CARDIOGENIC SHOCK

EPIDEMIOLOGY, PATHOPHYSIOLOGY, THERAPY

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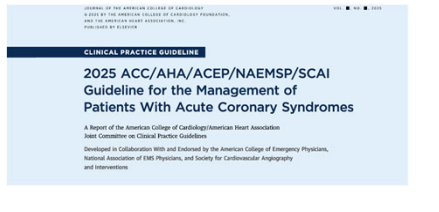
CONFLICTS OF INTEREST



CLINICAL PRACTICE GUIDELINE

2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes

A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by the American College of Emergency Physicians, National Association of EMS Physicians, and Society for Cardiovascular Angiography and Interventions

Guidelines

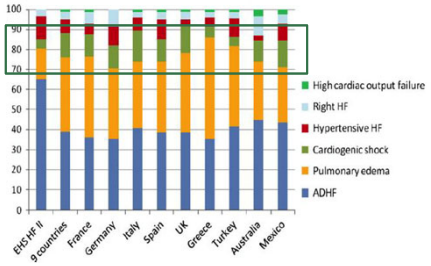
Experts' recommendations for the management of adult patients with cardiogenic shock☆

Arch Cardiovasc Dis 2026 Mar 31:S1875-2136(26)00051-3
Annals of Intensive Care 16 (2026) 100038

CLINICAL DEFINITION

- Life threatening end-organ hypoperfusion and tissular hypoxia from cardiac dysfunction
- Persistent low-cardiac output unresponsive to volume loading (fluid loading challenge+++)
- Both clinical and biochemical manifestation
- Majority of Cardiogenic Shock (≈80%) are caused by Acute Coronary Syndrome (STEMI+++)
- 5-10% of patients hospitalized for AMI (leading cause of mortality in these patients)
- Results from extensive damage to left ventricular myocardium or mechanical complications (**please check by repeating TTE exam!**)

ACUTE HEART FAILURE IS NOT CARDIOGENIC SHOCK…



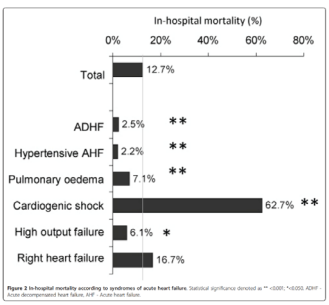
Follath F et al. Int Care Med 2011

RESEARCH Open Access

2011

Baseline characteristics and hospital mortality in the Acute Heart Failure Database (AHEAD) Main registry

Jindrich Spinar[1,2], Jiri Parenica[1,2*], Jiri Vitovec[2,3], Petr Widimsky[4], Ales Linhart[5], Marian Fedorco[6], Filip Malek[7], Cestmir Cihalik[8], Lenka Spinarova[2,3], Roman Miklik[1], Marian Felsoci[1], Miroslav Bambuch[9], Ladislav Dusek[9] and Jiri Jarkovsky[9]

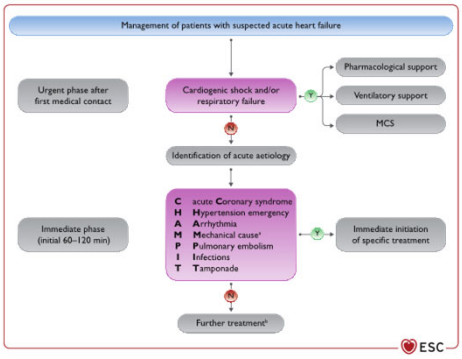


CAUSES OF CARDIOGENIC SHOCK (SPECIFIC PHARMACOLOGICAL AND MECHANICAL THERAPEUTIES)

- Acute myocardial infarction
  - Pump failure
    - Large infarction
    - Smaller infarction with preexisting left ventricular dysfunction
    - Infarction extension
    - Severe recurrent ischemia
    - Infarction expansion
  - Mechanical complications
    - Acute mitral regurgitation caused by papillary muscle rupture
    - Ventricular septal defect
    - Free-wall rupture
    - Pericardial tamponade
  - Right ventricular infarction

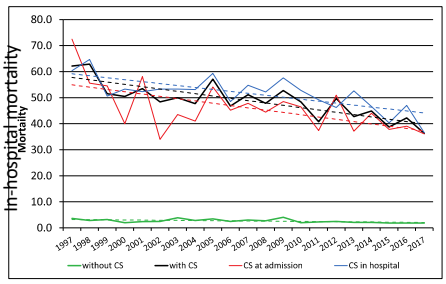
- Other conditions
  - End-stage cardiomyopathy
  - Myocarditis
  - Myocardial contusion
  - Prolonged cardiopulmonary bypass
  - Septic shock with severe myocardial depression
  - Left ventricular outflow tract obstruction
    - Aortic stenosis
    - Hypertropic obstructive cardiomyopathy
  - Obstruction to left ventricular filling
    - Mitral stenosis
    - Left atrial myxoma
  - Acute mitral regurgitation (chordal rupture)
  - Acute aortic insufficiency
  - Acute massive pulmonary embolism
  - Acute stress cardiomyopathy
  - Pheochromocytoma

Reynolds HR et al. Circulation 2008;117:686-97



Mc Donagh TA et al. Eur Heart J 2021; 42:3599-3726

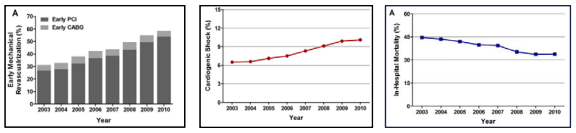
Poor prognosis of cardiogenic shock



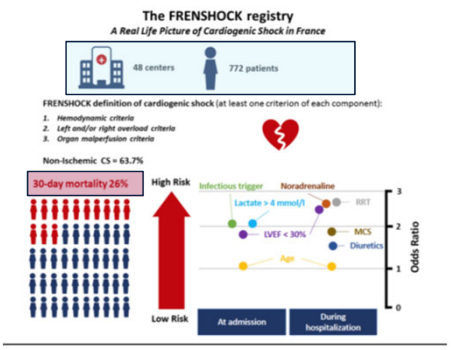
Hunziker L et al. Circ Cardiovasc Interv. 2019;12:e007293

TRENDS IN INCIDENCE AND OUTCOMES OF CARDIOGENIC SHOCK

- From 2003 to 2010, 1 990 486 patients (≥ 40 years) with STEMI
- Early revascularization increased from **30.4% to 50.7%**
- Overall incidence of cardiogenic shock was **7.9% (trends to increase...)**
- Global in-hospital mortality was **39% (trends to decrease...)**



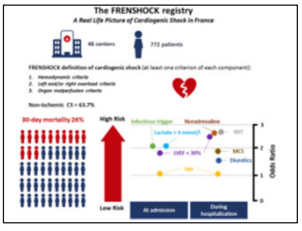
Kolte D et al. J Am Heart Assoc 2014



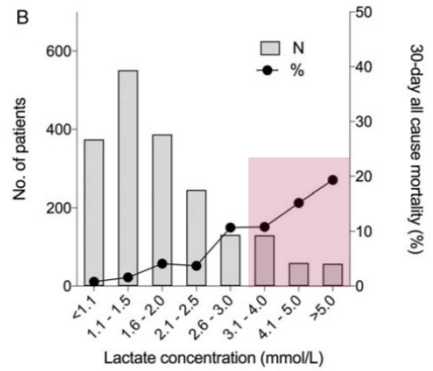
Delmas C et al. ECS Heart Failure 2022; 9:408-19

Delmas C et al. ECS Heart Failure 2022; 9:408-19

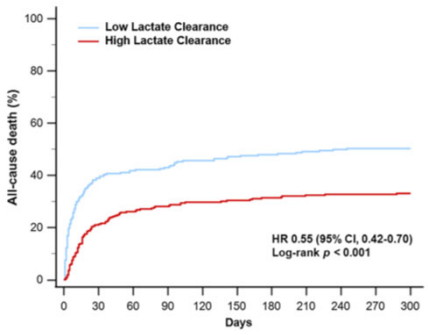
	Overall population (*n* = 772)
Medications used, *n* (%)	
Diuretics	633 (82.0)
Volume expander	321 (41.6)
Dobutamine	632 (81.9)
Maximum dose:	
5–10 µg/kg/min	405/632 (52.5)
10–15 µg/kg/min	136/632 (17.6)
>15 µg/kg/min	47/632 (6.1)
Unknown	184/632 (23.8)
Norepinephrine	410 (53.1)
Epinephrine	95 (12.4)
Norepinephrine and dobutamine combination	352 (45.6%)
Levosimendan	57 (7.4)
Dopamine	2 (0.3)
Isoprenaline	32 (4.1)
Antiarrhythmic	298 (38.6)
Transfusion	128 (16.6)
Fibrinolysis	13 (1.7)
Organ replacement therapies, *n* (%)	
Respiratory support	
Invasive	291 (37.9)
Non-invasive	199 (25.9)
Mechanical circulatory support	143 (18.6)
IABP	48/143 (34.3)
Impella	26/143 (18.6)
ECLS	85/143 (60.7)
Renal replacement therapy	122 (15.8)



Frydland M et al. Shock 2019;51:321-7

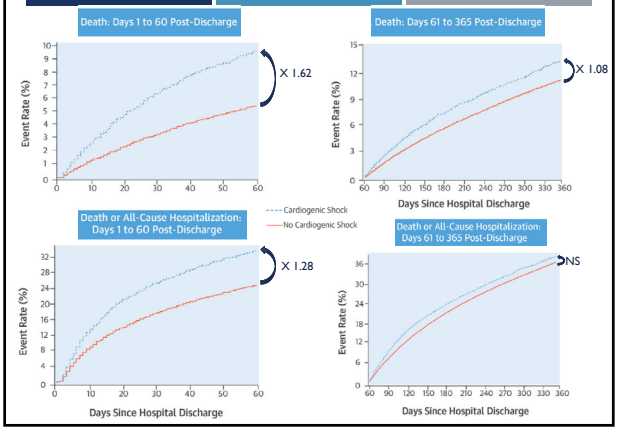


Park IH et al. J Intensive Care 2021;9:63

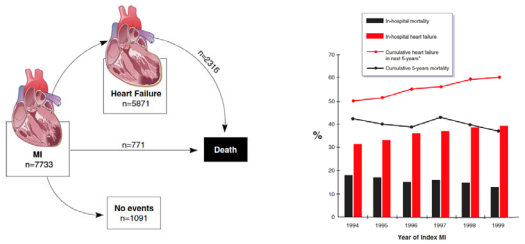


*High lactate clearance was defined as > 66% in 24 hours

Shah RU et al. JACC 2016;67:739-47



CHRONIC HEART FAILURE ATFER MYOCARDIAL INFARCTION



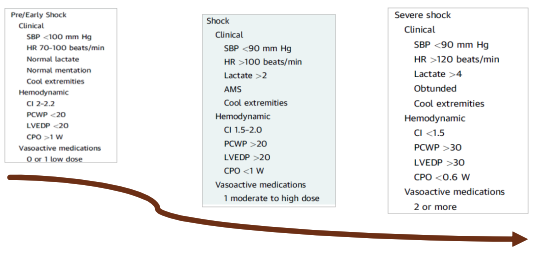
Ezekowitz JA et al. J Am Coll Cardiol 2009;53:13-20

A Practical Approach to Mechanical Circulatory Support in Patients Undergoing Percutaneous Coronary Intervention

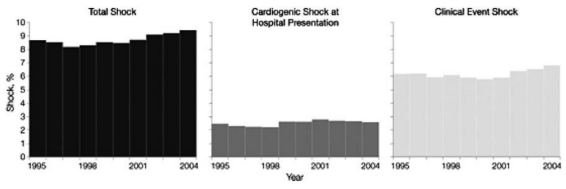
An Interventional Perspective

Atkinson TM et al. J Am Coll Cardiol Interv 2016;9:871–83

Spectrum of Cardiogenic Shock



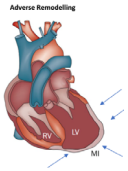
NATIONAL REGISTRY OF MYOCARDIAL INFARCTION



Topolian S et al. Crit Care Med 2008; 36[Suppl.]:S66-S74

VENTRICULAR REMODELING

- 30% of patients after MI (infarct size is the principal risk factor)
- Main origin of chronic heart failure after MI
- Increased parietal stress (+++) and myocardial work
- Increased myocardial oxygen consumption
- Decreased coronary blood flow (+++)
- Increased compliance of infarcted zone
  - Thinning of myocardial wall
  - Left ventricular dilation



Deja MA et al. Circulation 2012; 125:2639-48
Michler RE et al. J Thorac Cardiovasc Surg 2013;146:1139-45
Jackson BM JACC 2002; 40:1160-7

Van Diepen S et al. Circulation 2017;136:e232-68

SHOCK Trial[9]*	IABP-SHOCK II†	ESC HF Guidelines[15]
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·min^{-1}·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

Peripheral Circulation	Volume Status: Wet	Volume Status: Dry
Cold	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP) 64%	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP) 28%
Warm	Vasodilatory Cardiogenic Shock or Mixed Shock 5% (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) 3% (↑CI; ↓SVRI; ↓PCWP)

5% of patients presented end-organ hypoperfusion while SAP was > 90 mmHg

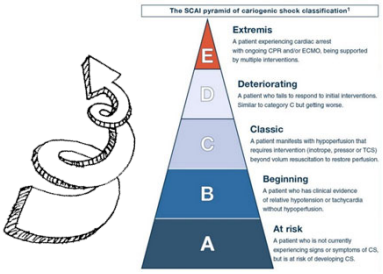
Table 4: INTERMACS Profiles

Profiles	Brief Description	Details
INTERMACS 1	Critical cardiogenic shock (Crash and burn)	Life-threatening hypotension despite rapidly escalating inotropic support.
INTERMACS 2	Progressive decline (Sliding fast on inotropes)	Declining function despite intravenous inotropic support.
INTERMACS 3	Stable but inotrope dependent (Dependent stability)	Stable on continuous intravenous inotropic support.
INTERMACS 4	Resting symptoms on oral therapy at home	Patient experiences daily symptoms of congestion at rest or during activities of daily living.
INTERMACS 5	Exertion intolerant	Patient is comfortable at rest and with activities of daily living but unable to engage in any other activity.
INTERMACS 6	Exertion limited (Walking wounded)	Patient has fatigue after the first few minutes of any meaningful activity.
INTERMACS 7	Advanced NYHA class III (Placeholder)	Patients living comfortably with meaningful activity limited to mild physical exertion.

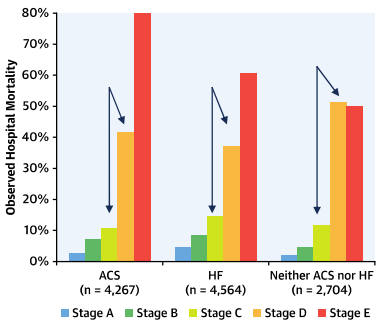
INTERMACS: Interagency Registry for Mechanically Assisted Circulatory Support; NYHA = New York Heart Association. Adapted from: Stevenson LW, et al.[28]

CALSSIFICATION SCAI DU CHOC CARDIOGÉNIQUE

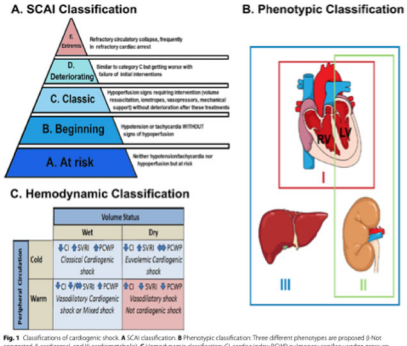
SCAI: Society for Cardiovascular Angiography and Interventions (SCAI)

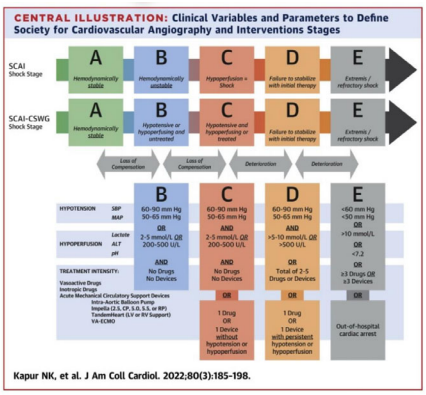


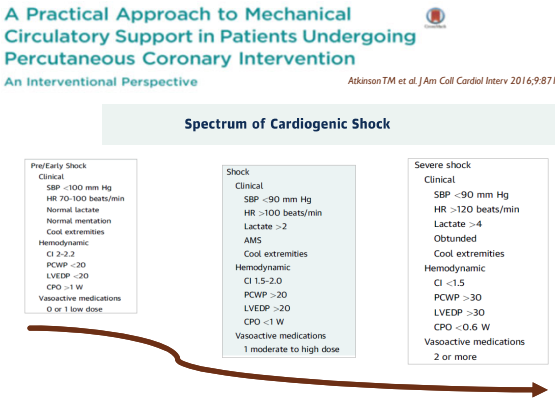
Baran DA, et al. Catheter Cardiovasc Interv. 2019;94:29–37



Jentzer JC et al.A Am Coll Cardiol 2019; 74:2117-28



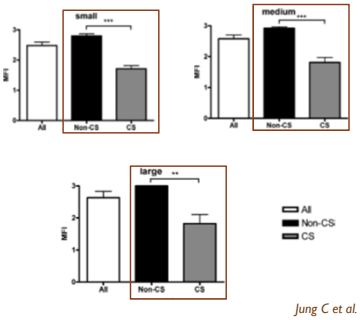




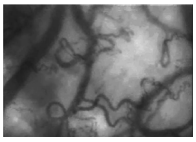
DISEASE OF ENTIRE CIRCULATORY SYSTEM

- Decreased stroke volume and thus reduced cardiac output while compensatory mechanism (tachycardia)
- Reduction myocardial contractility
- Inadequate oxygen delivery: mismatch between oxygen delivery and oxygen consumption
- Disturbances of entire circulatory system (peripheral vasculature)
- Adaptative vasoconstriction through increased afterload (neurohumoral system)
- Pathological vasodilation related to systemic inflammatory response (end organ hypoperfusion and/or ischemia reperfusion phenomenon)

ALTERATION OF MICROCIRCULATORY BLOOD FLOW



Microvascular flow index (MFI)



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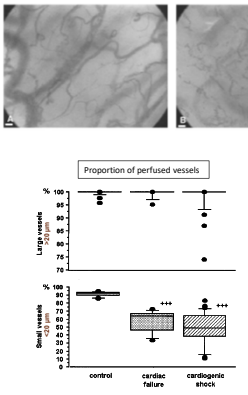


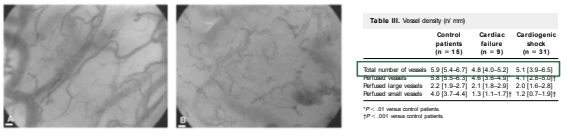


Table III. Vessel density (n/mm)

	Control patients (n = 15)	Cardiac failure (n = 9)	Cardiogenic shock (n = 31)
Total number of vessels	5.9 [5.4–6.7]	4.8 [4.0–5.2]	5.1 [3.9–6.5]
Perfused vessels	5.8 [5.5–6.3]	4.8 [3.6–5.9]	4.1 [2.8–5.0]†
Perfused large vessels	2.2 [1.9–2.7]	2.1 [1.8–2.9]	2.0 [1.6–2.8]
Perfused small vessels	4.0 [3.7–4.4]	1.3 [1.1–1.7]†	1.2 [0.7–1.9]†

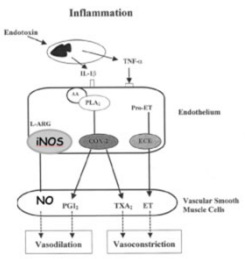
*P < .01 versus control patients.
†P < .001 versus control patients.

Table IV. Effects of topical acetylcholine (10−2M) application in 10 patients with cardiogenic shock

	Cardiogenic shock (n = 10)	
	Baseline	Acetylcholine 10-2M
Total number of vessels (n/mm)	5.0 [4.8–5.7]	5.8 [4.3–6.2]
Perfused vessels (%)	84 [80–89]†	98 [97–99]†
Perfused large vessels (%)	100 [95–100]	100 [100–100]
Perfused small vessels (%)	69 [59–73]†	91 [88–95]†

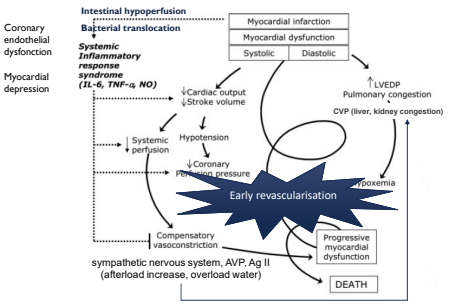
IMPAIRMENT OF MICROCIRCULATION

- Present early in cardiogenic shock
- Microcirculatory network seems to be initially present and functional
- A decreased in capillary density by reduced blood flow
- Organ dysfunction (liver, kidney…)
- Intestinal barrier (bacterial translocation)
- Lipopolysaccharide (endotoxins) in blood stream (endotoxemia)
- Inflammatory response (cytokine release) and multiorgan failure



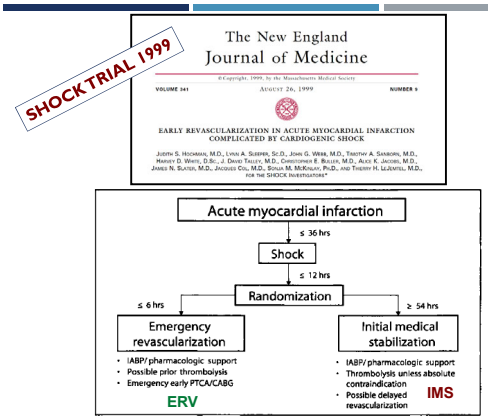
Bertini P et al; Curr Opin Crit Care 2021;27:409-415

SYSTEMIC CARDIOVASCULAR AND INFLAMMATORY DISEASE

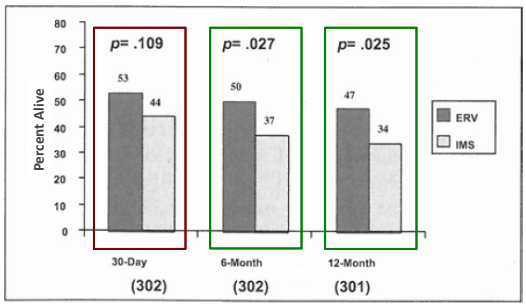


Reynolds HR et al. Circulation 2008; 117:686-97

SHOCK TRIAL 1999



Early myocardial revascularization (SHOCK Trial)



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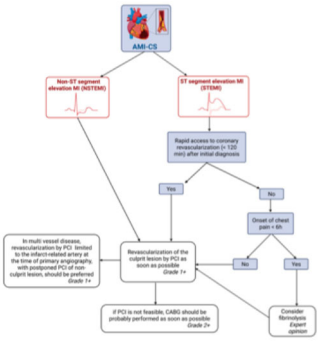

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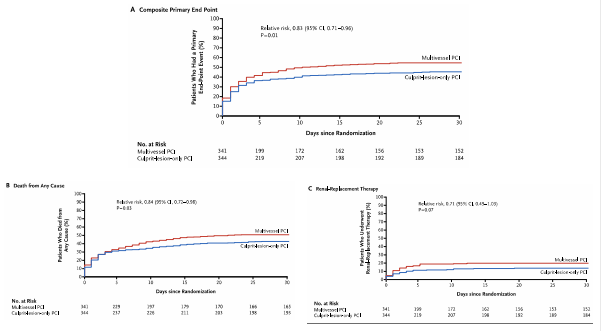
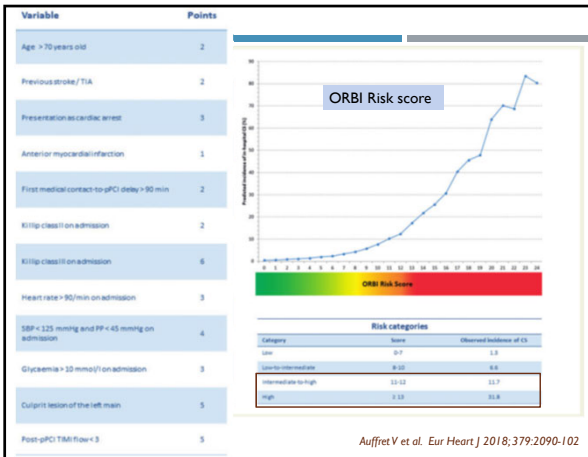
Annals of Intensive Care 16 (2026) 100038

PCI Strategies in Patients with Acute Myocardial Infarction and Cardiogenic Shock



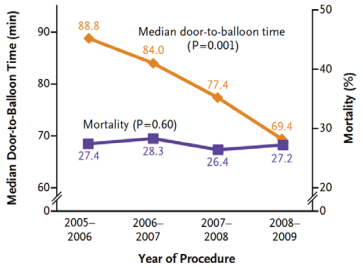
Thiele H et al. New Engl J Med 2017;377: 2419-32



Auffret V et al. Eur Heart J 2018;379:2090-102

IS « TIME IS MUSCLE » CONCEPT ENOUGH?

Cardiogenic Shock (N=9535)

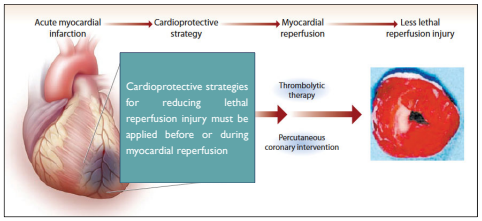


Menees DM. *et al. New Engl J Med 2013; 369:901-9*

From Door-to-Balloon to Door-to-Unload Time

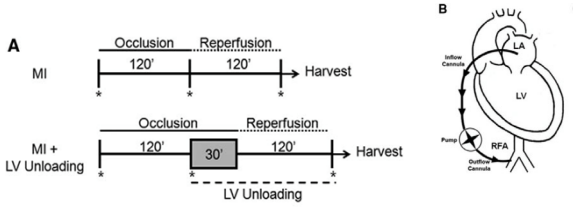
An emerging view on the management of STEMI complicated by cardiogenic shock.

BY NAVIN K. KAPUR, MD

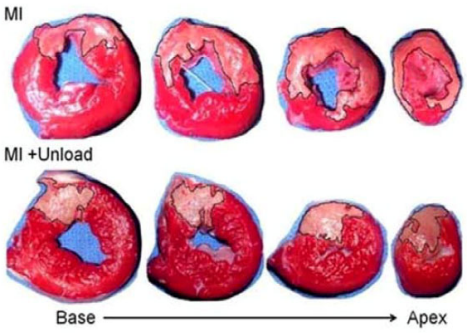


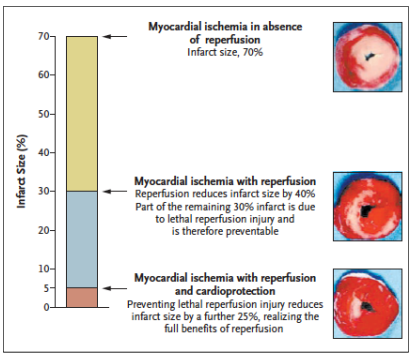
Yellon DM et al. N Engl J Med 2007;357:1121-35

Mechanically Unloading the Left Ventricle Before Coronary Reperfusion Reduces Left Ventricular Wall Stress and Myocardial Infarct Size



Kapur et al. Circulation 2013; 128:328-36

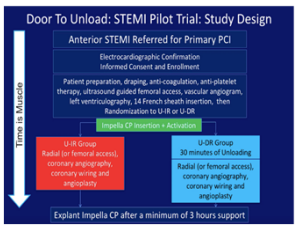


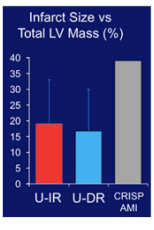


Yellon DM et al. N Engl J Med 2007;357:1121-35

Kapur NK *et al. Circulation* 2019

CLINICAL FAISABILITY

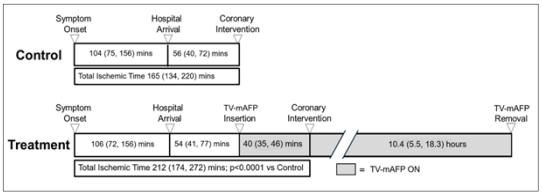


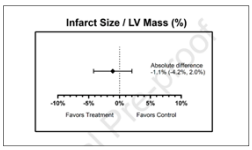


Clinical Variable	U-IR (n=25)	U-DR (n=25)*	p-value
Symptom to Unload Time, Min (mean_SD)	200±152	176±73	NS
Unload to First PTCA Time, Min (mean_SD)	11±7	34±3	<0.01

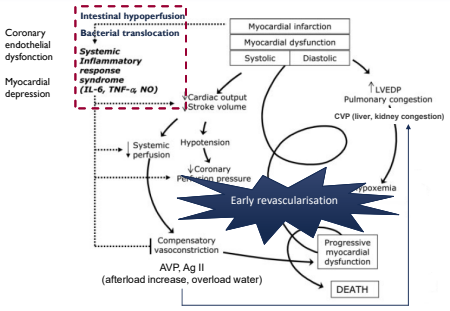
Left ventricular unloading in anterior STEMI without shock: The STEMI Door to unload (DTU) randomized

Kapur NK *et al. JACC 2026 (in press)*



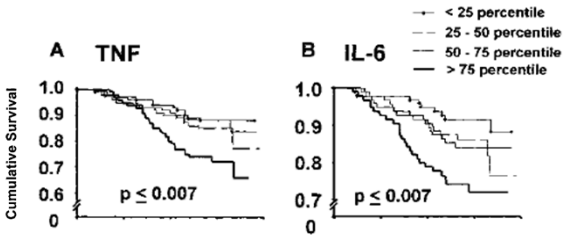


SYSTEMIC CARDIOVASCULAR AND INFLAMMATORY DISEASE



Reynolds HR et al. Circulation 2008; 117:686-97

SYSTEMIC INFLAMMATORY RESPONSE

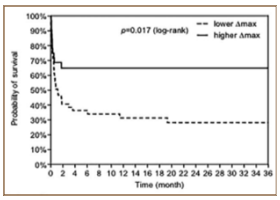


Deswal et al. Circulation 2001

PROGNOSTIC VALUE OF RELATIVE ADRENAL INSUFFICIENCY DURING CARDIOGENIC SHOCK: A PROSPECTIVE COHORT STUDY WITH LONG-TERM FOLLOW-UP

Bagate F et al. Shock 2017; 47:86-92

Plasma cortisol concentration, nmol/L	Survivors (n = 43)	Nonsurvivors (n = 49)	P
T0	666 (497–1,191)	819 (527–1,041)	0.34
T30	949 (603–1,588)	913 (690–1,212)	0.64
T60	1,332 (635–2,094)	1,155 (899–1,614)	0.89
Tmax	1,954 (719–1,659)	1,106 (866–1,451)	0.81
Δmax	369 (122–687)	232 (56–457)	0.04



Mekontso Dessap *et al. Trials* (2022) 23:4
https://doi.org/10.1186/s13063-021-05947-6

Trials

STUDY PROTOCOL Open Access

Low-dose corticosteroid therapy for cardiogenic shock in adults (COCCA): study protocol for a randomized controlled trial

Armand Mekontso Dessap[1,2,3*†], François Bagate[1,2,3†], Clément Delmas[4], Tristan Morichau-Beauchant[5], Bernard Cholley[6], Alain Cariou[7], Benoit Lattuca[8], Mouhamed Moussa[9], Nicolas Mongardon[10,11], Damien Fard[12], Matthieu Schmidt[13], Adrien Bouglé[14], Mathieu Kerneis[15], Emmanuel Vivier[16], François Roubille[17], Matthieu Duprey[18], Véronique Decaluf[19], Thibaud Genet[20], Messaouda Merzoug[21], Etienne Audureau[22] and Pierre Squara[21,23]

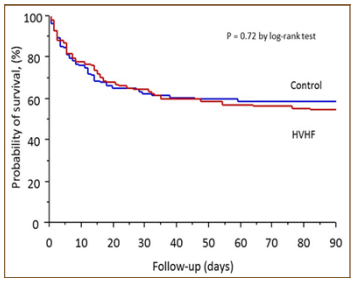
Completed

Low Dose of Hydrocortisone and Fludrocortisone in Adult Cardiogenic Shock. (COCCA)

ClinicalTrials.gov ID NCT03773822
Sponsor CMC Ambroise Paré
Information provided by CMC Ambroise Paré (Responsible Party)
Last Update Posted 2023-11-07

Early High-Volume Hemofiltration versus Standard Care for Post–Cardiac Surgery Shock

The HEROICS Study



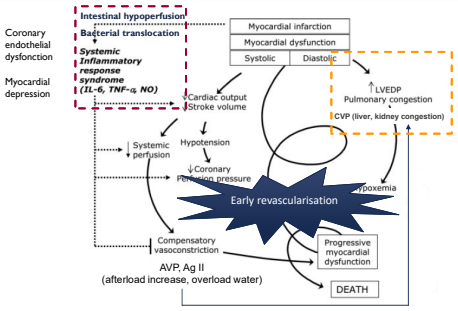
Combes A et al. Am J Respir Crit Care Med 2015;192: 1179-90

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

indicated according to blood gases.		
Fibrinolysis should be considered in patients		
Recommendations	**Class[a]**	**Level[b]**
Immediate PCI is indicated for patients with cardiogenic shock if coronary anatomy is suitable. If coronary anatomy is not suitable for PCI, or PCI has failed, emergency CABG is recommended.[248]	I	B
Invasive blood pressure monitoring with an arterial line is recommended.	I	C
Immediate Doppler echocardiography is indicated to assess ventricular and valvular functions, loading conditions, and to detect	I	C

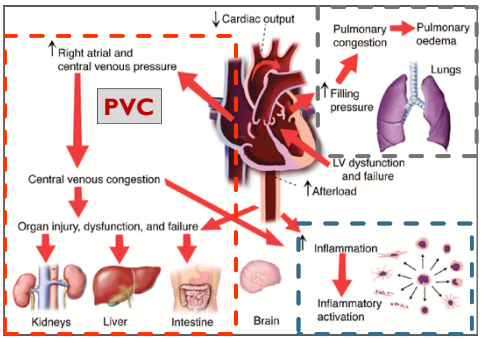
Ibanez B et al. Eur Heart J 2017

SYSTEMIC CARDIOVASCULAR AND INFLAMMATORY DISEASE



Reynolds HR et al. Circulation 2008; 117:686-97

Short term consequences of LV congestion



Harjola VP et al. Eur J heart Failure 2017;19:821-36

Risk indicators for acute kidney injury in cardiogenic shock

Johannes P.C. van den Akker [a,*], Jan Bakker [a,b,c,d], A.B.J Groeneveld [a,1], C.A. den Uil [a,e]

[a] Department of Intensive Care Adults, Erasmus University Medical Center, Dr. Molewaterplein 40, 3015GD, Rotterdam, the Netherlands
[b] Division of Pulmonary, Allergy and Critical Care, Columbia University Medical Center, New York, NY, USA
[c] Division of Pulmonary, Critical Care and Sleep Medicine, New York University Langone-Bellevue Hospital, New York, NY, USA
[d] Department of Intensive Care, Pontificia Universidad Católica de Chile, Santiago, Chile
[e] Department of Cardiology, Erasmus MC, University Medical Center, s-Gravendijkwal 230, Rotterdam 3015, the Netherlands

Univariate and multivariate regression analysis ($n = 62$).

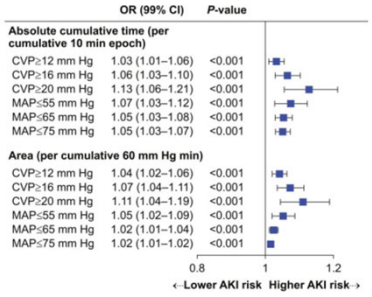
	Univariate			Multivariate		
	Odds ratio	95%CI	*P*	Odds ratio	95%CI	*P*
Central Venous Pressure, mmHg ($n = 38$)	1.199	1.007–1.428	0.041	1.241	1.030–1.495	0.023
Diastolic arterial blood pressure, mmHg	0.950	0.902–1.000	0.049	0.952	0.897–1.010	0.105
PEEP, cm H_2O[a]	1.180	1.017–1.369	0.029	–		
Dobutamine, $\mu g\ kg^{-1}\ min^{-1}$	1.239	0.985–1.559	0.067	1.264	0.976–1.639	0.076

CI: confidence interval. PEEP: positive end expiratory pressure.
[a] For non-ventilated patients we assumed a pressure of 0 cm H_2O.

Journal of Critical Care 50 (2019) 11–16

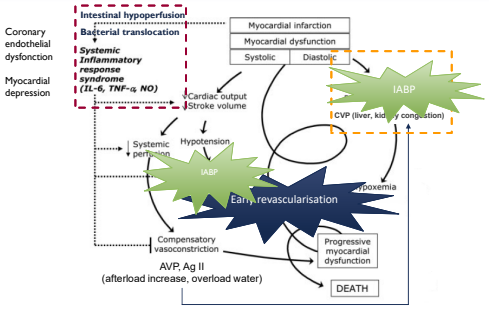
Intraoperative venous congestion rather than hypotension is associated with acute adverse kidney events after cardiac surgery: a retrospective cohort study

BJA



Chen L et al. Br J Anaesth 2022; 128:785-95

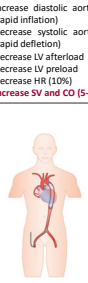
SYSTEMIC CARDIOVASCULAR AND INFLAMMATORY DISEASE

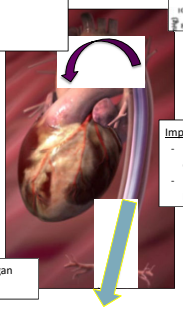


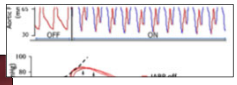
Reynolds HR et al. Circulation 2008; 117:686-97

Intra-aortic balloon pump (IABP)

- Increase diastolic aortic pressure from 30 to 70% (rapid inflation)
- Decrease systolic aortic pressure from 5 to 15% (rapid deflation)
- Decrease LV afterload
- Decrease LV preload
- Decrease HR (10%)
- **Increase SV and CO (5-10%).**





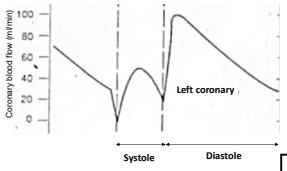


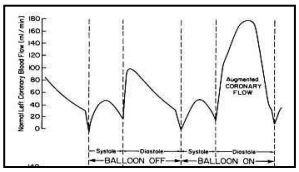
Improvement energetic balance of myocardium
- Increase myocardial oxygen supply (increase coronary perfusion)
- Decrease Myocardial oxygen demand (decrease loading conditions)

- Improvement of end-organ perfusion (Pulsatility)

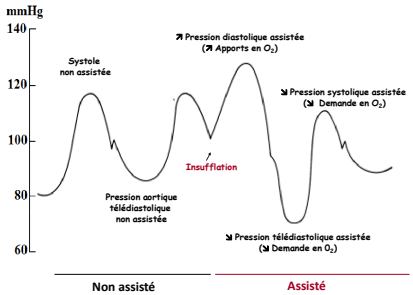
deWaha S et al.Vascular Pharmacology 2014;61:30-34
Ro SK et al. Eur J Cardiothorac Surg 2014; 46:186-92
Aso S et al. Crit Care Med 2016; 44:1974-9

Amélioration perfusion coronaire…





COURBES DE PRESSION ARTÉRIELLE

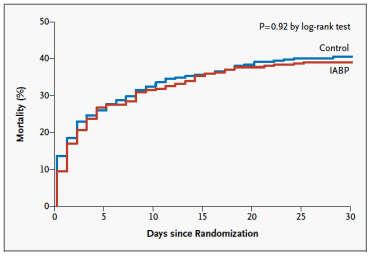


IABP Shock II trial

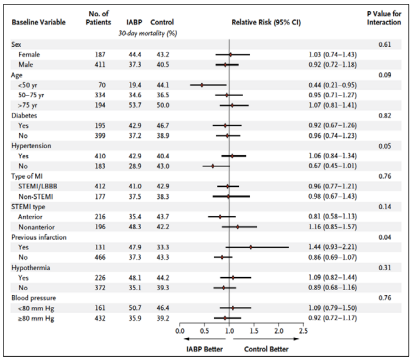
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 4, 2012 VOL. 367 NO. 14

Intraaortic Balloon Support for Myocardial Infarction with Cardiogenic Shock

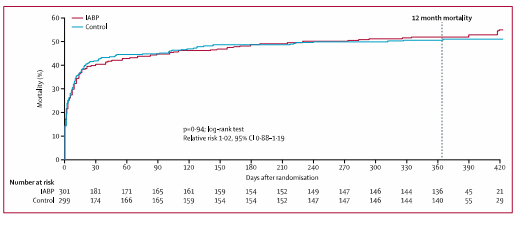


Thiele H et al. N Engl J Med 2012;367:1287-96



Thiele H et al. N Engl J Med 2012;367:1287-96

Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial



Thiele H et al. Lancet 2013; 382:1638-45

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

Recommendations	Class[a]	Level[b]
Short-term MCS should be considered in patients with cardiogenic shock as a BTR, BTD, BTB. Further indications include treatment of the cause of cardiogenic shock or long-term MCS or transplantation.	IIa	C
IABP may be considered in patients with cardiogenic shock as a BTR, BTD, BTB, including treatment of the cause of cardiogenic shock (i.e. mechanical complication of acute MI) or long-term MCS or transplantation.[450]	IIb	C
IABP is not routinely recommended in post-MI cardiogenic shock.[500–502]	III	B

McDonagh TA et al. Eur Heart J 2021; 42:3599-3726

Patients were not eligible for the study if they had undergone resuscitation for more than 30 minutes, had no intrinsic heart action, were in coma with fixed dilation of pupils that was not induced by drugs, had a mechanical cause of cardiogenic shock (e.g., ventricular septal defect or papillary muscle rupture);…

ADVANCES IN HEART FAILURE, MECHANICAL CIRCULATORY SUPPORT AND TRANSPLANT

Intra-Aortic Balloon Pumping in Acute Decompensated Heart Failure With Hypoperfusion: From Pathophysiology to Clinical Practice

Luca Baldetti, MD; Matteo Pagnesi, MD; Mario Gramegna, MD; Alessandro Belletti, MD; Alessandro Beneduce, MD; Vittorio Pazzanese, MD; Francesco Calvo, MD; Stefania Sacchi, MD; Nicolas M. Van Mieghem, MD, PhD; Corstiaan A. den Uil, MD; Marco Metra, MD; Alberto Maria Cappelletti, MD

ABSTRACT: Trials on intra-aortic balloon pump (IABP) use in cardiogenic shock related to acute myocardial infarction have shown disappointing results. The role of IABP in cardiogenic shock treatment remains unclear, and new (potentially more potent) mechanical circulatory supports with arguably larger device profile are emerging. A reappraisal of the physiological premises of intra-aortic counterpulsation may underpin the rationale to maintain IABP as a valuable therapeutic option for patients with acute decompensated heart failure and tissue hypoperfusion. Several pathophysiological features differ between myocardial infarction- and acute decompensated heart failure–related hypoperfusion, encompassing cardiogenic shock severity, filling status, systemic vascular resistances rise, and adaptation to chronic (if preexisting) left ventricular dysfunction. IABP combines a more substantial effect on left ventricular afterload with a modest increase in cardiac output and would therefore be most suitable in clinical scenarios characterized by a disproportionate increase in afterload without profound hemodynamic compromise. The acute decompensated heart failure syndrome is characterized by exquisite afterload-sensitivity of cardiac output and may be an ideal setting for counterpulsation. Several hemodynamic variables have been shown to predict response to IABP within this scenario, potentially guiding appropriate patient selection. Finally, acute decompensated heart failure with hypoperfusion may frequently represent an end stage in the heart failure history: IABP may provide sufficient hemodynamic support and prompt end-organ function recovery in view of more definitive heart replacement therapies while preserving ambulation when used with a transaxillary approach.

Key Words: cardiac output ■ cardiac output, low ■ heart failure ■ intra-aortic balloon pumping ■ patient selection ■ shock, cardiogenic ■ vascular resistance

Baldetti L. Circ Heart Fail. 2021;14:e008527

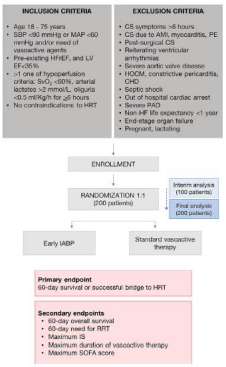
Early intra-aortic balloon pump in acute decompensated heart failure complicated by cardiogenic shock: Rationale and design of the randomized Altshock-2 trial

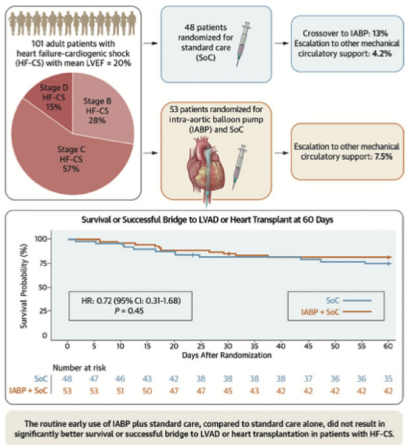
Nuccia Morici, MD[1], Claudia Mariel, MD[2], Alice Sacco, MD[3], Guido Tavazzi, MD[4], Marilia Cipriani, MD[5], Fabrizio Oliva, MD[1], Matteo Rota, MD[2], Gaetano Maria De Ferrari, MD[3], Jessica Campolo, MD[4], Gianfranco Frigerio, MD[5], Serafina Valente, MD[6], Sergio Leonardi, MD[7], Elena Corrado, MD[8], Maurizio Bonivoli, MD[9], Daniele Grossoni, MD[9], Luisa Cacciavillani, MD[10], Maria Felgerio, MD[11], and Federico Pappalardo, MD[12], for the Altshock-2 Group. *Milan, Italy; Pavia, Italy; Brescia, Italy; Verona, Italy; Siena, Italy; Bozzano, Italy; Rimini, Italy; Padova, Italy; Palermo, Italy*

Background Cardiogenic shock (CS) is a systemic disorder associated with dismal short-term prognosis. Given its time-dependent nature, mechanical circulatory support may improve survival. Intra-aortic balloon pump (IABP) had gained widespread use because of the easiness to implant and the low rate of complications; however, a randomized trial failed to demonstrate benefit on mortality in the setting of acute myocardial infarction. Acute decompensated heart failure with cardiogenic shock (ADHF-CS) represents a growing resource-intensive scenario with scant data and indications on the best management. However, a few data suggest a potential benefit of IABP in this setting. We present the design of a study aimed at addressing this research gap.

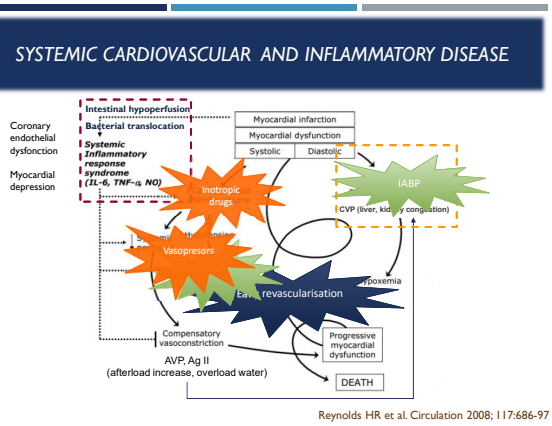
Methods and design The Altshock-2 trial is a prospective, randomized, multicenter, open-label study with blinded adjudicated evaluation of outcomes. Patients with ADHF-CS will be randomized to early IABP implantation or to vasoactive treatment. The primary end point will be 60 days patients' survival or successful bridge to heart replacement therapy. The key secondary end point will be 60-day overall survival; 60-day need for renal replacement therapy; in-hospital maximum inotropic score, maximum duration of inotropic/vasopressor therapy, and maximum sequential organ failure assessment score. Safety end points will be in-hospital occurrence of bleeding events (Bleeding Academic Research Consortium >3), vascular access complications and systemic (noncerebral) embolism. The sample size for the study is 200 patients.

Implications The Altshock-2 trial will provide evidence on whether IABP should be implanted early in ADHF-CS patients to improve their clinical outcomes. (Am Heart J 2021;233:39-47.)



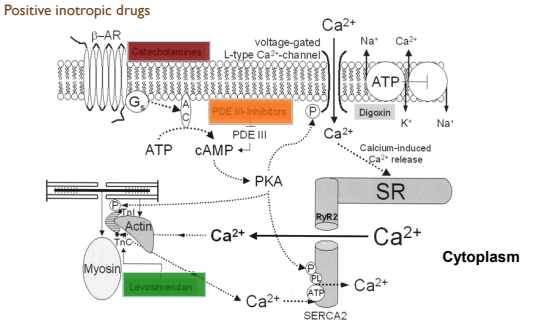


J Am Coll Cardiol. 2025

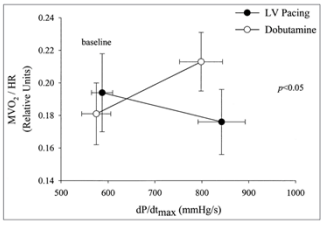


Reynolds HR et al. Circulation 2008; 117:686-97

Positive inotropic drugs



Toller WG. et al. Anesthesiology 2006

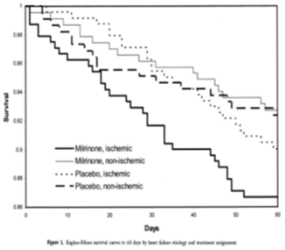


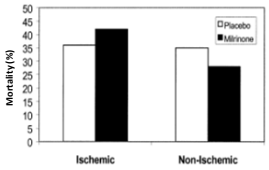
Dobutamine

- Increased myocardial oxygen consumption
- High incidence of arrhythmia
- Tolerance (receptor internalisation, decoupling….)
- Profound vasodilation

Nelson GS et al. Circulation 2000; 102: 3053-9

INOTROPES COULD BE HARMFUL
IN DECOMPENSATED HEART FAILURE FROM OPTIME-CHF STUDY?

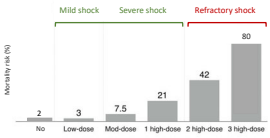




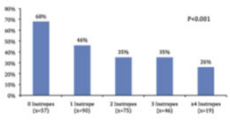
Felker GM et al. J Am Coll Cardiol 2003;41:997-1003

Refractory cardiogenic shock

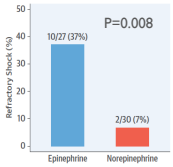
(from <1% to more than 15%...)



Samuels LE et al. J Card Surg 1999; 14:288-93



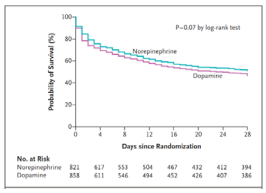
Basir MB et al. Am J Cardiol 2017;119:845-51

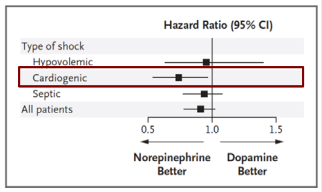


Levy B et al. J Am Coll Cardiol 2018; 72:173-81

The NEW ENGLAND JOURNAL of MEDICINE

Comparison of Dopamine and Norepinephrine in the Treatment of Shock





Adverse events			
Arrhythmias — no. (%)	207 (24.1)	102 (12.4)	<0.001
Atrial fibrillation	176 (20.5)	90 (11.0)	
Ventricular tachycardia	21 (2.4)	8 (1.0)	
Ventricular fibrillation	10 (1.2)	4 (0.5)	

De Backer D et al. N Engl J Med 2010;362:779-89

Perioperative Use of Dobutamine in Cardiac Surgery and Adverse Cardiac Outcome

Propensity-adjusted Analyses

Jean-Luc Fellahi, M.D., Jean-Jacques Parienti, M.D.,† Jean-Luc Hanouz, M.D., Ph.D.,‡ Benoît Plaud, M.D., Ph.D.,§ Bruno Riou, M.D., Ph.D.,‖ Alexandre Ouattara, M.D., Ph.D.#*

Table 4. Catecholamines: Crude and Adjusted Effects for Major Cardiac Morbidity and Global In-hospital Mortality Endpoints

Method	Odds Ratio	95% Confidence Interval	*P* Value
Major cardiac morbidity			
Crude	4.2	[2.5–7.3]	<0.001
Stratifying on PS quartiles*	2.1	[1.0–4.4]	<0.05
Covariate adjustment using PS†	2.3	[1.0–5.0]	<0.05
Marginal structural models‡	1.8	[1.3–2.5]	<0.001
PS matching	3.0	[1.2–7.3]	<0.02
In-hospital mortality			
Crude	12.9	[3.7–45.2]	<0.001
Stratifying on PS quartiles	—	—	—
Covariate adjustment using PS§	2.7	[0.4–20.5]	0.33
Marginal structural models	—	—	—
PS matching	2.0	[0.1–32.0]	0.63

* Model adjusted for higher cardiac troponin I level 24 h after surgery ($P < 0.001$) and longer cardiopulmonary bypass time ($P < 0.001$). † Model adjusted for higher cardiac troponin I level 24 h after surgery ($P < 0.001$), combined surgery ($P < 0.04$) and higher propensity score (PS) ($P < 0.15$). ‡ Model adjusted for higher cardiac troponin I level 24 h after surgery ($P < 0.001$), combined surgery ($P < 0.001$), longer aortic cross clamping time ($P < 0.009$), and lower left ventricular ejection fraction ($P < 0.001$). § Model adjusted for higher PS ($P < 0.01$).

Anesthesiology 2008; 108:979-87

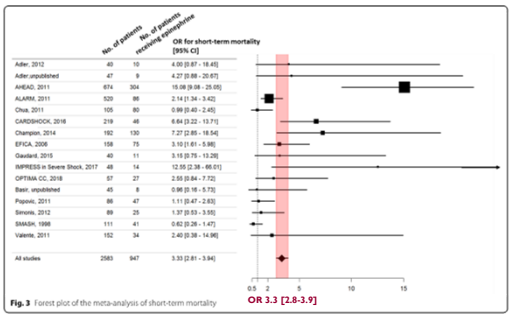


Fig. 3 Forest plot of the meta-analysis of short-term mortality

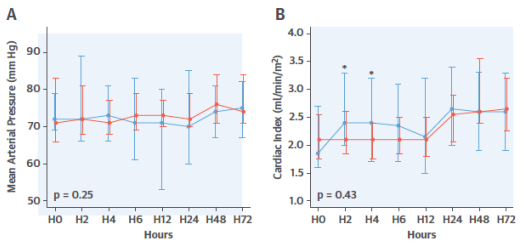
Léopold V et al. Intensive Care Med 2018;44:847-56

Epinephrine Versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction

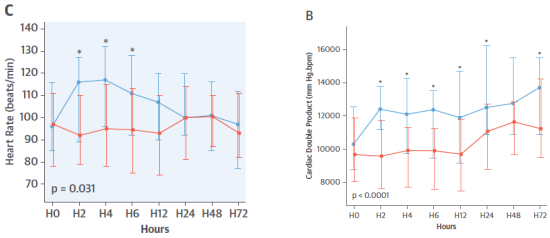
Levy B et al. J Am Coll Cardiol 2018; 72:173-81

OptimaCC

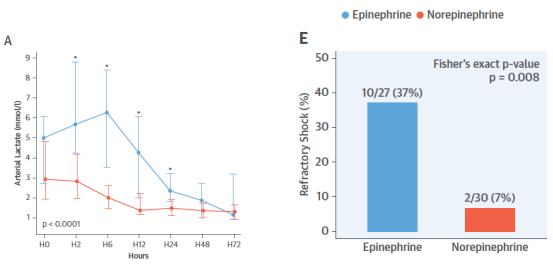
- Prospective, double-blinded, multicenter RCT
- Haemodynamic efficacy and tolerance of Epinephrine (n=27) and norepinephrine (n=30) in patients with CS due to MI successfully revascularized
- Tested drugs were increased by 0.02 µg/kg/min
- Primary efficacy endpoint was increase in CO
- Primary safety endpoint was the incidence of refractory CS (major cardiac dysfunction according to echocardiography, lactates levels, acute deterioration organ function despite 1 µg/kg/min Epinephrine or 10 µg/kg/min of dobutamine)
- Dobutamine was used in 67% of patients in both groups



Levy B et al. J Am Coll Cardil 2018; 72:173-81



Levy B et al. J Am Coll Cardil 2018; 72:173-81



Levy B et al. J Am Coll Cardil 2018; 72:173-81

ADVERSE EFFECTS OF CARDIOVASOACTIVE DRUGS

Increased myocardial oxygen demand

Myocardial ischemia

Tachycardia

Malignant arrhythmia

Increase left ventricular afterload

Pro-apoptotic effect

Valente S et al. Int J Cardiol 2007;114:176-82
Singh K et al. Cardiovasc Res 2000;45:713–9

Involvement of RV dysfunction or failure

Incidence of CS 33%

Primary predominant 3-5%

Higher intra-hospital mortality

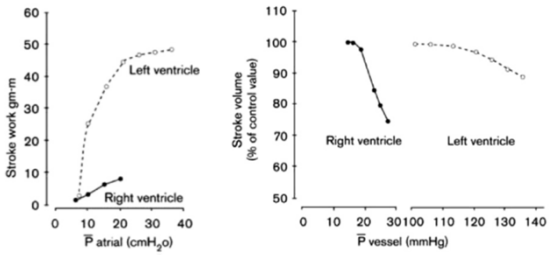
9.4% versus 3.0%

More difficult management

Cardiac index <2.2 L/min/m² despite continuous high dose inotropes or >1 inotrope or vasopressor medication + any of the following criteria:	
	CVP >10 mm Hg CVP/PCWP ratio >0.63 PAPi <2 RVSWI <450 mm Hg*mL/m² RV dysfunction and/or dilation on echocardiography: TAPSE <17 mm RV systolic TDI S' velocity <10 cm/sec RVFAC <35% RV free wall longitudinal strain <−20% RV basilar diameter >42 mm RV short axis (or mid cavity) diameter >35 mm
Severe RV dysfunction	CVP >15 mm Hg CVP/PCWP ratio >0.8 PAPi <1.5 RVSWI <300 mm Hg*mL/m²
Clinical	Ascites Edema Bilirubin elevation Creatinine elevation

Kapur NK et al. Circulation 2017; 136:314-26

Different adaptative response to changes in loading conditions



Ventetuolo CE et al. Ann Am Thorac Soc 2014; 11:811-22

RIGHT VENTRICULAR DYSFUNCTION

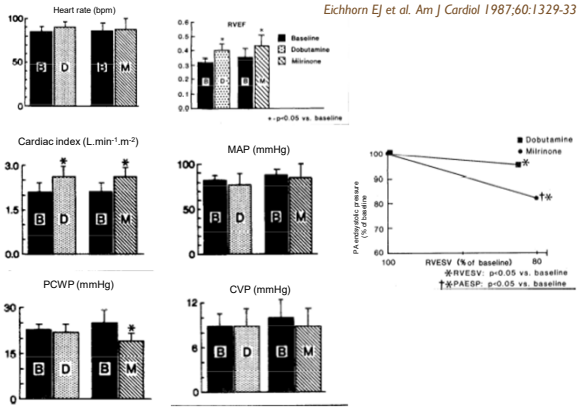
Pulmonary artery pulsatility index= (sPAP-dPAP)/RAP < 1.0

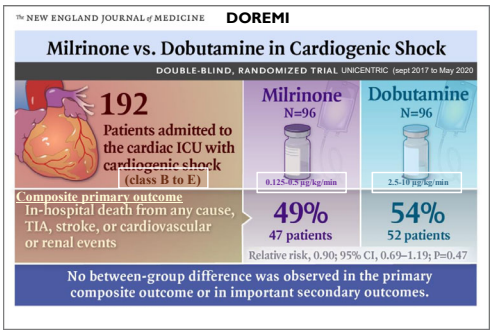
RAP/PCWP > 0.6

Korabathina R et al. Catheter Cardiovasc Interv 2012; 80:593-600

Tehrani BN et al. J Am Coll Cardiol 2019; 73:1659-69

Eichhorn EJ et al. Am J Cardiol 1987;60:1329-33





Mathew R et al. New Engl Med 2021 385; 516-25

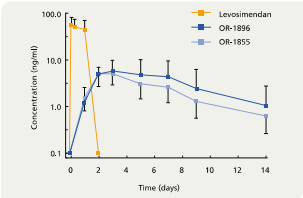
Levosimendan: Attracting properties...

- Inotropic effects:
  - Calcium sensitizer of myocardial contractile proteins
  - Improve cardiac contractility without increasing intracellular calcium concentration
  - Myocardial oxygen consumption unchanged
  - Diastolic function preserved
- Vasodilatory effects (openers K-ATP channels)
  - Coronary, pulmonary and systemic vasodilation
- Organ protective properties (myocardium, liver, kidney, digestive tractus…)
- Anti-apoptotic properties
- Anti-inflammatory effects

Rognoni et al. Recent Pat Cardiovascular Drug Discov 2011; 6:9-15

PHARMACOKINETICS

- Levosimendan: 0.2 µg/kg/min during 24h without initial bolus
- Active metabolites between 7 and 14 days



Levosimendan vs Dobutamine for Patients With Acute Decompensated Heart Failure

The SURVIVE Randomized Trial

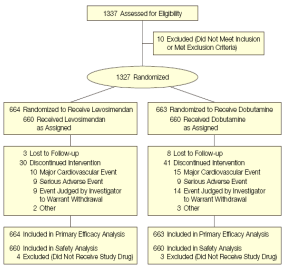
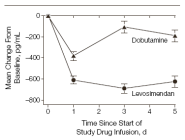
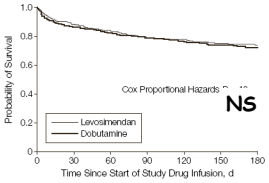
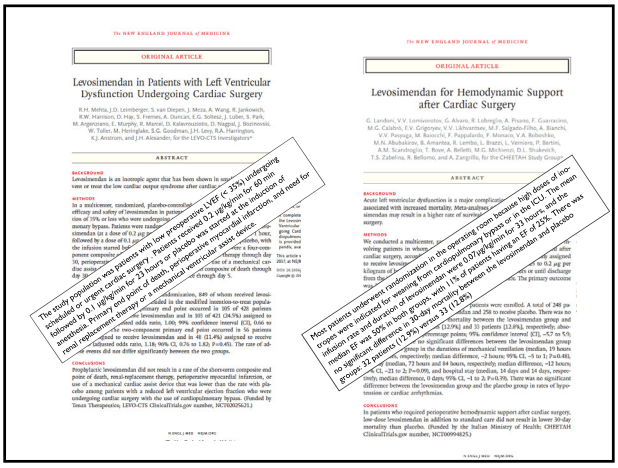


Figure 3. Mean Change From Baseline in B-Type Natriuretic Peptide Levels at 1, 3, and 5 Days by Treatment Group



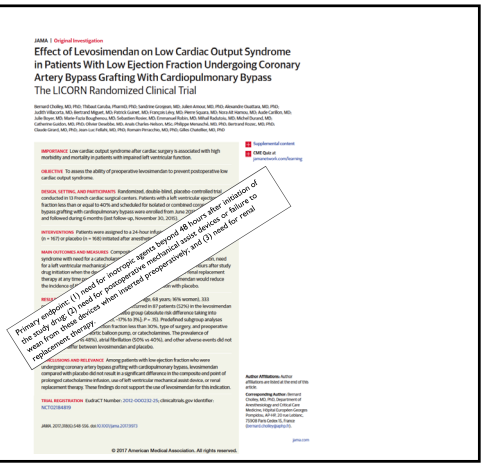


Mebazaa A et al. JAMA 2007; 297:1883-91



The study population was patients with low preoperative LVEF (< 35%) undergoing scheduled or urgent cardiac surgery. Patients received 0.2 µg/kg/min for 60 min followed by 0.1 µg/kg/min for 23 hours or placebo was started at the induction and need for anesthesiology. Primary point of death, perioperative myocardial infarction, renal replacement therapy or a mechanical ventricular assist device.

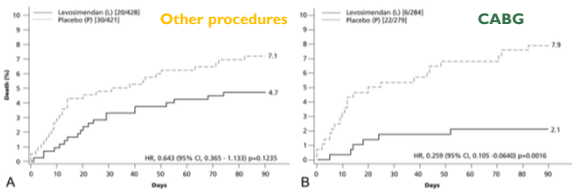
Most patients underwent randomization in the operating room because high doses of inotropes were indicated for weaning from cardiopulmonary bypass or in the ICU. The mean infusion rate and duration of levosimendan were 0.07 µg/kg/min for 33 hours and there was no significant difference in 30-day mortality between the levosimendan and placebo groups: 31 patients (12.9%) versus 33 (13.8%).



Primary endpoint: (1) need for inotropic agents beyond 48 hours after initiation of the study drug; (2) need for postoperative mechanical assist devices or failure to wean from these devices when inserted preoperatively; and (3) need for renal replacement therapy. These findings do not support the use of levosimendan for this indication.

Use of Levosimendan in Cardiac Surgery: An Update After the LEVO-CTS, CHEETAH, and LICORN Trials in the Light of Clinical Practice

Fabio Guarracino, MD, PhD, Matthias Heringlake, MD, PhD,† Bernard Cholley, MD, PhD,‡§ Dominique Bettex, MD, PhD,¶ Stefaan Bouchez, MD, PhD,‖ Vladimir V. Lomivorotov, MD, PhD,** Angela Rajek, MD,†† Matti Kivikko, MD, PhD,‡‡§§ and Piero Pollesello, PhD‡‡*



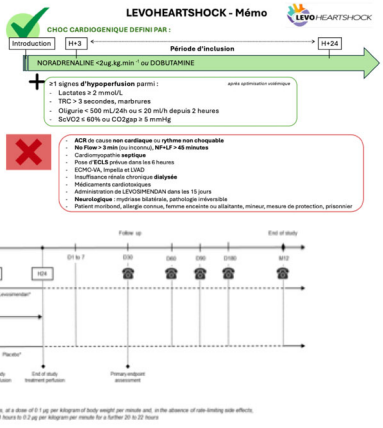
J Cardiovasc Pharmacol 2018; 71:1-9

LEVO-HEART SHOCK STUDY

- Effect of early use of levosimendan versus placebo on top of a conventional strategy of inotrope use on a combined morbidity-mortality endpoint in patients with cardiogenic shock
- The study goal is to evaluate the effect of the early use of levosimendan versus placebo on top of a conventional use of inotrope with regard to a composite endpoint of 30-day mortality and/or ExtraCorporeal Life Support (ECLS) requirement and/or dialysis.
- **Experimental group**: patients with cardiogenic shock treated with levosimendan in addition to the conventional strategy.
- **Control group**: Patients with cardiogenic shock treated with placebo for levosimendan in addition to the conventional strategy.
- **610 patients should be included**

PROTOCOLE LEVOHEARTSHOCK

Effect of early use of levosimendan versus placebo on top of a conventional strategy of inotrope use on a combined morbidity-mortality endpoint in patients with cardiogenic shock.
Eudra CT : 2019-001563-74
EUCT: 2024-513811-29-00.

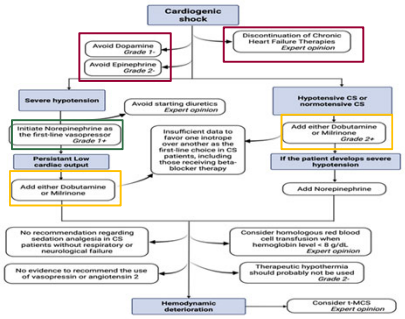


Inotropic agents		
Inotropic agents may be considered in patients with SBP <90 mmHg and evidence of hypoperfusion who do not respond to standard treatment, including fluid challenge, to improve peripheral perfusion and maintain end-organ function.[387]	IIb	C
Inotropic agents are not recommended routinely, due to safety concerns, unless the patient has symptomatic hypotension and evidence of hypoperfusion.[387,467,478]	III	C

Vasopressors		
A vasopressor, preferably norepinephrine, may be considered in patients with cardiogenic shock to increase blood pressure and vital organ perfusion.[485–487]	IIb	B

Mc Donagh TA et al. Eur Heart J 2021; 42:3599-3726

Arch Cardiovasc Dis 2026 Mar 31:S1875-2136(26)00051-3
Annals of Intensive Care 16 (2026) 100038



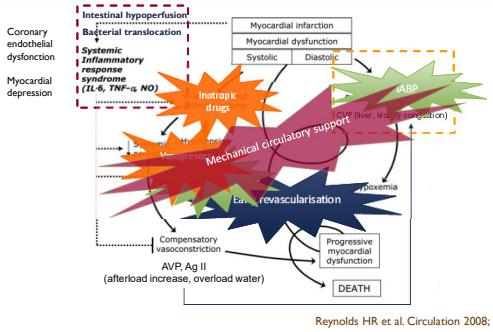
PLEASE, USE VASOACTIVE AND INOTROPICS AT THE LOWEST DOSE AND DURING THE SHORTEST TIME POSSIBLE …

Recruiting

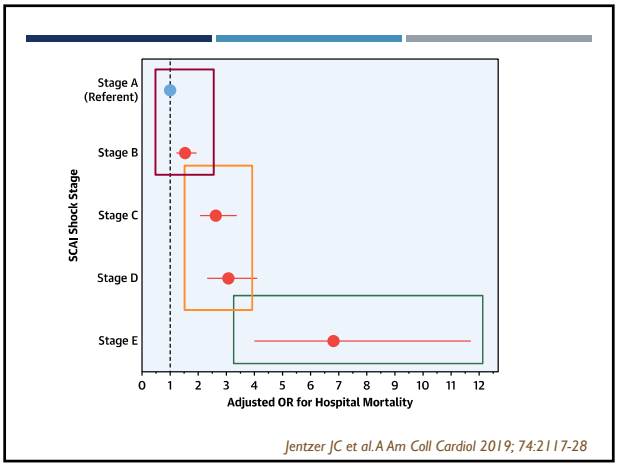
CAPITAL DOREMI 2: Inotrope Versus Placebo Therapy for Cardiogenic Shock (DOREMI-2)

ClinicalTrials.gov ID NCT05267886
Sponsor Ottawa Heart Institute Research Corporation
Information provided by Ottawa Heart Institute Research Corporation (Responsible Party)
Last Update Posted 2024-04-08

SYSTEMIC CARDIOVASCULAR AND INFLAMMATORY DISEASE



Reynolds HR et al. Circulation 2008; 117:686-97



Jentzer JC et al.A Am Coll Cardiol 2019; 74:2117-28

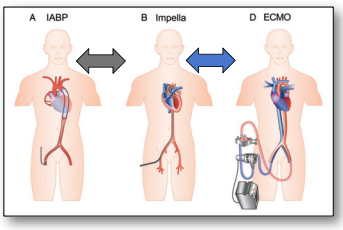
REVIEW Open Access

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

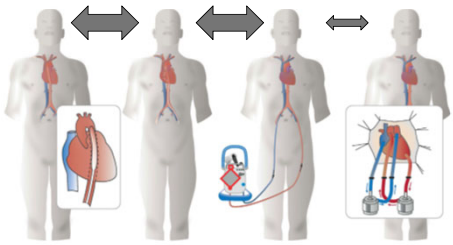
Annals of Intensive Care
a SpringerOpen Journal

Ann Intensive Care 2015; 5:17

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



SHORT TERM MECHANICAL CIRCULATORY SUPPPORT (STMCS)



IABP IMPELLA ECMO pVAD

Short term circulatory support and cardiogenic shock

IABP-SHOCK II

NEJM 2012

ECMO-CS

CIRCULATION 2023

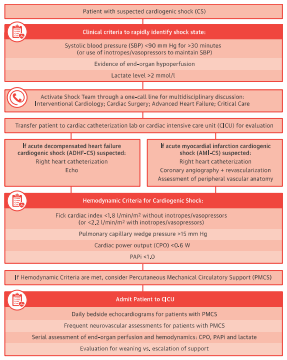
ECLS-SHOCK

NEJM 2023

DANGER

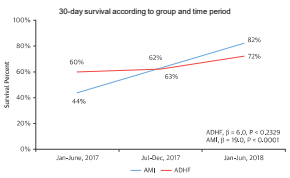
NEJM 2024

BY WHO? CARDIOGENIC SHOCK TEAM

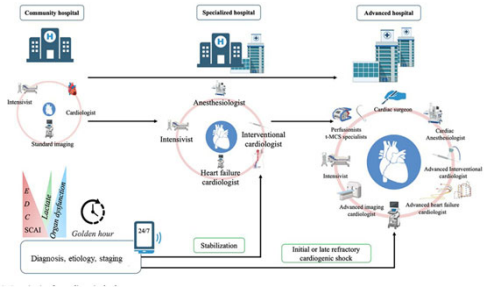


Multidisciplinary "Shock team" :

- Tertiary expert center
- Standardized protocol (clear objectives)
- Advanced hemodynamic monitoring modal)



Tehrani B et al. JACC 2019; 73:1659-69



Arch Cardiovasc Dis 2026 Mar 31:S1875-2136(26)00051-3
Annals of Intensive Care 16 (2026) 100038

Auteur	Type d'étude	Population	Composition de la team	Déclenchement	Modalité de réunion	Outcome
Garan et al, 2016	Lettre scientifique	Choc Cardiogénique	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel Réanimateur	Non rapporté	Réunion virtuelle Application smartphone	Non rapporté
Taleb et al, 2019	Monocentrique prospectif, contrôlé contre cohorte historique rétrospective	Choc Cardiogénique n= 123 (Shock team) n= 121 (control)	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel Réanimateur	Suspicion clinique de CC	Réunion virtuelle Appel téléphonique	Mortalité hospitalière 52% vs 39% (control vs Shock Team)
Tehrani et al, 2019	Monocentrique prospectif	Choc Cardiogénique n= 204 (Shock team)	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel Réanimateur	Suspicion clinique de CC	Réunion virtuelle Appel téléphonique	Mortalité à 30 jours 53% vs 39% (control vs Shock Team)
Lee et al, 2020	Monocentrique prospectif, contrôlé contre cohorte historique rétrospective	Choc Cardiogénique n= 64 (Shock team) n=36 (control)	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel Réanimateur	Cas validé par le Spécialiste de l'IC avancée	Réunion virtuelle Application smartphone	Mortalité à 8 mois 67% vs 43% (control vs Shock Team)
Gibbs et al, 2020	Etude qualitative	Choc Cardiogénique	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel ACT/ECMO spécialiste	Non rapporté	Réunion virtuelle Appel téléphonique	Non rapporté
Hernandez-Perez et al, 2021	Monocentrique rétrospectif	Choc Cardiogénique réfractaire n= 130 (Shock team)	Chirurgien cardiaque Spécialiste de l'IC avancée Cardiologue interventionnel Réanimateur	Choc cardiogénique réfractaire	Réunion virtuelle Appel téléphonique	Mortalité hospitalière 43%
Mannino et al, 2021	Communication, non revue par les pairs Monocentrique rétrospectif	Choc Cardiogénique ischémique Impella® seule n= 154 (Shock team) n= 37 (control)	Cardiologue interventionnel Réanimateur Spécialiste de l'IC avancée si besoin	Suspicion clinique de CC ischémique	Non rapporté	Mortalité hospitalière 56% vs 29% (control vs Shock Team)

Multidisciplinary cardiogenic shock team approach improves the long-term outcomes of patients suffering from refractory cardiogenic shock treated with short-term mechanical circulatory support

François-Xavier Hérion[1,2†], Antoine Bourton[1,2†], Claire Oddos[1], Karine Nubret[1], Clément Aguerreche[1], Astrid Quessard[1], Maxime Faure[1], Edouard Gerbaud[3,4], Mathieu Pernot[3,4], Julien Imbault[1,2], and Alexandre Ouattara[1,2]

ESC European Society of Cardiology
European Heart Journal. Acute Cardiovascular Care (2023) https://doi.org/10.1093/ehjacc/zuad108

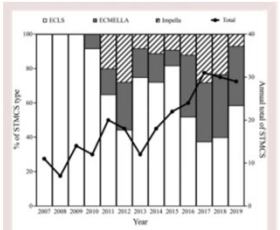
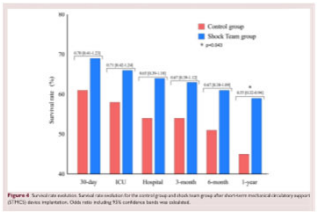


Figure 3 Trends in STMCS over the years. ECLS, extracorporeal life support; ECMELLA, Association of ECLS and Impella® 2.5, CP or 5.0; IABP, intra-aortic balloon pump; STMCS, short-term mechanical circulatory support. The 'Impella®' category includes only CP and 5.0 used alone.



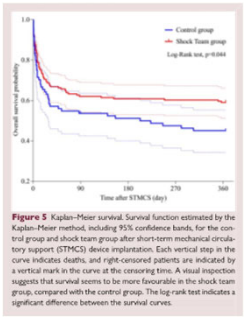
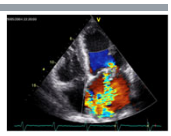
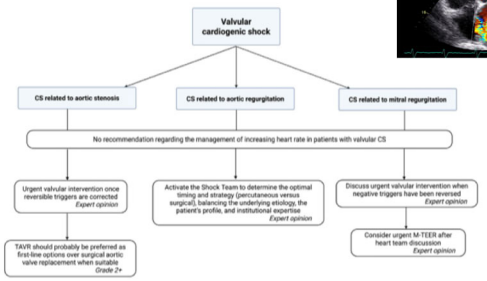


Figure 5 Kaplan–Meier survival. Survival function estimated by the Kaplan–Meier method, including 95% confidence bands, for the control group and shock team group after short-term mechanical circulatory support (STMCS) device implantation. Each vertical step in the curve indicates deaths, and right-censored patients are indicated by a vertical mark in the curve at the censoring time. A visual inspection suggests that survival seems to be more favourable in the shock team group, compared with the control group. The log-rank test indicates a significant difference between the survival curves.





Arch Cardiovasc Dis 2026 Mar 31:S1875-2136(26)00051-3
Annals of Intensive Care 16 (2026) 100038

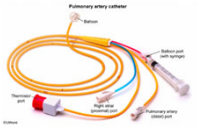
Use of pulmonary artery catheter?

Randomized studies and several meta-analyses have failed to confirm a clinical benefit of the PAC in a wide range of critically ill patient pathologies

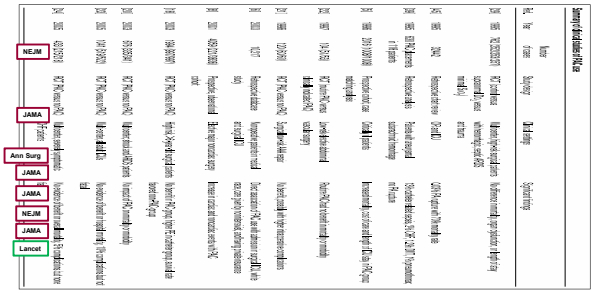
Current recommendations of the European Society of Intensive Care Medicine still consider PAC as a useful tool in some patients with severe CS

Especially in case of right ventricular dysfunction or CS unresponsive to initial therapies, reflecting standard practice in expert centers in the management of this condition.

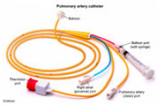
Mebazaa A et al. Intensive Care Med. 2018;44:760-73



Hadian M and Pinsky MR Crit care 2006;10(suppl3):S8



No study conducted in patients suffering from cardiogenic shock treated or not by mechanical circulatory support

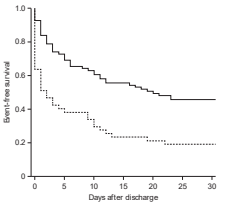


Impact of Pulmonary Artery Catheter Use on Short- and Long-Term Mortality in Patients with Cardiogenic Shock

Original Research

Rossello X et al. Cardiology 2017;136:61-9

- Prospective cohort (2005-2009) of patients suffering from CS (n=129) divided in two groups PAC + versus PAC –
- Logistic regression identifying risk factors of 30-day mortality
- 30-day mortality rate was 64%



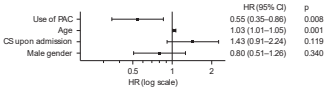


Table 3. In-Hospital Outcomes After Propensity Score Matching

	Cardiogenic Shock				Heart Failure			
Outcome	PAC (n = 11,139)	No PAC (n = 11,139)	OR (95% CI)*	*P* Value	PAC (n = 11,640)	No PAC (n = 11,640)	OR (95% CI)*	*P* Value
Mortality	34.9%	37.0%	0.91 (0.87–0.97)	.001	6.7%	2.4%	2.95 (2.56–3.39)	<.001
MCS use	39.0%	25.8%	1.84 (1.73–1.94)	<.001	3.0%	0.3%	11.18 (7.78–16.07)	<.001
AKI requiring hemodialysis	9.8%	6.1%	1.66 (1.51–1.84)	<.001	1.0%	3.6%	3.62 (2.95–4.45)	<.001
Acute kidney injury	56.4%	48.6%	1.37 (1.30–1.44)	<.001	32.0%	18.1%	2.13 (2.00–2.26)	<.001
Transfusion	27.9%	22.8%	1.31 (1.23–1.39)	<.001	13.3%	5.6%	2.57 (2.34–2.83)	<.001
Intubation	56.9%	50.8%	1.28 (1.21–1.35)	<.001	10.0%	2.1%	5.14 (4.47–5.91)	<.001
Vascular complications	19.6%	17.6%	1.14 (1.07–1.22)	<.001	5.9%	1.6%	3.84 (3.26–4.52)	<.001
Acute respiratory failure	45.9%	43.4%	1.11 (1.05–1.17)	<.001	13.4%	6.9%	2.07 (1.90–2.27)	<.001
Major Bleeding	7.8%	7.7%	1.03 (0.93–1.13)	.616	3.5%	2.2%	1.60 (1.37–1.87)	<.001
LOS ≥5 d	81.0%	67.7%	2.04 (1.92–2.17)	<.001	75.6%	43.0%	4.10 (3.88–4.34)	<.001
PCI	19.0%	22.4%	0.81 (0.76–0.87)	<.001	2.8%	1.5%	1.92 (1.60–2.31)	<.001
Cardiac arrest	15.1%	18.9%	0.76 (0.71–0.81)	<.001	1.5%	0.6%	2.67 (2.01–3.56)	<.001
Median LOS, d (IQR)	12 (6–20)	8 (3–15)	–	<.001	8 (5–14)	4 (2–7)	-	<.001
Median hospital costs	$50,911	$31,734	–	<.001	$20,168	$7,869	-	<.001
CABG	15.0%	13.2%	1.16 (1.08–1.25)	<.001	3.5%	0.4%	9.41 (6.91–12.81)	<.001
LVAD placement	4.5%	1.4%	3.27 (2.73–3.91)	<.001	2.2%	0.3%	7.19 (5.09–10.16)	<.001
Heart transplantation	2.0%	0.8%	2.45 (1.91–3.14)	<.001	2.1%	0.3%	8.54 (5.80–12.56)	<.001
Nursing home discharge	10.8%	9.5%	1.17 (1.03–1.33)	.013	15.0%	12.6%	1.90 (1.64–2.20)	<.001

AKI, acute kidney injury; CABG, coronary artery bypass graft; IQR, interquartile range; MCS, mechanical circulatory support; PCI, percutaneous coronary intervention; LVAD, left ventricular assist device; LOS, length of stay.

Hernandez GA et al. J Cardiac Fail 2019; 25:364-71

ESC European Society of Cardiology

European Journal of Heart Failure (2020) 22, 1315–1341 doi:10.1002/ejhf.1922

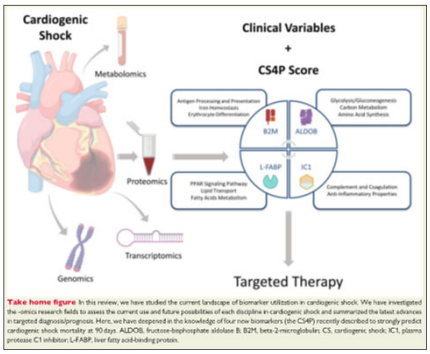
POSITION PAPER

Epidemiology, pathophysiology and contemporary management of cardiogenic shock – a position statement from the Heart Failure Association of the European Society of Cardiology

Ovidiu Chioncel[1,2*], John Parissis[3,4], Alexandre Mebazaa[5], Holger Thiele[6,7], Steffen Desch[6,7], Johann Bauersachs[8], Veli-Pekka Harjola[9], Elena-Laura Antohi[1,2], Mattia Arrigo[10], Tuvia B. Gal[11,12], Alena Celutkiene[13], Sean P. Collins[14], Daniel DeBacker[15], Vlad A. Iliescu[1,2], Ewa Jankowska[16], Tiny Jaarsma[17,18], Kalliopi Keramida[19,20], Mitja Lainscak[20,21], Lars H. Lund[22,23], Alexander R. Lyon[24,25], Josep Masip[26,27], Marco Metra[28], Oscar Miro[29,30], Andrea Mortara[31], Christian Mueller[32], W Wilfred Mullens[33,34], Maria Nikolaou[3], Massimo Piepoli[35], Susana Price[36], Giuseppe Rosano[37], Antoine Vieillard-Baron[38,39], Jean M. Weinstein[40], Stefan D. Anker[41,42], Gerasimos Filippatos[43,44], Frank Ruschitzka[45], Andrew JS Coats[46], and Petar Seferovic[46,47]

> "Based on expert opinion, PAC is currently recommended in selected patients who failed to respond to initial therapeutic interventions (persistence of hypotension and hypoperfusion)…"

Molecular signature of cardiogenic shock



Take home figure In this review, we have studied the current landscape of biomarker utilization in cardiogenic shock. We have investigated the -omics research fields to assess the current use and future possibilities of each discipline in cardiogenic shock and summarized the latest advances in targeted diagnosis/prognosis. Here, we have deepened in the knowledge of four new biomarkers (the CS4P) recently described to strongly predict cardiogenic shock mortality at 90 days. ALDOB, fructose-bisphosphate aldolase B; B2M, beta-2-microglobulin; CS, cardiogenic shock; IC1, plasma protease C1 inhibitor; L-FABP, liver fatty acid-binding protein.

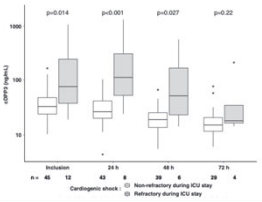
Iborra-Egea O et al. Eur heart J 2020;41:3839-48

ESC European Society of Cardiology

European Journal of Heart Failure (2020) 22, 279–286 doi:10.1002/ejhf.1600

RESEARCH ARTICLE

Circulating dipeptidyl peptidase 3 and alteration in haemodynamics in cardiogenic shock: results from the OptimaCC trial



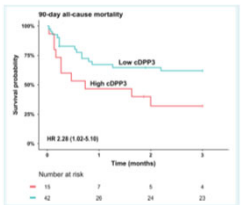
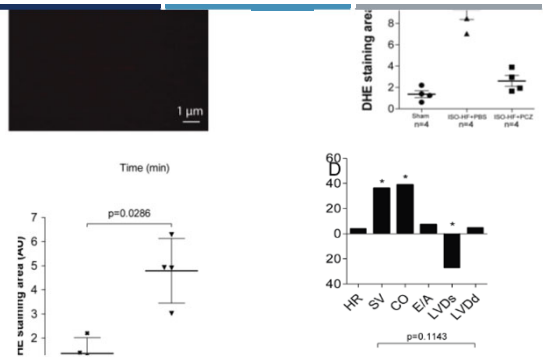
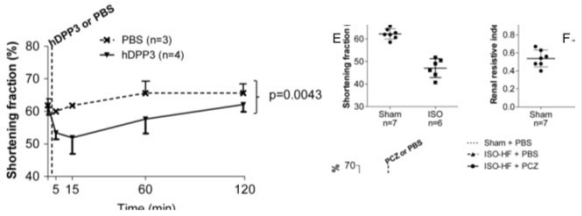


Figure 2 Kaplan–Meier analysis of 90-day all-cause mortality in cardiogenic shock patients with, at inclusion, high (circulating dipeptidyl peptidase 3 [cDPP3] ≥59.1 ng/mL, third quartile) vs. low cDPP3 values (cDPP3 <59.1 ng/mL). HR, hazard ratio.

Takagi K et al. Eur heart J 2020; 22: 279-86



Deniau B et al. Eur heart J 2019; 22:290-9



Deniau B et al. Eur heart J 2019; 22:290-9

CONCLUSION

- Pathophysiology of CS is complex and incompletely known (cardiovascular and inflammatory disease)
- High in-hospital mortality rate 30-40%
- Early myocardial revascularization improve outcomes but does not seem sufficient
- Steroids may have therapeutic place (???)
- Haemodynamic management of CS requires end-organ perfusion supply (organ hypoperfusion) as well as cardiac assist (LV and RV congestion)
- Use of inotropic agents did not demonstrate any outcome improvement but remain useful therapy for restoring haemodynamic
- Further randomized trials still required to define the optimal therapeutic approach

Many thanks…