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Recommended Citation

Abraham J, Blumer V, Burkhoff D, Pahuja M, Sinha SS, Rosner C, Vorovich E, Grafton G, Bagnola A, Hernandez-Montfort JA, and Kapur NK. Heart Failure-Related Cardiogenic Shock: Pathophysiology, Evaluation and Management Considerations: Review of Heart Failure-Related Cardiogenic Shock. J Card Fail 2021; 27(10):1126-1140.

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Review

Heart Failure-Related Cardiogenic Shock: Pathophysiology, Evaluation and Management Considerations

Review of Heart Failure-Related Cardiogenic Shock

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ABSTRACT

Despite increasing prevalence in critical care units, cardiogenic shock related to HF (HF-CS) is incompletely understood and distinct from acute myocardial infarction related CS. This review highlights the pathophysiology, evaluation, and contemporary management of HF-CS. (*J Cardiac Fail 2021;27:1126−1140*)

Key Words: Cardiogenic shock, Mechanical circulatory support, heart failure, Critical care.

Cardiogenic shock (CS) is an extreme manifestation of acute heart failure (HF); it carries in-hospital mortality rates of 30%–50%.[1−4] Although the pathophysiology and management of CS due to acute myocardial infarction (AMI-CS) has been the focus of intense investigation, CS due to nonischemic causes, including acute or chronic HF (HF-CS) is less well studied despite being more prevalent in the contemporary era. CS is 1 of the leading indications for admission to a cardiac intensive care unit (CICU).[5] Because of demographic trends and more widespread use of primary percutaneous coronary intervention (PCI), HF-CS has surpassed AMI-CS as the leading cause of cases of CS.[6−8] In this state-of-the-art review, we discuss the classification, pathophysiology and management of HF-CS, with emphasis on advanced chronic HF.

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Manuscript received June 6, 2021; revised manuscript received July 27, 2021; revised manuscript accepted August 9, 2021.

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1071-9164/$ - see front matter

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https://doi.org/10.1016/j.cardfail.2021.08.010

Definitions, Profiles and Staging

Based on studies of patients with AMI, CS is defined by a systolic blood pressure (SBP) <90 mm Hg or the need for pharmacological or mechanical support to maintain SBP > 90 mm Hg in combination with evidence of end-organ hypoperfusion.[9,4] Reduced cardiac output (CO) with normal or elevated filling pressures is a requisite hemodynamic condition. Ambulatory patients with HF who have been prescribed neurohormonal antagonists commonly have a SBP < 90 mm Hg, elevated intracardiac filling pressures[10,11] and depressed CO without hypoperfusion or end-organ dysfunction.[12] For these reasons, the clinical trajectory of a patient with AMI-CS often begins with hypotension leading to hypoperfusion and ending with congestion. In contrast, a patient with HF-CS commonly presents with acutely decompensated HF and congestion, leading to hypoperfusion and ending with hypotension (Fig. 1). These opposing clinical trajectories of AMI- and HF-CS raise important

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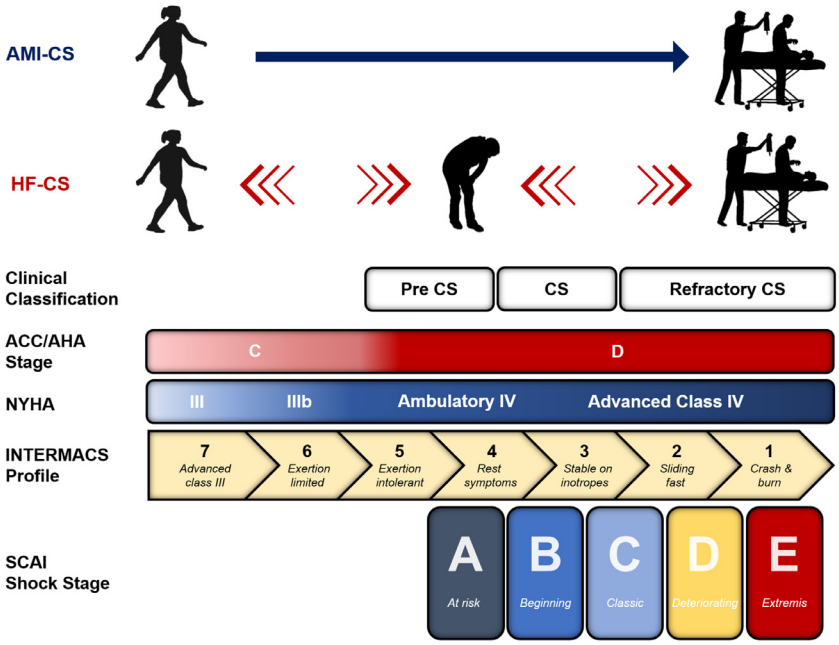

Fig. 1. Clinical trajectory and classification of cardiogenic shock. The clinical trajectory of acute myocardial infarction-cardiogenic shock (AMI-CS) differs significantly from heart failure-cardiogenic shock (HF-CS). AMI is characterized by an abrupt presentation that leads to CS in otherwise stable, ambulatory patients. Patients with chronic HF may have multiple, episodic decompensations that can progress to a pre-CS or CS state and with prompt interventions regress to an ambulatory state. Whereas in AMI-CS, the most common pathway is native heart recovery; in HF-CS, bridging to transplantation or left ventricular assist device therapy is common. In search of a unifying taxonomy to classify CS and guide therapies, the Society for Cardiovascular Angiography and Interventions (SCAI) proposed a 5-stage (A−E) classification system for CS inspired by the American College of Cardiology/American Heart Association (ACC/AHA) staging of HF and the INTERMACS classification.

questions about the appropriateness of our current definition of CS and suggest the need for further investigation to define distinct criteria for AMI-CS and HF-CS.

Hemodynamic profiles of CS further classify CS as having left ventricular (LV)-dominant, right ventricular (RV)-dominant or biventricular shock (Table 1).[13] These definitions are derived largely from clinical trials of patients with AMI and have limited validation in populations with HF.[2,14−16] More recently, 3 distinct CS phenotypes (noncongested, cardiorenal and cardiometabolic shock) were identified by using a supervised machine learning approach and were validated in both AMI-CS and HF-CS populations by 2 independent multicenter cohorts.[17] These phenotypes differed based on demographic, hemodynamic and metabolic profiles and were correlated with inpatient mortality. The cardiometabolic phenotype was associated with the highest mortality rate and was characterized clinically by venous congestion and low CO, right heart dysfunction and liver injury. Although further study is needed, risk stratification and treatment strategies based on the CS phenotype may enable more individualized therapy.

Table 1. Hemodynamic Profiles of Cardiogenic Shock Subtypes

Hemodynamic Variable	Preshock Normotensive Hypoperfusion	Preshock Hypotensive Normoperfusion	LV Dominant Shock	RV Dominant Shock	BiV Shock
Systolic arterial pressure, mmHg	>90	<90	< 90	< 90	< 90
CVP, mmHg	Variable	Variable	< 14	> 14	> 14
PCWP, mmHg	Variable	Variable	> 18	< 18	Variable
CVP/PCWP	Depends on degree of LV and RV involvement	Depends on degree of LV and RV involvement	< 0.86	> 0.86	> 0.86
PAPi (PAS-PAD)/RA	Depends on degree of RV involvement	Depends on degree of RV involvement	> 1.5	< 1.5*	< 1.5
Cardiac index, L/min/m^2	< 2.2	≥ 2.2	< 2.2	< 2.2	< 2.2
SVR, dynes-s/cm^{-5}	> 1600	800–1600	800−1600	800–1600	800–1600

BiV, biventricular; CVP, central venous pressure; LV, left ventricular; PAD, pulmonary artery diastolic pressure; PAPI, pulmonary artery pulsatility index; PAS, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; RA, right atrial pressure; RVR, systemic vascular resistance.
*Right ventricular (RV) dominant shock due primarily to RV dysfunction.

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The United Network of Organ Sharing (UNOS) defines CS using the following hemodynamic criteria for donor heart allocation[18]:

- cardiac index < 1.8 L/min/m^2, or <2.2 L/m/m^2 if the candidate is on inotropic or mechanical support;
- pulmonary capillary wedge pressure (PCWP) > 15 mmHg;
- SBP < 90 mmHg.

Under the UNOS criteria for heart transplantation (HT) implemented in October 2018, patients meeting these criteria are eligible for inotropes and status 3 listing. If the cardiac index is < 2.0 L/min/m^2 on inotropic support, then temporary mechanical circulatory support (tMCS) and more urgent listing status are justified. In patients for whom hemodynamic measurements are not obtained, the need for cardiopulmonary resuscitation, SBP < 70 mm Hg, arterial lactate > 4 mmol/L or liver transaminases > 1000 U/L within 24 hours qualify for tMCS. For these same hemodynamic criteria, various tMCS devices can be employed: intra-aortic balloon pumps (IABPs), nondischargeable endovascular left ventricular assist devices (LVADs) or extracorporeal membrane oxygenation (ECMO) provide distinct levels of hemodynamic support and confer different urgencies (Status 2 vs Status 1). By contrast, the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) profiles were proposed to characterize recipients of durable LVADs at the time of LVAD implant.[19] Within this nosology, patients are classified based on hemodynamic stability, inotrope use and functional capacity rather than on hemodynamic criteria. The sickest "crash-and-burn" patient with critical cardiogenic shock (INTERMACS 1 profile) is defined by subjective clinical criteria: organ hypoperfusion, escalating inotropic support or IABP and the need for definitive intervention within hours. The INTERMACS 2 profile includes patients who are declining despite inotropic support ("sliding on inotropes"). Within the INTERMACS classification, an arrhythmia modifier can be used to denote clinically significant ventricular arrhythmias at any INTERMACS level and a tMCS modifier for profiles 1–3 requiring nonelective hemodynamic support.

In search of a unifying taxonomy to classify CS, the Society for Cardiovascular Angiography and Interventions (SCAI) proposed a 5-stage (A−E) classification system for CS, inspired by the American College of Cardiology/American Heart Association (ACC/AHA) staging of HF and the INTERMACS classification (Fig. 1).[20] Each stage may have an arrhythmia modifier to signify the occurrence of a cardiac arrest. In the SCAI framework, the development of tissue hypoperfusion and end-organ dysfunction herald the transition from preshock (Stage B) to later stages (C–E). The SCAI classification robustly stratifies hospital mortality in unselected single-center CS cohorts that include AMI-CS and HF-CS and in a multicenter registry with invasive hemodynamic variables.[21−23]

Pathophysiology

Pressure-volume loop analysis provides a useful conceptual framework for understanding CS pathophysiology (see Supplementary videos). In acute CS such as AMI-CS or fulminant myocarditis, the end-systolic pressure volume relationship shifts downward and rightward, reflecting a sudden reduction in ventricular contractility, with attendant declines in stroke volume, CO and blood pressure and increases in PCWP and central venous pressure (CVP). In the transition from acute to chronic HF, neurohormonal activation leads to myocardial remodeling and intravascular volume expansion, resulting in higher ventricular volumes and end-diastolic pressure volume relationships.[24]

Chronic HF evolves from an initial hemodynamic disturbance into a multisystem disorder. Arterial vasoconstriction increases vascular resistance to peripheral organs and redistributes blood away from the splanchnic circulation. Venoconstriction increases stressed blood volume (SBV) that contributes significantly to increases in central venous and pulmonary venous pressures. Progressive elevations in CVP lead to visceral venous congestion.[25] In the kidney, reduced renal venous and lymphatic outflow impair glomerular filtration and alter tubular secretion and reabsorption.[25] Similarly, venous congestion contributes to a spectrum of hepatic dysfunction, most commonly cholestatic laboratory abnormalities.[26] If long-standing, both congestive nephropathy and hepatopathy lead to histologic and functional changes. These cardiovascular and end-organ adaptations allow a patient with chronic HF to tolerate hemodynamic conditions[27] that would cause critical illness if imposed acutely and also increase susceptibility to severe acute organ dysfunction in more advanced stages of HF-CS.

Chronic HF progresses to HF-CS when impaired ventricular contractility is severe enough to cause a critical reduction in mean arterial pressure (MAP) and CO (Fig. 2). End-organ hypoperfusion results in acute on chronic hepatic and renal insults, lactic acidemia, decreased coronary perfusion pressure, and further activation of baroceptors and chemoreceptors, all of which set up a vicious circle of worsening cardiac function. If the CS state persists, end-organ dysfunction worsens, lactic acidosis persists or worsens, and a state of systemic inflammatory response syndrome ensues. This inflammatory state can further worsen cardiac function and has a negative impact on prognosis.[28]

Initial Evaluation of Cardiogenic Shock

The initial assessment of patients presenting with CS should involve a focused history, physical examination and directed imaging (see Supplementary Tables 1 and 2). The nature and duration of symptoms may identify precipitating factors for decompensation and the rapidity of clinical deterioration. Acute myocarditis with high-risk presentations or fulminant myocarditis is especially important to recognize because endomyocardial biopsy and immunosuppressive treatment may be indicated.[29,30]

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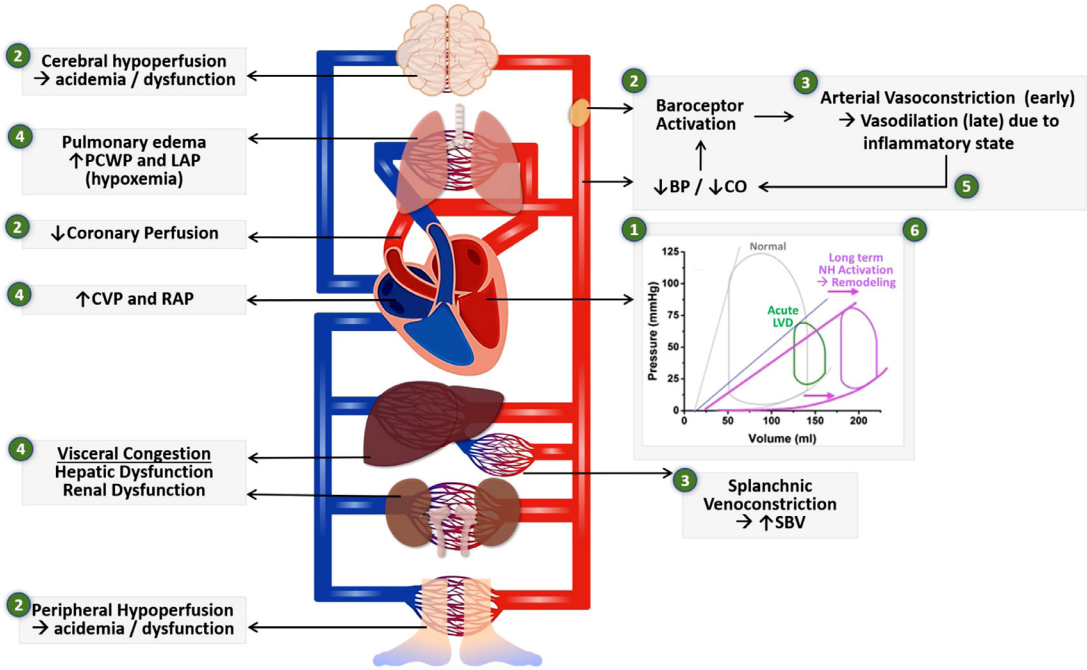

Fig. 2. Pathophysiology of heart failure-cardiogenic shock. Cardiogenic shock (CS) is initiated by a reduction of ventricular contractility (1) that is of sufficient severity to cause a reduction blood pressure and cardiac output. This results in peripheral and cerebral hypoperfusion, which results in acidemia, decreased coronary perfusion pressure (which can further compromise LV function) and activation of the baroceptors and chemoreceptors (2). In the early stages of CS, baroceptor and chemoreceptor activation results in neurohormonal stimulation, which has a multitude of systemic effects, including arterial and venoconstriction (3). Arterial vasoconstriction increases the resistance to blood flow through peripheral organs, whereas venoconstriction increases stressed blood volume (SBV) that contributes significantly to increases in central venous and pulmonary venous pressures (4); these factors further compromise end-organ perfusion and promote tissue congestion (including pulmonary edema), both of which exacerbate end-organ dysfunction. If the CS state persists, end-organ dysfunction worsens, lactic acidosis persists or worsens, and a systemic inflammatory response state (SIRS) ensues (5). SIRS results in a vasodilatory state (including arteries and veins) and can worsen cardiac function that further complicate management and has a negative impact on prognosis. In addition, over time with persistent neurohormonal activation, inflammation and hemodynamic forces, ventricular remodeling ensues (6), which underlies the development and progression of heart failure.

The physical examination is directed to the assessment of filling pressures and perfusion, allowing for bedside classification of hemodynamic profile. The echocardiogram examination should focus on assessment of left and right morphology and function, presence of intracardiac thrombus, regurgitant or stenotic valvular lesions, dynamic outflow tract obstruction, and surrogate estimates of filling pressures. Particular attention should be given to identifying RV dysfunction (RVD) because RVD is a harbinger of illness severity and mechanistically contributes to poor clinical outcomes by limiting total cardiac output while also promoting systemic venous congestion and end-organ failure. RVD is consistently associated with poor outcomes in acute and chronic HF and impacts the selection of tMCS devices and candidacy for durable LVADs.[31]

Laboratory evaluation should include serial arterial lactate measurements. Lactate clearance has emerged as an important prognostic indicator in CS, though further research is needed to delineate the optimal lactate thresholds for risk prediction in acute decompensated heart failure (ADHF).[1] Further advanced testing such as endomyocardial biopsy should be considered in appropriate clinical scenarios.[32] Invasive hemodynamic monitoring using a pulmonary artery catheter (PAC) is a useful tool in the diagnosis, phenotyping and management of patients with CS and is discussed further below

Multidisciplinary shock teams should be involved in the initial evaluation and ongoing management of CS. CS teams are ideally composed of an advanced HF cardiologist, cardiothoracic surgeon, interventional cardiologist, and intensivist, with additional members including a critical care nurse, perfusionist, respiratory therapist, and palliative care specialist. Specialized multidisciplinary teams that develop center-specific treatment algorithms to provide guidance in selecting patients with CS for tMCS have the potential to improve clinical outcomes. In the National Cardiogenic Shock Initiative, a single-arm, prospective, multicenter registry of AMI-CS in the United States, the use of a shared algorithm emphasizing early tMCS was associated with 72% survival to discharge with native heart recovery (NHR).[33] Similarly, in a large, single-center observational study, a standardized team-based approach emphasizing timely identification, early and complete hemodynamic profiling with a PAC and early selective tMCS for both AMI-CS and HF-CS was associated with improved survival at 30 days.[34]

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Clinical Trajectory and Outcomes

The clinical trajectory of CS follows 3 possible pathways: (1) NHR with sufficiency myocardial stabilization to allow for the weaning of vasoactive and/or tMCS support; (2) stabilization as a bridge to heart replacement therapy (HRT) with HT or VAD; or (3) death. Given the dynamic nature and rapid progression of CS, it is often not feasible to discern which pathway is most likely, so a "bridge-to-decision" strategy is employed. Throughout the clinical trajectory of the patient with CS, it is imperative for the shock team to consider and to reassess which pathway is most likely.

Based on underlying differences in pathophysiology and time course, AMI-CS and HF-CS have different hemodynamic profiles and clinical outcomes. In the Cardiogenic Shock Working Group (CSWG) registry, patients in the AMI-CS cohort had higher left ventricular ejection fraction and lower mean pulmonary artery pressures. Despite having similar CO, RAP, PCWP, MAP, and heart rate, in-hospital mortality was significantly higher in patients with AMI-CS (41%) than with HF-CS (23.5%).[23] Whereas NHR is the therapeutic endpoint in most cases of AMI-CS, LVAD or HT are more likely outcomes in HF-CS.[35,36] Of HF-CS patients in the CSWG registry, 39% underwent HRT and 37% experienced NHR. In contrast, among the estimated 70%–80% patients who survive AMI-CS, almost 90% experience NHR. Taken together, these findings suggest that HF-CS is a distinct clinical entity.

Critical Care Management of Shock

CICU Staffing

The care of patients with HF-CS is complex, challenging and resource-intensive. The optimal organizational structure and staffing models remain to be defined, but high-intensity staffing with a dedicated cardiac intensivist[37] or comanagement among cardiologists and intensivists may be associated with improved mortality rates.[38] Consensus documents from both the ACC/AHA and the European Society of Cardiology suggest that management of CS in the CICU requires 24/7 care in an advanced center capable of providing invasive hemodynamic monitoring and comprehensive, multiorgan system care.[2,39]

Respiratory Failure

Respiratory failure is common in patients with CS, and its prevalence has more than doubled over the past 10 years.[40] In a multivariable logistic regression analysis of HF trials, the requirement for mechanical ventilation (MV) was strongly associated with increased 30-day rehospitalization and all-cause mortality.[41]

Positive pressure ventilation (PPV), including MV and noninvasive positive pressure ventilation (NI-PPV), has complex effects on cardiopulmonary physiology. For the purposes of this review, clinicians should recognize that PPV reduces ventricular preload, decreases LV afterload and increases RV afterload.[42] Because of these hemodynamic effects, PPV should be used with caution in predominant RV failure or preload-dependent states. NI-PPV (CPAP, BiPAP and high-flow nasal cannula) improves cardiogenic pulmonary edema hypercapnia and acidosis[43] and reduces the need for intubation.[44] Adequate mentation to protect the airway is a prerequisite for use of NI-PPV.

For patients requiring MV, tidal volume (TV) should be set between 6 and10 mL/kg ideal body weight, with plateau pressure less than 30 cm H_2O to prevent complications associated with barotrauma and lung injury. There is no clear benefit of using low TV in patients with cardiogenic shock and with acute respiratory distress syndrome (ARDS). The PaO_2/fraction of inspired oxygen (FIO_2) (P/F ratio) is used to diagnosis and quantify the severity of acute lung injury.[45] Based on the definition of ARDS, a P/F ratio $\leq$ 300 defines mild ARDS, and $\leq$ 100 defines severe ARDS. The Extracorporeal Life Support Organization (ELSO) recommends ECMO for hypoxic respiratory failure at a threshold P/F ratio of $\leq$ 100 mmHg.[46]

Cardiac Arrest

CS and cardiac arrest (CA) confer significantly elevated mortality when occurring together.[47] Postresuscitation shock occurs in approximately two-thirds of patients after CA. The mortality rate remains > 60%, even for patients who achieve return of spontaneous circulation (ROSC). The most common causes of death for patients who achieve ROSC are CS and neurologic injury,[48] yet there are no randomized controlled trials evaluating the use of tMCS in patients with postarrest HF-CS. Selected patients with CS may benefit from early hemodynamic evaluation and tMCS. Postarrest patients with ST-elevation myocardial infarction (STEMI) should undergo emergent coronary angiography. Targeted hypothermia has been shown to have no benefit compared to targeted normothermia on 6-month survival.[49]

Hemodynamic Monitoring

Despite an absence of benefit of routine PAC use for HF, growing evidence supports the benefit of early invasive hemodynamic assessment in patients with HF-CS.[50] The Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial did not show benefit of PAC use in addition to clinician assessment in patients with severe, symptomatic, recurrent HF; importantly, however, very few of these patients met contemporary criteria for CS.[51] In a prospective, cohort study using case-matching and multivariable analysis, PAC-guided management in patients with HF (and other diseases) in the first 24 hours of ICU stay was of no benefit.[52] In a recent analysis from CSWG, outcomes were compared between groups with no PAC, incomplete profiling and complete profiling.[50] Mortality rates differed significantly between PAC-use groups within the overall cohort and each SCAI stage subcohort. Notably, those patients

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with complete PAC assessment group had the lowest in-hospital mortality rates across all SCAI stages. These data are the first to report an association of PAC use with improved survival in such a large and diverse population of patients with CS.

The use of invasive hemodynamics is often critical to identifying, phenotyping and managing HF-CS, especially in the setting of RVD. In the CSWG cohort, high rates of biventricular congestion (50%) and RV predominant congestion (8%) in AMI-CS and HF-CS were demonstrated.[23] Biventricular congestion was significantly associated with increased mortality as compared to euvolemia or LV congestion. Even after adjusting for SCAI classification, right atrial pressure (RAP) remained significantly associated with mortality, further highlighting the prognostic importance of RVD.[23] Multiple invasively derived measures of RVD have been used across the spectrum of cardiac diseases. The 4 most commonly used metrics include CVP, CVP/PCWP ratio, pulmonary artery pulsatility index (PAPI = PA pulse pressure/RAP), and RV stroke work index.[14] Comparative effectiveness data for these metrics in HF-CS is lacking, although PAPi is more commonly used and has more published algorithms.[53]

In sum, although contemporary randomized controlled trials are lacking, and residual confounding is possible with current evidence, PAC use may lead to earlier and more accurate identification of the CS phenotype so that drug- and device-based therapies may be applied in a tailored fashion.[50] The routine use of early invasive hemodynamics has been advocated as the standard of care in contemporary CS management.[2,54] Education about invasive hemodynamic data measurement and interpretation through dedicated training pathways and credentialing may be another strategy for improving patient management.[54]

Decongestive Strategies

Signs and symptoms of congestion complicate the overwhelming majority of acute decompensated heart failure (ADHF) hospitalizations and, not surprisingly, congestion is an important therapeutic target in HF-CS.[23] Increased SBV, defined as the volume of circulating blood above the amount required to fill a vessel to the point of increasing wall stress and intravascular pressure, is associated with increased rates of in-hospital mortality among patients with HF-CS. [55] Moreover, elevations in filling pressures are stronger predictors of outcomes than cardiac output in advanced HF.[56] Removing intravascular volume removal or reducing SBV reduction may, therefore, improve outcomes.

Currently available interventions to remove congestion in patients with ADHF are limited. Coadministration of loop diuretics with thiazide and thiazide-like diuretics can overcome diuretic resistance via sequential nephron blockade, leading to a synergistic natriuretic effect. Several prospective studies that used a stepped diuretic algorithm, though they excluded patients with HF-CS, provide a framework for escalating diuretic and adjunctive therapies.[57–59] These algorithms employ bolus and continuous infusions of loop diuretics at 2–2.5 times a patient's home regimen to achieve a goal output of urine.

Mechanical volume removal using ultrafiltration has been shown to be inferior to a stepped pharmacological approach in ADHF,[59] but the early use of continuous veno-venous hemofiltration (CVVH) was associated with better in-hospital and long-term survival in patients with postcardiotomy CS and acute kidney injury.[60] Several investigative device-based interventions that target mechanisms of congestion in HF may find application in HF-CS.[61]

Inotropes, Vasopressors and Vasodilators

Intravenous (IV) inotropes and vasopressors remain important therapies in the initial management of HF-CS and have a Class IC indication.[2] Despite being widely used in clinical practice, scant evidence is available to guide their use.[62] In particular, the optimal MAP and cardiac output are not known. Rational prescription of these agents is, therefore, based on pharmacologic principles that are tailored to patient physiology and response to treatment (Table 2). An important caveat is that these agents also increase myocardial oxygen demand (see Supplementary videos) and can provoke malignant arrhythmias and coronary or peripheral ischemia. Achieving hemodynamic targets must, therefore, be weighed against the inherent risks of high-dose or prolonged drug administration.

Norepinephrine may be the preferred first-line agent to treat hypotension in HF-CS. In a randomized trial in patients with AMI-CS, norepinephrine showed improvements in cardiac index and MAP similar to those of epinephrine but with a lower incidence of refractory shock (7% vs 37%; $P = 0.008$).[63] Vasopressin can be added if a target MAP is unachievable with norepinephrine. For inotropy, dobutamine is a reasonable first-line agent. Milrinone is a consideration if the patient has adequate MAP and low CO, especially if the patient has received beta-blockers chronically; milrinone should not be administered as a bolus, and caution is needed to prevent acute kidney injury due to its renal clearance. We recommend withholding oral HF medications during the acute phase of CS and gradually reintroducing these drugs as CS resolves.

Mobility and Nutrition

Because of the presence of monitoring devices, tMCS devices, or hemodynamic or electrical instability, patients with CS are often restricted to bedrest, resulting in limited mobility that can exacerbate underlying deconditioning and may have detrimental effects on various body systems.[64] Early mobilization can prevent or reduce these effects and is associated with improved outcomes in patients after critical illness. Early mobilization has recently been studied in a variety of critical illnesses and has been shown to limit or prevent physical and cognitive dysfunction and to provide various benefits in mechanically ventilated patients.[65] For patients with HF-CS who require tMCS, consideration

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Table 2. Cardiac Intensive Care Unit Care in Heart Failure-Cardiogenic Shock

	Preload	Afterload	Contractility	Heart Rate
Target	– Optimize preload when hypovolemic – Reduce congestion when volume overloaded RA 4–10 mmHgPCWP 10–18mmHg	– Optimize to maintain end organ and tissue perfusion – Reduce excessive resistance to ventricular emptying MAP 60–80 mmHgSBP 80–140 mmHgSVR 900–1600 mmHg	– Achieve adequate end-organ and tissue perfusion with least increase to myocardial oxygen demand End-organ perfusion: Lactate <2, normalize renal and hepatic function	– Optimize diastolic filling time to optimize left ventricular end-diastolic pressure HR 50–110 bpm
Increase	Intravascular volume −Careful fluid bolus or infusion −Blood transfusion Venous return - Passive leg raise - Compressive therapy - Ambulation/Mobility	Vasopressors Norepinephrine Mixed a, b_1 and b_2 agonist (a>b) 0.01–1 μg/kg/min Dopamine Lower dose: mild b_1 agonist Midrange dose: a, b_1 and b_2 agonist Higher dose: increased a and b_1 agonist 0.5–20 μg/kg/min Epinephrine Mixed a, b_1 and b_2 agonist 0.01–0.5 μg/kg/min Phenylephrine a agonist 0.1−10 μg/kg/min Vasopressin V1 receptor in vascular smooth muscle 0.02–0.04 U/min	Inodilators Phosphodiesterase-3 inhibitors Milrinone 0.125–0.5 μg/kg/min Levosimendan 0.05−0.2 μg/kg/min b-agonists Dobutamine a, b_1 and b_2 agonist 2.5–20 μg/kg/min	b-agonists Dopamine 0.5–20 μg/kg/min Dobutamine 2.5−20 μg/kg/min Epinephrine 0.01–0.5 μg/kg/min Isoproterenol 2.0−20 μg/min (Withdrawing b-blockers) Vagolytic Atropine 0.5 mg IVP q3-5 min Max dose 3 mg Pacing
Decrease	Decrease intravascular volume −Diuresis −Dialysis/CRRT Decrease venous return −Nitroglycerin 10–200 μg/min −Morphine −Positive pressure ventilation	Peripheral vasodilation Nitroprusside 0.5–10 μg/kg/min Milrinone 0.125–0.5 μg/kg/min RASS inhibitors ACE-I ARB ARNI Positive pressure ventilation	Sedatives/anesthesia medications Propofol 5-50 μg/kg/min Dexmedetomidine 0.2–1.4 μg/kg/min Prolonged tachycardia	PharmacologicIV AmiodaroneLoad: 150mg IV x 1 over 1 min THEN 1 mg/min IV x 6 h, then 0.5 mg/min IV x 18 h Sedation/anxiolytic– Propofol – Dexmedetomidine DCCV if appropriate AF with RVR, VT with pulse
Special considerations	−Use PA catheter guidance whenever possible to assess RA and PCWP −Caution in preload sensitive states (eg, mitral stenosis)	−Use PA catheter guidance whenever possible to assess SVR and PVR −Initiate GDMT when euvolemic and normotensive −Aortic stenosis −afterload sensitive −Mitral regurgitation−consider afterload reduction	−Withdrawal of GDMT with b-agonist −Milrinone does not require cessation of beta-blockers −Ventricular arrhythmias −Avoid increasing contractility in LVOT obstruction	– Aortic regurgitation - consider maintaining elevated HR

ACE-I, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; ARNI, angiotensin receptor neprilysin inhibitor; CCB, calcium channel blocker; CRRT, continuous renal replacement therapy; DCCV, direct current cardioversion; GDMT, guideline directed medical therapy; HR, heart rate; MAP, mean arterial pressure; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrial pressure; RASS, renin-angiotensin-aldosterone system; SBP, systolic blood pressure; SVR, systemic vascular resistance.

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should be given to use axillary tMCS devices because they enable patient mobility and are a more stable and durable device position for prolonged hemodynamic support.[66] Maximum mobility after Impella 5.0 implantation may be associated with improved survival and justifies further study of exercise as a therapeutic modality.[67] Mobilization is particularly important in patients with HF-CS, given the potential for prolonged support as a bridge to surgical HRT.

Patients with HF-CS often present after a long course that results in chronic organ hypoperfusion and dysfunction. Patients suffering from chronic HF are at high risk of malnutrition and loss of muscle mass due to cardiac cachexia. Optimizing nutrition is an important therapeutic goal that must be balanced with the goals of achieving hemodynamic targets and increasing tissue perfusion. Historically, enteral nutrition was started after achieving hemodynamic stability and at a low rate of infusion due to concerns about splanchnic hypoperfusion,[68] although enteral nutrition in patients with postcardiotomy CS has been shown to be safe.[69]

Temporary Mechanical Circulatory Support Devices

Percutaneous tMCS devices are used with increasing frequency to increase MAP and to maintain end-organ perfusion in patients with CS, despite lack of evidence demonstrating improved outcomes over the IABP. Each tMCS device has unique hemodynamic effects, risk profiles and clinical considerations (Table 3) (Supplementary videos).

Device selection should be based on the underlying pathophysiology, urgency, magnitude, and duration of hemodynamic support, device availability and operator/institutional experience. Several facility-specific algorithms have been developed for device selection in CS.[7,70] In the CSWG registry and a registry from a multicenter network of tertiary CICUs (Critical Care Cardiology Trials Network), the use of tMCS is highly variable.[23,71] IABP was the most commonly used tMCS device in both registries and represented more than half of all devices used in patients who ultimately received HRT in CSWG. Importantly, patients receiving more than 1 tMCS device had a significantly higher in-hospital mortality rate than those treated with 1 device and had the highest in-hospital mortality rate, irrespective of the number of vasoactive drugs used.

Several studies have demonstrated the feasibility of tMCS support as a bridge to HRT by using a variety of percutaneous and surgical devices in uni- and biventricular support configurations.[66,72,73] An increased proportion of patients are now supported with IABP prior to both durable LVAD implant and heart transplant.[74,75] Following UNOS policy changes, there has been a significant increase in the listing of patients supported by tMCS, and fewer patients are supported by inotropes and bridge-to-transplant LVADs.[75–78] Although early reports raised concern about increased post-transplant mortality,[79] subsequent studies have demonstrated comparable outcomes under old and new allocation systems.[80–83]

Intra-aortic Balloon Pump

Based on the concept of counter-pulsation, IABPs are balloon-mounted catheters with a capacity of 40–55 cc placed in the descending aorta. IABPs inflate during diastole, thereby augmenting central aortic root diastolic pressure and coronary perfusion, and they deflate during systole, thereby creating a negative pressure sink that reduces LV afterload. IABPs can decrease LV cardiac work and myocardial oxygen consumption and provide up to 0.5–1 L/min of augmented cardiac output. Randomized control studies have failed to show any survival benefit of IABP in AMI-CS[4,84]; however, smaller substudies suggest potential benefit associated with pre-PCI compared to post-PCI IABP support in AMI-CS.[85] By contrast, IABPs have been associated with improved outcomes among patients with HF-CS as a bridge to LVAD or HT (Supplementary Table 3). In a retrospective single-center study of IABP use in patients with chronic HF-CS, IABP insertion showed the greatest augmentation of CO in patients with nonischemic cardiomyopathy and higher PAPi scores, suggesting the potential to predict response to IABP in selecting a mechanical-support strategy in HF-CS.[86] Additionally, axillary IABP inserted percutaneously or via surgical graft enables mobilization and rehabilitation during prolonged support as bridge to HT or LVAD.[66,87] No randomized controlled studies have evaluated the use of IABPs in HF-CS. The Alt-shock-2 trial is a prospective, multicenter trial that will randomize patients with HF-CS to IABP or vasoactive therapy with a primary endpoint of 60-day survival or bridge to HRT.[88]

Impella

Use of transvalvular microaxial flow pumps such as the Impella devices (Abiomed, Danvers, MA) for both AMI-CS and HF-CS is growing.[89] Impella devices use the principle of an Archimedes screw in which rotational kinetic energy from an impeller is transferred to blood and displaces blood from the LV to the aorta. The net result is a decrease in LV pressure and volume, known as LV unloading, and reduced myocardial oxygen consumption. Increased blood delivery to the aorta increases mean arterial pressure and systemic tissue perfusion. The family of Impella devices (2.5, CP, LD, 5.0, and 5.5) provide varying degrees of blood flow. Impella 5.0 and 5.5 pumps require surgical cut-down and are inserted through a vascular graft into the axillary artery or, like the LD, directly into the aorta, and can provide up to 5−6.2 L/min of flow.

Currently available randomized controlled trials in patients with AMI-CS are small and showed improved hemodynamics without differences in outcomes.[90–92] Data from large U.S. registries showed an association between Impella use and adverse outcomes, including death and bleeding.[93,94] The Impella 5.0 or 5.5 may provide greater

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Table 3. Characteristics of Mechanical Circulatory Support Devices Used in Heart Failure-Cardiogenic Shock

	IABP	Impella CP/5.0/5.5	Tandem Heart	ECMO	Centrimag	Impella RP	Protek Duo
Mechanism	Aorta	LV to Aorta	LA to Aorta	RA to Aorta	LV to aorta RV to PA	IVC to PA	RA to PA
Sheath size	8F	Impella CP: 14F Impella 5.0/5.5: 21/ 23F	Inflow: 21F Outflow: 15-17F	Inflow: 18-21F Outflow: 14-16F	Variable	22F	29F
Cardiac flow (L/min)	0.5	CP−3.5 5.0−4.5 to 5 5.5−5.5 to 6.0	2.5−5.0	3−7	5–10	4.5	5
Pump location	Extracorporeal	Intracorporeal	Paracorporeal	Paracorporeal	Paracorporeal	Intracorporeal	Paracorporeal
Maximum implant days	Days to weeks	Days to weeks	Weeks	Weeks	Weeks-month	Days to weeks	Days to weeks
Afterload	â	â	à	á	à	-	-
Complications							
Bleeding	+	++	++	+++	+	++	++
Hemolysis	+	+++	++	+++	+	+++	++
Limb ischemia	+	++	++	++		++	+
Stroke	+	++	++	+++	+	+	+
Others	Thrombocytopenia Aortic rupture/ dissection	− Tamponade due to LV perforation − Ventricular arrhythmia − Pump migration	− ASD − Cannula migration - Tamponade due to perforation	− Harlequin syndrome − LV Dilation − Oxygenator leak − Systemic gas embolism	− Cannula migration and kink − Pump thrombosis	− Pump migration − Pulmonary or tricuspid valve injury	− Cannula migration − Oxygenator leak
Contraindications	− Aortic − Dissection − AAA − AR − PVD	− Severe AS or AR − Mechanical AoV - LV thrombus- Recent TIA/stroke	- AR - VSD - PVD	− PVD − Mod to Severe AR	− Contraindication for anticoagulants	− Severe TS/PS − Severe TR/PR − Mechanical TV or PV − Mural thrombus of RA or IVC − IVC filter	− Severe TS/PS − Mechanical TV or PV − Mural thrombus of RA or IVC

AAA, abdominal aortic aneurysm; AR, aortic regurgitation; AoV, aortic valve; AS, aortic stenosis; ECMO, extracorporeal membrane oxygenation; F, French; IABP, intraaortic balloon pump; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; PA, pulmonary artery; PR, pulmonic regurgitation; PS, pulmonic stenosis; PV, pulmonic valve; PVD, peripheral vascular disease; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; TS, tricuspid stenosis; TV, tricuspid valve; VSD, ventricular septal defect.

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hemodynamic support for LV failure with fewer complications.[95]

The Impella RP is used for RV support and has an inlet area in the inferior vena cava and an outlet into the pulmonary artery. When used in combination with left-sided Impella pumps, these pumps can provide biventricular support.[96] In a nonrandomized, prospective trial, the Impella RP acutely improved hemodynamics in patients with RV failure after cardiotomy, AMI or LVAD.[97] More recently, preemptive Impella RP support has been used to limit post-LVAD RV failure.

TandemHeart

The TandemHeart (TH) system (LivaNova, London, United Kingdom) employs trans-septal cannulation of the left atrium to bypass blood to the femoral artery. This configuration significantly decreases LV preload and stroke volume and reduces the LV work load.[98,99] A small randomized controlled trial comparing TH and IABP in CS showed hemodynamic superiority of TH yet no difference in 30-day mortality.[100] TH is often used if contraindications to transvalvular approaches, such as LV thrombus or significant aortic insufficiency, are present. An oxygenator can also to be spliced into the arterial return circuit to treat hypoxemia.

Venoarterial Extracorporeal Membrane Oxygenation

VA-ECMO employs an extracorporeal centrifugal flow pump to displace and oxygenate blood from venous to arterial circulation. VA-ECMO is capable of providing full cardiopulmonary support and can be placed at the bedside, making it especially useful in later stages of shock or CA. VA-ECMO is associated with a higher rate of complications than other tMCS devices.[101] VA-ECMO provides retrograde flow into the aorta, thereby increasing LV afterload and LV filling pressures and leading to pulmonary edema, aortic root or LV thrombus, mitral or aortic regurgitation, and reduced coronary flow. LV decompression strategies (Table 4) are, thus, recommended in patients with severely impaired LV function.[102] ECMO as a strategy of bridge to LVAD is associated with worse odds of survival and increased need for biventricular support compared to IABP and other tMCS,[103] yet importantly, bridging from ECMO to VAD has survival rates equivalent as bridging from ECMO to HT.[104]

TandemHeart RVAD and ProtekDuo

The TH RV assist device (TH-RVAD) provides RV support by using an extracorporeal centrifugal flow pump to displace blood from the RA to the PA. Several studies have demonstrated significant hemodynamic efficacy with the TH-RVAD in patients with AMI-CS and HF-CS.[105,106] The ProtekDuo is a dual-lumen cannula that can enables single venous access via the right internal jugular vein for use with the TH-RVAD. Similar to the TH-LVAD, an oxygenator can be added to the system in patients with hypoxemia.

Vascular Safety

The limb ischemia and bleeding associated with tMCS use in CS are major determinants of morbidity and mortality.[107] Best practices for vascular safety should be used whenever possible, including ultrasound and fluoroscopic guidance, micropuncture needle access, initial and final run-off angiography, use of distal perfusion catheters from ipsilateral or contralateral arteries, preclosure of arteriotomy sites, and dedicated vascular safety bundles to minimize harm from large-bore peripheral access.[7] CICU management of vascular access should encompass tailoring antiplatelet and anticoagulant therapy, monitoring for bleeding and device malposition, and device removal in procedure areas. Multidisciplinary collaboration among CICU cardiologists, intensivists, interventional cardiologists, and surgeons is critical.

Systems of Care

CS is a heterogenous, time-sensitive condition that demands complex decision making and specialized interventions. Early recognition and treatment are needed to avoid CS progression, which confers greater likelihood of mortality and may preclude or compromise outcomes of LVAD or transplant.[108] The use of standardized staging definitions facilitates communication and can provide criteria for the transfer of patients to higher levels of care.[2] The National Cardiogenic Shock Initiative outlined facility-level definitions for CS care: Level I centers provide PCI, tMCS, and HRT; Level II centers provide PCI for STEMI; and Level III are non-PCI-capable hospitals.[109] The AHA proposed a model of regional systems of CS care delivery, with Level I centers serving as the hub, and Level II and III centers as the spokes, with a focus on early recognition and timely transfer of patients with CS to provide access to a full range of CS management.[2] Outcomes in CS are associated with centers' case volumes, underscoring the importance of timely transfer to a level 1 CS center,[110] and several single-center studies have shown this model to be feasible and effective in improving survival, but randomized large-scale studies are lacking.

Palliative Care

Early involvement of palliative care in the clinical course of the patient with CS is appropriate to clarify the goals and limits of care and to provide patient and caregiver support. Despite the known high mortality rates of CS, palliative care is consistently underused.[111] Palliative care consultation has been shown to be associated with lower rates of invasive procedures, readmissions and hospital costs.[112] As in other areas of medicine, shared decision making remains pivotal to patient-centered health care delivery.

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Table 4. Venting Strategies for Extracorporeal Membrane Oxygenation

	Mechanism	Advantages	Disadvantages
Pharmacological venting			
Inotropes	Increase LV contractility	Noninvasive	– Increases LV work – Myocardial ischemia – Risk of arrhythmias
Passive venting			
Atrial septostomy	Allows for passive shunting of blood from the left to right atrium	No intracardiac device required	– Trans-septal puncture required – Limited durability – Cannot regulate flow
Venting with central (surgical) VA-ECMO			
Surgical cannulation of the left atrium, pulmonary vein, or left ventricle	Displaces blood from the left atrium or ventricle into the inflow segment of VA-ECMO (reduces LV pre-load)	High flow capacity and durable	– Requires surgical access – Risk of cardiac damage and arrhythmias
Venting with peripheral (nonsurgical) VA-ECMO			
Trans-septal inflow catheter or cannula	Displaces blood from the left atrium into the inflow segment of VA-ECMO (reduces LV preload)	High flow capacity and magnitude of flow can be regulated	– Trans-septal puncture required – Limited durability – Risk of cannula migration and vascular injury
Pulmonary artery cannula	Displaces blood from the pulmonary artery into the inflow segment of VA-ECMO (partially reduces LV preload)	High flow capacity and magnitude of flow can be regulated	– Requires cannulation of the pulmonary artery via the femoral vein – Limited efficacy and durability – Risk of cannula migration and vascular injury
Intra-aortic balloon pump	Reduces left ventricular afterload	Minimally invasive (8−9F sheath)	– Partial unloading effect – Fails with tachyarrhythmias – Risk of vascular injury
Impella	Displaces blood from the left ventricle into the aorta	– Nonsurgical – Direct LV unloading – Good for de-escalation from VA-ECMO to isolated LV support	– 13−14F sheath or surgical graft required (for Impella 5.0) – Risk of hemolysis – Aortic root or LV thrombus formation – North-south syndrome – Vascular injury

F, French; ECMO, extracorporeal membrane oxigenation; LV, left ventricle; VA, venoarterial.

Knowledge Gaps and Perspective

Although there have been important advances in contemporary HF-CS management, there is a paucity of data addressing extensive gaps in evidence. Given the difficulty in performing randomized trials in CS, current management recommendations are mostly empirical or extrapolated from AMI-CS trials. In general, there is a poor level of evidence for many of the interventions in AMI-CS, and this is especially true for HF-CS.[113]

High-priority areas for research include: (1) the role of PAC and invasive hemodynamic parameters in diagnosing and managing HF-CS; (2) comparison of outcomes with various tMCS devices in patients with similar CS stages and hemometabolic profiles to inform CS treatment algorithms; (3) drug and device strategies to treat venous and end-organ congestion in HF-CS; (4) diagnosing and managing RV/biventricular dysfunction; and (5) testing whether regionalized systems of CS care improve outcomes.

Conclusions

HF-CS is a complex syndrome that is increasingly prevalent in the modern CICU. Because of its distinctive presentation, pathophysiology, trajectory, treatment objectives, and outcomes, care of the critically ill patient with HF-CS requires a comprehensive, multidisciplinary approach and early referral of appropriate patients to advanced HF centers. Research to address major evidence gaps is urgently needed.

Disclosures

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Dr. Jacob Abraham has received speaker/consulting honoraria from Abbott and Abiomed.

Dr. Dan Burkhoff has received an unrestricted institutional educational grant from Abiomed and is a consultant to PVLoops LLC.

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Dr. Shashank S. Sinha is a consultant for Abiomed.

Dr. Jaime Hernandez-Montfort is a consultant for Abiomed.

Dr. Navin K. Kapur has received institutional research grants and speaker/consulting honoraria from Abbott, Abiomed, Boston Scientific, Edwards, Getinge, Medtronic, MD Start, NXT Technologies, LivaNova, Precardia, Stealth Peptides, and Zoll.

All other authors have no disclosures to report.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.cardfail.2021.08.010.

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