The Role of Mechanical Circulatory Support Devices in Heart Failure Cardiogenic Shock

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KEYWORDS

• Heart failure • LVAD • Heart transplant • Cardiogenic shock • Survival • Treatment
• Temporary mechanical circulatory support

KEY POINTS

- Mortality in heart failure cardiogenic shock (HF-CS) remains unacceptably high with limited randomized trial data supporting any particular temporary mechanical circulatory support device.
- The pathophysiology of HF-CS differs from acute myocardial infarction-CS in the primary problem being volume-driven rather than mechanical malfunction.
- Shock team-based care, including Palliative care early in disease development, has the most robust evidence in improving patient outcomes.

INTRODUCTION

Cardiogenic shock (CS) is a multisystem disorder resulting in both clinical and biochemical evidence of sustained hypoperfusion with impact on macrocirculatory and microcirculatory end-organ function. CS is often characterized as primary (ventricular failure) or secondary (valvular, post-cardiotomy, etc.). Primary CS is further differentiated as being due to an acute myocardial infarction (AMI-CS) or non-infarction heart failure (HF) cardiogenic shock (HF-CS). The focus of this article will be to provide an overview and update specifically on HF-CS (Fig. 1). We will review the epidemiologic trends of HF-CS, dive into the pathophysiologic profiles of varying phenotypes, and update the reader on classification and staging nomenclature, emphasizing key differences in many patients present in HF-CS versus AMI-CS. Finally, we will evaluate the literature for best practices in employing temporary mechanical circulatory support (tMCS) with a review of patient selection and device characteristics.

EPIDEMIOLOGY

Rates of CS tripled between 2004 and 2018, increasing from 122/100,000 hospitalizations to 408/100,000 hospitalizations in 2018[1] with some potential for data confounding due to improved diagnosis and coding of CS. Since 2018, rates appear to have plateaued.[2] Individuals with pre-existing HF from either chronic ischemic or non-ischemic disease now make up the largest proportion of CS patients.[3] While guideline-directed medical therapies have improved intermediate survival of patients with HF with reduced ejection fraction (HFrEF), this has led to a high prevalence of patients living

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Abbreviations	
AMI	acute myocardial infarction
CCCTN	Critical Care Cardiology Trials Network
CS	cardiogenic shock
CSWG	Cardiogenic Shock Working Group
HF	heart failure
HF-CS	heart failure-cardiogenic shock
HRT	heart replacement therapies
IABP	Intra-aortic balloon pump
INTERMACS	Interagency Registry for Mechanically Assisted Circulatory Support
ISHLT	International Society of Heart and Lung Transplantation
LAVA-ECMO	Left atrial VA-ECMO
LVAD	left ventricular assist device
PAC	pulmonary artery catheter
RA	right atrium
RV	right ventricular
RVF	Right ventricular failure
SCAI	Society for Cardiovascular Angiography and Intervention
SHARC	Shock Academic Research Consortium
tMCS	temporary mechanical circulatory support
UNOS	United Network for Organ Sharing
VA-ECMO	veno-arterial extracorporeal membrane oxygenation

with HF. As the HF disease inevitably progresses in many with HFrEF, the field lacks evidence-based medications for persons with predominant NYHA IV heart and/or ACC/AHA/HFSA stage D HF.[4] To this end, HF-CS is the terminal presentation of stage D HF for many.

There have been comparatively modest improvements in survival outcomes within HF-CS, especially when compared with AMI-CS.[5] Death from HF-CS remains unacceptably high with short-term survival of 25% to 60% depending on etiology and stage at presentation.[6,7] Differences in survival for HF-CS occur for a broad range of reasons, as pathophysiology and severity of HF-CS at presentation vary greatly for de novo HF-CS (eg, myocarditis) compared with acute on chronic HF-CS, especially with regards to Society for Cardiovascular Angiography and Intervention (SCAI) staging, rates of out-of-hospital cardiac arrest, and tMCS usage.[6] Sex is also a major determinant of outcomes in CS, with females having an absolute 5% to 10% increase in mortality vs males for both AMI-CS and HF-CS.[8,9] Mortality differences between AMI- and HF-CS are multifactorial and likely driven by differences in etiology, age of HF onset, delayed disease recognition, greater tendency for biventricular involvement in HF-CS, and disparate application of interventions (including Swan Ganz catheters and tMCS)[8–13] used for CS assessment and management. Finally, other patient-specific factors that correlate with inferior outcomes in CS include the presence of comorbidity, cardiac arrest, and longer time spent in HF-CS.

Another critical contribution to outcome differences in HF-CS is the lack of availability of Cardiac Critical Care and Advanced HF specialist and generalized access to advanced HF surgical therapies (eg, heart transplant and durable left ventricular assist device [LVAD]). While 94% of CS centers have round-the-clock PCI coverage, access to one or more certified Advanced HF Specialists is rare.[14] In the United States, there are only approximately 197 LVAD programs and 151 heart transplant centers, concentrating multidisciplinary shock specialty care (IC, Advanced HF, Critical Care) to a tiny proportion of US health care. Despite the thousands of patients who die from CS annually, only approximately 4000 heart transplants and 4000 durable LVADs are implanted annually in the US.

PATHOPHYSIOLOGY

Pathogenesis of CS stems from the heart's inability to maintain an adequate stroke volume despite ample preload, leading to a reduction in cardiac output and systemic hypoperfusion. The immediate compensatory effect of this is to increase sympathetic tone and neurohormonal activation thus maintaining perfusion pressure. Uncorrected, this compensation ultimately leads to pathologic peripheral vasoconstriction and fluid retention, worsening the preexisting preload-afterload mismatch, further compromising cardiac output and exaggerating intracardiac filling pressures. Persistent activation of renin-angiotensin-aldosterone system along with high adrenergic state and myocardial cell death via Damage-Associated Molecular Patterns, among others including TLR/IL-1

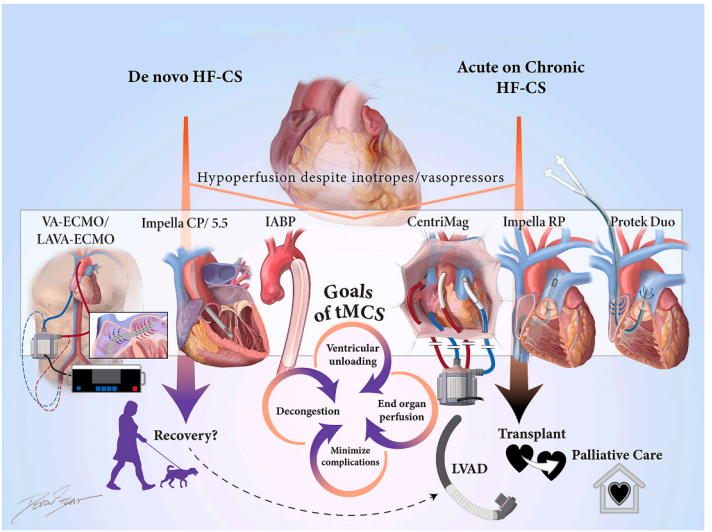

Fig. 1. Central illustration: The role of mechanical circulatory support devices in HF-CS. CS, cardiogenic shock; HF-CS, heart failure-cardiogenic shock. *Image credits* © Devon Medical Art, LLC.

signaling[15] can lead to paradoxical hypotension due to release of pro-inflammatory cytokines by ischemic tissue. Multiple pathways have been implicated in this process, with a unique biomarker profile for HF-CS compared with AMI-CS with notably higher levels of interleukin and Th1 pathways relative to TNF-a and oxidative pathways of AMI-CS.[16–18] High left ventricular end diastolic pressure or pulmonary capillary wedge pressure leads to pulmonary edema, which can further aggravate hypoxemia, leading to elevated pulmonary pressures and reduced right ventricular (RV) function from high afterload. Additionally, high RV and LV filling pressures compromise coronary perfusion pressure gradients and, therefore, microcirculatory perfusion of the heart. This can trigger microvascular coronary ischemia (even in the absence of obstructive coronary lesions), diastolic dysfunction, worsening systolic function, and arrhythmias. The RV, in particular, is sensitive to ischemia due to a relative reduction in capillary density and architectural design to be a high-capacitance yet low-afterload pump.[19,20] Early clinical signs of RV failure include a decrease in urine output or rising biochemical markers of kidney dysfunction and acute congestive hepatopathy, which is typically marked by a disproportionate rise of lactate dehydrogenase relative to transaminase levels (ALT>AST) with bilirubin lagging.[21] Bilirubin elevation is attributed to bile duct ischemia from reduced cardiac output or venous congestion. Generally, RV failure contributes to worsening cardiac output, tissue hypoperfusion, and ultimately multi-organ system failure.

Within HF-CS, the classic teaching is to differentiate clinically by volume (wet vs dry) and perfusion (cold vs warm) states. Refinement of the updated SCAI-CS staging to further identify 3 unique phenotypes from the Cardiogenic Shock Working Group (CSWG) database provides a new validated clinical tool in assessing HF-CS.[22–24] These profiles, non-congested (I) characterized by low output but relatively lower filling pressures and more stable vitals, cardiorenal (II) characterized by high wedge and predominant findings of left ventricular failure, and cardiometabolic (III) with high rates of multiorgan failure and marked lactic acidosis.[23,24] Clinically applying these phenotypes along with etiology of shock and SCAI classification provides nuanced prognostic information and physiological framework for targeting pharmacotherapy and/or tMCS (Fig. 2). Crucially, contemporary trials consistently demonstrate the survival benefit of early complete hemodynamic profiling with the use of invasive pulmonary artery (PA) catheters to expedite recognition of patients who are progressing to biventricular failure or failing to respond adequately to initial management require escalating care.[14,25–27]

CHARACTERIZING AND STAGING HEART FAILURE-CARDIOGENIC SHOCK

The standardization of the CS definition has been key to the ability of clinicians to recognize and treat the condition. The formation of consortia such as the National Cardiogenic Shock Initiative and the CSWG, Shock Academic Research Consortium (SHARC), along with a

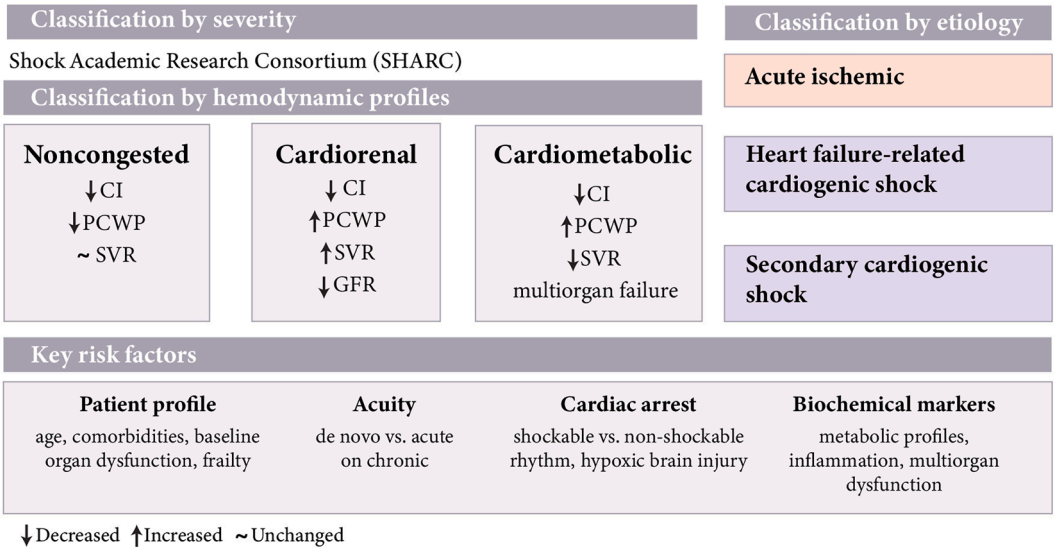

Fig. 2. Cardiogenic shock profiling.

focus on improving shock outcomes by the Critical Care Cardiology Trials Network (CCCTN), has been instrumental in this progress. CSWG along with SCAI helped define AMI-CS with the publication of SCAI staging and subsequent update.[22,28,29] The SCAI classification ranges from low-risk "A" (at risk for CS) through "E" (in extremis), which requires complete cardiovascular support with veno-arterial extracorporeal membrane oxygenation (VA-ECMO). While originally designed for AMI-CS, it has been applied to the HF-CS population with recognized limitations due to differences in these populations.

Robust data to support high level guideline management of HF-CS is presently limited by the degree of patient heterogeneity present in studies, largely due to differences in criteria used to define CS. While major societies[3,22,30–32] share common variables for HF-CS, thresholds for individual criterion and/or count of variables present to meet the HF-CS definition vary. Commonalities in HF-CS definitions include systolic blood pressure less than 90 mm Hg or the need for pharmacologic or mechanical support to maintain SBP $\geq$ 90 for more than 30 minutes, cardiac index $\leq$ 2.2 L/min/m^2, pulmonary capillary wedge pressure $\geq$15 mm Hg, and markers of end organ perfusion including reduced urine output, altered mentation, cool extremities and elevated lactate (>2 mmol/L) (Fig. 3). Most often cited are the definitions put forth by SHARC and CSWG in which patients are further sub-divided by etiology of shock and notably the SHARC definition recognizes the normotensive phenotype of CS.[3,22] This allows for creating a taxonomy of HF-CS for patients experiencing the state due to either de novo disease or acute on chronic HF. An additional staging system commonly used in Advanced Heart Failure centers is the INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support) staging system.[33] INTERMACS classification was originally designed with a similar intent of categorizing illness in patients undergoing durable LVAD, most of whom are in some state of CS in the preoperative phase. This staging system ranks patients through 7 possible phases of HF. INTERMACS stage 1 are the SCAI E equivalent and are patients in extremis despite vasoactive medications and tMCS, stage 2 correspond to SCAI D and are rapidly deteriorating on therapies and require escalation, and stage 3 are the "stabilized" shock patients of SCAI B-C on inotropic therapy (Fig. 4).

Further refinement of both the CSWG/SHARC and SCAI systems have included modifiers for cardiac arrest as these patients experience disproportionate death in all etiologies of CS.

PATIENT SELECTION AND SHOCK TEAMS

Essential to the management of CS patients is early identification of both short and medium/long-term treatment goals. Given the high mortality of CS, early assessment and prognostication aids the discussion between the care team and patient or patient surrogate about goals of care. Multidisciplinary shock teams have been shown to improve short-term survival by integrating expertise across the spectrum of

	Hypotension		Hypotension with Vasopressors	Hypoperfusion Clinical and Biochemical Signs
AHA/ACC/ HFSA 2022	**SBP** <90mmHg for ≥30min **or** **mean BP** <60mmHg for ≥30min	**OR**	**SBP** ≥90mmHg or **mean BP** ≥60mmHg	Poor mentation Cold extremities/livedo reticularis Urine output < 30 ml/h Lactate > 2 mmol/liter
ESC 2021	Not specified		Not specified	Mental confusion Cold extremities Oliguria Dizziness Narrow pulse pressure Elevated lactate Elevated serum creatinine CO_2 Metabolic acidosis
CSWG *Any of the following:*	**SBP** <90mmHg for >30min	**OR**	Need vasopressors to maintain BP	CI < 2.2 liter/min/m² due to cardiac dysfunction Use of tMCS
CCCTN *All of the following:*	**SBP** <90mmHg for >30min	**OR**	Need vasopressors to maintain BP	Altered mental state Oliguria Acute kidney/ hepatic injury **OR** Lactate > 2 mmol/liter **OR** If available, CI < 1.8 or < 2.2 liter/min/m² *(on inotrope) with elevated filling pressures*
Mayo Clinic (SCAI C) *Any of the following:*	Not specified		Not specified	Urine output < 720 ml *during 1st 24 hours* Lactate > 2 mmol/liter Creatinine increase ≥ 0.3 mg/dL *during 1st 24 hours*

Fig. 3. Defining cardiogenic shock. AHA/ACC/HFSA, American Heart Association/American College of Cardiology/Heart Failure Society of America; CCCTN, Critical Care Cardiology Trials Network; CI, cardiac index; CS, cardiogenic shock; CSWG, Cardiogenic Shock Working Group, ESC, European Society of Cardiology; MCS, mechanical circulatory support; SBP, systolic blood pressure.

multiorgan failure often seen with CS.[34–38] Data are confounded by most studies examining the impact of shock teams at quaternary centers with established advanced HF programs that offer heart replacement therapies (HRT) such as LVAD and orthotopic heart transplant (OHT).

In addition to SCAI and INTERMACS classification, use of the vasoactive inotrope score[39]

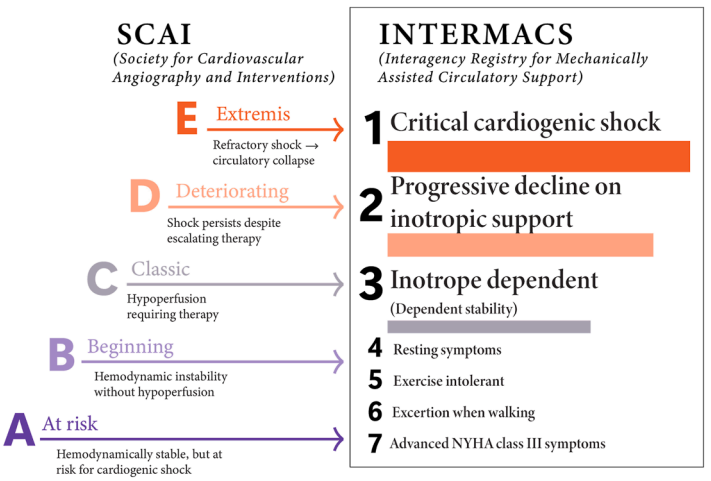

Fig. 4. SCAI and INTERMACS staging in cardiogenic shock. CS, cardiogenic shock; SCAI, Society for Cardiovascular Angiography and Intervention.

can provide additional prognostic information. Early patient selection guided by pulmonary artery catheter (PAC) use to guide care escalation from medical therapy to tMCS can also save progression to other end organ injury, which can limit HF-CS patients from HRT. Exploratory analysis of a study looking at PAC use and mortality in the CICU[40] found that management with a PAC among those with CS was associated with a higher rate of durable LVAD implantation. Single-center registry of 1043 CS patient (AMI or HF) outcomes were studied. Patients classified as SCAI D and E who received PACs were more likely to receive Impella, durable LVAD, and OHT.[41] Importantly, no risk scores or hemodynamic criterion have sufficient discrimination and calibration to be used for routine guidance on whether to apply or not apply support in HF-CS. Care decisions require multifaceted assessment of patients other comorbidities, baseline degree of CS and SCAI trajectory, severity of end-organ involvement, oxygenation status, and the presence or absence of biventricular failure and/or malignant arrhythmias.

Imperative to the decision to escalate patients to tMCS is having an exit strategy devised with multidisciplinary discussion and clear communication with the patient and family about prognosis and care termination protocols (as applicable). Identifying goals of tMCS upfront as bridge to recovery, durable LVAD/OHT, or decision can help the shock team pursue optimal therapy for each patient and can help patients/surrogates establish realistic expectations on risks. It is critical to be aware that few programs in the US will transplant patients greater than or equal to 72 years of age, and those that do tend to select patients without major comorbidity. Thus, elderly patients or younger patients with notable end-organ dysfunction or other medical comorbidities may not achieve transplant candidacy. Durable LVAD support is applied to an older patient population, but again local LVAD patient selection criteria will apply. Given variability in programmatic tolerance of renal failure, RV dysfunction, and social risk factors, there is a critical need to engage Advanced HF specialists early in the care process, even if done via teleconference with a local program.

Expert consensus is lacking in creating any clear absolute contraindications for tMCS. Often advanced age, pre-existing end-stage organ dysfunction or life limiting condition (metastatic cancer, cirrhosis, disseminated infection, active illicit substance abuse) and severe frailty are cited.[42] However, setting age-specific or organ-specific cut offs are complicated by variation between centers offering HRT and consideration for multi-organ transplant. Finally, there is recent evidence to suggest no increase in early mortality difference in use of durable LVAD in patients with advanced or end stage chronic kidney disease, albeit at a cost of increased index hospital length of stay, cost and rates of subarachnoid hemorrhage as well as reduced long-term survival.[43,44] In those patients who have clear contraindications to long-term therapy and presenting in ACHF-CS, early involvement of Palliative Care is integral.

To overcome the lack of data, multidisciplinary shock teams have emerged as a cornerstone in the contemporary management of CS. These teams integrate advanced HF specialists, interventional cardiologists, cardiothoracic surgeons, critical care physicians, and perfusionists to enable rapid diagnosis, hemodynamic profiling, and early initiation of tMCS.[28,45] Observational studies from large tertiary centers demonstrate that shock team implementation is associated with improved coordination of care, higher rates of timely MCS placement, and reduced in-hospital mortality.[34,35] Shock teams facilitate structured escalation and de-escalation of therapy, emphasize the discussion for exit strategies including weaning, recovery, HRT and involvement of Palliative medicine. Importantly, consensus statements from the American Heart Association, International Society of Heart and Lung Transplantation (ISHLT), and European Society of Cardiology highlight that shock team models may bridge the current gap between clinical trial evidence and real-world practice, emphasizing protocolized care, hemodynamic monitoring, and early multidisciplinary involvement as best practices in HF-CS.[14,45,46]

CLEAR CONVERSATIONS

CS is one of the highest acuity diseases in the medical field. Thus, one of the most important aspects of patient care is communicating about prognosis, expectations, complications, and potential for care termination from the beginning. Data show that clear conversations about mortality and complications from the CS syndrome and tMCS can improve patient/family trust, avoid litigation, and may improve the process of withdrawal if care futility is reached. Engagement of Palliative care is critical to ensure open discussions are had, and prognosis expectations are communicated. For complicated situations where care is futile, and family members will not assent to care withdrawal, ethics consultations may be

necessary. To date, there are no clear, standardized legal protections for providers who withdraw MCS in the setting of futile care without family assent.[47]

MECHANICAL UNLOADING

As stated above, HF-CS represents a distinct phenotype from AMI-CS. However, in both *acute* entities, ventricular remodeling takes place with the initiation of myocardial injury, leading to metabolic derangements followed by myocyte death. Myocyte hypertrophy and fibrosis ensue with the viable myocardium, which eventually results in a reduction in contractile force, neurohormonal activation, chronic ventricular dilation, and often biventricular dysfunction.[48] HF-CS is marked by high cardiac filling pressures, low blood pressure, and abnormal markers of end-organ perfusion, also recognized as predictors of in-hospital mortality.

In HF-CS, mechanical unloading aims to reduce the workload of the left ventricle, offer concomitant reduction in heart rate and contractility, ultimately decreasing myocardial oxygen consumption. LV unloading has also been shown to reduce neurohormonal activation and increase beta adrenergic receptors responsiveness, promoting reverse remodeling.[49,50] In fact, the rationale for mechanical unloading extends beyond temporary hemodynamic stabilization: it seeks to reduce filling pressures, lower wall stress, minimize ventricular remodeling, and create conditions for myocardial recovery or durable therapy. Anatomic changes are also accompanied by molecular and biochemical changes.[51–54] It is important to keep in mind that although recovery of myocardial function may be achieved, metabolic and molecular derangements may persist.[55]

DEVICES IN HEART FAILURE-CARDIOGENIC SHOCK

Most patients in CS are initially supported with inotropes or vasopressors which may not be adequate to improve tissue hypoperfusion and metabolic derangements. A class IC indication, these medications increase cardiac output and normalize blood pressure, but at the known expense of increased myocardial oxygen consumption and arrhythmias.[18,56] Vasopressors are also associated with increased LV afterload, which can ultimately reduce cardiac output and perpetuate worsening of CS.

Recommendations on the use of tMCS devices in HF-CS are extrapolated data from observational studies and randomized controlled trials in AMI-CS, even though there are differences in pathophysiology and management.[57,58] Moreover, outcome data in HF-CS are derived from predominantly registry cohorts of patients receiving OHT or durable LVAD.[59] Using data from the CSWG registry, Hernandez and colleagues demonstrated that there is significant heterogeneity with the use of tMCS in HF-CS, and the use of multiple devices and higher-flow devices is associated with higher mortality. Often, tMCS provides support as a bridge to HRT.[60] With the current United Network for Organ Sharing (UNOS) policy, a significant number of patients are listed for transplant supported by variety of tMCS devices and configurations as opposed to inotropes and LVADs who are designated bridge to transplant.[61–63]

The ISHLT along with HFSA provide a class I level B recommendation for initiating MCS as early as possible in patients who fail to stabilize on medical therapy with that recommendation weakening to class II level C for patients with multi-organ system failure in attempting to stabilize and optimize patient status prior to determining candidacy for advanced therapies.[64] This consensus document provides the same level of recommendation for VA-ECMO consideration of patients undergoing active cardiopulmonary resuscitation.

Ultimately, tMCS devices provide hemodynamic stability and maintain end-organ perfusion. tMCS comprises numerous devices that can be utilized to provide univentricular and biventricular support (Fig. 5). Each device has its unique unloading profile, hemodynamic effects, and risk, along with clinical considerations. Device selection needs to account for underlying pathophysiology, magnitude of hemodynamic support needed to reverse tissue hypoperfusion, exit strategy, operator and institutional experience while minimizing potential device and vascular access complications. Furthermore, it is imperative to perform a comprehensive hemodynamic assessment in the management of CS when tMCS is being considered—also associated with improved survival.[27]

INTRA-AORTIC BALLOON PUMP

Intra-aortic balloon pump (IABP) remains the most commonly used mechanical support device used in HF-CS[59] and as a bridge to durable LVAD and OHT.[65] IABP, a balloon-mounted catheter, is placed in the descending aorta and mechanistically works on the concept of counter-pulsation. IABPs inflate during diastole augmenting

Device	Access	Flow (L/Min)	Location	Mechanism	Unloading	Contraindications
IABP	Femoral/axillary artery 7-8 Fr	0.5 - 1	Decending aorta	Aortic counterpulsation: Balloon inflation-deflation	↓Afterload ↑Coronary Perfusion	Severe AR, aortic dissection, aortic aneurysm, PVD
Impella CP/5.5	Femoral/axillary artery across aortic valve CP (percutaneous): 13-14 Fr 5.5 (surgical): 21 Fr	CP: 3.5 5.5: 5.5	LV to aorta	Axial-flow continuous pump	Direct LV unloading	Severe AS or AR, mechanical AV, LV thrombus
VA-ECMO/ LAVA-ECMO	Percutaneous or central cannulation Femoral/jugular vein + femoral artery Venous: 17-28 Fr Arterial: 14-22 Fr	3-7	RA to aorta	Centrifugal-flow continuous pump	Biventricular support;↑ afterload; need venting strategies	PVD, moderate to severe AR
	Left atrial VA-ECMO configuration: fenestrated cannula via femoral vein → transseptal puncture		Transseptal inflow cannula to drain left and right atria		LAVA: Biventricular support, indirect unloading of LV and biatrial drainage; no need for venting	Severe LA pathology
CentriMag	Central cannulation (surgical)	Up to 10	Direct LV (LV to aorta) RV bypass (RV to PA)	Centrifugal-flow continuous pump	Uni- or biventricular support; can add oxygenator	Contraindication for anticoagulation
Impella RP	Femoral vein→ PA 22 Fr	2 - 4	RA to PA	Axial-flow continuous pump	RV support	Severe TR/PR/ TS/PS, mechanical TV/PV, RA/IVC mural thrombus IVC filter
Protek Duo	IVC → PA 29-31 Fr	4 - 5	RA to PA	Can function as centrifugal-flow continuous pump when connected to TandemHeart or CentriMag	RV support; can add oxygenator	Severe TS/PS, mechanical TV/PV, RA/IVC mural thrombus

Fig. 5. Mechanical circulatory support devices used in HF-CS. AR, aortic regurgitation; AS, aortic stenosis; AV, aortic valve; HF-CS, heart failure-cardiogenic shock; IABP, intra-aortic balloon pump; IVC, inferior vena cava; LAVA-ECMO, left atrial VA-ECMO; LV, left ventricle; PA, pulmonary artery; PR, pulmonic regurgitation; PS, pulmonic stenosis; PVD, peripheral vascular disease; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; TS, tricuspid stenosis; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

coronary perfusion by increasing pressure in the proximal aorta during diastole and deflate during systole causing reduction in left ventricular afterload and myocardial oxygen consumption. These devices augment cardiac output by 0.5 to 1 L/min. The IABP is usually gated to the electrocardiogram thus tachycardia reduces diastolic augmentation.[64] IABP can be placed percutaneously in the femoral, brachial or axillary arteries with 7 to 8 Fr access. Axillary access offers ability for mobility and rehabilitation.

Major complications of IABP include vascular injury, atheroembolic events, balloon and catheter occlusion leading to ischemia, and renal failure due to impaired renal perfusion secondary to IABP malposition.

There has been no survival benefit demonstrated in randomized controlled studies with the routine use of IABP in AMI-CS.[66–68] The Alt-Shock 2 trial, a multicenter randomized trial, evaluated the effect of early IABP use versus standard care on 60 day survival or bridging to HRT.[69] IABP was not associated with improved survival or successful bridge compared with standard care. Fried and colleagues retrospectively studied hemodynamic and clinical response to IABP in chronic HF patients with CS, which was found to be highly variable.[70] Patients with nonischemic cardiomyopathy and those with higher PA pulsatility index experienced a robust response with augmentation of cardiac output, termed "super-responders."

AXIAL FLOW PUMPS (IMPELLA CP, 5.5)

Impella (Abiomed, Danvers, MA) is a continuous transvalvular microaxial flow pump that can provide varying degrees of blood flow based on subtype: CP—3.5 L/min, 5.5—5.5 L/min, RP and RP Flex—4 L/min. Impella CP is placed percutaneously in the femoral arteries with 13 to 14 Fr access, while Impella 5.5 requires a surgical cut down with graft in the axillary artery or directly into the aorta. Impella RP/Flex is discussed under RV support devices.

Impella is positioned across the aortic valve with the inlet and pigtail positioned in the left ventricle and the outlet in the proximal ascending aorta. Rotational kinetic energy from the impeller allows for displacement of blood from the left ventricle to the aorta, allowing for decrease in pressure and volume subsequently unloading the ventricle and reducing myocardial oxygen consumption. There is also increase in mean arterial pressure and systemic tissue perfusion.[60] As opposed to the IABP, the Impella is not dependent on intrinsic cardiac rhythm or LV function. Current literature, which includes randomized controlled trials in AMI-CS, has demonstrated improvement in hemodynamics with no difference in outcomes.[71–74] Several studies have shown Improved clinical outcomes associated with the Impella 5.5 compared with 5.0, specifically with regards to higher survival through explant and/or successful bridge to HRT.[75,76]

VENO-ARTERIAL EXTRACORPOREAL MEMBRANE OXYGENATION

VA-ECMO utilizes an extracorporeal centrifugal pump and a membrane oxygenator, providing full cardiopulmonary support up to 7 L/min, supporting end organ function and simultaneous gas exchange for acute cardiorespiratory failure. It can be implanted percutaneously or surgically via 17 to 28 Fr venous cannula and 14 to 22 Fr arterial cannula. Venous blood is oxygenated and displaced to the arterial circulation, providing retrograde flow to the aorta. This can result in increased left ventricular afterload and filling pressures leading to ventricular distension, elevated PA pressures, and pulmonary edema.[77] Therefore, left ventricular venting/decompression strategies are recommended in patients with severely reduced LV systolic function. This can be in the form of pharmacologic venting with inotropes (increase contractility), passive venting with atrial septostomy (shunting of blood from left to right atrium, RA), devices such as IABP (reduce LV afterload), Impella (move blood from LV to aorta), PA cannula (drain blood from PA).[60] Femoral cannulation can also lead to upper body hypoxemia, commonly referred to as *Harlequin Syndrome or North-South Syndrome*, when pulmonary function is compromised. To mitigate these issues, alternative strategies such as venous drainage through the internal jugular vein with arterial return via the axillary artery can be utilized.

Another option for VA-ECMO is to implant a trans-septal inflow cannula to drain blood from the left and right atria while providing biventricular support. Left atrial VA-ECMO (LAVA-ECMO) utilizes a fenestrated cannula placed via the femoral vein using intracardiac ultrasound to guide transseptal puncture. Blood is displaced from the left and RA and guided toward the aorta via femoral artery. Significant benefit of this configuration allows for indirect unloading of the left ventricle, reduction of preload to the LV subsequently protecting the lung from pulmonary edema and decreasing left atrial pressure using one large bore access without need for additional MCS devices.[78]

VA-ECMO support has been shown to stabilize hemodynamics and preserve end organ function in patients being evaluated for myocardial recovery, LVAD implantation, or heart transplantation.[79] Outcomes of patients on ECMO within INTERMACS and UNOS database were studied.[80] There was no difference in mortality on pump support compared with post-transplant mortality among those bridged from ECMO to LVAD or heart transplant. Due to the critical and acuity of illness, well-powered randomized controlled trials have been difficult to perform and provide positive results. The ECLS-SHOCK trial found no survival benefit for routine early ECMO in AMI-CS compared with medical therapy alone.[81] The EURO SHOCK trial aimed to determine if early use of VA-ECMO could improve outcomes in patients with persistent CS following primary percutaneous coronary intervention[82] and no definitive conclusions could be drawn.

Central VA-ECMO cannulation, beyond the scope of this article, can also be employed, requiring an open sternum in the operating room. This can be used as an upgrade from peripheral cannulation when support for end organ function is not sufficient or to address inherent issues to peripheral VA-ECMO such as small vessels, upper or lower limb complications.[64] Ascending aorta (outflow) and RA (inflow) are the preferred cannulation sites.

VA-ECMO cannulation requires operator and center expertise along with dedicated

perfusionists to manage the circuit. Large bore cannulas can inevitably lead to increased bleeding risk, vascular injury, limb ischemia, clot formation, and strokes.

CentriMag

The CentriMag (Abbott, Abbott Park, IL) is a surgically implanted centrifugal blood pump with a magnetically levitated impeller. It has a maximal speed of 5500 rpm, which allows blood flow of up to 9.9 L/min.[83] CentriMag can provide mechanical support for right (RV to PA), left (LV to aorta) or biventricular support. Moreover, a membrane oxygenator can be added, providing pulmonary support, acting as extracorporeal membrane oxygenation circuit system.

RIGHT VENTRICULAR ASSIST DEVICES

Right ventricular failure (RVF) is associated with increased mortality.[84] It is present in 40% to 50% of patients with left ventricular failure, AMI-CS, acute durable LVAD, acute pulmonary embolism, and pulmonary arterial hypertension.[85] MCS devices are used to manage RVF, although practices are not standardized. The following devices directly unload the right ventricle. VA-ECMO, discussed in the section above, provides indirect RV unloading.

Impella RP (Abiomed, Danvers, MA) is a continuous flow axial flow pump with an inlet in the inferior vena cava and outlet in the PA, delivering up to 4 L/min flow from the RA to the PA. It is percutaneously implanted via 22 Fr venous access. This can be used in combination with left-sided Impella pumps (section above), providing biventricular support. RECOVER RIGHT was a non-randomized prospective trial that showed that Impella RP improved hemodynamics in RV failure after LVAD implantation and cardiotomy or myocardial infarction.[86] RP Flex is similar to Impella RP and utilizes a *flexible* cannula implanted in the internal jugular vein via an 11Fr catheter, allowing for mobilization.

Protek-Duo dual-lumen cannula is a percutaneous device with 2 lumens within one 29Fr or 31Fr cannula placed in the internal jugular vein. When connected to a pump, such as the TandemHeart (LivaNova, London, UK) or CentriMag (Abbott, Abbott Park, IL), it can function as RV mechanical circulatory support. It displaces blood from the RA to the extracorporeal pump and delivers it to the PA, providing flows of 4 to 5 L/min. It can be combined with an oxygenator to provide ECMO support. Increasing evidence supports its role in RV infarction and RV failure after LVAD placement.[45] Surgical RV unloading (eg, CentriMag) is also an option with direct surgical RA and PA cannulation.

DEVICE-RELATED COMPLICATIONS

tMCS support devices offer tremendous support in critically ill patients but can be associated with major complications such as bleeding, infection, thromboembolism, hemolysis, arrhythmias, cannula malposition, vascular compromise leading to limb ischemia, and mechanical myocardial/valvular injury. Care teams must be diligent in serial assessment of the patient, devices, support requirements, and goals of care. Ultimately, hemodynamic profiling with support devices needs to be weighed against individual patient risks and benefits.

SUMMARY

HF-CS continues to be associated with high mortality despite advances in recognition, staging, and the use of tMCS. The therapeutic goal of mechanical circulatory support is not only immediate stabilization and survival but also provision of a platform for potential myocardial recovery, protecting end-organ function, and guiding transition to durable LVAD, transplantation, or palliation when appropriate. While survival outcomes in AMI-CS have improved over the past 2 decades, progress in HF-CS has been modest, reflecting the heterogeneous pathophysiology, delayed recognition, and inconsistent access to advanced HF expertise. Development of standardized classification systems such as the SCAI shock stages and the INTERMACS profiles has provided a common language for risk stratification, yet their application to HF-CS remains imperfect. The integration of shock teams has demonstrated advancement in coordination of care and expediting escalation to advanced therapies, but widespread implementation across centers remains a need.

Future studies must address the HF-CS population directly, as extrapolation from AMI-CS underestimates the complexity and chronicity of this disease. Improving survival in HF-CS will require a multifaceted approach, including standardized protocols, early hemodynamic profiling with PAC, refining patient selection for tMCS with individualized device strategies, shock phenotype, and equitable resource allocation. Efforts must focus on addressing disparities in outcomes, particularly among women and patients without access to specialized HF programs. Continued collaboration through national and

international shock registries and networks is essential to uniform practice recommendations, generating high-quality evidence, and ensuring that patients with HF-CS receive timely, effective, and equitable care.

CLINICS CARE POINTS

- Differentiate HF-CS from AMI-CS.
- Invasive hemodynamic assessment should be performed early.
- Integrate SCAI stage with shock phenotype to better risk stratify patients and individualize treatment decisions.
- Select mechanical circulatory support devices based on physiology and anticipate device-specific hemodynamic effects and possible device-related complications.
- Establish an exit strategy prior to initiating tMCS.
- Assess candidacy for advanced therapies early.
- Activate and engage in multidisciplinary shock teams.

DISCLOSURE

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