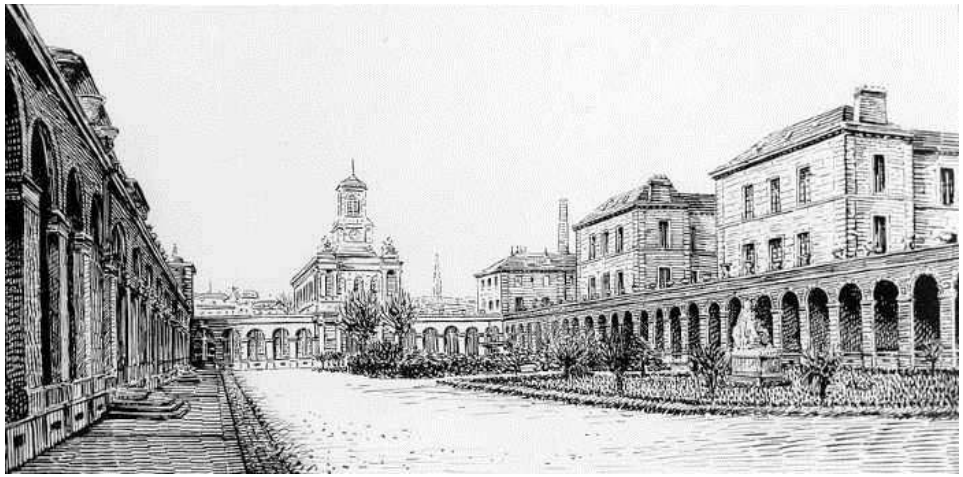
Insuffisance cardiaque aiguë : présentation clinique et biologique



Alexandre Mebazaa

Département d'Anesthésie-Réanimation

Hôpitaux Universitaires Saint Louis – Lariboisière, APHP

Université Paris 7; INSERM – UMR 942

ED: Dyspnea and/or Other Signs of Congestion + Elevated SBP (> 150 mmHg)

Acute pulmonary edema

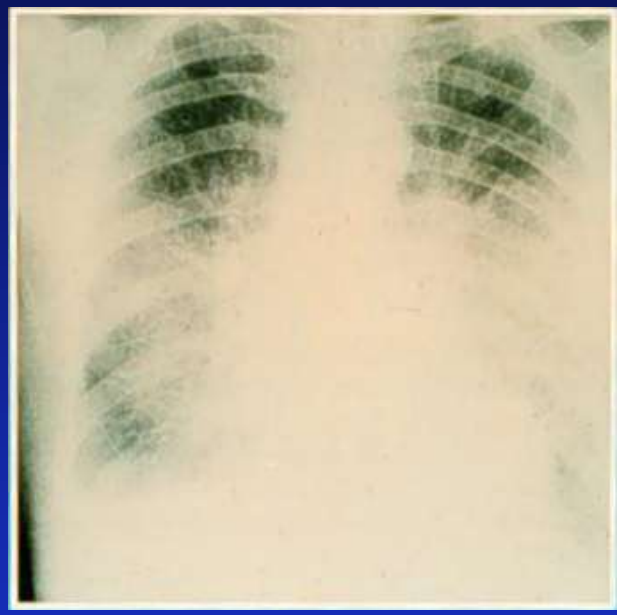
+

- Dyspnea develops abruptly
- Diffuse pulmonary edema
- Minimal systemic edema

It is a <u>vascular</u> illness

+ Warning !
Patient is very often
normovolemic
or hypovolemic

always

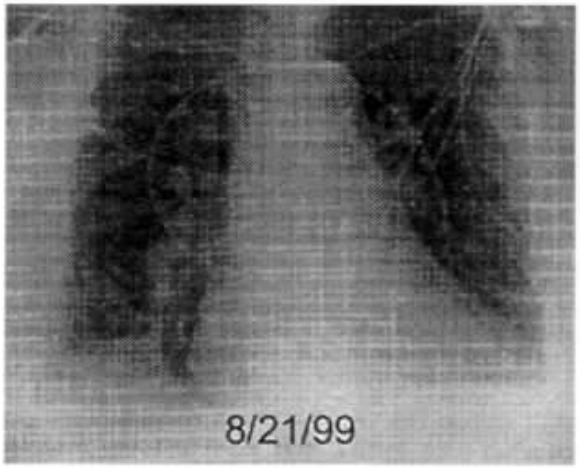


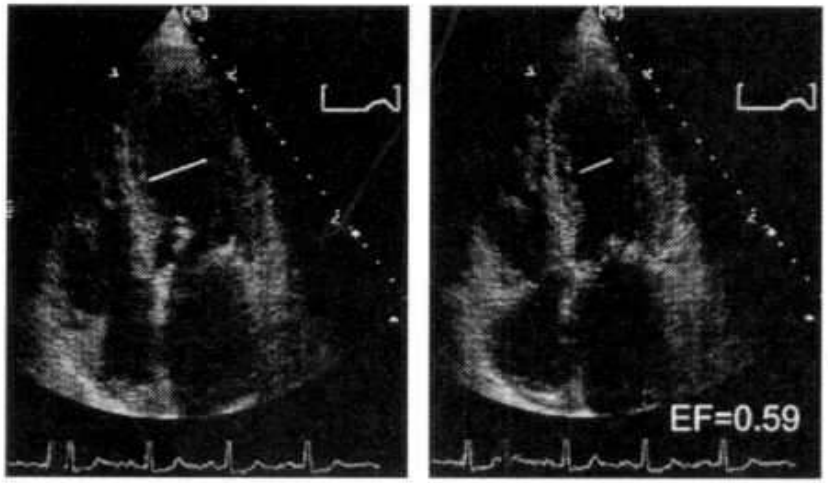
During Acute Pulmonary Edema

Blood pressure, 240/144 mm Hg

After Treatment

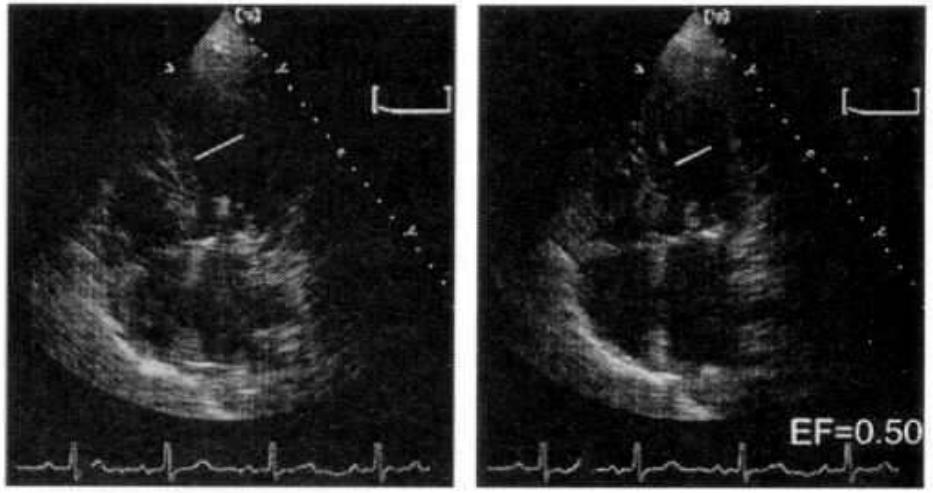
Blood pressure, 149/75 mm Hg





End Diastole

End Systole

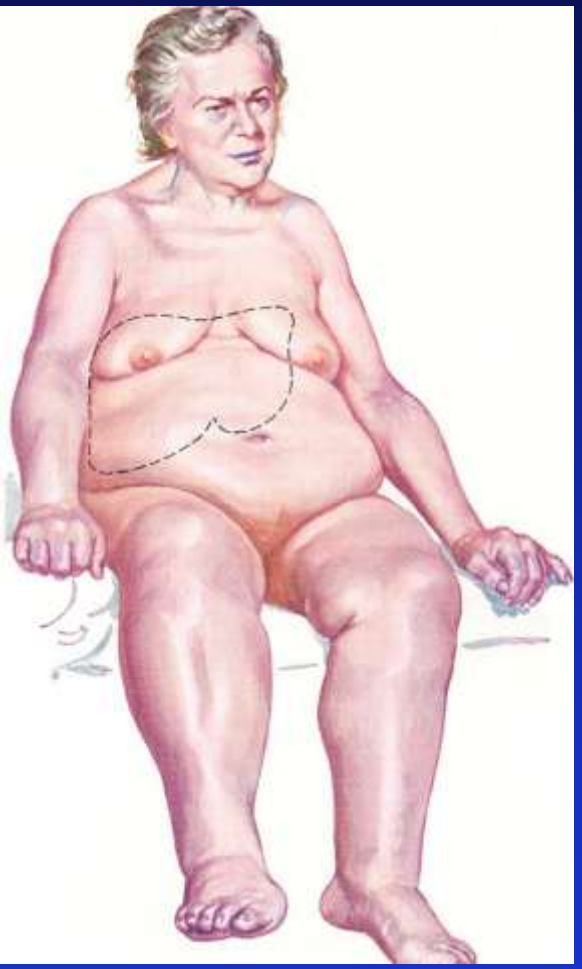


End Diastole

End Systole

SK Ghandi, NEJM 2001, 344: 17-22

CCU: Dyspnea + SBP 110 – 150 mmHg



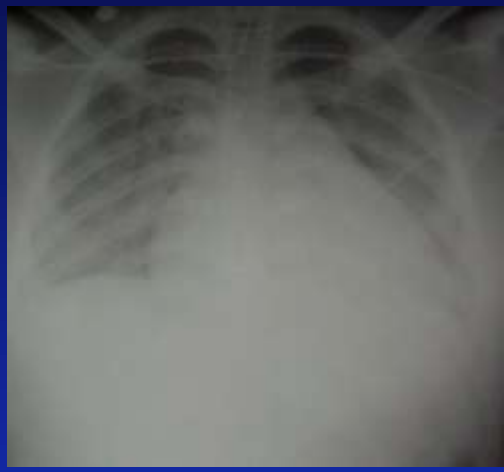
Decompensated chronic heart failure

+

- Dyspnea develops gradually
- Gradual increase in body weight
- Systemic edema
- Minimal pulmonary edema

It is a systemic illness:

- Possible Renal dysfunction
- Anemia
- Low albumin
- Increased Pulmonary Congestion
- Systemic Congestion



or

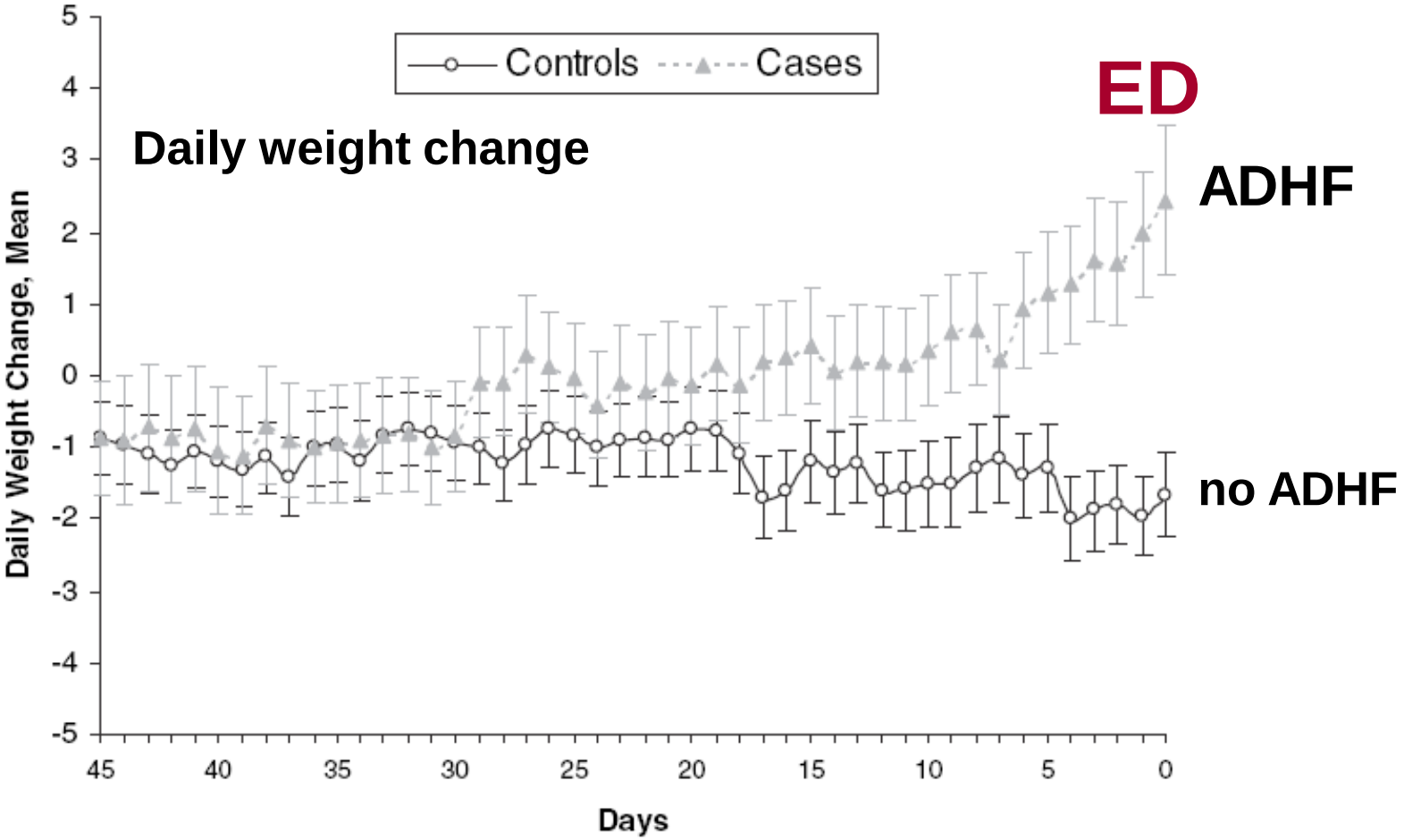


Figure 1. Daily weight change before heart failure hospitalization: cases vs controls. n=268. "Days" on the x-axis denotes days before hospital admission in case patients. The difference in daily weight changes between case and control patients within 30 days before (case) hospitalization was statistically 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

ICU: Cardiogenic Shock: EFICA study
Symptoms on Admission

	All patients	Cardiogenic shock		p^a
	($n=581$)	Yes ($n=166$)	No ($n=415$)	
Symptoms on admission (%)				
Cardiogenic shock	29	100	0	<0.0001
Pulmonary oedema	82	60	91	<0.0001
Peripheral oedema	27	20	30	0.02
Angina	14	17	13	0.29
Hepatomegaly	20	24	18	0.09
Syncope	4	9	2	0.0002
Arrhythmia	23	26	21	0.17
Stroke	1	1	1	1.00
SBP mmHg	**126**	**93**	**139**	**<0.0001**
DBP mmHg	**71**	**54**	**77**	**<0.0001**

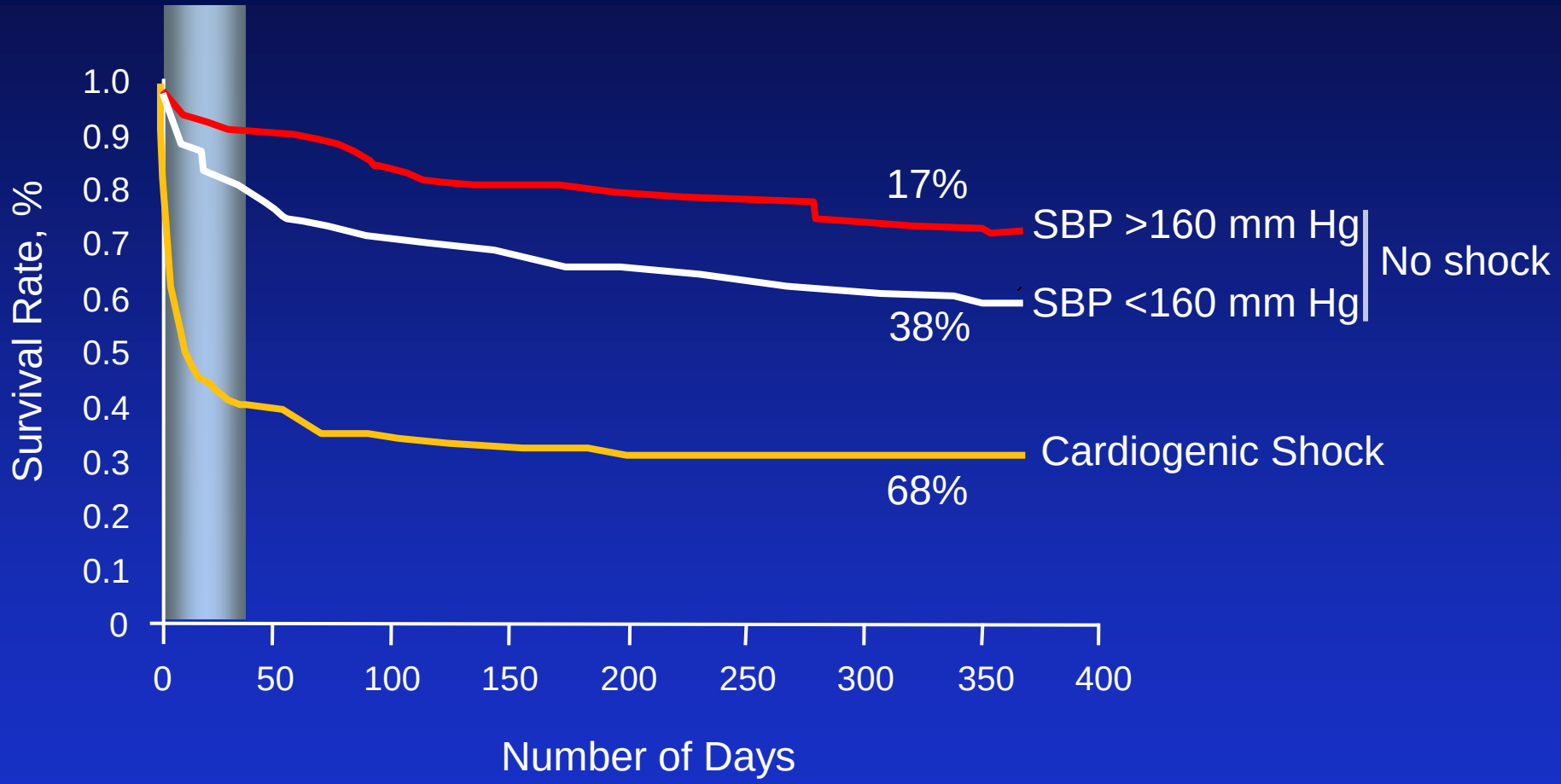
Zannad F, Mebazaa A, et al. Eur J Heart Fail. 2006

Cardiogenic Shock: *Laboratory Tests on Admission*

	All patients	Cardiogenic shock		p^a
	($n=581$)	Yes ($n=166$)	No ($n=415$)	
Laboratory tests (%)				
Hyponatraemia	29	39	25	0.005
Abnormal serum K^+	33	45	28	<0.0001
Creatinine (>200 μmol/L)	53	71	45	<0.0001
Liver dysfunction	61	83	51	<0.0001
Biological marker of cardiac injury (%)				
At least one available‡	87	89	87	0.98
At least one raised	42	56	36	<0.0001

Zannad F, Mebazaa A, et al. Eur J Heart Fail. 2006

EFICA Study
Predictive Factors of Mortality



Zannad F, Mebazaa A, et al. Eur J Heart Fail. 2006

Assessing and grading congestion in acute heart failure: a scientific statement from the Acute Heart Failure Committee of the Heart Failure Association of the European Society of Cardiology and endorsed by the European Society of Intensive Care Medicine

Mihai Gheorghiade[1], Ferenc Follath[2], Piotr Ponikowski[3], Jeffrey H. Barsuk[4], John E.A. Blair[5], John G. Cleland[6], Kenneth Dickstein[7,8], Mark H. Drazner[9], Gregg C. Fonarow[10], Tiny Jaarsma[11], Guillaume Jondeau[12], Jose Lopez Sendon[13], Alexander Mebazaa[14,15], Marco Metra[16], Markku Nieminen[17], Peter S. Pang[18], Petar Seferovic[19], Lynne W. Stevenson[20], Dirk J. van Veldhuisen[21], Faiez Zannad[22], Stefan D. Anker[22], Andrew Rhodes[23], John J.V. McMurray[24], and Gerasimos Filippatos[25]*

Gheorghiade et al. Eur J Heart F 2010

Abstract of the review

Patients with acute heart failure (AHF) require urgent in-hospital treatment for relief of symptoms. The main reason for hospitalization is congestion, rather than low cardiac output. Although congestion is associated with a poor prognosis, many patients are discharged with persistent signs and symptoms of congestion and/or a high left ventricular filling pressure. Available data suggest that a pre-discharge clinical assessment of congestion is often not performed, and even when it is performed, it is not done systematically because no method to assess congestion prior to discharge has been validated. Grading congestion would be helpful for initiating and following response to therapy. We have reviewed a variety of strategies to assess congestion which should be considered in the care of patients admitted with HF. We propose a combination of available measurements of congestion. Key elements in the measurement of congestion include bedside assessment, laboratory analysis, and dynamic manoeuvres. These strategies expand by suggesting a routine assessment of congestion and a pre-discharge scoring system. A point system is used to quantify the degree of congestion. This score offers a new instrument to direct both current and investigational therapies designed to optimize volume status during and after hospitalization. In conclusion, this document reviews the available methods of evaluating congestion, provides suggestions on how to properly perform these measurements, and proposes a method to quantify the amount of congestion present.

« The main reason for hospitalization for acute heart failure is CONGESTION, rather than low cardiac output ».

Gheorghiade et al. Eur J Heart F 2010

EUROPEAN SOCIETY OF CARDIOLOGY®

European Heart Journal (2010) **31**, 703–711
doi:10.1093/eurheartj/ehp507

CLINICAL RESEARCH
Heart failure/cardiomyopath

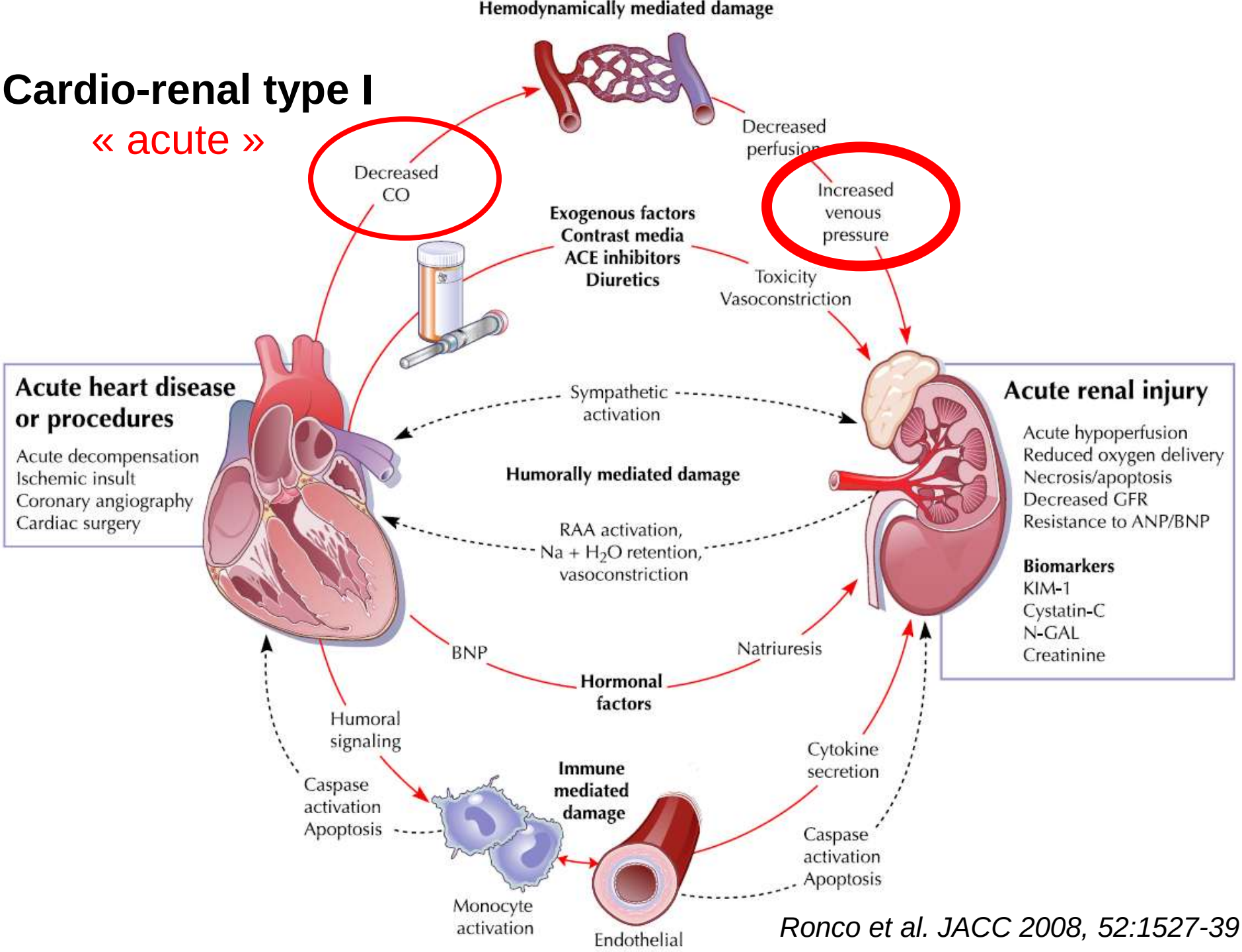
Cardio-renal syndromes: report from the consensus conference of the Acute Dialysis Quality Initiative

Claudio Ronco[1,2*], Peter McCullough[3], Stefan D. Anker[4,5], Inder Anand[6], Nadia Aspromonte[7], Sean M. Bagshaw[8], Rinaldo Bellomo[9], Tomas Berl[10], Ilona Bobek[1], Dinna N. Cruz[1,2], Luciano Daliento[11], Andrew Davenport[12], Mikko Haapio[13], Hans Hillege[14], Andrew A. House[15], Nevin Katz[16], Alan Maisel[17], Sunil Mankad[18], Pierluigi Zanco[19], Alexandre Mebazaa[20], Alberto Palazzuoli[21], Federico Ronco[11], Andrew Shaw[22], Geoff Sheinfeld[23], Sachin Soni[1,24], Giorgio Vescovo[25], Nereo Zamperetti[26], and Piotr Ponikowski[27] for the Acute Dialysis Quality Initiative (ADQI) consensus group

Ronco et al. Eur Heart Journal 2010

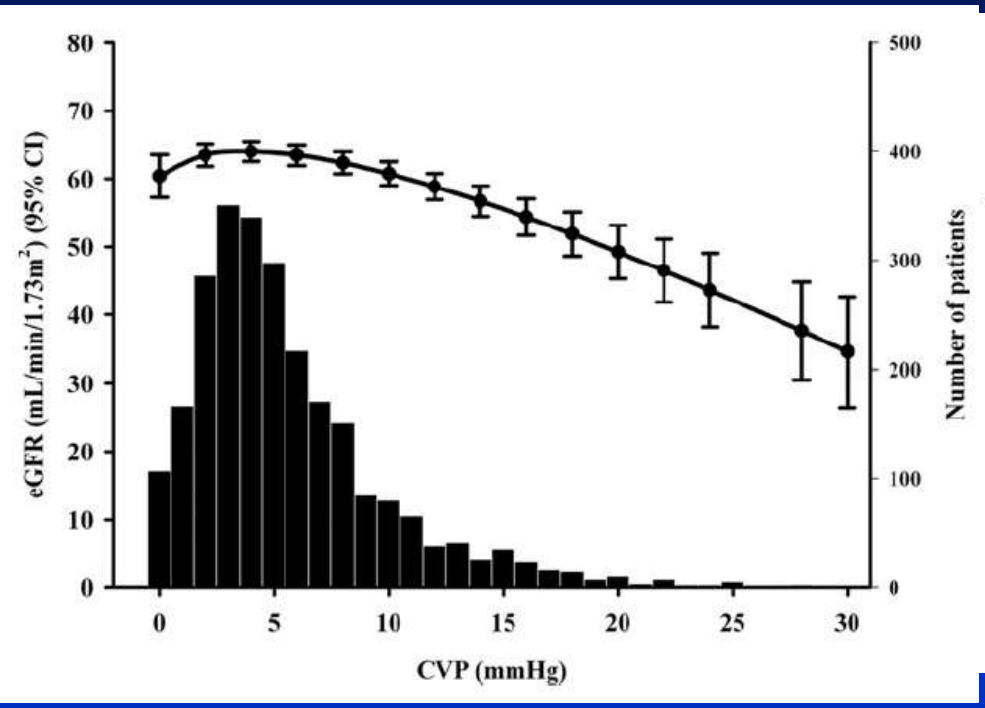
Cardio-renal type I

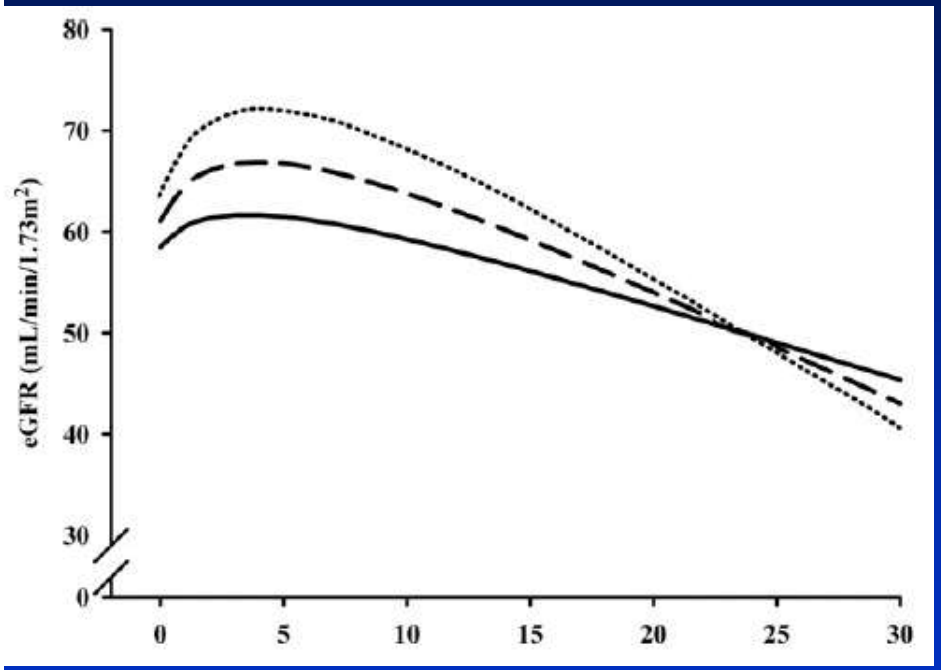
« acute »



Ronco et al. JACC 2008, 52:1527-39

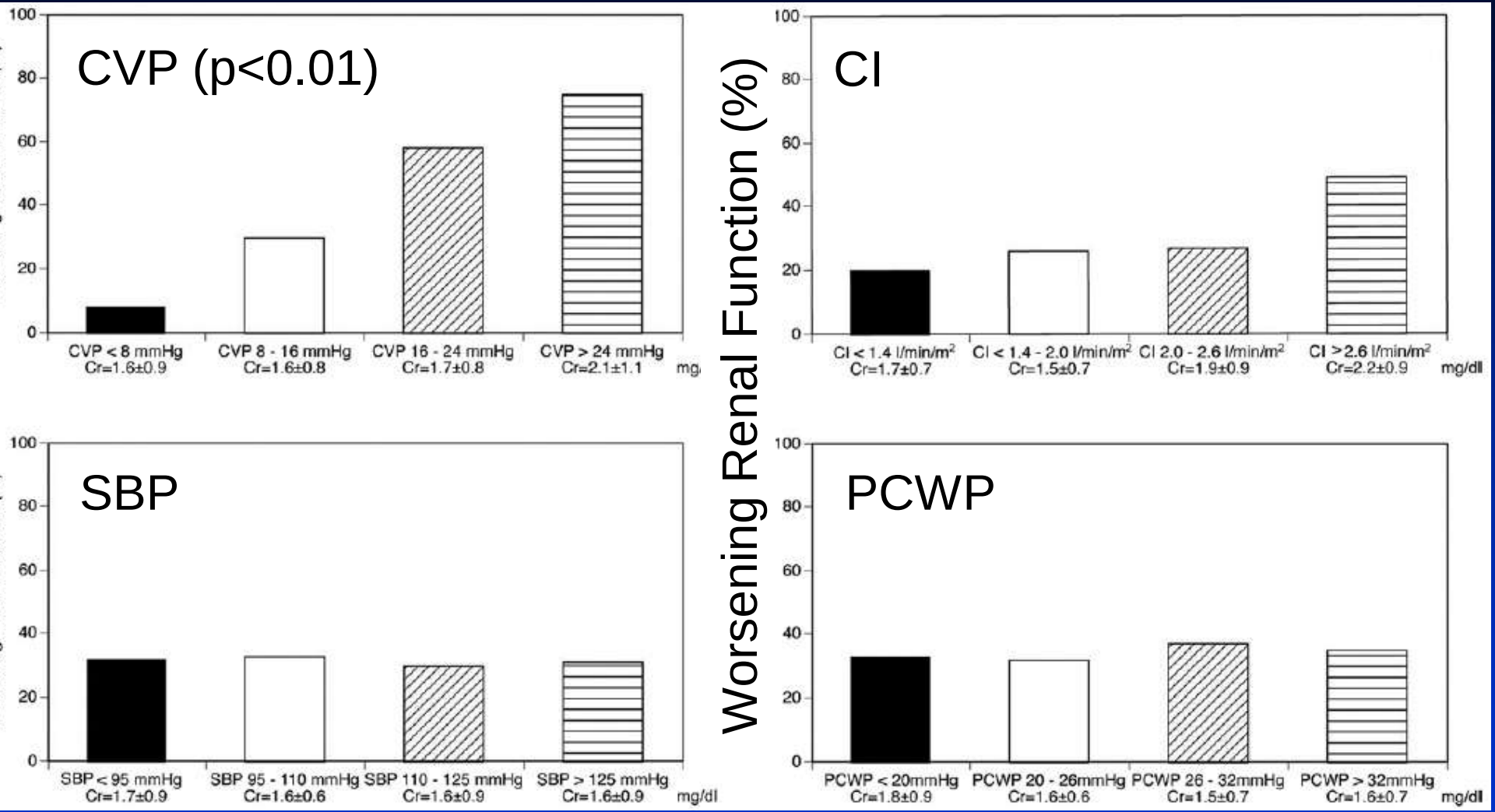
Renal function is impaired as a function of Central Venous Pressure (CVP) in Chronic HF





Damman et al. JACC 2009, 53:582-8

Effects of CVP, CI, SBP and PcwP on worsening renal functionIn Acute Heart Failure patients



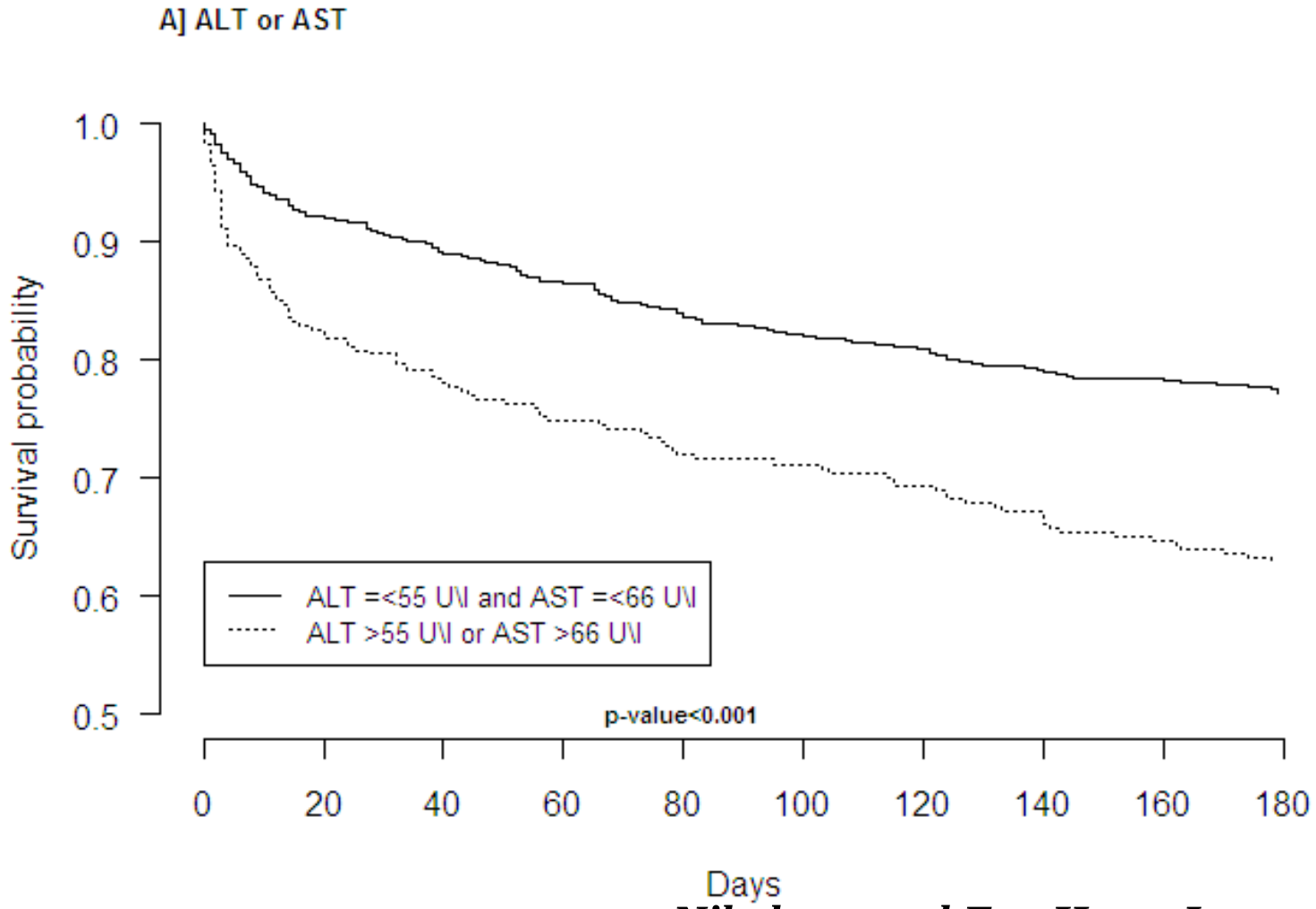
Mullens et al. JACC 2009, 53:589-596

Liver dysfunction in AHF:
Clinical characteristics

	Alk Phosphatase			***Transaminases***		
	normal	abnormal		normal	abnormal	
N	944	261 **(22%)**		740	427 **(37%)**	
Clinical signs at baseline						
SBP (mmHg)	117	114	0.013	117	114	0.012
DBP (mmHg)	71	69	0.073	70	71	NS
HR (bpm)	83	83	NS	81	87	<0.001
Peripheral edema (%)	65.8	79.3	<0.001	70.0	63.7	0.023
Ascites **(%)**	16.9	31.0	<0.001	22.0	17.1	0.049
Cold extremities (%)	20.8	26.1	0.076	19.6	25.5	0.022
Biological parameters at baseline						
BNP (pg/mL)	1465.1	2250.9	<0.001	1464	1918	<0.001
Initial hospitalization characteristics (%)						
Acute MI	19.0	10.7	0.002	11.1	30.1	<0.001
LVEF	24.0	23.3	0.071	24.1	23.5	0.048
Tricuspid regurgitation	45.8	52.9	0.04	51.6	40.8	<0.001
All-cause mortality (%)						
at 31 d	11.1	14.6	NS	8.4	17.6	<0.001
at 180 d	23.5	34.9	0.001	22.4	31.6	<0.001

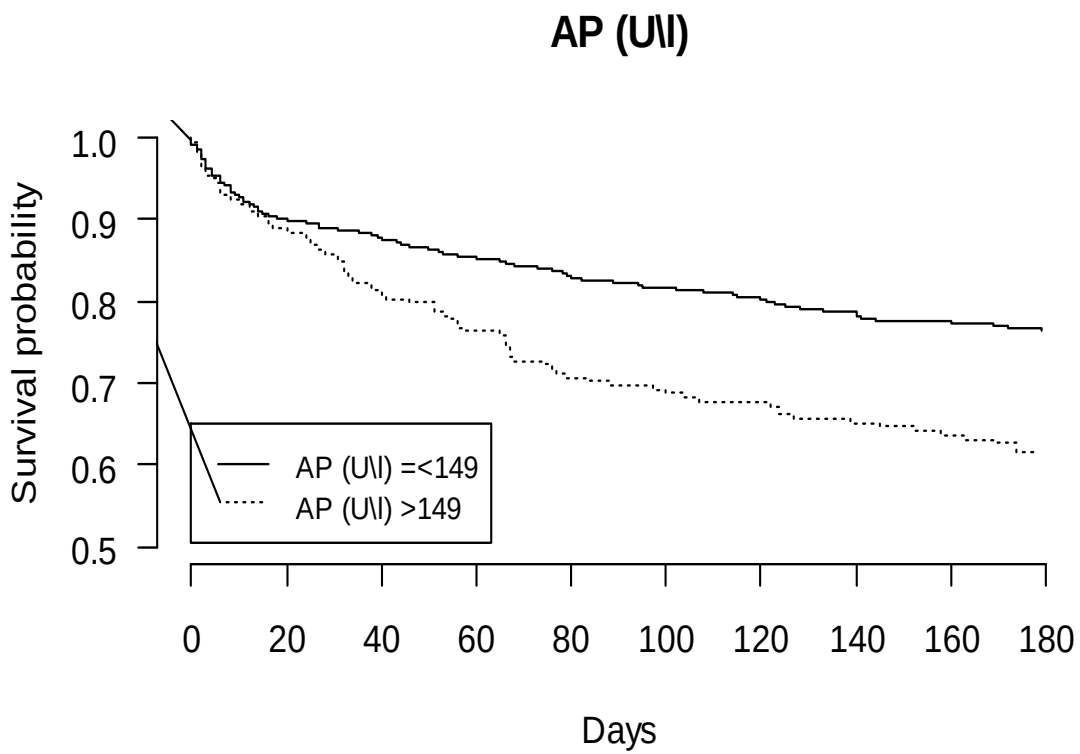
Nikolaou et al Eur Heart Journal (in press)

6-month mortality as a function of liver cytolysis



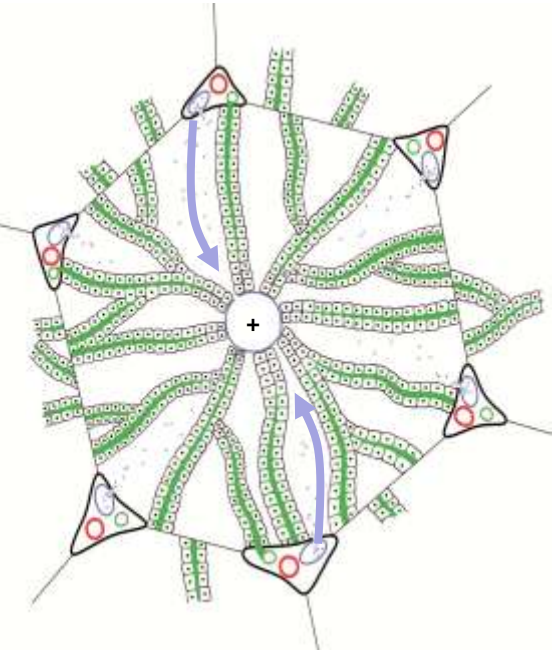
Nikolaou et al Eur Heart Journal (in press)

6-month mortality as a function of cholestatis



Nikolaou et al Eur Heart Journal (in press)

Normal liver lobule

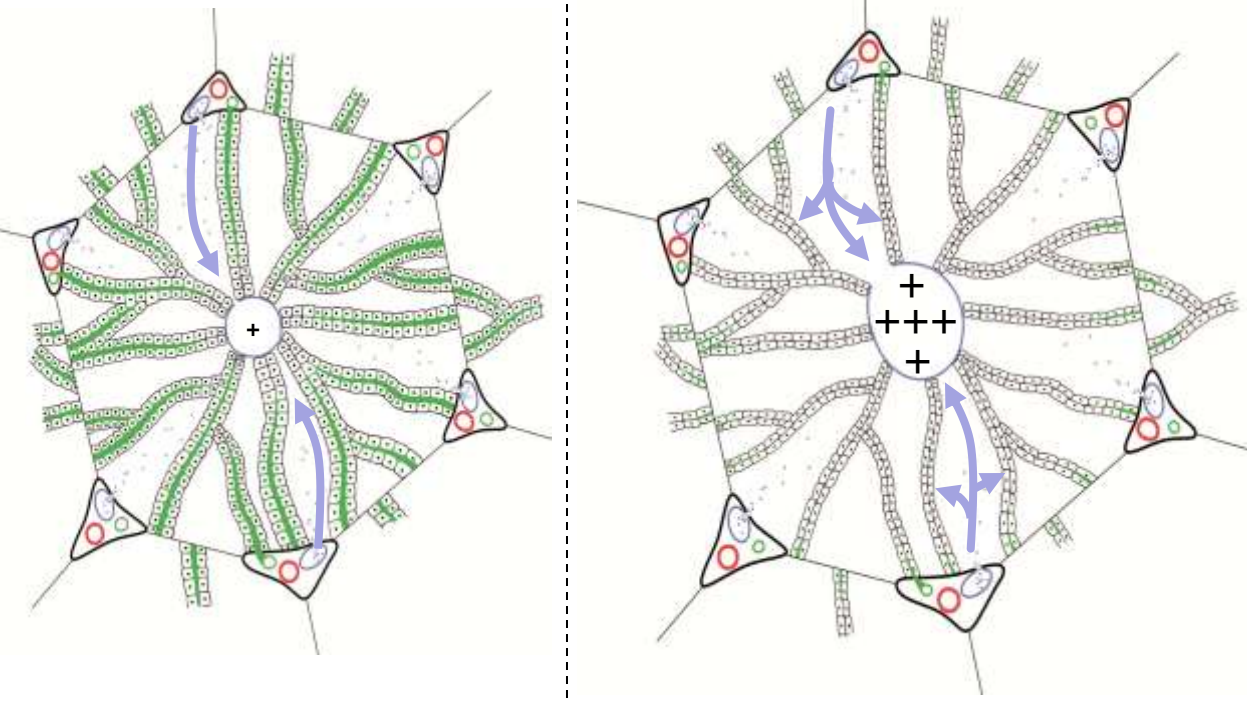


Nikolaou et al Eur Heart Journal (in press)

AHF-induced liver congestion
(increased BNP)

Normal liver lobule

bile duct compression
(increased AP)



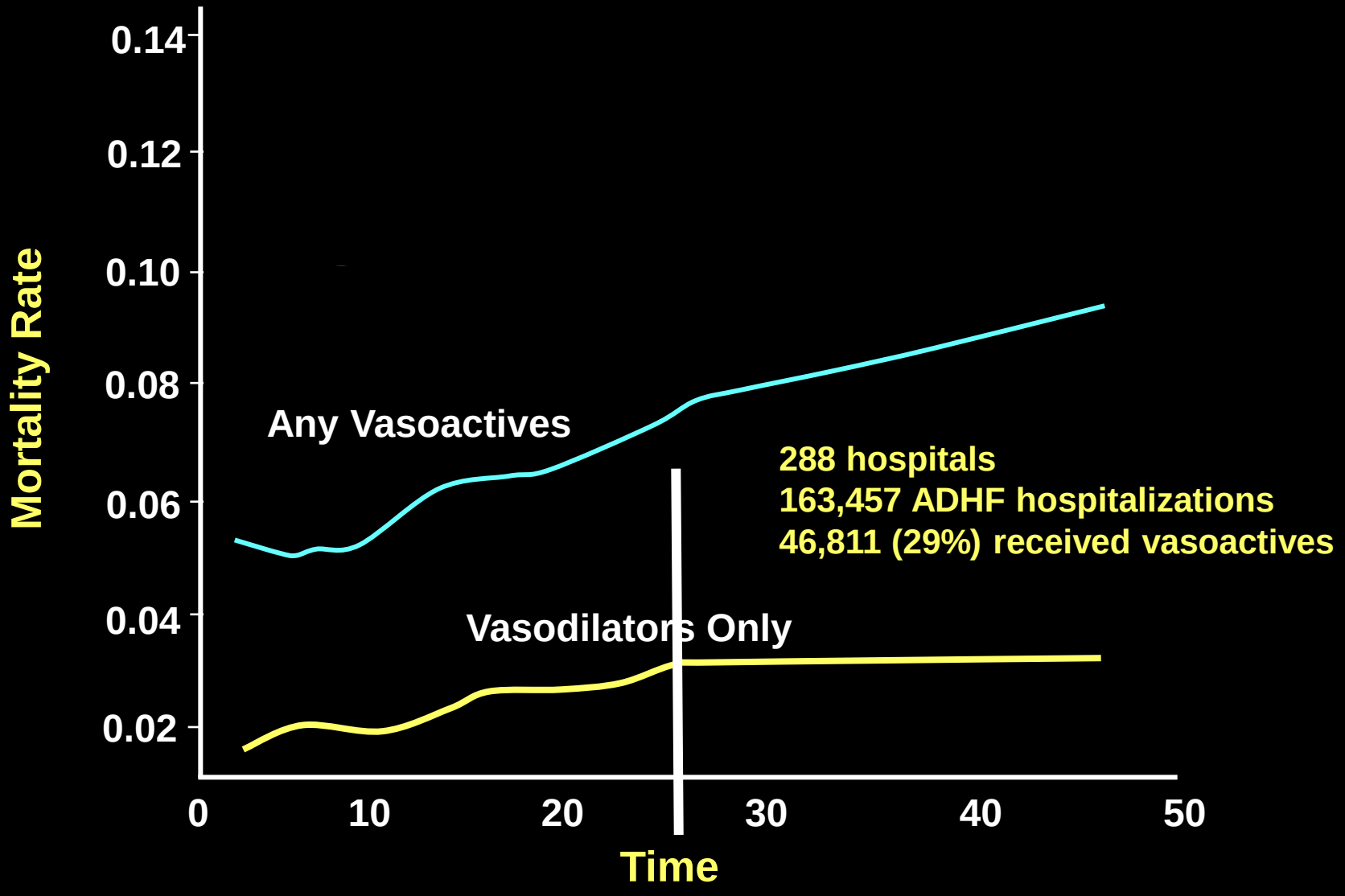
Nikolaou et al Eur Heart Journal (in press)

There are

GOLDEN HOURS

for AHF management

Time to Vasoactives vs. Mortality



Peacock WF. *Ann Emerg Med*. 2003;42(4):S26.

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)

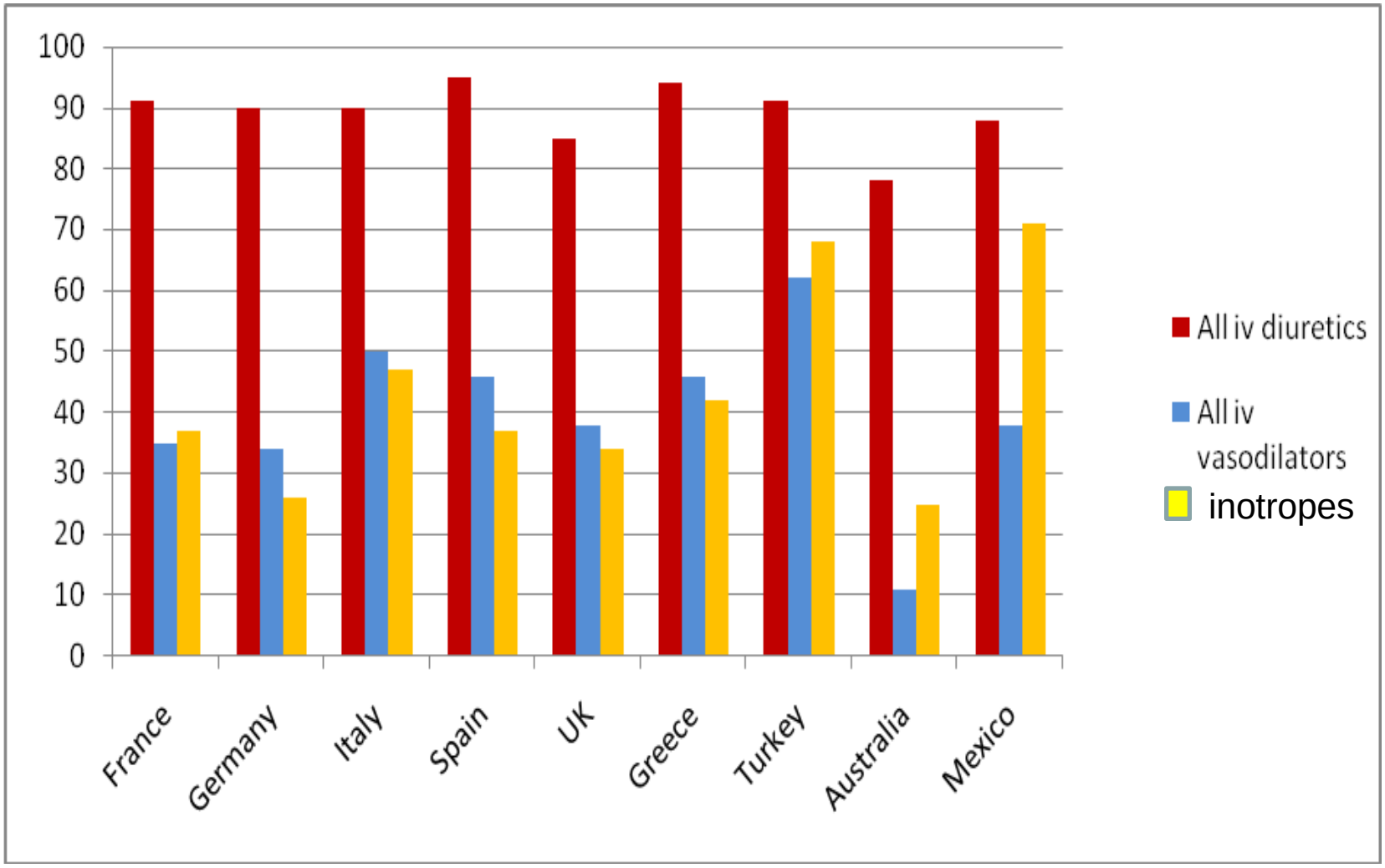
Intensive Care Med (2011) 37:290–301
DOI 10.1007/s00134-010-2073-4

ORIGINAL

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

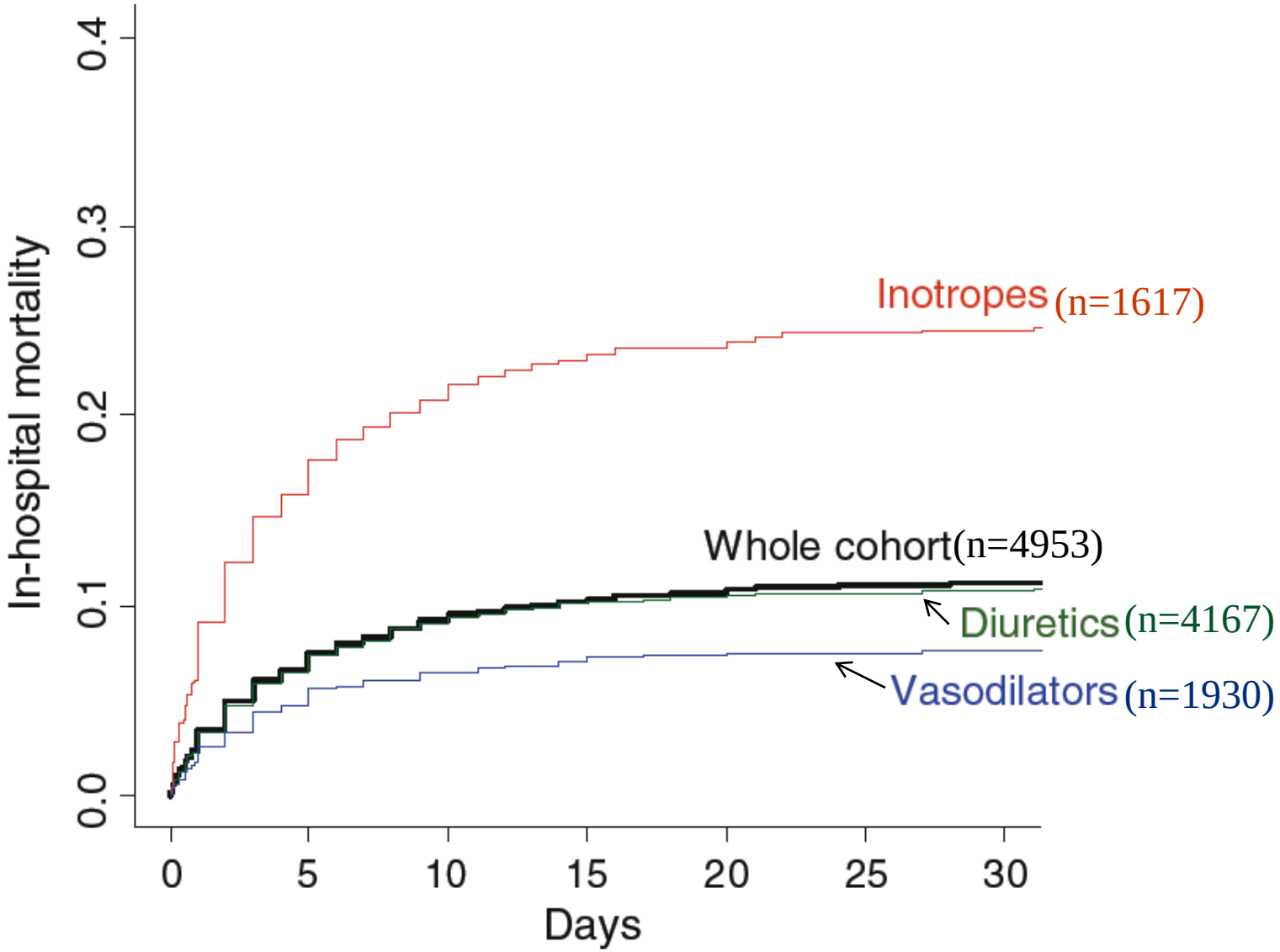
Short-term survival by treatment among patients hospitalized with acute heart failure: the global ALARM-HF registry using propensity scoring methods

ALARM-HF: IV treatment at admission

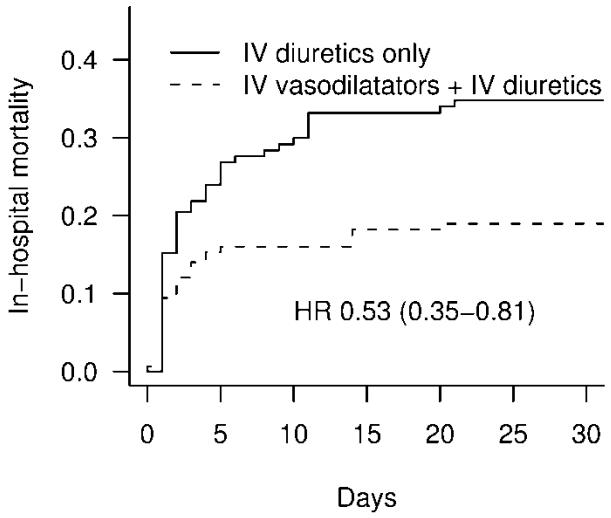


Mebazaa et al Intensive Care Medicine 2011

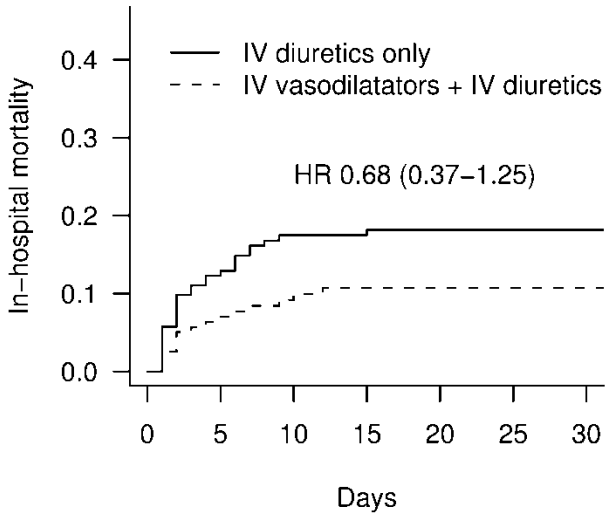
Effect of IV drugs given during the first 48 hours in AHF patients on in-hospital mortality



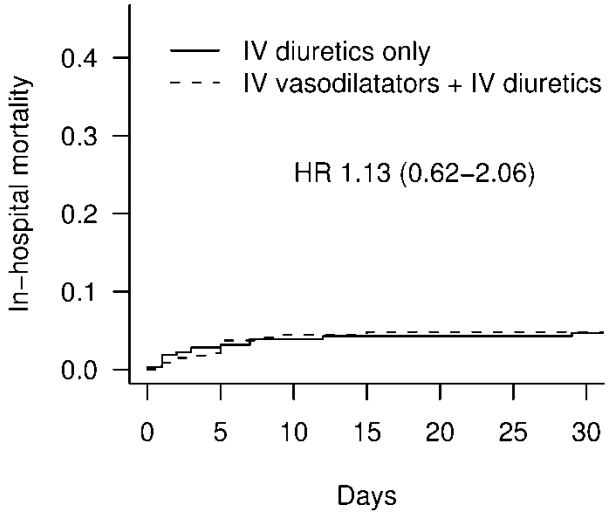
SBP < 100 mmHg (n=318)



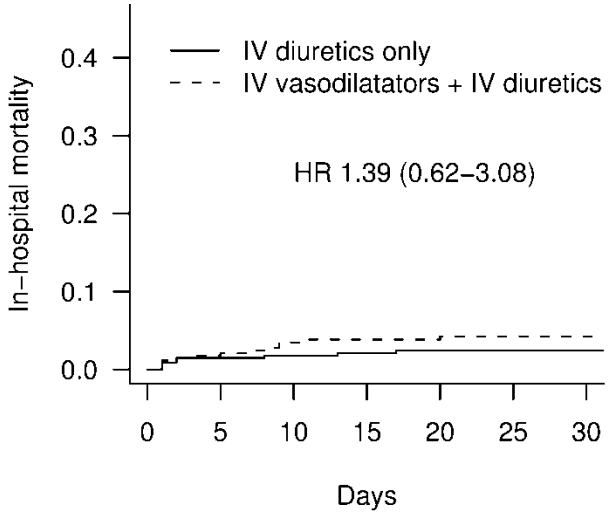
SBP 100-119 mmHg (n=334)



SBP 120-159 mmHg (n=618)



SBP > 160 mmHg (n=694)



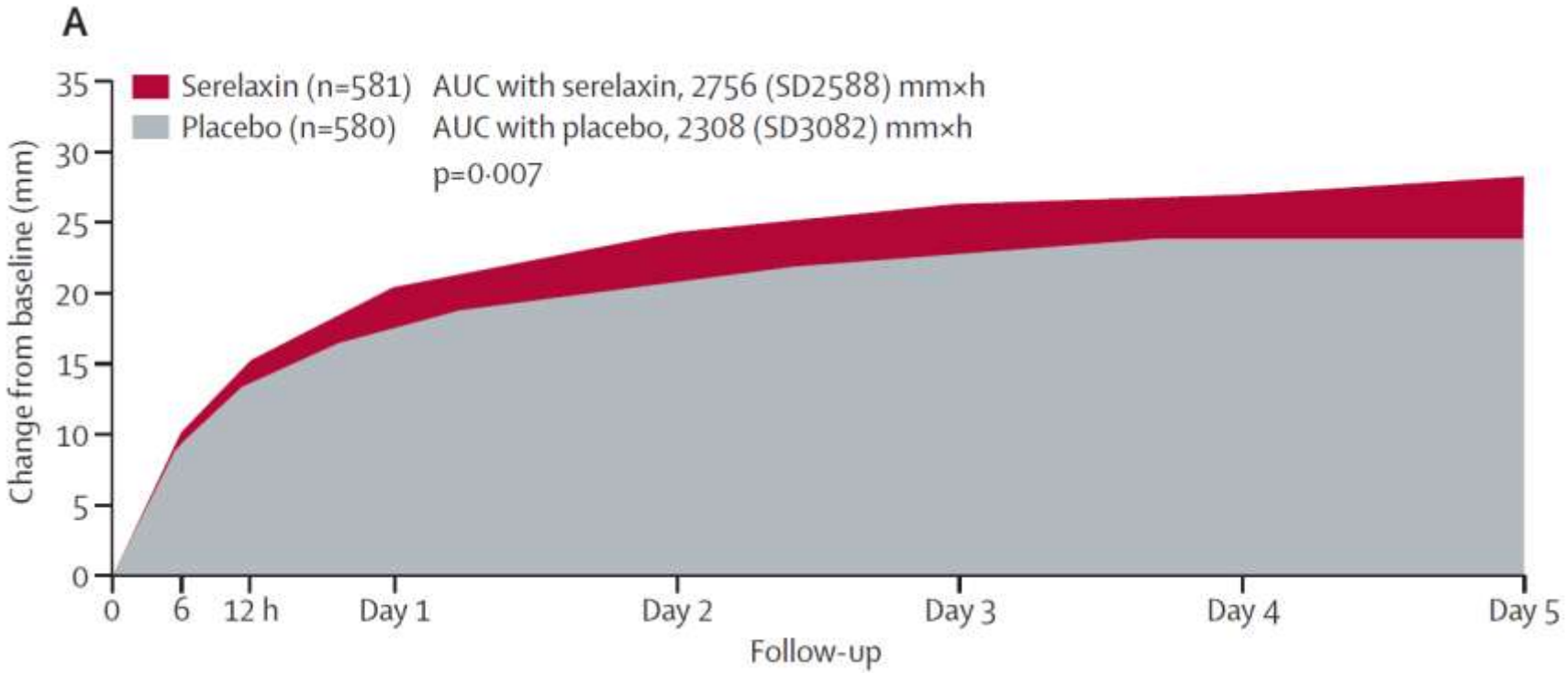
Mebazaa et al Intensive Care Medicine 2011

Serelaxin, recombinant human relaxin-2, for treatment of acute heart failure (RELAX-AHF): a randomised, placebo-controlled trial

John R Teerlink, Gad Cotter, Beth A Davison, G Michael Felker, Gerasimos Filippatos, Barry H Greenberg, Piotr Ponikowski, Elaine Unemori, Adriaan A Voors, Kirkwood F Adams Jr, Maria I Dorobantu, Liliana R Grinfeld, Guillaume Jondeau, Alon Marmor, Josep Masip, Peter S Pang, Karl Werdan, Sam L Teichman, Angelo Trapani, Christopher A Bush, Rajnish Saini, Christoph Schumacher, Thomas M Severin, Marco Metra, for the RELAXin in Acute Heart Failure (RELAX-AHF) Investigators

Teerlink et al, Lancet 2012

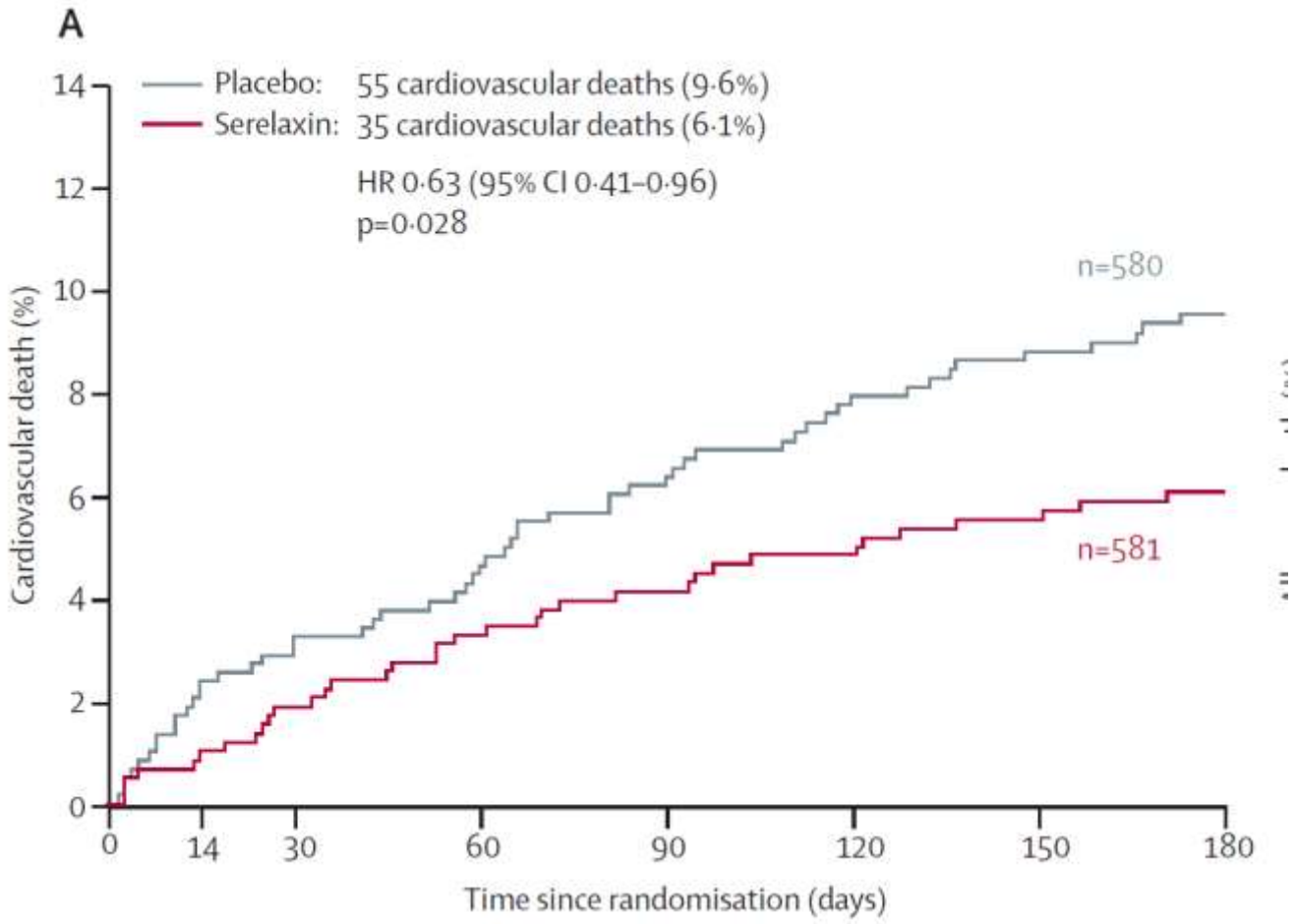
Improvement in dyspnea



Door-to-serelaxin/SOC : 7.2 hours

Teerlink et al, Lancet 2012

Improvement in 180-d cardiovascular mortality



Teerlink et al, Lancet 2012

Clevidipine Improves Dyspnea in ED Acute Heart Failure: A Randomized, Open Label Study

Peacock WF, Baylor College of Medicine, Houston, TX

Chandra A, Kaiser Permanente, Sacramento, CA

Collins S, Vanderbilt University, Nashville, TN

Fonarow G, University of California LA, Los Angeles, CA

Garrison N, Drug Research & Analysis, Montgomery, AL

Mebazaa A, University Paris, Paris, France

TREATMENT

- Most SOC patients 86.8% (46/53) received:
  - Nitroglycerin 56.6% (30/53)
  - Nicardipine 30.2% (16/53)
- 13.2% in the SOC group received
  - IV ISDN 7.5% (4/53)
  - Hydralazine 1.9% (1/53)
  - Diltiazem 1.9% (1/53)
  - NP 1.9% (1/53)

Door to Drug Time

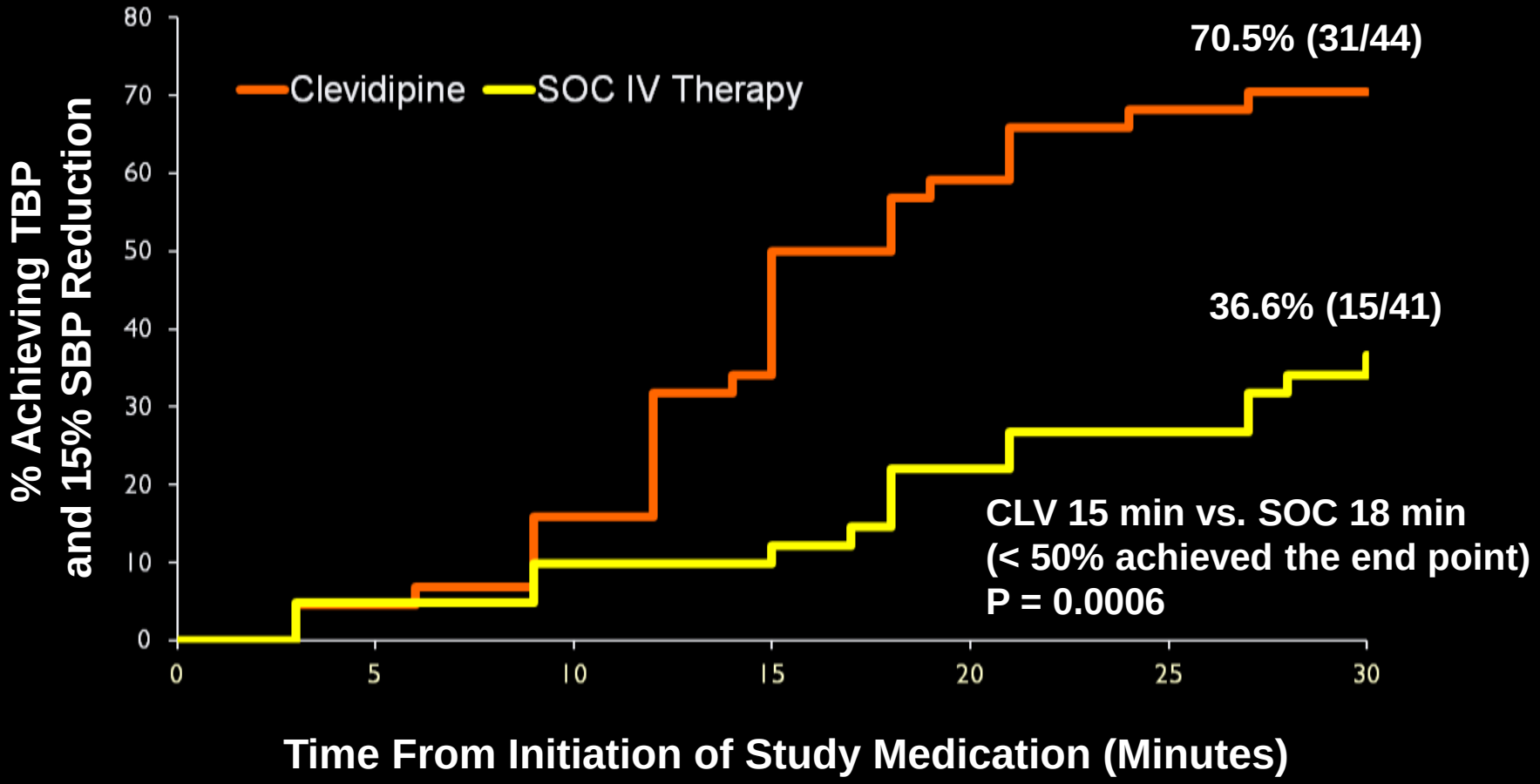
CLV	**2.6 hrs (1.8, 3.7)[1]**
SOC	**2.2 hrs (1.5, 3.2)[1]**

p > 0.05 CLV vs. SOC

[1] Median (Q1, Q3)

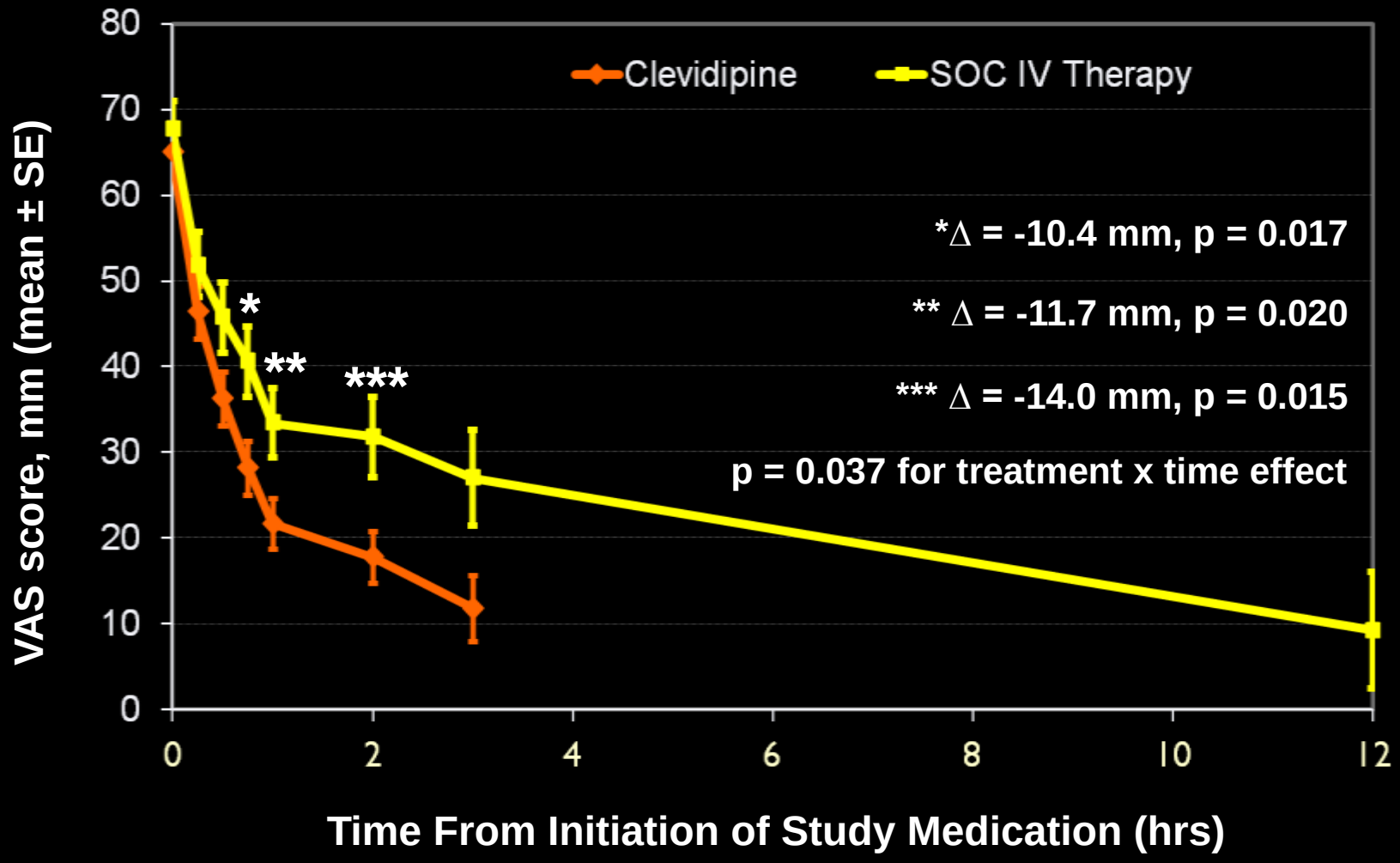
PRIMARY ENDPOINT

- SBP control in 30 minutes (confirmed AHF) (time to target AND 15% SBP reduction)



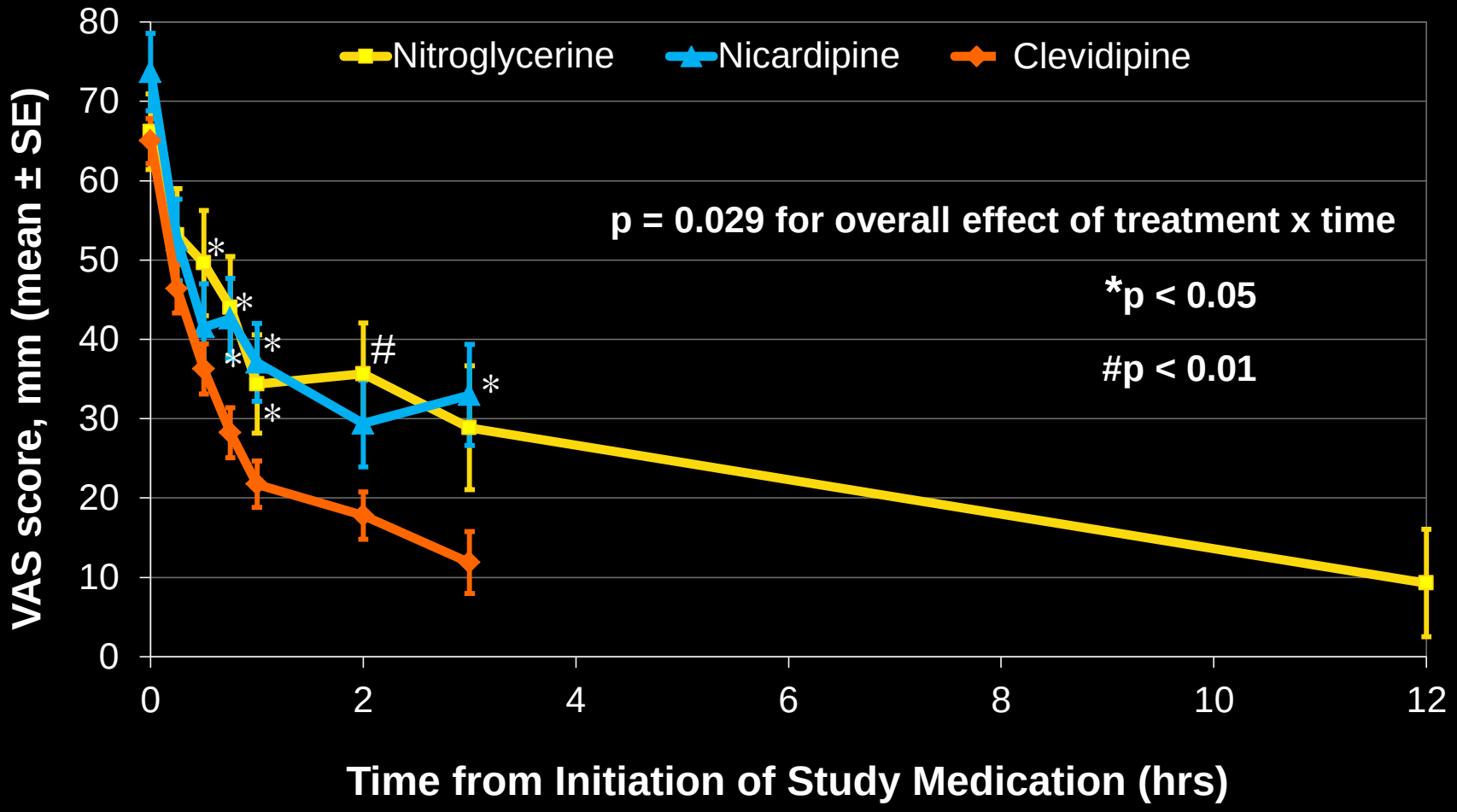
SECONDARY ENDPOINT

VAS score over time (Confirmed AHF)



DYSPNEA REDUCTION

VAS score over time (Confirmed AHF)



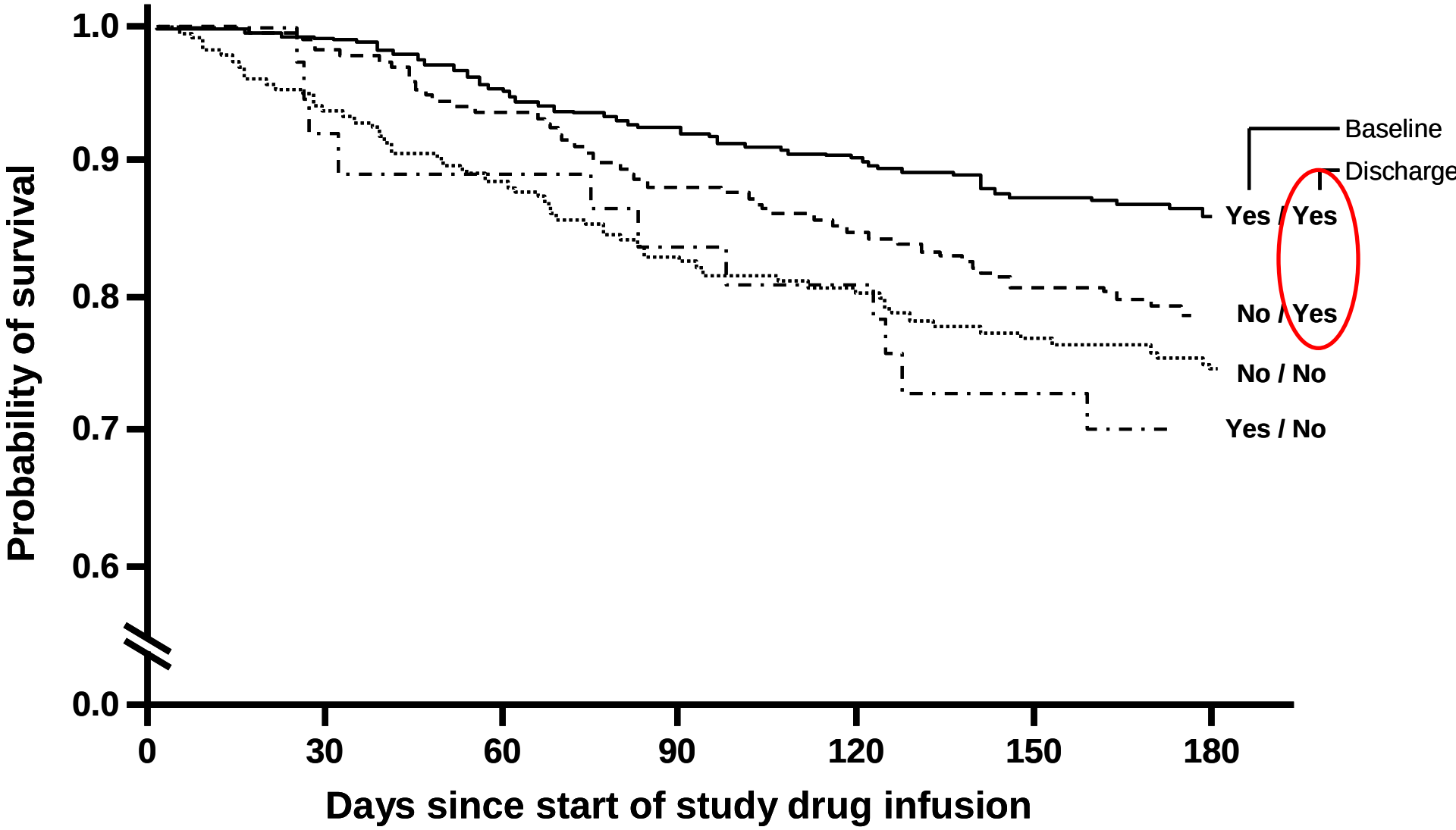
Pre-discharge therapy determines long term outcome

Beneficial association of β-blocker therapy on recovery from severe acute heart failure treatment: Data from the Survival of Patients With Acute Heart Failure in Need of Intravenous Inotropic Support trial*

Michael Böhm, MD; Andreas Link, MD; Danlin Cai, MD; Markku S. Nieminen, MD; Gerasimos S. Filippatos, MD; Reda Salem, MD; Alain Cohen Solal, MD; Bidan Huang, PhD; Robert J. Padley, MD; Matti Kivikko, MD; Alexandre Mebazaa, MD, PhD

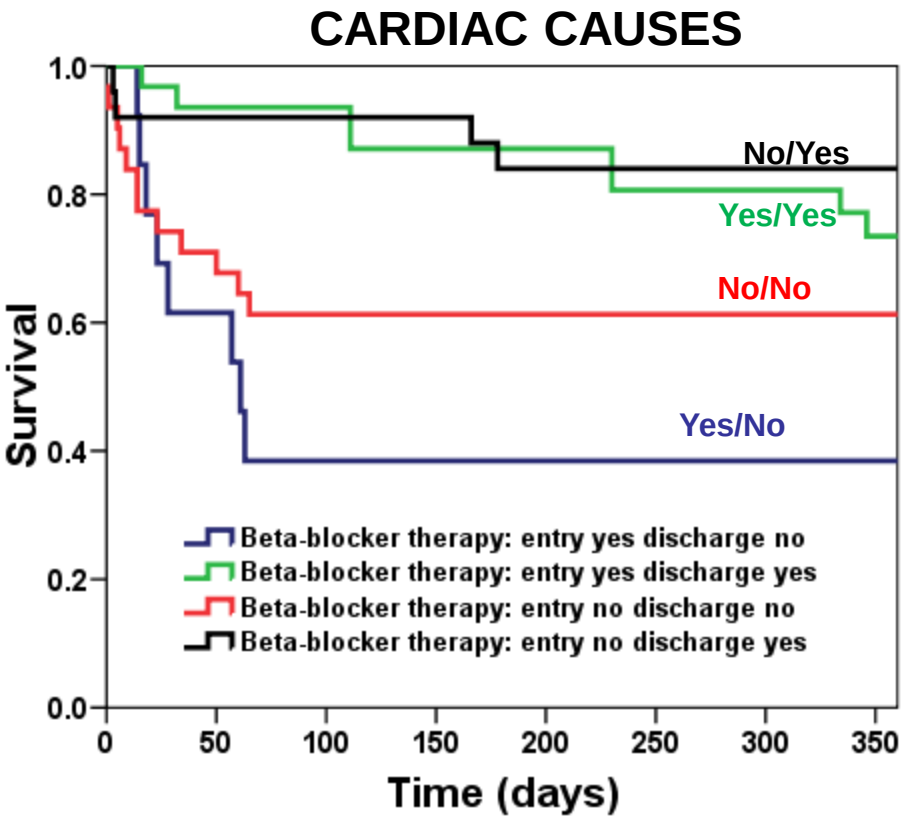
(Crit Care Med 2011; 39:940–944)

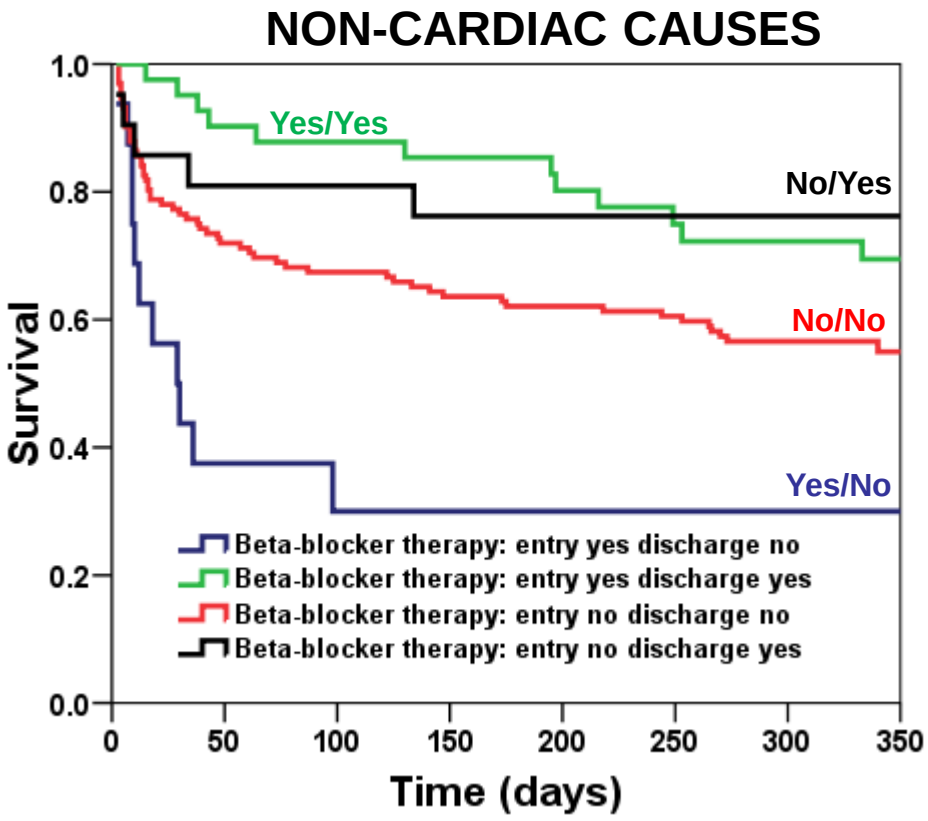
All-Cause Mortality by Beta-Blocker Use at Baseline and Discharge



M. Boehm et al. Crit Care Medicine 2011

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

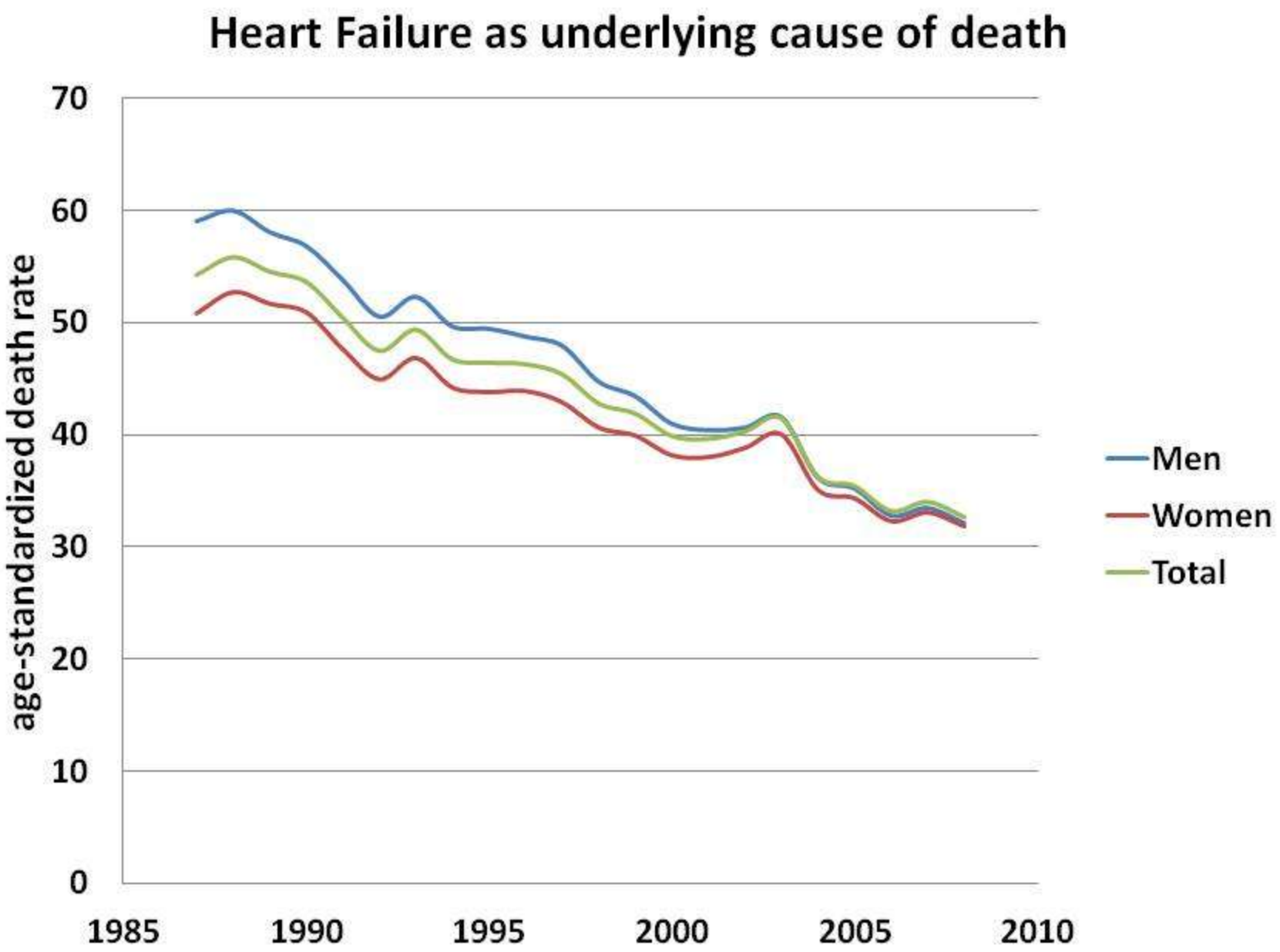




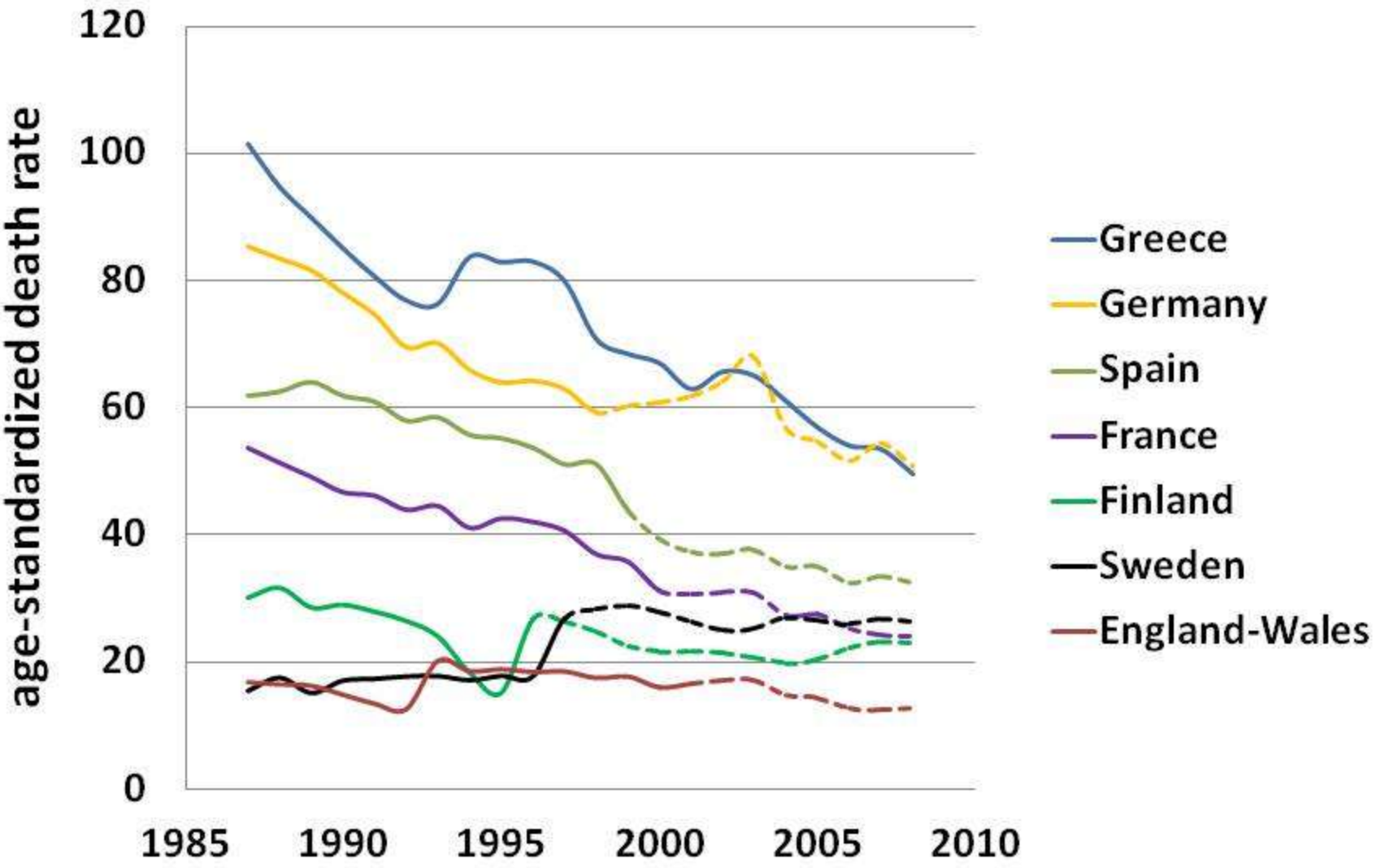
Noveanu et al Crit Care 2011

In summary

- Moderate doses of diuretics
- Higher use of vasodilators as early as possible; it is safe!!
- Much lesser use of catecholamines
- Please, keep or introduce beta-blockers when patient is stabilize and leaving the hospital.



S Laribi et al . Eur J Heart Failure, 2012



S Laribi et al . Eur J Heart Failure, 2012