CRITICAL CARE

REVIEW

Clinical review: mechanical circulatory support for cardiogenic shock complicating acute myocardial infarction

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Abstract

Acute myocardial infarction is one of the 10 leading reasons for admission to adult critical care units. In-hospital mortality for this condition has remained static in recent years, and this is related primarily to the development of cardiogenic shock. Recent advances in reperfusion therapies have had little impact on the mortality of cardiogenic shock. This may be attributable to the underutilization of life support technology that may assist or completely supplant the patient's own cardiac output until adequate myocardial recovery is established or long-term therapy can be initiated. Clinicians working in the intensive care environment are increasingly likely to be exposed to these technologies. The purpose of this review is to outline the various techniques of mechanical circulatory support and discuss the latest evidence for their use in cardiogenic shock complicating acute myocardial infarction.

Introduction

The in-hospital mortality for acute myocardial infarction (AMI) is currently around 7% [1]. Death is related predominantly to the development of cardiogenic shock, which affects 5% to 10% of all cases of AMI and has a mortality rate of 50% to 90% [2,3]. Patients who develop cardiogenic shock frequently require critical care services, and AMI is one of the 10 leading causes for admission to adult critical care units [4]. Over the past three decades, revascularization therapy has revolutionized care for these patients. Recent studies support prompt percutaneous coronary intervention (PCI) when there is electrocardiographic evidence of an AMI [5], and if PCI is not available within 90 minutes, fibrinolysis should be delivered within 30 minutes [6,7].

Despite these developments, there has been little progress in reducing mortality from cardiogenic shock complicating an AMI [8]. Part of the reason for this is that impaired cardiac contractility may persist many hours after revascularization, an observation described as myocardial stunning [9]. Interventions that can assist or completely supplant the patient's own cardiac output may support these patients until the stunned myocardium recovers (bridge to recovery). Recovery can be predicted using peak serum creatinine kinase levels [10] or contrast echocardiography [11], but even when recovery does not occur, mechanical circulatory support may provide time to determine whether longer-term therapies are appropriate (bridge to decision). In this review, we will outline the various techniques of mechanical circulatory support and discuss the evidence for their use in cardiogenic shock complicating AMI.

Initial management

Effective treatment of cardiogenic shock begins with early recognition, prompt pharmacological intervention, and appropriate respiratory support. Cardiogenic shock is defined by evidence of tissue hypoperfusion, such as cool peripheries, oliguria, and elevated lactate, in the setting of cardiac dysfunction and adequate filling pressures (Table 1). Hemodynamic criteria include a systolic blood pressure of less than 90 mm Hg for more than 30 minutes, a cardiac index of less than 2.2 L/min per m^2, and a pulmonary artery occlusion pressure of greater than 15 mm Hg [12]. An in-depth review of pharmacological and respiratory support for cardiogenic shock is beyond the scope of this article and can be found elsewhere [13]. However, pharmacological interventions predominately involve inotropic support that may perpetuate ischaemia by increasing myocardial oxygen demand. Therefore, mechanical circulatory support should be considered early when inotropes have been initiated.

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Table 1. Cardiogenic shock criteria

Hemodynamic criteria
Systolic blood pressure (SBP) of less than 90 mm Hg for greater than 30 minutes
SBP drop of greater than 30 mm Hg below basal for greater than 30 minutes in patients with hypertension
Use of vasopressors and inotropes to keep SBP greater than 90 mm Hg
Cardiac index of less than 2.2 L/min per m^2
Pulmonary artery occlusion pressure of greater than 15 mm Hg
Signs of tissue hypoperfusion
Pale, cool, and clammy peripheries
Prolonged capillary refill times
Altered mental status/confusion
Oliguria
Pulmonary congestion
Tachycardia
Elevated lactate
Mixed venous saturation of less than 65%

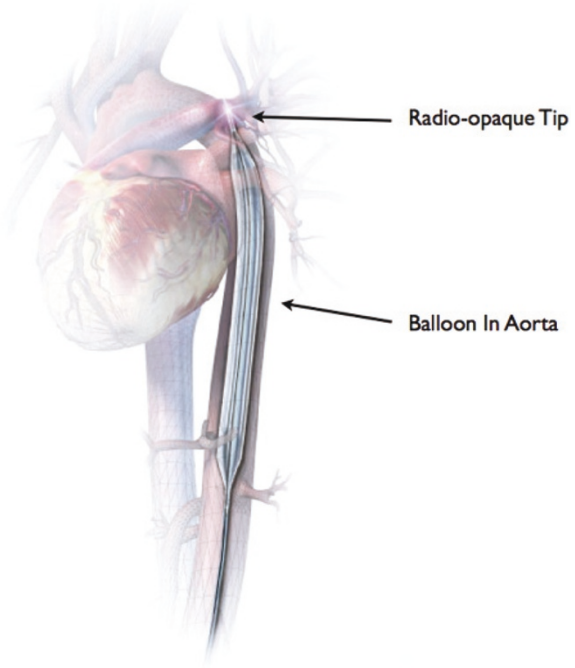

Figure 1. Pictorial representation of intra-aortic balloon pump within the aorta, showing placement just distal to subclavian artery. Reprinted with permission from Maquet GmbH & Co. KG (Rastatt, Germany).

Intra-aortic balloon pumps

Intra-aortic balloon pumps (IABPs) are the most commonly used form of mechanical circulatory support [14]. They were first used in humans in 1968 [15], and percutaneous devices were introduced in 1980 [16]. The device consists of a balloon catheter and a pump console that inflates the balloon with helium. The balloon catheter is placed in the aorta, with the tip just distal to the origin of the left subclavian artery (Figure 1). The balloon is inflated during diastole, displacing aortic blood and augmenting diastolic pressure. Prior to systole, the balloon is deflated, reducing afterload and facilitating left ventricular emptying. In cardiogenic shock, these hemodynamic effects result in reduced myocardial oxygen demand, enhanced coronary blood flow, and increased cardiac output.

Inflation timing is determined using either the electrocardiogram or the arterial pressure waveform. In the latest devices, inflation timing can be controlled with a physiologic timing algorithm that predicts aortic valve closure. When combined with R wave or pressure predictive deflation, this method maintains balloon synchrony even in patients with severe tachyarrhythmias [17].

As well as providing improved synchrony, modern IABPs have reduced vascular complications. Data from the Benchmark registry, which has collected outcomes for over 37,000 patient episodes [18], demonstrate that smaller (8 to 9.5 French) catheter sheaths have reduced the total complication rate to 2.6% and cut major complications, including limb, bowel, and renal ischemia, to under 0.5%. As a result, mortality directly attributable to IABP use is currently less than 0.05% [19]. Owing to a higher risk of limb ischemia, these devices, even with smaller catheters, should be used cautiously in patients with severe peripheral vascular disease. IABPs are not suitable for all patients and are specifically contraindicated in those with severe aortic regurgitation, aortic dissection, or large aneurysms.

Clinical evidence supporting intra-aortic balloon pump in cardiogenic shock

Attempts to study IABP use in cardiogenic shock have been affected by poor recruitment. This may reflect the difficulty of obtaining timely consent and randomization in the critically ill. For example, the SMASH (Swiss Multicenter Evaluation of Early Angioplasty for Shock Following Myocardial Infarction) study was stopped after recruiting only 55 patients during a 4-year period [20]. Similarly, the TACTICS (Thrombolysis and Counterpulsation to Improve Survival in Myocardial Infarction Complicated by Hypotension and Suspected Cardiogenic Shock) trial was stopped after 3 years when only 57 out of a planned 538 patients were randomly assigned [21].

Early experiences using IABPs in the treatment of cardiogenic shock secondary to AMI were disappointing. Two studies published prior to the availability of reperfusion therapy reported no benefit (Table 2) [22,23]. This is not surprising since patients who develop

Table 2. Mortality evidence supporting intra-aortic balloon pump use in cardiogenic shock complicating an acute myocardial infarction

Study	Patients	IABP[a]	No IABP[a]	*P* value
Pre-thrombolysis				
O'Rourke et al. [22] (1981)	30	50%	43%	0.09
Flaherty et al. [23] (1985)	20	52%	53%	-
Thrombolysis era				
Kovack et al. [25] (1997)	335	93%	37%	0.0002
GUSTO-I [26] (1997)	310	47%[b]	60%[b]	0.06
SHOCK [27] (2000)	856	47%	63%	0.007
NRMI-2 [28] (2001)	12,054[c]	49%	67%	-
Reperfusion by PCI				
NRMI-2 [28] (2001)	7,881[c]	47%	42%	-

[a]In-hospital mortality expressed as a percentage. [b]Thirty-day mortality. [c]Total number of patients in the study, including those with no reperfusion therapy, was 23,180. GUSTO-I, Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries; IABP, intra-aortic balloon pump; NRMI-2, National Registry of Myocardial Infarction 2; PCI, percutaneous coronary intervention; SHOCK, Should We Emergently Revascularize Occluded Coronary Arteries for Cardiogenic Shock.

cardiogenic shock have typically infarcted greater than 40% of their left ventricle [24]. It is therefore unlikely that IABP support would be successful without definitive reperfusion therapy.

In 1997, Kovack and colleagues [25] demonstrated that patients who developed cardiogenic shock complicating an AMI were twice as likely to survive when an IABP was used in conjunction with pharmocological reperfusion strategies (Table 2). In the same year, the GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial reported that early IABP use was associated with a trend toward lower 30-day (47% versus 60%; $P = 0.06$) and 1-year (57% versus 67%; $P = 0.04$) mortality rates in patients who presented with cardiogenic shock complicating an AMI [26]. Analysis of the larger SHOCK (Should We Emergently Revascularize Occluded Coronary Arteries for Cardiogenic Shock) trial (1,190 patients) confirmed this benefit, demonstrating statistically significant lower in-hospital mortality for cardiogenic shock patients who received IABP verses those who did not (50% versus 72%; $P \leq 0.0001$) [27]. However, a significant confounding factor in these studies was a higher number of revascularization procedures in the IABP group.

To eliminate confounding, the SHOCK data were re-evaluated comparing IABP plus fibrinolysis with fibrinolysis alone. In this analysis, in-hospital mortality was still improved by 25% (47% versus 63%; $P = 0.007$) [27]. A similar benefit was observed in the larger National Registry of Myocardial Infarction 2 (NRMI-2) (n = 23,180 patients), in which the use of IABP as an adjunct to fibrinolysis, in cardiogenic shock, reduced in-hospital odds of death by 18% (odds ratio (OR) 0.82, 95% confidence interval (CI) 0.72 to 0.93) [28]. A recent meta-analysis by Sjauw and colleagues [29] demonstrated that this benefit remains statistically significant beyond the in-hospital period, with an absolute decrease in 30-day mortality of 18% (95% CI 16% to 20%; $P < 0.0001$).

IABP benefits are less clear for cardiogenic shock patients who undergo primary PCI. In the SHOCK trial, revascularization with PCI resulted in a significant reduction of mortality when compared with medical therapy, including fibrinolysis. Importantly, IABP use was 86% in both groups, and mortality in the medical therapy group was lower than expected [8]. This suggests that IABP plus medical therapy may result in lower mortality and that IABP plus PCI further improves mortality. In contrast, the NRMI-2 study observed that IABP as an adjunct to primary PCI resulted in a higher mortality (OR 1.27, 95% CI 1.07 to 1.50) in patients with cardiogenic shock complicating an AMI [28]. This negative association is also evident in the recent meta-analysis by Sjauw and colleagues [29]. However, for this part of their analysis, only two registries were used: the NRMI-2 study and the data of Sjauw and colleagues. In the absence of randomization, the trend may be confounded since patients receiving both PCI and an IABP in the NRMI-2 study were more likely to have cardiogenic shock complicated by previous PCI (OR 1.85, CI 1.64 to 2.09) and experience an inter-hospital transfer (OR 2.57, CI 2.40 to 2.75) [28].

A more recent study that randomly assigned patients with AMI complicated by cardiogenic shock to either IABP plus PCI or PCI alone did not demonstrate significant improvement in APACHE II (Acute Physiology and Chronic Health Evaluation II) scores or mortality over the first 4 days of admission (36.8% in the IABP group versus 28.6%) [30]. However, this study was

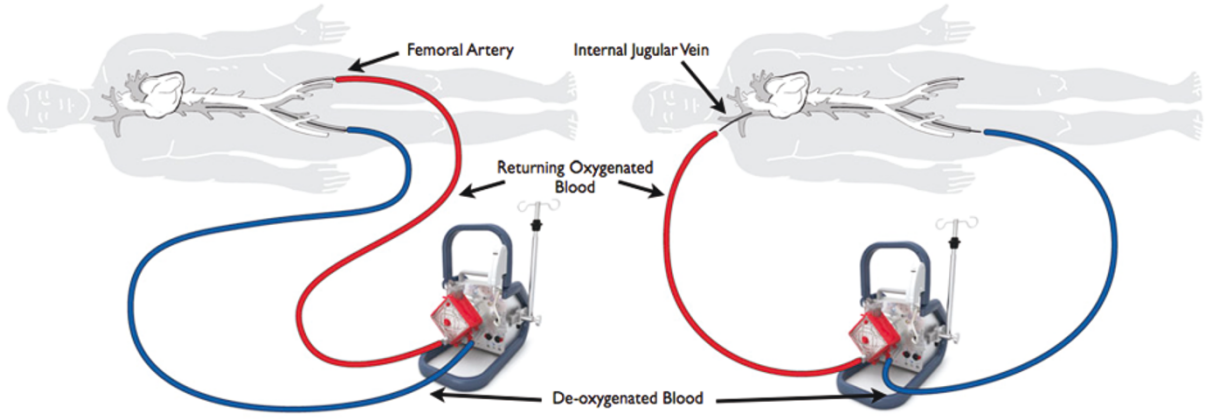

Figure 2. Diagrammatic representation of peripheral veno-venous (VV-ECMO) and peripheral veno-arterial (VA-ECMO) extracorporeal membrane oxygenation. Reprinted with permission from Maquet GmbH & Co. KG (Rastatt, Germany).

not powered to assess mortality. Large randomized clinical trials are required to resolve this issue and address whether the current American Heart Association and American College of Cardiology guidelines recommending PCI and IABP in the setting of cardiogenic shock complicating AMI require revision [31]. In the meantime, we recommend IABP use in any patient meeting the criteria for cardiogenic shock in the setting of an AMI when inotrope therapy has been initiated, whether the patient has received PCI or thrombolysis or neither. IABPs are more widely available than more complex forms of mechanical circulatory support, have a low complication rate, and decrease myocardial oxygen demand. IABPs should be routinely available at centers treating patients with AMI.

Extracorporeal membrane oxygenation

When there is evidence of inadequate tissue oxygen delivery despite IABP, invasive ventilation, and inotropes, full circulatory support should be considered. Extracorporeal membrane oxygenation (ECMO) can subsume the function of both heart and lungs and was first successfully used in adults in 1972 [32]. De-oxygenated blood is removed from the body, pumped through an artificial oxygenator, and returned to the circulation. Modern oxygenators consist of multiple, small hollow fibers lined with polymethylpentene and allow gas but not liquid transfer. Oxygen and carbon dioxide exchange is achieved as blood runs through the center of the fibers and an oxygen/air mix flows on the outside. Blood flow is generated by a centrifugal pump, where a rotating impeller spins blood outwards, creating centrifugal acceleration. Since no compression is involved, high flow rates can be generated with minimal trauma to blood components.

ECMO can be broadly categorized into two types: veno-venous ECMO (VV-ECMO) and veno-arterial ECMO (VA-ECMO) (Figure 2). The type selected depends on therapeutic goals. VV-ECMO is appropriate only for respiratory failure. VA-ECMO is used for cardiogenic shock and is currently the fastest growing indication for ECMO worldwide [33]. In adults, blood is usually removed through a femoral vein and returned through a femoral artery (peripheral ECMO). Occasionally, other cannulation strategies, such as directly cannulating the right atrium and aorta (central ECMO), may be employed.

Peripheral ECMO is less invasive, is easier to place, and can be placed percutaneously by surgeons or intensivists. It can be initiated quickly, making it more appropriate in emergencies. However, the cardiac output of the failing heart competes with retrograde ECMO flow from the femoral aortic cannula, producing admixing in the thoracic aorta and an increase in left ventricular wall tension. If there is concomitant respiratory failure, this can result in the delivery of inadequately oxygenated blood to the coronary and cerebral circulations and hinder recovery [34]. Central ECMO is not associated with this problem but is slower to initiate and may have a higher complication rate with bleeding and infection. It is usually confined to the support of patients after surgical revascularization. Peripheral VA-ECMO is adequate for most forms of cardiogenic shock, but frequent echocardiography is necessary to monitor for progressive ventricular dilatation. If this develops, the left atrium can be vented either by changing the ECMO circuit

configuration or by performing a percutaneous atrial septostomy [35-37].

VA-ECMO is associated with bleeding in 30% to 60% of cases [38,39], sometimes requiring massive transfusions. New pumps and improved circuit biocompatibility allow lower levels of anticoagulation to be used and should reduce the impact of this complication. Clotting abnormalities predispose to hemorrhagic stroke, which, combined with circuit embolic complications such as air bubbles or clots, results in an overall stroke rate of 3% to 12% [33,40,41]. Other complications include nosocomial infection in 50% to 60% [40,41] and multi-organ dysfunction in 33% [39], although the contribution of ECMO is not easy to separate from the complications of severe critical illness. Device and circuit complications appear to be declining [33].

Table 3. Evidence supporting extracorporeal membrane oxygenation use in cardiogenic shock complicating an acute myocardial infarction

Study	Patients	Survival rate[a]	Cardiogenic shock etiology
Golding *et al*. [42] (1992)	91	25.3%	Post-CABG[b]
Muehrcke *et al*. [43] (1996)	23	30.4%	Post-CABG[b]
Magovern *et al*. [44] (1999)	27	85%	UA or CHF
Formica *et al*. [39] (2008)	18	27.8%	AMI/Post-CABG
Combes *et al*. [38] (2008)	16	31.3%	AMI
ELSO [33] (2009)	153[c]	39%	Not defined

[a]Survival to hospital discharge. [b]Postcardiotomy patients who were unable to wean off bypass or developed postoperative cardiogenic shock. [c]Number of extracorporeal membrane oxygenation runs. AMI, acute myocardial infarction; CABG, coronary artery bypass graft; CHF, congestive heart failure; ELSO, Extracorporeal Life Support Organization; UA, unstable angina.

Evidence supporting extracorporeal membrane oxygenation in cardiogenic shock complicating acute myocardial infarction

In 1992, the Cleveland Clinic reported their experience with adult ECMO in postcardiotomy patients, of whom 25.3% survived to discharge (Table 3) [42]. Two years later, this improved to 30.4% [43]. In 1999, Pittsburgh's Allegheny Hospital reported ECMO use in high-risk patients undergoing PCI, of whom 85% survived to hospital discharge [44]. In 2008, two studies from Europe (Formica and colleagues [39] and Combes and colleagues [38]) demonstrated survival to discharge rates of 28% to 31% when ECMO was used for postcardiotomy cardiogenic shock or cardiogenic shock complicating AMI (Table 3). Patients were selected for ECMO if they failed conventional treatment, including inotropes, ventilation, or IABP. PCI was frequently used in the AMI patients.

The variable survival rates reflect that fact these are small single-center studies. The Extracorporeal Life Support Organization (ELSO) registry addresses this limitation by recording the experience of over 170 ECMO centers worldwide. ELSO has accumulated data on over 40,000 ECMO cases, of whom approximately 3,000 are adults. In 2009, ELSO reported a survival rate of 39% for adult cardiogenic shock [33].

The timing of ECMO is controversial, given the absence of guidelines. We recommend considering this therapy in patients with ongoing tissue hypoperfusion despite escalating inotropes, appropriate ventilatory support, and initiation of IABP. Evidence of tissue hypoperfusion includes worsening organ dysfunction, rising lactate, or falling central venous oxygen saturation. Inotrope scores [45,46] approaching 40 to 50 also indicate that mechanical circulatory support may be required (Figure 3). Additional considerations include the rate of decompensation as well as local resources (for example, how quickly ECMO can be initiated or whether the patient has to be transferred). Delaying ECMO until the inotrope score is 60 may be associated with poorer outcomes [46].

Candidates should be selected only if significant organ recovery is expected and there is no contraindication to long-term mechanical support or transplant (Table 4). Up to 60% of survivors cannot be weaned and require a ventricular assist device (VAD) or transplantation [38,41]. ECMO may therefore provide a bridge to decision; it is less costly than VADs, can be initiated quickly, and offers biventricular and respiratory support, thereby stabilizing patients while their suitability for a VAD or transplant is evaluated [47]. Institutions that do not provide this therapy should consider referring patients to an experienced center once IABP support has been initiated. In these situations, expert retrieval teams from the specialist center should provide transport [48,49].

Ventricular assist devices

VADs were first used successfully in humans in 1966 [50]. Three types are used: left ventricular assist (LVAD), right ventricular assist, or biventriciular assist (BiVAD) device. LVAD is the one most commonly used in cardiogenic shock complicating an AMI. Blood is removed from a cannula in the left atrium, or apex of the left ventricle, and pumped into the ascending aorta. Depending on the pump, flow will be pulsatile or continuous. In pulsatile pumps, also known as first-generation VADs, blood fills a compliant, collapsible chamber that is intermittently compressed. Continuous flow pumps use an internal rotating impeller and these newer devices are referred to as second-generation pumps. They may be centrifugal (see 'Extracorporeal membrane oxygenation' section above) or axial, where the impeller is cylindrical with helical blades, similar to an Archimedes' screw. The latest devices, third-generation VADs, spin and levitate the impeller within an electromagnetic field, reducing blood trauma and prolonging serviceable life [51]. A range of

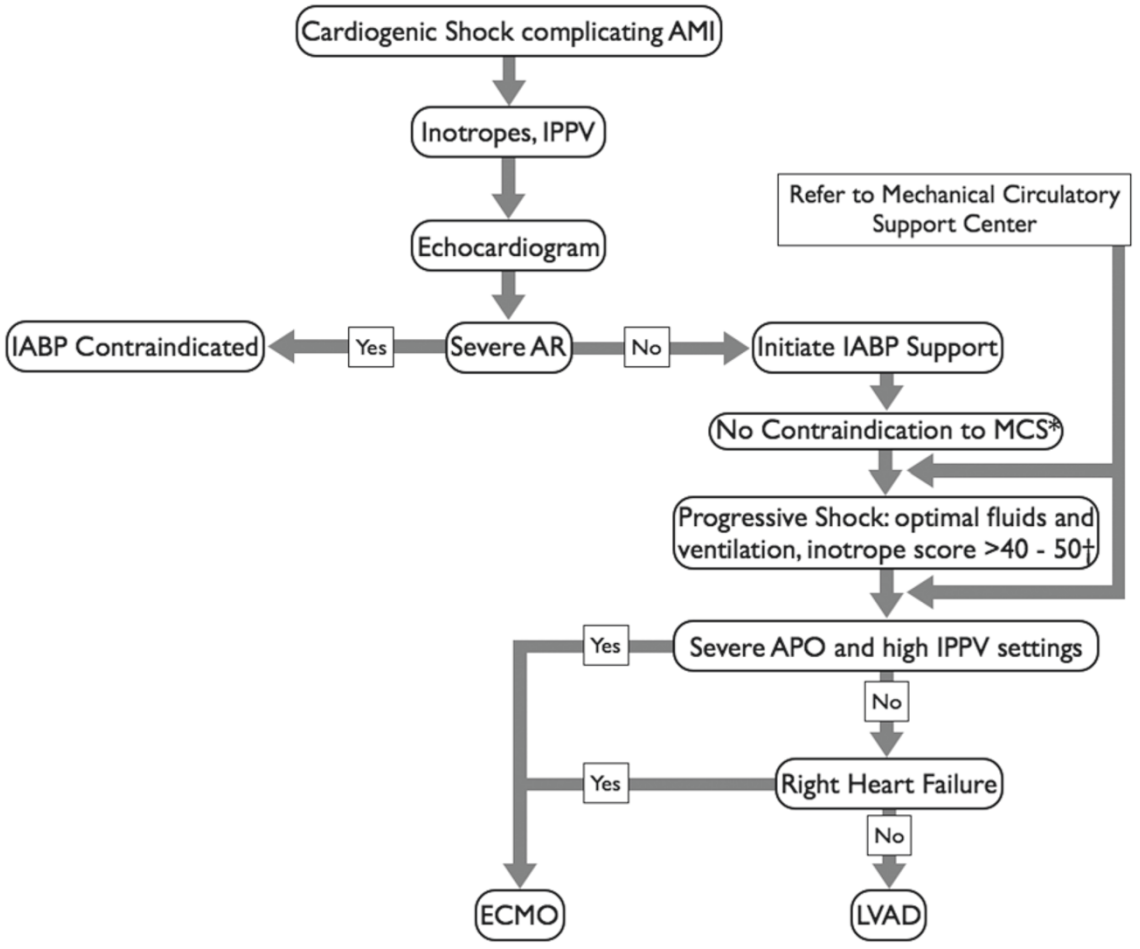

Figure 3. Simplified flow diagram of initiation of mechanical circulatory support. Patients requiring full mechanical circulatory support should be referred to experienced, high-volume centers. *See Table 4 for contraindications to mechanical circulatory support. †Inotrope score = doses of dopamine + dobutamine μg/kg per min + [(epinephrine + norepinephrine + isoproterenol μg/kg per min) × 100] + [milrinone μg/kg per min × 15]. AMI, acute myocardial infarction; APO, acute pulmonary edema; AR, aortic regurgitation; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; IPPV, invasive positive pressure ventilation; LVAD, left ventricular assist device; MCS, mechanical circulatory support.

LVADs are available and can be broadly distinguished by whether cannulation is achieved percutaneously or centrally via a surgical sternotomy (Table 5).

Percutaneous left ventricular assist device

In acute cardiogenic shock complicating an MI, percutaneous LVADs (pLVADs) hold the most promise. They can be initiated quickly and do not require a sternotomy. The two most studied devices are the TandemHeart (CardiacAssist, Inc., Pittsburgh, PA, USA) and Impella (Abiomed, Aachen, Germany).

The TandemHeart removes blood from the left atrium by means of a catheter that is transeptally placed in the left atrium via a femoral vein and returns it to the circulation through a femoral artery by means of a centrifugal pump (Figure 4). This device has been compared with an IABP (Table 6). In one study, 41 patients presenting with cardiogenic shock following an AMI were randomly assigned to receive an IABP or the TandemHeart prior to PCI. The TandemHeart resulted in a larger improvement in the cardiac power index compared with IABP (0.37 versus 0.28, $P = 0.004$) but did not translate into improved 30-day mortality (IABP 45% versus VAD 43%, $P = 0.86$) [52]. In another study, 30 patients presenting with cardiogenic shock were randomly assigned, and 70% of them had cardiogenic shock secondary to an AMI. In that study, the TandemHeart also improved hemodynamics more than the IABP did (Δ cardiac output 1.2 L/min versus 0.6 L/min, $P < 0.05$) [53]. Again, this did not confer a significant 30-day survival advantage (53% survival for TandemHeart versus 64% for IABP). However, in both of these studies, a larger number of hemorrhagic complications and ischemic limbs were seen in the TandemHeart groups.

Table 4. Contraindications to full mechanical circulatory support

Prolonged cardiopulmonary resuscitation with inadequate perfusion
Advanced age
Advanced malignancy
Existing organ dysfunction
Advanced chronic obstructive pulmonary disease
Interstitial lung disease
Liver cirrhosis
Previous stroke with significant disability
Dementia
End-stage renal failure (relative)
Contraindication to anticoagulation (relative)
Contraindication to transplant (relative)

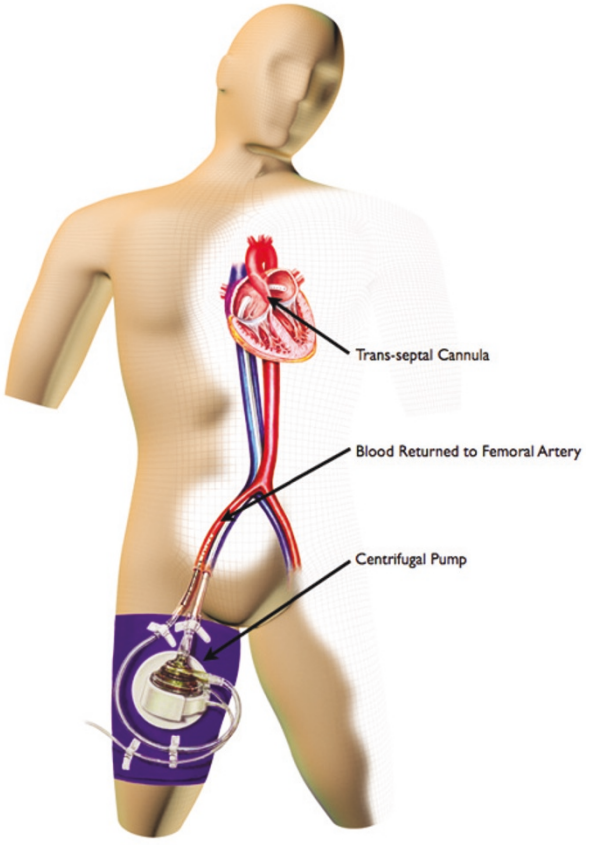

Figure 4. Diagram of the TandemHeart percutaneous left ventricular assist device *in situ* in an adult. Reprinted with permission from CardiacAssist, Inc. (Pittsburgh, PA, USA), the manufacturer of this device.

The Impella percutaneous pump has been recently studied under conditions similar to those of the TandemHeart. Impella uses an axial pump that is placed across the aortic valve via one of the femoral arteries (Figure 5). In 2008, the ISAR-SHOCK study (Impella LP2.5 versus IABP in Cardiogenic SHOCK) randomly assigned 25 patients with cardiogenic shock following an AMI to receive the Impella or an IABP. Investigators found that the cardiac index after 30 minutes of support was significantly increased in patients with the Impella LP2.5 compared with patients with IABP (Impella: Δ cardiac index = 0.49 ± 0.46 L/min per m^2; IABP: Δ cardiac index = 0.11 ± 0.31 L/min per m^2; P = 0.02) [54]. The mortality rate was 43% for both groups, and of particular note, there was no difference in major bleeding or distal limb ischemia between the two groups.

When these three studies are combined in a meta-analysis, it is still not possible to detect a mortality benefit [55]. However, it is arguable that an overall number of 100 patients is still too small. In addition to offering no clear survival benefits, pLVADs provide only left

Table 5. Classification of ventricular assist devices

Access	Ventricular support	Abbreviation	Examples[a]
Percutaneous	Left	pLVAD	TandemHeart (CardiacAssist, Inc., Pittsburgh, PA, USA)
			Impella 2.5L (Abiomed, Aachen, Germany)
			Impella 5L (Abiomed)
Surgical			
Extracorporeal	Right/Left/Biventricular	RVAD/LVAD/BiVAD	CentriMag (Levitronix LLC, Waltham, MA, USA)
			Bio-Medicus (Eden Prairie, MN, USA)
			DeltaStream (Medos Medizintechnik AG, Stolberg, Germany)
Implantable	Left	LVAD	HeartMate II (Thoratec Corporation, Pleasanton, CA, USA)
			Jarvik 2000 (Jarvik Heart Inc., New York, NY) and Incor (Berlin Heart AG, Berlin, Germany)
			HeartAssist 5 (MicroMed Cardiovascular, Inc., Houston, TX, USA) and DuraHeart (Terumo Heart Inc., Ann Arbor, MI, USA)

[a]This list is not exhaustive and includes only a few continuous flow devices. BiVAD, biventricular assist device; LVAD, left ventricular assist device; pLVAD, percutaneous left ventricular assist device; RVAD, right ventricular assist device.

Table 6. Comparative data of studies into ventricular assist device use in cardiogenic shock complicating an acute myocardial infarction.

		Δ Cardiac output, L/min			30-day survival, percentage	
Study	Patients	VAD	IABP	*P* value	VAD	IABP
TandemHeart (pLVAD)						
Thiele *et al.* [52] (2005)	41	1.0	0.3	0.007	57	55
Burkhoff *et al.* [53] (2006)	42	1.2	0.6	-	53	64
Impella (pLVAD)						
Seyfarth *et al.* [54] (2008)	25	0.5	0.1	0.18	54	54
Centrimag (eBiVAD)						
John *et al.* [61] (2007)	12	Titrated for cardiac index >2.2 L/min per m^2			75	N/A

eBiVAD, extracorporeal biventricular assist device; IABP, intra-aortic balloon pump; N/A, not applicable; pLVAD, percutaneous left ventricular assist device; VAD, ventricular assist device.

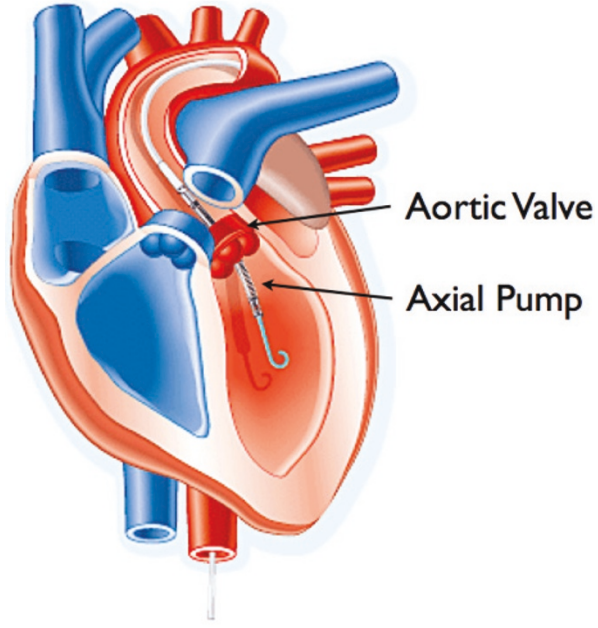

Figure 5. Diagram demonstrating the Impella LP2.5 axial flow left ventricular assist device sitting across the aortic valve. Reprinted with permission from Abiomed (Aachen, Germany), the manufacturer of this device.

ventricular support. Thus, they are inappropriate for cardiogenic shock due to right ventricular ischemia, and although successful cases of percutaneous right ventricular assist [56,57] and even biventricular assist [58] have been reported, they required substantial modification of existing technology. Despite this, the improved hemodynamics are impressive and percutaneous devices are set to become increasingly important in the management of acute cardiogenic shock [59,60], especially if larger studies demonstrate that these hemodynamic benefits translate into significant survival benefits.

Surgically placed ventricular assist device (extracorporeal and implantable)

In the acute setting of cardiogenic shock complicating AMI, surgical VAD placement has proven to be challenging. The additional trauma of surgery compounds the multi-organ dysfunction and coagulopathy associated with extracorporeal circuits. However, third-generation pumps have been successfully surgically placed in the acute setting by means of cannulas tunneled through the chest wall. In one study, the Centrimag (Levitronix LLC, Waltham, MA, USA) was used to provide temporary BiVAD for 12 patients presenting with refractory shock following AMI. Eight patients were successfully bridged to an implantable VAD, and two patients recovered allowing device explantation. Overall 1-year survival was 62.5% [61].

Implantable VADs allow patients to be discharged home, providing a bridge to transplant, bridge to recovery, or destination therapy. Destination therapy is particularly attractive since transplant demand greatly exceeds donor availability. Studies in the last decade have demonstrated that implantable pulsatile LVADs are superior to medical therapy in end-stage heart failure patients who are ineligible for a transplant [62,63]. Recently, it was demonstrated that third-generation devices result in decreased mortality and greater reliability when compared with pulsatile LVADs [64].

VADs are susceptible to complications similar to those experienced with ECMO. Neurological insults affect 4% to 12% of patients, infection 20% to 30%, and bleeding 30% to 40% [52,53,65]. Device malfunction rates are improving; over a 2-year period, less than 10% of implantable third-generation pumps require replacement [64].

The decision to initiate VAD therapy should be made under the same circumstances as those described above for ECMO. The precise modality chosen depends on institutional experience and patient factors. For isolated left ventricular failure, with minimal respiratory

disturbance, a pLVAD may be sufficient. Where there is concomitant respiratory failure or high ventilatory settings or when biventricular support is desired through a percutaneous approach, ECMO is more appropriate (Figure 3). Occasionally, the two may be used together [66]. In patients between these extremes, the factors of institutional experience, likelihood of recovery, and whether surgical revascularization is required will dictate choice. Finally, pursuing this technology is not without controversy in terms of resource allocation and ethics [67]. These issues vary substantially depending on health-care infrastructure, financing sources, and donor (as well as blood product) availability.

Conclusions

When cardiogenic shock complicating AMI is refractory to medical therapy, the only options available for survival are mechanical support strategies. Mechanical support can be applied in a stepwise progression starting with IABP support, followed by either ECMO or an LVAD. In the acute setting, these devices may provide circulatory support until the benefits of revascularization are realized. In the event that weaning is not possible, these devices serve as a bridge to decision or transplant. In patients who are ineligible for transplant, implantable VADs hold the promise of viable destination therapy.

Abbreviations

AMI, acute myocardial infarction; BiVAD, biventricular assist device; CI, confidence interval; ECMO, extracorporeal membrane oxygenation; ELSO, Extracorporeal Life Support Organization; IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; NRMI-2, National Registry of Myocardial Infarction 2; OR, odds ratio; PCI, percutaneous coronary intervention; pLVAD, percutaneous left ventricular assist device; SHOCK, Should We Emergently Revascularize Occluded Coronary Arteries for Cardiogenic Shock; VAD, ventricular assist device; VA-ECMO, veno-arterial extracorporeal membrane oxygenation; VV-ECMO, veno-venous extracorporeal membrane oxygenation.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

MEC contributed to the writing and editing of the text and produced the figures and tables. GM contributed to the writing and editing of the text. Both authors read and approved the final manuscript.

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