PLACE

PLATFORM OF LABORATORIES FOR ADVANCES IN CARDIAC EXPERIENCE

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9ª Edizione

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SHOCK CARDIOGENO

APPROCCIO ALLO SHOCK CARDIOGENO: PATOGENESI E CLASSIFICAZIONE

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Definition

SHOCK Trial	IABP-SHOCK II Trial	CULPRIT-SHOCK Trial	ESC Heart Failure Guidelines
Clinical criteria SBP < 90 mmHg for ≥30 min or need for support to maintain SBP ≥ 90 mmHg -AND- End-organ hypoperfusion (cool extremities or UOP < 30 ml/h, and HR ≥ 60 bpm) *Hemodynamic criteria* CI ≤ 2.2 l/min/m^2 and PCWP ≥ 15 mmHg	SBP < 90 mmHg for ≥30 min or need for catecholamines to maintain SBP ≥ 90 mmHg -AND- Clinical signs of pulmonary congestion -AND- Impaired end-organ perfusion requiring at least one of the following: Altered mental status Cold, clammy skin and extremities Oliguria with UOP < 30 ml/h Serum lactate > 2.0 mmol/l	SBP < 90 mmHg for ≥30 min or use of catecholamines to maintain SBP ≥ 90 mmHg -AND- Clinical signs of pulmonary congestion -AND- Impaired end-organ perfusion requiring at least one of the following: Altered mental status Cold, clammy skin and limbs Oliguria with UOP < 30 ml/h serum lactate > 2.0 mmol/l	SBP < 90 mmHg 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

CI, cardiac index; HR, heart rate; PCWP, pulmonary capillary wedge pressure; UOP, urine output.

Berg DD et al Curr Opin Crit Care 2021

Definition

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

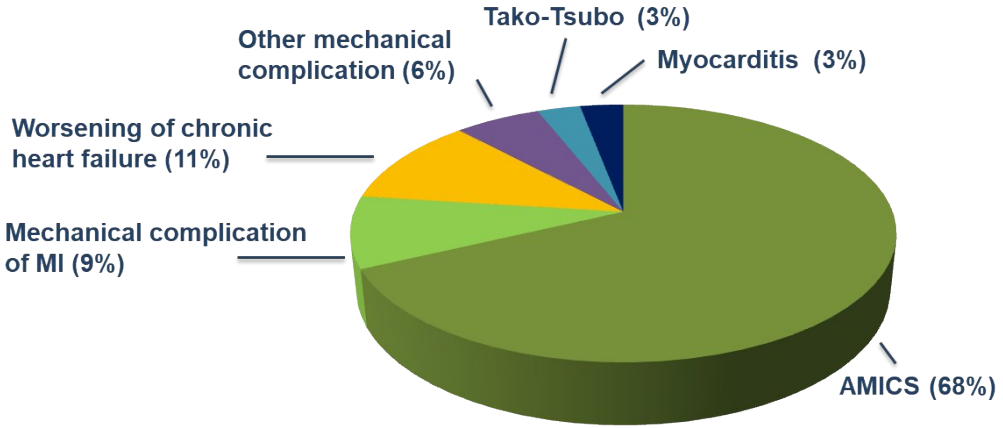
A syndrome caused by a primary cardiovascular disorder in which inadequate cardiac output results in a life-threatening state of tissue hypoperfusion associated with impairment of tissue oxygen metabolism and hyperlactatemia which, depending on its severity, may result in multi-organ dysfunction and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

Chioncel O et al Eur J Heart Fail. 2020

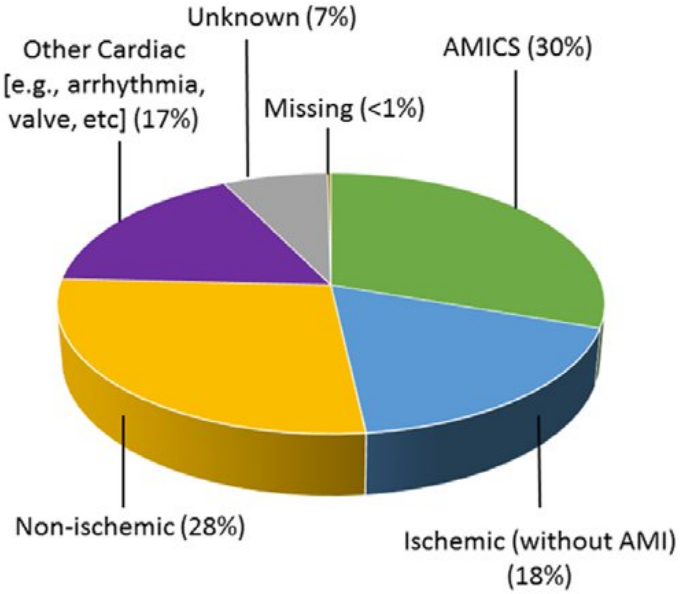
Etiology

Causes of cardiogenic shock

Data from the CardShock study investigators and the GREAT network (2010-2012)

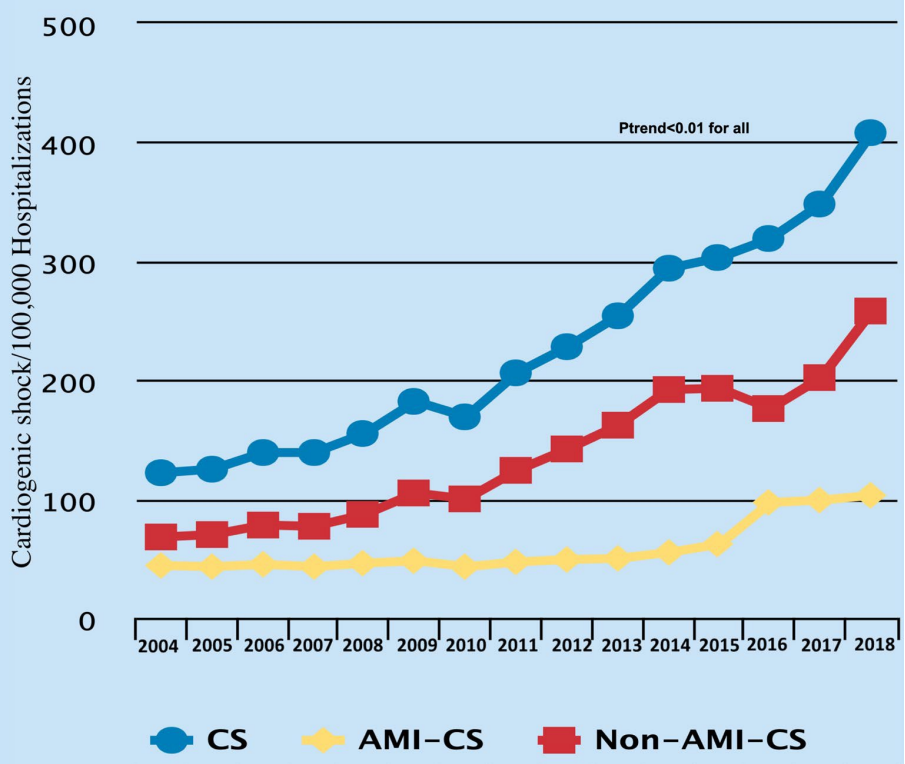


Data from observational study from the Critical Care Cardiology Trials Network (CCCTN), a collaborative research network of advanced cardiac ICUs (CICUs) in North America (2017-2018)



Epidemiology

15-Year Trends in Incidence of Cardiogenic Shock Hospitalization in the United States



From January 2004 to December 2018:

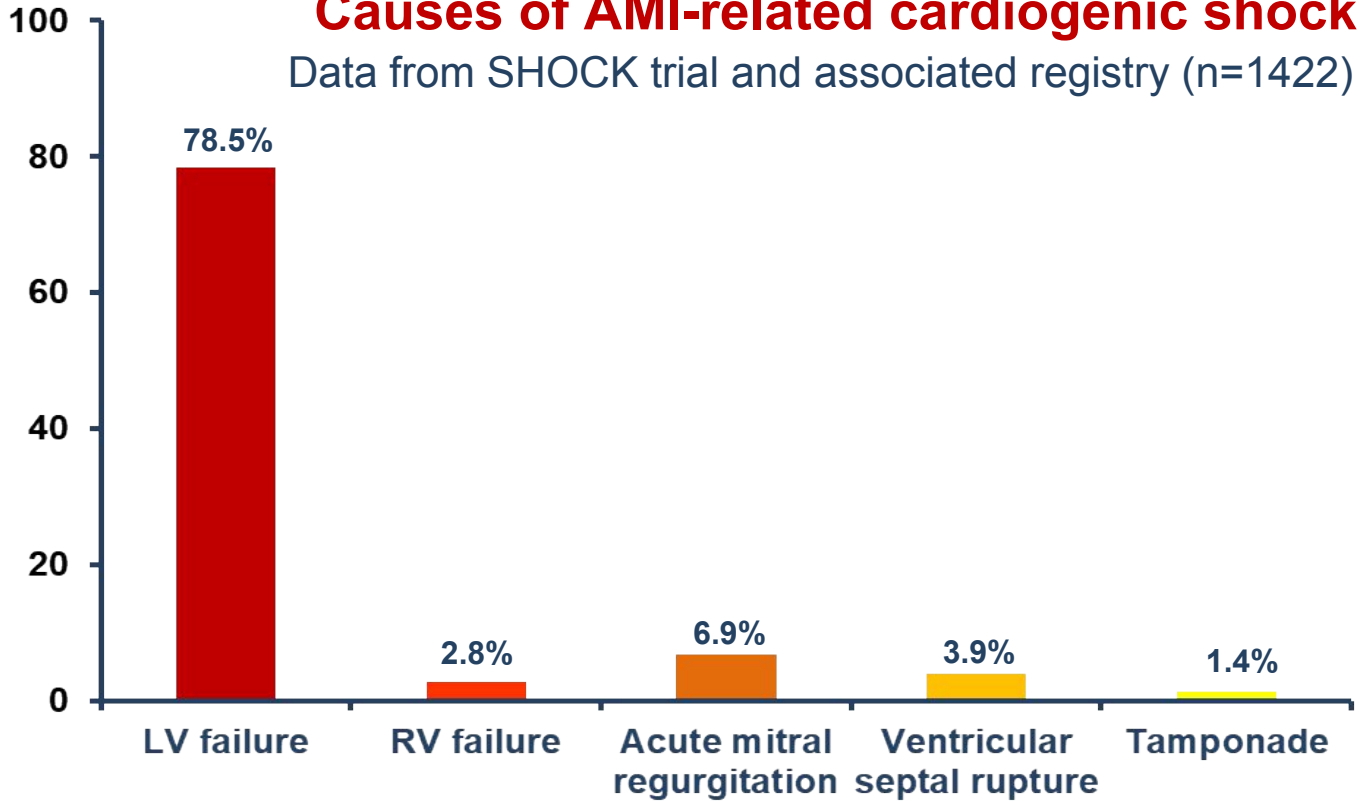
- a steady increase in hospitalizations for CS from 122 per 100 000 hospitalizations in 2004 to 408 per 100 000 hospitalizations in 2018
- AMI-CS from 44 per 100 000 hospitalizations in 2004 to 103 per 100 000 hospitalizations in 2018
- Non AMI-CS from 68 per 100 000 hospitalizations in 2004 to 258 per 100 000 hospitalizations in 2018

Osman M et al Am Heart Assoc. 2021

Etiology

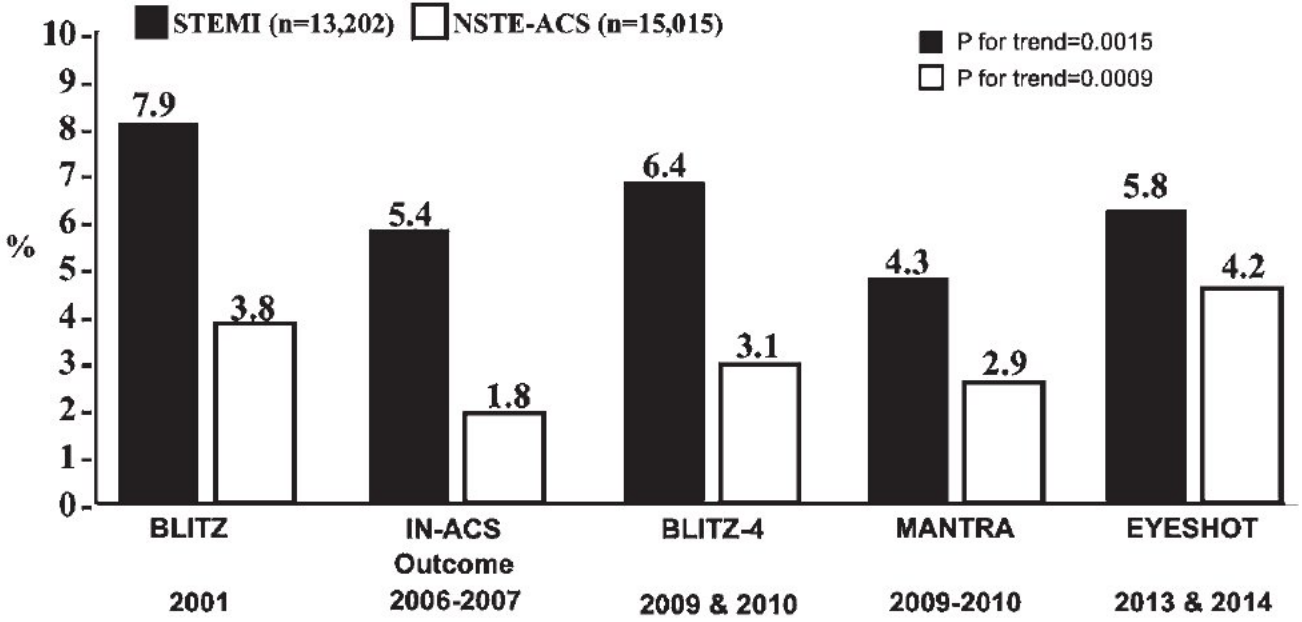
Causes of AMI-related cardiogenic shock

Data from SHOCK trial and associated registry (n=1422)



Etiology

AMI-related cardiogenic shock

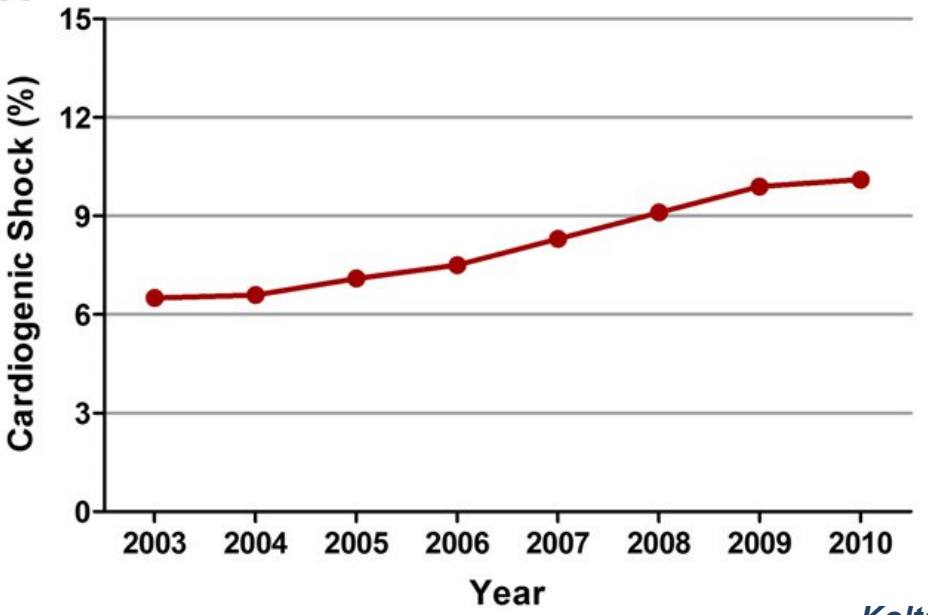


De Luca L et al Eur J Heart Fail 2015

Epidemiology

AMI-related Cardiogenic Shock

Data 2003–2010 Nationwide Inpatient Sample USA databases
(1.990.486 patients aged ≥40 years with STEMI, 157.892 (7.9%) with cardiogenic shock



Kolte D et al J Am Heart Assoc. 2014

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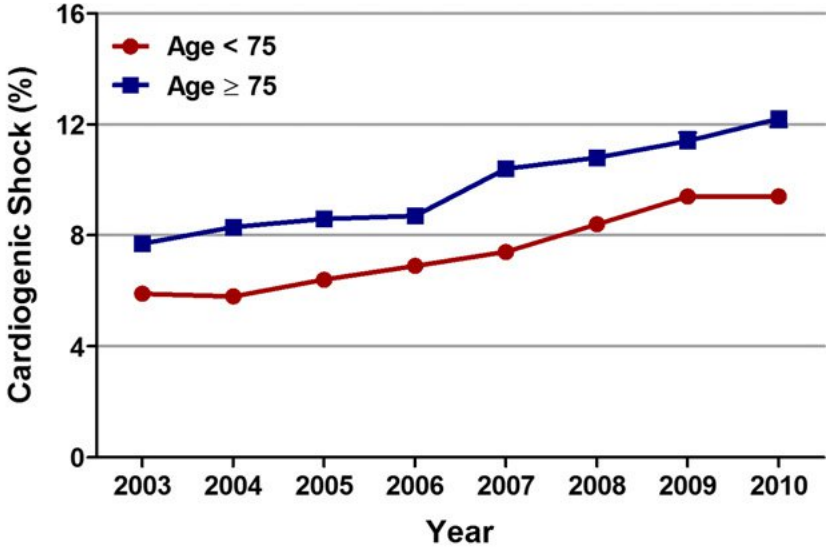

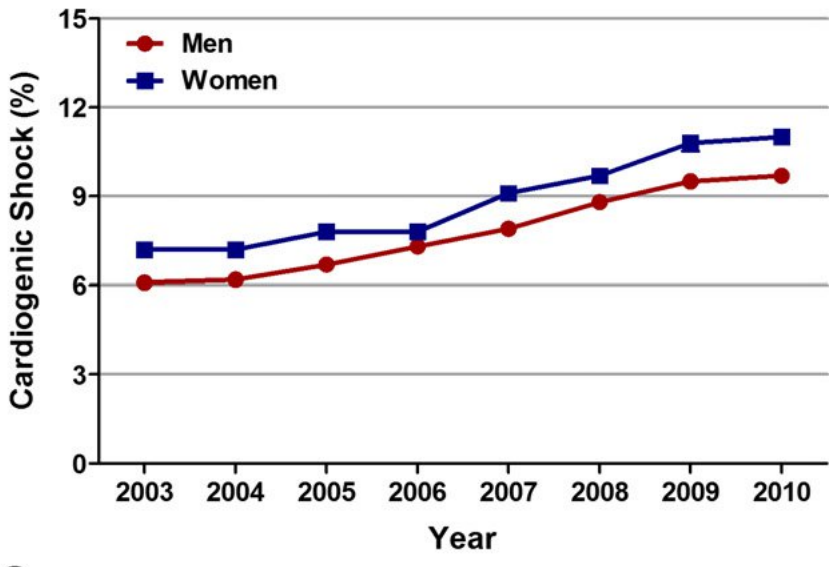

Kolte D et al J Am Heart Assoc. 2014

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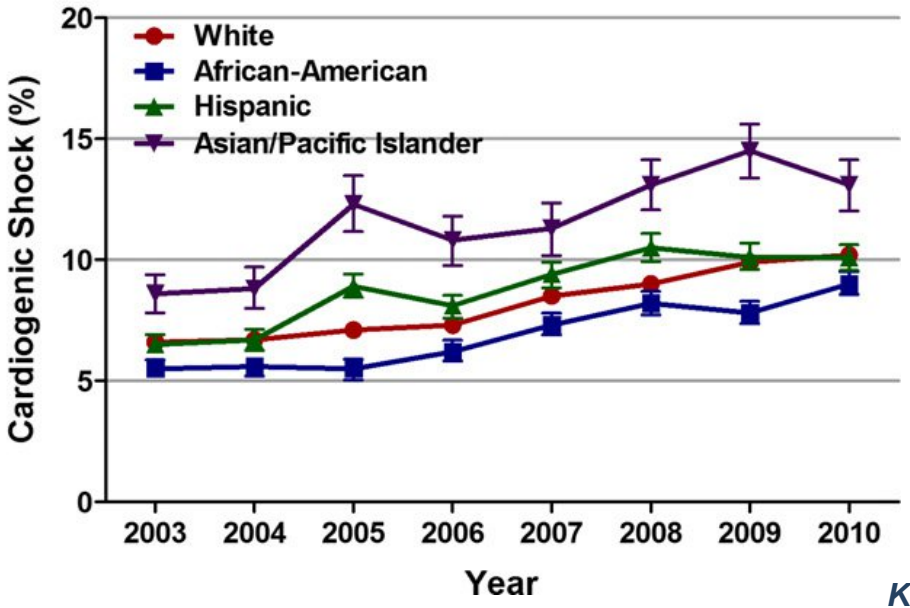
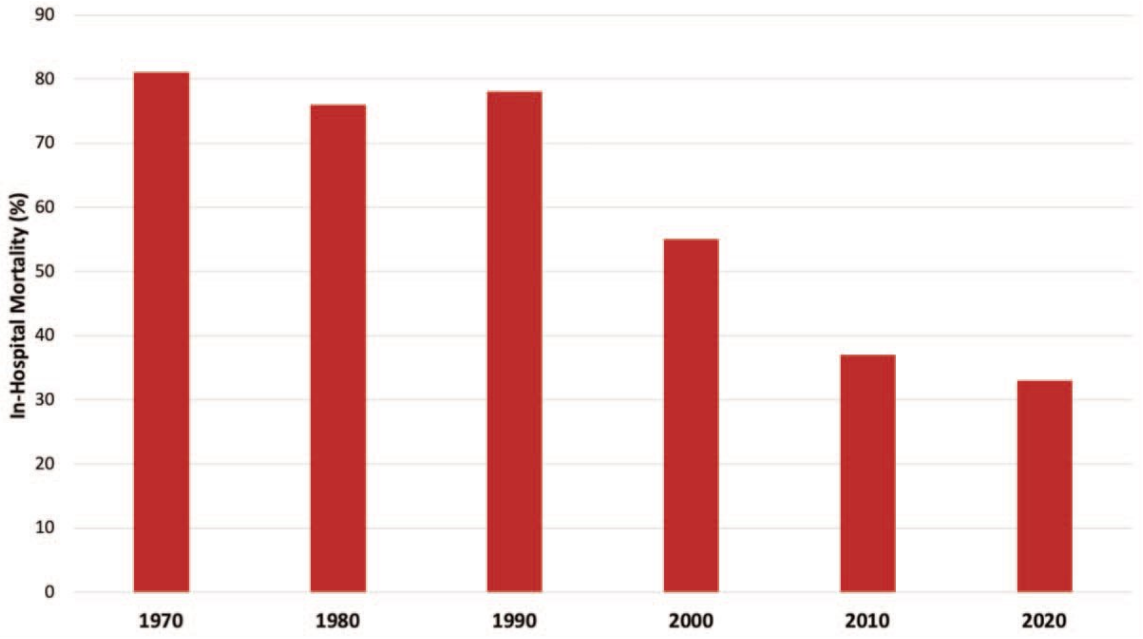


Kolte D et al J Am Heart Assoc. 2014

Prognosis

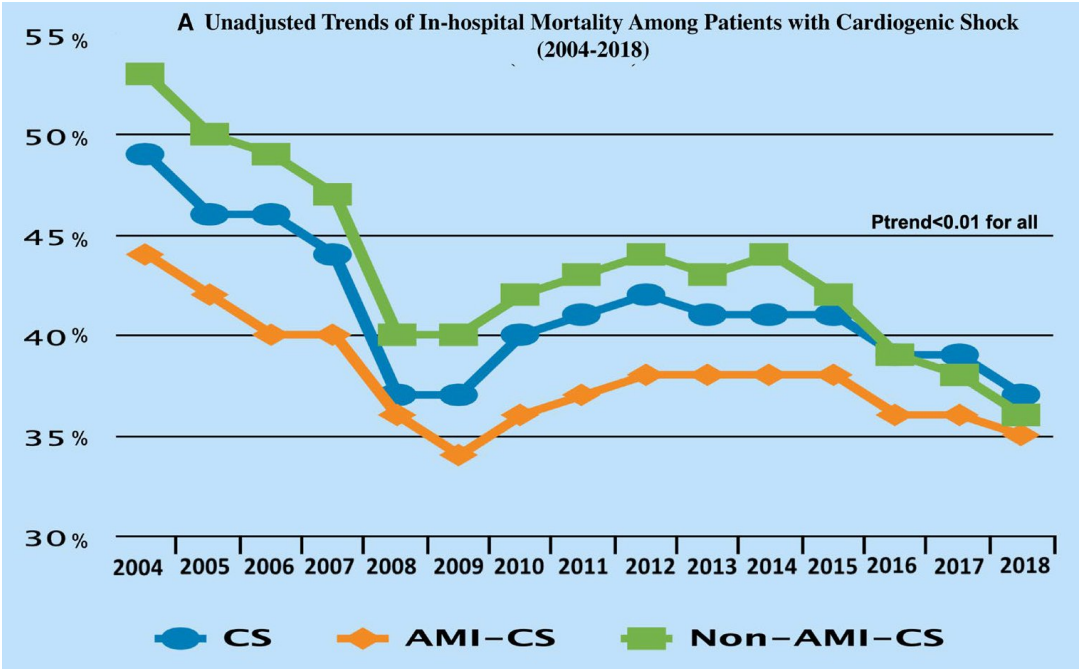
Temporal trends in cardiogenic shock in-hospital mortality



Berg D D et al Curr Opin Crit Care 2021

Prognosis

15-Year Trends in Trends in in-hospital mortality among patients with Cardiogenic Shock in the United States



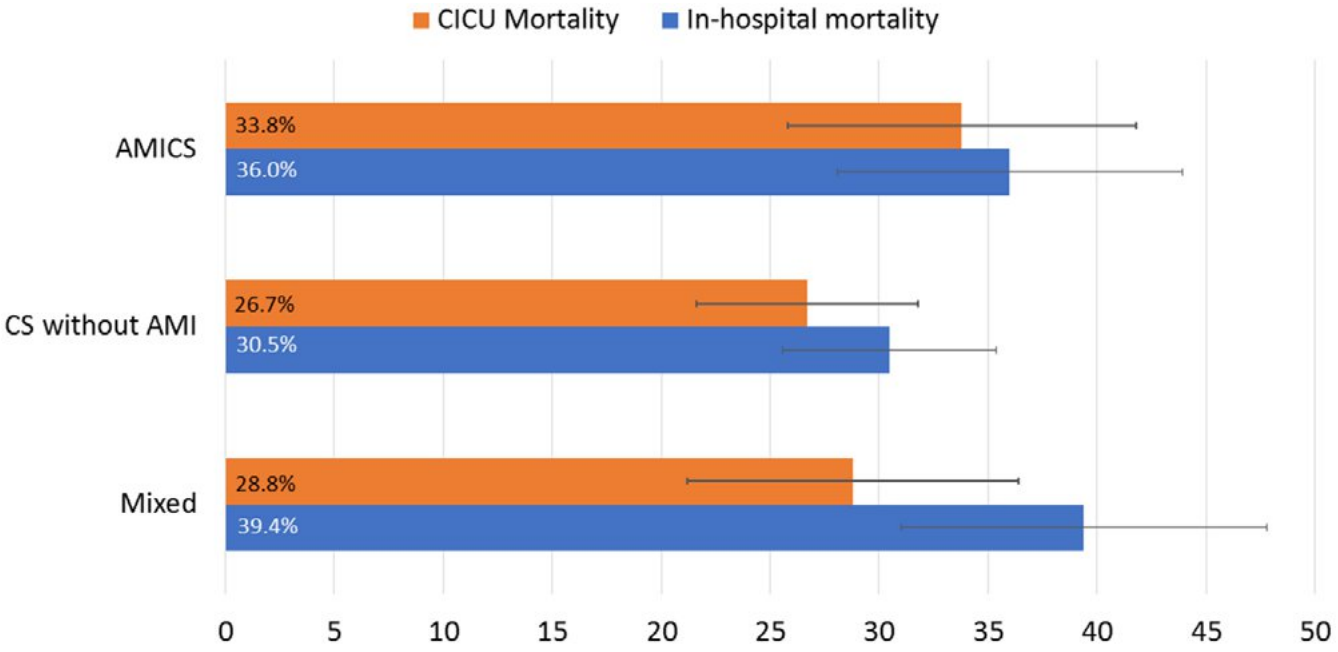
From January 2004 to December 2018:

- a decline in in-hospital mortality during the study periods from 49% in 2004 to 37% in 2018
- AMI-CS from 44% in 2004 to 35% in 2018
- Non AMI-CS from 53% in 2004 to 36% in 2018.

Osman M et al Am Heart Assoc. 2021

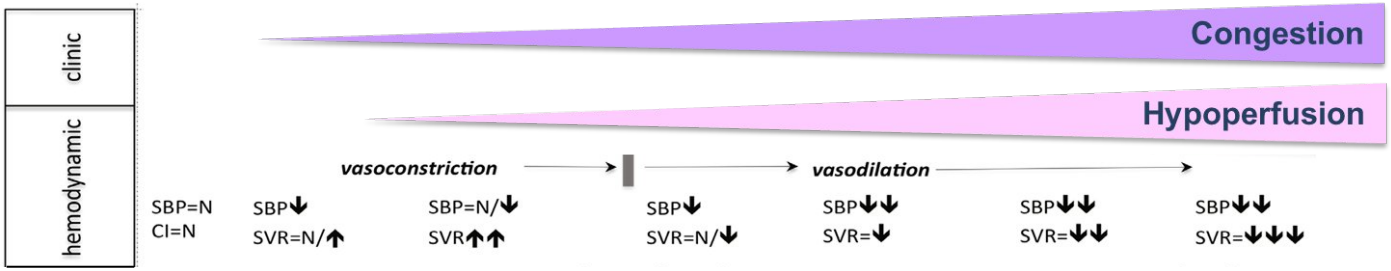
Prognosis

Data from observational study from the Critical Care Cardiology Trials Network (CCCTN), a collaborative research network of advanced cardiac ICUs (CICUs) in North America (2017-2018)

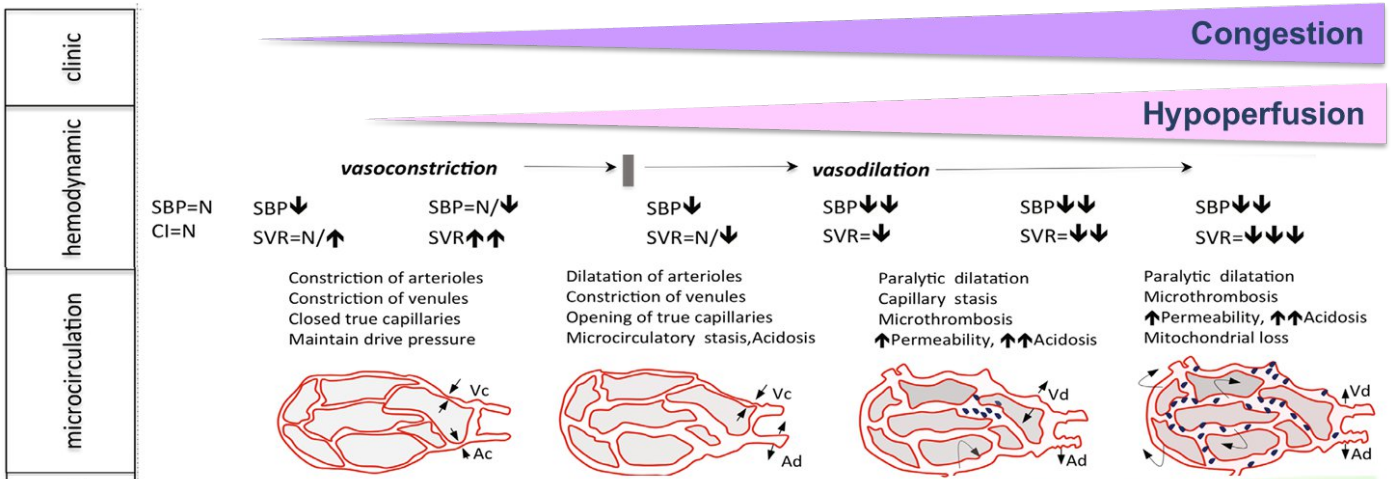


Berg D D et al Circ Cardiovasc Qual Outcomes. 2019

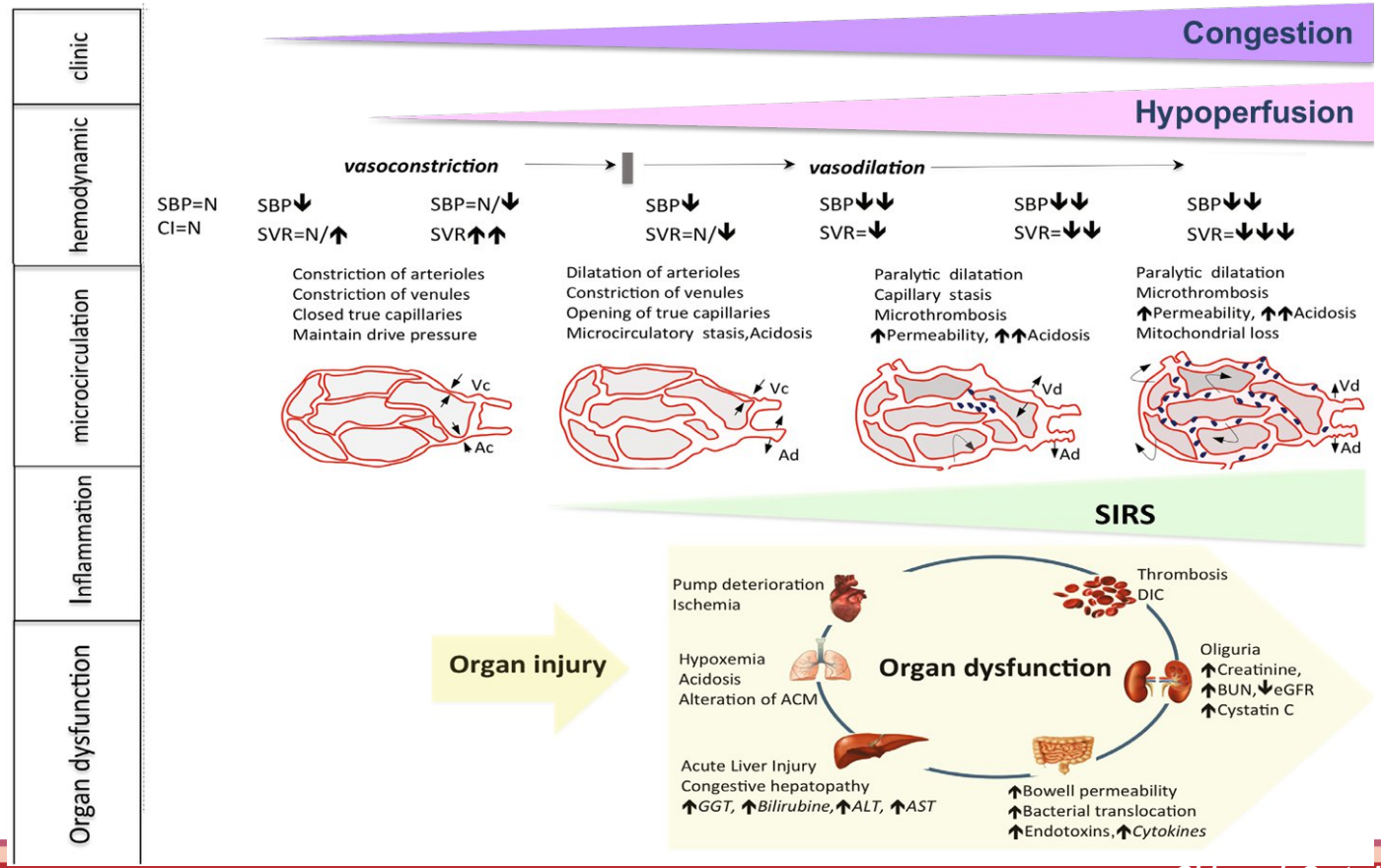
Pathophysiology



Pathophysiology



Pathophysiology



Chioncel O et al Eur J Heart Fail. 2020

Classification

Clinical Classification

preCS

Hypoperfusion and SBP> 90 mmHg without circulatory support

CS

Hypoperfusion and SBP <90 for >30 min or the need for pharmacologic or IABP to maintain SBP >90 mm Hg or MAP with 30 mm Hg lower than baseline.

Refractory CS

Ongoing evidence of tissue hypoperfusion despite administration of adequate doses of 2 vasoactive medications and treatment of the underlying etiology

Chioncel O et al Eur J Heart Fail. 2021

Classification

Clinical Classification

preCS

Hypoperfusion and SBP> 90 mmHg without circulatory support

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Hypoperfusion and SBP <90 for >30 min or the need for pharmacologic or IABP to maintain SBP >90 mm Hg or MAP with 30 mm Hg lower than baseline.

Refractory CS

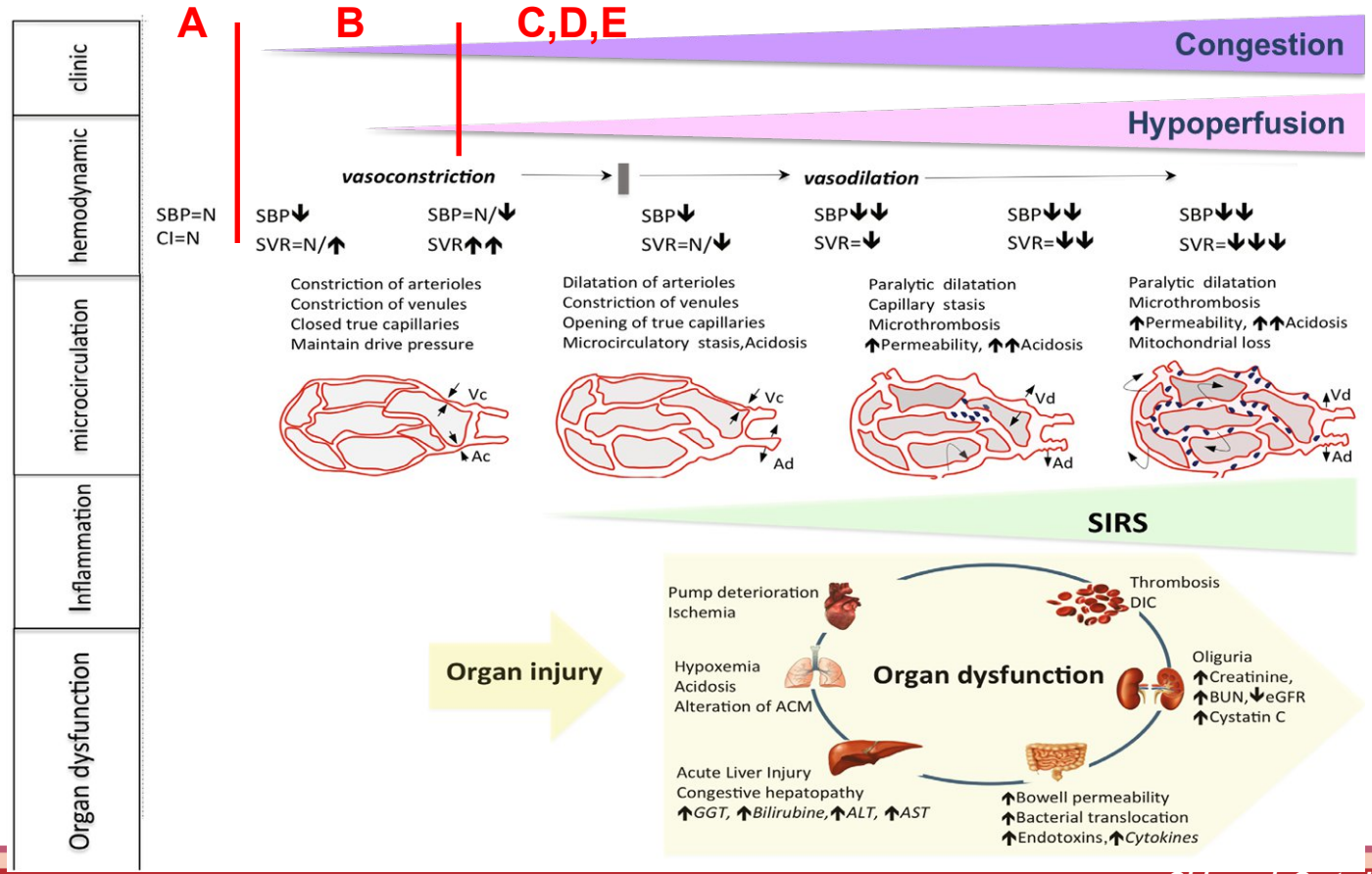
Ongoing evidence of tissue hypoperfusion despite administration of adequate doses of 2 vasoactive medications and treatment of the underlying etiology

Hemodynamic Classification

SVR ↓;PCWP N ↓;CVP N ↓ "warm-dry"	SVR ↓;PCWP ↑;CVP ↑ "warm-wet"
SVR ↑;PCWP N ↓;CVP N ↓ "cold-dry"	SVR ↑;PCWP ↑;CVP ↑ "cold-wet"

Chioncel O et al Eur J Heart Fail. 2021

Classification



Chioncel O et al Eur J Heart Fail. 2020

SCAI Shock Stage Classification

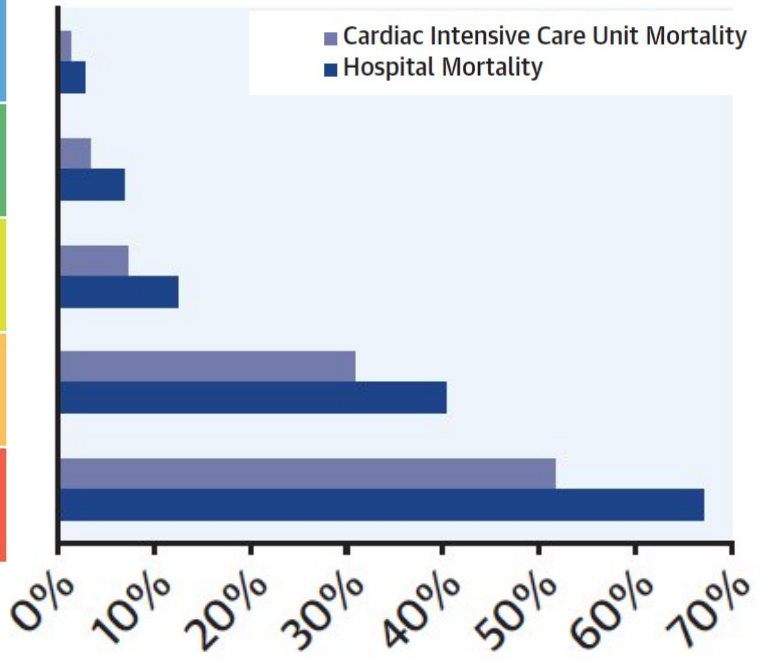
SCAI Shock Stage Classification

Stage	Description	Physical exam/bedside findings	Biochemical markers	Hemodynamics
A At risk	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for its development. These patients may include those with large acute myocardial infarction or prior infarction acute and/or acute on chronic heart failure symptoms.	Normal JVP Lung sounds clear Warm and well perfused • Strong distal pulses • Normal mentation	Normal labs • Normal renal function • Normal lactic acid	Normotensive (SBP≥100 or normal for pt.) If hemodynamics done • cardiac index ≥2.5 • CVP <10 • PA sat ≥65%
B Beginning CS	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.	Elevated JVP Rales in lung fields Warm and well perfused • Strong distal pulses • Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP <90 ***OR*** MAP <60 ***OR*** >30 mmHg drop from baseline Pulse ≥100 If hemodynamics done • cardiac index ≥2.2 • PA sat ≥65%
C Classic CS	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support, including ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension.	***May Include Any of:*** Looks unwell Panicked Ashen, mottled, dusky Volume overload Extensive rales Killip class 3 or 4 BiPap or mechanical ventilation Cold, clammy Acute alteration in mental status Urine output <30 mL/h	***May Include Any of:*** Lactate ≥2 Creatinine doubling ***OR*** >50% drop in GFR Increased LFTs Elevated BNP	***May Include Any of:*** SBP <90 ***OR*** MAP <60 ***OR*** >30 mmHg drop from baseline ***AND*** drugs/device used to maintain BP above these targets Hemodynamics • cardiac index <2.2 • PCWP >15 • RAP/PCWP ≥0.8 • PAPI <1.85 • cardiac power output ≤0.6
D Deteriorating/ doom	A patient that is similar to category C but are getting worse. They have failure to respond to initial interventions.	***Any of stage C***	***Any of Stage C AND:*** Deteriorating	***Any of Stage C AND:*** Requiring multiple pressors ***OR*** addition of mechanical circulatory support devices to maintain perfusion
E Extremis	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions.	Near Pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	**"Trying to die"** CPR (A-modifier) pH ≤7.2 Lactate ≥5	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

SCAI Shock Stage Classification & Mortality

Cardiogenic Shock Stage	Study Definition
Stage A ("At risk")	Neither hypotension/tachycardia nor hypoperfusion
Stage B ("Beginning")	Hypotension/tachycardia WITHOUT hypoperfusion
Stage C ("Classic")	Hypoperfusion WITHOUT deterioration
Stage D ("Deteriorating)"	Hypoperfusion WITH deterioration NOT refractory shock
Stage E ("Extremis")	Hypoperfusion WITH deterioration AND refractory shock

Observed Mortality in Overall Cohort

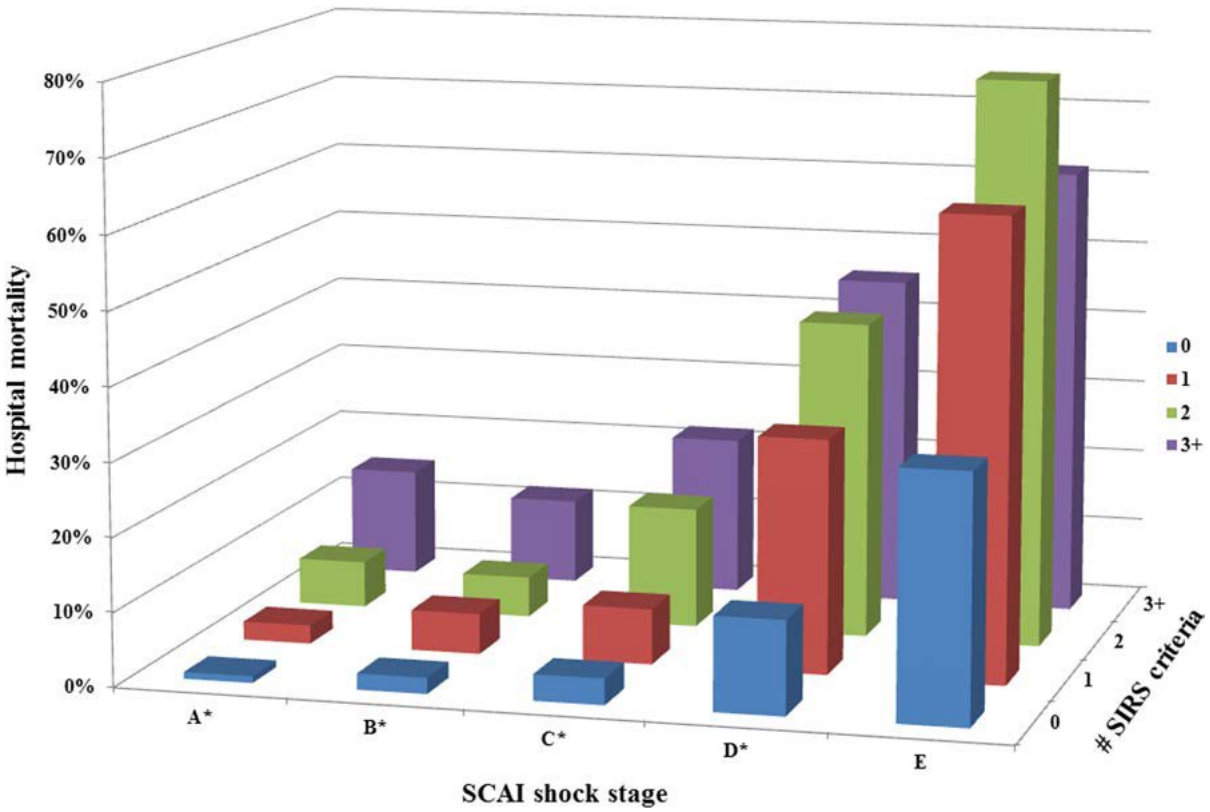


10.004 patients admitted between 2007 and 2015 in Mayo Clinic CICU, 43.1% had acute coronary syndrome, 46.1% had heart failure, and 12.1% had CA. The proportion of patients in SCAI CS stages A through E was 46.0%, 30.0%, 15.7%, 7.3%, and 1.0%

Jentzer JC et al J Am Coll cardiol 2019

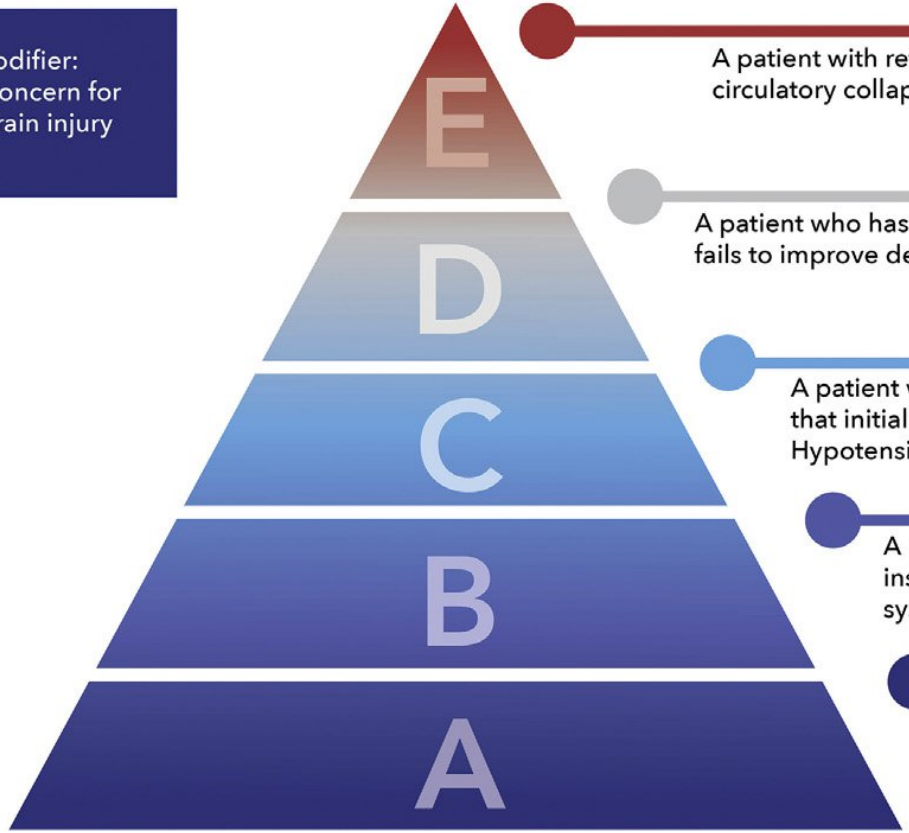
SCAI Shock Stage SIRS & Mortality

8.995 unique patients admitted to the Mayo Clinic CICU between 2007 and 2015. One-third of CICU patients meet clinical criteria for SIRS (patients with ≥2/4 SIRS criteria based on admission laboratory and vital sign data)



SCAI Shock Stage Classification

(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).

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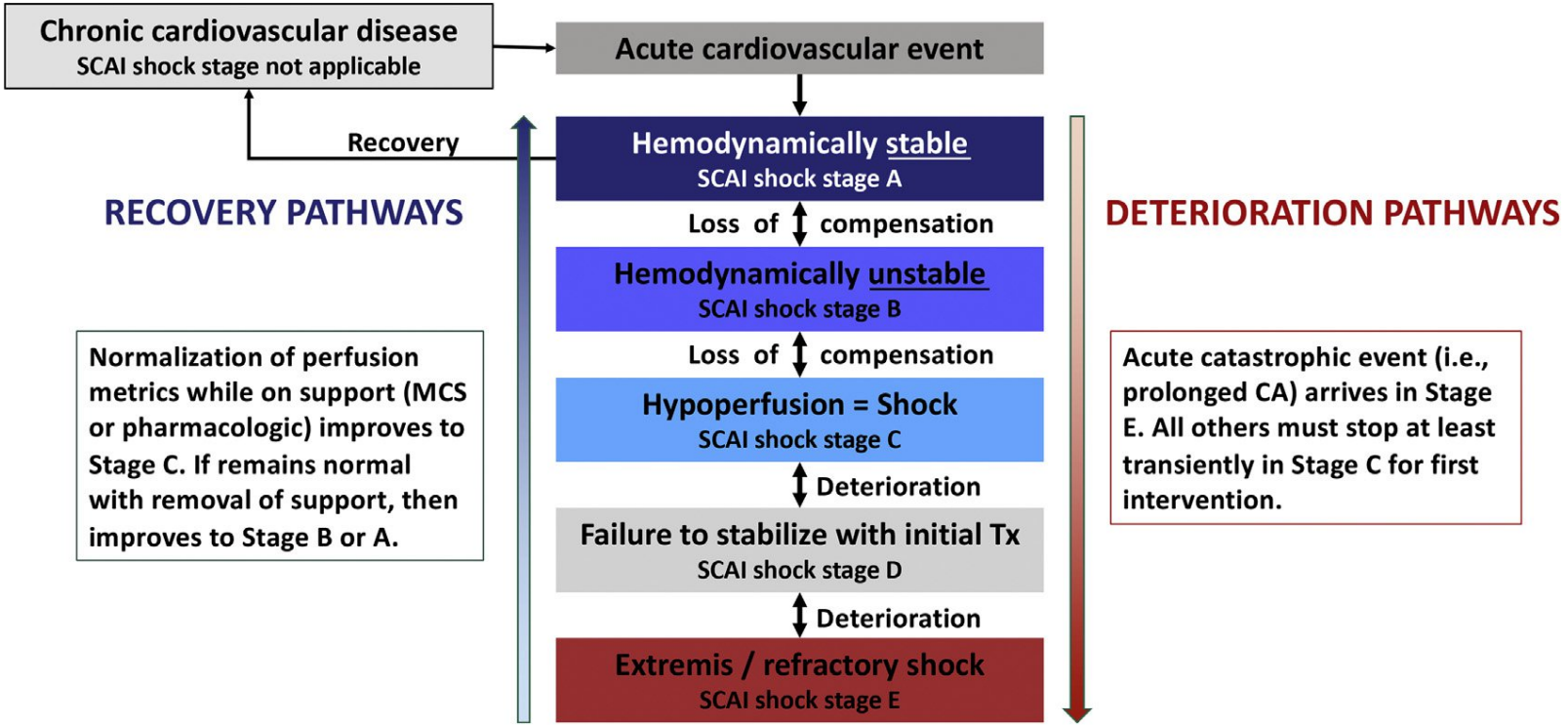
SCAI Shock Stage Classification

		Physical examination/ bedside findings		Biochemical markers		Hemodynamics	
Stage	Description	Typically includes	May include	Typically includes	May include	Typically includes	May include
A At risk	A patient who is **not currently experiencing signs or symptoms of CS, but is at risk for its development.** These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Clear lung sounds	**Normal lactate**	Normal labs • Normal (or at baseline) renal function	**Normotensive** (SBP ≥100 mmHg or at baseline)	If invasive hemodynamics are assessed: • Cardiac Index ≥2.5 L/min/m^2 (if acute) • CVP ≤10 mmHg • PCWP ≤15 mmHg • PA saturation ≥65%
B Beginning CS	A patient who has **clinical evidence of hemodynamic instability** (including relative hypotension or tachycardia) **without hypoperfusion.**	**Elevated JVP** **Warm and well-perfused** • Strong distal pulses • Normal mentation	Rales in lung fields	**Normal lactate**	Minimal acute renal function impairment Elevated BNP	**Hypotension** • SBP <90 mmHg • MAP <60 mmHg • >30 mmHg drop from baseline **Tachycardia** • Heart rate ≥100 bpm	
C Classic CS	A patient who manifests with **hypoperfusion and who requires one intervention (pharmacological or mechanical) beyond volume resuscitation.** These patients typically present with relative hypotension (but hypotension is not required).	**Volume overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output <30 mL/h	**Lactate ≥2 mmol/L**	Creatinine increase to 1.5 x baseline (or 0.3 mg/dL) or >50% drop in GFR Increased LFTs Elevated BNP	If invasive hemodynamics assessed (**strongly recommended**) • Cardiac index <2.2 L/min/m^2 • PCWP >15 mmHg	
D Deteriorating	A patient who is similar to category C but is getting worse. **Failure of initial support strategy to restore perfusion** as evidenced by worsening hemodynamics or rising lactate.	**Any of stage C and worsening (or not improving) signs/ symptoms of hypoperfusion despite the initial therapy.**		**Any of stage C and lactate rising and persistently >2 mmol/L**	Deteriorating renal function Worsening LFTs Rising BNP	**Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of a mechanical circulatory support device to maintain perfusion**	
E Extremis	**Actual or impending circulatory collapse**	**Typically unconscious**	Near pulselessness Cardiac collapse Multiple defibrillations	**Lactate ≥8 mmol/L**[a]	CPR (A-modifier) Severe acidosis • pH <7.2 • Base deficit >10 mEq/L	**Profound hypotension despite maximal hemodynamic support**	Need for bolus doses of vasopressors

SCAI Shock Stage Classification

The Dynamic Process of Cardiogenic Shock



SCAI Shock Stage Classification

A-At risk
At risk of CS;
SBP=N;CI>2.5;
Lactate=N
No hypoperfusion

B-Beginning
Relative hypotension or tachycardia without hypoperfusion;
CI<2.2,PA sat ≥65%.
Lactate<2mmol/l

C-Classic
Hypoperfusion that requires an initial set of Interventions (inotropes, pressor, MCS, or ECMO) beyond volume resuscitation to restore perfusion; CI<2.2,PA sat <65%,CP ≤0.6. Lactate>2mmol/l

D-Deteriorating
Failed to stabilize despite intense initial efforts. After >30 minutes the patient has not responded with resolution of hypotension or end-organ hypoperfusion. Further escalation (increase in the number and intensity of IV therapies, or addition of MCS) is required;
Lactate>5mmol/l

E-Extremis
refractory cardiac arrest with ongoing CPR or are being supported by multiple acute interventions including ECMO-facilitated CPR

Lactate<2mmol/L ← → Lactate>2mmol/L

Hypoperfusion

MODS

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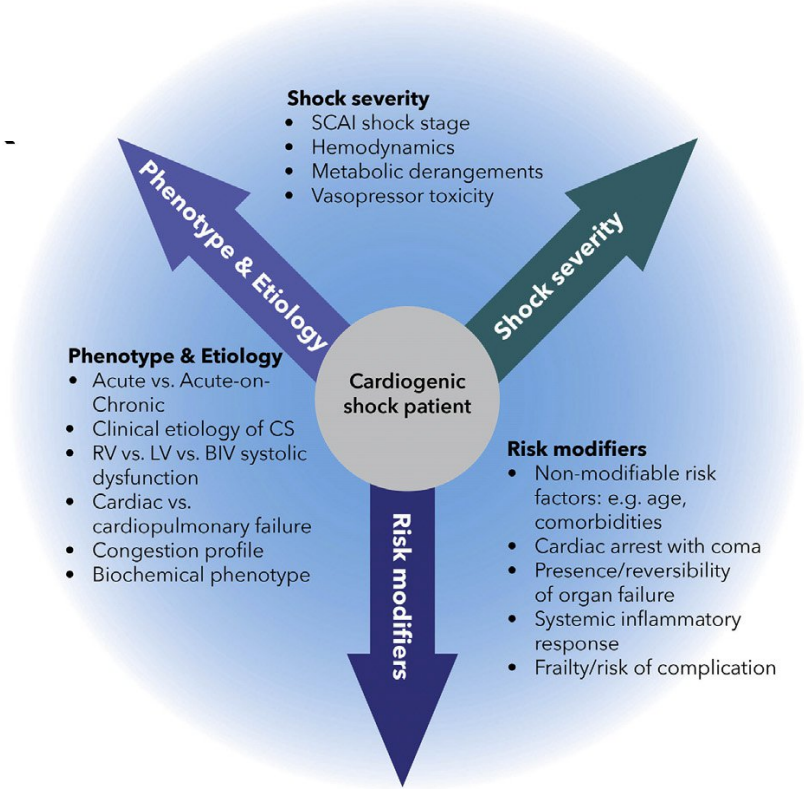
SCAI Shock Stage Classification

3-axis model of cardiogenic shock evaluation and prognostication



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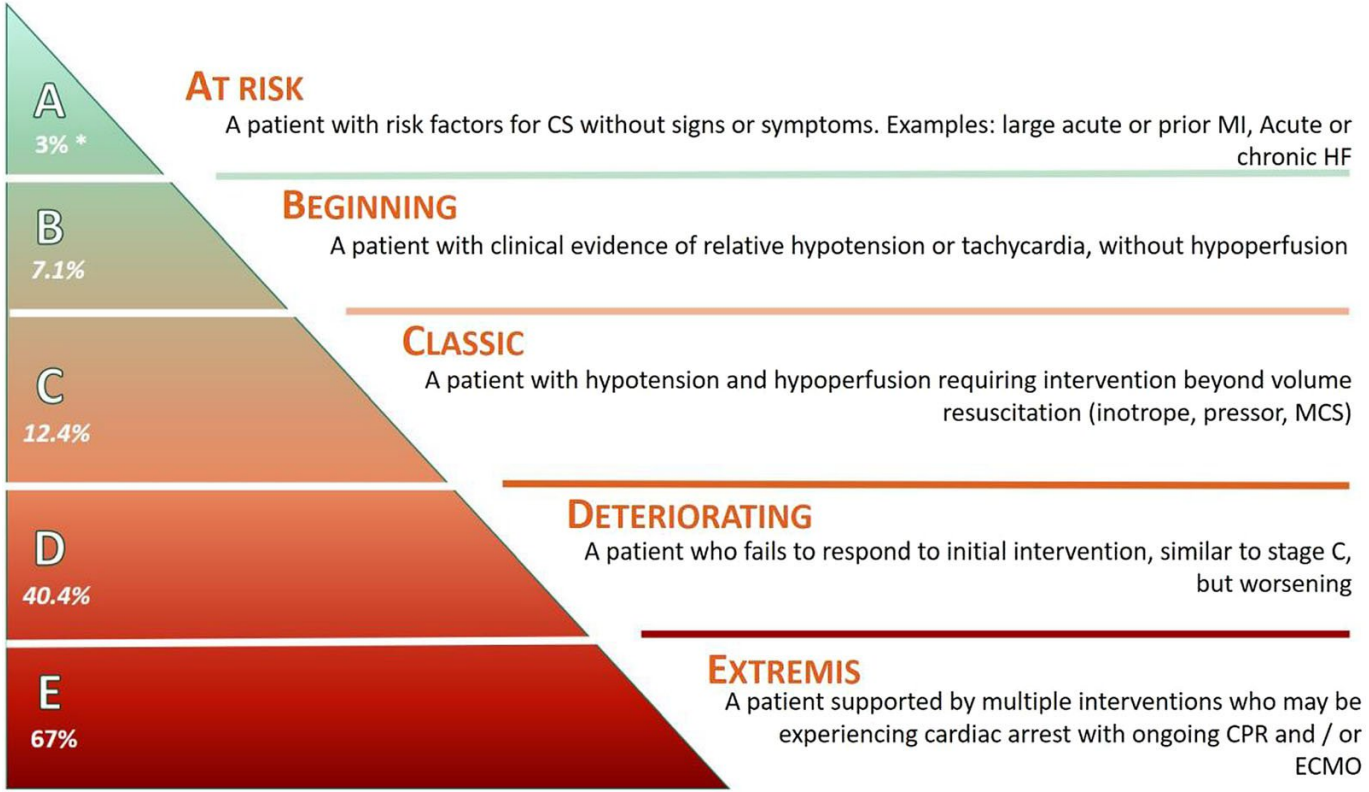

Conclusions

- ✓ Cardiogenic shock is a heterogeneous and dynamic condition representing a major health challenge
- ✓ A one-size fits all approach to CS is inadequate for addressing the needs of this grievously ill patient population
- ✓ The SCAI shock classification system with a multi-domain methodology allows for flexibility in incorporating the diversity of presentation
- ✓ Improved clinical characterization and risk assessment of patients with cardiogenic shock may facilitate more effective triage and clinical care, clinical, and translational investigations of this disorder, and ultimately the development of more targeted therapies