Cardiol Ther (2025) 14:123–139
https://doi.org/10.1007/s40119-025-00413-9

REVIEW

Left Ventricular Mechanical Circulatory Support Devices for Cardiogenic Shock After Myocardial Infarction

Duc M. Giao · Robert P. Giugliano

Received: September 7, 2024 / Accepted: April 10, 2025 / Published online: May 9, 2025

ABSTRACT

Cardiogenic shock is the most common cause of mortality in patients with acute myocardial infarction (AMI). Historically, AMI complicated by cardiogenic shock was associated with in-hospital survival of only ~50%. Recent advances in mechanical circulatory support have allowed for improved survival rates compared with only conventional medical treatment. However, the management strategy for AMI-related cardiogenic shock remains largely empirical due to limited high-quality evidence-based studies. In this review, we provide an overview of the four types of left ventricular mechanical circulatory support currently available, review new guideline updates from the American College of Cardiology Foundation/American Heart Association and European Society of Cardiology, and discuss recent and ongoing studies and registries in cardiogenic shock following AMI.

Keywords: Left ventricular mechanical circulatory support; Cardiogenic shock; Acute myocardial infarction

Key Summary Points

Cardiogenic shock most commonly presents secondary to acute myocardial infarction (AMI) and remains the leading cause of in-hospital mortality after AMI.

Despite advances in treatments for cardiogenic shock, no consensus exists for optimal therapy.

Current commonly used mechanical circulatory support devices for the left ventricle include intra-aortic balloon pumps, percutaneous ventricular assist devices, and veno-arterial extracorporeal membrane oxygenation.

Several recent trials in mechanical circulatory support devices for cardiogenic shock provide new data to guide clinical practice.

D. M. Giao
Department of Cardiac Surgery, Smidt Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, USA

R. P. Giugliano (✉)
Division of Cardiovascular Medicine, Brighamand Women's Hospital, Harvard Medical School, Boston, MA 02115, USA
e-mail: rgiugliano@bwh.harvard.edu

INTRODUCTION

Cardiogenic shock (CS) most commonly occurs secondary to acute myocardial infarction (AMI), comprising ~50% of all cases of CS [1]. CS occurs as a sequela in 5–10% of all patients presenting with an AMI [2] and is one of the leading causes of death, with in-hospital mortality historically exceeding 50% [3]. CS is defined by low cardiac index (< 1.8 L/min/m^2 without support, < 2.0 to 2.2 L/min/m^2 with support), systolic blood pressure < 90 mmHg, and signs of systemic hypoperfusion such as elevated lactic acid in the absence of hypovolemia or other causes of lactic acidosis [4]. Despite the development of advanced treatment therapies to treat CS and reduce mortality, no universal consensus exists for the use of left ventricular mechanical circulatory support (MCS) devices in the management of CS.

The primary treatment for CS involves addressing the root cause and providing supportive care of ventricular function, initially with intravenous inotropic and vasoactive drugs. [5] When medical therapy is insufficient, MCS is often necessary to prevent further end-organ injury and death. Current MCS options include left ventricular, right ventricular, and biventricular support, and may be temporary or durable [5, 6]. The goal of temporary MCS is to bridge a patient to recovery from the instigating event. Other therapies (medical, coronary artery revascularization, durable ventricular assist devices [VADs], or heart transplantation) are implemented to reverse the underlying cause of CS [5].

In this review, we focus on temporary left ventricular MCS (intra-aortic balloon pumps (IABP) [5], VADs such as TandemHeart® [7] and Impella® [8], and veno-arterial extracorporeal membrane oxygenation (VA-ECMO)® [9], discussing their mechanisms of action, hemodynamic effects, and relevant guideline updates, clinical trials, and registries.

This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

CLASSIFICATION OF CARDIOGENIC SHOCK

The Society for Cardiovascular Angiography and Intervention (SCAI) has developed a classification system for CS that categorizes the severity of cardiogenic shock ranging from “at risk” to “extremis” (Table 1). Stage A (**A**t risk) is characterized by patients who are not currently experiencing signs or symptoms of CS, but remain at risk for its development (e.g., following a large AMI, prior infarction, acute or acute-on-chronic heart failure). Stage B (**B**eginning shock) includes patients who have clinical hemodynamic instability (e.g., hypotension and/or tachycardia) without hypoperfusion. Stage C (**C**lassic shock) is described as patients with hypoperfusion who require intervention (pharmacologic or mechanical) beyond volume resuscitation to restore end-organ perfusion. Patients with stage C who are clinically worsening with failure of initial support strategy to restore perfusion as assessed by worsening hemodynamics and/or rising lactate are considered to have progressed to stage D (**D**eteriorating shock). Stage E (**E**xtremis) is defined as actual or impending circulatory collapse [4].

MEDICAL TREATMENT

Delayed recognition and treatment of CS may lead to rapid deterioration with high in-hospital mortality despite advances in medical therapies, including prompt mechanical revascularization. The mainstay of initial treatment for AMI-related CS (AMI-CS) involves reversal of the underlying cause and the use of vasopressors and inotropes [10]. The need for vasoactive medications is associated with increased risk of short-term mortality, and an increase in the likelihood of in-hospital death has been found with the use of more vasoactive medications [11]. Vasopressors are used in approximately 90% of patients with CS with refractory hypotension to maintain mean arterial pressure and tissue perfusion pressures [10]. The most commonly used vasopressors are

Table 1 Society for Cardiovascular Angiography and Intervention stages of shock

Stage	Description	Hemodynamics
A (At risk)	A patient who is not currently experiencing signs or symptoms of CS, but is at risk for development. May include patients with large AMI or prior infarction and/or acute on chronic heart failure symptoms	Normotensive (SBP ≥ 100 mmHg or normal for the patient) If invasive hemodynamics measured: • Cardiac index ≥ 2.5 L/min • CVP < 10 mmHg • PA saturation ≥ 65%
B (Beginning shock)	A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion	SBP < 90 mmHg or MAP < 60 mmHg or > 30 mmHg drop from baseline Pulse ≥ 100 beats/min If hemodynamics measured: • Cardiac index ≥ 2.2 L/min • PA sat > 65%
C (Classic shock)	A patient that manifests with hypoperfusion that requires intervention (inotrope, pressor or mechanical support) beyond volume resuscitation to restore perfusion Patients typically present with relative hypotension	May include: SBP < 90 or MAP < 60 OR > 30 mmHg drop from baseline and drugs/device used to maintain BP above these targets If hemodynamics taken: • Cardiac index < 2.2 L/min • PCWP > 15 mmHg • RAP/PCWP ≥ 0.8 • PAPi < 1.85 • Cardiac power output ≤ 0.6 Watts
D (Deteriorating)	A patient that is similar to category C but is getting worse, and have not responded to initial interventions	Any of stage C and either or both of the following: Require multiple pressors or addition of mechanical circulatory support devices to maintain perfusion
E (Extremis)	A patient that is experiencing cardiac arrest with ongoing CPR and/or ECMO, being supported by multiple interventions	No SBP without resuscitation PEA or refractory VT/VF Hypotension despite maximal support

AMI acute myocardial infarction, *BP* blood pressure, *CPR* cardiopulmonary resuscitation, *CS* cardiogenic shock, *CVP* central venous pressure, *ECMO* extracorporeal membrane oxygenation, *MAP* mean arterial pressure, *PA* Pulmonary artery, *PAPI* pulmonary artery pulsatility index (PA systolic pressure - PA diastolic pressure / RAP), *PCWP* pulmonary capillary wedge pressure, *PEA* pulseless electrical activity, *RAP* right atrial pressure, *SBP* systolic BP, *VF* ventricular fibrillation, *VT* ventricular tachycardia

Permission was obtained to reproduce this table from Baran, D.A., et al., *SCAI clinical expert consensus statement on the classification of cardiogenic shock: This document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019*. Catheter Cardiovasc Interv, 2019. **94**(1): p. 29–37

vasopressin and phenylephrine; both increase systemic vascular resistance and mean arterial pressure albeit through different mechanisms of smooth muscle vasoconstriction [12].

Inotropes improve the contractility of the myocardium by increasing the force of myocardial contraction and as such are indicated in patients with tissue hypoperfusion despite volume resuscitation [13]. The most frequently used inotropic agents are adrenergic agonists (e.g., dobutamine, dopamine, epinephrine, norepinephrine), which exert positive inotropic effects by acting on beta-adrenergic receptors to increase the heart rate, stroke volume, and cardiac output [10]. Milrinone and levosimendan can further augment cardiac output especially in patients treated with beta blockers, as their mechanisms of action are independent of the beta-adrenergic receptor [12]. The optimal selection and dosing of inotropes is often guided by invasive hemodynamic monitoring with a pulmonary artery catheter [13].

Randomized data comparing inotropes in cardiogenic shock are sparse. The SOAP II trial was a large, randomized comparison in 1679 patients comparing dopamine versus norepinephrine as the initial vasopressor to manage various types of shock. This trial showed no difference between dopamine and norepinephrine in the primary outcome of death at 28 days. [14] However, a prespecified secondary analysis limited to 280 patients with CS revealed that dopamine was associated with higher mortality at 28 days compared to norepinephrine (p=0.03). One potential explanation is that dopamine doubled the rate of arrhythmic events compared to norepinephrine (24.1% vs. 12.4%; p<0.001) [14]. Additional support for norepinephrine as the initial vasopressor in CS comes from a small (n=57) randomized controlled trial that found refractory CS after AMI was more common in patients treated with epinephrine compared to norepinephrine (37.0% vs. 27.0%; p=0.01), although there was no difference in the primary endpoint of change in cardiac index change at 72 h [12].

A randomized controlled trial comparing two inodilators, milrinone versus dobutamine, in 192 patients with CS found no significant difference in the primary composite of in-hospital death, receipt of a heart transplant or MCS, resuscitated cardiac arrest, nonfatal MI, transient ischemic attack, or stroke between the two groups (49% vs. 54%; p=0.47). Additionally, there were no differences between these two inodilators in the rates of the individual elements of the composite endpoint [15].

ROLE OF MECHANICAL CIRCULATORY SUPPORT (MCS)

For patients with AMI-CS refractory to vasopressors, temporary MCS devices can improve hemodynamics by restoring systemic perfusion [16]. The early initiation of MCS in AMI-CS unloads the left ventricle, increases systemic perfusion, enhances coronary myocardial perfusion, and provides hemodynamic support, which can prevent the progression of shock to end-organ injury and death [5]. The first documented clinical application of MCS was in 1967, where an IABP was successfully used in the treatment of a 45-year-old female patient who had sustained a MI and had severe CS [17]. Temporary MCS has been increasingly used in patients with AMI-CS, particularly devices that can provide greater support (i.e., higher cardiac output) including peripheral VADs (pVADs), such as the Impella 5.5® and TandemHeart®. Meanwhile, VA-ECMO can provide complete cardiopulmonary support [18]. However, in an analysis of clinical practice patterns of MCS from North American centers, the most common temporary MCS device was IABP (70%), which provides only modest support, while other MCS options were less common [Impella® (16%), VA-ECMO (11%)] [19]. Notably, IABP use varied widely between centers, ranging from 40% to 100%.

TEMPORARY MECHANICAL CIRCULATORY SUPPORT DEVICES (MCS)

Temporary MCS is summarized in Table 2, and options include the following:

Table 2 Types of temporary mechanical cardiac support devices

MCS device	IABP	Tandem heart®	Impella 2.5/CP/5.5®	VA-ECMO
Mechanism	Pulsatile	Centrifugal (continuous)	Axial (continuous)	Centrifugal (continuous)
CO (L/min)	0.5–1.0	4.0–5.0	2.5/3.0–4.0/5.0	4.0–10.0
Size	7–8 Fr	Arterial: 15–19 Fr; Venous: 21 Fr	12–14 Fr	Arterial: 14–19 Fr
Hemodynamic effects	LV pressure or volume unloading	LV volume unloading	LV pressure or volume unloading	Biventricular pressure and volume unloading
Advantages	Readily available Bedside insertion Easy to adjust No anticoagulation No extracorporeal blood	Addition of pulmonary support	Direct ventricular unloading Independent of rhythm Easy insertion No extracorporeal blood	Independent of rhythm Robust CO support Pulmonary support
Disadvantages	Minimal hemodynamic support Requires stable rhythm No effect on mean BP or lactate	Immobilization Difficult insertion Requires transseptal puncture Vascular complications	Mandatory anticoagulation; Hemolysis; Vascular complications	Incomplete LV unloading Vascular complications Regional hypoxemia
Contraindications	Severe PAD; AAA; significant AI	VSD; significant AI; left atrial thrombus	LV thrombus; mechanical AV; severe PAD	Severe PAD; significant AI; aortic dissection

AAA abdominal aortic aneurysm, *AI* aortic insufficiency, *AV* aortic valve, *BP* blood pressure, *CO* cardiac output, *ECMO* extracorporeal membrane oxygenation, *IABP* intra-aortic balloon pump, *LV* left ventricle, *MCS* mechanical cardiac support, *PAD* peripheral arterial disease, *VA* venoarterial, *VSD* ventricular septal defect

Intra-Aortic Balloon Pump (IABP)

The IABP consists of an expandable balloon mounted on a vascular 7F to 8F catheter and is most commonly introduced via the femoral artery and positioned in the descending thoracic aorta distal to the left subclavian artery. Depending on factors such as the balloon size (which varies according to the patient's height), positioning, and vascular compliance, the cardiac output provided is up to 1 L/min via the femoral approach. However, it may also be surgically placed via an axillary artery conduit if needed due to patient anatomy, which allows greater augmentation of cardiac output and mobility of the patient—this approach has gained favor to support patients with end-stage heart failure who are awaiting a durable LV assist device or heart transplant. The IABP is timed to inflate and deflate synchronously with the cardiac cycle, increasing the diastolic blood pressure and reducing the systolic blood pressure to improve coronary perfusion and cardiac output [5]. More specifically, the IABP acts passively by inflating during diastole and then deflating prior to systole. When inflated, there is an increase in aortic pressure (referred to as

"augmented diastolic pressure") that results in greater coronary blood flow. Then when the balloon deflates just prior to the onset of systole, there is an abrupt lowering of aortic pressure that results in a reduction in work for the LV during systole [20]. However, the IABP might cause a drop in mean arterial pressure, requiring the addition of vasopressor agents. Thus, IABP can be used to augment coronary and systemic perfusion but it may not provide enough support as a standalone MCS device. Some registry studies have reported no short- or long-term improvement in cardiac output or hemodynamic parameters with IABP use [8, 21]. Contraindications to IABP include moderate to severe aortic regurgitation, uncontrolled sepsis, uncontrolled bleeding diathesis, aortic aneurysm, aortic dissection, and severe peripheral artery disease [22].

In the large randomized multicenter IABP-SHOCK II trial, which randomized 600 patients with AMI-CS and early revascularization to IABP or medical therapy, IABP was not associated with any short-term mortality benefit [23]. Furthermore, the 1-year and 6-year data demonstrated no benefit of IABP on long-term outcomes (Fig. 1) [21, 22].

Percutaneous Ventricular Assist Devices (pVADs): Impella 2.5/CP/5.5®, TandemHeart®, and Magenta Elevate

Currently available pVADs include the TandemHeart® (Cardiac Assist, Inc, Pittsburgh, PA) and the micro-axial Impella® (Abiomed Europe, Aachen, Germany). With the aid of fluoroscopic guidance, a transseptal puncture is required between the right and left atria via the femoral vein for placement of TandemHeart®. TandemHeart® bypasses the LV by taking arterialized blood from the left atrium from a catheter that crosses the atrial septum and circulated through an external centrifugal pump, and diverts it to the lower abdominal aorta or iliac arteries via a femoral artery cannula (15–17 Fr) with retrograde perfusion of the abdominal and thoracic aorta allowing for blood flow rates of up to 4 L/min [5].

Contraindications for pVADs include significant peripheral artery disease, moderate-to-severe aortic valve insufficiency, mechanical tricuspid or pulmonary valve, severe tricuspid valve stenosis, severe pulmonary valve stenosis or insufficiency, thrombosis in the vena cava

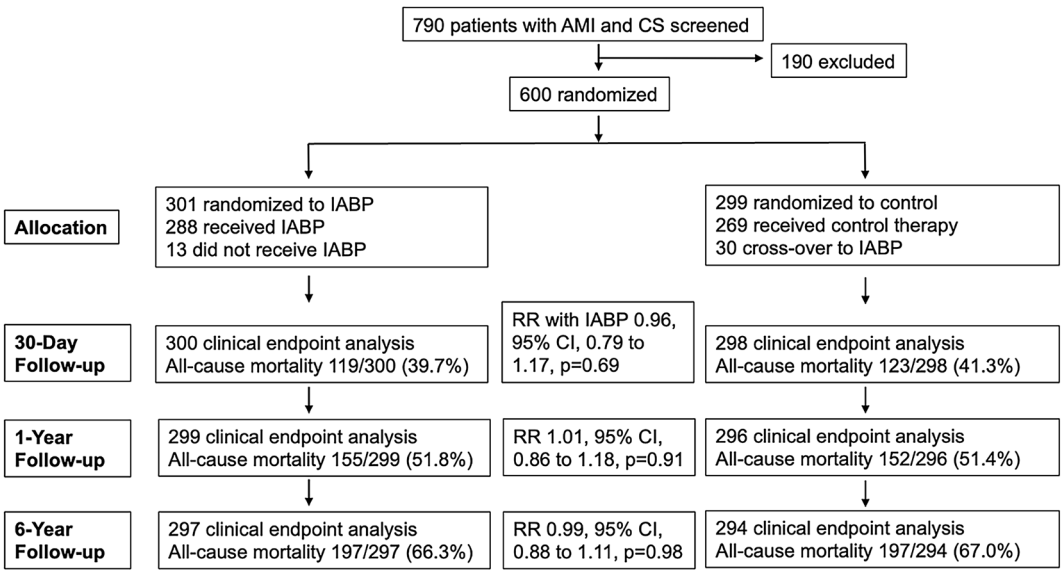

Fig. 1 IABP SHOCK Trial II results. Screening, randomization, management strategy, and follow-up at 30 days, 1 year, and 6 years. *AMI* acute myocardial infarction, *CI* confidence interval, *CS* cardiogenic shock, *IABP* intra-aortic balloon pump, *RR* relative risk. Permission was obtained to adapt this figure from references 20, 21, and 22

or right atrium or ventricle, and superior vena cava or internal jugular vein stenosis or occlusion [24]. Patients with a TandemHeart are more likely to experience vascular complications such as significant bleeding. This may be linked to the requirement for transseptal puncture, having two sites for percutaneous vascular access, the large sheath needed for insertion, and the necessity for anticoagulation to mitigate thromboembolism risk [25].

A small study of 42 patients that compared TandemHeart® to IABP in patients with CS found that patients randomized to TandemHeart® had a significantly improved cardiac index and pulmonary capillary wedge pressure, with no change in clinical outcomes, but with a higher frequency of complication including severe bleeding ($n=19$ vs. $n=8$, $p=0.002$) and limb ischemia ($n=7$ vs. $n=0$, $p=0.009$) with TandemHeart® support [26].

Currently, three classes of Impella® LVAD support are available in the US: 2.5, cardiac power (CP), and 5.5. These three classes differ in the peak flows offered for cardiac output, as 2.5 provides flows up to 2.5 L/min, CP provides flows up to 4.3 L/min, and 5.5 provides flows up to 6 L/min. The Impella 2.5® is now rarely used given the availability of the Impella CP® and 5.5 that offer higher flow rates. The Impella Expandable Cardiac Power® (ECP) is a smaller version of the Impella CP® that provides up to 5.5 L/min support, but is not approved by the US Food and Drug Administration for clinical use. It has a 21 Fr pump which is compressible to 9 Fr that is designed to be implanted and removed using small bore access and closure techniques. It is delivered wirelessly across the aortic valve into the LV [27]. The Impella® devices support hemodynamics by increasing cardiac output and unloading the heart, reducing LV end-diastolic pressure, thereby improving coronary flow and myocardial perfusion, and decreasing myocardial oxygen demand. To avoid malfunction and device-related complications, it is critical that the Impella® device be placed in the proper position and monitored with ultrasound. Importantly, the inlet area of the catheter should be well below the aortic outflow tract (approximately 3.5 cm in adults), free of the papillary muscle and mitral valve apparatus, in a free-floating position. In addition, adequate right ventricular function and filling pressures are necessary to avoid device suctioning [20]. Improper rotation of the device has been linked to poorer outcomes. Both the Impella®support devices depend on adequate right ventricular function to maintain sufficient LV preload and require stable oxygenation for optimal performance. Contraindications include the presence of left ventricular thrombus and mechanical aortic valves [8]. It is important to note that for axillary devices, insertion of a perfusion catheter distally can help to mitigate distal limb complications. More specifically, this is done using ultrasound-guided antegrade access with a 5- or 6-F braided sheath [28].

Data comparing different types of percutaneous MCS devices for CS are sparse. A meta-analysis from 2009 combined data from three randomized trials (two involving TandemHeart® and one with Impella 2.5®, each compared to IABP). Patients receiving percutaneous MCS exhibited higher cardiac indices, elevated mean arterial pressures, lower pulmonary capillary wedge pressures, and a higher incidence of bleeding complications, but no significant difference in mortality rates between MCS devices versus IABP [29]. Two small studies with < 50 patients with CS showed that Impella 2.5® improved cardiac index compared to IABP, but there were no differences in mortality at 1 or 6 months [30, 31]. The Impella®-EUROSHOCK, a European registry comprised of 120 patients enrolled between 2005 and 2010 who had Impella 2.5® for CS, showed that the device improved hemodynamics compared to no MCS support in this high-risk cohort [32]. The available data suggest Impella® devices offer reasonable balance between greater hemodynamic support and ease of use; however, randomized data comparing various types of MCS in CS are limited.

For patients whose hemodynamics remain stable or improve, assessing readiness to wean from MCS should be pursued. The wean allows for evaluation of whether the heart can provide the necessary cardiac output to match demand. If hemodynamic, metabolic, and end-organ perfusion instability or ventricular dysfunction ensues during the wean, device

settings should resume at the prior level of support before a further weaning attempt. If stability is maintained at the lowest device support, explantation of the device can be considered. However, if a patient's clinical condition deteriorates (e.g., low cardiac indices or requirement of multiple vasoactive medications), the use of higher forms of support such as Impella 5.5® or VA-ECMO should be considered [16]. The Danish Cardiogenic Shock Trial (DanGerShock) was a multicenter study of 355 patients with CS following STEMI that were randomized to receive conventional circulatory support with Impella CP® with standard of care or standard of care alone. The study demonstrated a significant reduction in mortality with the routine use of MCS in patients with CS following STEMI [33]. Further details are discussed in a later section.

A first-in-human feasibility study with a small peripheral LVAD known as the Magenta Elevate demonstrated initial safety and feasibility of this device in 14 patients undergoing high-risk PCI. This self-expanding, catheter-mounted pump is inserted through a 10-F femoral catheter and can provide 5.4 L/min flow [34].

Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO)

VA-ECMO provides both cardiovascular and pulmonary support in acute cardiorespiratory failure and has also been used to assist cardiopulmonary resuscitation in cardiac arrest [9]. For CS, VA-ECMO is most commonly used, since isolated right ventricular failure (for which veno-venous [VV]-ECMO could be considered) due to AMI is rare. VA-ECMO supports both ventricles and lung function, while VV-ECMO supports lung function and the right ventricle only.

In VA-ECMO, blood is removed from the right atrium (V), then circulated through a heat exchanger and membrane oxygenator (ECMO), before returning oxygenated blood to the femoral artery (A). This greatly enhances the flow of oxygenated blood to the body's extremities. Peripheral VA-ECMO can be initiated percutaneously using ultrasound guidance, typically via the femoral vein and artery. Alternatively, it can be established through surgical means. With VA-ECMO, deoxygenated (venous) blood is withdrawn from either a peripheral (e.g., femoral) or central (e.g., superior vena cava) vein, oxygenated in an external circuit, and returned through an arterial cannula. With peripheral V-A ECMO, to avoid leg ischemia due to a large bore arterial catheter in the femoral artery, an antegrade perfusion catheter is advised [20]. Absolute contraindications to VA-ECMO include limited life expectancy (<1 year); irreversible neurologic injury; severe peripheral arterial disease or other limitation to vascular access with large bore catheters; respiratory failure for>1 week requiring very high levels of inhaled oxygen or high-pressure ventilation; advanced liver disease; and severe coagulopathy, contraindication to systemic anticoagulation, or refusal to receive blood products. Relative contraindications to VA-ECMO include advanced age, cognitive impairment, severe non-cardiac comorbid conditions, poor adherence to treatment, and insufficient social support [24].

A meta-analysis of five randomized trials in 567 patients with CS complicating AMI compared early, routine VA-ECMO with optimal medical therapy. There was no significant difference in 30-day mortality between treatments (OR for VA-ECMO vs. optimal medical therapy 0.93, 95% CI 0.66–1.29). However, VA-ECMO was associated with significantly higher complication rates, including major bleeding (OR 2.44 [1.55, 3.84]) and peripheral ischemic vascular complications (OR 3.53 [1.70, 7.34]) [33].

GUIDELINES AND EXPERT STATEMENTS

Early management of AMI-CS should consist of coronary revascularization (if appropriate), diuretics or renal replacement therapy for treatment of congestion or volume overload, and the use of vasoactive agents to support the mean arterial pressure and cardiac output [5]. MCS may be a reasonable option in patients with CS shock who do not respond to initial medical therapy or have contraindications to inotropes (e.g., refractory arrhythmias), and who have ongoing end-organ signs of hypoperfusion or worsening hemodynamics. Criteria for severe CS include

(1) persistent cardiac index < 2.2 L/min/m^2; (2) low cardiac power output < 0.6 W; (3) persistent hypotension (mean arterial pressure < 65 mm Hg); (4) failure to adequately clear lactate with levels consistently > 2 mmol/L); (5) right ventricular failure confirmed by hemodynamic measurements (e.g., right atrial to pulmonary capillary wedge pressure ratio > 0.86, a pulmonary artery pulsatility index [PAPi] ≤ 0.9 calculated as [PA systolic minus PA diastolic pressure]/RA pressure; (6) ongoing severe hypoxemia (e.g., PaO_2:FiO_2 ratio < 200 mm Hg); or (7) recurrent sustained ventricular arrhythmias [16]. Early initiation of MCS is recommended with the goal of limiting the potential adverse consequences of escalating doses of vasoactive medications and preventing further decline of end-organ function [5]. The selection MCS device should be based on device availability, multidisciplinary team familiarity, and patient-specific factors. Temporary MCS should be considered over durable devices as the first-line approach in the following circumstances: (1) immediate stabilization is needed to enable recovery of the heart and other organ systems, (2) risk of coronary revascularization is prohibitory, (3) ventricular support is needed to facilitate a definitive intervention, or (4) when additional time is necessary to permit an evaluation for cardiac transplantation or durable MCS [36]. The strategy for the use of MCS is described in professional society guidelines in Table 3.

Table 3 Key guideline recommendations for temporary mechanical cardiac support in cardiogenic shock complicating acute myocardial infarction

Recommendation	Organization	Year	Class of recommendation	Level of evidence
Short-term mechanical support, including ECMO, should be used in acutely decompensated patients who fail to respond to maximal medical therapy. [40]	ISHLT	2013	I	C
The use of temporary MCS should be strongly considered in patients with multiorgan failure, sepsis, or on mechanical ventilation to allow successful optimization of clinical status and neurological assessment prior to placement of a long-term MCS device. [40]	ISHLT	2013	I	C
In select patients with AMI-CS, short-term MCS may be considered, depending on patient age, comorbidities, neurological function, and the prospects for long-term survival and predicted quality of life. [41]	ESC	2016	IIa	C
IABP can be useful for patients with AMI-CS who do not quickly stabilize with pharmacological therapy. [37]	ACC/AHA	2013	IIa	B
IABP insertion should be considered in patients with hemodynamics due to mechanical complications. [41]	ESC	2016	IIa	C
pVADs for circulatory support may be considered in patients with refractory CS. [37]	ACC/AHA	2013	IIb	C
Routine use of IABP in patients with CS and no mechanical complications due to MI is not recommended. [41]	ESC	2016	III	B

ACC/AHA American College of Cardiology/American Heart Association, *AMI-CS* acute myocardial infarction-related cardiogenic shock, *ECMO* extracorporeal membrane oxygenation, *ESC* European Society of Cardiology, *IABP* Intra-aortic balloon pump, *ISHLT* International Society of Heart and Lung Transplantation, *MCS* mechanical cardiac support, *pVAD* percutaneous ventricular assist device. Level of evidence: B = limited populations evaluated and/or data derived from a single randomized trial or nonrandomized studies; C = Very limited populations evaluated and/or only consensus opinion of experts, case studies, or standard of care

Briefly, the 2013 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guidelines recommend a stepwise strategy of treatment for patients with AMI-CS, starting with vasoactive medications, and followed by implantation of percutaneous MCS devices if the initial medical therapy does not improve hemodynamics [37]. Patients with AMI-CS who have spontaneous circulation should be transported to the cardiac catheterization lab at a percutaneous coronary intervention (PCI)-capable hospital as quickly as possible [38]. Similarly, the 2021 European Society of Cardiology (ESC) position statement did not recommend MCS as the first-line treatment for CS [39]. The International Society of Heart and Lung Transplantation (ISHLT) 2013 guidelines recommend short-term MCS for acutely decompensated patients refractory to medical treatment [40]. Routine use of IABP for patients with CS in the setting of ST-segment elevation myocardial infarction (STEMI) was formerly a class 1 recommendation, but the 2013 AHA/ACC and 2014 ESC guidelines were revised to a class IIa due to the results of the IABP-SHOCK II trial results (see above) [41]. The use of MCS devices may be considered (class IIb, for TandemHeart® or Impella®) for patients with AMI-CS refractory to medications [36]. The 2017 ESC guidelines do not recommend the routine use of an IABP in patients with AMI-CS (class III, level of evidence B), but provide a class IIa level-of-evidence C recommendation for use in patients with AMI-CS and mechanical complications [42].

MCS DEVICE COMPLICATIONS

Potential complications of MCS devices should be balanced against their hemodynamic and clinical benefits. Complications can arise from either direct cardiac or vascular damage [24]. Rarely, cardiac complications such as valve injury or perforation of the left ventricle may occur, which can lead to life-threatening issues including intracardiac shunting, pericardial effusion, and cardiac tamponade. Vascular complications may involve distal limb ischemia and dissection of the cannulated vessels [43]; thus, careful management of distal limb perfusion is necessary, particularly when large femoral arterial cannulas are used [44].

Hematological complications from MCS devices are not uncommon and include hemorrhage, anemia, platelet dysfunction, thrombocytopenia, and thrombosis. In addition, MCS, and in particular ECMO, can lead to complications such as increased bleeding risk, device-related thrombosis, and thromboembolic events. Infectious complications may also occur with MCS due to multiple external cannulation sites, critical illness, and extended hospital stays [43].

Neurological damage may result from the migration of microemboli within the MCS device. Patients requiring MCS, who are often hemodynamically unstable, may experience episodes of cerebral hypoperfusion, hypoxia, or metabolic disturbances, which can lead to stroke (either thromboembolic or hemorrhagic) and neurological injury. While the stroke rate is comparable among patients using IABP, TandemHeart®, or Impella® devices, rates of stroke (both thromboembolic and hemorrhagic) are significantly higher in patients on ECMO [43].

UPDATES OF RECENT AND CURRENT TRIALS AND REGISTRIES

The key findings from several recently completed studies published after the aforementioned guidelines are summarized in Table 4 and detailed as follows:

Recently Completed Trials

The Extracorporeal Life Support in Infarct-Related Cardiogenic Shock trial (ECLS-Shock; NCT03637205) was a multicenter trial consisting of 420 patients with AMI-CS that compared VA-ECMO with medical treatment alone and found that there was no statistically significant difference between the two groups in 30-day mortality. However, VA-ECMO had higher rates of moderate to severe bleeding (23.4% vs. 9.6%,

Table 4 Recent studies on temporary mechanical cardiac support

Trial/registry	Participants	Patient cohort	Intervention	Design	Primary endpoint	Results
ECLS-SHOCK (NCT03637205) [45]	420	MI with severe CS	Peripheral ECMO + PCI/CABG vs. PCI/CABG	RCT	Mortality at 6 months	No mortality difference and associated with increased complications
ECMO-CS (NCT02301819) [46]	122	Rapidly deteriorating or severe CS	Immediate ECMO vs. conservative treatment (delayed ECMO if needed)	RCT	Mortality, cardiac arrest, or additional MCS at 1 month	No differences in mortality, cardiac arrest, or additional MCS between groups
DanGer Shock (NCT01633502) [33]	360	STEMI with CS	Impella CP® vs. standard of care	RCT	Mortality at 6 months	Mortality reduction with use of MCS in patients with CS

CABG coronary artery bypass grafting, *CS* cardiogenic shock, *ECMO* extracorporeal membrane oxygenation, *MCS* mechanical circulatory support, *MI* myocardial infarction, *PCI* percutaneous coronary intervention, *RCT* randomized controlled trial, *STEMI* ST-segment elevation myocardial infarction

RR 2.44 [1.50 to 3.95]) and peripheral vascular complications requiring intervention (11.0% vs. 3.8%, RR 2.86 [1.31, 6.25]) [45].

The Extracorporeal Membrane Oxygenation in the Therapy of Cardiogenic Shock trial (ECMO-CS; NCT02301819) was a multicenter, randomized clinical trial that included 117 patients with either rapidly deteriorating or severe CS and compared immediate implementation of VA-ECMO versus no immediate VA-ECMO [46]. There was no significant difference in the primary composite endpoint of all-cause mortality, resuscitation from circulatory arrest, or use of another type mechanical circulatory support device (which included VA-ECMO in the no immediate use group) at 30 days, in patients randomized to immediate versus no immediate VA-ECMO (63.8% vs. 71.2%; $p = 0.21$). No significant differences were observed in the individual rates of resuscitated cardiac arrest or all-cause mortality; however, the use of another MCS device was significantly reduced by 62% in patients randomized to immediate VA-ECMO (17.2% vs. 42.4%). No significant differences were observed in the rates of serious adverse events (including sepsis, pneumonia, stroke, bleeding, and leg ischemia) at 30 days between groups. Overall, immediate use of VA-ECMO in patients with rapidly worsening or severe CS post-AMI did not show any clinical benefit over an initial conservative approach.

The Danish Cardiogenic Shock Trial (DanGerShock; NCT 01633502) was a multicenter study of 355 patients with CS following STEMI that were randomized to receive conventional circulatory support with Impella CP® with standard of care or standard of care alone. [33] The primary endpoint of death from any cause at 180 days was significantly reduced by 26% [1–45%] from 58.5% in the standard-care group to 45.8% in the Impella®

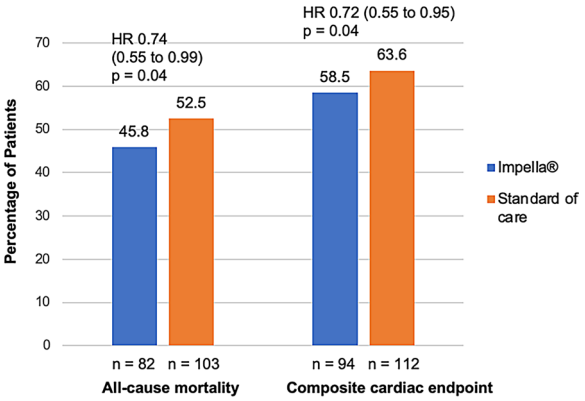

Fig. 2 DanGer Shock Results at 180 Days. Blue represents Impella® + standard of care group and orange represents standard of care group. Comparison of primary (left) and secondary (right) endpoints at 180 days. Primary endpoint is all-cause mortality, and composite cardiac endpoint includes escalation of treatment to additional MCS (short- or long-term), heart transplantation, or death from any cause, whichever came first. Figure was created from data from Ref. [33]. *HR* hazard ratio, *MCS* mechanical circulatory support

group ($p = 0.04$) (Fig. 2). The two secondary endpoints were (1) a cardiovascular composite endpoint consisting of escalation of treatment to additional MCS, heart transplantation, or death from any cause, and (2) days alive and out of the hospital. The composite cardiovascular endpoint event was significantly reduced in the Impella® versus standard-care group 63.6% to 52.5% (HR, 0.72 [0.55, 0.95]). However, the mean number of days alive and out of the hospital were not different between groups (82 vs. 73 days). The composite safety endpoint of severe bleeding, limb ischemia, hemolysis, device failure, or worsening aortic regurgitation event occurred significantly more often in the Impella® group (24.0% vs. 6.2%); RR 4.74 [2.36 to 9.55]). Furthermore, the need for renal replacement therapy was significantly increased in the Impella group® (41.9% vs. 26.7%; RR 1.98 [1.27 to 3.09]). To date, this is the first randomized trial to demonstrate a significant mortality reduction with the routine use of MCS in patients with CS following STEMI [33].

Ongoing Trials and Registries

Currently ongoing trials and registries are summarized in Table 5.

Established in 2017 with 16 medical centers, the Critical Care Cardiology Trial Network (CCCTN) has expanded to a research network

Table 5 Ongoing registries on temporary mechanical cardiac support

Trial/registry	Participants	Patient cohort	Intervention	Design	Primary endpoint	Results
Altshock-2 (NCT04369573) [48]	200	ADHF with CS	Early IABP vs. vasoactive medications	RCT	Survival or heart transplant at 2 months	Results expected in 2025
PROTECT IV (NCT04763200) [49]	1252	CAD with LV dysfunction	Impella 2.5® or CP® vs. standard of care	RCT	Mortality at 3 years	Results expected in October 2027
Cardiogenic Shock Working Group Registry (CSWG) (NCT04682483) [50]	5000	CS	MCS or vasoactive medications	Observational	Mortality at 1 month	Results expected in June 2025

ADHF acute decompensated heart failure, *CAD* coronary artery disease, *CS* cardiogenic shock, *IABP* intra-aortic balloon pump, *LV* left ventricular, *MCS* mechanical circulatory support, *RCT* randomized controlled trial

comprising 49 academic and clinical centers in North America and Europe. The registry now includes >35,000 unique admissions to cardiac intensive care units (CICUs) and is currently in its eighth annual cycle. It captures data on variations in care, epidemiology, and outcomes for CICU patients, covering specific conditions such as shock, heart failure, renal dysfunction, and respiratory failure. Additionally, the CCCTN has examined patterns of care utilization, including the use of MCS in response to changes in heart transplantation allocation and the impact of multidisciplinary shock teams. The CCCTN has developed a comprehensive research network to support multicenter registry-based randomized controlled trials for patients with severe cardiovascular conditions [47].

Between September 2017 and September 2018, 16 centers each provided a 2-month snapshot of consecutive CICU admissions. Out of 270 admissions involving temporary MCS, 33% were for AMI-CS. Among the 585 admissions with CS or mixed shock, 34% utilized temporary MCS during their CICU stay, with usage varying significantly between centers (ranging from 17% to 50%). The most frequently used MCS devices were IABP (72%), followed by Impella (17%) and VA-ECMO (11%), although IABP use varied widely across centers (40–100%). Patients treated with IABP, compared to those with more advanced MCS devices, had lower Sequential Organ Failure Assessment (SOFA) scores and less severe metabolic issues [19].

The Altshock-2 (NCT04369573) trial is enrolling 200 patients with acute decompensated heart failure-cardiogenic shock (ADHF-CS) who are randomized to receive early IABP versus vasoactive medications without MCS. The study aims to determine whether early IABP implantation improves clinical outcomes in patients with ADHF-CS using a PROBE (**P**rospective, **R**andomized, **O**pen-label study with **B**linded **E**ndpoint adjudication) design. Enrollment began in May 2020, and is expected to conclude in April 2025. The primary outcome is patient survival or successful support to heart transplantation within 60 days. Secondary outcomes include 60-day survival, the need for renal replacement therapy, the maximum in-hospital inotropic score, the duration of inotropic/vasopressor therapy, and the maximum sequential organ failure assessment score. Safety endpoints include the occurrence of bleeding events, vascular access complications, and systemic embolism during hospitalization [48].

PROTECT IV (Impella®-Supported PCI in High-Risk Patients With Complex Coronary Artery Disease and Reduced Left Ventricular Function; NCT04763200) is randomized controlled trial in 1252 patients with left ventricular dysfunction undergoing high-risk PCI that is comparing the Impella CP® or Impella 2.5® device to standard of care without MCS. Cardiogenic shock is an exclusion criterion for the trial. The trial began enrollment in April 2021 and is expected to run until October 2027. The primary outcome is a composite measure of all-cause mortality, stroke, MI, unplanned clinically driven revascularization, durable LVAD implantation or heart transplant, or other cardiovascular hospitalizations over 3 years [49].

The Cardiogenic Shock Working Group Registry (CSWG, NCT04682483) is a multicenter registry whereby clinical variables are collected from multiple institutions and includes a retrospective arm to collect data during a hospital stay and a prospective arm for assessment of long-term outcomes. The registry plans to enroll approximately 5000 patients between December 2017 and June 2025 [50]. In a recent study of data from this registry using the CSWG-modified Society for Cardiovascular Angiography (CSWG-SCAI) staging system, the authors sought to risk-stratify the severity of CS and examine clinical changes and outcomes at 24, 48. and 72 h. The study involved 3268 patients (average age 60.3 years; 70.3% male), and among them, 27% had AMI-CS while 57% had CS due to heart failure (HF-CS). At the time of presentation, most patients (50.8%) were in CS stage D, followed by 18.1% in stage B, 16.2% in stage C, and 14.9% in stage E [51].

Within the first 24 h, most patients transitioned to a different CSWG-SCAI stage, with stages B and C showing the most movement (54.5% and 50.9%, respectively) worsening to a more severe stage. After 24 h, the majority of surviving patients (70.6%) were in stage D. The highest

mortality rates occurred in patients who had either remained in or moved to stage E at 24 h: 71.4% for those transitioning from stage B, 80% from stage C, 62.2% from stage D, and 59.7% for those staying in stage E [51]. Mortality rates and the use of temporary MCS were similar in stage E for patients with or without cardiac arrest, although those with cardiac arrest required fewer vasoactive medications. The authors underscored the importance of early CS diagnosis, stage classification, and identification of predictors of mortality in patients with CS [51].

CONCLUSION

MCS offers a potentially lifesaving intervention for patients with AMI-CS that is refractory to medical therapy. Several options exist for temporary MCS of the left ventricle in CS to provide a bridge to recovery, coronary revascularization, or heart transplantation. However, patients with AMI-CS comprise a heterogeneous population; therefore, the selection of MCS device type depends on the severity of impaired cardiac output, urgency of initiation, and anatomic considerations. Current use of MCS is guided by expert consensus and professional society statements and guidelines, but there are limited data to make recommendations based on randomized controlled trials. Ongoing clinical trials and population-based registries will provide further evidence to inform clinical practice regarding the use of MCS in AMI-CS.

Author Contributions. Project Conceptualization—Duc M. Giao, Robert P. Giugliano; Drafting—Duc M. Giao; Revision—Duc M. Giao, Robert P. Giugliano; Supervision—Robert P. Giugliano.

Funding. No funding or sponsorship was received for publication of this article.

Data Availability. Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Declarations

Conflict of Interest. Robert P. Giugliano is a member of the TIMI Study Group of the Brigham and Women's Hospital, which has received research grants to support clinical trials from Amgen, Anthos Therapeutics, Daiichi Sankyo, and Ionis; and has received honoraria for CME program, lectures and/or consulting from Amgen, Artivion, Beckman Coulter, Daiichi Sankyo, Dr. Reddy's Laboratories, Gilead, Inari, Inventiva, Medical Education Resources, Menarini, PhaseBio, SAJA Pharmaceutics, Samsung, Sanofi Aventis, Servier, and Summeet. Robert P Giugliano was previously an Editor-in-Chief of *Cardiology and Therapy*. Robert P Giugliano was not involved in the selection of peer reviewers for the manuscript nor any of the subsequent editorial decisions. Duc M. Giao has no disclosures.

Ethical Approval. This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

REFERENCES

1. Berg DD, et al. Epidemiology of shock in contemporary cardiac intensive care units. Circ Cardiovasc Qual Outcomes. 2019;12:3.
2. Samsky MD, et al. Cardiogenic shock after acute myocardial infarction: a review. JAMA. 2021;326(18):1840–50.
3. Lim HS, Howell N. Cardiogenic shock due to end-stage heart failure and acute myocardial infarction: characteristics and outcome of temporary mechanical circulatory support. Shock. 2018;50(2):167–72.
4. Baran DA, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. Catheter Cardiovasc Interv. 2019;94(1):29–37.
5. Fernando SM, et al. Mechanical circulatory support in the treatment of cardiogenic shock. Curr Opin Crit Care. 2022;28(4):434–41.
6. Atti V, et al. Comprehensive review of mechanical circulatory support devices. Heart Int. 2022;16(1):37–48.
7. Telukuntla KS, Estep JD. Acute mechanical circulatory support for cardiogenic shock. Methodist Debakey Cardiovasc J. 2020;16:27–35.
8. Ramzy D, Soltesz E, Anderson M. New surgical circulatory support system outcomes. Asaio j. 2020;66(7):746–52.
9. Keebler ME, et al. Venoarterial extracorporeal membrane oxygenation in cardiogenic shock. JACC Heart Fail. 2018;6(6):503–16.
10. Shankar A, et al. A clinical update on vasoactive medication in the management of cardiogenic shock. Clin Med Insights Cardiol. 2022;16:11795468221075064.
11. Jentzer JC, et al. Temporal trends and clinical outcomes associated with vasopressor and inotrope use in the cardiac intensive care unit. Shock. 2020;53:452–9.
12. Tehrani BN, et al. A standardized and comprehensive approach to the management of cardiogenic shock. JACC Heart Fail. 2020;8(11):879–91.
13. Levy B, Buzon J, Kimmoun A. Inotropes and vasopressors use in cardiogenic shock: when, which and how much? Curr Opin Crit Care. 2019;25(4):384–90.
14. De Backer D, et al. Comparison of dopamine and norepinephrine in the treatment of shock. N Engl J Med. 2010;362(9):779–89.
15. Mathew R, et al. Milrinone as compared with dobutamine in the treatment of cardiogenic shock. N Engl J Med. 2021;385(6):516–25.
16. Geller BJ, et al. Escalating and de-escalating temporary mechanical circulatory support in cardiogenic shock: a scientific statement from the American Heart Association. Circulation. 2022;146(6):e50–68.
17. Parissis H, et al. IABP: history-evolution-pathophysiology-indications: what we need to know. J Cardiothorac Surg. 2016;11(1):122.
18. Vallabhajosyula S, et al. Ten-year trends, predictors and outcomes of mechanical circulatory support in percutaneous coronary intervention for acute myocardial infarction with cardiogenic shock. EuroIntervention. 2021;16(15):e1254–61.
19. Berg DD, et al. Clinical practice patterns in temporary mechanical circulatory support for shock in the critical care cardiology trials network (CCCTN) registry. Circ Heart Fail. 2019;12(11):e006635.
20. Møller JE, et al. Mechanical circulatory support: when, how, and for whom. Eur Heart J. 2025;10:925. https://doi.org/10.1093/eurheartj/ehae925.
21. Thiele H, et al. Intraaortic balloon pump in cardiogenic shock complicating acute myocardial infarction: long-term 6-year outcome of the randomized IABP-SHOCK II trial. Circulation. 2019;139(3):395–403.
22. Thiele H, et al. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. Lancet. 2013;382(9905):1638–45.
23. Thiele H, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med. 2012;367(14):1287–96.
24. Salter BS, et al. Temporary mechanical circulatory support devices: practical considerations for all stakeholders. Nat Rev Cardiol. 2023;20(4):263–77.

25. Ali JM, Abu-Omar Y. Complications associated with mechanical circulatory support. Ann Transl Med. 2020;8(13):835.

26. Thiele H, et al. Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock. Eur Heart J. 2005;26(13):1276–83.

27. Kaki A. Mechanical circulatory support of high-risk PCI with a novel, low-profile pMCS: first results of the impella ECP pivotal study. TCT World Innovation Forum 2024, October 27. 2024. Washington D.C.

28. Damluji AA, et al. Position statement on vascular access safety for percutaneous devices in AMI complicated by cardiogenic shock. JACC Cardiovasc Interv. 2022;15(20):2003–19.

29. Thiele H, et al. Percutaneous short-term active mechanical support devices in cardiogenic shock: a systematic review and collaborative meta-analysis of randomized trials. Eur Heart J. 2017;38(47):3523–31.

30. Cheng JM, et al. Percutaneous left ventricular assist devices vs intra-aortic balloon pump counterpulsation for treatment of cardiogenic shock: a meta-analysis of controlled trials. Eur Heart J. 2009;30(17):2102–8.

31. Seyfarth M, et al. A randomized clinical trial to evaluate the safety and efficacy of a percutaneous left ventricular assist device versus intra-aortic balloon pumping for treatment of cardiogenic shock caused by myocardial infarction. J Am Coll Cardiol. 2008;52(19):1584–8.

32. Lauten A, et al. Percutaneous left-ventricular support with the Impella-2.5-assist device in acute cardiogenic shock: results of the Impella-EUROSHOCK-registry. Circ Heart Fail. 2013;6(1):23–30.

33. Møller JE, et al. Microaxial flow pump or standard care in infarct-related cardiogenic shock. N Engl J Med. 2024;390(15):1382–93.

34. Schwammenthal E, et al. High-output mechanical circulatory support in left main interventions: the magenta elevate high-risk PCI first-in-human study. JACC Cardiovasc Interv. 2024;17(24):2851–62.

35. Zeymer U, et al. Venoarterial extracorporeal membrane oxygenation in patients with infarct-related cardiogenic shock: an individual patient data meta-analysis of randomised trials. Lancet. 2023;402(10410):1338–46.

36. van Diepen S, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. Circulation. 2017;136(16):e232–68.

37. O'Gara PT, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;127(4):e362-425.

38. Henry TD, et al. Invasive management of acute myocardial infarction complicated by cardiogenic shock: a scientific statement from the American Heart Association. Circulation. 2021;143(15):e815–29.

39. Cormican DS, et al. Acute myocardial infarction complicated by cardiogenic shock: analysis of the position statement from the European Society of Cardiology Acute Cardiovascular Care Association, with perioperative implications. J Cardiothorac Vasc Anesth. 2021;35(10):3098–104.

40. Feldman D, et al. International Society for Heart and Lung Transplantation. The 2013 International Society for Heart and Lung Transplantation Guidelines for mechanical circulatory support: executive summary. J Heart Lung Transplant. 2013;32(2):157–87.

41. Ponikowski P, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2016;37(27):2129–200.

42. Collet JP, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021;42(14):1289–367.

43. Subramaniam AV, et al. Complications of temporary percutaneous mechanical circulatory support for cardiogenic shock: an appraisal of contemporary literature. Cardiol Ther. 2019;8(2):211–28.

44. Patton-Rivera K, et al. Using near-infrared reflectance spectroscopy (NIRS) to assess distal-limb perfusion on venoarterial (V-A) extracorporeal membrane oxygenation (ECMO) patients with femoral cannulation. Perfusion. 2018;33(8):618–23.

45. Thiele H, et al. Extracorporeal life support in infarct-related cardiogenic shock. N Engl J Med. 2023;389(14):1286–97.

46. Ostadal P, et al. Extracorporeal membrane oxygenation in the therapy of cardiogenic shock: results of the ECMO-CS randomized clinical trial. Circulation. 2023;147(6):454–64.

47. Metkus TS, et al. Critical care cardiology trials network (CCCTN): a cohort profile. Eur Heart J Qual Care Clin Outcomes. 2022;8(7):703–8.

48. Morici N, et al. Early intra-aortic balloon pump in acute decompensated heart failure complicated by cardiogenic shock: rationale and design of the randomized Altshock-2 trial. Am Heart J. 2021;233:39–47.

49. Impella®-Supported PCI in High-Risk Patients With Complex Coronary Artery Disease and Reduced Left Ventricular Function (PROTECT IV), NCT04763200. Clinicaltrials.gov. 2024. https://classic.clinicaltrials.gov/ct2/show/NCT04763200 (2024-02-24)

50. Cardiogenic Shock Working Group Registry, NCT04682483. Clinicaltrials.gov. 2024. https://clinicaltrials.gov/study/NCT04682483 (2024-02-24)

51. Ton VK, et al. Serial shock severity assessment within 72 hours after diagnosis: a cardiogenic shock working group report. J Am Coll Cardiol. 2024;84(11):978–90.

52. Estep JD. Percutaneous mechanical circulatory support technologies. In: Karimov J, Fukamachi K, Starling R, editors. Mechanical support for heart failure. Cham: Springer; 2020.