



Brigham and Women's Hospital
Founding Member, Mass General Brigham

Cardiogenic Shock

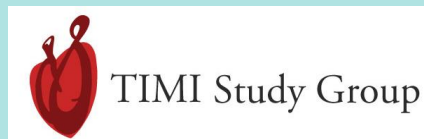
Erin Bohula, MD DPhil FACC

*Critical Care Cardiologist, Brigham & Women's
Hospital*

Investigator, TIMI Study Group

Assistant Professor, Harvard Medical School

**CONTINUING MEDICAL EDUCATION
DEPARTMENT OF MEDICINE**



**HARVARD MEDICAL SCHOOL
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Biography



- Training
 - Medicine Residency @ BWH
 - Cardiology and Critical Care Medicine Fellowships @ BWH
- Critical Care Cardiologist, Brigham & Women's Hospital
- Founding Member, Critical Care Cardiology Trials Network
- Senior Investigator, TIMI Study Group



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Relevant Disclosures

- Research grant to my institution
 - Amgen, Merck, Eisai, Astra Zeneca, Novartis, The Medicines Company
- Honoraria for CME programs
 - Medscape, Merck, Servier
- Consultant
 - Scleroderma Foundation, Novo Nordisk, CeleCor, Kowa



Objectives

- Review the definitions, epidemiology, and pathophysiology of cardiogenic shock
- Discuss my approach to management



Case

- 45yo M with recent smoking history (1 pack per wk) admitted with SOB, orthopnea, PND, abd distension.
- PE: HR 105, BP 120/92, 95% RA, JVP 15cm, no murmurs, +S3, b/l LE edema
- Labs: Cr 2.5, AST 750, TB 2, Lactate 8
- EKG: ST, Q in I, aVL, V1-3
- Echo: EF 20%, mild LV dilation, mod central MR, mod RV dysfunction, severe TR





RCTs for P2Y12 inhibition in ACS/PCI

CURE (N=12,562)

The New England Journal of Medicine

EFFECTS OF CLOPIDOGREL IN ADDITION TO ASPIRIN IN PATIENTS WITH ACUTE CORONARY SYNDROMES WITHOUT ST-SEGMENT ELEVATION

PLATO (N=18,624)

The NEW ENGLAND
JOURNAL of MEDICINE

ESTABLISHED IN 1812

SEPTEMBER 10, 2009

VOL. 361 NO. 11

CLARITY-TIMI 28 (N=3,491)

JO

ESTABL

Addition
for M

N=59,430

N=0 with Cardiogenic Shock

(N=11,145)

JOURNAL of MEDICINE

TRITON-TIMI 38 (N=13,608)

The NEW ENGLAND
JOURNAL of MEDICINE

ESTABLISHED IN 1812

NOVEMBER 15, 2007

VOL. 357 NO. 20

Prasugrel versus Clopidogrel in Patients
with Acute Coronary Syndromes

ORIGINAL ARTICLE

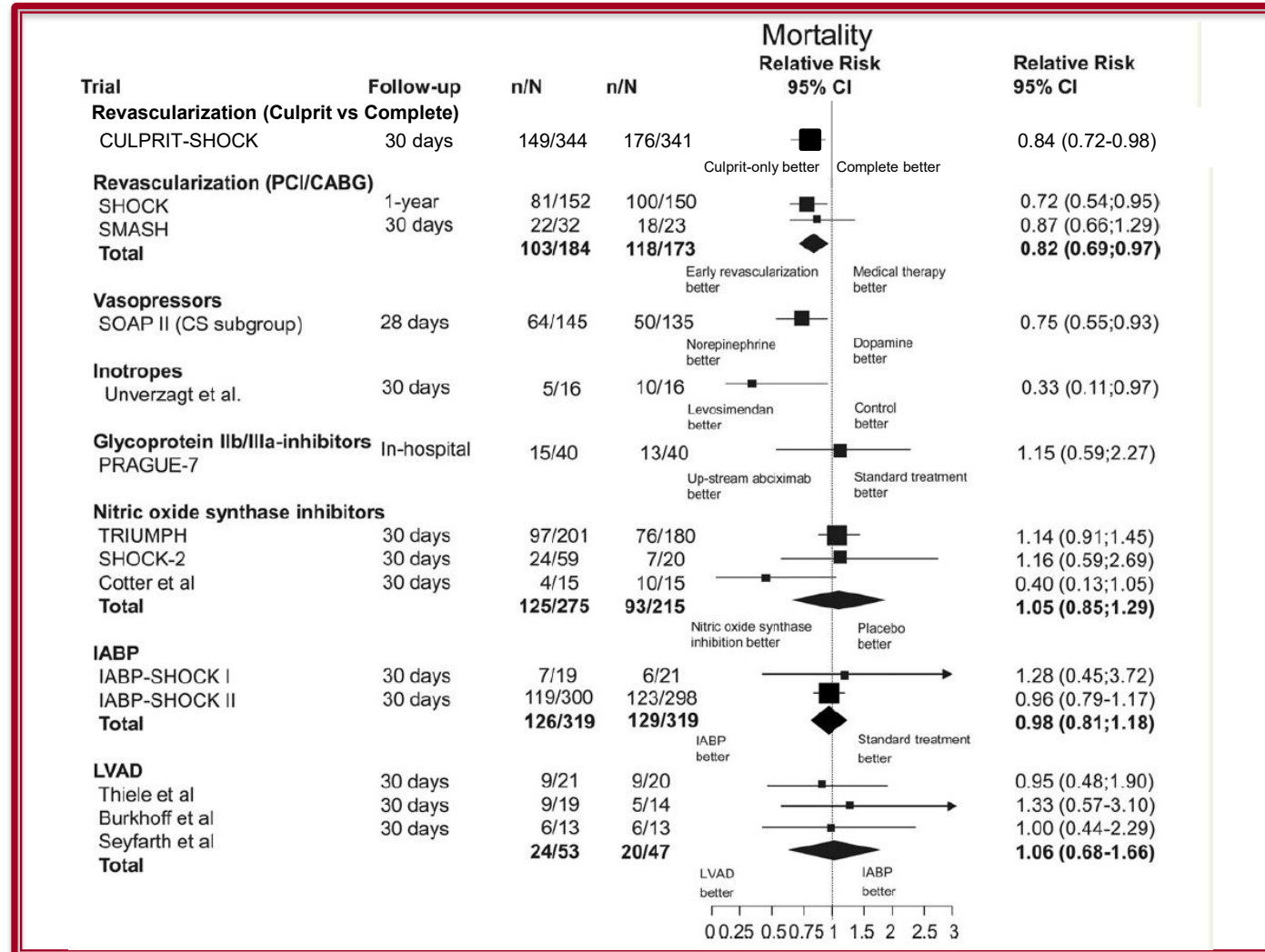
Effect of Platelet Inhibition with Cangrelor
during PCI on Ischemic Events



RCTs in Cardiogenic Shock

Total N~2,700

Currently ~3,800:
+ *DOREMI* (192)
+ *ECMO trials* (~560)
+ *DanGer Shock* (355)



Where Does That Leave Us?

- Pathophysiologic principles
- Extrapolation from non-critical CV pts
- Extrapolation from MICU or SICU pts



What is the current status for cardiogenic shock?



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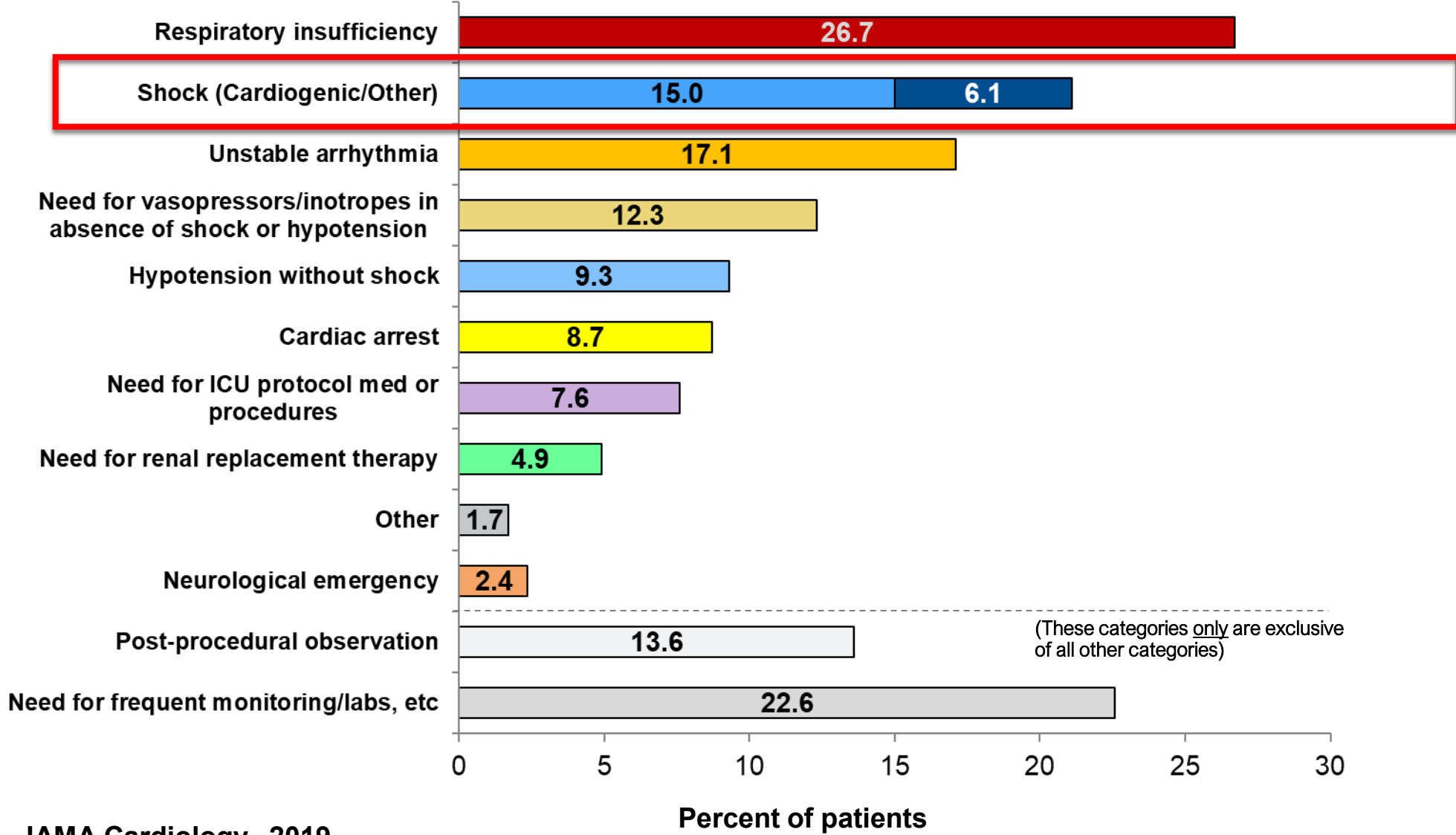


Critical Care Cardiology
Trials Network



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Indications for Intensive Care



Historical [Trial] Definitions of CS

	Hypotension	No Hypotension
Hypoperfusion	✓	X
No Hypoperfusion	X	X

Van Diepen S et al. Circ 2017;136.



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SHARC Criteria for CS

CS Definition

Cardiac disorder with evidence of hypoperfusion

Hypo- perfusion Definition

- Elevated lactate (>2 mmol/L)
- Acute kidney injury (creatinine $\geq 2\times$ ULN) or oliguria (UOP $< 0.5\text{ml/kg/hr}$)
- Acute hepatic injury (ALT $>3\times$ ULN)
- Cool or mottled extremities
- Altered mental status not explained by an alternative cause

Waksman R et al. Circ 2023;148(14).



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SHARC Criteria for CS

CS
Definition

Cardiac disorder with evidence of hypoperfusion

Subtypes

Hypotensive CS

- SBP <90 mmHg for ≥30 min, or
- Vasopressors, inotropes and/or MCS to maintain SBP ≥90 mmHg

Normotensive CS

- SBP ≥90 mmHg without vasopressors, inotropes or MCS

Optional
Criteria

$CI \leq 2.2 \text{ L/min/m}^2$

$CI \leq 2.2 \text{ L/min/m}^2$ and SVR
index >2200 dynes/cm/sec-
5/m²

Waksman R et al. Circ 2023;148(14).



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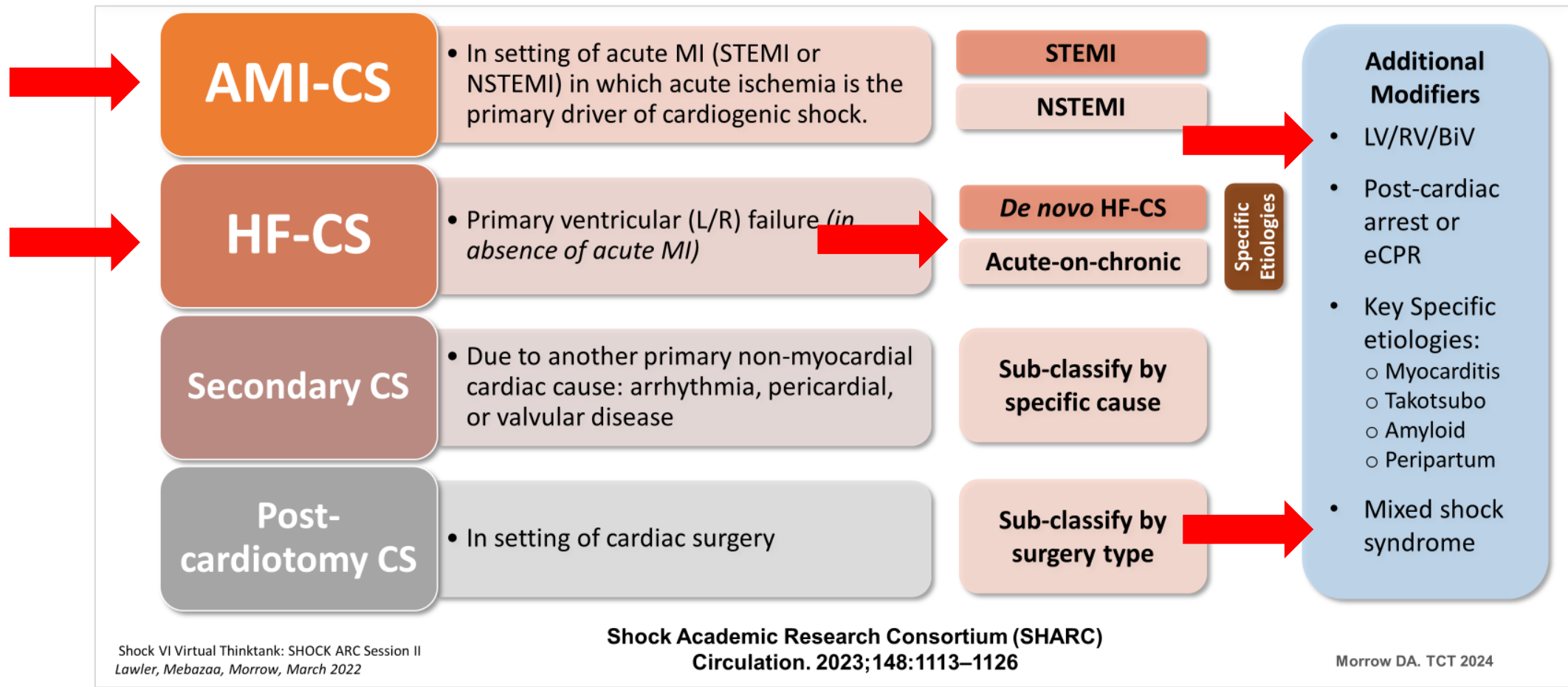


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Cardiogenic Shock Etiologies/Subtypes



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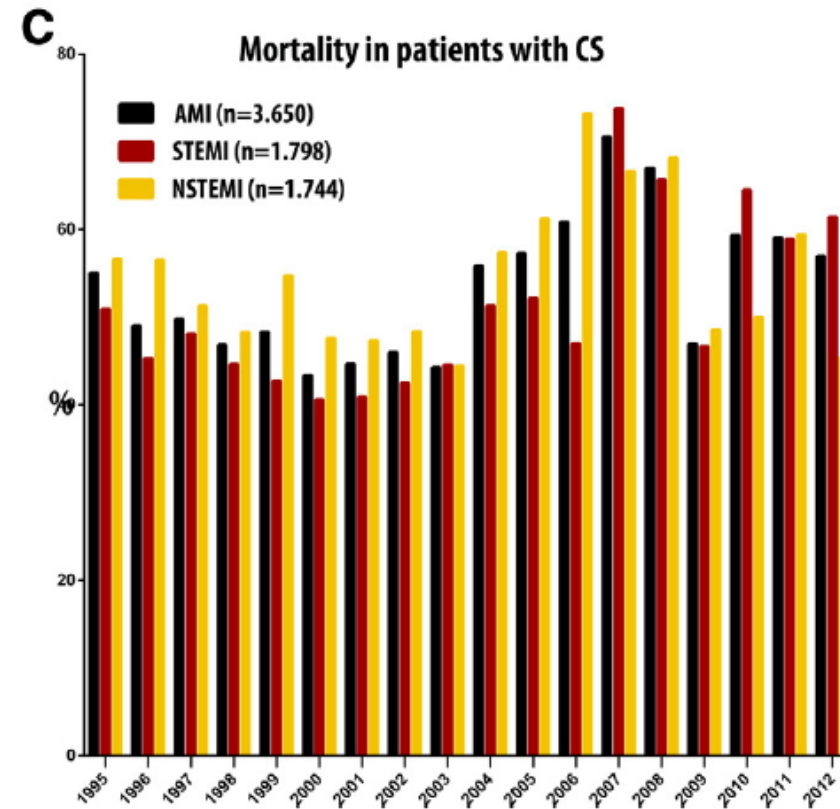
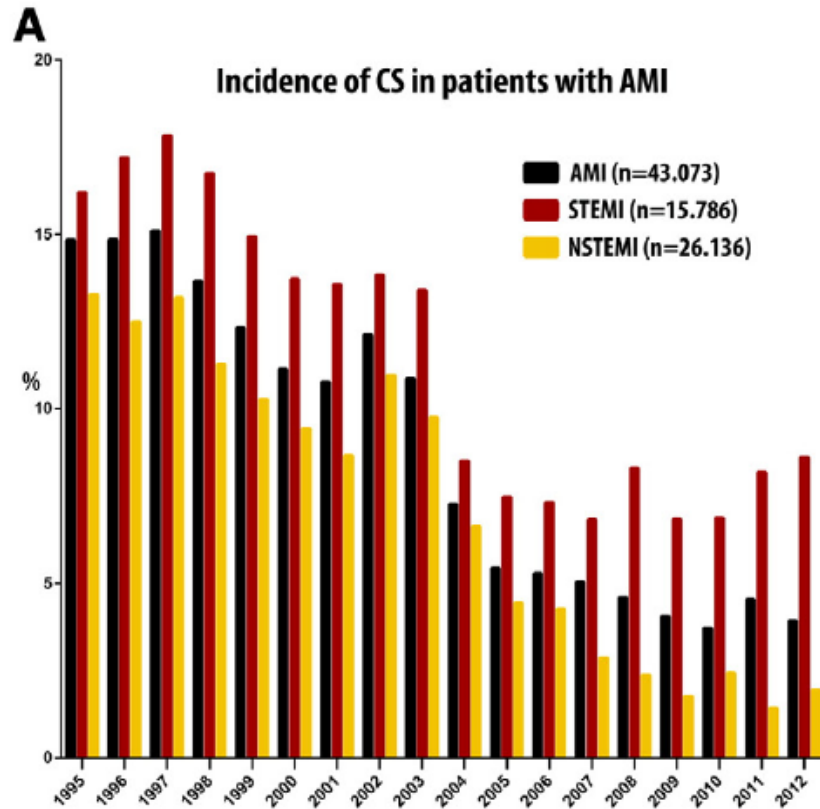


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Epidemiology



Patients are older and with more comorbidities

Redfors. Int J Cardiol. 2015.



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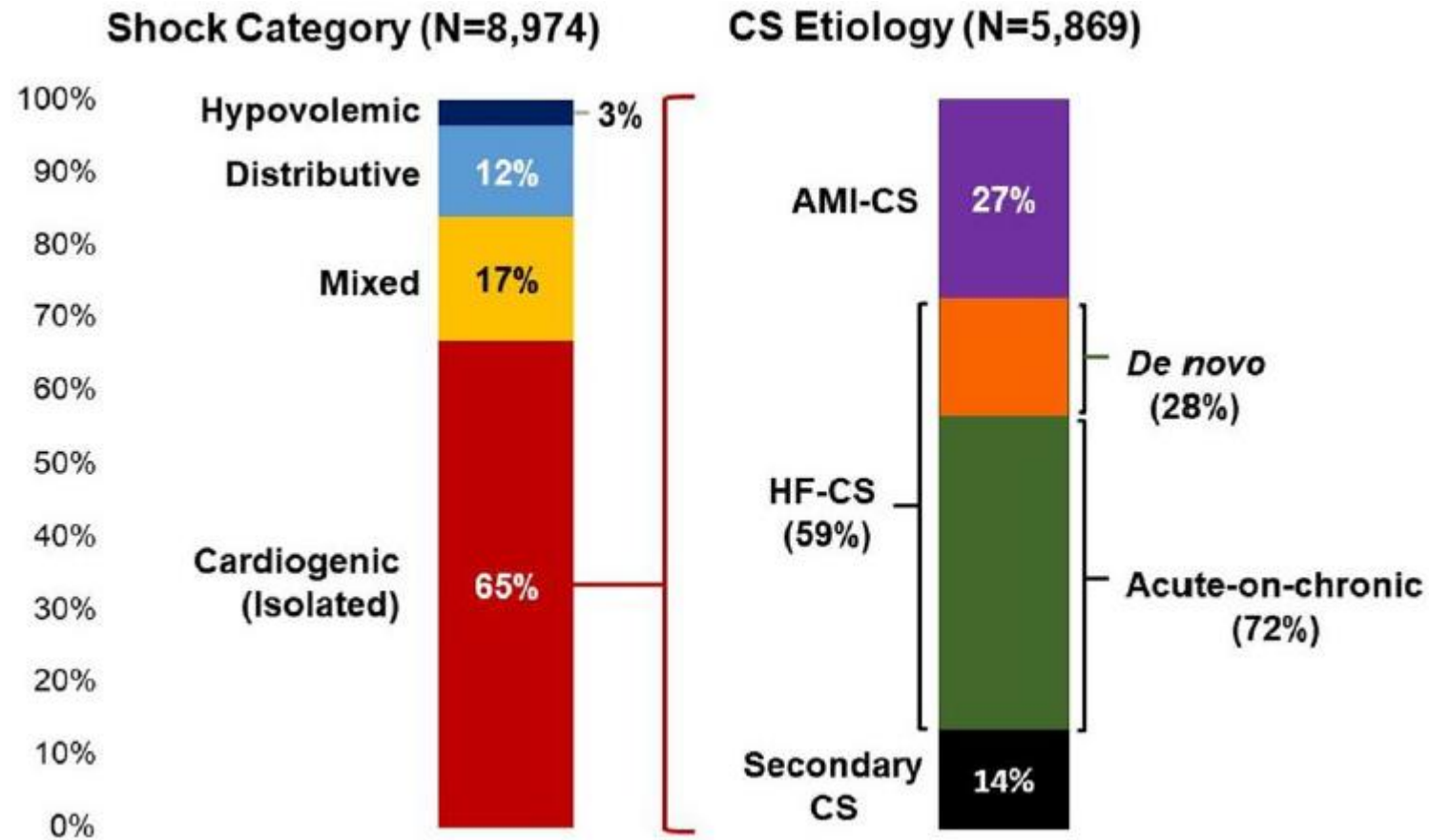


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Contemporary Epidemiology



European Heart Journal: Acute Cardiovascular Care (2024) **13**, 709–714



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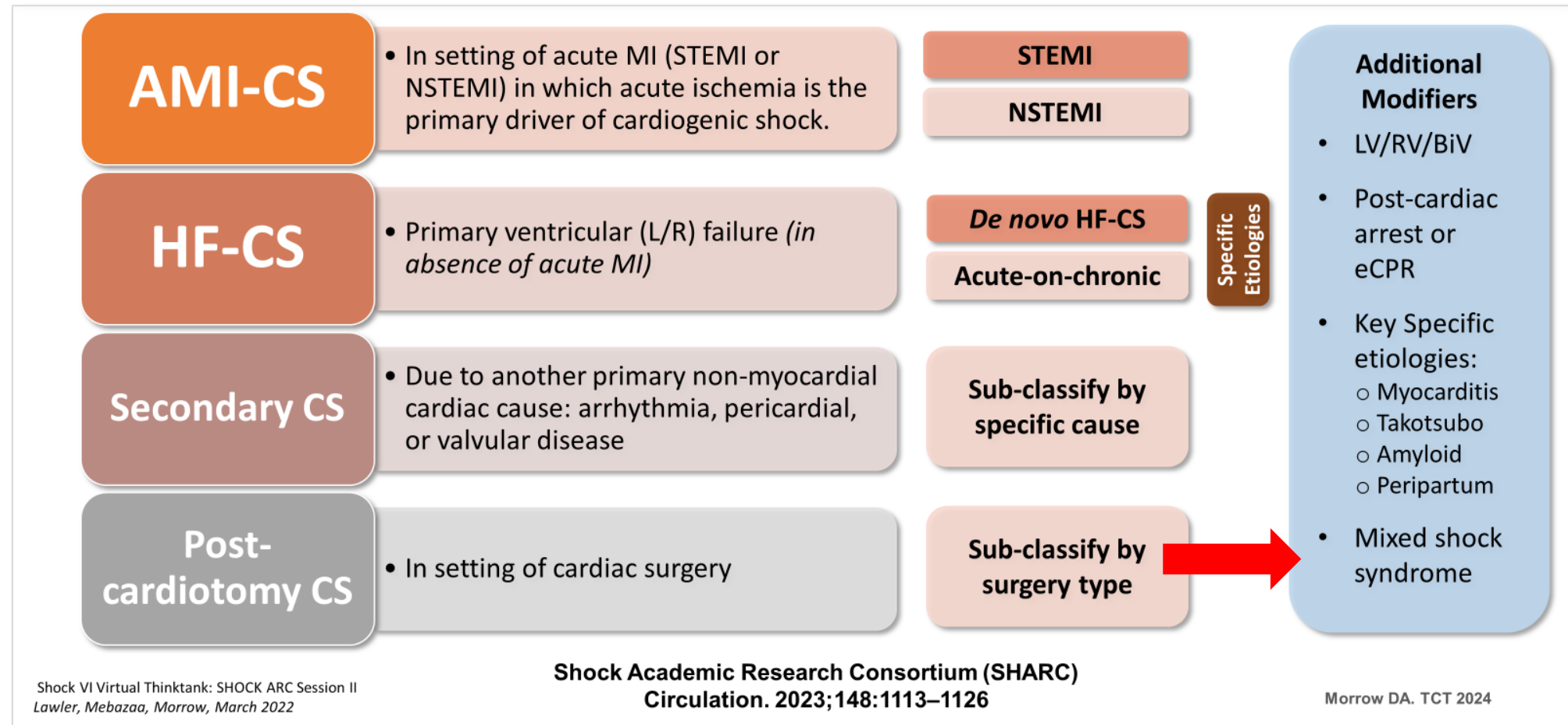


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Understanding the Heterogeneity of Shock



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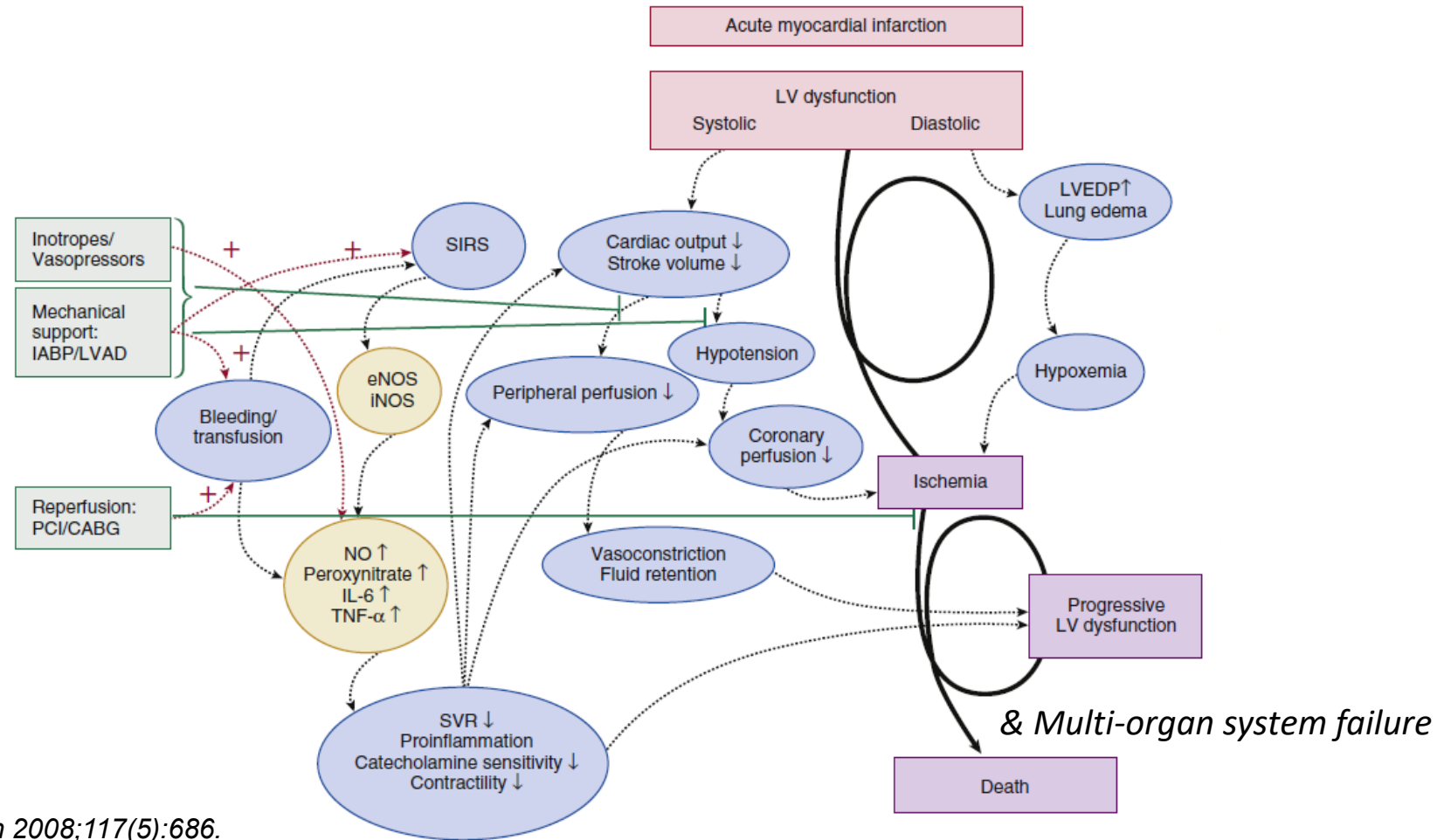


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Shock Spiral



Adapted from Reynolds et al. *Circulation* 2008;117(5):686.



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High vs Low SVR State

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

Van Diepen. Circulation. 2017.



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Uni- or Bi-Ventricular Failure?

Hemodynamic Profiles of Various Forms of Shock

Type of shock	RAP	PCWP	RAP/ PCWP	CO	SVR	CPO	PAPi
1° L-sided	nl or ↑	↑	<0.8	↓	↑	≤0.6	>0.9
1° R-sided	↑	nl or ↓	>0.8	↓	↑	≤0.6	≤0.9
Biventricular	↑	↑	>0.8	↓	↑	≤0.6	≤0.9

- Cardiac power output (CPO) (W) = $\text{MAP} \times \text{CO} / 451$
- Pulmonary artery pulsatility index (PAPi) = $(\text{PA systolic} - \text{PA diastolic}) / \text{RA mean}$

Pocket Cardiology, Ginder, Bohula and Sabatine



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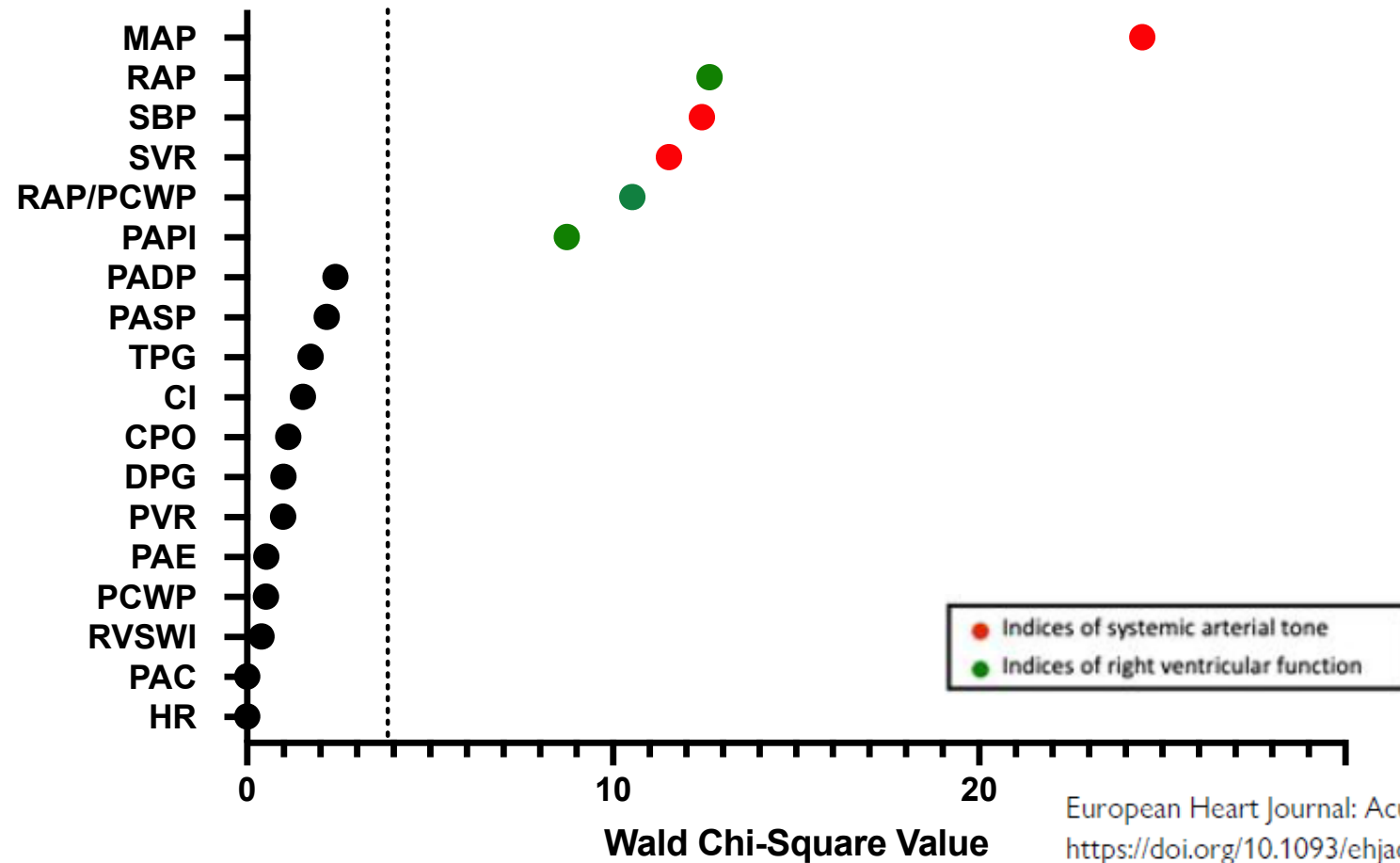


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Predictors of CS Mortality: Arterial Tone and RV Function



European Heart Journal: Acute Cardiovascular Care (2023) **12**, 651–660
<https://doi.org/10.1093/ehjacc/zuad095>



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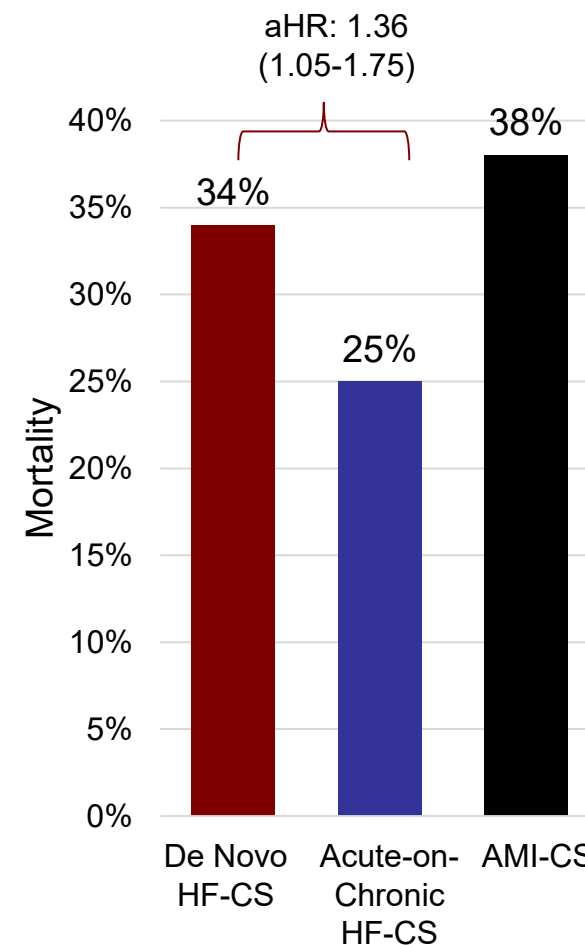
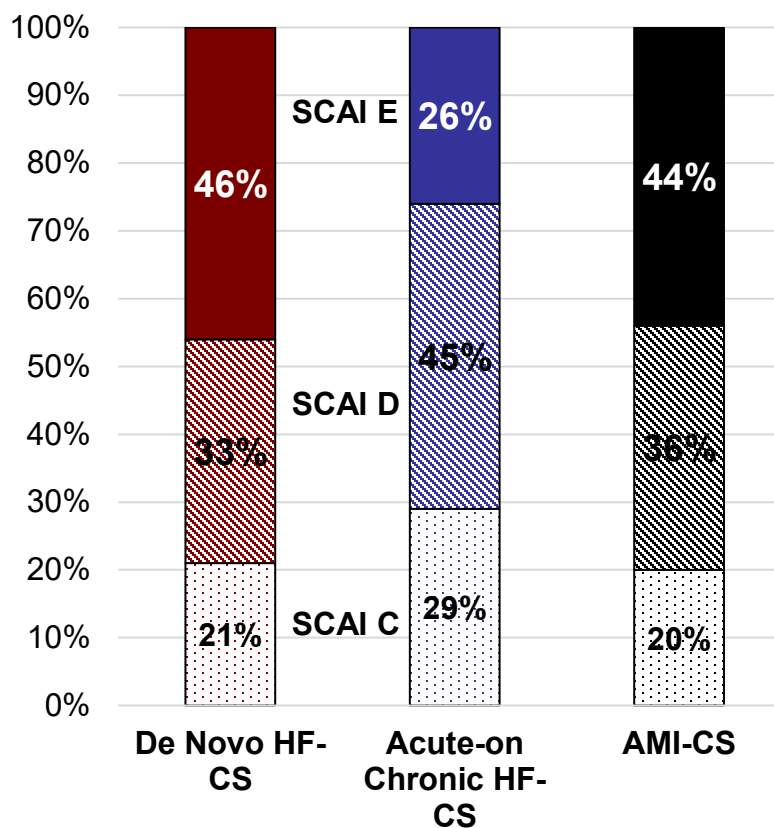
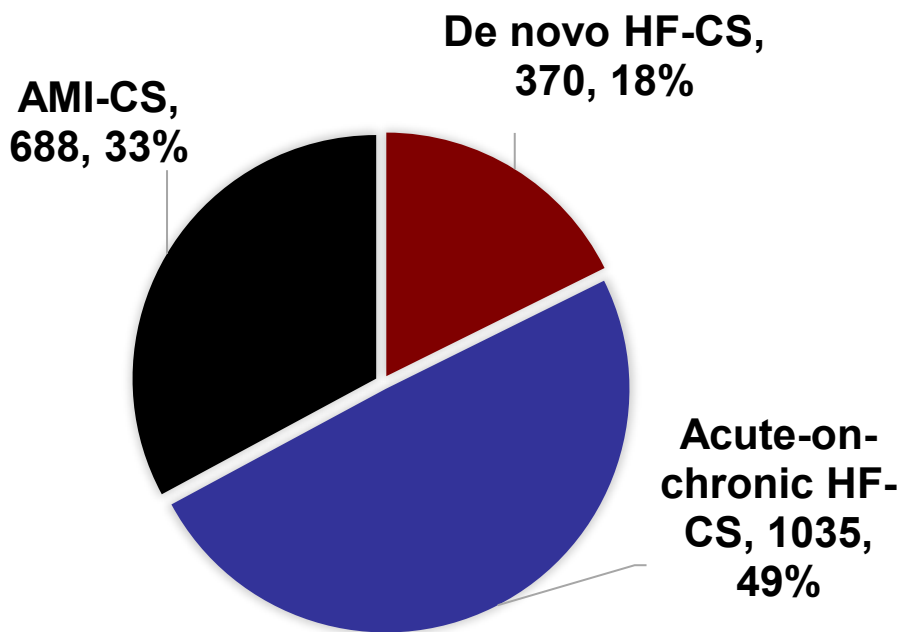
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Deeper Dive into CS Epidemiology



Cardiogenic Shock Admissions (N=2093 from 2017-2020)



An Academic Research Organization of
Brigham and Women's Hospital and Harvard Medical School

Bhatt A*, Berg D* et al. JCF. 2021.

Cardiogenic Shock Staging

SCAI Shock Stages (Cath CVI 2019;94:29)					
Stage	Description	BP	Exam	Labs	Hemodyn.
At risk	MI, ADHF	nl	nl	nl	nl
Beginning	Relative HoTN; tachycardia; w/o hypoperfusion	SBP <90, MAP <60, or >30 mmHg ↓ from baseline	↑ JVP, crackles, extrem warm	nl	↑ PCWP CI ≥2.2
Classic	Hypoperfusion that requires intervention	SBP <90, MAP <60, or requiring drugs/device to maintain BP	↑ JVP, crackles, extrem cool & mottled, ↓ UOP	↑ Cr lactate ≥2 ↑ LFTs	↑ PCWP CI <2.2 CPO ≤0.6
Deteriorating	Failing to respond			↑↑ Cr, lactate, & LFTs	
Extremis	Near or in cardiac arrest			lactate ≥5 pH <7.2	

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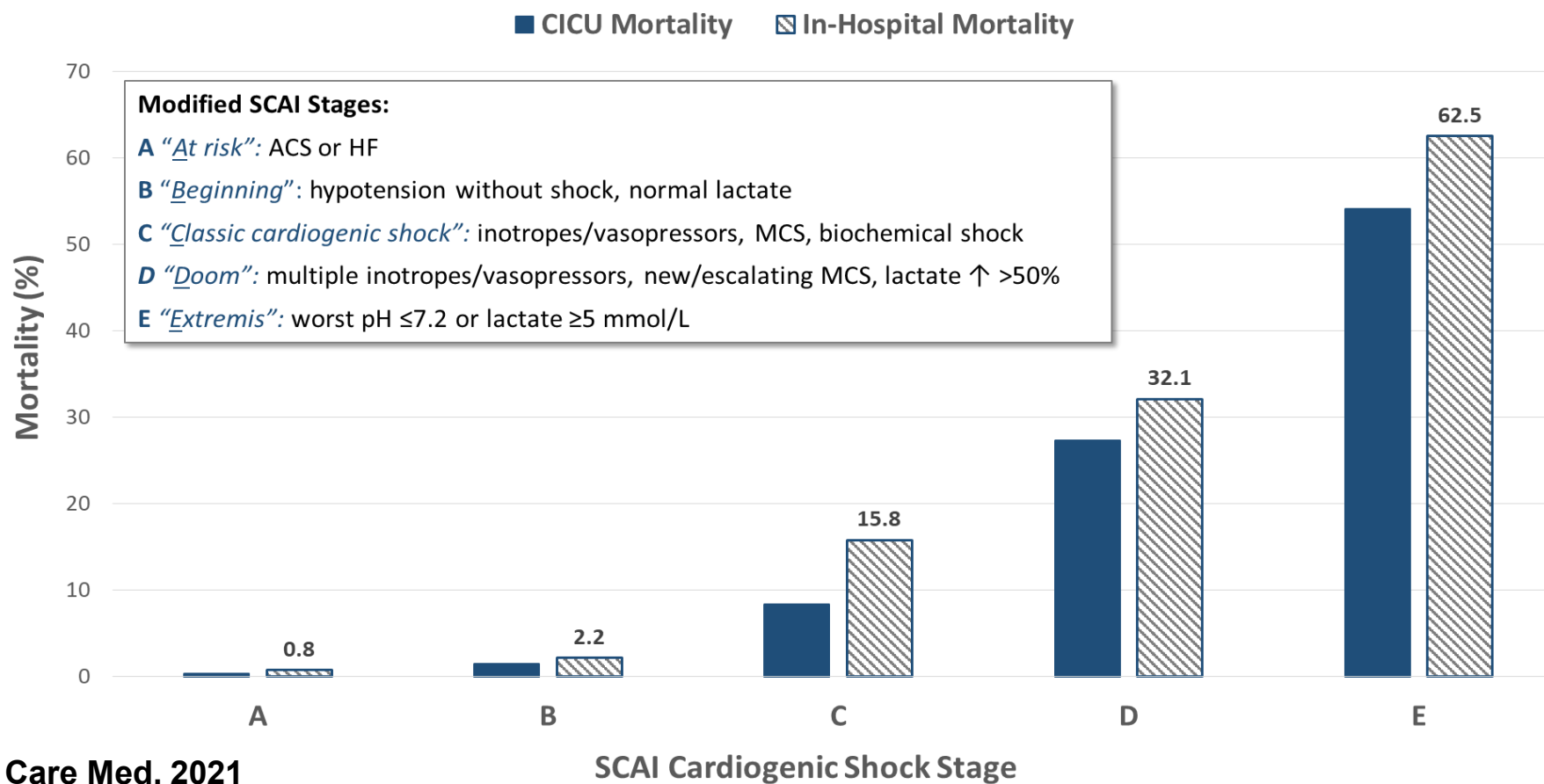


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Mortality by SCAI Classification



Lawler et al. Critical Care Med. 2021



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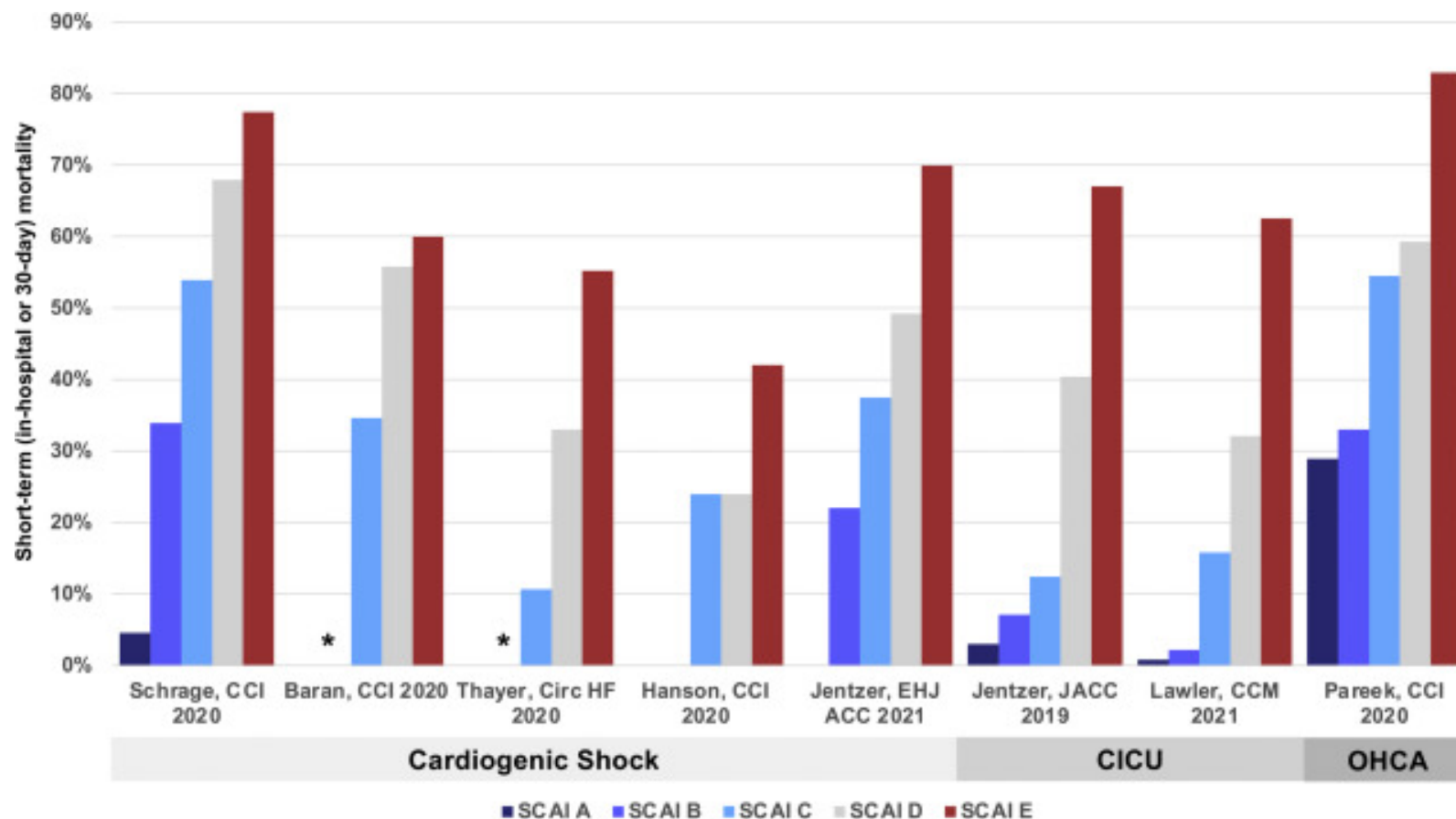


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Mortality by SCAI Classification



SCAI 2022



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What evidence-based interventions can we offer?



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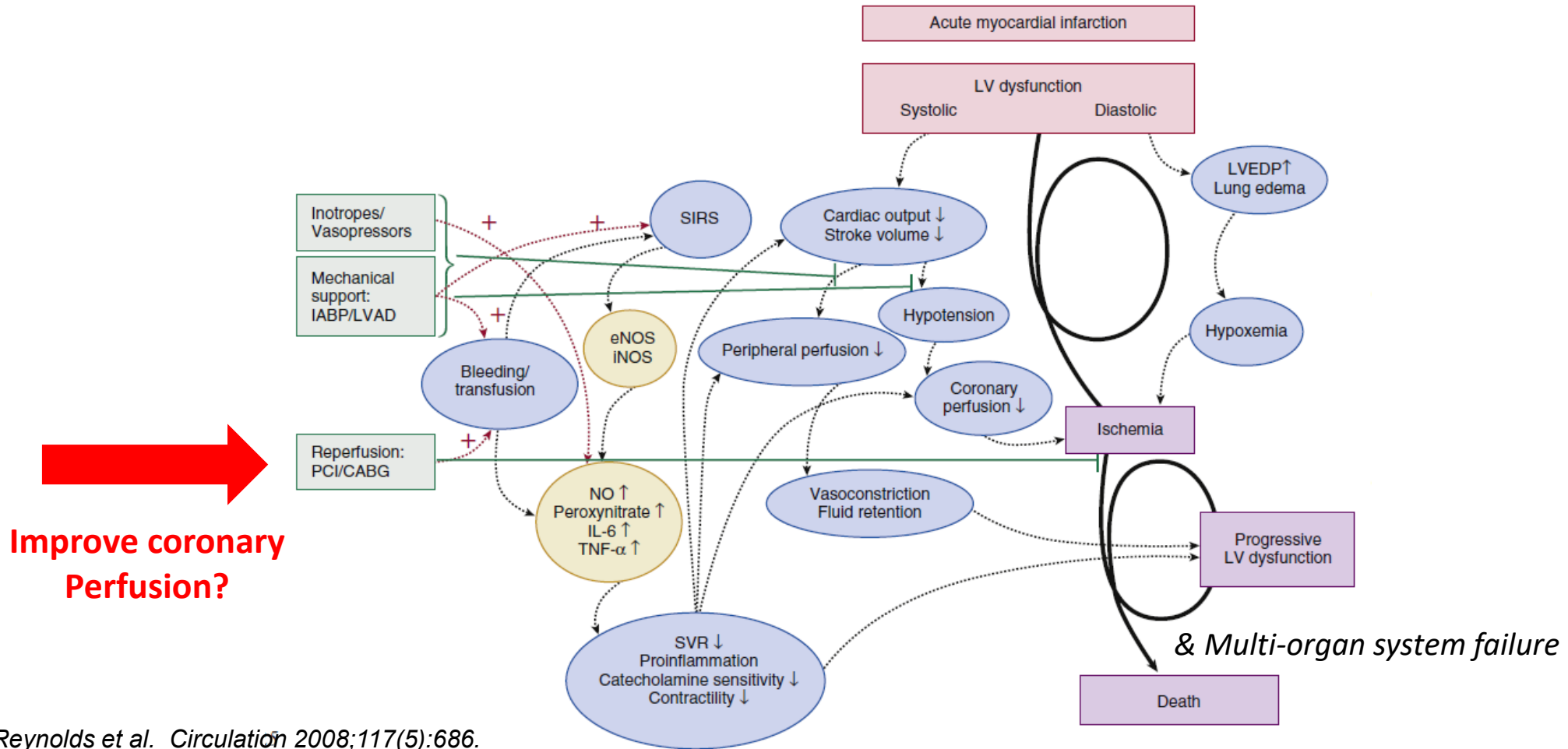


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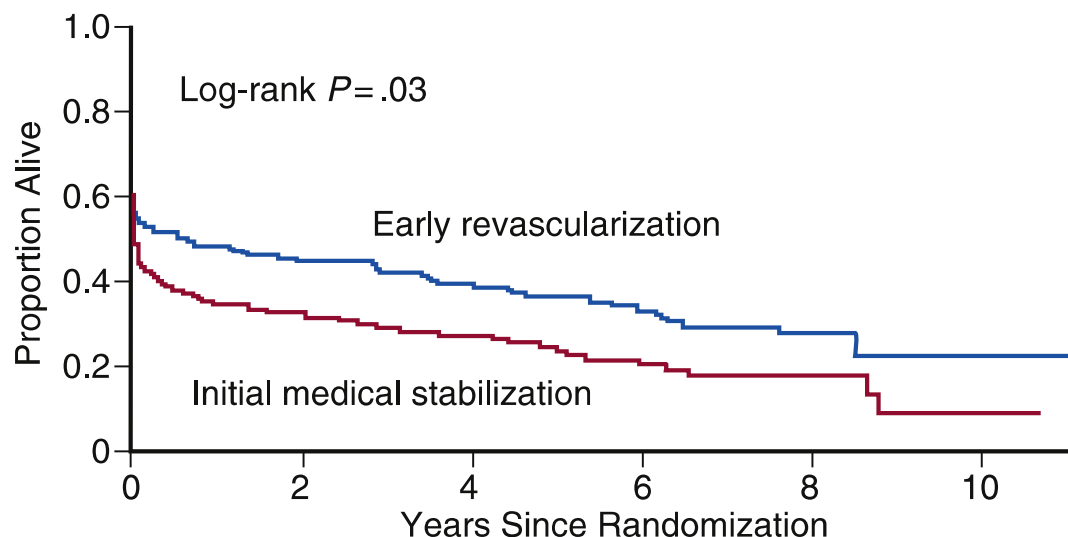
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Interrupting the Shock Spiral



Adapted from Reynolds et al. Circulation 2008;117(5):686.

Mortality Benefit of Early Revascularization in CS



No. at risk

Early revascularization	152	56	42	33	18	3
Initial medical stabilization	150	38	29	18	9	2

- 302 pts with STEMI and CS
- Early revasc w/in 6 hrs vs med Rx followed by prn revasc
- Survival
 - 30 d: 53.3% vs 44.0% ($p=0.11$)
 - 1 yr: 46.7% vs 33.6% ($p<0.03$)
 - 6 yr: 32.8% vs 19.6% ($p=0.03$)

Hochman JS, Sleeper LA, Webb JG, et al: JAMA 295(21):2511, 2006



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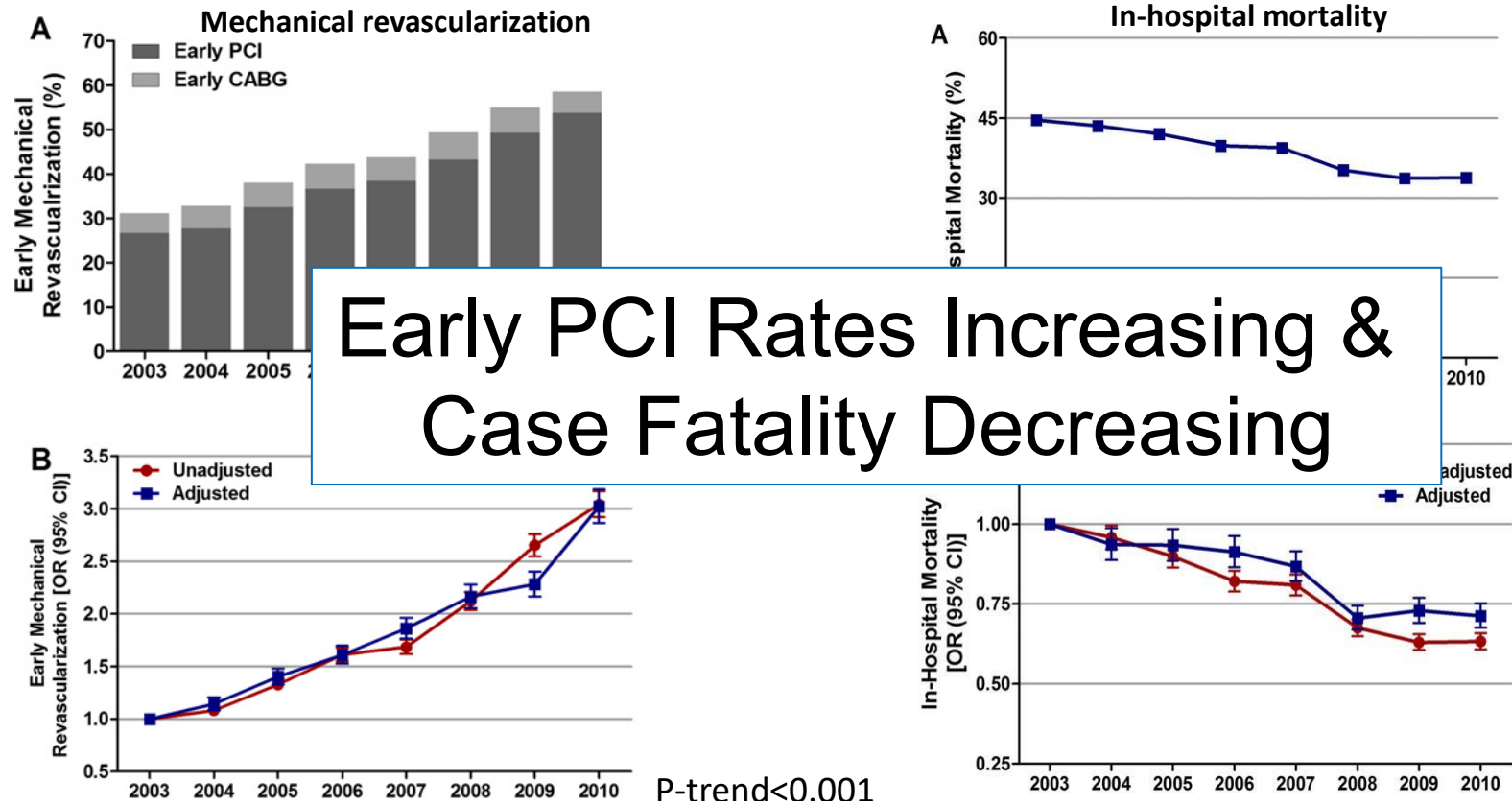
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CS due to STEMI Case Fatality Rate Decreasing Over Time

157,892 pts with CS due to STEMI in Nationwide Inpatient Sample from 2003-2010



Kolte D et al: JAMA 2014



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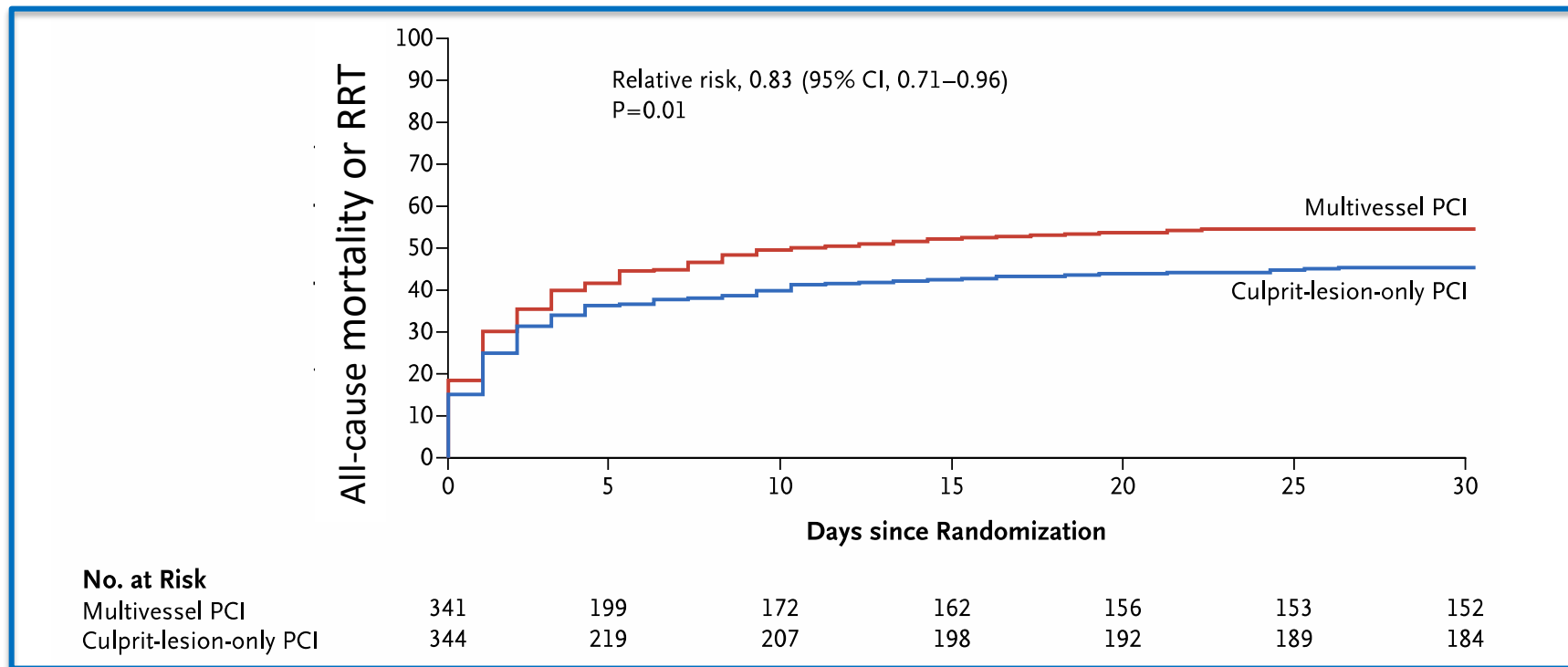
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CULPRIT-SHOCK

706 pts with CS due to AMI (61% STEMI/39% NSTEMI) and MVD
Rx: Immediate MV PCI vs Culprit-Only +/- Staged PCI



Thiele H et al. NEJM 2017;377(25).



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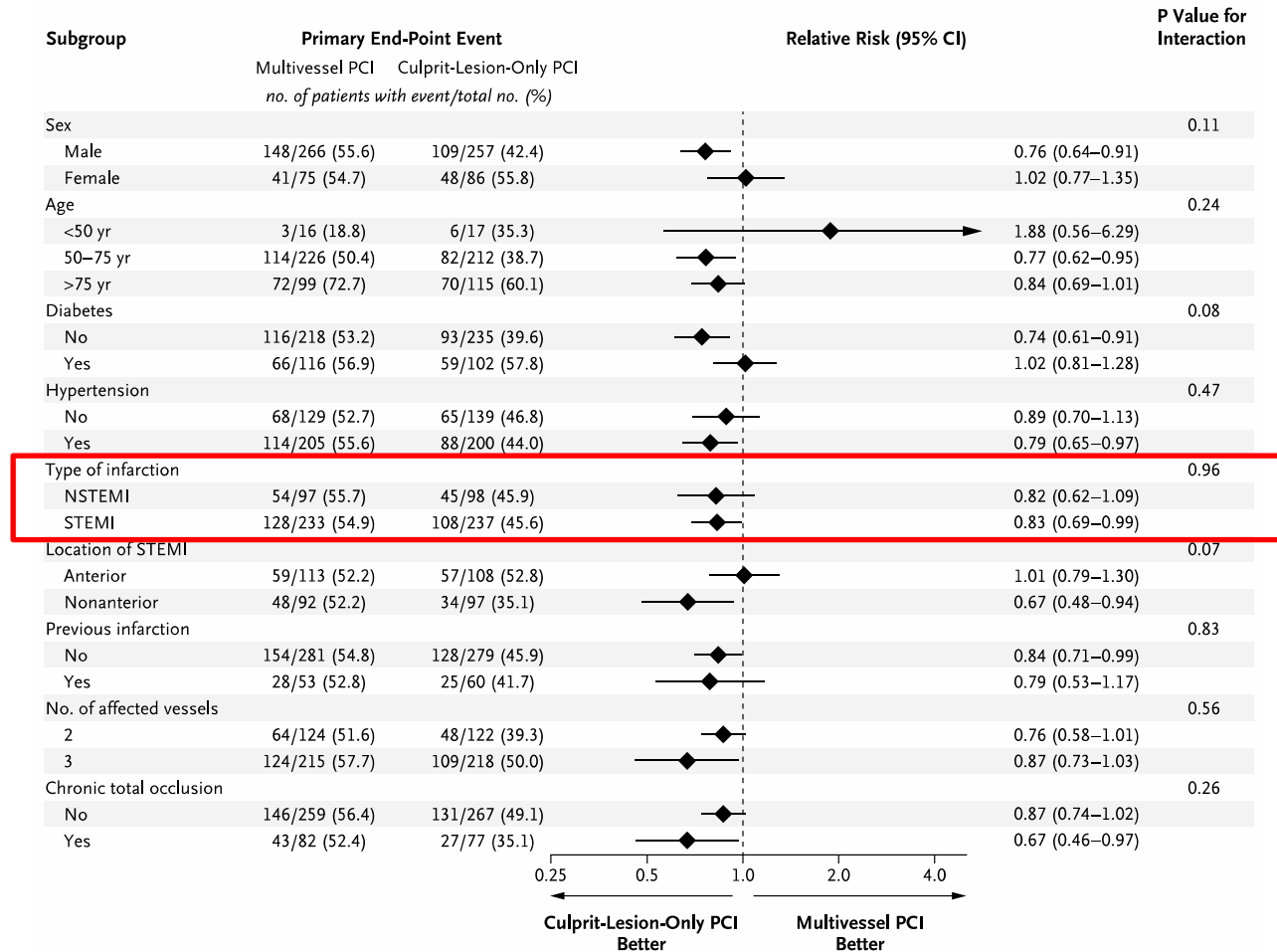


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CULPRIT-SHOCK



Thiele H et al. NEJM 2017;377(25).



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Immediate Multi-vessel PCI in Shock

ESC STEMI Guidelines 2017 → Revascularization Guidelines 2018

STEMI (NSTEMI), Cardiogenic Shock

2017

2018



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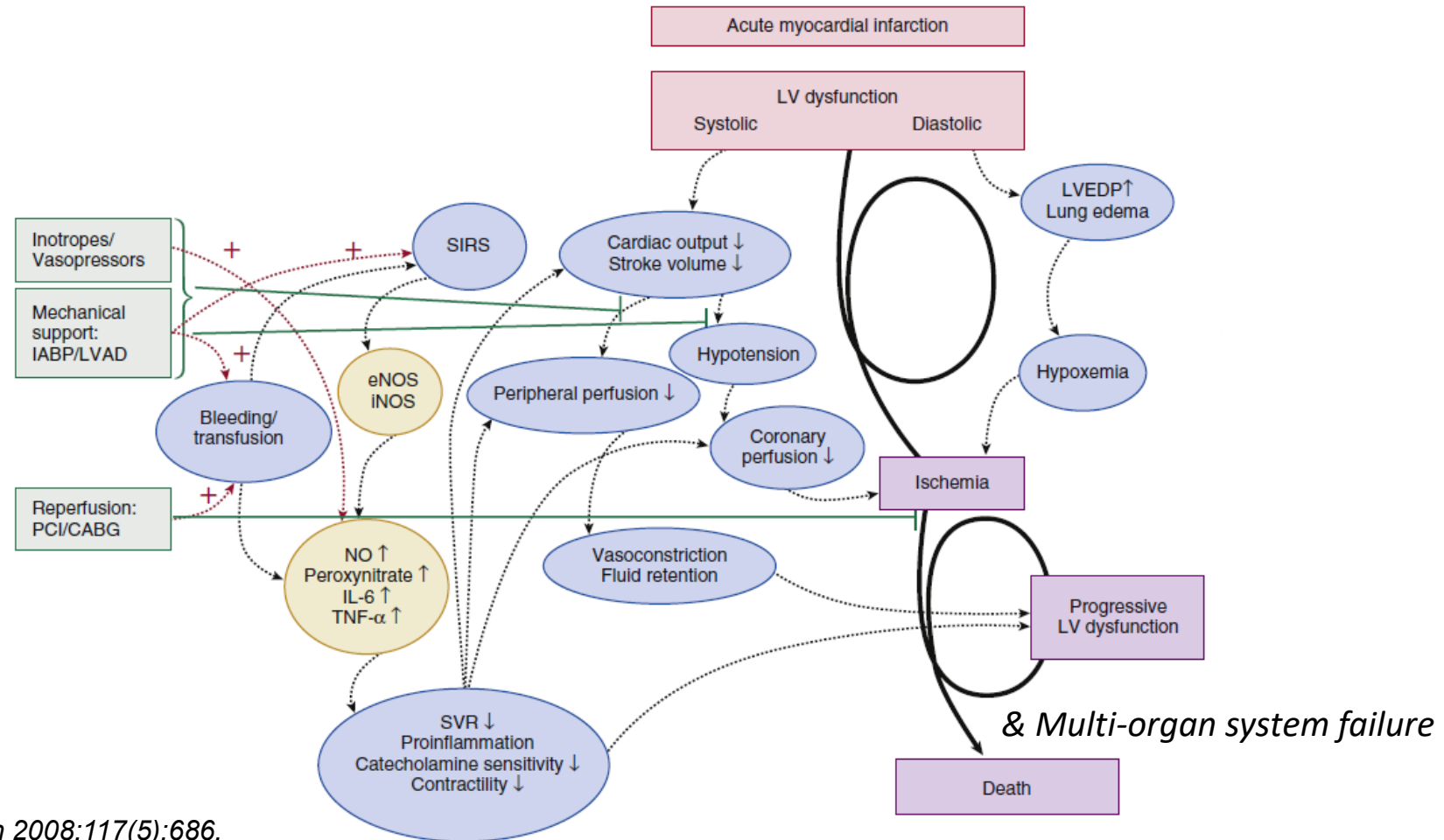
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Interrupting the Shock Spiral

Improve global
perfusion?



Adapted from Reynolds et al. *Circulation* 2008;117(5):686.



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Vasoactives

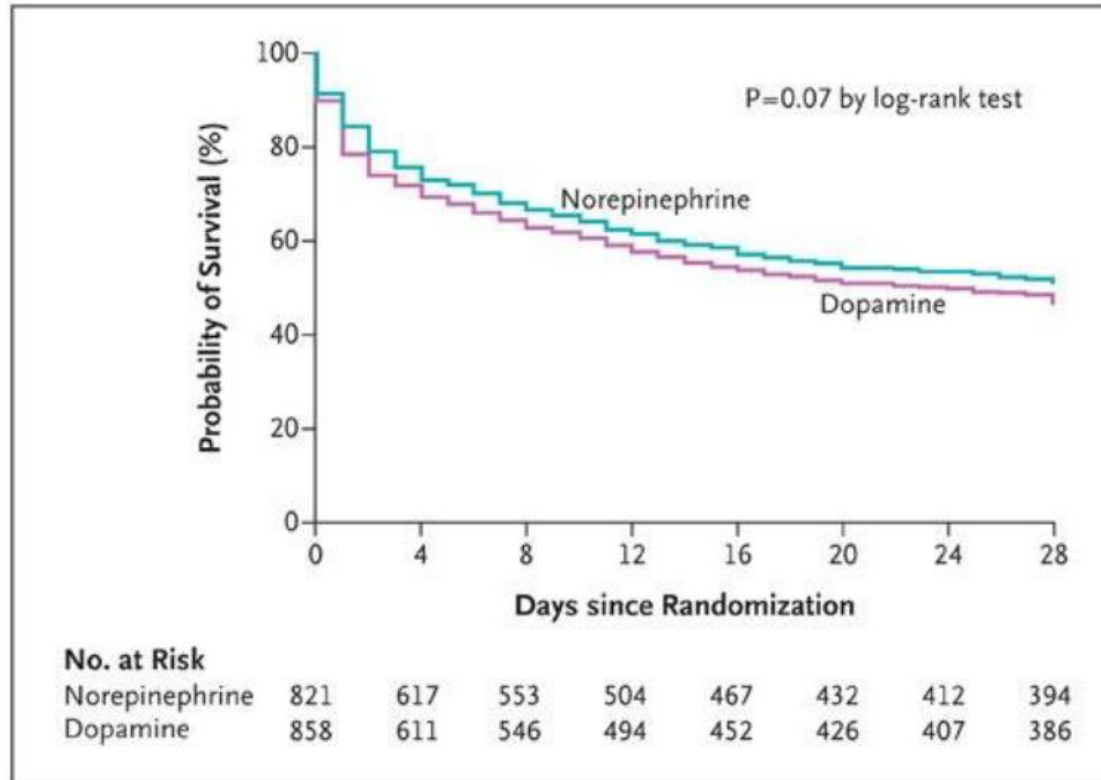
Pure vasopressors – Incr SVR

Inopressors – Incr CO, Incr SVR

Inodilators – Incr CO, decr SVR

Vasoactive Drugs							
Drug	Receptors	MAP	HR	CO	SVR	PVR	Comment
Pure vasopressors							
Phenylephrine	Pure α_1	$\uparrow\uparrow$	$\downarrow\downarrow^a$	$\downarrow^a$	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$	
Vasopressin	V_1 & V_2	$\uparrow\uparrow$	$\downarrow\downarrow^a$	$\downarrow^a$	$\uparrow\uparrow\uparrow$	$\leftrightarrow$	Consider if refractory to catechols. Attractive if RV dysfxn or PHT.
Inopressors (relative pressor vs. inotropy depends on drug & dose)							
Norepinephrine	$\alpha \gg \beta_1$	$\uparrow\uparrow$	$\leftrightarrow/\uparrow$	$\leftrightarrow/\uparrow$	$\uparrow\uparrow\uparrow$	$\leftrightarrow/\uparrow$	More pressor than inotrope. Fewer tachyarrhythmias than w/ dopa and mortality at least as good if not better.
Epinephrine							
Low-dose	β_1 & $\beta_2 > \alpha$	$\uparrow$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\downarrow$	$\leftrightarrow$	Inotrope
High-dose	$\alpha > \beta$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\uparrow$	Inotrope+pressor
Dopamine ^b							
Low-dose	D	$\leftrightarrow$	$\leftrightarrow/\uparrow$	$\leftrightarrow/\uparrow$	$\leftrightarrow/\downarrow$	$\leftrightarrow$	
Medium-dose	$\beta_1 > D, \alpha$	$\leftrightarrow/\uparrow$	$\uparrow$	$\uparrow\uparrow$	$\leftrightarrow$	$\leftrightarrow$	
High-dose	$\alpha > \beta_1, D$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\uparrow$	$\uparrow\uparrow$	$\uparrow$	
Inodilators							
Dobutamine	$\beta_1 \gg \beta_2, \alpha_1$	$\leftrightarrow/\downarrow$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\downarrow$	$\downarrow$	$\downarrow$ PCWP. Fast onset. Tachyphylaxis.
Milrinone	PDE ₃ inhib	$\downarrow\downarrow$	$\uparrow$	$\uparrow\uparrow\uparrow$	$\downarrow\downarrow$	$\downarrow\downarrow$	$\downarrow\downarrow$ PCWP; $\downarrow$ PVR; $\therefore$ attractive if RV dysfxn or PHT. Slow onset. Renally cleared.
Isoproterenol	β_1 & β_2	$\downarrow$	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$	$\downarrow\downarrow$	$\downarrow$	$\oplus$ chronotrope
Pure vasodilators							
Nitroglycerin	NO $\rightarrow$ sGC	$\downarrow$	$\uparrow$	$\leftrightarrow$	$\downarrow$	$\downarrow$	Venodilator $\gg$ arteriolar dilator
Nitroprusside ^c	NO $\rightarrow$ sGC	$\downarrow\downarrow\downarrow$	$\uparrow$	$\uparrow\uparrow^c$	$\downarrow\downarrow\downarrow$	$\downarrow\downarrow$	Arteriolar dilator $\geq$ venodilator

SOAP II: Dopamine vs Norepinephrine



- **28d mortality:**
 - **52.5% for DA vs 48.5% for norepi**
 - **OR 1.17 (0.97-1.42), p=0.10**
- **Arrhythmias: 24.1% vs 12.4%**

De Backer et al. NEJM 2010;362:779.



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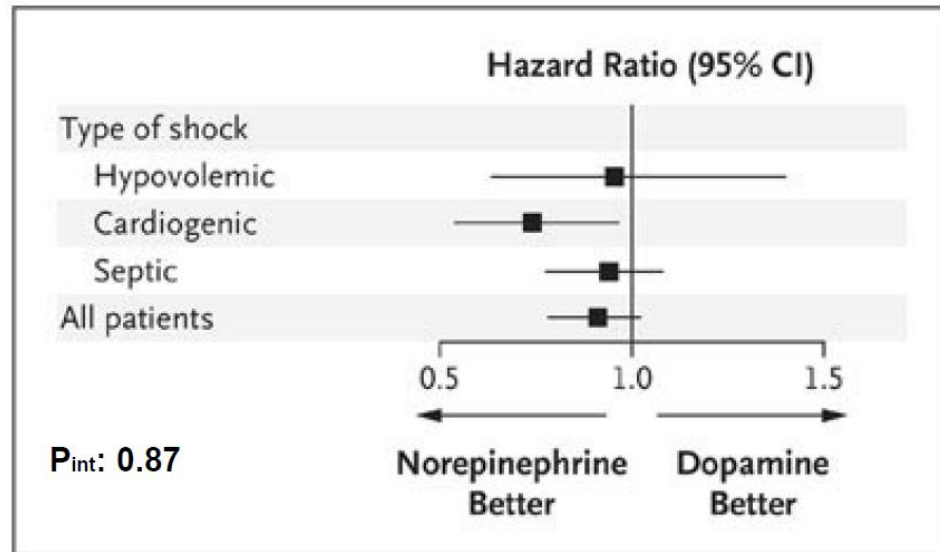


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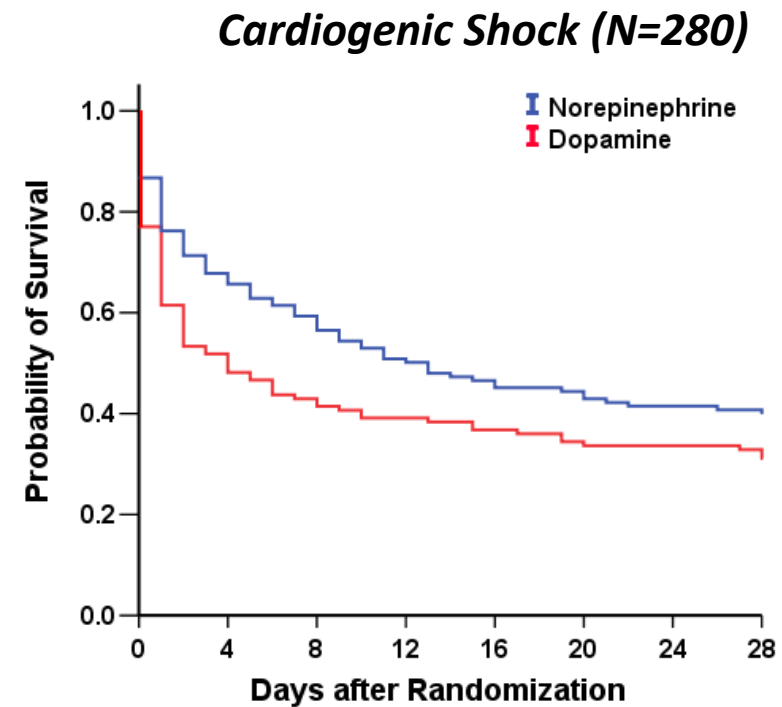


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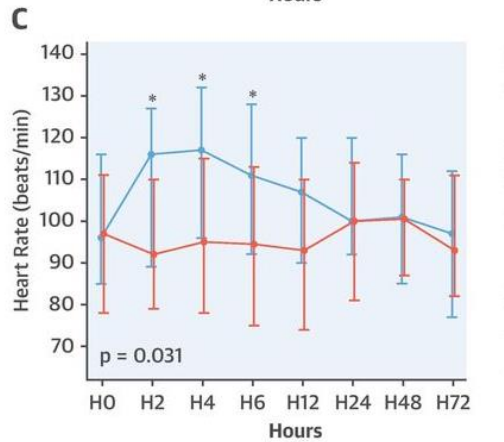
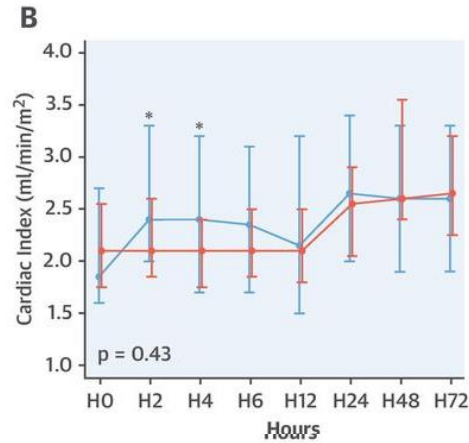
SOAP II: Dopamine vs Norepinephrine



Increased signal of harm with dopamine?



Epinephrine vs Norepinephrine



● Epinephrine

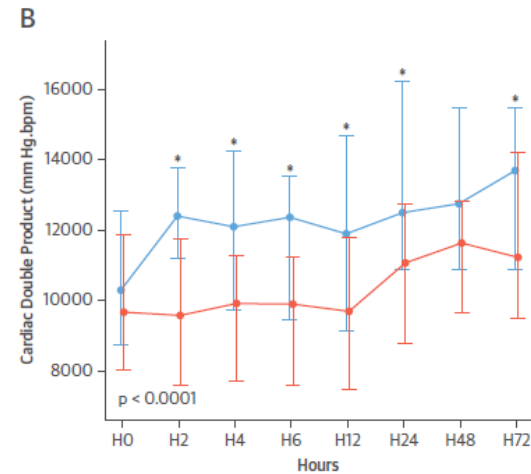
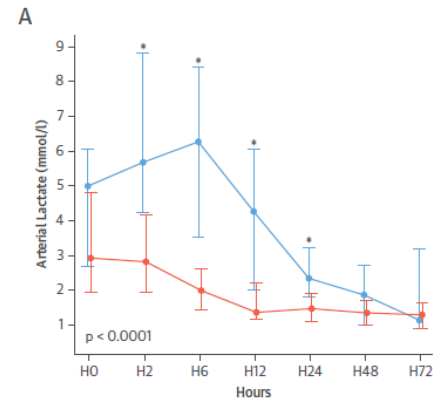


TABLE 2 Serious Adverse Events and Outcomes

	Epinephrine (n = 27)	Norepinephrine (n = 30)	p Value*	Odds Ratio (95% Confidence Interval)	p Value†
Refractory shock	10 (37)	2 (7)	0.008	8.24 (1.61–42.18)	0.011
Arrhythmia	11 (41)	10 (33)	0.59	1.37 (0.47–4.05)	0.56
ECLS	3 (11)	1 (3)	0.34	3.62 (0.35–37.14)	0.28
Death	14 (52)	11 (37)	0.29	1.86 (0.65–5.36)	0.25
Death within 7 days	8 (30)	3 (10)	0.093	3.79 (0.89–16.17)	0.072
Death within 28 days	13 (48)	8 (27)	0.11	2.55 (0.84–7.72)	0.097

Values are n (%) unless otherwise indicated. Odds ratios were expressed by using the norepinephrine group as reference. *p value from the Fisher exact test. †p value from the Wald test.

ECLS = extracorporeal life support.

Increased signal of harm with epinephrine?

Levy et al. JACC 2018;72:173-82.



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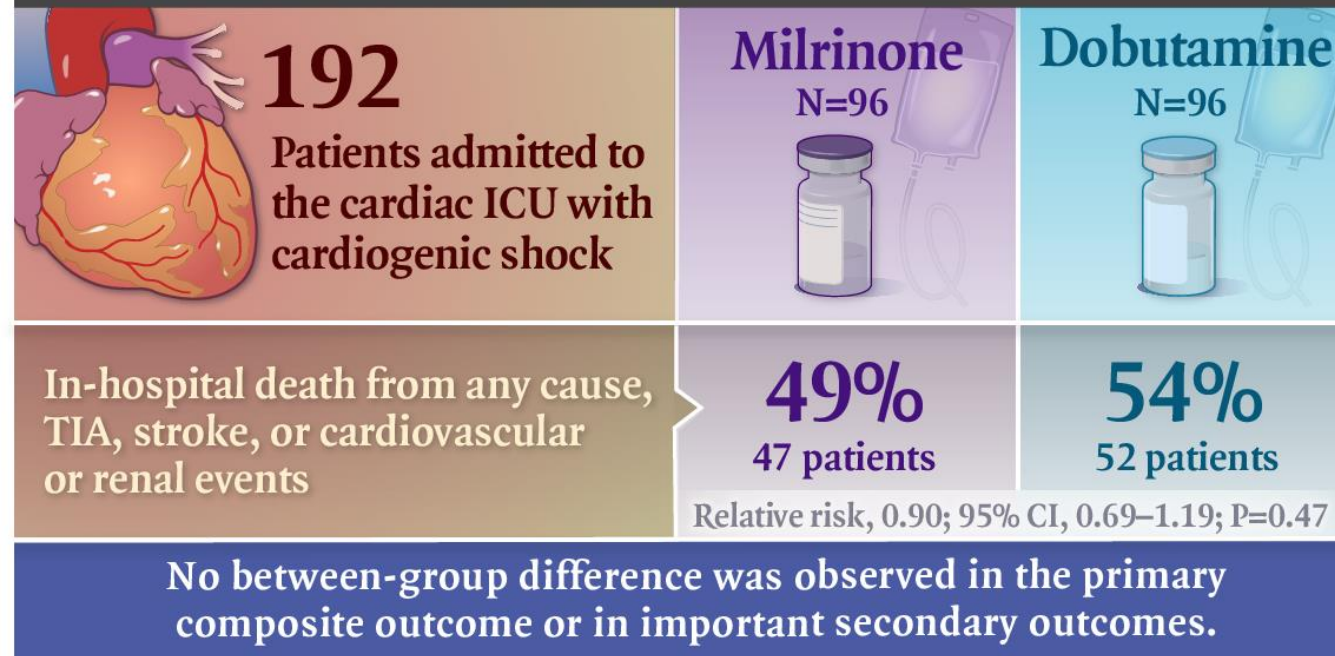
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DOREMI Trial

The NEW ENGLAND JOURNAL of MEDICINE

Milrinone vs. Dobutamine in Cardiogenic Shock

DOUBLE-BLIND, RANDOMIZED TRIAL



R. Mathew et al. 10.1056/NEJMoa2026845

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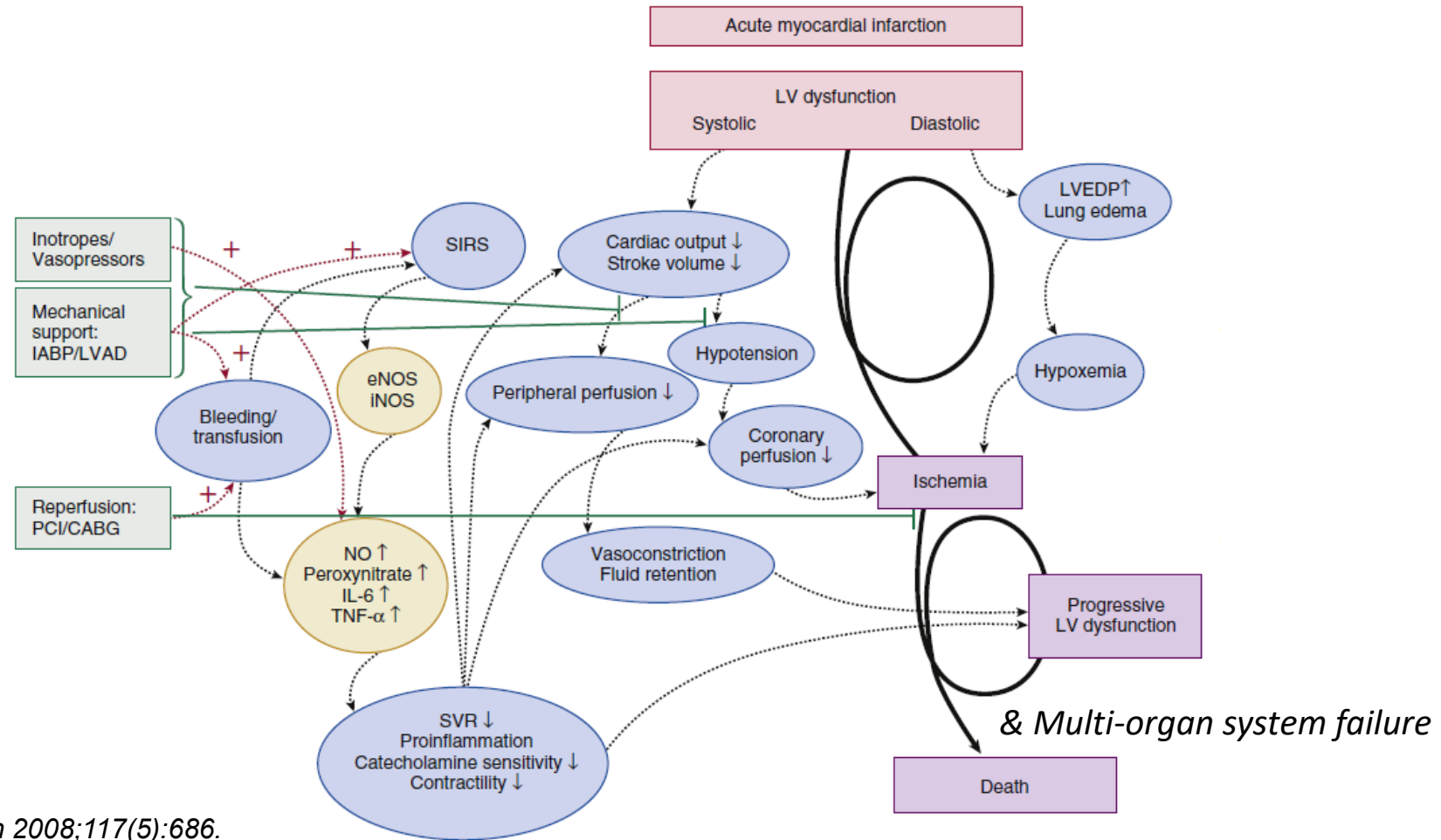
Vasopressor Take Home

- Catecholamines have not demonstrated improved survival, but in theory may “break” the shock spiral
- Very limited data suggests:
 - Norepinephrine may be better than dopamine and epinephrine



Interrupting the Shock Spiral

Improve global
perfusion?



Adapted from Reynolds et al. *Circulation* 2008;117(5):686.



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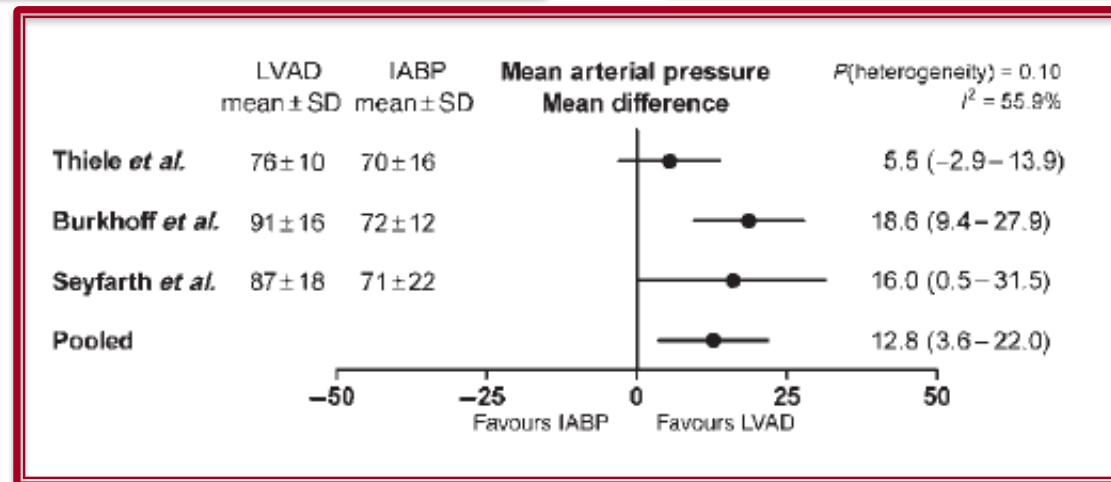
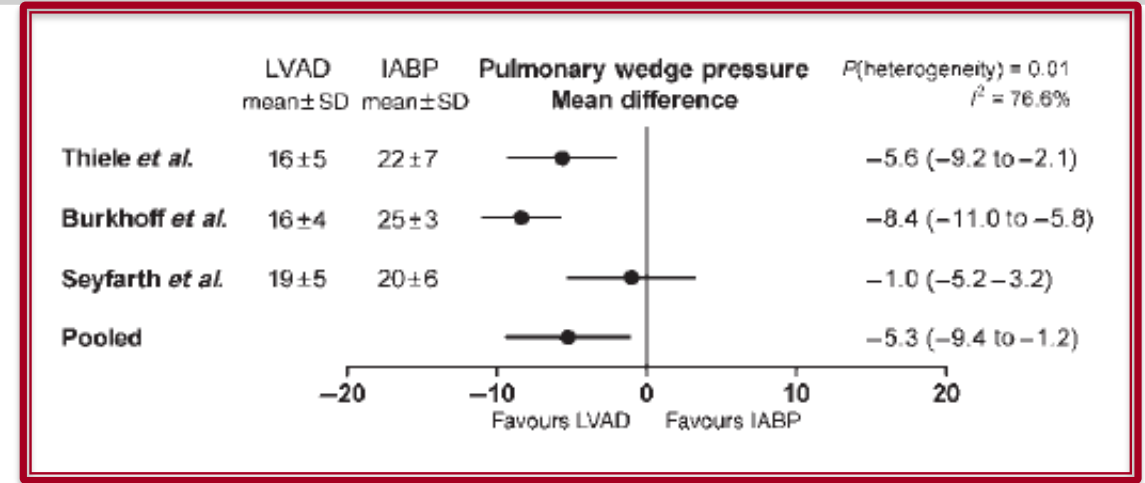
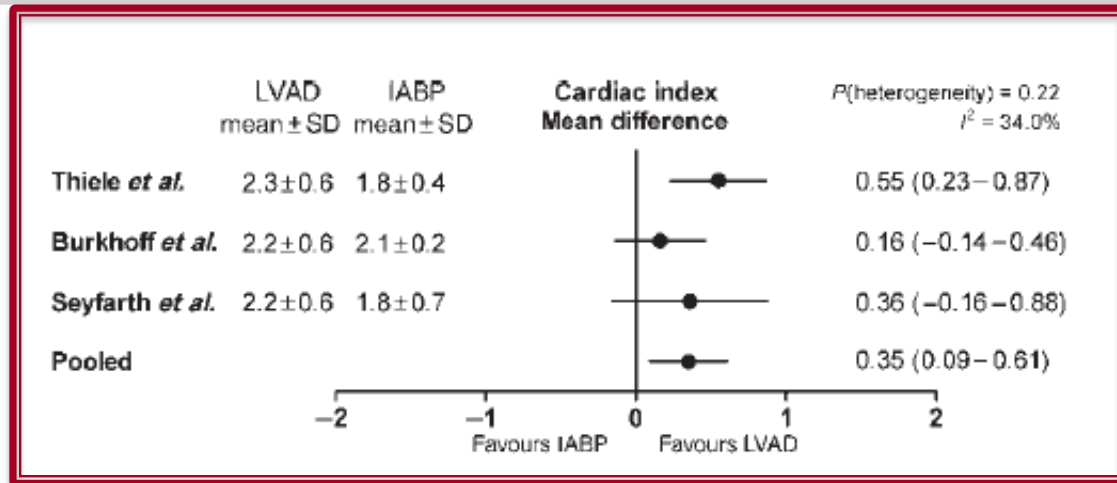
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Potential Mechanisms of Benefit with MCS

- ↓ intracardiac filling pressures and volumes →
 - ↓ PCWP → ↓ hypoxia
 - ↓ wall stress → ↓ myocardial oxygen demand → ↓ ischemia
- ↓ ventricular afterload →
 - ↓ myocardial oxygen demand → ↓ ischemia
 - ↑ SV → ↑ CO → ↓ hypotension and end-organ hypoperfusion
- Directly ↑ CO → ↓ hypotension and end-organ hypoperfusion
- ↑ coronary perfusion → ↓ ischemia



Devices Improve Hemodynamics



Cheng *et al. EHJ.* 2009;30:2102-2108.



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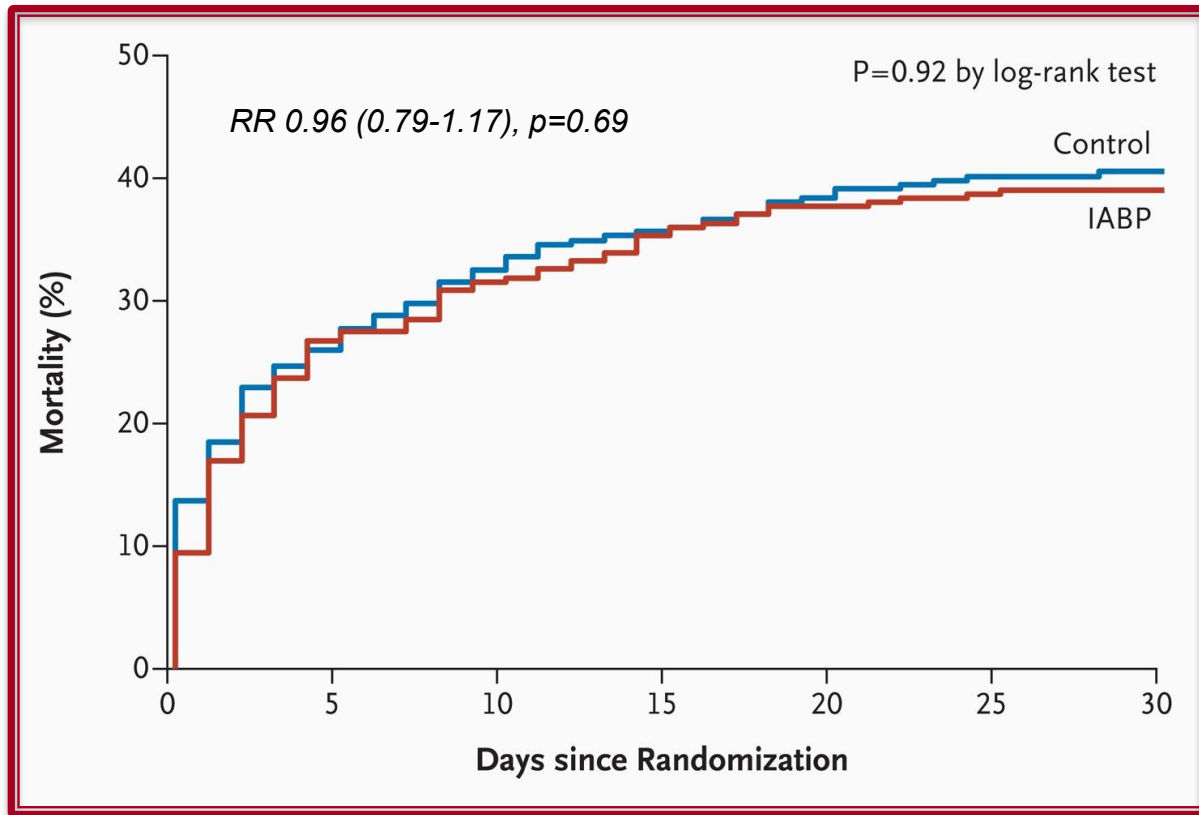
TIMI Study Group



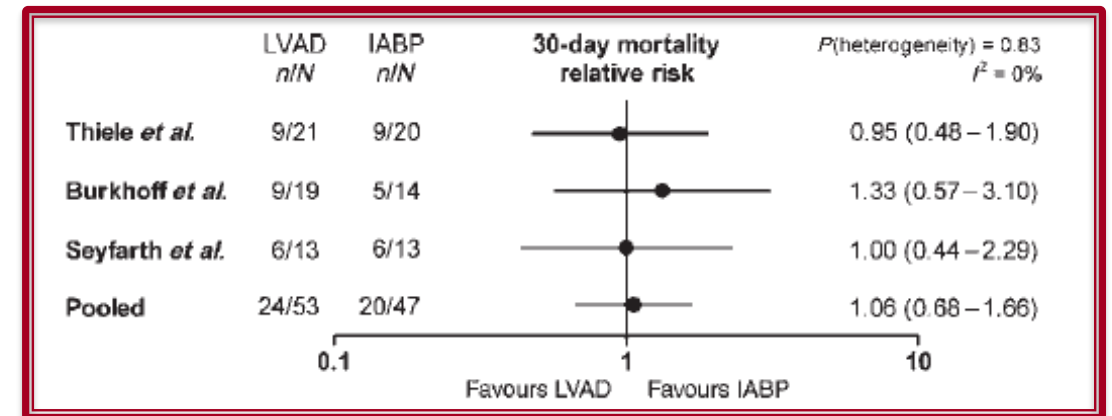
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Lack of Mortality Benefit

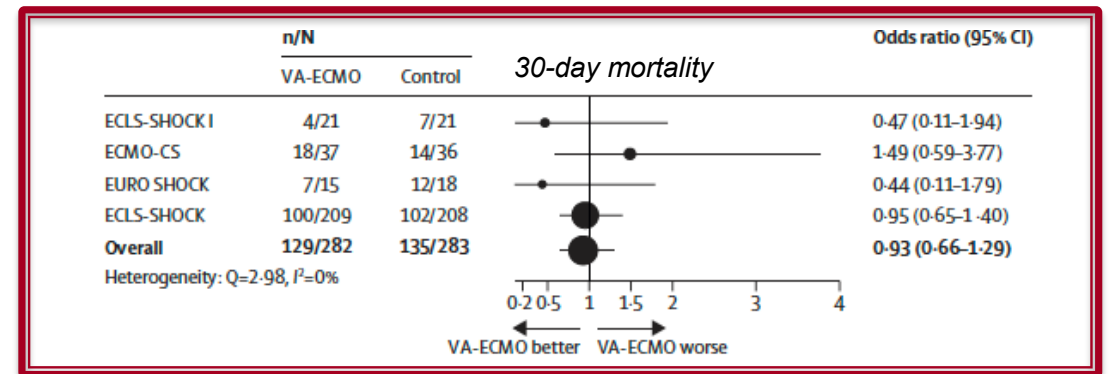
IABP-SHOCK II (AMI-CS, N=598)



LVAD vs IABP (N=100)



VA-ECMO vs Control (N=567)



Thiele *et al.* NEJM. 2012; Cheng *et al.* EHJ. 2009;30:2102-2108; Zeymer *et al.* Lancet 2023.



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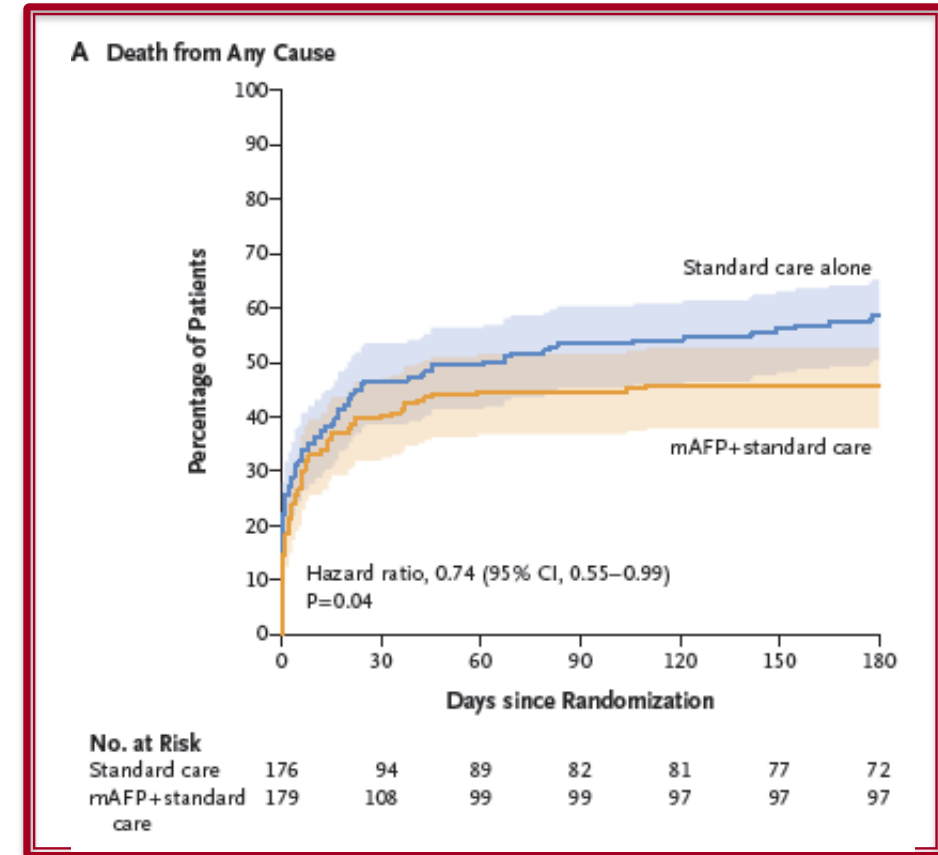
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DanGer Shock

- 355 pts with STEMI and AMI-CS, LA ≥ 2.5 , LV EF $< 45\%$, & not comatosed
- Randomized to SOC or impella CP w/in 12 hrs of PCI, run at highest P-level for 48 hours
- Median time from symptom onset to randomization was 4.8 hrs
- Median duration of support 2.5 days
- 16% vs 21% escalated to other MCS



Moller et al. EHJ. 2024;390.



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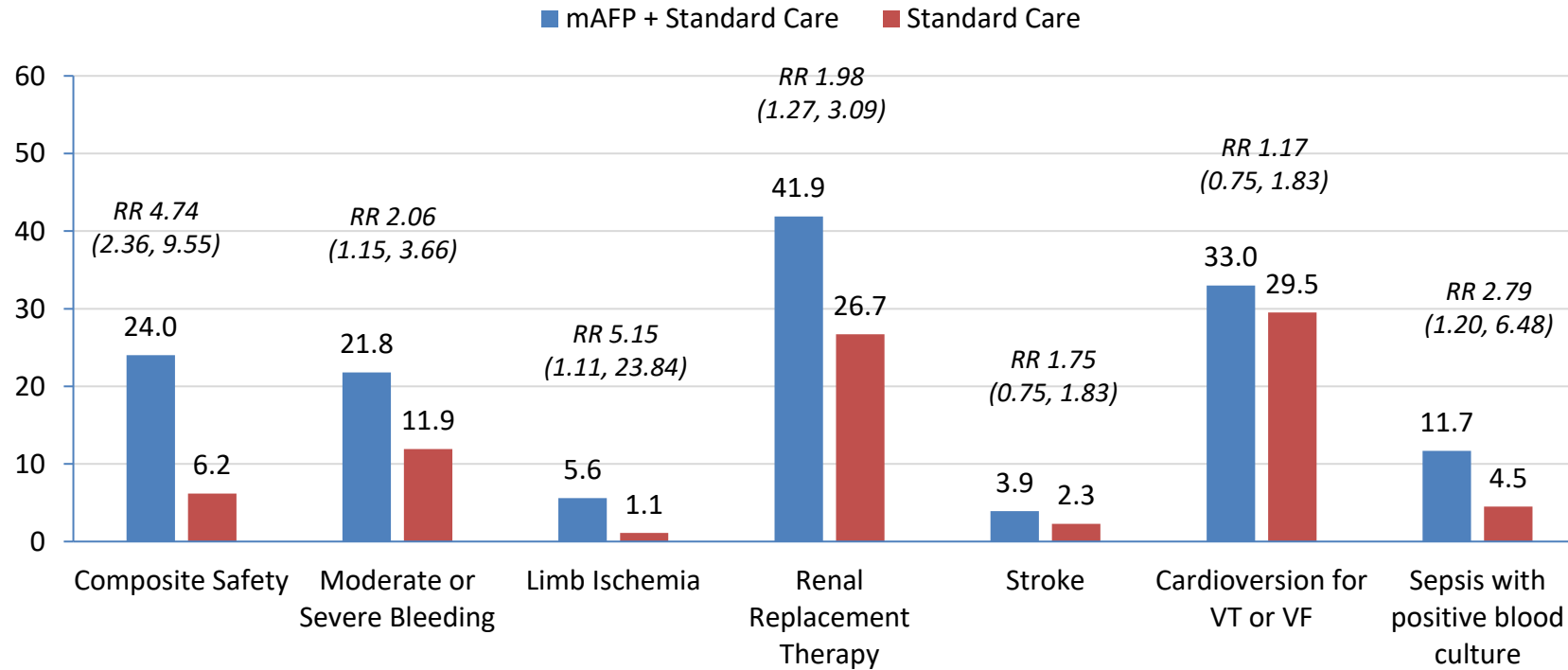


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DanGer Shock



Moller et al. EHJ. 2024;390.



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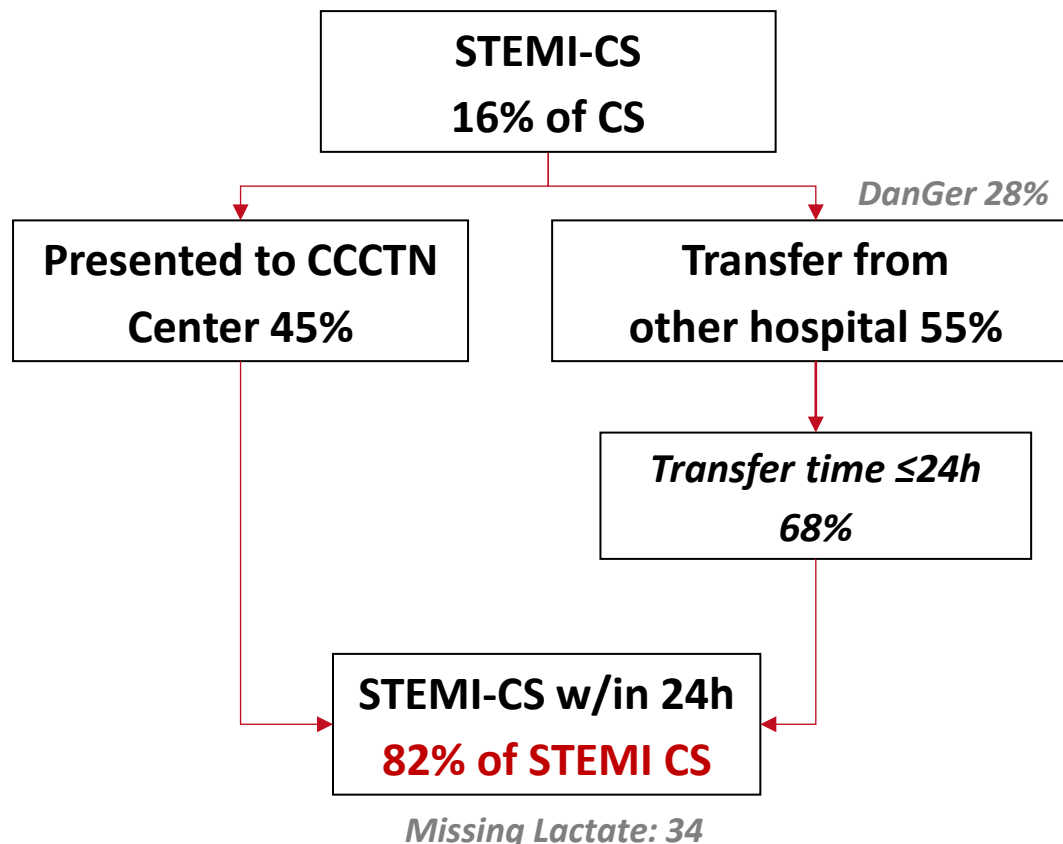
UNDERSTANDING APPLICATION OF DATA TO CLINICAL PRACTICE



All cardiogenic shock
4842

AMI-Shock
24% of CS

STEMI-CS
16% of CS



Presenting Lactate ≥ 2.5 mmol/L
73% of STEMI-CS w/ lactate

LVEF $< 50\%$
86% STEMI-CS w/lactate > 2.5

Trial Exclusion Criteria (-34%)

- VSD: 10
- OHCA & TTM: 86
- RV failure: 12
- Severe Valve Dysf: 12
- Severe dementia: 10

DanGer Eligible
32% of STEMI-CS
5% of all CS admissions

Practical Guidance for Management of CS



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General Principles of CS Management

- Stabilize HD and improve end-organ perfusion via optimization of MAP and CO, while minimizing risk of pulmonary edema and systemic venous congestion
- Allow for time to treat underlying driver(s) of shock (ACS, arrhythmia, sepsis, etc.) and facilitate cardiac recovery.



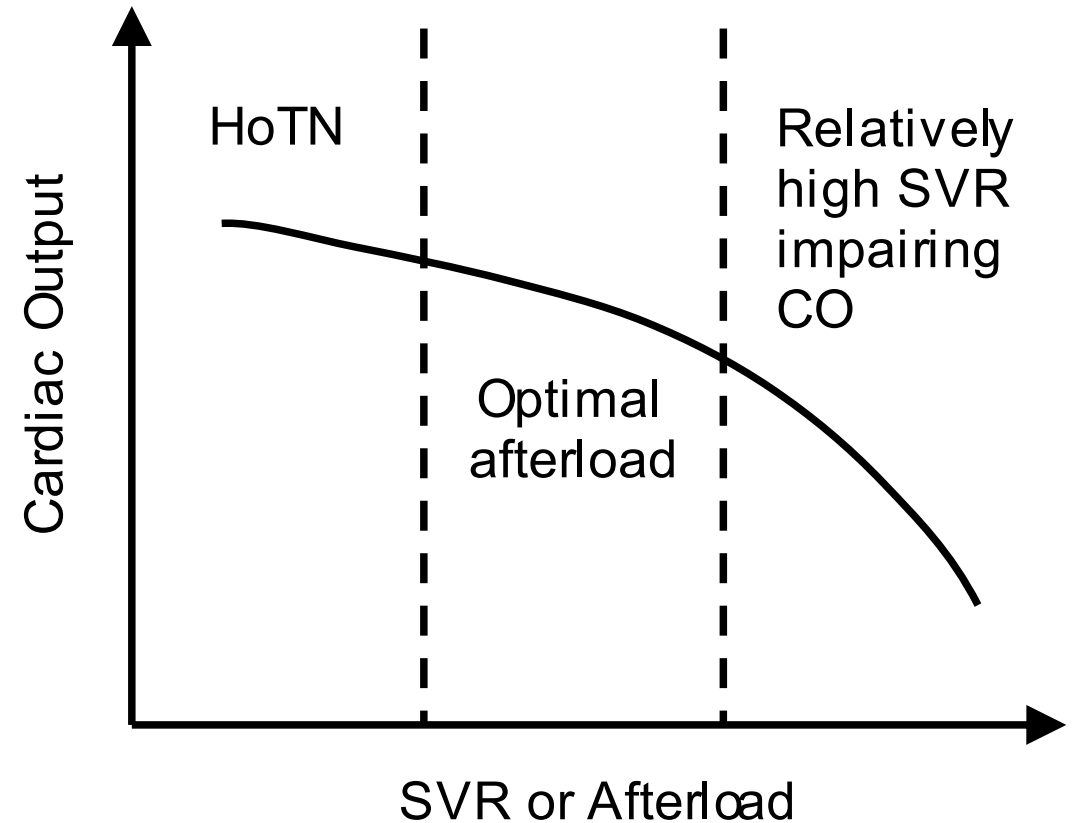
Step-Wise Approach to CS Management

- Initially must correct hypotension and MAP (goal ≥ 65 mmHg), typically with inopressor
- Assess degree of congestion (preload) & adequacy of perfusion (CO)
- Assess and treat reversible causes of cardiogenic shock:
 - Acute ischemia
 - Address other potential contributors: dysrhythmias, acid/base disturbances, negative inotropes (bB, CCB) and antihypertensives
- Optimize hemodynamics, **often with PAC to guide therapy**



Optimizing Hemodynamics

- **Contractility: Goal CI ≥ 2.2**
- **Afterload**
 - Minimize excess afterload to maximize SV while maintaining adequate perfusion pressure.
 - ***Typical goals: SVR ~800-1000, SBP 65-75***



My Strategy for Vasoactives in CS

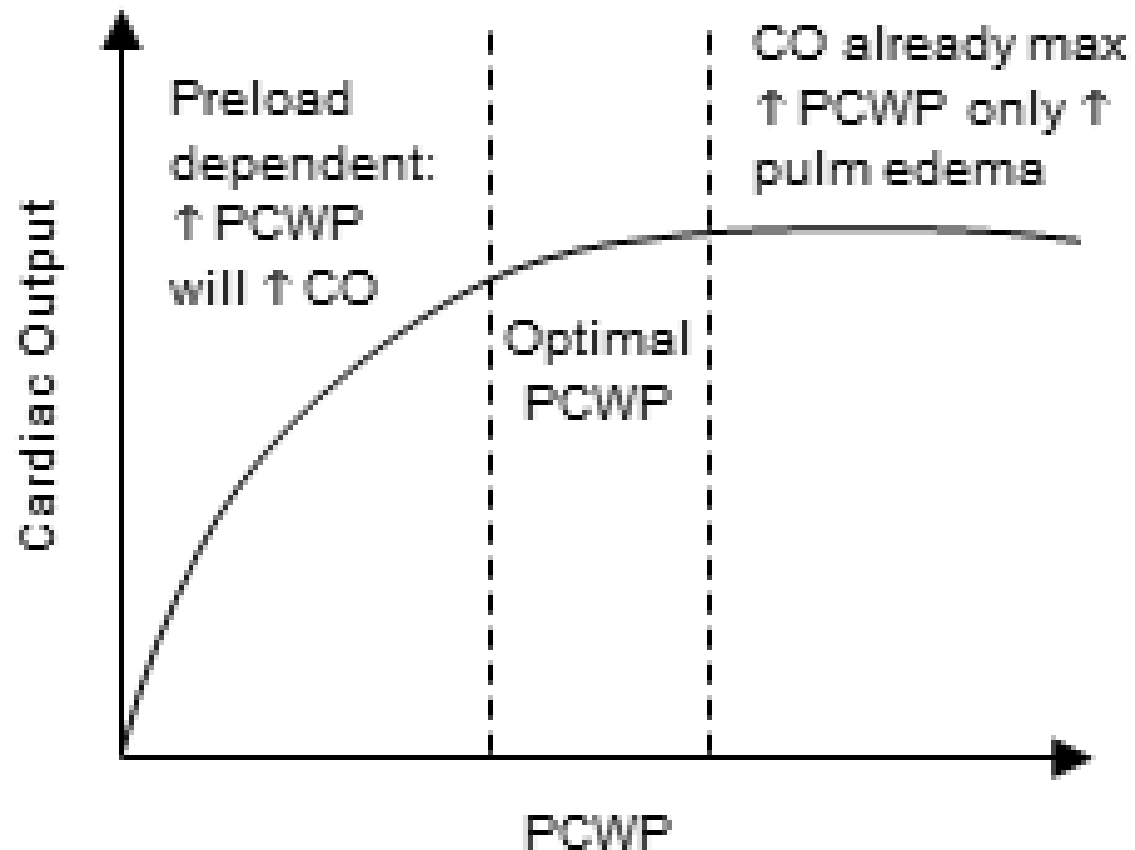
Phenotype	Vasoactive Choice
Classic CS with hypotension (low CO, high SVR, low MAP)	Inopressor (NE/epi/DA) to maintain MAP, then inodilator
Vasodilatory CS with hypotension (low CO, low SVR, low MAP)	Inopressor (NE/epi/DA) +/- vasopressin to maintain MAP, then inodilator if BP room for better CO
Normotensive cardiogenic shock (low CO, high SVR, nl MAP)	Inodilator and/or pure vasodilator



Optimizing Hemodynamics

- **Preload**

- Target a preload that makes CO near maximal (ie, flat part of Starling curve) while avoiding pulmonary edema, hepatic & renal congestion
- **Typical goal PCWP <15-20** but ↑ levels may be tolerated in chronic HF
- Prefer diuretic gtt to bolus



Other Things to Consider

- When to escalate beyond medical therapy
 - Aggressive up-front trial of medical therapy
 - Simultaneously collect data (eg place PAC at bedside or in cath lab)
 - If refractory/declining, consider rapid escalation to MCS
- ADHF involvement
 - Are there advanced options (eg durable VAD, OHT)?
- Shock team involvement
 - CICU, iCards, ADHF, CSS, ECMO

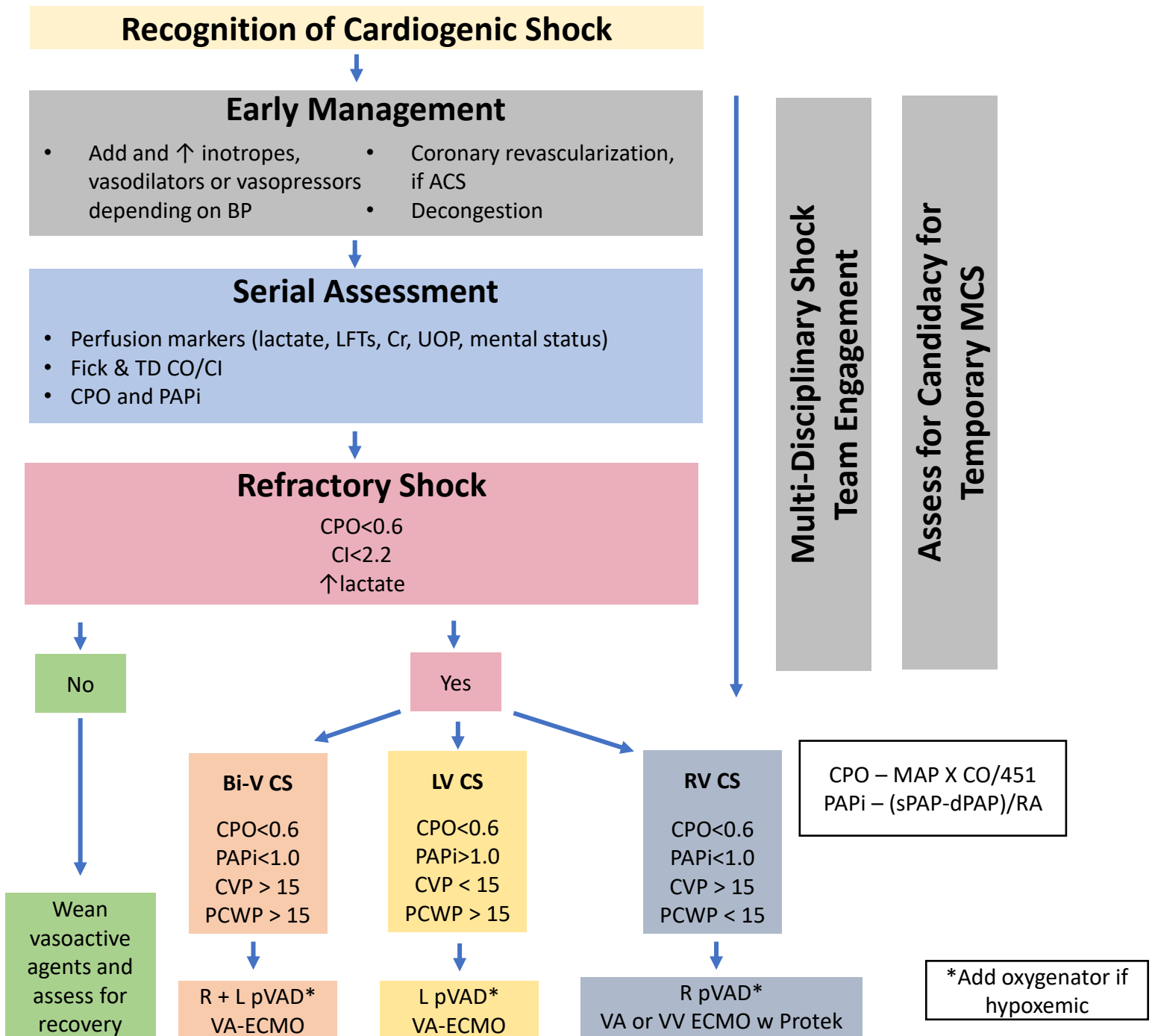


Considerations When Choosing MCS

- Physiology
 - Ventricular involvement (left vs right vs biventricular) 
- Shock severity → degree of CO support needed 
- Requirement for respiratory support
- Duration of support and desire for ambulation
- Technical & Logistical Aspects
 - Adequate access (eg, PAD)
 - Specific contraindications (eg, LV thrombus, mechanical AVR)
 - Operator availability (eg, iCards, surgery)
- Additional considerations 
 - AMI-CS vs acute decompensated HF-CS
 - Cardiogenic ($\uparrow$ SVR) vs mixed ($\downarrow$ SVR)

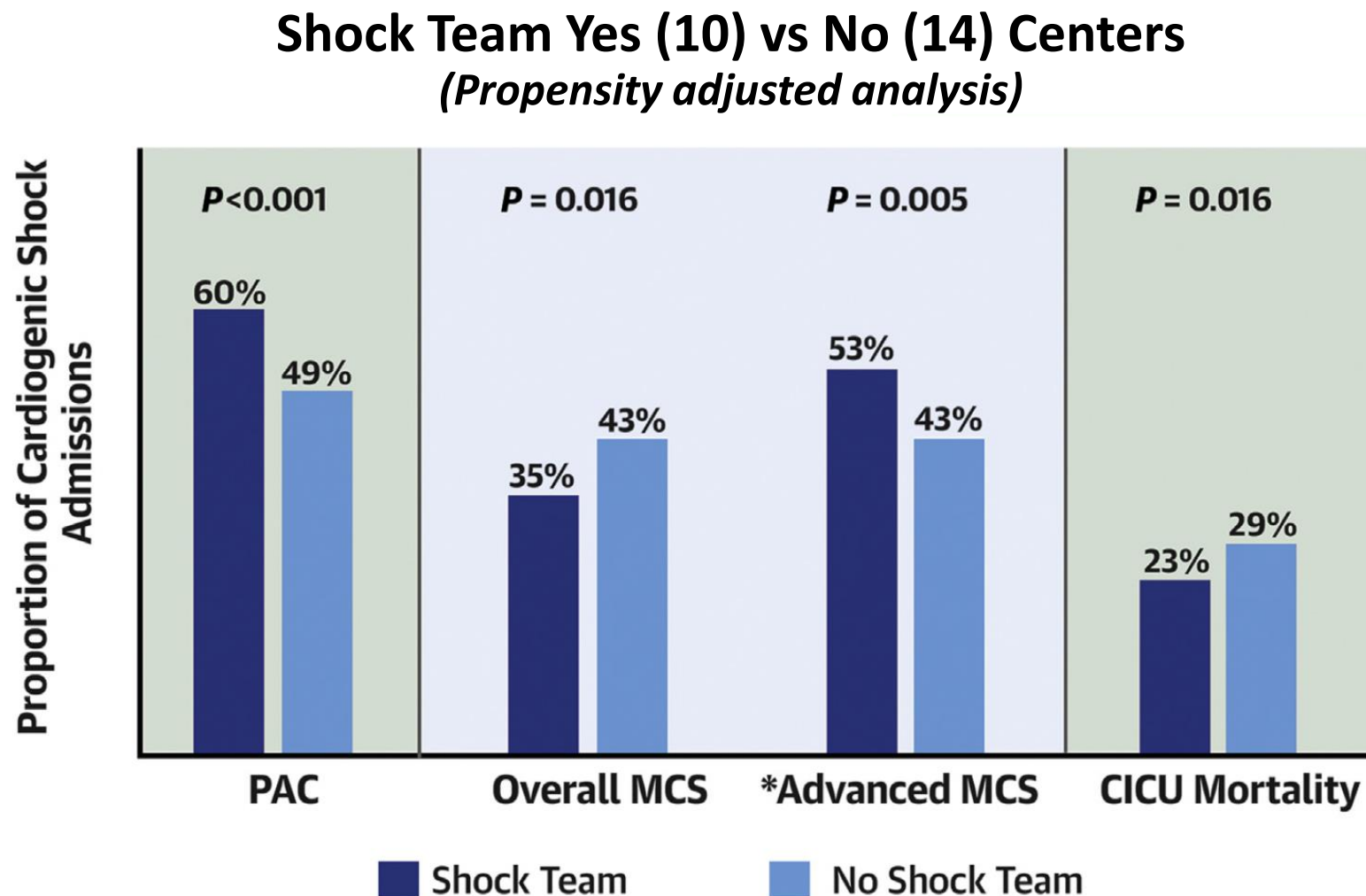


Protocol-Based Approach



Shock Team: Multicenter Observational Study

Shock Team vs No Shock Team Center Population Characteristics	
Cardiogenic shock admissions (n)	546 vs 696
AMI-CS (%)	27 vs 28
Admission lactate (mmol/L)	2.3 vs 2.3
PCWP (mm Hg)	25 vs 22
CI (L/min/m ²)	1.9 vs 2.0
CPO (W)	0.62 vs 0.64



Case Continuation

- Remote/virtual Dx: Normotensive cardiogenic shock
 - Initial treatment: Dobutamine initiation, diuresis, transfer
- Additional Studies:
 - RHC with elevated BiV filling pressures, low CO (RA 26, PCWP 25, SVR 1800, CI 1.5, PAPi 0.8, CPO 0.5).
 - Coronary angio w/o coronary disease. No EMBx done.
 - Added milrinone
 - CO, SVR, filling pressures improved. Cr, lactate and LFTs normalized
- Refined diagnosis
 - De novo HF-CS, SCAI Stage E -> D with Biventricular failure
- Course: Unable to wean off dual inotropes → advanced therapies (R tandem, surgical LVAD (HM3) with plan for bridge to OHT



Take Aways

- Highly mortal condition with significant heterogeneity
- Phenotyping is helpful to understand prognosis (e.g., SCAI Stage, ventricular involvement, etc)
- Early recognition is critical to minimize multi-organ system failure and death
- Team-based approach is optimal



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