Initial Evaluation and Management of Cardiogenic Shock

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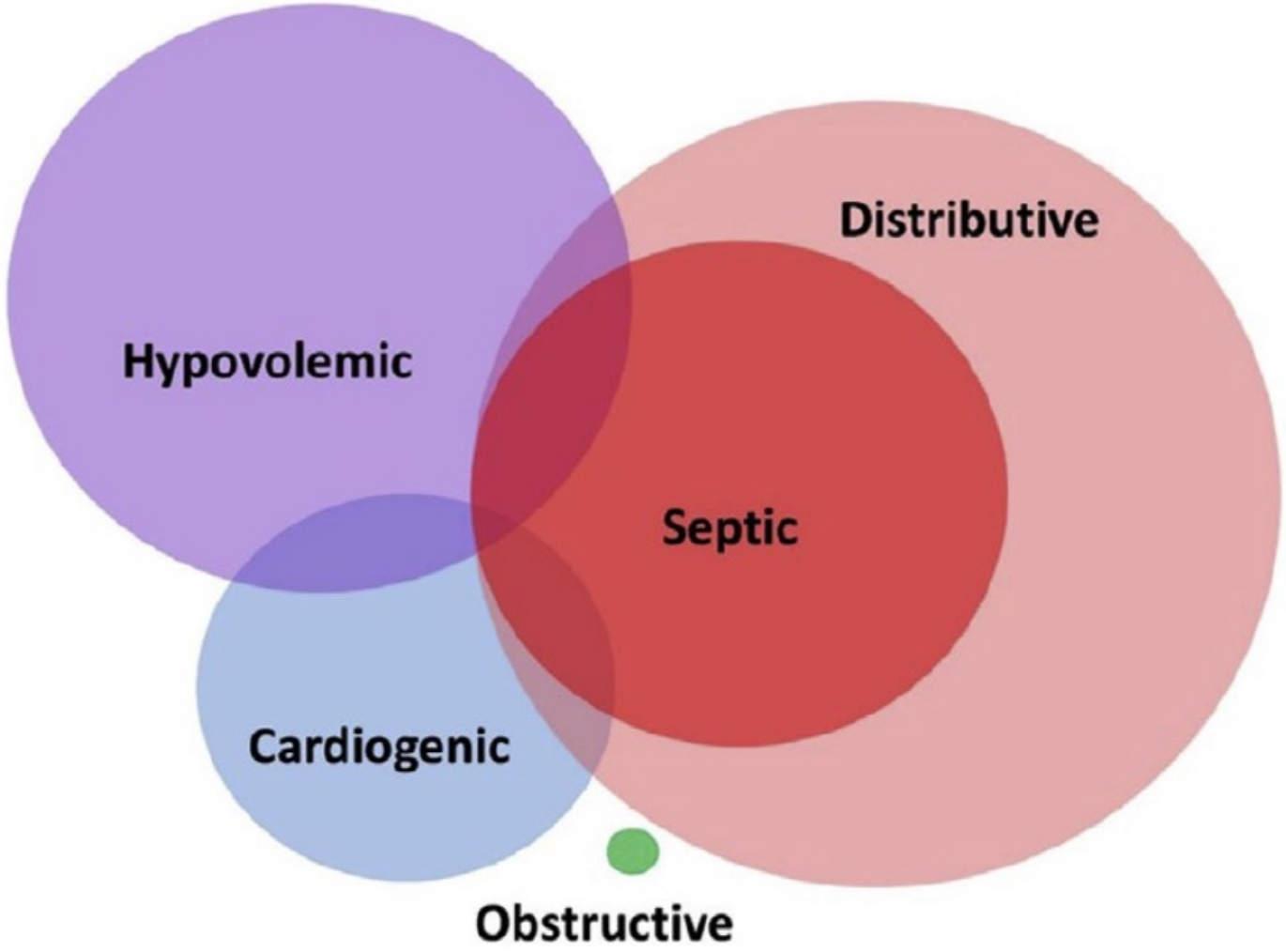
Disclosures: None

Objectives of Talk

- Understand the epidemiology of Cardiogenic Shock
- Understand the assessment of Cardiogenic Shock
- Discuss the current treatment options of Cardiogenic Shock
- Future Directions for management of Cardiogenic Shock

Heart Failure patients can develop any kind of Shock



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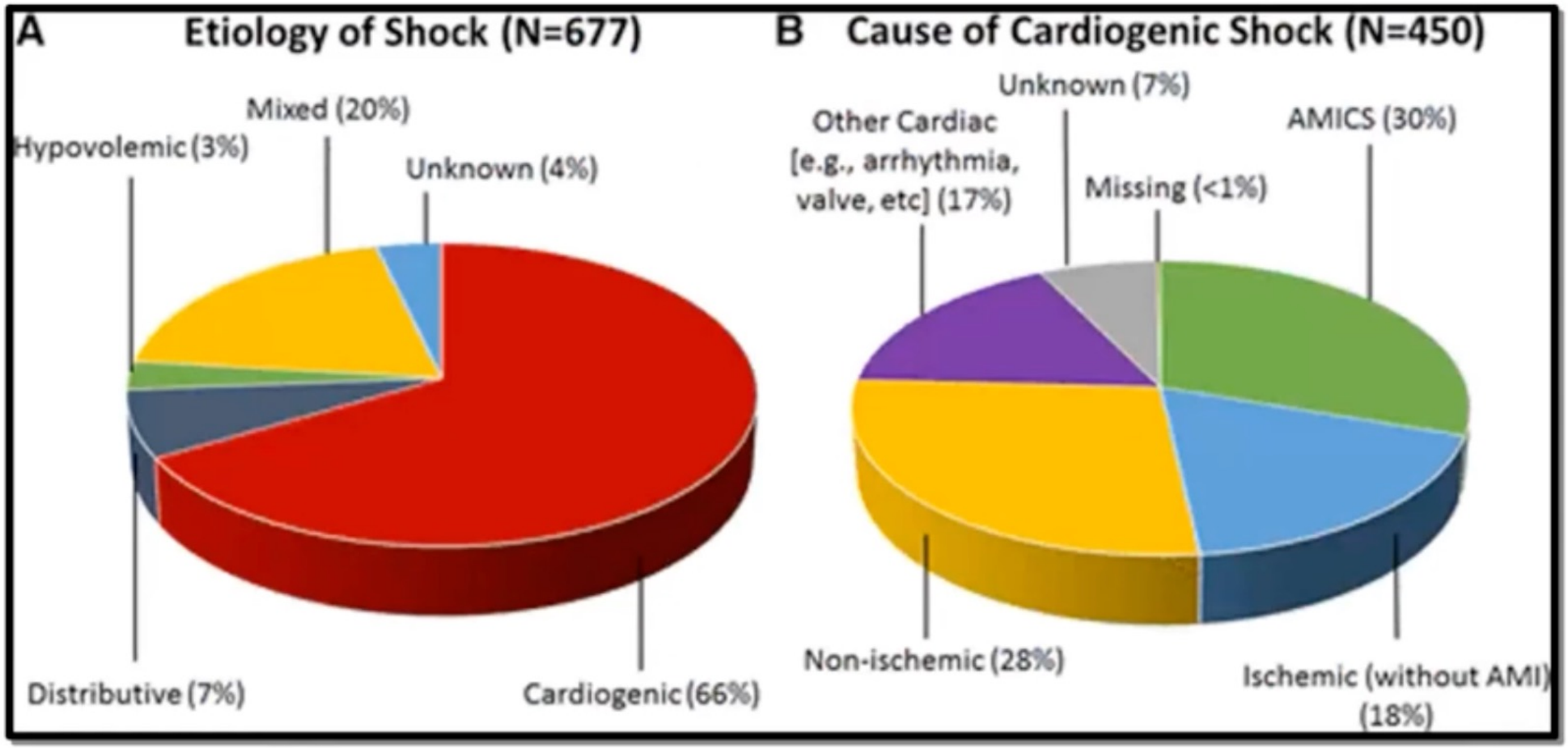

Tehrani, Rosner, Sinha JACC HF 2019
Berg et al Circulation Cardiovascular Quality & Outcomes 2019

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Definition of Cardiogenic Shock

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

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Van Diepen, S., et al. *Circulation 2017*

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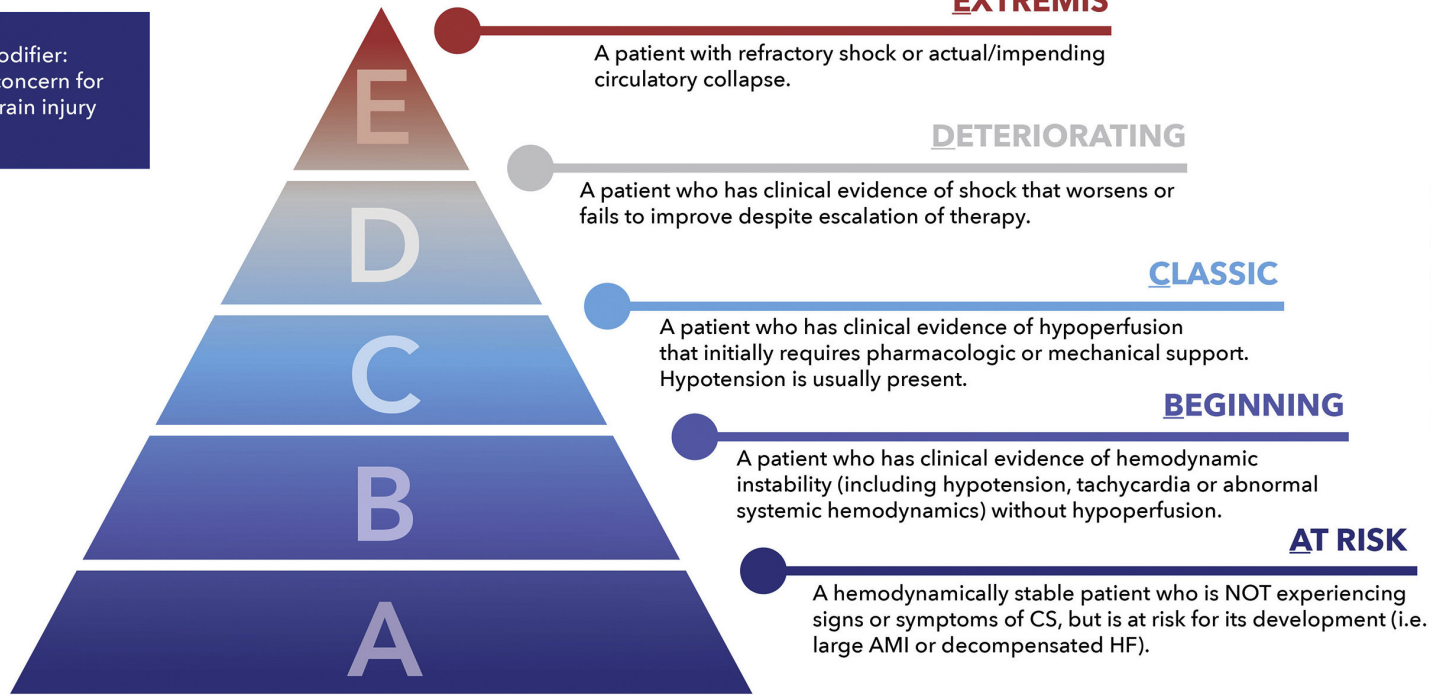
SCAI Clinical Definitions of Cardiogenic Shock

(A) Modifier:
CA with concern for anoxic brain injury



EXTREMIS

A patient with refractory shock or actual/impending circulatory collapse.

DETERIORATING

A patient who has clinical evidence of shock that worsens or fails to improve despite escalation of therapy.

CLASSIC

A patient who has clinical evidence of hypoperfusion that initially requires pharmacologic or mechanical support. Hypotension is usually present.

BEGINNING

A patient who has clinical evidence of hemodynamic instability (including hypotension, tachycardia or abnormal systemic hemodynamics) without hypoperfusion.

AT RISK

A hemodynamically stable patient who is NOT experiencing signs or symptoms of CS, but is at risk for its development (i.e. large AMI or decompensated HF).

Stage A At risk	Stage B Beginning CS	Stage C Classic CS	Stage D Deteriorating	Stage E Extremis
• SBP >100 • CI ≥2.5 • RAP <10 • PA Sat ≥65% • Normal renal function + lactate	• SBP <90 or MAP <60 (or ≥30 drop from baseline) • Pulse ≥100 • CI ≥2.2 • PA Sat ≥65% • Normal lactate • Mild renal impairment • Elevated BNP	• BP criteria of Stage B + drugs/devices used to maintain BP above target • CI <2.2 • PCWP >15 • RAP/PCWP ≥0.8 • PAPI ≤1.85 • CPO ≤0.6 • Lactate ≥2 • >50% drop in GFR • Increased LFTs • Elevated BNP	• Any of stage C AND • Multiple pressors OR • Mechanical circulatory support device • Lactate rising and persistently >2 • Deteriorating renal function • Worsening LFTs • Rising BNP	• No SBP without resuscitation • PEA or refractory VT/VF • Hypotension with maximum support • pH ≤7.2 • Lactate ≥8 • Base deficit >10

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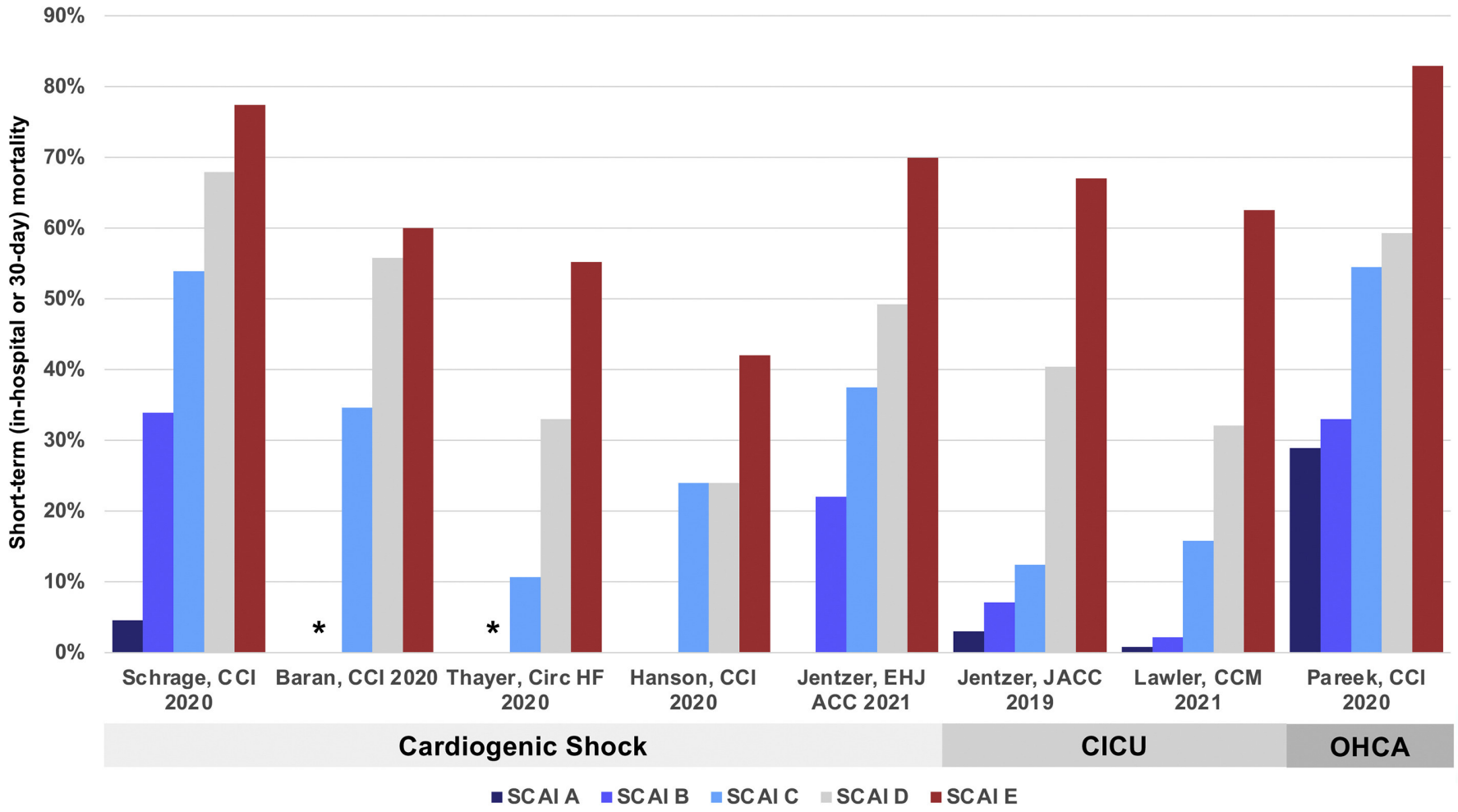
SCAI Clinical Evaluation of Cardiogenic Shock



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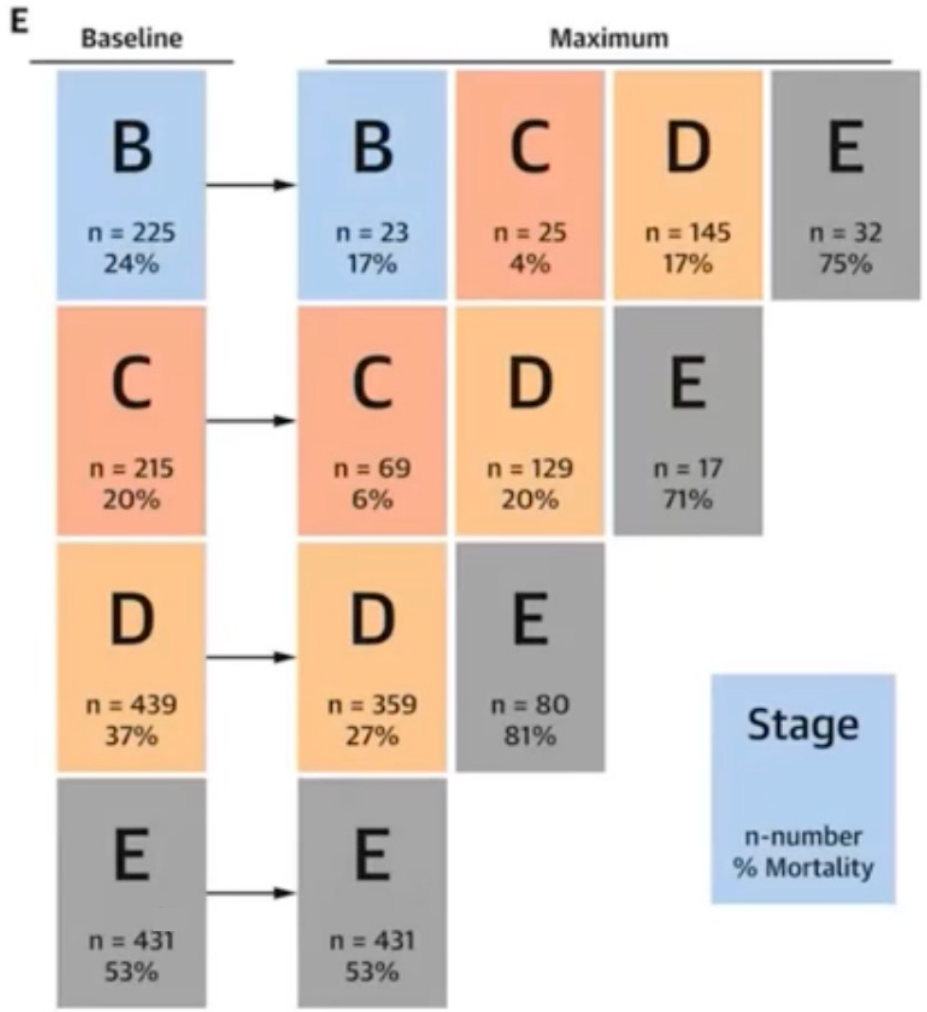
Clinical Course Predicts Mortality



Kapur, Kanwar, Sinha et al JACC 2022

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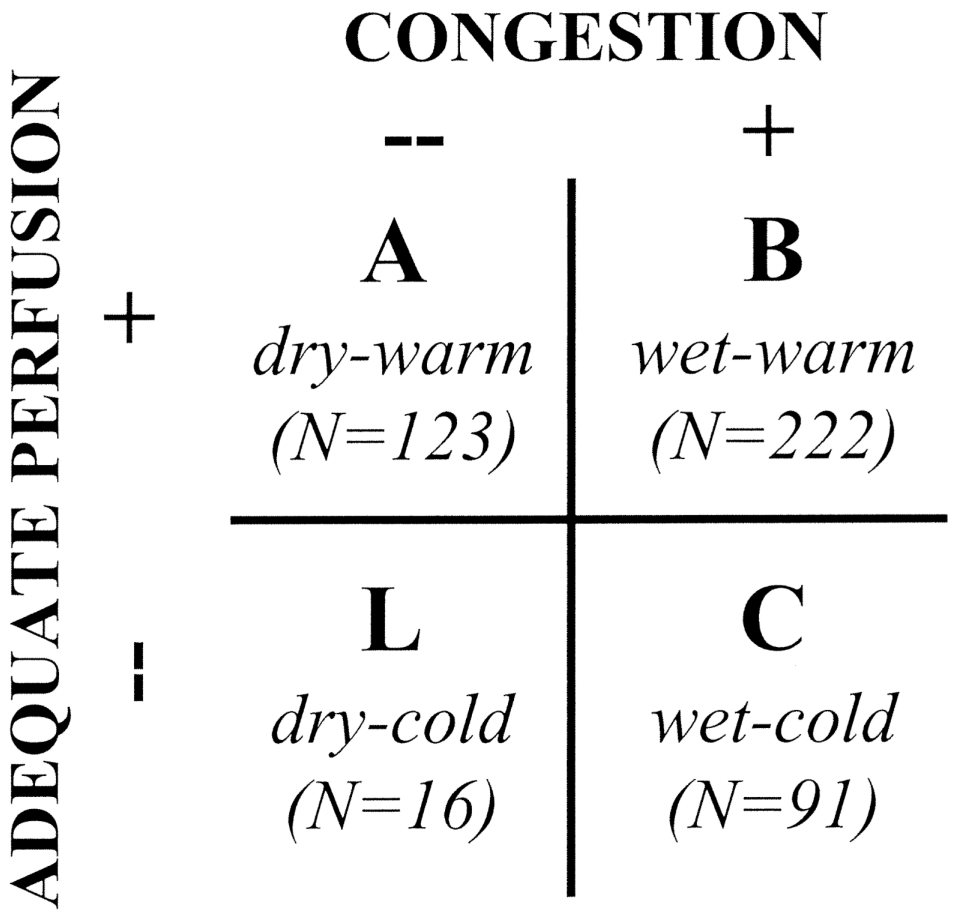
Etiologies of Cardiogenic Shock

- Acute Myocardial Infarction and/or mechanical complications
- Decompensation of Advanced Heart Failure
- Acute Myocarditis
- Post Cardiotomy Shock
- Hypertrophic Cardiomyopathy with obstruction
- Pulmonary Embolism
- Electrical Instability
- Tamponade
- PPCM
- Acute Valve Disease

Physical Exam



Diuretics

Diuretics + Inotrope / Vasodilator

Nohria, A et al. JACC 2003

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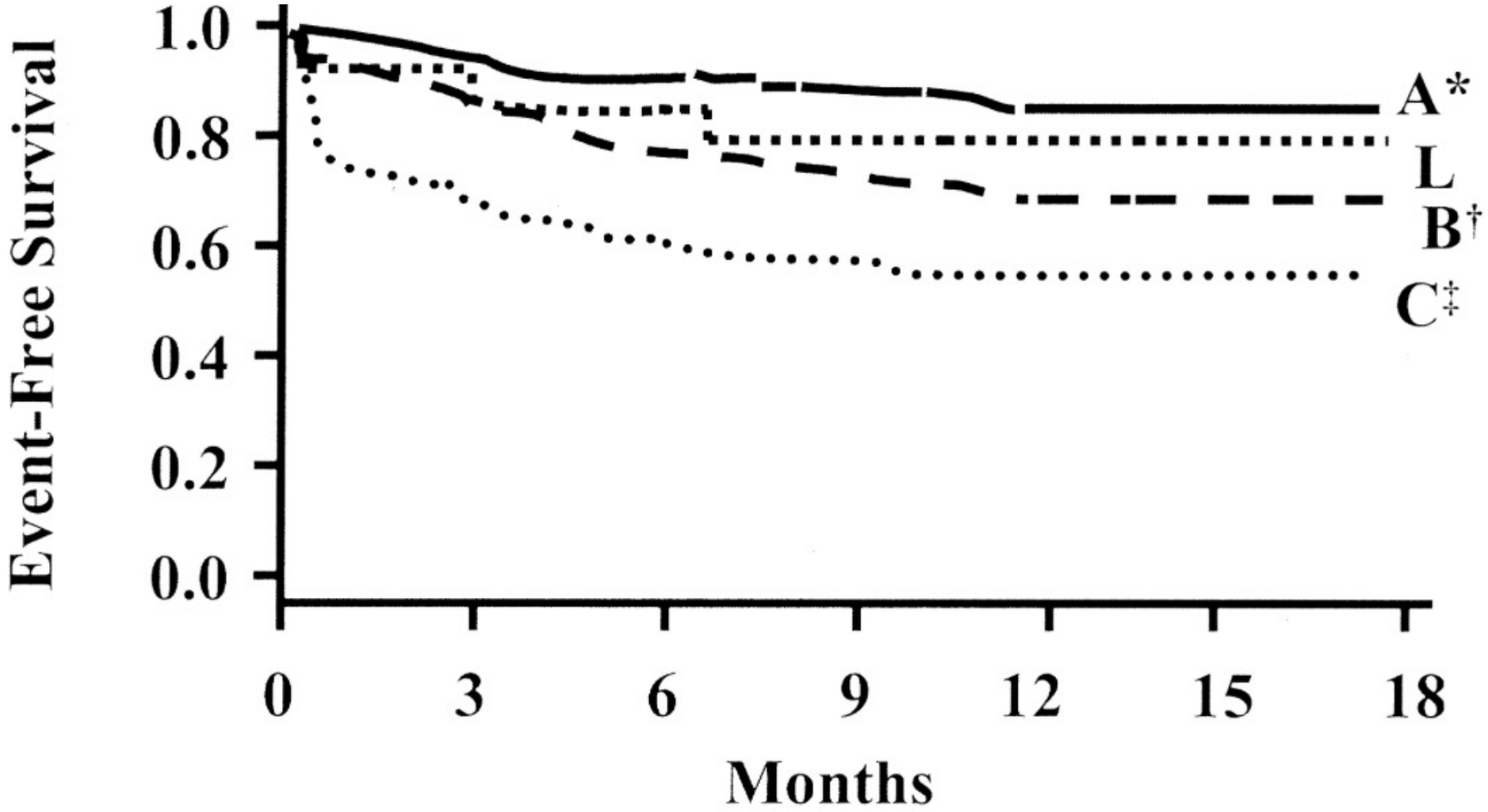

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Nohria, A et al. JACC 2003

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Value of Clinician Assessment of Hemodynamics in Advanced Heart Failure

Estimated Cardiac Index	N	Measured Cardiac Index, L/(min·m^2)
<1.8	32	1.85 (1.50, 2.25)
1.8–2.2	102	1.90 (1.60, 2.20)
>2.3	55	2.00 (1.70, 2.40)

Clinical finding	Sensitivity (%)
Cool extremities	20
Proportional pulse pressure < 25%	10
Overall assessment of “cold” profile	33

If they are cold, they are cold; but if they are warm, they may still be cold.

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Drazner, M. H., et al. Circulation HF 2008.

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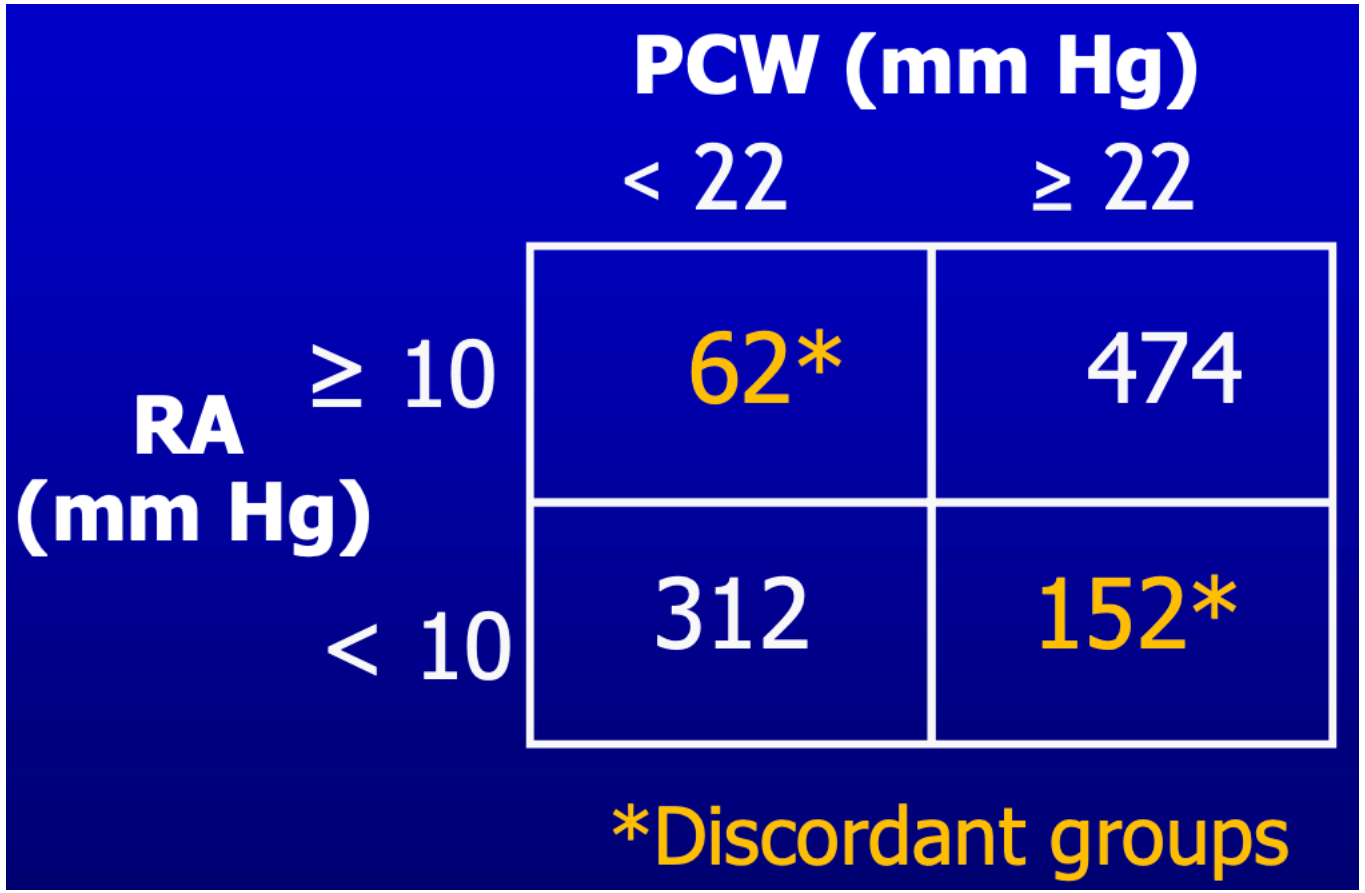
RA and PCWP are not always concordant in Advanced Heart Failure



Concordant	Discordant
79%	21%

Drazner, M. H., et al. JHLT 1999

Laboratory Evaluation

- Troponin, ABG w/ lactate, NT-proBNP, CMP, coagulation studies, etc…
- CXR, TTE, ECG, TEE, CTA, LHC, etc…

2022 ACC/AHA/HFSA Guidelines on the Management of Cardiogenic Shock

COR	LOE	RECOMMENDATIONS
1	B-NR	1. In patients with cardiogenic shock, intravenous inotropic support should be used to maintain systemic perfusion and preserve end-organ performance (1-8).
2a	B-NR	2. In patients with cardiogenic shock, temporary MCS is reasonable when end-organ function cannot be maintained by pharmacologic means to support cardiac function (9-17).
2a	B-NR	3. In patients with cardiogenic shock, management by a multidisciplinary team experienced in shock is reasonable (17-22).
2b	B-NR	4. In patients presenting with cardiogenic shock, placement of a PA line may be considered to define hemodynamic subsets and appropriate management strategies (23-27).
2b	C-LD	5. For patients who are not rapidly responding to initial shock measures, triage to centers that can provide temporary MCS may be considered to optimize management (17-22).

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Heidenreich P et al. JACC 2022

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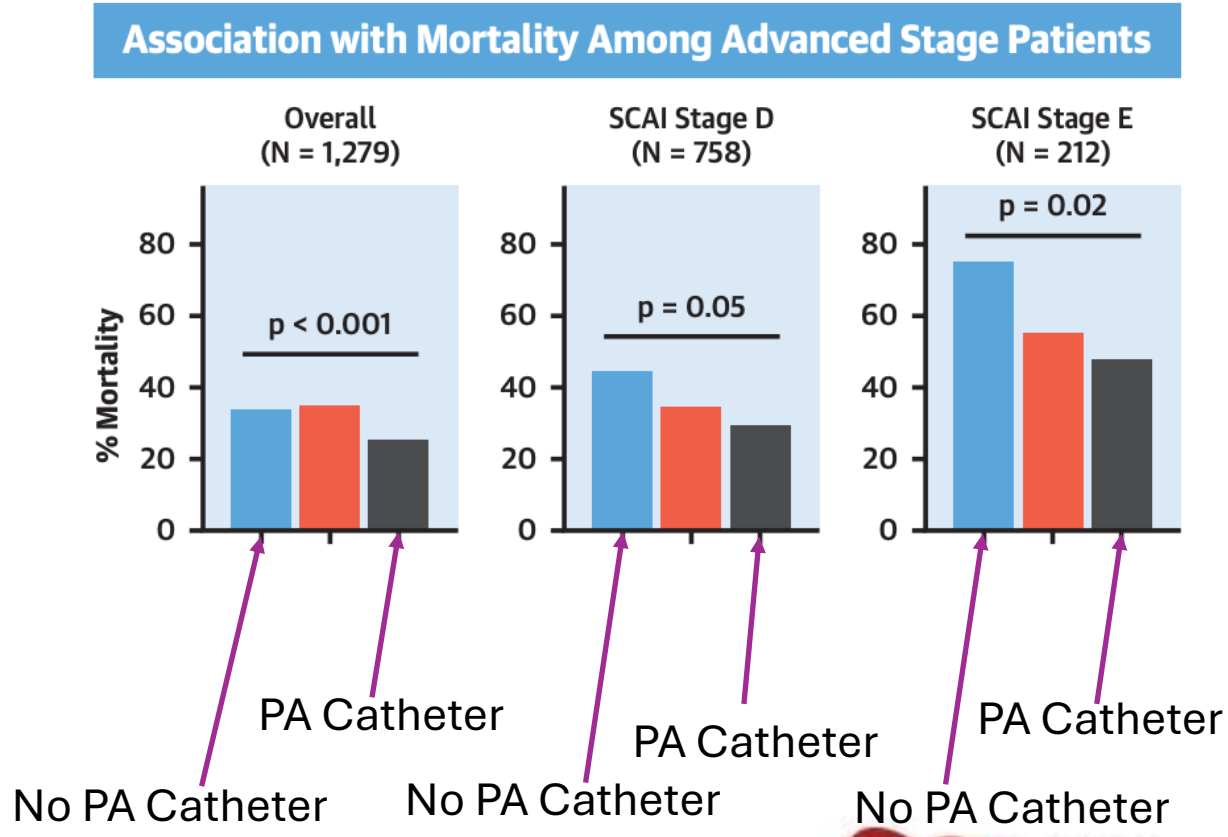
Initial Stabilization

- Includes IVF, oxygenation, mechanical ventilation and vasopressor/inotrope use

Medication	Classification	Clinical Uses in CS
Norepinephrine	Vasoconstrictor Mild Inotrope	First-line vasopressor for CS in ED
Phenylephrine	Vasoconstrictor	Increased afterload (syst & pulm), generally not used in CS Useful to decrease LVOT gradients
Vasopressin	Vasoconstrictor	Increased afterload of systemic but not pulm circulation Useful in isolated RV disease or vasoplegia
Epinephrine	Inoconstrictor	Arrhythmia, recommended for symptomatic bradycardia or refractory hypotension
Dopamine	Inoconstrictor	Arrhythmia, inferior to norepi in trial, use reserved for symptomatic bradycardia
Dobutamine	Inodilator	First-line inotrope for CS in ED Fast acting, easily titratable
Milrinone	Inodilator	Slower acting and longer half-life than dobutamine, renally cleared, can accumulate, less arrhythmogenic, more pulmonary vasodilation

Use of Pulmonary Artery Catheters in Cardiogenic shock

- Initial trials performed 20 years ago suggested limited benefit of PA catheters in the management of cardiogenic shock[1].
- Further studies have shown that refined use of PA catheters is associated with improved mortality[2].



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1. Binanay, Cynthia, et al. JAMA 2005
2. Garan, A.R. et al. JACC HF. 2020

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Hemodynamic Profiling via RHC

Metric	Calculation	Markers of cardiogenic shock
Cardiac index (CI)	CO/body surface area	≤2.2 L/min/m^2
Cardiac power output (CPO)	(MAP x CO)/451	<0.6 W
Cardiac power index (CPI)	(MAP x CI)/451	<0.4 W/m^2
Pulse pressure	systolic – diastolic blood pressure	<25 mmHg
Systemic vascular resistance (SVR)	[(MAP – CVP) / CO] x 80	variable

Right Ventricular Metrics	Calculation	Markers of RV dysfunction
Right atrial pressure (RAP)		>10/15 mmHg
Right atrial pressure (RAP) / Pulmonary capillary wedge pressure (PCWP)		>0.86 (in acute MI) >0.63 (after LVAD)
Pulmonary artery pulsatility index (PAPi)	(PASP-PADP) / RAP	≤0.9 (in acute MI) <1.85 (after LVAD)
Right ventricular stroke work index (RVSWI)	0.0136 x SVi x (mPAP–RAP)	<6 g/m/beat/m^2

Pulmonary Vascular Metrics	Calculation	Markers of pulmonary vascular disease
Transpulmonary pressure gradient (TPG)	mPAP-PCWP	≥12 mmHg
Diastolic pulmonary gradient (DPG)	PADP-PCWP	≥7 mmHg

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Hemodynamic and Physiologic Treatment Goals

TABLE 1 Perfusion Targets From Highlighted Guidelines and Consensus Statements

Perfusion Targets	2017 ACC/AHA Scientific Statement on CS[8]	2022 AHA/ACC Guideline for the Management of Heart Failure[a]	Management of CS Complicated MI: An Update 2019[7]	2021 ESC Guidelines for Diagnosis and Treatment of Acute and Chronic Heart Failure[b]
Hemodynamic targets	No clear sBP or MAP recommendations	No clear sBP or MAP recommendations	No clear sBP or MAP recommendations. Suggest that MAP >65 mmHg probably not required	No clear sBP or MAP recommendations. In AHF with sBP >110 mmHg, IV vasodilators may be considered as initial therapy to improve symptoms and reduce congestion (Class IIb)
Physical exam targets	Use cold/warm and wet/dry descriptors to highlight hemodynamic phenotypes. Longitudinal CVP trends may provide information on trends in fluid status	Severity of congestion and adequacy of perfusion should be assessed to guide triage and initial therapy (Class I)	Not specified	Use wet/dry and warm/cold, as well as mental confusion, dizziness, and narrow pulse pressure. Emphasize that hypoperfusion is not always accompanied by hypotension
Renal targets	Suggest serial monitoring of urine output and creatinine. Include KDIGO guidelines that CRRT be considered when "life-threatening changes in fluid, electrolyte, and acid-base balance" exist	Not specified	Suggest serial monitoring of urine output and creatinine RRT initiated with AKI and uremia, refractory volume overload, metabolic acidosis, and/or refractory hyperkalemia (Class IIb)	Suggest serial monitoring of urine output and creatinine
Lactate targets	Suggest serial monitoring of arterial lactate q1-4 h	Not specified	Not specified	Suggest serial monitoring and when peripheral hypoperfusion is suspected
Additional variables for serial monitoring	Suggest using serial perfusion markers including SvO_2 or $ScvO_2$ LFTs, mental status, and other invasive hemodynamic variables	Not specified	Not specified	NT-pro-BNP recommended at admission, predischarge
Vasoactive agent selection	Norepinephrine may be vasopressor of choice as associated with fewer arrhythmias Note that optimal first-line vasoactive medication in CS remains unclear Provides pragmatic considerations based on etiology and phenotype of shock	In patients with CS, intravenous inotrope support should be used to maintain systemic perfusion and preserve end-organ performance (Class I) Choice of inotrope guided by blood pressure, concurrent arrhythmias, and availability	Norepinephrine is vasoconstrictor of choice when low BP and insufficient tissue perfusion pressure (Class IIb) Inotropes (ie, dobutamine) may be given simultaneously to norepinephrine to improve cardiac contractility (Class IIb)	Consider inotropes and/or vasopressors for sBP <90 mmHg and hypoperfusion who do not respond to standard treatment, including fluid challenge to improve peripheral perfusion and maintain end-organ function (Class IIb) Inotropic agents not recommended routinely, due to safety concerns, unless patient has symptomatic hypotension and evidence of hypoperfusion (Class III) Vasopressor therapy, preferably norepinephrine, may be considered in patients with CS to increase BP and vital organ perfusion (Class IIb) Consider RRT for persistent hypoperfusion and organ dysfunction (Class IIa)

Mathew R, et al. JACC Adv. 2022

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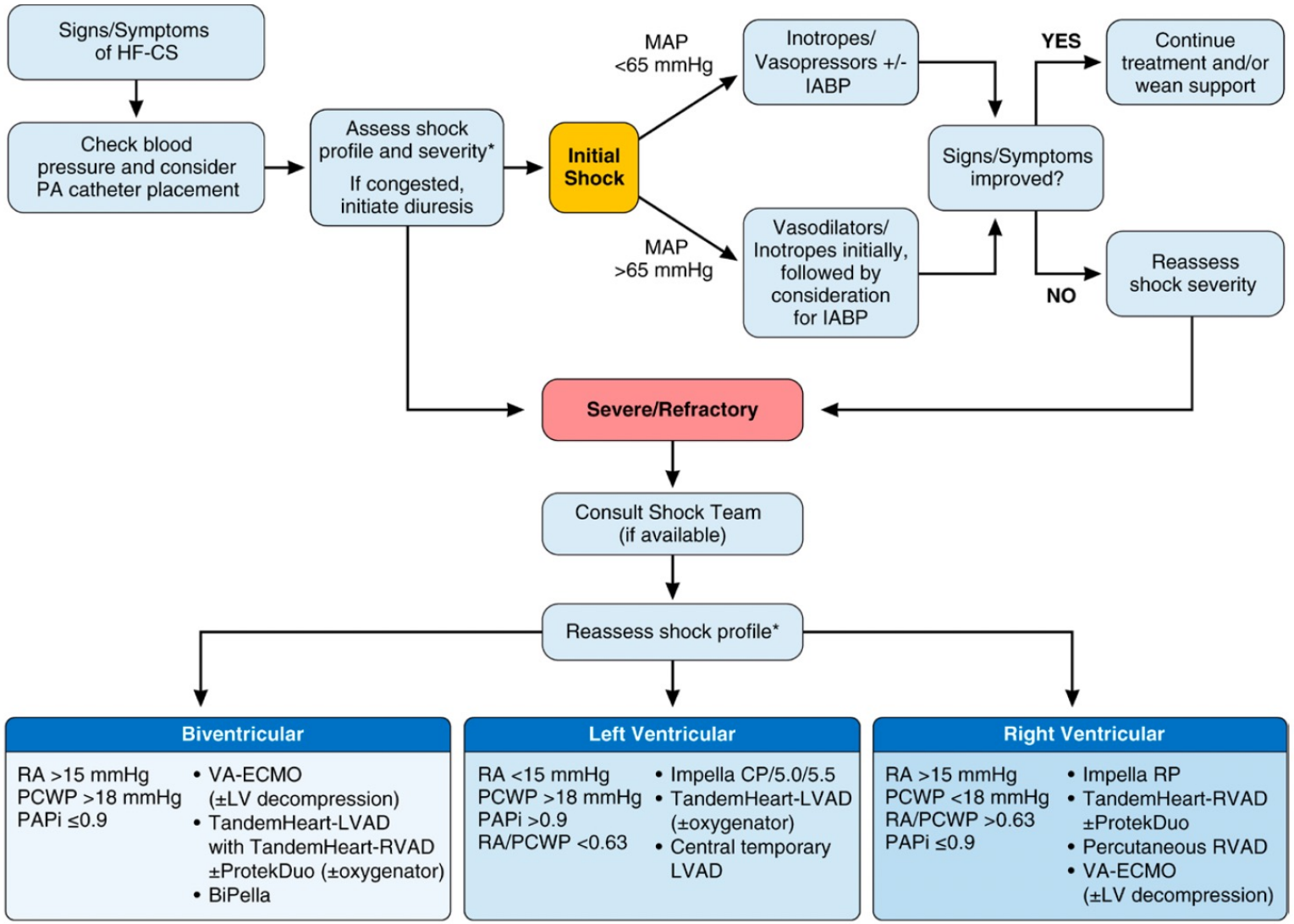
Cardiogenic Shock Treatment Algorithm



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Geller B, et al. Circulation 2022

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DanGer Shock Trial

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

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

J.E. Møller, T. Engstrøm, L.O. Jensen, H. Eiskjær, N. Mangner, A. Polzin, P.C. Schulze, C. Skurk, P. Nordbeck, P. Clemmensen, V. Panoulas, S. Zimmer, A. Schäfer, N. Werner, M. Frydland, L. Holmvang, J. Kjærgaard, R. Sørensen, J. Lønborg, M.G. Lindholm, N.L.J. Udesen, A. Junker, H. Schmidt, C.J. Terkelsen, S. Christensen, E.H. Christiansen, A. Linke, F.J. Woitek, R. Westenfeld, S. Möbius-Winkler, K. Wachtell, H.B. Ravn, J.F. Lassen, S. Boesgaard, O. Gerke, and C. Hassager, for the DanGer Shock Investigators*

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Moller JE et al. NEJM 2024

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DanGer Shock Trial

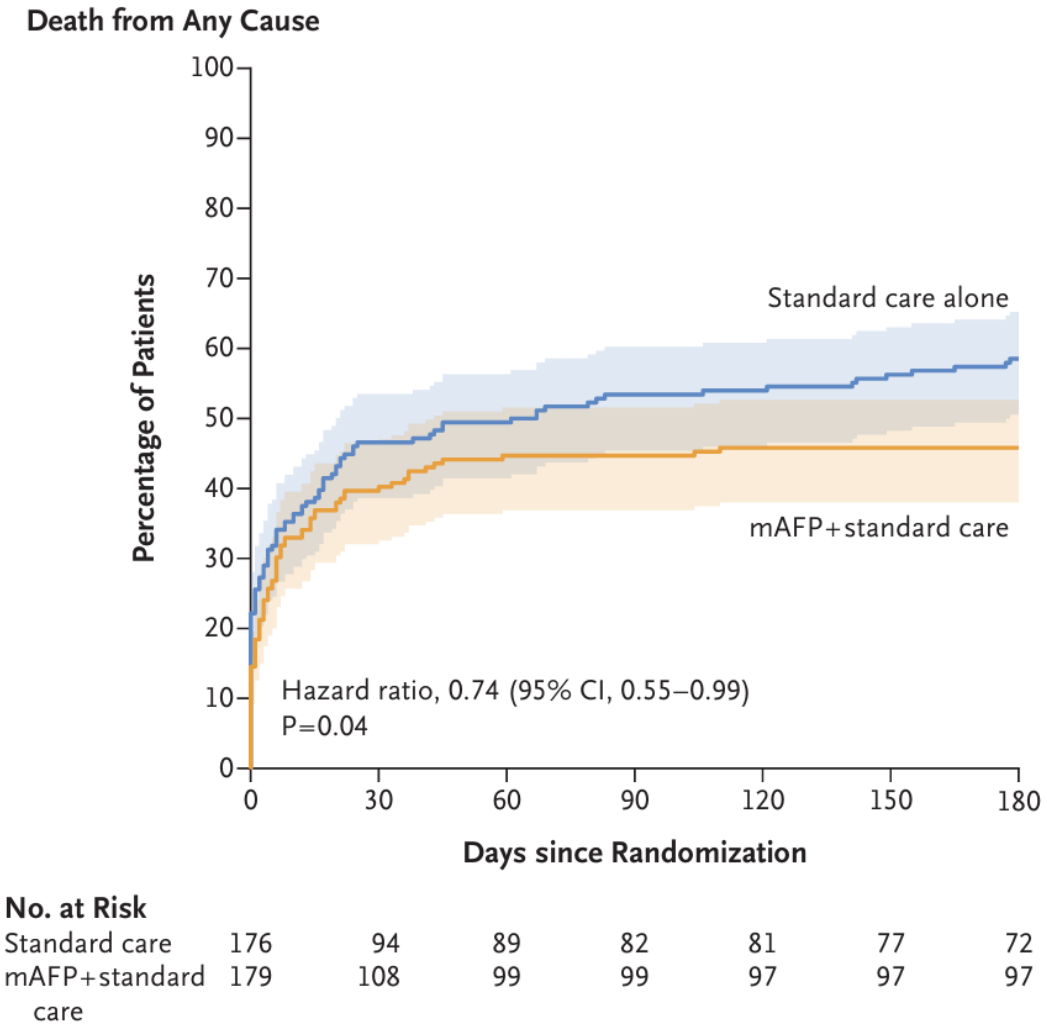

Table 3. End Points and Adverse Events in the Intention-to-Treat Population.*

Event	Microaxial Flow Pump plus Standard Care (N=179)	Standard Care Alone (N=176)	Effect Size (95% CI)†
Primary end point: death from any cause at 180 days — no. (%)	82 (45.8)	103 (58.5)	0.74 (0.55 to 0.99)‡
Secondary end point			
Composite cardiac end point — no. (%)§	94 (52.5)	112 (63.6)	0.72 (0.55 to 0.95)
No. of days alive and out of the hospital (range)¶	82 (0 to 177)	73 (0 to 179)	8 (–8 to 25)
Adverse events			
Composite safety end point — no. (%)‖	43 (24.0)	11 (6.2)	4.74 (2.36 to 9.55)
Moderate or severe bleeding — no. (%)**	39 (21.8)	21 (11.9)	2.06 (1.15 to 3.66)
Limb ischemia — no. (%)	10 (5.6)	2 (1.1)	5.15 (1.11 to 23.84)
Renal-replacement therapy — no. (%)	75 (41.9)	47 (26.7)	1.98 (1.27 to 3.09)
Stroke — no. (%)	7 (3.9)	4 (2.3)	1.75 (0.50 to 6.01)
Cardioversion after ventricular tachycardia or fibrillation — no. (%)	59 (33.0)	52 (29.5)	1.17 (0.75 to 1.83)
Sepsis with positive blood culture†† — no. (%)	21 (11.7)	8 (4.5)	2.79 (1.20 to 6.48)

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Shock Teams

Standardized Team-Based Care for Cardiogenic Shock

Behnam N. Tehrani, MD,[a] Alexander G. Truesdell, MD,[a,b] Matthew W. Sherwood, MD,[a] Shashank Desai, MD,[a] Henry A. Tran, MD,[a] Kelly C. Epps, MD,[a] Ramesh Singh, MD,[a] Mitchell Psotka, MD, PhD,[a] Palak Shah, MD,[a] Lauren B. Cooper, MD,[a] Carolyn Rosner, NP,[a] Anika Raja, BS,[a] Scott D. Barnett, PhD,[a] Patricia Saulino, RN, MPA,[a] Christopher R. deFilippi, MD,[a] Paul A. Gurbel, MD,[a] Charles E. Murphy, MD,[a] Christopher M. O'Connor, MD[a]

ABSTRACT

BACKGROUND Cardiogenic shock (CS) is a multifactorial, hemodynamically complex syndrome associated with high mortality. Despite advances in reperfusion and mechanical circulatory support, management remains highly variable and outcomes poor.

OBJECTIVES This study investigated whether a standardized team-based approach can improve outcomes in CS and whether a risk score can guide clinical decision making.

METHODS A total of 204 consecutive patients with CS were identified. CS etiology, patient demographic characteristics, right heart catheterization, mechanical circulatory support use, and survival were determined. Cardiac power output (CPO) and pulmonary arterial pulsatility index (PAPi) were measured at baseline and 24 h after the CS diagnosis. Thresholds at 24 h for lactate (<3.0 mg/dl), CPO (>0.6 W), and PAPi (>1.0) were determined. Using logistic regression analysis, a validated risk stratification score was developed.

RESULTS Compared with 30-day survival of 47% in 2016, 30-day survival in 2017 and 2018 increased to 57.9% and 76.6%, respectively ($p < 0.01$). Independent predictors of 30-day mortality were age ≥71 years, diabetes mellitus, dialysis, ≥36 h of vasopressor use at time of diagnosis, lactate levels ≥3.0 mg/dl, CPO <0.6 W, and PAPi <1.0 at 24 h after diagnosis and implementation of therapies. Either 1 or 2 points were assigned to each variable, and a 3-category risk score was determined: 0 to 1 (low), 2 to 4 (moderate), and ≥5 (high).

CONCLUSIONS This observational study suggests that a standardized team-based approach may improve CS outcomes. A score incorporating demographic, laboratory, and hemodynamic data may be used to quantify risk and guide clinical decision-making for all phenotypes of CS. (J Am Coll Cardiol 2019;73:1659–69) © 2019 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation.

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Volume 140, Issue 1, 2 July 2019; Pages 98-100
https://doi.org/10.1161/CIRCULATIONAHA.119.040654

RESEARCH LETTER

Shock Team Approach in Refractory Cardiogenic Shock Requiring Short-Term Mechanical Circulatory Support

A Proof of Concept

Iosif Taleb, MD, Antigone G. Koliopoulou, MD, Anwar Tandar, MD, Stephen H. McKellar, MD, MSc, Joseph E. Tonna, MD, Jose Nativi-Nicolau, MD, Miguel Alvarez Villela, MD, Frederick Welt, MD, Josef Stehlik, MD, MPH, Edward M. Gilbert, MD, Omar Wever-Pinzon, MD, Jack H. Morshedzadeh, MD, Elizabeth Dranow, PhD, Craig H. Selzman, MD, James C. Fang, MD, and Stavros G. Drakos, MD, PhD

CJC Open 2 (2020) 249–257

Original Article

Multidisciplinary Code Shock Team in Cardiogenic Shock: A Canadian Centre Experience

Felicity Lee, MBBS,[a,‡] Jordan H. Hutson, MD,[a,‡] Munir Boodhwani, MD,[b] Bernard McDonald, MD, PhD,[c] Derek So, MD, MSc,[a] Sophie De Roock, MD,[a] Fraser Rubens, MD, MSc,[b] Ellamae Stadnick, MD, MSc,[a] Marc Ruel, MD, MPH,[b] Michel Le May, MD,[a] Marino Labinaz, MD,[a] Kevin Chien,[a] Habibat A. Garuba, MD,[a] Lisa M. Mielniczuk, MD, MSc,[a] and Sharon Chih, MBBS, PhD[a]

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Future Directions

- What are the components of a Shock Team?
- What are the best practices?
- What is the right device at the right time?
- Hub and Spoke Model?
- Financial Cost?