Mechanical circulatory support in acute cardiogenic shock

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Abstract

Cardiogenic shock complicates about 5% to 8% of all admissions for acute myocardial infarction, and despite advancement in treatment over the past 50 years, mortality remains unacceptably high. Management with vasoactive agents after revascularization can have its limitations and thus mechanical circulatory support is often initiated. Intra-aortic balloon pumps (IABPs) are the devices most commonly used worldwide. IABPs appeared to improve mortality when used along with fibrinolytic therapy but may not when used along with percutaneous coronary interventions. Extracorporeal membrane oxygenation (ECMO) is utilized in the setting of worsening tissue perfusion despite inotropes and IABP utilization. Although retrospective studies show some mortality benefit, randomized prospective studies have not yet demonstrated ECMO to be advantageous either with or without IABP. Percutaneous left ventricular assist devices such as TandemHeart® and Impella are easier to institute than ECMO and are better for hemodynamics compared with the IABP but also have not yet shown a mortality benefit. More randomized studies are needed to define the most appropriate role of the various mechanical support devices in cardiogenic shock.

Introduction

Cardiogenic shock is a physiologic state in which end-organ tissue hypoperfusion is a result of cardiac dysfunction, where the heart is unable to maintain adequate cardiac output despite adequate intravascular volume [1]. It complicates about 5% to 8% of all ST segment elevation myocardial infarctions (STEMIs) as well as other primary cardiac abnormalities including myocardial disease, valvular heart disease, and congenital heart disease [2]. Despite many advances in the early diagnosis, treatment, and management of cardiogenic shock, mortality remains unacceptably high [2,3]. Recent advances in mechanical cardiac support (MCS) of the failing ventricle present the potential for improvements in outcome. The aim of this review is to assess the role of MCS in acute cardiogenic shock.

Cardiogenic shock manifests clinically as hypotension without hypovolemia with signs of poor tissue perfusion such as oliguria, cyanosis, cool extremities, and altered mentation [1]. A more concrete hemodynamic definition consists of persistent hypotension (systolic blood pressure of less than 80 to 90 mm Hg or mean arterial pressure 30 mm Hg lower than baseline) with severe reduction in cardiac index (less than 1.8 L/min per m^2 without support or less than 2.0 to 2.2 L/min per m^2 with support) and adequate or elevated filling pressure (for example, left ventricular [LV] end-diastolic pressure of less than 18 mm Hg or right ventricular end-diastolic pressure of less than 10 to 15 mm Hg) [4].

The most common cause of cardiogenic shock is acute myocardial infarction (MI). Myocardial ischemia leads to both systolic and diastolic compromise; the resulting myocardial dysfunction can worsen ischemia, creating a downward spiral. When a critical mass of myocardium is affected, stroke volume and cardiac output decrease, leading to hypotension. This in turn lowers coronary perfusion pressure, potentiating ischemia, and induces compensatory vasoconstriction to maintain blood pressure, which can worsen cardiac dysfunction. If this cycle is not reversed, death is the result.

Coronary revascularization is the mainstay of therapy for cardiogenic shock caused by myocardial infarction, but after reperfusion, areas of the myocardium may have myocardial stunning, post-ischemic dysfunction that persists despite restoration of normal blood flow. Other myocardial segments may have persistently impaired function at rest because of severely reduced coronary blood flow, which is referred to as hibernating myocardium. These areas may improve over time with revascularization. Regardless of the mechanism, the concept that areas of the myocardium may have reversible dysfunction and may recover over time provides a strong rationale for supportive hemodynamic therapies in cardiogenic shock.

To address the hemodynamic abnormalities in cardiogenic shock, initial therapy involves careful infusion of fluids, as patients with acute MI can have intravascular fluid loss consequent to diaphoresis or vomiting and may be either absolutely or relatively hypovolemic. If shock is persistent despite fluid resuscitation, initial therapy is pharmacologic, with a delicate balance of inotropic or vasopressor agents or both [5]. Dobutamine, a β_1 adrenoreceptor agonist, is often the first inotropic agent utilized, as it can enhance contractility and can provide support until stunned or reperfused myocardium recovers [6]. Owing to dobutamine's arrhythmogenic properties, other inotropic agents such as milrinone can be considered, but these agents can cause significant hypotension and have not been shown to improve mortality [7]. In patients requiring vasopressors to support systemic blood pressure, norepinephrine is preferred to dopamine [8]. Use of inotropes and vasopressors in cardiogenic shock increases myocardial oxygen demand, and studies of their efficacy have been small and have not necessarily demonstrated that their use improves mortality. Mechanical circulatory support devices can provide substantial cardiovascular support without potentiating myocardial ischemia and may also decrease myocardial oxygen demand, which renders their use theoretically attractive.

## Intra-aortic balloon pump counterpulsation

Counterpulsation with an IABP is the oldest and most commonly used mechanical circulatory support mechanism worldwide [9]. The concept of counterpulsation was devised based on the fact that coronary flow occurs during diastole and thus diastolic augmentation might improve coronary artery flow, improve systemic blood pressure, and decrease myocardial oxygen demand [10]. The IABP was pioneered in the early 1960s by Dr. Adrian Kantrowitz at Maimonides Hospital in Brooklyn, NY. His first patient, described in 1968, was a 45-year-old woman in cardiogenic shock, who was supported with IABP for 7 hours, survived, and was subsequently

discharged [11]. Over 3 million patients have been treated with IABPs since the 1980s, when they came into general use [12].

The IABP consists of a balloon catheter and a pump console that inflates the balloon with helium at the end of systole. The catheter is commonly placed in the cardiac catheterization lab under fluoroscopic guidance but may also be placed at bedside with confirmation on chest X-ray, with the proximal tip distal to the origin of the left subclavian artery and the distal aspect of the balloon proximal to the renal artery (Figure 1). Inflation timing is based on electrocardiography or arterial pressure waveform or more commonly now on programmed algorithms that determine aortic valve closure in the context of the possibility of arrhythmias [13].

The hemodynamic advantages with balloon counterpulsation include the following: an increase in stroke volume and cardiac output; an increase in systemic blood pressure with increased coronary blood flow; a decrease in LV wall stress and myocardial oxygen demand; a reduction in LV preload, LV end-diastolic pressure, and pulmonary capillary wedge pressure; and a mild improvement in microcirculatory flow [14].

Studies conducted to test the clinical effectiveness of IABP counterpulsation have had varied endpoints. Prior to the age of coronary revascularization in the setting of myocardial infarction, IABPs were not shown to modify infarct size or alter morbidity or mortality when compared with standard therapy in cardiogenic shock, likely because IABPs do not increase flow distal to a critical coronary stenosis [15,16]. Not until coronary reperfusion therapy was common did data suggest that IABPs might improve mortality, possibly by supporting patients through a period of myocardial stunning. In 1997, an analysis of patients in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries (GUSTO-1) trial showed a trend toward lower 30-day and 1-year mortality in patients treated with an IABP and fibrinolytic therapy compared with fibrinolytic therapy alone [17]. Another retrospective study showed that combined therapy with IABP and fibrinolytics improved in-hospital and 1-year survival (67% versus 32%, $P = 0.019$) versus fibrinolytic therapy alone [18]. The Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock? (SHOCK) trial registry controlled for confounding by reperfusion therapy and showed that in-hospital mortality was lower in patients supported with IABP (lytics + IABP, 47%; IABP only, 52%; lytics only, 63%; no lytics, no IABP, 77%; $P <0.0001$) [19]. Comparable results were found in an analysis of 23,000 patients in the

Figure 1. Intra-aortic balloon pump
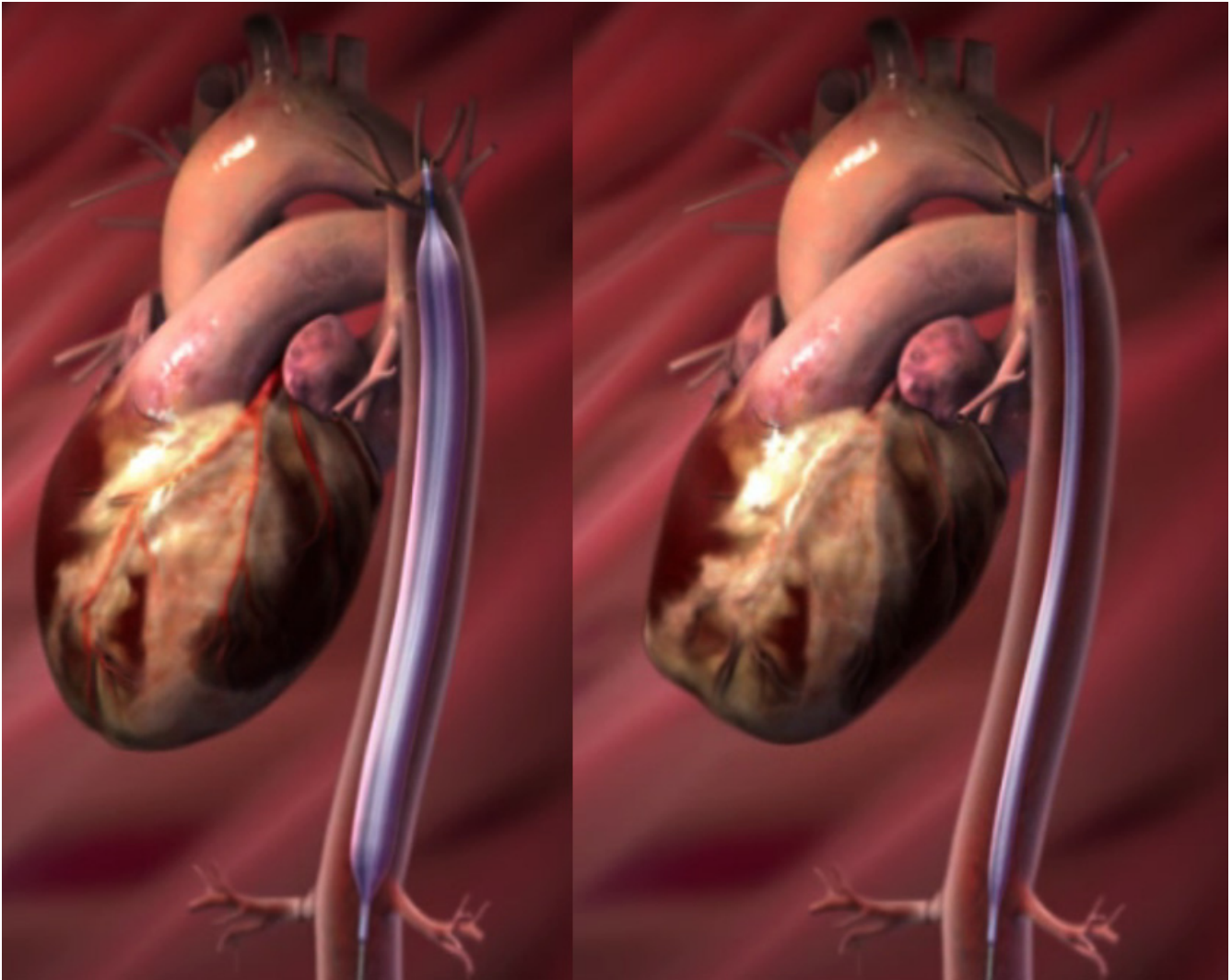
A computer-controlled mechanism inflates a cylindrical polyethylene balloon with helium during diastole (left) and then actively deflates the balloon in systol (right). The tip of the balloon should be approximately 2 cm from the left subclavian artery. Image provided with permission from MAQUET Cardiac Assist, Datascope Corp.

National Registry of Myocardial Infarction 2 (NRMI-2), where IABP plus fibrinolytic therapy decreased the odds of death by 18% (odds ratio 0.82, 95% confidence interval [CI] 0.72 to 0.93) [20].

The mortality benefit conferred by IABP counterpulsation in patients treated with fibrinolytics is evident, but when it comes to percutaneous coronary intervention (PCI), studies have been mixed. Use of IABP in the NRMI-2 study was not associated with any mortality benefit in patients treated with primary angioplasty (45% versus 47%) [20]. A meta-analysis showed that IABP was associated with an 18% (95% CI 16% to 20%;

P <0.0001) decrease in 30-day mortality in patients treated with fibrinolysis but only a 6% decrease (95% CI 3% to 10%; P <0.0008) in patients treated with primary PCI [21]. It should be recognized that observational data concerning IABP therapy in the setting of cardiogenic shock are importantly hampered by bias and confounding [21].

The IABP-SHOCK trial randomly assigned 45 patients with an acute myocardial infarction and cardiogenic shock to IABP + PCI versus PCI alone and was not powered for mortality benefit but did not show either a decrease in mortality or a significant improvement in multi-organ dysfunction score with the addition of IABP

counterpulsation to standard therapy [22]. The IABP-SHOCK II study, a much larger prospective, randomized, open-label, multicenter trial enrolling 600 patients with cardiogenic shock complicating acute myocardial infarction, randomly assigned patients to an IABP group or a non-IABP group. There was no significant difference in the primary endpoint of 30-day all-cause mortality (39.7% versus 41.3%; $P = 0.69$) nor was there a significant difference in any of the subgroups. Secondary safety endpoints such as major bleeding, peripheral ischemic complications, sepsis, and stroke did not differ between the two groups [23]. Subsequent follow-up showed no 12-month mortality benefit with the use of IABP [24]. The decision to use an IABP based upon this study may still be up to the clinician as some randomly assigned patients received an IABP when they were hemodynamically stable and might not have been expected to derive great benefit. In addition, 10% of patients in the control group crossed over to IABP therapy.

Despite the lack of a demonstrated mortality benefit in randomized control trials, it is the opinion of the authors that IABP counterpulsation still has a significant role for the stabilization of patients with cardiogenic shock. Owing to its widespread availability, safety, lower cost compared with other support devices, and ease of placement in many health-care facilities, IABP counterpulsation can be utilized as adjunctive therapy in cardiogenic shock. Insertion of an IABP for patients with hemodynamic instability was given a class IIa recommendation in the American College of Cardiology/American Heart Association (ACC/AHA) guidelines and a class IIb recommendation by the European Society of Cardiology (ESC) (Table 1) [25,26]. Whether its use will be superseded by other mechanical circulatory support devices remains to be seen.

Extracorporeal membrane oxygenation

The mechanism behind ECMO involves draining blood from the venous system, oxygenating it through an oxygenator, and returning this oxygenated blood into the systemic circulation by a centrifugal pump. Artificial oxygenation and perfusion support was used for the first successful open heart operation by Gibbon in 1953 [27]. ECMO was subsequently used predominantly in a pediatric population [28,29]. ECMO was used outside of these settings in 1972 when partial veno-arterial (VA) perfusion with peripheral cannulation, with a Bramson membrane heart-lung machine, was initiated and continued for 75 hours on a 24-year-old male with respiratory failure 4 days after repair of a transected thoracic aorta [30].

VA ECMO can be delivered centrally, which involves cannulation of the right atrium and aorta [31], or peripherally with femoral artery and venous cannulation. Although peripheral VA ECMO can reduce LV preload, it can in turn lead to increased ventricular wall tension due to retrograde flow from the femoral arterial cannulation and thus needs to be monitored more closely as opposed to central VA ECMO (Figure 2). Other potential ECMO complications include a risk of bleeding (upwards of 50%) as well as clotting abnormalities, with a stroke incidence as high as 10%.

Initial survival from ECMO was reported to be 25% to 30%, although the indication for ECMO varied from post-cardiotomy LV failure to cardiogenic shock [32-34]. Whereas earlier studies showed improved survival with complicated PCI, a 2008 study did not show remarkably improved survival with ECMO after post-cardiotomy cardiogenic shock, with survival at about 40% [33]. In a recent retrospective study, the addition of ECMO to patients in cardiogenic shock improved survival at

Table 1. Guideline recommendations in cardiogenic shock [25,26]

Assist device	ACC/AHA/SCAI guidelines		ESC/EACTS guidelines	
IABP	Class IIa	A hemodynamic support device is recommended for patients with cardiogenic shock after STEMI who do not quickly stabilize with pharmacological therapy.	Class IIb	IABP insertion is recommended in patients with hemodynamic instability (particularly those in cardiogenic shock and with mechanical complications).
ECMO	No recommendation	No recommendation	No recommendation	ECMO implantation should be considered for temporary support in patients with acute heart failure with potential for functional recovery following revascularization.
TandemHeart®	Class IIb	Same as IABP	Class IIb	Routine use of percutaneous centrifugal pumps is not recommended.
Impella	Class IIb	Same as IABP	No recommendation	No recommendation

Abbreviations: ACC/AHA, American College of Cardiology/American Heart Association; EACTS, European Association for Cardio-Thoracic Surgery; ECMO, extracorporeal membrane oxygenation; ESC, European Society of Cardiology; IABP, intra-aortic balloon pump; SCAI, Society for Cardiovascular Angiography and Interventions; STEMI, ST segment elevation myocardial infarction.

Figure 2. Extracorporeal membrane oxygenation
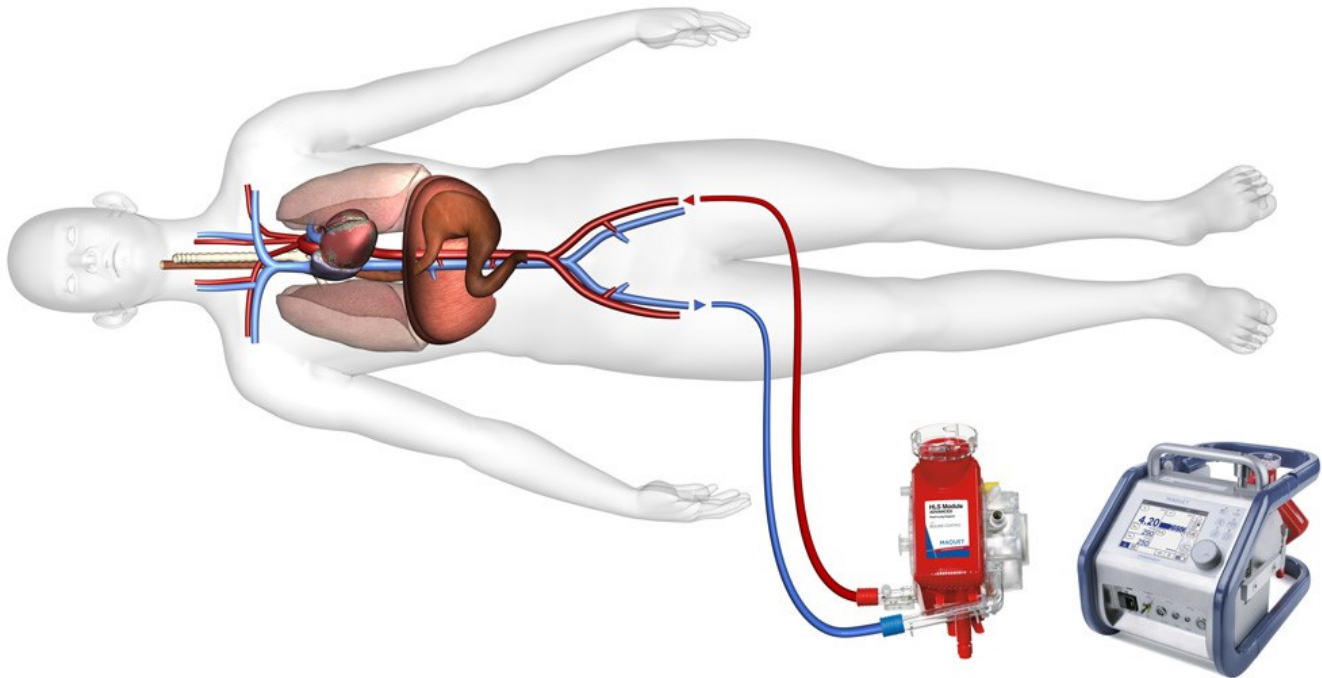
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A catheter is placed in a central vein from which a mechanical pump draws out venous blood and passes it into an oxygenator. This oxygenated blood is then warmed or cooled and returned to the arterial system. Images provided with permission from MAQUET Cardiac Assist, Datascope Corp.

discharge and at 1 year (63% versus 24%; $P = 0.001$) [35]. This study, however, was small and potentially confounded since the comparison group was not contemporaneous.

Approximately 13,000 patients have been treated with ECMO, and the survival to discharge rate was 39% in adults when ECMO was used for cardiac support [36]. When to initiate ECMO in cardiogenic shock is difficult to assess, and the scarcity of patients in randomized trials may explain why there are still no consensus recommendations by the ACC/AHA [25]. The European STEMI guidelines give a class IIb recommendation for the use of ECMO in patients with refractory cardiogenic shock [26]. ECMO may be considered in the context of refractory cardiogenic shock with continued end-organ tissue hypoperfusion and as a bridge to emergent transplantation or ventricular assist device (VAD) placement.

Ventricular assist devices
VADs are mechanical circulatory support devices that are designed to assist/mimic a normally functioning heart. DeBakey described the first device in the late

1960s: "A gas-energized, synchronized, hemispherical pump, of Dacron®-reinforced Silastic®, has been used effectively to relieve the strain on the failing left ventricle during the early recuperative period after open heart surgery" [37]. The pump propels blood forward from the left ventricle (left ventricular assist device [LVAD]) or the right ventricle (right ventricular assist device [RVAD]) or both (biventricular assist device [BIVAD]). The pump technology was originally pulsatile, which attempted to mimic the normal function of the heart. Often a vent outside the body was required to store blood between the pulsations. Current technology provides continuous flow, using either centrifugal pumps or axial flow pumps. Both are more durable and smaller in design than pulsatile pumps, using electric currents in the coil to spin magnetic rotors as much as 15,000 times per minute and expel blood forward [38]. VADs can be used temporarily with the intent that they are a bridge to recovery or a bridge to decision. More recently, implantable VADs have been employed as destination therapy as opposed to being only a bridge to transplant.

In the context of acute cardiogenic shock, percutaneous LVADs (pLVADs) have recently been developed and

implanted temporarily as a bridge to recovery or decision. One of the main advantages for VADs is their ability to operate independent of a patient's intrinsic cardiac rhythm or output and thus provide more cardiac support via increased systemic flow [39].

TandemHeart®

The TandemHeart® (CardiacAssist, Pittsburgh, PA, USA) is an extracorporeal pLVAD device with a centrifugal pump first conceived as short-term support for a patient in cardiogenic shock by researchers at Allegheny General Hospital (Pittsburgh, PA, USA). It consists of a cannula that is inserted in the femoral vein and advanced across the atrial septum into the left atrium where oxygenated blood is obtained (Figure 3). This oxygenated blood is then aspirated and pumped into one or both femoral arteries at a rate of 4.0 L/min [40]. By unloading the left ventricle, it reduces pulmonary capillary wedge pressure and LV preload and subsequently improves peripheral tissue perfusion, despite a mild increase in afterload due to the pumping of blood back into the femoral arteries (Table 2). The TandemHeart® is usually placed to allow

Figure 3. TandemHeart®
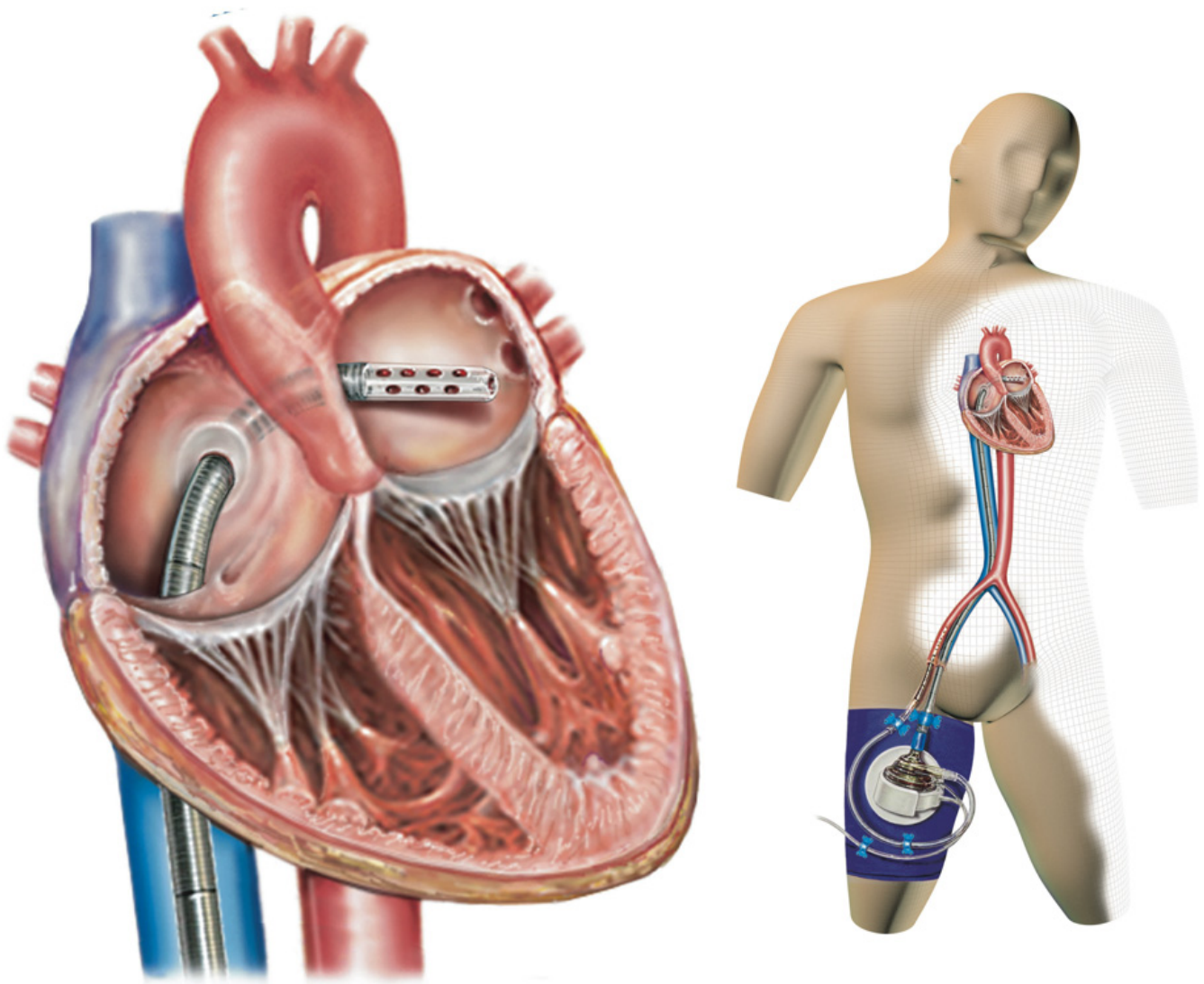
The TandemHeart® transseptal cannula withdraws oxygenated blood from the left atrium to bypass the left ventricle, the target for work reduction. The centrifugal pump provides up to 5 L/min of forward and uniform flow in a percutaneous configuration as well as full pressure support. The arterial cannula returns oxygenated blood to the femoral artery, completing the extracorporeal circuit. Images provided with permission from CardiacAssist (Pittsburgh, PA, USA).

Table 2. Hemodynamic effects of mechanical circulatory support devices [50-53]

	IABP	ECMO	TandemHeart®	Impella
PAOP	Slightly ↓	↓	↓	↓
LV preload	Slightly ↓	↓[a]	↓	↓
Afterload	Slightly ↓	↑	↑	↔
Mean Arterial Pressure	↑	↑	↑↑	↑↑
LV stroke volume	Slightly ↑	↓	↓	↓
Cardiac Output	Slightly ↑	↑	↑↑	↑↑
Coronary perfusion	Slightly ↑	–	↑	↑
Oxygen Consumption	Slightly ↓	↓	↓	↓
Peripheral tissue perfusion	↔	↑	↑	↑

[a] In LV failure, ECMO reduces LV preload. In other settings, the effect of ECMO on LV preload can be variable. ECMO flow rates can also affect LV preload. Key: ↓= reduced, ↑= increased or improved, ↑↑= largely increased, ↔= neutral.

Abbreviations: ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LV, left ventricular; PAOP, pulmonary artery occlusion pressure.

support for a few hours to days, but its use has been reported up to 14 days.

The TandemHeart® was shown in a randomized 2005 trial to improve cardiac power index, as well as other hemodynamic and metabolic variables, compared with IABP counterpulsation, in patients with an acute MI and intended PCI and cardiogenic shock [41]. Similar hemodynamic improvement was shown by the TandemHeart® Investigators Group in 2006; TandemHeart® percutaneous VAD (pVAD), compared with IABP counterpulsation, achieved significantly greater increases in cardiac index and mean arterial blood pressure and significantly greater decreases in pulmonary capillary wedge pressure [42]. Neither study, however, showed a 30-day mortality benefit with the TandemHeart® [41,42]. In 2011, another study showed that TandemHeart® placed in patients with severe cardiogenic shock rapidly reversed the terminal hemodynamic compromise seen in patients with severe cardiogenic shock refractory to IABP and vasopressor support; cardiac index improved from a median of 0.52 to 3.0 L/min per m^2 (P <0.001) [43].

The ACC/AHA guidelines give a class IIb recommendation for LVADs in the context of cardiogenic shock in ST elevation MI [25]. Despite significantly improved hemodynamic parameters compared with IABP counterpulsation, no study has yet shown a mortality benefit. In addition, the complexity of inserting a large-bore catheter (21 French [Fr]) as well as a transseptal puncture requires specialized skills and may not be able to be performed as routinely as one would hope or as quickly as needed in emergency situations.
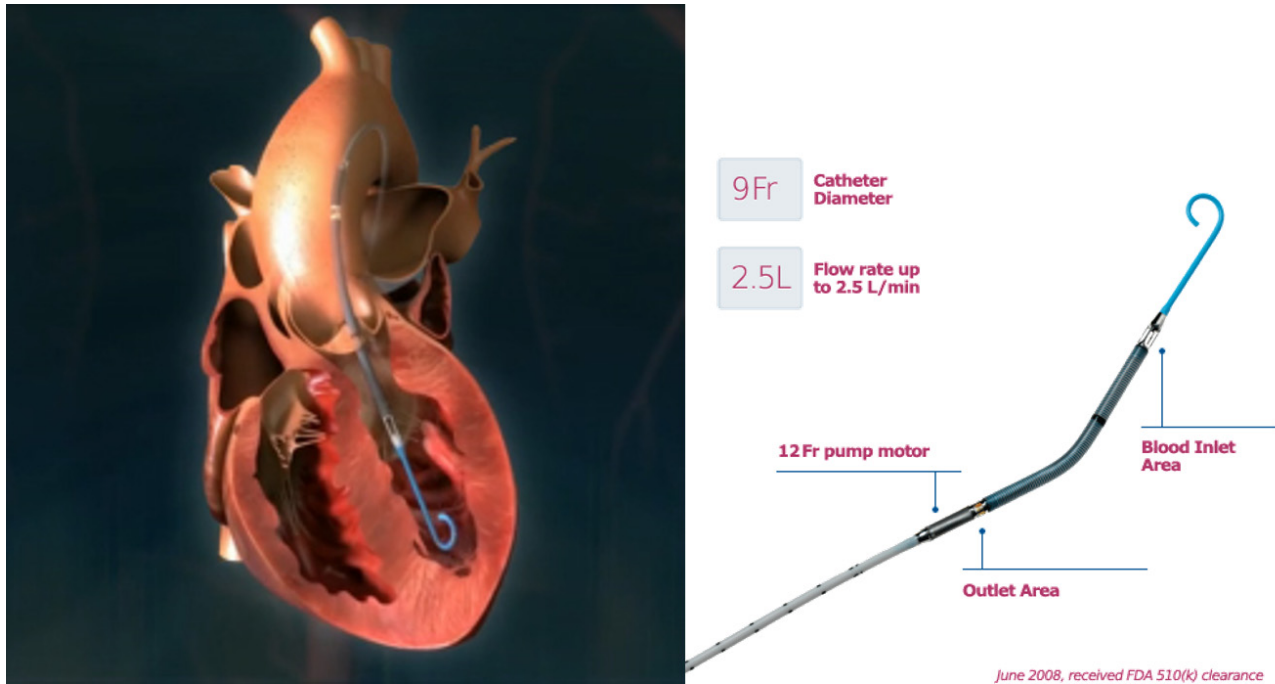
Impella
The Impella (Abiomed, Danvers, MA, USA) device is a pVAD with an axial flow pump that is inserted via the femoral artery and across the aortic valve that aspirates

blood from the left ventricle (Figure 4). The Impella 2.5 and Impella CP devices are inserted percutaneously and provide a cardiac output from 2.5 to 4.0 L/min. A larger device, the Impella 5.0, is able to maintain a cardiac output of 5.0 L/min but requires a large 22-Fr sheath and cannula as well as a surgical cutdown of the femoral artery [44]. The Impella device improves hemodynamics by directly unloading the left ventricle, reducing end-diastolic wall stress and immediately decreasing pulmonary capillary wedge pressure without elevating afterload [45]. Contraindications to Impella device use include moderate to severe aortic insufficiency or aortic valve stenosis, a mechanical aortic valve, and severe peripheral arterial disease.

The Impella device was approved by the US Food and Drug Administration in 2008 on the basis of hemodynamic studies. In 2003, use of the Impella device in refractory cardiogenic shock, when added to the maximum regimens of inotropic and IABP support, showed hemodynamic improvement by increasing cardiac output and reducing pulmonary capillary wedge pressure [46]. In the Efficacy Study of LV Assist Device to Treat Patients with Cardiogenic Shock (ISAR-SHOCK), patients in cardiogenic shock were randomly assigned to either an IABP or an Impella 2.5 device. Patients who received the Impella 2.5 had significantly improved hemodynamic results compared with an IABP; however, like the TandemHeart®, the Impella 2.5 did not demonstrate a 30-day mortality benefit compared with the IABP [47]. The European Shock Control (EUROSHOCK) Registry trial revealed similar hemodynamic improvement in patients in refractory cardiogenic shock but again no mortality benefit, although the study was retrospective and lacked a control group [48]. One small non-randomized study comparing the Impella 5.0 with the Impella 2.5 showed a trend to improved survival in cardiogenic shock complicating STEMI with the Impella 5.0, but there was no statistical

Figure 4. Impella 2.5

The Impella pulls blood from the left ventricle through an inlet area near the tip and expels blood from the catheter into the ascending aorta. The pump can be inserted via a standard catheterization procedure through the femoral artery, into the ascending aorta, across the valve, and into the left ventricle. Images provided with permission from Abiomed (Danvers, MA, USA).
Abbreviation: Fr, French.

Table 3. Comparison of devices

	Impella 2.5	Impella CP	Impella 5.0	ECMO	TandemHeart®	IABP
Pump mechanism	Axial flow/ Transvalvular	Axial flow/ Transvalvular	Axial flow/ Transvalvular	Centrifugal/Bypass	Centrifugal/Bypass	Pneumatic Counterpulsation
Cannula size	12 Fr	14 Fr	21 Fr	18-21 Fr inflow 15-22 Fr outflow	21 Fr inflow 15-17 Fr outflow	7-9 Fr
Cardiac output	2.5 L/min	3.7 L/min	5.0 L/min	>4.5 L/min	4-5 L/min	0.5 L/min
Vascular Surgery	No	No	Yes	Yes	No	No
Complexity of Insertion	Medium	Medium	High	Medium	High	Low
Wall Puncture	No	No	No	No	Yes	No
Hemorrhagic Complications	Medium	Medium	Medium	High	High	Low

Abbreviations: ECMO, extracorporeal membrane oxygenation; Fr, French; IABP, intra-aortic balloon pump.

significance and the patients were not contemporaneous [49]. In 2010, the trial using the Impella LP 2.5 system in patients with acute myocardial infarction induced hemodynamic instability (RECOVER II) and was unfortunately terminated due to the lack of power to reach its primary goal (NCT00972270). There is a paucity of data for the Impella device for acute treatment of acute MI, especially when it is complicated by cardiogenic shock. Like the TandemHeart®, it is given a class IIb recommendation by the ACC/AHA but no recommendation by the ESC/ European Association for Cardio-Thoracic Surgery guidelines. Unlike the TandemHeart®, we do recommend the Impella device be considered for use in preference to IABP for acute cardiogenic shock in experienced institutions (Tables 2 and 3).

Conclusions
Despite multiple medical advances, acute cardiogenic shock has a poor prognosis. Management should begin with fluid resuscitation, inotrope/vasopressor therapy,

and, in patients with an ischemic etiology, prompt revascularization. In patients with refractory cardiogenic shock, concurrent mechanical circulatory support should also be initiated. For a long time, IABP counterpulsation was considered to have a mortality benefit until the IABP SHOCK-II trial was performed. Comparison with the other mechanical support devices has shown a hemodynamic advantage for VADs; however, a significant improvement in survival with VADs has not yet been demonstrated. We believe that the IABP should be considered the initial mechanical circulatory support device in acute cardiogenic shock because it is easy to insert. Peripheral VADs may be considered in experienced centers in preference to IABP for patients with more severe shock. Further randomized studies are needed to properly evaluate outcomes after use of mechanical support devices in cardiogenic shock and to define their most appropriate role.

Abbreviations

ACC/AHA, American College of Cardiology/American Heart Association; CI, confidence interval; ECMO, extracorporeal membrane oxygenation; ESC, European Society of Cardiology; Fr, French; IABP, intra-aortic balloon pump; LV, left ventricular; LVAD, left ventricular assist device; MCS, mechanical cardiac support; MI, myocardial infarction; NRMI-2, National Registry of Myocardial Infarction 2; PCI, percutaneous coronary intervention; pLVAD, percutaneous left ventricular assist device; pVAD, percutaneous ventricular assist device; SHOCK, Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock?; STEMI, ST segment elevation myocardial infarction; VA, veno-arterial; VAD, ventricular assist device.

Disclosures

The authors declare that they have no disclosures.

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