Heart rescue: the role of mechanical circulatory support in the management of severe refractory cardiogenic shock

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Purpose of review
Cardiogenic shock is present in 3.5% of patients presenting with acute decompensated heart failure. Despite advances in therapy, mortality remains high, approaching 70% in some settings. Recent management strategies have incorporated the use of mechanical circulatory support (MCS), which has been associated with better survival in nonrandomized trials. MCS is increasingly used in the acute setting and has become an important treatment modality for cardiogenic shock.

Recent findings
Small studies have demonstrated improved survival when MCS is instituted early in the management of cardiogenic shock. Numerous case reports support the benefit of MCS for various causes of cardiogenic shock, including acute myocardial infarction, cardiac allograft rejection, myocarditis and refractory arrhythmias.

Summary
This article will review novel strategies in the management of cardiogenic shock including percutaneous MCS (intra-aortic balloon pump, Impella, TandemHeart, venoarterial extracorporeal membrane oxygenation) and surgically implanted devices (CentriMag) that are used for short-term management. We will review the mechanisms involved in cardiogenic shock and discuss management and device selection strategies.

Keywords
cardiogenic shock, mechanical circulatory support, percutaneous ventricular assist device, severe refractory shock

INTRODUCTION

Cardiogenic shock is a common endpoint of multiple disease processes that is characterized by myocardial dysfunction, depressed cardiac output (*CO*) and end-organ hypoperfusion. Cardiogenic shock is associated with significant morbidity and mortality, and conventional medical support such as inotropic agents or intra-aortic balloon counter pulsation is often insufficient to reverse the hemodynamic changes seen in cardiogenic shock. Advances in management, including early revascularization have led to a reduction of in-hospital mortality of more than 10% [1]. A further reduction may be seen with the advancement of mechanical circulatory support (MCS), which provides a means for patients to recover or transition to long-term therapies for management of their underlying cardiac disease. In particular, the development of percutaneous MCS options has facilitated rapid resuscitation of the cardiogenic shock patient potentially interrupting the characteristic systemic inflammatory response before it can cause irreversible harm.

DEFINITION AND CAUSES OF CARDIOGENIC SHOCK

Cardiogenic shock is defined as sustained hypotension [SBP <90 mmHg or mean arterial pressure

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KEY POINTS

- Patients with acute refractory cardiogenic shock have a poor prognosis and medical therapy using inotropic agents and vasopressors is often insufficient for adequate hemodynamic support in these patients.
- MCS offers the ability to restore hemodynamics and prevent end organ damage associated with severe cardiogenic shock.
- There is a 'window of opportunity' for rescue intervention with MCS, beyond which a patient is too ill to benefit, thus early intervention is crucial.
- Device selection is based on the degree of hemodynamic support needed, whether right ventricular failure or lung injury is present, individual patient factors and the availability of interventionalists/cardiac surgeons.

(MAP) that is 30 mmHg below baseline], depressed cardiac index (<2.2 l/min/m^2) with a pulmonary capillary wedge pressure at least 12 mmHg, and evidence of diminished tissue perfusion (decreased urine output, altered mental status, cool extremities). Patients who require inotropes/vasopressors or an intra-aortic balloon pump (IABP) to maintain a normal SBP or *CO* are also considered to have cardiogenic shock.

Cardiogenic shock often occurs as the result of an acute event that precipitates rapid cardiovascular collapse. Among patients with an acute ST-elevation myocardial infarction (MI), 8% will develop cardiogenic shock [2] typically within 24 h of the onset of symptoms [3]. In these patients, cardiogenic shock is typically a direct consequence of regional myocardial dysfunction and diminished contractility. Mechanical complications of MI including ventricular septal defect, papillary muscle rupture producing acute mitral regurgitation, and free left ventricular wall rupture can also cause cardiogenic shock.

Many nonischemic disease processes may present acutely or subacutely and result in cardiogenic shock. Acute valvular regurgitation, regardless of cause, can rapidly progress to severe heart failure. Several types of cardiomyopathies can present with a fulminant course, including viral myocarditis, giant-cell myocarditis, peripartum and Takotsubo cardiomyopathy. Extracardiac disease may also result in cardiogenic shock, as with a massive pulmonary embolism or pericardial tamponade. Finally, 3–4% of patients admitted to the hospital for acute decompensation of chronic heart failure will present with shock [4].

PATHOPHYSIOLOGY

With the exception of acute valvular disease, cardiogenic shock typically occurs in the setting of pronounced myocardial dysfunction and low *CO*. The reduction in MAP results in poor systemic perfusion and end-organ ischemia. Low coronary perfusion pressure may exacerbate ischemia. Catecholamine release attempts to compensate for the low-output state by increasing inotropy and peripheral vasoconstriction at the cost of increasing myocardial oxygen demand. Upregulation of the neurohormonal systems promotes sodium and fluid retention, potentially increasing blood pressure but worsening congestion. There are increased cytokine levels and expression of inducible nitric oxide synthase [2], which can exacerbate hypotension and further worsen myocardial function, causing a deterioration of cardiovascular hemodynamics.

Cardiogenic shock has been divided into four stages to demonstrate severity and progression of disease: preshock, mild shock, profound shock and severe refractory cardiogenic shock [5]. The progression from mild cardiogenic shock to severe refractory cardiogenic shock reflects the severity of hemodynamic compromise and is reflected by the number of vasoactive medications required to maintain reasonable *CO* and MAP. In mild shock, the cardiovascular system may not require support or can be easily supported with low doses of one inotrope or vasopressor. Patients with profound shock require moderate-to-high doses of a single agent, whereas patients with severe refractory cardiogenic shock remain hemodynamically compromised, despite high doses of multiple vasoactive medications. Mortality increases progressively with each stage, and patients with severe refractory cardiogenic shock generally have a very poor prognosis in the absence of MCS.

INITIAL MANAGEMENT OF CARDIOGENIC SHOCK

The initial management goals of cardiogenic shock include cardiovascular resuscitation and identification of the underlying cause. Reversible cardiac causes, including arrhythmias and conduction disturbances, should be identified and treated. If myocardial ischemia or infarction is suspected by history or ECG, patients should rapidly undergo coronary angiography and either percutaneous or surgical revascularization. In the SHOCK (One-year survival following early revascularization for cardiogenic shock) trial, early revascularization in those presenting with cardiogenic shock reduced 1-year mortality from 66 to 53% [6]. A similar trend has been noted in a Canadian database over time with

greater adoption of aggressive revascularization strategies [7].

Medical therapy of cardiogenic shock is directed at normalizing hemodynamic parameters, correcting metabolic disarray and minimizing end-organ dysfunction. Vasoactive agents (inotropes, vasopressors) are often required to augment *CO* but at the expense of worsening myocardial oxygen demand, exacerbation of ischemia and potentiation of arrhythmias. Correction of acidosis may help to prevent damage to end-organs and to promote the effects of vasoactive agents. Those patients with continued worsening or lack of improvement of hemodynamics despite escalation of medical therapy are considered to have severe refractory shock and should immediately be considered for placement of MCS.

MECHANICAL CIRCULATORY SUPPORT IN THE TREATMENT OF CARDIOGENIC SHOCK

The key concept is to quickly identify patients in need of more support than medical management and/or an IABP can achieve, as early intervention with MCS in the patients at highest risk is most effective when done early. MCS can interrupt the inflammatory cascade initiated by the onset of shock and prevent progression to irreversible end-organ damage and subsequent death; however, there remains a window of opportunity during which rescue is possible. An IABP is typically the first line of mechanical support used due to ease of insertion and minimal risk, but it is often insufficient in providing adequate support in patients with severe cardiogenic shock. Other options for temporary support include the Impella percutaneous ventricular assist device (PVAD), TandemHeart PVAD, venoarterial extracorporeal membrane oxygenation (ECMO) and the CentriMag device, which can be placed surgically or percutaneously (Table 1). Device selection is based on a number of factors including the degree of hemodynamic support needed, whether right ventricular failure or lung injury is present, individual patient factors (e.g. mechanical valves, peripheral vascular disease) and the availability of interventionalists/cardiac surgeons.

At our institution we have created a multidisciplinary team that includes heart failure cardiologists, interventional cardiologists and cardiac surgeons who work together to rapidly assess and triage patients to the appropriate form of mechanical assistance required for each clinical situation (Fig. 1). This 'shock team' is activated for patients with profound cardiogenic shock, and an immediate bedside evaluation is made.

Table 1. Characteristics of various devices used in the management of severe refractory cardiogenic shock

Device	IABP	Impella 2.5	Tandem	Impella 5.0	VA ECMO	Centrimag
Maximum support	Add 0.5 l/min	2.5 l/min	4.5 l/m	5.0 l/min	Up to 6 l/min	Up to 10 l/min
Contraindications	AI, aortic aneurysm or dissection, severe PAD (relative), coagulopathy	LV thrombus, mechanical aortic valve severe AS, HOCM, VSD, severe PAD (relative)	Bleeding diathesis, uncontrolled sepsis, severe PAD, LV thrombus	LV thrombus, mechanical aortic valve severe AS, HOCM, VSD, severe PAD (relative)	Irreversible brain injury, contraindication to anticoagulation	Irreversible brain injury, contraindication to anticoagulation
Ease of insertion (1 very easy, 5 most technically challenging)	1	2	4	3	2	5
LV support	+	++	+++	+++	++++	++++
RV support			++ (when placed in a right sided configuration)		++	+++ (when placed in a right sided configuration)
Respiratory support	–	–	–	–	+	–
Hemolysis	–	+++	++	++	+	+
Bleeding	–	–	++	+	+++	++
TCP	++	+	+?	+	++	+
Duration of use	Up to 90 days	Up to 4 weeks	Days to weeks	Up to 4 weeks	Up to 60 days or more	Months

AI, aortic insufficiency; AS, aortic stenosis; ECMO, extracorporeal membrane oxygenation; HOCM, hypertrophic obstructive cardiomyopathy; IABP, intra-aortic balloon pump; LV, left ventricular; PAD, peripheral artery disease; RV, right ventricular; TCP, thrombocytopenia; VA, venoarterial; VSD, ventricular septal defect.

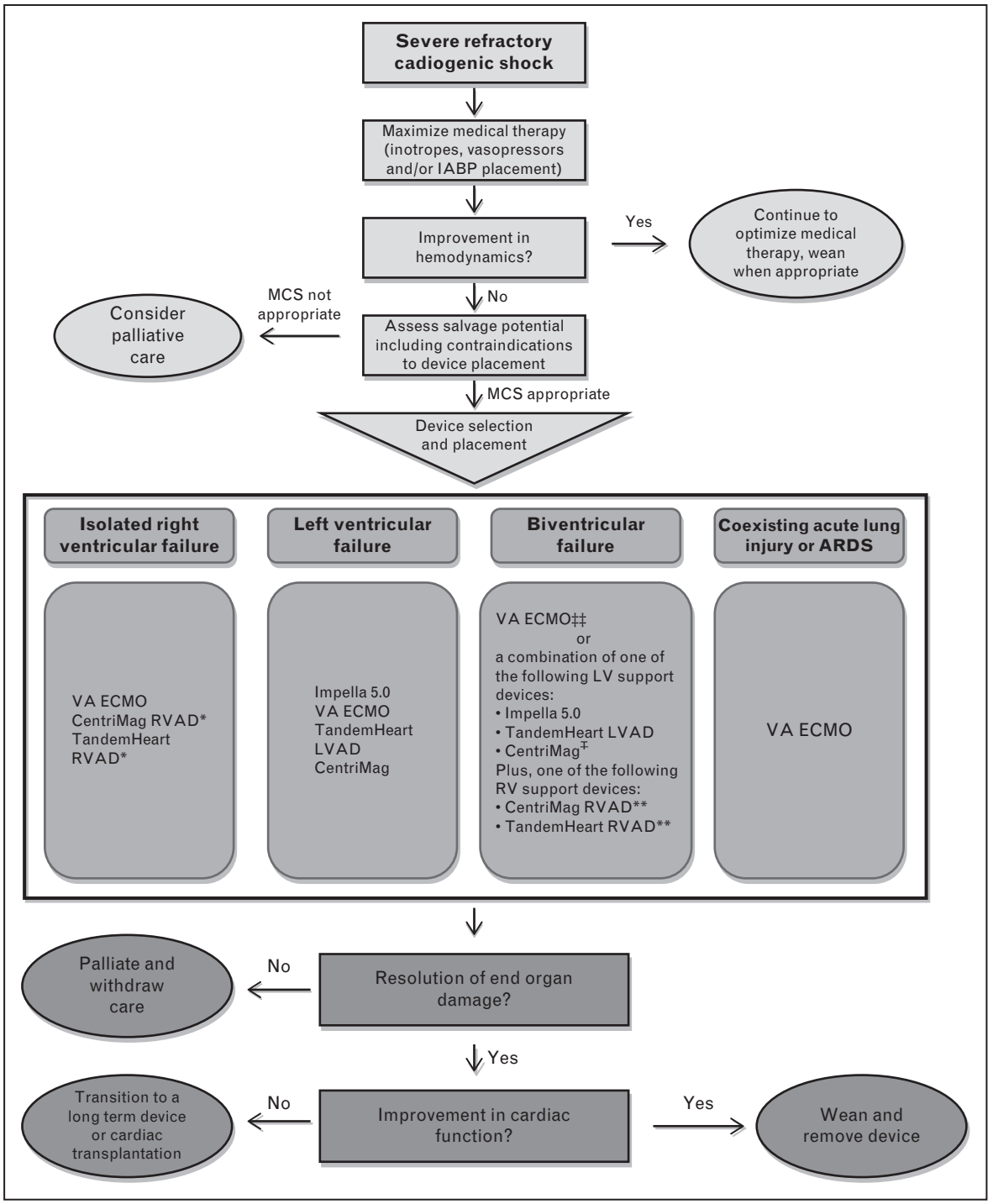

FIGURE 1. Heart rescue algorithm for patients with severe refractory cardiogenic shock. IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; MCS, mechanical circulatory support; RVAD, right ventricular assist device; VA ECMO, venoarterial extracorporeal membrane oxygenation. ‡The CentriMag device can be placed percutaneously or via open surgical technique. ‡‡VA ECMO is the preferred device in patients with biventricular failure, but other options can be used. *The CentriMag and TandemHeart should not be used in the setting of acute pulmonary embolism. **The CentriMag and TandemHeart can be used to support the right ventricle, with cannula placement into the right atrium (inflow) and main pulmonary artery (outflow). Right-sided support devices should be reserved for patients with severe right ventricular failure by hemodynamic criteria.

INTRA-AORTIC BALLOON COUNTERPULSATION

The IABP is the most commonly used form of MCS and has ACC/AHA class I indication in the adjunctive management of acute MI complicated by cardiogenic shock [8]. The IABP is a percutaneously inserted balloon that lies in the descending aorta and augments coronary blood flow by inflating during diastole, while also assisting myocardial function through reduced afterload by deflating during systole. The ultimate effect on *CO* is limited to an increase of 0.5–1.0 l/min, thus it is often insufficient to support patients with profound shock. It is relatively easy to insert, has a low rate of complications, and is low cost. The IABP can be placed in the cardiac catheterization laboratory or at the bedside in less than 15 min.

Outcomes data using the IABP are scarce. In the SHOCK trial, rapid improvement in hemodynamics following IABP placement was associated with a survival benefit [9]. However, a recent single-center trial did not find a difference in *CO*, cardiac power or systemic vascular resistance between patients randomized to IABP or medical therapy alone [10]. A meta-analysis identified only three randomized studies comparing IABP to medical therapy, comprising less than 200 patients [11▪], and among these limited samples no mortality benefit was found. Despite lingering questions about the efficacy of IABP therapy, it remains the first-line therapy for the treatment of cardiogenic shock at most centers.

TANDEMHEART PERCUTANEOUS VENTRICULAR ASSIST DEVICE

The TandemHeart PVAD (CardiacAssist, Inc., Pittsburgh, Pennsylvania, USA) is an external centrifugal blood pump with percutaneous cannulae. The inflow cannula is placed in the left atrium via a transseptal puncture. Pump outflow is returned to the body through a 17 French cannula in the femoral artery. It typically augments *CO* up to 3.0–4.0 l/min. Its use is limited by access site complications, limb ischemia and bleeding. Implantation is more time-consuming and requires specialized expertise, due to the need for a transseptal puncture. The presence of a cannula in the left atrium can be a nidus for thrombus formation. One significant advantage of the TandemHeart is that it can be configured to provide right ventricular support with inflow cannula placement into the right atrium and outflow cannula placement into the main pulmonary artery [12]. The TandemHeart is FDA-approved for up to 6 h of use, but successful use has been reported for greater than 1 week [13].

Two randomized trials have demonstrated superior hemodynamic support with the TandemHeart PVAD as compared to an IABP, while also showing an increase in complications with the TandemHeart [14,15]. Neither study showed a mortality advantage for the TandemHeart, although the ability to do so may have been limited by small sample sizes. In the largest reported series, 117 patients with refractory cardiogenic shock were implanted with the TandemHeart for an average of 5.8 days [16▪▪]. The population was critically ill, with a MAP of 45 mmHg, cardiac index of 0.5 l/min/m^2, and lactic acid level of 24.5 mg/dl. TandemHeart support provided rapid reversal of the hemodynamic abnormalities, increasing the MAP to 81 mmHg, cardiac index to 3.0 l/min/m^2, and decreasing the lactic acid level to 11.0 mg/dl. Although there was no control group, the 30-day mortality of 40% was considerably better than expected outcomes in this population. The most common complications were bleeding and sepsis.

IMPELLA RECOVER PERCUTANEOUS VENTRICULAR ASSIST DEVICE

The Impella Recover PVAD (AbioMed Inc, Danvers, Massachusetts, USA) comes in two sizes: the 2.5, which is percutaneously inserted, and the 5.0, which is surgically implanted. The Impella 2.5 is an axial flow motor that pumps blood from the left ventricle into the ascending aorta. The catheter is placed percutaneously through a tapered 13 or 14 French sheath and is connected to an external power source. Flow is less robust than the TandemHeart, averaging less than 2.5 l/min. However, implantation is quicker and there are fewer access site complications due to the smaller sheath size. A comparison of the Impella 2.5 with IABP showed better initial hemodynamic support with the Impella PVAD, but no difference in mortality or support after 6 h [17]. The primary complication of the Impella 2.5 is hemolysis, which can be severe and often limits the duration of use. Another common issue is pump migration from its intended position, which may lead to poor support or contribute to hemolysis.

The Impella 5.0 is a larger device, providing flows up to 5.0 l/min. Due to its size, it must be implanted surgically, either directly into the ascending aorta or through a vascular graft to the axillary artery. Due to the larger size of the inflow, hemolysis is a less frequent complication. A recent report of one center's experience with the Impella in patients with acute MI complicated by cardiogenic shock showed better early outcomes in patients who initially received the Impella 5.0 instead of the

Impella 2.5 [18▪▪]. These authors also demonstrated a frequent need to upgrade the Impella 2.5 to an Impella 5.0, suggesting that it is a reasonable approach to start with the Impella 5.0 in severe cardiogenic shock. In a series of postcardiotomy shock patients supported, the Impella 5.0 provided significant improvements in cardiac index, MAP, and pulmonary artery pressures. Survival at 30 days and 1 year were 94 and 75%, respectively [19].

Our institutional preference is to opt for the Impella 5.0 rather than the 2.5 when profound cardiogenic shock is present, as the Impella 2.5 is often inadequate in providing the desired level of support for these patients.

EXTRACORPOREAL MEMBRANE OXYGENATION

ECMO circuits include a centrifugal blood pump, a membrane oxygenator and inflow, and a circuit consisting of inflow and outflow cannulae. Venoarterial ECMO can provide support for patients with lung injury as well as either univentricular or biventricular failure. In the most commonly used percutaneous configuration, the inflow cannula is inserted into the right atrium through either the femoral or jugular vein and the outflow cannula is placed in the lower descending aorta via the femoral artery. Due to the large size of the arterial cannula (18 French), an antegrade catheter is often placed in the ipsilateral femoral artery to provide adequate perfusion to the leg. A percutaneous circuit can be established in less than 30 min, and it is feasible to put patients on ECMO at the bedside during an emergency. When percutaneous access is not possible, the ECMO circuit can be placed centrally, with direct cannulation of the right atrium and aorta.

Of the percutaneous MCS options, ECMO provides the most cardiac support, with the ability, based on cannula size and position, to achieve flow of greater than 6.0 l/min. However, ECMO is resource intensive, requiring continuous monitoring by nursing and trained perfusion staff. Complications include limb ischemia, bleeding, stroke, and infection. High levels of anticoagulation (activated clotting time 180–220 s) must be maintained to prevent thromboembolic complications. In patients with pulsatility during support, care must be taken to ensure that blood leaving the heart is adequately oxygenated, as perfusing the coronary arteries and brain with deoxygenated blood may result in catastrophic anoxic injury. Alternatively, with severe ventricular dysfunction, the left ventricle (LV) may not be adequately decompressed due to return of blood to the left atrium through the bronchial circulation. Left ventricular distension can lead to excess wall stress and may impede ventricular recovery. Several methods of decompressing the LV have been described, including a transseptal catheter [20], a pulmonary artery cannula [21▪], and minimally invasive placement of an apical vent [22]. Several recent reports have described successful use of an Impella Recover 2.5 as a vent for the LV [23,24▪,25].

Following institution of ECMO, there is often a rapid reversal of hemodynamics with a decrease in inotrope/vasopressor requirement, improvement in gas exchange, and reduction in markers of end-organ failure. With meticulous care, ECMO support can be maintained for weeks. No randomized trials have been performed to evaluate the efficacy of ECMO. However, retrospective evaluations of patients with cardiogenic shock due to an acute coronary syndrome have demonstrated reasonable outcomes in a critically ill population [26▪▪,27▪,28]. In a cohort of 15 patients with cardiogenic shock due to a variety of causes, ECMO support was maintained for an average of 11.5 days, and 12 were successfully weaned from ECMO with seven patients surviving to discharge [29]. Although results have been promising for the use of ECMO in acute cardiomyopathies, there are limited data about supporting patients with chronic cardiomyopathies who develop cardiogenic shock. One report showed a dismal 11% 1-year survival among this patient population [30▪▪].

CENTRIMAG VENTRICULAR ASSIST DEVICE

The Thoratec CentriMag VAD (Thoratec Corporation, Pleasanton, California, USA) is a centrifugal pump with a magnetically levitated rotor that can provide up to 10 l/min of blood flow. The CentriMag can be connected to many different types of circuits, including ECMO, but is designed as an extracorporeal, surgically implanted VAD for short-term or intermediate-term support. For left ventricular support, an inlet cannula is placed in the left ventricular apex (the left atrium is not recommended due to the potential for thromboembolic complications) with the outlet cannula delivering blood to the aorta. The CentriMag can also provide right ventricular support with inflow from the right atrium and outflow into the pulmonary artery. Two CentriMags can also be configured to provide biventricular support.

The primary advantage of the CentriMag system is its ability to deliver high-flow rates and to completely unload the LV. The system is relatively easy to use and has a low rate of thromboembolism when high-flow rates are maintained The CentriMag is

more durable than PVADs and can provide effective support for weeks to months [31]. A multicenter investigation of the CentriMag in 38 patients demonstrated a 47% 30-day survival. The major complications included infection and neurological dysfunction [32▪]. The CentriMag can be configured to support the right ventricle percutaneously with an inflow cannula placed in the right atrium via the femoral vein and the outflow cannula placed in the pulmonary artery via the internal jugular vein [33▪].

CONCLUSION

Cardiogenic shock is a systemic illness associated with rapid progression of multiorgan dysfunction. Early intervention is crucial to prevent the irreversible consequences of multiorgan failure. Developments in MCS technology have facilitated more widespread use of both percutaneous and surgically implanted devices for short-term support until myocardial recovery, transition to a long-term MCS device, or cardiac transplantation. Future developments, including better patient selection and safer percutaneous devices, should continue to improve outcomes in high-risk patients with cardiogenic shock.

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Conflicts of interest

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There are no conflicts of interest.

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- ▪ of special interest
- ▪▪ of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 555–559).

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