Risk stratification Acute MI cardiogenic shock

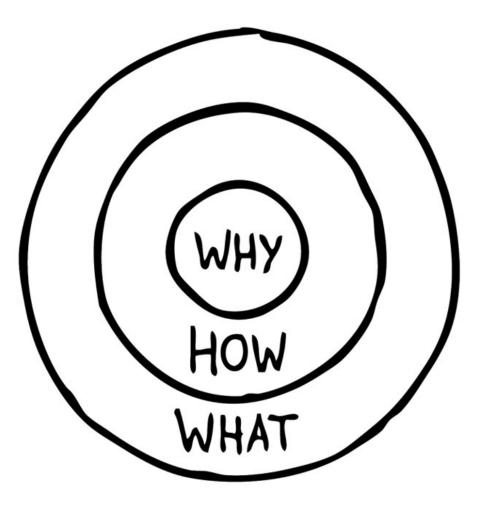
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• No disclosures



The Golden Circle





Start with 'WHY'

- Pathophysiology of acute MI CS
- Hemodynamic shock \rightarrow Hemo metabolic Shock
- Spectrum of Cardiogenic Shock



Start with 'WHY'

CONGESTION Pulmonary veins

Cardiogenic shock due to acute myocardial infarction

Myocardial

Occlusion of epicardial infarction TRIAL coronary artery Coronary MYOCARDIUM artery Progressive worsening Progressive worsening Myocardial ischemia **Decreased cardiac** Hypoxemia contractile mass Cardiomyocyte necrosis **Pulmonary congestion Decrease in ventricular function** Blood backup in the heart due to left Decreased cardiac output leads to insufficent ventricular systolic and diastolic dysfunction perfusion to end organs increases left atrial pressure and pulmonary HYPOPERFUSION capillary pressure, causing pulmonary edema Increased left atrial and pulmonary venous pressure Ventricular End-organ dysfunction or injury Decreased dysfunction cardiac output Decreased cardiac output leads to tissue injury Liver Gastrointestinal Brain Kidneys
Lungs tract Systemic hypotensio Systemic Systemic inflammatory response Decreased coronary hypoperfusion perfusion pressure Pulmonary edema impairs gas exchange Increased circulating at the capillary-alveolar interface proinflammatory mediators Increased pulmonary capillary pressure Vasoplegia Capillary. Sympathetic and neurohormonal activation Systemic hypotension Carotid baroreceptors and kidney juxtaglomerular ce perceive hypoperfusion, causing reflexive responses Edema fluid Renin-angiotensin-aldosterone LVEOLUS (RAAS) cascade Catecholamine Volume overload Increased circulating

Cross-section view

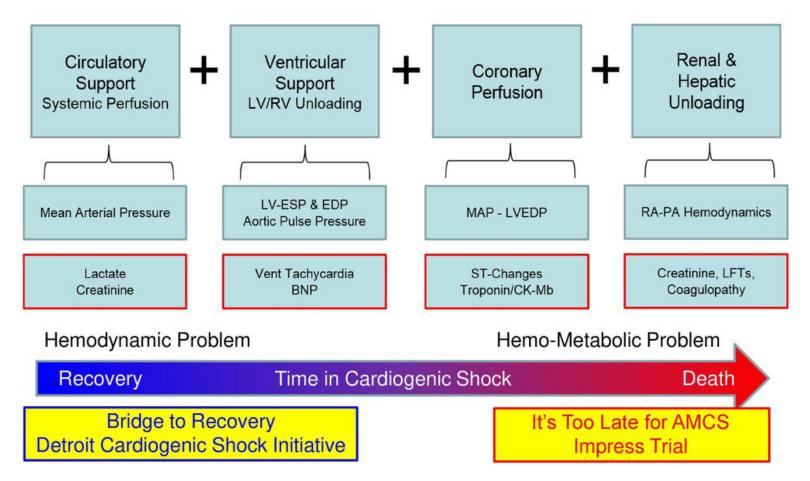
of coronary artery

SHOCK

HYPOTENSION



The Hemodynamic Support Equation for Acute MCS From Arithmetic to Calculus



Kapur and Esposito Curr Cardio Risk 2016 & F1000 2017



SCAI stages of CS

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.

<u>C</u>LASSIC

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).

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



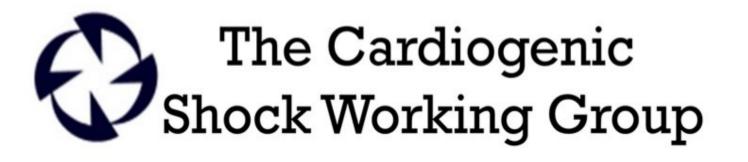
Mortality increases with SCAI staging





Cardiogenic Shock Working Group (CSWG)

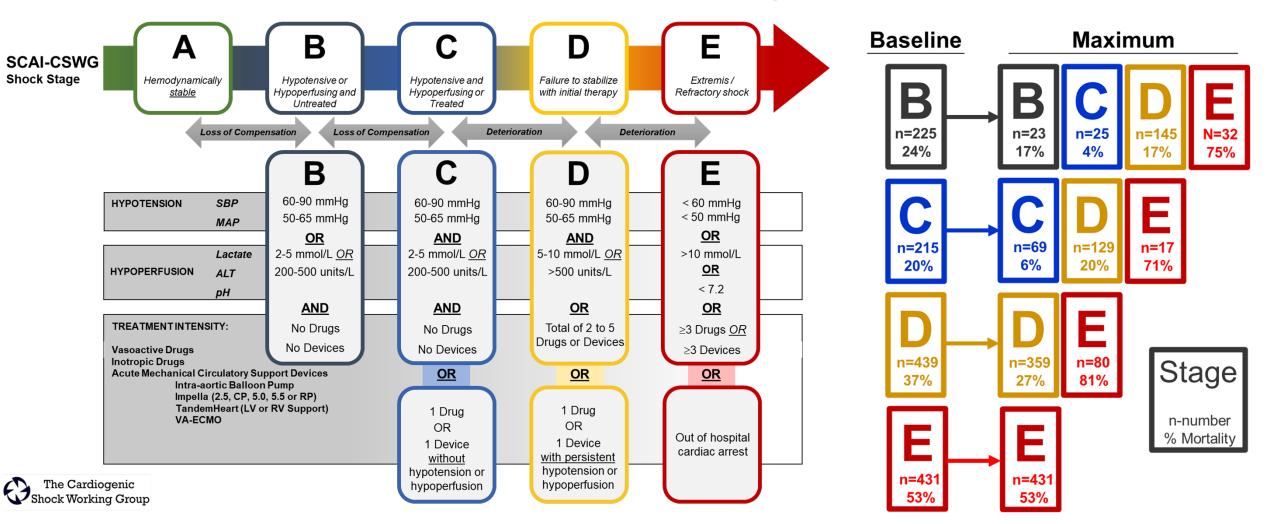
 A goal of the Cardiogenic Shock Working Group is to create a centralized registry, compiled of data from multiple institutions, to analyze clinical outcomes.



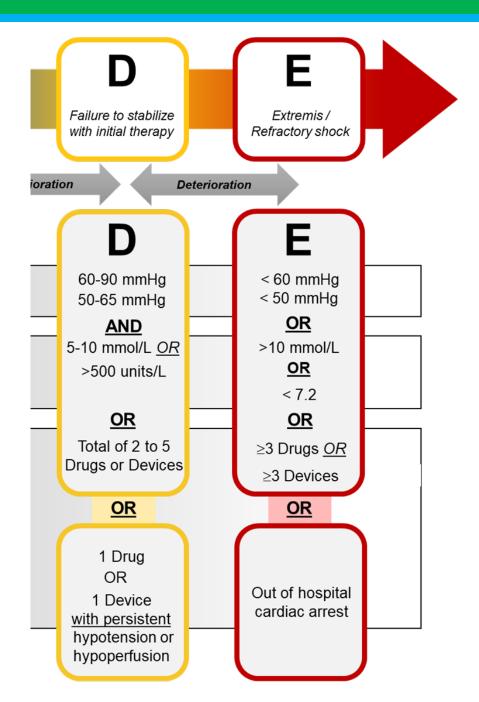




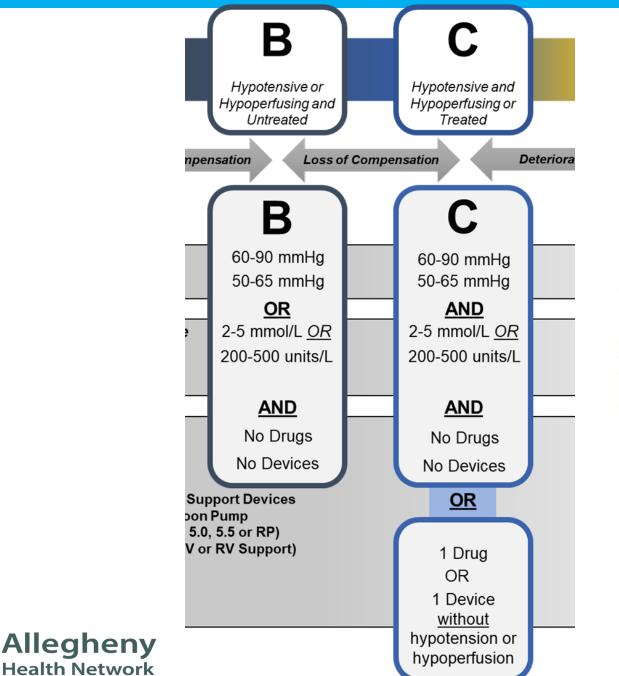
How to assess severity?



Allegheny Kapur, Kanwar, Sinha et al, *Criteria for Defining Stages of Cardiogenic Shock Severity*, JACC 2022:185-198 Health Network







REPLY: "B" Is for Bad in SCAI Shock Staging

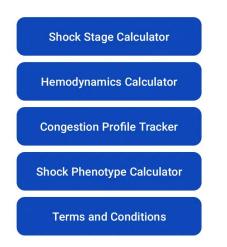
The Need for Early Diagnosis and Intervention

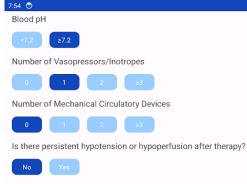
clinical deterioration. We agree that patients with SCAI B shock are a particularly vulnerable cohort that may be easily overlooked because of the lack of hypotension and as a result may not receive early intervention. This expanded definition for SCAI B is

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The Cardiogenic Shock Working Group

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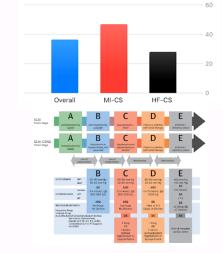




Out of Hospital Cardiac Arrest



The CSWG-SCAI shock stage is D Predicted in-hospital mortality: 36.45% Predicted in-hospital mortality for MI-CS: 46.9% Predicted in-hospital mortality for HI-CS: 28.16% Predicted probability of stage escalation: 18%



▼⊿∎

Kapur NK, Kanwar M, Sinha SS, et al. Criteria for Defining Stages of Cardiogenic Shock Severity. J Am Coll Cardiol. 2022;80(3):185-198 doi:10.1016/j.jacc.2022.04.049

John KJ, Stone SM, Zhang Y, et al. Application of Cardiogenic Shock Working Group defined Society for Cardiovascular Angiography and Interventions (CSWG SCAI) Staging of Cardiogenic Shock to the Medical Information Mart for Intensive Carel V (MIMIC-IV) database. Cardiovasc Revasc Med. 2023;51553-8389(23):00666-8. doi:10.1016/j.carev.2023.06.019

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Factors to be considered when choosing tMCS

Proposed 3-axis model of cardiogenic shock evaluation and prognostication

Shock severity

- SCAI shock stage
- Hemodynamics
- Metabolic derangements
- Vasopressor toxicity shock seventry

Phenory pe & Eriology **Phenotype & Etiology**

 Acute vs. Acute-on-Chronic

dysfunction

Congestion profile

• Cardiac vs.

Clinical etiology of CS

• RV vs. LV vs. BIV systolic

cardiopulmonary failure

Biochemical phenotype

Cardiogenic shock patient

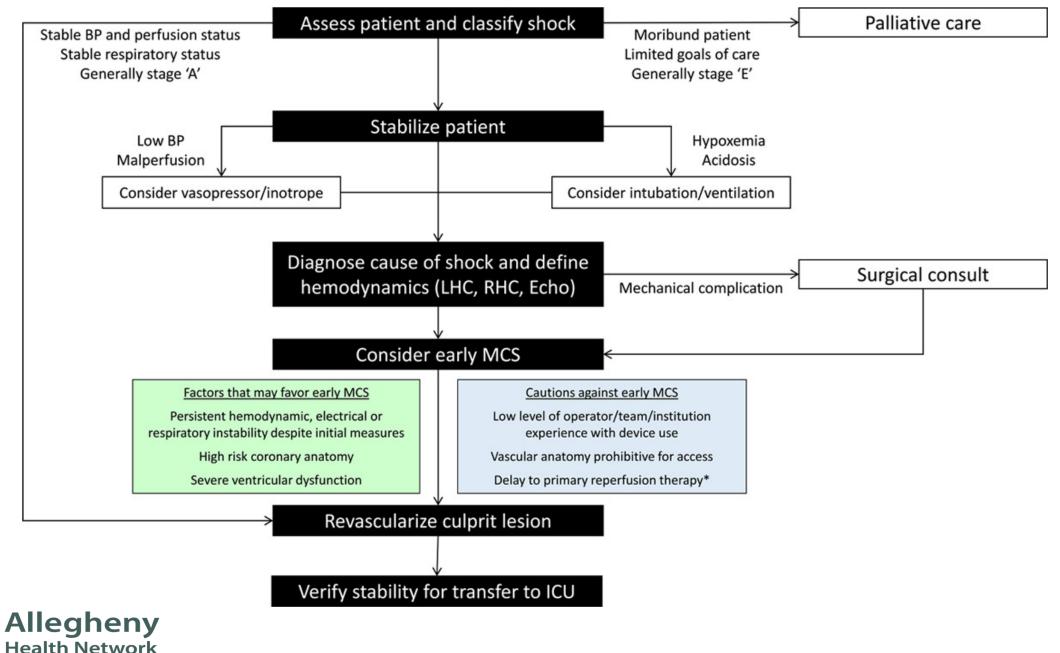
Risk modifiers

Risk modifiers

- Non-modifiable risk factors: e.g. age, comorbidities
- Cardiac arrest with coma
- Presence/reversibility of organ failure
- Systemic inflammatory response
- Frailty/risk of complication



What to do in practice?



Take home messages

- Cardiogenic Shock is a spectrum (Stage A \rightarrow Stage E)
- Hemodynamic \rightarrow Hemo metabolic \rightarrow Death
- Vasopressors in CS can normalize BP = Normalize perfusion
- TTE/LHC/RHC essential to phenotype CS
- Serum lactate, ABG, CMP metabolic derangements
- Severity of CS SCAI CSWG chart/app
- Non modifiable risk factors (age, frailty, co morbidities)

