Insuffisance cardiaque aiguë post-opératoire

Alexandre Mebazaa

Hôpital Lariboisière,

University Paris 7, U942 Inserm; Paris, France

En résumé
ICA post-opératoire

- Se passe en SSPI ou en étage plus tard
- Trouver le mécanisme de la décompensation cardiaque
  - Dysfonction diastolique du VG
  - Dysfonction systolo-diastolique du VG
  - Dysfonction du VD
- ECG, radio du thorax
- Biomarqueurs : BNP(ou NT-Pro-BNP)/troponine
- Echocardiographie
- éviter
  - variation de PA et survenue ischémie
  - Pour le VD : altération de la gazométrie, remplissage excessif
- Favoriser les vasodilatateurs, éviter les catécholamines
- ET SURTOUT reprendre vite le(s) traitements au long cours

Il existe 3 types d'IC chronique :

- IC chronique avec dysfonction VG systolique et diastolique : bas débit circulatoire
- IC chronique avec dysfonction diastolique isolée : OAP
- IC droite avec ou sans HTAP

Oedème pulmonaire aiguë

Dyspnée +
PAS > 140 mmHg

TOUJOURS

ProblèmeVasculaire Aiguë +
Dysfonction Diastolique VG

Variable	During Acute Pulmonary Edema	After Treatment
	mean ±SD	
Blood pressure (mm Hg)		
Systolic	200±26	139±17*
Diastolic	100±25	64±15*
Heart rate (beats/min)	83±14	72±12*
Mitral flow velocity (cm/sec)		
E wave	98±33	98±28
A wave	88±33	78±26*
E wave:A wave	1.31±0.80	1.51±0.97*
E-wave deceleration time (msec)	174±62	194±62*
Isovolumic relaxation time (msec)	78±19	75±25
Left ventricular volume (ml)		
End diastolic	109±43	117±50
End systolic	58±32	61±37
Left ventricular ejection fraction	0.50±0.15	0.50±0.13
Left ventricular wall thickness (mm)		
Posterior	12.8±2.9	12.8±3.1
Septal	12.5±3.7	12.9±3.6
Left ventricular dimension (mm)		
End diastolic	49.7±9.5	49.4±9.8
End systolic	38.3±10.1	38.3±10.7

*P<0.05 for the comparison with the value during the acute episode.

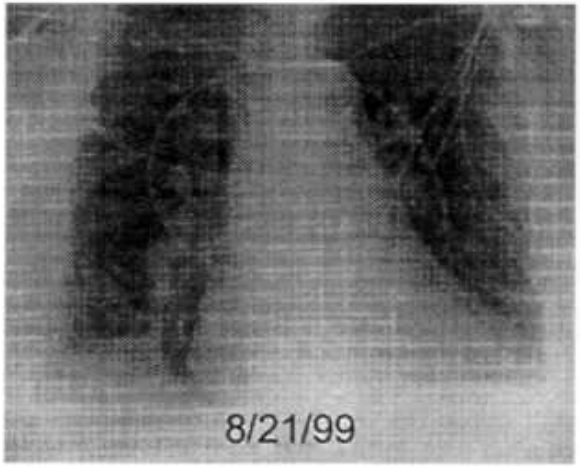
SK Ghandi, NEJM 2001, 344: 17-22

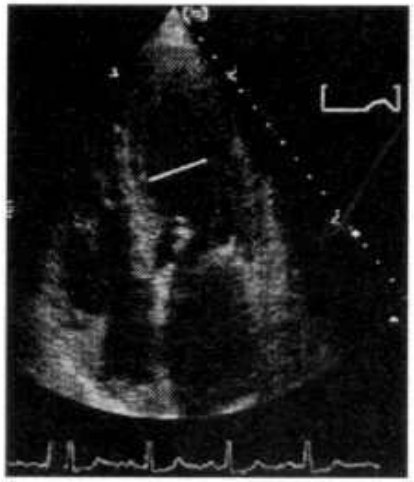
During Acute Pulmonary Edema

Blood pressure, 240/144 mm Hg

After Treatment

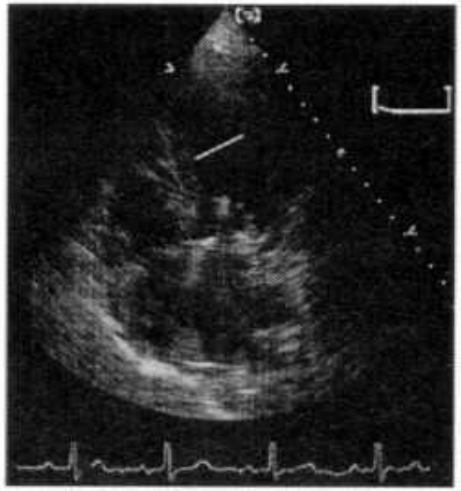
Blood pressure, 149/75 mm Hg



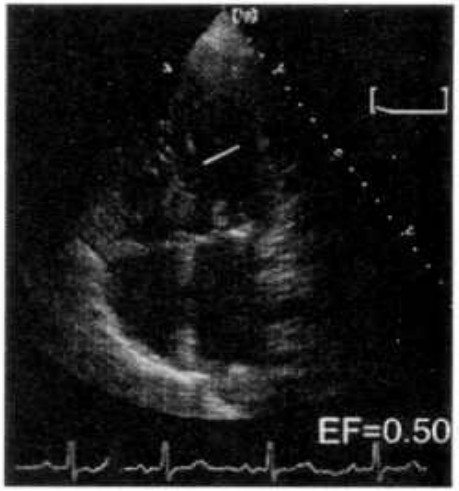


End Diastole

End Systole



End Diastole



End Systole

SK Ghandi, NEJM 2001, 344: 17-22

Pathogénie de l'OAP

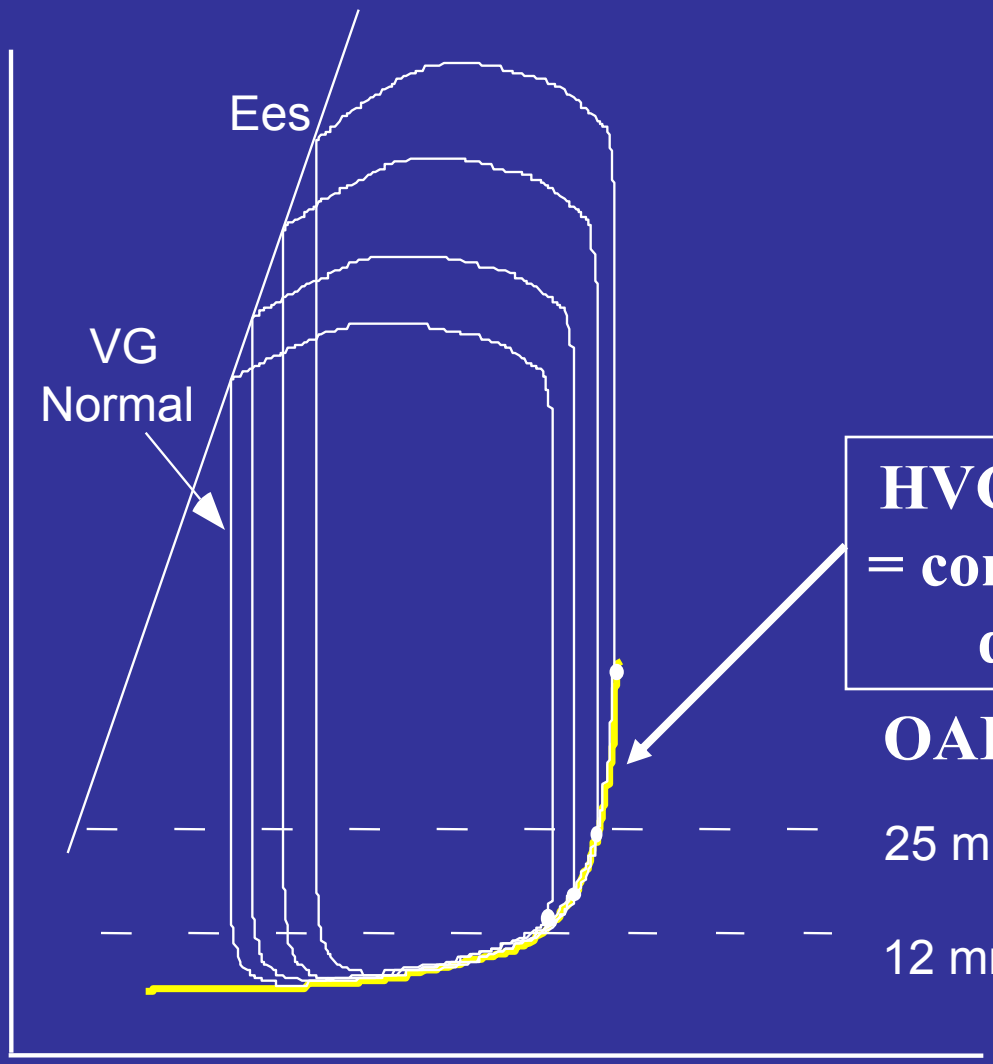
L'oedème aiguë du poumon :

- Est un signe de congestion pulmonaire = élévation de la PAPo = POG = PTDVG

- Est un signe de dysfonction diastolique et non systolique

SK Ghandi, NEJM 2001, 344: 17-22
Pirracchio et al. Br. J. Anaesth.2007; 98: 707-721

Œdème aiguë du poumon sans état de choc : crise hypertensive, FEVG normale!

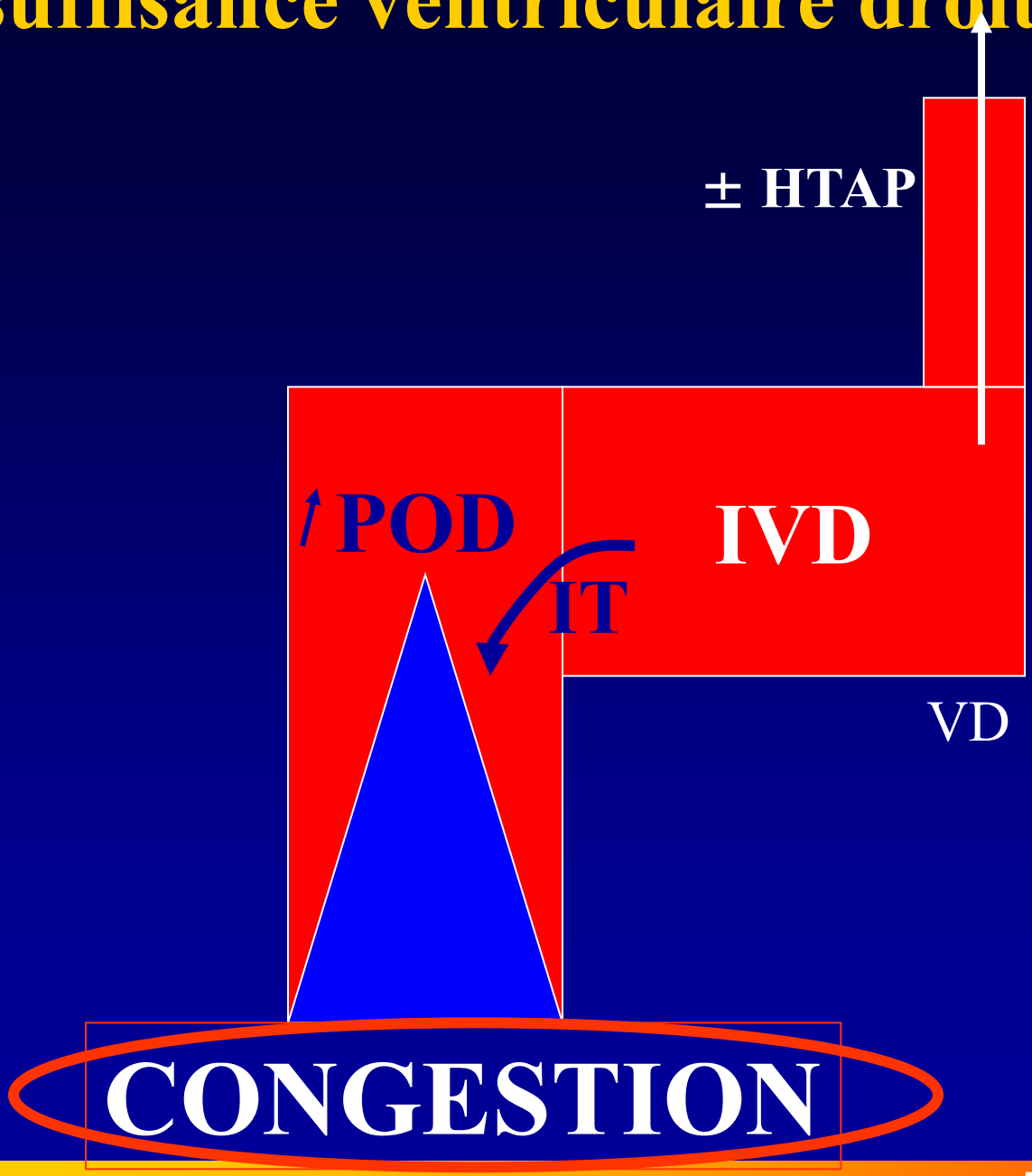


HVG + ischémie = compliance VG diminuée

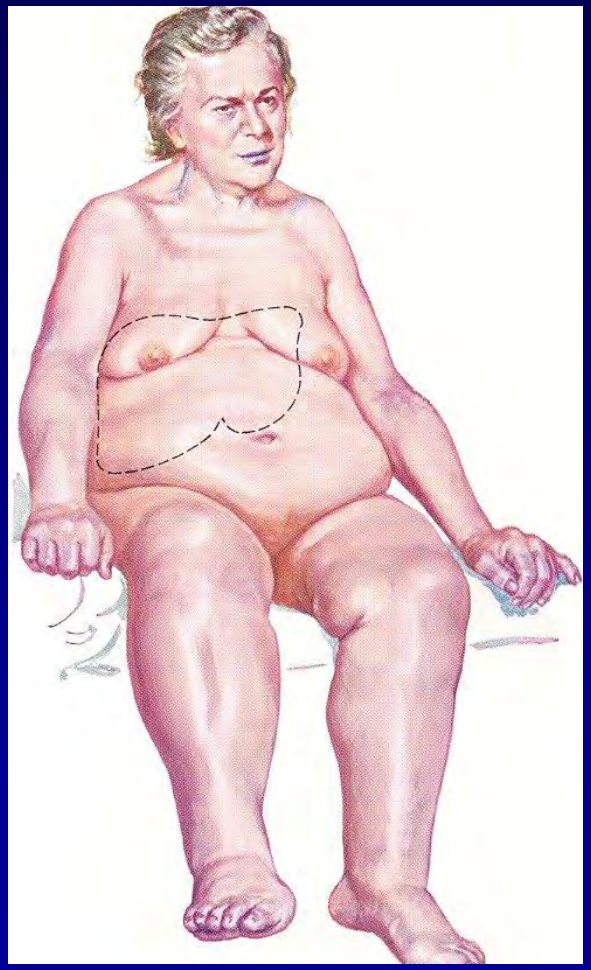
OAP !

Pirracchio et al. *Br. J. Anaesth.* 2007; 98: 707-721

Insuffisance ventriculaire droite



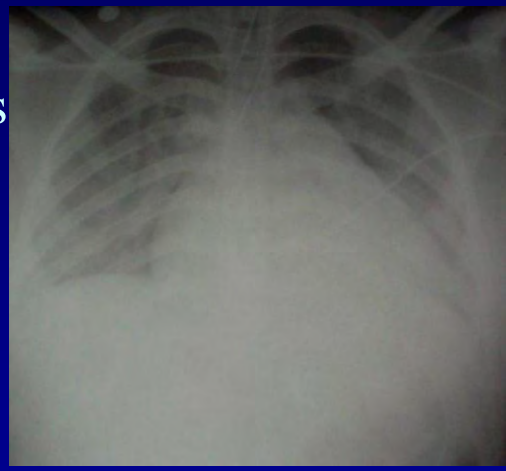
Décompensation d'une ICC



Dyspnée
+ PAS <140 mmHg

+

- Gêne respiratoire depuis plusieurs jours
- Elévation du poids
- OMI



+ Maladie systémique !

- Insuffisance rénale
- Anémie
- Hypoalbuminémie

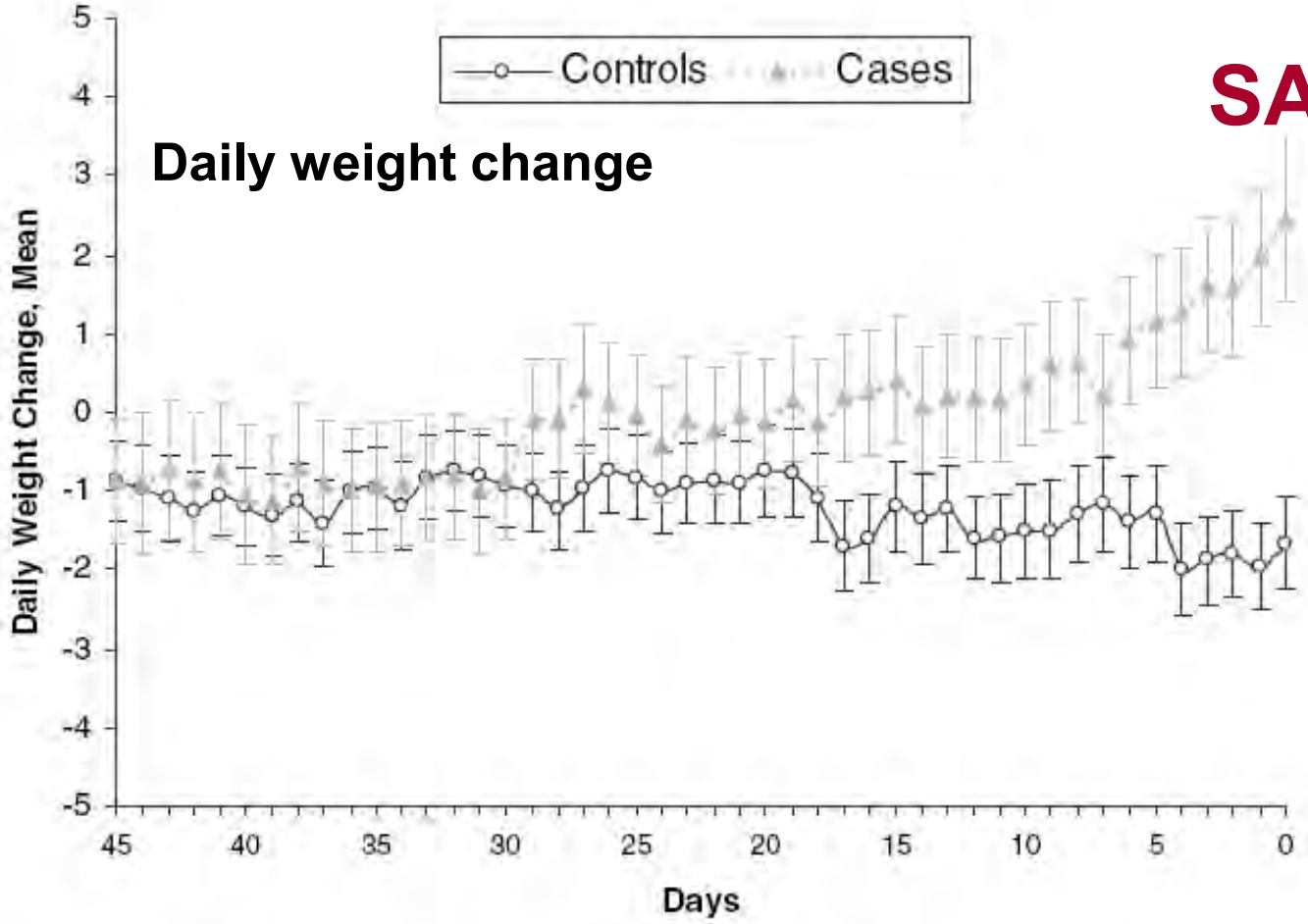
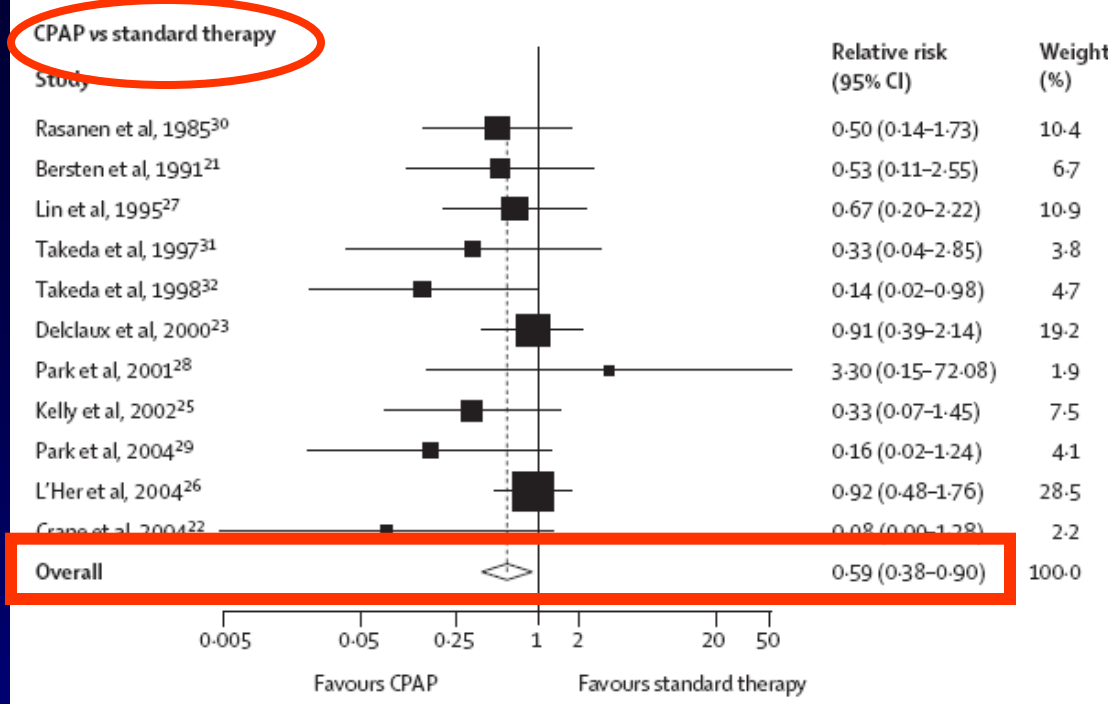
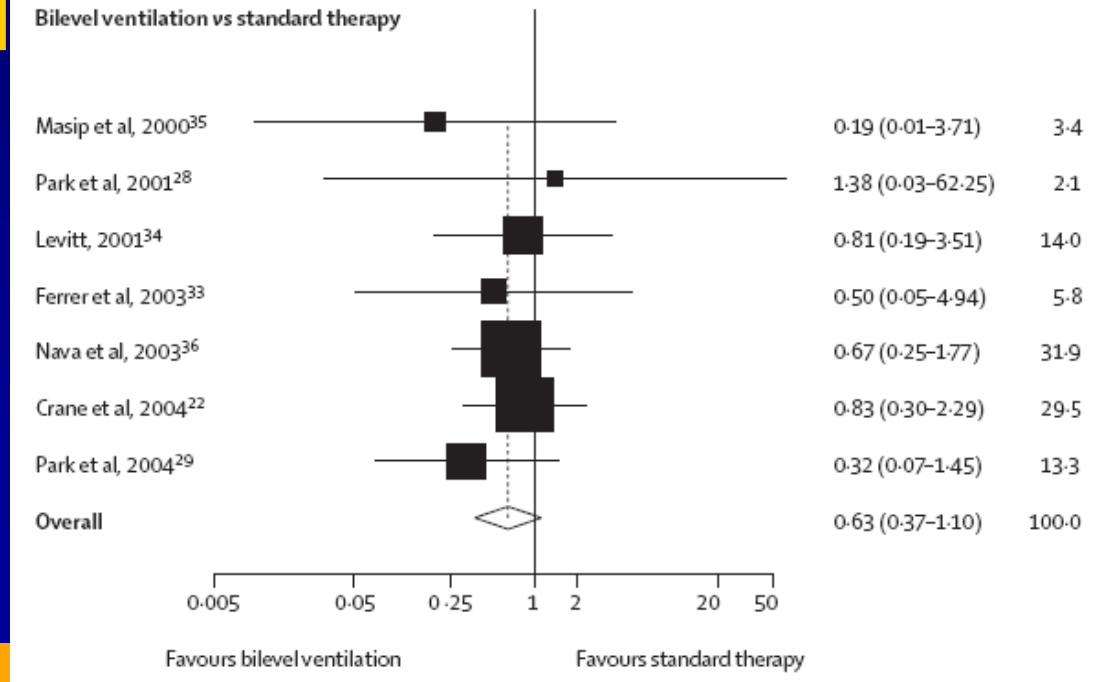


Figure 1. Daily weight change before heart failure hospitalization: cases vs controls. n=268. "Days" on the x-axis denotes days before hospital admission in case patients. The difference in daily weight changes between case and control patients within 30 days before (case) hospitalization was statistically significant ($P<0.001$) on the basis of a generalized linear model with daily weight change as the dependent variable.

Chaudhry E al. Circulation 2007; 116: 1549-15

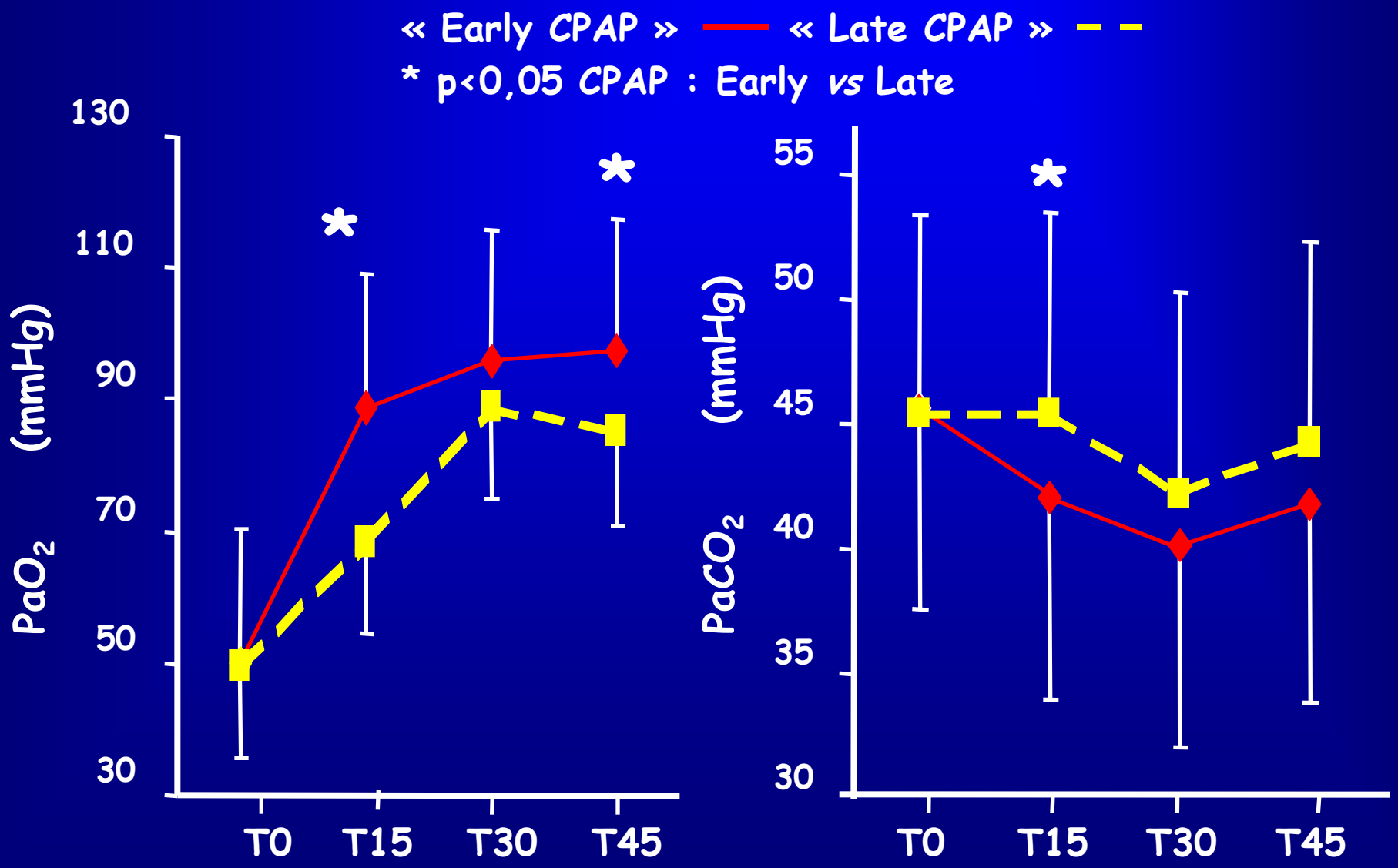
CPAP mostly reduces the need of mechanical ventilation





Peter JV, Lancet 2006;367:1155
Masip J et al. JAMA. 2005;294:3124
Winck JC et al. Crit Care. 2006;10:R69

Blood Gases Evolution



Plaisance et al EHJ 2007

Therapy and strength of evidence

Therapy	Level of recomm	Level of evidence	Comment
Diuretics	I	B	If fluid retention To reduce dyspnoea
Vasodilators	I	B (NTG) C (NTP)	Pulmon. congestion Hypertension To reduce dyspnoea
Inotropics agents	II a/b	B	Hypotension, Hypoperfusion

ESC/ESICM guidelines on AHF; European Heart Journal 2005

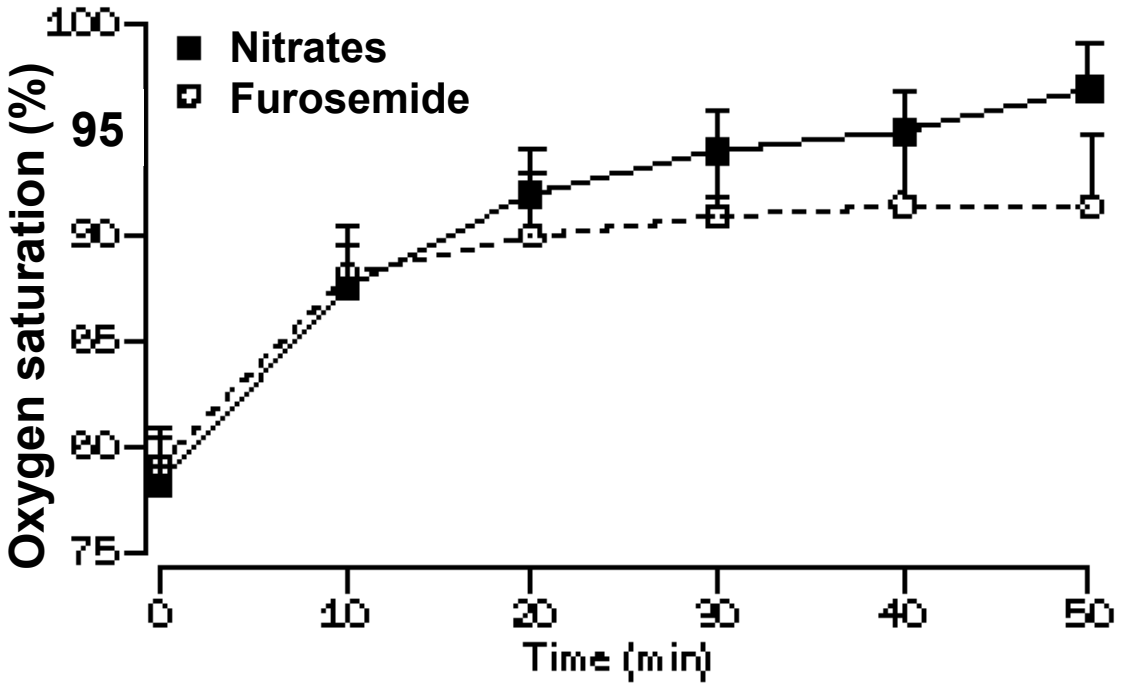


Figure 2: **Change in oxygen saturation during treatment in group A (predominant isosorbide dinitrate) and group B (predominant furosemide)**

Primary outcome	Group A (n=52)	Group B (n=52)	p
Died	1 (2%)	3 (6%)	0·61
Required mechanical ventilation	7 (13%)	21 (40%)	0·0041
Myocardial infarction	9 (17%)	19 (37%)	0·047
Any adverse event	13 (25%)	24 (46%)	0·041

Cotter et al. Lancet 1998; 351:389-393

Intensive Care Med (2011) 37:619–626
DOI 10.1007/s00134-010-2113-0

ORIGINAL

F. Follath
M. B. Yilmaz
J. F. Delgado
J. T. Parissis
R. Porcher
E. Gayat
Nigel Burrows
A. Mclean
F. Vilas-Boas
A. Mebazaa

Clinical presentation, management and outcomes in the Acute Heart Failure Global Survey of Standard Treatment (ALARM-HF)

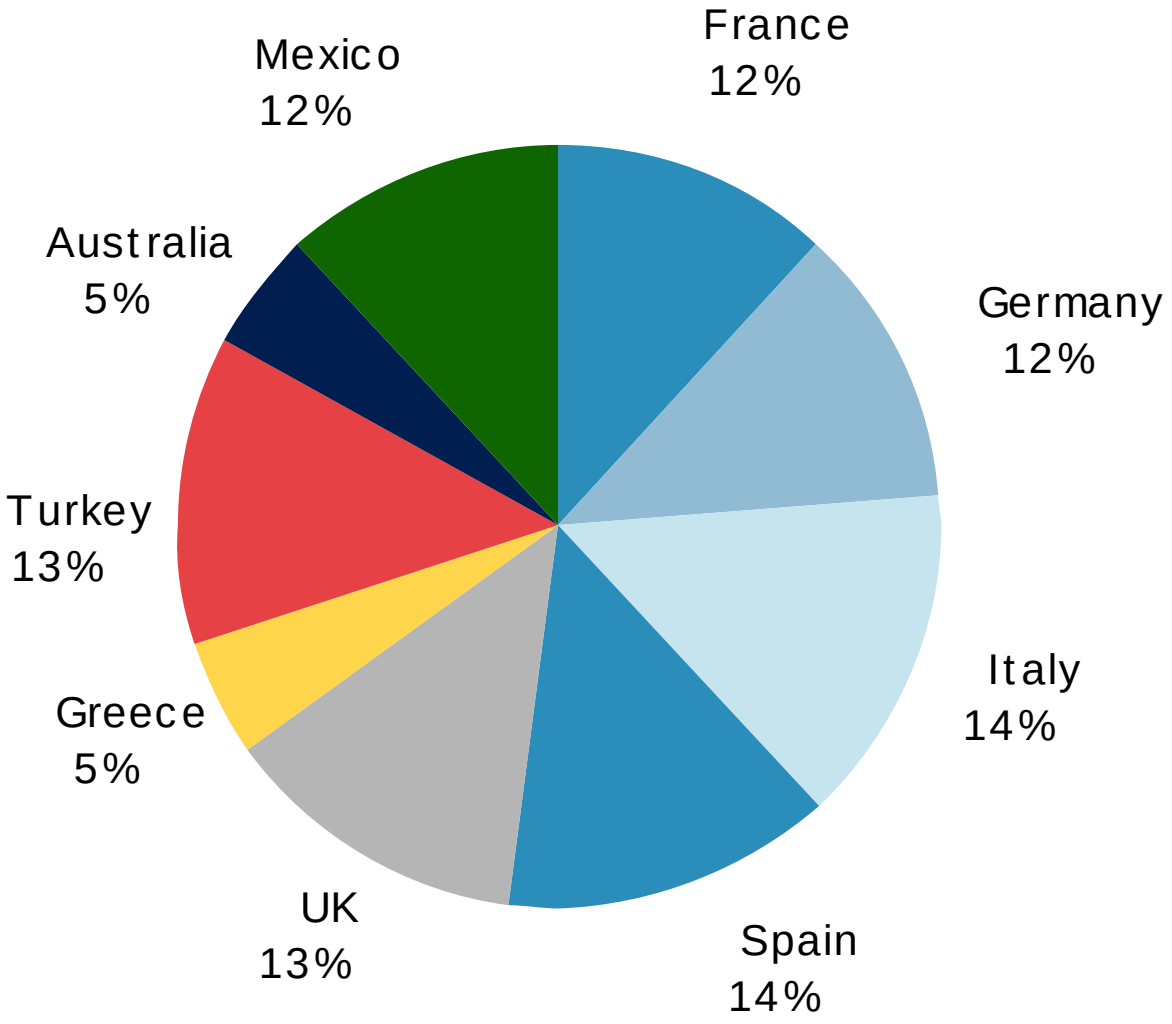
Intensive Care Med (2011) 37:290–301
DOI 10.1007/s00134-010-2073-4

ORIGINAL

Alexandre Mebazaa
John Parissis
Raphael Porcher
Etienne Gayat
Maria Nikolaou
Fabio Vilas Boas
J. F. Delgado
Ferenc Follath

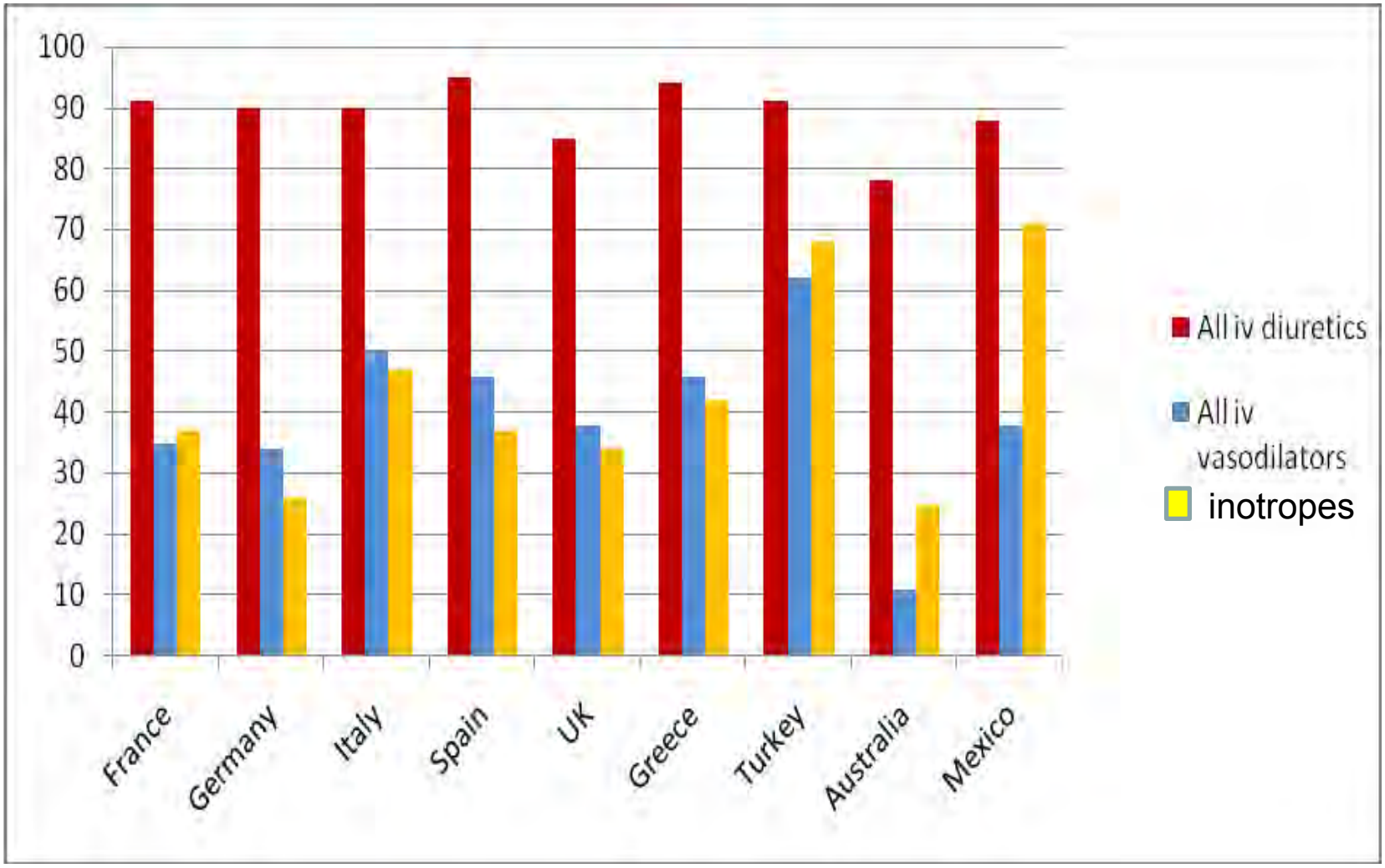
Short-term survival by treatment among patients hospitalized with acute heart failure: the global ALARM-HF registry using propensity scoring methods

ALARM-HF provides a unique view of AHF management across a wide variety of countries



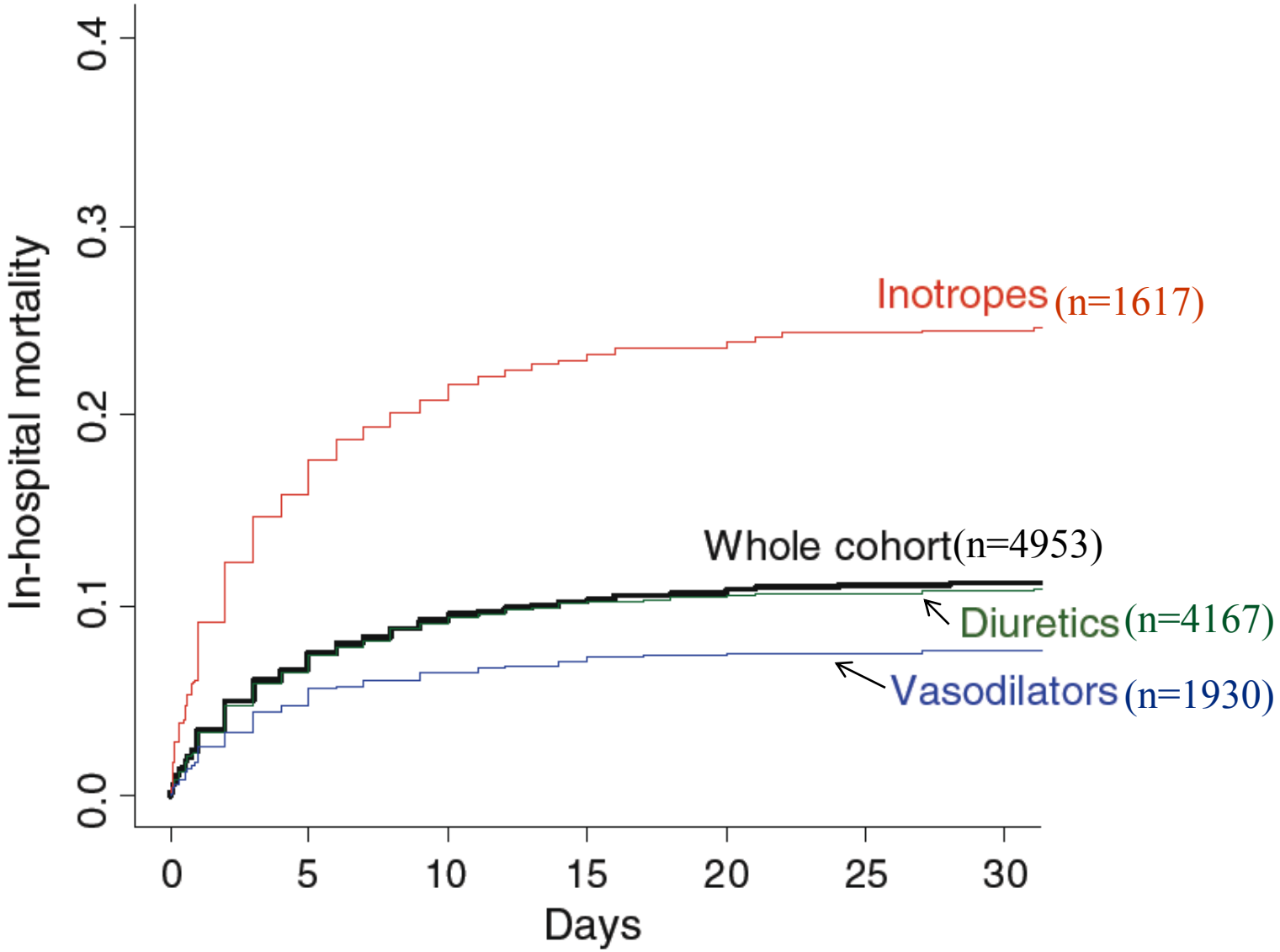
ALARM-HF 9 Countries (4,953 patients)

ALARM-HF: IV treatment at admission



Mebazaa et al Intensive Care Medicine 2011

Effect of IV drugs given during the first 48 hours in AHF patients on in-hospital mortality



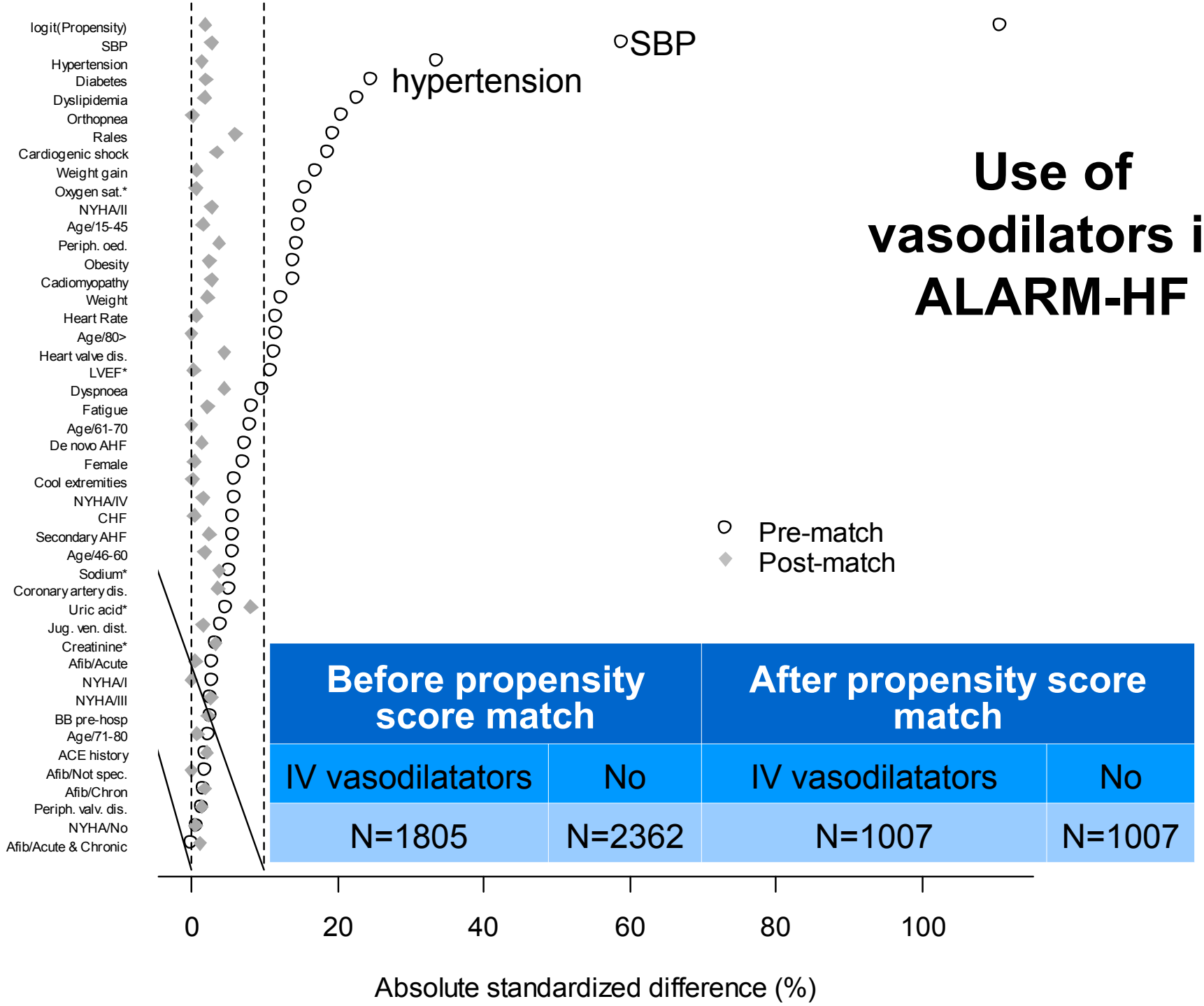
Intensive Care Med (2010) 36:1993–2003
DOI 10.1007/s00134-010-1991-5

REVIEW

Etienne Gayat
Romain Pirracchio
Matthieu Resche-Rigon
Alexandre Mebazaa
Jean-Yves Mary
Raphaël Porcher

Propensity scores in intensive care and anaesthesiology literature: a systematic review

Use of vasodilators in ALARM-HF



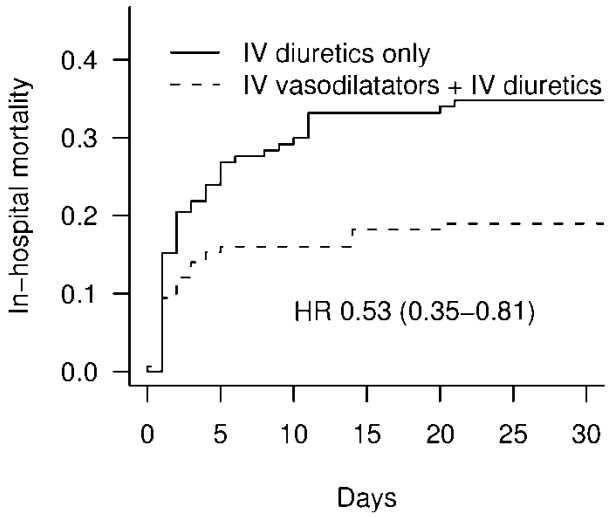
Before propensity score match		After propensity score match	
IV vasodilatators	No	IV vasodilatators	No
N=1805	N=2362	N=1007	N=1007

Results

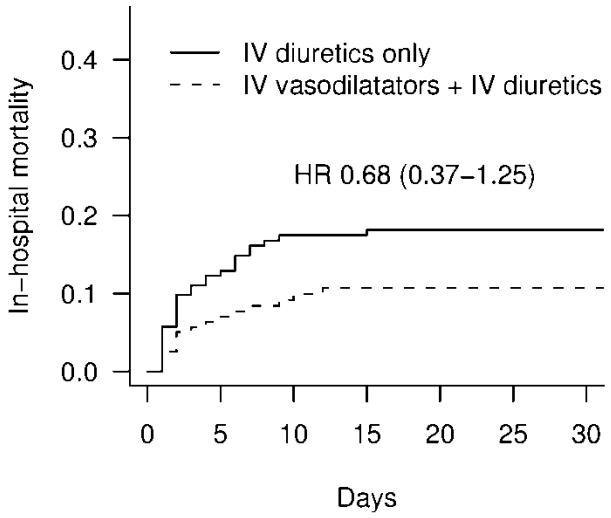
- IV diuretics and IV vasodilators were started at a median of 0.5 [0.0 – 1.0] hour and 0.5 [0.0 – 2] hour respectively after admission.
- IV vasodilators were quasi-exclusively nitrates: nitroglycerine in 76 % and isosorbite dinitrate 19 %
- In-hospital mortality:

- *Before matching* **7.6** vs 14.2 % with and without vasoD
- *After matching* **7.8** versus 11 % with and without vasoD

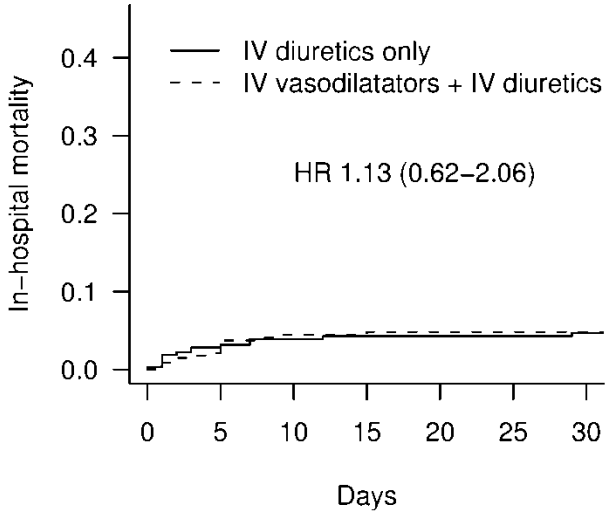
SBP < 100 mmHg (n=318)



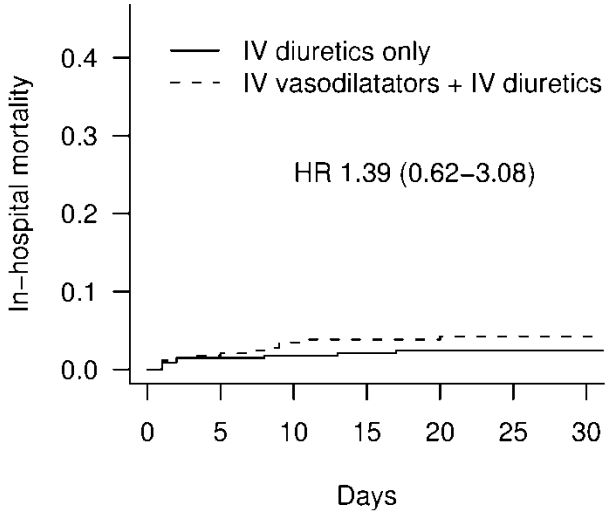
SBP 100-119 mmHg (n=334)



SBP 120-159 mmHg (n=618)



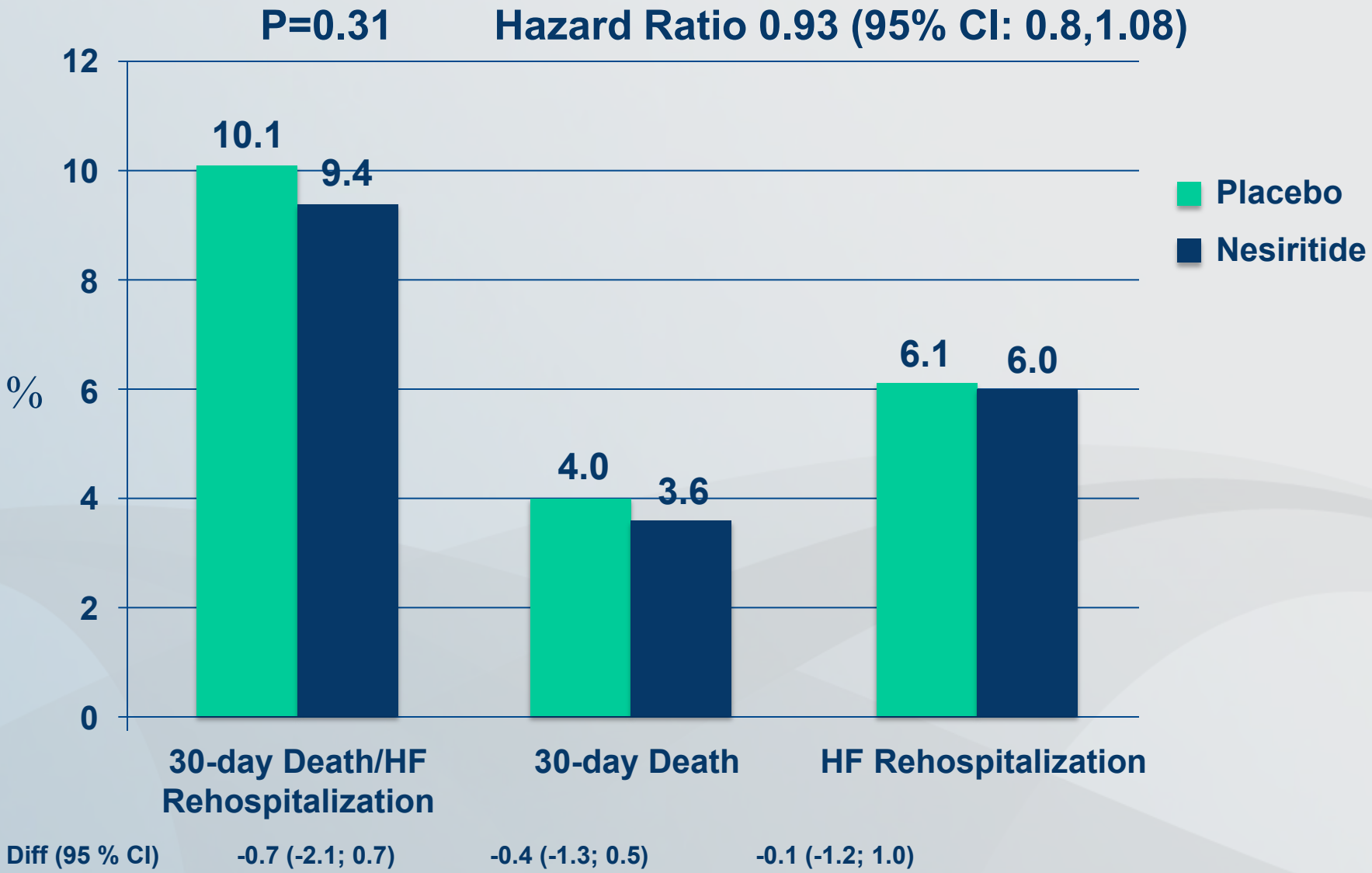
SBP > 160 mmHg (n=694)



Mebazaa et al Intensive Care Medicine 2011

Co-Primary outcome: 30-day all-cause mortality or HF rehospitalization (n=6836)

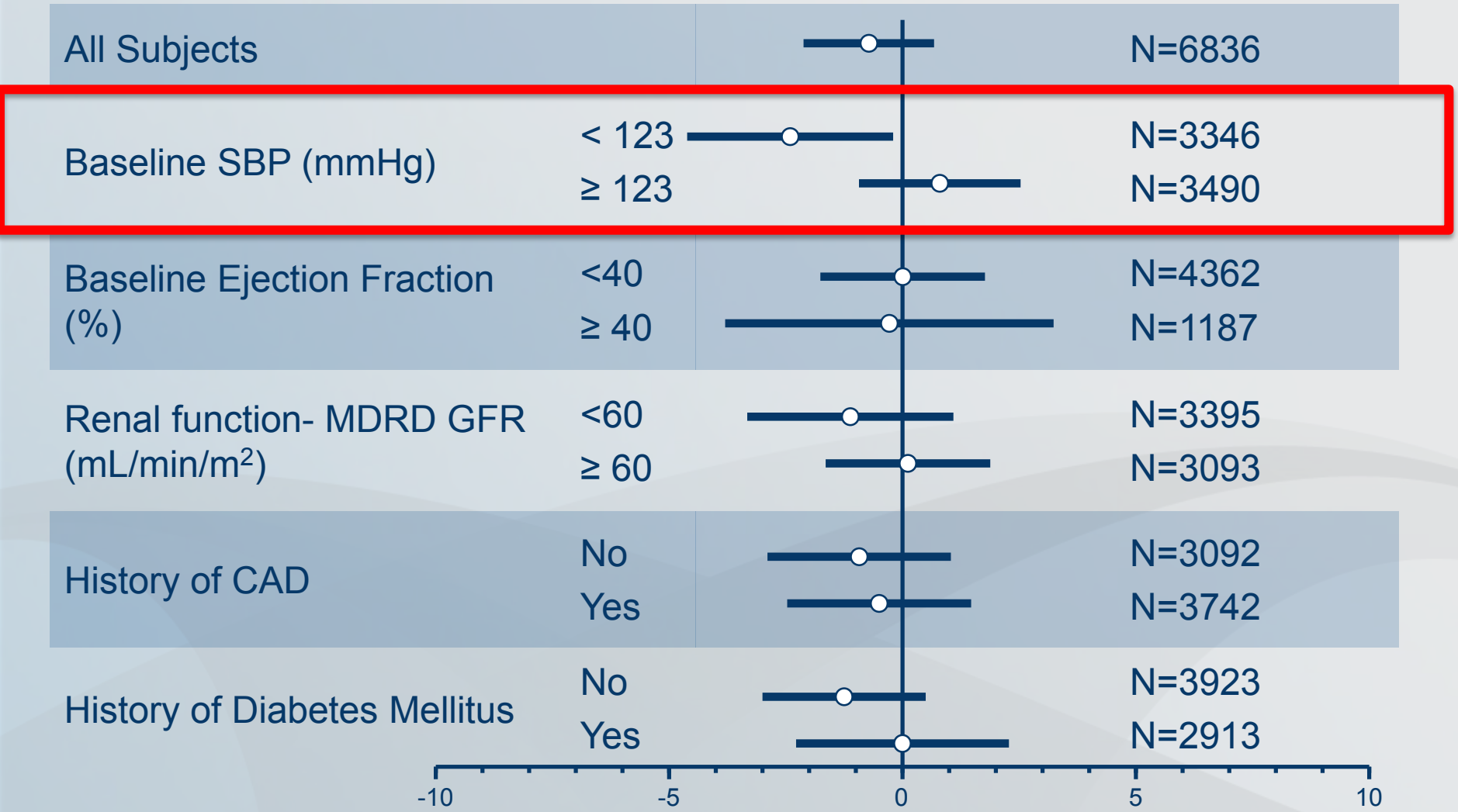
ASCEND-HF
Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure



Hernandez AHA 2010

30 day death/HF readmission subgroups

ASCEND-HF
Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure



Risk Difference <0: Favors Nesiritide;
Risk Difference >0: Favors Placebo

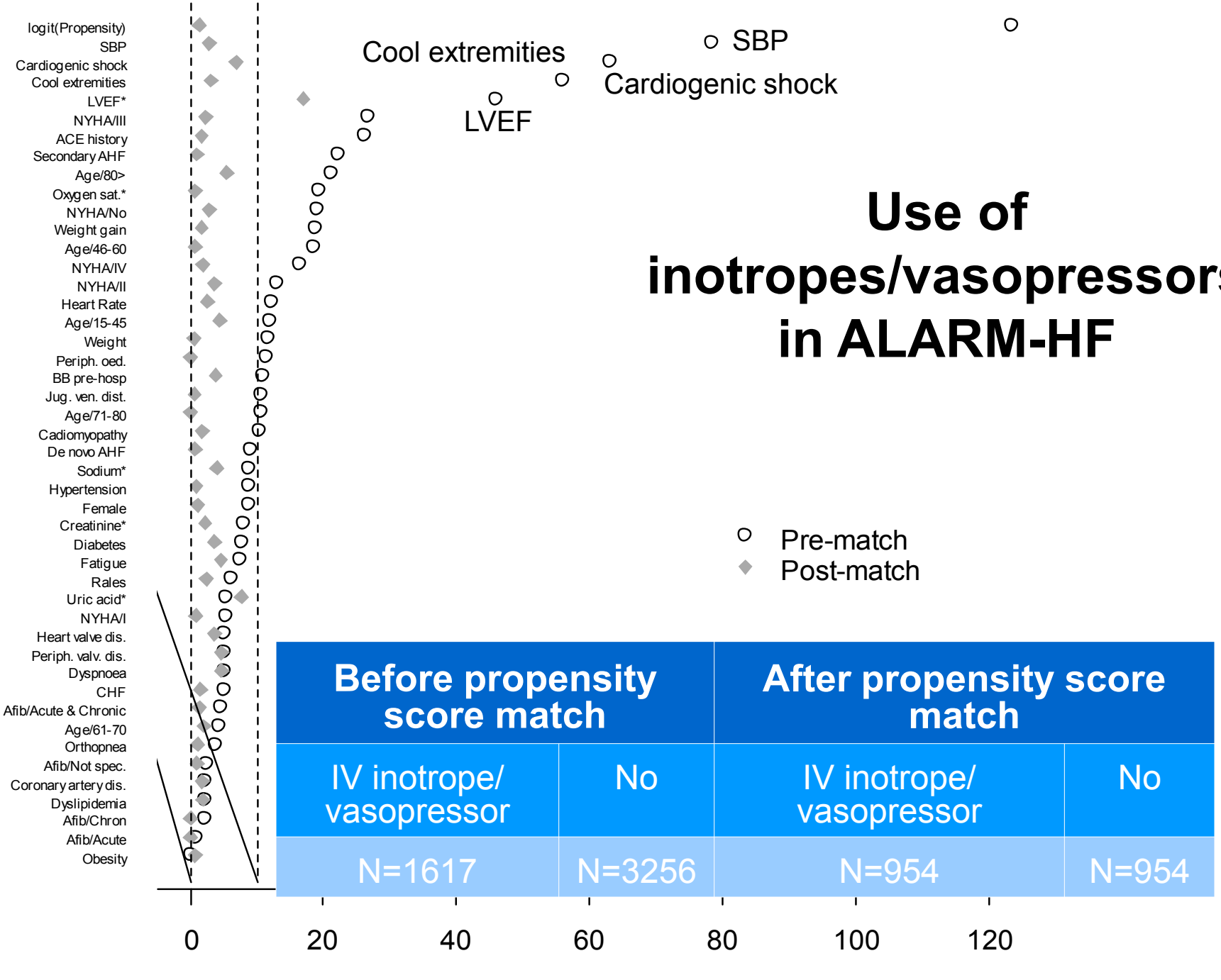
Hernandez AHA 2010

Catecholamine treatment for shock—equally good or bad?

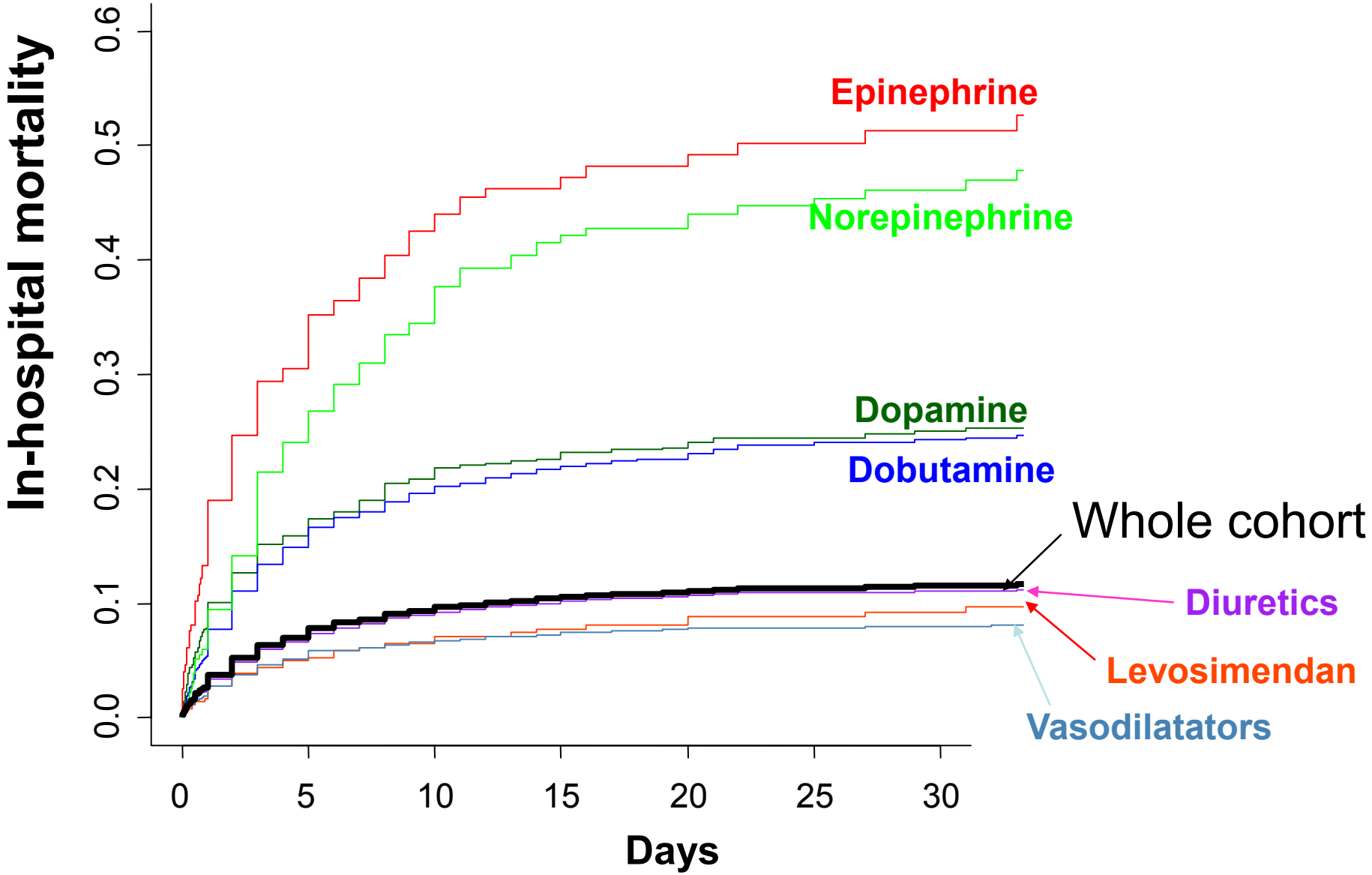
Mervyn Singer
Bloomsbury Institute of Intensive Care Medicine, Wolfson Institute for Biomedical Research and Department of Medicine, University College London, London WC1E 6BT, UK
m.singer@ucl.ac.uk

www.thelancet.com Vol 370 August 25, 2007

Use of inotropes/vasopressors in ALARM-HF

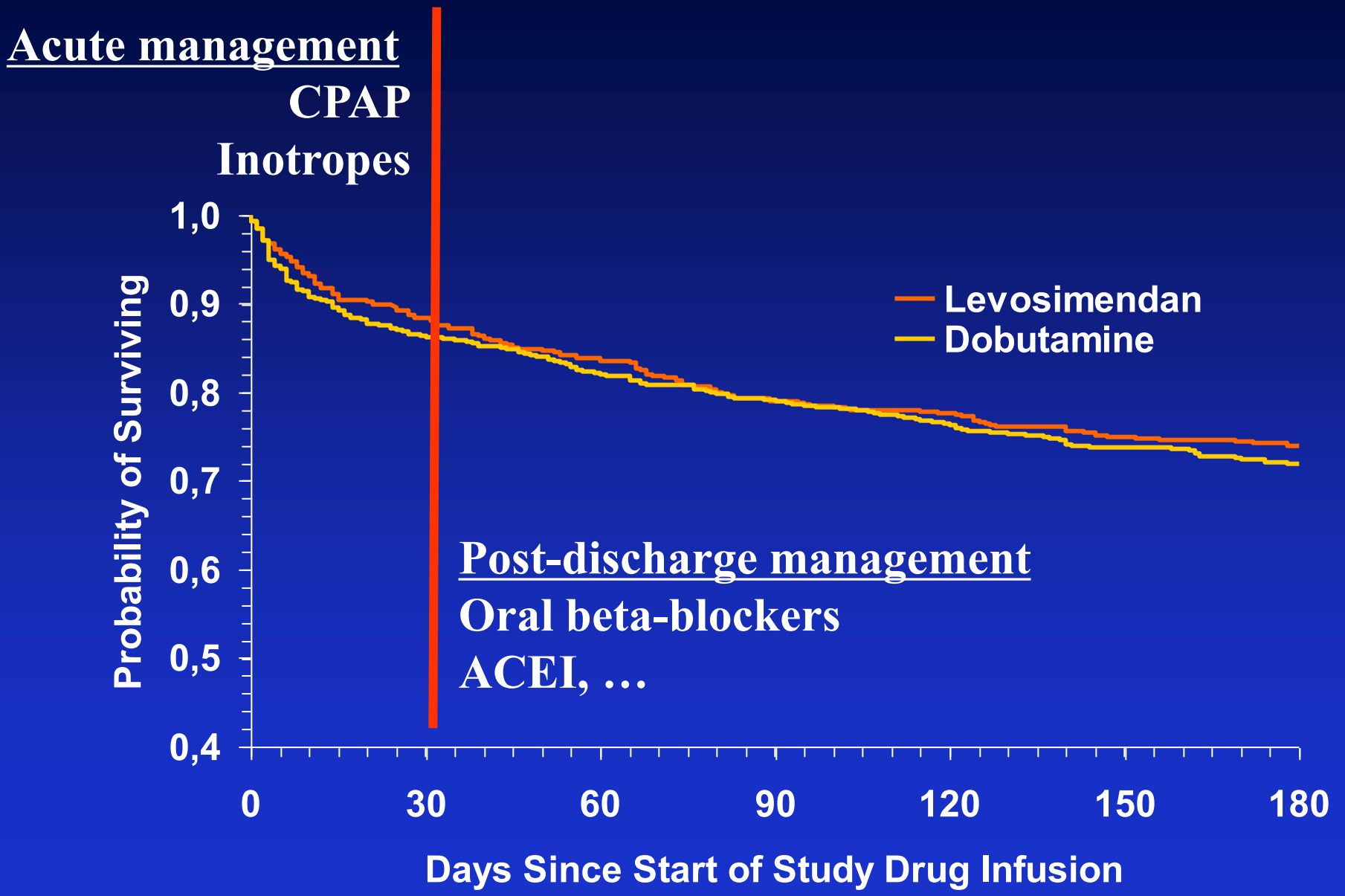


Before propensity score match		After propensity score match	
IV inotrope/ vasopressor	No	IV inotrope/ vasopressor	No
N=1617	N=3256	N=954	N=954


In-hospital mortality
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0
5
10
15
20
25
30
Days
Epinephrine
Norepinephrine
Dopamine
Dobutamine
Whole cohort
Diuretics
Levosimendan
Vasodilatators

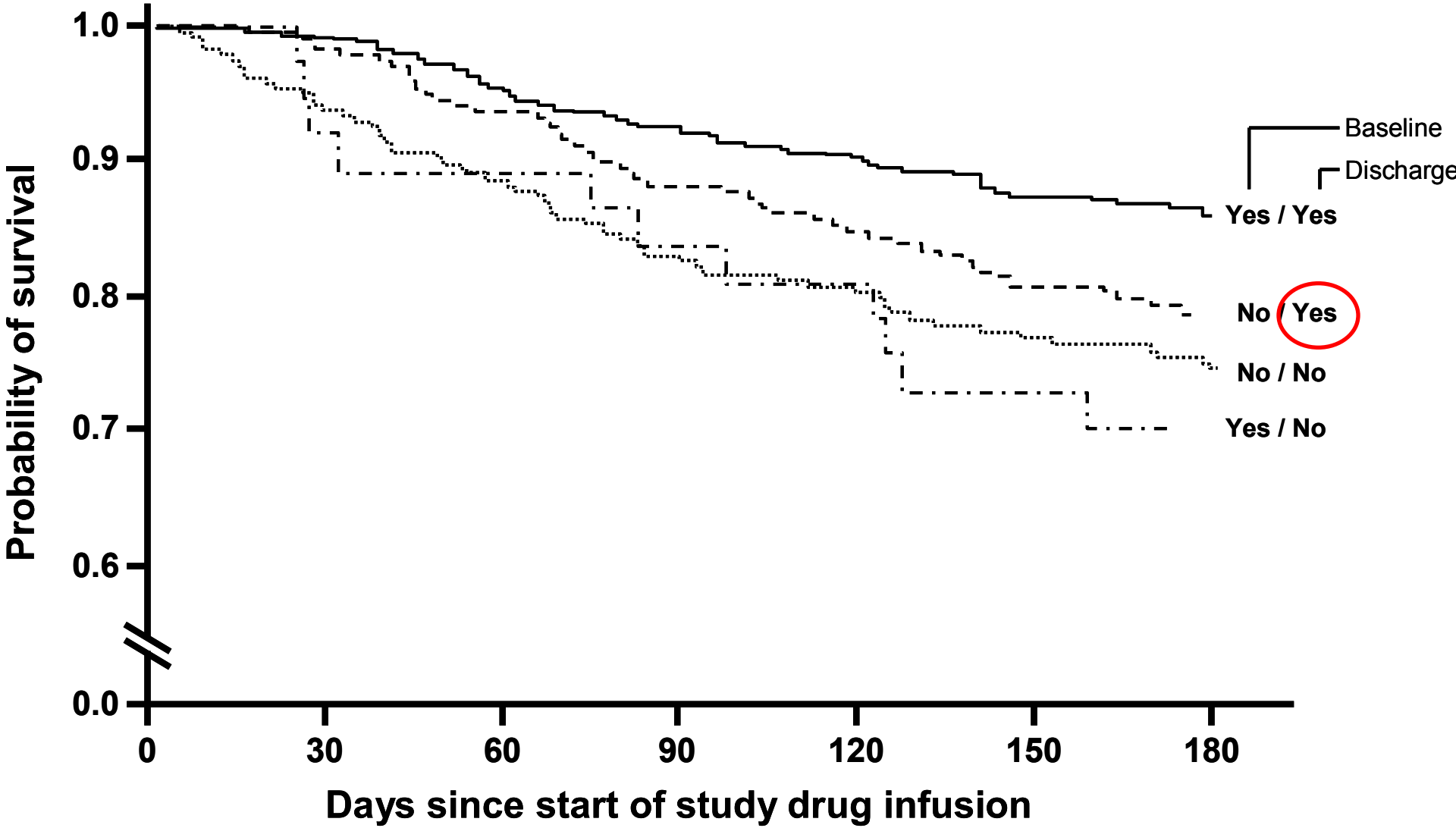
Post-discharge treatement after discharge from Acute Heart Failure

Determinants of 180-day All-Cause Mortality



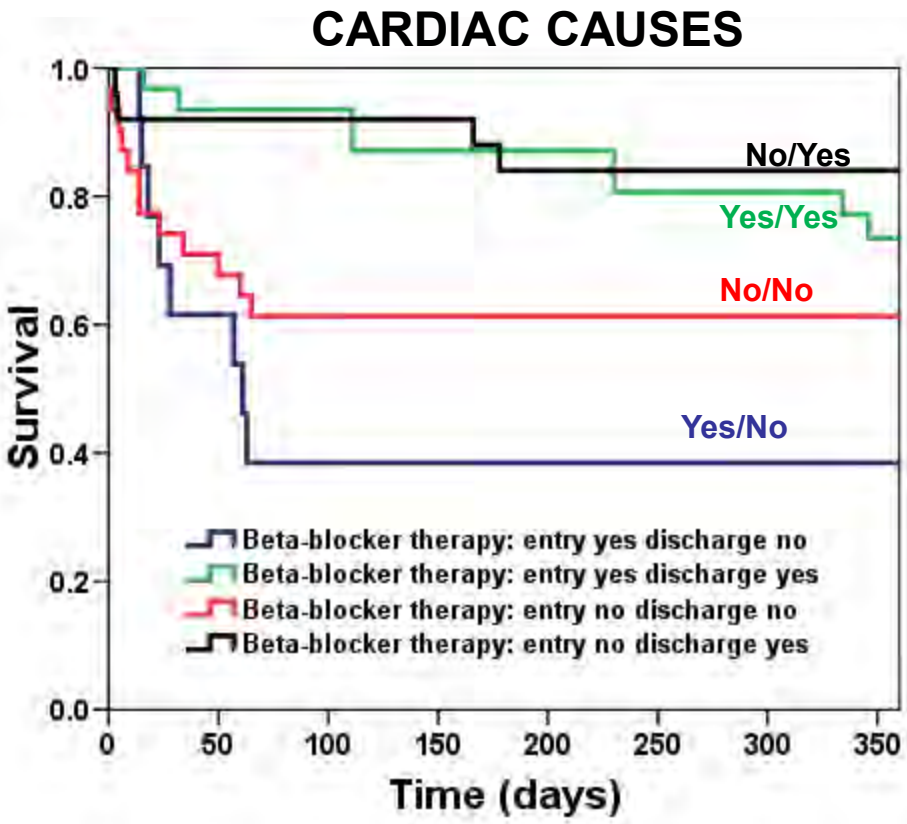
Mebazaa et al. JAMA 2007;297,1883-1891

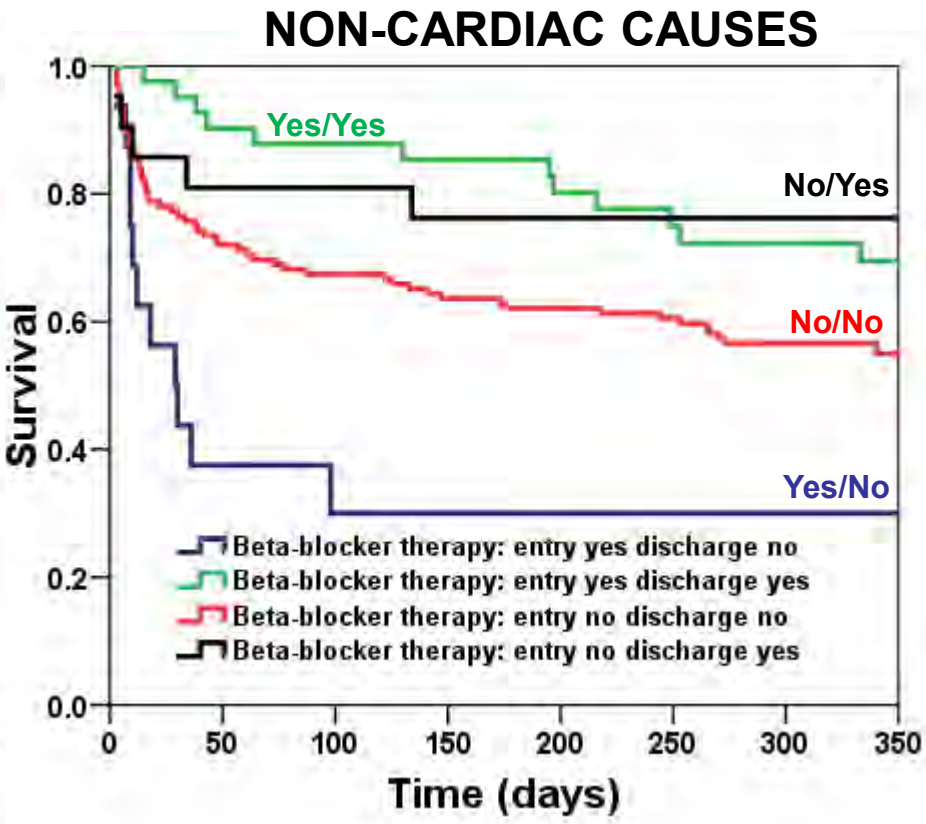
All-Cause Mortality by Beta-Blocker Use at Baseline and Discharge



M. Boehm et al. Crit Care Medicine 2011

Effects of beta-blockers on patients admitted for acute respiratory failure





Noveanu et al Crit Care 2011

Practical recommendations for prehospital and early in-hospital management of patients presenting with acute heart failure syndromes

Alexandre Mebazaa, MD, PhD; Mihai Gheorghiade, MD, FACC; Ileana L. Piña, MD, FACC; Veli-Pekka Harjola, MD; Steven M. Hollenberg, MD; Ferenc Follath, MD; Andrew Rhodes, MD; Patrick Plaisance, MD; Edmond Roland, MD; Markku Nieminen, MD; Michel Komajda, MD; Alexander Parkhomenko, MD; Josep Masip, MD; Faiez Zannad, MD, PhD; Gerasimos Filippatos, MD

Mebazaa et al. Critical Care Medicine, 2008, Suppl 36:129-139

Management at admission

Tailored therapy

- **CS1 (SBP > 140 mmHg):** NIV and Nitrates; diuretics are rarely indicated unless volume overload
- **CS2 (SBP 100-140 mmHg):** NIV and Nitrates; diuretics if systemic chronic fluid retention
- **CS3 (SBP < 100 mmHg):** Volume loading with initial fluid challenge if no overt fluid retention; inotrope; PAC if no improvement; if BP fails to improve above 100 mmHg and hypoperfusion persits, then consider vasoconstrictors
- **CS4 (ACS):** NIV; Nitrates; Cardiac catheterization lab, follow guideline recommended management for ACS (aspirin, heparin, reperfusion therapy); IABP
- **CS5 (RVF):** Avoid volume loading; diuretics if SBP >90 mmHg and systemic chronic fluid retention; inotropes if SBP <90 mmHg; If SBP fails to improve above 100 mmHg, then begin vasoconstrictors

Initial 90-120 minutes

Next 6 – 12 hours

•Additional diagnostic studies
•Transfer to tertiary care center

•Transfer to tertiary care center

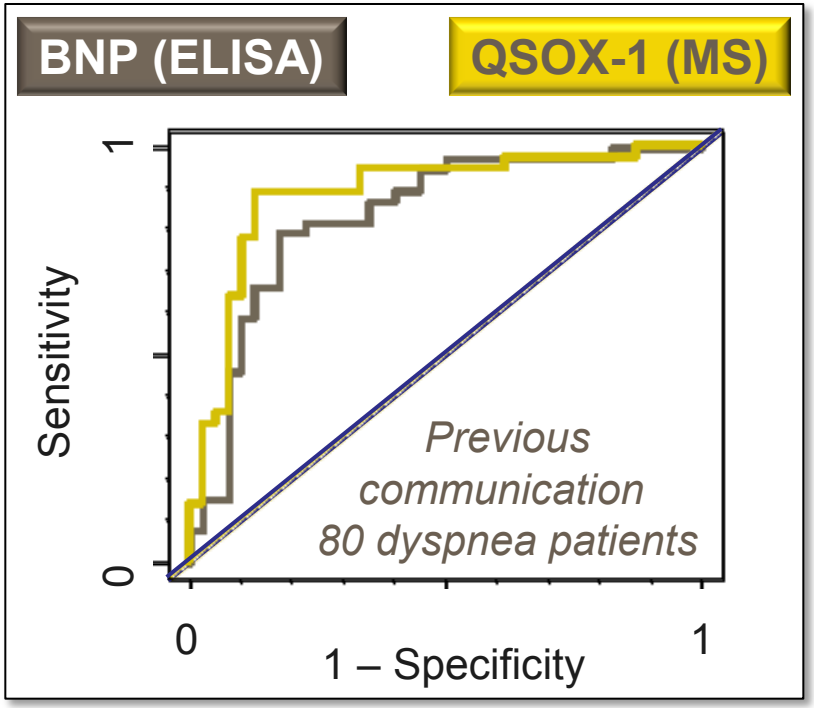
Mebazaa et al. Critical Care Medicine, 2008, Suppl 36:129-139

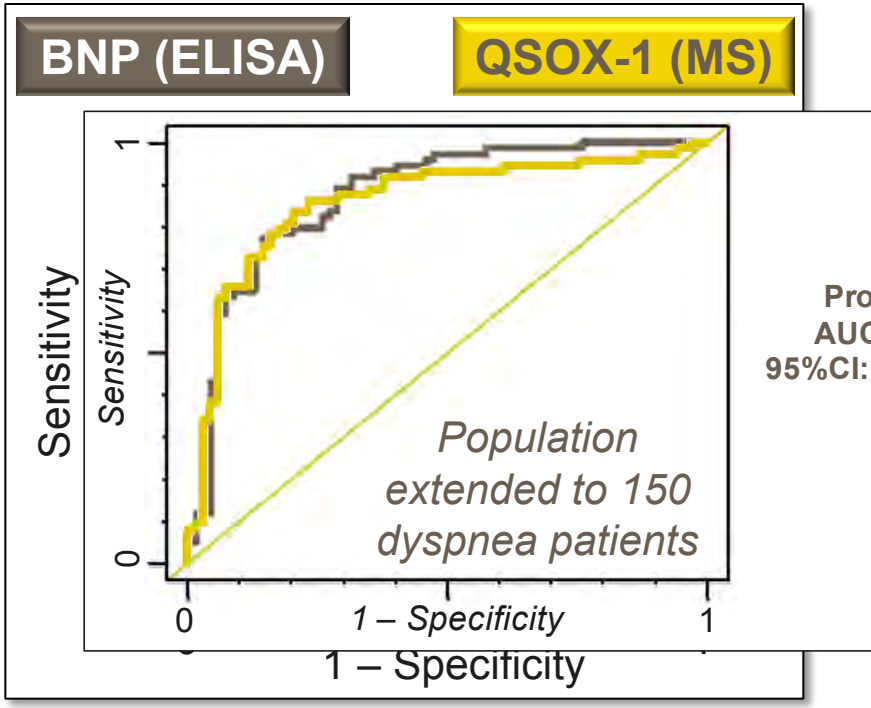
Autres nouveautés ?

-Biomarqueurs du diagnostic de l'ICA

-Mortalité de l'ICA

Unbaised proteomics: QSOX-1

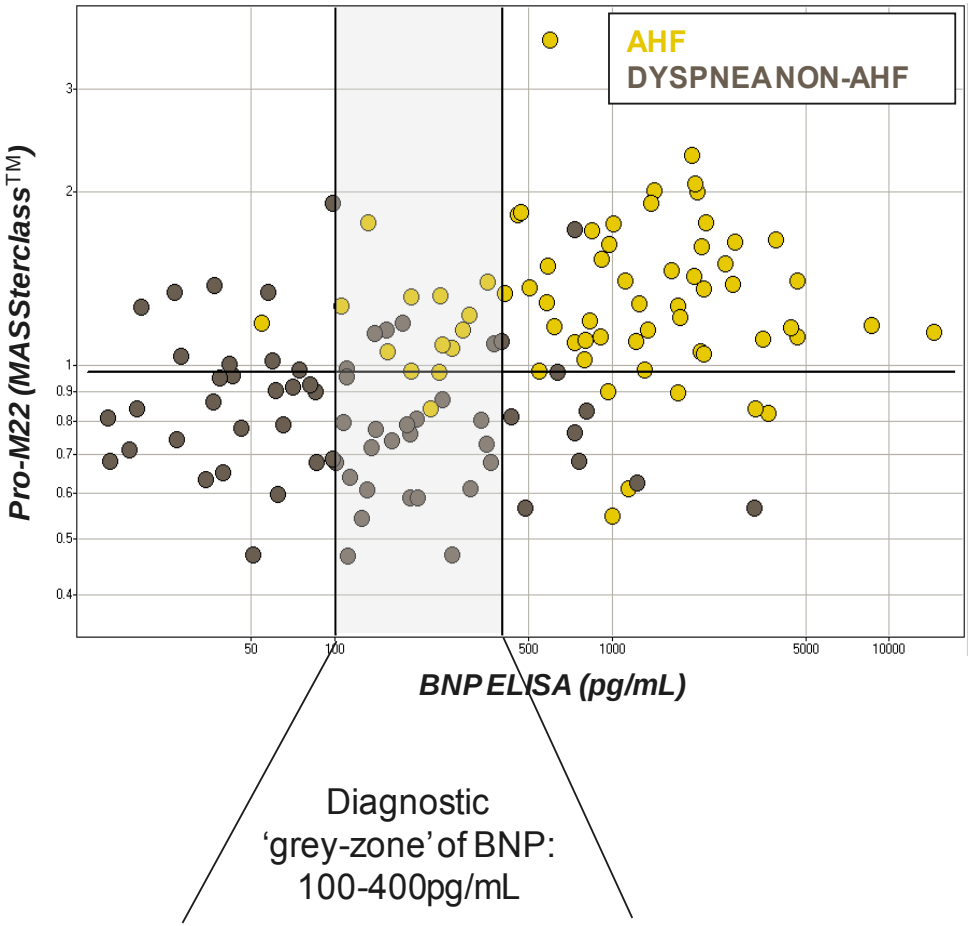




	BNP		QSOX-1	
	n = 80	n = 150	n = 80	n = 150
Median AUC	0.84	0.88	0.89	0.86
95% CI	0.74 – 0.92	0.81 – 0.93	0.79 – 0.96	0.79 – 0.92

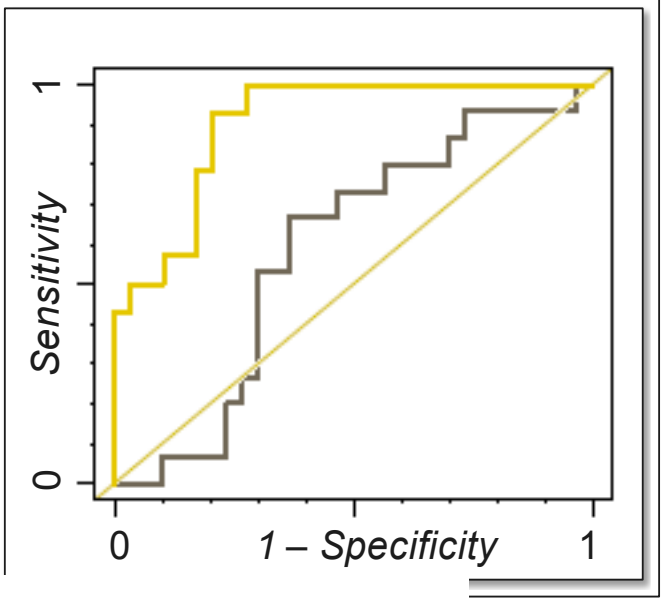
Performance of QSOX-1 in the BNP diagnostic "grey-zone"

Pronota

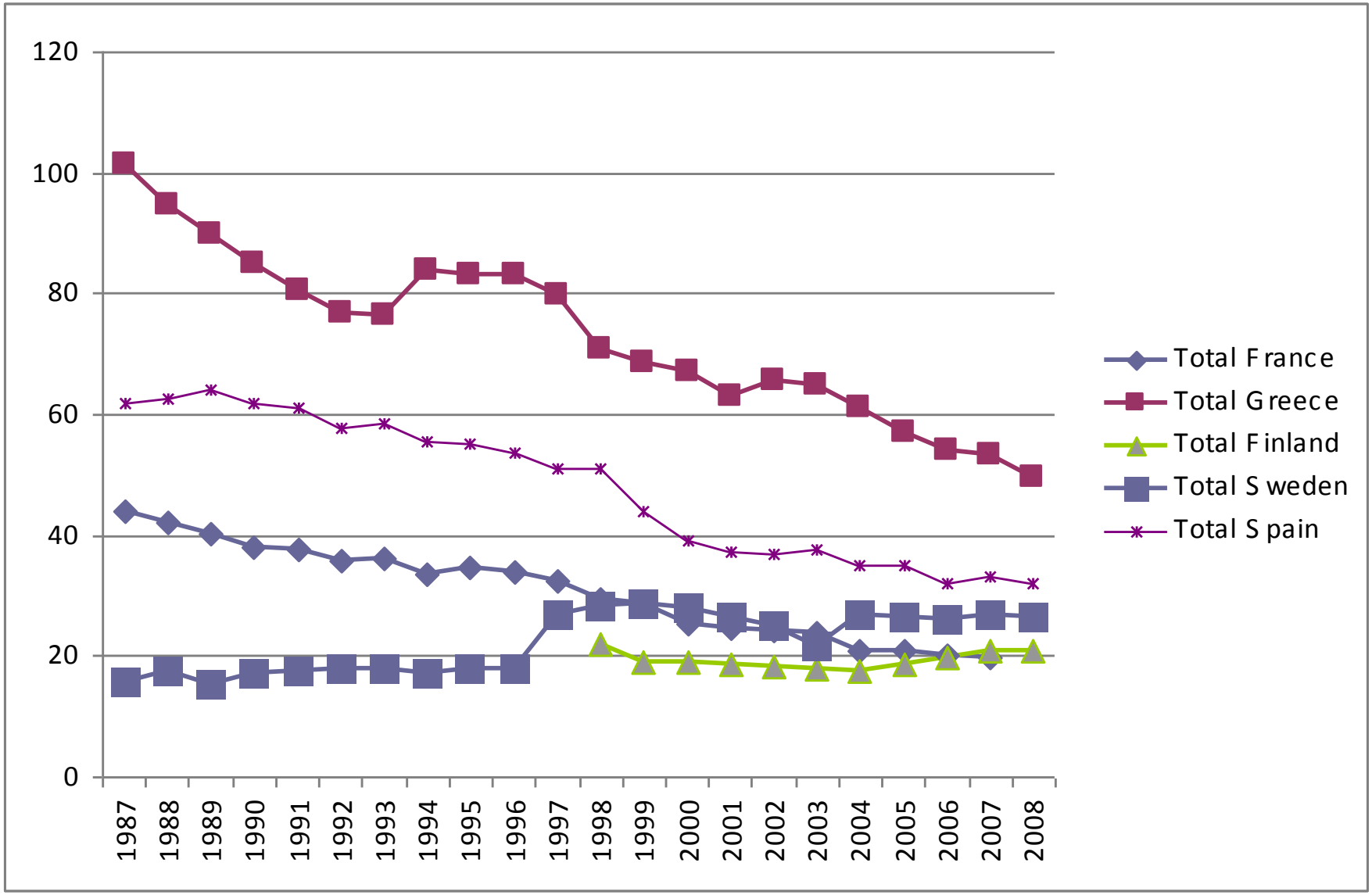


Diagnostic 'grey-zone' of BNP: 100-400pg/mL

Pro-M22	BNP
AUC 0.91	AUC 0.58
95%CI 0.8-097	95%CI 0.42-0.75



Standardized death rates per 100 000 hab



S Laribi et al.

En résumé
ICA post-opératoire

Se passe en SSPI ou en étage plus tard

Trouver le mécanisme de la décompensation cardiaque

- Dysfonction diastolique du VG
- Dysfonction systolo-diastolique du VG
- Dysfonction du VD

ECG, radio du thorax

Biomarqueurs : BNP(ou NT-Pro-BNP)/troponine

Echocardiographie

éviter

- variation de PA et survenue ischémie
- Pour le VD : altération de la gazométrie, remplissage excessif

Favoriser les vasodilatateurs, éviter les catécholamines

ET SURTOUT reprendre vite le(s) traitements au long cours