2026 SCAI SHOCK Classification Expert Consensus Update: Fine-Tuning the Stages and Incorporating Specific Patient Populations and Clinical Phenotypes

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Main text: 7453 words

Figures: 10

Tables: 6

Introduction

Since its introduction in 2019, the SCAI SHOCK stage classification system has seen increased adoption across diverse clinical settings, valued for its simplicity, ease-of-use, and ability to meaningfully discriminate patient risk across the spectrum of cardiogenic shock (CS).[1,2] Further, the SCAI SHOCK classification has been widely accepted and utilized in clinical, regulatory, and research settings, including both observational studies and clinical trials. A defining and essential feature of the SCAI SHOCK stage classification has been and must remain that mortality increases incrementally as patients progress through advancing stages of CS, reflecting a greater degree of shock severity and associated risk. Real-world experience, however, has highlighted areas for refinement, balancing ease of use in real time clinical settings and the rigor suitable to support research and regulatory applications, which may require supplementary granular elements to facilitate comparability between data sets.

The goals of this document are as follows: (1) to clarify and add granularity to definitions of SCAI SHOCK stages in response to practical questions encountered in varied clinical environments, (2) to facilitate use of SCAI SHOCK staging across various disease states and phenotypes by highlighting differences between these settings, and (3) to examine the perspectives of differing clinicians that encounter CS across the spectrum of care. The overall purpose of this document is to improve the classification system's usefulness for both clinical practice and research and re-affirm consensus opinion across broad stakeholders after evaluating the plethora of interim data, while preserving the classification system's consistency, clarity and rapid usability.

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Methods

This document has been developed according to SCAI Standards and Guidelines Committee policies for writing group composition, disclosure and management of relationships with industry (RWI), internal and external review, and organizational approval.[3] The writing group has been organized to prioritize diversity of perspectives and demographics, ensure multi-stakeholder representation, and maintain an appropriate balance of RWI. Relevant author disclosures are included in **Supplemental Table 1**. Before the appointment, members of the writing group were asked to disclose financial and intellectual relationships from the 12 months before their nomination. A majority of the writing group disclosed no relevant, significant financial relationships. Financial and intellectual disclosure information was periodically reviewed by the writing group during document development and updated as needed. SCAI policy requires that writing group members with current, relevant financial interests be recused from participating in related discussions or voting on recommendations. The work of the writing committee was supported exclusively by SCAI, a nonprofit medical specialty society, without commercial support. Writing group members contributed to this effort on a volunteer basis and did not receive payment from SCAI.

Narrative literature searches were performed by group members designated to lead each section and initial findings were synthesized in section drafts authored primarily by the section leads in collaboration with other members of the writing group. The content of this document was iteratively discussed by the full writing group in a series of five virtual meetings.

The draft manuscript was peer reviewed in July, 2026 and the document was revised to address pertinent comments. The writing group unanimously approved the final document. The

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SCAI Executive Committee and Standards and Guidelines Committee endorsed the document as official society guidance in [MONTH, 2026].

SCAI statements are primarily intended to help clinicians make decisions about treatment options. Clinicians also must consider the clinical presentation, setting, and preferences of individual patients to make judgments about the optimal approach to treatment.

Updated SCAI Shock Stage Definitions

In pursuit of clarity and consistency, it is important to recognize that staging adaptations which define SCAI SHOCK stages based primarily on the intensity of therapies used to achieve hemodynamic stability introduce significant subjectivity. Variability in resources, clinical expertise, team preferences, and institutional practices can influence treatment decisions independent of patient-specific or disease-severity variables. In the absence of a standardized escalation framework, differences in the timing, sequence, and selection of vasoactive-inotrope therapies or mechanical circulatory support (MCS) alone or in combinations, therefore, should not solely determine SCAI SHOCK stage assignment. Instead, a synthesis of objective patient-level data including vital signs, physical exam, metabolic (biochemical) parameters, and hemodynamic data should serve as the foundation for staging and subsequent treatment decisions. (**Figure 1 and Tables 1 and 2**). We further propose a basic core dataset for rapid clinical assessment in cases of suspected CS that is broadly applicable to the clinical, research and quality objectives of pre-hospital, emergency room (ER), intensive care unit (ICU), Cath Lab, and operating room (OR) environments.[4] This includes heart rate, blood pressure, respiratory rate, oxygen saturation, physical assessment of pulmonary and systemic volume status and

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perfusion (recognizing limitations), and a focused point-of-care cardiac ultrasound (POCUS), lactate, and right heart catheterization as available.

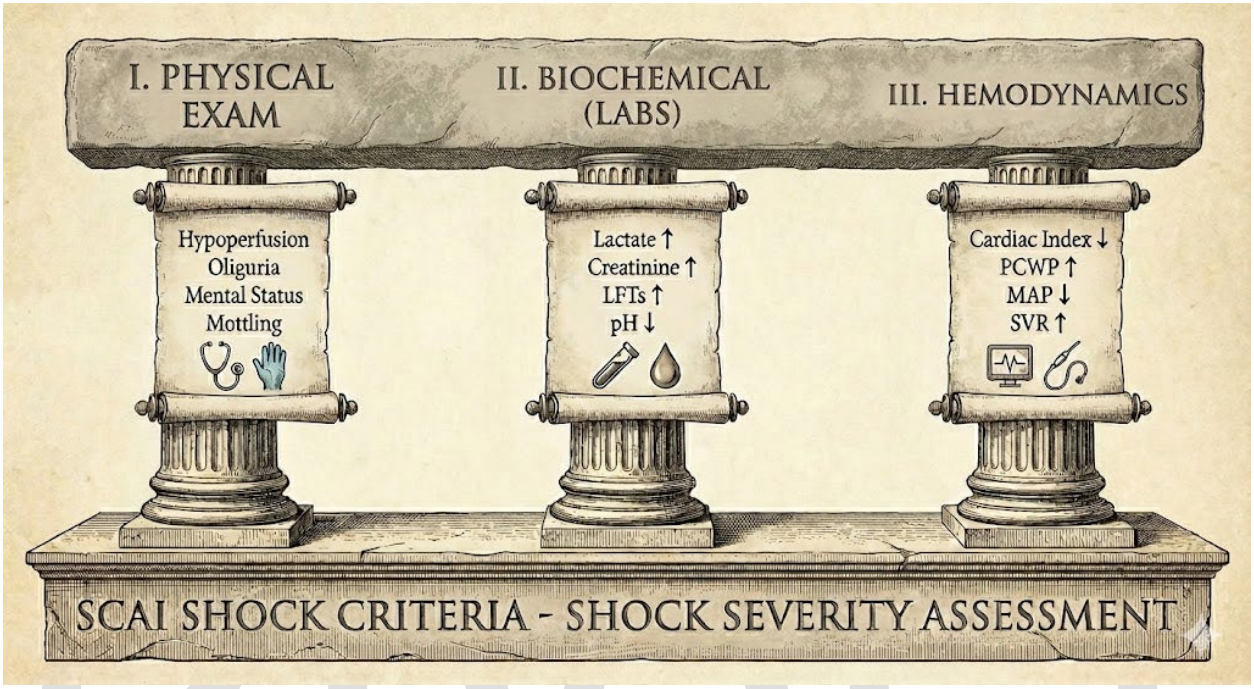

Figure 1: Central Figure SCAI SHOCK Objective Criteria in Context **NOTE: STILL IN DEVELOPMENT. WILL INCLUDE 3 ADDITIONAL COMPONENTS 1) SHOCK pyramid, 2) dynamic nature of CS (reuse of Fig 3 from 2022 publication) and elements of Table 4 below.**

76 **Table 1**: Pillars of Evaluation to Assess SCAI SHOCK Stage

Physical Exam	**Biochemistry (Labs)**	**Hemodynamics / Supports**
Cool extremities	Elevated lactic acid / anion gap	Narrow pulse pressure < 25% of SBP
Pale / cold / mottled skin	Elevated serum creatinine / BUN	Heart rate >=100 bpm
Delayed capillary refill		Vasoactive drug requirement (VIS Score)
Mental status change	Elevated transaminases (AST/ALT)	Systolic blood pressure < 90 mmHg or > 30 mmHg drop from baseline
Oliguria	Elevated BNP	MAP <60 mmHg
Tachypnea (increased respiratory rate)	Acidemia (low pH) on arterial blood gas analysis	Low cardiac index < 2.2 L/min/m2
Lung congestion / low O2 saturation	Reduced serum bicarbonate / base excess	Elevated filling pressures PAWP / PADP > 15 CVP > 10 Cardiac Power Output <= 0.75

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Weak pulses	Low central or mixed venous oxygen saturation	Requiring pressors or inotropes to maintain clinical stability
Edema / volume overload		Requirement for new mechanical circulatory support based on objective data of worsening severity

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80 **Table 2**--- Descriptors of Shock Stages: Physical Exam, Biochemical Markers, and Hemodynamics

Stage	**Description**	**Physical Exam / Bedside Findings**		**Biochemical Markers**		**Hemodynamics**	
		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**.	**Normal JVP** **Warm and well-perfused** • Strong distal pulses	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

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	These patients may include those with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic heart failure symptoms.	• Normal mentation					• 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 that manifests with **hypoperfusion that requires one intervention (pharmacological or mechanical) beyond volume resuscitation**.	**Volume Overload**	Looks unwell Acute alteration in mental status Feeling of impending doom Cold and clammy	**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/m²** • **PCWP > 15 mmHg**	

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	These patients typically present with relative hypotension (but hypotension is not required).		Extensive rales Ashen, mottled, dusky, or cool extremities Delayed capillary refill Urine Output < 30 mL/h				
D Deteriorating	A patient that 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 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	**Lactate ≥ 8 mmoL/L***	CPR (A-Modifier) Severe acidosis	**Profound hypotension despite maximal hemodynamic**	Need for bolus doses of vasopressors

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			Collapse Multiple Defibrillations		• pH ≤ 7.2 • Base deficit > 10 mEq/L	**support**	

BNP, B-type natriuretic peptide; CPR, cardiopulmonary resuscitation; CVP, central venous pressure; GFR, glomerular filtration rate; JVP, jugular venous pressure; LFT, liver function tests; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; SVP, systolic ventricular pressure.

***Stage E prospectively is a patient with cardiovascular collapse or ongoing CPR.**

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An important consideration is that the SCAI SHOCK stages are intended for patients presenting to the hospital with an acute cardiac event or for a defined high-risk cardiac procedure. The SCAI SHOCK classification is not intended to replace existing chronic HF classification systems for stable ambulatory patients such as Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) or the New York Heart Association classification.

SCAI A

The 2022 SCAI SHOCK stage classification update defined stage A as "stable patients who have acute cardiac diagnoses that place them at risk for cardiogenic shock" and included patients "with large acute myocardial infarction or prior infarction and/or acute or acute-on-chronic HF symptoms".[2] While extensive registry studies have validated and demonstrated the prognostic value of SCAI stages B through E, comparatively little evidence has focused on SCAI stage A. Often SCAI A is defined by the lack of criteria for SCAI B or higher, but with significant cardiac risk substrate. Available studies in unselected CICU populations have shown low overall risk of progression to shock or death in the SCAI A cohort, suggesting the need to better identify relevant risk factors to help classify which patients should be more appropriately assigned to SCAI SHOCK stage A. Given the dynamic nature of CS, including transitions between stages, it seems appropriate that SCAI A be refined to identify vulnerable patients at clear and actionable risk, as opposed to theoretical risk. By redefining SCAI A, we put forth the possibility of shifting the therapeutic paradigm from *reversing* established CS to *preventing* the onset of the CS cascade in some SCAI A patients. Such clearly defined SCAI A populations may well be suited for

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the investigation of strategies and technologies to prevent progression to SCAI B pre-shock or SCAI C shock, or catastrophic decompensation to SCAI E.

Although the spectrum of potential SCAI A patients includes a broad array of cofactors as outlined in **Table 3**, the essence of SCAI A is best conceptualized in **Figure 2**. SCAI A describes patients with a baseline physiological state that is already compromised ("at risk") or patients with very poor cardiovascular reserve. **Figure 2** illustrates the intersection between a vulnerable baseline state, the physiologic stress imposed by a disease state or invasive procedure and the additional effect of unforeseen but not uncommon acute and procedural events. The patient with preserved physiologic reserve might tolerate these combined stressors without deterioration, whereas a SCAI A patient with limited reserve may be more likely to progress to SCAI B or more advanced shock. Importantly, this definition does not rely on subjective or treatment-based criteria but is instead grounded in objective patient physiology, which is a recurring and central focus of this document.

The authors propose several key phenotypes of SCAI A patients, while recognizing that additional categories will likely emerge over time. The phenotypes discussed below include selected patients with coronary artery disease (CAD), atrial or ventricular tachyarrhythmias, valvular heart disease, and acute or advanced HF.

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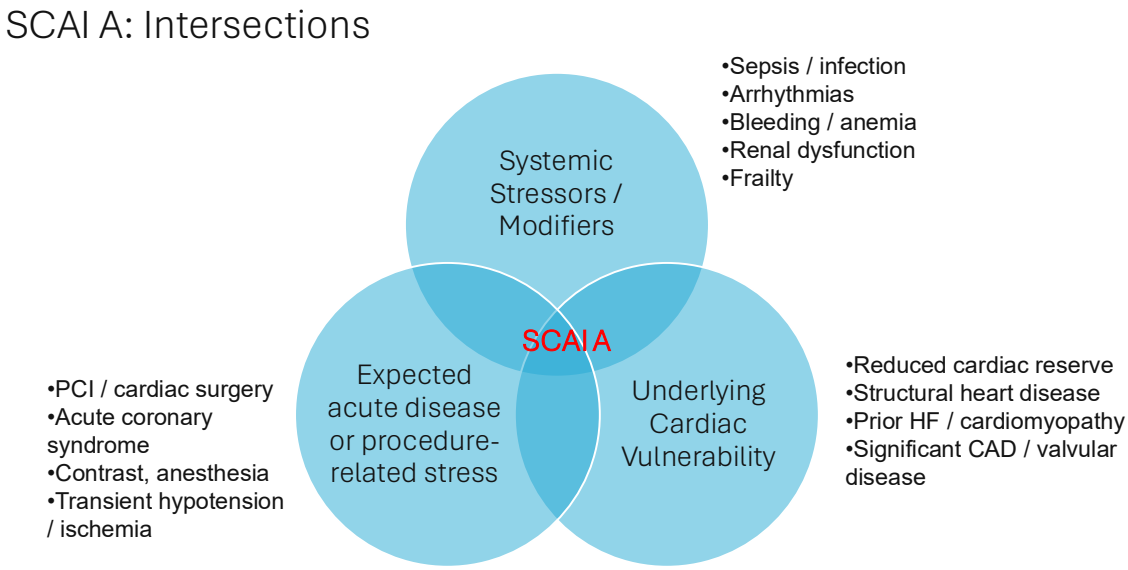

Figure 2: SCAI SHOCK Stage A Intersections

CAD Risk Group: Acute syndromes and chronic disease

As noted in prior SCAI SHOCK statements, patients with acute coronary syndromes with large territories at risk may have adequate perfusion and blood pressure immediately after intervention but be at risk of deterioration in the hours following the procedure. Risk scores for prediction of CS in patients with acute coronary syndromes can separate patients into high-risk (typically those with poor reperfusion, significant vital sign abnormalities, and evidence of HF) and low-risk (stable patients without the above features) groups.[5,6]

The other subset of patients – typically termed high-risk PCI – is those with chronic significant CAD, reduced ejection fraction, adverse hemodynamics, and multiple significant cardiac and non-cardiac comorbidities, who may be particularly vulnerable to the development of CS or cardiac arrest from recurrent procedural ischemic insults.[7,8] Further defining the

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characteristics of the most vulnerable SCAI A CAD cohort and elucidating ways to reduce procedural risk has been the subject of intense investigation in registry analyses[9,10] as well as the recent CHIP-BCIS3[11] and the ongoing PROTECT IV (NCT04763200) randomized trials.

TABLE 3: Contributing Factors affecting the SCAI A Patient (examples, not exhaustive list)

Category	Examples
Non-Cardiac Comorbidities	• Advanced Lung Disease • Kidney Disease • Vascular Disease • Anemia • Frailty
Procedural Components	• Coronary revascularization (PCI, CABG) • Arrhythmia ablation(s) • Catheter-based valvular heart interventions • Advanced heart failure interventions • Valvular and other cardiac surgery
Cardiac Vulnerability	• Low LVEF • Reduced cardiac index • Pulmonary or systemic congestion • Pulmonary hypertension • Right heart failure • Severe valvular heart disease • Prior HF/cardiomyopathy • Critical multivessel CAD • ACS involving large myocardium at risk

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Clinical features that may indicate advanced disease progression or intolerance to invasive procedures. These factors should be considered when evaluating candidacy for further interventions or advanced therapies.
Abbreviations: PCI = percutaneous coronary intervention; CABG = coronary artery bypass grafting; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; HF = heart failure; LVEF = left ventricular ejection fraction.

Electrophysiology Risk Group

Patients undergoing radiofrequency catheter ablation (RFA) for VT often have underlying myocardial substrates and comorbid conditions, along with procedural factors such as use of general anesthesia and the need for persistent time in the targeted arrhythmia, which render them vulnerable to the development and/or delayed recognition of CS. The heart team, led by the electrophysiologist, can assess when risk benefit ratio favors interventions to prevent marked deterioration. Risk scores such as the PAINESD score have been developed to assess risk in such situations.[12]

Structural Heart Disease Risk Group

Patients with severe aortic stenosis or mitral, tricuspid, or aortic regurgitation undergoing trans-catheter valvular intervention may be at elevated risk for CS, particularly in the context of decompensated HF and/or right ventricular (RV) dysfunction, and likely merit SCAI A designation.[13] These patients frequently have multiple severe cardiovascular and non-cardiovascular comorbidities, including pulmonary hypertension, renal insufficiency, frailty, and hepatic dysfunction, that further elevate procedural risk.

Heart Failure Risk Group

Heart failure is a very heterogeneous group with distinctions between acute *de novo* heart failure CS, chronic heart failure (well compensated) as well as those with acute

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exacerbations of chronic heart failure. While all patients with heart failure have some risk of developing shock, we choose to limit SCAI A to patients with increased risk to avoid making the category so broad that it loses utility. **Figure 3** illustrates groups of particularly high risk who merit close observation and treatment. These factors overlap with the INEEDHELP mnemonic[14] and the forthcoming PREVENT 2-LATE mnemonic (Mehra et al, under review). These patients are admitted to the hospital for management of their condition but may appear deceptively stable. Regardless of compensation, chronic heart failure often leads to physiologic adaptations which allow normal or near-normal lactic acid and preserved blood pressure with significantly abnormal cardiac filling pressures and depressed cardiac output. Importantly, SCAI A is reserved for patients without hemodynamic instability (SCAI B) or hypoperfusion (which warrants SCAI C).

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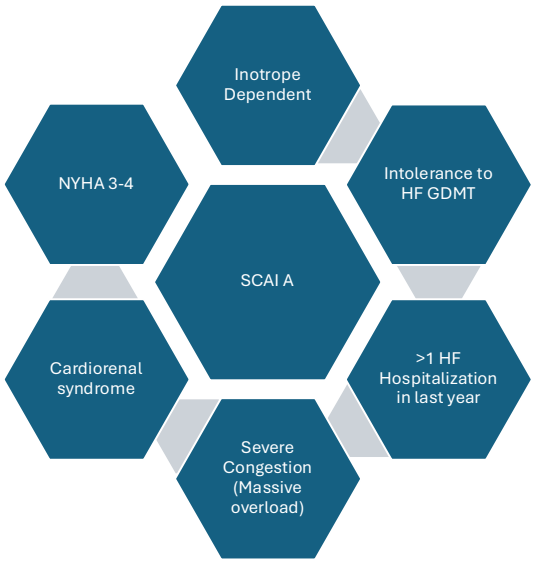

Figure 3: Additional factors which identify the admitted heart failure patient as SCAI A CS.

SCAI B

The hallmark of SCAI B shock is the activation of CS compensatory pathways which are evidenced by findings such as resting hypotension (SBP < 90, MAP < 60, > 30 mmHg drop from baseline blood pressure) and/or sinus tachycardia (heart rate > 100 beats per minute) due to compromised stroke volume, decreased cardiac output and/or clinical congestion, but without evidence of hypoperfusion as assessed by the integration of physical exam, hemodynamics and biochemical indices. These features are illustrated in **Figure 4.** SCAI B represents a high risk of progression to SCAI C and especially in the case of chronic HF, these compensatory mechanisms may fail with little warning, with the patient subsequently progressing to frank hypoperfusion which defines SCAI C.

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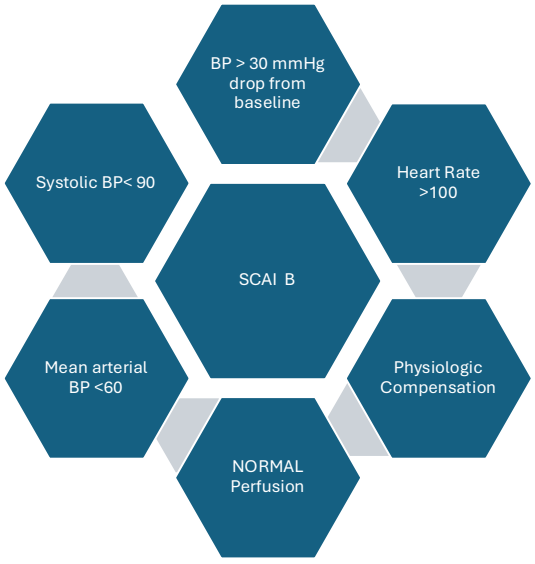

Figure 4: SCAI B Shock criteria

SCAI C

The dividing line which indicates presence of SCAI C or higher CS is evidence of end-organ hypoperfusion. Crucially, patients with SCAI C CS may transiently have normal or higher blood pressure due to effective compensatory mechanisms, resulting in the phenomenon of normotensive hypoperfusion which may later progress to hypotension all in the domain of SCAI C shock.

This can be determined by evaluation of the 3 pillars of SCAI SHOCK CS assessment (**Figure 1, Table 2**): physical, biochemical and hemodynamic assessments. There appears to be a dose-response between the number and magnitude of hypoperfusion markers and the risk of mortality, emphasizing the spectrum of shock that exists within SCAI Stage C. Oftentimes, patients with SCAI stage C shock will be on inotropes and/or temporary MCS and retrospective studies have used the presence of these therapies as an indicator of SCAI C shock. It is imperative to note that the chosen therapy does not define the SCAI stage but is a marker of clinician judgement that perfusion was inadequate prior to its use. Potential therapeutic options to improve perfusion once Stage C is determined are listed in **Table 4**.

Recently, the Cardiogenic Shock Working Group (CSWG) published a granular construct to allow assignment of SCAI stage via a computer or smartphone application.[15] Notably, in this interpretation of CSWG-SCAI stages, isolated normotensive hypoperfusion was included in SCAI B, and the mortality of such patients was higher than SCAI C, in contrast to other studies which uniformly observe progressive increases in mortality with advancing SCAI Stage. It is the opinion of this writing group that normotensive patients with hypoperfusion are classified as SCAI C, which is supported by their higher risk profile and mortality than those with hypotension and normal perfusion (SCAI B). Maintaining the higher mortality with successive stages of shock, and

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prioritizing hypoperfusion as an important binary marker of risk is a defining component of this update, and allows the SCAI SHOCK stages to be intuitively usable across the broad spectrum of stakeholders.

Table 4: Potential Therapies to Improve Perfusion in Stages C through E

Noninvasive	**Percutaneous**	**Surgical**
Diuretics	PCI	Surgical Intervention
Fluid Resuscitation	Percutaneous MCS (LV, RV, Bi-V)	Central ECMO
Inotropes	Peripheral ECMO	Surgically placed catheter based mechanical circulatory support device
Vasopressors	Primary Structural Intervention	OHT
Vasodilators (afterload reduction)	IABP	Durable VAD
Anti-arrhythmics or Direct current cardioversion	RRT (CVVHD or HD)	
Antibiotics (mixed shock with sepsis component)		

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Non-invasive ventilation / Intubation / Alteration of ventilator settings		

SCAI D

SCAI D shock is identified when a patient is worsening in the composite of physical exam, hemodynamics, and biochemical indices despite initial treatments and an aliquot of time. Often such patients are on increasing doses of vasoactive medications or different temporary mechanical support devices are placed in such patients. There is a critical distinction between lack of improvement and worsening status. For example, a patient may have SCAI C shock and treatment begins without significant improvement. The transition to SCAI D shock occurs when the patient has worsening perfusion deficits as often evidenced by objective hemodynamic and metabolic data and resultant additional therapies chosen by the treatment team. Both renal and hepatic injury may reflect damage already done rather than ongoing hypoperfusion. This distinction encompasses the risk profile for the patient who is deteriorating and requiring more therapy and is consequently at higher risk than a patient who is not requiring escalation of treatment. As with SCAI C, the chosen therapy does not define the SCAI stage but is a marker of clinician judgement that perfusion was inadequate prior to its use.

Patients with SCAI D shock are heterogeneous by nature with various combinations of drugs and devices, reflecting differences in regional availability, institutional preference and regulatory limitations in the absence of any consistent evidence-based guidelines. For example, microaxial flow pumps are preferred in some centers, particularly following emerging data in

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selected AMI-CS patients.[16] However, these devices are not licensed or readily available in all countries, often due to cost constraints. Therefore, while it is simple to categorize patients based on the burden of drugs and devices, it oversimplifies a complex situation and is most suited for retrospective or research studies where fixed rules are necessary.

Time is an important aspect of management of CS. It is anticipated that patients will transit between SCAI C and D as they are treated and thus serial monitoring is critical to the clinical care of these individuals. Once shock is recognized and treatment (ideally of the underlying cause) has begun, clinicians will observe and wait to assess patient response which is appropriate. Although the optimal interval of serial assessment remains uncertain, the group proposes that reassessment by 2-3 hours may be appropriate to identify clear worsening, whereas up to 4-6 hours may better determine failure to improve.[17] The recently introduced Door to Lactate Clearance (DLC) initiative similarly recommends 2-3 hour lactate checks.[18] This has been further validated in a large population based study.[19] Ultimately, the clinical team is best positioned to assess the trajectory of such patients, integrating the multitude of hemodynamic and laboratory data along with clinical bedside assessment.

SCAI E

SCAI E represents patients in hemodynamic and often metabolic extremis who have an imminent risk of mortality (minutes). **Figure 5** illustrates common findings in such patients. Clinical trials such as ECMO-CS[20] have defined SCAI E patients as ones requiring large or repeated bolus doses of vasoactive drugs (e.g., epinephrine or norepinephrine) to maintain

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mean arterial pressure. Additional markers such as a lactate ≥ 8 mmol/L or severe multiorgan failure contribute to the assessment of a patient as SCAI E shock.

The use of extracorporeal life support (ECLS), or any other temporary MCS device(s) by itself does not define SCAI stage E in the absence of consideration of the underlying hemodynamics, physical findings and biochemical evaluation. As such, SCAI E should instead be objectively defined by the hemodynamic and metabolic state independent of the specific therapy selected by an individual operator or team. Often, emergent placement of ECLS is selected for SCAI E patients given the rapidity of their clinical decline and the desire for full hemodynamic and respiratory support. In this example, a patient receiving ECLS with normalization of perfusion including markers such as arterial pH and lactate would subsequently be classified as SCAI C shock.

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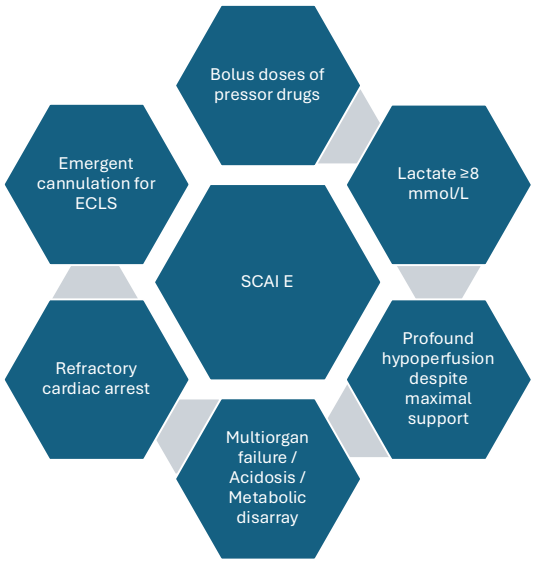

Figure 5: Markers of SCAI E cardiogenic shock

Objective Measurements in Cardiogenic Shock Patients

Intensity of Support

While the current framework of clinical, hemodynamic, and biochemical abnormalities used to define SCAI stages of CS has been widely adopted in clinical practice, there is growing interest in enhancing these classifications with objective measures. **Figure 6** shows the calculation of the Vasoactive inotropic score (VIS), which uses vasoactive drug dose equivalencies to objectively quantify the intensity of pharmacologic cardiovascular support regardless of more subjective individual vasoactive agent selection, and has consistently demonstrated correlation with morbidity and mortality across cardiogenic shock, cardiac surgery, and post-cardiac arrest cohorts.[21–24] Both higher absolute VIS values and rising VIS trajectories independently predict mortality, providing prognostic information beyond static hemodynamic or laboratory parameters, but must be taken in the context of the achieved hemodynamic goals.

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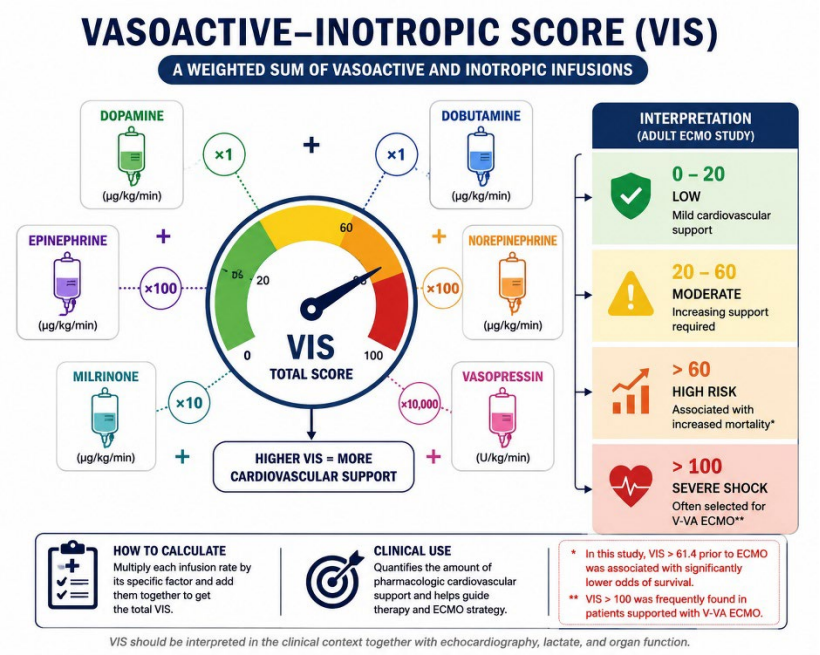

Figure 6: Calculation of the Vasoactive-Inotropic Score (VIS)

Trajectory of Shock

Trajectory-based assessment of perfusion markers and hemodynamic parameters over time, such as lactate, cardiac filling pressures, and vasoactive medication requirements, further enhances the early identification of evolving CS. Serial lactate measurements, for example, demonstrate that both persistently elevated or rising values predict mortality more accurately than baseline levels, while improvements or clearance over time correlates with improved outcomes.[18,25,26] SCAI Shock stage trajectory over time is preferred over single-time assessment given the dynamic nature of these patients.[27] Hemodynamic and metabolic trajectories show stronger associations with survival than single time point measurements, emphasizing the importance of repeated assessments at frequent intervals to detect progression from compensated states to overt hypoperfusion.[26,28] These observations led to the recent SCAI "door-to-lactate clearance" call-to-action aimed at early assessment, frequent reassessment,

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and optimization of end-organ perfusion (assessed in part via lactate measurement) within the first 24 hours of diagnosis of Stage C or higher shock.[18]

Cardiac Arrest

There is only one modifier in the SCAI Shock construct and that is "A" for cardiac arrest, in recognition of the critical importance of this event. The modifier may be applied across all SCAI stages. Cardiac arrest does not determine SCAI SHOCK stage—it modifies risk across the spectrum with greater incremental impact on mortality in the lower stages versus the higher stages. The "A" modifier notably applies to cardiac arrest with uncertain neurologic recovery status. A patient who has an arrhythmia requiring electrical cardioversion or brief cardiopulmonary resuscitation who is neurologically intact, conversant, and protecting their own airway should not be assigned the "A" modifier. Cardiac arrest adds additional complexity to both the management and outcomes of CS.[29–31] Post-arrest physiology often reflects a combined state of myocardial dysfunction, global ischemia-reperfusion injury, vasodilatory shock, temporary or permanent neurologic injury, and sometimes superimposed systemic inflammatory response syndrome (SIRS).[32,33]

Phenotypes of CS: Pathology Matters

CS arises from diverse etiologies, making accurate identification critical for both treatment and prognostication. Indeed, while prognosis worsens with each SCAI SHOCK stage, the absolute mortality tracks as much with phenotype as it does with stage. In addition, each predominant phenotype carries distinct pathophysiologic features that influence response to therapy. Recognizing the underlying etiology enables targeted interventions, improving survival

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and quality of care for this high-risk population. **Figure 7** graphically illustrates some of the most common phenotypes of CS. Thorough assessment and individualized management remain cornerstones of effective care in critical care settings.

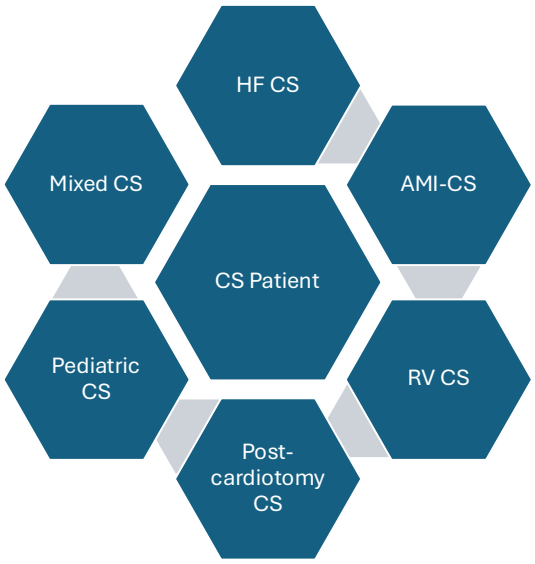

Figure 7- Phenotypes of CS

AMI-CS

CS following an acute coronary syndrome reflects the acuity of the cardiac dysfunction and the degree of impaired myocardial function, some of which might be pre-existing from prior MI or other cardiac disease. Unlike some patients with acute exacerbations of chronic heart failure CS (HF-CS) where chronic myocardial dysfunction is present, the AMI-CS patient often has no or few compensatory mechanisms already activated. In addition, while PCI is often associated with rapid improvement in myocardial function, interrupting the developing cascade

of CS, in some patients, post-reperfusion myocardial dysfunction may predominate instead. The length of time in shock prior to intervention as well as the severity of shock both contribute to the outcome after coronary intervention and are important for the clinical team to assess beyond simply treating the acute coronary occlusion. It is also not uncommon for the patient to initially stabilize with coronary artery intervention but develop worsening CS after leaving the catheterization laboratory due to the complex cascade unleashed by the initial myocardial insult with ischemia-reperfusion injury.

Heart Failure-related CS

Heart failure-related cardiogenic shock (HF-CS) represents a distinct pathophysiologic entity within the shock spectrum.[34] These patients can be further divided into multiple subcategories including *de novo* acute HF-CS, acute on chronic decompensated HF, and those receiving continuous inotropic therapy for treatment of advanced HF. Patients in each group are meaningfully different with different physiological reserve, different recovery potential, and different downstream decisions. Recent publications review these complex issues in great detail.[35–38]

Patients with acute exacerbation of chronic HF often have chronic adaptations to impaired cardiac output including skeletal muscle tissue oxygen extraction which allows them to be minimally symptomatic with chronic poor cardiac performance. Patients may have hemodynamics that meet the definition of CS in their compensated state, but this is dissociated from normal or near normal end organ function/perfusion in these patients due to existing compensatory mechanisms.

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These compensatory adaptations help explain why traditional indicators of hypoperfusion—such as rising lactate, cool extremities, or altered mental status—often emerge late, after compensatory reserves have been exhausted. Consequently, some acute on chronic HF-CS patients may remain well perfused (SCAI stage B) for prolonged periods before progressing to stage C. This underscores the slower, congestion-dominant and right heart–involved nature of HF-CS. Because compensation can mask severity, accurate assessment requires serial hemodynamic monitoring, serial assessment, and interpretation of trends rather than reliance on isolated/single measurements of CI, MAP, or lactate. In HF patients, a near normal lactate does not rule out hypoperfusion as lactate cut-points are not absolute. Neither does preserved blood pressure, as these patients may be significantly hypoperfused (SCAI C) despite unremarkable vital signs. When managing HF-CS patients, trends matter far more than any single value, and a confluence of perfusion markers may be better suited to determine perfusion than an isolated lactate alone.

On the other hand, such patients may improve dramatically and clear markers of hypoperfusion with decongestion alone, afterload reduction, and/or minimal hemodynamic support, highlighting the role of chronic compensatory mechanisms being reinstated and once again sufficient, and reinforcing that there is no “one-size-fits-all” therapy for HF-CS.

RV Predominant Failure

The right ventricle is often challenging to assess in terms of function, anatomy, and hemodynamics. In settings of CS, biventricular HF (right and left) is not uncommon and often more difficult to manage. It is important to assess both right and left ventricular contributions to the hemodynamics of CS patients, including both HF-CS (in all its forms) and AMI-CS as well as

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non-cardiac conditions such as pulmonary embolism (PE). The latter create acute strains on the right ventricle, leading to effects on the left ventricle due to ventricular independence mediated by the interventricular septum. **Figure 8** illustrates multiple factors which are relevant to consideration of RV function in CS.

RV predominant failure may be precipitated by multiple pathophysiologic processes including primary pump failure or high RV afterload states, particularly in acute onset syndromes such as PE. These concepts are illustrated by **SUPPLEMENTAL FIGURE 1**. In the SHOCK trial registry of acute coronary syndrome patients, RV failure was the predominant phenotype in only 5% of patients. However, mortality risk was similar compared to those with predominant LV dysfunction; other studies have shown worse outcomes in patients with RV-predominant CS.[39]

Recently, the AHA and ACC published new guidelines on the management of acute PE and have adopted a SCAI-like schema of categories from A to E with increasing risk of mortality. The PE stages, which are defined differently than the SCAI stages, reflect clinical severity as well as imaging of the right ventricle with PE Category D reflecting normotensive shock and PE Category E reflecting cardiogenic shock.[40]

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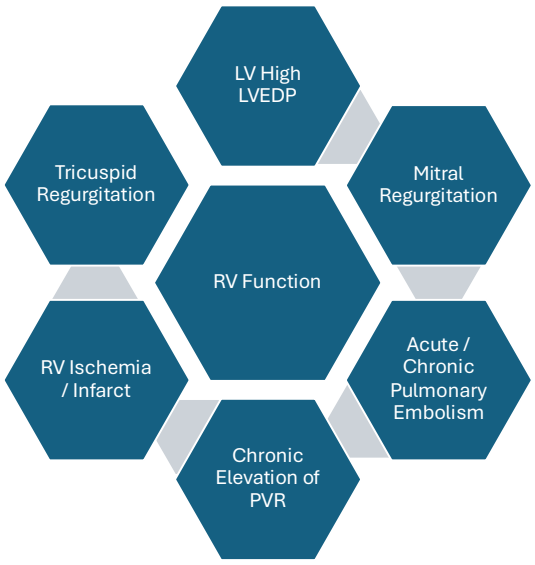

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420 **Figure 8:** Pathologic factors affecting RV function

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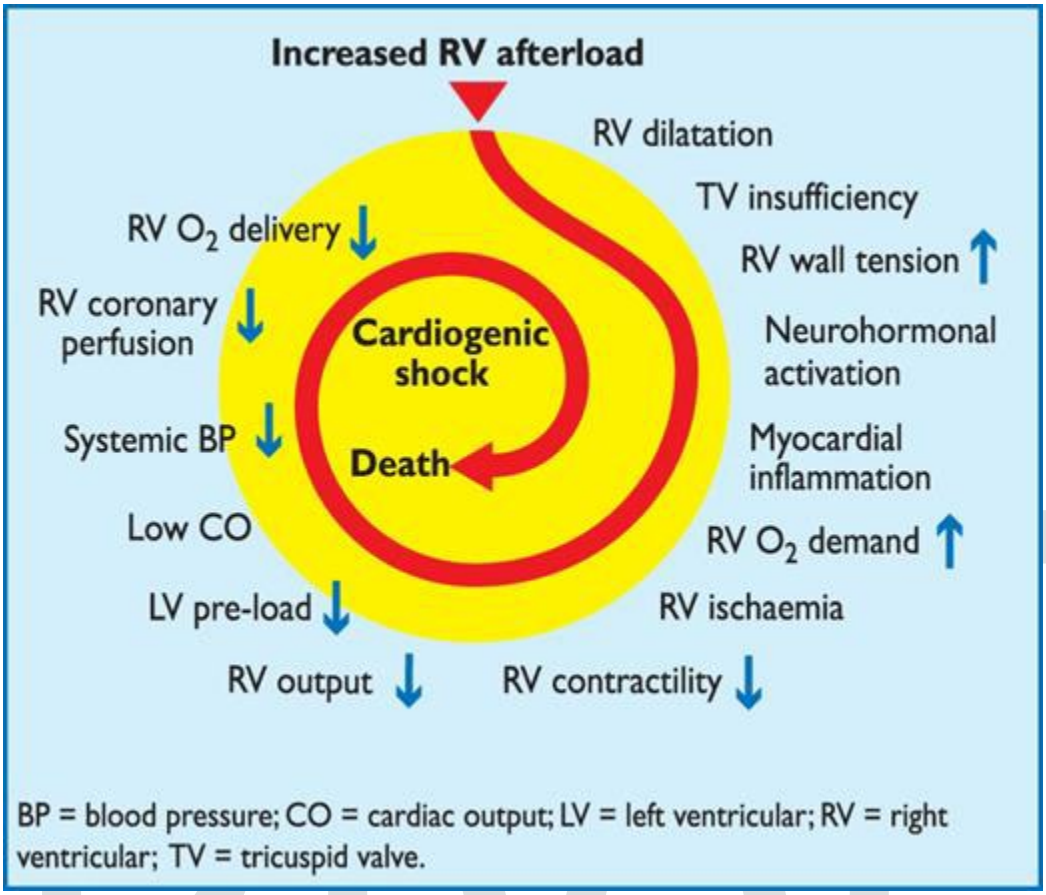
SUPPLEMENTAL Figure 1: Graphical schematic of the effects of increased RV afterload



Mixed CS

Mixed CS is among the leading causes of CS in contemporary CICUs, accounting for 20% of CS admissions.[41] Primary cardiogenic with secondary vasodilatory shock is likely the most common mixed shock state managed in CICUs. The seminal "SHould we emergently revascularize Occluded Coronaries for cardiogenic shock" (SHOCK) trial reported that 18% of all AMI-CS patients had reduced systemic vascular resistance (SVR), among whom 80% had a sterile (e.g., culture-negative) vasodilated state[42–45]; this phenomenon has been attributed to a cytokine-mediated systemic inflammatory response syndrome triggered by myonecrosis.

The SCAI writing group endorses previously published definitions of mixed CS as CS with at least one additional shock etiology (e.g., vasodilatory, hypovolemic/ hemorrhagic, and/or obstructive) contributing to hypotension and hypoperfusion.[46] Among these, cardio-vasodilatory shock can be conceptualized as an inappropriate compensatory hemodynamic response to a reduced cardiac output (CO). Although no absolute hemodynamic threshold exists, a low or inappropriately normal SVR (<1000–1200 dynes*sec/cm^5; or SVR index <2000–2400 dynes*sec/cm^5/m^2) supports a mixed cardio-vasodilatory shock state. Similarly, in patients with a primary vasodilatory shock, a low or inadequately compensatory CO (e.g., < 6L/min) despite adequate preload as assessed by central venous and/or pulmonary capillary wedge pressure) should raise consideration of a secondary cardiac component to mixed-CS. Importantly, all invasive hemodynamics must be interpreted in the context of concurrent vasoactive therapies, which may influence both CO and SVR.

The writing group endorses the use of SCAI SHOCK stages outlined in this manuscript as a practical and prognostication framework for both isolated CS and mixed CS states including those associated with sepsis.[44,47] In addition, the writing group endorses previously proposed mixed CS clinical classification framework of either *primary* or *secondary* CS (**Figure 9**).[46] Standardizing this clinical reporting framework may allow communication of etiologies, hemodynamic dominance, and sub-phenotyping within mixed CS states. In primary mixed CS, the cardiac pathology (e.g., AMI-CS or HF-CS) represents the primary or hemodynamically dominant shock in mixed CS, followed by the secondary cause of shock (e.g., sterile vasoplegia, sepsis, or hemorrhage). In secondary CS, the primary non-cardiac shock state (e.g., sepsis) precedes and precipitates cardiac dysfunction (e.g., septic cardiomyopathy).

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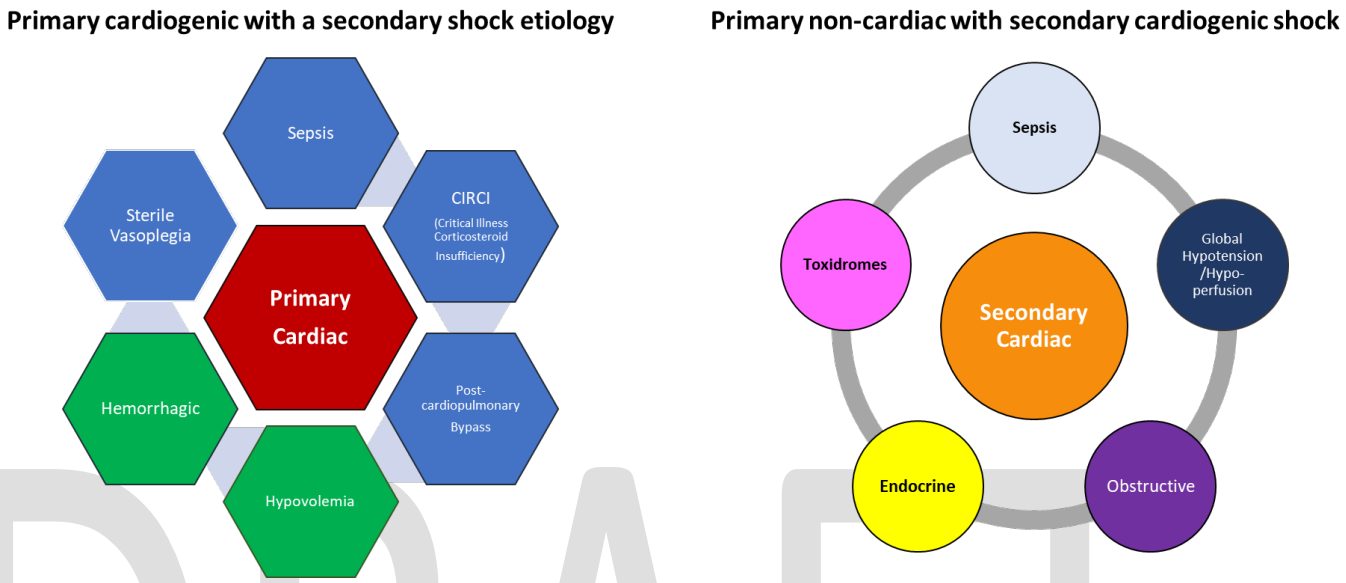

Figure 9. Common causes of mixed cardiogenic shock states managed in contemporary cardiac intensive care units.

Post-Cardiotomy CS

Post-Cardiotomy shock (PCCS) or Post-Cardiotomy Low Output Syndrome (PCLOS) continues to be a serious complication in patients undergoing cardiac surgery. It is an important peri-operative challenge due to the increasing prevalence of risk factors including previous surgeries, older age, and more advanced stages of HF. Although frequently seen in high-risk patients undergoing cardiac surgery, it can also develop unexpectedly due to intraoperative complications. In these cases, PCCS can develop because of inadequate myocardial protection, prolonged cardiopulmonary bypass (CPB) and aortic cross clamp time, technical complications, persistent ischemia, or progressive myocardial dysfunction in patients with poor pre-operative cardiac function or reserve.

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Reported incidence ranges from 2-20% with mortality rates approaching 50-70% in high-risk patients requiring ECLS.[48] A recent guideline statement from the European Association for Cardio-thoracic Surgery (EACTS) addresses the use of temporary MCS (tMCS) in patients with PCCS and PCLOS and provides a diagnostic algorithm.[49] Traditionally, PCCS has been defined as the inability to separate from CPB requiring ECLS or tMCS, or progressive post-operative CS despite high doses of inotropes and/or vasopressors. However, this binary construct does not include many patients with transient and evolving stages of "low CO" or CS that may or may not recover with standard therapeutic options.

Two large single-center retrospective analyses of cardiac surgical patients evaluated outcomes based on the presence of "standard" postoperative SCAI SHOCK Stages.[50,51] Postcardiotomy SCAI-derived criteria for CS severity showed a good correlation with in-hospital mortality. The adapted SCAI staging can be found in **Supplemental Figure 2.** These findings indicate that SCAI SHOCK classification effectively risk-stratifies postoperative CSICU patients for mortality, postoperative complications, and organ dysfunction. The analyses also examined outcomes by surgery type and time of PCCS onset (early vs. delayed), suggesting the use of SCAI staging as a triage and prognostic tool in preoperative planning of postoperative care as well. Finally, applying SCAI staging in the perioperative setting may improve risk stratification, support earlier consideration of tMCS, to include prophylactic use, and more accurately reflect the full spectrum of PCCS severity. Moreover, dynamic assessment of shock progression, rather than a binary definition based on ECLS use, provides better prognostic discrimination.

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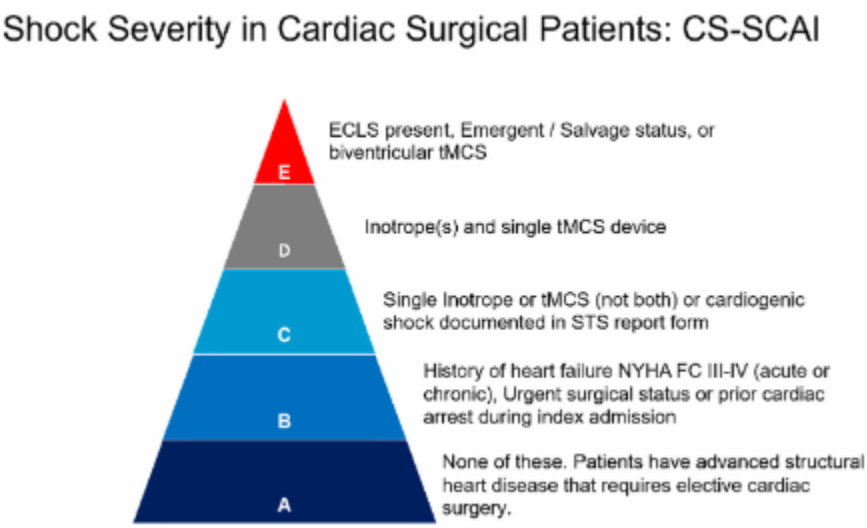
Supplemental Figure 2: SCAI staging adapted to cardiac surgical patients from Brozzi et al. [PMID: 40644812]



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Perspectives Across Specialties

The management of CS by necessity involves many specialties, and it is helpful to outline the differences in perspective to better allow team members to understand each other's approach. **Table 5** outlines the varied perspectives of the different members of the clinical team. It is organized into areas of focus, primary tools utilized and goals for each practice area. It is not meant to be exhaustive, but instead to highlight the varying perspectives with the goals often being aspirational and subject to future development. **Figure 10** illustrates the varied lenses of clinician viewpoints through which CS is seen and managed.

Pediatric CS: Unique patients

CS in children has traditionally been studied in etiologic or therapeutic silos such as post-cardiotomy, myocarditis, or multisystem inflammatory syndrome in children or according to the use of ECLS or MCS.[52] The etiologies of CS in children are quite different than those of adults and the thresholds for abnormal vital signs vary by significantly by age. The SCAI SHOCK staging has recently been adapted for pediatric use and validated in a large retrospective single center study of pediatric HF-CS, demonstrating increasing hospital mortality across stages (6% for stage A, 13% for stage B, 26% for stage C, and 39% for stage E).[52] As the study of pediatric CS grows, a specific adaptation of SCAI stages to pediatrics and multicenter validation of CS will be needed to improve risk stratification and outcomes.

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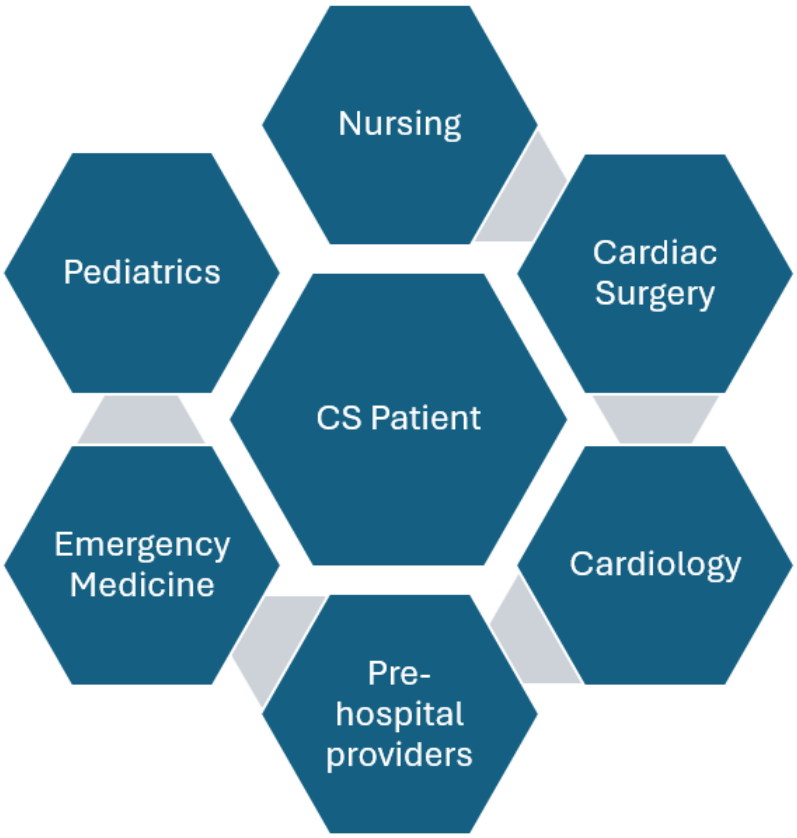

Figure 10: The varied lenses of clinician viewpoints through which CS is seen and managed.

519 **TABLE 5:** Interdisciplinary clinical perspectives

Practice Area	Focus	Primary Tools	Goals
Prehospital	• Triage • Assessment • Stabilization • En-route care	• Stethoscope • Vital signs • Skin examination • ECG/Telemetry • Vasoactive Drugs • Intubation • CPR • POCUS where available	• Assessment of perfusion along with other vital signs • Identification of CS • Reduce disparities in the delivery of pre-hospital care across geographic settings
Emergency Medicine/ Hospital	• Triage • Identification of etiologies • Initial Treatment • Stabilization	• Labs • Imaging—Xray, POCUS, CT scan • Vasoactive Drugs • Ventilatory support • Consult Shock team if available	• Rapid identification of shock • Earlier call for shock / ECLS team • Serial assessment
Critical Care Medicine	• Identification of etiologies • Initial Treatment • Stabilization • Assess response to therapies delivered prior to ICU • Trajectory assessment • Short term planning	• Labs • Continuous monitoring • Invasive hemodynamic monitoring • Multimodality imaging • Ventilatory support • Shock Team support	• Fully define SCAI SHOCK stage • Prediction of trajectory • Early decisions regarding support requirements
Interventional Cardiology	• Revascularization for AMI-CS • Assessment of feasibility of tMCS and device selection • Placement of tMCS devices and ongoing device site management	• Catheterization lab imaging • Echocardiography • Invasive hemodynamic assessment (including indwelling PA catheter)	• Coronary revascularization • Assessment of structural causes of CS • Appropriate tMCS function • Reduction of complications of tMCS

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Heart Failure Cardiology	• Assessment of SCAI SHOCK stage • Consideration of options if myocardial recovery does not recur • Management of vasoactives and tMCS in coordination with team	• Labs • Continuous monitoring • Invasive hemodynamic monitoring (including indwelling PA catheter) • Multimodality Imaging • Ventilatory support • Shock Team support	• Discriminate between advanced heart failure and temporary hemodynamic disturbance • Assess for tMCS, as well as candidacy for heart replacement therapy
CT surgery	• Assessment of SCAI SHOCK stage • Define candidacy for surgical options • Management of vasoactives and tMCS in coordination with team	• Labs • Continuous monitoring • Invasive hemodynamic monitoring • Multimodality imaging • Ventilatory support • Shock Team support • Surgical risk assessment	• Assess for surgically treatable causes of CS • Define feasibility of surgical tMCS and durable options • Assess role of valvular interventions to treat CS
Nursing	• Physical assessment including vitals • Review of clinical reports • Trending results of bloodwork • Trending hemodynamics • Early identification of deterioration	• Vital signs • Invasive hemodynamics • Physical assessment / pulses, Input /output • Infusions (based on orders) • Response to therapies	• Include SCAI SHOCK assessment as part of shift summary • Routine use of VIS to quantitate degree of pressor support • Empower nurses to recognize transitions of SCAI stage and alert team
Pediatrics	• Unique presentation of pediatric patients who are on a spectrum based on age	• Echo-guided surveillance • Physical exam • Inotropes	• Development of a pediatric adaptation of SCAI SHOCK criteria • Define outcomes of pediatric CS on

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		• Mechanical ventilation as tool for cardiac support	the spectrum of patient age and severity

Implementation of SCAI SHOCK Classification — How do we operationalize SCAI SHOCK more broadly?

Clinical implementation

Bedside application of the SCAI SHOCK Classification is inherently simple when experienced clinicians use the SCAI SHOCK pyramid and guidance table[53] as well as the core clinical data set, and can succeed even without the need for detailed granular criteria.[54] The writing group advocates for maintaining the simplicity of SCAI SHOCK staging as much as possible for more widespread local pre-hospital and in-hospital implementation, with a greater focus on increasing awareness, early identification (and rapid detection of deterioration), and education than on precision. A key question when applying any algorithm for disease detection or diagnosis relates to the balance between sensitivity (avoiding underdiagnosis) and specificity (avoiding overdiagnosis)—for a disease as dangerous as CS, the authors prefer approaches that optimize sensitivity at the expense of specificity (although specificity may improve with increasing individual and team use and experience). The common language of SCAI SHOCK should facilitate communication across centers, disciplines and care settings.

Clinically indicated laboratory data should be combined with physical assessment, vital signs and other metabolic and hemodynamic data to assess clinical trajectory. Point-of-care lactate measurements are a useful tool for early CS recognition in multiple settings from prehospital and ED to the cardiac catheterization laboratory and ICU environment. POCUS may

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be similarly useful in multiple settings. Despite its ubiquity, lactate has important limitations and should not be used as a standalone marker of perfusion. The response to treatment must be considered (e.g., washout from ischemic tissues with reperfusion causing transient elevation, nonischemic lactic acidosis due to medications such as epinephrine and albuterol, compartment syndrome or gut ischemia, and alternate clearance through hemodialysis), so integration with hemodynamics and other markers of perfusion is essential when lactate levels fail to improve as expected.[55] Likewise, biochemical markers of kidney and liver injury may have a significantly delayed rise and/or fall in response to the development and resolution of shock, often in excess of 12-24 hours, making serial values less closely tied to ongoing hypoperfusion.

User-friendly checklists, whether paper-based, mobile App-based, or embedded within the EMR, can promote earlier and more consistent staging and restaging.[56] However, these efforts alone are unlikely to improve outcomes unless integrated into structured shock team models, consensus-based treatment protocols, and education about the importance of early recognition and treatment of CS.[57–59] An effective EMR based detection system should be transparent, reproducible, simple, user-friendly, publicly available, and integrate both clinical decision support and research capabilities.[19,56,60] An EMR-based SCAI SHOCK tool alone is unlikely to provide a substantial benefit without integration into clinical pathways of care that integrate rapid assessment, evaluation, diagnosis, and treatment.

For CS to be fully evaluated and managed by all stakeholders, the classification should be incorporated into clinical care at all critical junctures. This includes the pre-hospital setting, ER, catheterization laboratory, OR, and ICU. A proposal for utilization and education around this topic for broader incorporation is represented in **Table 6**.

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565 **TABLE 6**: Recommendations for the future to enhance the earlier detection of shock

Recommendation	Details
Implement SCAI Shock stage assessment as part of EMS pre-hospital assessment	May include vital signs, physical exam findings, and preliminary lactate levels, need for simple prehospital perfusion monitors POCUS dependent on training and availability
Optimize assessment of CS by SCAI SHOCK Criteria in the emergency department	Emphasize routine use of point of care cardiac ultrasound and point of care lactate in the emergency department
Assess intravascular volume status	Avoid large fluid bolus for patients with CS who have compromised cardiac function and are not volume depleted
Implement SCAI Shock stage assessment part of serial nursing shift assessment	Broaden education to Nursing academic meetings and CME symposia
Adopt SCAI Shock stage assessment as a fundamental part of Cath lab operations	Including initial and final lactate levels for selected patients and/or hemodynamics, part of cardiac catheterization laboratory assessment of all relevant procedures (SCAI A) or presentations (AMI, decompensated HF). Low threshold to leave PA catheter indwelling to monitor serial hemodynamics.
Adopt SCAI Shock stage assessment as part of the routine provider description of (C)ICU patients each shift	Accomplish via collaboration with professional societies and training program leaders, and incorporate SCAI DLC methodology in all patients diagnosed with CS

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Support research into non-invasive correlates of reduced perfusion—the "pulse oximeter" of shock	Identify shock earlier—before the ICU, before the PAC is placed, before the arterial line

Research implementation

While SCAI SHOCK staging of CS was developed primarily as a pragmatic bedside tool, it has also proved valuable in observational studies and clinical trials to characterize severity, define prognosis and facilitate interpretation within and between research populations.[15,16,54,61–63] In clinical research, SCAI staging can be used to define eligibility criteria (e.g., enrolling stages C and D but excluding stage E), support stratified analyses based on initial stage, evaluate heterogeneity of treatment effect, serve as a serial outcome measure to quantify therapeutic response, and improve matching of illness severity when comparing practice patterns and outcomes. Ideally, SCAI stage should be assigned prospectively by clinicians at the point of enrollment in trials using prespecified criteria.

To date, applications of SCAI SHOCK staging within clinical datasets have used diverse algorithms.[2] A recent analysis by Jentzer and colleagues compared six methodologies for retrospectively assigning SCAI stage and evaluating in-hospital mortality.[64] The best predictive value was observed with the original approach, which incorporated blood pressure, heart rate, lactic acid, kidney injury, and use of vasopressors as quantified by VIS.[63]

For use in clinical practice, the criteria for each stage leverage the clinician's integrated assessment of physical findings (which are rarely available in research data sets), biochemical markers, hemodynamics and, importantly, trajectory to classify the patient with CS. Notably,

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clinician-assigned SCAI SHOCK stage has performed as well for risk stratification as more complex research definitions.[54] However, reproducible application in clinical investigation necessitates discrete evidence-based quantitative thresholds (e.g., biochemical markers and hemodynamics) and clearly defined clinical elements (e.g., congestion) to determine SCAI stage algorithmically as well as to support consistent clinician assessment across providers and sites.

Lastly, research is needed to connect SCAI SHOCK staging to guidance for specific treatment approaches. There is tremendous heterogeneity inherent in all forms of CS, combined with institutional specific approaches with significant diversity and varying resource utilization. Ultimately, providing simplified diagnostic and treatment algorithms to medical teams based on SCAI SHOCK stage and trajectory would be of tremendous utility.

Conclusion

The rapid adoption of the SCAI Shock classification system since 2019 has led to greater recognition of CS and a greater appreciation of the dynamic nature of CS. By developing and refining stages rather than a binary shock / no shock schema, the classification has facilitated a deeper understanding of the disease state and trajectory, even though treatment choices remain relatively limited. This updated consensus document further refines and sharpens definitions to clarify distinctions between stages while adding greater granularity within stages. It also emphasizes additional diverse pathologies that lead to shock including pediatric CS, post-cardiotomy CS, and mixed CS in addition to HF-CS and AMI-CS and highlights the different perspective of various clinicians. In doing so, this document serves to reinforce the necessity for a broad multidisciplinary team to achieve better outcomes for this high mortality condition.

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In the future, it is hoped better outcomes for CS patients will be observed by virtue of improved prevention, detection and more effective and timely interventions. If this is realized, then the SCAI Shock construct will have achieved one of its foundational goals: transforming the care and outcome of patients with CS.

Supplemental Materials

Supplemental Table 1: Author COI disclosure summary

Group Member	Description of Relevant Relationship(s)	Management Strategy and Rationale
Srihari Naidu, MD, MSCAI (Chair)	•Advisory and Speaker: Bristol Meyers Squibb, ZollTherox (<$10,000) •Advisory: Cytokinetics (<$10,000)	All relationships with industry fall below the $10,000 threshold for significance. This member is part of the unconflicted majority.
David Baran, MD, FSCAI (Vice-Chair)	•Nirsense: Consulting ($5,000), Stock Options (50 shares starting Jan. 2026) •Site PI, REVAMP	Nirsense manufactures vital sign monitoring equipment which is not relevant to the scope of the document. This member is part of the unconflicted majority.
Babar Basir, DO, FSCAI	•Abiomed (consulting) •Boston Scientific (consulting) •Chiesi (consulting) •Zoll (consulting)	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Edmund Bermudez, MD, MPH, FACC, FSCAI	•Abiomed/Breethe (unpaid medical advisor)	This member is part of the unconflicted majority.
Phyllis Billia, MD, PhD	•PI: Nitric Oxide in Venoarterial Extracorporeal Membrane Oxygenation (VA ECMO) (NOVICE), sponsored by Abbott. Linked clinical trial information. •PI: Proteomic profiling of RV failure, sponsored by SEER Bio ($320,000)	National PI for two industry-funded research studies in Canada. This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Vanessa Blumer, MD	No reported relationships with industry.	This member is part of the unconflicted majority.

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Group Member	Description of Relevant Relationship(s)	Management Strategy and Rationale
Deborah B. Diercks, MD	• Abbott (unpaid Scientific Advisory Board member) •Celecor (unpaid Scientific Advisory Board member) • Tosoh (research study adjudicator for BNP test) •Site PI: PGY2 inhibitor drug, Celecor •Site collaborator: Cardiac troponin, Siemens	This member is part of the unconflicted majority.
Allison Dupont, MD, FSCAI, FACC	•Getinge (consulting) •Medtronic (speaker)	All relationships with industry fall below the $10,000 threshold. This member is part of the unconflicted majority.
Jason Grady, FSCAI	• Abiomed (speaker)	All relationships with industry fall below the $10,000 threshold. This member is part of the unconflicted majority.
Timothy D. Henry, MD, MSCAI	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Steven Hollenberg, MD, FACC, FCCM, FAHA	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Abhishek Jaiswal, MD	•Bristol Myers Squibb (consulting, speaker) •Cytokinetics (consulting) •Site PI: Astrazeneca, Alleviant Medical, Outcome Sciences LLC, Medtronic, Novartis, Tenax, Endotronix	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Jacob C. Jentzer, MD	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Jason N. Katz MD, MHS	•Abiomed (speaker) •Zoll (speaker)	All relationships with industry fall below the $10,000 threshold. This member is part of the unconflicted majority.

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Group Member	Description of Relevant Relationship(s)	Management Strategy and Rationale
Mitchell W. Krucoff MD, FSCAI, FACC, FAHA	•Abbott Vascular (consulting) •Boston Scientific (consulting) •Johnson & Johnson (consulting •Medtech (consulting) •Medtronic (consulting) •Nipro (consulting) •PercAssist (consulting) •Terumo ($0) •Industry-Sponsored research: Abbott Vascular), Boston Scientific, Medtronic, Johnson & Johnson, Medtech (Abiomed, Shockwave)	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
David A. Morrow, MD, MPH	•Abbott Laboratories (consulting) •Merck & Co (consulting) •Regeneron (consulting) •Roche Diagnostics (consulting) •UpToDate (honoraria) •Industry-Sponsored research: Abbott Laboratories- Biomarkers in CV disease (PI); Abiomed- MCS in shock (Steering Committee Member); Anthos- FXI inhibitor in atrial fib (Coordinating Center Co-investigator); AstraZeneca- SGLT2i in CV disease & anti-inflamm rx in ASCVD (Coordinating Center Co-investigator); Daiichi Sankyo- Biomarkers in atrial fib (Co-PI); 4TEEN4- Biomarkers in CV disease (Steering Committee Member); Novartis- Lipid lowering in ASCVD (Steering Committee Member); Pfizer- GDF-15 inhibition in heart failure (Co-PI); Regeneron- Biomarkers in CV disease (PI); Roche Diagnostics- Biomarkers in CV disease (PI)	While the amounts received to the individual are below the $10,000 threshold, this member is the primary investigator of industry-funded research which may represent a COI.
Sandeep Nathan, MD, MSc, FSCAI	•Abbott (consulting) • Magenta Medical (consulting) • Merit Medical (consulting)	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Evjenij Potapov, MD	•Abbott (advisory and speaker) •Johnson & Johnson (speaker) •Recovery Therapeutics (speaker) •Industry-Sponsored research: Johnson & Johnson (Site Investigator); Abbott (PI & Site Investigator)	While the amounts received to the individual are below the $10,000 threshold, this member is the primary investigator of industry-funded research which may represent a COI.

Group Member	Description of Relevant Relationship(s)	Management Strategy and Rationale
Susanna Price, MBBS, BSC, FRCP, PhD, FFICM, FESC	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Kriti Puri, MBBS, FAAP, FACC	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Saraschandra Vallabhajosyula, MD MSc	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.
Sanjum S. Sethi MD	•Boston Scientific (advisory and consulting) •Conavi (consulting) •Penumbra (honoraria) •Terumo (consulting) • NAMSA (Data Safety Monitoring Board)	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Shashank Sinha, MD, MSc, FACC, FAHA	•CardiacBooster (consulting) •Industry-Sponsored research: J&J Med Tech/Abiomed- OASIS AMICS (National PI)	While the amounts received to the individual are below the $10,000 threshold, this member is the primary investigator of industry-funded research which may represent a COI.
Alexander G. Truesdell, MD	•Abiomed (consulting, speakers Bureau, and honoraria) •Getinge (honoraria) •Zoll (consulting and honoraria)	This relationship is significant because the amount received in the prior 12 months is >$10k and may represent a COI.
Sean van Diepen, MD, MSc, FAHA	No reported relationships with industry in the past 12 months.	This member is part of the unconflicted majority.

Supplemental Table 2: Study definitions of SCAI SHOCK stage B, as explicitly described in published studies

Study	Definition
Jentzer, et al. 2019[2]	**Any CICU patient meeting ALL of the following:** 1. One or more criterion for hypotension and/or tachycardia during first 1 hour after admission* a. Minimum systolic BP <90 mmHg b. Minimum mean BP <60 mmHg c. Maximum heart rate >100 BPM d. Admission heart rate > admission systolic BP e. Mean heart rate > mean systolic BP 2. No criteria for hypoperfusion a. Admission lactate <=2 mmol/L b. 24-hour urine output >=720 ml c. 24-hour creatinine increase <0.3 mg/dL

	* A subsequent study[9] using similar criteria defined the use of vasoactive drugs as a criterion for hypotension
Schrage, et al.[4]	**Signs/symptoms of cardiogenic shock OR large MI with heart rate > systolic BP plus ALL of the following:** 1. No vasoactive drugs 2. Arterial lactate <2 mol/L (or venous lactate <2.5 mmol/L)
Thayer, et al.[6] *If lactate NOT available*	**Cardiogenic shock patients NOT receiving any vasoactive drugs or MCS devices**
Thayer, et al.[6] *If lactate available*	**Cardiogenic shock patient meeting ALL of the following:** 1. No vasoactive drugs or MCS devices 2. Lactate <2 mmol/L
Pareek, et al.[8]	**OHCA patient meeting ALL of the following:** 1. Either of the following criteria for Hypotension and/or tachycardia a. Systolic BP >90 mmHg and heart rate >100 BPM b. Low-dose bolus vasopressor to maintain systolic BP >90 mmHg 2. GFR >60 ml/min
Lawler, et al. 2021[10]	**CICU patient with acute coronary syndrome or heart failure meeting ALL of the following:** 1. Hypotension 2. GFR >60 ml/min 3. Normal lactate
Kapur, et al. 2022	1. EITHER mild hypotension or hypoperfusion a. SBP 60-90 mmHg or MAP 50-65 mmHg b. Lactate 2-5 mmol/L or ALT 200-500 IU/L 2. No drugs/devices

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Supplemental Table 3: Study definitions of SCAI SHOCK stage C, as explicitly described in published studies.

Study	Definition
Jentzer, et al. 2019[2]	**Any CICU patient meeting ALL of the following:** 1. One or more criterion for hypoperfusion a. Admission lactate >2 mmol/L b. 24-hour urine output <720 ml c. 24-hour creatinine increase >=0.3 mg/dL 2. No criteria for deterioration a. Admission lactate = maximum lactate b. Maximum # vasoactive drugs during first 1 hour = maximum # vasoactive drugs during first 24 hours c. Maximum total vasoactive drugs dosage during first 1 hour = maximum total vasoactive drugs dosage during first 24 hours
Schrage, et al.[4]	**Signs/symptoms of CS with EITHER of the following:** 1. Need for vasoactive drugs 2. Arterial lactate >=2 mmol/L (or venous lactate >=2.5 mmol/L)
Thayer, et al.[6] *If lactate NOT available*	**Cardiogenic shock patient receiving only one vasoactive drug OR one MCS device**
Thayer, et al.[6] *If lactate available*	**Cardiogenic shock patient with ALL of the following criteria:** 1. One vasoactive drug OR one MCS device 2. Lactate <5 mmol/L
Pareek, et al.[8]	**OHCA with ALL of the following criteria:** 1. Requiring low-dose bolus vasopressor or one vasopressor infusion to maintain systolic BP >90 mmHg 2. GFR <60 ml/min
Lawler, et al. 2021[10]	**Cardiogenic shock patient with ALL of the following:** 1. Requiring either a single vasoactive drug or MCS support 2. Abnormal lactate, GFR (<60 ml/min) or LFT's 3. No criteria for deterioration a. <50% increase from admission lactate to peak lactate b. No new or escalating MCS >24 hours after admission 4. No criteria for refractory shock 1. Worst pH >7.2 2. Highest lactate <5
Kapur, et al. 2022	1. EITHER mild hypotension AND hypoperfusion WITH no drugs/devices a. SBP 60-90 mmHg or MAP 50-65 mmHg b. Lactate 2-5 mmol/L or ALT 200-500 IU/L 2. One drug/devices WITHOUT hypotension/hypoperfusion

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TBD, placeholder: Supplement 4: Updated checklist, based on prior:

https://scai.org/sites/default/files/2023-04/SCAI%20SHOCK%20Bedside%20Checklist%202022.pdf

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References

1. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. *Catheter Cardiovasc Interv*. 2019;94(1):29-37. doi:10.1002/ccd.28329

2. Naidu SS, Baran DA, Jentzer JC, et al. SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies. *J Soc Cardiovasc Angiogr Interv*. 2022;1(1):100008. doi:10.1016/j.jscai.2021.100008

3. Szerlip M, Al-Azizi K, Alasnag M, et al. SCAI Publications Committee Manual of Standard Operating Procedures: 2022 Update. *J Soc Cardiovasc Angiogr Interv*. 2022;1(4):100389. doi:10.1016/j.jscai.2022.100389

4. Simonato M, Vemulapalli S, Ben-Yehuda O, et al. Minimum Core Data Elements for Evaluation of TAVR: A Scientific Statement by PASSION CV, HVC, and TVT Registry. *JACC Cardiovasc Interv*. 2022;15(7):685-697. doi:10.1016/j.jcin.2022.01.014

5. Kalra S, Ranard LS, Memon S, et al. Risk Prediction in Cardiogenic Shock: Current State of Knowledge, Challenges and Opportunities. *J Card Fail*. 2021;27(10):1099-1110. doi:10.1016/j.cardfail.2021.08.003

6. Ng GWY, Low CJW, Ng KJ, et al. Predictive Models for Mortality in Cardiogenic Shock: A Systematic Review and Meta-Analysis. *Crit Care Med*. 2026;54(1):142-153. doi:10.1097/CCM.0000000000006937

7. Kirtane AJ, Doshi D, Leon MB, et al. Treatment of Higher-Risk Patients With an Indication for Revascularization: Evolution Within the Field of Contemporary Percutaneous Coronary Intervention. *Circulation*. 2016;134(5):422-431. doi:10.1161/CIRCULATIONAHA.116.022061

8. Bharadwaj AS, Truesdell AG, Lemor A, et al. Characteristics of Patients Undergoing High-Risk Percutaneous Coronary Intervention in Contemporary United States Practice. *Am J Cardiol*. 2024;222:8-10. doi:10.1016/j.amjcard.2024.04.042

9. Basir MB, Bentley D, Truesdell AG, et al. Clinical Outcomes of Patients Experiencing Transient Loss of Pulse Pressure During High-Risk PCI with Impella. *J Card Fail*. 2024;30(10):1287-1299. doi:10.1016/j.cardfail.2024.07.016

DRAFT

10. Protty M, Sharp ASP, Gallagher S, et al. Defining Percutaneous Coronary Intervention Complexity and Risk. *JACC Cardiovasc Interv*. 2022;15(1):39-49. doi:10.1016/j.jcin.2021.09.039

11. Perera D, Ryan M, Ezad SM, et al. Left Ventricular Unloading in High-Risk Percutaneous Coronary Intervention. *N Engl J Med*. 2026;394(18):1779-1789. doi:10.1056/NEJMoa2515704

12. Pothineni NVK, Enriquez A, Kumareswaran R, et al. Outcomes of a PAINESD score-guided multidisciplinary management approach for patients with ventricular tachycardia storm and advanced heart failure: A pilot study. *Heart Rhythm*. 2023;20(1):134-139. doi:10.1016/j.hrthm.2022.08.037

13. Hahn RT, Lindenfeld J, Lim SD, Mack MJ, Burkhoff D. Structural Cardiac Interventions in Patients With Heart Failure. *J Am Coll Cardiol*. 2024;84(9):832-847. doi:10.1016/j.jacc.2024.05.061

14. Baumwol J. "I Need Help"—A mnemonic to aid timely referral in advanced heart failure. *J Heart Lung Transplant*. 2017;36(5):593-594. doi:10.1016/j.healun.2017.02.010

15. 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

16. Møller JE, Engstrøm T, Jensen LO, et al. Microaxial Flow Pump or Standard Care in Infarct-Related Cardiogenic Shock. *N Engl J Med*. 2024;390(15):1382-1393. doi:10.1056/NEJMoa2312572

17. Jentzer JC, Van Diepen S, Patel PC, et al. Serial Assessment of Shock Severity in Cardiac Intensive Care Unit Patients. *J Am Heart Assoc*. 2023;12(23):e032748. doi:10.1161/JAHA.123.032748

18. Naidu SS, Nathan S, Basir MB, Baran DA, Marbach JA, Grines CL. SCAI Door to Lactate Clearance (SCAI DLC) Cardiogenic Shock Initiative: Definition, Hypothesis, and Call to Action. *J Soc Cardiovasc Angiogr Interv*. Published online September 2025:103996. doi:10.1016/j.jscai.2025.103996

19. Mehta C, Has P, Asnani H, et al. Impact of "Door-to-Lactate Clearance" on Clinical Outcomes in Cardiogenic Shock. *JACC Heart Fail*. Published online May 14, 2026:103104. doi:10.1016/j.jchf.2026.103104

20. Ostadal P, Rokyta R, Karasek J, et al. Extracorporeal Membrane Oxygenation in the Therapy of Cardiogenic Shock: Results of the ECMO-CS Randomized Clinical Trial. *Circulation*. 2023;147(6):454-464. doi:10.1161/CIRCULATIONAHA.122.062949

DRAFT

21. Vallabhajosyula S, Faugno AJ, Li B, et al. Prognostic Implications of Quantifying Vasoactive Medications in Cardiogenic Shock. *J Card Fail*. 2024;30(11):1516-1521. doi:10.1016/j.cardfail.2024.06.010

22. Patel SM, Berg DD, Bohula EA, et al. Early Serial Assessment of Aggregate Vasoactive Support and Mortality in Cardiogenic Shock: Insights From the Critical Care Cardiology Trials Network Registry. *Circ Heart Fail*. 2024;17(5):e011736. doi:10.1161/CIRCHEARTFAILURE.124.011736

23. Jentzer JC, Patel PC, Van Diepen S, Morrow DA, Barsness GW, Kashani KB. CHANGES IN VASOACTIVE DRUG REQUIREMENTS AND MORTALITY IN CARDIAC INTENSIVE CARE UNIT PATIENTS. *Shock Augusta Ga*. 2023;59(6):864-870. doi:10.1097/SHK.0000000000002123

24. Choi KH, Yang JH, Park TK, et al. Differential Prognostic Implications of Vasoactive Inotropic Score for Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock According to Use of Mechanical Circulatory Support. *Crit Care Med*. 2021;49(5):770-780. doi:10.1097/CCM.0000000000004815

25. Pagnesi M, Riccardi M, Sacco A, et al. Lactate Values and Mortality in Patients With Cardiogenic Shock: Insights From the Altshock-2 Registry. *Crit Care Med*. 2025;53(8):e1620-e1629. doi:10.1097/CCM.0000000000006738

26. Khalife W, Kanwar MK, Abraham J, et al. Association of Hemometabolic Trajectory and Mortality: Insights From the Cardiogenic Shock Working Group Registry. *J Card Fail*. 2024;30(10):1196-1207. doi:10.1016/j.cardfail.2024.06.019

27. Ton VK, Li S, John K, et al. Serial Shock Severity Assessment Within 72 Hours After Diagnosis: A Cardiogenic Shock Working Group Report. *J Am Coll Cardiol*. Published online August 1, 2024:S0735-1097(24)07740-4. doi:10.1016/j.jacc.2024.04.069

28. Marbach JA, Di Santo P, Kapur NK, et al. Lactate Clearance as a Surrogate for Mortality in Cardiogenic Shock: Insights From the DOREMI Trial. *J Am Heart Assoc*. 2022;11(6):e023322. doi:10.1161/JAHA.121.023322

29. Vallabhajosyula S, Payne SR, Jentzer JC, et al. Long-Term Outcomes of Acute Myocardial Infarction With Concomitant Cardiogenic Shock and Cardiac Arrest. *Am J Cardiol*. 2020;133:15-22. doi:10.1016/j.amjcard.2020.07.044

30. Vallabhajosyula S, Dunlay SM, Prasad A, et al. Cardiogenic shock and cardiac arrest complicating ST-segment elevation myocardial infarction in the United States, 2000-2017. *Resuscitation*. 2020;155:55-64. doi:10.1016/j.resuscitation.2020.07.022

DRAFT

31. Vallabhajosyula S, Jentzer JC, Prasad A, et al. Epidemiology of cardiogenic shock and cardiac arrest complicating non-ST-segment elevation myocardial infarction: 18-year US study. *ESC Heart Fail*. 2021;8(3):2259-2269. doi:10.1002/ehf2.13321

32. Vallabhajosyula S, Verghese D, Henry TD, et al. Contemporary Management of Concomitant Cardiac Arrest and Cardiogenic Shock Complicating Myocardial Infarction. *Mayo Clin Proc*. 2022;97(12):2333-2354. doi:10.1016/j.mayocp.2022.06.027

33. Jentzer JC, Berg DD, Chonde MD, et al. Mixed Cardiogenic-Vasodilatory Shock: Current Insights and Future Directions. *JACC Adv*. 2025;4(1):101432. doi:10.1016/j.jacadv.2024.101432

34. Abraham J, Blumer V, Burkhoff D, et al. Heart Failure-Related Cardiogenic Shock: Pathophysiology, Evaluation and Management Considerations: Review of Heart Failure-Related Cardiogenic Shock. *J Card Fail*. 2021;27(10):1126-1140. doi:10.1016/j.cardfail.2021.08.010

35. Kanwar MK, Billia F, Randhawa V, et al. Heart failure related cardiogenic shock: An ISHLT consensus conference content summary. *J Heart Lung Transplant Off Publ Int Soc Heart Transplant*. 2024;43(2):189-203. doi:10.1016/j.healun.2023.09.014

36. Baran DA, Billia F, Randhawa V, et al. Consensus statements from the International Society for Heart and Lung Transplantation consensus conference: Heart failure-related cardiogenic shock. *J Heart Lung Transplant Off Publ Int Soc Heart Transplant*. 2024;43(2):204-216. doi:10.1016/j.healun.2023.10.007

37. Sinha SS, Morrow DA, Kapur NK, Kataria R, Roswell RO. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. 2025;85(16):1618-1641. doi:10.1016/j.jacc.2025.02.018

38. Yilmaz MB, Kilic ME, Biegus J, et al. Acute myocardial infarction vs acute decompensated heart failure in cardiogenic shock: a systematic review and meta-analysis of clinical phenotypes and mortality. *Eur J Heart Fail*. Published online June 9, 2026:xuag186. doi:10.1093/ejhf/xuag186

39. Jacobs AK, Leopold JA, Bates E, et al. Cardiogenic shock caused by right ventricular infarction. *J Am Coll Cardiol*. 2003;41(8):1273-1279. doi:10.1016/S0735-1097(03)00120-7

40. Creager MA, Giri J, Mukherjee D, et al. 2026 AHA/ACC/ACCP/ACEP/CHEST/SCAI/SHM/SIR/SVM/SVN Guideline for the Evaluation and Management of Acute Pulmonary Embolism in Adults: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines.

DRAFT

Circulation. Published online February 19, 2026:CIR.0000000000001415. doi:10.1161/CIR.0000000000001415

41. Berg DD, Bohula EA, Van Diepen S, et al. Epidemiology of Shock in Contemporary Cardiac Intensive Care Units: Data From the Critical Care Cardiology Trials Network Registry. *Circ Cardiovasc Qual Outcomes*. 2019;12(3):e005618. doi:10.1161/CIRCOUTCOMES.119.005618

42. Van Diepen S, Vavalle JP, Newby LK, et al. The Systemic Inflammatory Response Syndrome in Patients With ST-Segment Elevation Myocardial Infarction*: *Crit Care Med*. 2013;41(9):2080-2087. doi:10.1097/CCM.0b013e31828a67b2

43. Thiele H, Allam B, Chatellier G, Schuler G, Lafont A. Shock in acute myocardial infarction: the Cape Horn for trials? *Eur Heart J*. 2010;31(15):1828-1835. doi:10.1093/eurheartj/ehq220

44. Jentzer JC, Lawler PR, Van Diepen S, et al. Systemic Inflammatory Response Syndrome Is Associated With Increased Mortality Across the Spectrum of Shock Severity in Cardiac Intensive Care Patients. *Circ Cardiovasc Qual Outcomes*. 2020;13(12):e006956. doi:10.1161/CIRCOUTCOMES.120.006956

45. Malik AO, Nallamothu BK, Trumpower B, et al. Association Between Hospital Debriefing Practices With Adherence to Resuscitation Process Measures and Outcomes for In-Hospital Cardiac Arrest. *Circ Cardiovasc Qual Outcomes*. 2020;13(11):e006695. doi:10.1161/CIRCOUTCOMES.120.006695

46. Van Diepen S, Pöss J, Senaratne JM, Gage A, Morrow DA. Mixed Cardiogenic Shock: A Proposal for Standardized Classification, a Hemodynamic Definition, and Framework for Management. *Circulation*. 2024;150(18):1459-1468. doi:10.1161/CIRCULATIONAHA.124.069508

47. Jentzer JC, van Diepen S, Hollenberg SM, Lawler PR, Kashani KB. Shock Severity Assessment in Cardiac Intensive Care Unit Patients With Sepsis and Mixed Septic-Cardiogenic Shock. *Mayo Clin Proc Innov Qual Outcomes*. 2022;6(1):37-44. doi:10.1016/j.mayocpiqo.2021.11.008

48. Lorusso R, Whitman G, Milojevic M, et al. 2020 EACTS/ELSO/STS/AATS expert consensus on post-cardiotomy extracorporeal life support in adult patients. *Eur J Cardiothorac Surg*. 2021;59(1):12-53. doi:10.1093/ejcts/ezaa283

49. Potapov EV, Whitman G, John R, et al. EACTS/STS/AATS Guidelines on temporary mechanical circulatory support in adult cardiac surgery. *Ann Thorac Surg*. Published online October 2025:S000349752500949X. doi:10.1016/j.athoracsur.2025.09.005

DRAFT

50. Wang Y, Li C, Wang L, et al. Using a Cardiogenic Shock Classification System for Predicting Postcardiotomy Shock Mortality. *JACC Asia*. 2025;5(5):663-676. doi:10.1016/j.jacasi.2025.01.007

51. Roeschl T, Hinrichs N, Hommel M, et al. Systematic Assessment of Shock Severity in Postoperative Cardiac Surgery Patients. *J Am Coll Cardiol*. 2023;82(17):1691-1706. doi:10.1016/j.jacc.2023.08.031

52. Puri K, Jentzer JC, Spinner JA, et al. Clinical Presentation, Classification, and Outcomes of Cardiogenic Shock in Children. *J Am Coll Cardiol*. 2024;83(5):595-608. doi:10.1016/j.jacc.2023.11.019

53. Baran DA, Long A, Badiye AP, Stelling K. Prospective validation of the SCAI shock classification: Single center analysis. *Catheter Cardiovasc Interv Off J Soc Card Angiogr Interv*. 2020;96(7):1339-1347. doi:10.1002/ccd.29319

54. Patel SM, Berg DD, Bohula EA, et al. Clinician and Algorithmic Application of the 2019 and 2022 Society of Cardiovascular Angiography and Intervention Shock Stages in the Critical Care Cardiology Trials Network Registry. *Circ Heart Fail*. 2023;16(1):e009714. doi:10.1161/CIRCHEARTFAILURE.122.009714

55. Kattan E, Hernández G, Ospina-Tascón G, et al. A lactate-targeted resuscitation strategy may be associated with higher mortality in patients with septic shock and normal capillary refill time: a post hoc analysis of the ANDROMEDA-SHOCK study. *Ann Intensive Care*. 2020;10(1):114. doi:10.1186/s13613-020-00732-1

56. Hill KL, Rustin MA, Asche MA, Bennett CE, Patel PC, Jentzer JC. Cardiogenic Shock Classification and Associated Mortality Risk. *Mayo Clin Proc*. 2023;98(5):771-783. doi:10.1016/j.mayocp.2022.12.007

57. Blumer V, Hanff TC, Gage A, Schrage B, Kanwar MK. Cardiogenic Shock Teams: Past, Present, and Future Directions. *Circ Heart Fail*. 2025;18(5). doi:10.1161/CIRCHEARTFAILURE.124.011630

58. Senman B, Jentzer JC, Barnett CF, et al. Need for a Cardiogenic Shock Team Collaborative-Promoting a Team-Based Model of Care to Improve Outcomes and Identify Best Practices. *J Am Heart Assoc*. 2024;13(6):e031979. doi:10.1161/JAHA.123.031979

59. Truesdell AG, Tehrani B, Singh R, et al. “Combat” Approach to Cardiogenic Shock. *Interv Cardiol Lond Engl*. 2018;13(2):81-86. doi:10.15420/icr.2017:35:3

DRAFT

60. Vallabhajosyula S, Mehta C, Jindal A, et al. Cardiogenic Shock Detection Using Electronic Medical Records: A Review and Blueprint for Clinical Implementation and Future Research. *J Am Heart Assoc*. 2026;15(4):e047049. doi:10.1161/JAHA.125.047049

61. Pöss J, Jentzer J, Desch S, et al. Prognostic impact of SCAI shock severity classes in AMI-related cardiogenic shock: A sub-study of the ECLS-SHOCK Trial. *ESC Heart Fail*. Published online October 13, 2025. doi:10.1002/ehf2.15446

62. Morici N, Sacco A, Frea S, et al. Early Intra-Aortic Balloon Support for Heart Failure-Related Cardiogenic Shock. *J Am Coll Cardiol*. 2025;85(16):1587-1597. doi:10.1016/j.jacc.2025.03.003

63. Jentzer JC, Van Diepen S, Barsness GW, et al. Cardiogenic Shock Classification to Predict Mortality in the Cardiac Intensive Care Unit. *J Am Coll Cardiol*. 2019;74(17):2117-2128. doi:10.1016/j.jacc.2019.07.077

64. Jentzer JC, Naidu SS, Henry TD, Baran DA. Comparison of Different SCAI SHOCK Stage Classification Systems in Patients With Cardiogenic Shock. *J Soc Cardiovasc Angiogr Interv*. Published online January 2026:104119. doi:10.1016/j.jscai.2025.104119

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