Cardiogenic Shock and Temporary Mechanical Circulatory Support

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KEYWORDS

• Cardiogenic shock • Acute myocardial infarction • Heart failure
• Mechanical circulatory support • Critical care cardiology

KEY POINTS

- Cardiogenic shock is a clinical syndrome that commonly occurs due to myocardial infarction, heart failure decompensation, and de novo heart failure.
- Identification of cardiogenic shock stage and type is critical in guiding management including the use of revascularization and mechanical circulatory support.
- Biventricular failure is associated independently with higher hospital mortality.
- Mechanical circulatory support is a critical tool in the treatment of cardiogenic shock; patient selection is key and is not without complications.

INTRODUCTION

Cardiogenic shock (CS) is a syndrome that arises from severe cardiac dysfunction and leads to hypotension, systemic hypoperfusion, and ultimately often spirals to death. Acute myocardial infarction (AMI) and heart failure-related CS (HF-CS) are the most common causes of CS.[1] Other causes include postcardiotomy, postcardiac arrest, pericardial disease, valvular disease, arrhythmia, right ventricular (RV) failure, peripartum cardiomyopathy (PPCM), and myocarditis.[2] Early recognition, hemodynamic profiling, use of mechanical circulatory support (MCS), and multidisciplinary teams

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Med Clin N Am 109 (2025) 1255–1272
https://doi.org/10.1016/j.mcna.2025.04.007 **medical.theclinics.com**

Abbreviations	
AMI	acute myocardial infarction
cAMP	cyclic adenosine monophosphate
CI	confidence interval
CS	cardiogenic shock
ECMO	extracorporeal membrane oxygenation
ECPR	extracorporeal cardiopulmonary resuscitation
EPI	epinephrine
HF	heart failure
HF-CS	heart failure-related CS
IABP	intra-aortic balloon pump
LVOT	left ventricular outflow tract
MAP	mean arterial pressure
MCS	mechanical circulatory support
NE	norepinephrine
PACs	pulmonary arterial catheters
PDE3	phosphodiesterase 3
PPCM	peripartum cardiomyopathy
pVADs	percutaneously ventricular assist devices
RV	right ventricular
SCAI	Society for Cardiovascular Angiography
VA	veno arterial

are crucial for better outcomes. However, despite improved diagnostics and treatments, short-term mortality for CS remains in excess of 30%.[3]

HISTORY

First classified in 1940, CS was defined as failure of the circulation attributable to primary disease of the heart predominately caused by AMI-CS.[4] Standard therapies beyond oxygen and morphine included ethyl alcohol vapor, digitalis, and quinidine. Cortisone and intra-arterial infusions of blood and plasma were also used without marked success.[4] The use of norepinephrine was found to lower mortality to 68% and became the vasopressor drug of choice. When patients with CS received care in a designated coronary care unit, it improved their in-hospital mortality rate largely due to increased access to closed chest cardiac massage and prompt bedside defibrillation. Development of the Swan–Ganz catheter later led to further hemodynamic profiling. Dopamine was also introduced in patients with AMI-CS and found to improve peripheral perfusion but at the expense of myocardial oxygen consumption.[4] In 1962, the intra-aortic balloon pump (IABP) was first created and tested in dogs using carbon dioxide, and then successfully in humans with helium-filled balloons in 1968 and demonstrated improved cardiac output. Early revascularization, either with coronary artery bypass grafting or percutaneous coronary intervention, became a cornerstone therapy for AMI-CS after the publication of the SHOCK (SHould we emergently revascularize Occluded Coronaries for Cardiogenic ShocK) trial in 1999. The development of several percutaneous mechanical support devices followed in the decades later providing clinicians with an array of options to provide hemodynamic support with the first positive trial in 2024.[5] Although the mortality from CS remains high, especially in women and the elderly populations, modern therapies improve the chance of survival. CS, once associated with near 100% mortality, is now associated with survival rates of greater than 60% to 70%.[3]

Definitions

There have been several proposed definitions listed in **Table 1**.

Table 1
Organizational difference in cardiogenic shock definition

Year	Organization	Definition
2023	Shock Academic Research Consortium[6]	*Pragmatic definition*: A cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion irrespective of underlying blood pressure *Comprehensive definition:* A cardiac disorder that results in a systolic blood pressure (SBP) less than 90 mm Hg for ≥30 min (or the need for vasopressors, inotropes, or MCS to maintain SBP ≥90 mm Hg) with evidence of tissue hypoperfusion
2022	Cardiogenic Shock Working Group[7]	A sustained episode of at least 1 of the following: SBP <90 mm Hg for at least 30 min, use of vasoactive agents to maintain SBP, cardiac index <2.2 L/min/m^2 in the absence of hypovolemia, each determined to be secondary to cardiac dysfunction, or use of an MCS device for clinically suspected CS
2017	American Heart Association[8]	*Pragmatic definition*: A state in which ineffective cardiac output caused by a primary cardiac disorder results in both clinical and biochemical manifestations of inadequate tissue perfusion. The clinical presentation is typically characterized by persistent hypotension unresponsive to volume replacement and is accompanied by clinical features of end-organ dysfunction
1999	SHOCK trial[9]	*Clinical criteria*: Hypotension (a SBP of <90 mm Hg for at least 30 min or the need for supportive measures to maintain a SBP of ≥90 mm Hg) and end-organ hypoperfusion (cool extremities or a urine output of <30 mL per hour, and a heart rate of ≥60 beats per minute). *Hemodynamic criteria*: Cardiac index of no more than 2.2 L per minute per square meter of body-surface area and a pulmonary-capillary wedge pressure of at least 15 mm Hg

However, shock exists on a spectrum. One classification system is the Society for Cardiovascular Angiography (SCAI) stages of CS.[10] This staging system classifies patients from A to E, with stage A representing "at-risk," stage B "preshock," stage C "classic shock," stage D "deteriorating," and stage E "refractory shock or extremis."

BACKGROUND

Etiology and Pathogenesis

CS has numerous etiologies with the ultimate pathway resulting in inadequate cardiac output, which results in hemodynamic derangements and subsequent hypoperfusion.[2] To compensate for early hypotension and reduced cardiac output, systemic vasoconstriction and fluid retention occur secondary to increased catecholamine state and activation of the renin-angiotensin aldosterone system to maintain perfusion pressure. These are ultimately maladaptive as increased volume and afterload lead to worsening of cardiac output. Worsening cardiac output leads to hypoxic tissue damage, which begins to trigger hemo-metabolic derangements such as increasing lactate and end-organ damage such as renal and liver failure.[8,11] These are associated with the release of proinflammatory cytokines that can induce a vasodilatory component to CS. Ultimately, this cyclical maladaptive response results in refractory hypotension and hypoperfusion and death. CS is a dynamic state and often progresses during one's hospitalization.[12] Thus, early recognition and serial assessments are key in these patients.[12]

One of the most common causes of CS is acute myocardial infarction (AMI). AMI typically results in a left ventricular predominant shock, though as much as 38% of patients with AMI demonstrate RV dysfunction, and over 50% of patients have biventricular dysfunction.[13] Patients with AMI-CS typically present in a more advanced shock state and have a higher mortality as compared with HF-CS.[14] Recently, the incidence of HF-CS due to new onset or de novo heart failure (HF) and acute on chronic decompensated HF has surpassed AMI-CS.[1] Despite having lower cardiac index, more frequent biventricular failure, and higher pulmonary capillary wedge pressures, patients with HF-CS have lower morbidity and mortality.[15] This may be because patients with chronic heart failure become compensated and accustomed to a low flow state and thus suffer less hemo-metabolic consequences when cardiac output drops further.

Mixed cardiogenic-vasodilatory shock cannot be uniformly defined rather hypothesized as hypotension with end-organ hypoperfusion occurring in the context of both acute cardiac insufficiency and systemic vasodilation, resulting in 3 distinct phenotypes: cardiogenic-vasodilatory, vasodilatory-cardiogenic, and primary mixed.[16] Cardiogenic-vasodilatory shock occurs when CS is complicated by inappropriate vasodilation, impairing compensatory mechanisms and contributing to worsening shock. Vasodilatory-CS occurs when vasodilatory shock is complicated by myocardial dysfunction, resulting in low cardiac output. Primary mixed shock occurs when a systemic insult triggers both myocardial dysfunction and vasoplegia. It can be seen in patients after cardiotomy, after cardiac arrest syndrome, whereby the reperfusion injury insult triggers low cardiac output and vasoplegia followed by rapid clinical decline. Regardless of the etiology of mixed shock, the hemodynamic profile can be similar, and outcomes tend to be poor.[16] **Fig. 1** highlights types of CS; however, there are multiple other etiologies of CS including cardiac tamponade, cor pulmonale, valvular disease, arrhythmias, infiltrative disease, and many others.

Clinical Presentation

Signs and symptoms

The spectrum of CS is wide and thus the signs and symptoms vary in different stages of shock.[17] SCAI A patients, or "at risk" of shock, do not demonstrate any overt physical signs or laboratory abnormalities consistent with shock. The overall trajectory

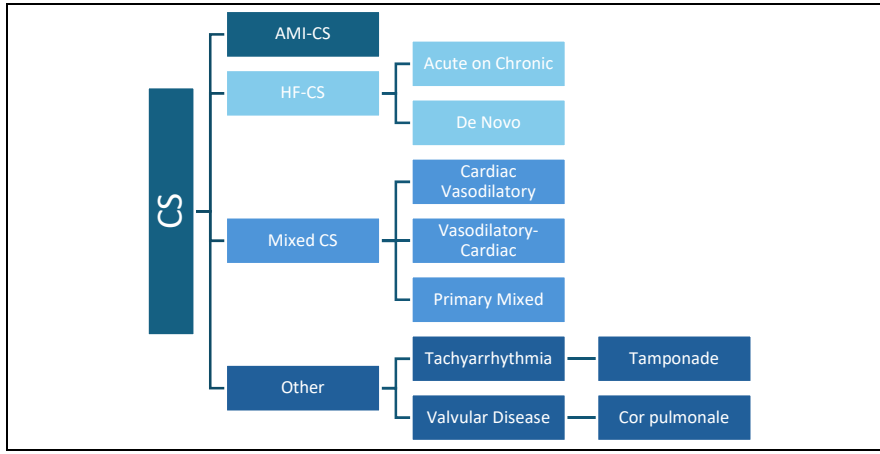


Fig. 1. Types of cardiogenic shock.

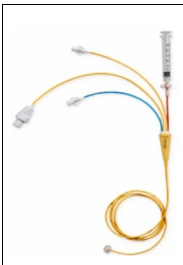
differs between AMI-shock and HF-shock but typically, as patients progress into SCAI stage B, they first begin to exhibit hemodynamic derangements such as tachycardia and hypotension defined by an SBP below 90 mm Hg or need for vasopressor therapy to keep the blood pressure above 90 mm Hg. However, they typically remain well compensated with minimal clinical or laboratory evidence of end-organ hypoperfusion. Their physical examination typically demonstrates warm extremities with some potential rales or elevated jugular venous pressures. Distinguishing patients in preshock versus patients in CS can often be difficult; the sine qua non of shock physiology is hypoperfusion. SCAI stage B, also commonly referred to as "preshock," is hemodynamic instability such as isolated hypotension or tachycardia without hypoperfusion.[18] Preshock can progress to shock and so, identifying patients in this stage is key. Patients with SCAI stages C and D present with continued hemodynamic derangements and additionally progressive laboratory and clinical evidence of hypoperfusion.[19] Clinically, the hypoperfusion may present as altered mental status, malaise, nausea, tachypnea, orthopnea, or cool extremities and oliguria. Hypoperfusion is also reflected in laboratory values such as elevated creatinine, lactate, liver function tests, anemia, thrombocytopenia, and acidosis all of which are associated with a higher risk of decompensation and mortality.[20,21] SCAI stage E reflects refractory hypotension despite support including active cardiopulmonary resuscitation and thus can be characterized by shock liver, need for renal replacement therapy, and ultimately cardiac arrest.

Imaging

Dynamic bedside assessment and the use of echocardiography in patients with CS represent an additive and indispensable tool to assess organ function and to identify the underlying mechanism of CS. The FoCUS examination (focused cardiac ultrasound) demonstrates the presence of pericardial effusion/cardiac tamponade, left and right ventricular size (acute vs chronic) and function, valvular function, and intravascular volume status.[22] In an analysis comparing echocardiographic findings for AMI-CS versus HF-CS, AMI-CS had better left ventricular filling pressures (35% vs 28%) and lower biventricular filling pressures as well as higher stroke volume when compared with HF-CS.[23] Utilization of early Doppler transthoracic echocardiogram was studied in patients with acute coronary syndrome with specific measurement of left ventricular outflow tract (LVOT) diameter and velocity-time integral, which was the single best predictor of hospital mortality when compared with left ventricular ejection fraction.[24] Global longitudinal strain has independently predicted short-term and long-term mortality in patients with CS and is a superior mortality measure specifically in AMI-CS.[25] Other measures like low stroke volume index and high E/e' ratio have demonstrated strong association with high hospital mortality.[26]

Roles for invasive monitoring

Proactive use of pulmonary arterial catheters (PACs) may aid in earlier detection of clinical decline when assessed every 4 to 6 hours when treating HF-CS.[27] There are proposed recommendations for use in CS listed in **Fig. 2**.[28] PACs can delineate CS phenotypes and identifying biventricular or single ventricular dysfunction.[28] A study on PAC defined "early use" as within 2 days of admission and found that PAC were associated with an increased use of MCS in those with mixed CS and resulted in lower in-hospital mortality.[29] However, if PAC cannot be readily placed, use of a central line and corresponding right atrial pressures, central venous oxygen saturations measurement, and continuous blood pressure monitoring via arterial lines may be a reasonable approach.[30]



1.PACs should be used in patients presenting with CS that is not resolved within 30 to 60 minutes with the use of a recommended dose of one inotrope or vasopressor.

2.PACs should be used in patients who present with CS that appeared to resolve based on blood pressure in whom clinical parameters (eg, urine output, mental status, lactate levels) fail to improve.

3.PACs should be used in all patients undergoing MCS to monitor effectiveness, optimize device settings, assess the need for escalation (including the need for device combinations) and guide timing and rate of weaning [28]

Fig. 2. Recommendations for clinical use of pulmonary arterial catheter in cardiogenic shock.

Detection and risk prediction models

Regarding clinical presentations and their role in triage, several classifications have been studied. The first is the Killip Classification, which is used to stratify the severity of heart failure in patients with AMI. There are 4 classes, with class 4 being the most severe with presence of CS, hypotension less than 90 mm Hg, and evidence of hypoperfusion such as altered mental status, cold extremities, and oliguria.[31] Alternatively, the Interagency Registry for Mechanically Assisted Circulatory Support profiles aim to assess severity of heart failure for patients who potentially need MCS. There are 7 profiles, profile 1 is the most severe with criticdal CS and life-threatening symptoms that require intervention compared with profile 7 characterized as advanced New York Heart Association (NYHA) Class III symptoms but without instability.[32]

According to the IABP-SHOCK II randomized trial substudy, serum creatinine levels were shown as a significant independent predictor of 1 year mortality in a multivariable analysis compared to glomerular filtration rate.[33] Lactate clearance was also studied as a surrogate mortality marker in the IABP-SHOCK II substudy trial and determined to be an important clinical predictor, further validated by post hoc analysis of the Capital DOREMI (DObutamine CompaREd with MIlrinone) trial.[34]

There are scoring methods for patients to aid with mortality prediction. Patients with AMI-CS in the SHOCK trial were used to formulate the IABP SHOCK II risk score to predict in-hospital mortality accounting for clinical and hemodynamic variables, found to also be useful after percutaneous coronary intervention.[35] The CardSHOCK risk score was developed for more broad application in CS with MCS accounting for more short-term mortality and accounted for patients' lactate.[36]

There are several risk scores that have been proposed to aid clinicians in evaluating likelihood of survival specific to extracorporeal membrane oxygenation (ECMO), such as the Survival After Veno-Arterial ECMO score assesses prognosis after ECMO initiation. The PREDICT VA-ECMO can be utilized as a dynamic assessment to predict short-term outcomes and is used to adjust treatment strategies.[37,38]

Treatment

Overview

Given that the mechanism of CS involves hypoperfusion of end organs secondary to decreased cardiac output, treatments are targeted at either augmenting perfusion or increasing cardiac output.

Pharmacologic therapy

Vasopressors. The goal of pharmacologic management is to raise the mean arterial pressure and augment cardiac output. Vasopressors induce arterial vasoconstriction

by acting on different combinations of alpha, beta, and dopaminergic receptors. Phenylephrine acts on alpha-1 adrenergic receptors, causing peripheral vasoconstriction with minimal effects on beta receptors. Outside of CS secondary to dynamic LVOT obstruction as seen in hypertrophic obstructive cardiomyopathy, phenylephrine is very rarely used as its strong peripheral vasoconstriction dramatically increases afterload, which can subsequently worsen cardiac output and raise pulmonary capillary wedge pressure.[39] Vasopressin acts on V1a (smooth muscle) and V2 (renal) receptors to cause peripheral vasoconstriction by increasing smooth muscle cytosolic calcium. Vasopressin is typically used as an adjunct to mechanical support and inotropes in patients who are persistently hypotensive. Vasopressin has limited impact on hemodynamics and wedge pressure.[40]

Catecholamines act by stimulating G protein-coupled beta 1 receptors in the left ventricle leading to increased contractility, faster relaxation (lusitropy), and a higher heart rate through enhanced calcium cycling.[41,42] In blood vessels, α1 receptor activation raises intracellular calcium in arteriolar smooth muscle, causing contraction and higher vascular resistance (and blood pressure).[43] In contrast, beta 2 receptor stimulation triggers vasodilation by activating inhibitory pathways in smooth muscle, reducing vascular resistance.[44] Norepinephrine, epinephrine, and dopamine are potent catecholamines typically used in CS as they theoretically can improve cardiac output while at the same time raise blood pressure.

Although direct comparative data among catecholamines in CS remain limited, the available evidence suggests that norepinephrine may be the preferred first-line vasoactive agent. In a prespecified CS subgroup analysis of the SOAP II trial, patients receiving dopamine experienced a higher risk of mortality and an almost 2 fold increase in arrhythmic events as compared with patients treated with norepinephrine.[45] The Optima CC trial was a small-randomized trial that compared epinephrine and norepinephrine in CS. Similar amounts of vasopressors between the 2 groups were required to achieve similar-to achieve target pressures and increases in cardiac output. However, the epinephrine arm had greater increases in heart rate, lactate levels, and odds of developing refractory shock.[46] **Table 2** describes the major trials of vasopressors in patients with shock, specifically highlighting the populations, sample sizes, and agents studied in these trials.

Inotropes. Inotropes increase cardiac output by increasing myocardial contractility and heart rate. Dobutamine and milrinone are most often used as inotropes in the United States and are more specifically inodilators as they also decrease afterload through peripheral vasodilation. They are most commonly used in patients with low cardiac output but adequate blood pressures or in combination with vasopressors.[50] Dobutamine works primarily by direct stimulation of beta 1 receptors increasing cardiac contractility of the myocytes and exhibiting a positive inotropic effect. Milrinone is a phosphodiesterase 3 (PDE3) inhibitor. PDE3 biologically increases the hydrolysis of cyclic adenosine monophosphate (cAMP), thus, the inhibition of PDE3 leads to increased cytosolic cAMP. In cardiac tissue, increased cAMP causes increased inotropy and increased vasodilation within blood vessels. This manifests clinically as increased cardiac output, decreased afterload, and decreased vascular resistance. Notably, milrinone is excreted renally and should be used with caution in patients with renal impairment. Dobutamine was compared to milrinone in the CAPITAL DOREMI study, which showed that there was no difference in the primary composite outcome.[51] A follow-up study, DOREMI-2, is underway comparing use of milrinone or dobutamine to placebo in CS.[52]

Although not currently available in the United States, calcium sensitizers, such as levosimendan, act as inodilators. They enhance cardiac contractility and provide

Table 2
Major trials of vasopressors in patients with cardiogenic shock

Title	Population	Sample Size	Agents Studied	Major Finding
Epinephrine versus Norepinephrine for Cardiogenic Shock After Acute Myocardial Infarction[46]	CS after AMI	57	EPI NE	Similar effects on arterial pressure and cardiac index, but use of epinephrine was associated with a higher incidence of refractory shock
Norepinephrine use in cardiogenic shock patients is associated with increased 30-day mortality[47]	CS	927	NE	Associated with increased short-term mortality but no significant difference in long-term mortality
Dopamine versus norepinephrine as the first-line vasopressor in the treatment of cardiogenic shock[48]	CS	520	Dopamine NE	Those receiving norepinephrine first had a smaller percentage of patients requiring an additional vasopressor
Comparison of Dopamine and Norepinephrine in the Treatment of Shock[45]	Shock patients	1679	Dopamine NE	No significant difference in rate of death but those treated with dopamine first line had a higher risk of adverse effects
A comparison of epinephrine and norepinephrine in critically ill patients[49]	Patients requiring vasopressors	280	EPI NE	No difference in achievement of MAP goal.

Abbreviations: AMI, acute myocardial infarction; CS, cardiogenic shock; EPI, epinephrine; MAP, mean arterial pressure; NE, norepinephrine.

peripheral vasodilation by vascular smooth muscle potassium channel binding and cardiac myofilament calcium sensitization by calcium-dependent troponin C binding. Levosimendan has a half-life over 80 hours making its effects long-lasting even after discontinuation of infusion.[53,54]

Revascularization

Early revascularization is one of the few therapies that has randomized evidence shown to improve mortality in patients with AMI-CS. The SHOCK trial was a multicenter RCT that randomized 302 patients with AMI who developed CS secondary to predominantly left ventricular failure within 36 hours of MI onset to either early revascularization (within 6 hours of randomization) or initial medical management with delayed revascularization (minimum 54 hours after randomization). The early revascularization arm demonstrated a 13.2% absolute risk reduction in mortality at 6 months and a 67% relative risk reduction compared to initial medical management.[55]

Patients with AMI-CS present with multivessel coronary artery disease in over 70% of cases.[56] The CULPRIT-SHOCK (Culprit Lesion Only PCI versus Multivessel PCI in Cardiogenic Shock) trial randomized patients with multivessel disease and AMI-CS to either culprit lesion-only PCI or immediate multivessel PCI. The composite endpoint of death or renal-replacement therapy at 30 days was found to be lower in the group that underwent culprit lesion-only PCI (45.9%) as compared with immediate multivessel PCI (55.4%).[57]

Mechanical Circulatory Support

Indications

The past few decades have seen substantial rise in the utilization of MCS devices and several technological innovations. Currently available MCS devices can be implanted percutaneously or surgically. They can also be configured to support the left, right, or both ventricles, offering varying levels of circulatory support.

Intra-aortic balloon pump

IABP is the most commonly used MCS device. The device is inserted in the femoral artery or axillary artery with the distal tip positioned 1 to 2 cm from the aortic knob with the proximal portion above the renal arteries. The physician can set the IABP to trigger every beat (1:1), every other beat (1:2), or every third beat (1:3) depending on the level of support desired.[58] By inflating during diastole with the aortic valve closed, the device displaces blood from the thoracic aorta into the peripheral circulation. This increases diastolic pressure and subsequently increases coronary perfusion pressure. When the device rapidly deflates just before the onset of systole, it decreases afterload and increases stroke volume. In practice, this translates to an increase in cardiac output of approximately 0.5L to 1 L/min.[59]

The IABP has several attractive features including being technically easy to insert, relatively small vascular access size allowing placement in most anatomies while minimizing risk of distal limb ischemia, and a strong theoretic foundation. Its contraindications are moderate-to-severe aortic regurgitation, active bleeding, aortic dissection, and severe peripheral artery disease.[58]

Despite these advantages, the IABP SHOCK II trial findings suggested that the routine use of IABP in AMI-CS did not result in any mortality benefit. Despite the lack of randomized evidence for IABPs in AMI-CS, they are still frequently used in clinical practice. Specifically, IABP may have a role in the management of severe mitral regurgitation, heart failure in patients awaiting cardiac transplantation, and as a bridge to coronary artery bypass surgery.[43,60]

Percutaneous inserted ventricular assist device

Percutaneously ventricular assist devices (pVADs) are nonpulsatile axial flow pumps intended to provide ventricular support. These devices can provide significantly more support than an IABP with some devices generating as much as 5.0 L/min of flow.[61] Their use has increased significantly over the last decade.[62] There are a variety of devices commercially available and can be classified based on the type of support they can provide: left ventricular, RV, or biventricular support.

Left ventricular percutaneously ventricular assist device

Left ventricle pVADs are primarily used in LV predominant shock. The pVAD decreases left ventricular load and myocardial oxygen consumption, thus increasing cardiac output and decreasing left ventricular end-diastolic pressure.

The most widely used left ventricular pVAD device is the Impella (Abiomed, Inc., Danvers, MA, USA). The Impella is a nonpulsatile axial-flow pump that is advanced retrograde across the aortic valve and positioned in the left ventricle. The Impella is currently available in 2 models: Impella CP and Impella 5.5.[63] While the Impella 5.5 can generate flows as high as 5.5 L/min, it requires a surgical cutdown of the axillary artery to be placed.[64] Alternatively, the Impella CP, which is placed in the common femoral artery or axillary artery, typically provides 3.5 to 4.0 L/min of flow at the highest level of support. The pump performance is also affected by the native cardiovascular physiology; these pVADs are preload dependent and afterload sensitive. The console can also provide information on blood pressure, estimated left ventricular end diastolic pressure. Contraindications to Impella use include the presence of mechanical aortic valves, left ventricular thrombus, ventricular septal defect, and severe peripheral arterial disease.[63]

Initial randomized control studies evaluating the efficacy of Impella support in CS such as ISAR-SHOCK (Impella LP2.5 vs. IABP in Cardiogenic SHOCK) and IMPRESS (IMPella versus IABP Reduces mortality in STEMI patients treated with primary PCI in Severe cardiogenic SHOCK) were limited by small sample sizes and thus were often underpowered to show significant differences in outcomes.[65,66] However, in 2024, the DanGer-SHOCK trial randomized 360 noncomatose patients with ST-segment-elevation myocardial infarction (STEMI) and CS to receive Impella CP plus standard care or standard care alone.[5] The primary end point was death from any cause at 180 days; the Impella CP arm showed a statistically significant reduction in mortality 45.8% (Impella) versus 58.5% (standard-care group), the hazard ratio was 0.74 (95% confidence interval [CI], 0.55–0.99; $P = .04$). However, there was a high rate of complications in the pVAD group including severe bleeding, renal replacement therapy, and infection.

Left atrial to femoral artery bypass

The TandemHeart (CardiacAssist Inc., Pittsburgh, PA, USA) is a percutaneous transeptal extracorporeal centrifugal pump. The device consists of an arterial cannula inserted through the femoral vein and passes through the interatrial septum via transseptal puncture into the left atrium.[67] Like the Impella device, the TandemHeart has been used in both shock and high-risk PCI.[68]The TandemHeart has the benefit of indirectly offloading the LV and can be placed in a patient in the presence of a left ventricular thrombus or mechanical aortic valves. However, it necessitates operator experience with transseptal techniques and is not placed as rapidly in case of an emergency. While there are no randomized trials in CS evaluating the TandemHeart, some registry data have been encouraging with one suggesting a 74% 30 day survival and a 66% 180 day survival in a real-world experience cohort.[69]

Right ventricular support

In patients with isolated RV failure seen in conditions such as RV infarcts, patients after durable left ventricular assist device placement, right-sided valvular disease, congenital disorders, or long-standing pulmonary hypertension, isolated RV support may be necessary. Two commonly used percutaneous RV bypass systems typically inserted through the internal jugular vein or common femoral vein are the Protek Duo (TandemLife, Pittsburg, PA, USA) and the Impella RP.[70] These devices are approved for support for as long as 14 days for the Impella RP and 30 days for the Protek Duo. As they can frequently be inserted through the internal jugular vein, this allows for patients to be ambulatory. The devices are prone to clotting as they are placed in the venous system and are much larger access points when compared to the left ventricular assist devices increasing the risk of vascular injury and bleeding albeit in a vein. In a systematic review of RV assist devices, successful device weaning ranged between 23% and 100% and 30 day survival posttemporary right ventricular assist device (RVAD) implantation ranged from 46% to 100%.[71] These large ranges reflect the paucity of data and heterogeneity of the patients studied with these devices.

Extracorporeal membrane oxygenation

ECMO has increasingly been utilized as a temporary bridge treatment in CS or refractory cardiac arrest.[72] Veno arterial (VA)-ECMO is designed to increase global systemic perfusion and mean arterial pressure; however, it also leads to increased LV afterload, subsequent increase in left ventricular end diastolic pressure, pulmonary capillary wedge pressure, and decreased stroke volume.[19] VA-ECMO circuits consist of a venous (inflow, drainage) cannula, a pump, an oxygenator, and an arterial (outflow, return) cannula.[73] VA-ECMO can be established via peripheral or central access. Peripheral VA-ECMO can be initiated percutaneously or by surgical cut-down via femoral artery and femoral or internal jugular vein access. Another configuration uses the standard venous access (either via the femoral or internal jugular vein) with arterial return to a graft placed on the subclavian artery. This latter strategy has been introduced to ensure perfusion of the cerebral circulation with oxygenated blood and to allow for the possibility for patients to ambulate while on ECMO.[73]

The Advanced Reperfusion Strategies trial studied patients with out-of-hospital cardiac arrest and refractory ventricular fibrillation. It showed a significant increase in survival for patients randomized to extracorporeal cardiopulmonary resuscitation (ECPR) and was stopped early after unanimous recommendation from the data safety monitoring board for benefit. The INCEPTION trial, a noninferiority study on ECPR but did not reveal an improvement in clinical outcomes.[74] The extracorporeal life support (ECLS)-Shock trial showed that for AMI-CS patients with planned early revascularization, 30 day mortality was not lower with ECLS therapy compared to medical therapy alone.[75]

The ECMO-CS trial aimed to compare immediate implementation of VA-ECMO versus an initially conservative therapy (allowing downstream use of VA-ECMO) in patients with rapidly deteriorating or severe CS.[76] The study found the immediate implementation of VA-ECMO in patients with rapidly deteriorating or severe CS did not improve clinical outcomes compared with an early conservative strategy that permitted downstream use in case of worsening hemodynamic status and complications.[76]

Special Populations in Mechanical Circulatory Support

Pregnancy

The leading cause of heart failure and CS in the peripartum period is PPCM, an idiopathic and often dilated cardiomyopathy that occurs late in pregnancy or in the

postpartum period.[77] Although there are not robust clinical trials, MCS use in pregnancy and the postpartum period have been successfully utilized as a bridge to recovery, and outcomes are most favorable when implemented early after initial medical therapies have failed to maintain adequate hemodynamics. When CS is diagnosed in the peripartum period, 33% of patients receive MCS in the form of an IABP, left ventricular assist device (LVAD), or ECMO, with LVAD being the most common. Women with PPCM who received MCS, 48% underwent transplant at 36 months.[77]

Older adult

Contemporary ACC and AHA documents designate 75 years or greater as geriatric. Age is an important risk factor for adverse events in CS. Additionally, frailty—an aging-related biological construct reflecting a vulnerability to an acute stressor—is likely an important effect modifier of outcomes. While age alone should not be used to disqualify patients from potentially lifesaving therapies, it should be incorporated into informed decision-making with patients and family members. SHOCK trial registry data suggest that early revascularization, even among older adults, resulted in reduced mortality. A subanalysis from the DanGer Shock trial, on the other hand, suggests that the benefit of a percutaneous VAD attenuates with age and beyond 77 years may confer harm.[78]

Complications

Although MCS devices are important tools for the management of CS, they are associated with a high risk of complications. These complications involve various systems: hematologic, vascular, neurologic, infectious, and mechanical, see **Fig. 3**.[79,80] The most common include major bleeding defined as the need for blood transfusion or bleeding, stroke often the highest risk in ECMO patients, thromboembolism, and acute limb ischemia. The development of these complications results in increased length of stay and is often associated with higher mortality.

Shock teams

CS teams leverage the expertise of a multidisciplinary panel of clinicians to provide optimal care to patients with CS. The pertinent specialties are often interventional cardiology, critical care medicine/cardiology, advanced heart failure, anesthesiology, cardiothoracic surgery, perfusion services, nursing, pharmacy, rehabilitation, and palliative care.

The International Society for Heart and Lung Transplantation defined 4 tiers of Shock Teams with tier 1 being the highest level of care, offering a full shock team including interventional cardiology, intensivists, heart failure, and cardiothoracic surgery, also included is LVAD and Heart transplant with full support capabilities to initiate, manage,

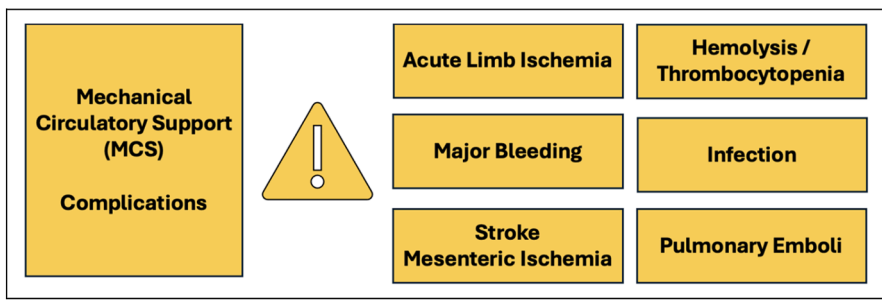


Fig. 3. MCS complications.

escalate, and recover using MCS. Multidisciplinary shock teams have been implemented to facilitate the rapid identification and phenotyping of CS, aid in treatment plans development, and assist in the decision-making surrounding MCS.[80] The multi-institutional Critical Care Cardiology Trials Network reported an improved intensive care unit survival for centers with and without shock teams (mortality of 23% vs 29%, respectively, adjusted odds ratio [OR]: 0.72; 95% confidence interval [CI]: 0.55–0.94; P = .016) in North American centers.[38] Palliative care involvement is a notable requirement of the Centers for Medicare and Medicaid service and the Joint Commission for durable left ventricular assist devices.[68]

SUMMARY

CS is a life-threatening and dynamic disease state primarily caused by acute myocardial infarction or decompensated heart failure. It is characterized by hypotension, reduced organ perfusion, and diminished cardiac output resulting in patients progressing to severe shock stages during their hospitalization. MCS therapies provide hemodynamic support but at the cost of numerous complications. The cornerstone of AMI-CS is early revascularization, with trial data to support noncomatose STEMI shock MCS implementation may result in reduced mortality endorsed by the most recent acute coronary syndrome guidelines. Despite significant advancements in the field, high mortality rates still exist.

CLINICS CARE POINTS

- Cardiogenic shock is a multisystem disease state that needs great attention to central hemodynamics and organ perfusion.
- Restoration of normal hemodynamics and support of organ function constitute the cornerstone of cardiogenic shock management.

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