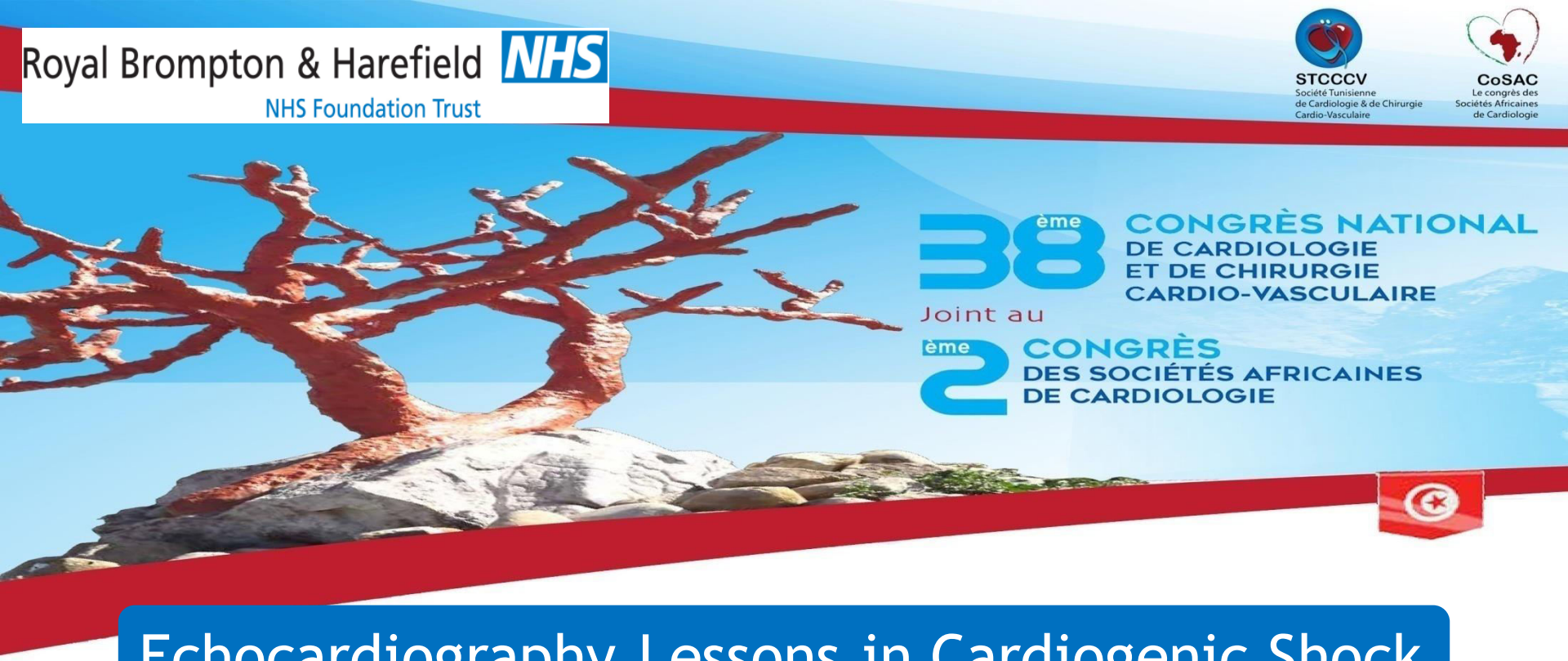


Echocardiography Lessons in Cardiogenic Shock

Dr. Hatem Soliman Aboumarie

MBBS, MRCP, MSc (ICM), PgDip (Cardio), EDICM, ASCeXAM

Senior Clinical Fellow
Adult Intensive Care
Royal Brompton Hospital, London, UK
hatem.soliman@gmail.com
@hatemsoliman

www.stcccv.org

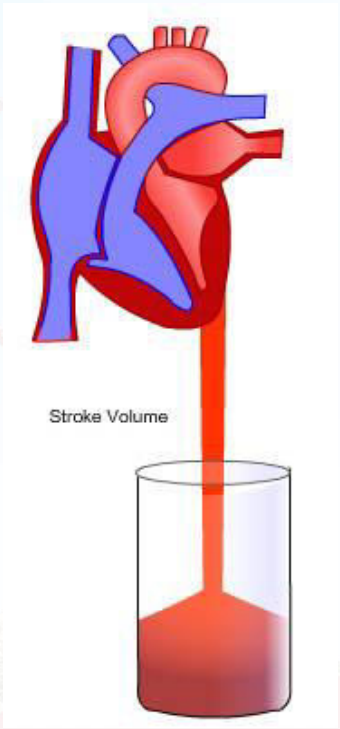
HIT
EACVI's Heart Imagers of Tomorrow

Nothing to disclose

Overview

- Cardiac output estimation
- Monitoring patients on pVA ECMO
- Echo-guided pacing optimization
- Integrated approach
- Take home messages

Cardiac output



SV = LVOT CSA x LVOT VTI

CO = HR x Stroke Volume

Cardiogenic Shock

- Stroke Volume
- LV Systolic Function
- Valvular Pathology
- LV Diastolic Function

Stroke Volume

(LVOT area x LVOT VTI)
VTI = Velocity Time Integral

1. Parasternal long axis view

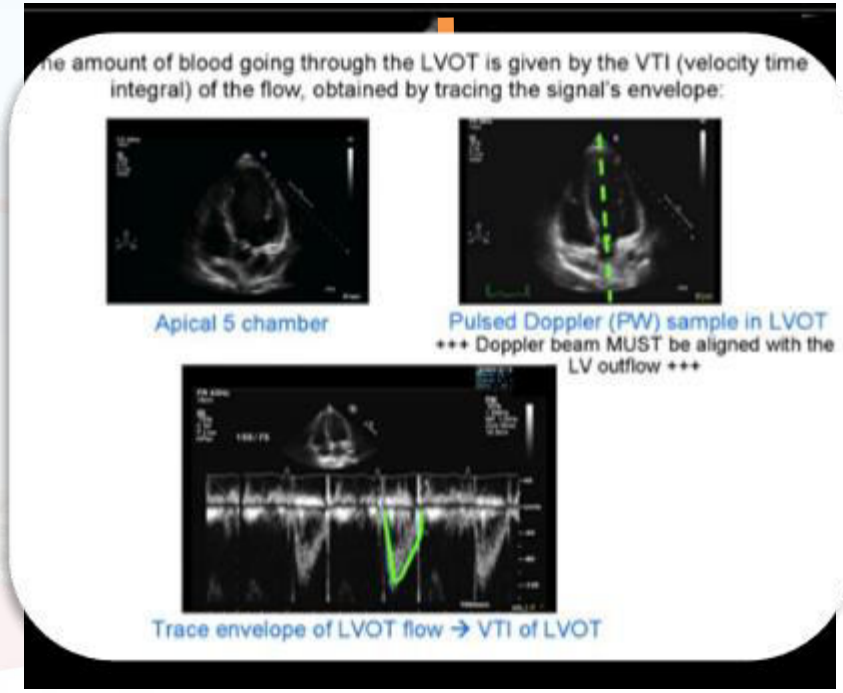
2. Zoom on LVOT

++ good visualization of LVOT and of long axis of the aortic root ++

3. Measure LVOT diameter

++ Cusps insertion on aortic annulus, one or two frames after maximal systolic leaflet separation (**systole**) ++

LVOT Area = 3.14 x (1/2 LVOT diameter)^2



Pearls

The LVOT VTI a surrogate for the stroke volume

Normal value >20 cm

Record the measured LVOT area in the pt. records

Average of 5-10 beats in AF

Pitfalls

SV variations are exaggerated with:

- Hypovolaemia
- Larger Tidal Volumes
- Cardiac tamponade

Presumes LVOT is circular. It isn't!

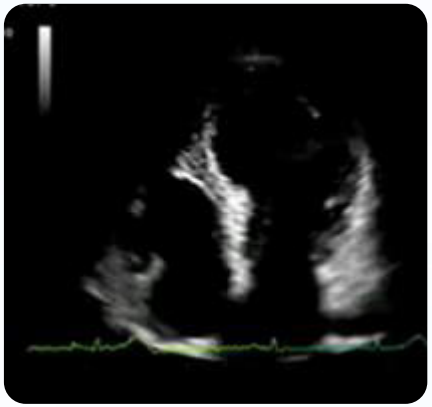
Non-alignment of Doppler beam: VTI will be underestimated

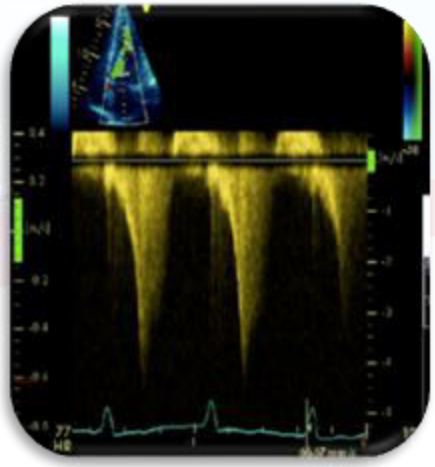
Error in LVOT diameter will be squared

Dynamic LVOT Obstruction

Echo Findings

- Typical with basal septal hypertrophy
- Close approximation of lateral wall and septum
- Systolic anterior motion of the anterior mitral leaflet.
- Dagger-shaped Doppler pattern of LVOT flow





Dynamic LVOT Obstruction

Causes

- Acute MI in the apical and mid segments
- Hypertrophic Cardiomyopathy
- Dobutamine in patients with small LV cavity (concentric LVH)
- Hyperdynamic states (Sepsis, severe anemia)
- Stress Cardiomyopathy (Takatsubo)
- Mitral valve surgery

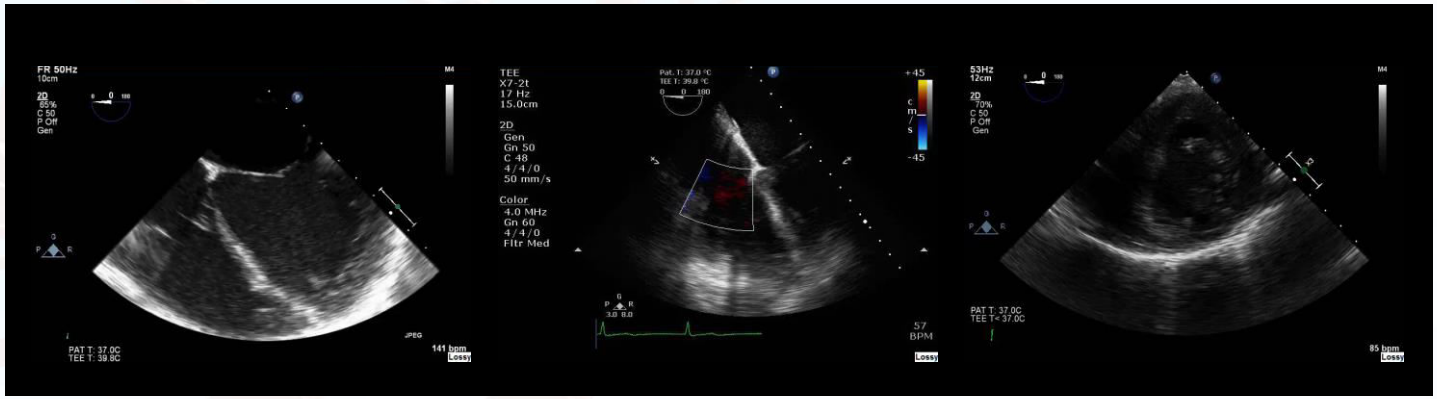
Dynamic LVOT Obstruction

Pitfall

Tachycardia, hypovolemia, and inotropes makes critically ill more prone to it

Decision for MCS

- **Left heart?**
- **Right heart?**
- **Both hearts?**



VA ECMO
(Impella)
(TH)

VA ECMO
Impella RP
Protek Duo
(NovaLung)

VA ECMO

Intensive Care Med (2011) 37:1738–1745
DOI 10.1007/s00134-011-2358-2

ORIGINAL

Nadia Aissaoui
Charles-Edouard Luyt
Pascal Leprince
Jean-Louis Trouillet
Philippe Léger
Alain Pavie
Benoit Diebold
Jean Chastre
Alain Combes

Predictors of successful extracorporeal membrane oxygenation (ECMO) weaning after assistance for refractory cardiogenic shock

Step 1 : The **etiology** of cardiac failure must be compatible with myocardial recovery

Step 2 : **Hemodynamic stability** :
- The patient should have recovered a pulsatile arterial waveform for at least 24 hours
- Baseline MAP > 60 mmHg in the absence or with low doses catecholamine
- The patient should have recovered from major metabolic disturbances

Step 3 : **Pulmonary function** should not be severely impaired
If PaO2/FiO2 <100 mmHg when FiO2 of the ECMO gas flow is set at 21%, consider bridging the patient from VA- to VV-ECMO

Step 4 : The patient **must tolerate a full weaning** trial
** Hemodynamic and echocardiographic assessment whereas ECMO flow is gradually decreased to 66%, and to 33% of its baseline value and then to a minimum of 1–1.5 L/n*

If steps 1, 2, 3 and 4 are validated and the patient has under minimal ECMO support :
- LVEF of ≥ 20–25%, an aortic VTI of ≥ 12 cm and a TDSa ≥ 6 cm/s
- or CI > 2.4 liters/min/m2, PCWP < 18 mmHg and CVP < 18 mmHg

ECMO removal should be considered

Combes 2016

Monitoring of the pt on pVA-ECMO

Underlying LV dysfunction

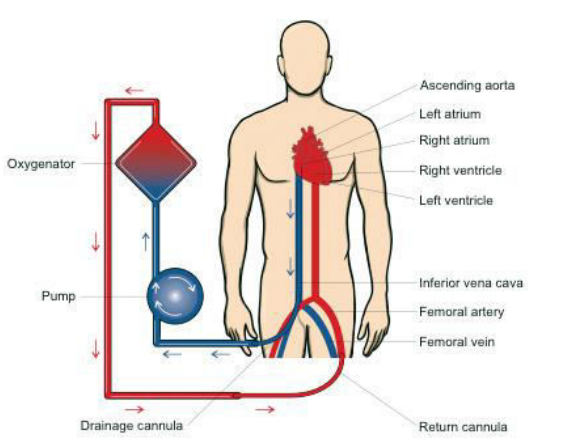
↗ afterload due to retrograde VA ECMO flow

Insufficient unloading of LV

↓

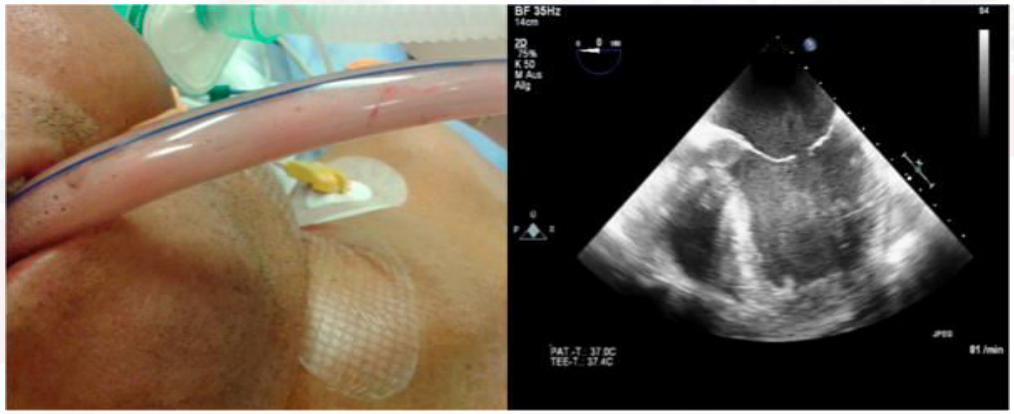
Blood stagnation in LV

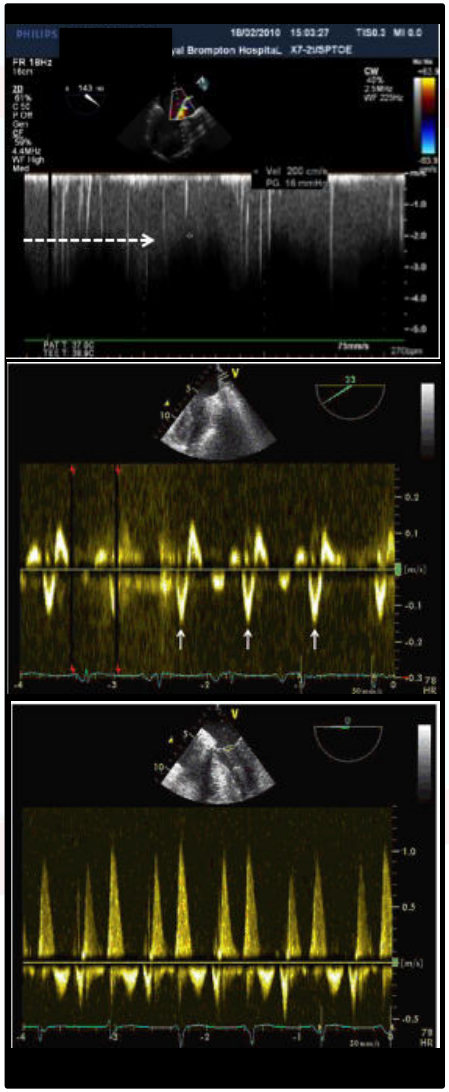
Pulmonary congestion, edema, hemorrhage.



Inadequate LV Decompression

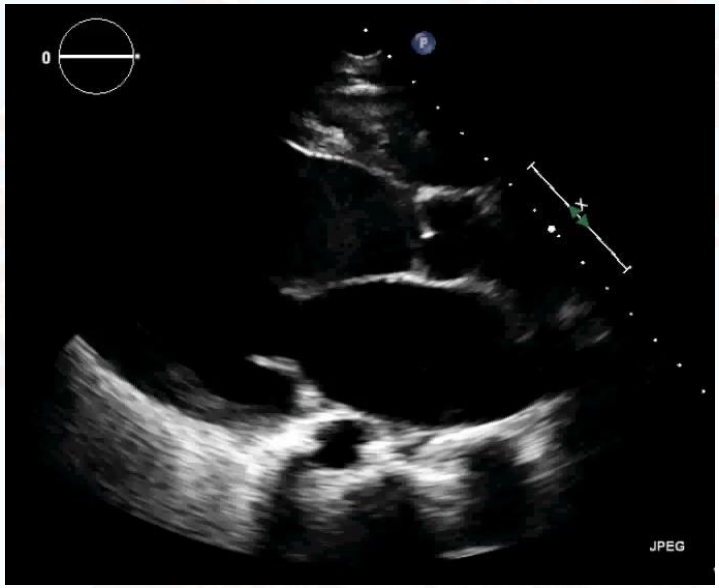
- Progressive ventricular dilatation
- Worsening mitral regurgitation
- Retrograde pulmonary vein systolic flow
- Lack of aortic valve opening
- Intraventricular stasis and thrombus formation
- Retrograde diastolic mitral flow



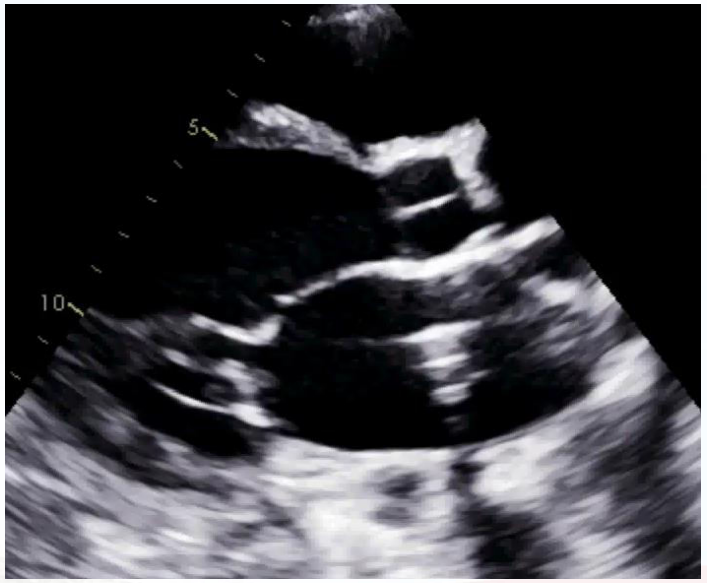


De Backer 2011, Price 2016

LV unloading

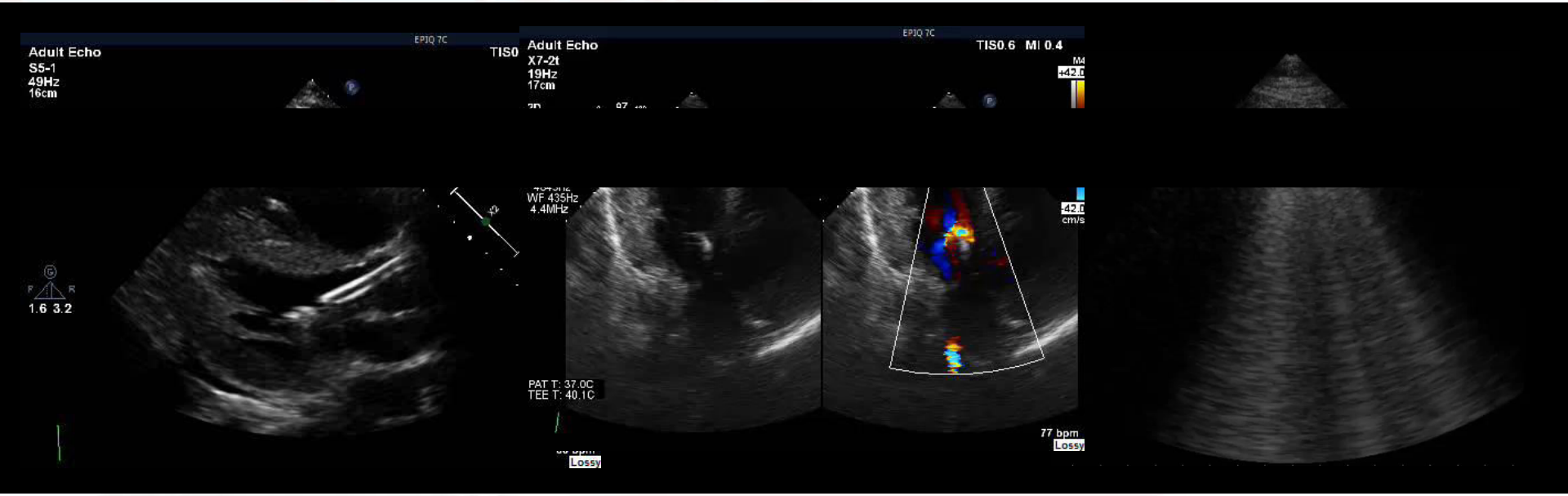


Before septostomy



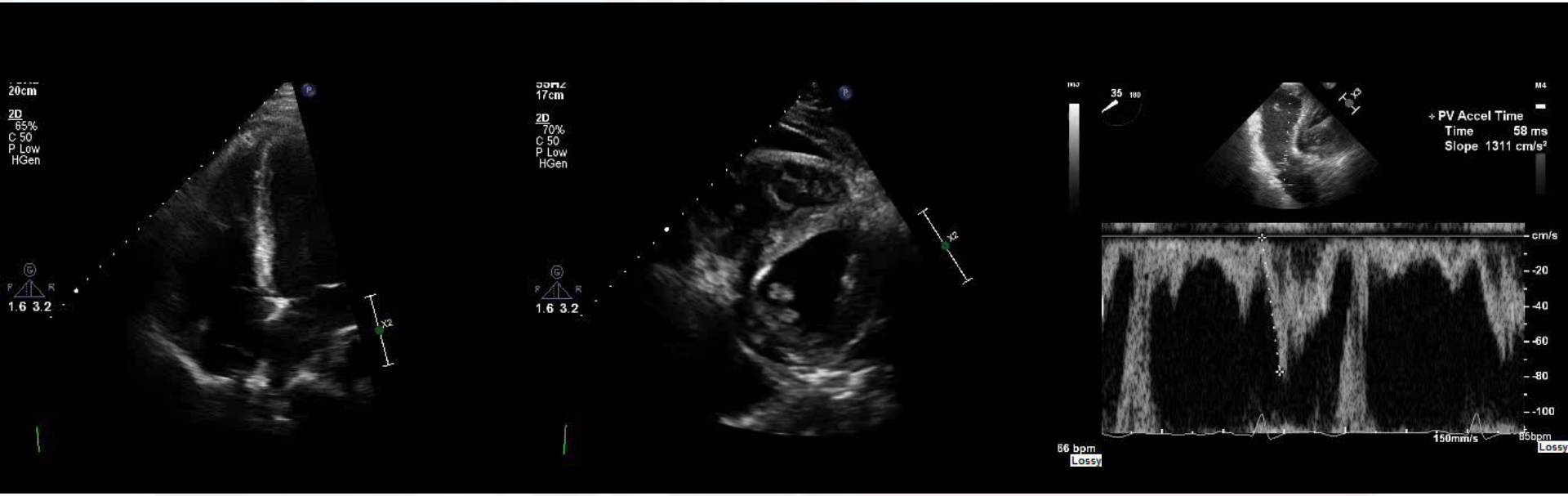
After septostomy

Targeted echo: “why O_2 worse?”



Impella displacement, significant MR, pulmonary oedema

Other important findings?

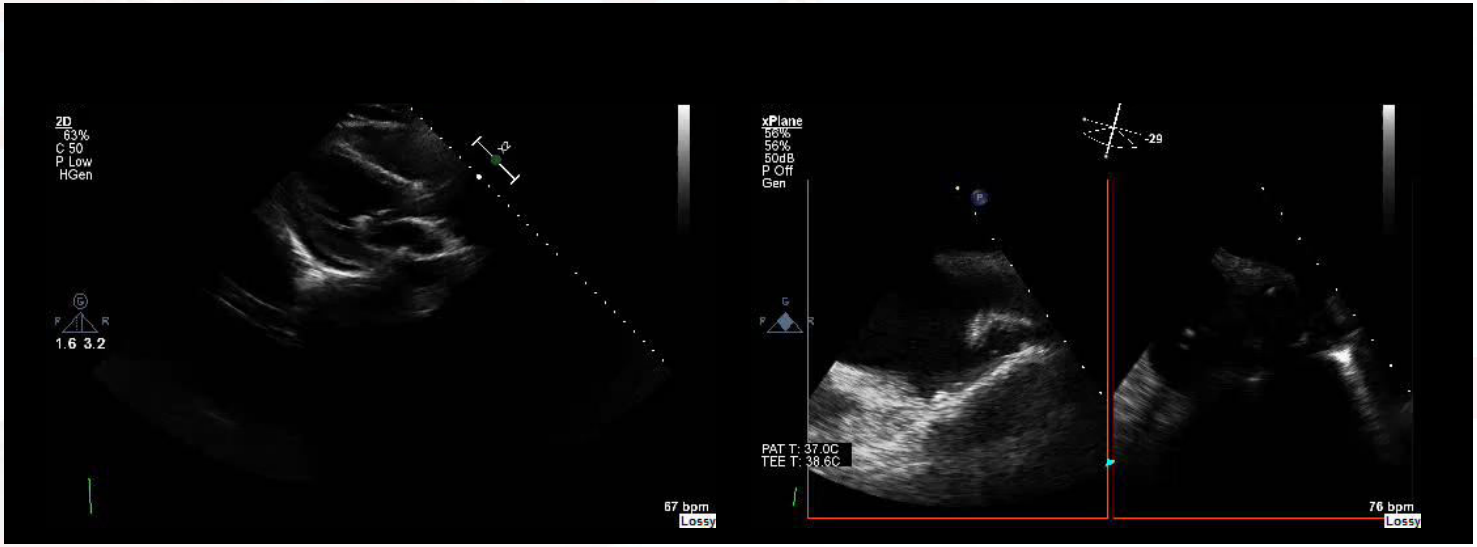


+ Right heart dilatation + high PVR + RV restriction

Interventions

Severe MR – pulmonary oedema – increased PVR: reposition Impella

Inadequate ECMO drainage of right heart: reposition inlet cannula



Resolution of Cardiogenic Shock Using Echocardiography-Guided Pacing Optimization in Intensive Care: A Case Series

Guido Tavazzi, MD[1,2]; Andy Kontogeorgis, MBBch, PhD[3]; Niels P. Bergsland, MSc[4]; Susanna Price, MD, PhD[5]

An iterative method is used whereby the AV delay is changed in increments of 10 to 20 ms depending on the native AV delay and mitral inflow patterns.

The optimal AVD was selected on the basis of the best diastolic LV filling pattern and CO, and smaller mitral regurgitation (MR) in terms of severity and duration.

The optimal diastolic pattern was defined as E and A wave separation, the shortest isovolumic contraction phase, and maximal SV while avoiding diastolic MR

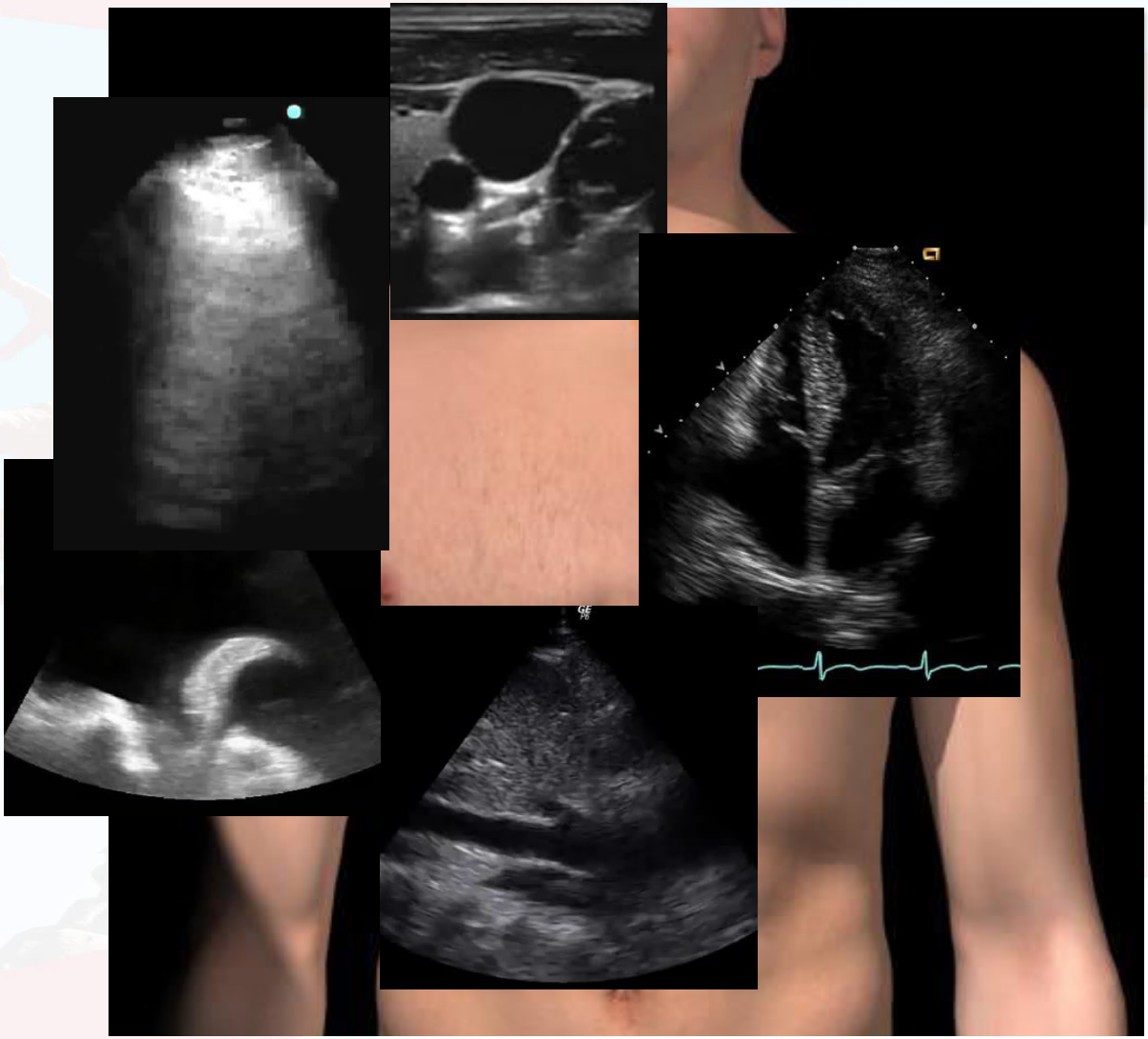
Resolution of Cardiogenic Shock Using Echocardiography-Guided Pacing Optimization in Intensive Care: A Case Series

Guido Tavazzi, MD[1,2]; Andy Kontogeorgis, MBBch, PhD[3]; Niels P. Bergsland, MSc[4]; Susanna Price, MD, PhD[5]

The cardiac output ↗ from 3.2 (2.3/3.8) to 5.7 L/min (4.85/7.1) and cardiac index from 1.64 (1.1/1.9) to 2.68 L/min/m2 (2.1/3.2) and the total isovolumic time reduced from 22.8 to normal values (<14).

The glomerular filtration rate (GFR) ↗ significantly except in one patient with stage IV chronic kidney disease. All inotropes and vasopressors were discontinued within 12 hours of pacemaker optimization on cardiac output, and all patients were discharged from the ICU within 1 week.

Critical Care Medicine. 44(8):e755–e761, AUG 2016

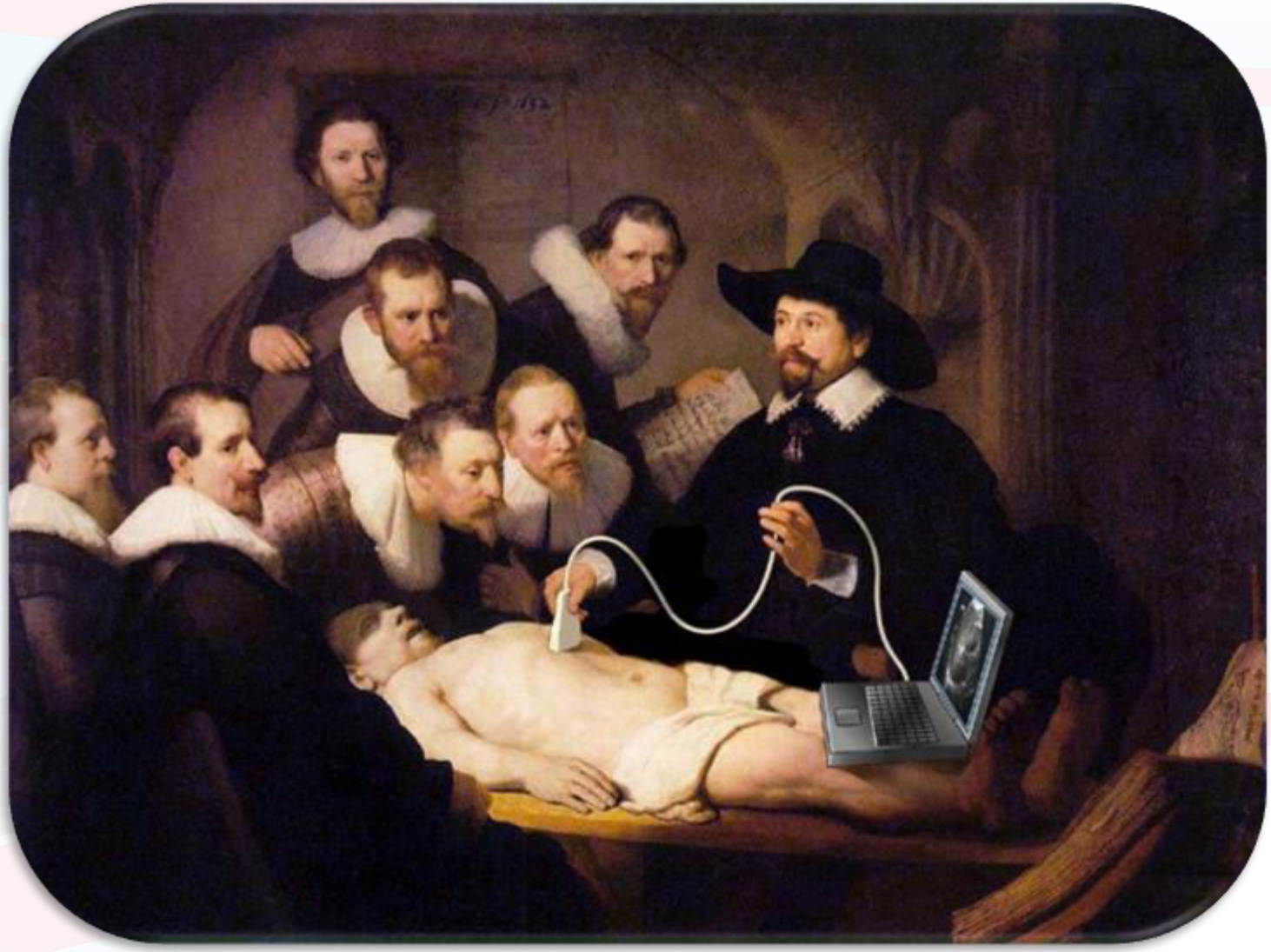


Integrated Approach

5 take-home messages

1. SV estimation is more reliable than EF in critically ill patients
2. LVOT VTI can be a useful surrogate for LV Stroke Volume
3. Echo is paramount in managing patients with pMCS
4. Echo-guided pacing optimization may improve outcomes in cardiogenic shock
5. Integrated approach is the key to proper management

The Anatomy Lesson of Dr. Nicolaes Tulp. 1632. Rembrandt



Merci pour votre attention

hatem.soliman@gmail.com

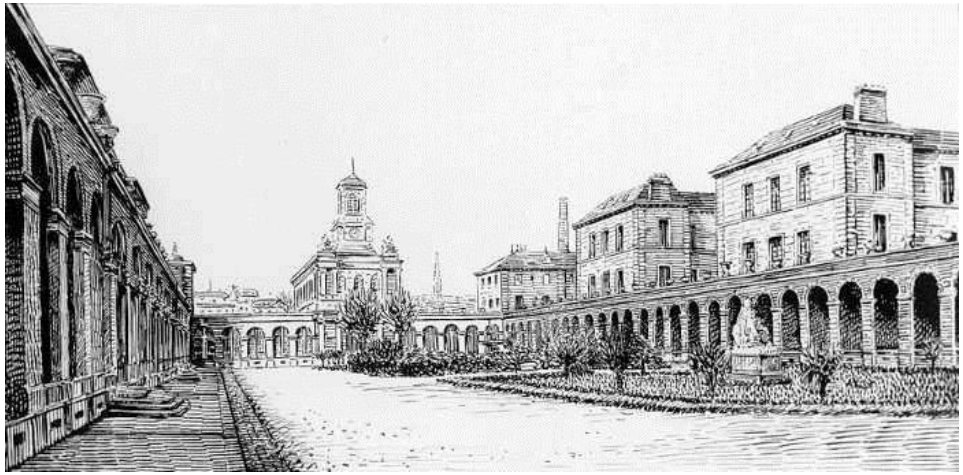
@hatemsoliman

Royal Brompton & Harefield NHS
NHS Foundation Trust

CoSAC
Le congrès des Sociétés Africaines de Cardiologie

STCCCV
Société Tunisienne de Cardiologie & de Chirurgie Cardio-Vasculaire

Le choc cardiogénique, pas si simple …:
dobutamine, noradrénaline ou adrénaline ?



Alexandre Mebazaa

Département d'Anesthésie-Réanimation

Hôpitaux Universitaires Saint Louis – Lariboisière

Université Paris 7; INSERM – UMR 942

Conflicts of interest

Honorarium for lectures

- Orion, Abbott, Novartis, Roche

Consultant:

- BMS, Cardiorentis, Novartis, Sphingotec, Sanofi

Messages principaux

- **ICA sans choc:**
  - *Mécanisme* : « congestion »
  - *Traitement initial* : Diurétiques/dérivés nitrés pas d'inotropes

- **Choc cardiogénique**
  - 1) **si bas débit cardiaque + infarctus du myocarde/Post-Arrêt cardiaque**
    - Premier médicament : norépinéphrine +/- inotrope si besoin
  - 2) **si insuffisance ventriculaire droite**
    - Premier médicament : norépinéphrine +/- inotrope si besoin
  - 3) **pas d'adrénaline : dans tous les cas!**

Intensive Care Med
DOI 10.1007/s00134-015-3659-7

Mattia Arrigo
Alexandre Mebazaa

Understanding the differences among inotropes

	Dobutamine	Adrenaline	Milrinone	Levosimendan
Substance class	Catecholamines	Catecholamines	PDE III inhibitor	Calcium sensitizer
Mechanism of inotropic effect	Beta-adrenergic receptor-mediated increase of cAMP synthesis	Beta-adrenergic receptors-mediated increase of cAMP synthesis	Decreased breakdown of cAMP through inhibition of PDE III	Enhanced troponin C sensitivity to intracellular calcium
Half-life	2–3 min	2 min	2 h	1 h, metabolite (OR-1896) up to 80 h
Common IV infusion (mcg/kg/min)	2–20	0.01–0.10	0.375–0.750	0.05–0.20
Frequent adverse effects	Hypotension (14 %), ventricular arrhythmia (7 %), chest pain (7 %), atrial fibrillation (6 %) [15]	Supraventricular tachycardia (12 %), ventricular arrhythmia (7 %), acute coronary events (3 %) [12]	Hypotension (7 %), atrial fibrillation (3 %), ventricular arrhythmia (2–4 %); increased events in ischaemic heart disease [3]	Hypotension (15 %), atrial fibrillation (9 %), ventricular arrhythmia (8 %), headache (8 %) [15]

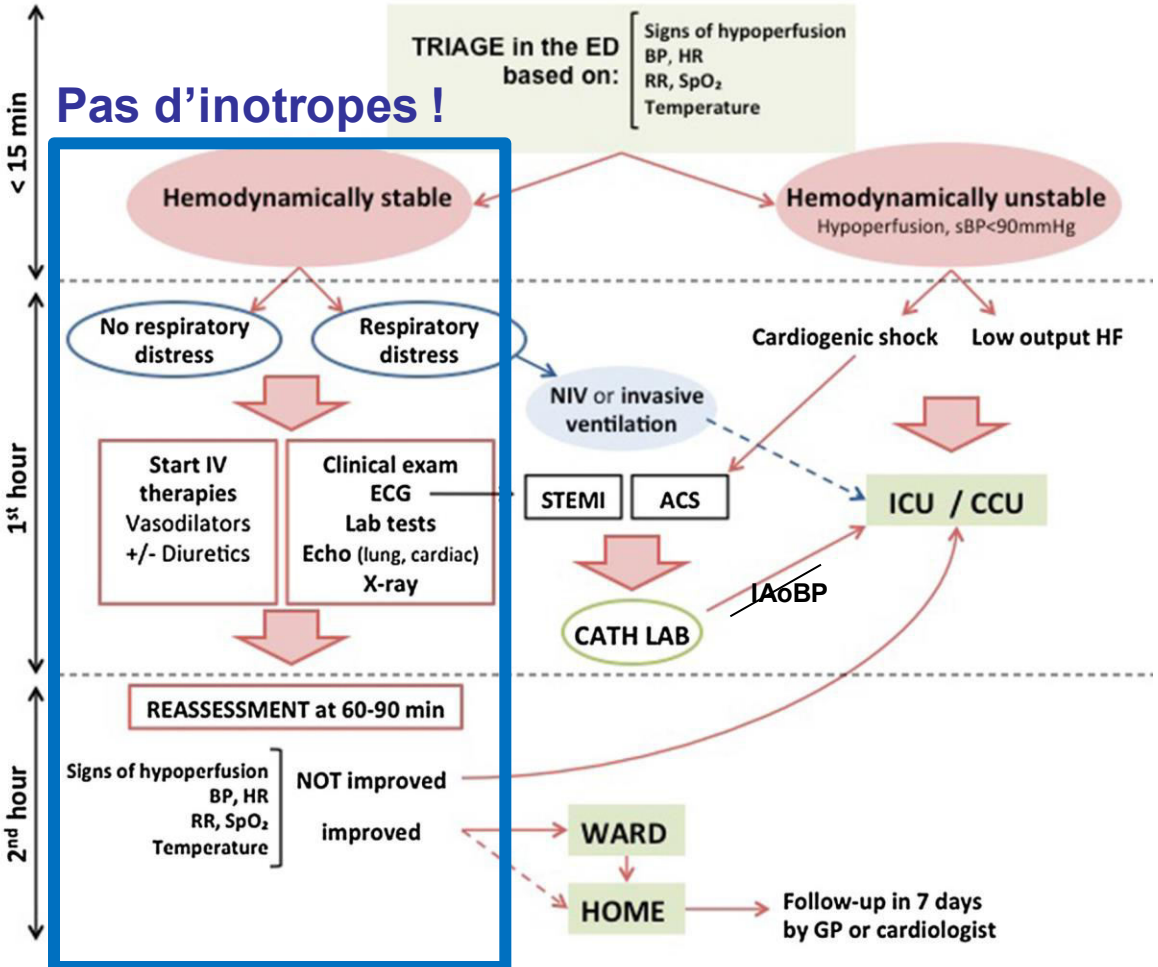
Arrigo M & Mebazaa A; Intensive Care Med, 2015; 5: 912-915

Intensive Care Med
DOI 10.1007/s00134-015-4041-5

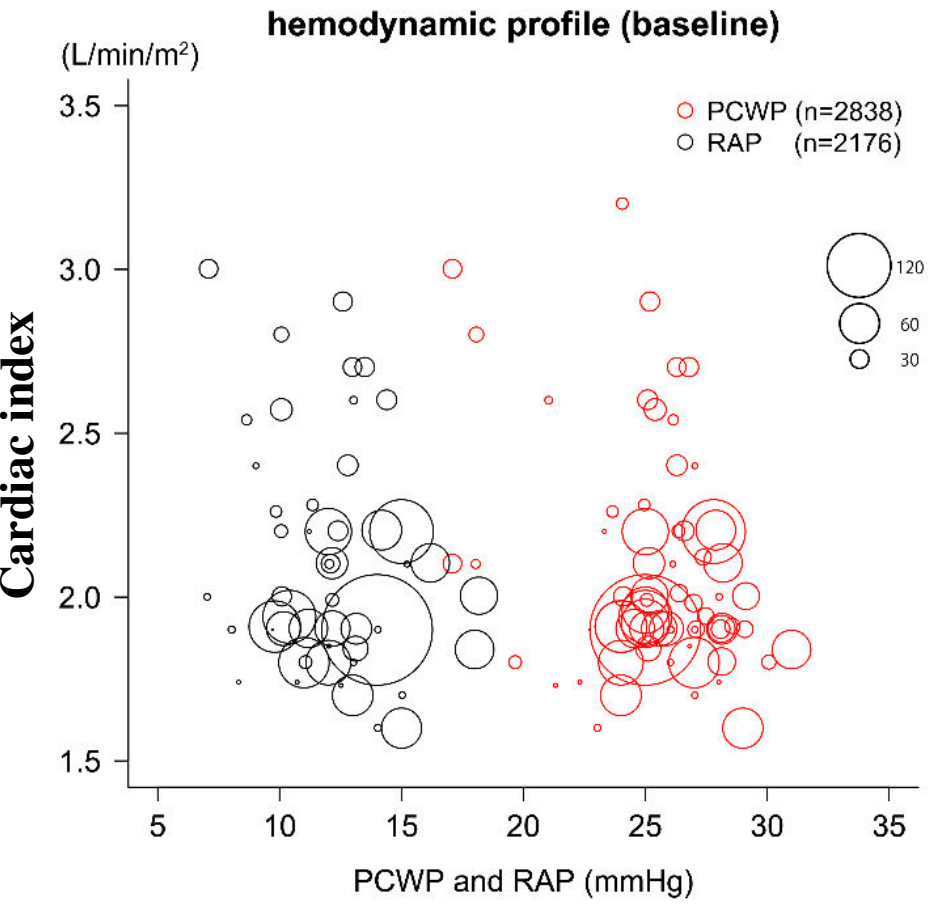
REVIEW

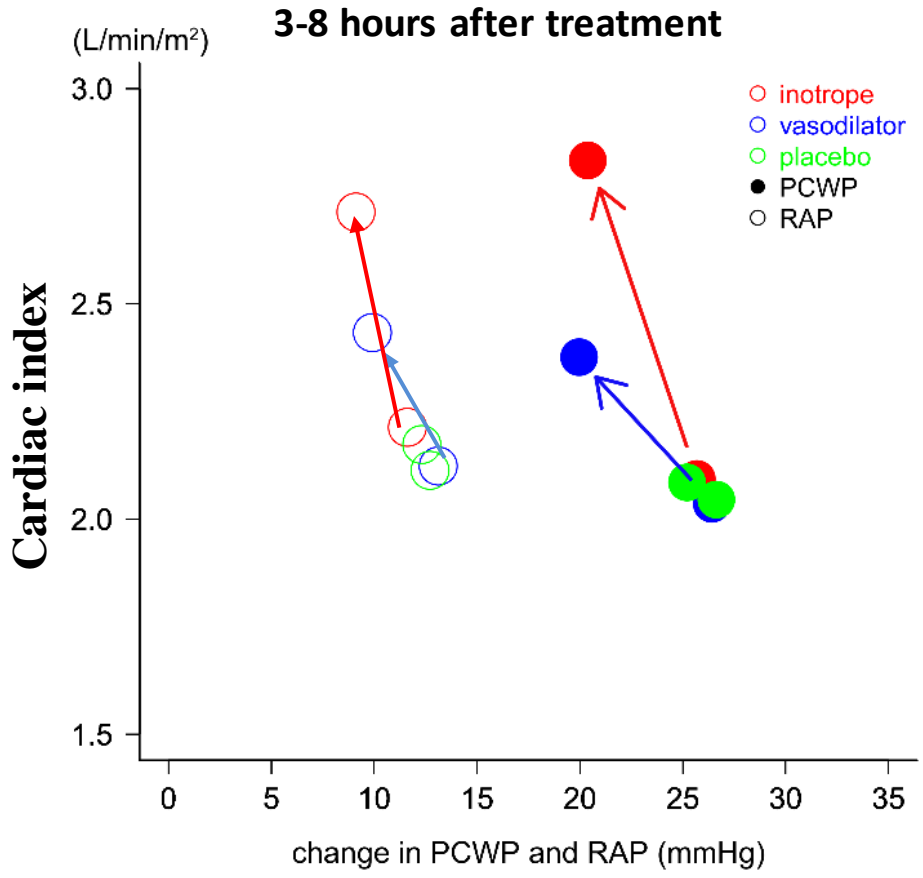
A. Mebazaa
H. Tolppanen
C. Mueller
J. Lassus
S. DiSomma
G. Baksyte
M. Cecconi
D. J. Choi
A. Cohen Solal
M. Christ
J. Masip
M. Arrigo
S. Nouira
D. Ojji
F. Peacock
M. Richards
N. Sato
K. Sliwa
J. Spinar
H. Thiele
M. B. Yilmaz
J. Januzzi

Acute heart failure and cardiogenic shock: a multidisciplinary practical guidance



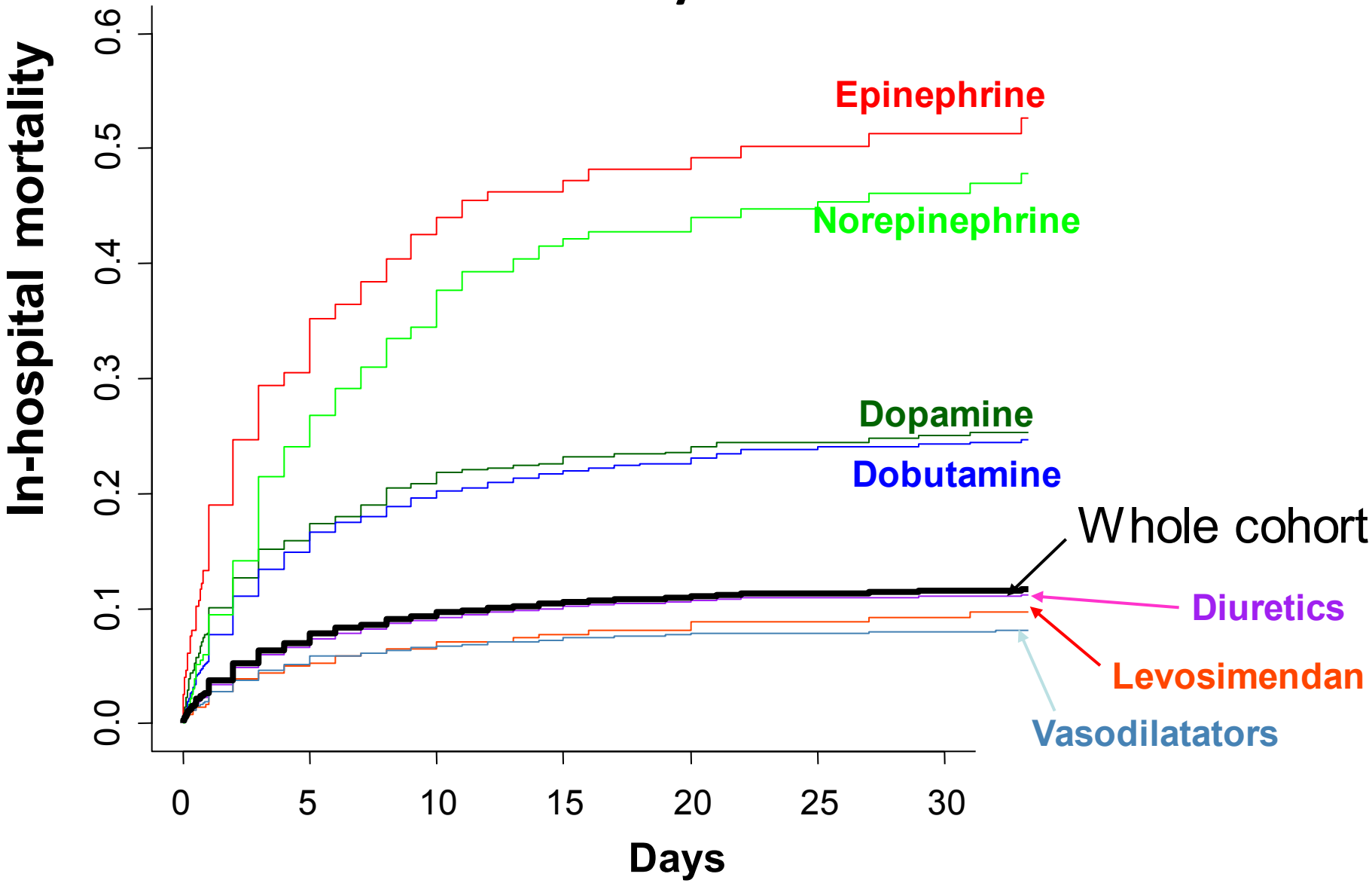
Invasive hemodynamics at baseline and after treatment in AHF: results of a meta-analysis





Ishihara et al Clin Res Cardiol 2016.

Effect of IV drugs in-hospital mortality: propensity score analysis



Mebazaa et al Intensive Care Medicine 2011

EUROPEAN SOCIETY OF CARDIOLOGY®

European Journal of Heart Failure (2017)
doi:10.1002/ejhf.991

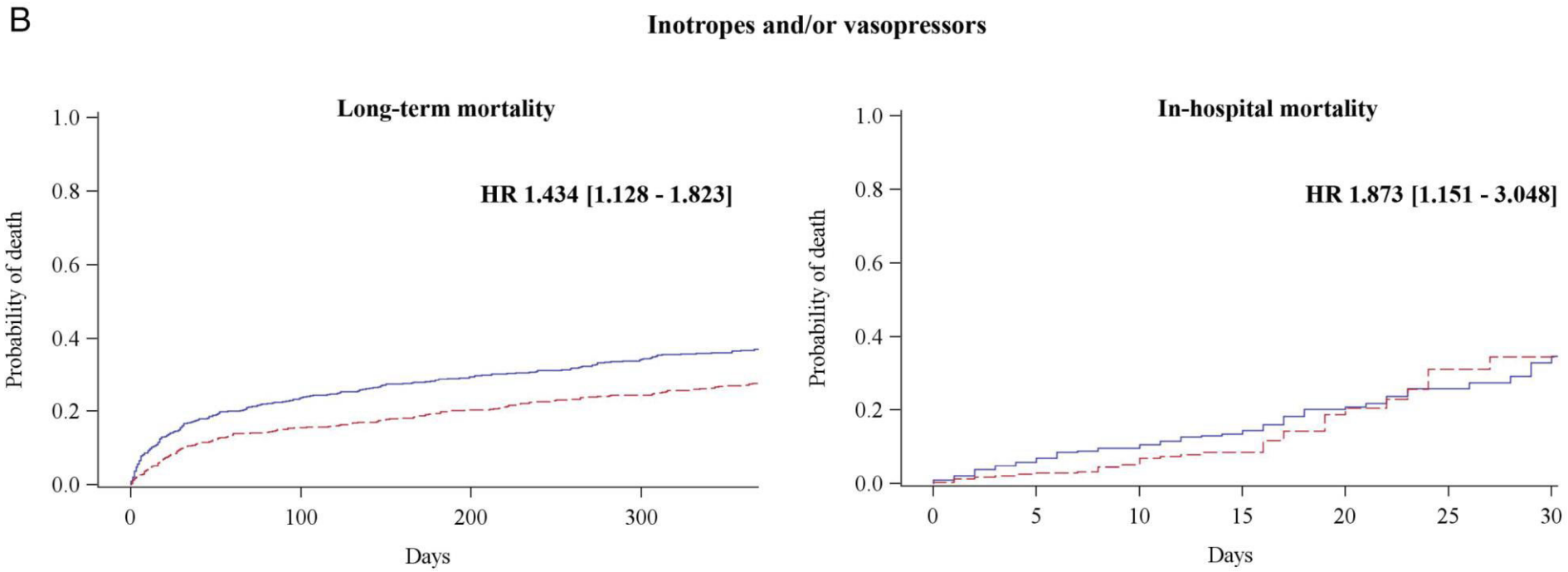
RESEARCH ARTICLE

Long-term safety of intravenous cardiovascular agents in acute heart failure: results from the European Society of Cardiology Heart Failure Long-Term Registry

Alexandre Mebazaa[1,2,3]*, Justina Motiejunaite[1,2,4], Etienne Gayat[1,2,3], Maria G. Crespo-Leiro[5], Lars H. Lund[6], Aldo P. Maggioni[7], Ovidiu Chioncel[8], Eiichi Akiyama[1,9], Veli-Pekka Harjola[10], Petar Seferovic[11], Cecile Laroche[12], Marisa Sanz Julve[13], Eulalia Roig[14], Frank Ruschitzka[15], and Gerasimos Filippatos[16], on behalf of the ESC Heart Failure Long-Term Registry Investigators

Mebazaa A et al Eur J Heart Failure 2017

Heart Failure Association:ESC-Long term registry: propensity matching



Mebazaa A et al Eur J Heart Failure 2017

All catecholamines are not equal!

Table 3 Duration and dosage of treatment with intravenous inotropes and/or vasopressors and their association with long-term all-cause death

Inotrope/vasopressor (whole cohort, *n* = 833)	**Dobutamine (*n* = 354)**	**Dopamine (*n* = 206)**	**Levosimendan (*n* = 109)**	**Norepinephrine (*n* = 45)**	**Epinephrine (*n* = 14)**
Hours of treatment					
Mean ± SD	42.5 ± 29.9	43.4 ± 32.3	24.8 ± 6.3	40.2 ± 28.3	37.6 ± 41.7
Median (IQR)	36.0 (23.0–72.0)	36.0 (20.0–72.0)	24.0 (24.0–24.0)	35.0 (17.0–60.0)	22.0 (1.0–72.0)
Long-term all-cause death, %	37.9	49.0	38.5	55.6	64.3
Inotrope/vasopressor (matched cohort, *n* = 606)	**Dobutamine (*n* = 512)**	**Dopamine (*n* = 314)**	**Levosimendan (*n* = 168)**	**Norepinephrine (*n* = 36)**	**Epinephrine (*n* = 16)**
HR (95% CI) for long-term all-cause death	1.055 (0.727–1.531)	**1.628 (1.031–2.572)**	1.229 (0.618–2.445)	3.762 (0.903–15.663)	NA

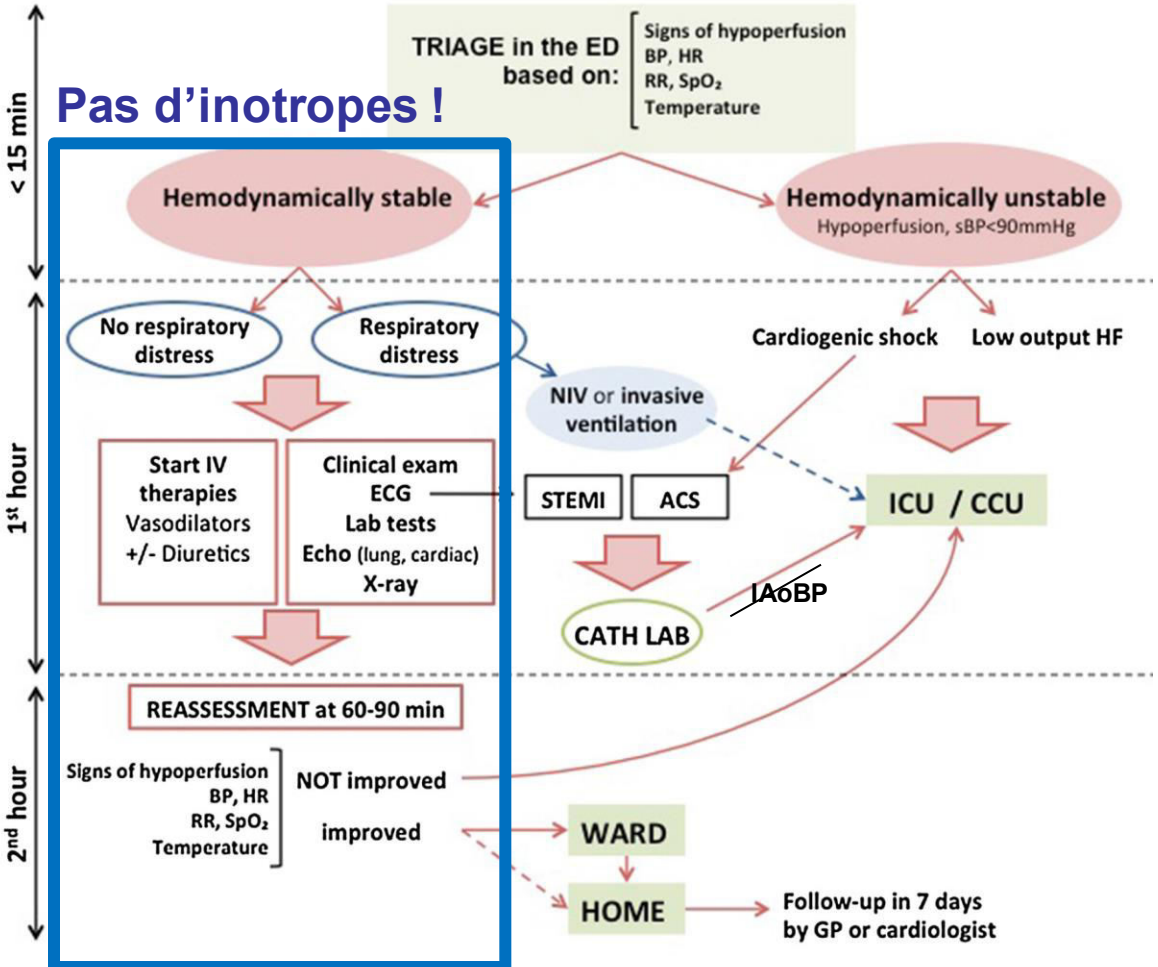
Mebazaa A et al Eur J Heart Failure 2017

Intensive Care Med
DOI 10.1007/s00134-015-4041-5

REVIEW

A. Mebazaa
H. Tolppanen
C. Mueller
J. Lassus
S. DiSomma
G. Baksyte
M. Cecconi
D. J. Choi
A. Cohen Solal
M. Christ
J. Masip
M. Arrigo
S. Nouira
D. Ojji
F. Peacock
M. Richards
N. Sato
K. Sliwa
J. Spinar
H. Thiele
M. B. Yilmaz
J. Januzzi

Acute heart failure and cardiogenic shock: a multidisciplinary practical guidance

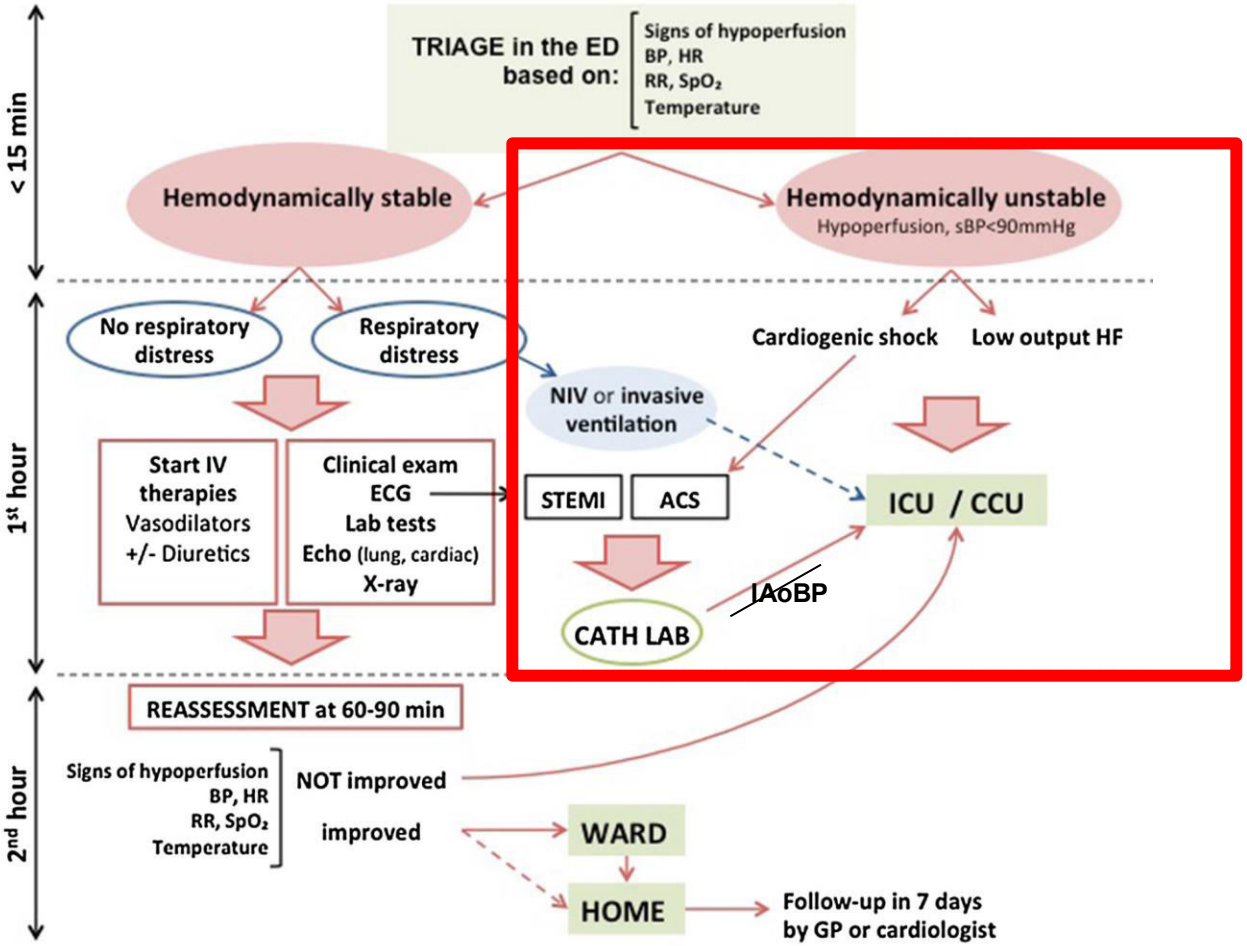


Intensive Care Med
DOI 10.1007/s00134-015-4041-5

REVIEW

A. Mebazaa
H. Tolppanen
C. Mueller
J. Lassus
S. DiSomma
G. Baksyte
M. Cecconi
D. J. Choi
A. Cohen Solal
M. Christ
J. Masip
M. Arrigo
S. Nouira
D. Ojji
F. Peacock
M. Richards
N. Sato
K. Sliwa
J. Spinar
H. Thiele
M. B. Yilmaz
J. Januzzi

Acute heart failure and cardiogenic shock: a multidisciplinary practical guidance



EUROPEAN SOCIETY OF CARDIOLOGY®

European Journal of Heart Failure (2015) **17**, 501–509
doi:10.1002/ejhf.260

Clinical picture and risk prediction of short-term mortality in cardiogenic shock

Veli-Pekka Harjola[1*,†], Johan Lassus[2†], Alessandro Sionis[3], Lars Køber[4], Tuukka Tarvasmäki[5], Jindrich Spinar[6], John Parissis[7], Marek Banaszewski[8], Jose Silva-Cardoso[9], Valentina Carubelli[10], Salvatore Di Somma[11], Heli Tolppanen[2], Uwe Zeymer[12], Holger Thiele[13], Markku S Nieminen[2], and Alexandre Mebazaa[14], for the CardShock study investigators and the GREAT network

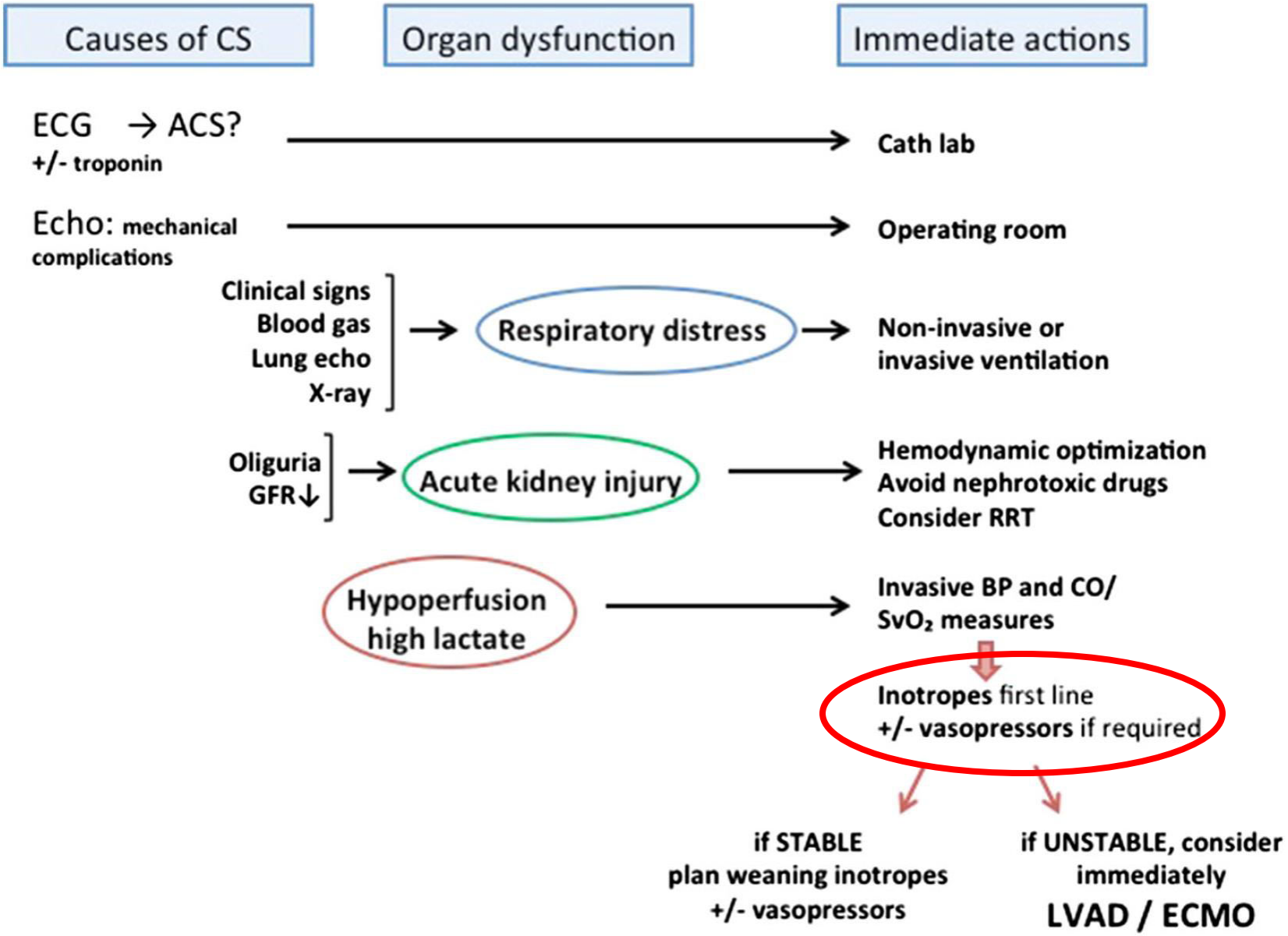
VP Harjola et al, Eur J Heart Failure 2015

CardShock: patients characteristics

Characteristic	All ($n = 219$)
Systolic blood pressure, mmHg	78 (14)
Diastolic blood pressure, mmHg	47 (10)
Mean arterial pressure, mmHg	57 (11)
Heart rate, b.p.m.	90 (28)
Sinus rhythm	170 (78)
Clinical findings, *n* (%)	
Cold periphery	207 (95)
Confusion	148 (68)
Oliguria	121 (55)
Lactate >2 mmol/L	155 (71)

VP Harjola et al, Eur J Heart Failure 2015

CARDIOGENIC SHOCK (CS)



Mebazaa et al. Intensive Care Medicine 2016

HF guidelines 2016

Inotropic agents – dobutamine, dopamine, levosimendan, phosphodiesterase III (PDE III) inhibitors
Short-term, i.v. infusion of inotropic agents may be considered in patients with hypotension (SBP <90 mmHg) and/or signs/ symptoms of hypoperfusion despite adequate filling status, to increase cardiac output, increase blood pressure, improve peripheral perfusion and maintain end-organ function.
An intravenous infusion of levosimendan or a PDE III inhibitor may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypotension with subsequent hypoperfusion.
Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.
Vasopressors
A vasopressor (norepinephrine preferably) may be considered in patients who have cardiogenic shock, despite treatment with another inotrope, to increase blood pressure and vital organ perfusion.
It is recommended to monitor ECG and blood pressure when using inotropic agents and vasopressors, as they can cause arrhythmia, myocardial ischaemia, and in the case of levosimendan and PDE III inhibitors also hypotension. In such cases intra-arterial blood pressure measurement may be considered.

Ponikowski P et al, Heart Failure Guidelines, Eur Heart J, 2016

Tarvasmäki *et al. Critical Care* (2016) 20:208
DOI 10.1186/s13054-016-1387-1

Critical Care

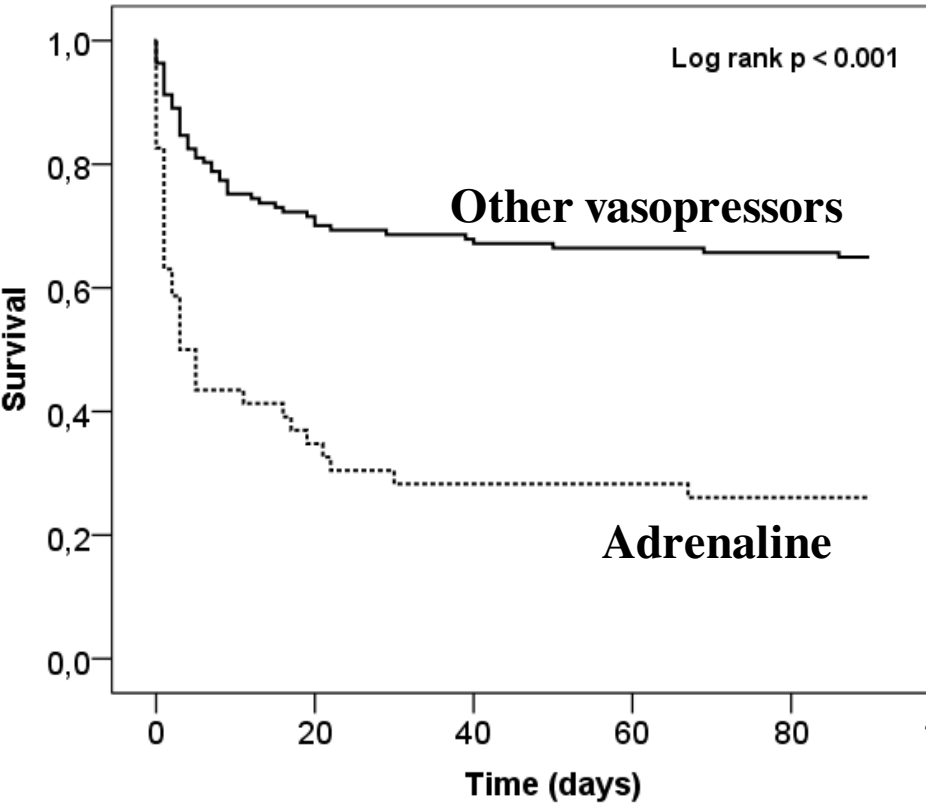
RESEARCH Open Access

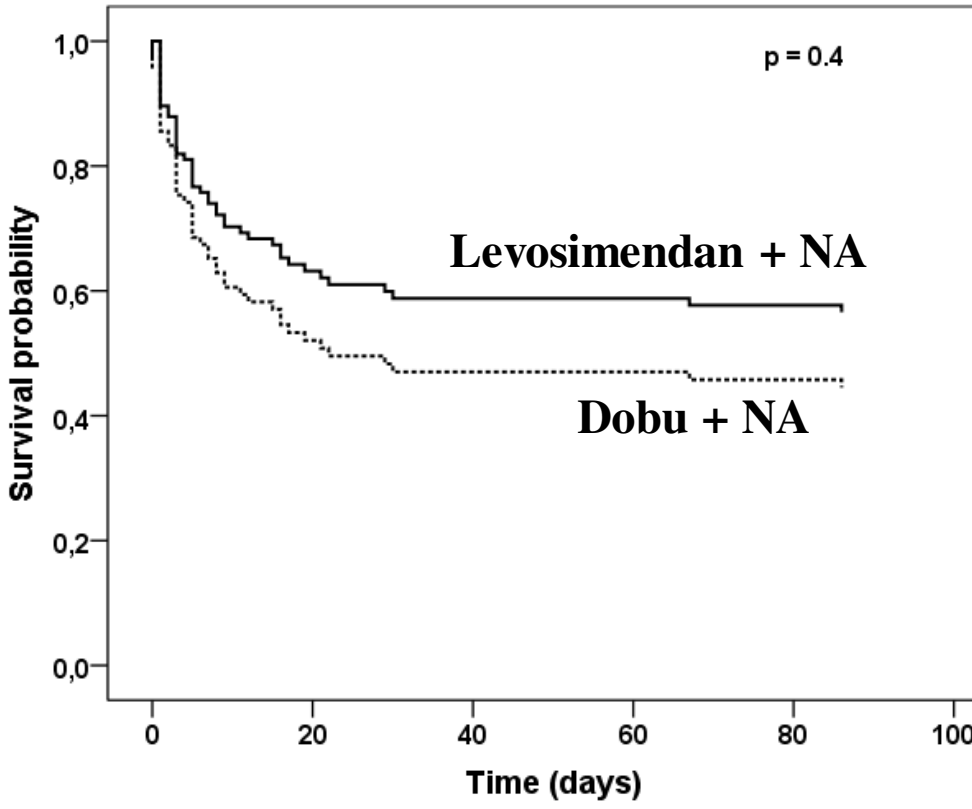
Current real-life use of vasopressors and inotropes in cardiogenic shock - adrenaline use is associated with excess organ injury and mortality

Tuukka Tarvasmäki[1*], Johan Lassus[2], Marjut Varpula[2], Alessandro Sionis[3], Reijo Sund[4], Lars Køber[5], Jindrich Spinar[6], John Parissis[7], Marek Banaszewski[8], Jose Silva Cardoso[9], Valentina Carubelli[10], Salvatore Di Somma[11], Alexandre Mebazaa[12], Veli-Pekka Harjola[1] and for the CardShock study investigators

Tarvasmäki T et al. Crit Care 2016

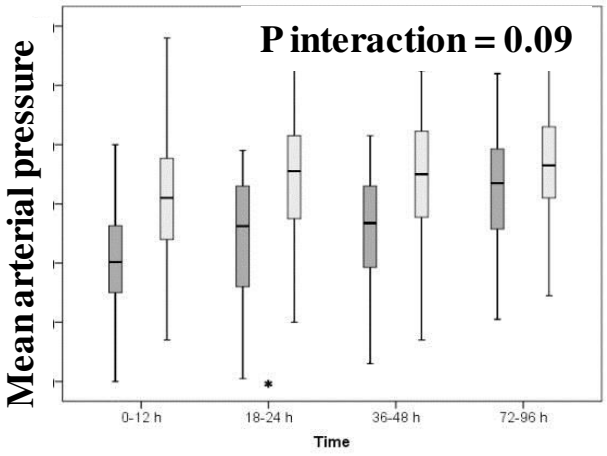
CardShock: Adrenaline is the worse vasopressor in cardiogenic shock secondary to ACS

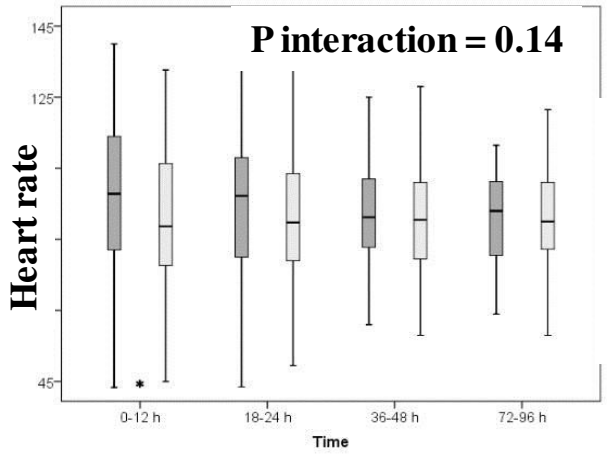


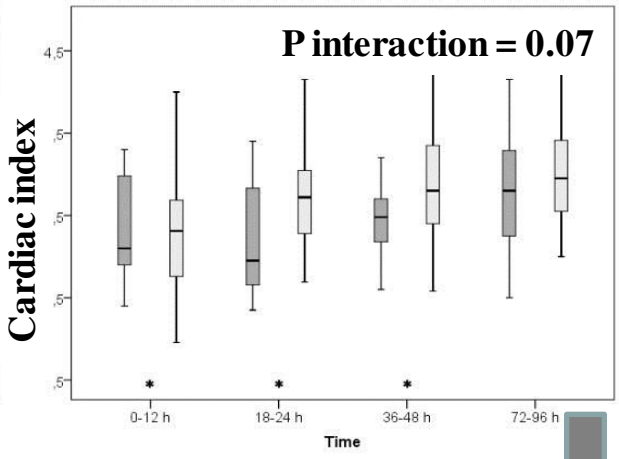


Tarvasmäki T et al. Crit Care 2016

CardShock: Detrimental effect of adrenaline on organ function (regardless to resuscitation)

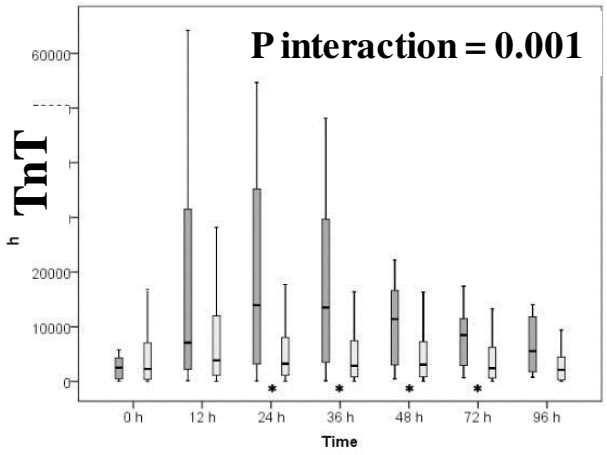


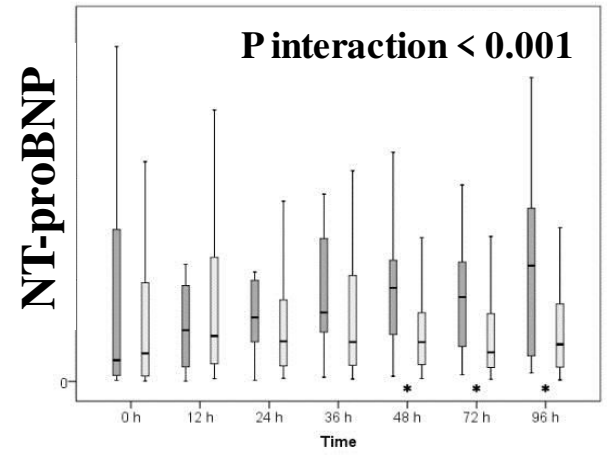


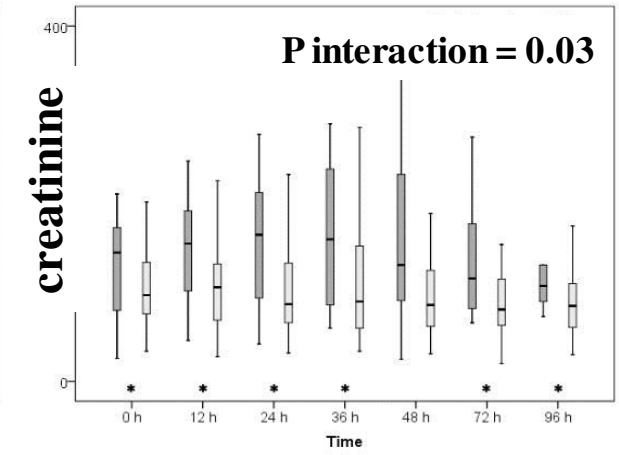


A

NA



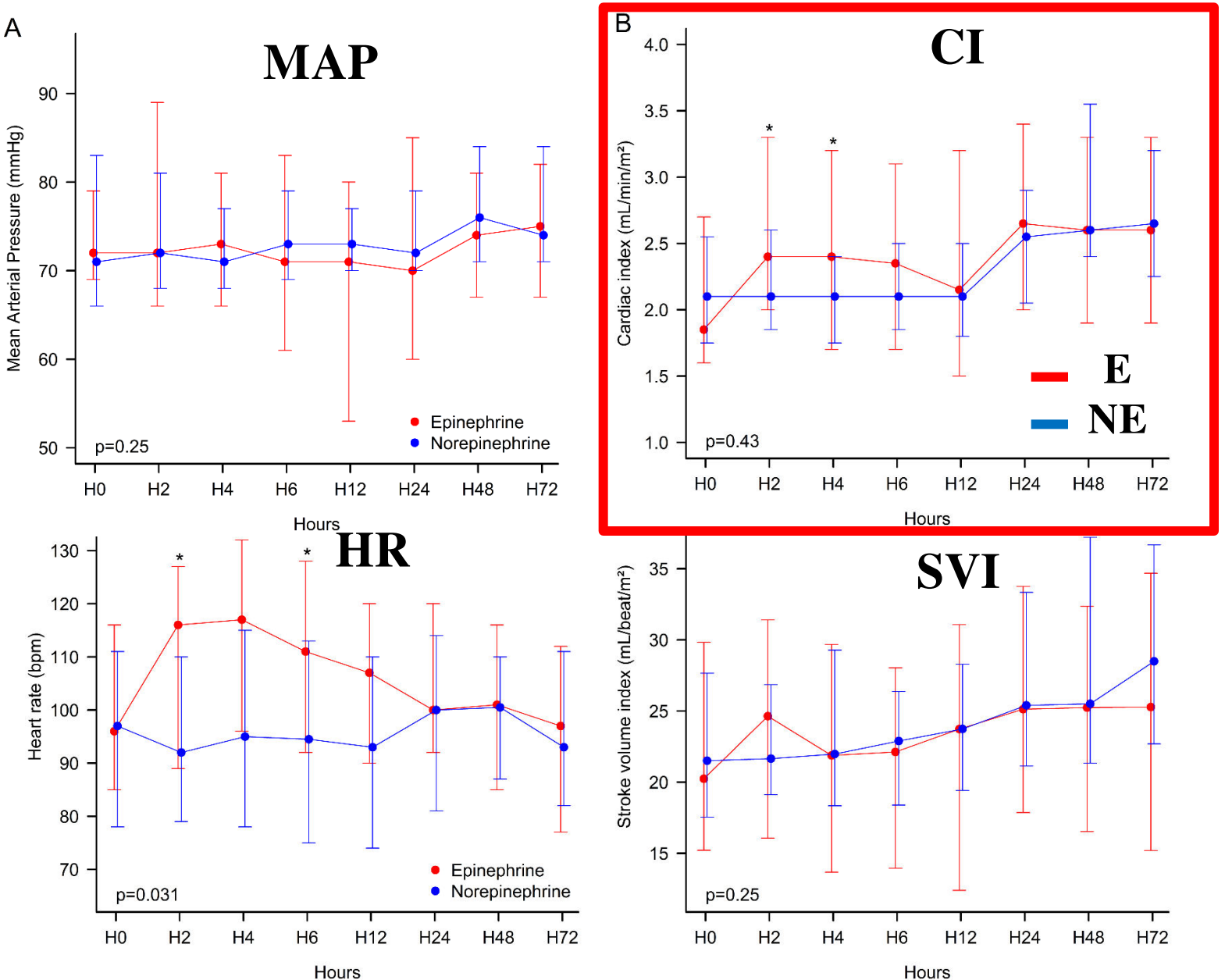




Tarvasmäki T et al. Crit Care 2016

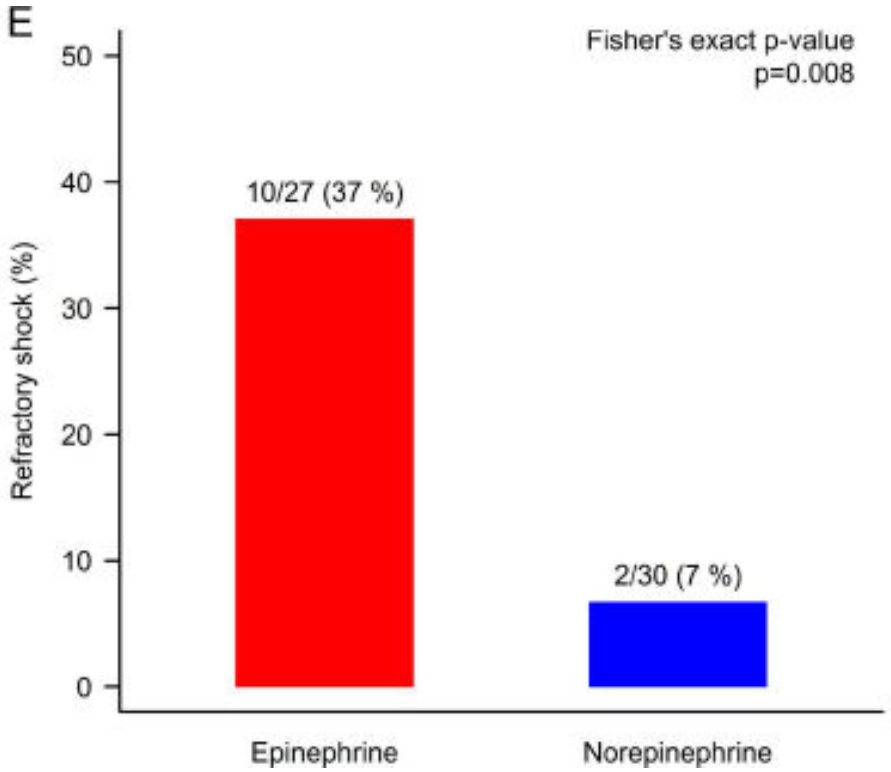
Epinephrine vs NE in CS: primary endpoint

B Levy et al. Epinephrine vs NE in CS. J Am Coll Cardiol, 2018

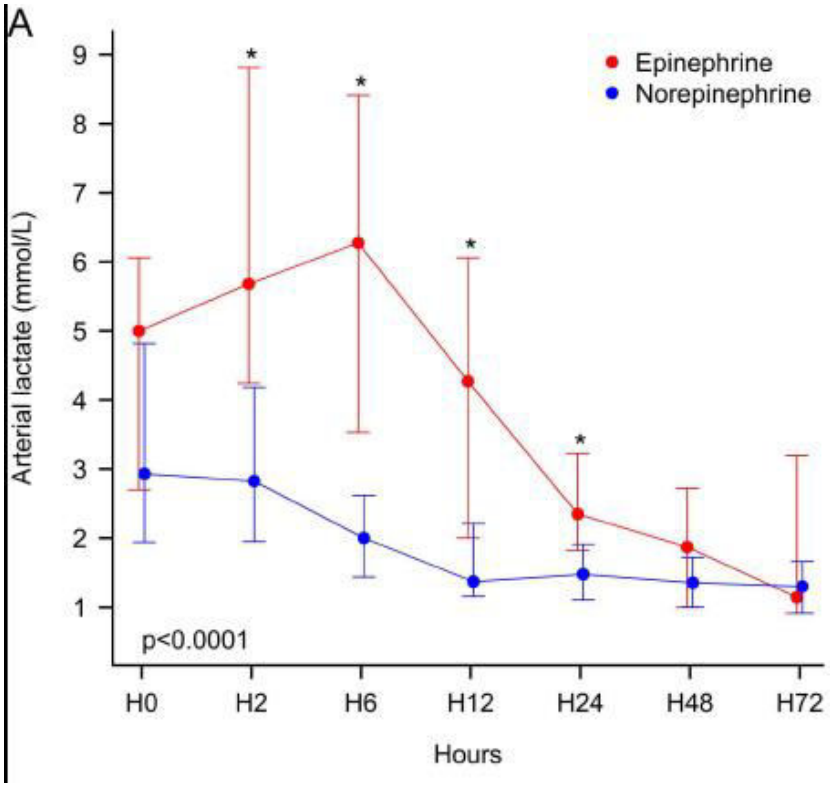


OPTIMA – CC: Epinephrine versus norepinephrine in cardiogenic shock

% Refractory Shock



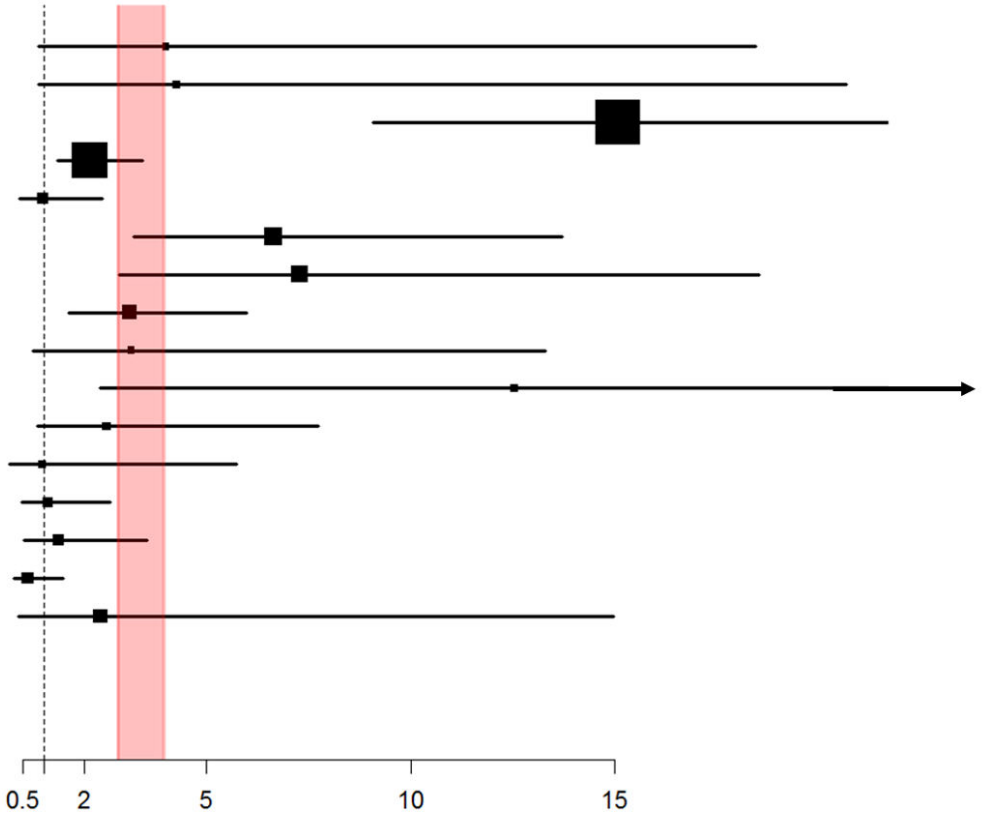
Arterial lactate



Levy B et al, J American Coll Cardiol, 2018

Epinephrine and short-term survival in cardiogenic shock: An individual data meta-analysis of 2,583 patients.

	No. of patients	No. of patients receiving epinephrine	OR for short-term mortality [95% CI]
Adler, 2012	40	10	4.00 [0.87 - 18.45]
Adler,unpublished	47	9	4.27 [0.88 - 20.67]
AHEAD, 2011	674	304	15.08 [9.08 - 25.05]
ALARM, 2011	520	86	2.14 [1.34 - 3.42]
Chua, 2011	105	80	0.99 [0.40 - 2.45]
CARDSHOCK, 2016	219	46	6.64 [3.22 - 13.71]
Champion, 2014	192	130	7.27 [2.85 - 18.54]
EFICA, 2006	158	75	3.10 [1.61 - 5.98]
Gaudard, 2015	40	11	3.15 [0.75 - 13.29]
IMPRESS in Severe Shock, 2017	48	14	12.55 [2.38 - 66.01]
OPTIMA CC, 2018	57	27	2.55 [0.84 - 7.72]
Basir, unpublished	45	8	0.96 [0.16 - 5.73]
Popovic, 2011	86	47	1.11 [0.47 - 2.63]
Simonis, 2012	89	25	1.37 [0.53 - 3.55]
SMASH, 1998	111	41	0.62 [0.26 - 1.47]
Valente, 2011	152	34	2.40 [0.38 - 14.96]
All studies	2583	947	3.33 [2.81 - 3.94]



Levy B et al, J American Coll Cardiol, 2018

Intensive Care Med
https://doi.org/10.1007/s00134-018-5214-9

REVIEW

Management of cardiogenic shock complicating myocardial infarction

Alexandre Mebazaa[1,2,3,4*], Alain Combes[5*], Sean van Diepen[6], Alexa Hollinger[2,3,7], Jaon N. Katz[8], Giovanni Landoni[9,10], Ludhmila Abrahao Hajjar[11], Johan Lassus[12], Guillaume Lebreton[13,14], Gilles Montalescot[14,15], Jin Joo Park[16], Susanna Price[17], Alessandro Sionis[18,19], Demetris Yannopolos[20], Veli-Pekka Harjola[21], Bruno Levy[22,23,24] and Holger Thiele[25*]

Mebazaa… Thiele. Intensive care Medicine 2018

< 2 hours

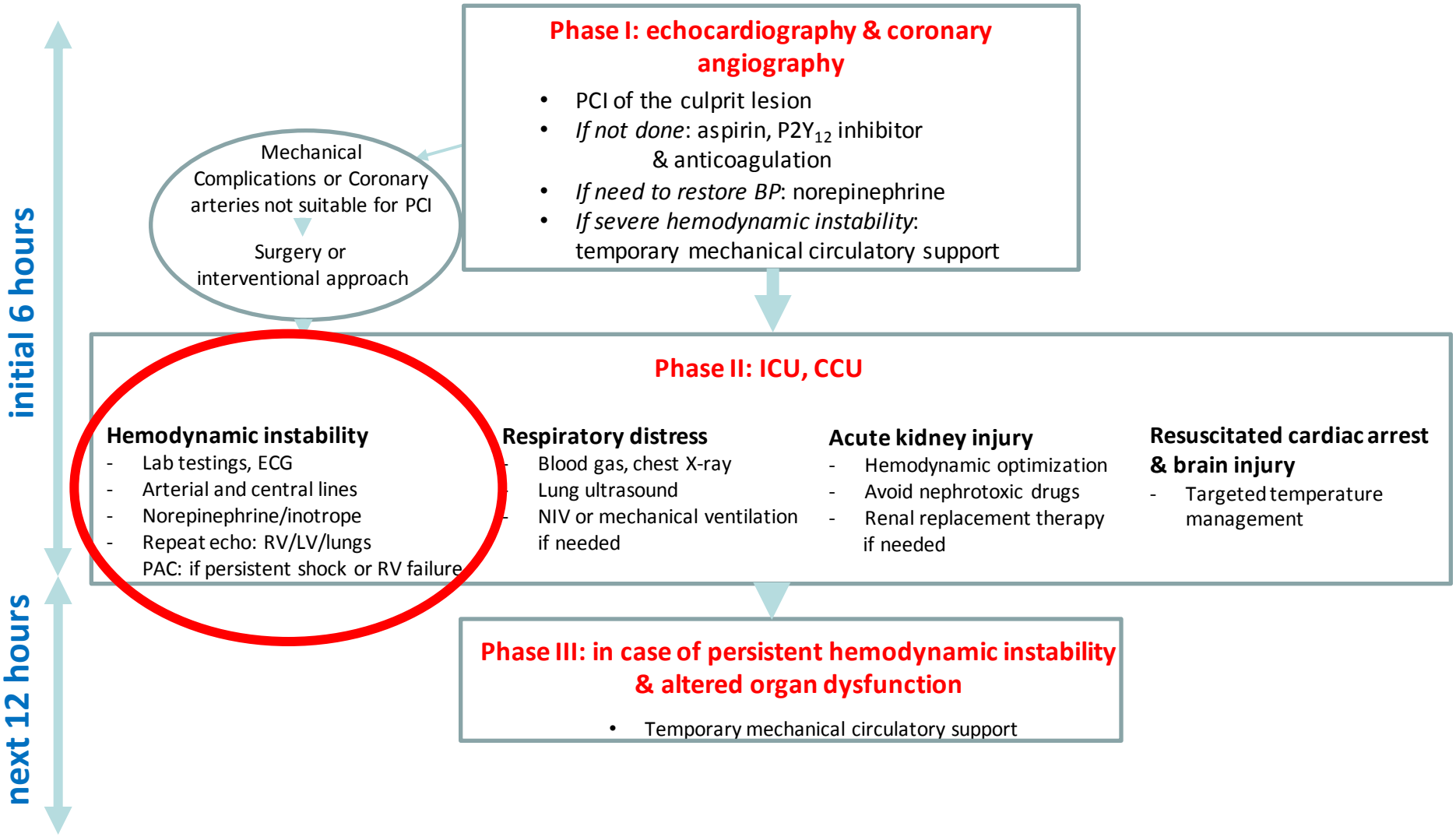
STEMI or high-risk ECG signs or symptoms suggesting AMI, < 3 hours & hemodynamic instability or resuscitated cardiac arrest

PCI should be performed < 120 min:
alert & transfer to cardiac shock center for primary PCI

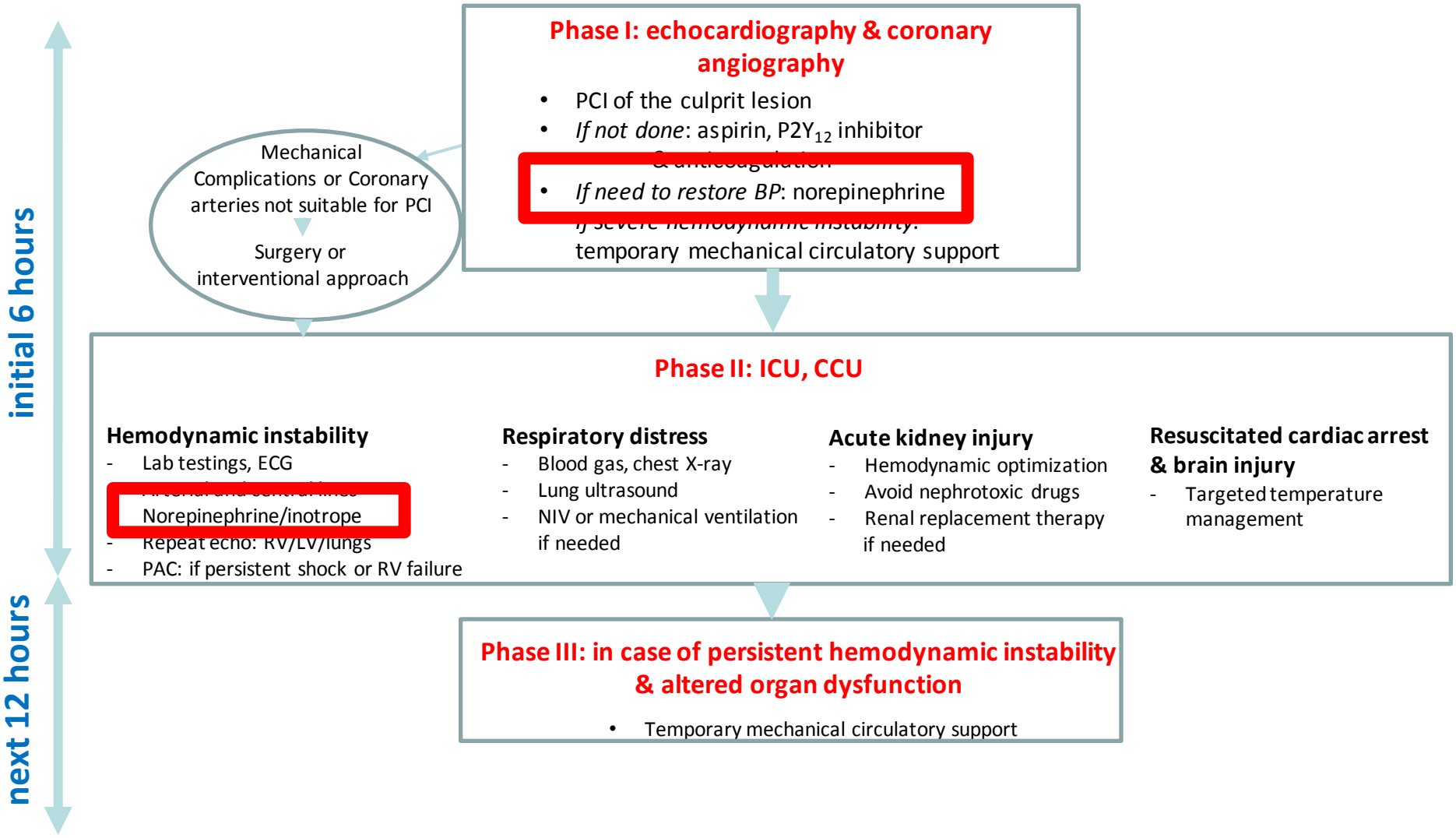
- *During the transport*, start:
  - Aspirin orally or iv
  - Consider potent $P2Y_{12}$ inhibitor (prasugrel or ticagrelor), or clopidogrel if contraindicated, orally
  - Anticoagulation iv
- *If hemodynamic instability*: volume loading < 250 ml & norepinephrine on peripheral line
- *If SpO_2 < 90%:* O_2 therapy

Cardiac Shock Center

Mebazaa… Thiele. Intensive care Medicine 2018



Mebazaa… Thiele. Intensive care Medicine 2018



Mebazaa… Thiele. Intensive care Medicine 2018

In case of severe Right Ventricular dysfunction

Management of Right Ventricular dysfynction

Vasopressors and inotropes	
Noradrenaline, 0.2–1.0 µg/kg.min[30]	Increases RV inotropy, systemic blood pressure, promotes positive ventricular interactions, restores coronary perfusion gradient
Dobutamine, 2–20 µg/kg.min[30]	Increases RV inotropy, lowers filling pressures
Levosimendan, 0.1–0.2 µg/kg.min (6–12 µg/kg bolus over 10 min is optional and not recommended if SBP <90 mmHg). Infusion can be decreased to 0.05 µg/kg.min or increased to 0.2 µg/kg.min)[30]	Combines RV inotropy and pulmonary vasodilation; favourably affects right ventricular–arterial uncoupling

VP Harjola et al. Eur J Heart Failure 2016

Messages principaux

- **ICA sans choc:**
  - *Mécanisme* : « congestion »
  - *Traitement initial* : Diurétiques/dérivés nitrés pas d'inotropes

- **Choc cardiogénique**
  - 1) **si bas débit cardiaque + infarctus du myocarde/Post-Arrêt cardiaque**
    - Premier médicament : norépinéphrine +/- inotrope si besoin
  - 2) **si insuffisance ventriculaire droite**
    - Premier médicament : norépinéphrine +/- inotrope si besoin
  - 3) **pas d'adrénaline : dans tous les cas!**