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CLINICAL DECISION MAKING

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SCAI clinical expert consensus statement on the classification of cardiogenic shock

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Abstract

Background: The outcome of cardiogenic shock complicating myocardial infarction has not appreciably changed in the last 30 years despite the development of various percutaneous mechanical circulatory support options. It is clear that there are varying degrees of cardiogenic shock but there is no robust classification scheme to categorize this disease state.

Methods: A multidisciplinary group of experts convened by the Society for Cardiovascular Angiography and Interventions was assembled to derive a proposed classification schema for cardiogenic shock. Representatives from cardiology (interventional, advanced heart failure, noninvasive), emergency medicine, critical care, and cardiac nursing all collaborated to develop the proposed schema.

Results: A system describing stages of cardiogenic shock from A to E was developed. Stage A is "at risk" for cardiogenic shock, stage B is "beginning" shock, stage C is "classic" cardiogenic shock, stage D is "deteriorating", and E is "extremis". The difference between stages B and C is the presence of hypoperfusion which is present in stages C and higher. Stage D implies that the initial set of interventions chosen have not restored stability and adequate perfusion despite at least 30 minutes of

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observation and stage E is the patient in extremis, highly unstable, often with cardiovascular collapse.

Conclusion: This proposed classification system is simple, clinically applicable across the care spectrum from pre-hospital providers to intensive care staff but will require future validation studies to assess its utility and potential prognostic implications.

KEYWORDS
cardiogenic shock, heart failure, hemodynamics

1 | INTRODUCTION

The treatment of acute myocardial infarction (MI) and heart failure (HF) has advanced exponentially over the last 50 years. One of the greatest advances has been the routine use of immediate percutaneous coronary intervention (Primary PCI) for ST segment elevation MI (STEMI) which has reduced mortality and subsequent HF substantially.[1] However, cardiogenic shock (CS) may occur prior to or following reperfusion. Even those who survive acute intervention may later develop CS and the overall 30-day mortality for patients with CS in association with MI is approximately 40–50%. Unfortunately, this incidence has not changed in the past 20 years since the publication of the landmark SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK) trial.[2–5]

The SHOCK trial was conducted when the only percutaneous form of cardiopulmonary support was the intra-aortic balloon pump (IABP). Since then, multiple devices (e.g., left atrial to femoral artery bypass devices [TandemHeart left ventricular assist device, LivaNova, London, UK], axial left ventricular—aorta pumps [Impella, Abiomed, Danvers, MA]), as well as similar devices for right ventricular support and veno-arterial (VA) extracorporeal membrane oxygenation (ECMO) have been developed and studied in the setting of CS.

Unfortunately, despite these efforts, CS mortality remains unacceptably high, and there are no prospective randomized trials showing that percutaneous mechanical circulatory support devices change the mortality in this clinical state.[3–9] It has been difficult to prove therapeutic benefit, in part, because CS patients are a heterogeneous population, and prognosis may vary widely based on etiology, severity of illness and comorbidities. CS encompasses a spectrum spanning from those at high risk of developing shock due to isolated myocardial dysfunction to those critically ill patients with severe multi-organ dysfunction and hemodynamic collapse to those with ongoing cardiac arrest. It is logical to expect that treatments may have widely varying outcomes in different patient subsets, including nonischemic subsets, and therefore a more granular classification of the CS spectrum is urgently needed to guide treatment and predict outcome.

1.1 | Purpose of a new definition

The purpose of the proposed SCAI Classification of CS is to provide a simple schema that would allow clear communication regarding patient status and to allow clinical trials to appropriately differentiate patient subsets. A few guiding principles served to organize the deliberations of the multidisciplinary team. First, the classification must be simple and intuitive without the need for calculation. Next, a new schema must be suitable for rapid assessment. Shock patients often deteriorate abruptly and therefore it is important that the schema be applied rapidly at the bedside upon patient presentation by a wide range of clinicians, as well as allowing reassessment as the patient progresses. In addition, a robust classification should be applicable to retrospective datasets or prior trials to examine whether the different shock categories correlate with definitive patient outcomes. Application of the schema may potentially identify differences between trials and perhaps explain why device-based therapies were or were not beneficial in those trials. This information would potentially inform the development of future trials. The writing group felt it critical that the schema had multidisciplinary applicability. We endeavored to develop a dynamic classification system that would be usable across all clinical settings including emergency departments, intensive care units, catheterization laboratories and others. It was equally important that the new system be actionable. An ideal schema would lead to changes in behavior such as facilitating the "hub-and-spoke" model of shock care, based on recognition of risk of deterioration and further adverse outcomes.[10] Lastly, the schema should have prognostic discriminatory potential. In other words, the different shock groups should reflect different morbidity or mortality rankings.

In the development of a new clinical acuity taxonomy for CS, we took inspiration from the American College of Cardiology/American Heart Association (ACC/AHA) classification of HF and the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) classification.[11,12] The INTERMACS classification is particularly useful due to key "tags" which serve as memorable ways to categorize patients. INTERMACS profile 1 is annotated "crash and burn", 2 is "sliding on inotropes", and profile 3 is "dependent stability". There is a temporary circulatory support modifier, but the INTERMACS classification does not distinguish between patients who were placed on ECMO support for refractory cardiac arrest, those who are stable on multiple inotropes and an IABP and those who received an Impella catheter to improve cardiac output while on inotropes. INTERMACS also does not have a construct to account for stability versus clinical deterioration, having been designed to classify patients at the single timepoint of durable mechanical circulatory support. The heterogeneity of patients described as INTERMACS 1 renders it difficult to compare outcomes across retrospective reports.

1.2 | Methodology

By design, the writing group included multidisciplinary representation reflecting the composition of teams which care for critically ill CS patients including active representation from cardiology (interventional, advanced heart failure, noninvasive), emergency medicine, critical care, and cardiac nursing. Cardiac surgery representation was sought and ultimately involved via peer review of the completed document. Broad involvement of the major professional societies was sought through representation on the writing group and peer review.

In accordance with SCAI Publications Committee policies on relationships with industry and other entities (RWI), relevant author disclosures are included in Supplemental Table S1. Before appointment, members of the writing group were asked to disclose all relevant financial relationships (>$25,000) with industry from the 12 months before their nomination. A majority of the writing group disclosed no relevant financial relationships. Disclosures were periodically reviewed during document development and updated as needed. The work of the writing committee was supported exclusively by SCAI without commercial support.

2 | THE CLASSIFICATION SCHEMA

There are five stages of shock labeled A-E in our proposed schema (Table 1, Figure 1).

Stage A: "At Risk" for CS describes a patient who is not experiencing signs or symptoms of CS but is at risk for its development. The Stage A patient may appear well and may have normal laboratories as well as physical examination. Patients with non-STEMI, prior MI as well as those with decompensated systolic or diastolic heart failure may fall into this classification which is quite broad. In general, anterior wall and large distribution infarcts carry a higher risk of cardiogenic shock but some patients may manifest shock with smaller infarcts in the setting of preexisting left ventricular dysfunction. A recent study notes the increasing incidence of shock in the ICU without myocardial infarction.[13]

Stage B: "Beginning" CS (Pre-shock/compensated shock) describes a patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion. Hypotension is defined as systolic blood pressure (SBP) <90 mmHg OR mean arterial blood pressure (MAP) <60 mmHg or >30 mmHg drop from baseline. Hypoperfusion is defined by clinical signs such as cold, clamped extremities, poor urine output, mental confusion, and the like. The physical exam of the Stage B patient may demonstrate mild volume overload and laboratories may be normal.

Stage C: "Classic" CS is a patient with hypoperfusion that requires an initial set of interventions (inotropes, pressor, mechanical support, or ECMO) beyond volume resuscitation to restore perfusion. These patients typically present with relative hypotension, with the majority manifesting the classic shock phenotype of mean arterial blood pressure (MAP) ≤60 mmHg or systolic blood pressure ≤90 mmHg along with hypoperfusion. The laboratory findings may include impaired kidney function, elevated lactate, brain natriuretic peptide, and/or liver enzymes. Invasive hemodynamics (if available) demonstrates the classic depressed cardiac index that is associated with CS.

Stage D: "Deteriorating" or "Doom" CS describes a patient who has failed to stabilize despite intense initial efforts and further escalation is required. Classification in this stage requires that the patient has had some degree of appropriate treatment/medical stabilization. In addition, at least 30 minutes have elapsed but the patient has not responded with resolution of hypotension or end-organ hypoperfusion. Escalation is an increase in the number or intensity of intravenous therapies to address hypoperfusion, or addition of mechanical circulatory support after the initial period of observation and treatment.

Stage E: "Extremis" CS is the patient with circulatory collapse, frequently (but not always) in refractory cardiac arrest with ongoing cardiopulmonary resuscitation (CPR) or are being supported by multiple simultaneous acute interventions including ECMO-facilitated CPR (eCPR). These are patients with multiple clinicians at bedside laboring to address multiple simultaneous issues related to the lack of clinical stability of the patient.

3 | DOMAINS OF PATIENT CHARACTERISTICS

We also categorized patients in three domains: biochemical (laboratory) findings, clinical bedside findings, and hemodynamics. Our classification does not legislate the presence of a particular number of findings but instead describes the common features that are prototypical of each stage.

3.1 | The arrest modifier-A

Cardiac arrest, however brief, is a significant event and usually worsens the clinical trajectory in ways that may be unforeseen. The $_{(A)}$ modifier is applied to describe patients who have had a cardiac arrest irrespective of duration (treated with chest compressions or direct current cardioversion). Accordingly, a patient may be in stage B_A shock, indicating stage B with a cardiac arrest complicating the clinical picture. This is distinct from the clinical picture of a stage E_A patient with prolonged cardiac arrest, severe clinical instability, often with numerous simultaneous interventions to maintain circulation. Whether a patient who presents with ventricular fibrillation in the setting of AMI and rapidly stabilizes with prompt defibrillation (stage B_A) has a similar or disparate survival as stage E_A will need to be examined in the future. Cardiac arrest and CS frequently occur together and the prognosis for the patient with both is worse than the presence of either cardiac arrest or CS alone.[14]

Two key components are the presence or absence of neurologic recovery and return of spontaneous circulation (ROSC). For example, a patient with out of hospital cardiac arrest (OHCA) intubated and sedated but with ROSC could be Stage A, B, C, D, or E. The prognosis for this patient may depend more on neurologic recovery than on myocardial failure.

TABLE 1 Descriptors of shock stages: physical exam, biochemical markers and hemodynamics

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

3.2 | Biomarkers

Biomarkers assist in assessing myocardial dysfunction severity as well as the response of peripheral organs and tissue in the setting of hypoperfusion. While no specific biomarker is diagnostic of shock due to a cardiac etiology, they do serve to support the diagnosis of a cardiac mechanism and provide information regarding the state of the patient at presentation as well as prognostic data as the care of the patient progresses. Frequency of testing will vary depending on the clinical scenario, the availability of rapid testing (or point-of-care testing) and the trajectory of the clinical course.

3.2.1 | Chemistry studies

Measurement of electrolytes, renal function parameters, specifically blood urea nitrogen and creatinine, and liver function tests are markers of vital organ hypoperfusion. Changes in creatinine provide important clinical prognostic features. It may be necessary to utilize the first measured value as previous baseline data may not be available. A creatinine of greater than 1.33 had a significantly higher mortality in the Intra-aortic Balloon Pump in CS (IABP-SHOCK II) trial.[15] Admission hyperglycemia, especially in patients without a known diagnosis of diabetes was also shown in this same trial to have a worse prognosis.[16]

3.2.2 | Creatine kinase and troponin

AMI is a common cause of CS. This complication may occur as a consequence of any type of acute coronary syndrome but occurs most frequently in STEMI.

If AMI is suspected, the diagnosis can be defined further using a variety of serum markers, which include creatine kinase (CK) and its subclasses (CKMB), and troponin (both I and T). Troponin T is an independent prognostic indicator of adverse outcomes and can be used as

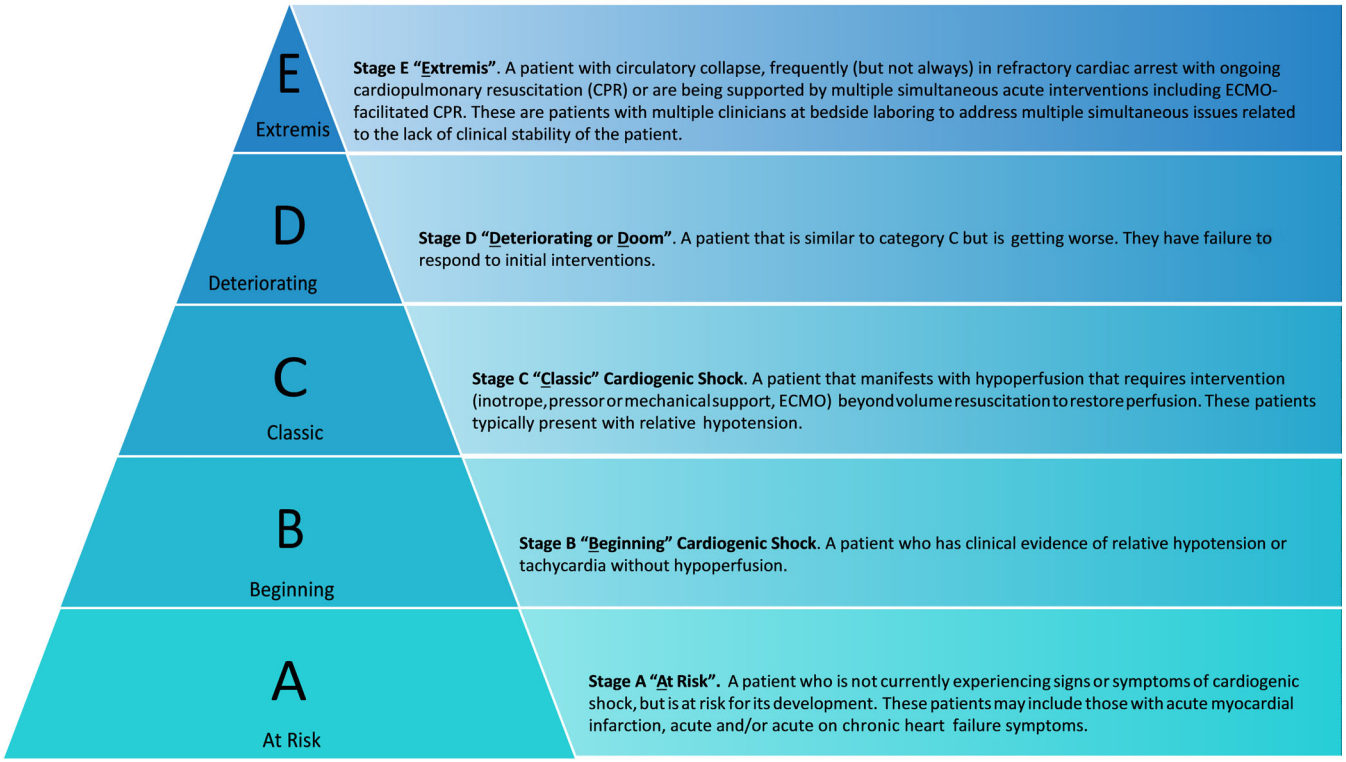

FIGURE 1 The pyramid of CS classification [Color figure can be viewed at wileyonlinelibrary.com]

a patient risk-stratifying tool.[17–21] Elevation of troponin in CS may identify patients who present late.

3.2.3 | Lactate

Lactate (whether measured from arterial, venous or capillary blood) is an early marker of mitochondrial dysfunction and cellular hypoperfusion. Since it is commonly available, it has been extensively used in studies regarding the treatment of cardiogenic shock with evidence that increased levels are associated with adverse outcomes, but without consensus on a specific discriminatory value.[16,22–24] In general, arterial lactate is preferable since venous lactate is generally higher than arterial lactate and the 2.0 mmol/L cut-off is best established for arterial lactate. The interval of assessment is uncertain and has not been systematically evaluated but most commonly occurs at 1–4 hours. In stages C or higher patients, hourly or more frequent point-of-care testing may be more appropriate.[25]

3.2.4 | Blood gas measurements

Arterial blood gas determinations of acid–base status and the level of arterial blood oxygenation offer timely assessment of the patient's clinical status. Importantly, severe acidosis has a deleterious effect on myocardial contractility and response to certain vasopressors. A base deficit abnormality correlates with the occurrence and severity of shock. It is also an important marker to follow during resuscitation of a patient from shock to assess response to therapy.[26] Central venous and pulmonary artery oxygen saturations offer insight into tissue oxygen extraction, though pulmonary artery saturation is far preferable.[27–29] Serial evaluations are essential to determine clinical severity and response to therapy.

3.2.5 | Serum bicarbonate

Serum bicarbonate, especially when assessed early in the course of patients at risk of CS may provide information regarding prognosis. In a recent study by Wigger et al[30] serum bicarbonate decreased prior to significant elevation of lactate. A low bicarbonate level was a better predictor of 30-day mortality than the highest recorded lactate level.

3.2.6 | Brain natriuretic peptide (BNP) and emerging biomarkers

Brain natriuretic peptide (BNP) may be useful as an indicator of HF and as an independent prognostic indicator of survival in CS.[31,32] A low BNP level argues against CS in the setting of hypotension; however, an elevated BNP level does not establish the diagnosis as any form of cardiac ventricular or atrial stress may elevate levels of this peptide.

Although a number of biomarkers are under investigation, there are limited data to support their use in the acute evaluation of severity of CS. These include markers of inflammation such as fibroblast growth factor-23 (FGF-23),[33] GDF-15[15] high-sensitive C-reactive

protein (hsCRP), soluble tumor necrosis factor receptor-1 (sTNFR1), and angiopoietin-2.[34] As well, markers of apoptosis including sFas and sFasL, endothelin-1 (marker of neurohumoral axis activation), and procollagen II N-Terminal Pro-peptide (PIINP) as a marker of extracellular matrix turnover are all novel markers under study but not appropriate for routine clinical use.[32]

3.3 | Physical examination

In **Stage A** (*At risk*), patients typically have an unremarkable physical examination often with no signs of volume overload. They are warm, well perfused, with normal mentation. In **Stage B** (*Beginning*), patients have clinical manifestations of elevated right or left sided filling pressures as evidenced by an elevated jugular venous pressure and/or rales on auscultation, or a low BP but preserved end-organ and peripheral perfusion. The hallmark of **Stage C** (*Classic*) and **Stage D** (*Deteriorating / Doom*) is impaired end-organ perfusion. Patients in these categories appear in obvious distress and may exhibit impaired mental status, cold/mottled extremities, volume overload, reduced urine output (<30 mL/h), and/or respiratory failure requiring mechanical ventilatory support. The final **Stage E** (*Extremis*) manifests with cardiovascular collapse with a pulseless (or near pulseless) state and respiratory failure requiring mechanical ventilation.

3.4 | Hemodynamics

3.4.1 | Hemodynamic diagnosis of CS

Although all forms of shock are diagnosed by a relative reduction in systemic blood pressure with tissue hypoperfusion, labeling it *cardiogenic* implies that shock is due to a low cardiac output/index in the absence of hypovolemia. Although CS may be diagnosed clinically, it is often difficult to distinguish it from other forms of shock without invasive hemodynamic monitoring. It is essential to measure intracardiac pressures and cardiac output in patients where the diagnosis of CS is being considered. Intriguing new data suggests that use of PA catheter may be associated with lower mortality in CS patients.[35] Echocardiography may be a valuable adjunct, in particular to identify mechanical complications of myocardial infarction, acute valvular regurgitation and to identify signs of right or left ventricular volume or pressure overload. Other conditions such as pericardial tamponade can also be rapidly identified and may significantly affect management strategies.

3.4.2 | Blood pressure measurements

Systemic hypotension (defined as a sustained systolic blood pressure (SBP) less than or equal to 90 mmHg or a mean arterial pressure at least 30 mmHg lower than baseline) due to CS occurs after a reduction in stroke volume and cardiac output. SBP may be obtained by brachial cuff (cuff measurements in thigh or ankle may be artificially higher or lower), but an arterial line may be preferable to continuously monitor pressure and facilitate frequent arterial blood gas and lactate measurements. However, systolic amplification may occur when measuring arterial pressure in a distal location compared to central aortic pressure. An underestimate of central arterial pressure using a distal arterial line is also possible with peripheral arterial disease or with peripheral vasoconstriction either due to the shock state itself or the vasoactive drugs administered.

3.4.3 | Pulmonary artery catheter measurements

Pulmonary artery (PA) catheters can directly measure right atrial (RA), PA and pulmonary capillary wedge pressures (PCWP), mixed venous oxygenation, cardiac output (CO) and allows calculation of CI, systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), pulmonary artery pulsatility index (PAPI), and cardiac power output (CPO). Recent reviews of the hemodynamics of CS provide further details on the derived values and interpretation of these indices in this setting.[36,37]

Although hemodynamic definitions of CS may vary, the National Cardiovascular Data Registry defines CS as systolic blood pressure ≤ 90 and cardiac index <2.2 L/min/m^2 and/or the requirement for parenteral inotropic or vasopressor agents or mechanical support to maintain BP and CI above these levels.[38] Although classic "cold, wet" CS is associated with low CI and high SVR and PCWP, there are four different common hemodynamic types of CS which are difficult to determine without invasive hemodynamic monitoring, and importantly, the patient may go from one category to another (Figure 2). There are two other uncommon types of CS (approximately 5% of cases): right ventricular shock and normotensive shock.[10]

The use of PA catheters can be critically important to establish the diagnosis of CS versus other causes of shock, unmask normotensive CS in patients with clinical hypoperfusion and SBP >90 mmHg, as well as accurately determining filling pressures. PA catheter hemodynamics are also helpful to assess right ventricular involvement in MI, distinguish classic cardiogenic from a mixed shock picture, assist in choice or titration of vasopressor or inotropic drugs, select patients who may benefit from mechanical circulatory support and guide weaning of pharmacological or mechanical support. Measurement of the saturation of PA blood, as well as CI and CPO are also very helpful to determine prognosis.

Despite these potential benefits, the use of PA catheters remains controversial in the wider setting. A recent analysis of the National Inpatient Sample of 89,718 AMI patients with CS who underwent cardiac catheterization revealed that only 6.1% received a PA catheter.[39] This retrospective report and others have not found a mortality benefit for CS patients who received PA catheters, although interpretation is limited given selection bias to use hemodynamic monitoring in sicker patients. The prospective, randomized ESCAPE trial in patients with decompensated HF showed no benefit, and was stopped early due to safety concerns (infection, ICD firing).[40] However, these patients did not have acute coronary syndromes or CS and all patients were enrolled with clinical equipoise. Accordingly, results of the ESCAPE study do not apply to patients with CS. There is no other randomized trial to evaluate the utility of PA catheters in cardiac patients, especially in those with

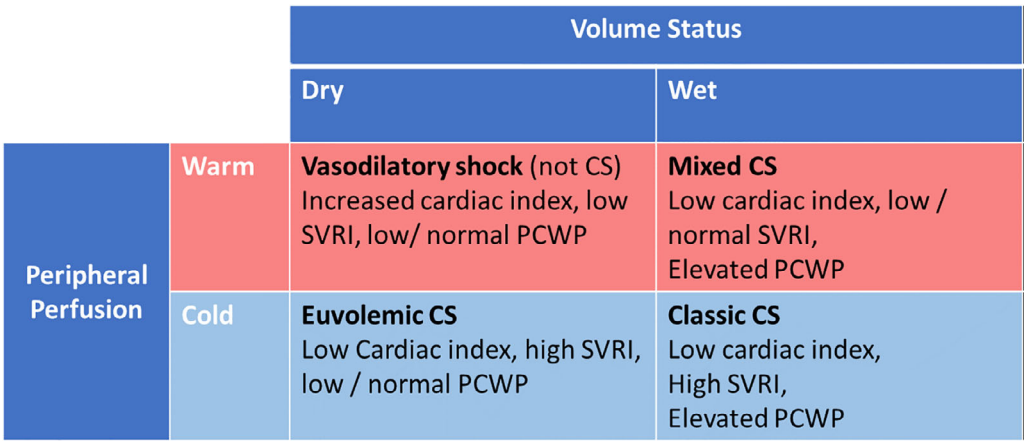

FIGURE 2 Different hemodynamic presentations of CS [Color figure can be viewed at wileyonlinelibrary.com]

CS and those being supported by mechanical support devices. We recommend the use of a PA catheter to diagnose and/or manage patients with CS, along with consideration of rapid transfer to experienced shock centers in the case of patients who require a higher level of care.

4 | MIXED SHOCK

The underlying cause of CS is by definition failure of myocardial function, and prompt measures to identify and address the underlying cause are of paramount importance. Other hemodynamic forms of shock can contribute to myocardial failure; however, as shock progresses, common pathways emerge leading to tissue and organ dysfunction, often involving inflammation and microcirculatory dysfunction.[41] These pathways can alter the hemodynamic profile of CS.

An analysis of hemodynamics in the SHOCK trial revealed that about 20% of patients had low SVR at the onset of CS.[42] Most of these patients had fever and leukocytosis suggestive of systemic inflammation, but not all of them were proven to have infection.[42] Such vasodilation can further exacerbate impaired systemic perfusion and decreased coronary perfusion pressure resulting from the initial CS state.

Distinguishing infection from systemic inflammation without infection can be challenging. Procalcitonin, an acute phase reactant released in response to endotoxin and other cytokines, is a highly sensitive marker for bacterial infection, and thus low levels may identify patients who do not require antibiotics.[43] Procalcitonin, however, has been shown to be elevated in HF[44] and so elevated levels may not be entirely specific for infection in patients with CS.

The potential for mixed shock emphasizes the importance of invasive hemodynamic monitoring in patients with CS. If patients do not respond rapidly to therapy based on the assumption that CO is low and filling pressures are high, mixed shock merits urgent consideration.

4.1 | Transitions of shock stage

Patients with CS often have dynamic clinical symptomatology and hemodynamics. In designing this classification, the authors acknowledge this and note that a patient may start at a stage B_A (beginning CS with a cardiac arrest) and then worsen over time to a higher stage. Whether transitions to higher or lower grade stages change the prognosis is unknown. For example, a patient who presents with Stage C shock, and rapidly improves following PCI of a proximally occluded left anterior descending artery might regress to stage B, but it is unknown whether the clinical trajectory is the same as a stage B patient who never develops hypoperfusion. Similarly, does the prognosis of a Stage C patient who deteriorates into Stage D but stabilizes on mechanical support and inotropes and can be weaned after 48 hours equal that of a stage C patient who never progressed in this fashion?

It is hoped that the use of the shock classification and application to patient datasets will allow such insights to be gleaned. Clearly validation in clinical datasets will be necessary to establish the utility of this proposed classification schema.

5 | PRACTICAL UTILITY OF SHOCK CLASSIFICATION

The authors recognize that the proposed classification schema presented (like most) is arbitrary and fairly simple. Some may wish for stricter definitions of stages, or to tie stages to laboratory values or some kind of a scoring mechanism. However, we feel that the elegance of the classification resides with its simplicity and that it is designed to be applicable across the care spectrum. The prognostic and therapeutic merits of the proposed classification schema are expected to be retrospectively and possibly prospectively validated.

5.1 | Example case

Mr. SL is a 67-year-old man with diabetes, hypertension, hypercholesterolemia and tobacco use who underwent coronary artery bypass grafting 10 years prior for severe three vessel coronary disease. He presents with vague chest pain which woke him from sleep. Further questioning indicates a crescendo pattern to angina and troponin T measured in the emergency department is positive. His blood pressure is 94/70 mmHg

and heart rate 100 beats per minute (BPM) but he normally has a blood pressure of 140/70 mmHg. He is scheduled to undergo diagnostic catheterization later in the day. In the new classification he would be assessed as **Stage B**. Later that day, in the catheterization laboratory, he is noted to be more tachycardic (heart rate 110 BPM), with reduced urine output. A PA catheter is placed and his cardiac index is 1.8/m^2 with a wedge pressure of 29 mmHg. He would be judged to be **Stage C** at this point. The team considers putting an IABP in but instead decides to intervene on a thrombosed saphenous vein graft to the right coronary artery. During thrombectomy, the patient has ventricular fibrillation and requires a single 200 joule shock by external pads. Now the patient would have the **A-modifier (Stage C_A)**. Low dose inotrope is started and the intervention completed successfully. An IABP is placed at the end of the case. Later that night in the intensive care unit, the patient's urine output continues to decline and the continuous cardiac index assessment remains below 2 L/min/m^2 despite increasing inotropes and IABP 1:1 counter-pulsation. The patient is now in **Stage D_A** and plans are made to escalate percutaneous support.

6 | CONCLUSION

Despite intense study, the mortality of CS in association with MI remains approximately 50% even with the development of percutaneous mechanical circulatory support devices. It is likely that prior trials have not been successful partially because some patients were “too sick” to benefit from the studied intervention. Others may do well with or without an intervention, and in the absence of a standardized classification system, it may be impossible to ascertain which groups may benefit. The schema outlined is a result of a broad multidisciplinary collaboration of experts to define the groups of patients who suffer from CS. The criteria are simple and clinically based, and if validated, this classification may become the “lingua franca” for the field. By having a common language, we hope to support communication at the bedside, in the catheterization laboratory, at the level of shock teams across institutions, and with clinical trialists as new approaches are tested to reduce the high mortality of CS.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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