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The Management of Cardiogenic Shock From Diagnosis to Devices:

A Narrative Review

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Abstract

Cardiogenic shock (CS) is a heterogenous syndrome broadly characterized by inadequate cardiac output leading to tissue hypoperfusion and multisystem organ dysfunction that carries an ongoing high mortality burden. The management of CS has advanced rapidly, especially with the incorporation of temporary mechanical circulatory support (tMCS) devices. A thorough understanding of how to approach a patient with CS and to select appropriate monitoring and treatment paradigms is essential in modern ICUs. Timely characterization of CS severity and hemodynamics is necessary to optimize outcomes, and this may be performed best by multidisciplinary shock-focused teams. In this article, we provide a review of CS aimed to inform both the cardiology-trained and non-cardiology-trained intensivist provider. We briefly describe the causes, pathophysiologic features, diagnosis, and severity staging of CS, focusing on gathering key information that is necessary for making management decisions. We go on to provide a more detailed review of CS management principles and practical applications, with a focus on tMCS. Medical management focuses on appropriate medication therapy to optimize perfusion—by enhancing contractility and minimizing afterload—and to facilitate decongestion. For more severe CS, or for patients with decompensating hemodynamic status despite medical therapy, initiation of the appropriate tMCS increasingly is common. We discuss the most common devices currently used for patients with CS—phenotyping patients as having left ventricular failure, right

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ventricular failure, or biventricular failure—and highlight key available data and particular points of consideration that inform tMCS device selection. Finally, we highlight core components of sedation and respiratory failure management for patients with CS.

Keywords

cardiogenic shock; cardiogenic shock severity classification; mechanical circulatory support

Introduction

Cardiogenic shock (CS) is a state of inadequate cardiac output (CO) that results in tissue hypoperfusion, pulmonary and venous congestion, and often multisystem organ dysfunction.[1] CS is a heterogenous syndrome, with variable causes, hemodynamic profiles, and clinical presentations. Despite notable advances in treatment, mortality remains unacceptably high, with figures ranging from 30% to 60%.[2–4] It is increasingly important for all critical care providers to be familiar with the management of CS and options for mechanical circulatory support. In this review, we summarize the basic pathophysiologic principles of CS and its diagnosis, as well as provide a more extensive discussion of contemporary CS management, with a particular emphasis on reviewing options for temporary mechanical circulatory support (tMCS).

Part I: Principles of Cardiogenic Shock

Definition, Classification of Severity, and Clinical Phenotyping

Clinical trials and society guidelines use varying definitions of CS, but most rely on clinical criteria, namely impaired CO resulting in hypotension (systolic BP of < 90 mm Hg or need for vasopressors or mechanical support) as well as clinical or laboratory evidence of end-organ hypoperfusion.[5–8] Commonly referenced hemodynamic criteria include cardiac index of ≤ 2.2 L/min/m^2 and elevated intracardiac filling pressures with pulmonary capillary wedge pressure of > 15 mm Hg.[9] Nonclassical presentations increasingly have been recognized, including normotensive CS, which is associated with similarly high mortality, and right ventricular-predominant CS.[1,10]

To standardize the language surrounding CS, and for prognostication, the Society of Cardiovascular Angiography and Interventions (SCAI) proposed a CS classification scheme in 2019.[11] The SCAI classification uses physical examination findings, hemodynamic parameters, and biochemical markers to categorize patients into five stages of shock severity (SCAI stages A through E). SCAI stage A refers to patients with no signs or symptoms of CS but who are at risk of CS developing because of underlying cardiac pathologic features. SCAI stage B refers to patients who are beginning to show signs of CS, such as tachycardia or relative hypotension, but without end-organ hypoperfusion. SCAI stage C defines patients with classic CS who show clinical evidence of end-organ hypoperfusion requiring pharmacologic or mechanical support, or both. SCAI stage D refers to patients with CS whose condition is deteriorating despite initiation of therapy. Finally, SCAI stage E refers to patients with CS in extremis with refractory shock who are at impending risk of

death (Fig 1).[12] Importantly, the SCAI stages are dynamic and were designed purposefully to allow patient movement between classes as interventions are applied. The SCAI stages are associated with mortality across various cohorts with CS, including those with acute myocardial infarction (AMI) and without AMI.[2,13,14] Biochemical phenotypes of CS (eg, noncongested, cardiorenal, and cardiometabolic shock) also have been defined and validated to risk-stratify patients further within the SCAI staging system.[15]

Cause and Pathophysiologic Features

The causes of CS can be classified according to the site of initial insult: myocardium, conduction system, pericardium, or valves (Table 1). Historically, most cases of CS have been precipitated by AMI.[16,17] However, the epidemiologic features of CS have shifted in recent decades such that decompensated heart failure (HF) has overtaken AMI as the leading cause.[4] In addition, recognition of mixed cardiogenic-distributive shock as a particularly high-risk clinical entity has been growing.[4]

Although the inciting causes of CS may differ, the resultant pathophysiologic features of CS center around reduced CO leading to inadequate tissue perfusion. This triggers a cycle of maladaptive and unsustainable compensatory hemodynamic changes, including increased systemic vascular resistance and fluid retention, which further decrease stroke volume and propagates myocardial ischemia, leading to death if the cycle is not interrupted.[1,16] CS, especially after AMI, also can produce profound systemic inflammation leading to pathologic vasodilation and capillary leakage as opposed to, or after, vasoconstriction, which can exacerbate the deleterious hemometabolic cascade and lead to worsening multiorgan failure.[18]

Part II: Initial Evaluation

The initial evaluation of patients with CS should focus on identification of end-organ hypoperfusion and categorization of severity based on the SCAI classification system. A careful assessment and focused history to identify precipitating factors is essential, because certain underlying causes can alter the initial course of management and the selection of appropriate tMCS devices significantly.

The bedside physical examination provides an important clinical assessment of perfusion and congestion. Common signs of hypoperfusion (so-called cold profile) include altered mental status, oliguria, and cool extremities. Hemodynamically, low output often manifests as relative hypotension (commonly with narrow pulse pressure) and compensatory tachycardia. Common signs of congestion (so-called wet profile) include tachypnea, pulmonary edema, jugular venous distention, and peripheral edema. Although the presence of these signs can be helpful, their absence does not exclude CS, which can present with a so-called cold and dry profile (euvolemic CS) or with a so-called warm and wet profile (vasodilatory CS or mixed shock).[1]

Noninvasive testing initially should include ECG, chest radiography, and a complete transthoracic echocardiography. Point-of-care ultrasound also is used commonly to identify CS cause and complicating factors, such as valvular disease, while awaiting formal

echocardiography.[19] Laboratory testing provides additional information about end-organ dysfunction. All patients should undergo arterial blood gas, baseline lactic acid, complete metabolic panel, complete blood count, and cardiac biomarker testing. Lactic acidosis is an important marker of tissue hypoxia, and both the baseline lactate and subsequent trend are strong predictors of mortality at every SCAI stage.[11,20–24]

Part III: Management

Early Recognition, Regionalized Systems of Care, and Shock Teams

A critical element of successful CS management is early recognition. Efforts to reverse the underlying cause and to restore tissue perfusion rapidly should take place urgently to prevent the deterioration of CS from a hemodynamic problem to a potentially irreversible hemometabolic process.[25,26]

As soon as CS has been recognized, continuous assessment of illness acuity and appropriate repeat triaging is another crucial aspect of early management. Patients treated at institutions with higher CS case volumes and more contemporary CS management strategies have lower mortality, highlighting the impact of experience and expertise.[27] As such, establishing regional systems of care and expedited transfer protocols between smaller community hospitals and high-volume CS centers within a hub-and-spoke model is recommended to improve CS outcomes.[1,28]

In addition to regional care networks, institutional multidisciplinary shock teams have the potential to enhance the quality of early CS management significantly.[10,29,30] Shock teams typically consist of an advanced HF cardiologist, interventional cardiologist, cardiothoracic surgeon, and intensivist. In prospective observational studies, use of a standardized shock team and local treatment algorithms that focus on rapid identification of CS, invasive hemodynamic monitoring, and early, appropriate escalation to tMCS result in improved survival.[10,31]

Hemodynamic Assessment and Monitoring

Initial noninvasive assessments, specifically Doppler echocardiography, can be used to estimate hemodynamic parameters and have been shown to correlate well with those obtained invasively.[32] Several echocardiographic parameters have been shown to be associated with higher hospital mortality in patients with CS at each SCAI stage, including LV ejection fraction, stroke volume index, cardiac index, cardiac power output, and and E/e’ ratio (ratio of early diastolic mitral inflow velocity to early diastolic mitral annulus velocity).[33] The LV outflow tract velocity-time integral is a surrogate measure of stroke volume and has been shown to be a predictor of in-hospital mortality in CS.[34,35] These relatively easily obtained measurements may aid in prognostication and risk stratification of patients with CS and can be performed during point-of-care ultrasound as well as during formal echocardiography. Another noninvasive tool that is being adopted increasingly in the care of patients with CS is lung ultrasound. Lung ultrasound can be used to assess for B-lines that, in the setting of CS, reflect pulmonary vascular congestion and elevated left atrium (LA) pressures.[36] This is superior to chest radiography in patients with chronic HF,

which lacks sensitivity in detecting pulmonary edema, and is less invasive than pulmonary artery catheters (PACs).[36] Notably, other pulmonary pathologic features also may result in B-lines on lung ultrasound, and clinical context must be taken into consideration in interpreting this finding.

Invasive hemodynamic monitoring with PACs provides direct information about biventricular filling pressures, PA pressures, and CO, allowing for the calculation of vascular resistances. These hemodynamic parameters are useful prognostically and are paramount for clinical decision-making for patients with CS. The usefulness of PACs has been debated based on prior studies demonstrating lack of benefit in various clinical contexts, including broad populations of critically ill patients and in acute decompensated HF without shock.[37,38]

Recently, however, evidence to support the clinical benefit of PAC-derived hemodynamic data in guiding CS treatment has been growing, especially in the setting of tMCS.[39–41] An early knowledge of CO and filling pressures allows providers to select the appropriate device(s), and the continuous feedback allows for fine-tuning of treatment decisions. In a large multicenter registry of patients with CS, hemodynamic profiling with PACs before tMCS was associated with improved outcomes, including mortality, particularly in advanced stages of shock.[39] In an observational study of patients with CS resulting from HF (HF-CS), PAC use was associated with decreased hospital mortality, particularly when performed early.[42] A randomized trial of patients with CS currently is underway to assess the impact of early PAC on in-hospital mortality.[43] From a safety standpoint, PAC use can be associated with a small incidence of complications related to central venous access, infection, catheter manipulation, and misinterpretation of the data.[44] However, clinical trial and registry data show that the complication rates are low (< 5%), especially when PAC insertion and management are performed by experienced operators and centers, highlighting the need for procedural expertise.[41,45]

Treating the Underlying Cause

Early identification of the underlying cause of CS is crucial, because certain causes may require specific medical or interventional therapies, or both. In CS resulting from AMI (AMI-CS), emergent revascularization of the occluded coronary artery significantly improves survival, as was demonstrated first in the landmark Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial.[9] Although early revascularization is essential, the optimal revascularization strategy for nonculprit lesions in AMI-CS remains an area of active investigation.[46] Urgent revascularization also should be considered in high-risk patients with non-ST-segment elevation myocardial infarction as well as in patients with known ischemic cardiomyopathy who have progressive shock or refractory arrhythmia.[47]

Many other causes of CS may warrant specific interventions to address the underlying process. For example, acute myocarditis requires prompt immunosuppression, hemodynamically unstable bradyarrhythmias require pacing, unstable tachyarrhythmias require pharmacologic or electrical cardioversion, acute valvular disease may require emergent percutaneous or surgical valvular repair or replacement, tamponade requires

pericardiocentesis or pericardial window, and high-risk pulmonary embolism may require thrombolysis or thrombectomy.

Mixed cardiogenic-septic shock can be caused either by sepsis-induced cardiomyopathy or by an HF exacerbation in a patient with sepsis with preexisting cardiomyopathy, and it poses unique treatment challenges.[48] Although antimicrobial therapy and achieving infection source control are of utmost importance in all patients, the challenges arise primarily regarding fluid balance. Rapid fluid resuscitation is a cornerstone of sepsis management, but it may be detrimental in a patient with decompensated HF and impending CS. In these patients, careful monitoring of respiratory status and fluid balance is crucial, as well as the ongoing reassessment of the relative contribution of septic vs cardiogenic shock to the patient's hemodynamics.[49]

Medical Management and Treatment Targets

Medical management of CS is focused on rapidly restoring tissue perfusion and beginning the process of decongestion to mitigate end-organ damage (Fig 2).[1,2,10,12,50–54] Treatment targets in CS are not defined clearly in guideline recommendations because of limited data and may vary based on the underlying cause of CS.[1,55,56] Generally speaking, hemodynamic targets include achieving a perfusing mean arterial pressure (MAP) and normalization of cardiac index (> 2.2 L/min/m^2) and filling pressures (right atrial pressure, ≤ 10-12 mm Hg; PA diastolic pressure, ≤ 20 mm Hg; pulmonary capillary wedge pressure, ≤ 15-18 mm Hg).[55,57] Notably, although a MAP target of > 65 mm Hg is recommended in septic shock, a lower MAP may be adequate in CS, particularly in the presence of pathologic features that may be exacerbated by excess afterload, such as severe mitral regurgitation.[55,58] Clinical targets include restoration of baseline mental status, improved urine output and respiratory status, and relief of congestion symptoms.[55] Biochemical targets include improvement in markers of end-organ perfusion, including serum creatinine, liver enzymes, and particularly lactate.[55] Indeed, lactate should be monitored every 1 to 4 h until normalization, given the association between lack of lactate clearance and mortality in CS.[1,59]

Early use of vasoactive agents is recommended to optimize perfusion in CS. These agents are of four varieties: vasopressors, inopressors, inodilators, and vasodilators (Table 2).[60] Nearly all of these medications contribute to increased myocardial oxygen consumption and may provoke atrial or ventricular arrhythmias, which can be detrimental in CS.[61] Thus, although the value of these medications cannot be overstated in the acute management of CS, their use ideally should be limited to the lowest doses necessary and for the shortest duration possible.

Inopressors are the preferred first-line agents in hypotensive patients because they increase MAP and provide inotropic support. Several expert consensus documents recommend norepinephrine as the initial agent of choice, and a subgroup analysis of patients with CS enrolled in the Sepsis Occurrence in Acutely Ill Patients (SOAP) II trial showed that norepinephrine was associated with lower rates of death and arrhythmia in CS than dopamine.[62] However, no singular approach exists, and often norepinephrine is used concurrently with the inodilator dobutamine for greater β-agonist effect, or epinephrine can be used as monotherapy. It should be noted that in a small randomized trial comparing

norepinephrine with epinephrine in AMI-CS, norepinephrine use was associated with lower rates of refractory shock, lower lactate levels, and less tachycardia than epinephrine.[63] However, these findings are controversial, and at low doses, epinephrine represents a reasonable, primarily inotropic, agent that may be highly effective and has been used widely in care after cardiotomy.[64,65]

In normotensive CS and hypotensive CS stabilized with pressors, the inodilators dobutamine or milrinone can be used effectively.[66] Notably, the MAP should be adequate (with or without vasopressor support) before the initiation of either agent because of their vasodilatory properties and difficulty predicting individual patient responses to therapy. Despite physiologic and pharmacokinetic differences, a randomized controlled trial comparing dobutamine to milrinone in patients with CS showed no significant difference in outcomes, arrhythmias, or hypotension.[66] Commonly, it is the local practice pattern that dictates which is initiated and patient-specific factors such as renal dysfunction (milrinone being primarily renally cleared). Finally, in normotensive or hypertensive patients with CS, vasodilators such as nitroprusside can be an effective bridge to guideline-directed therapy.[67]

Vascular congestion is common in both AMI-CS and HF-CS and is associated with worse shock severity and increased in-hospital mortality, especially biventricular congestion.[15,57] Persistent congestion at 24 h is associated with a worse prognosis, indicating that PAC-guided decongestion and relief of renovascular congestion should be therapeutic targets in CS.[68] Decongestion can minimize secondary organ dysfunction and can help to enhance perfusion by optimizing ventricular stroke volume and performance. Loop diuretics in combination with thiazide or thiazide-like diuretics should be initiated in patients to achieve sequential nephron blockade synergistically and to achieve more effective diuresis.[69–71] Of note, although sequential nephron blockade can overcome diuretic resistance and can serve as a highly effective therapeutic strategy, it frequently is associated with electrolyte disturbances including hypokalemia, hypomagnesemia, and hyponatremia, and thus electrolytes should be monitored carefully and should be replaced as necessary during treatment.[71] Volume removal using ultrafiltration should be reserved for patients showing inadequate diuretic response. It should be noted that data are insufficient regarding ultrafiltration in CS, as well as evidence that it is inferior to pharmacotherapy in patients with acute decompensated HF.[72]

Mechanical Circulatory Support

The use of tMCS in the management of CS has expanded rapidly despite a paucity of high-quality evidence to support and guide its routine use.[73] Although the development of randomized trials using tMCS in CS has been challenging, equipoise to use tMCS in patients refractory to medical management remains.[74] Multidisciplinary shock teams aid in both patient and device selection, ideally achieving at least center-specific standardization to the use of these high-intensity, high-resource therapies.

The general role of tMCS is to provide hemodynamic support and end-organ perfusion without increasing myocardial oxygen demand, serving as a bridge to recovery, to intervention, or to advanced HF therapies, such as durable ventricular assist device or heart transplantation. Numerous tMCS devices now have entered the market, complicating the

device decision tree (Table 3), but in general, appropriate device selection should be made based on a few key considerations. These include the cause of CS, the type of support needed (ie, LV, RV, or biventricular); the amount of anticipated CO augmentation required; the need for decompression of the LV, RV, or both; the need for oxygenation support; feasibility and safety of placement; and patient-specific potential complications. Device selection also is dictated often by the local expertise and comfort.

LV Predominant Failure

Intra-Aortic Balloon Pump

The most conservative LV-only support device, and the most frequently used nationally, is the intra-aortic balloon pump (IABP), which consists of a conical balloon attached to a peripherally inserted catheter. The appropriate position of the IABP is within the descending aorta, between the renal and the left subclavian arteries.[75] The IABP provides hemodynamic support through counterpulsation, with inflation of the balloon during early diastole and deflation just before systole, gated by either electrocardiography or a fiber-optic arterial pulse wave sensor. Inflation increases diastolic BP in the aorta, augmenting coronary perfusion pressure and MAP, whereas deflation before systole creates a vacuum effect, thus reducing afterload and improving the myocardial oxygen supply to demand ratio.[76] The IABP generally provides only a modest increase in carbon monoxide of 0.5 to 1 L/min, but may be more efficacious in select patients with decompensated HF.[76] Limitations include the device's poor performance in the setting of tachyarrhythmias as well as its contraindication in patients with significant aortic regurgitation.[76]

IABP was studied prospectively in patients with AMI-CS in the IABP SHOCK II trial and was not associated with decreased mortality.[6] However, observational data in HF-CS are encouraging, with some patients being identified as so-called super-responders, augmenting the CO significantly and many achieving stabilization without the need for further tMCS escalation.[76,77] Given the ease of IABP deployment—a viable bedside procedure—and lower vascular complication rates compared with other tMCS devices, the IABP remains a practical option and is a reasonable first step in the management of CS, especially when end-organ function is relatively preserved and hemodynamic collapse is not imminent.[78]

Percutaneous Ventricular Assist Device: Impella: The Impella (Abiomed, Inc.) is a percutaneously implanted, transvalvular, axial flow pump.[79] Axial flow is produced by an impeller that traverses the aortic valve and expels blood from the LV into the ascending aorta. The two available models for LV support are Impella CP (providing up to 4 L/min of flow) and Impella 5.5 (providing up to 5.5 L/min of flow). The CP is inserted femorally via a 14-F sheath, whereas the 5.5 is inserted via a 23-F sheath through a surgical axillary cutdown and graft. Hemodynamically, the Impella directly unloads the LV and augments CO, reducing wall stress and end-diastolic pressure and volume. Limitations of the Impella include access site complications resulting from large-bore cannulation, as well as significant risk for hemolysis (with associated renal failure) and thrombocytopenia, especially with the Impella CP.[79] Impella placement requires fluoroscopy and, in the case of

the Impella 5.5, surgical cutdown and graft anastomosis; thus, the Impella typically is not an option for emergent bedside placement.

It is the opinion of the authors that, given the modest CO support and high rates of hemolysis and kidney injury, the Impella CP is no longer considered to be an adequate longitudinal shock device at many shock referral centers. Instead, the axillary-placed Impella 5.5, which also enables ongoing rehabilitation before heart replacement therapy, is preferred for more sustained support.[80–82]

In patients with AMI-CS, the Impella was compared with the IABP in the Impella vs IABP Reduces Mortality in STEMI Patients Treated With Primary PCI in Severe Cardiogenic Shock (IMPRESS) trial, which showed no mortality benefit for the Impella over the IABP.[7] However, it is important to note that the trial design included patients who had progressed to profound hemometabolic CS, with > 90% of patients having experienced cardiac arrest. Observational data show inconsistent findings. Although the National Cardiogenic Shock Initiative touts early deployment of the Impella and demonstrated astounding inpatient survival rates of > 70%, a propensity-matched cohort study showed worse mortality with the Impella compared with the IABP and increased vascular complications.[83,84] Additional randomized controlled trials are underway to assess the efficacy of the Impella in AMI-CS better.[85,86] The lack of randomized data supporting the efficacy and usefulness of the Impella 5.5 device is notable.

Venoarterial ECMO: Venoarterial ECMO provides cardiopulmonary support to patients in CS, with or without concomitant respiratory failure.[87] Blood is removed from the venous circulation via a drainage cannula in the right atrium, circulated via a centrifugal pump through an oxygenator, and then returned to the arterial system either peripherally via a cannula in the femoral or subclavian artery or, less commonly, centrally into the aorta.

Hemodynamically, venoarterial ECMO provides circulatory support and improved end-organ perfusion and unloads the RV, but it significantly increases LV afterload resulting from retrograde arterial flow.[88] In cases of severe LV dysfunction, venoarterial ECMO can lead to increased LV pressures, myocardial oxygen demand, pulmonary edema, and potentially LV to LA stasis. As such, venoarterial ECMO for LV-predominant CS often is used in tandem with an LV so-called vent to maintain aortic valve opening, to reduce LV end-diastolic pressure, and to protect the lungs. Venting strategies commonly include concurrent Impella, IABP, atrial septostomy, or transeptal LA-RA venous limb drainage.[89] Although venting data are sparse, mechanical unloading may be associated with improved survival compared with venoarterial ECMO alone.[90,91] However, a recent randomized controlled trial showed that early unloading was not superior to a rescue strategy, so upfront venting may not be necessary for all patients.[92] One particular challenge in the management of venoarterial ECMO is maintaining the delicate balance between the need to vent the LV, which outputs relatively deoxygenated blood in the setting of respiratory failure, and the need to circulate oxygenated blood provided by the ECMO circuit. The location where this blood mixes within the aorta, known as the mixing cloud, is of particular clinical importance to avoid differential oxygenation of tissue beds (ie, north-south syndrome).

Results of the first randomized controlled trial for venoarterial ECMO in patients with AMI-CS who were planned for revascularization were published in 2023 (Extracorporeal Life Support-Shock [ECLS-Shock]).[93] Four hundred patients with AMI-CS were randomized to early tMCS support with venoarterial ECMO (with predefined criteria for LV venting) or medical management alone. The patients enrolled in ECLS-Shock were critically ill, with a median lactate of 6.9 mM, 78% having received CPR before enrollment, and 50% categorized as having SCAI stage D or E disease. Death resulting from any cause at 30 days was not different between the two study groups, and both bleeding and peripheral ischemia were more prevalent in the venoarterial ECMO group.

Overall, the results of ECLS-Shock suggest that nonselective early use of venoarterial ECMO should not be standard first-line care in AMI-CS. However, a number of questions remain to be answered, and it can be argued that venoarterial ECMO has never been considered (nor should it be) a first-line therapy to begin with. Within the trial protocol, patients in the treatment arm were not assessed for alternative methods of tMCS that may have been more appropriate on an individual patient basis. Additionally, only 5.8% of patients supported with venoarterial ECMO received mechanical LV unloading, which may have impacted the efficacy of the intervention on reducing LV demand and filling pressures. The trial population was both generally older and severely ill, with 78% having experienced a cardiac arrest. In considering translatability to a real-world cohort of patients with AMI-CS, > 60% would not have met eligibility for the trial, so there may be populations in which venoarterial ECMO could be expected more reasonably to alter outcomes.[94] Venoarterial ECMO should be reserved for carefully selected patients with AMI-CS, and especially those whose disease is refractory to medical management alone.[95] Finally, the trial results are limited in their generalizability and should not be taken to apply to all patients with CS, such as those with HF-CS.

TandemHeart: TandemHeart (LivaNova, Inc.) is a percutaneously placed extracorporeal centrifugal pump that uses a 21-F transeptal inflow cannula—blood is removed from the LA—and either a 15-F or 17-F arterial return cannula in the aorta to deliver 4 to 6 L/min of flow.[96] This allows for circulatory support with concomitant LV unloading, but without RA drainage, thus providing minimal RV support. Right-sided configurations with RA drainage and PA return can be used as well. One limitation of this device is the requirement for transeptal puncture, which is a technically challenging process requiring specific interventional cardiology expertise.

LV Failure Device Selection: The choice of tMCS device in LV-predominant shock is dictated by the cause and severity of CS, rapidity of progression, the presence of concurrent respiratory failure, and perhaps most significantly, by local expertise and comfort. Indeed, in the absence of high-quality data or recognized guidelines, device selection is driven by expert opinion. The following represents one approach, supported by the authors.

As stated, no clear mortality benefit to Impella CP over IABP has been shown in randomized trials, and an association with increased vascular complications exists.[97] With this in mind, IABP often is the device of choice in the absence of profound hemometabolic shock.[77] In these cases, the IABP is selected with the aim of providing sufficient CO and perfusion

support while also avoiding larger devices with higher-risk profiles.[76,83] However, Impella CP inarguably provides substantially more CO support and LV unloading compared with IABP, can be placed under fluoroscopy similarly, and has been used with astounding success in some national shock registries.[83] When placed for AMI-CS, the Impella CP typically is considered a short-term support device with the aim of stabilizing a patient until they can be weaned off the Impella CP or the device can be upgraded to a more sustainable LV support device.[86] In practice, the Impella CP often is placed at referring centers as a means of patient stabilization until they can be transferred to a hub shock center. In the setting of profound hemometabolic shock or progressive shock despite IABP or Impella CP, full LV support can be provided by venoarterial ECMO or the Impella 5.5. The decision between these two devices often is determined by the perceived risk of subsequent RV failure after Impella 5.5 placement (akin to RV failure after durable LV assist device placement), the need for oxygenation, and the urgency of cannulation.[81] Venoarterial ECMO can be placed readily at the bedside, and thus often is preferred when a patient is in extremis. However, the Impella 5.5 is preferable for long-term support because it allows for ongoing rehabilitation. If necessary, a patient can be cannulated to venoarterial ECMO emergently, with the Impella 5.5 subsequently placed as a venting strategy and venoarterial ECMO as an exit strategy.

RV Predominant Failure

Several tMCS options are available for RV-predominant CS, although they are fewer and less well studied compared with those used for LV-predominant CS.[98] Venoarterial ECMO often is the most reliable and readily available device because it bypasses the dysfunctional RV, dramatically decreasing RV preload without increasing RV afterload. In addition to venoarterial ECMO, RV-only support can be provided with a right-sided Impella device called the Impella RP, as well as the Protek Duo cannula (LivaNova), which can function in line with the TandemHeart pump or an ECMO circuit.[99] The Impella RP aspirates blood from the RA and expels it into the PA, using an axial transvalvular impeller similar to other Impella devices.[100] The Protek Duo is a dual-lumen cannula that also aspirates blood from the RA and ejects it into the PA. However, unlike the Impella, blood is removed from the body via a centrifugal pump before reinfusion, allowing for flexibility of pump types as well as the insertion of an oxygenator.[101] Both the Protek Duo and, more recently, the novel Impella RP flex can be placed via internal jugular access, thus eliminating the need for femoral access and allowing for patient mobilization.

RV Failure Device Selection

Venoarterial ECMO often is the device of choice in patients with RV-predominant failure, when escalation to tMCS is needed rapidly. If oxygenation is a concern, then only venoarterial ECMO or the Protek Duo in line with an oxygenator are feasible choices.[102] The other main determinant of RV support selection concerns the status of RV afterload and pulmonary vascular resistance (PVR). Minimal data are available to support the use of RA-PA flow devices in the setting of severely elevated PVR and increased RV afterload, such as in acute PE or long-standing pulmonary arterial hypertension. Reinfusion into a noncompliant pulmonary circulation can impact the amount of generated flow negatively as well as lead to complications such as pulmonary hemorrhage, and in these cases,

venoarterial ECMO is the preferred tMCS.[103] Although reports have indicated that these devices can be used successfully with elevated PVR, RA-PA flow devices are best suited to situations of primary RV failure, including RV AMI, RV failure after heart transplant, or LV assist device implantation.[104,105]

Biventricular Failure

AMI-CS as well as various forms of CS not owing to AMI can result in biventricular failure, which is associated with worse mortality than isolated LV failure.[106,107] When selecting a tMCS strategy for the patient with biventricular failure, it is important to consider the hemodynamic impact of the selected device on both left-side and right-side circulation. Options for biventricular support include venoarterial ECMO, biventricular extracorporeal centrifugal pumps such as the TandemHeart or Protek Duo, the biventricular Impella (BiPella), or the LV Impella plus Protek Duo.[108] As in other cases, the decision here is driven largely by institutional preference and comfort, with venoarterial ECMO—often requiring an LV vent—being the simplest and most rapidly deployed option.

Respiratory Failure and Sedation in CS

Respiratory failure is common in CS because of elevated left-sided filling pressures resulting in pulmonary vascular congestion and cardiogenic pulmonary edema. Respiratory support for patients with CS is aimed at minimizing hypoxemia, correcting respiratory acidosis and hypercapnia, and minimizing the work of breathing and high catecholamine states.[109] Noninvasive ventilatory support with high-flow nasal cannula or bilevel positive pressure ventilation should be attempted; however, in more severe CS with progressive acidosis, altered mental status, and hemodynamic instability, invasive mechanical ventilation often is required.[110] Positive pressure ventilation, either via noninvasive or invasive strategies, can impact CO and myocardial oxygen demand significantly, and the specific impact depends largely on the relative RV and LV dysfunction.[111] Broadly, high transpulmonary pressures, either at end expiration (positive end-expiratory pressure) or with inspiration, lead to increased PVR and to increased RV afterload.[110] In the same circumstances, the LV experiences decreased preload and filling and decreased afterload because of reflex vasodilation and decreased transmural pressures, resulting in decreased myocardial oxygen demand.[110] With these considerations, the ventilator parameters should be adjusted to optimize CO with PAC feedback while maintaining gas exchange.

For patients with CS requiring invasive mechanical ventilation, the priorities should be analgosedation with opioids, and delirium control with primary goals of reducing pain, achieving light sedation, and minimizing sympathetic stimulation,[112] which can contribute to arrhythmias and hemodynamic instability. When deeper sedation is required, sedative hypnotics and anxiolytics should be used. Although propofol often is avoided because of concerns about its cardiodepressive effects, data are emerging that it likely is safe and may even be associated with improved outcomes as compared with benzodiazepines.[113] Notably, data addressing optimal sedation in CS remain largely observational.

Conclusions

CS is a heterogeneous syndrome that requires prompt recognition and urgent management. The first stage of approaching a patient with CS is to determine the underlying driving insult, the hemodynamic state, and the severity of shock. This is achieved best with a combination of noninvasive and invasive hemodynamic testing and multidisciplinary discussion. As soon as a diagnosis is reached, the cornerstones of management include medication therapy to optimize perfusion and decongestion and initiation of tMCS for patients with severe or worsening CS.

ABBREVIATIONS:

AMI	acute myocardial infarction
AMI-CS	cardiogenic shock resulting from acute myocardial infarction
CS	cardiogenic shock
ECMO	extracorporeal membrane oxygenation
HF	heart failure
HF-CS	cardiogenic shock resulting from heart failure
IABP	intra-aortic balloon pump
LA	left atrium
LV	left ventricle
MAP	mean arterial pressure
PA	pulmonary artery
PAC	pulmonary artery catheter
PCI	percutaneous coronary intervention
PVR	pulmonary vascular resistance
RA	right atrium
RV	right ventricle
SCAI	Society of Cardiovascular Angiography and Interventions
tMCS	temporary mechanical circulatory support

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CLINICAL QUESTION

A 42-year-old woman whose toddler recently received a diagnosis of Coxsackie A virus demonstrated upper respiratory symptoms and was in the ICU with cardiogenic shock (CS). The echocardiogram revealed normal left ventricular volume, global hypokinesis (ejection fraction, 15%), and severe right ventricular dysfunction. A pulmonary artery catheter was placed and showed a right atrial pressure of 24 mm Hg, pulmonary artery pressure of 68/35/46 mm Hg, and pulmonary capillary wedge pressure of 32 mm Hg, with a cardiac output of 2.8 L/min and a cardiac index of 1.6 L/min/m^2. Despite inopressor support, she shows rising lactate and persistent hypoxia and is becoming more encephalopathic with frequent episodes of nonsustained ventricular tachycardia.

What is the next best step in her management?

A: Place an Impella 5.5 B: Place an intra-aortic balloon pump C: Cannulate for venoarterial extracorporeal membrane oxygenation (ECMO) D: Place an Impella CP E: Serum C-reactive protein

Answer: C, Venoarterial ECMO

The patient demonstrated severe CS likely secondary to myocarditis with evidence of biventricular failure based on echocardiography and right heart catheterization findings. She demonstrated Society of Cardiovascular Angiography and Interventions stage C shock at presentation and deteriorated to Society of Cardiovascular Angiography and Interventions stage D shock given the inadequate response to medical therapy. At this stage, temporary mechanical circulatory support (tMCS) is warranted based on the worsening clinical status, young age, and absence of contraindications to therapy. The selection of the appropriate tMCS device is dependent on the acuity of the presentation, the type of hemodynamic support required, and local comfort and expertise. In this situation, the patient required biventricular support and additional oxygenation support, making venoarterial ECMO the only appropriate choice listed. Impella CP, Impella 5.5, and intra-aortic balloon pump all provide left ventricle (LV)-only support and do not provide additional oxygenation, making them inadequate support devices in this case (choices A, B, and C are incorrect). Given the severe LV dysfunction, she requires close monitoring for need of LV venting, and after a period of stabilization, she may warrant transition to a more sustainable tMCS option as a bridge to recovery, durable mechanical support, or heart transplantation.

Key Points

1. Cardiogenic shock (CS) is defined by impaired cardiac output resulting in hypotension, clinical or laboratory evidence of end-organ hypoperfusion, or both; Society of Cardiovascular Angiography and Interventions classifications should be used in the evaluation of a patient with CS to standardize language, to help with prognostication, and to identify appropriate treatment pathways.
2. Multidisciplinary shock teams and experienced regional care centers can enhance the recognition and initial management of patients with CS and can improve outcomes.
3. Pulmonary artery catheters provide valuable hemodynamic information during CS, informing shock phenotypes and allowing for the monitoring of treatment response; increasing evidence suggests clinical benefit when used early in the course of disease and in severe shock.
4. Medical management of CS should focus on reversing the underlying cause and using pharmacologic agents to optimize perfusion and to reduce congestion; when vasoactive agents are inadequate to restore systemic perfusion, mechanical circulatory support should be considered.
5. The method of temporary mechanical circulatory support should be selected based on the type of support that is necessary (ie, left ventricular, right ventricular, biventricular), the anticipated amount of necessary cardiac output support, the need for oxygenation, the feasibility and safety of placement, and the potential for patient-specific complications.

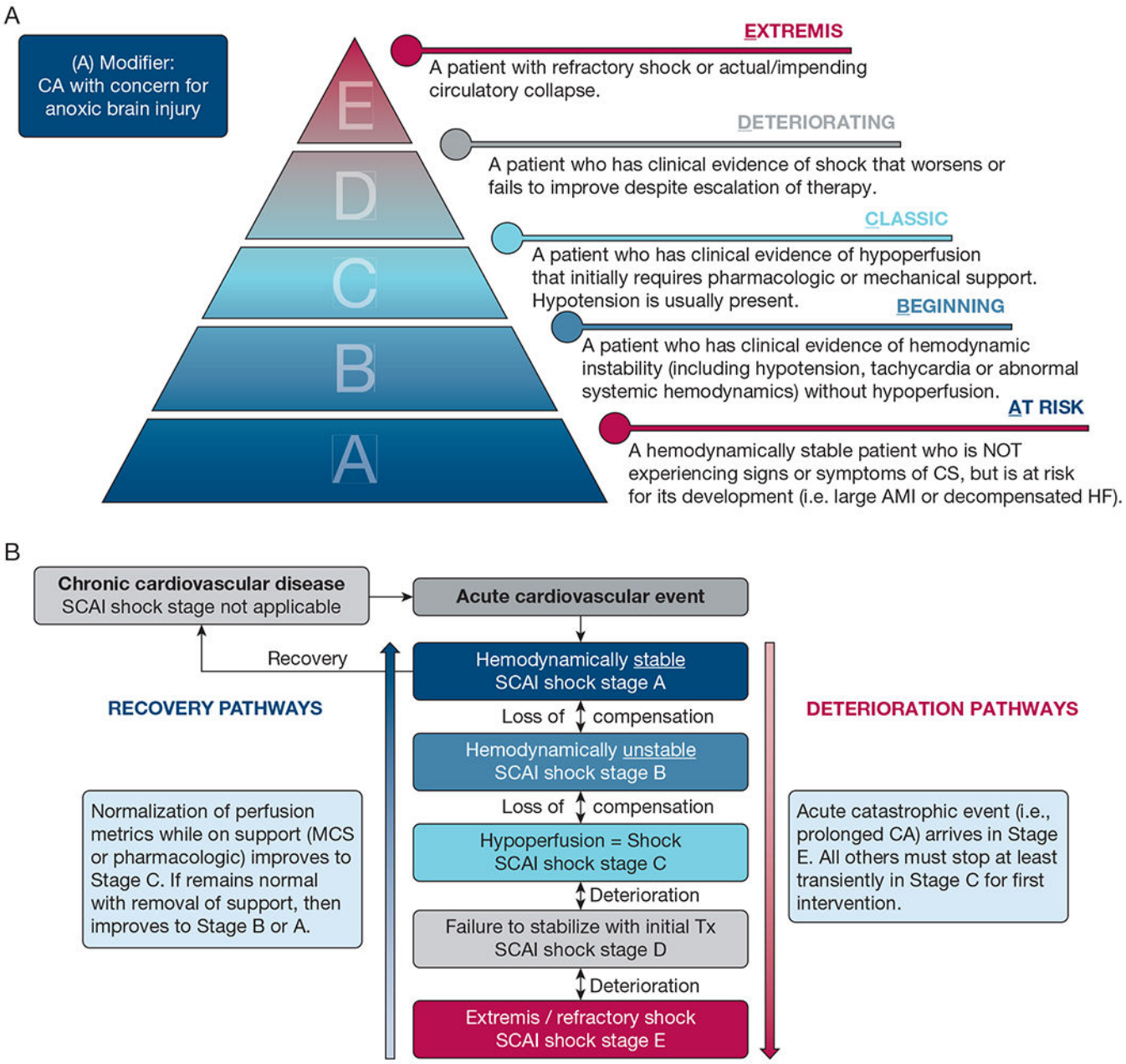

Figure 1 –.

A, B, Diagrams showing SCAI shock classification pyramid (A) and the dynamic evolution of cardiogenic shock and progression or recovery through SCAI shock stages (B). AMI = acute myocardial infarction; CA = cardiac arrest; CS = cardiogenic shock; HF = heart failure; MCS = mechanical circulatory support; SCAI = Society for Cardiovascular Angiography and Interventions; Tx = treatment. (Reproduced with permission from Naidu et al.[12])

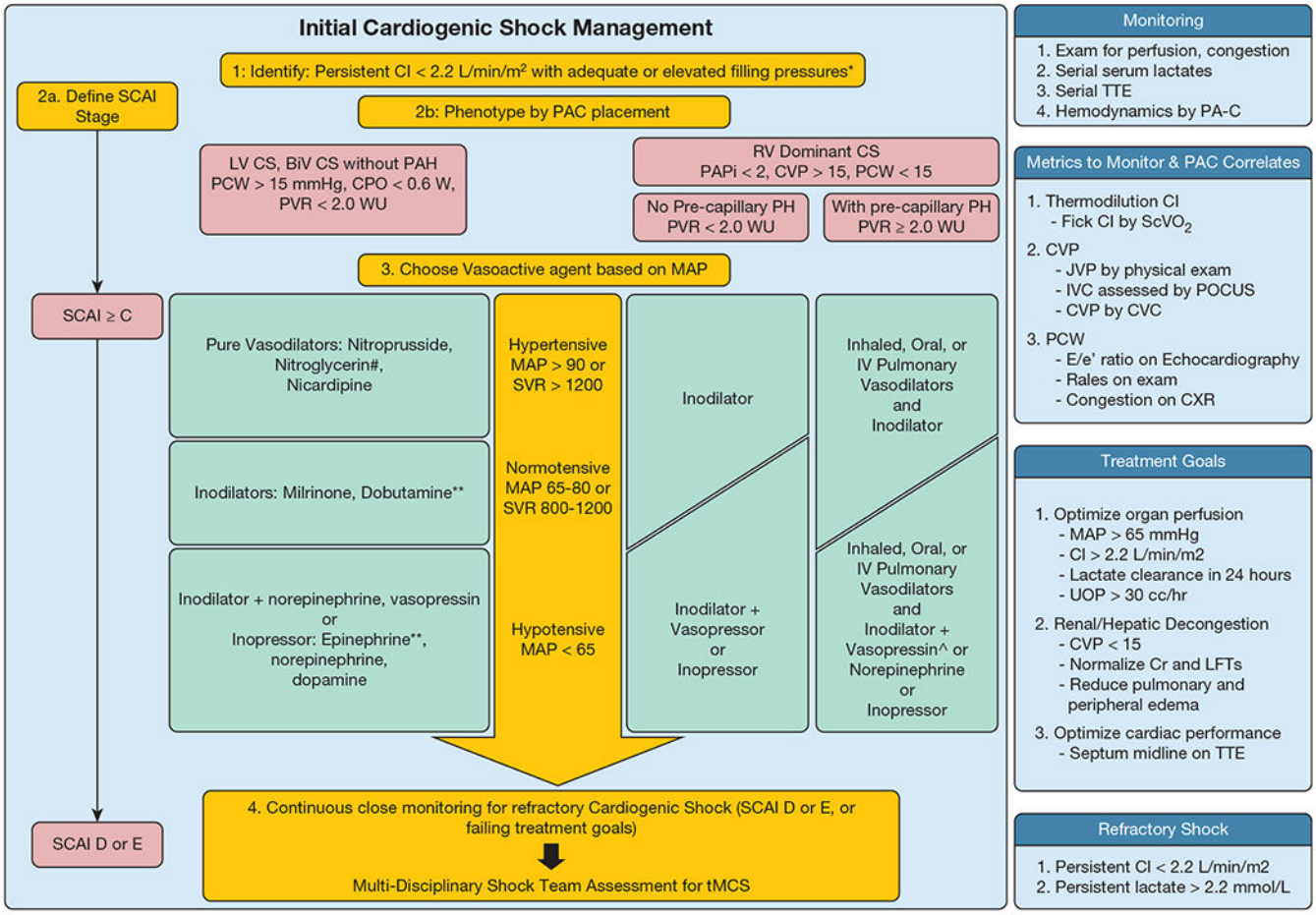

Figure 2 –.

Flowchart showing medical management of cardiogenic shock. This management algorithm represents the expert opinions of the authors and is informed by trial data and society guidelines.[1,2,10,12,50–54] No singular approach to treating CS exists, and this is meant to serve as a guide, not the only definitive approach. Key components in the management of CS include early recognition, SCAI staging, and phenotyping by PAC. Early treatments for CS prioritize prompt resolution of organ hypoperfusion using vasoactive agents with close monitoring for deterioration. Vasoactive agent use is tailored to an individual patient based on the hemodynamic phenotype. Please see Table 2 for more information on vasoactive agents. BiV = biventricular; CI = cardiac index; CPO = cardiac power output; Cr = creatinine; CS = cardiogenic shock; CVC = central venous catheter; CVP = central venous pressure; CXR = chest radiograph; E/e' ratio = ratio of early diastolic mitral inflow velocity to early diastolic mitral annulus velocity; IVC = inferior vena cava; JVP = jugular venous pressure; LFT = lung function test; LV = left ventricle; MAP = mean arterial pressure; PAC/PA-C = pulmonary artery catheter; PAH = pulmonary arterial hypertension; PAPi = pulmonary artery pulsatility index; PCW = pulmonary capillary wedge pressure; PH = pulmonary hypertension; POCUS = point-of-care utrasound; PVR = pulmonary vascular resistance; RV = right ventricle; SCAI = Society for Cardiovascular Angiography and Interventions; $ScVO_2$ = central venous oxygen saturation; SVR =

systemic vascular resistance; tMCS = temporary mechanical circulatory support; TTE = transthoracic echocardiography; UOP = urine output; W = watts; WU = Wood unit. [a]Patients who are hypovolemic and have inadequate preload can have low CI and a high SVR mimicking CS. Patients should show adequate filling pressures and be resuscitated as needed before proceeding with CS diagnosis. [b]Nitroglycerin provides more venous than arterial vasodilation and often is used in patients with volume overload, acute coronary syndrome, or both. [c]In CS resulting from acute myocardial infarction, inotropes can increase myocardial oxygen demand and increase or provoke ischemia. These agents should be used with caution and in expert centers. [d]Evidence exists that vasopressin has more impact on SVR than PVR, but norepinephrine often is used also in patients with pulmonary arterial hypertension in shock given the larger titratable range and also a favorable SVR to PVR impact.

TABLE 1]

Common Causes of CS

Variable	Myocardial		Pericardial	Valvular	Conduction
	RV	LV			
CS resulting from an acute insult	• Acute pulmonary embolism • RV AMI	• AMI • Myocarditis • Stress CM • Peripartum CM • Ventricular septal rupture • LVFWR • Postcardiotomy	Cardiac tamponade	• Acute valvular regurgitation ○ Endocarditis ○ Cord rupture ○ Dissection • Mechanical valve thrombosis	• Ventricular arrhythmia • Unstable supraventricular arrhythmia • Bradycardia ○ AV node block ○ Sinus dysfunction
CS resulting from decompensated chronic disease	• Pulmonary arterial hypertension • Chronic thromboembolic pulmonary hypertension	• Ischemic CM • Nonischemic CM • Restrictive CM • Hypertrophic CM	Constrictive pericarditis	• Aortic stenosis • Mitral stenosis • Mitral regurgitation	• Tachycardia mediated cardiomyopathy

CS is a heterogeneous condition with varying causes. It is helpful to categorize CS based on the driving pathologic feature, but it is important to recognize that multiple interacting pathologic drivers are common. AMI = acute myocardial infarction; AV = atrioventricular; CM = cardiomyopathy; CS = cardiogenic shock; LV = left ventricle; LVFWR = left ventricular free wall rupture; RV = right ventricle.

TABLE 2]

Vasoactive Agents Used in CS

Category	Agent	Receptor Affinity/Target	Hemodynamics	Special Considerations
Vasopressors	Vasopressin	V_{1a}, V_2	Increases SVR, and thus MAP, through V_{1a}-mediated vascular smooth muscle vasoconstriction	• No chronotropic or inotropic effects • May result in digital ischemia
	Phenylephrine	α-1	Increases SVR, and thus MAP, through α-1 mediated vasoconstriction	• No chronotropic or inotropic effects • May result in reflex bradycardia • Can be agent of choice in hypotension because of hypertrophic obstructive cardiomyopathy
Inopressors	Norepinephrine	α-1 ++++ β-1 +++ β-2 ++	Increases SVR and augments CO by increasing HR and contractility	• Increased risk of atrial and ventricular arrhythmias
	Epinephrine	α-1 ++++ β-1 ++++ β-2 ++	Increases SVR and augments CO by increasing HR and contractility	• Increased risk of atrial and ventricular arrhythmias and lactic acidosis
	Dopamine	α-1 +++ β-1 +++ β-2 ++ DA ++++	Acts in a dose-dependent fashion to increase HR, contractility, and SVR at higher doses	• Increased risk of atrial and ventricular arrhythmias
Inodilators	Dobutamine	β-1 ++++ β-2 +++	Increases contractility and reduces SVR	• Increased risk of atrial and ventricular arrhythmias • Vasodilatory and can lower MAP in patients with mixed shock
	Milrinone	PDE-3 inhibitor	Increases contractility and reduces SVR	• Caution in patients with worsening renal function resulting from predominant renal excretion • Increased risk of atrial and ventricular arrhythmias
Vasodilators	Sodium nitroprusside	Generates NO in circulation	Promotes both arterial and venous vasodilation; reduces ventricular afterload and preload	• No chronotropic or inotropic effects • May result in marked hypotension in patients without contractile reserve • Requires close monitoring of thiocyanate toxicity especially with prolonged use or in patients with renal dysfunction
	Nitroglycerin	Generates NO in circulation	Promotes primarily venous vasodilation and reduces ventricular preload	• No chronotropic or inotropic effects

Number of + signs indicates the strength that drug acts on the described receptor. CO = cardiac output; CS = cardiogenic shock; DA = dopamine; HR = heart rate; MAP = mean arterial pressure; NO = nitric oxide; PDE = phosphodiesterase; SVR = systemic vascular resistance; V = vasopressin.

TABLE 3]

Summary of Temporary Mechanical Circulatory Support Devices

Device	Pump Mechanism	Configuration	Access Point(s)	Lumen Size	Duration of Use (FDA Approved)[a]	Advantages	Limitations
Left-sided support							
IABP	Counterpulsation	Ao	Femoral or axillary artery	7-8 F	9 d	• Ease of placement • Low-profile vascular access • Favorable safety profile	• No oxygenation capacity • Modest cardiac output augmentation and hemodynamic support • Poor performance in arrhythmias • Contraindicated in severe aortic regurgitation
Impella CP	Axial flow	LV to Ao	Femoral artery	14 F	14 d	• Single arterial access • Direct LV support and decompression	• No oxygenation capacity • Unstable positioning • Frequent hemolysis • Requires AC
Impella 5.5	Axial flow	LV to Ao	Axillary artery, via graft	23 F	14 d	• Single arterial access • Direct LV support and decompression	• No oxygenation capacity • Requires surgical placement • Requires AC
Right-sided Support							
Impella RP (Flex)	Axial flow	RA to PA	Right IJ or femoral vein	11 F	14 d	• Single venous access	• No oxygenation capacity • Contraindicated with right sided valvular disease • Contraindicated with IVC or right-sided clots, or IVC filter • Risk of pulmonary hemorrhage if high PVR • Requires AC
Protek Duo	Centrifugal flow	RA to PA	Right IJ vein	29 F or 31 F	< 24 h	• Single IJ vein access allows patient to remain ambulatory • Allows addition of oxygenator	• Contraindicated with IJ stenosis or thrombosis • Contraindicated with right-sided valvular disease • Risk of pulmonary hemorrhage if high PVR
Biventricular support							
TandemHeart	Centrifugal flow	LA to Ao or RA to PA	Left-sided support: Femoral vein + femoral artery Right-sided support: Bilateral femoral veins or femoral	Inflow: 21F Outflow: 15-19F	30 d	• Allows addition of oxygenator • Provides LV unloading	• Need for atrial transseptal puncture for left-sided support • Limited patient mobility • Requires AC

Device	Pump Mechanism	Configuration	Access Point(s)	Lumen Size	Duration of Use (FDA Approved)[a]	Advantages	Limitations
			vein and right IJ vein				
Venoarterial ECMO	Centrifugal flow	RA and IVC to Ao	Femoral or IJ vein to femoral artery	Varies by patient size; venous cannula, 21-29 F; arterial cannula, 15-21 F	9 d	• Provides gas exchange • Ease of cannulation: can be performed at bedside without fluoroscopy depending on institutional experience • Bypasses RV	• Frequently causes LV distention and requires secondary device—LV vent—for mechanical unloading • Risk of differential oxygenation (north-south syndrome) • Contraindicated in severe peripheral arterial disease • Contraindicated with aortic regurgitation • Requires AC

AC = anticoagulation; Ao = aorta; FDA = US Food and Drug Administration; IABP = intraaortic balloon pump; IJ = internal jugular; IVC = inferior vena cava; LA = left atrium; LV = left ventricle; PA = pulmonary artery; PVR = pulmonary vascular resistance; RA = right atrium; RV = right ventricle; ECMO = extracorporeal membrane oxygenation.

[a]Although these are the FDA-approved durations, temporary mechanical circulatory support devices commonly are used off-label and courses may extend well beyond these durations.