PLACE 2026
PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

CONGRESSO
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Sistema Socio Sanitario
Regione Lombardia
ASST Spedali Civili

UNIVERSITÀ DEGLI STUDI DI BRESCIA

Shock cardiogeno e shock misto: diagnosi differenziali e implicazioni terapeutiche

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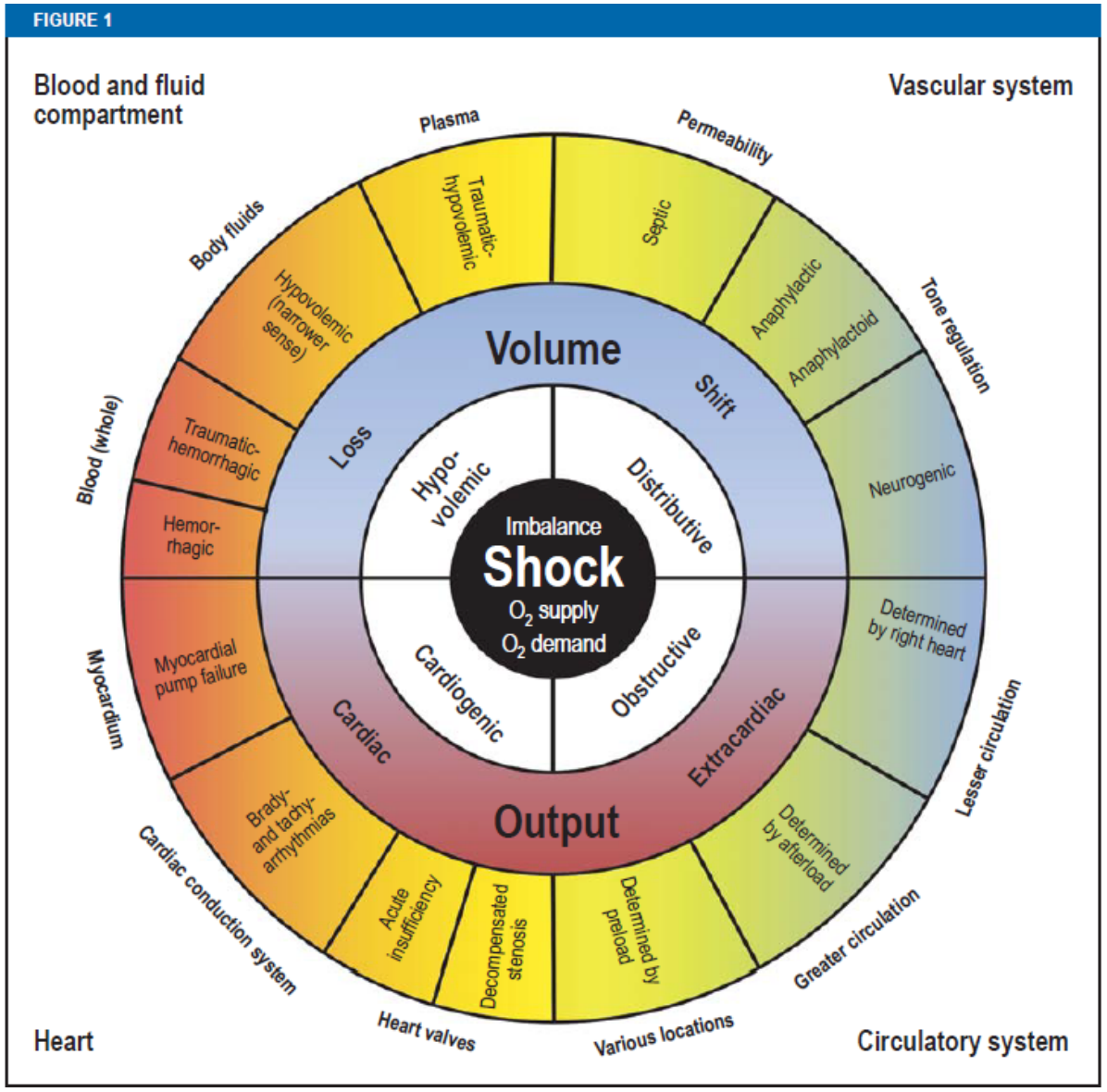
Synoptic view of the four types of shock



- **Hypovolemic shock**
- **Distributive shock**
- **Cardiogenic shock**
- **Obstructive shock**

Standl T, et alThe Nomenclature, Definition and Distinction of Types of Shock. Dtsch Arztebl Int. 2018 Nov 9;115(45):757-768

CARDIOGENIC SHOCK

Impaired contractility

- Myocardial ischemia and compliations
- Myocarditis
- Myocardial contusion
- Septic shock
- Poisoning or toxic exposure
- End stage cardiomyopathy

Dysrhythmia

- Tachycardias
- Bradycardias

Valvular dysfunction

- Severe aortic regurgitation
- Severe aortic or mitral stenosis

Left ventricular outflow obstruction

- Hypertrophic cardiomyopathy

OBSTRUCTIVE SHOCK

Within the circulatory system

- Massive pulmonary embolus
- atrial thrombus or myxoma
- occulsive valvular lesion
- Cardiac tamponade
- abdominal compartment syndrome
- Tension pneumothorax
- Dynamic hyperinflation (e.g. severe asthma)
- Tension pneumomedistinum
- caval compression (e.g. supine hypotension syndrome in the pregnant female)

HYPOVOLAEMIC SHOCK

Hemorrhage

- Traumatic/Non-traumatic

Fluid loss

- GI losses (vomiting, diarrhoea, short gut, etc)
- Excessive diuresis (diabetes insipidus, diuretics)
- Excessive diaphoresis (heat-related illness)
- Diabetic ketoacidosis
- Burns
- “Third spacing” (pancreatitis, severe sepsis, anaphylaxis)
- Iatrogenic (post-dialysis)

DISTRIBUTIVE SHOCK

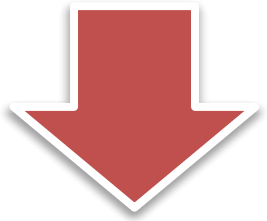
- neurogenic shock
- liver failure
- adrenal insufficiency
- anaphylaxis
- septic shock
- post-bypass vasoplegia
- drugs and toxic exposures, e.g. calcium channel blockers, epidural anaesthesia

Adrenal insufficiency

- Hypothyroidism
- Hyperthyroidism
- Diabetic ketoacidosis
- Severe acidosis/ alkalosis and electrolyte

Step by step approach to CS Definition & Intervention

1. CS Clinical Definition/Identification
2. CS Etiologycal Definition
3. CS Definition/Phenotypization
4. CS Staging Definition (Stratification)



Intervention

Step by step approach to CS Definition & Intervention

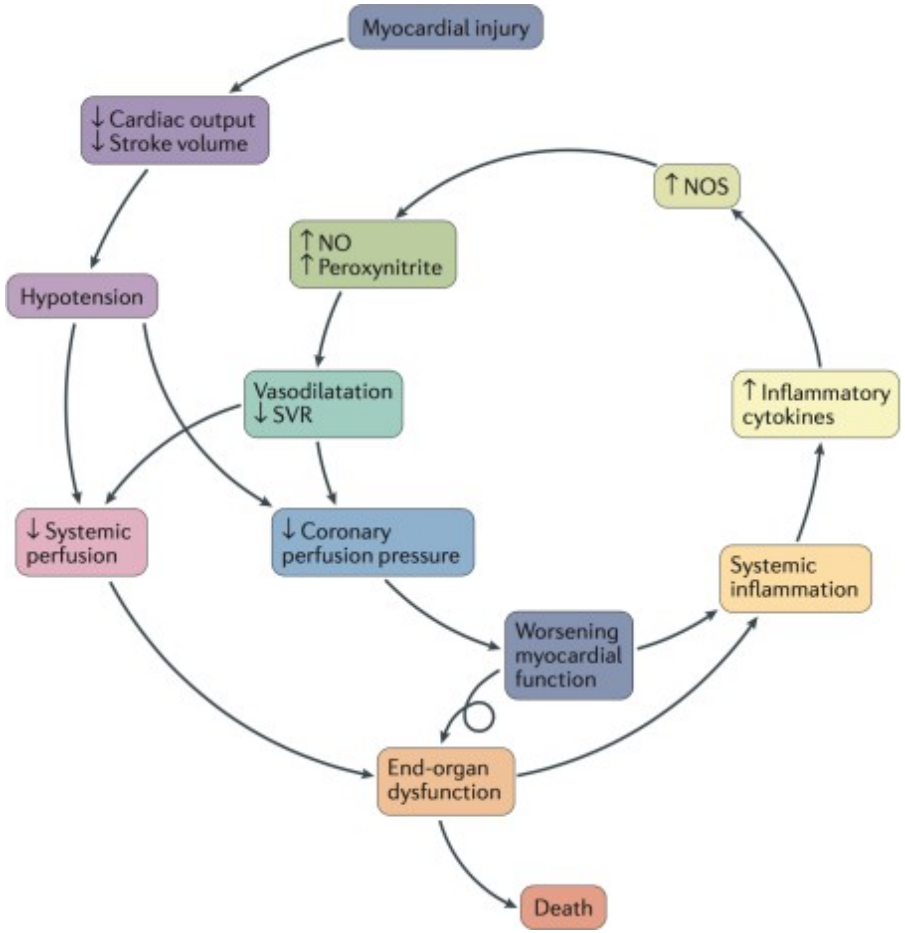
1. CS Clinical Definition/Identification
2. CS Etiologycal Definition
3. CS Haemodynamic Definition/Phenotypization
4. CS Staging Definition (Stratification)

Intervention

CS Definition & Clinical Identification

A syndrome caused by a primary cardiovascular disorder in which **inadequate CO** results in a life-threatening state of **tissue hypoperfusion** associated with impairment of tissue oxygen metabolism and hyperlactatemia which, depending on its severity, may result in multi-organ dysfunction and death.

30-days death 45-75%



ESC HFA Position Statement 2020 Eur J Heart Fail. 2020 Aug;22(8):1315-1341

CS Definition & Clinical Identification (II)

ESC Heart Failure Guidelines 2021	• Diagnosis of cardiogenic shock mandates the presence of clinical signs of hypoperfusion (cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure).	• Biochemical manifestations of hypoperfusion (elevated serum creatinine and lactate, metabolic acidosis). • Hypoperfusion is not always accompanied by hypotension
ACVC position statement 2020	All of the following: • Systolic blood pressure <90mmHg for >30 min **OR** need of vasopressors to maintain systolic pressure >90mmHg	Clinical signs/ symptoms (At least one of the following): • Altered mental statuS **OR** Cold, clammy skin and extremities **OR** Oliguria with urine output <30 ml/h **OR** **Arterial lactate >2.0 mmol/l** And elevated left ventricular filling pressures: • Pulmonary congestion **OR** PCWP/LVEP >20 mm Hg
Shock Academic Research Consortium *(Clinical trial def)*	• Systolic blood pressure <90 mm Hg for ≥30 min (or the need for vasopressors, inotropes or mechanical circulatory support to maintain systolic blood pressure ≥90 mm Hg) with evidence of hypoperfusion.	Hemodynamic criteria (optional) • Cardiac index <2.2L/min/m2 **AND** • Hypoperfusion (1 of the following): **arterial lactate >2mmol/L**, Acute kidney injury (creatinine ≥2× upper limit of normal) **OR** oliguria (e.g. urine output <0.5mL/(kg·h), Acute hepatic injury (eg, ALT >3× upper limit of normal), Cool or mottled extremities, Altered mental status not explained by an alternative cause

[1]*Eur Heart J. 2021 Sep 21;42(36):3599-3726*

[2]*Eur Heart J Acute Cardiovasc Care. 2020 Mar;9(2):183-197*
[3]*Circulation. 2023 Oct 3;148(14):1113-1126*

CS Definition & Clinical Identification (II)

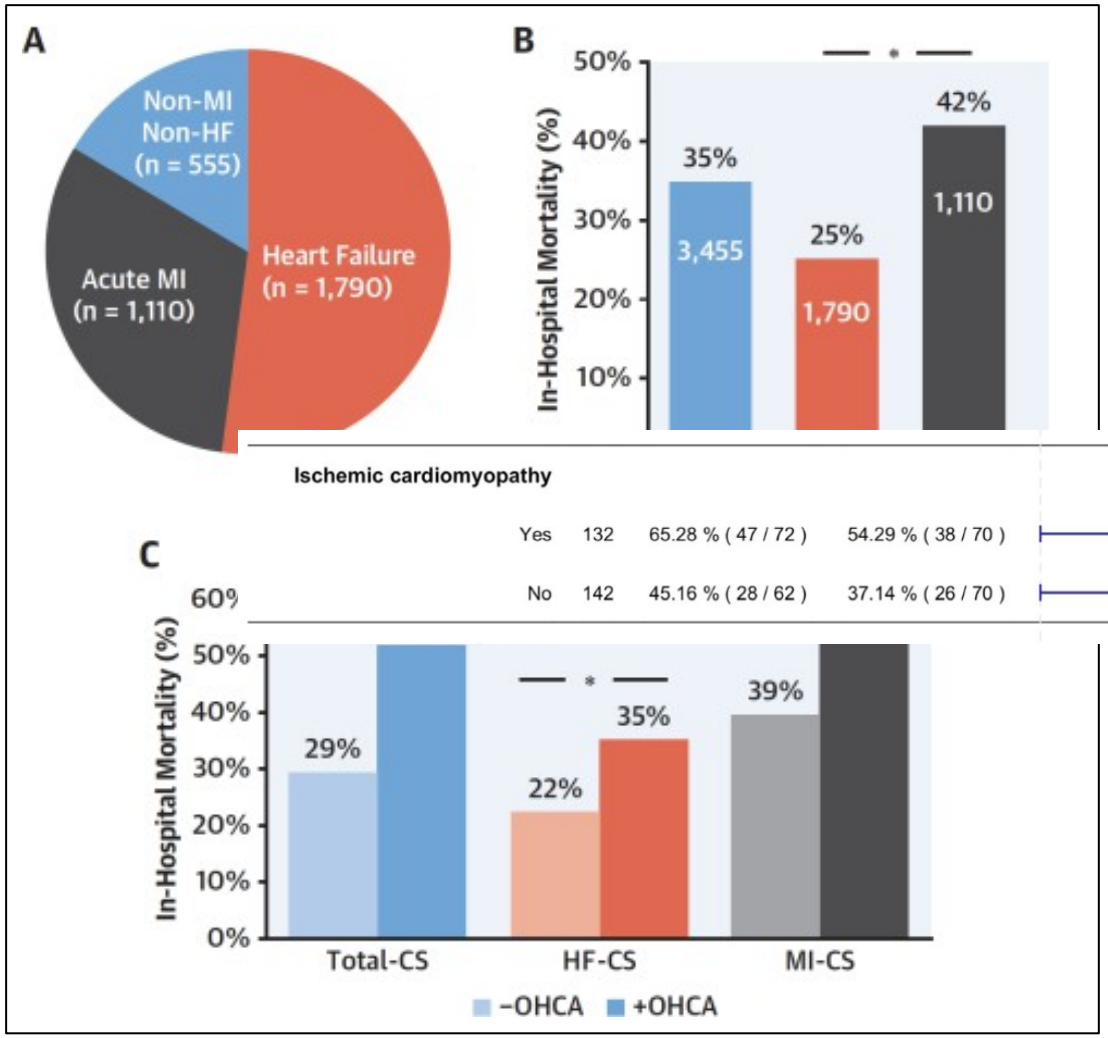
IABP-SHOCK II[1]	• Acute myocardial infarction • Systolic blood pressure <90 mmHg for more than 30 minutes or needed infusion of catecholamines to maintain a systolic pressure above 90 mmHg, • Clinical signs of pulmonary congestion, and had impaired end-organ perfusion.	• The diagnosis of impaired end-organ perfusion required at least one of the following: Altered mental status, Cold, clammy skin and extremities • Oliguria with urine output <30 ml per hour; or **Serum lactate level >2.0 mmol/L.**
ECLS Shock[2]	• Acute myocardial infarction • Systolic blood pressure <90 mmHg for >30 min or catecholamines required to maintain systolic blood pressure >90 mmHg	• Signs of impaired organ perfusion with at least one of the following criteria: Altered mental status, Cold, clammy skin and extremities • Oliguria with urine output <30 ml/h • **Arterial lactate >3 mmol/l**
DanGer Shock[3]	• Acute myocardial infarction • systolic blood pressure less than 100mmHg and/or need for vasopressor therapy (dopamine/ norepinephrine or epinephrine)	• peripheral signs of tissue hypoperfusion (**arterial blood lactate ≥2.5mmol/l** and/or **SvO2 <55% with a normal PaO2**)

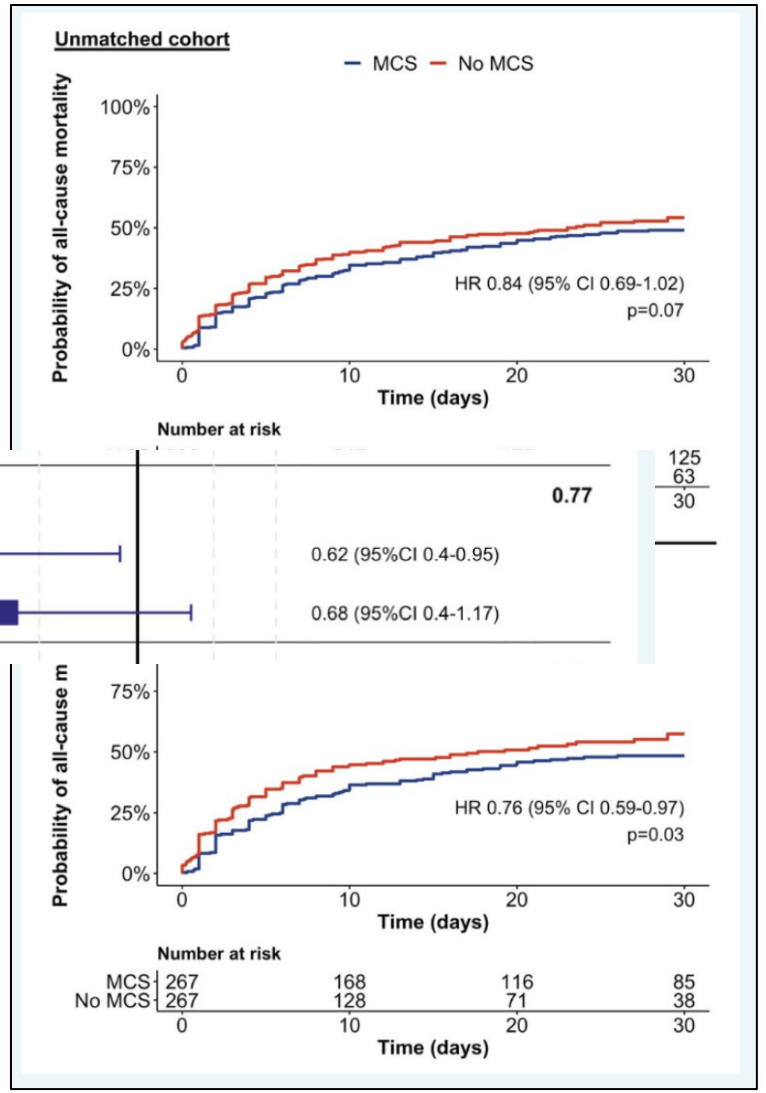
[1] *N Engl J Med. 2012 Oct 4;367(14):1287-96*

[2] *N Engl J Med. 2023 Oct 5;389(14):1286-1297*

[3] *N Engl J Med. 2024 Apr 18;390(15):1382-1393*

Cardiogenic Shock Etiological Definition



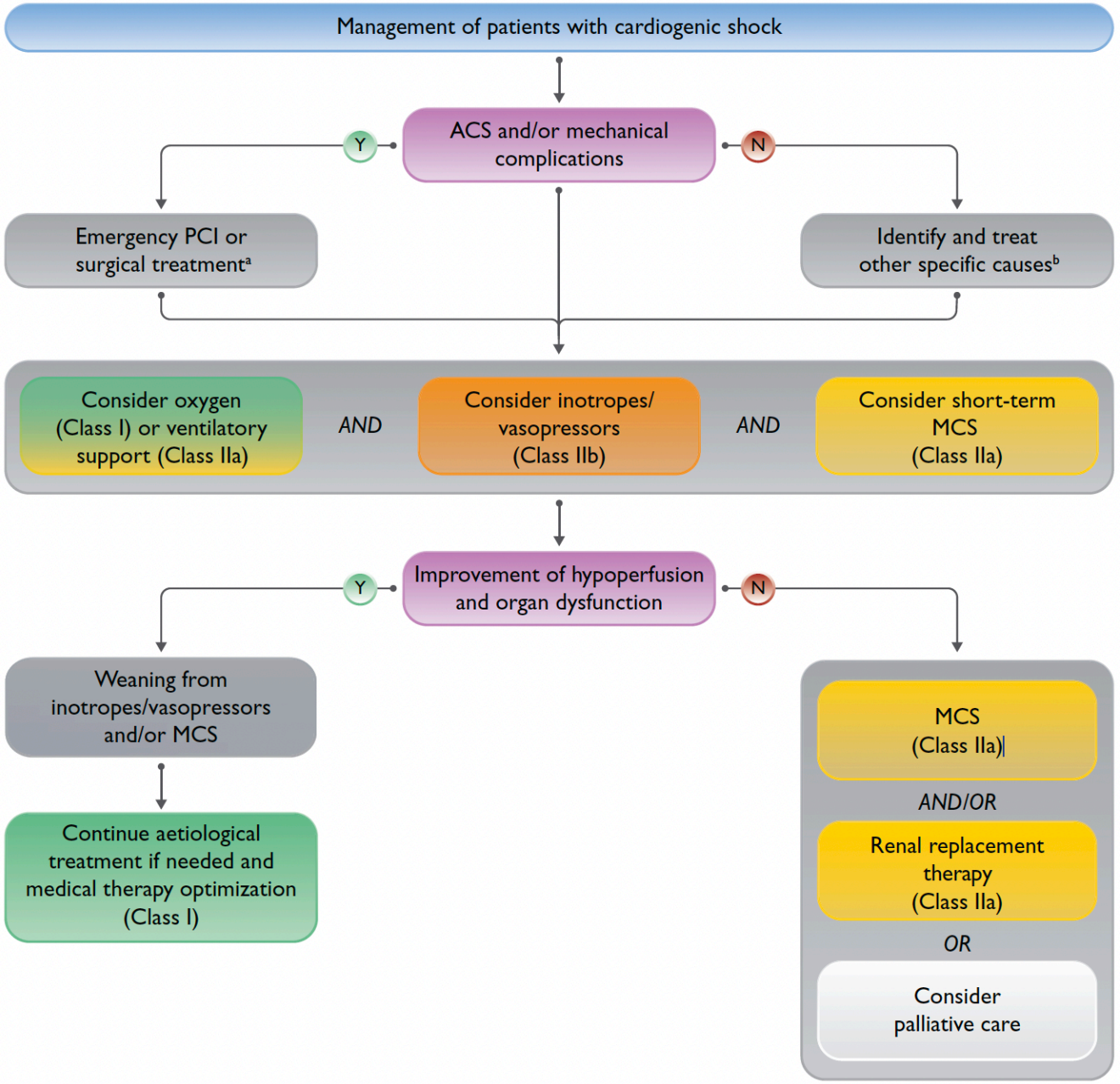


JACC. 2022 Jul, 80 (3) 185–198
Eur J Heart Fail. 2023 Apr;25(4):562-572

CS – more questions than answers

- Few solid evidence-based data on inotropes/vasopressors in CS
- Interplay between medical therapy and MCS
- Different CS phenotypes – one size does not fit all
- Challenges in performing proper RCTs in this setting
- Adverse effects of common adrenergic drugs

2021 ESC Guidelines



McDonagh T, Metra M et al. Eur Heart J 2021;42:3599-3726

2021 ESC Guidelines

Table 22 Inotropes and/or vasopressors used to treat acute heart failure

Drug	Infusion rate
Dobutamine	2–20 µg/kg/min (beta+)
Dopamine	3–5 µg/kg/min; inotropic (beta+) >5 µg/kg/min: inotropic (beta+), vasopressor (alpha+)
Milrinone	0.375–0.75 µg/kg/min
Enoximone	5–20 µg/kg/min
Levosimendan	0.1 µg/kg/min, which can be decreased to 0.05 or increased to 0.2 µg/kg/min
Norepinephrine	0.2–1.0 µg/kg/min
Epinephrine	0.05–0.5 µg/kg/min

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

McDonagh T, Metra M et al. Eur Heart J 2021;42:3599-3726

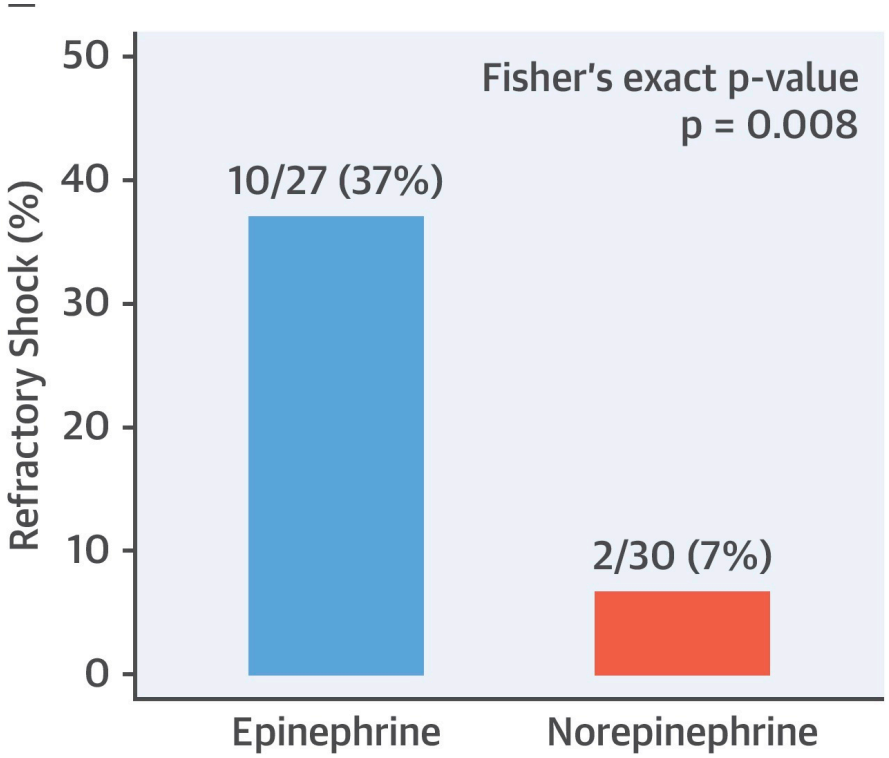
Key recommendations by different guidelines

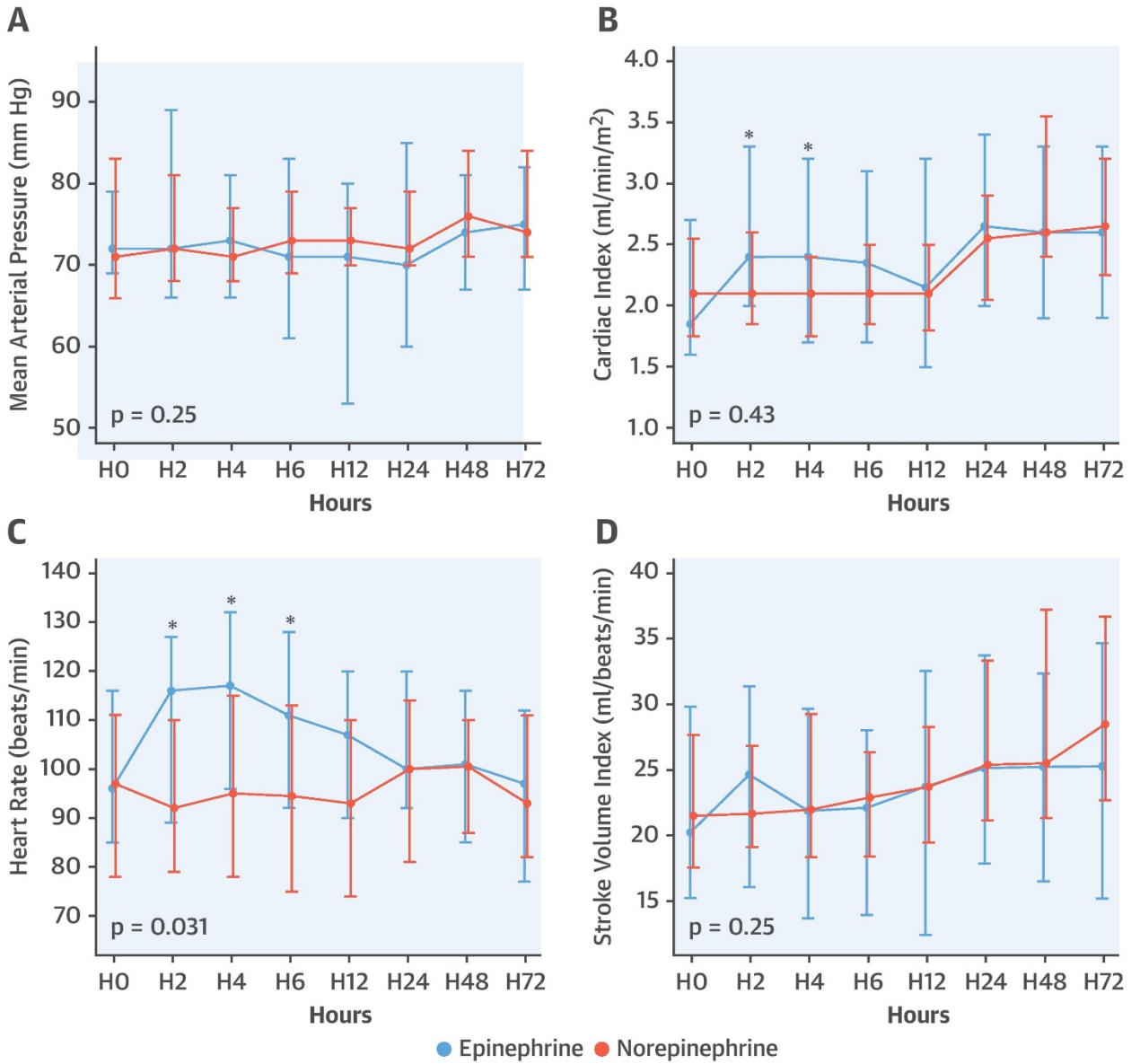
Indication to commence therapy	Hypotension (systolic blood pressure <90 mm Hg) and/or evidence of end-organ hypoperfusion				
Societal guideline	**ESC HF**	**ACC/AHA HF**	**ESC STEMI**	**AHA CS**	**AHA AMI-CS**
Recommendation and level of evidence	**IIb - B/C**	**IIa - B**	**IIb - C**	**N/A**	**N/A**
First-line agent	**Hypotensive: Norepinephrine** **Low output: No recommendation**	**Hypotensive: Clinician preference** **Low output: Clinician preference**	**Hypotensive: Norepinephrine** **Low output: Dobutamine**	**Hypotensive: Norepinephrine or Dopamine** **Low output: Norepinephrine or Dopamine**	**Hypotension: Norepinephrine**

Bloom JE et al. JAHA 2023; 12:e029787

OptimaCC trial

- N = 57 AMICS patients



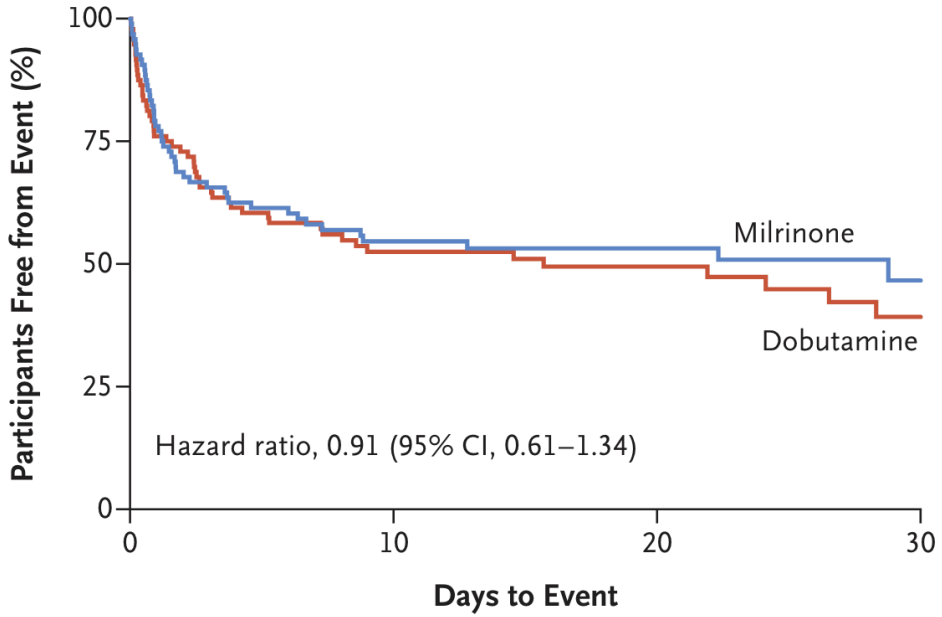


Levy B et al. J Am Coll Cardiol 2018;72:173-182

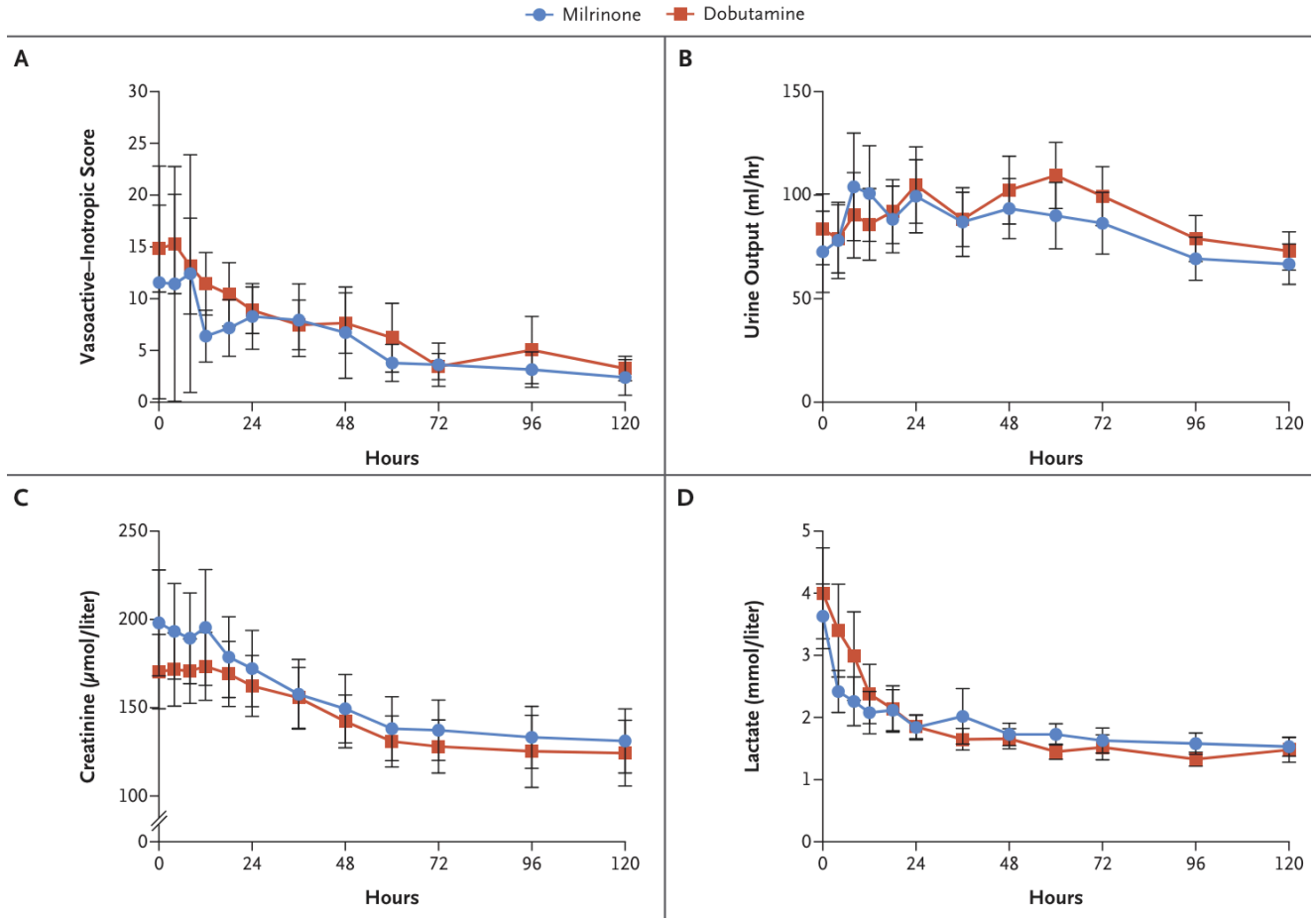
DOREMI trial

- N = 192 CS patients (67% ischemic cause)

A **Primary Composite Outcome**



No. at Risk				
Milrinone	96	42	26	7
Dobutamine	96	43	25	13

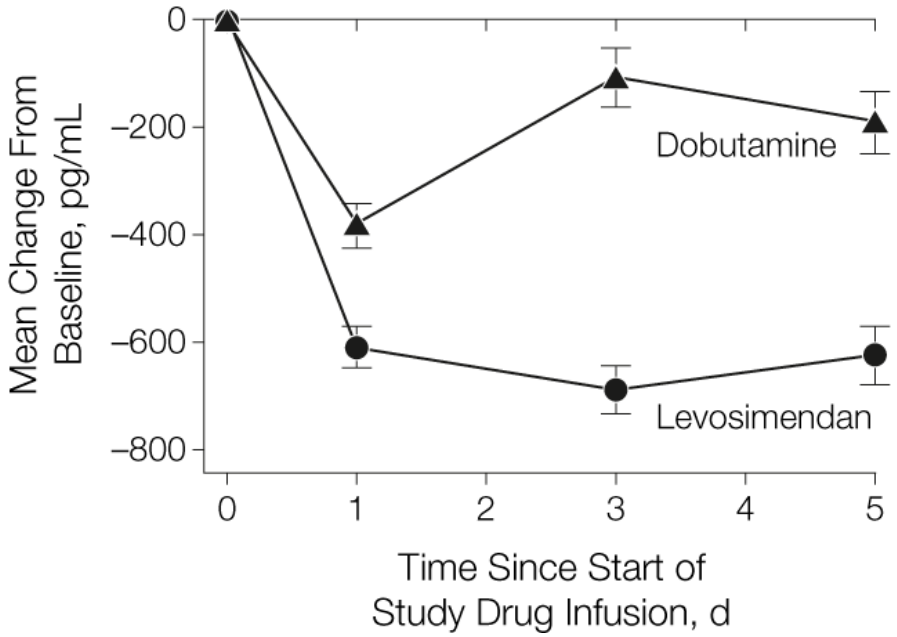


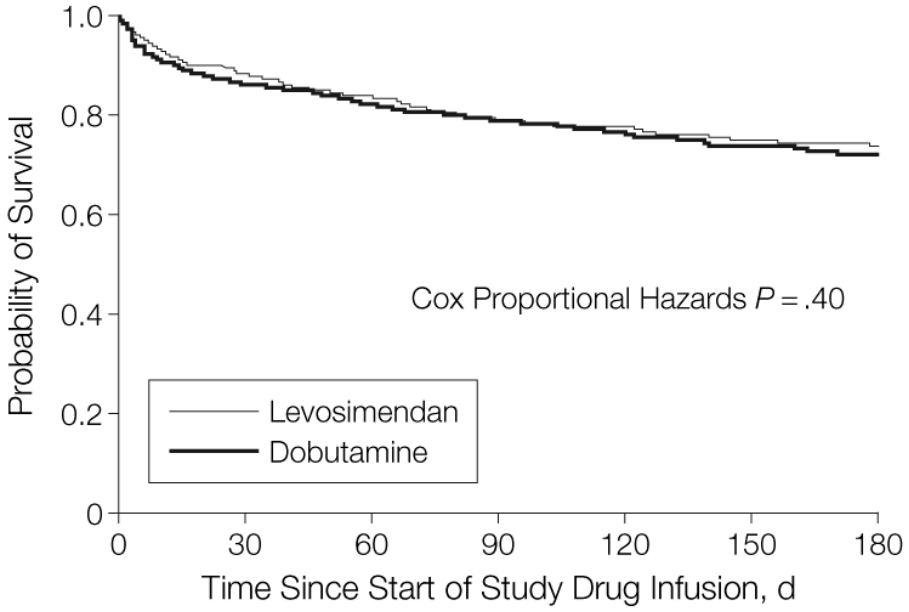
Outcome	Milrinone (N=96)	Dobutamine (N=96)	Relative Risk or Hazard Ratio (95% CI)†	P Value‡
Primary outcome: composite of in-hospital death from any cause, resuscitated cardiac arrest, receipt of cardiac transplant or mechanical circulatory support, nonfatal myocardial infarction, transient ischemic attack or stroke diagnosed by a neurologist, or initiation of renal replacement therapy — no. (%)	47 (49)	52 (54)	0.90 (0.69–1.19)	0.47
Secondary outcomes				
In-hospital death from any cause — no. (%)	35 (37)	41 (43)	0.85 (0.60–1.21)	

Mathew R et al. NEJM 2021;385:516-525

SURVIVE trial

- N = 1372 patients with ADHF "*who required inotropic support*"
- Need of inotropic support based on insufficient response to IV diuretics and/or vasodilators and at least 1 of the following:
  (1) dyspnea at rest or mechanical ventilation for ADHF
  (2) oliguria not as a result of hypovolemia
  (3) PCWP ≥ 18 mmHg and/or CI ≤ 2.2 L/min/m^2

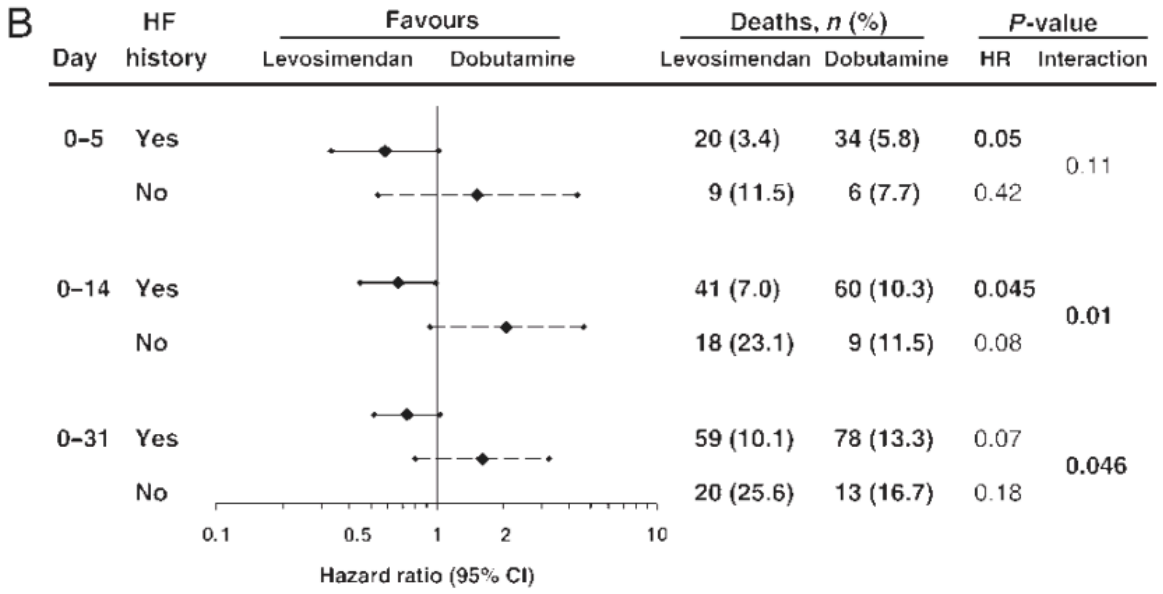


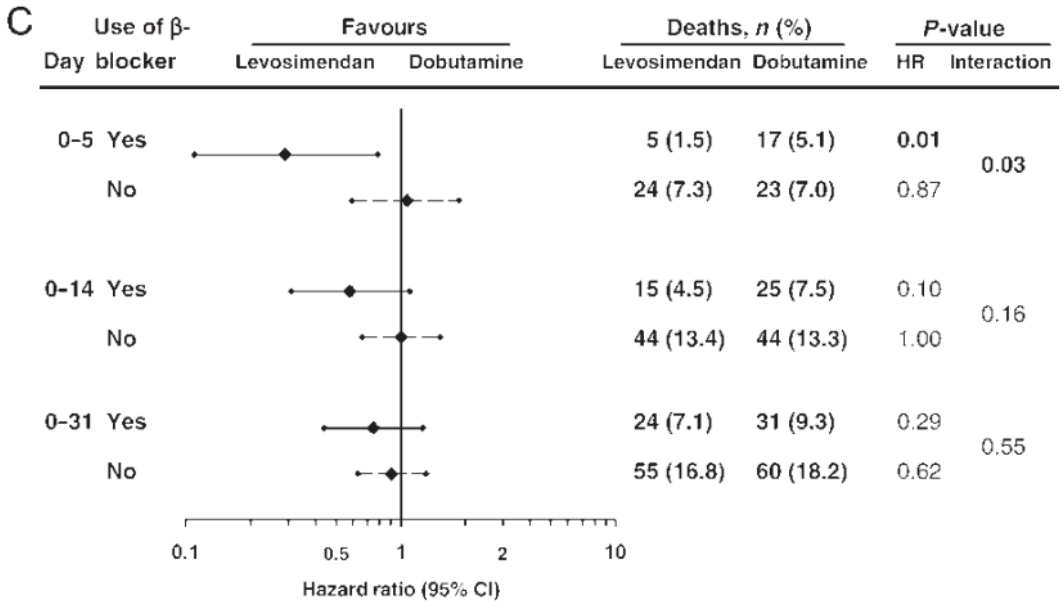


Mebazaa A et al. JAMA 2007;297:1883-91

SURVIVE trial

- N = 1372 patients with ADHF "*who required inotropic support*"
- Substudy according to history of previous HF and previous use of beta-blockers



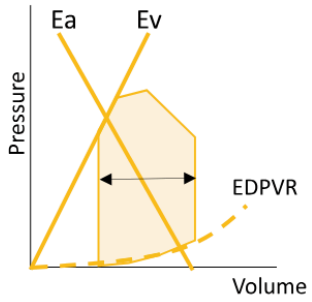


Mebazaa A et al. EJHF 2009;11:304-311

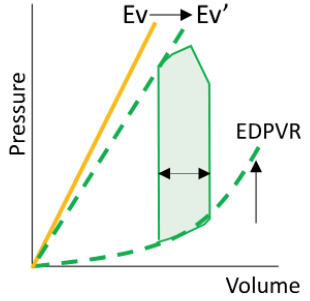
Pharmacodynamic effects

Pathophysiology

Normal

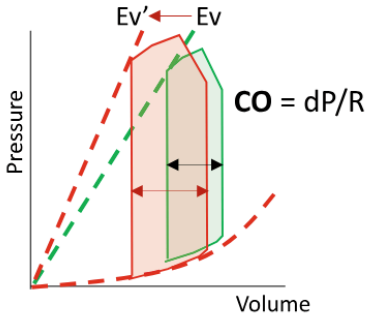


Cardiogenic shock

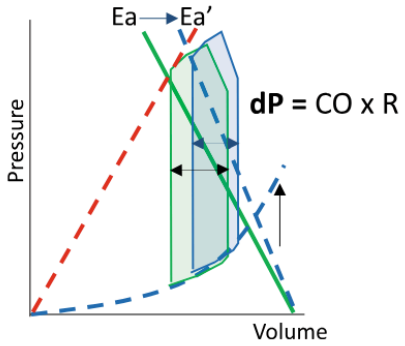


Inotropes

Increase in CO



Increase in MAP



Adrenergic

Isoproterenol

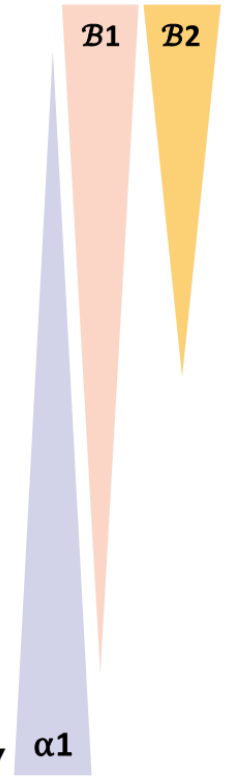
Dobutamine

Epinephrine

Dopamine

Norepinephrine

Phenylephrine



Non adrenergic

Inodilators
PDE3 inhibitors
Levosimendan

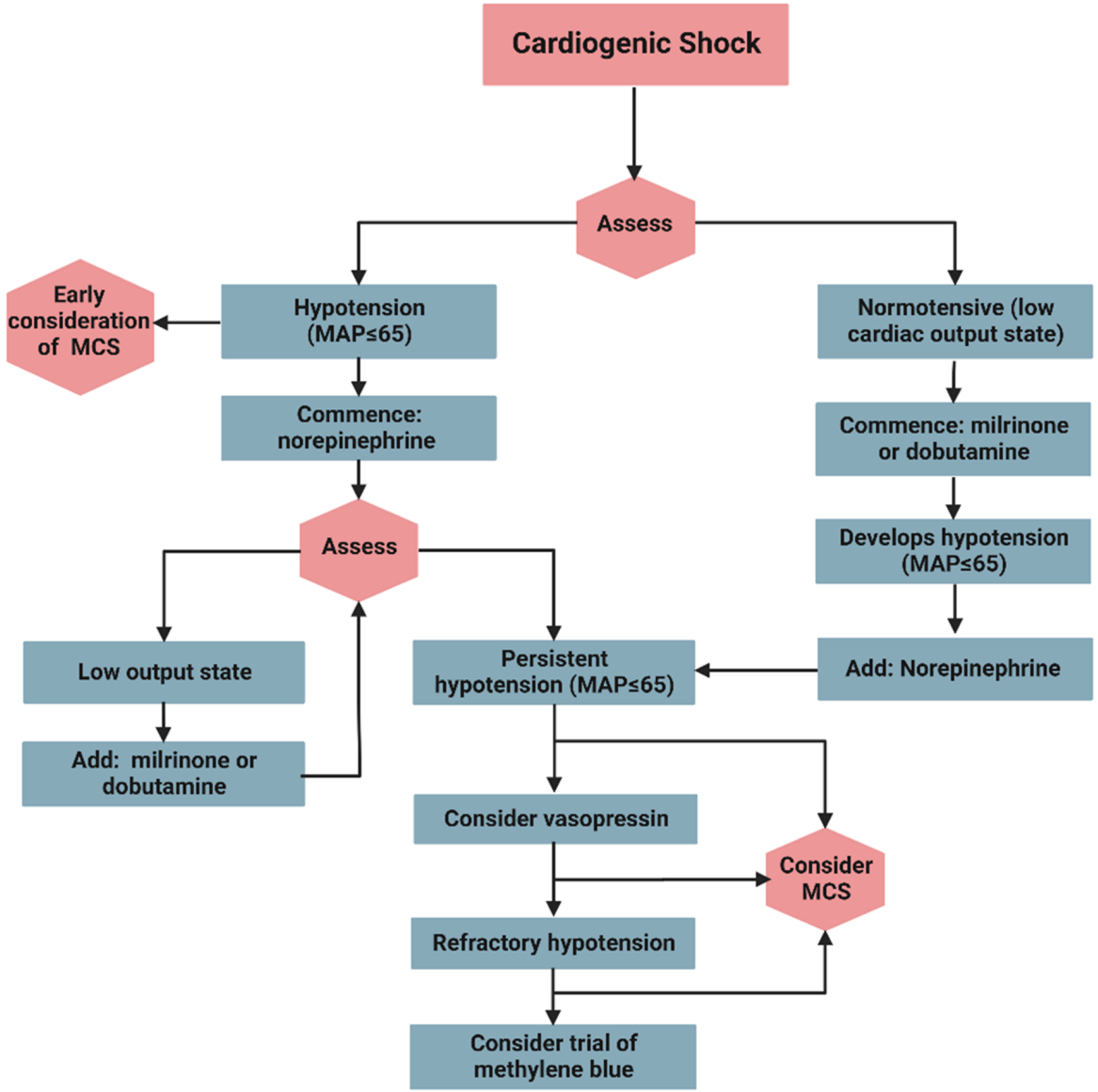
Others
Istaroxime

Vasopressin
Methylene blue
Angiotensin II

Vasopressors

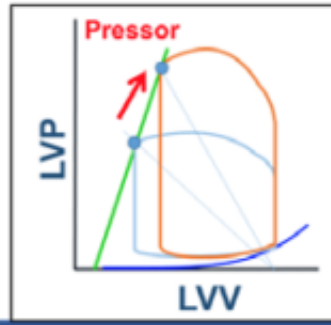
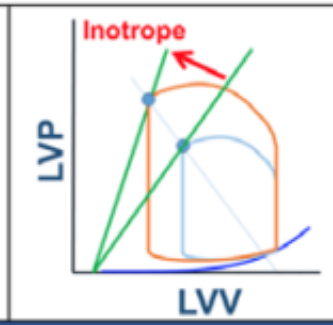
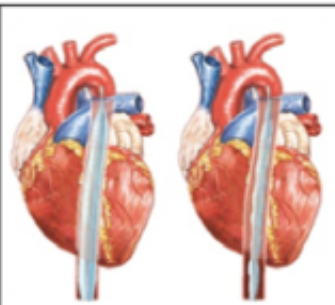
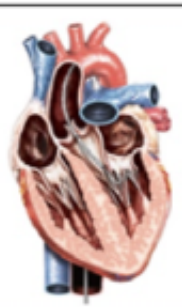
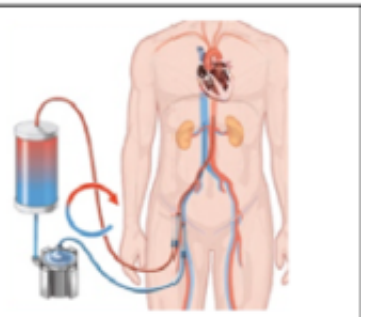
Riccardi M, Pagnesi M et al. EJHF 2024;26:411-431

Blood pressure-based approach?



Bloom JE et al. JAHA 2023; 12:e029787

Cardiogenic Shock Hemodynamic Phenotypization (III)

					
	Vasopressor	**Inotrope**	**IABP**	**IMPELLA**	**VA-ECMO**
Mechanism	Peripheral vasoconstriction	Increased myocyte calcium cycling	Aortic counter-pulsation	Left ventricle (LV) to aorta transvalvular circulatory support (LVADs)	Right atrium (RA) to peripheral artery circulatory support with gas exchange unit
LV Contractility	↔ or ↑	↑	↔	↔	↔
TPR	↑	↔ or ↑ or ↓	↔	↔	↔
LV Flow	↓	↑	↑	↓	↓
Total CO	↓	↑	↑	↑↑	↑↑↑
CVP	↔ or ↑	↔	↔ or ↓	↔ or ↓	↓
PCWP	↔ or ↑	↔ or ↓	↔ or ↓	↓	↔ or ↑
MAP	↑	↑	↑	↑↑	↑↑
Total CPO	↔ or ↑	↑	↑	↑↑	↑↑
PVA	↑	↑	↔ or ↓	↓↓	↑↑
MVO2	↑	↑	↓	↓↓	↑↑
Sheath size	NA	NA	7-8 French arterial	14 French arterial	15 – 17 French arterial 21-23 French venous

No effect on SpO2 | No effect on SpO2 | ↑↑ SpO2

Phenotype-based approach?

CARDIOGENIC SHOCK

Inadequate cardiac output
PLUS
Clinical signs of hypoperfusion
AND/OR
Biochemical manifestations of hypoperfusion

Few evidence-based data

AMI-CS

First-line therapy:
- Norepinephrine (if hypotension prevails)
- Dobutamine OR Milrinone OR Levosimendan (if CS «normotensive»)

Second-line therapy:
- Norepinephrine + Dobutamine/Levosimendan

ADHF-CS

First-line therapy:
- Norepinephrine (if hypotension prevails)
- Dobutamine OR Milrinone OR Epinephrine + Sodium Nitroprusside OR Levosimendan (if CS «normotensive»)

Second-line therapy:
- Norepinephrine + Dobutamine/Levosimendan

RV-predominant CS

First-line therapy:
- Norepinephrine (if hypotension prevails)
- Milrinone OR Levosimendan (if CS «normotensive»)

Second-line therapy:
- Norepinephrine + Milrinone/Levosimendan/Dobutamine

Riccardi M, Pagnesi M et al. EJHF 2024;26:411-431

Multi-parametric approach?

Cardiogenic shock, general rules derived from AMI

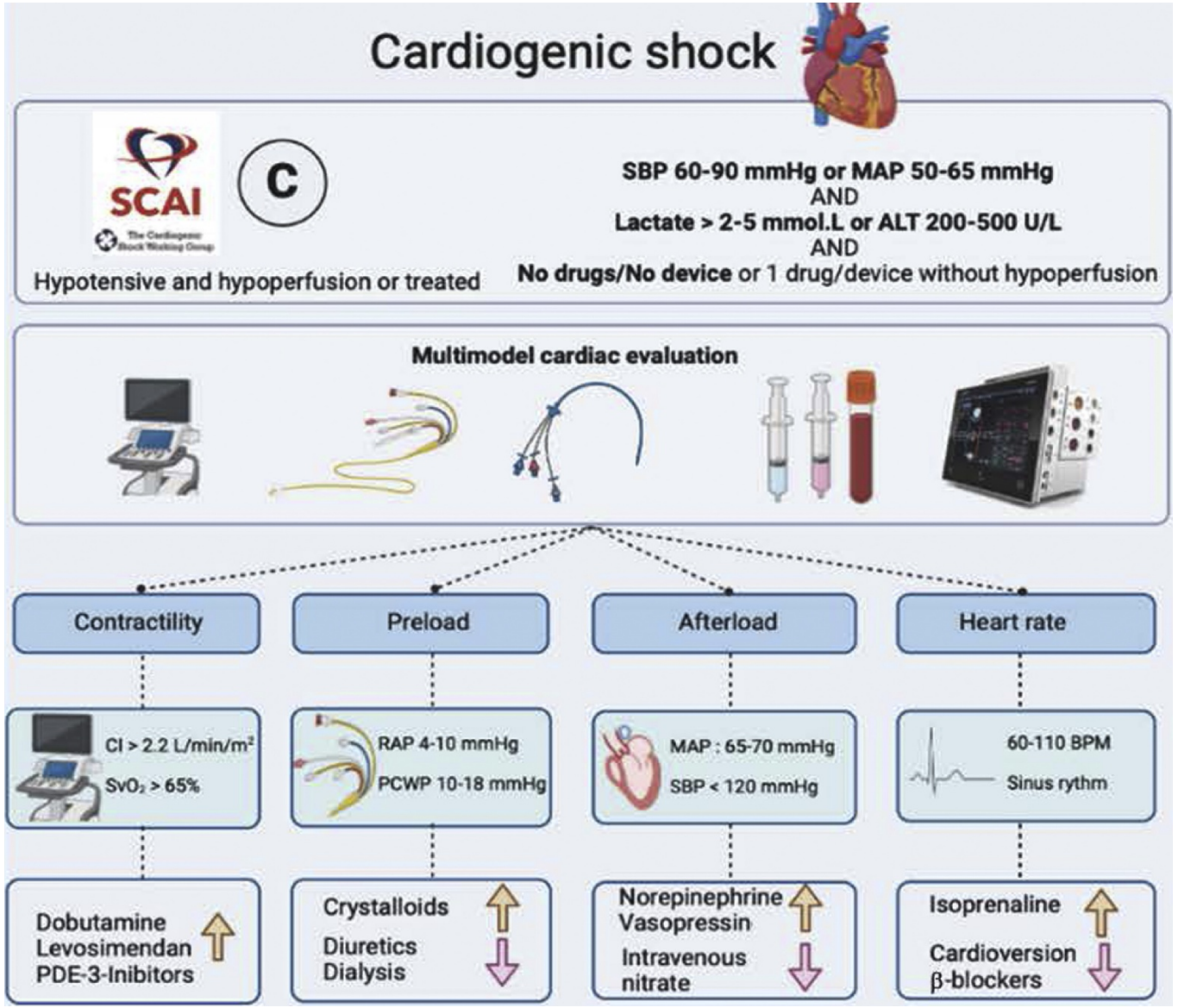
- Early revascularisation of the culprit artery
- Inotropic drugs (dobutamine) to target effective CO
- Consider vasopressors to target effective MAP
- Consider LV unloading (IABP) for mechanical complications of AMI in bridge to surgery

Tako-Tsubo syndrome

- Cessation of sympathomimetic drugs
- Consider non-adrenergic inotropic drug
- Identify LVOT obstruction
- β-blockers

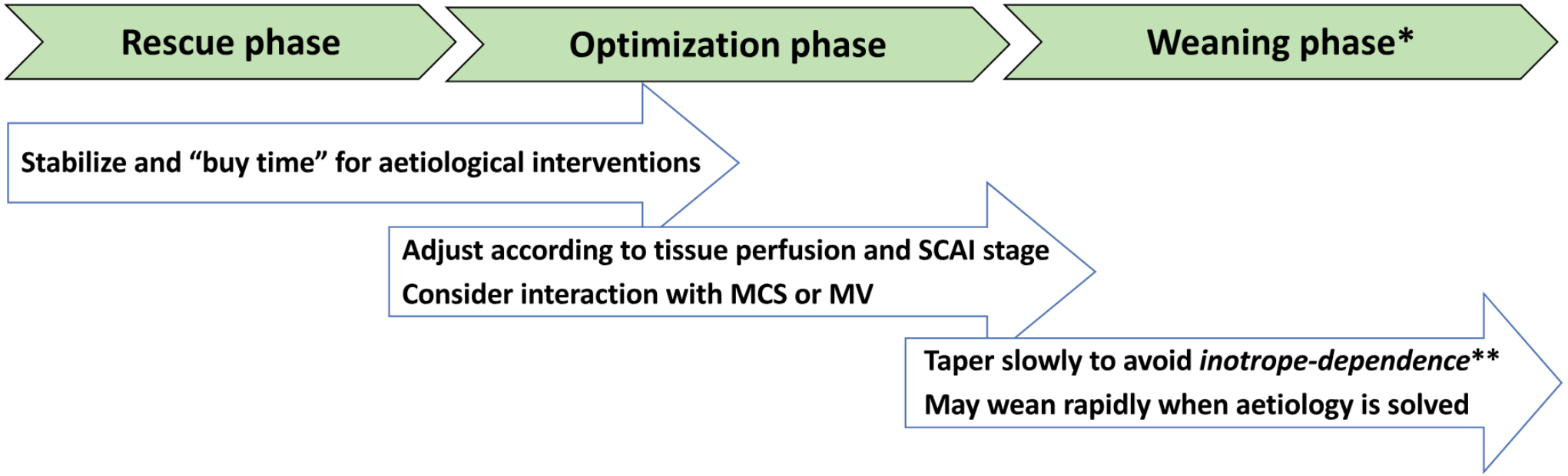
Hypertrophic cardiomyopathy with LV obstruction

- Stop inotropic drugs
- Identify LVOT obstruction
- β-blockers or verapamil
- Fluid challenge



Lescroart M et al. J Intensive Med 2023;3:114-123

Multi-step tailored strategy

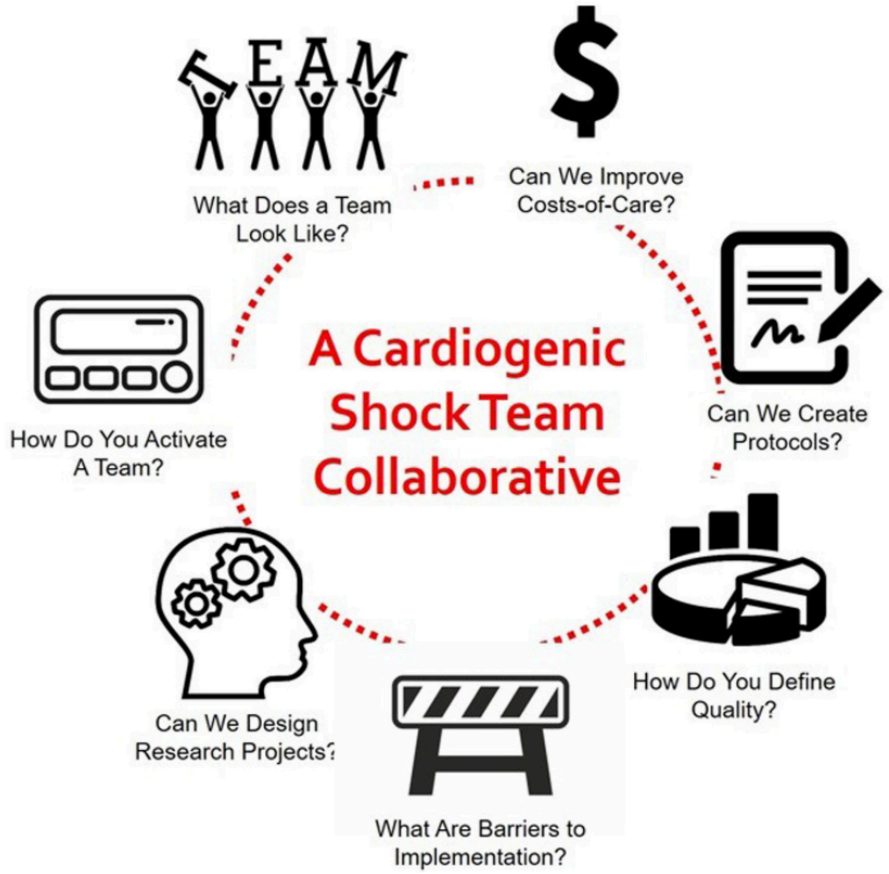


*Dopa, Dob: 1 µg/kg/min per hour; NE: 0.1 µg/kg/min per hour
**Worsening end-organ function, deteriorating hemodynamics, exacerbation of HF symptoms, or significant hypotension on attempted inotrope wean

Riccardi M, Pagnesi M et al. EJHF 2024;26:411-431

Strategy-based rather than drug-based approach?

- Develop and test a pharmacological care strategy (rather than compare 1:1 different single drugs)
- Develop and test a team-based comprehensive approach (shared decisions, drugs/MCS escalation/de-escalation)
- Develop and test *different* strategies based on patients' phenotype, hemodynamic/metabolic data, stage and trajectory
- Better define endpoints and therapeutic goals/needs



Senman et al. JAHA 2024; 13:e031979

Potential new targets/treatments

Treatment	Pathophysiology and benefits	Study population	Main results
Adrecizumab ACCOST HH[70]	Endovascular chelation of adrenomedullin to enhance its vascular and myocardial protective effects	CS defined as low SBP + clinical pulmonary congestion + organ dysfunction (lactate, oliguria, altered mental status, clammy skin and limbs) Exclusion criteria: cardiac arrest with CPR >60 min, sustained bradycardia or tachycardia.	On day-30, number of days free from any CV support (vasopressor, inotropes, mechanical circulatory support) Failed to improve CV support free survival days.
OM ATOMIC HF[74,75]	Cardiac myosin activator. Increases systolic ejection time and inotropism without increasing MVO_2	Elevated BNP/NTBNP and reduced LVEF and dyspnea refractory to IV loop diuretics Exclusion criteria: eGFR <20 mL/min/m^2, stenotic valvular disease, recent AMI (<30 day), uncontrolled blood pressure	Primary outcome: dyspnea relief after OM infusion *vs.* placebo. Only the highest dose (targeting blood concentration ≈ 310 ng/mL) improved dyspnea relief at 48 h
iDPP3	Increased DPP3 found in CS DPP3 injection in murine models induces myocardial depression Murine model of isuprel induced heart failure, injection of iDPP3 resolve cardiac function	Pre-clinical studies only	–
Istaroxime SEISMIC trial[79]	Positive stimulation of SERCA Ca^{2+} re-uptake and Na/K ATPase plasmatic membrane pump Increases inotropism and lusitropism without increased heart rate.	SCAI stage B pre-CS: acute HF + LVEF <40% + SBP <75–90 mmHg Exclusion criteria: recent AMI < 3 months; IV drugs; venous lactate >2 mmol/L; eGFR <30 mL/min/m^2; Tp° >38 °C; recent stroke.	Primary outcome: AUC of SBP along the first 6 h of infusion was significantly higher in the Istaroxime group TTE ΔCO improvement: +0.2 L/min/m^2 Adverse events: nausea, vomiting

Lescroart M et al. J Intensive Med 2023;3:114-123

Available phase 2 trials on istaroxime

Etiology-based management algorithms?

Pagnesi et al. Heart Fail Rev 2025;30:1559-1589

Ongoing RCTs in CS

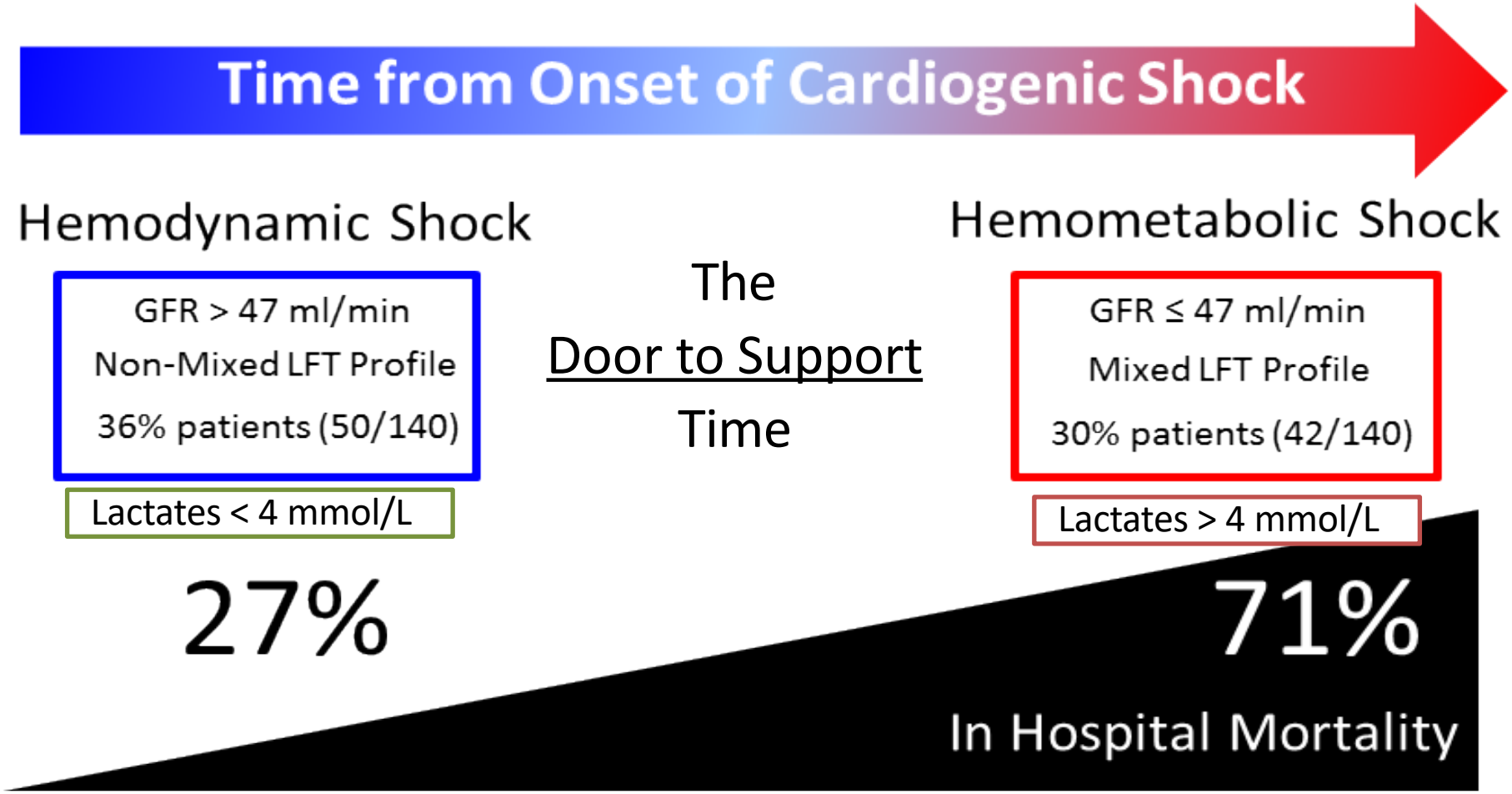
Mechanical circulatory support						
ECMO-RRT (NCT02870946)	AMI and HF	VA-ECMO plus RRT	VA-ECMO only	362	Mortality at 30 days	
HEMO-ECMO (NCT03729765)	AMI and HF	VA-ECMO plus hemoperfusion	VA-ECMO	60	Change in IL-6 level at 3 days	
ANCHOR (NCT04184635)	AMI	VA-ECMO plus IABP	Best medical therapy	400	Mortality or VA-ECMO at 30 days	
UNLOAD-ECMO (NCT05577195)	AMI and HF	VA-ECMO plus Impella CP	VA-ECMO	198	Mortality at 30 days	Lactate level >5 mmol/liter for inclusion
ULYSS (NCT05366452)	AMI	Impella CP	Best medical therapy	204	Either composite of all-cause death, ECMO, LVAD, or heart transplantation at 30 days	
RECOVER IV (NCT05506449)	AMI	Impella CP	Best medical therapy including IABP	558	Mortality at 30 days	Stopped early for safety
ICONE (NCT05699005)	AMI and HF	Individualized transfusion during VA-ECMO	Conventional transfusion during VA-ECMO	138	Total number of PRBCs transfused during support, adjusted for VA-ECMO duration	
REMAP ECMO (NCT05913622)	AMI and HF	VA-ECMO plus IABP	VA-ECMO	430	Successful weaning from VA-ECMO at 30 days	Bayesian analysis, adaptive design
Transcatheter interventions						
MINOS (NCT05298124)	AMI and HF	M-TEER	Best medical therapy	144	Composite of in-hospital death from any cause, cardiac transplantation, implantation of durable LVAD, or discharge with palliative inotropic therapy	Mitral regurgitation grade 3+ or 4+
RESCUE-SHOCK (NCT05527717)	AMI	Immediate multivessel PCI with VA-ECMO	Culprit-lesion only PCI with VA-ECMO	560	Composite of death from any cause, LVAD, or heart transplantation at 90 days	

Inotropes, vasopressors, and hemodynamic agents						
LevoHeartShock (NCT04020263)	AMI and HF	Levosimendan	Placebo	610	Composite of death from any cause, VA-ECMO, or dialysis at 30 days	
CAPITAL DOREMI-2 (NCT05267886)	AMI and HF	Dobutamine or milrinone	Placebo	346	Composite of in-hospital death from any cause and the occurrence of any of the following ≤12 hr after start of intervention: • Sustained hypotension (MAP ≤55 mm Hg) or sustained use of high-dose vasopressors • Lactate level >3.5 mmol/liter at 6 hr or thereafter • Need for MCS • Atrial or ventricular arrhythmia leading to emergent electrical cardioversion • Cardiac arrest	SCAI shock C or D
NorShock (NCT05168462)	AMI	MAP ≥55 mm Hg	MAP ≥65 mm Hg	776	Composite of death from any cause and severe renal failure leading to renal replacement therapy	
PACCS (NCT05485376)	HF	PAC	No PAC	400	In-hospital mortality	Lactate level >2 mmol/liter
Systemic approaches and antiplatelet agents						
DAPT-SHOCK (NCT03551964)	AMI	Cangrelor plus DAPT with ticagrelor	DAPT with ticagrelor	605	Laboratory end point, platelet reactivity index; clinical end point, MACE	Primary laboratory end point met; primary clinical end point not met
DOBERMANN (NCT05350592)	AMI preshock	Tocilizumab or dobutamine (or both)	Placebo		Pro–B-type natriuretic peptide plasma concentration <48 hr	2×2 factorial design; patients with preshock, no overt cardiogenic shock; double-blind
COCCOA (NCT03773822) — Completed	AMI and HF	Combination of hydrocortisone and fludrocortisone	Placebo	380	Free of corticosteroid therapy at day 7	

Thiele and Hassager. NEJM 2026; 394 :62-77

Hemo-Metabolic Shock has a Poor Prognosis

Treat Shock Before Metabolic Failure Begins



Morine & Kapur et al. Shock Working Group

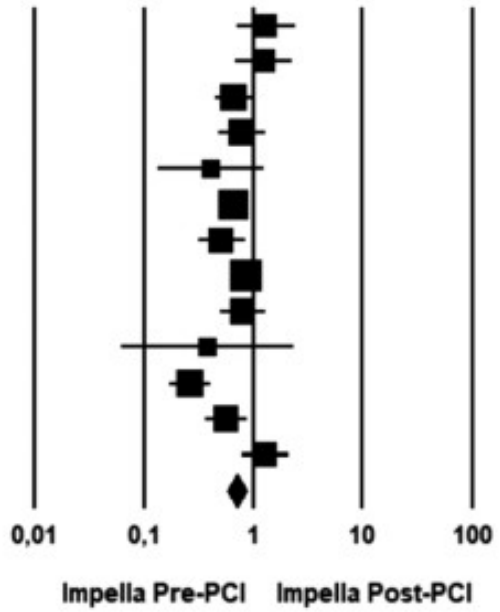
Timing of Intervention

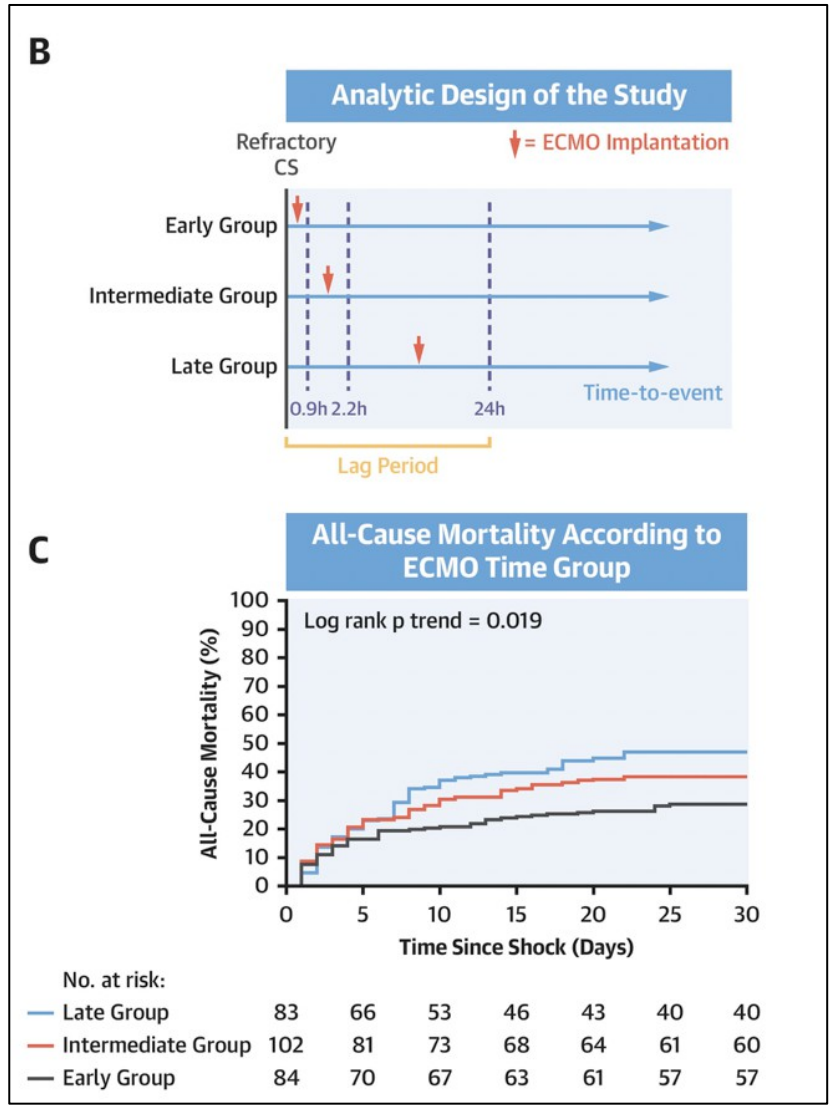
Short term mortality

Study name	Risk ratio	Lower limit	Upper limit	Z-Value	p-Value
Basir, M. B., et al. (2019)	1,302	0,709	2,393	0,851	0,395
Boshara, A., et al. (2021)	1,238	0,679	2,256	0,698	0,485
Chatzis, G., et al. (2021)	0,657	0,437	0,989	-2,014	0,044
Hemradj, V. V., et al. (2020)	0,778	0,478	1,267	-1,009	0,313
Jensen, P. B., et al. (2018)	0,409	0,134	1,246	-1,573	0,116
Joseph, S. M., et al. (2016).	0,659	0,489	0,887	-2,746	0,006
Loehn, T., et al. (2020).	0,510	0,309	0,841	-2,636	0,008
O'Neill, W. W., et al. (2018).	0,855	0,805	0,907	-5,140	0,000
Ouweneel, D. M., et al. (2019).	0,788	0,488	1,271	-0,977	0,329
Ouweneel, D. M., et al. (2017).	0,380	0,063	2,309	-1,051	0,293
Schäfer, A., et al. (2020).	0,264	0,172	0,405	-6,102	0,000
Tarantini, G., et al. (2021)	0,558	0,353	0,880	-2,507	0,012
Wilkins C. E., et al. (2019)	1,277	0,770	2,120	0,947	0,344
	0,702	0,561	0,878	-3,093	0,002

Statistics for each study

Risk ratio and 95% CI





Iannaccone et al Int J Cardiol 2022

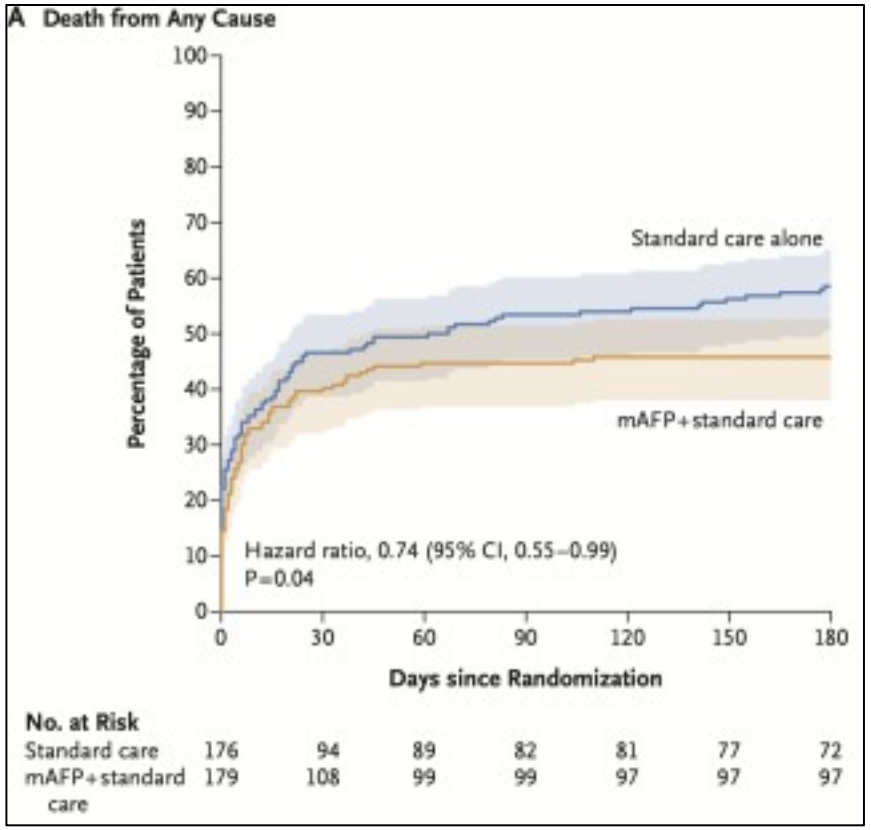
Lee H-H et al, J Am Cardiolg Intv 2021;14(10):1109-19

Timing of Intervention (II)

Management	Microaxial Flow Pump plus Standard Care (N=179)	Standard Care Alone (N=176)
Revascularization		
PCI — no. (%)	171 (95.5)	172 (97.7)
Non–culprit vessel PCI — no./no. of patients with multivessel disease (%)	59/127 (46.5)	55/129 (42.6)
Immediate CABG — no. (%)	1 (0.6)	4 (2.3)
Median time from admission to balloon inflation (IQR) — min	58 (36–114)	45 (31–81)
Mechanical circulatory support		
Placement of Impella CP device — no. (%)†	170 (95.0)	3 (1.7)
Randomization occurred before PCI and microaxial flow pump placed before PCI — no./total no. (%)	84/99 (84.8)	3/3 (100)
Median time from randomization to placement of microaxial flow pump (IQR) — min	14 (8–29)	15 (8–31)

ORIGINAL ARTICLE

Microaxial Flow Pump or Standard Care in Infarct-Related Cardiogenic Shock



N Engl J Med 2024;390:1382-93

Conclusions

1. **Cardiogenic shock represents a complex clinical condition characterized by a common definition but varying diagnostic criteria.**

2. Accurate identification of **CS etiology, clinical/hemodynamic phenotype, stratification, and risk modifiers** is essential to determine which patients will most likely benefit from an interventional approach.

3. In **SCAI Stage B**, the "B" stands not for *benign*, but for *beginning*—a stage that is **not clinically stable or safe** (*strict monitoring*).

4. When **percutaneous mechanical circulatory support** is indicated, **early initiation is critical** to stop or reverse the progression from **cardiogenic shock to hemo-metabolic shock.**

Thanks for your attention!