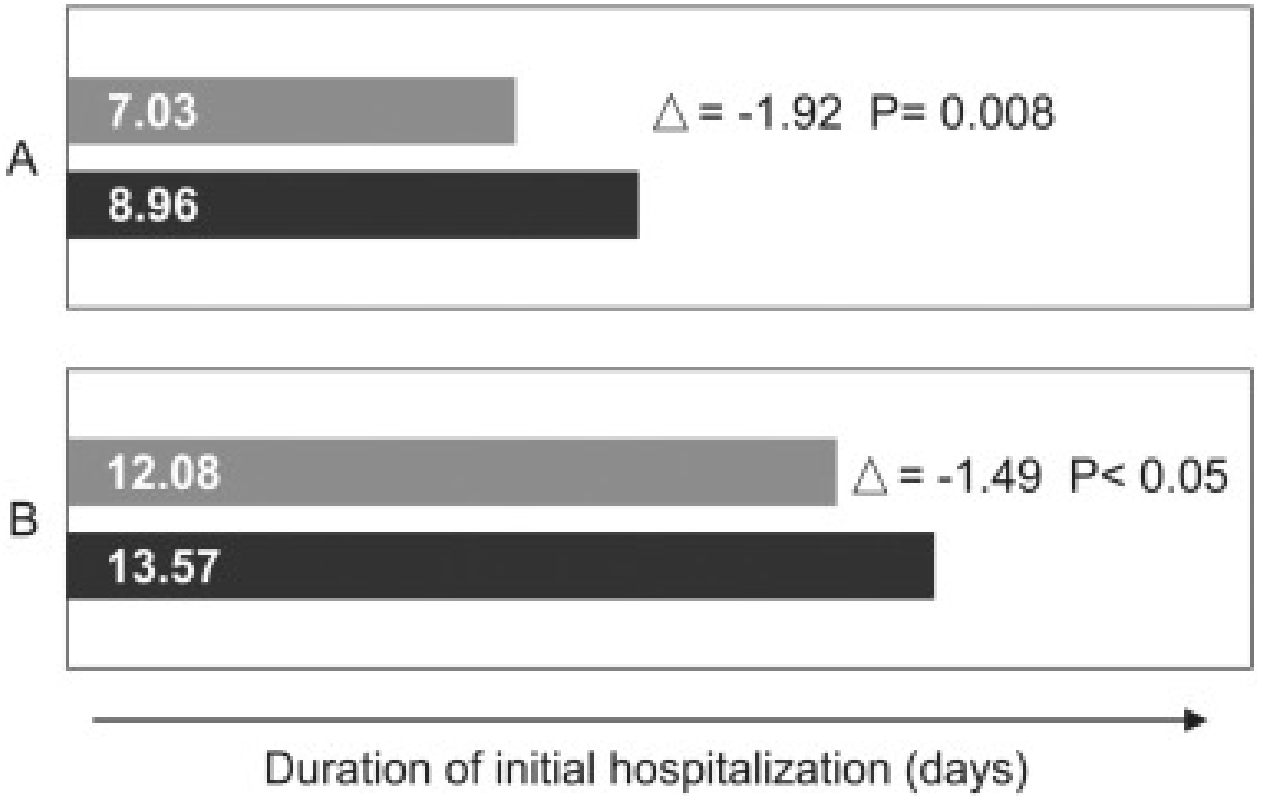


1 flacon 12,5mg/5mL
1000 euros HT

International Journal of CARDIOLOGY

Pharmaco-economics of levosimendan in cardiology: A European perspective

M.S. Nieminen



Levosimendan, on top of standard of care, shortened the hospital stay for patients with acute heart failure in a phase III study vs. placebo (panel A, from de Lissavoy), and in a single center large registry study vs. dobutamine (panel B, from Fedele). Levosimendan (gray), dobutamine (black)

Table 1
Costs and savings of levosimendan treatment of patients hospitalized for acute heart failure.

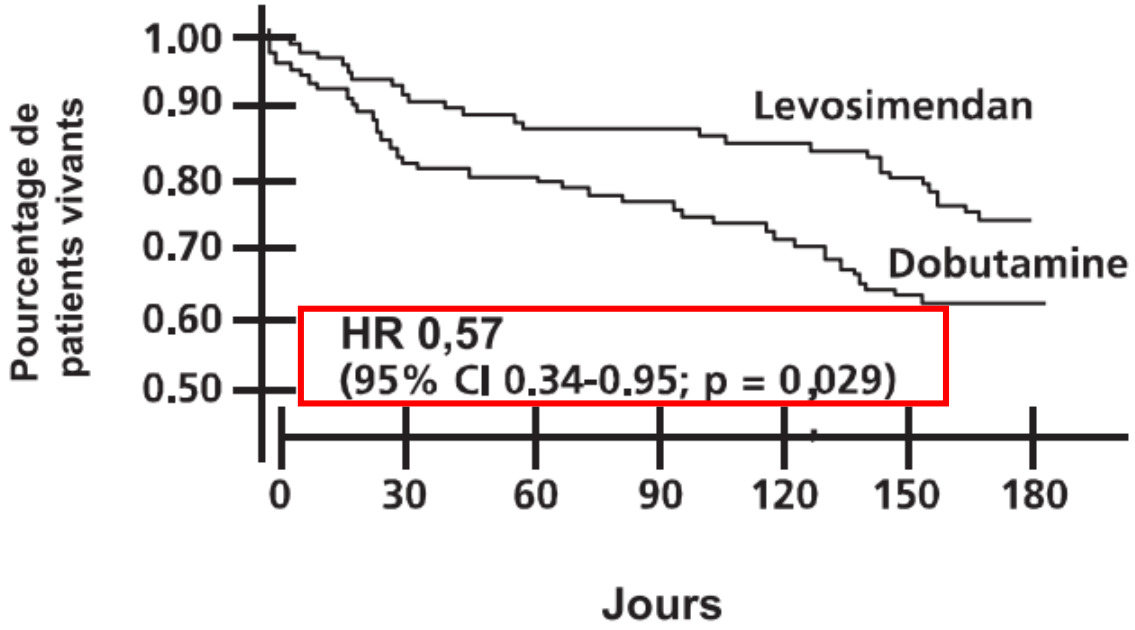
Country	Costs (€)		Net savings (€)
	C_L^a	Cardiac ward (per day)[b]	By using levosimendan[c]
Italy	648.86	416[d]	−12.58
Spain	598.79	421[e]	−70.60
Greece	487.07	361[f]	−86.92
Germany	715.00	450[g]	−0.50
Sweden	648.65	631[h]	−354.64
Finland	659.18	450[i]	−56.32
Israel	561.57	368[j]	−23.55

Additional drug costs of a levosimendan treatment vs. comparator treatment calculated according to the text; b excluding the costs for levosimendan; c net savings considering a reduction of 1.59 days of stay in the cardiac ward obtained by levosimendan treatment; d calculated from DRG 127 in the ICD9-CM DRG classification; e in 2013 according to RECH; f calculated from the data by Parissis et al. [24]; g data calculated from the Fallpauschalen-Katalog for the relevant DRG codes (InEK 2014); h average cardiac ward cost at 'Akademiska sjukhuset', Uppsala, Sweden; i from HUS Palveluhinnasto 2015; j according to the Israeli Ministry of Health

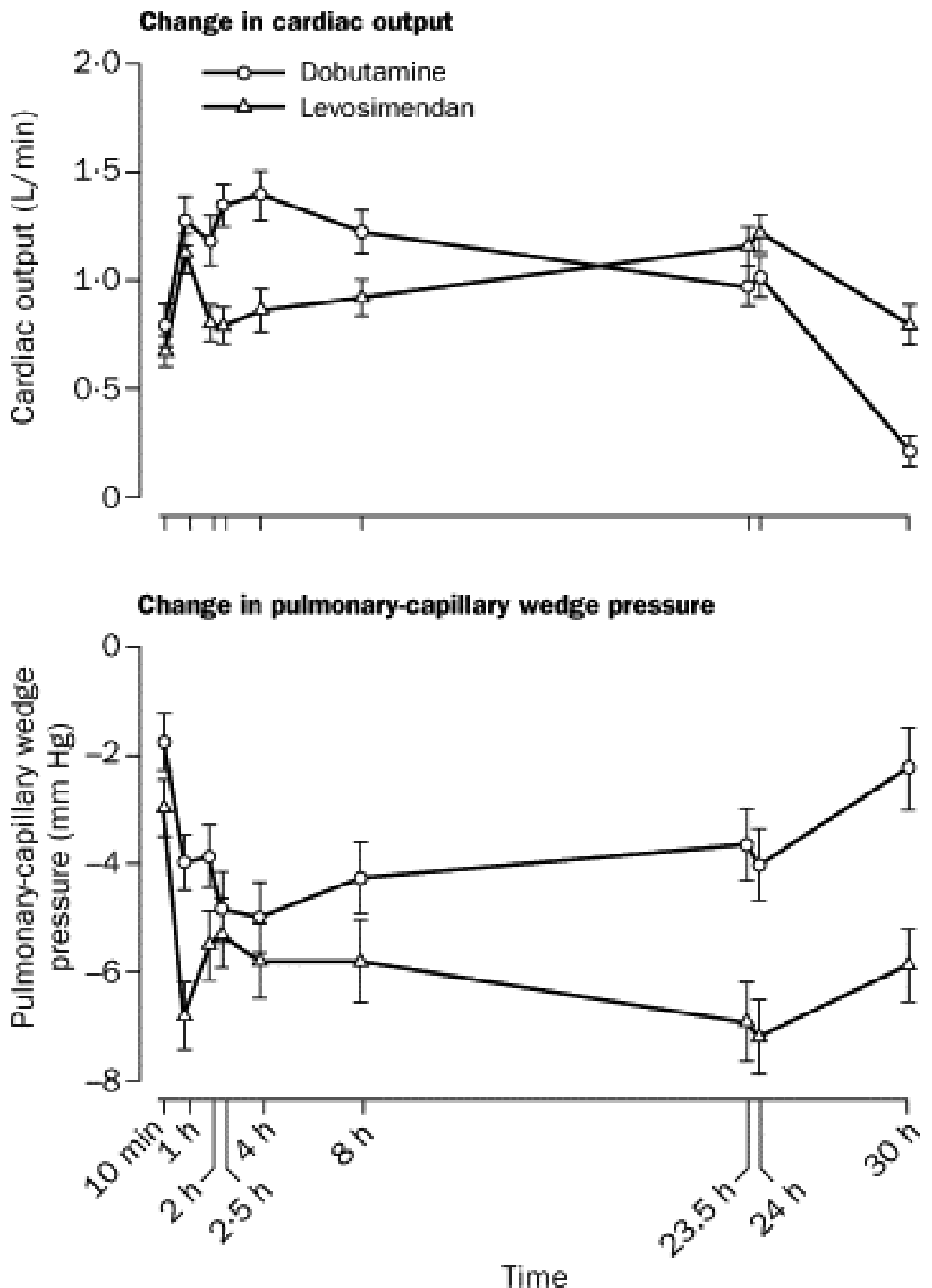
THE LANCET

Efficacy and safety of intravenous levosimendan compared with dobutamine in severe low-output heart failure (the LIDO study): a randomised double-blind trial

Follath F, 2012



At 180 days, 27 (26%) levosimendan-group patients had died, compared with 38 (38%) in the dobutamine group

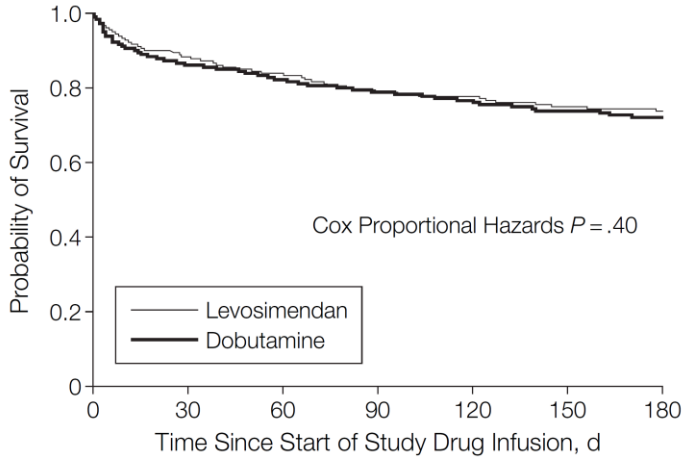

Change in cardiac output
Dobutamine
Levosimendan
Cardiac output (L/min)
2·0
1·5
1·0
0·5
0
Change in pulmonary-capillary wedge pressure
Pulmonary-capillary wedge pressure (mm Hg)
0
−2
−4
−6
−8
10 min
1 h
2 h
2·5 h
4 h
8 h
23·5 h
24 h
30 h
Time

JAMA®
The Journal of the American Medical Association

Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure

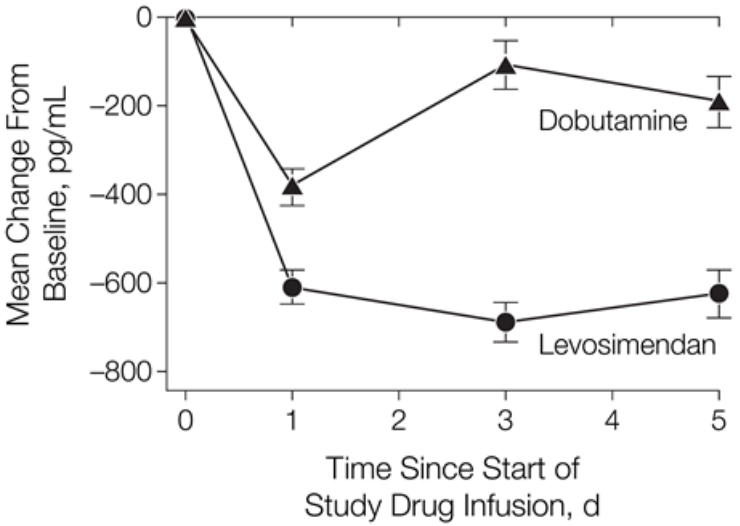
The SURVIVE Randomized Trial

Figure 2. Effect of Dobutamine and Levosimendan Treatment on All-Cause Mortality During 180 Days Following the Start of Study Drug Infusion



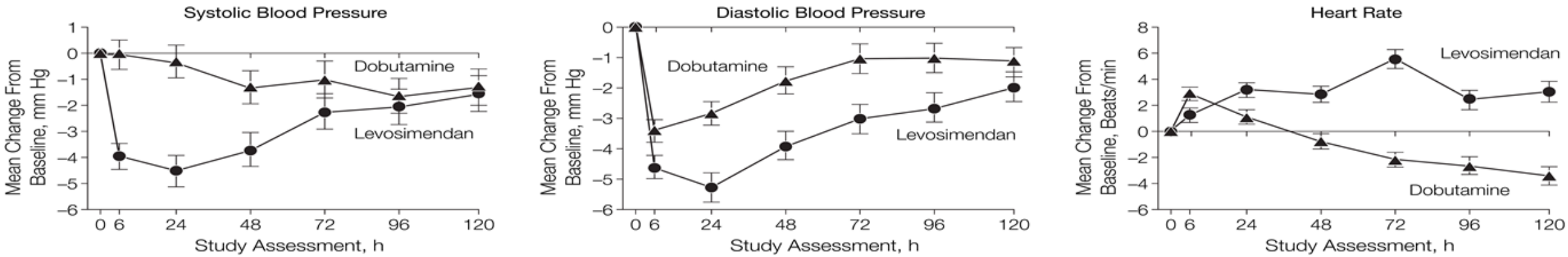
No. at Risk					
Levosimendan	664	608	586	525	462
Dobutamine	663	596	568	519	454

Figure 3. Mean Change From Baseline in B-Type Natriuretic Peptide Levels at 1, 3, and 5 Days by Treatment Group



There was a significantly greater mean (SE) change from baseline in plasma B-type natriuretic peptide levels in the levosimendan group compared with the dobutamine group at 1, 3, and 5 days after initiation of study drug infusion. $P<.001$ at all 3 time points. Statistical significance was determined using Kruskal-Wallis test with treatment effect.

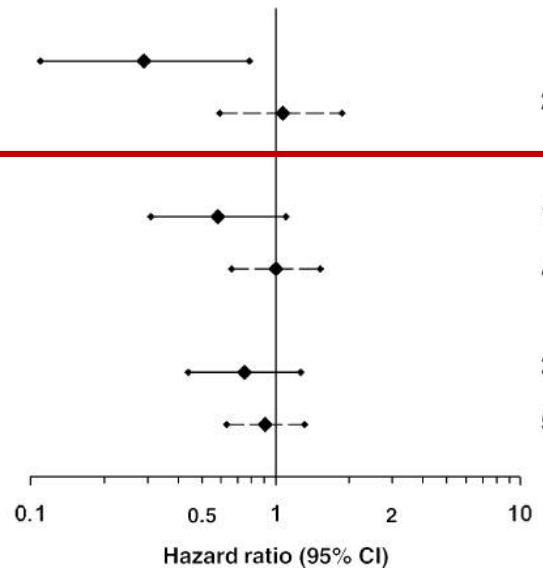
Figure 4. Mean Change From Baseline in Systolic Blood Pressure, Diastolic Blood Pressure, and Heart Rate Through 5 Days by Treatment Group

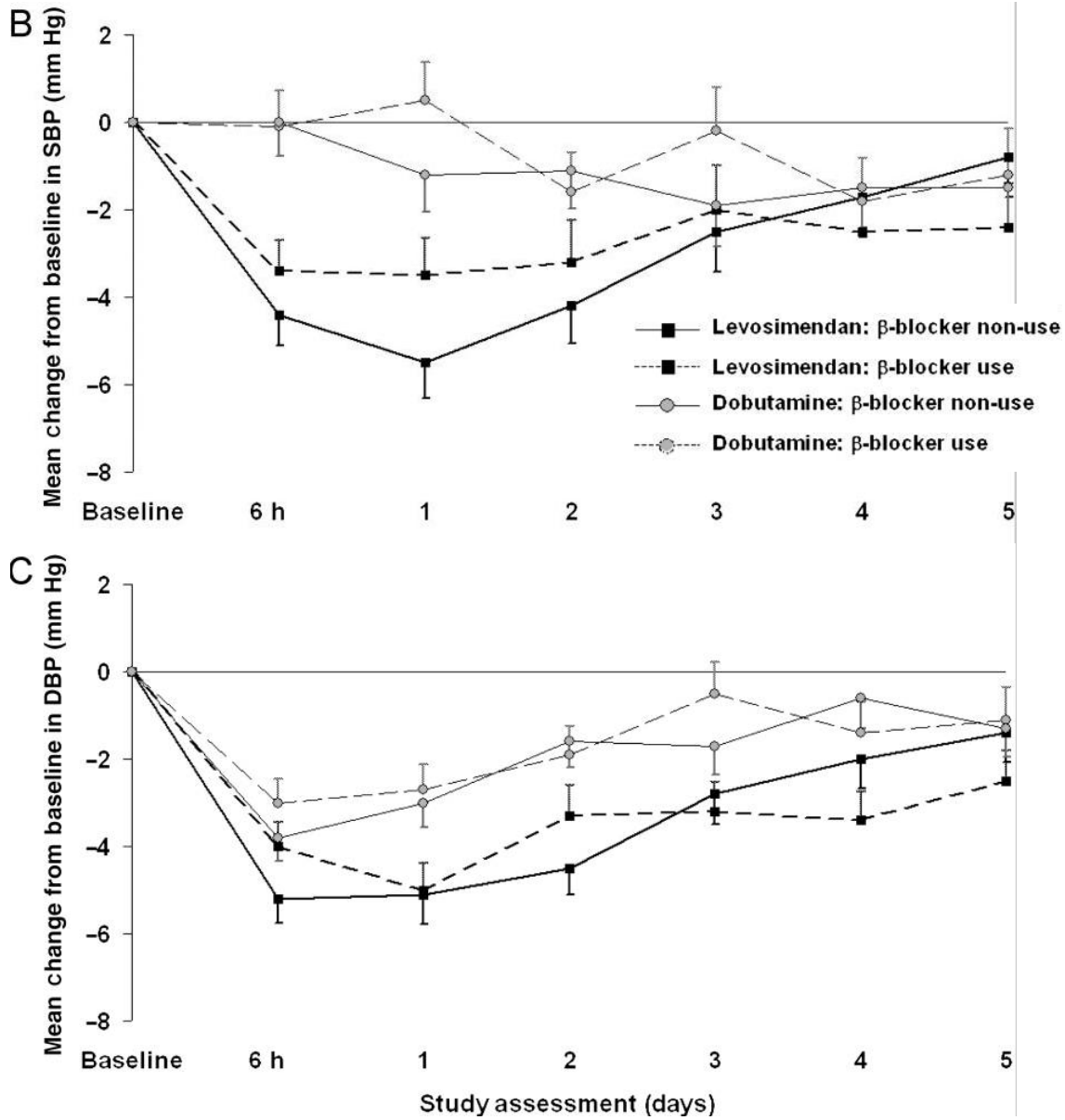


Levosimendan vs. dobutamine: outcomes for acute heart failure patients on beta-blockers in SURVIVE

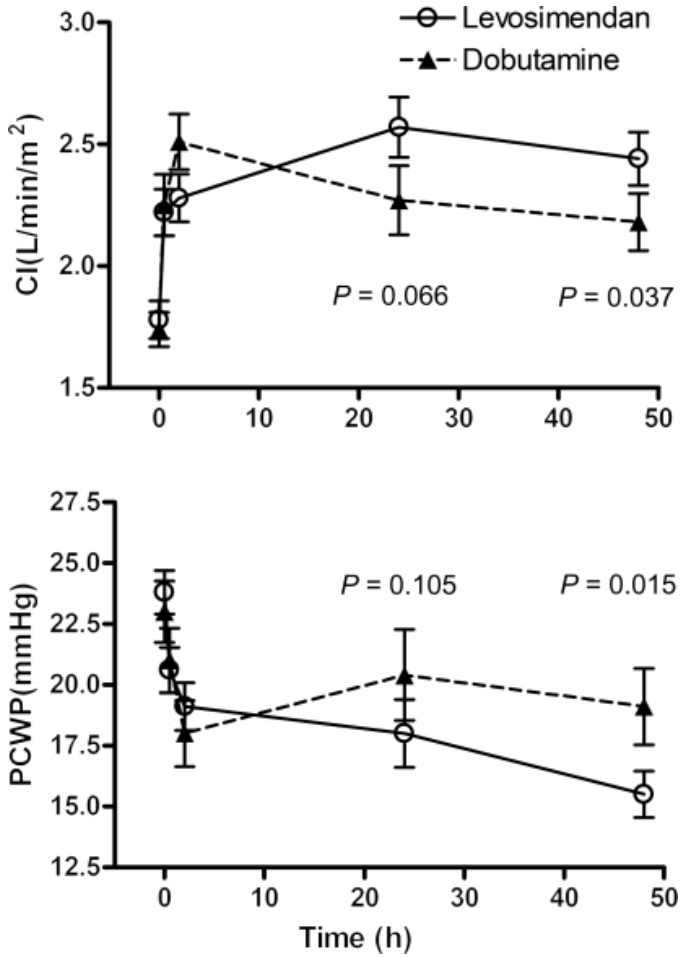
C

Day	Use of β-blocker	Favours Levosimendan	Favours Dobutamine	Deaths, *n* (%) Levosimendan	Deaths, *n* (%) Dobutamine	*P*-value HR	*P*-value Interaction
0–5	Yes			5 (1.5)	17 (5.1)	0.01	0.03
	No			24 (7.3)	23 (7.0)	0.87	
0–14	Yes			15 (4.5)	25 (7.5)	0.10	0.16
	No			44 (13.4)	44 (13.3)	1.00	
0–31	Yes			24 (7.1)	31 (9.3)	0.29	0.55
	No			55 (16.8)	60 (18.2)	0.62	





Intravenous levosimendan vs. dobutamine in acute decompensated heart failure patients on beta-blockers



Mean cardiac index and pulmonary capillary wedge pressure from baseline to 48 h after the start of the study drug infusion [intention-to-treat (ITT) population]

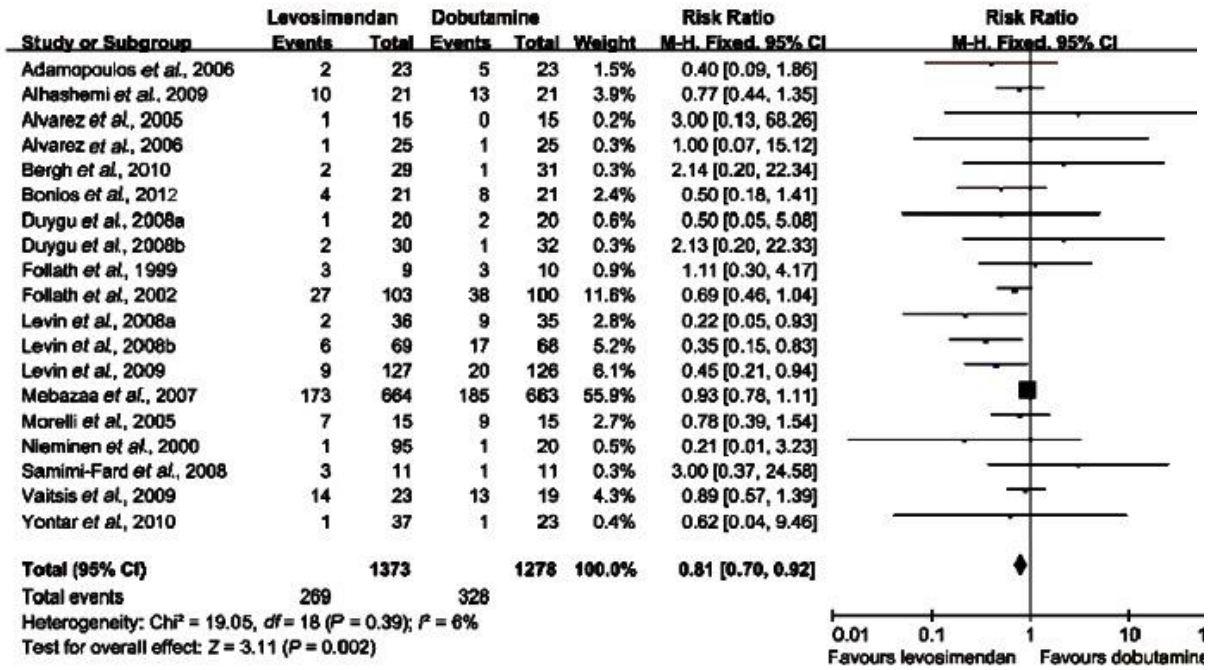
Time point	Levosimendan (*n* = 29)	Dobutamine (*n* = 31)	*P*-value[a]
BNP (ng/mL)			
Baseline, mean (SD)	1114 (1214)	979 (748)	
24 h mean change (SD)	−432 (727)	−324 (533)	0.248
48 h mean change (SD)	−507 (785)	−260 (475)	0.029
1 month mean change (SD)	−206 (1094)	52 (471)	0.138

B-type natriuretic peptide values (ng/mL) at baseline, change from baseline to 24, and 48 h after the start of the infusion, and at the 1-month follow-up (ITT population)

Bergh, 2010

Journal of Zhejiang University SCIENCE B

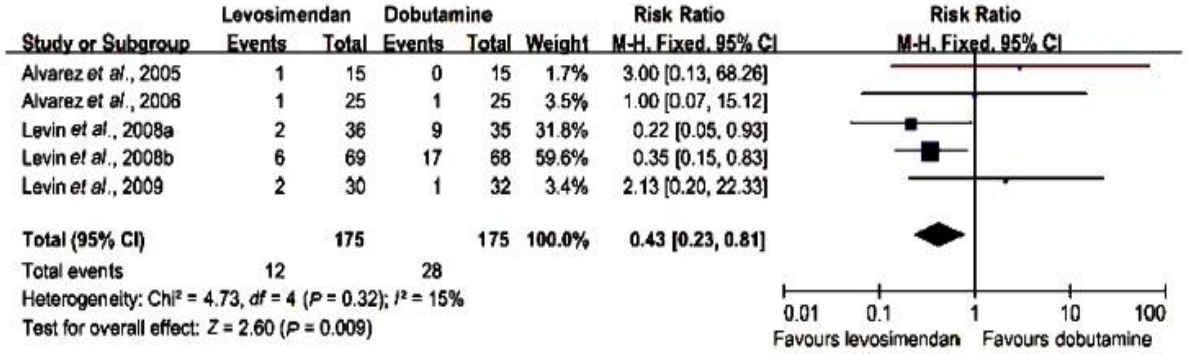
Levosimendan versus dobutamine in critically ill patients: a meta-analysis of randomized controlled trials



Study or Subgroup	Levosimendan Events	Total	Dobutamine Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Adamopoulos *et al.*, 2006	2	23	5	23	1.5%	0.40 [0.09, 1.86]
Alhashemi *et al.*, 2009	10	21	13	21	3.9%	0.77 [0.44, 1.35]
Alvarez *et al.*, 2005	1	15	0	15	0.2%	3.00 [0.13, 68.26]
Alvarez *et al.*, 2006	1	25	1	25	0.3%	1.00 [0.07, 15.12]
Bergh *et al.*, 2010	2	29	1	31	0.3%	2.14 [0.20, 22.34]
Bonios *et al.*, 2012	4	21	8	21	2.4%	0.50 [0.18, 1.41]
Duygu *et al.*, 2008a	1	20	2	20	0.6%	0.50 [0.05, 5.08]
Duygu *et al.*, 2008b	2	30	1	32	0.3%	2.13 [0.20, 22.33]
Follath *et al.*, 1999	3	9	3	10	0.9%	1.11 [0.30, 4.17]
Follath *et al.*, 2002	27	103	38	100	11.6%	0.69 [0.46, 1.04]
Levin *et al.*, 2008a	2	36	9	35	2.8%	0.22 [0.05, 0.93]
Levin *et al.*, 2008b	6	69	17	68	5.2%	0.35 [0.15, 0.83]
Levin *et al.*, 2009	9	127	20	126	6.1%	0.45 [0.21, 0.94]
Mebazaa *et al.*, 2007	173	664	185	663	55.9%	0.93 [0.78, 1.11]
Morelli *et al.*, 2005	7	15	9	15	2.7%	0.78 [0.39, 1.54]
Nieminen *et al.*, 2000	1	95	1	20	0.5%	0.21 [0.01, 3.23]
Samimi-Fard *et al.*, 2008	3	11	1	11	0.3%	3.00 [0.37, 24.58]
Vaitsis *et al.*, 2009	14	23	13	19	4.3%	0.89 [0.57, 1.39]
Yontar *et al.*, 2010	1	37	1	23	0.4%	0.62 [0.04, 9.46]
Total (95% CI)		**1373**		**1278**	**100.0%**	**0.81 [0.70, 0.92]**
Total events	269		328			

Heterogeneity: Chi² = 19.05, *df* = 18 (*P* = 0.39); I^2 = 6%
Test for overall effect: *Z* = 3.11 (*P* = 0.002)

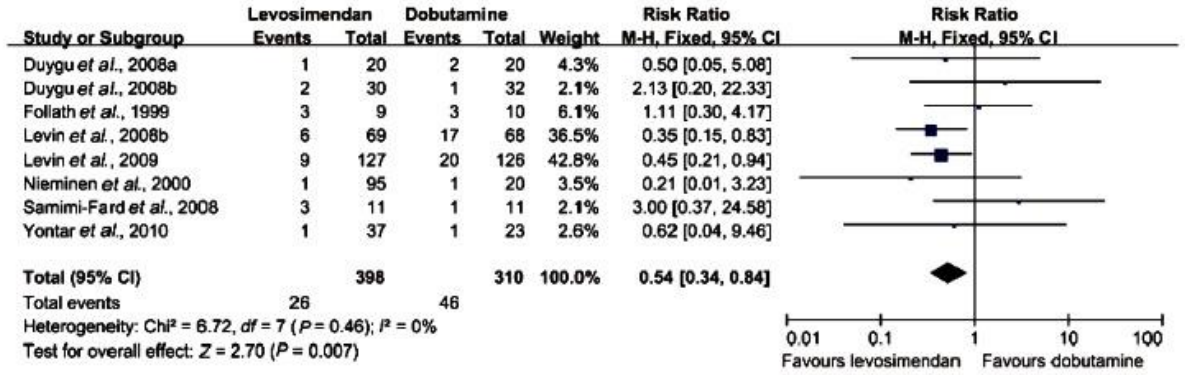
Forest plot for the risk of mortality in the overall population



Study or Subgroup	Levosimendan Events	Total	Dobutamine Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Alvarez *et al.*, 2005	1	15	0	15	1.7%	3.00 [0.13, 68.26]
Alvarez *et al.*, 2006	1	25	1	25	3.5%	1.00 [0.07, 15.12]
Levin *et al.*, 2008a	2	36	9	35	31.8%	0.22 [0.05, 0.93]
Levin *et al.*, 2008b	6	69	17	68	59.6%	0.35 [0.15, 0.83]
Levin *et al.*, 2009	2	30	1	32	3.4%	2.13 [0.20, 22.33]
Total (95% CI)		**175**		**175**	**100.0%**	**0.43 [0.23, 0.81]**
Total events	12		28			

Heterogeneity: Chi² = 4.73, *df* = 4 (*P* = 0.32); I^2 = 15%
Test for overall effect: *Z* = 2.60 (*P* = 0.009)

Forest plot for the risk of mortality in the cardiac surgery setting



Study or Subgroup	Levosimendan Events	Total	Dobutamine Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Duygu *et al.*, 2008a	1	20	2	20	4.3%	0.50 [0.05, 5.08]
Duygu *et al.*, 2008b	2	30	1	32	2.1%	2.13 [0.20, 22.33]
Follath *et al.*, 1999	3	9	3	10	6.1%	1.11 [0.30, 4.17]
Levin *et al.*, 2008b	6	69	17	68	36.5%	0.35 [0.15, 0.83]
Levin *et al.*, 2009	9	127	20	126	42.8%	0.45 [0.21, 0.94]
Nieminen *et al.*, 2000	1	95	1	20	3.5%	0.21 [0.01, 3.23]
Samimi-Fard *et al.*, 2008	3	11	1	11	2.1%	3.00 [0.37, 24.58]
Yontar *et al.*, 2010	1	37	1	23	2.6%	0.62 [0.04, 9.46]
Total (95% CI)		**398**		**310**	**100.0%**	**0.54 [0.34, 0.84]**
Total events	26		46			

Heterogeneity: Chi² = 6.72, *df* = 7 (*P* = 0.46); I^2 = 0%
Test for overall effect: *Z* = 2.70 (*P* = 0.007)

Forest plot for the risk of mortality in the studies including all patients who had ischemic HF

Huang, 2013

Levosimendan dans le choc septique

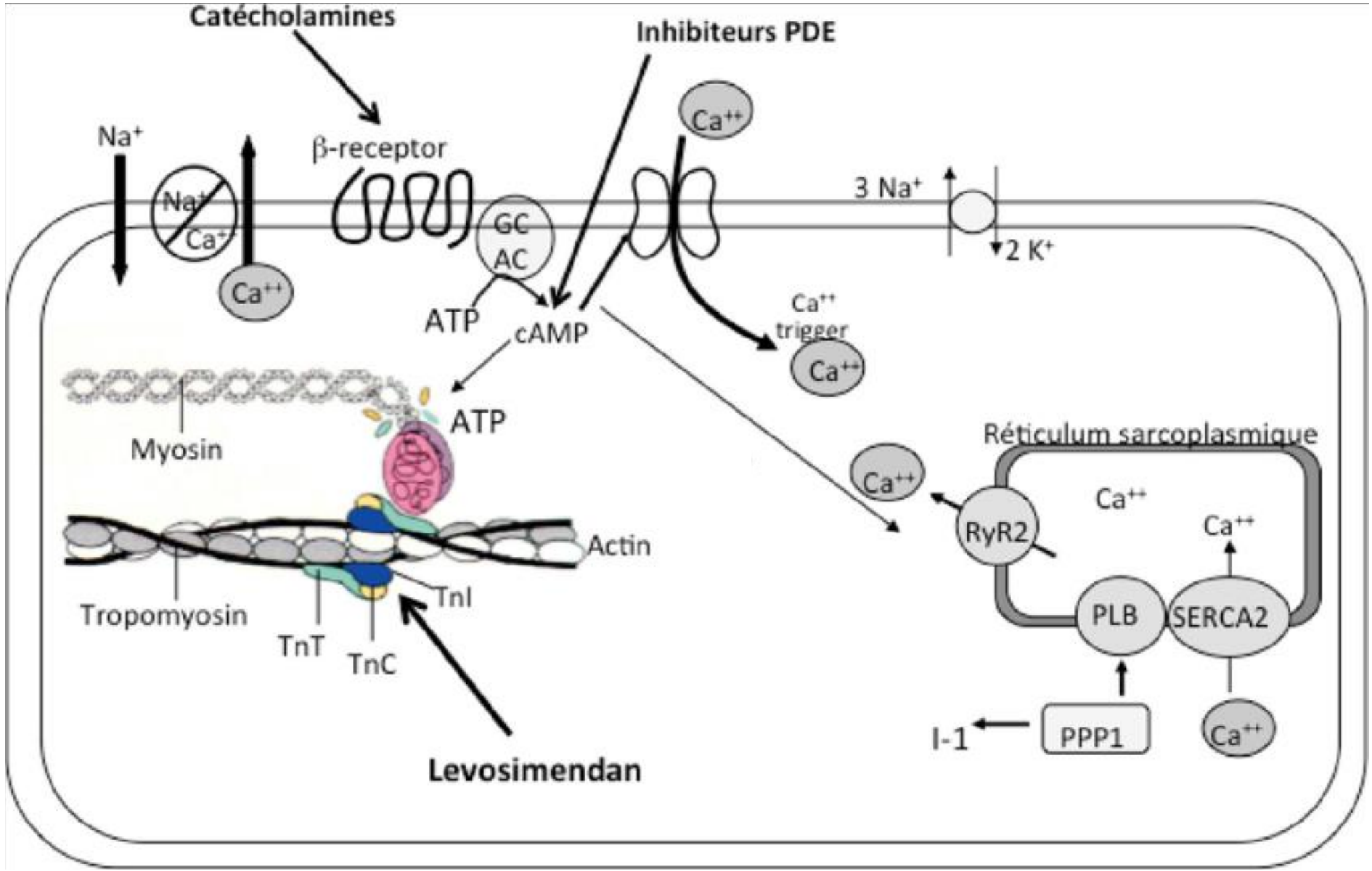
Le choc septique on observe des anomalies circulatoires, une hypotension artérielle résistante au remplissage secondaire à une combinaison: vasodilatatrice, d'hyporéactivité aux catécholamines, et de dépression myocardique (action des IL1 et TNFα).

60% des patients présentent une hypokinésie ventriculaire gauche.

En contraste avec les catécholamines le lévosimendan majore la fonction inotrope avec une faible majoration de la consommation en oxygène.

Il a une composante anti-ischémique, anti-inflammatoire et antiapoptotique

Il semble pouvoir augmenter la survie au décours d'un choc septique


Catécholamines
Inhibiteurs PDE
Na+
β-receptor
Ca++
3 Na+
2 K+
GC
AC
ATP
cAMP
Ca++
trigger
Ca++
ATP
Myosin
Réticulum sarcoplasmique
Ca++
Ca++
RyR2
Ca++
Actin
PLB
SERCA2
Tropomyosin
TnI
TnT
TnC
I-1
PPP1
Ca++
Levosimendan

Effects of levosimendan on systemic and regional hemodynamics in septic myocardial depression.

Morelli A, 2005

Table 3 Echocardiographic measurements (*EDVI* end diastolic volume index, *ESVI* end systolic volume index, *LVEF* left ventricular ejection fraction)

	Levosimendan		Dobutamine	
	Baseline	24 h	Baseline	24 h
EDVI (ml m^{-1})	75.8±23.8	66.2±24.6*,**	84.2±25.1	82.9±26.4
ESVI (ml m^{-1})	46.7±21.9	36.9±19.4*,**	52.4±25.8	50.5±25.3
LVEF (%)	37.1±3.0	45.4±8.4*	37.3±2.6	40.8±11.3

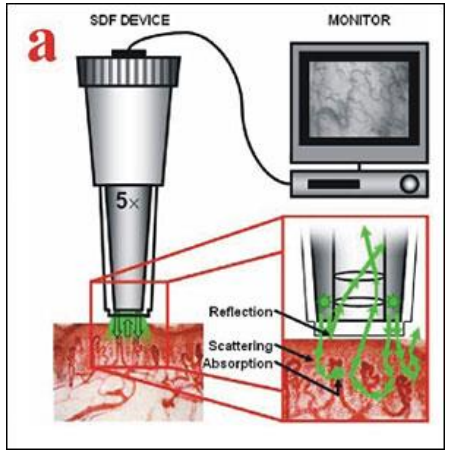
*$p<0.05$ baseline vs. 24 h, **$p<0.05$ levosimendan vs. dobutamine after 24 h

Table 4 Gastric mucosal perfusion and biological variables; fluid perfused included all fluids administered during the 24-h study period (*GMP* gastric mucosal perfusion expressed as percentage, $\Delta P_{g\text{-}a}CO_2$ gastric PCO_2–arterial PCO_2)

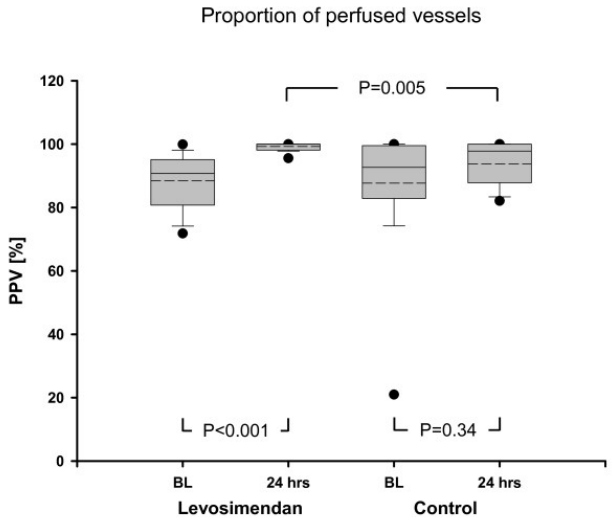
	Levosimendan		Dobutamine	
	Baseline	24 h	Baseline	24 h
Troponin cTnI (ng/ml)	0.14±0.07	0.13±0.06***	0.14±0.08	0.15±0.06
GMP (%)	–	+55.3±20.1***	–	2.5±4.7
$\Delta P_{g\text{-}a}CO_2$ (mmHg)	15.3±1.1**	11.9±1.3*,***	14.2±1.2	14.4±1.2
Arterial lactate (mmol l^{-1})	4.9±1.2	3.7±0.7*,***	5.2±1.1	5.2±1.0
Creatinine clearence (ml min^{-1})	43.9±12.8	72.1±16.2*,***	51.2±17.0	51.3±13.3
Urinary output (ml 24 h^{-1})	–	2028±461***	–	1521±302
Fluid perfused (ml 24 h^{-1})	–	5907±330***	–	4311±136
Norepinephrine rate (μg kg^{-1} min^{-1})	0.22±0.07	0.22±0.06	0.23±0.05	0.23±0.06

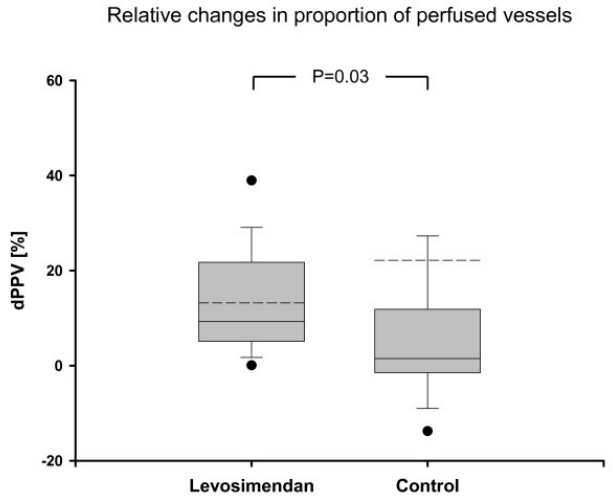
Levosimendan for resuscitating the microcirculation in patients with septic shock: a randomized controlled study

Morelli, 2010



Sidestream Dark Field Imaging (SDF, imagerie en champ sombre avec éclairage de côté)





The relative increase of perfused vessel density vs. baseline was significantly higher in the levosimendan group than in the control group (dMFIm 10 (3; 23)% vs. 0 (-1; 9)%; *P* = .007; dMFIs 47 (26; 83)% vs. 10 (-3; 27); *P* < .001)

This effect was not correlated with changes in systemic flow variables

Absolute and relative changes in microcirculatory variables. BL, baseline; dPPV, relative changes in proportion of perfused vessels; PPV, proportion of perfused vessels

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Levosimendan for the Prevention of Acute Organ Dysfunction in Sepsis

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Etude prospective multicentrique randomisée en double aveugle contre placébo.

Critère d'inclusion: patient en choc septique à plus de 4h du début des vasopresseurs.

Critère d'évaluation principal: le « daily SOFA score ».

Critères d'évaluations secondaires: nombre de jours sans catécholamines, le nombre de jours sans ventilation mécanique, la durée de sevrage ventilatoire, la mortalité à J28

Résultats de l’étude

Le « daily SOFA score » moyen cardiovasculaire est plus élevé dans le groupe lévosimendan que placébo (difference moyenne, 0.25; 95% CI, 0.04 - 0.46; P = 0.01)

Il n’est pas retrouvé de différence significative sur la mortalité à J28 entre le groupe levosimendan (34,5%) et le groupe placebo (30,9%) (95% CI,−4.5 to 11.7; P = 0.43).

Le succès de sevrage ventilatoire dans les 28 jours est inferieur dans le groupe lévosimendan (HR, 0.77; 95% CI, 0.60 to 0.97; P = 0.03)

Table 3. Clinical Outcomes.*

Outcome	Levosimendan (N=258)	Placebo (N=257)	Absolute Difference (95% CI)†	P Value
Primary outcome				
Mean daily total SOFA score	6.68±3.96	6.06±3.89	0.61 (−0.07 to 1.29)	0.053
Respiratory	1.70±1.18	1.56±1.15	0.14 (−0.06 to 0.34)	0.23
Coagulation	0.75±1.05	0.75±1.02	0.00 (−0.18 to 0.17)	0.55
Hepatic	0.51±0.84	0.45±0.77	0.06 (−0.08 to 0.19)	0.65
Cardiovascular	2.27±1.20	2.02±1.20	0.25 (0.04 to 0.46)	0.01
Renal	1.46±1.49	1.28±1.38	0.18 (−0.07 to 0.42)	0.32
Mean daily SOFA score excluding cardiovascular score	4.41±3.13	4.05±3.07	0.36 (−0.17 to 0.90)	0.12
Mean daily total SOFA score in the sensitivity analysis‡	7.19±3.72	6.78±3.74	0.41 (−0.24 to 1.06)	—
Secondary outcomes				
Death — no./total no. (%)§				
At 28 days	89/258 (34.5)	79/256 (30.9)	3.6 (−4.5 to 11.7)	0.43
At ICU discharge	83/258 (32.2)	76/257 (29.6)	2.6 (−5.4 to 10.6)	0.59
At hospital discharge	97/258 (37.6)	84/256 (32.8)	4.8 (−3.5 to 13.0)	0.30
Median no. of catecholamine-free days (IQR)	22 (0 to 26)	23 (0 to 26)	−1.0 (−4.5 to 1.0)	0.09
Median no. of ventilation-free days (IQR)	16 (0 to 25)	19 (0 to 25)	−3.0 (−9.5 to 1.0)	0.14
Major acute kidney event over period of 28 days — no./total no. (%)	148/258 (57.4)	139/256 (54.3)	3.1 (−5.5 to 11.6)	0.54
Need for new renal-replacement therapy	62/257 (24.1)	62/257 (24.1)	0.0 (−7.4 to 7.4)	>0.99
Sustained renal failure at day 28 or ICU discharge if before 28 days	118/258 (45.7)	108/257 (42.0)	3.7 (−4.9 to 12.3)	0.45
Median duration of renal-replacement therapy (IQR) — days	3.0 (1.0 to 8.0)	5.0 (2.0 to 9.0)	−2.0 (−3.0 to 0.0)	0.24
Median length of ICU stay (IQR) — days				
All patients	7.3 (3.2 to 14.8)	8.3 (3.9 to 13.5)	−1.0 (−2.6 to 0.8)	0.66
Survivors	9.1 (5.0 to 16.1)	9.0 (4.9 to 14.1)	0.2 (−2.0 to 2.7)	0.31
Nonsurvivors	3.2 (1.4 to 8.9)	5.7 (2.2 to 11.7)	−2.6 (−5.7 to −0.8)	0.09
Median length of hospital stay (IQR) — days				
All patients	19.6 (10.1 to 40.4)	22.7 (11.7 to 42.3)	−3.1 (−7.0 to 2.2)	0.24
Survivors	30.1 (16.8 to 48)	27.7 (18 to 52.3)	2.5 (−5.9 to 8.2)	0.81
Nonsurvivors	8.2 (3.4 to 18.6)	11.3 (5.1 to 25.7)	−3.1 (−6.5 to 0.7)	0.25
Safety outcomes				
Any serious adverse event — no. (%)	32 (12.4)	23 (8.9)	3.5 (−2.3 to 9.2)	0.26
Any life-threatening arrhythmia — no. (%)	15 (5.8)	6 (2.3)	3.5 (−0.3 to 7.3)	0.08
Supraventricular tachyarrhythmia	8 (3.1)	1 (0.4)	2.7 (0.1 to 5.3)	0.04
Bradycardia	0	2 (0.8)	−0.8 (−2.2 to 0.7)	0.48
Ventricular fibrillation or tachycardia	7 (2.7)	3 (1.2)	1.5 (−1.2 to 4.3)	0.34
Myocardial infarction or acute coronary syndrome — no. (%)	3 (1.2)	1 (0.4)	0.8 (−1.1 to 2.7)	0.62
Other — no. (%)¶	18 (7.0)	17 (6.6)	0.4 (−4.3 to 5.1)	>0.99

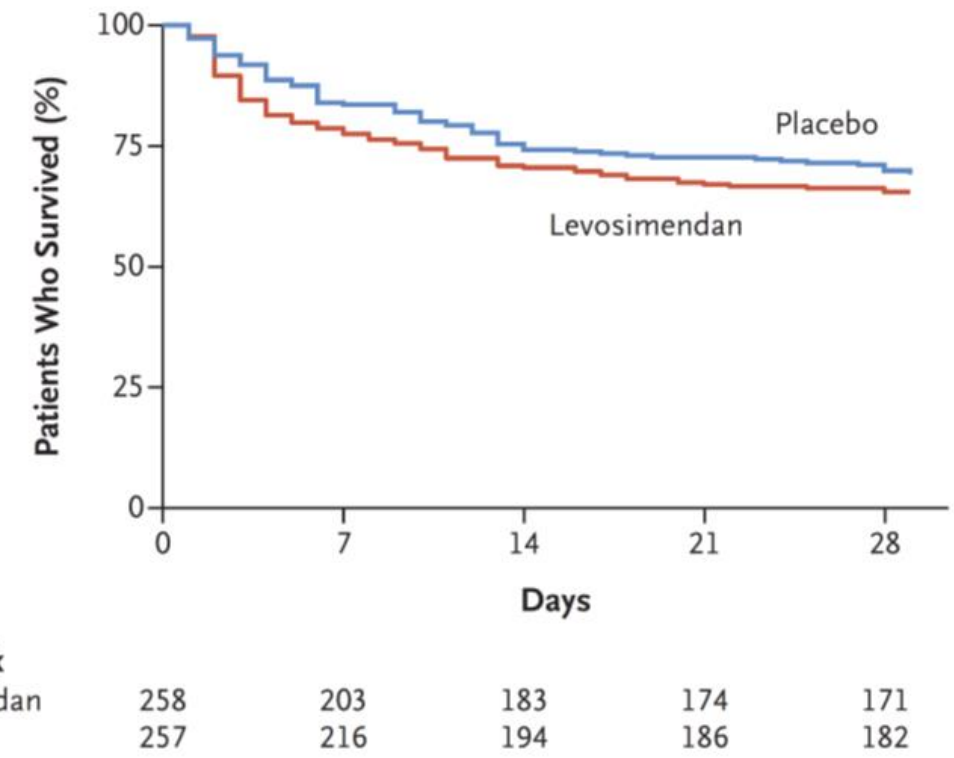


Figure 2. Kaplan–Meier Estimates of the Probability of Survival to Day 28.

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).

Discussion de l’étude

L’augmentation du score SOFA cardio-vasculaire peut s’expliquer par l’hypotension artérielle favorisée par le lévosimendan.

La dobutamine était ajoutée lorsqu’il était retrouvé des critères de bas débit cardiaque.

La dysfonction myocardique était surestimée par rapport aux résultats de l’étude.

La majoration du risque de trouble du rythme supraventriculaire peut expliquer le bénéfice retrouvé dans les études antérieures à l’adjonction de béta-bloquant.

Etudes antérieures de faible puissance

Efficacité du lévosimandan prouvée pour lors d’un faible index cardiaque et une majoration des RVS

- **Points forts**

Etude randomisée, multicentrique

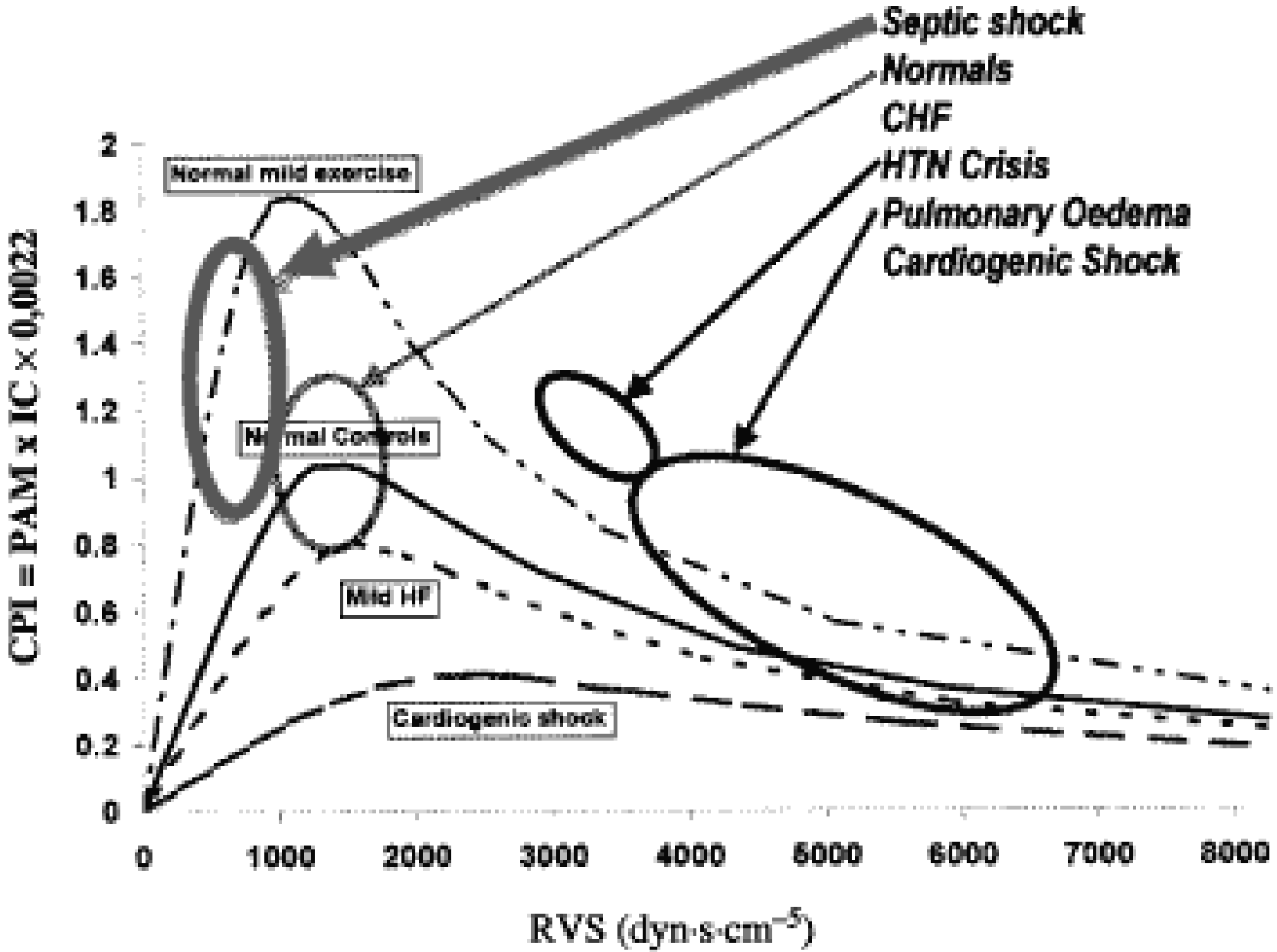
Double aveugle vrai et maintenu

Peu de perdus de vue, le nombre de patients a été respecté

- **Points faibles**

Pas de monitorage hémodynamique systématique

Le choix arbitraire du score SOFA moyen comme objectif primaire est discutable.


Septic shock
Normals
CHF
HTN Crisis
Pulmonary Oedema
Cardiogenic Shock
Normal mild exercise
Normal Controls
Mild HF
Cardiogenic shock
CPI = PAM x IC x 0,0022
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
0
1000
2000
3000
4000
5000
6000
7000
8000
RVS (dyn·s·cm^{-5})

Conclusion

L’utilisation de lévosimendan dans le choc septique ne permet pas de diminuer la mortalité ni la dysfonction d’organe, elle nécessite une majoration des doses de noradrénaline, enfin elle favorise les troubles du rythme supraventriculaire.

La dobutamine reste l’agent inotrope de première intention dans la myocardite septique.

Le levosimendan doit être réservé aux défaillances myocardiques avérées notamment lorsqu’il y a des critères d’HTP post capillaire ou d’élévation des RVS, en cas d’echec de réponse à la dobutamine, ou chez les patients décompensant sous traitement de fond par béta-bloquant.

Merci pour votre attention