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EXPERT CONSENSUS DOCUMENTS

Definitions of Cardiogenic Shock and Indications for Temporary Mechanical Circulatory Support: Joint Consensus Report of the PeriOperative Quality Initiative and the Enhanced Recovery After Surgery Cardiac Society

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ABSTRACT

BACKGROUND The management of patients with cardiogenic shock (CS) is complex and resource intensive, particularly given the recent surge in temporary mechanical circulatory support (tMCS) devices. This document was created to establish an approach to the assessment of CS to provide early and targeted therapies, including tMCS.

METHODS An interdisciplinary, international panel of experts, using a structured appraisal of the literature and a modified Delphi method, derived consensus regarding the assessment of CS based on pathophysiologic severity, etiology, and phenotypic clustering to guide escalation of care as well as identify those patients who might benefit from tMCS.

RESULTS Key principles included early and continuous assessment for the evolution of shock severity to guide the escalation of care as well as establishment of the cause of CS to facilitate triage and assignment of initial therapies. Phenotypic clustering is complementary and aids in prognosis. tMCS provides the greatest benefit in CS for relief of congestion refractory to medical therapy, ideally when initiated before the development of organ injury. The use of tMCS should be preceded by an interdisciplinary discussion as part of the informed consent process to establish therapeutic goals, including exit strategies.

CONCLUSIONS Based on the available literature and expert consensus, there is an opportunity to further standardize the approach to CS, including characterization based on the severity of the shock state, etiology, and further enhancement by phenotyping. Monitoring, early triage, and timely escalation of care, including the targeted initiation of tMCS, can minimize organ injury and in-hospital mortality.

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Cardiogenic shock (CS) is a heterogeneous syndrome with highly variable clinical manifestations, pathophysiology, severity,

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Abbreviations and Acronyms

AMI = acute myocardial infarction
AMI-CS = acute myocardial infarction–related cardiogenic shock
CS = cardiogenic shock
CSWG = Cardiogenic Shock Working Group
ERAS = Enhanced Recovery After Surgery
HF = heart failure
HF-CS = heart failure–related CS
IABP = intraaortic balloon pump
LVAD = left ventricular assist device
NSTEMI = non-ST elevation myocardial infarction
PAC = pulmonary artery catheter
P-CS = postcardiotomy shock
POQI = Perioperative Quality Initiative
RCT = randomized controlled trial
SCAI = Society for Cardiovascular Angiography and Interventions
tLVAD = temporary left ventricular assist device
tMCS = temporary mechanical circulatory support
VA-ECMO = veno-arterial extracorporeal membrane oxygenation

in-hospital trajectories, and outcomes. Therefore, it is necessary for the assessment and phenotyping of CS to capture this multidimensional heterogeneity to enhance the care of patients with CS. This includes timely and patient-specific delivery of appropriate interventions based not only on disease-specific mechanisms but also on the individual's physiological and biological responses. This approach increases the likelihood of success of treatment and minimizes treatment-related complications. There is growing evidence to support a more systematic approach to defining CS, both to enhance communication for clinical support and research and to individualize care. The goal, ultimately, is to establish an approach for dynamic risk stratification and tailoring of resuscitative efforts, including both pharmacology and temporary mechanical circulatory support (tMCS) strategies.

For related articles, see pages 194, 213, 225

Owing to the inherent complexity of caring for patients with CS as well as the burgeoning nature of research associated with the topic, a joint conference between the PeriOperative Quality Initiative (POQI) and Enhanced Recovery After Surgery (ERAS) Cardiac Society was convened to appraise and consolidate the available literature as well as provide practical guidance on phenotyping CS and establishing criteria for escalation of care, including the consideration for tMCS. We hypothesized that a formal review and appraisal of the existing literature would reveal comprehensive strategies to assess patients with CS, the evidence behind them, and facilitate triage of patients who benefit from tMCS.

MATERIAL AND METHODS

POQI is a nonprofit organization that assembles international, multidisciplinary groups to develop consensus statements on key topics pertinent to perioperative medicine. On January 24 through 26, 2024, the 14th POQI meeting convened in person in conjunction with the ERAS Cardiac Society to address topics relevant to the management of CS and MCS. A group of experts were identified with clinical backgrounds in anesthesiology, surgery, cardiology, and nursing, with a particular focus on CS and tMCS. For the purposes of this effort, tMCS includes any nondurable device designed to support cardiac function, including intra-aortic balloon pump (IABP), left ventricular assist device (tLVAD [ie, Impella; Abiomed]), right ventricular assist device (ie, ProtekDuo [LivaNova], CentriMag [Abbott]), and venoarterial extracorporeal membrane oxygenation (VA-ECMO).

The joint meeting produced a series of manuscripts, each detailing a different aspect of the clinical management of cardiogenic shock and tMCS. This report is the result of the subgroup that appraised the literature to establish clinical definitions of CS and indications for tMCS. Subsequent parts of the series will outline the clinical approach to the escalation and de-escalation of tMCS for patients in shock (Escalation and De-escalation of Temporary Mechanical Circulatory Support) as well as best practices for patients managed with tMCS in the intensive care unit (Best Management Practices on Temporary Mechanical Cardiac Support).

To assess the literature on clinical definitions of CS and indications for tMCS, 4 predetermined topics were addressed by the subgroup and later refined during the conference and subsequent proceedings:

1. How do we define CS and its severity?
2. What is the impact of the etiology of CS on prognosis, in-hospital trajectories, and outcomes?
3. What are the specific clinical phenotypes of CS?
4. Which CS patients (severity/etiology/phenotype) benefit from the use of tMCS?

The joint POQI/ERAS Cardiac Society conference was a consensus-building initiative using the modified Delphi process, with participants selected to ensure representation from numerous backgrounds based on expertise in principles of perioperative management for the cardiac surgical patient. Original questions were refined, statements were developed over several days using alternating plenary sessions and small group

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TABLE 1 Summary of Questions, Statements, and Quality of Evidence

Question	Statement	Quality of Evidence
1. How do we define cardiogenic shock and its severity?	Initial and ongoing assessment for severity and progression of shock is necessary to determine escalation in care.	Moderate
2. What is the impact of etiology of cardiogenic shock on prognosis, in-hospital trajectories, and outcomes?	Identifying the etiology of underlying cardiac dysfunction and mechanism of acute cardiac injury improves risk stratification and initiates a specific therapeutic pathway.	Moderate
3. What are the phenotypes of cardiogenic shock?	Phenotyping cardiogenic shock, regardless of the etiology, is a complementary method to assess severity.	Low
4. Which cardiogenic shock patients (ie, severity, etiology, phenotype) stand to benefit from tMCS?	Patients with evidence of cardiogenic shock should be considered for tMCS.	Moderate
	An interdisciplinary discussion prior to non-emergent initiation of tMCS establishes therapeutic goals, including exit strategies.	Moderate
	Patients with increased risk undergoing non-emergent cardiac procedures as well as those patients admitted with cardiogenic shock benefit from incorporating tMCS within the informed consent process.	Moderate

tMCS, temporary mechanical circulatory support.

discussions, and consensus was reached on the main issues within each topic (Supplemental Figure). The modified Delphi method used has been previously described in POQI consensus statements and includes iterative steps from an initial literature review to building consensus around key statements related to the central topic.[1]

Content refinement continued until agreement was achieved, resulting in a formal consensus document. Quality of evidence was assessed, and designations were rendered based on whether additional research on the topic is unlikely (high quality), likely (moderate quality), or very likely (low quality) to have an important impact on the statement of the effect of the intervention.[2] Final questions and accompanying statements were endorsed after large group discussion, with a majority vote representing confirmation.

For content to be included in the consensus statement, we reviewed the literature and searched PubMed from inception to January 2024. All coauthors participated in the literature search and consolidated relevant references to a central repository. The search was limited to human trials and articles published in English. During the conference and thereafter as a writing group, reference applicability to the topic was discussed, and disagreements were resolved by group consensus. No human subject research occurred, so institutional review board approval was not required.

RESULTS

The results and synopsis of the evidence review and modified Delphi process are below. Statements are summarized in Table 1, along with quality of evidence designations.

Question 1: How do we define cardiogenic shock and its severity?

Statement 1: Initial and ongoing assessment for severity and progression of shock is necessary to determine escalation in care.

Quality of evidence: Moderate

Rationale: Before initiating treatment pathways for a patient in shock, it is necessary to recognize whether the primary process leading to the shock state is of cardiogenic origin and determine its severity. Broadly, CS is a maladaptive state in which cardiac dysfunction leads to sustained inadequate perfusion at both the tissue and cellular levels.[3] There was a paucity of standardized literature defining CS, especially in the absence of an acute myocardial infarction (AMI).[4,5] Reduced cardiac output with normal or elevated filling pressures is a requisite hemodynamic condition.[6] However, traditional definitions of CS focused on reduced systolic blood pressure, cardiac index, or elevated pulmonary capillary wedge pressure run the risk of underdiagnosing or overdiagnosing shock states in patients whose chronic condition (ie, HF with reduced ejection fraction) may alter the presenting clinical picture.[7] For this reason, CS is defined as "circulatory failure attributable to cardiac

TABLE 2 Clinical Assessment, Laboratory Findings, and Hemodynamics of SCAI-CSWG Cardiogenic Shock Stages

Stage	Clinical Impression	Physical Assessment	Biochemical Markers	Noninvasive Hemodynamics	Invasive Hemodynamics
A At Risk	Hemodynamically stable	Normal JVP Lung sounds clear Strong distal pulses Normal mentation	Lactate <2 mmol/L Normal renal function	SBP >100 mm Hg MAP >60 mm Hg HR <100 beats/min	CI ≥2.5 L/min/m^2 CVP ≤10 mm Hg PA sat ≥65%
B Beginning	Hypotensive or hypoperfusing **and** untreated	Elevated JVP Rales in lung fields Strong distal pulses Normal mentation	Lactate < 2 mmol/L Minimal renal impairment +/− Elevated BNP	SBP <90 mm Hg MAP <60 mm Hg >30 mm Hg reduction from baseline HR >100 beats/min	CI ≥2.2 L/min/m^2 PA sat >65% PCWP <15 mm Hg
C Classic	Hypotensive and hypoperfusing **and** treated	Ashen, mottled, dusky Volume overload Extensive rales BiPap or mechanical ventilation Altered mentation	Lactate >2 mmol/L **or** Creatinine doubling **or** >50% reduction in GFR **or** Increased LFTs **or** Elevated BNP	SBP <90 mm Hg MAP <60 mm Hg >30 mm Hg reduction from baseline HR >100 beats/min **and** vasopressor **or** inotrope	CI <2.2 L/min/m^2 PCWP >15 mm Hg RAP/PCWP ≥0.8 mm Hg PAPi <1.85 Cardiac power output ≤0.6 **and** vasopressor **or** inotropic agent
D Deteriorating	Continued shock despite initial therapy	Stage C **and** further deteriorating	Stage C **and** further deteriorating	Stage C **and** multiple vasopressors/ inotropic agents **or** MCS device(s)	Stage C **and** multiple vasopressors/ inotropes **or** MCS device(s)
E Extremis	Refractory shock despite maximal therapy	Near pulselessness/ PEA Cardiac collapse Mechanical ventilation Defibrillation	Lactate ≥5 mmol/L pH ≤7.2	No SBP PEA or refractory VT/VF **and** maximal support	No SBP PEA or refractory VT/VF **and** maximal support

BiPap, Bilevel positive airway pressure; BNP, B-type natriuretic peptide; CI, cardiac index; CSWG, Cardiogenic Shock Working Group; CVP, central venous pressure; GFR, glomerular filtration rate; HR, heart rate; JVP, jugular venous pressure; LFTs, liver function tests; MAP, mean arterial pressure; MCS, mechanical circulatory support; PA, pulmonary artery; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; PEA, pulseless electrical activity; RAP, right atrial pressure; SBP, systolic blood pressure; SCAI, Society for Cardiovascular Angiography and Interventions; VF, ventricular fibrillation, VT, ventricular tachycardia.

dysfunction that results in abnormal tissue perfusion," with stages of shock severity then defined by worsening hypotension, impaired perfusion, and the associated congestion profile.[8]

The progression of CS has been defined with a validated staging system, the Society for Cardiovascular Angiography and Interventions (SCAI) criteria, which differentiates CS stages by presenting clinical signs, laboratory findings, and noninvasive and invasive testing.[9] The initial assessment focuses on the patient's presenting clinical examination to quickly identify CS. Where feasible, patients at risk for and with evidence of CS should receive continuous assessment and treatment.[10] According to the SCAI classification criteria,[11] CS stages are defined as:

Stage A ("at risk"): Patients at risk for developing CS due to disease states such as AMI, prior infarction, or acute/acute-on-chronic heart failure. Patients in stage A are not acutely experiencing any symptoms of hypotension or showing signs of hypoperfusion.

Stage B ("beginning"): Patients who have clinical signs of hemodynamic instability; however, are not showing signs of end-organ hypoperfusion.

Stage C ("classic"): Patients who show signs of hypoperfusion and require an intervention other than volume resuscitation (ie, pharmacologic, tMCS) to maintain end-organ perfusion. Hypotension is typically present but is not required.

Stage D ("deteriorating" or "decompensating"): Stage C with continued worsening of end-organ perfusion as evidenced by physical examination, biochemical, and hemodynamic indicators.

Stage E ("extremis"): Actual or impending circulatory collapse despite maximal hemodynamic support.

The SCAI Shock Classification system has become a useful guide for clinical practice, streamlining communication of CS severity, and facilitating timely decision-making regarding triage and treatment.[10] More recently, the Cardiogenic Shock Working Group (CSWG) sought to address HF-related CS (HF-CS) patients who may present in stage B cardiogenic shock without hypotension.[8] Additionally, the SCAI-CSWG Shock Classification staging system provides clinicians with clinical variables to aid in diagnosing CS and detecting its progression (Table 2).[8-10] By providing clinicians with indicator-based guidelines coupled with clinical descriptions, the aim was to reduce the subjective

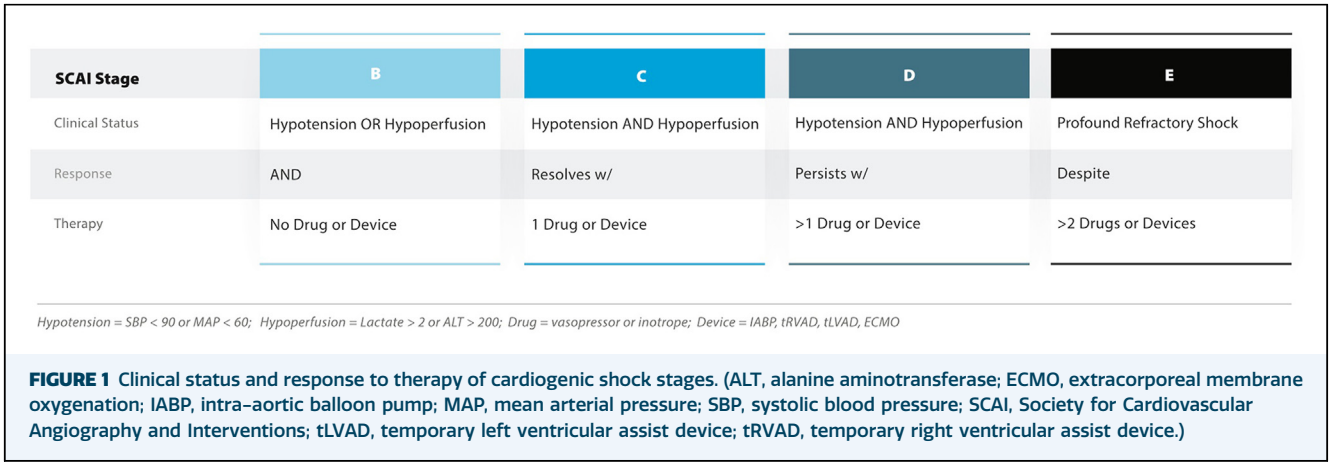

FIGURE 1 Clinical status and response to therapy of cardiogenic shock stages. (ALT, alanine aminotransferase; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; MAP, mean arterial pressure; SBP, systolic blood pressure; SCAI, Society for Cardiovascular Angiography and Interventions; tLVAD, temporary left ventricular assist device; tRVAD, temporary right ventricular assist device.)

nature of the original SCAI guidelines (Figure 1). The SCAI-CSWG stages include:

Stage A: Hemodynamically stable.
Stage B: Hypotensive or hypoperfusing and untreated.
Stage C: Hypotensive and hypoperfusing and treated.
Stage D: Failure to stabilize with initial therapy.
Stage E: Extremis/refractory shock.

ROLE OF INVASIVE HEMODYNAMICS IN DEFINING CS. Landmark trials have previously adjudicated presence of CS based on hypotension and hypoperfusion indicators without the need for further corroboration using invasive or noninvasive methods.[12] Alternatively, it is increasingly described that management of congestion and device escalation can be achieved with meticulous invasive and noninvasive hemodynamic profiling and monitoring in distinct types of CS.[13]

Contemporary classification of CS now includes specific congestion-associated indicators derived from the use of a pulmonary artery catheter (PAC) (Table 2).[14] The early and comprehensive use of a PAC has been associated with improved survival irrespective of severity staging and etiology.[15,16] Hemodynamic profiles used to classify CS include right heart, left heart, and biventricular, for which right heart and biventricular were associated with increased mortality among both AMI and HF-CS clinical subtypes.[14,17] However, a PAC is an invasive tool that may not be available in community hospitals and requires requisite experience to calculate and interpret hemodynamics.

Alternately, the use of echocardiography has shown value in risk-stratification of CS in addition to providing valuable insight toward early stages of disease etiology. It may also aid in the diagnosis of potentially reversible and/or mechanical complications, including myocardial rupture, valvular heart disease, and/or restrictive heart disease as well as confirmation of successful therapy.

Recognizing CS, especially in the early stages, allows clinicians to appropriately escalate interventions in a timely fashion and also permits providers to make informed decisions regarding the appropriateness of patient transfer to a tertiary care center. It is, however, important to realize that transfers to tertiary care centers are not a panacea and have been associated with increased mortality despite similar clinical disease severity.[18] Individual hospital resources and expertise, therefore, may limit the diagnostic methods used to determine the progression of CS.

Question 2: What is the impact of the etiology of CS on prognosis, in-hospital trajectories, and outcomes?

Statement 2: Identifying the etiology of underlying cardiac dysfunction and the mechanism of acute cardiac injury improves risk stratification and initiates a specific therapeutic pathway.

Quality of evidence: Moderate

Rationale: Clinical presentations of CS stratified by etiology are characterized in 2 groups based on recent registry-based data: AMI-related CS (AMI-CS) and HF-CS.[7,19,20] Each clinical subgroup has distinct subcategories that allow for a more refined prediction of disease trajectory, treatment options, and prognosis. AMI-CS, including acute coronary syndrome in the form of non-ST-elevation myocardial infarction and ST-elevation myocardial infarction has also been shown to have different disease trajectories at

different levels of severity and treatment intensity.[8] HF-CS, which includes patients with newly diagnosed disease (described as de novo CS, although this term does not exclude the previously undiagnosed presence of disease), encompasses etiologies such as postpartum cardiomyopathy, myocarditis, and arrhythmia-related entities.[21] Alternatively, acute-on-chronic HF decompensating into a shock state has also been identified and broadly described with its own specific pathophysiologic considerations.[6]

A potential third category altogether, post-cardiotomy shock (P-CS), remains an understudied heterogenous subgroup with high mortality that requires separate considerations to best risk stratify and define, including perioperative details related with surgical-, anesthesia-, and perfusion-associated techniques and complications.[22] P-CS may have certain causes and clinical features that overlap those of other subgroups (ie, coronary malperfusion, valvular or ventricular dysfunction), but more research is required to determine the etiology, risk factors, and the degree to which patients with P-CS respond to similar therapeutic interventions. Other emerging clinical subtypes associated with CS that might impact treatment strategies include restrictive cardiomyopathy, congenital heart disease, and spontaneous coronary dissection.[23]

Each individual CS entity is likely to present and, hence, respond quite differently to initial therapeutic interventions. The clinical trajectory of a patient with AMI-CS often begins with hypotension, which then leads to hypoperfusion and eventually, congestion.[6] In contrast, a patient with HF-CS presents with acutely decompensated HF and congestion, leading to hypoperfusion and ending with hypotension. In fact, the traditional definition of CS can be applicable to many ambulatory patients with HF with reduced ejection fraction on guideline-directed medical therapy, who often have a systolic blood pressure <90 mm Hg, elevated intracardiac filling pressures, and depressed cardiac output—the key distinction from a CS profile being that they are without hypoperfusion or end-organ dysfunction.[24]

Overwhelmingly, the literature has focused on survivability associated with AMI-CS, in large part due to the extensive research, clinical resources, and success of various forms of coronary revascularization over recent decades.[25] Comparatively, other forms of CS are associated with discouragingly high mortality rates.[6,24] Time to intervention, regardless of the cause, remains a critical predictor of survival. Thus, early establishment of etiology allows for assignment of initial therapies (ie, triage/transport, revascularization, organ support) as well as initial stratification of risk (ie, mortality vs recoverability).

Question 3: What are the phenotypes of cardiogenic shock?

Statement 3: Phenotyping CS, regardless of the etiology, is a complementary method to assess severity.

Quality of evidence: Low

Rationale: Thus far, this document has established CS phenotypes based on etiology as well as pathophysiologic severity by using clinical indicators and hemodynamic data. However, currently used variables as markers for CS severity (ie, vital signs, hemodynamic measurements, lactate, vasoactive drug requirement) are relatively nonspecific individually and fail to adequately capture the spectrum of shock severity. Therefore, a combination of clinical presentation (ie, hypotension) and markers of CS severity (ie, hypoperfusion and congestion) may improve risk stratification. Even though the SCAI Shock Classification defines discrete stages, that the severity of CS exists on a continuous and dynamic spectrum is well recognized.[4,26] To address this clinical reality, a 3-axis model for conceptualizing CS patient trajectories was proposed.[9] The CSWG further refined SCAI staging by defining specific ranges of each clinical indicator to make these definitions readily and uniformly applicable in real-world clinical practice.[8] Furthermore, they presented the incidence and degree of progression of CS severity and its association with in-hospital mortality from baseline, at 24 hours, and along the course of hospitalization.

Within each SCAI stage as defined by risk-based indicators, subgroups of CS patients remain quite heterogeneous. Defining these subgroups using insight into underlying pathophysiologic mechanisms may allow us to transition from the typical risk-based approach to a mechanistic approach that embraces the heterogeneity within the CS population.[27] Unsupervised machine learning clustering is increasingly used to identify subgroups within heterogenous populations of patients with the overall aim to stratify risk.[28] An analysis by Zweck and colleagues[29,30] used unsupervised clustering to identify and characterize 3 distinct CS phenotypes based on admission characteristics: noncongested, cardiorenal, and cardiometabolic.

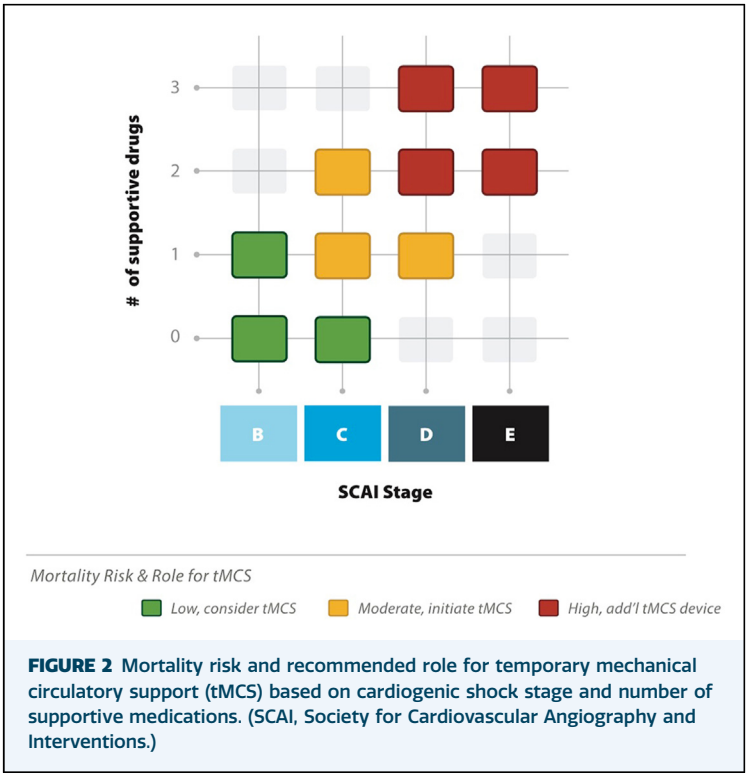

FIGURE 2 Mortality risk and recommended role for temporary mechanical circulatory support (tMCS) based on cardiogenic shock stage and number of supportive medications. (SCAI, Society for Cardiovascular Angiography and Interventions.)

These phenotypes can be assigned prospectively, were compatible with SCAI Shock Classification, and were increasingly associated with risk of death. Moreover, these phenotypes were derived in patients with AMI-CS and validated in patients with HF-CS, suggesting conservation across etiologies of CS. These phenotypes have since been further validated in other contemporary CS data sets.[29-31]

Phenotyping in CS can also provide a framework for prospective stratification of patients by shock severity in clinical research. High variance in the patient samples might be one reason previous randomized clinical trials (RCTs) have inconsistently identified survival benefit using tMCS devices in AMI-CS patients.[29] For example, if the benefit of a certain intervention only exists in low-severity patients, then a hypothetical RCT enrolling a nonstratified cohort of patients with CS across the spectrum of shock severity could yield an overall neutral result despite a meaningful effect in this subgroup.[27] The identification of widely applicable phenotypes of CS may facilitate the development of more targeted RCTs tailored to a specific phenotype and not just etiology of CS. Further classification of CS subphenotypes using metabolomic profiles is ongoing and will continue to improve the approach to CS management.[32]

Question 4: Which CS patients (ie, etiology, severity, and phenotype) will benefit from tMCS?

Statement 4A: All patients with evidence of CS should be considered for tMCS.

Quality of evidence: Moderate

Rationale: Within each respective SCAI Shock Classification stage, escalation of care involves increasing levels of surveillance (ie, shorter intervals between clinical and laboratory assessments and hemodynamic data), pharmacologic intervention (ie, vasopressors and/or inotropic support), or mechanical support to maintain or treat end-organ hypoperfusion (Table 2).[3] The decision to escalate care is guided by a comprehensive assessment of the patient's clinical presentation and shock severity progression.[3,9] The frequency of patient assessments should depend on their perceived stability based on the assessment of their response to escalating clinical intervention.

Signs of classic CS (stage C) include hypotension (systolic blood pressure <90 mm Hg or mean arterial pressure <60 mm Hg), persistently low cardiac index (<2.2 L/min/m^2), inadequate lactate clearance (lactate >2 mmol/L), refractory hypoxemia, cardiac power output <0.6 W, evidence of right ventricular failure (including a ratio of right atrial pressure to pulmonary capillary wedge pressure >0.8 or pulmonary artery pulsatility index ≤1.85), and/or recurrent ventricular arrhythmias (Table 2).[4] CS can progress rapidly and has a short-term mortality that ranges from 40% to 50%.[11,33] Beyond the initial escalation of care for patients in stage C CS, which typically involves the provision of diuretics, inotropic agents, and vasopressors, early consideration should be given to initiating tMCS (Figure 2).[34-36]

In contrast to vasopressors and inotropic agents, which may provide only partial resolution of CS derangements, tMCS can allow for clinical stabilization and provide a bridge to decision, recovery, more durable MCS, or even heart transplantation.[4,37] Early initiation of tMCS has been independently associated with reduction in in-patient death, length of stay, respiratory failure, and kidney injury requiring renal replacement therapy.[34-37] As a result, it may be prudent to establish the infrastructure and begin planning for tMCS when patients present in SCAI Shock Classification Stage B to prevent delays in care when patients begin to encounter organ injury and irreversible complications associated with greater shock severity (Figure 3).

Statement 4B: An interdisciplinary discussion prior to nonemergent initiation of tMCS establishes therapeutic goals, including exit strategies.

Quality of evidence: Moderate

Rationale: Care escalation for CS involves the engagement of an interdisciplinary heart team, including cardiac intensivists, cardiac surgeons, interventional and clinical cardiologists, cardiac anesthesiologists, palliative care specialists, nursing, and other consultants. Interdisciplinary heart teams are well established at many institutions to encourage clinicians with different perspectives to collaborate and assist patients and their caregivers in making informed decisions regarding their care.[38] Patient-centered team discussions are focused on the critical appraisal of the underlying disease state and subsequent recommendation of the best treatment strategy. Previously described for transcatheter aortic valve replacement or high-risk cardiovascular interventions, the heart team is increasingly used for a growing list of treatments and new devices to determine care options for a broad range of heart conditions.

Until more high-quality data for different types of patients with CS are available, decisions regarding the indication and timing for tMCS initiation are best made based on a risk-benefit assessment, incorporating etiology, severity of shock, and the burden of comorbidity, with careful evaluation in the context of a heart team discussion.[39] Preliminary evidence from small, single-center studies demonstrates improved rates of 30-day survival after the implementation of a CS team.[40-42] Further, consultation between the initial treating physicians and experts with greater experience in treatment with tMCS is useful to establish therapeutic goals and potential exit strategies after CS diagnosis.[43]

Statement 4C: Patients with increased risk undergoing nonemergent cardiac procedures as well as those patients admitted with CS benefit from incorporating tMCS within the informed consent process.

Quality of Evidence: Low

Rationale: CS after cardiac procedures is a common complication, especially in patients with a reduced ejection fraction, as well as those undergoing a complex cardiac procedure, with increased age, and with other comorbidities.[44-48] The incidence of CS in this patient population is reported to be up to 10% and is associated with increased morbidity and mortality.[48] Early initiation of tMCS, during or immediately after an inciting procedure, has been shown to stabilize hemodynamics, lessen the severity of subsequent organ injury, and is associated with greater likelihood of survival.[34-37] It may be logistically challenging, if not impossible, to properly inform patients and their loved ones of the need for tMCS given the emergent nature of the therapy. Yet, even in those circumstances, the decision to pursue tMCS should still be heavily influenced by the likelihood of therapeutic success and incorporating shared decision-making principles, including the goals, expectations, and desires of the patient and their loved ones. Therefore, particularly for high-risk patients, the potential for tMCS should ideally be introduced as part of the informed consent process before nonemergent procedures and for any patients who present in even early stages of CS.

Although this poses a logistical challenge, and despite the concern that discussions of this nature may negatively influence patients considering high-risk surgery, the process will likely engender greater acceptance in the event tMCS is required. One potential surrogate can be found in research involving an LVAD population, where health care providers endorsed a shared decision-making process, used nonbiased educational materials, and involved a multidisciplinary team sensitive to the tension between conveying enough detail about the therapy yet not overwhelming patients.[49] Researchers demonstrated that this shared decision-making intervention improved concordance between caregiver values and treatment choice for their loved ones.[50] However, despite greater therapeutic concordance, patients and

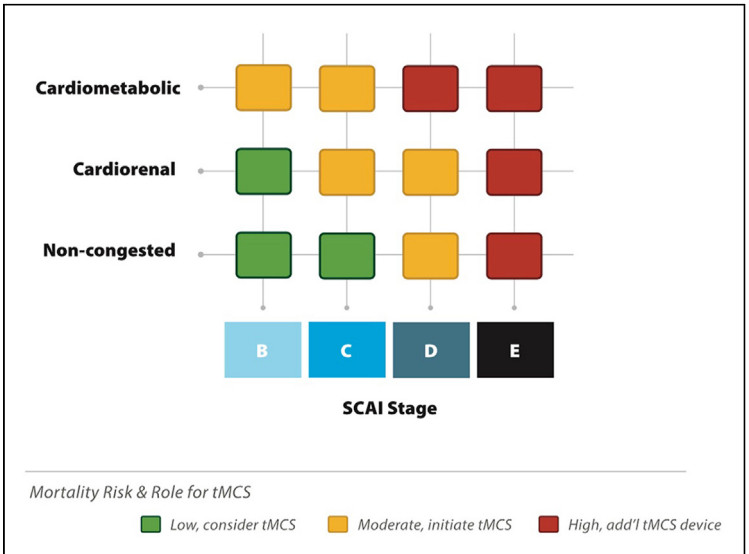

FIGURE 3 Mortality risk and recommended role for temporary mechanical circulatory support (tMCS) based on cardiogenic shock stage and phenotypic classification. (SCAI, Society for Cardiovascular Angiography and Interventions.)

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their caregivers also found the decision aids led to higher initial conflict, without significantly impacting knowledge transfer.[50] This further illustrates the inherent complexity of shared decision-making and the informed consent process as well as highlights important areas for future clinical investigation.

COMMENT

A number of key points have emerged regarding the clinical assessment and phenotyping of CS. Vital to the comprehensive care of patients with CS is the initial and continuous assessment of shock severity using a battery of clinical tools, establishment of etiology, and greater awareness and application of advanced analytics to cluster patients to guide therapeutic intervention and inform research. Ultimately, heart teams are encouraged to provide early consideration for tMCS, coupled with a proactive informed consent process grounded in shared decision-making principles.

LIMITATIONS. Although based on available literature and expert consensus, the guidance provided by this document has inherent limitations. These include the potential of uncaptured published or unpublished data regarding certain interventions owing to the scoping rather than systematic nature of the literature review. Observations are largely isolated to practice patterns in North America and Europe and reflect the expertise and experience of the individuals of the authorship and working group, through discussion focused on establishing pragmatic statements grounded in the available evidence.

Additional studies are necessary to investigate the therapeutic response and safety profile within various etiologies, especially among those with P-CS, which poses a particular challenge to design high-quality, prospectively enrolled trials. This efficacy/safety profile is highlighted in a recent landmark trial that showed a mortality benefit among patients with AMI-CS supported by microaxial-flow pump tMCS, but also an increased risk of adverse events, including severe bleeding, limb ischemia, and hemolysis, among others.[51] Whereas guidance of care based on shock severity has proven useful, the translation of that work to development of machine learning–derived phenotype clusters is in its relative infancy. Future research will likely enhance our ability to prospectively assign patients to individual phenotypic subtypes and assess responses to individual therapies.

Guidance in this document may not be suitable for certain patients, including those with congenital and/or restrictive etiologies, and additional research designed to specifically address those populations is warranted. Although this work introduces a more systematic approach to identifying patients with CS who may benefit from additional support, the role of tMCS in this patient population requires a great deal more attention, particularly to establish algorithms for escalation and de-escalation and identify the appropriate device-pathology relationship.

SUMMARY. CS is a heterogeneous disease with highly variable clinical manifestations, pathophysiology, severity, and outcomes. The aim of assessment and phenotyping of CS is to capture this multidimensional heterogeneity, allowing for improved care of patients and the tailoring of pharmacologic and tMCS strategies.

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