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Principaux messages

- **ICA sans choc :**
  - **la congestion** est la principale cause d'admission
  - Les vasodilateurs sont les médicaments de choix
- **Choc cardiogénique**
  - Le bas débit cardiaque et l'ischémie myocardique sont en première ligne
  - Privilégier NA+inotrope; ne pas donner d'adrénaline
- « **le temps est du muscle** »

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

Guideline recommendations for the prehospital and early in-hospital (first 6–12 hrs after presentation) management of acute heart failure syndromes are lacking. The American College of Cardiology/American Heart Association and European Society of Cardiology guidelines direct the management of these acute heart failure patients, but specific consensus on early management has not been published, primarily because few early management trials have been conducted. This article summarizes practical recommendations for the prehospital and early management of patients with acute heart failure syndromes; the recommendations were developed from a meeting of experts in cardiology, emergency medicine, and intensive care medicine from Europe and the United States. The recommendations are based on a unique clinical classification system considering the initial systolic blood pressure and other symptoms: 1) dyspnea and/or congestion with systolic blood pressure >140 mm Hg; 2) dyspnea and/or congestion with systolic blood pressure 100–140 mm Hg; 3) dyspnea and/or congestion with systolic blood pressure <100 mm Hg; 4) dyspnea and/or congestion with signs of acute coronary syndrome; and 5) isolated right ventricular failure. These practical recommendations are not intended to replace existing guidelines. Rather, they are meant to serve as a tool to facilitate guideline implementation where data are available and to provide suggested treatment approaches where formal guidelines and definitive evidence are lacking. (Crit Care Med 2008; 36[Suppl.]:S129–S139)

KEY WORDS: heart failure; acute; emergency treatment

Mebazaa et al, Crit Care Medicine 2008

The 3 clinical scenarios

Clinical Scenario	Characteristics
CS1	SBP >140 mm Hg
	Symptoms develop abruptly
	Predominantly diffuse pulmonary edema
	Minimal systemic edema (patient may be euvolemic or hypovolemic)
	Acute elevation of filling pressure often with preserved LVEF
	Vascular pathophysiology
CS2	SBP 100–140 mm Hg
	Symptoms develop gradually, together with a gradual increase in body weight
	Predominantly systemic edema
	Minimal pulmonary edema
	Chronic elevation of filling pressure, including increased venous pressure and elevated pulmonary arterial pressure
	Manifestations of organ dysfunction (renal impairment, liver dysfunction, anemia, hypoalbuminemia)
CS3	SBP <100 mm Hg
	Rapid or gradual onset of symptoms
	Predominantly signs of hypoperfusion
	Minimal systemic and pulmonary edema
	Elevation of filling pressure

Mebazaa et al, Crit Care Medicine 2008

Who are the 3 clinical scenarios?

CS1: ED: Dyspnea and/or Other Signs of Congestion + Elevated SBP (> 150 mmHg)

always

Acute pulmonary edema

+

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

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

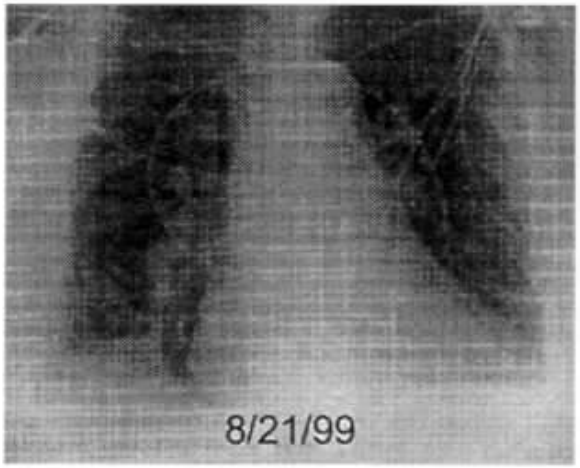
+ Warning !
Patient is very often
normovolemic
or hypovolemic

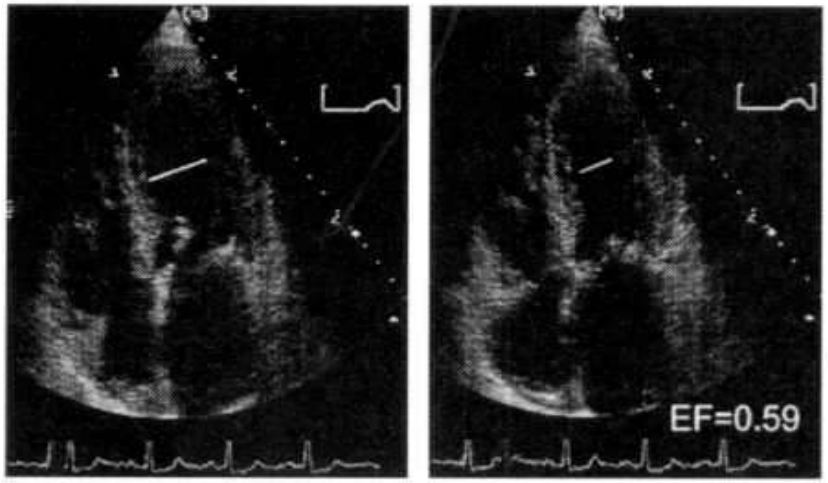
During Acute Pulmonary Edema

Blood pressure, 240/144 mm Hg

After Treatment

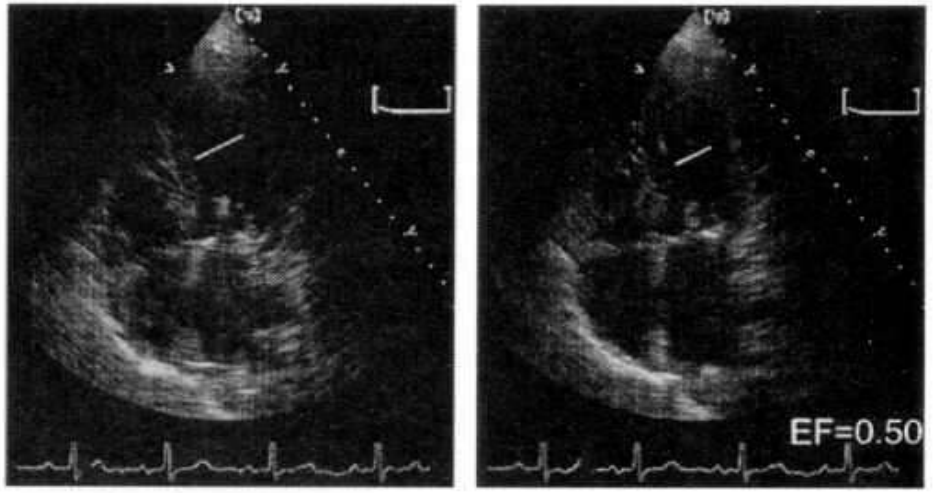
Blood pressure, 149/75 mm Hg





End Diastole

End Systole



End Diastole

End Systole

SK Ghandi, NEJM 2001, 344: 17-22

CS2: CCU, Dyspnea+SBP 110 – 150mmHg

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

CS3: 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**

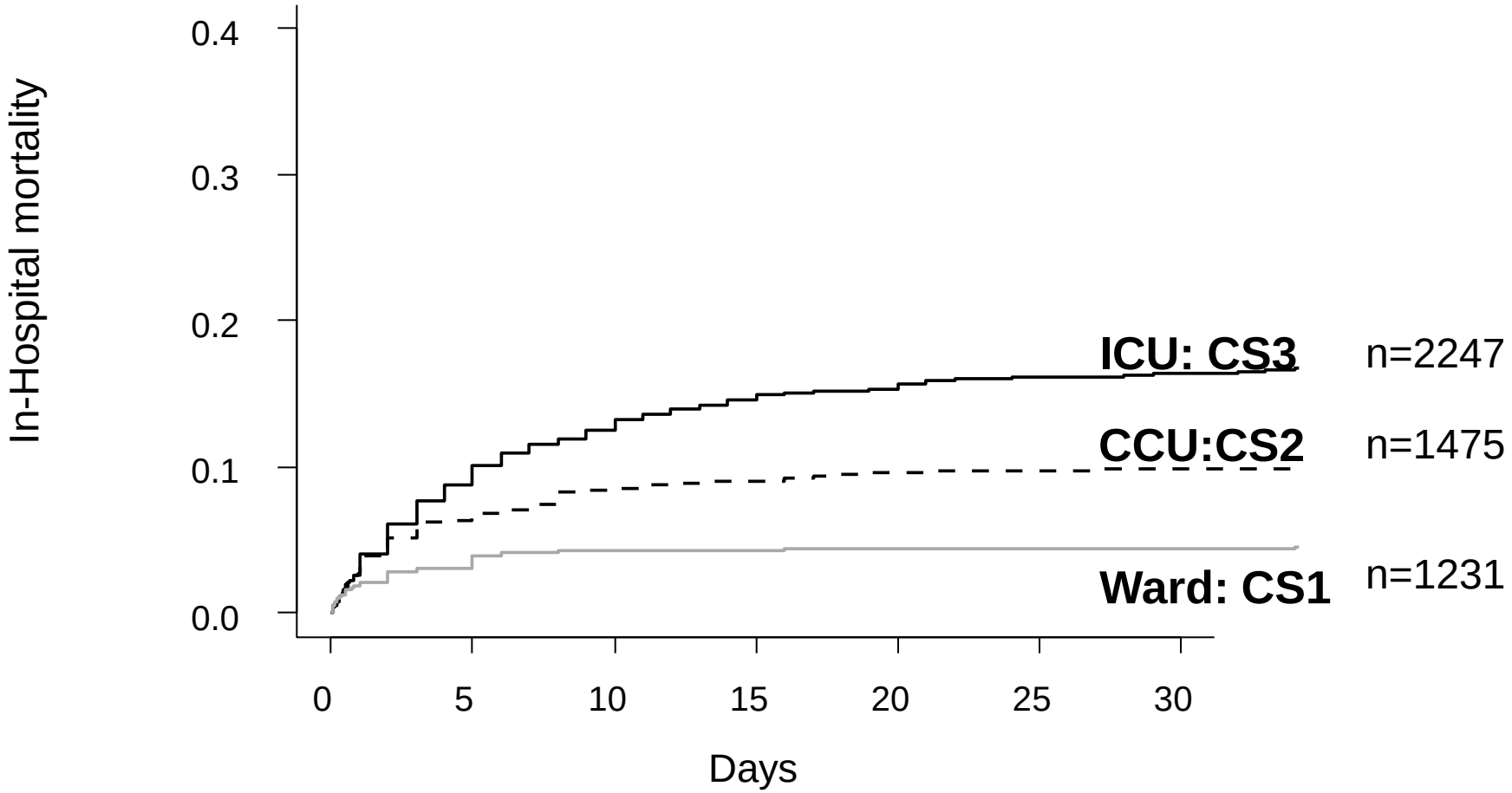
Zannad F, Mebazaa A, et al. Eur J Heart Fail. 2006

ALARM-HF: Patients characteristics at admission

Variable	Patients hospitalized in			p value[b]
	ICU, **CS3** $n = 2,247$	CCU, **CS2** $n = 1,475$	Ward, **CS1** $n = 1,231$	
Median SBP (mmHg) (IQR)	120 (95–160)	130 (100–160)	140 (110–160)	<0.0001
SBP < 100 (mmHg), no. (%)	617 (27.7)	298 (20.3)	100 (8.3)	<0.0001
Median DBP (mmHg) (IQR)	78 (58–95)	80 (60–94)	80 (70–95)	<0.0001
Heart rate, median (IQR)	110 (90–125)	110 (90–120)	100 (86–118)	<0.0001
Cardiogenic shock (%)	16.2	12.3	2.9	<0.0001
Pulmonary edema (%)	38.1	42.8	27.0	<0.0001
Cold extremities (%)	33.1	26.2	13.2	<0.0001
Normal diuresis at baseline (%)	47.2	54.3	65.5	<0.0001
Median BNP (IQR)[a]	1108 (552–1,995)	1045 (642–2,136)	700 (313–1,640)	0.009

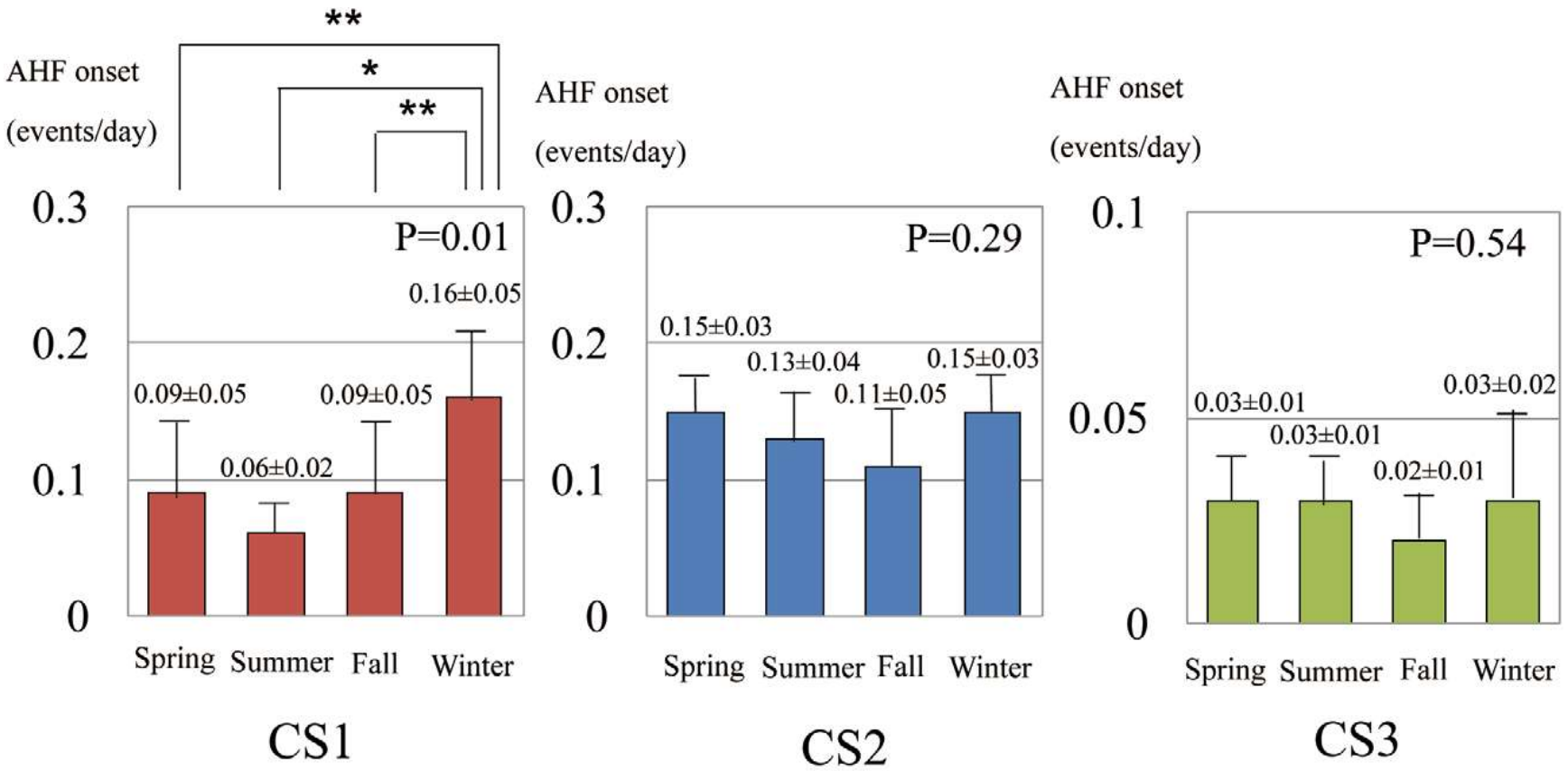
Follath et al Intensive Care Medicine 2011

ALARM-HF: In hospital mortality of acute heart failure in ICU or CCU hospitalization (n=4953)



Follath et al Intensive Care Medicine 2011

Clinical Scenario 1 Is Associated With Winter Onset of AHF



Hirai M et al, Circ J in press 2015

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

RELAX trial: patients have preserved CI but a high RAP and very high PCWP

Pulmonary edema

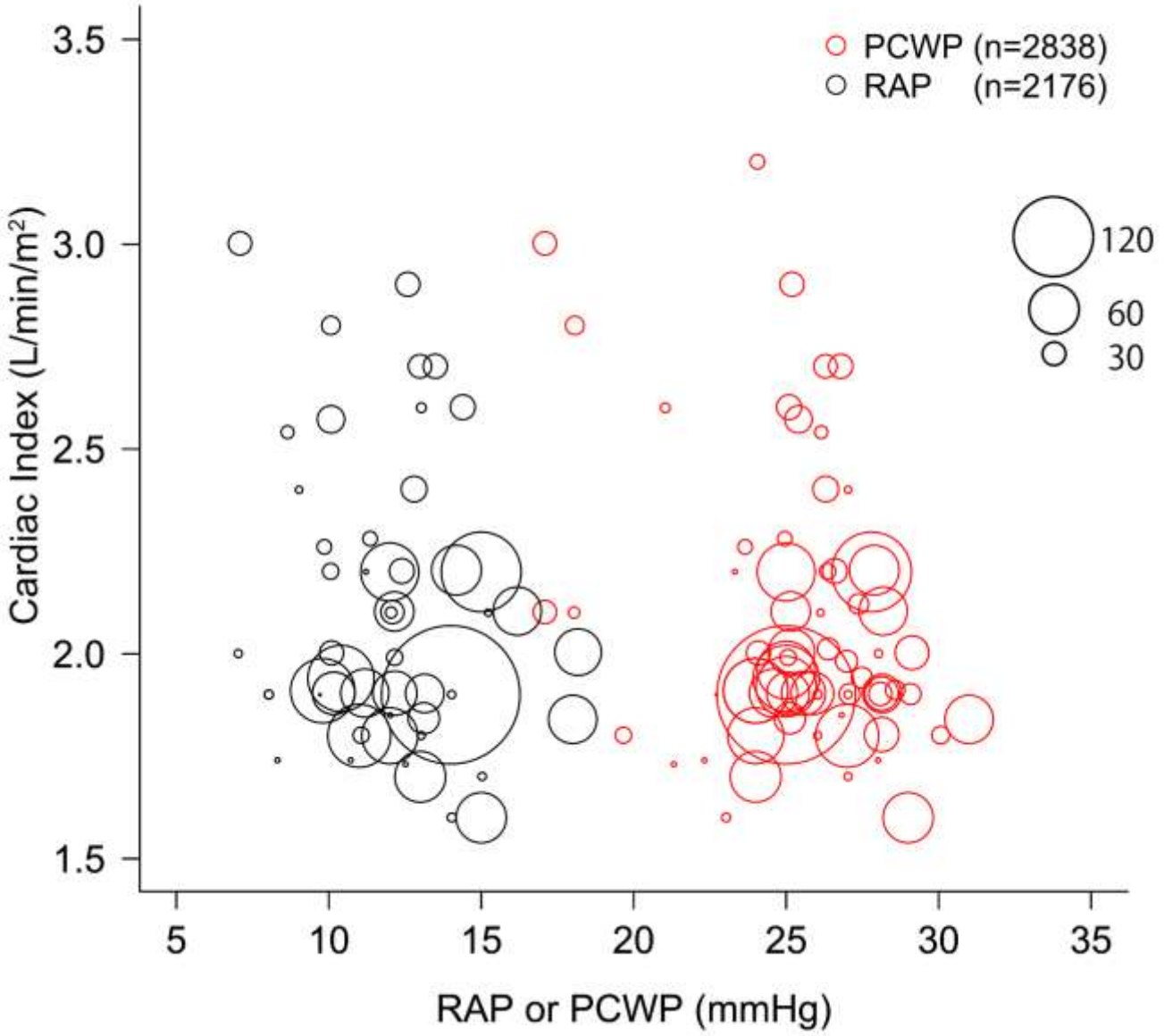
Baseline haemodynamic parameters[d]	Normal values	Serelaxin (n=34)	Placebo (n=37)
PCWP (mmHg)	**3 - 5**	26.2 (5.9)	26.5 (5.2)
CI (L/min/m^2)	**2.5 – 3.0**	2.4 (0.7)	2.2 (0.6)
Systolic PAP (mmHg)		56.1 (13.0)	58.0 (13.8)
Diastolic PAP (mmHg)		27.3 (6.2)	28.8 (6.9)
Mean PAP (mmHg)		36.9 (7.9)	38.5 (8.1)
RAP (mmHg)	**0 - 2**	12.7 (5.9)	12.3 (5.5)
SVR (dynes × s/cm^5)		1530 (462)	1720 (607)
PVR (dynes × s/cm^5)		210 (161)	243 (166)

Kidney & liver dysfunction

Ponikowski et al Eur Heart J 2014 in press

Acute heart failure = Right & Left ventricular failure

Results of meta-analysis



Shiro Ishohara et al.

EUROPEAN SOCIETY OF CARDIOLOGY®

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

CLINICAL RESEARCH
Heart failure/cardiomyopath

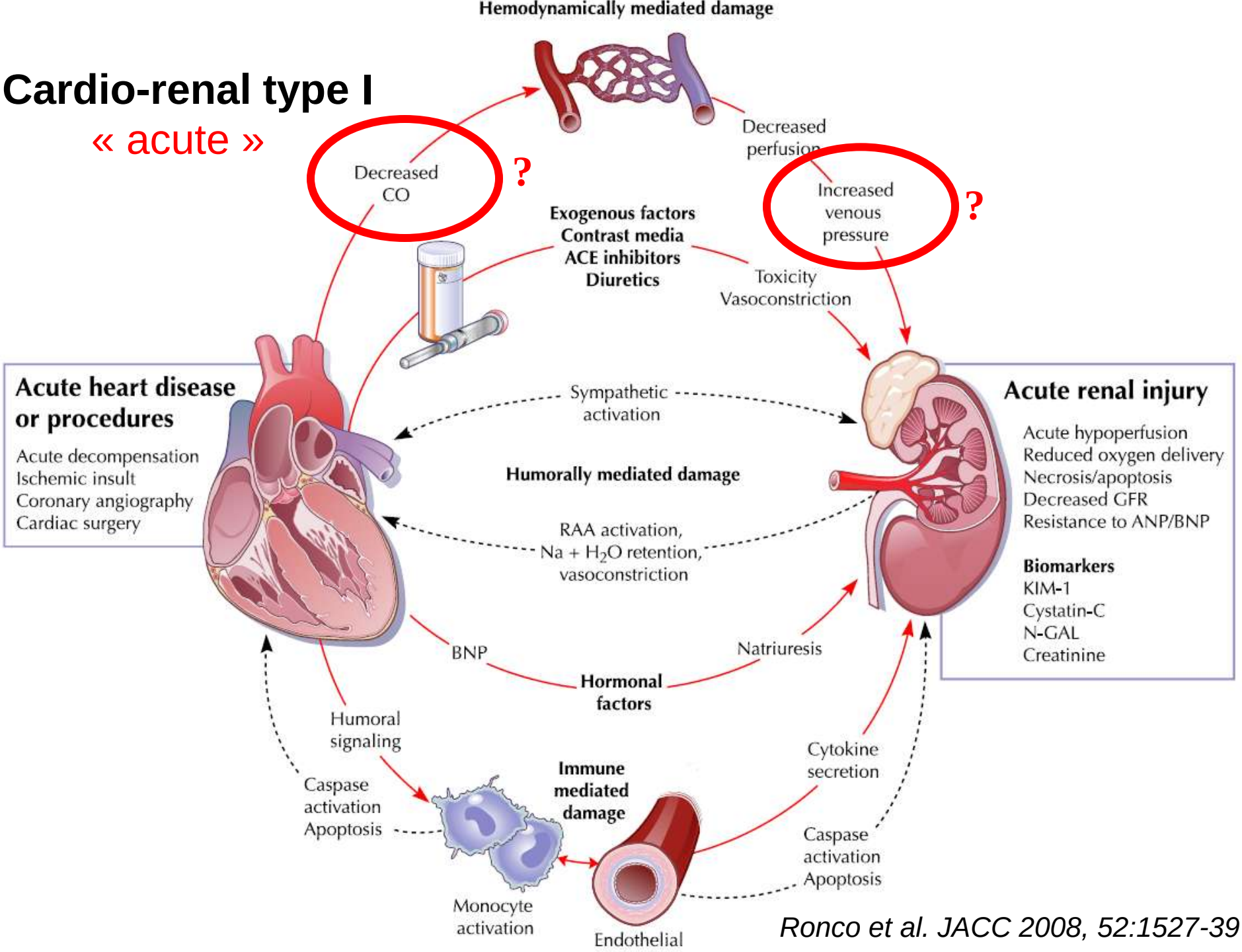
Cardio-renal syndromes: report from the consensus conference of the Acute Dialysis Quality Initiative

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

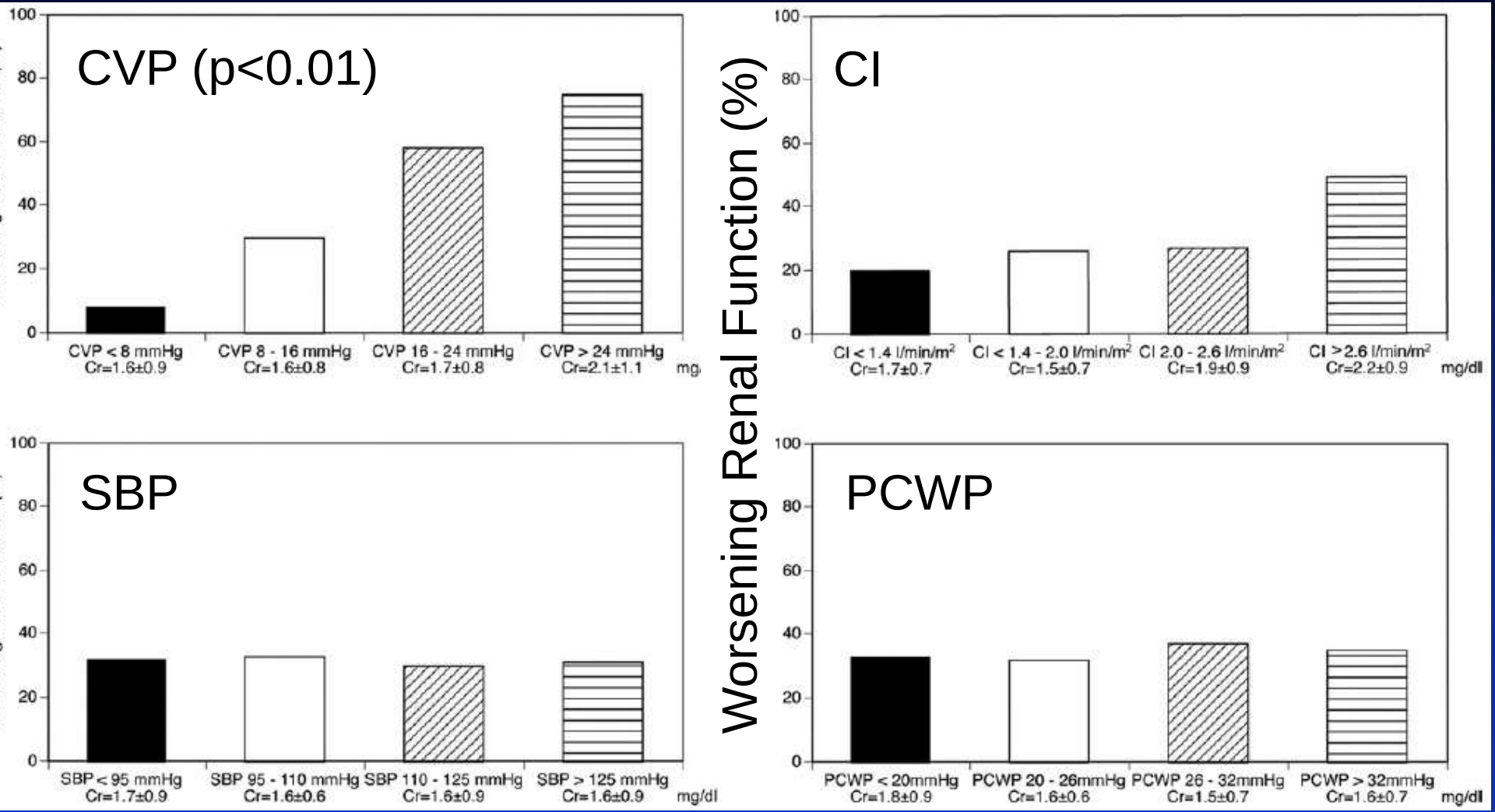
Cardio-renal type I

« acute »



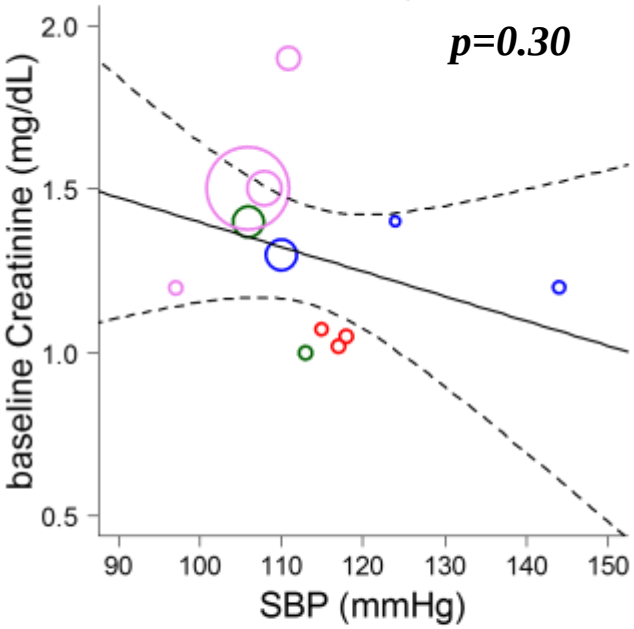
Ronco et al. JACC 2008, 52:1527-39

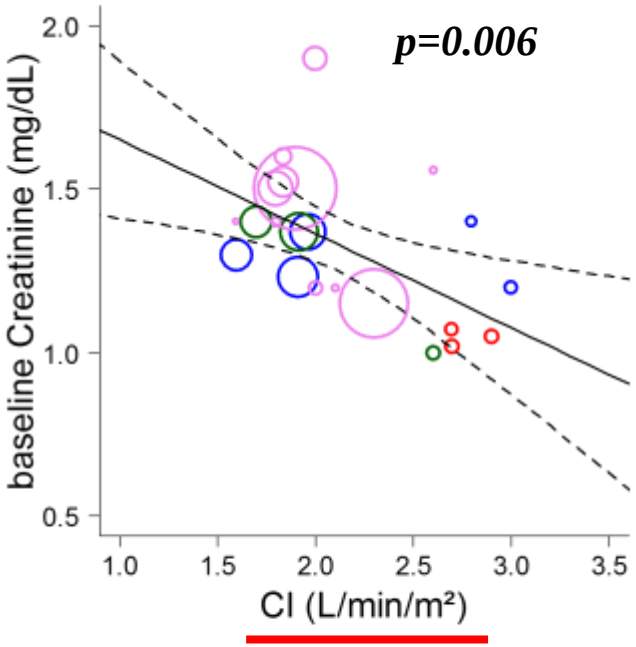
Effects of CVP, CI, SBP and PcwP on worsening renal function in Acute Heart Failure patients

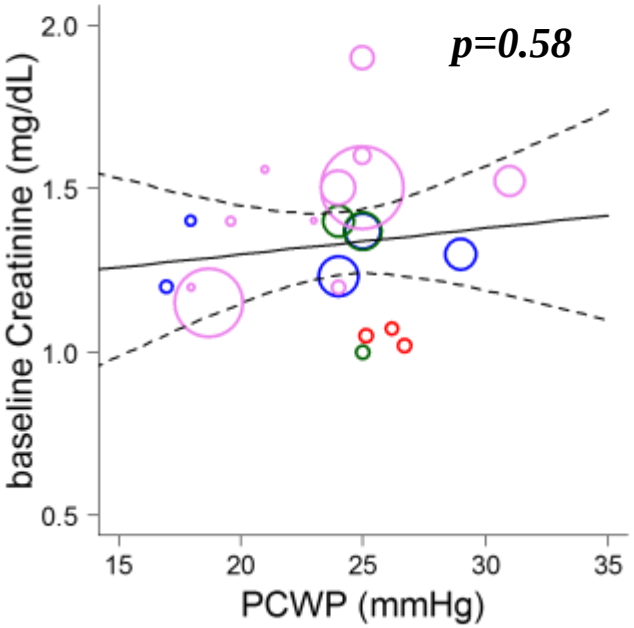


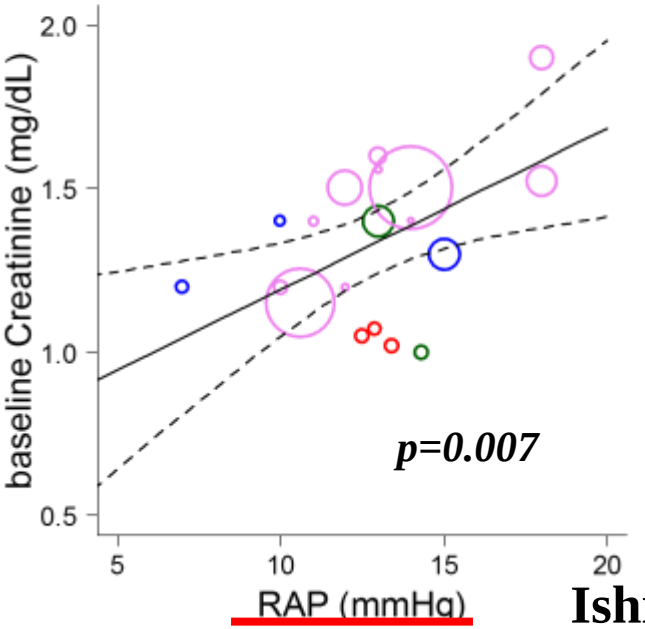
Mullens et al. JACC 2009, 53:589-596

Association between baseline creatinine level and invasive hemodynamics in AHF: results of a meta-analysis









Ishihara S et al.

European Heart Journal Advance Access published October 22, 2012

European Heart Journal
doi:10.1093/eurheartj/ehs332

CLINICAL RESEARCH

Liver function abnormalities, clinical profile, and outcome in acute decompensated heart failure

Maria Nikolaou[1,2,3], John Parissis[3], M. Birhan Yilmaz[1,15], Marie-France Seronde[1,2,4], Matti Kivikko[5,6], Said Laribi[1,2,7], Catherine Paugam-Burtz[2,8], Danlin Cai[9], Pasi Pohjanjousi[6], Pierre-François Laterre[10], Nicolas Deye[1,11], Pentti Poder[12], Alain Cohen Solal[1,2,13], and Alexandre Mebazaa[1,2,14]*

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Received 14 March 2012; revised 21 August 2012; accepted 12 September 2012

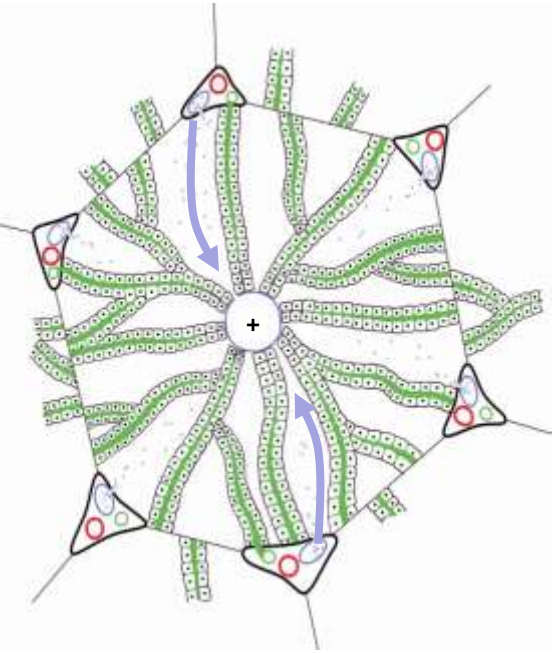
Liver dysfunction in AHF:

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 2013

Normal liver lobule

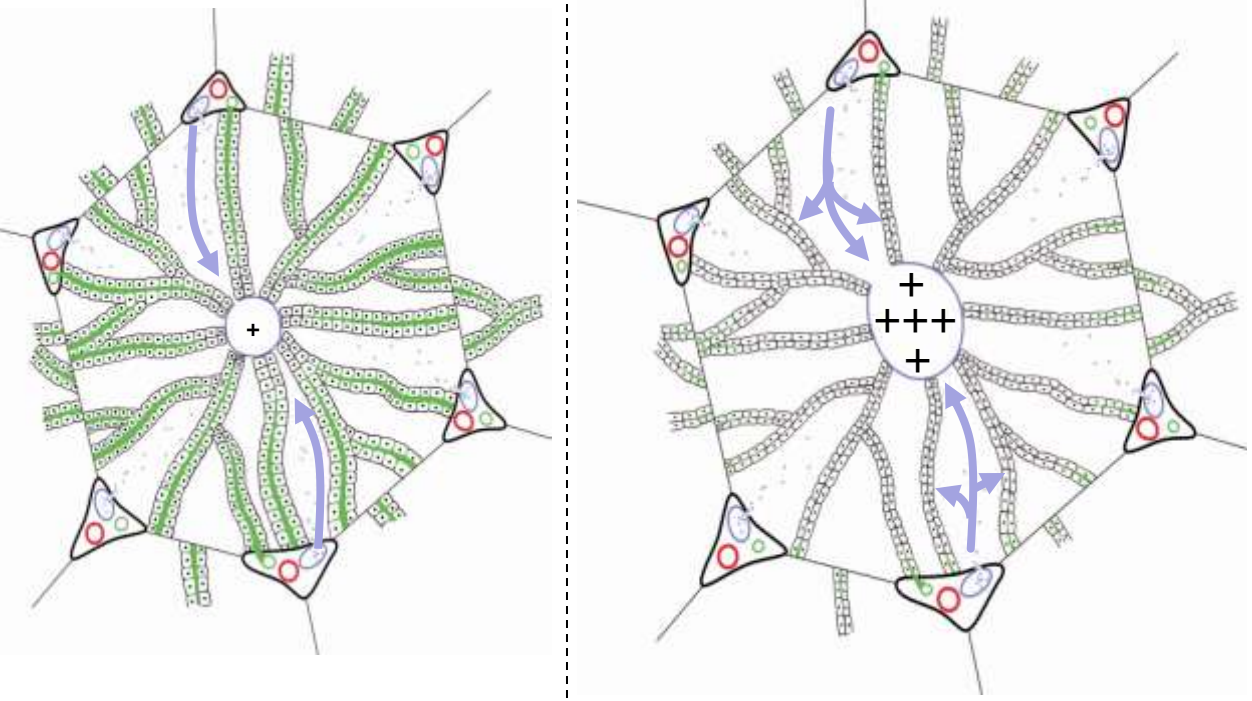


Nikolaou et al Eur Heart Journal 2013

AHF-induced liver congestion
(increased BNP)

Normal liver lobule

bile duct compression
(increased AP)



Nikolaou et al Eur Heart Journal 2013

What is the **safety/efficacy ratio** for diuretics, vasodilators, catecholamines, vasopressors ?

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)

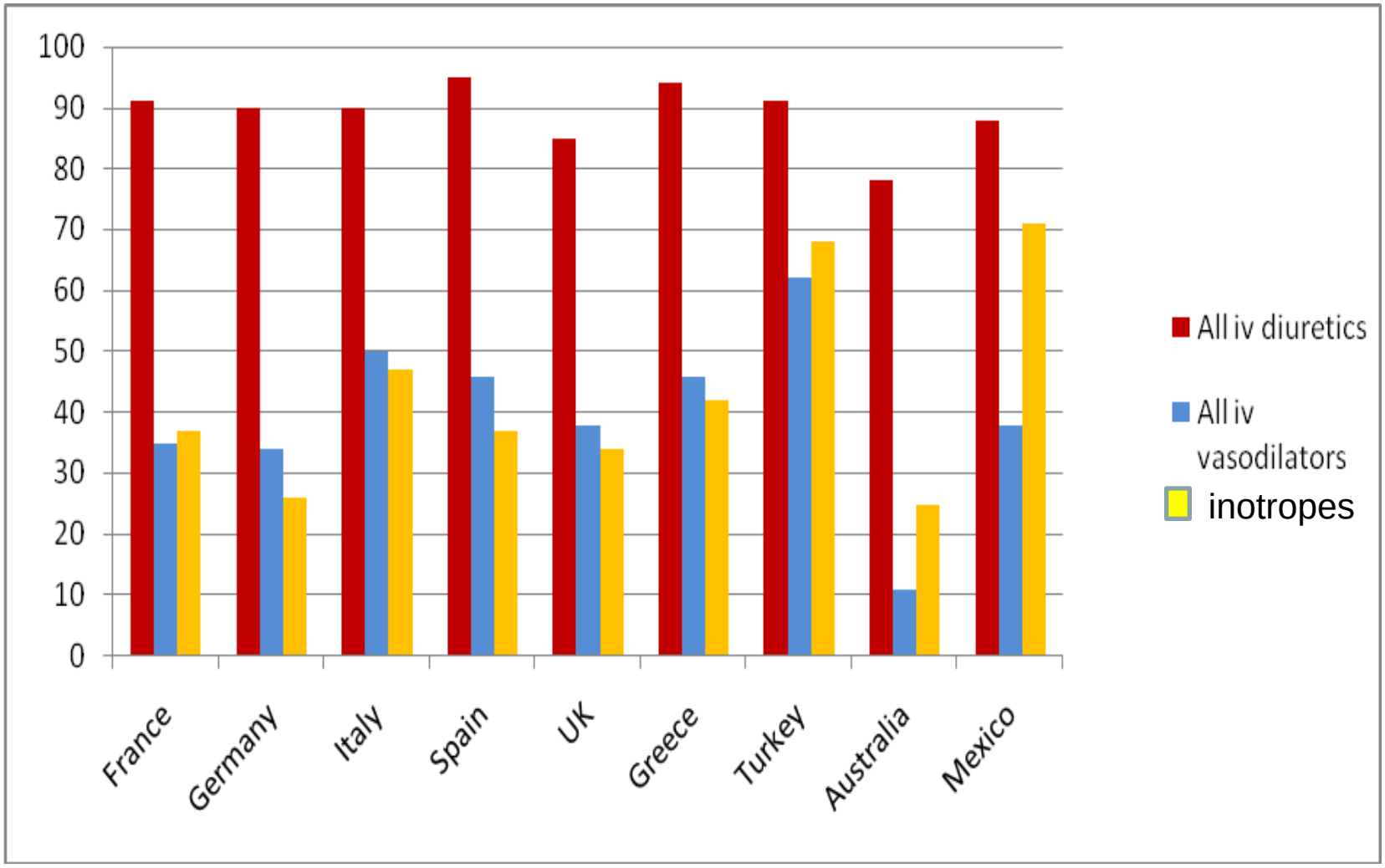
Intensive Care Med (2011) 37:290–301
DOI 10.1007/s00134-010-2073-4

ORIGINAL

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

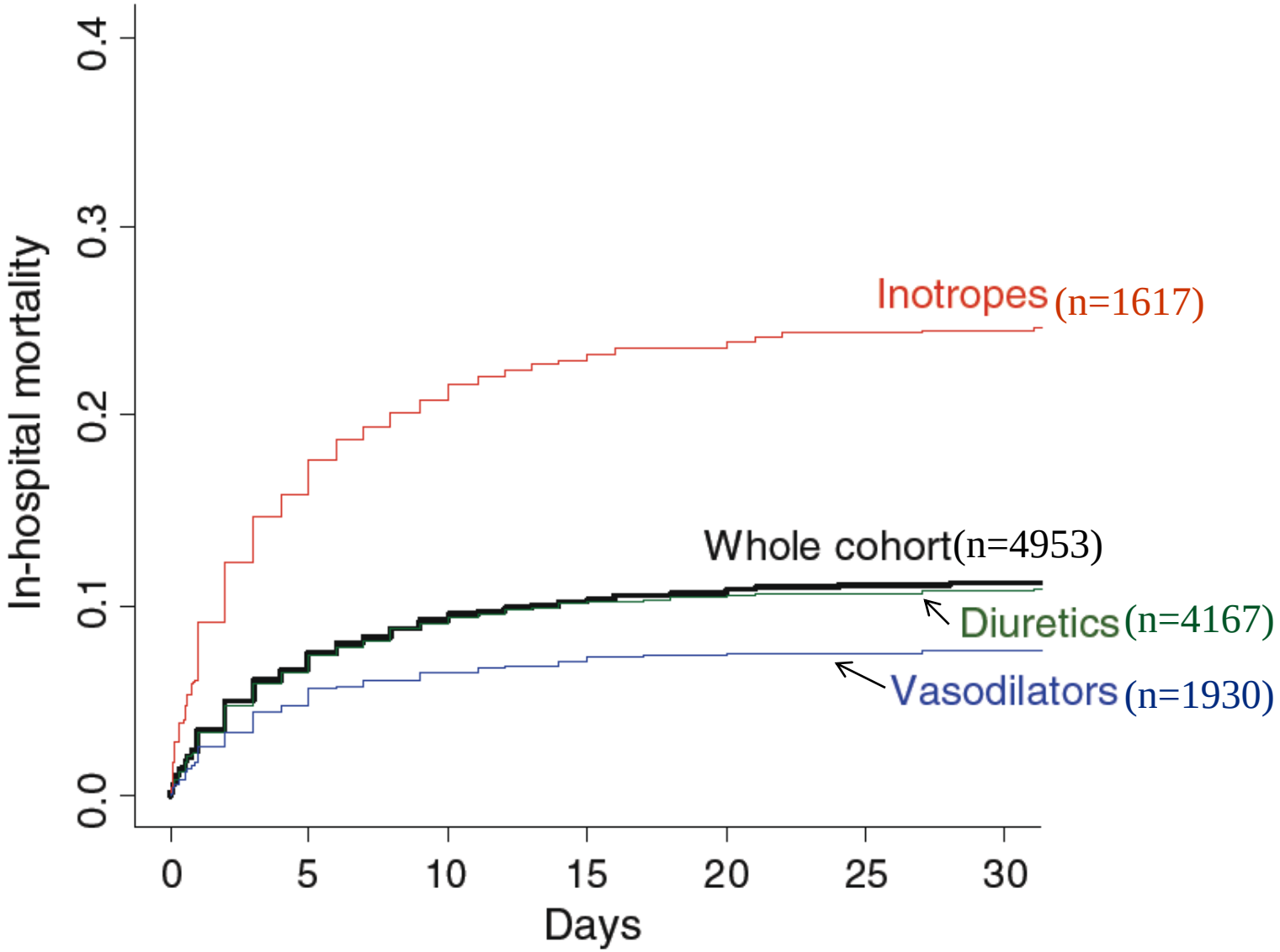
Short-term survival by treatment among patients hospitalized with acute heart failure: the global ALARM-HF registry using propensity scoring methods

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

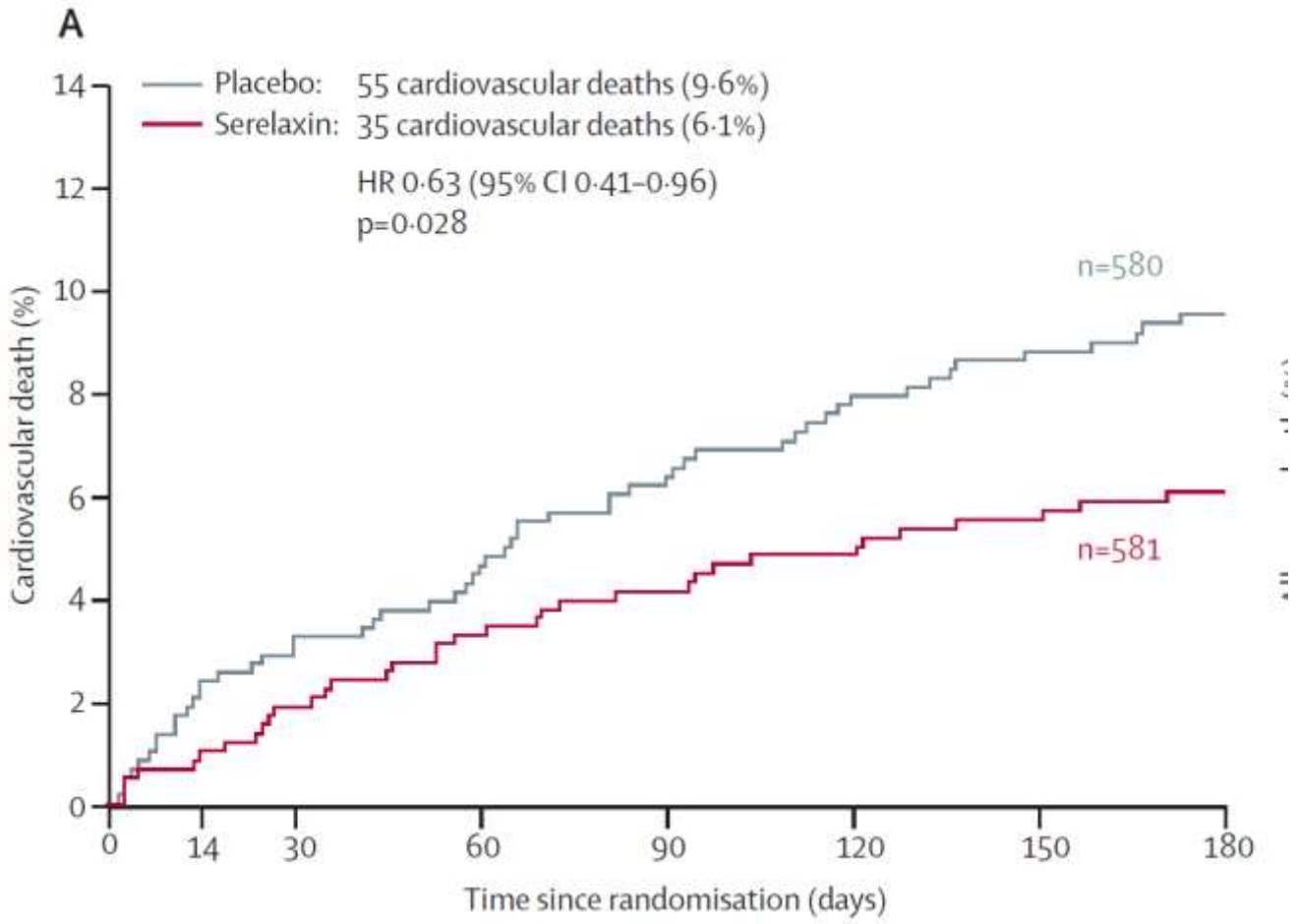


Two large trials to assess beneficial effects of vasodilators in AHF

- TRUE-AHF: ularitide administered within 12 hours after admission

- RELAX-2: serelaxin in AHF

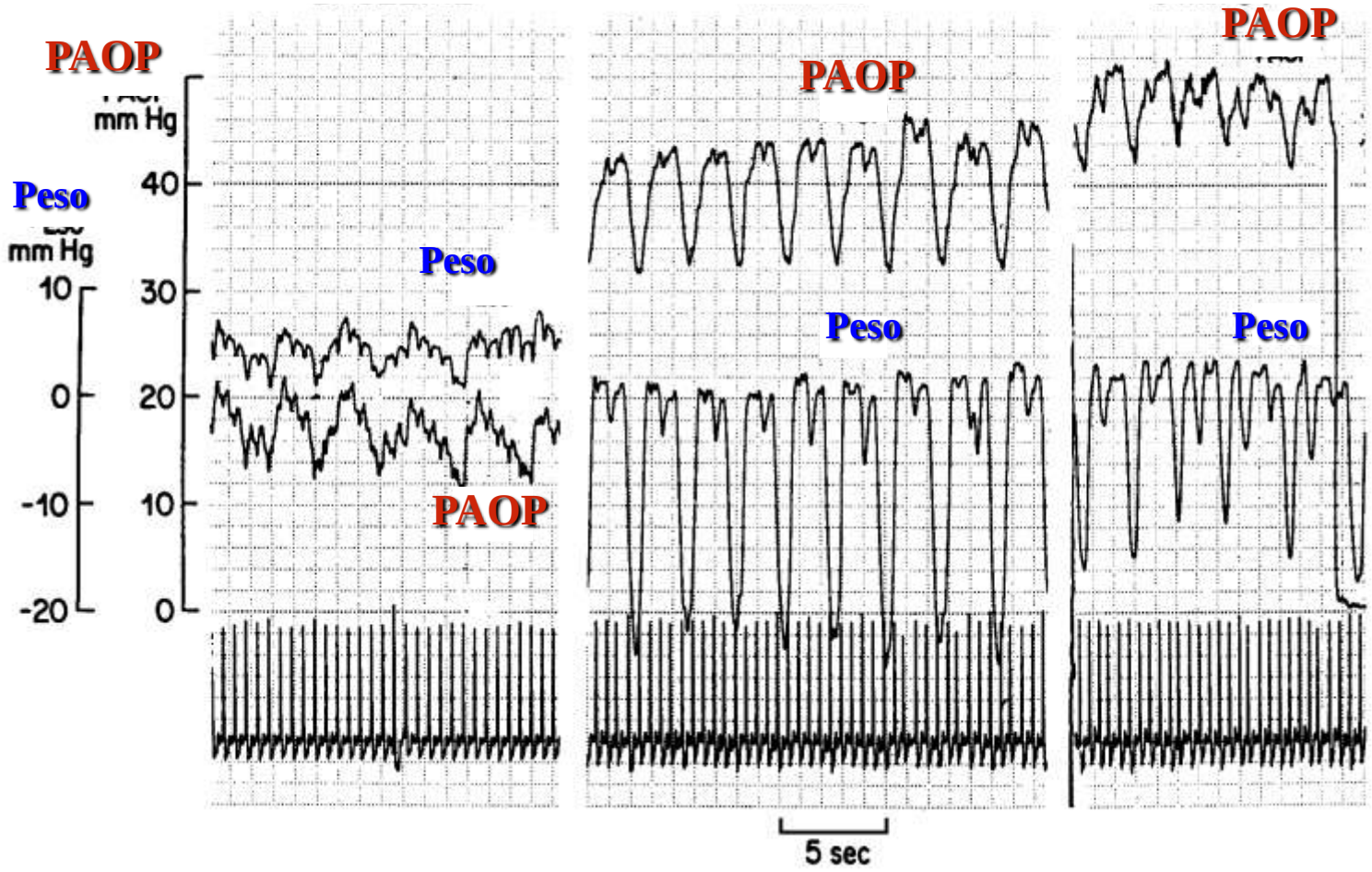
Improvement in 180-d cardiovascular mortality



Teerlink et al, Lancet 2012

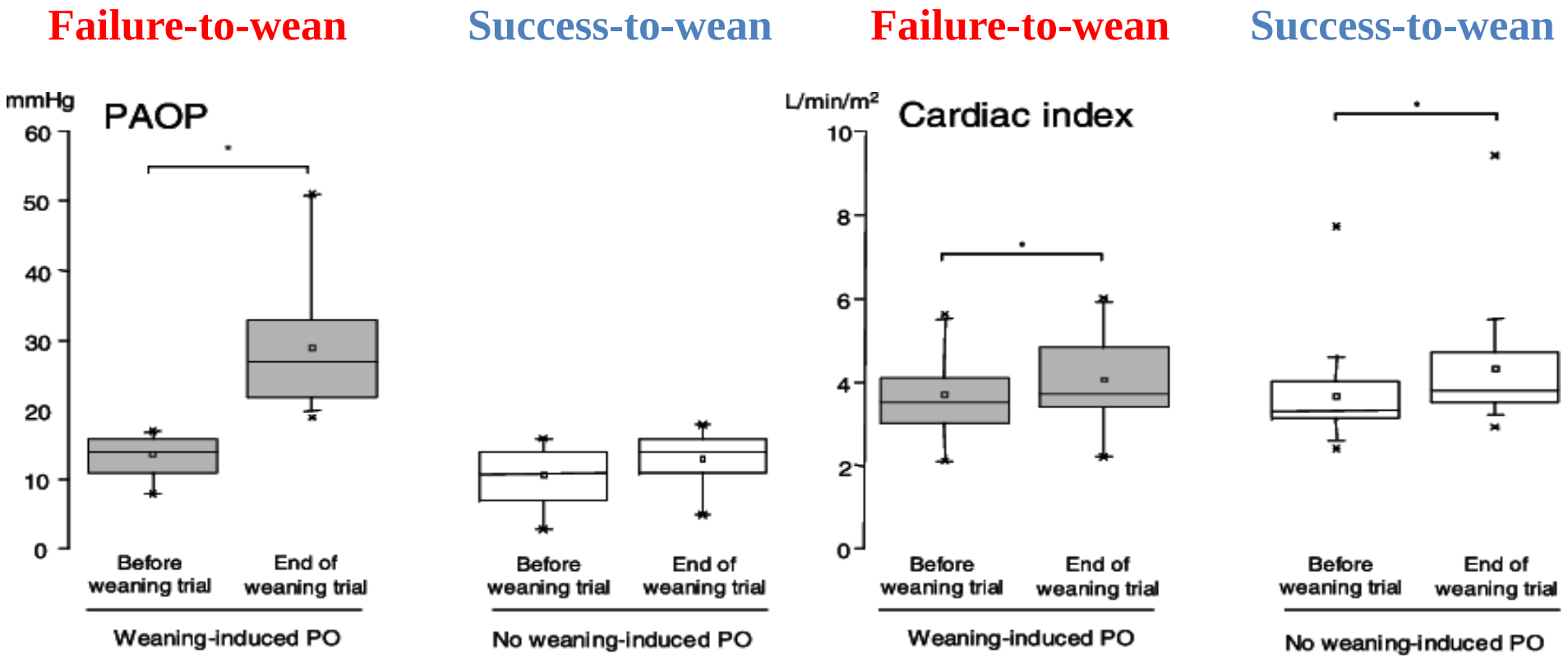
Beijing …….

Acute LV dysfunction during failure-to-wean COPD



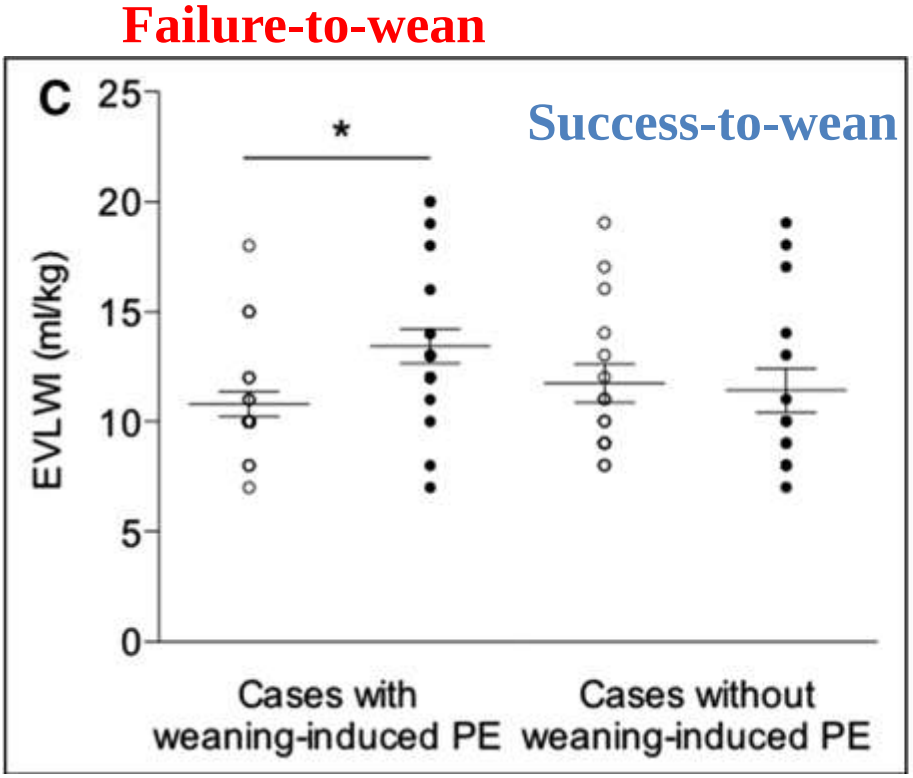
Lemaire F, Teboul JL, Cinotti L et al. *Anesthesiology 1988 ; 69 : 171-9*

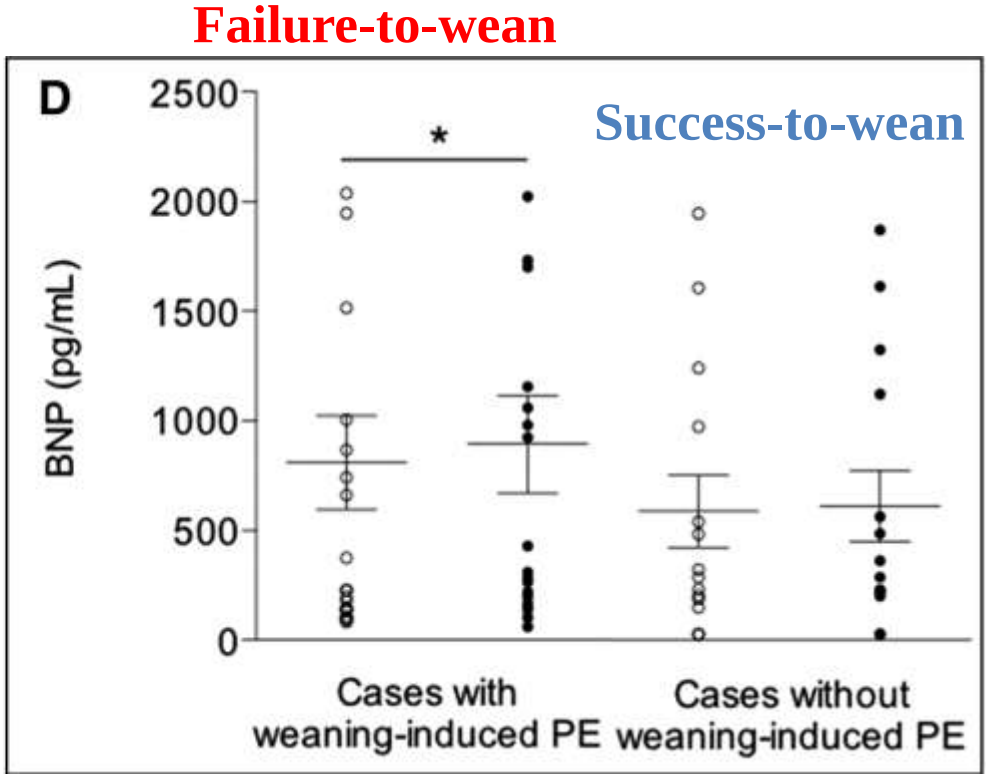
Failure-to-wean in COPD is related to congestion rather low cardiac output



Anguel et al. Increase in plasma protein concentration for diagnosis weaning-induced pulmonary oedema. Intensive Care Medicine 2008

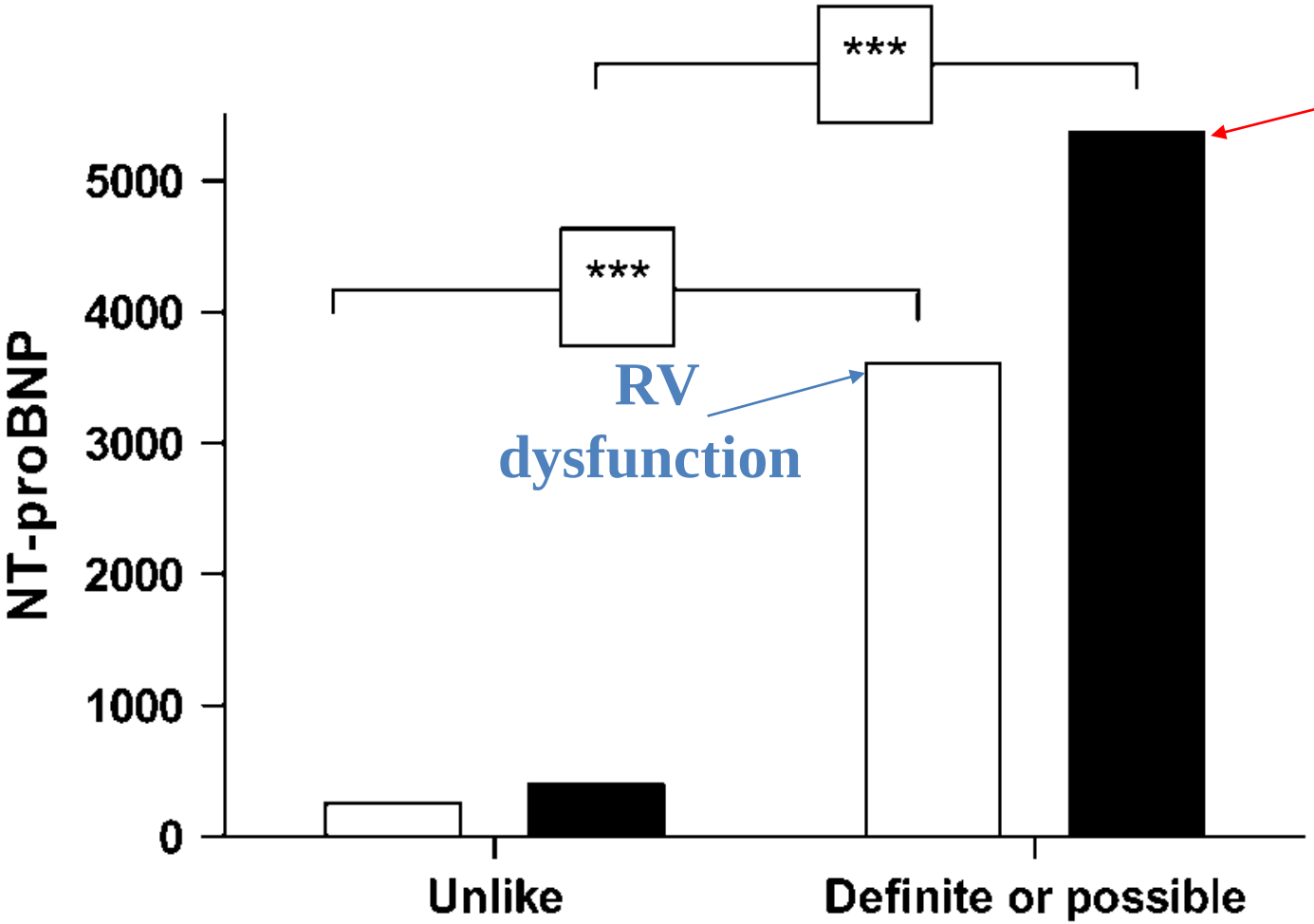
Failure-to-wean is associated with increase EVLW and plasma BNP





Dres et al, Crit Care Med 2014

Plasma levels of NT-proBNP associated to the level of heart dysfunction



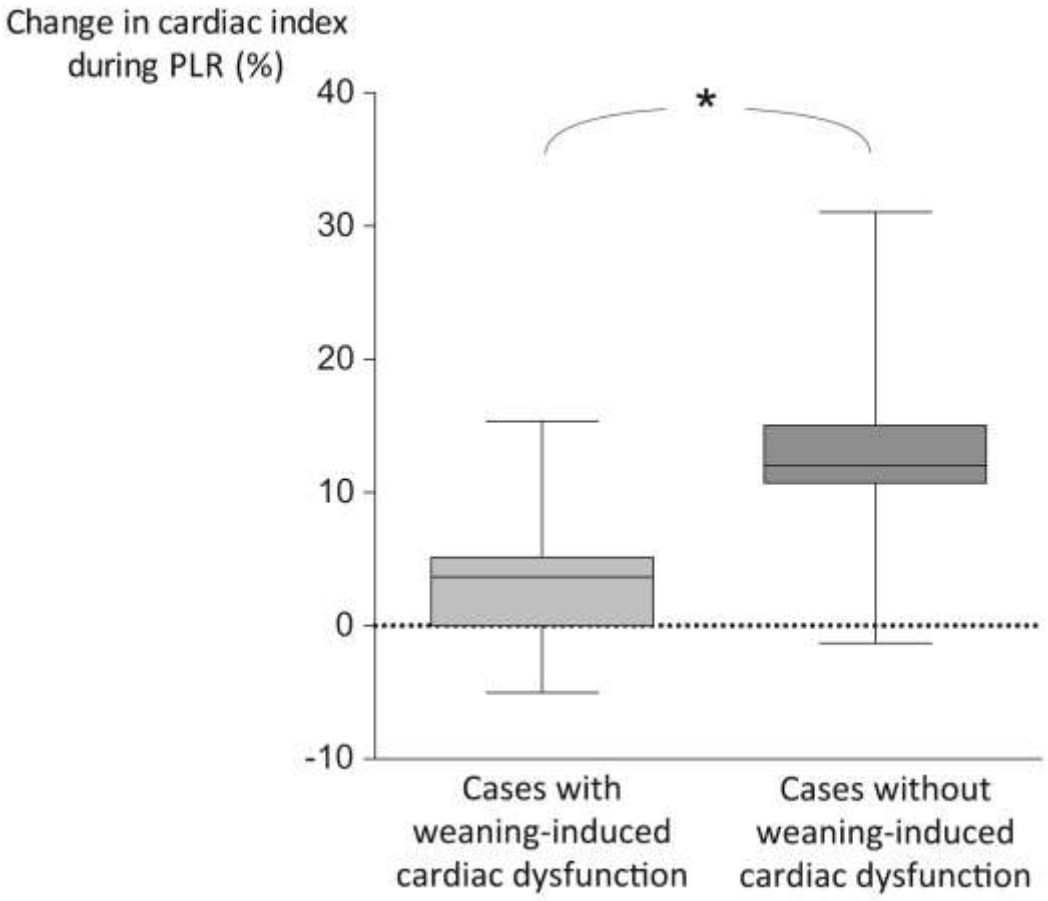
Abroug et al AJRCCM 2006

Intensive Care Med
DOI 10.1007/s00134-015-3653-0

ORIGINAL

Martin Dres
Jean-Louis Teboul
Nadia Anguel
Laurent Guerin
Christian Richard
Xavier Monnet

Passive leg raising performed before a spontaneous breathing trial predicts weaning-induced cardiac dysfunction

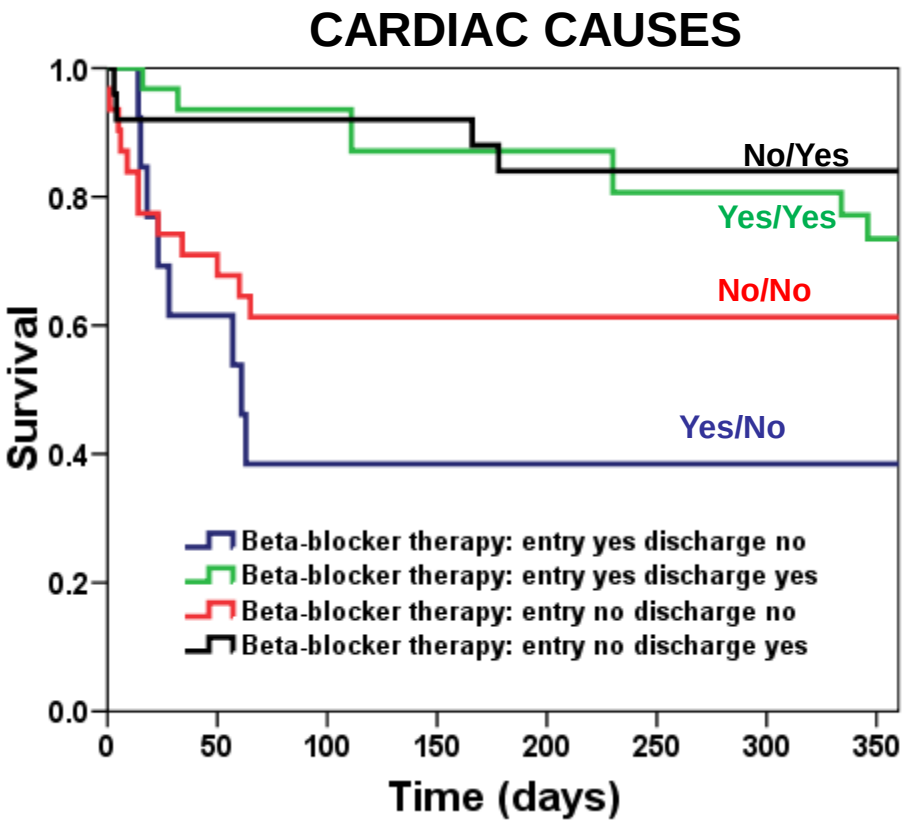


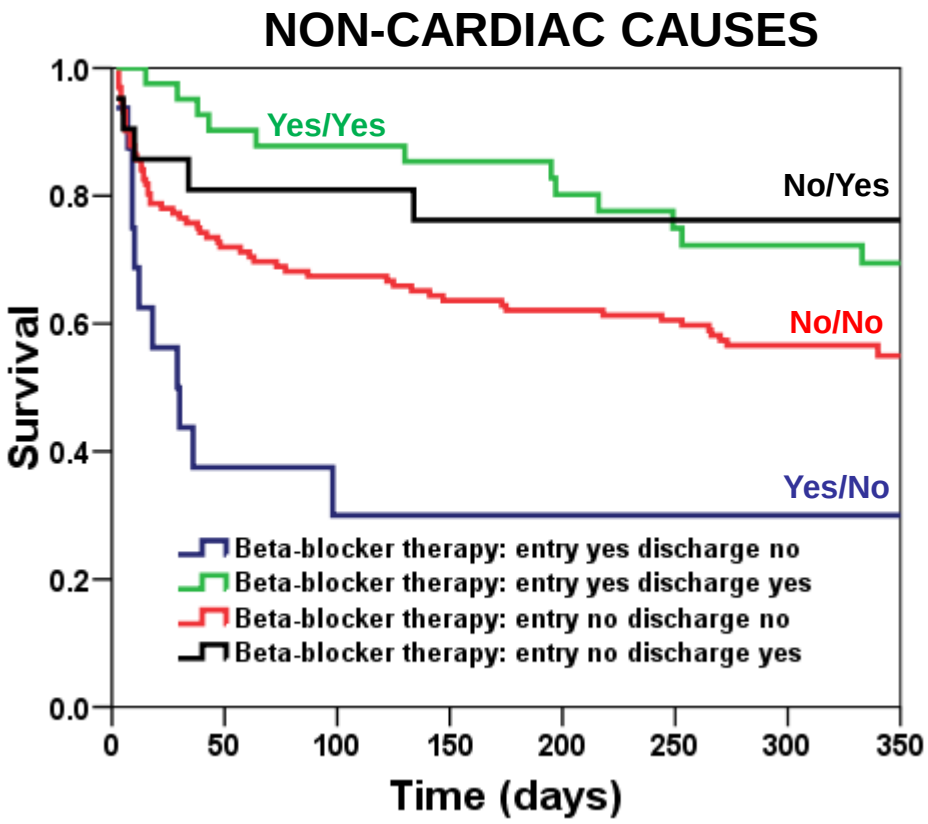
Dres et al, Intensive Care Med 2015

浅草寺

Pre-discharge therapy determines long term outcome

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





Noveanu et al Crit Care 2011

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Que retenir ?

European Heart Journal
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CURRENT OPINION

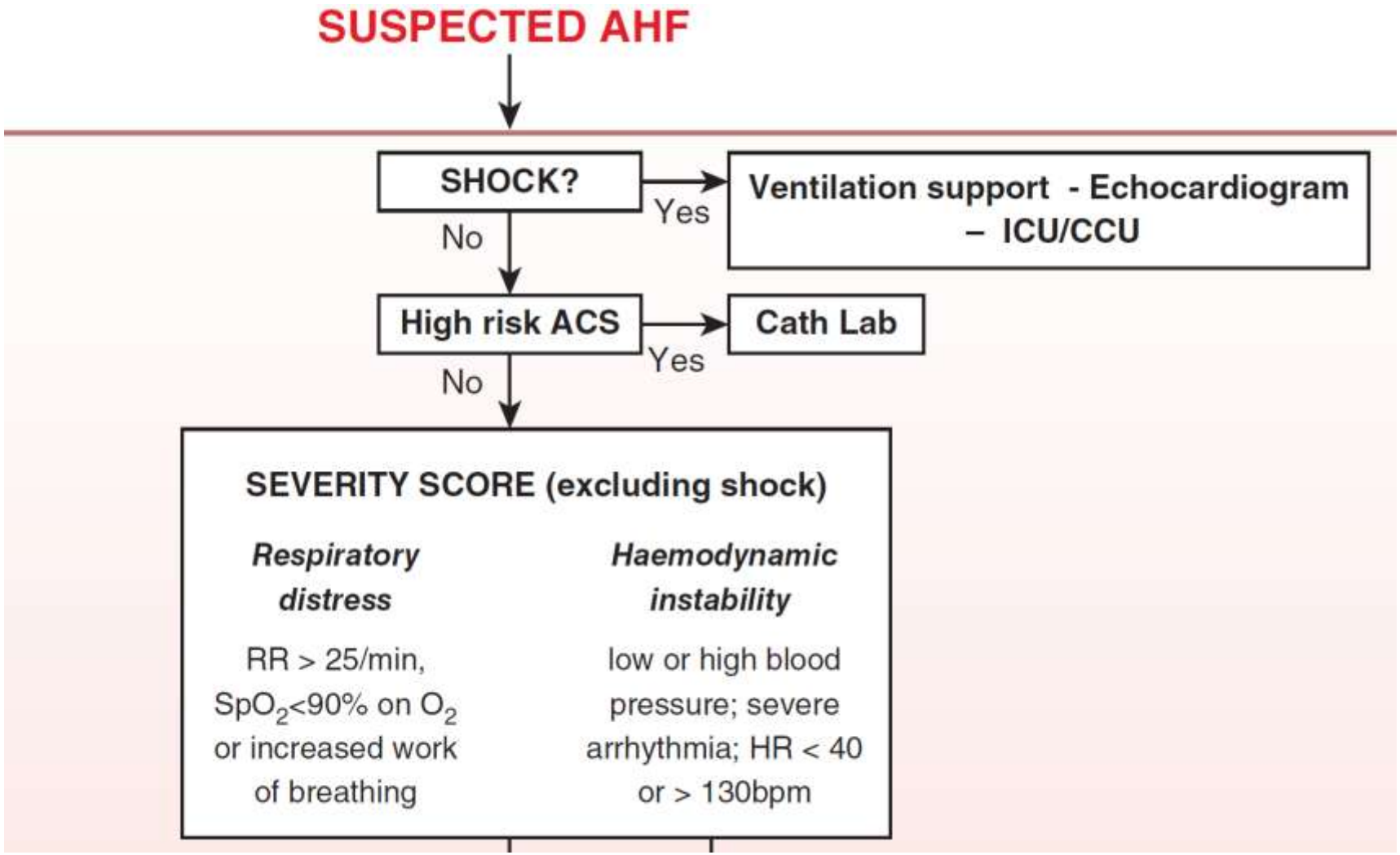
Online, free access

Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine – short version

Alexandre Mebazaa[1]*, M. Birhan Yilmaz[2], Phillip Levy[3], Piotr Ponikowski[4], W. Frank Peacock[5], Said Laribi[6], Arsen D. Ristic[7], Ekaterini Lambrinou[8], Josep Masip[9], Jillian P. Riley[10], Theresa McDonagh[11], Christian Mueller[12], Christopher deFilippi[13], Veli-Pekka Harjola[14], Holger Thiele[15], Massimo F. Piepoli[16], Marco Metra[17], Aldo Maggioni[18], John J.V. McMurray[19], Kenneth Dickstein[20], Kevin Damman[21], Petar M. Seferovic[22,23], Frank Ruschitzka[24], Adelino F. Leite-Moreira[25,26], Abdelouahab Bellou[27,28], Stefan D. Anker[29,30], and Gerasimos Filippatos[31]

HFA/EUSEM/SAEM Eur Heart Journal 2015

Que faire dans les premières 30-60 min (1)



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Que faire dans les premières 30-60 min (2)

SEVERITY SCORE

Yes

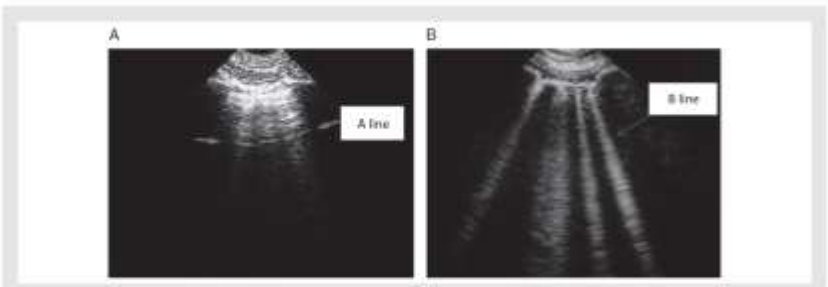
RESUSCITATION AREA/CCU/ICU
To stabilize vital signs (echo if needed) and/or immediate non-invasive ventilation

No

DIAGNOSTIC TESTS
- ECG,
- Lab tests (refer to text)
- Echo (lung, heart)
- Chest Xray

IV THERAPY

SBP<110 mmHg	***SBP>110 mmHg***
• Diuretics, 1st line therapy	• Vasodilators / Diuretics



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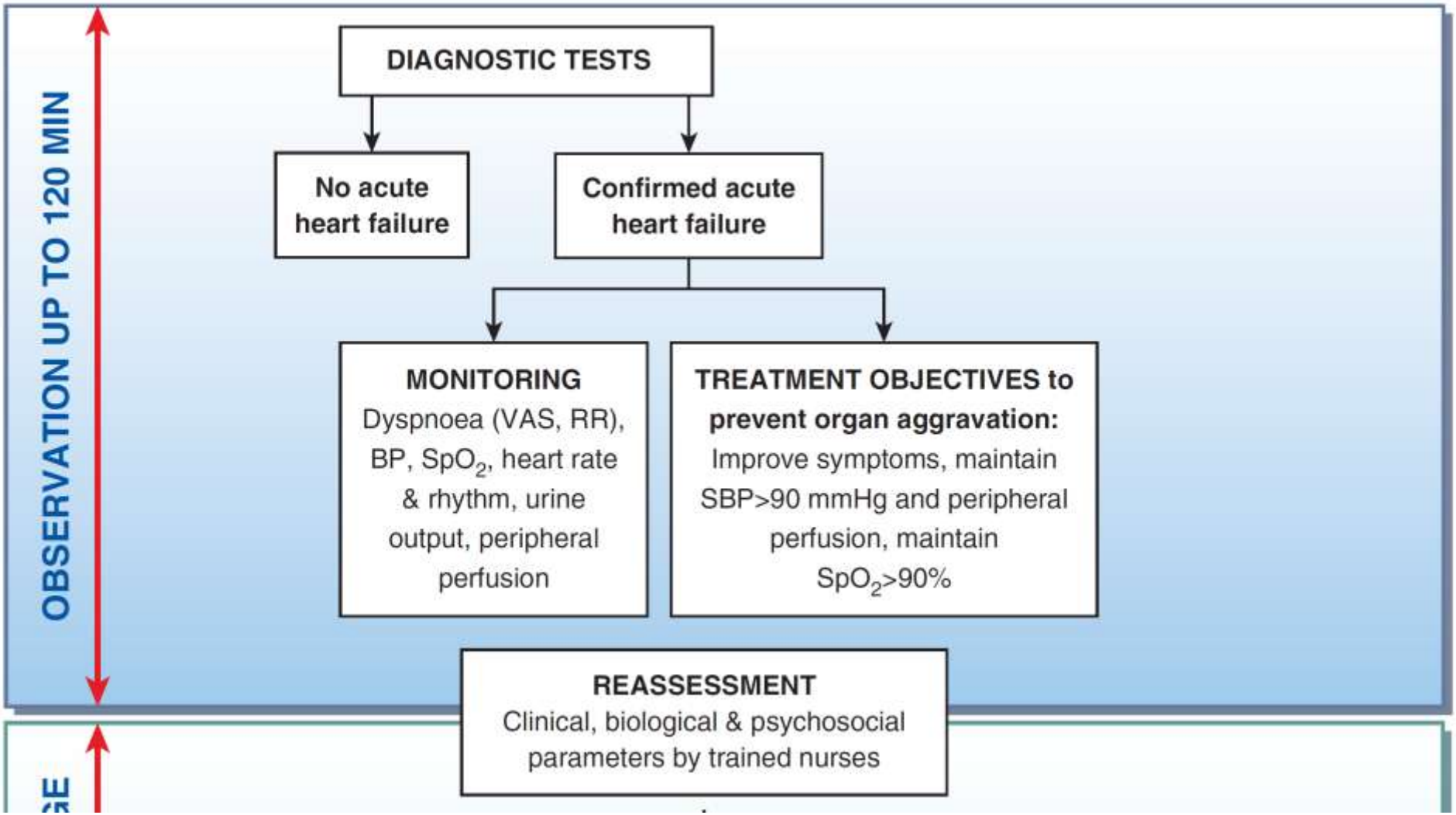
120 min suivantes



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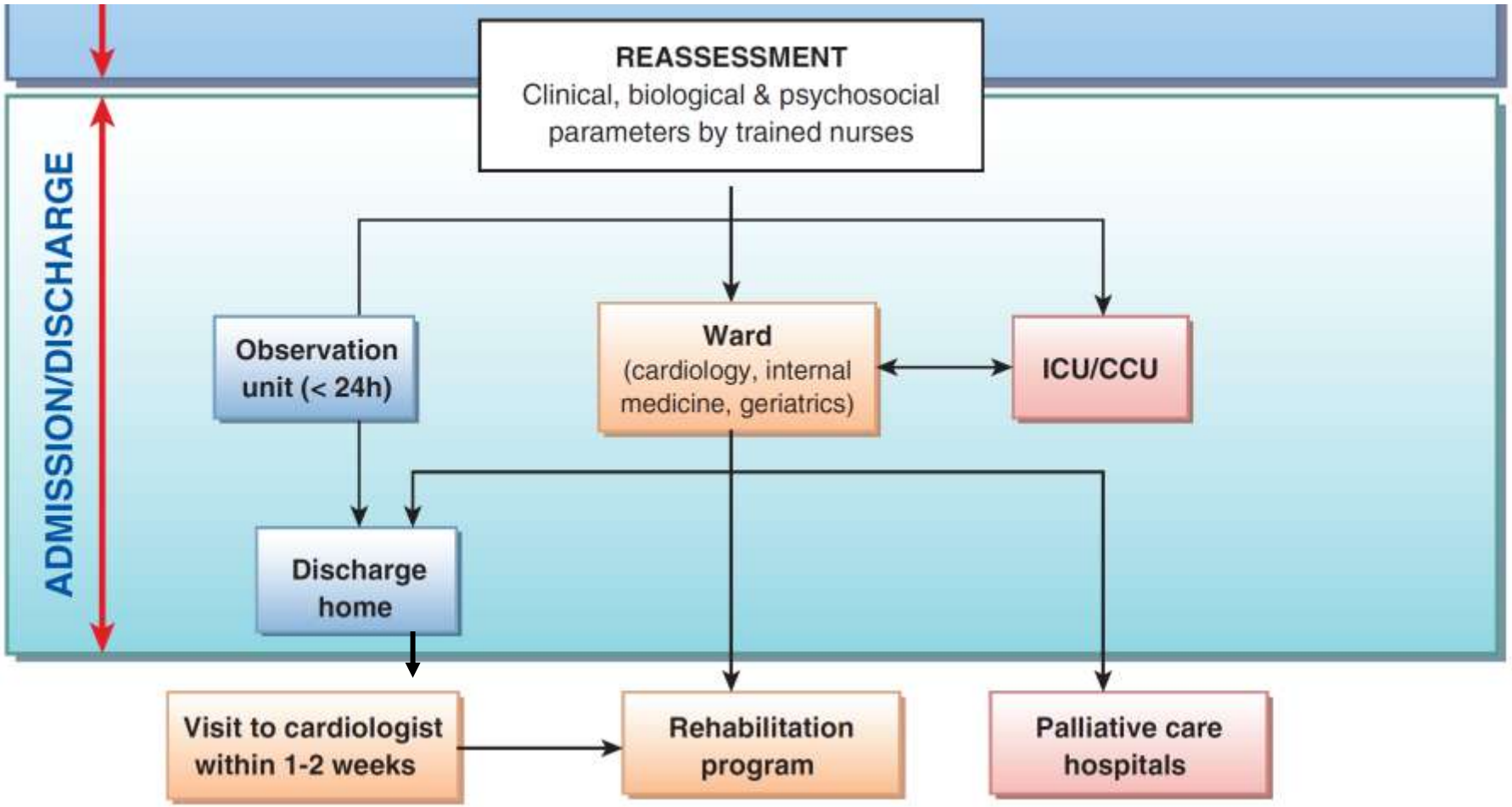
Suivi hospitalier et critères de sortie



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Principaux messages

- **ICA sans choc :**
  - **la congestion** est la principale cause d'admission
  - Les vasodilateurs sont les médicaments de choix
- **Choc cardiogénique**
  - Le bas débit cardiaque et l'ischémie myocardique sont en première ligne
  - Privilégier NA+inotrope; ne pas donner d'adrénaline
- « **le temps est du muscle** »