Le Choc Cardiogénique en 2019

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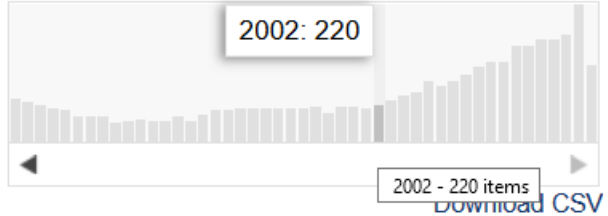

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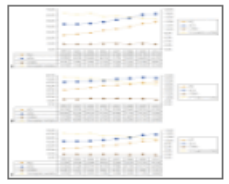
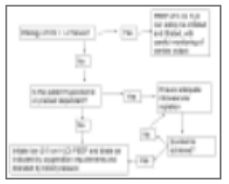

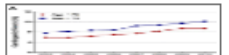

1. Rationale and design of DanGer **shock**: Danish-German **cardiogenic shock** trial.
Udesen NJ, Møller JE, Lindholm MG, Eiskjær H, Schäfer A, Werner N, Holmvang L, Terkelsen CJ, Jensen LO, Junker A, Schmidt H, Wachtell K, Thiele H, Engstrøm T, Hassager C; DanGer **Shock** investigators.
Am Heart J. 2019 May 6;214:60-68. doi: 10.1016/j.ahj.2019.04.019. [Epub ahead of print]
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J Pediatr (Rio J). 2019 Jun 6. pii: S0021-7557(19)30059-2. doi: 10.1016/j.jped.2019.04.006. [Epub ahead of print]
PMID: 31176690

2. Accuracy and precision of non-invasive **cardiac output** monitoring by electrical cardiometry: a systematic review and meta-analysis.
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J Clin Monit Comput. 2019 Jun 7. doi: 10.1007/s10877-019-00330-y. [Epub ahead of print] Review.
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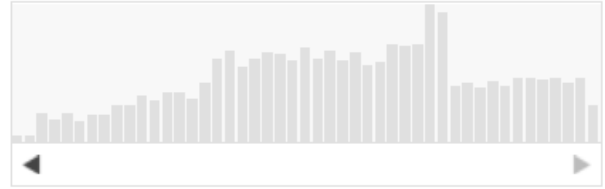

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European Journal of Heart Failure (2016)
doi:10.1002/ejhf.646

Cardiogenic shock in intensive care units: evolution of prevalence, patient profile, management and outcomes, 1997–2012

Etienne Puymirat[1], Jean Yves Fagon[2], Philippe Aegerter[3,4,5], Jean Luc Diehl[2], Alexandra Monnier[2], Caroline Hauw-Berlemont[2], Florence Boissier[2], Gilles Chatellier[6], Bertrand Guidet[7], Nicolas Danchin[1] Nadia Aissaoui[2]*, on behalf of the Collège des Utilisateurs de Bases de données en Réanimation (CUB-Réa Group [Intensive Care Database User Group])

1997-2012 Cub-Réa French database

	1997–2000 (*n* = 3248)	2001–2004 (*n* = 4602)	2005–2008 (*n* = 5179)	2009–2012 (*n* = 6387)	*P*-value for trends
Age, years	66.4 ± 16.1	65.1 ± 16.5	63.6 ± 16.9	63.7 ± 16.6	<0.001
SAPS-II	58.7 ± 25.3	59.3 ± 24.7	63.8 ± 23.7	64.5 ± 23.3	<0.001

European Journal of Heart Failure (2016)
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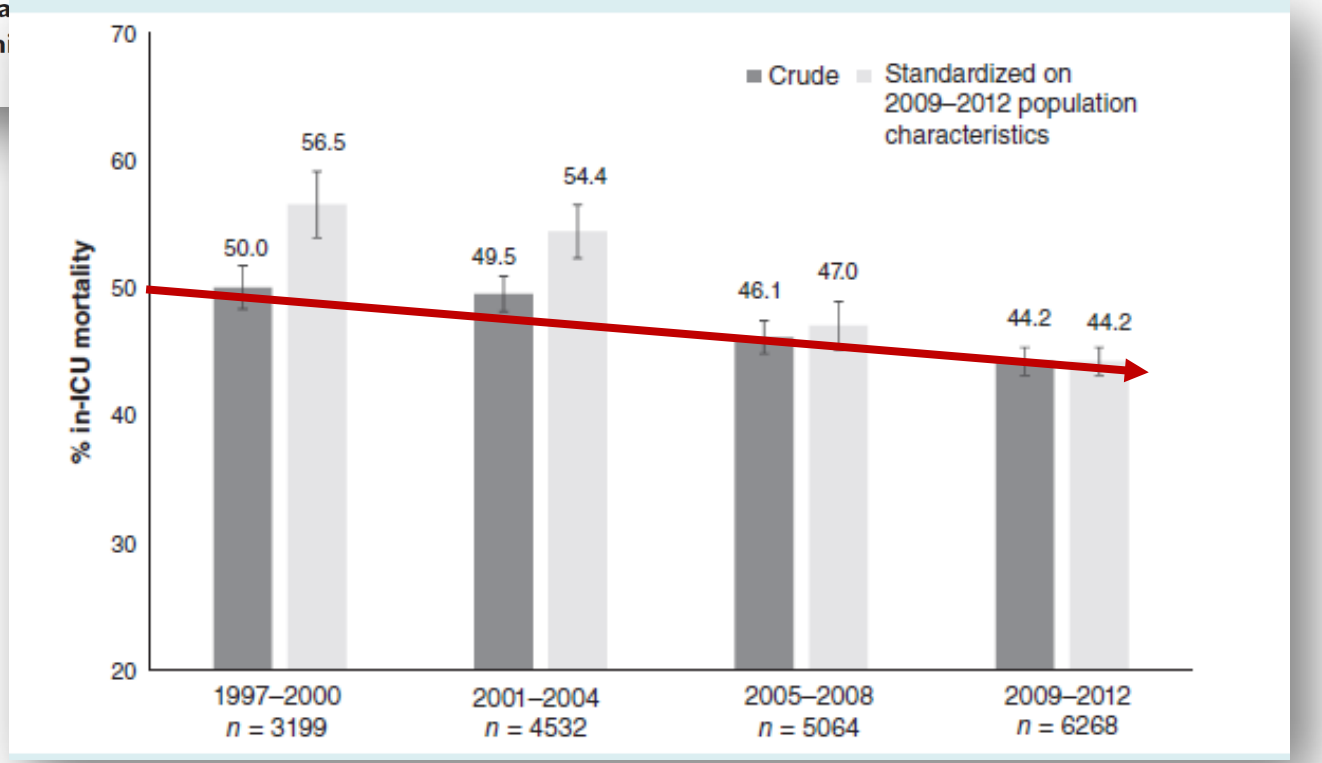

European Heart Journal
Acute Cardiovascular Care

ESC European Society of Cardiology

Original scientific paper

European Heart Journal: Acute Cardiovascular Care
2018, Vol. 7(1) 16–27

DOI: 10.1177/2048872617741735
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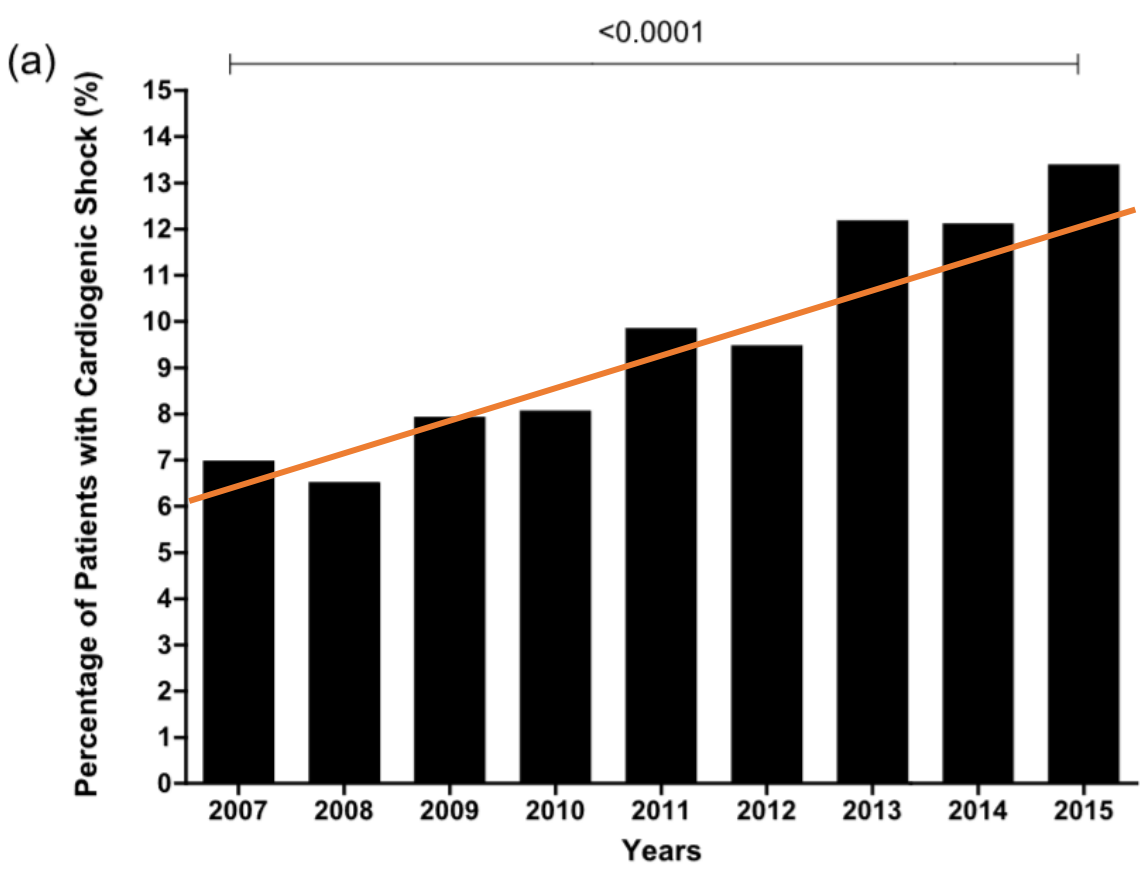
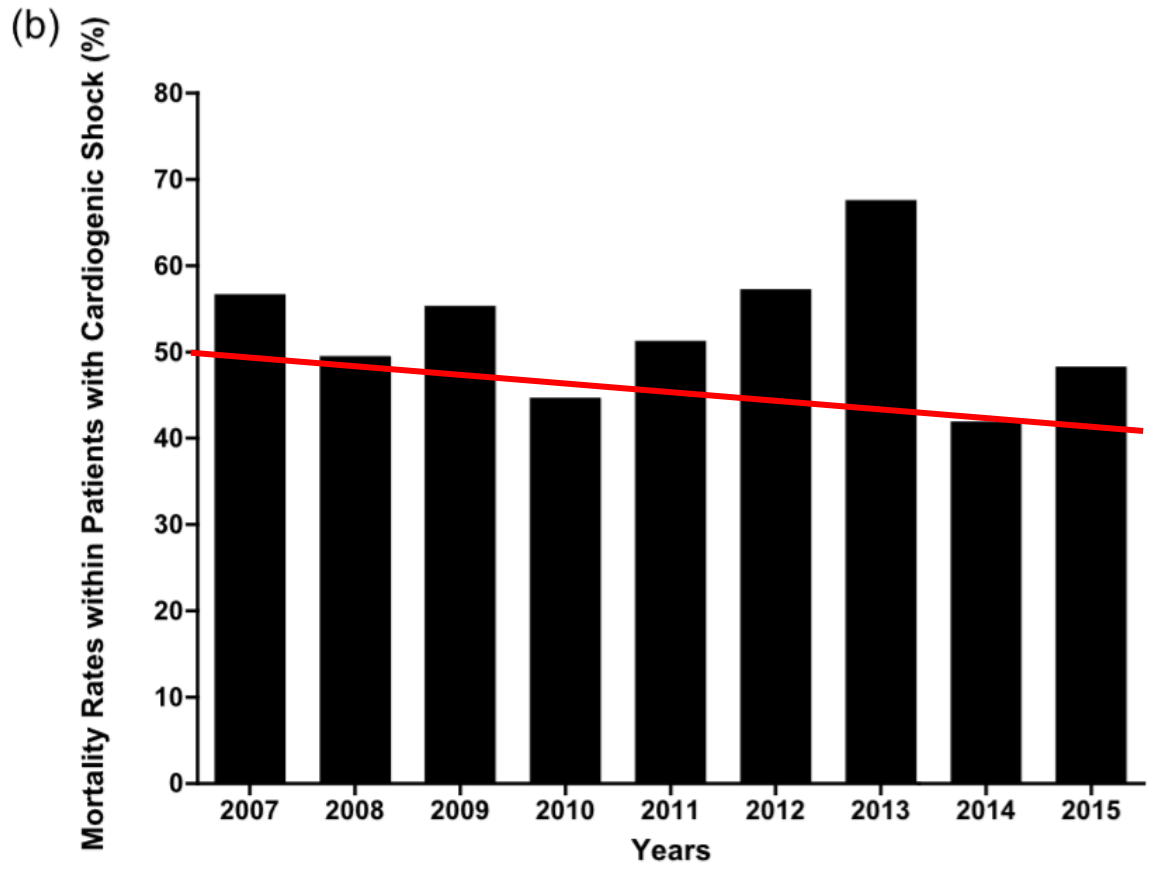
Contemporary trends in cardiogenic shock: Incidence, intra-aortic balloon pump utilisation and outcomes from the London Heart Attack Group

Krishnaraj S Rathod[1], Sudheer Koganti[1], M Bilal Iqbal[2], Ajay K Jain[1], Sundeep S Kalra[3], Zoe Astroulakis[4], Pitt Lim[4], Roby Rakhit[5], Miles C Dalby[2], Tim Lockie[5], Iqbal S Malik[7], Charles J Knight[1], Mark Whitbread[6], Anthony Mathur[1], Simon Redwood[8], Philip A MacCarthy[3], Alexander Sirker[1], Constantinos O'Mahony[1], Andrew Wragg[1] and Daniel A Jones[1]



	2007	2008	2009	2010	2011	2012	2013	2014	2015
Number of patients with Cardiogenic Shock	105	110	172	189	227	228	292	272	117
Total number of patients that year	1506	1691	2170	2345	2306	2407	2398	2246	874



	2007	2008	2009	2010	2011	2012	2013	2014	2015
Number of patients dying with cardiogenic shock	51	47	84	75	82	111	79	85	14
Total number of patients that year	90	95	152	168	160	194	117	203	29

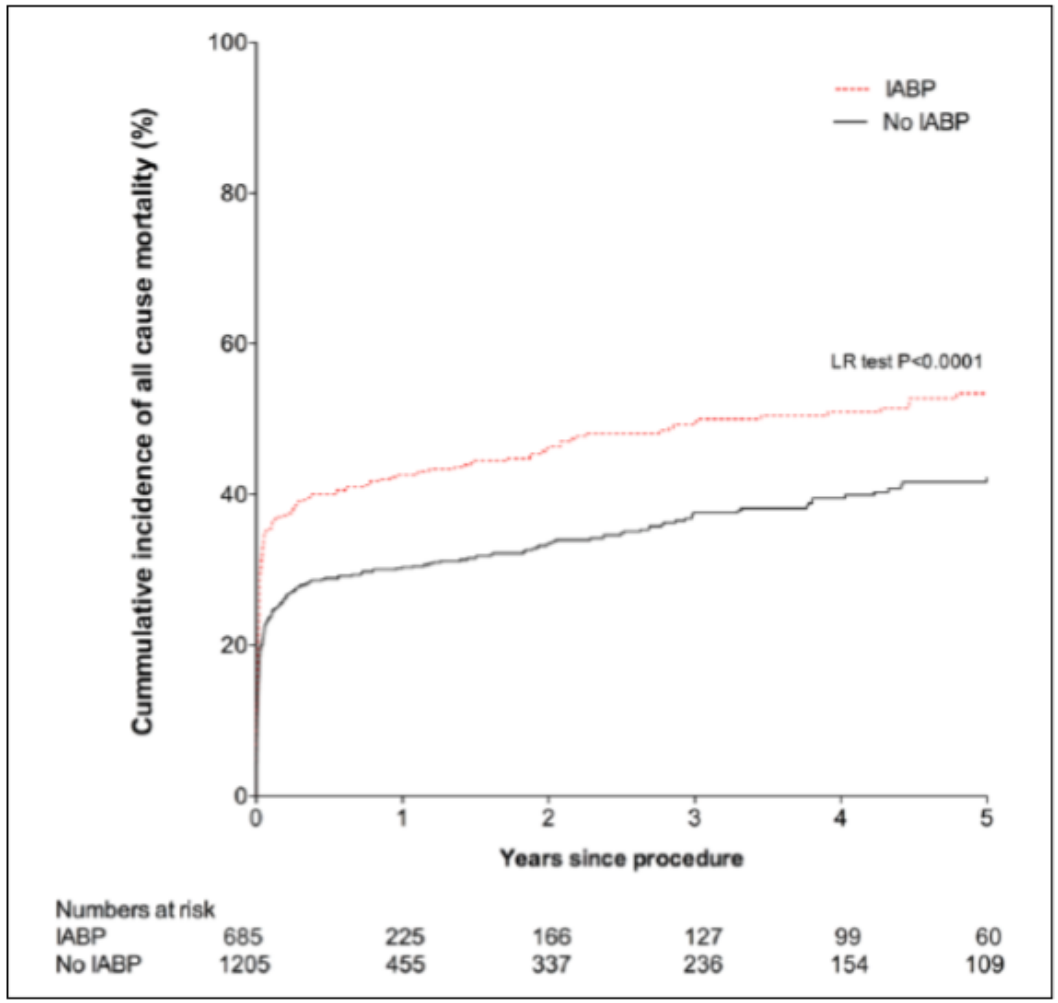


Figure 4. Kaplan-Meier curves showing cumulative probability of all-cause mortality after primary percutaneous coronary intervention (PCI) according to group. IABP: intra-aortic balloon pump; LR: log-rank.

Table 1. Pragmatic and Clinical Trial Definitions of CS

Clinical Definition	SHOCK Trial[9]*	IABP-SHOCK II[1]†	ESC HF Guidelines[15]
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Support to maintain SBP ≥90 mm Hg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of ≤2.2 $L \cdot min^{-1} \cdot m^{-2}$ AND PCWP ≥15 mm Hg	Clinical criteria: SBP <90 mm Hg for ≥30 min OR Catecholamines to maintain SBP >90 mm Hg AND Clinical pulmonary congestion AND Impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 mL/h, or lactate >2.0 mmol/L)	SBP <90 mm Hg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: Cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: Metabolic acidosis, elevated serum lactate, elevated serum creatinine

CI indicates cardiac index; CS, cardiogenic shock; ESC, European Society of Cardiology; HF, heart failure; IABP-SHOCK II, Intraaortic Balloon Pump in Cardiogenic Shock II; LV, left ventricular; MI, myocardial infarction; PCWP, pulmonary capillary wedge pressure; SBP, systolic blood pressure; and SHOCK, Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock.

*In setting of MI complicated by predominantly LV dysfunction.

†In setting of acute MI.

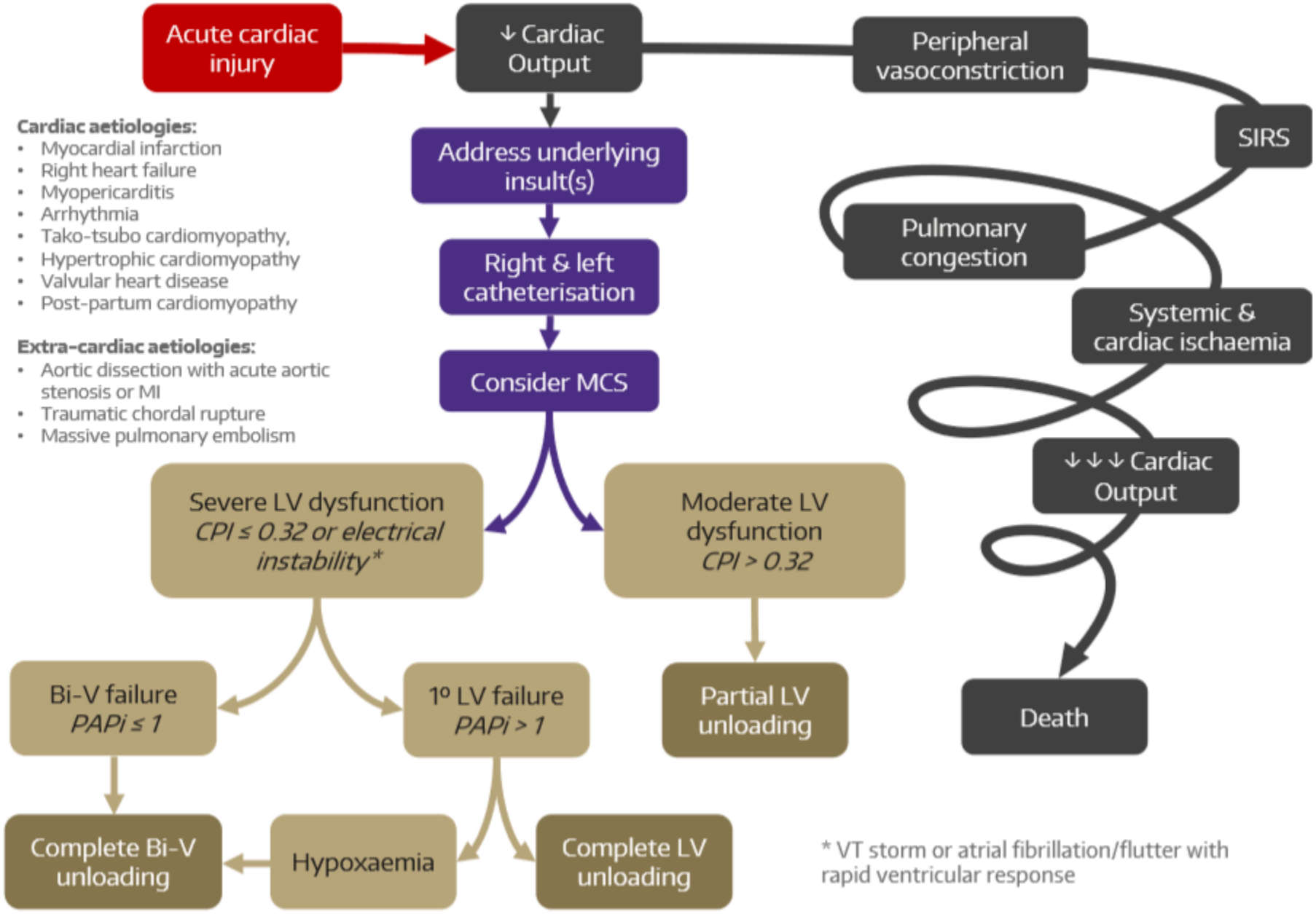
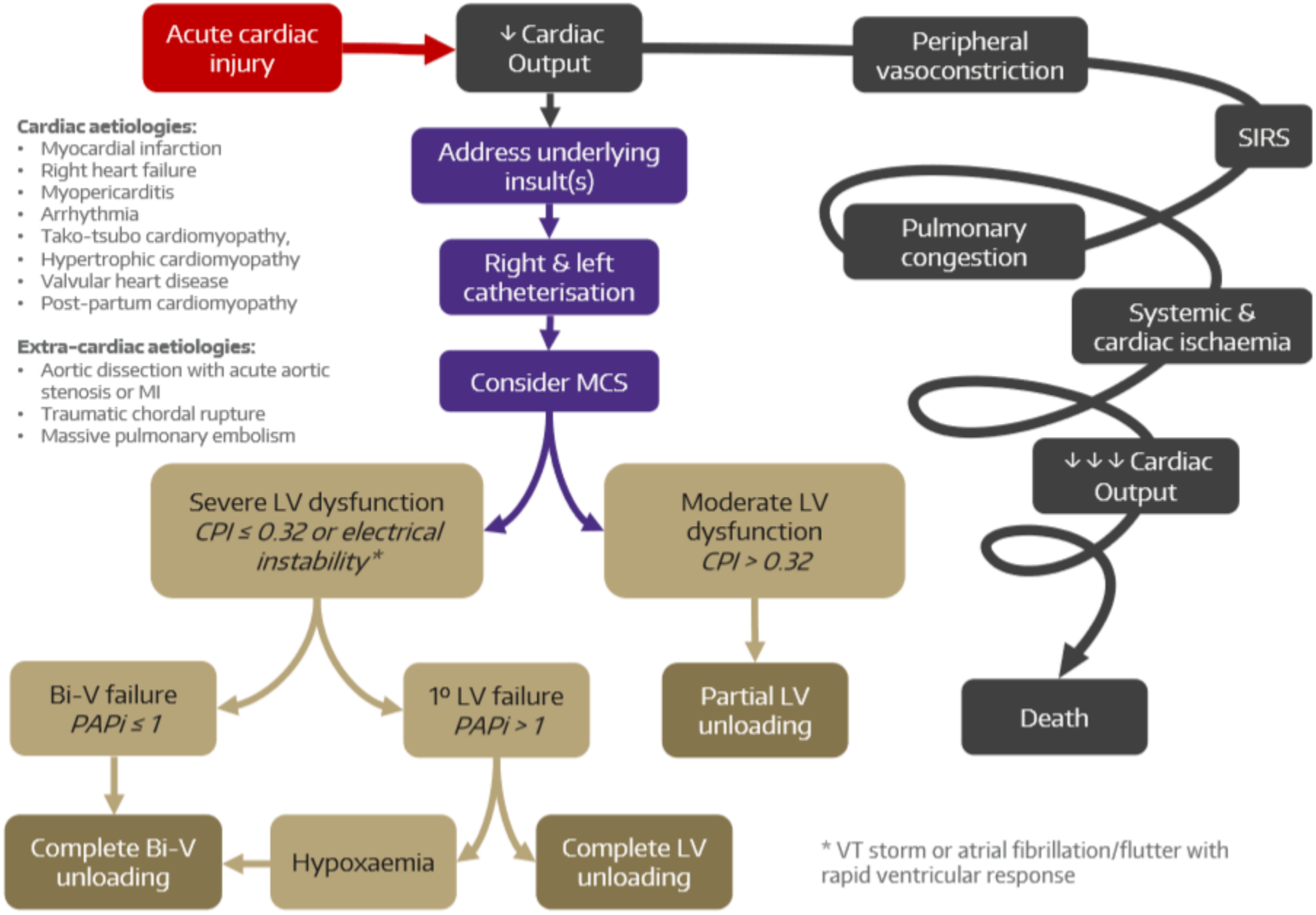


Figure 1

Conceptual algorithm for the management of cardiogenic shock (CS). The pathophysiology of CS is characterised by impaired cardiac output, SIRS, end-organ hypoperfusion and maladaptive compensatory mechanisms. Prevention of progressive cardiac and systemic compromise requires early recognition typically requiring right and left catheterisation and interruption of the vicious cycle by addressing underlying insults and initiation of mechanical circulatory support matched to the degree of clinical decompensation. Clinical indices such as CPI for LV function, PAPi for right ventricular function, and presence of malignant clinical features such as arrhythmia and hypoxaemia may help guide the decision for the most appropriate MSC modality. Bi-V, biventricular; CPI, Cardiac Power Index; LV, left ventricular; MCS, mechanical circulatory support; MI, myocardial infarction; PAPi, Pulmonary Artery Pulsatility Index; SIRS, systemic inflammatory response syndrome.



Vicious cycle of injury!

Cardiac and systemic decompensation

Maladaptive compensatory mechanisms

Prompt and appropriately tailored medical and mechanical support

1356 THE NEW ENGLAND JOURNAL OF MEDICINE Dec. 9, 1976

Classic mechanistic model based on hemodynamic subsets

Flow-based approach

MEDICAL PROGRESS

MEDICAL THERAPY OF ACUTE MYOCARDIAL INFARCTION BY APPLICATION OF HEMODYNAMIC SUBSETS (First of Two Parts)

JAMES S. FORRESTER, M.D., GEORGE DIAMOND, M.D., KANU CHATTERJEE, M.B., M.R.C.P., AND H. J. C. SWAN, M.D., PH.D.

VS

AHA SCIENTIFIC STATEMENT

Pressure-based approach

Phenotypic classification based on Blood Pressure

CLINICAL STATEMENTS AND GUIDELINES

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

Réanimation (2014) 23:548-557
DOI 10.1007/s13546-014-0915-8

RÉFÉRENTIEL / *GUIDELINES*

Prise en charge du choc cardiog

Management of Cardiogenic Shock in Ad

Recommandations formalisées d'experts

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Levy *et al. Annals of Intensive Care* (2015) 5:17
DOI 10.1186/s13613-015-0052-1

REVIEW

Experts' recommendat

ESC European Society of Cardiology
European Heart Journal (2017) **00**, 1–8
doi:10.1093/eurheartj/ehx393

ESC GUIDELINES

2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation – Web Addenda

rdial infarction
he European

Current Cardiology Reports (2019) 21:17
https://doi.org/10.1007/s11886-019-1102-3

MANAGEMENT OF ACUTE CORONARY SYNDROMES (H JNEID, SECTION EDITOR)

AHA SCIENTIFIC STATEMENT

CLINICAL STATEMENTS AND GUIDELINES

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

Ashish H. Shah | Rishi Puri | Ankur Kalra

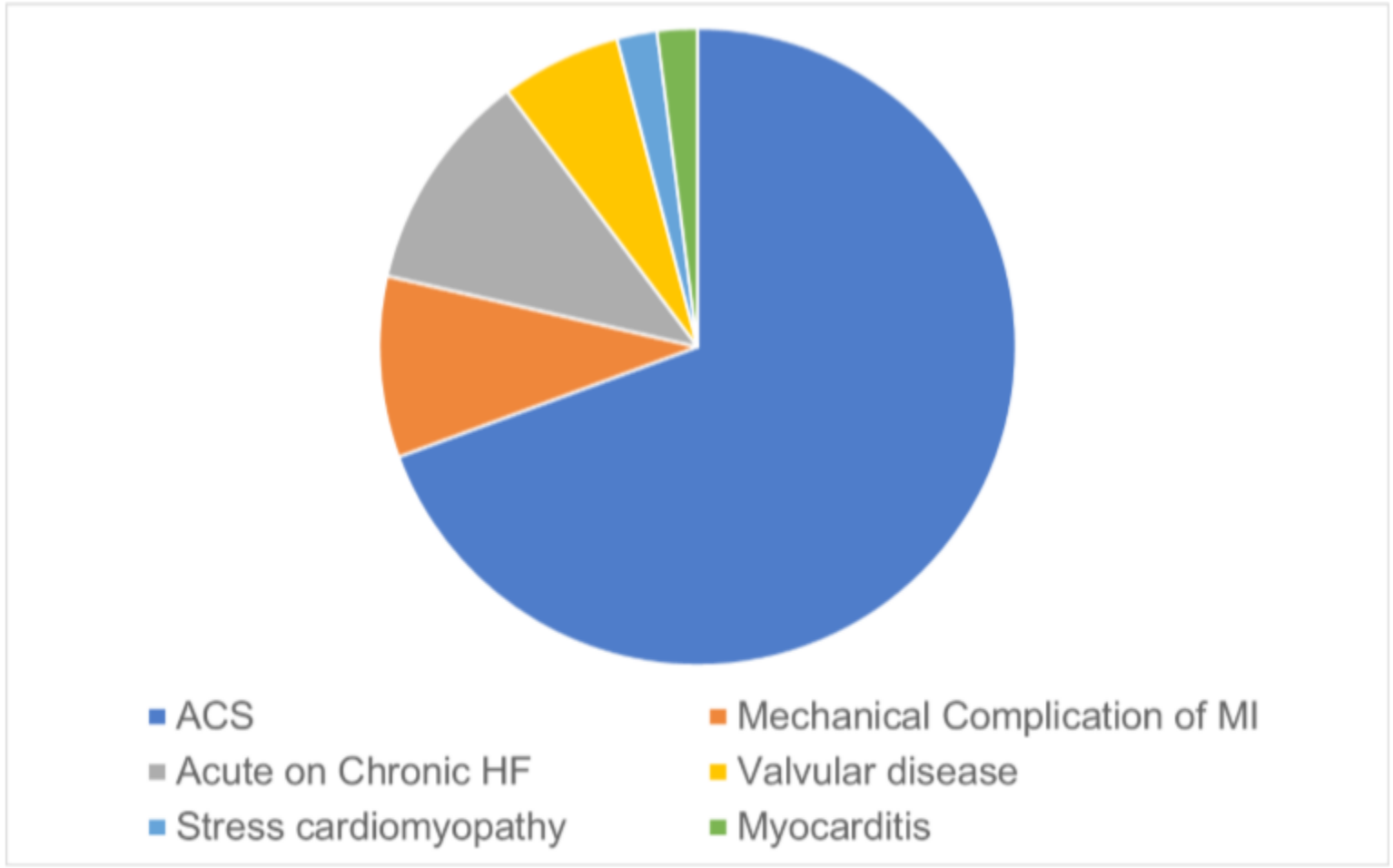
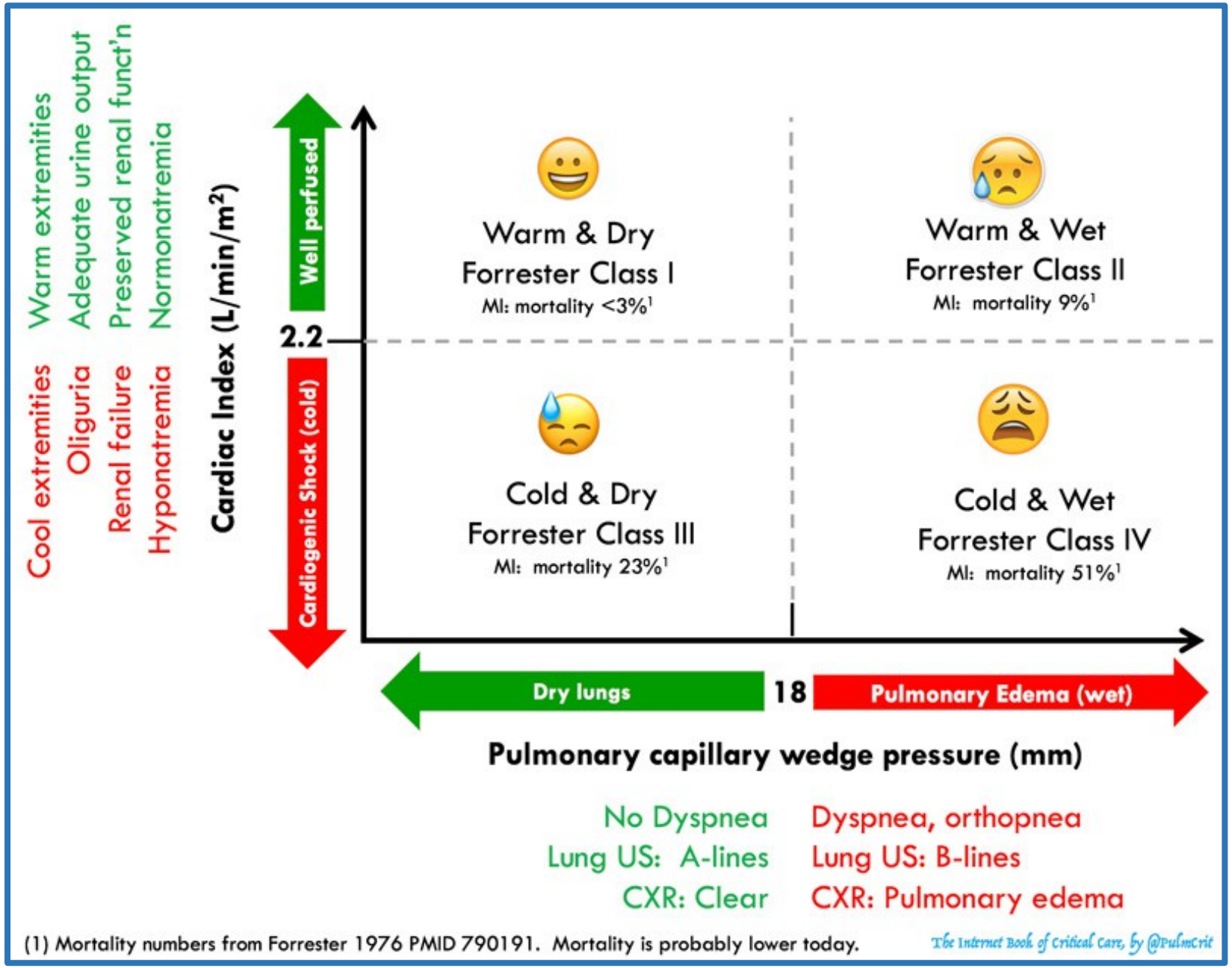


Figure 2 Causes of cardiogenic shock (adapted from Harjola *et al* [10]). ACS, acute coronary syndrome; MI, myocardial infarction.


Warm extremities
Adequate urine output
Preserved renal funct'n
Normonatremia
Cool extremities
Oliguria
Renal failure
Hyponatremia
Cardiac Index (L/min/m²)
Well perfused
2.2
Cardiogenic Shock (cold)
Warm & Dry
Forrester Class I
MI: mortality <3%[1]
Warm & Wet
Forrester Class II
MI: mortality 9%[1]
Cold & Dry
Forrester Class III
MI: mortality 23%[1]
Cold & Wet
Forrester Class IV
MI: mortality 51%[1]
Dry lungs
18
Pulmonary Edema (wet)
Pulmonary capillary wedge pressure (mm)
No Dyspnea
Lung US: A-lines
CXR: Clear
Dyspnea, orthopnea
Lung US: B-lines
CXR: Pulmonary edema
(1) Mortality numbers from Forrester 1976 PMID 790191. Mortality is probably lower today.
The Internet Book of Critical Care, by @PulmCrit

CURRENT OPINION

Clinical examination for diagnosing circulatory shock

Bart Hiemstra, Ruben J. Eck, Frederik Keus, and Iwan C.C. van der Horst

Purpose of review
In the acute setting of circulatory shock, physicians largely depend on clinical examination and basic laboratory values. The daily use of clinical examination for diagnostic purposes contrasts sharp with the limited number of studies. We aim to provide an overview of the diagnostic accuracy of clinical examination in estimating circulatory shock reflected by an inadequate cardiac output (*CO*).

Recent findings
Recent studies showed poor correlations between *CO* and mottling, capillary refill time or central-to-peripheral temperature gradients in univariable analyses. The accuracy of physicians to perform an educated guess of *CO* based on clinical examination lies around 50% and the accuracy for recognizing a low *CO* is similar. Studies that used predefined clinical profiles composed of several clinical examination signs show more reliable estimations of *CO* with accuracies ranging from 81 up to 100%.

Summary
Single variables obtained by clinical examination should not be used when estimating *CO*. Physician's educated guesses of *CO* based on unstructured clinical examination are like the 'flip of a coin'. Structured clinical examination based on combined clinical signs shows the best accuracy. Future studies should focus on using a combination of signs in an unselected population, eventually to educate physicians in estimating *CO* by using predefined clinical profiles.

Keywords
cardiac output, circulatory shock, clinical examination, critical illness, diagnostic accuracy, physical examination, shock

Table 2. Physician's capacity to estimate cardiac output based on cl

Author, year	Patients	Setting	Varia Classification	Results Estimation	Diagnostic accuracy for low *CO* (95% CI)
Connors *et al.* 1983 [13]	62[a]	ICU	*CI* categorical: <2.5; 2.5–3.5; >3.5	44% correct estimation	Sens 58% (45–68%); Spec 60% (48–71%) PPV 58% (49–65%); NPV 60% (52–67%) LR+ 1.43 (1.02–2.00); LR– 0.71 (0.51–0.98)
Eisenberg *et al.* 1984 [14]	97	ICU	*CO* categorical: <4.5; 4.5–7.5; >7.5	51% correct estimation	Sens 71% (54–85%); Spec 56% (43–69%) PPV 48% (39–57%); NPV 78% (66–86%) LR+ 1.64 (1.15–2.33); LR– 0.51 (0.29–0.89)
Tuchschmidt *et al.* 1987 [15]	35	ICU	*CO* continuous	$r=0.72$	–
Connors *et al.* 1990 [17]	461	ICU	*CI* dichotomous: <2.2; ≥2.2 *CI* continuous	64% correct estimation Mean CI-difference in *CI* = 1.0 ± 0.9	Sens 49% (40–57%); Spec 70% (65–75%) PPV 43% (38–49%); NPV 74% (71–77%) LR+ 1.62 (1.28–2.05); LR– 0.73 (0.62–0.87)
Celoria *et al.* 1990[b] [16]	114	Surgical ICU	*CO* categorical: <4; 4–8; >8	51% correct estimation $r=0.47$	Sens 67% (30–93%); Spec 80% (71–87%) PPV 22% (14–34%); NPV 97% (92–99%) LR+ 3.33 (1.83–6.07); LR– 0.42 (0.16–1.05)
Steingrub *et al.* 1991[b] [53]	152	Surgical and medical ICU	*CO* categorical: <4; 4–8; >8	51% correct estimation	Sens 54% (37–70%); Spec 73% (63–81%) PPV 40% (31–51%); NPV 82% (76–87%) LR+ 1.96 (1.29–2.98); LR– 0.64 (0.44–0.91)
Mimoz *et al.* 1994 [18]	112	ICU	Combinations of *CI*, PAOP and SVRI	56% correct estimation	–
Staudinger *et al.* 1998 [54]	149	ICU	*CI* categorical: <2.0; 2.0–4.0; >4.0	62% correct estimation	–
Rodriguez *et al.* 2000 [55]	33	ED + respiratory distress or hypotension	*CI* categorical: <2.6; 2.6–4.0; >4.0.	$\kappa 1 = -0.04$ (95% CI–0.31–0.24) $\kappa 2 = 0.07$ (95% CI –0.17–0.31)	–
Linton *et al.* 2002 [56]	50	Post cardiac surgery	*CI* categorical: <1.9; 1.9–3.5; >3.5	54% correct estimation	Sens 42% (15–72%); Spec 74% (57–87%) PPV 33% (18–54%); NPV 80% (71–87%) LR+ 1.58 (0.67–3.72); LR– 0.79 (0.47–1.32)
Iregui *et al.* 2003 [57]	105	ICU	*CI* categorical: <2.5; 2.5–4.5; >4.5	44% correct estimation	–
Veale *et al.* 2005 [58]	68	ICU	*CI* categorical: <2.5; 2.5–4.2; >4.5	42% correct estimation	Sens 22% (6–48%); Spec 66% (51–79%) PPV 19% (8–38%); NPV 70% (63–76%) LR+ 0.65 (0.25–1.68); LR– 1.18 (0.86–1.62)
Rodriguez *et al.* 2006 [59]	31	ED + endotracheal intubation	*CI* categorical: ranges not specified	$\kappa = 0.57$ (95% CI 0.36–0.77)	–
Nowak *et al.* 2011 [60]	38	ED + respiratory distress	*CO* categorical <4.0; 4.0–8.0; >8.0	50% correct estimation $\kappa = -0.02$ (95% CI –0.25–0.20)	Sens 33% (4–78%); Spec 63% (44–79%) PPV 14% (5–36%); NPV 83% (73–90%) LR+ 0.89 (0.26–3.00); LR– 1.07 (0.57–2.00)
Duan *et al.* 2014 [61]	132	ICU	*CI* categorical: <3; 3–5; >5	50% correct estimation	–
Perel *et al.* 2016 [62▪▪]	206[a]	ICU	*CO* continuous	Percentage error = 66% Absolute mean difference in *CO* = –1.5 ± 2.2	–

Table 3. Combined signs of clinical examination for estimation of *CO*

Author, year	Patients	Population	Variables of interest: Clinical profile	Variables of interest: Clinical profile based on	*CO*-measurement	Results
Combined clinical profiles						
Ramo *et al.* 1970 [63]	98	AMI	I (normal *CI*): no signs of HF II (normal *CI*): mild-to-moderate HF III (low *CI*): overt pulmonary edema IV (low *CI*): cardiogenic shock	Mean arterial pressure, cool extremities, urine output, mental status, third heart sound gallop rhythm and rales	PAC, indicator-dilution technique	I (normal *CI*): 23 of 45 (51%) II (normal *CI*): 19 of 30 (63%) III (low *CI*): 10 of 10 (100%) IV (low *CI*): 13 of 13 (100%)
Forrester *et al.* 1977 [64]	200	AMI	I (normal *CI*): no pulmonary congestion or peripheral hypoperfusion II (normal *CI*): pulmonary congestion only III (low *CI*): hypoperfusion only IV (low *CI*): both	Heart rate, blood pressure, cool extremities, urine output and mental status	PAC, thermodilution	Overall: 81% correct estimations of *CI* I & II (normal *CI*): 84 of 95 (88%) III & IV (low *CI*): 76 of 105 (72%)
Grissom *et al.* 2009 [65]	405	ALI	I: All three clinical signs aberrant II: Any one clinical sign aberrant	Capillary refill time, knee mottling and cool extremities	PAC, thermodilution	92% correct estimations of *CI* in class I: Sens 12% (3–28%); Spec 98% (97–99%) PPV 40% (17–69%); NPV 93% (92–93%) LR+ 7.52 (2.23–25.3); LR– 0.89 (0.79–1.01) 75% correct estimations of *CI* in class II: Sens 52% (34–69%); Spec 78% (73–82%) PPV 17% (12–23%); NPV 95% (93–96%) LR+ 2.31 (1.58–3.38); LR– 0.62 (0.44–0.89)
Multivariable analysis						
Sasse *et al.* 1996 [66]	23[a]	ICU patients	*CO* continuous	Heart rate, respiratory rate, mean arterial pressure and temperature	PAC, thermodilution	Heart rate: $R^2 = 0.05$ Respiratory rate: $R^2 = 0.14$ Mean arterial pressure: $R^2 = 0.03$

[a]=repeated measurements in each patient.

ALI, acute lung injury; AMI, acute myocardial infarction; *CI*, cardiac index (l/min/m^2); *CO*, cardiac output (l/min); HF, heart failure; LR–, negative likelihood ratio; LR+, positive likelihood ratio; NPV, negative predictive value; PAC, pulmonary artery catheter; PPV, positive predictive value; Sens, sensitivity; Spec, specificity.

KEY POINTS

- Clinical examination findings are poorly associated with *CO* in single-variable and multivariable analyses.
- The physician's accuracy to subjectively estimate *CO* based on clinical examination equals the flip of a coin.
- Physicians are likely insufficiently capable to recognize a low *CO* by using clinical examination.
- Estimating *CO* by using a predefined combination of clinical signs seems the most accurate method to diagnose shock.
- Future studies on estimating *CO* should be conducted in a representative population, use standardized clinical examination and use appropriate statistical indices of diagnostic accuracy.

- **Interrupt cellular, metabolic and inflammatory pathways**

« The vicious Cycle »

General supportive measures, pharmacological and MCS

Must focus on quantitative and modifiable parameters of CS from invasive procedures

- **Prompt etiological tailored treatment**

- **A compromise best tissue perfusion/lowest MVO2**

Open access Heart failure and cardiomyopathies

openheart

Cardiogenic shock: evolving definitions and future directions in management

Tara L Jones,[1] Kenta Nakamura,[2] James M McCabe[2]

Flow-based approach

CPI = CI x MAP

Cardiac Power index

Pressure-based approach

Table 4. Mechanism of Action and Hemodynamic Effects of Common Vasoactive Medications in CS

Medication	Usual Infusion Dose	Receptor Binding				Hemodynamic Effects
		α_1	β_1	β_2	Dopamine	
Vasopressor/inotropes						
Dopamine	0.5–2 μg·kg^{-1}·min^{-1}	–	+	–	+++	↑CO
	5–10 μg·kg^{-1}·min^{-1}	+	+++	+	++	↑↑CO, ↑SVR
	10–20 μg·kg^{-1}·min^{-1}	+++	++	–	++	↑↑SVR, ↑CO
Norepinephrine	0.05–0.4 μg·kg^{-1}·min^{-1}	++++	++	+	–	↑↑SVR, ↑CO
Epinephrine	0.01–0.5 μg·kg^{-1}·min^{-1}	++++	++++	+++	–	↑↑CO, ↑↑SVR
Phenylephrine	0.1–10 μg·kg^{-1}·min^{-1}	+++	–	–	–	↑↑SVR
Vasopressin	0.02–0.04 U/min	Stimulates V_1 receptors in vascular smooth muscle				↑↑SVR, ↔PVR
Inodilators						
Dobutamine	2.5–20 μg·kg^{-1}·min^{-1}	+	++++	++	–	↑↑CO, ↓SVR, ↓PVR
Isoproterenol	2.0–20 μg/min	–	++++	+++	–	↑↑CO, ↓SVR, ↓PVR
Milrinone	0.125–0.75 μg·kg^{-1}·min^{-1}	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Enoximone	2–10 μg·kg^{-1}·min^{-1}	PD-3 inhibitor				↑CO, ↓SVR, ↓PVR
Levosimendan	0.05–0.2 μg·kg^{-1}·min^{-1}	Myofilament Ca^{2+} sensitizer, PD-3 inhibitor				↑CO, ↓SVR, ↓PVR

CO indicates cardiac output; CS, cardiogenic shock; PD-3, phosphodiesterase-3; PVR, pulmonary vascular resistance; and SVR, systemic vascular resistance.

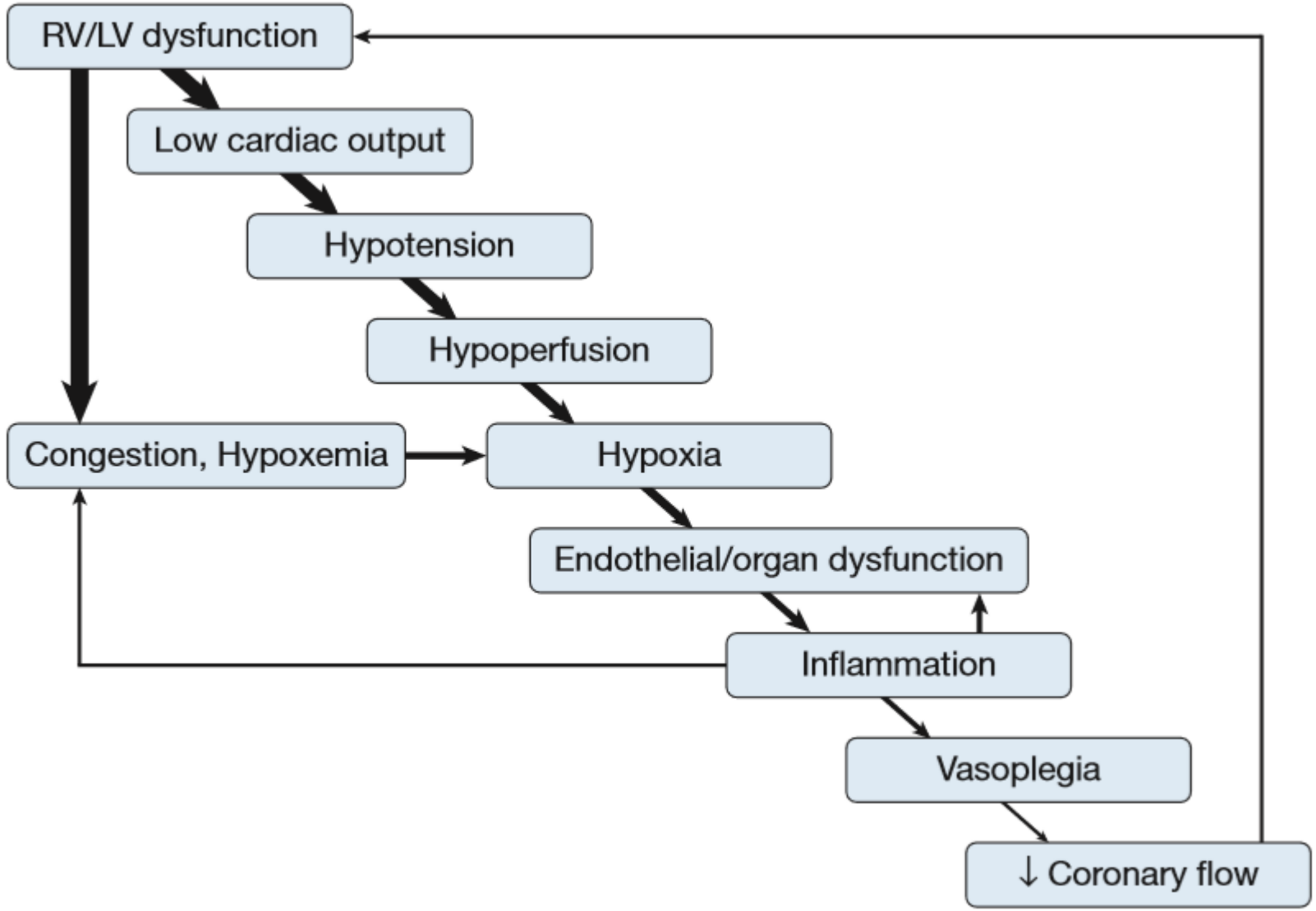


Figure 1 – *Cardiogenic shock downward cascade. The thickness of the arrows schematically represents the strength of the linkage depending on the severity and duration of the disorder and on the patient's physiologic reserve. LV = left ventricle; RV = right ventricle.*

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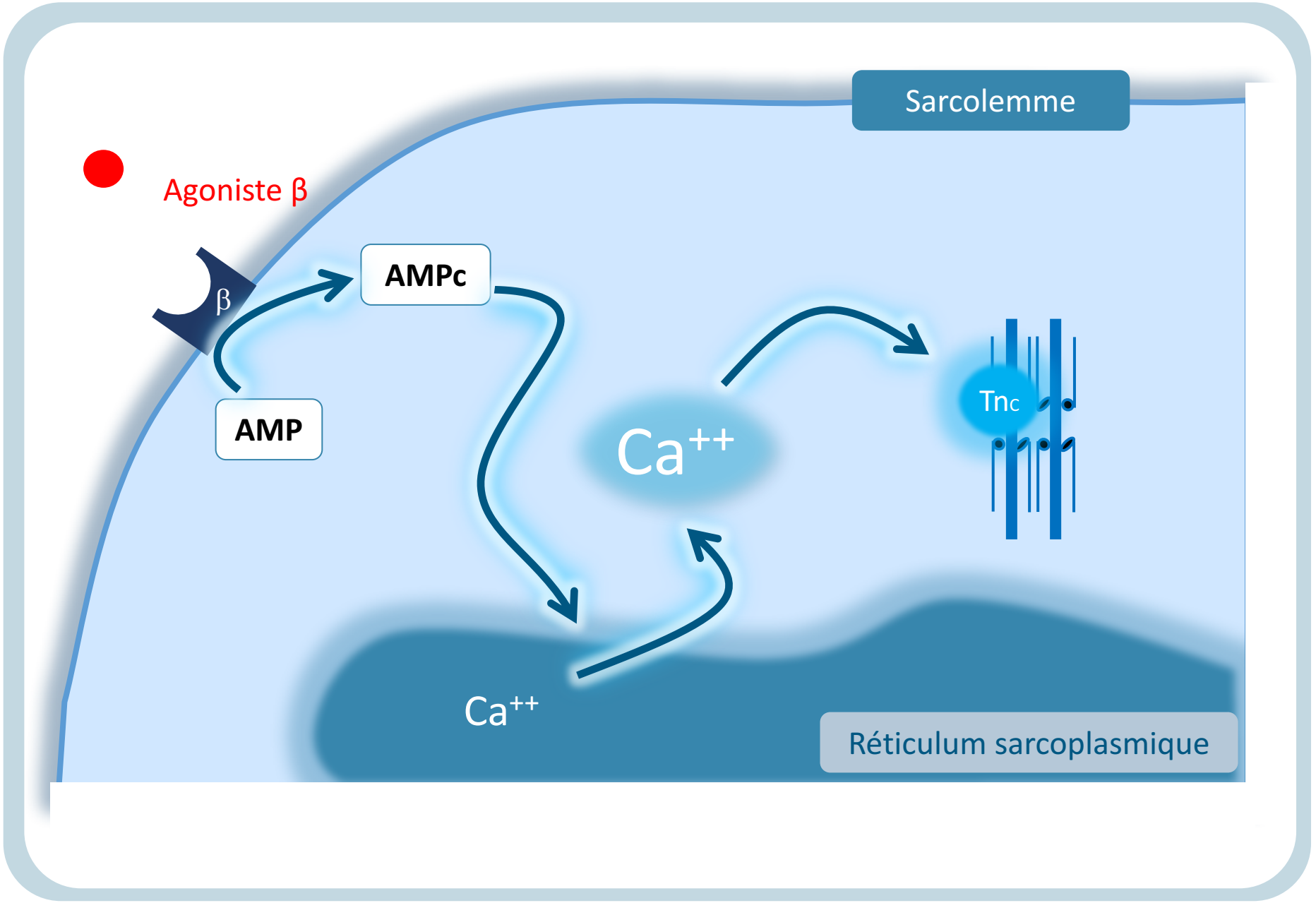
Schumann J, Henrich EC, Strobl H, Prondzinsky R, Weiche S, Thiele H, Werdan K, Frantz S, Unverzagt S


Sarcolemme
Agoniste β
β
AMPc
AMP
Ca++
Tnc
Ca++
Réticulum sarcoplasmique

Comparison of the Occurrence of Ventricular Arrhythmias in Patients With Acutely Decompensated Congestive Heart Failure Receiving *Dobutamine* Versus *Nesiritide* Therapy

Am J Cardiol 2001;88:35–39

Andrew J. Burger, MD, Uri Elkayam, MD, Mathew T. Neibaur, MD, Herbert Haught, MD, Jalal Ghali, MD, Darlene P. Horton, MD, and Doron Aronson, MD

309 pts with decompensated AHF
standard therapy or neseritide 0.015 µkd or neseritide 0.030 µkd
(dobutamine in 58 pts)

Arrhythmic events by initial treatment assignment during study drug

Characteristics	Dobutamine (n = 58)	Nesiritide (µg/kg/min) 0.015 n = 103	Nesiritide (µg/kg/min) 0.030 n = 100	Overall p Value*
Cardiac arrest	3 (5%)	0 (0%)	0 (0%)	0.011
VT	13 (22%)	17 (17%)	8 (8%)	0.032
Nonsustained	10 (17%)	17 (17%)	6 (6%)	0.029
Sustained	4 (7%)	0 (0%)	2 (2%)	0.014

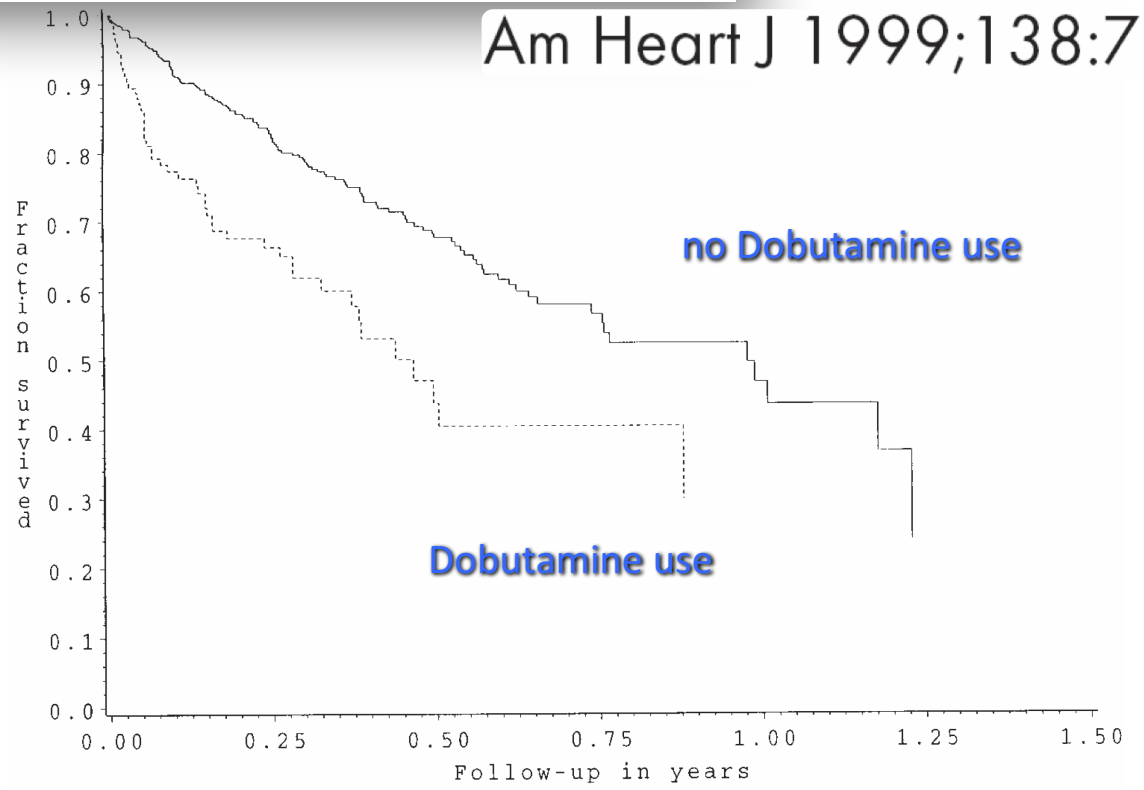
Continuous intravenous dobutamine is associated with an increased risk of death in patients with advanced heart failure: Insights from the Flolan International Randomized Survival Trial (FIRST)

Christopher M. O'Connor, MD, FACC,[a] Wendy A. Gattis, PharmD,[a] Barry F. Uretsky, MD, FACC,[b] Kirkwood F. Adams Jr, MD, FACC,[c] Steven E. McNulty, MS,[a] Steven H. Grossman, MD,[d] William J. McKenna, MD, FACC,[e] Faiez Zannad, MD,[f] Karl Swedberg, MD, FACC,[g] Mihai Gheorghiade, MD, FACC,[h] and Robert M. Califf, MD, FACC,[a] for the FIRST Investigators *Durham, Chapel Hill, and Research Triangle Park, NC; Galveston, Texas; London, United Kingdom; Nancy, France; Goteborg, Sweden; and Chicago, Ill*

471 pts from the FIRST study

480 pts with dobutamine at randomization vs 91 pts without dobutamine

Am Heart J 1999;138:78-86



Use and impact of inotropes and vasodilator therapy in hospitalized patients with severe heart failure

Uri Elkayam, MD,[a] Gudaye Tasissa, PhD,[b] Cynthia Binanay, RN, BSN,[b] Lynne W. Stevenson, MD,[c] Mihai Gheorghiade, MD,[d] J. Wayne Warnica, MD,[e] James B. Young, MD,[f] Barry K. Rayburn, MD,[g] Joseph G. Rogers, MD,[b] Teresa DeMarco, MD,[h] and Carl V. Leier, MD[i] *Los Angeles and San Francisco, CA; Durham, NC; Boston, MA; Chicago, IL; Calgary, Alberta, Canada; Cleveland and Columbus, OH; and Birmingham, AL*

Am Heart J 2007;153:98-104

433 pts from the ESCAPE study (decompensated HF)
Mortality at 6 months

Table IV. Propensity score–adjusted multivariable risk factors for mortality

433 pts	HR (95% CI)	P
IV vasoactive therapy		
Vasodilator	1.39 (0.64-3.00)	.403
Inotrope	2.14 (1.10-4.15)	.024
Both	4.81 (2.34-9.90)	<.001
Sodium	0.92 (0.87-0.98)	.006
RAP*	1.03 (1.01-1.05)	.008
BNP (100 U)	1.02 (1.01-1.04)	.006
SUN (10 U)	1.13 (1.02-1.26)	.018
Age (10 y)	1.21 (0.97-1.51)	.087
Ischemic etiology	1.58 (0.91-1.73)	.104
Systolic blood pressure <100 (dichotomous)	1.60 (0.99-2.58)	.055

Levy *et al. Annals of Intensive Care* (2015) 5:17
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Annals of Intensive Care
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REVIEW Open Access

Experts' recommendations for the management of adult patients with cardiogenic shock

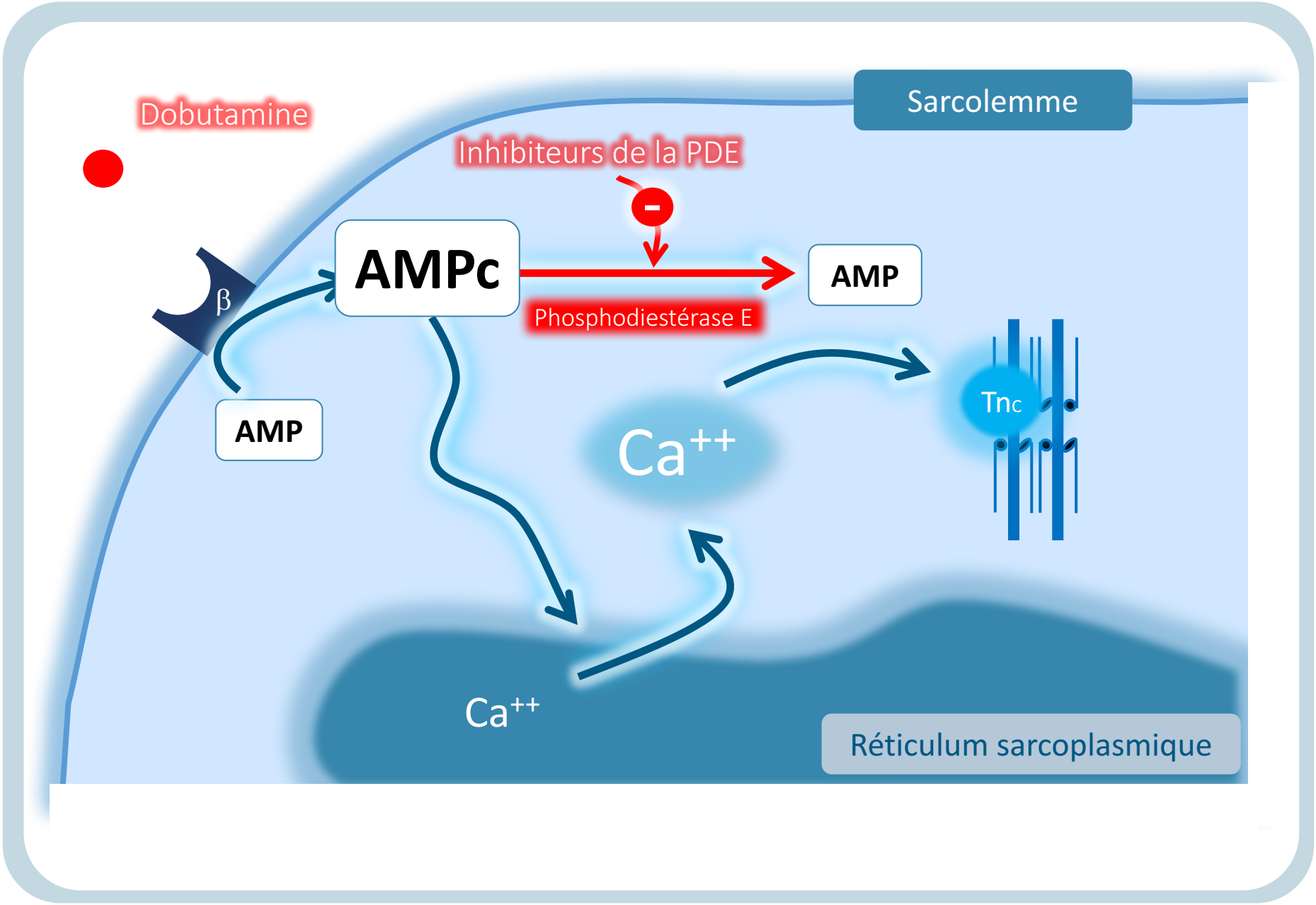
Bruno Levy[1*], Olivier Bastien[2], Karim Bendjelid[3], Alain Cariou[4], Tahar Chouihed[5], Alain Combes[6], Alexandre Mebazaa[7], Bruno Megarbane[8], Patrick Plaisance[9], Alexandre Ouattara[10], Christian Spaulding[11], Jean-Louis Teboul[12], Fabrice Vanhuyse[13], Thierry Boulain[14] and Kaldoun Kuteifan[15]

Area 4: management of blood pressure and cardiac output in intensive care

1- A MAP of at least 65 mmHg should be reached using inotropic treatment and/or vasopressor

4- Dobutamine should be used to treat low cardiac output in cardiogenic shock (strong agreement).

Dobutamine must be used at the lowest possible dose, starting at 2 µg/kg/min. Its titration is based on cardiac index and SvO_2. Dopamine must never be used.


Sarcolemme
Dobutamine
Inhibiteurs de la PDE
AMPc
AMP
Phosphodiestérase E
β
AMP
Ca++
TnC
Ca++
Réticulum sarcoplasmique

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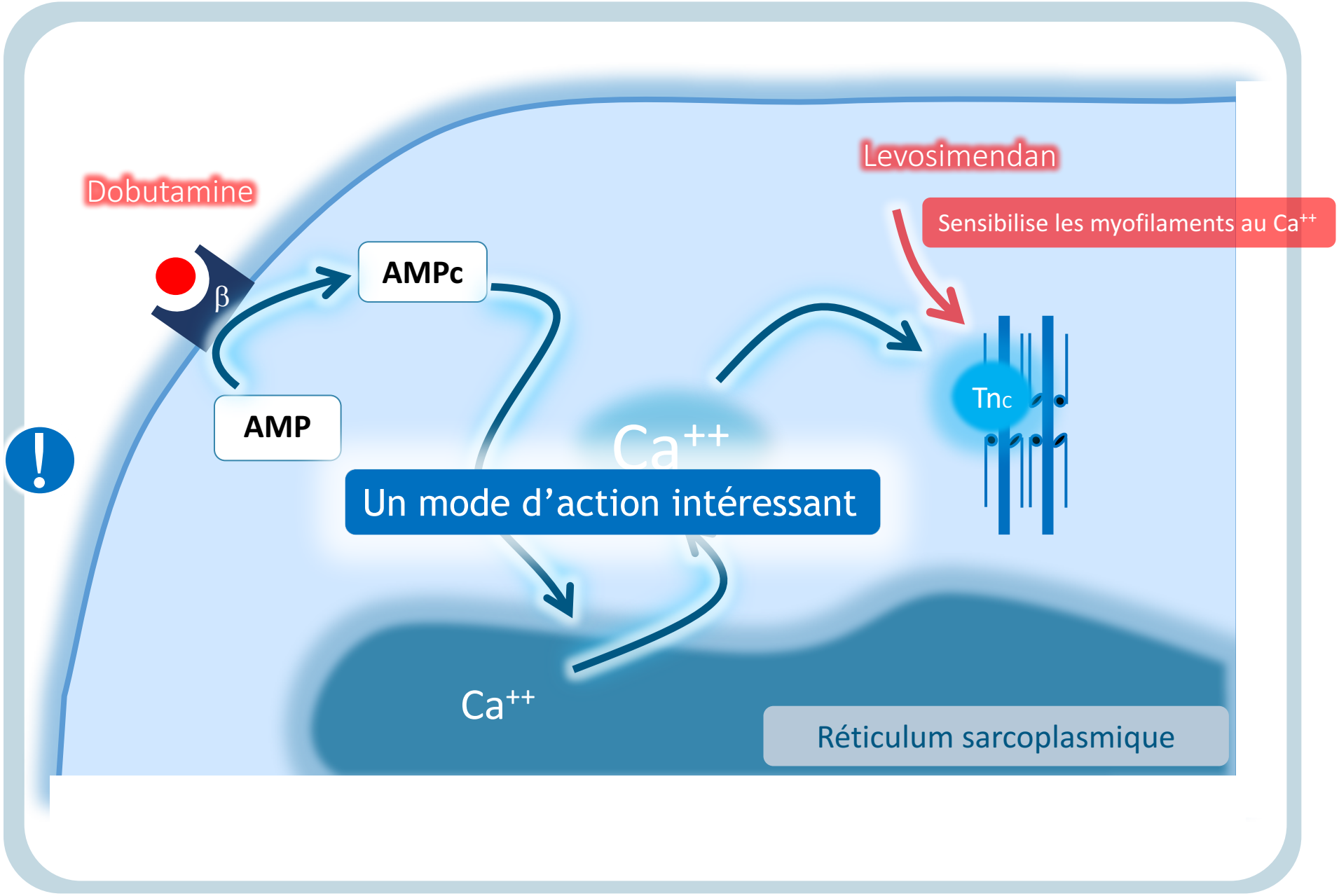
1- A MAP of at least 65 mmHg should be reached using inotropic treatment and/or vasopressor

5- Phosphodiesterase inhibitors should not be used first-line (strong agreement).

tachycardia, and hyperlactatemia (weak agreement).

4- Dobutamine should be used to treat low cardiac output in cardiogenic shock (strong agreement).

5- Phosphodiesterase inhibitors or levosimendan should not be used first-line (strong agreement).


Dobutamine
β
AMPc
AMP
Levosimendan
Sensibilise les myofilaments au Ca++
Tnc
Ca++
Un mode d'action intéressant
Ca++
Réticulum sarcoplasmique

Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure

The SURVIVE Randomized Trial

JAMA. 2007;297:1883-1891

Alexandre Mebazaa, MD, PhD
Markku S. Nieminen, MD, PhD
Milton Packer, MD
Alain Cohen-Solal, MD, PhD
Franz X. Kleber, MD
Stuart J. Pocock, PhD
Roopal Thakkar, MD
Robert J. Padley, MD
Pentti Põder, MD, PhD
Matti Kivikko, MD, PhD
for the SURVIVE Investigators

Context Because acute decompensated heart failure causes substantial morbidity and mortality, there is a need for agents that at least improve hemodynamics and relieve symptoms without adversely affecting survival.

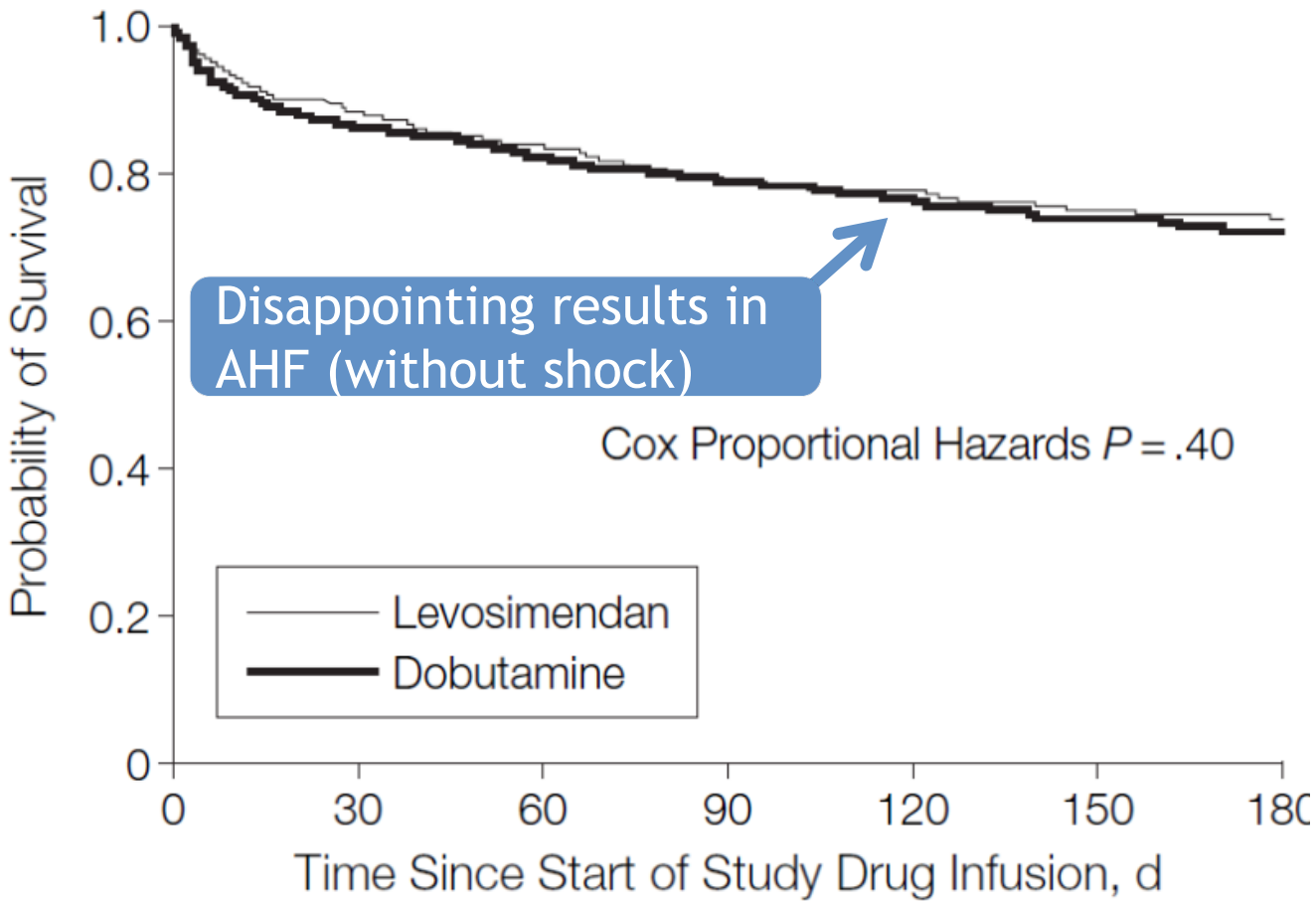
Objective To assess the effect of a short-term intravenous infusion of levosimendan or dobutamine on long-term survival.

Design, Setting, and Patients The Survival of Patients W in Need of Intravenous Inotropic Support (SURVIVE) study was blind trial comparing the efficacy and safety of intravenous lev mine in 1327 patients hospitalized with acute decompensate quired inotropic support. The trial was conducted at 75 centers in were randomized between March 2003 and December 2004.

Interventions Intravenous levosimendan (n=664) or int (n=663).

Main Outcome Measure All-cause mortality at 180 days.

1 327 patients with severe congestive heart failure
dobutamine vs. levosimendan



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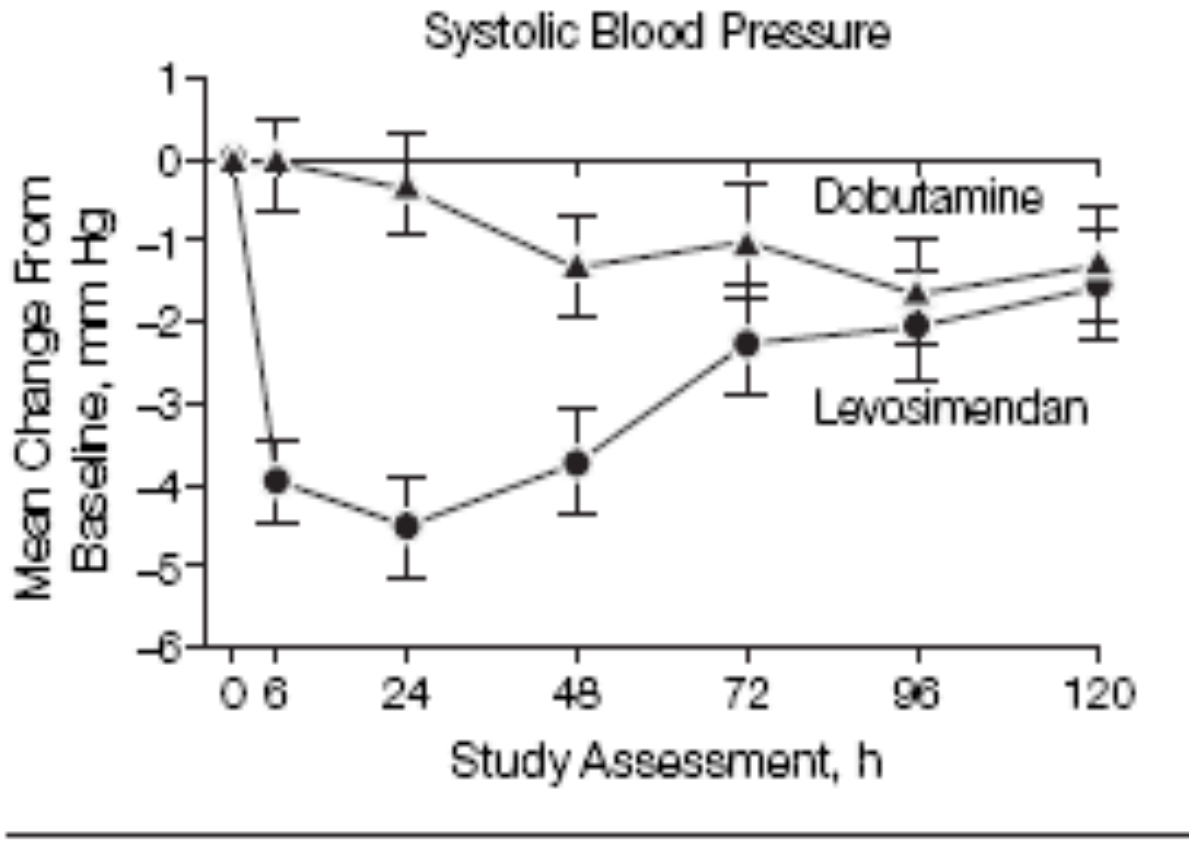

Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure

The SURVIVE Randomized Trial

JAMA. 2007;297:1883-1891

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Interventions Intravenous levosi
(n=663).

Main Outcome Measure All-cau

1 327 patients with severe congestive heart failure
dobutamine vs. levosimendan

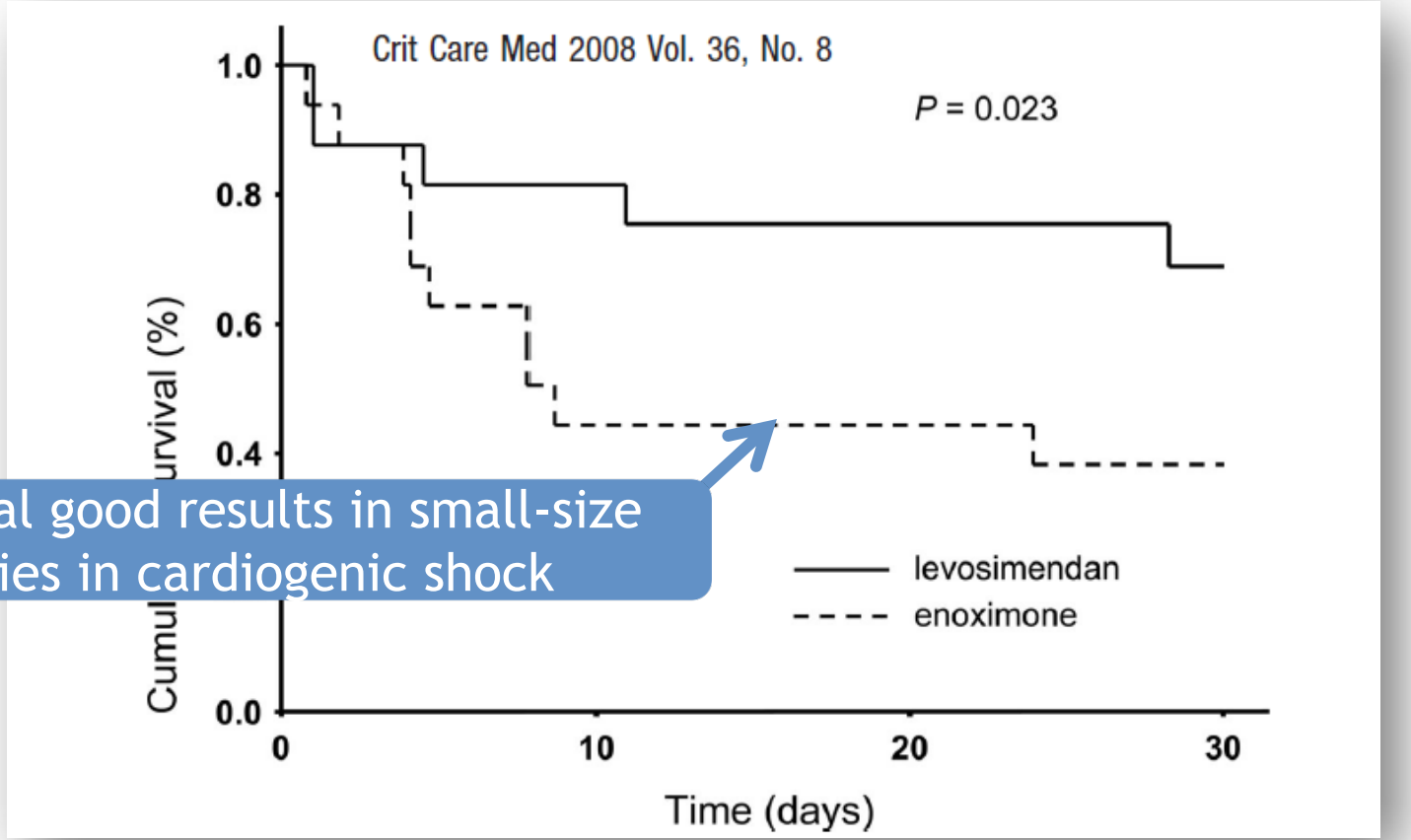
Table 5. Treatment-Emergent Adverse Events*

	No. (%) of Patients		
	Levosimedan (n = 660)	Dobutamine (n = 660)	*P* Value†
Any adverse event	518 (78.5)	502 (76.1)	.32
Any serious adverse event‡	195 (29.5)	217 (32.9)	.21
Hypotension	102 (15.5)	92 (13.9)	.48
Cardiac failure§	81 (12.3)	112 (17.0)	.02
Hypokalemia	62 (9.4)	39 (5.9)	.02
Atrial fibrillation	60 (9.1)	40 (6.1)	.05
Headache	55 (8.3)	31 (4.7)	.01
Ventricular tachycardia	52 (7.9)	48 (7.3)	.76
Nausea	45 (6.8)	49 (7.4)	.75
Ventricular extrasystoles	40 (6.1)	24 (3.6)	.05

Levosimendan is superior to enoximone in refractory cardiogenic shock complicating acute myocardial infarction*

Joerg T. Fuhrmann, MD; Alexander Schmeisser, MD; Matthias R. Schulze, MD; Carsten Wunderlich, MD; Steffen P. Schoen, MD; Thomas Rauwolf, PhD; Christof Weinbrenner, MD; Ruth H. Strasser, MD

32 patients with refractory cardiogenic shock
Levosimendan or enoximone
In addition to revascularisation, inotropes and CPBIA



Initial good results in small-size studies in cardiogenic shock

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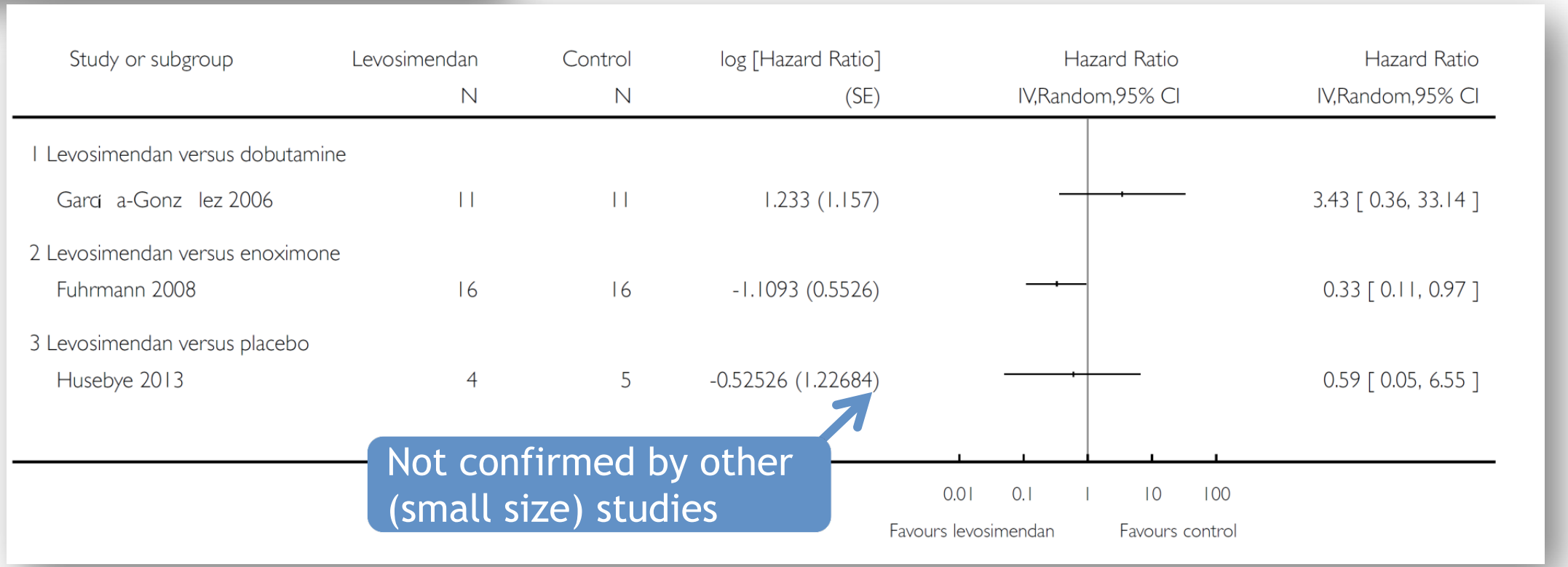
Schumann J, Henrich EC, Strobl H, Prondzinsky R, Weiche S, Thiele H, Werdan K, Frantz S, Unverzagt S



Not confirmed by other (small size) studies

Levy et al. Annals of Intensive Care (2015) 5:17
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Experts' recommendations for the management of adult patients with cardiogenic shock

Bruno Levy[1*], Olivier Bastien[2], Karim Bendjelid[3], Alain Cariou[4], Tahar Chouihed[5], Alain Combes[6], Alexandre Mebazaa[7], Bruno Megarbane[8], Patrick Plaisance[9], Alexandre Ouattara[10], Christian Spaulding[11], Jean-Louis Teboul[12], Fabrice Vanhuyse[13], Thierry Boulain[14] and Kaldoun Kuteifan[15]

cardial oxygen consumption. There is a pharmacodynamic rationale for the use of levosimendan in patients on chronic beta-blocker treatment. In CS refractory to catecholamines, it seems logical to consider the use of circulatory support rather than increased pharmacological support.

Area 4: management of blood pressure and cardiac output in intensive care

1- A MAP of at least 65 mmHg should be reached using inotropic treatment and/or vasopressor

4- Dobutamine should be used to treat low cardiac output in cardiogenic shock (strong agreement).

5- Phosphodiesterase inhibitors or levosimendan should not be used first-line (strong agreement).

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Area 4: management of blood pressure and cardiac output in intensive care

1- A MAP of at least 65 mmHg should be reached using inotropic treatment and/or vasopressor

2- Norepinephrine should be used to restore perfusion pressure during cardiogenic shock (strong agreement).

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4- Dobutamine should be used to treat low cardiac output in cardiogenic shock (strong agreement).

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The NEW ENGLAND JOURNAL of MEDICINE

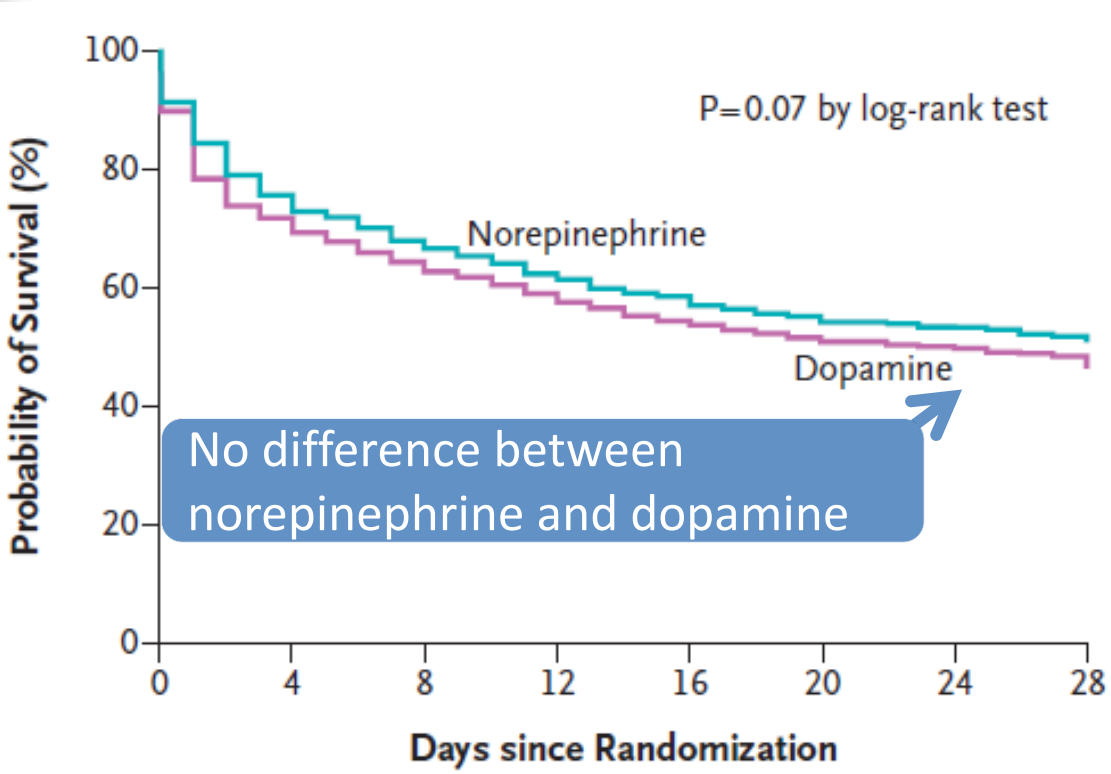
ESTABLISHED IN 1812 MARCH 4, 2010 VOL. 362 NO. 9

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*

1 679 pts with shock

Dopamine vs. norepinephrine



No difference between norepinephrine and dopamine

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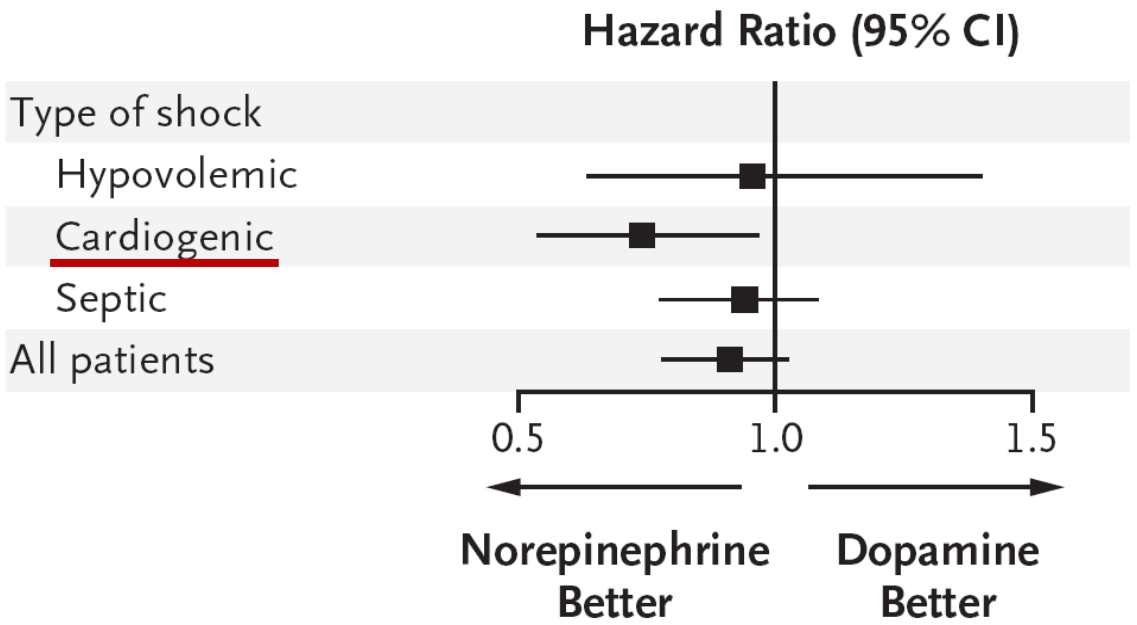

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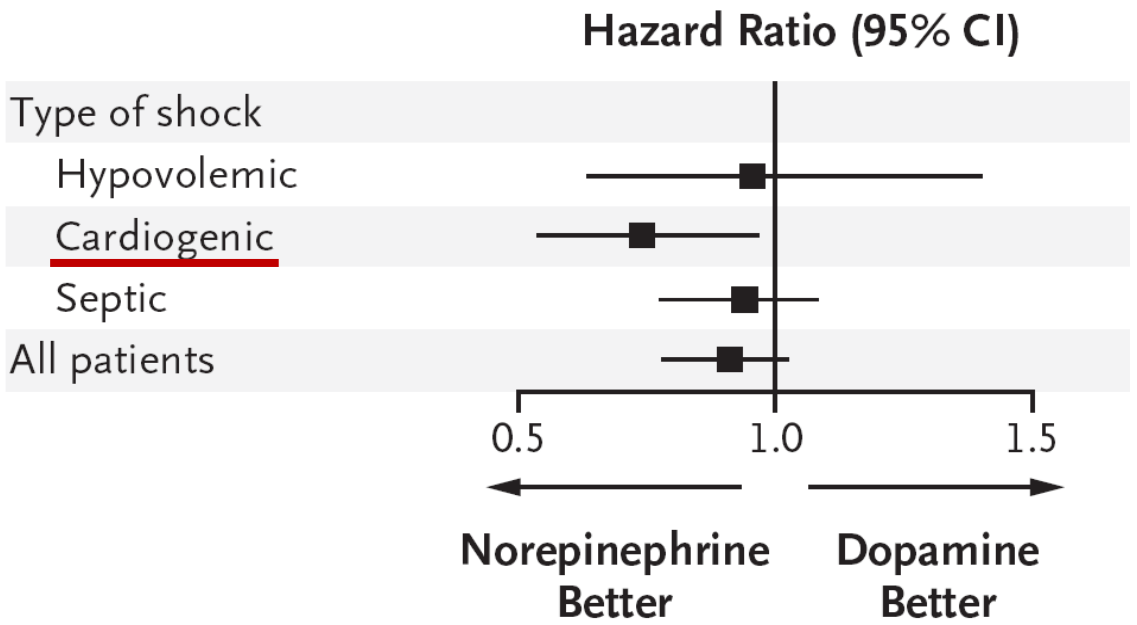
Dopamine vs. norepinephrine



Critical Care

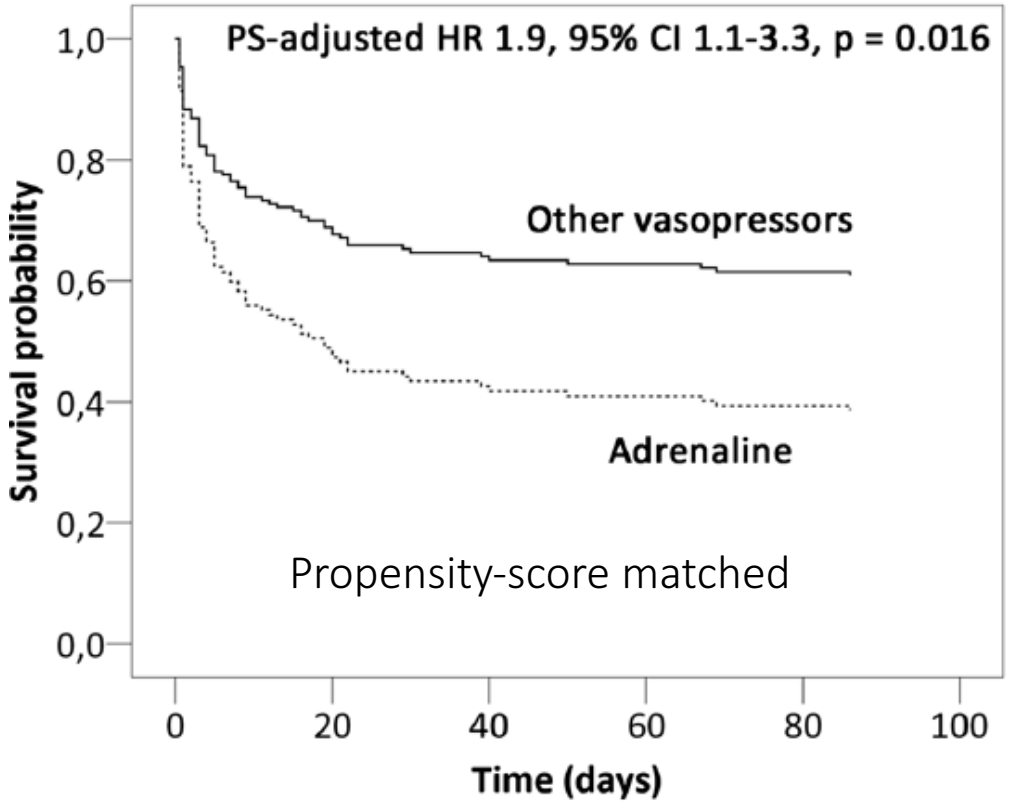
RESEARCH Open Access

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

Critical Care (2016) 20:208

219 patients with cardiogenic shock



Propensity-score matched

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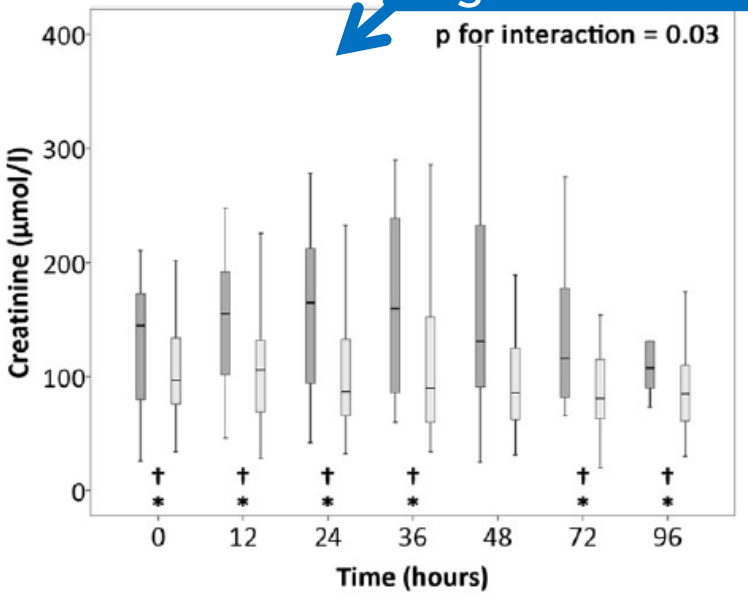
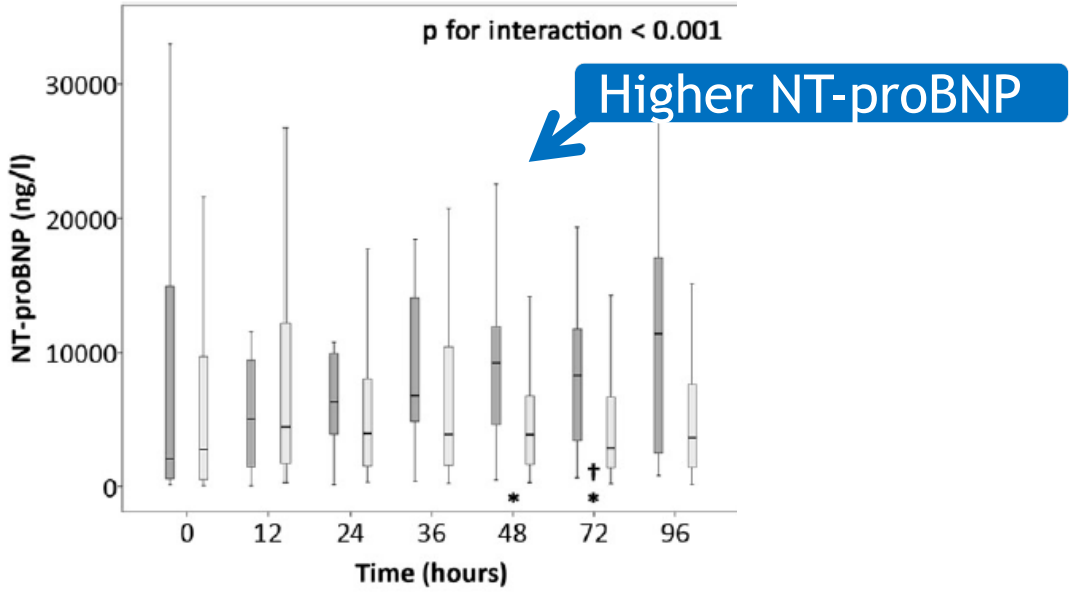
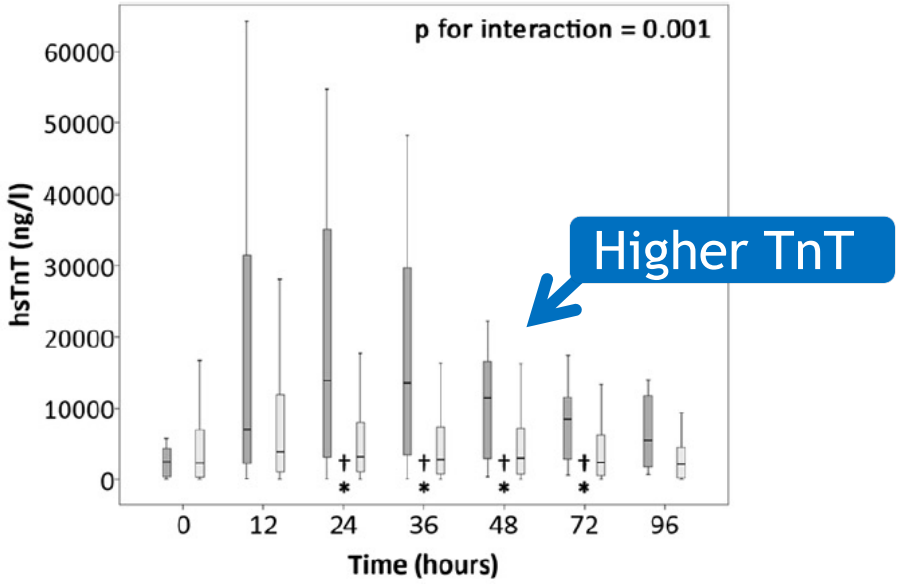
Higher Creat



Higher NT-proBNP



Higher TnT

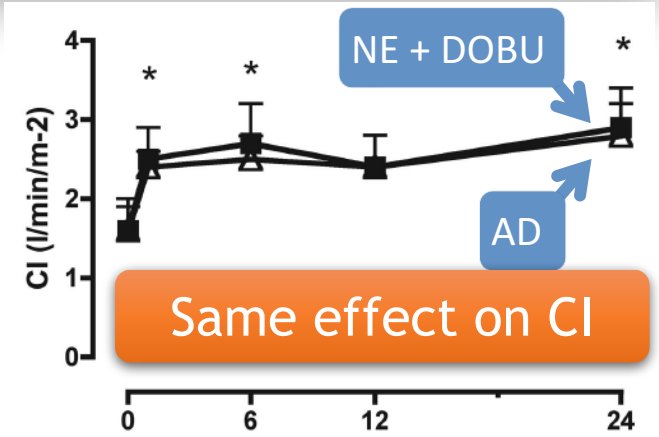


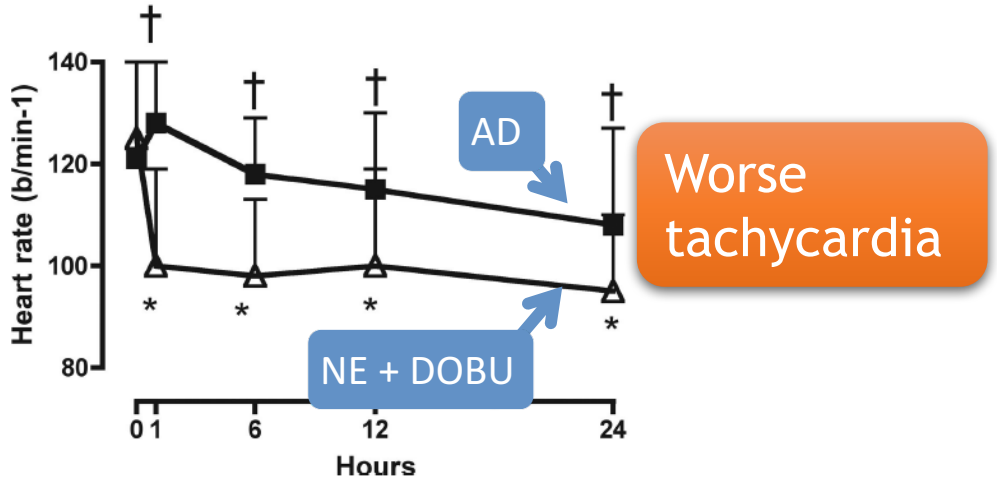
Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study*

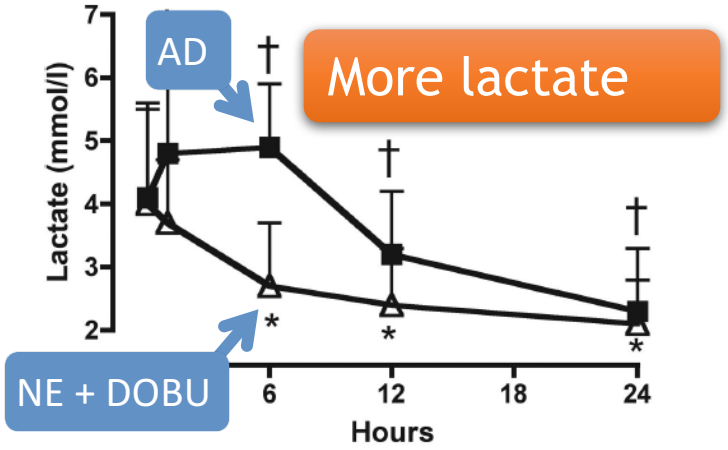
Bruno Levy, MD, PhD; Pierre Perez, MD; Jessica Perny, MD; Carine Thivilier, MD; Alain Gerard, MD

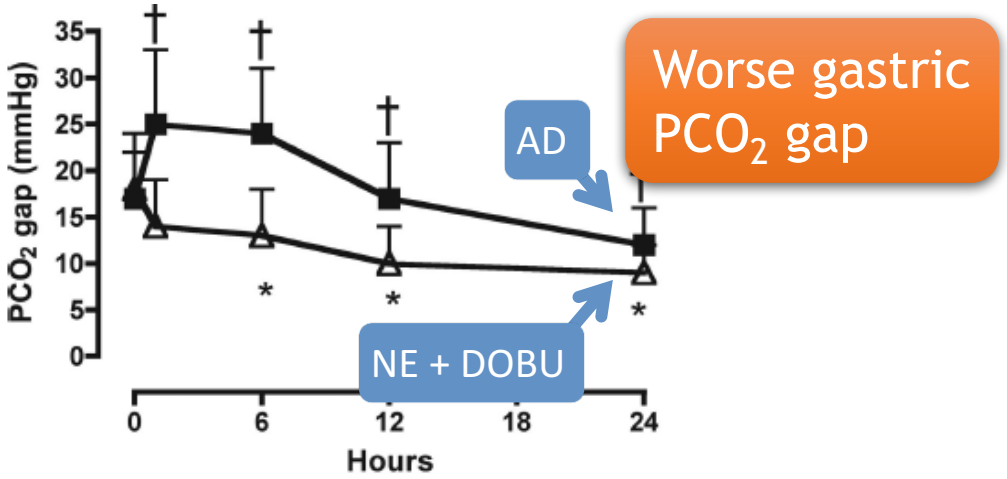
30 patients with cardiogenic shock resistant to dobutamine + NE

Dobu + NE vs. epi









Levy et al. Annals of Intensive Care (2015) 5:17
DOI 10.1186/s13613-015-0052-1

Annals of Intensive Care
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REVIEW Open Access

Experts' recommendations for the management of adult patients with cardiogenic shock

Bruno Levy[1*], Olivier Bastien[2], Karim Bendjelid[3], Alain Cariou[4], Tahar Chouihed[5], Alain Combes[6], Alexandre Mebazaa[7], Bruno Megarbane[8], Patrick Plaisance[9], Alexandre Ouattara[10], Christian Spaulding[11], Jean-Louis Teboul[12], Fabrice Vanhuyse[13], Thierry Boulain[14] and Kaldoun Kuteifan[15]

Area 4: management of blood pressure and cardiac output in intensive care

1- A MAP of at least 65 mmHg should be reached using inotropic treatment and/or vasopressor

2- Norepinephrine should be used to restore perfusion pressure during cardiogenic shock (strong agreement).

3- Epinephrine can be a therapeutic alternative to the combination of dobutamine and norepinephrine,

3- Epinephrine can be a therapeutic alternative to the combination of dobutamine and norepinephrine, but is associated with a greater risk of arrhythmia, tachycardia, and hyperlactatemia (weak agreement).

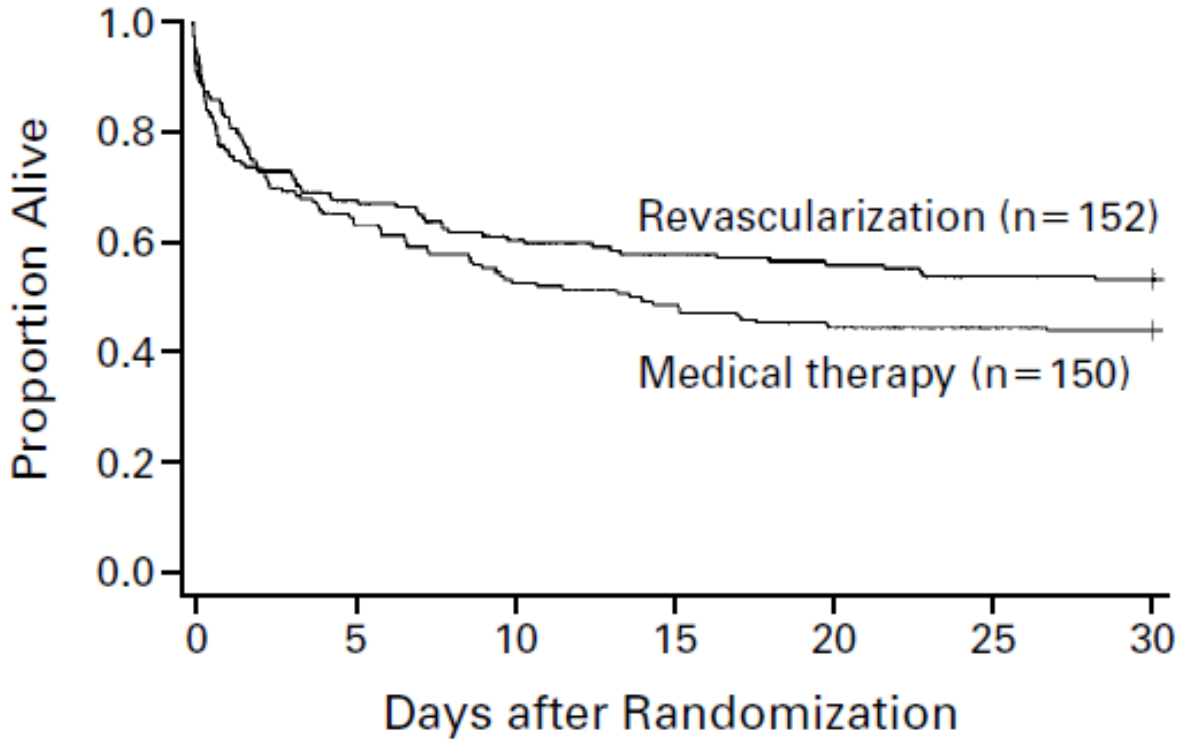
The New England
Journal of Medicine

VOLUME 341 AUGUST 26, 1999 NUMBER 9

EARLY REVASCULARIZATION IN ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

JUDITH S. HOCHMAN, M.D., LYNN A. SLEEPER, SC.D., JOHN G. WEBB, M.D., TIMOTHY A. SANBORN, M.D., HARVEY D. WHITE, D.SC., J. DAVID TALLEY, M.D., CHRISTOPHER E. BULLER, M.D., ALICE K. JACOBS, M.D., JAMES N. SLATER, M.D., JACQUES COL, M.D., SONJA M. MCKINLAY, PH.D., AND THIERRY H. LEJEMTEL, M.D., FOR THE SHOCK INVESTIGATORS*

302 patients with AMI and CS
Early revascularisation or medical stabilisation



Judith S. Hochman, MD
Lynn A. Sleeper, ScD
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Vladimir Dzavik, MD
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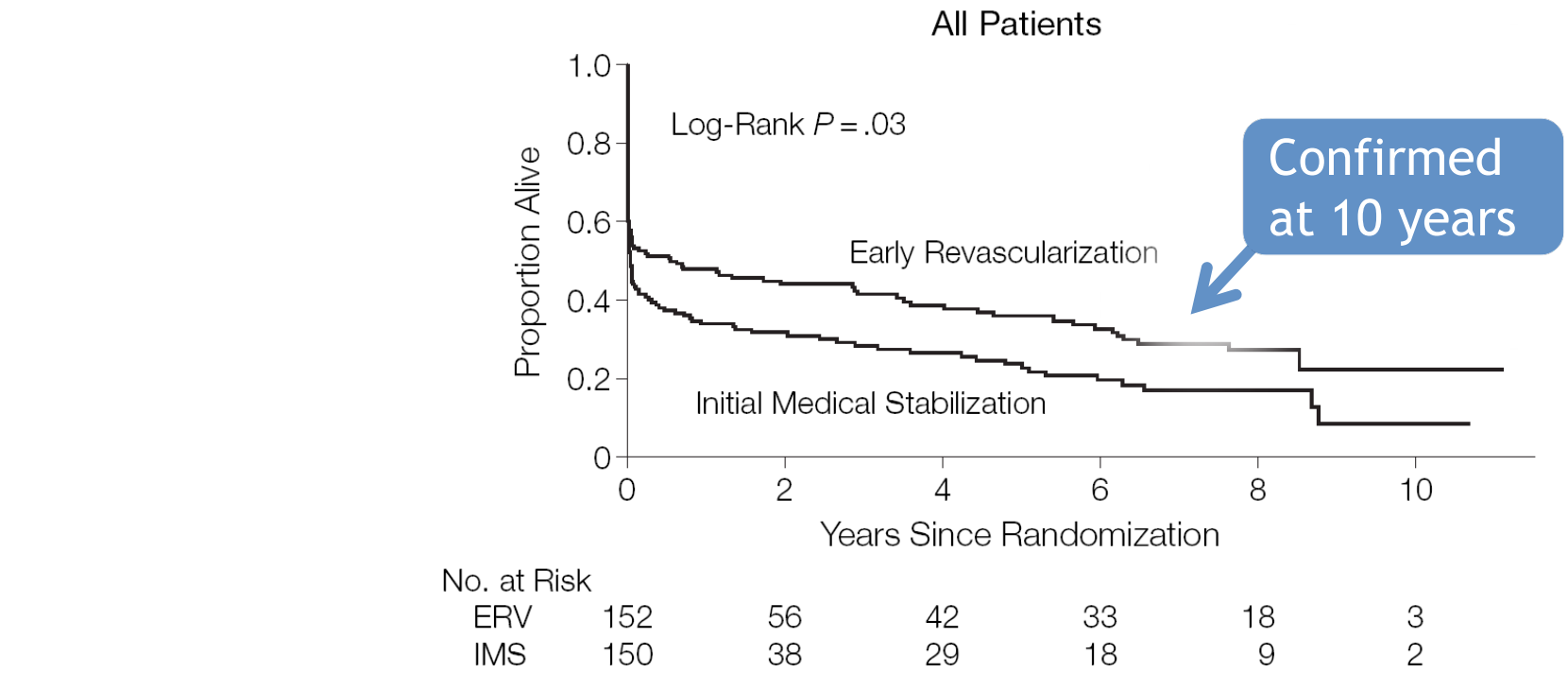
Philip Aylward, MD
Jacques Col, MD
Harvey D. White, DSc
for the SHOCK Investigators

Early Revascularization and Long-term Survival in Cardiogenic Shock Complicating Acute Myocardial Infarction

JAMA, June 7, 2006—Vol 295, No. 21

420 septic shock patients
Retrospective analysis



ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

European Heart Journal (2012) **33**, 2569–2619

Recommendations	Class [a]	Level [b]
Indications for primary PCI		
Primary PCI is the recommended reperfusion therapy over fibrinolysis if performed by an experienced team within 120 min of FMC.	I	A
Primary PCI is indicated for patients with severe acute heart failure or cardiogenic shock, unless the expected PCI related delay is excessive and the patient presents early after symptom onset.	I	B
Procedural aspects of primary PCI		
Stenting is recommended (over balloon angioplasty alone) for primary PCI.	I	A
Primary PCI should be limited to the culprit vessel with the exception of cardiogenic shock and persistent ischaemia after PCI of the supposed culprit lesion.	IIa	B
If performed by an experienced radial operator, radial access should be preferred over femoral access.	IIa	B
If the patient has no contraindications to prolonged DAPT (indication for oral anticoagulation, or estimated high long-term bleeding risk) and is likely to be compliant, DES should be preferred over BMS.	IIa	A
Routine thrombus aspiration should be considered.	IIa	B
Routine use of distal protection devices is not recommended.	III	C
Routine use of IABP (in patients without shock) is not recommended.	III	A

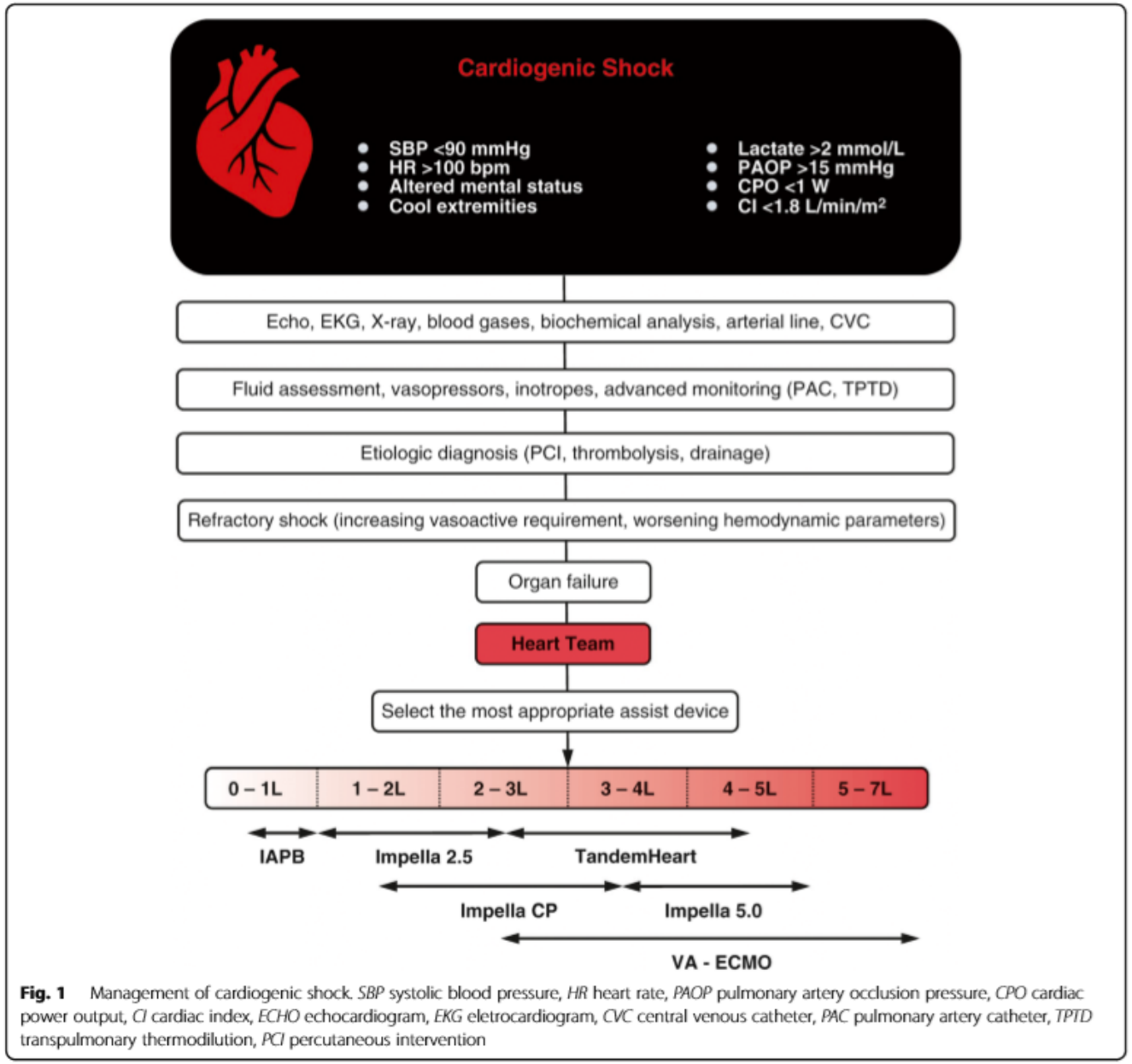
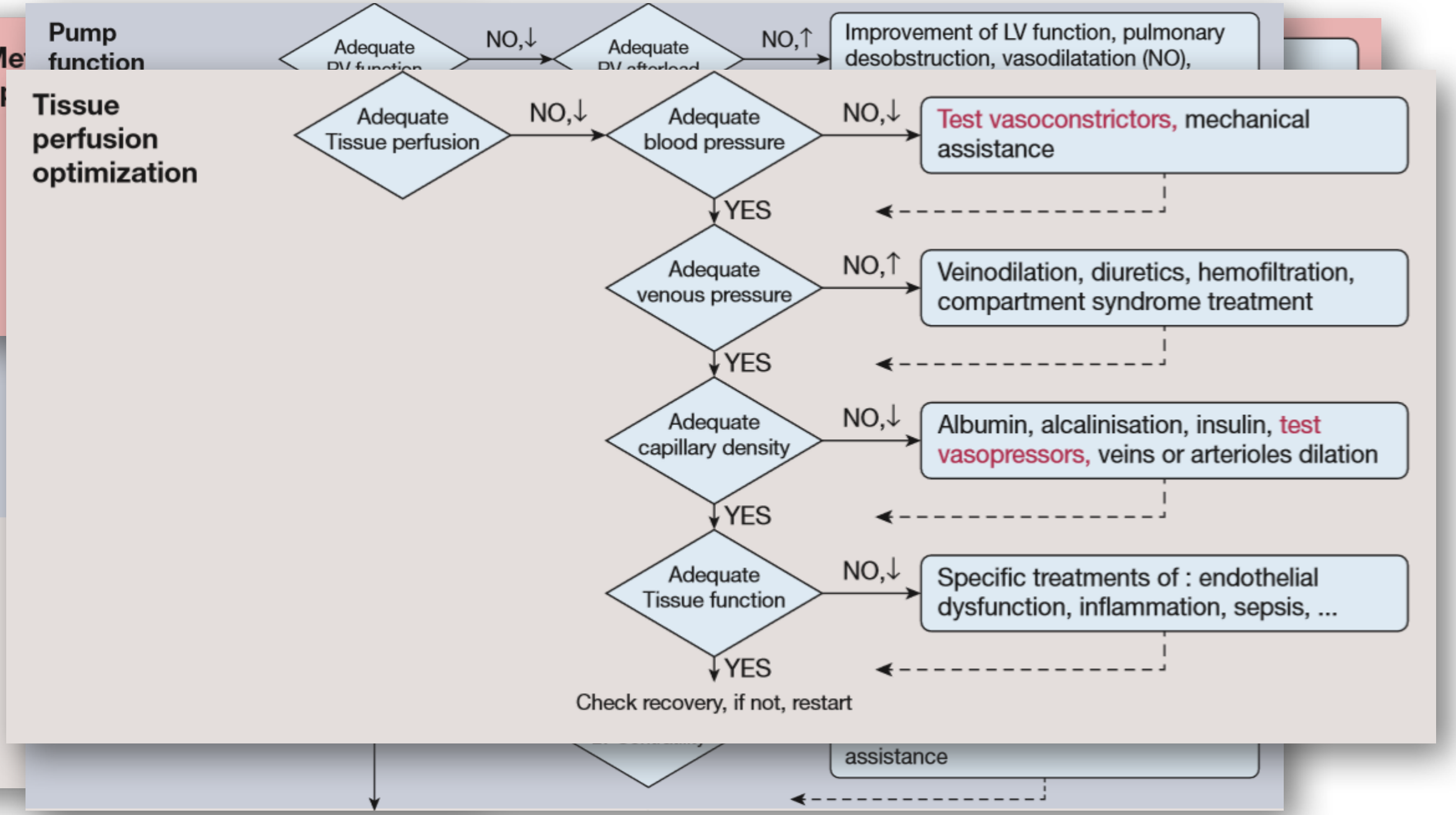


Fig. 1 Management of cardiogenic shock. *SBP* systolic blood pressure, *HR* heart rate, *PAOP* pulmonary artery occlusion pressure, *CPO* cardiac power output, *CI* cardiac index, *ECHO* echocardiogram, *EKG* eletrocardiogram, *CVC* central venous catheter, *PAC* pulmonary artery catheter, *TPTD* transpulmonary thermodilution, *PCI* percutaneous intervention

Table 1 Technical properties of percutaneous circulatory assist devices

	IAPB	IMPELLA 2.5	IMPELLA CP	IMPELLA 5.0	TandemHeart	VA-ECMO
Mechanism	Aorta	LV → aorta	LV → aorta	LV → aorta	LA → aorta	RA → aorta
Flow (L/min)	0.3–0.5	1.0–2.5	3.7–4.0	Max. 5.0	2.5–5.0	3.0–7.0
Cannula size (Fr)	7–8	13–14	13–14	21	15–17 arterial 21 venous	14–16 arterial 18–21 venous
Femoral artery size (mm)	> 4.0	5.0–5.5	5.0–5.5	8.0	8.0	8.0
Cardiac synchrony or stable rhythm	Yes	No	No	No	No	No
Maximum implant days	TBD	7–10 days	7–10 days	2–3 weeks	2–3 weeks	3–4 weeks
Cardiac power	↑	↑↑	↑↑	↑↑	↑↑	↑↑↑
Afterload	↓	↓	↓	↓	↑	↑↑↑
MAP	↑	↑↑	↑↑	↑↑	↑↑	↑↑
LVEDP	↓	↓↓	↓↓	↓↓	↓↓	↔
PAOP	↓	↓↓	↓↓	↓↓	↓↓	↔
LV preload	–	↓↓	↓↓	↓↓	↓↓	↓
Coronary perfusion	↑	↑	↑	↑	–	–

IABP intraaortic balloon pump, *VA-ECMO* veno-arterial extracorporeal membrane oxygenation, *LV* left ventricle, *LA* left atrium, *RA* right atrium, *MAP* mean arterial pressure, *LVEDP* left ventricular end-diastolic pressure, *PAOP* pulmonary artery occlusion pressure


Tissue perfusion optimization
Adequate Tissue perfusion
NO,↓
Adequate blood pressure
NO,↓
Test vasoconstrictors, mechanical assistance
YES
Adequate venous pressure
NO,↑
Veinodilation, diuretics, hemofiltration, compartment syndrome treatment
YES
Adequate capillary density
NO,↓
Albumin, alcalinisation, insulin, test vasopressors, veins or arterioles dilation
YES
Adequate Tissue function
NO,↓
Specific treatments of : endothelial dysfunction, inflammation, sepsis, ...
YES
Check recovery, if not, restart
Pump function optimization
Adequate
NO,↓
Adequate
NO,↑
Improvement of LV function, pulmonary desobstruction, vasodilatation (NO),
assistance
Metabolic optimizati
Pump function optimizati
Tissue perfusion optimization

Levy *et al. Annals of Intensive Care* (2015) 5:17
DOI 10.1186/s13613-015-0052-1

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AHA SCIENTIFIC STATEMENT

CLINICAL STATEMENTS AND GUIDELINES

Contemporary Management of Cardiogenic Shock

A Scientific Statement From the American Heart Association

Table 5. Initial Vasoactive Management Considerations in Types of CS

Cause or Presentation of CS	Vasoactive Management Considerations	Hemodynamic Rationale
Classic wet and cold	Norepinephrine or dopamine[144] Inotropic agent[210,211]*	This subtype has low CI and high SVR. Consider hemodynamic stabilization with norepinephrine (preferred in ↑HR or arrhythmias) or dopamine (↓HR preferred but associated with higher risk of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only)
Euvolemic cold and dry	Norepinephrine or dopamine[144] Inotropic agent[210,211] Small fluid boluses	Consider hemodynamic stabilization with norepinephrine (preferred in ↑HR or arrhythmias) or dopamine (↓HR preferred but associated with higher risk of arrhythmias) Consider addition of inotropic agent when stabilized and after revascularization (MI only) LVEDP may be low, and patients may tolerate fluid boluses
Vasodilatory warm and wet or mixed cardiogenic and vasodilatory	Norepinephrine Consider hemodynamics-guided therapy	This subtype has low SVR
RV shock	Fluid boluses[144,145] Norepinephrine, dopamine, or vasopressin[144,212,213] Inotropic agents[144]* Inhaled pulmonary vasodilators[214]	Hemodynamic goals include maintaining preload, lowering RV afterload (PVR), treating absolute or relative bradycardias, and maintaining atrioventricular synchrony Dopamine (↓HR preferred but associated with arrhythmia risk) Vasopressin may raise SVR and have neutral effect on PVR Consider adding or transitioning to inotrope after initial hemodynamic stabilization and revascularization
Normotensive shock	Inotropic agent or vasopressor	Initial inotropic therapy may be appropriate given that this subtype has SBP >90 mm Hg and relatively high SVR
Aortic stenosis	Phenylephrine or vasopressin In patients with reduced LVEF, echocardiography- or PAC-guided dobutamine titration	Shock caused by aortic stenosis is an afterload-dependent state Inotropy may not improve hemodynamics if LVEF is preserved Definitive therapies will be defined by underlying cause and may include surgical aortic valve replacement or balloon valvuloplasty and/or transcatheter aortic valve replacement

Aortic regurgitation	Dopamine Temporary pacing	Maintaining an elevated HR may shorten diastolic filling time and reduce LVEDP Definitive therapies will be defined by underlying cause and may include surgical aortic valve replacement
Mitral stenosis	Phenylephrine or vasopressin Esmolol or amiodarone	Shock resulting from mitral stenosis is a preload-dependent state Avoiding chronotropic agents, slowing the HR (and thereby increasing diastolic filling time), and maintaining atrioventricular synchrony may improve preload Definitive therapies will be defined by underlying cause and may include surgical mitral valve replacement or balloon valvuloplasty
Mitral regurgitation	Norepinephrine or dopamine Inotropic agents* Temporary MCS, including IABP[144]	After hemodynamic stabilization with vasopressor, consider addition of inotropic agent Afterload reduction may help reduce LVEDP IABP may reduce regurgitation fraction by reducing afterload and increasing CI Definitive therapies will be defined by underlying cause and may include surgical mitral valve replacement/repair and percutaneous edge-to-edge repair
Postinfarction ventricular septal defect	See classic wet and cold considerations Temporary MCS, including IABP[144]	IABP may reduce shunt fraction by reducing afterload and increasing CI Cardiac surgical referral for repair or percutaneous interventional umbrella closure
Dynamic LVOT obstruction	Fluid boluses[215,216] Phenylephrine or vasopressin[215,216] Avoid inotropic agents[215,216] Avoid vasodilating agents[215,216] Esmolol or amiodarone[215] RV pacing	Dynamic gradients may be reduced by increasing preload and afterload, reducing inotropy and ectopy, maintaining atrioventricular synchrony, and inducing ventricular dyssynchrony
Bradycardia	Chronotropic agents or Temporary pacing	Treatment should also focus on identifying and treating underlying cause of bradycardia Chronotropic agents may include atropine, isoproterenol, dopamine, dobutamine, and epinephrine
Pericardial tamponade	Fluid bolus Norepinephrine	Pericardiocentesis or surgical pericardial window required for definitive therapy

[Contemporary Reviews in Critical Care Medicine]

CHEST

Reconsidering Vasopressors for Cardiogenic Shock

Everything Should Be Made as Simple as Possible, but Not Simpler

Pierre Squara, MD; Steven Hollenberg, MD; and Didier Payen, MD, PhD

Scientific statements and publications have recommended the use of vasoconstrictors as the first-line pharmacologic choice for most cases of cardiogenic shock (CS), without the abundance of strong clinical evidence. One challenge of guidelines is that the way recommendations are stated can potentially lead to oversimplification of complex situations. Except for acute coronary syndrome with CS, in which maintenance of coronary perfusion pressure seems logical prior to revascularization, physiologic consequences of increasing afterload by use of vasoconstrictors should be analyzed. Changing the CS conceptual frame, emphasizing inflammation and other vasodilating consequences of prolonged CS, mixes causes and consequences. Moreover, the considerable interpatient differences regarding the initial cause of CS and subsequent consequences on both macro- and microcirculation, argue for a dynamic, step-by-step, personalized therapeutic strategy. In CS, vasoconstrictors should be used only after a reasoning process, a review of other possible options, and then should be titrated to reach a reasonable pressure target, while checking cardiac output and organ perfusion.

CHEST 2019; ■(■):■-■

Open access Heart failure and cardiomyopathies

openheart

Cardiogenic shock: evolving definitions and future directions in management

Tara L Jones,[1] Kenta Nakamura,[2] James M McC

Cardiac Power Index = $\frac{\text{MAP x CI}}{451}$

TABLE: Cardiac Power Index (Cutoff: 0.32 W/m2)

MAP ≤55

	Mean Arterial Pressure						
Fick CI	85	80	75	70	65	60	55
2.6	0.49	0.46	0.43	0.40	0.37	0.35	0.32
2.4	0.45	0.43	0.40	0.37	0.35	0.32	0.29
2.2	0.41	0.39	0.37	0.34	0.32	0.29	0.27
2.0	0.38	0.35	0.33	0.31	0.29	0.27	0.24
1.8	0.34	0.32	0.30	0.28	0.26	0.24	0.22
1.6	0.30	0.28	0.27	0.25	0.23	0.21	0.20

CI ≤1.7

ESHF

AMI

Figure 3 Cardiac index (CI) and mean arterial pressure (MAP) correlation to Cardiac Power Index (CPI).

Table 3. Considerations for Initial Critical Care Monitoring in Patients With CS

Monitoring Parameter	Frequency	Comment/Rationale
Noninvasive monitoring		
Telemetry, pulse oximetry, respiratory rate	Continuous	High incidence of arrhythmias, ventilator failure, and pulmonary edema
Critical care unit monitoring	1:1 Nurse-to-patient ratio	High incidence of hemodynamic deterioration and multisystem organ failure
Invasive monitoring		
Arterial BP monitoring	Continuous	Consider continuing until vasoactive medications have been discontinued for 12–24 h
CVP	Continuous	A central line is required for delivery of vasoactive medications; single-point-in-time CVP measurements may be unreliable measures of fluid status, but longitudinal CVP trends may provide information on trends in fluid status
Central venous oxygen saturation	Every 4 h	Trends in central venous oxygen saturation in patients with a central line can be used to help monitor trends in cardiac output
Urine output	Every hour	Urine output and serum creatinine monitoring are markers of renal perfusion and acute kidney injury
PAC or noninvasive cardiac output monitor	Selected use	Consider using early in the treatment course in patients not responsive to initial therapy or in cases of diagnostic or therapeutic uncertainty
Laboratory investigations		
Complete blood counts	Every 12–24 h	Consider more frequently in patients with CS with, or at high risk for, bleeding
Serum electrolytes	Every 6–12 h	Frequency should be tailored to risks or presence of renal failure and electrolyte dyscrasias
Serum creatinine	Every 12–24 h	Urine output and serum creatinine monitoring are markers of renal perfusion and acute kidney injury
Liver function tests	Daily	Monitoring for congestive hepatopathy and hypoperfusion
Lactate	Every 1–4 h	Lactate clearance is a marker of resolving end-organ hypoperfusion, and lack of clearance is associated with a higher risk of mortality
Coagulation laboratories	Every 4–6 h for those on anticoagulants until therapeutically stable, every 24 h if patient is not on anticoagulants	Altered drug elimination and frequent use of mechanical support devices often necessitate antithrombotic monitoring

BP indicates blood pressure; CS, cardiogenic shock; CVP, central venous pressure; and PAC, pulmonary artery catheter.

Arterial pressure allows monitoring the changes in cardiac output induced by volume expansion but not by norepinephrine*

Xavier Monnet, MD, PhD; Alexia Letierce, PhD; Olfa Hamzaoui, MD; Denis Chemla, MD, PhD; Nadia Anguel, MD; David Osman, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD

Crit Care Med 2011

No change
in arterial resistance

228 patients receiving volume expansion

145 patients with increase of NE

Change
in arterial resistance

Arterial pressure allows monitoring the changes in cardiac output induced by volume expansion but not by norepinephrine*

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Crit Care Med 2011

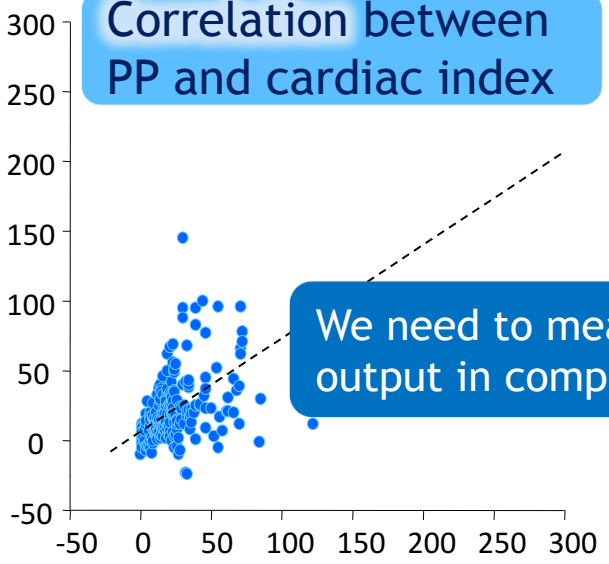
228 pts receiving volume expansion
145 patients with increase of NE

Volume expansion

Changes in PP (%)

r = 0.56

Correlation between PP and cardiac index



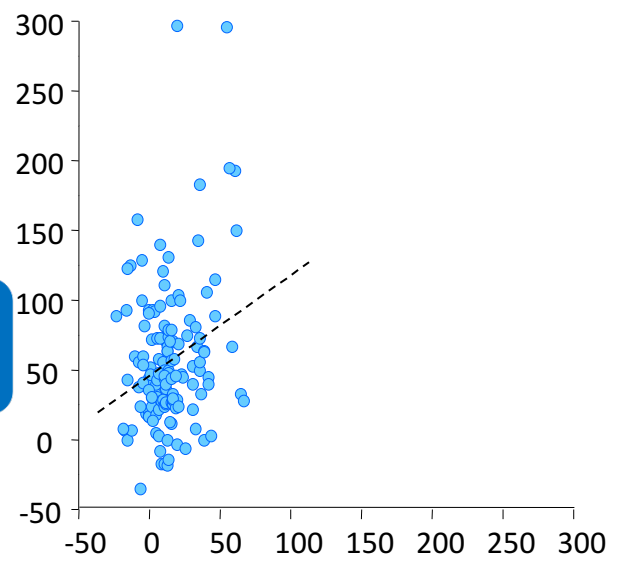
We need to measure cardiac output in complex patients

No correlation between PP and cardiac index

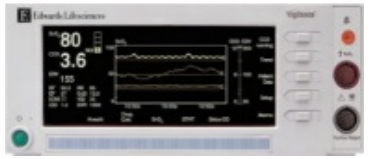
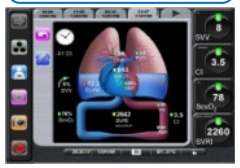
↗ norepinephrine

Changes in PP (%)

r = 0.21



Advanced monitoring

	PA catheter	PiCCO	EV 1000
			
How much is cardiac output?	Cardiac output	Cardiac output	
Is cardiac output adequate?	SvO_2	$ScvO_2$	
Should we give fluids?	PAOP	EEO test PLR test	
Should we NOT give fluids	PAOP	Lung water Lung permeability	
Is there RV dysfunction	↗ pulmonary vasc. resistance	↘ cardiac output ↗ CVP	
How is the LV contractility	↘ cardiac output ↗ PAOP	Global ejection fraction	

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6- In shock refractory to empirical treatment, cardiac output as well as mixed venous oxygen saturation (SvO_2) or $ScvO_2$ should be continuously monitored

7- We suggest pulmonary artery catheterization in patients with refractory cardiogenic shock and right ventricular dysfunction

8- We suggest the use of a transpulmonary thermodilution monitor/pulse wave analysis plus measurement (continuous or intermittent) of mixed venous oxygen saturation (SvO_2) or $ScvO_2$ when cardiogenic shock is refractory to initial treatment, in the absence of mechanical assistance and of predominant right ventricular dysfunctior

1. La dobutamine doit être utilisée précautionneusement, à la plus petite dose possible
2. Il n'y a pas de place en 1ère intention pour les inhibiteurs de la PDE et pour le levosimendan
3. La noradrénaline est le vasopresseur à choisir en 1ère intention. Les risques liés à l'adrénaline sont bien démontrés
4. Il faut mesurer directement le débit cardiaque des patients en choc cardiogénique. La pression artérielle seule ne peut suffire
5. La thermodilution transpulmonaire et le cathéter artériel pulmonaire sont conseillés pour le monitoring