

Defining Cardiogenic Shock

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Cardiogenic Shock Working Group (CSWG)

The mission of the CSWG is to improve clinical outcomes for cardiogenic shock by promoting rigorous scientific investigation inclusive of a prospective multicenter registry to generate real-world evidence for this deadly problem



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Cardiogenic Shock

>50% in-hospital
Mortality rate





Prolonged MCS without a clear Transition Bridge





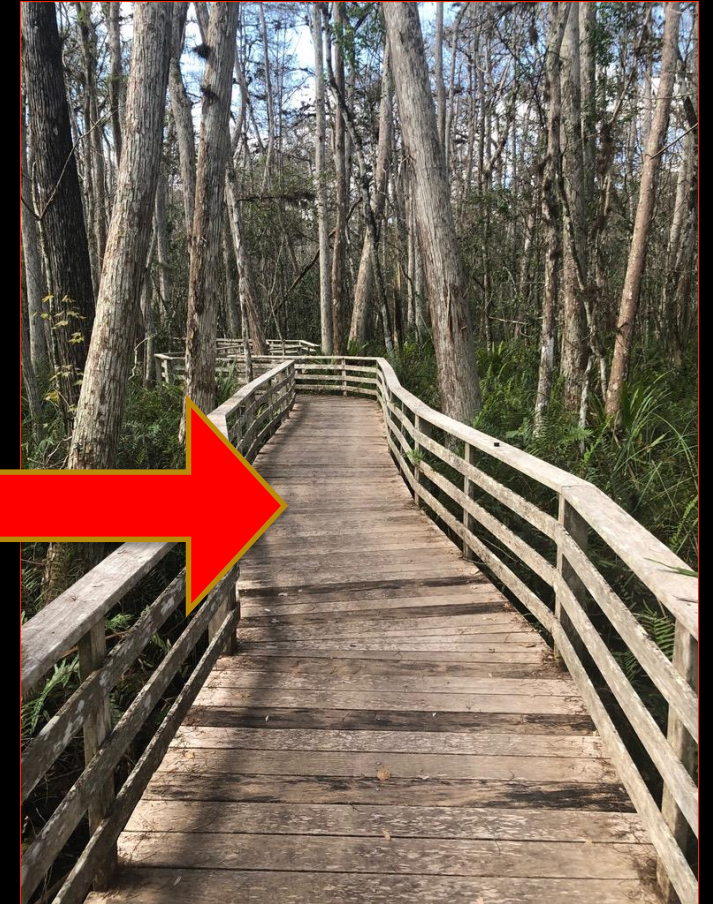
Guided approach:

- Early Recognition
- Assess shock severity
- LV/ RV or both
- Timely management

Multi-disciplinary care

Exit strategy

- Recovery
- Replacement
- Palliation



Key Considerations in the Diagnosis & Management of Cardiogenic Shock



Defining Cardiogenic Shock

- A Cardiac disorder presenting with:
 - SBP <90mm Hg
 - CI <1.8 L/min/m² without hemodynamic support
 - CI <2.2 L/min/m² with hemodynamic support
 - PCWP >15

Clinical Definition	SHOCK Trial ^{9*}	IABP-SHOCK II†	ESC HF Guidelines ¹⁵
Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion	Clinical criteria: SBP <90 mmHg for ≥30 min OR Support to maintain SBP ≥90 mmHg AND End-organ hypoperfusion (urine output <30 mL/h or cool extremities) Hemodynamic criteria: CI of ≤2.2 L·min ⁻¹ ·m ⁻² AND PCWP ≥15 mmHg	Clinical criteria: SBP <90 mmHg for ≥30 min OR Catecholamines to maintain SBP >90 mmHg 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 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

Early identification

Step 1: Suspect shock

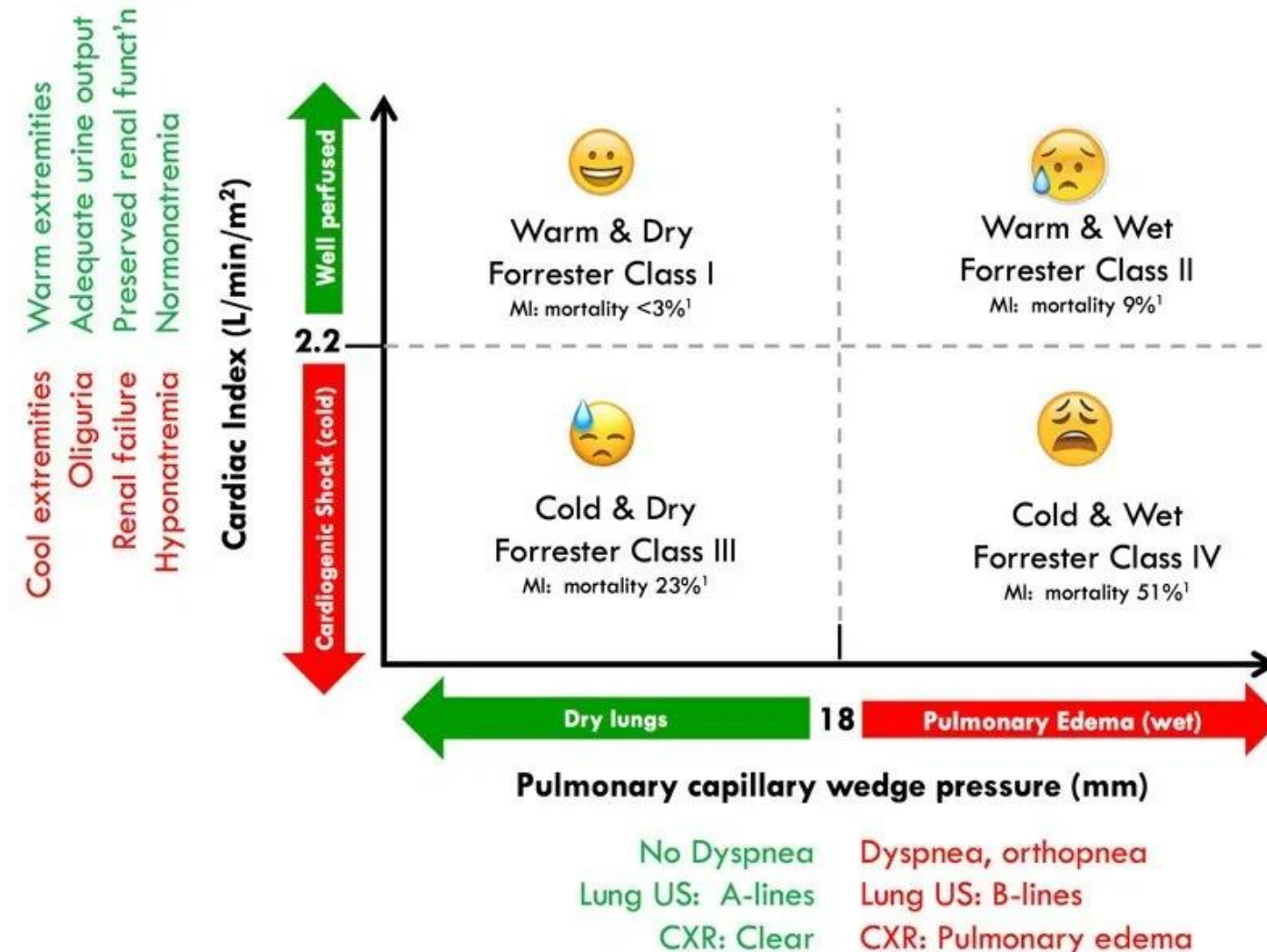
- SBP < 90 mm Hg for > 30 mins
- need for inotrope/ pressor/ IABP to maintain SBP > 90 mm Hg
- decrease in UO to < 0.5 cc/kg/h
- lactic acid > 2 mmol/L

Step 2: Confirm Cardiogenic shock

- Check ECG, Troponin, TTE, rhythm
- Swan/ PAC
 - $CI < 2.2$ and $PCWP > 15$ mm Hg
 - Check SVR and CVP

	CAUSED BY	SKIN	PCWP (PRELOAD)	CO	SVR (AFTERLOAD)
Hypovolemic	Hemorrhage, dehydration, burns	Cold, clammy	↓↓	↓	↑
Cardiogenic Obstructive	Acute MI, HF, valvular dysfunction, arrhythmia Cardiac tamponade, pulmonary embolism, tension pneumothorax	Cold, clammy	↑ or ↓	↓↓	↑
Distributive	Sepsis, anaphylaxis CNS injury	Warm Dry	↓ ↓	↑ ↓	↓↓ ↓↓

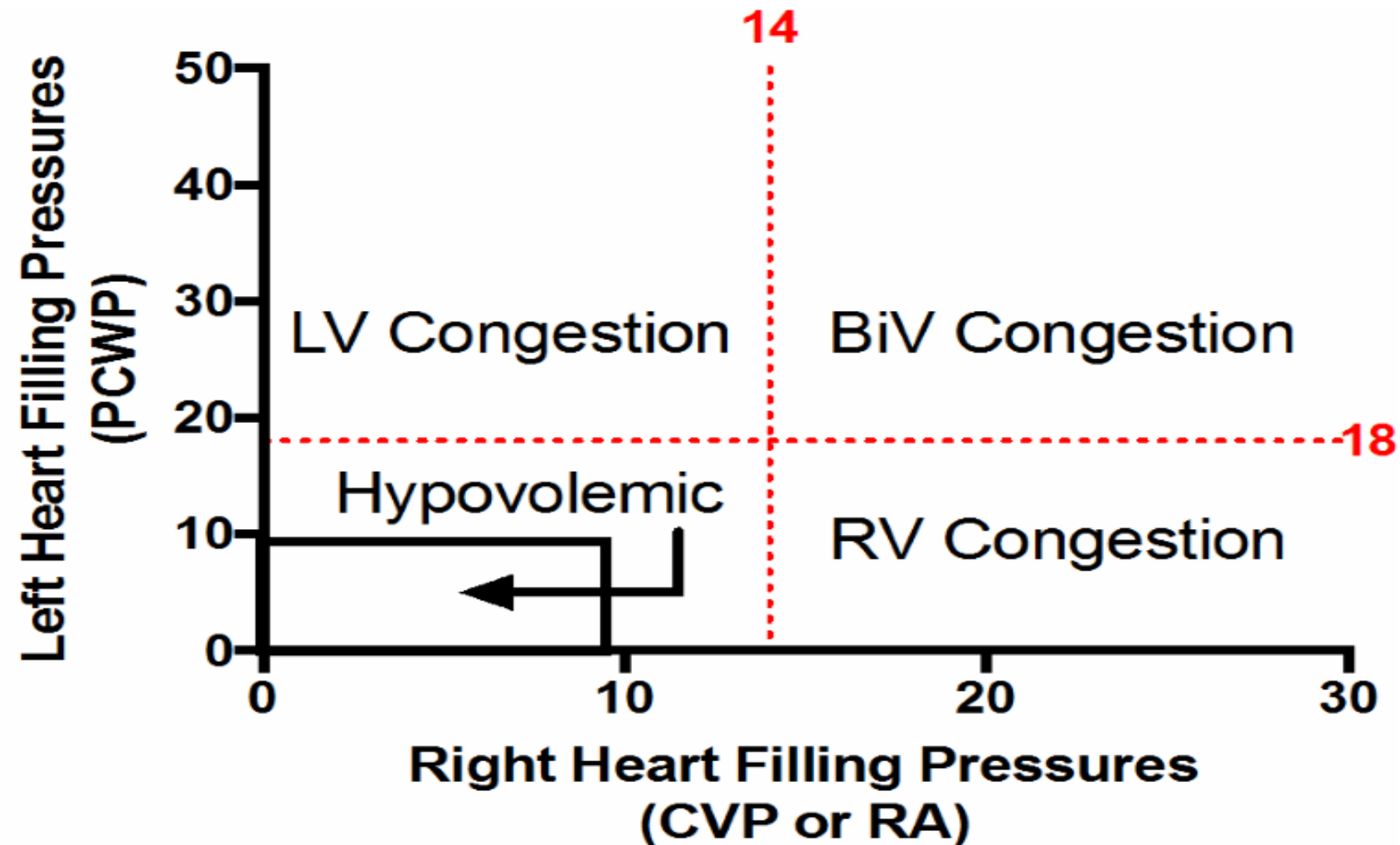
Step 3: Patient Assessment in Cardiogenic shock



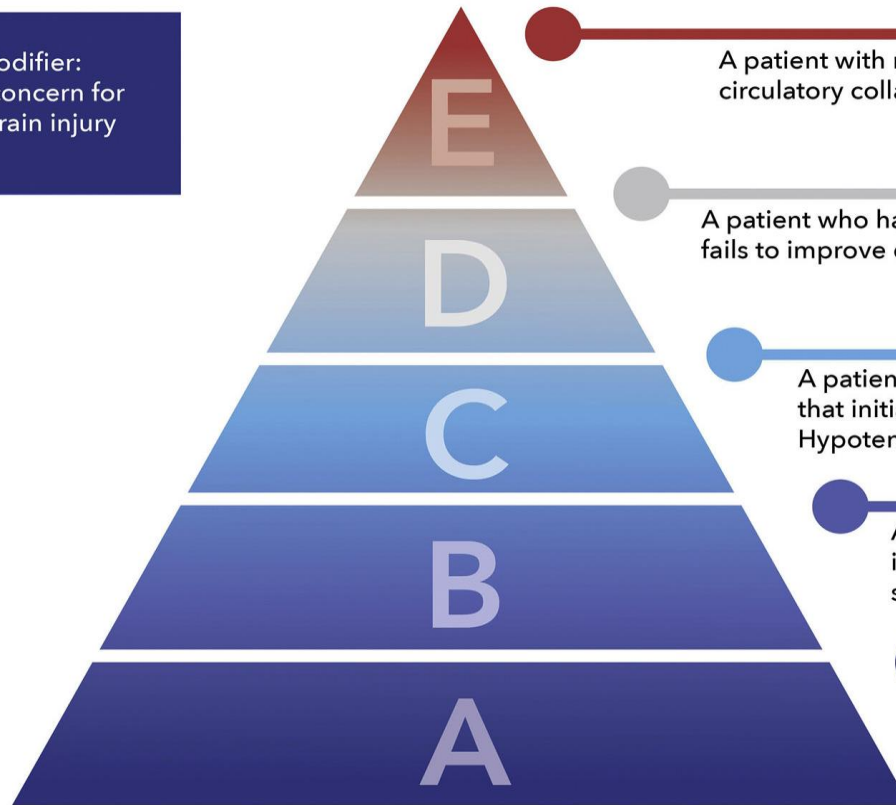
(1) Mortality numbers from Forrester 1976 PMID 790191. Mortality is probably lower today.

The Internet Book of Critical Care, by @pulmcrit

Hemodynamic Profiles in 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.

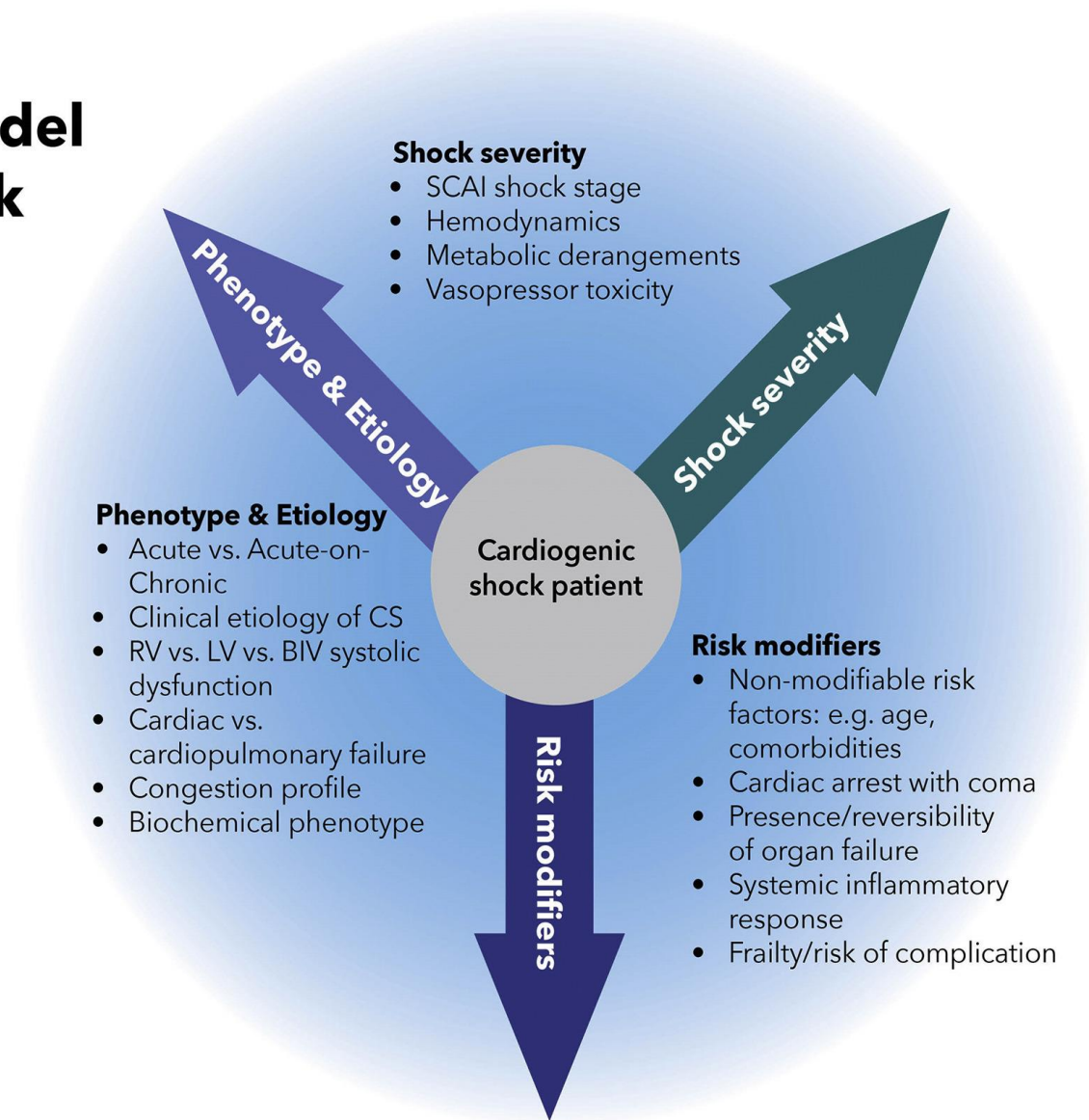
BEGINNING

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

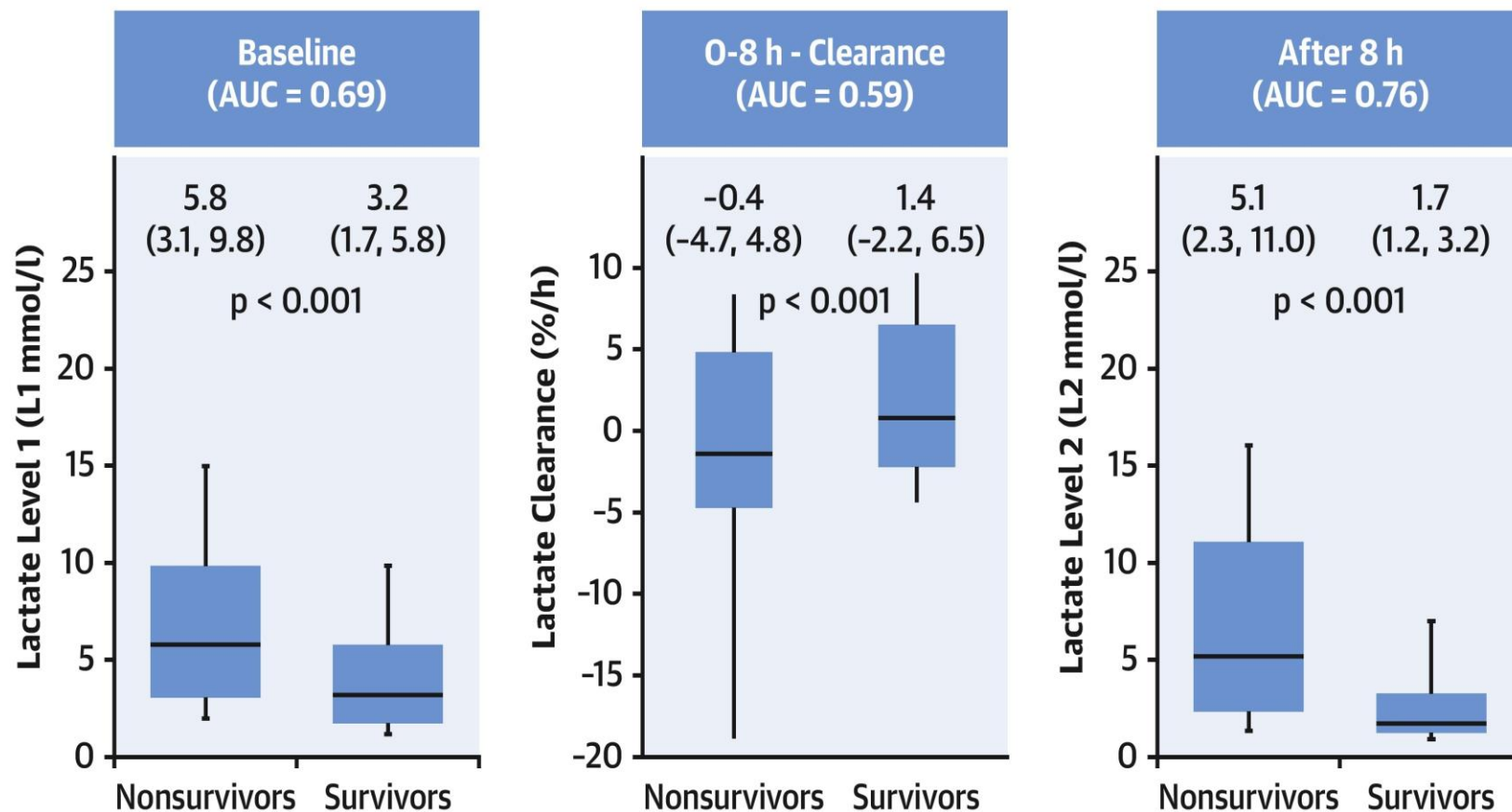
AT RISK

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).

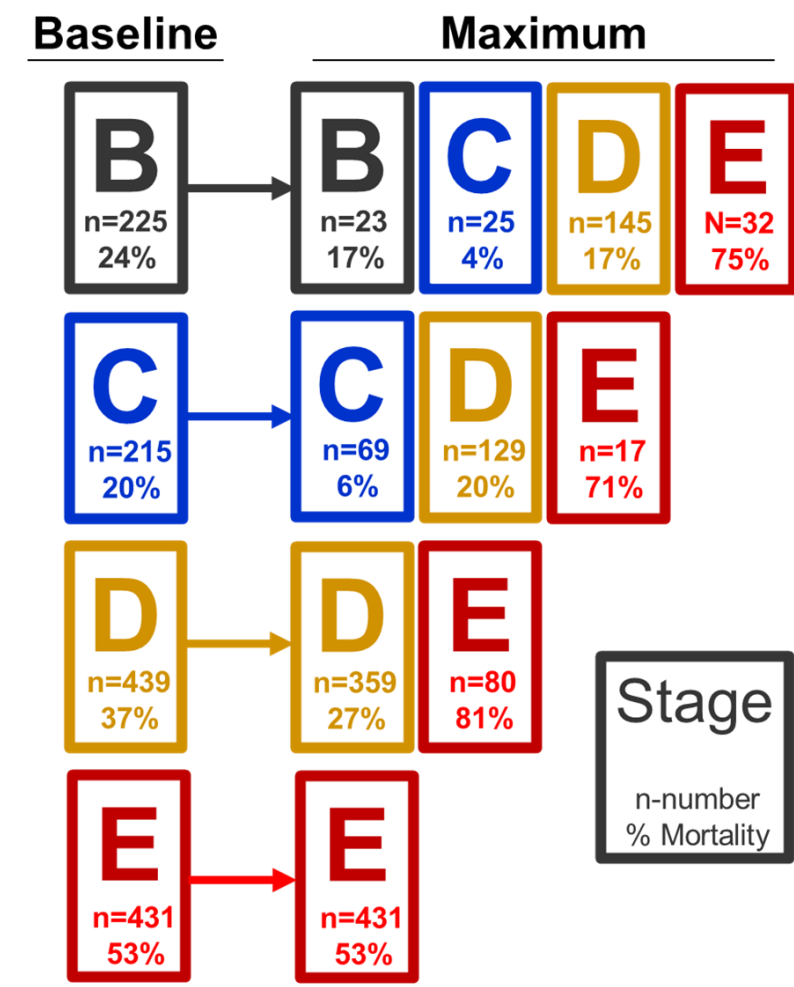
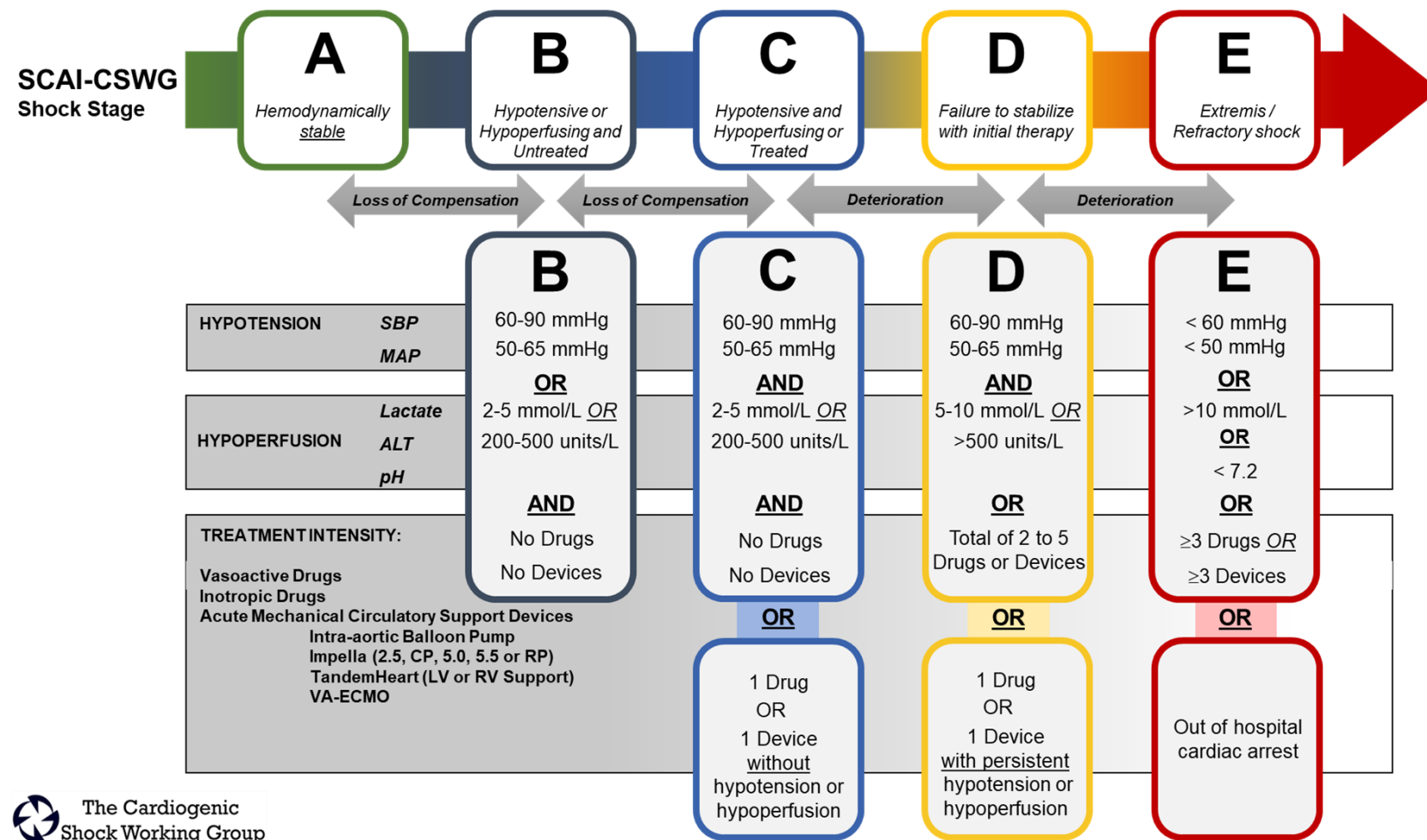
Proposed 3-axis model of cardiogenic shock evaluation and prognostication



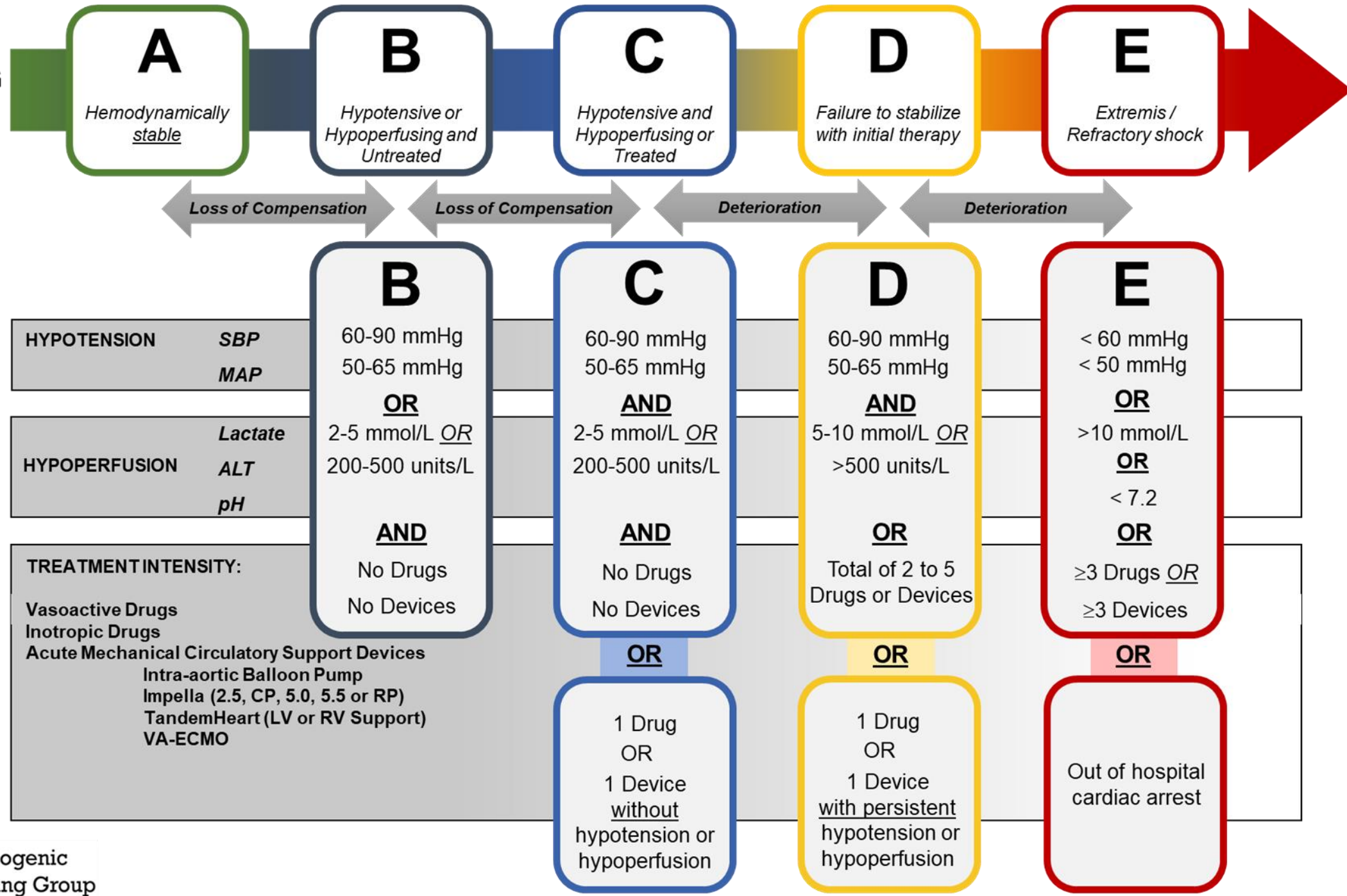
CENTRAL ILLUSTRATION: Arterial Lactate in Cardiogenic Shock



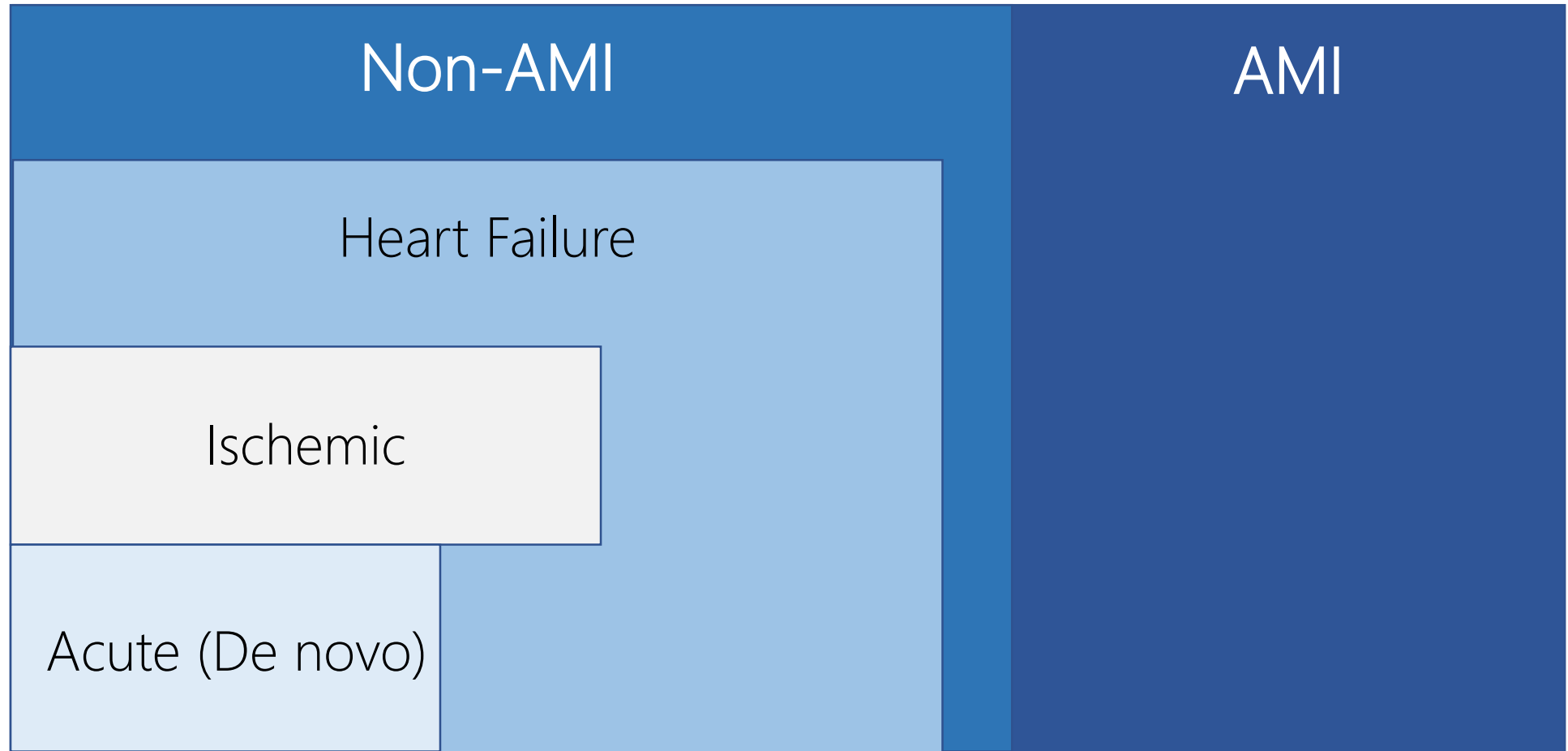
Fuernau, G. et al. J Am Coll Cardiol Interv. 2020;13(19):2208-16.



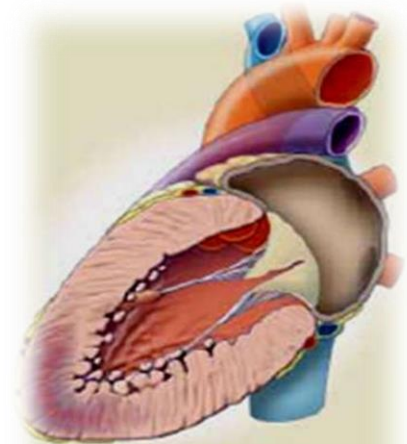
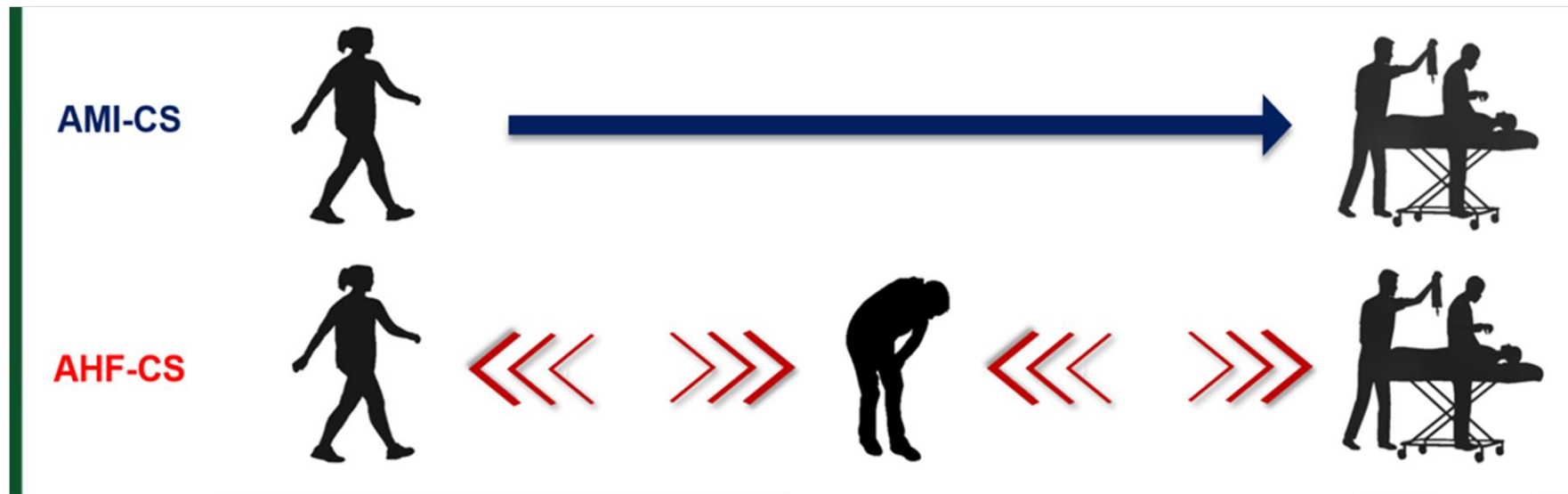
SCAI-CSWG
Shock Stage



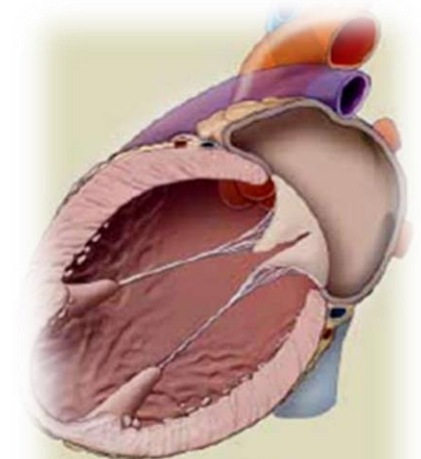
Phenotypes of Cardiogenic Shock



Not all shock is equal



Initial infarct

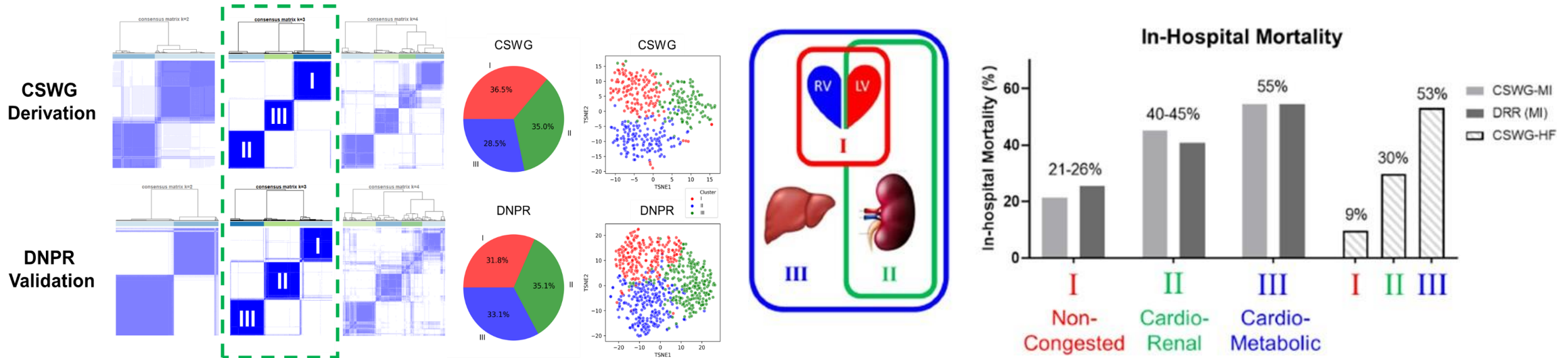


Global remodeling
(days to months)



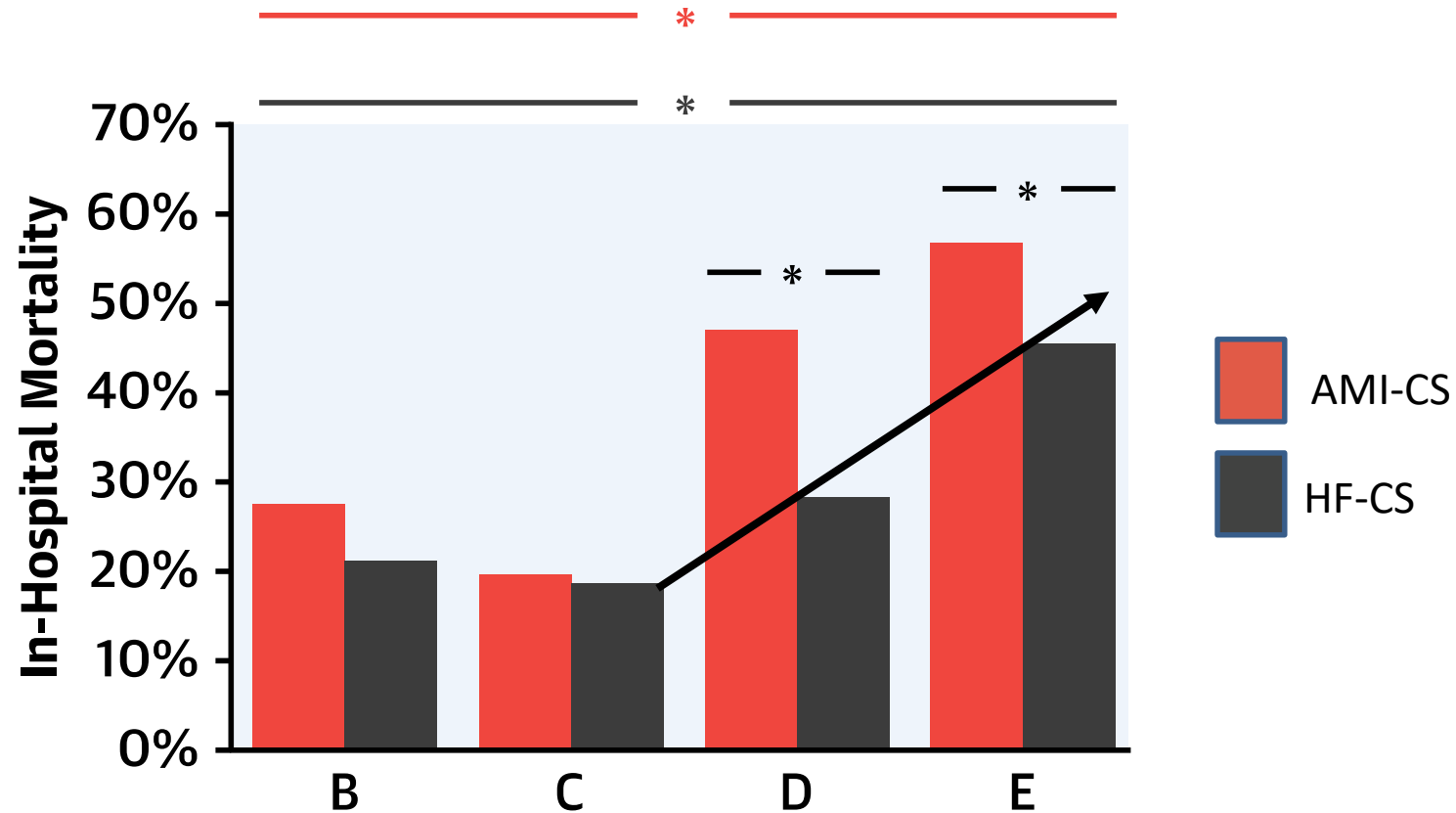
The Cardiogenic Shock Working Group

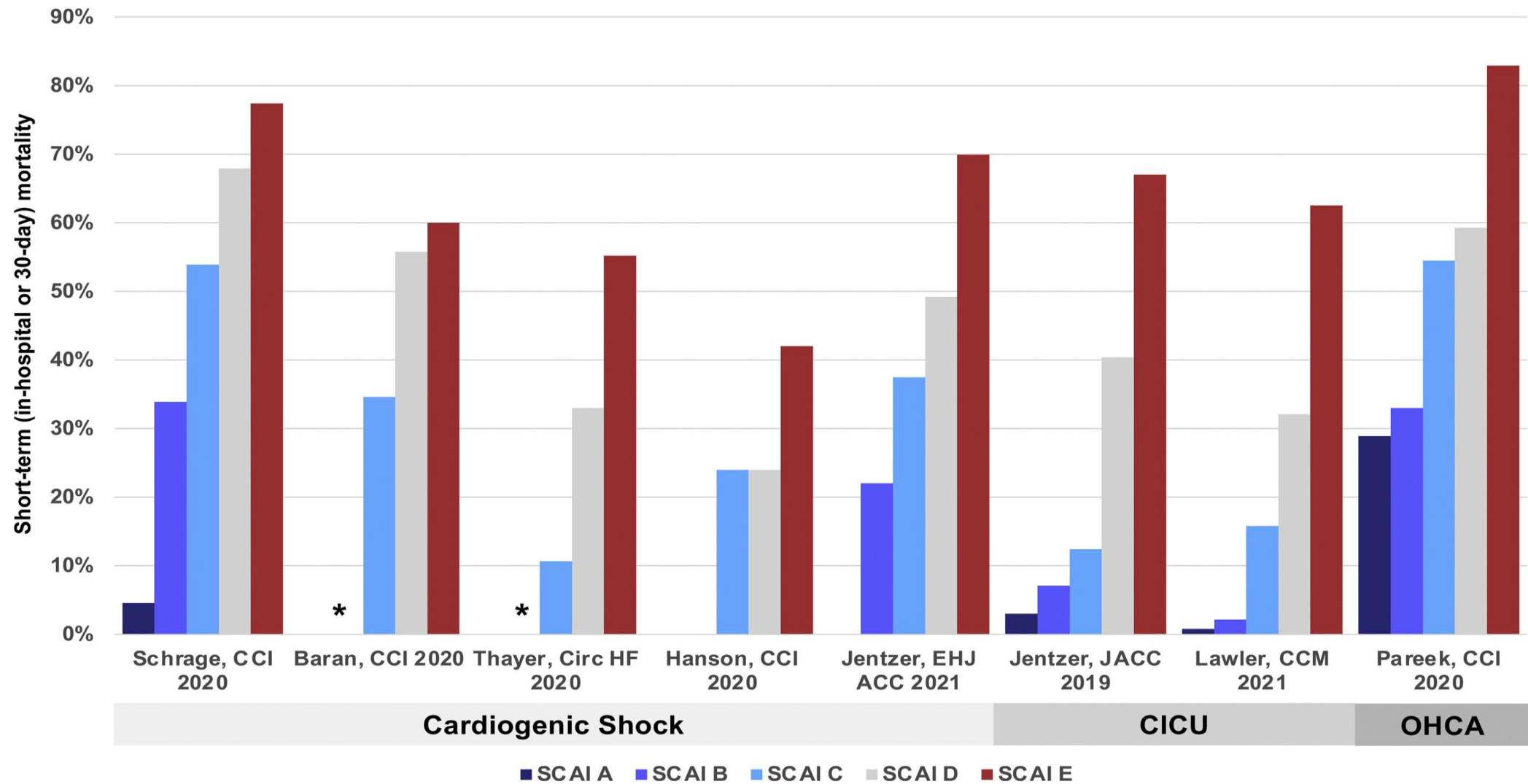
Machine learning algorithms identified 3 distinct phenotypes associated with increasing mortality in the CSWG dataset, which were then validated in collaboration with a Danish Shock Registry



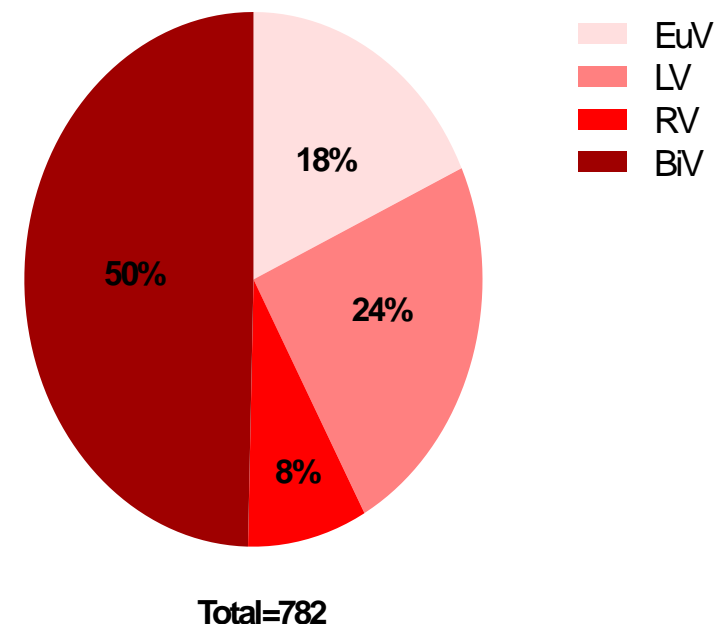
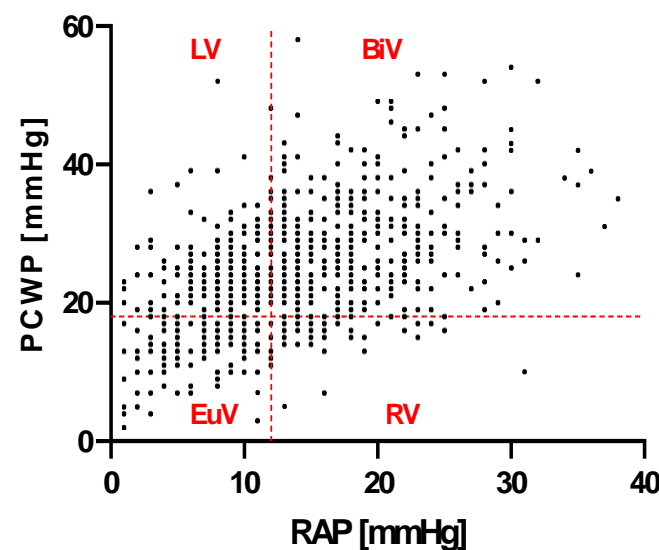
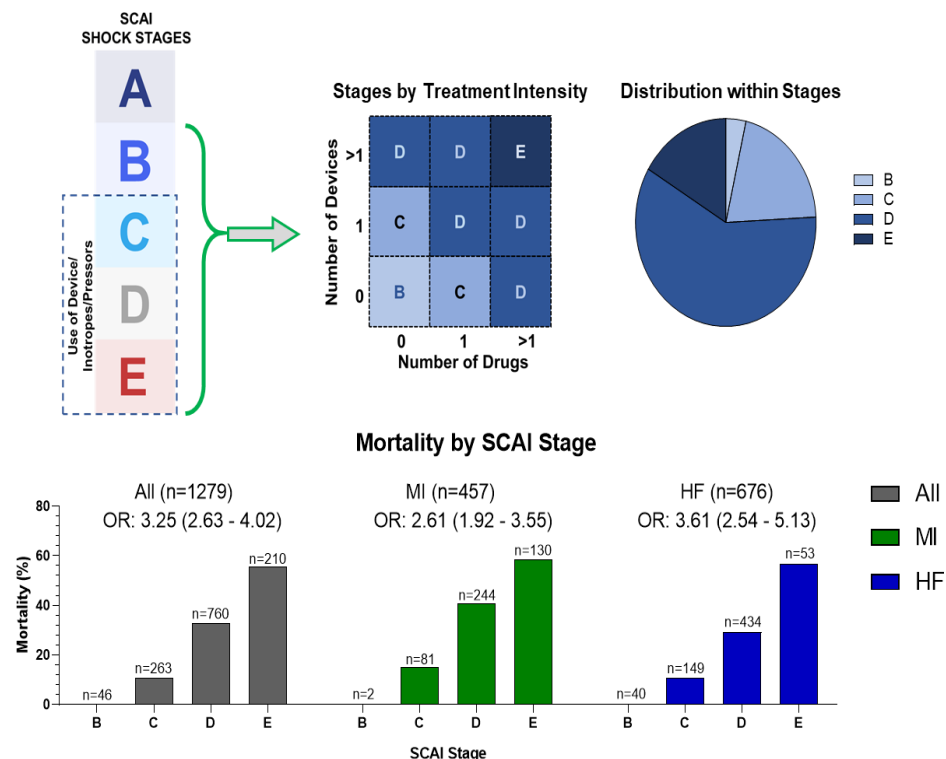
Zweck E, Kanwar M, Kapur N JAHA 2021

We must identify and treat CS in early stages





Biventricular congestion is commonly observed and associated with increased shock severity and higher in-hospital mortality. Congestion is an important target of therapy.



Hemodynamic Problem

Hemo-Metabolic Problem

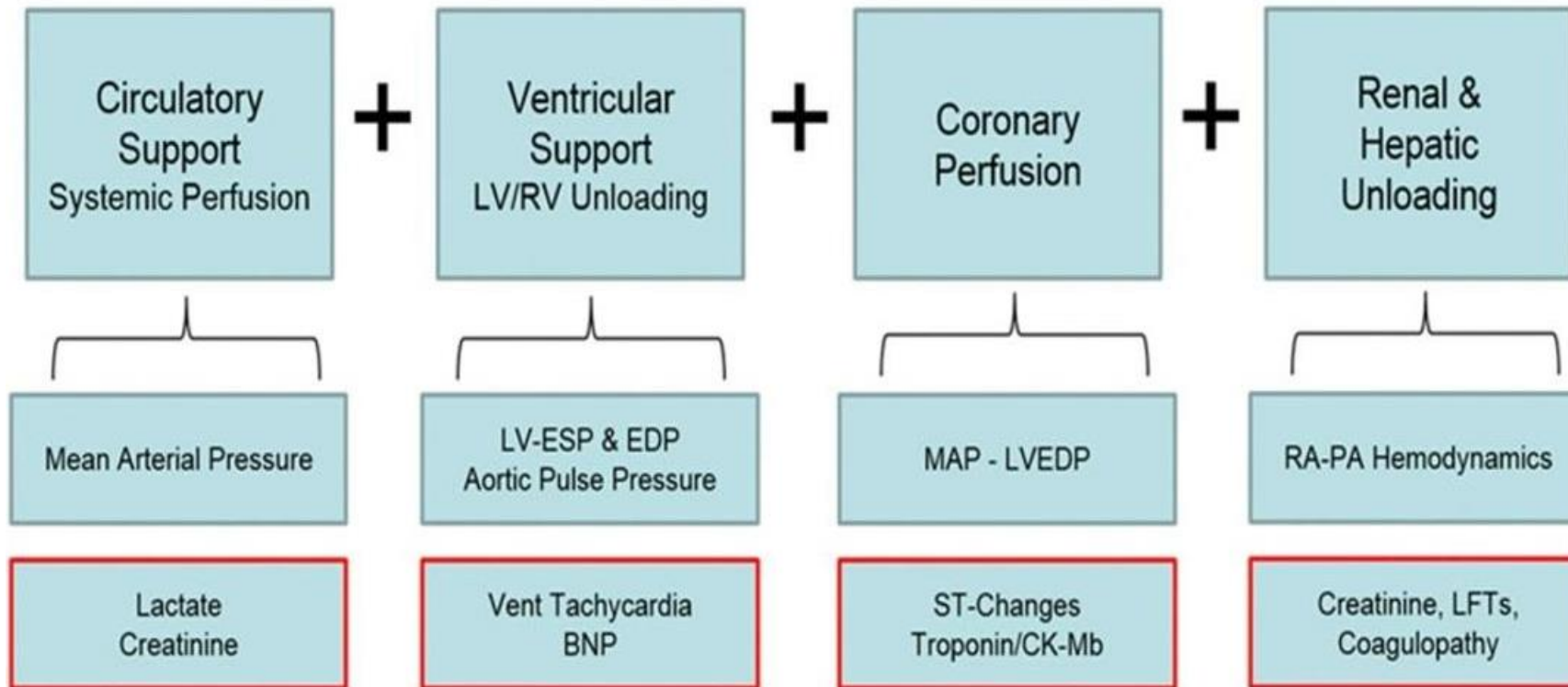
Recovery

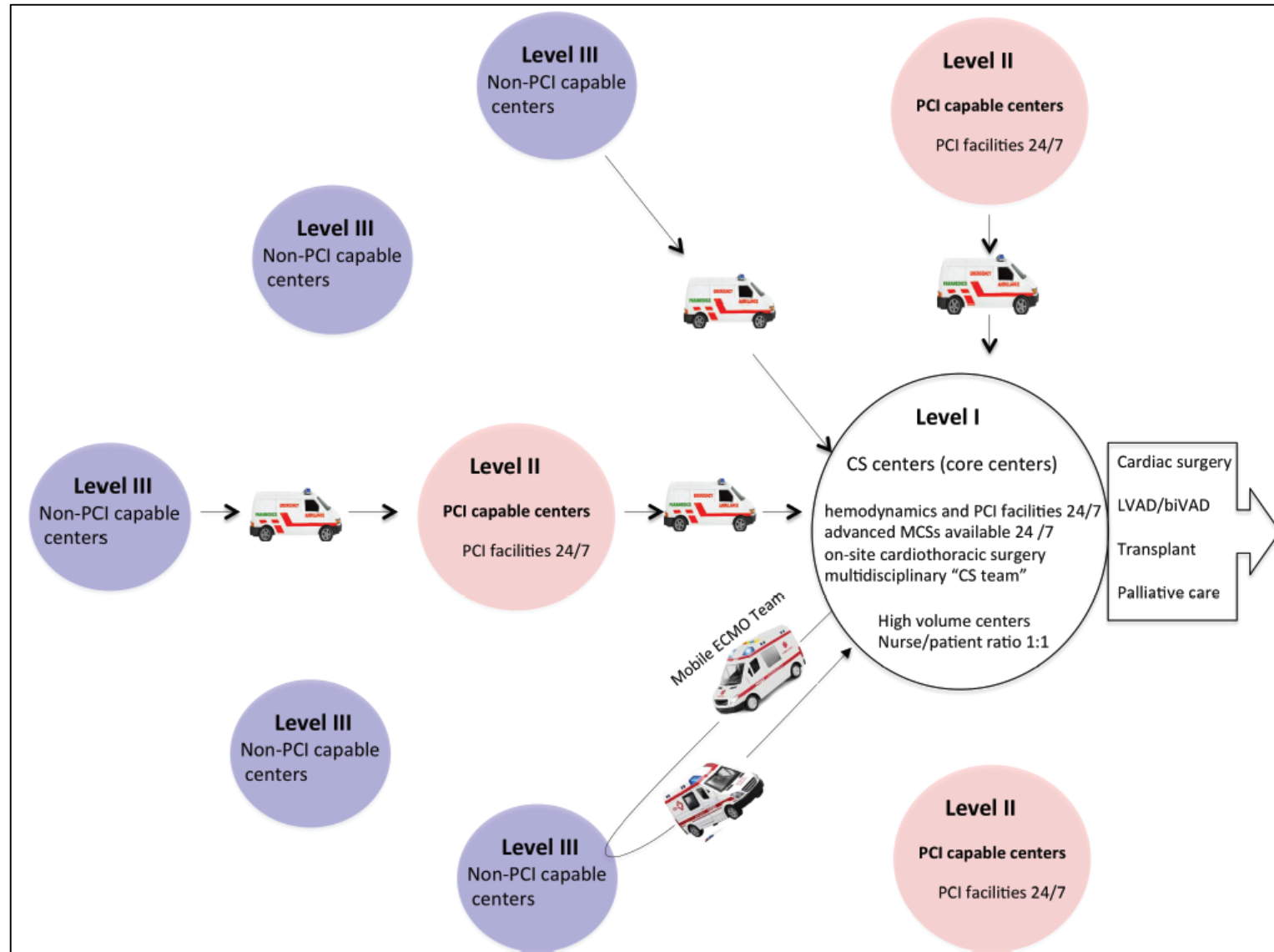
Time in Cardiogenic Shock

Death

Therapy: Hemodynamic Support

Therapy: Multi-organ Support



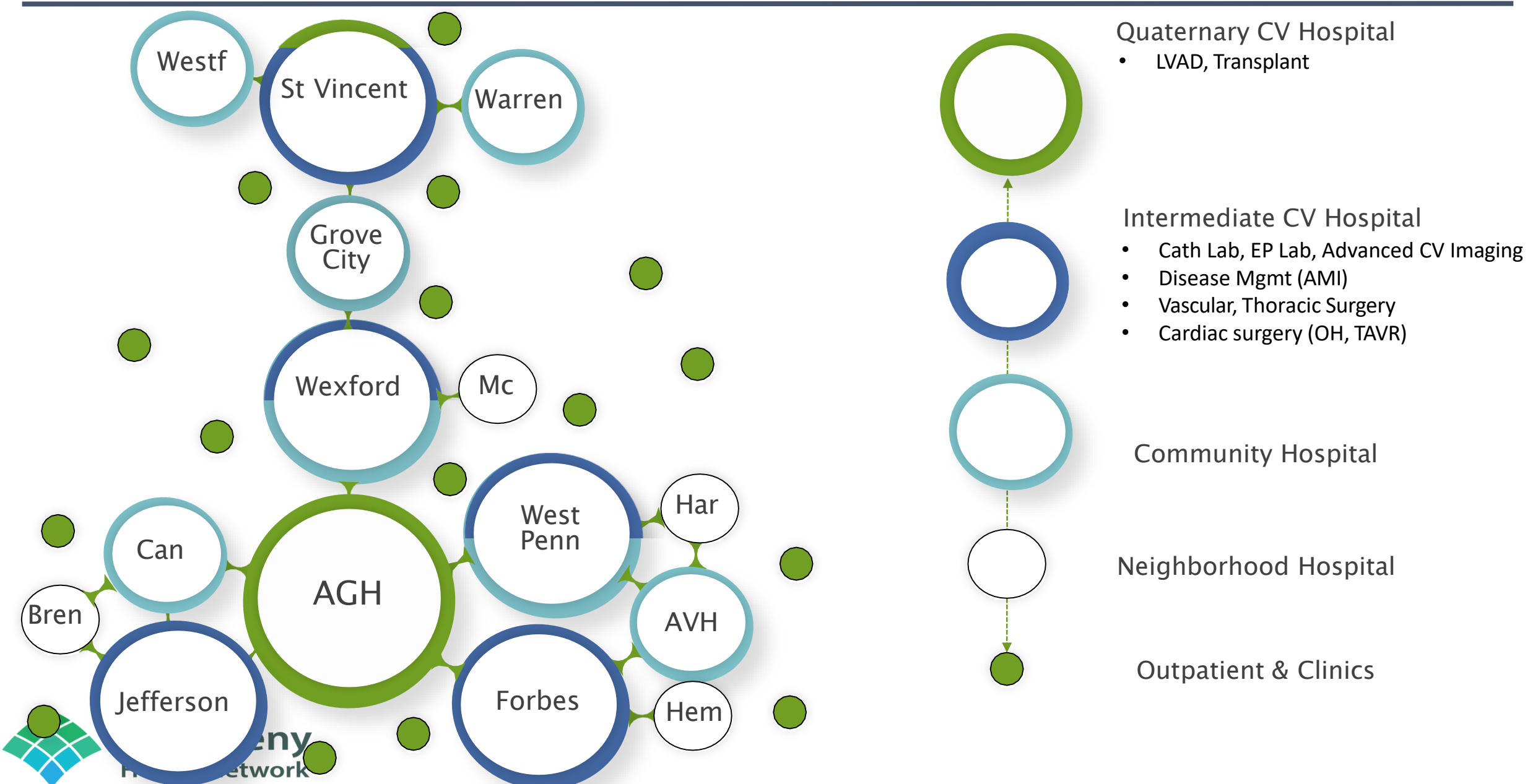


When to transfer a patient to a 'hub' hospital

- Escalating dose of 1st pressor / inotrope
- Adding a second pressor or inotrope
- Not improving / worsening on temporary MCS
- Lactate > 2 or rising

@AGH

- About 200 patients/ year in CS
- 60% are transferred in from OSH
- 55% are non AMI shock
- 60% supported on temporary MCS
- Top 3 t-MCS devices used: Impella, IABP and ECMO
- Survival to hospital discharge = 63% (increased from 53% in 2020)



Thank you

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