4-1-2021

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Recommended Citation
Lemor A, Ya'qoub L, and Basir MB. Mechanical Circulatory Support in Acute Myocardial Infarction and Cardiogenic Shock. Interv Cardiol Clin 2021; 10(2):169-184.

Mechanical Circulatory Support in Acute Myocardial Infarction and Cardiogenic Shock

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KEYWORDS

• Acute myocardial infarction • Cardiogenic shock • Mechanical circulatory support
• Intra-aortic balloon pump • Extracorporeal membrane oxygenation • TandemHeart • Impella

KEY POINTS

- Acute myocardial infarction complicated by cardiogenic shock is a deadly condition associated with significant morbidity and mortality.
- Despite 20 years of medical advancements, early revascularization remains the sole therapy proven to improve outcomes.
- Mechanical circulatory support devices provide a physiologically plausible mechanism of improving outcomes by offering hemodynamic stability for revascularization and improving end-organ perfusion. Results from well-powered randomized controlled trials, however, are not yet available.
- Randomized controlled trials have been difficult to conduct in this patient population; until such trials are performed, implementing shock teams and protocols has been associated with improved outcomes in observational studies and may be considered.
- Technological advancements will lead to continued development of more mobile, smaller-caliber, and more powerful mechanical circulatory support devices. Understanding the mechanisms of action and physiologic effects of these devices, therefore, is critically important.

INTRODUCTION

Acute myocardial infarction (AMI) can result in diastolic dysfunction and an increase in left ventricular end-diastolic pressure. If not treated promptly, AMI can progress to systolic dysfunction and decreasing stroke volume, which can lead to cardiogenic shock (CS). CS is a low-output state resulting in decreased systemic and coronary perfusion. Decreased systemic perfusion results in end-organ injury, whereas decreased coronary perfusion results in further ischemia, leading to a vicious cascade that ultimately can lead to death. The cascade of events results in a complex neurohumoral cascade referred to as the systemic inflammatory response syndrome. The goals for treating AMI and CS (AMICS), therefore, are to relieve ischemia and improve perfusion to end organs.[1]

AMICS is a deadly condition associated with significant morbidity and mortality. Patients presenting with AMICS who do not receive invasive

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Intervent Cardiol Clin 10 (2021) 169–184
https://doi.org/10.1016/j.iccl.2020.12.005

therapies have less than 20% survival.[2] The Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial demonstrated improved survival in patients presenting with AMICS treated with early mechanical revascularization.[3] Unfortunately, further revascularization does not lead to further improvements in short-term survival, as was demonstrated in the CULPRIT-SHOCK trial.[4] In the past 2 decades, there has been little advancement made to improving outcomes further. This is of great concern because the prevalence of AMICS is growing in the aging population.[5] Patients frequently present with more comorbidities and are more likely to experience cardiac arrest and CS.[5]

Given the high mortality associated with AMICS despite revascularization, clinicians have looked to other forms of therapies in the hope of improving outcomes. Technological advancements have resulted in an increased availability of temporary mechanical circulatory support (MCS) devices, which can improve systemic and coronary perfusion. These devices are reviewed herein.

INTRA-AORTIC BALLOON PUMP

Intra-aortic balloon pump (IABP) counterpulsation is the oldest and most common form of MCS.[6–8] Since its inception in 1967, several observational studies have suggested improved survival with the use of IABP in patients with AMICS[9–21] (**Table 1**). IABPs have been demonstrated to improve systemic hemodynamics and improve coronary perfusion, are easy to use, and are inexpensive. Until recently, there was 1 alternative device, venoarterial (VA)–extracorporeal membrane oxygenation (ECMO), which was more invasive, associated with more complications, and utilized primarily in select tertiary care centers. Therefore, the use of IABPs was questioned infrequently for decades.

Randomized controlled trials (RCTs), however, failed to show survival benefit[22–26] (**Table 2**). In the Thrombolysis and Counterpulsation to Improve Cardiogenic Shock (TACTICS) trial, 57 patients with AMICS were randomized after thrombolytic therapy to 48 hours of IABP therapy or optimal medical therapy. The investigators found no significant difference in 6-month mortality between the 2 groups.[22] Prondzinsky and colleagues[24] randomized 45 patients with AMICS after percutaneous coronary intervention (PCI) to IABP therapy or optimal medical therapy. They found no difference in Acute Physiology and Chronic Health Evaluation II scores, interleukin-6 levels, and cardiac index (CI) between the groups. In-hospital mortality also was similar between the groups (38.6% vs 28.6%, respectively).[24] The largest trial conducted evaluating the efficacy of IABP in AMICS was the IABP-SHOCK II trial; 300 patients were randomized to IABP and 298 patients to the control group. There was no difference in outcomes, including secondary endpoints, such as time to hemodynamic stabilization, length of stay in the intensive care unit, serum lactate levels, dose and duration of catecholamine therapy, renal function, major bleeding, peripheral ischemic complications, and stroke.[25,26]

Furthermore, numerous meta-analyses have investigated the role of routine IABP in AMICS.[27–29] The largest analysis was performed by Ahmad and colleagues,[27] who analyzed patients presenting with AMI from 12 RCTs, including 2123 patients, and 15 observational studies, including 15,530 patients. They found no difference in 30-day mortality in patients with AMI who received IABP, regardless of the presence (odds ratio [OR] 0.94; 95% CI, 0.69–1.28) or absence (OR 0.98; 95% CI, 0.57–1.69) of CS.[28] As a result of these randomized trials and meta-analyses, the European guidelines downgraded IABP use in AMICS from a previous class I to a class III recommendation,[30] whereas the US guidelines downgraded IABP use to a class II recommendation.[31]

This review focuses on patients with AMICS. Patients who present with CS from decompensated heart failure CS, however, differ in their response to IABPs. Malick and colleagues[32] have demonstrated that patients with decompensated heart failure CS had a 5-fold greater cardiac output augmentation with IABP compared with patients with AMICS.

VENOARTERIAL–EXTRACORPOREAL MEMBRANE OXYGENATION

VA-ECMO uses a centrifugal pump and a membrane oxygenator, to provide flows of up to 3 L/min to 7 L/min. There are few retrospective observational studies evaluating the use of ECMO in AMICS (**Table 3**). These studies demonstrate a survival rate ranging from 47% to 60.9% in patients who have a mean age of 54 years to 60 years.[33,34] In 2010, Sheu and colleagues[35] studied 115 patients with AMICS from 1993 to 2002 without ECMO support and compared them with 219 patients with AMICS from 2002 to 2009 with ECMO support. The 30-day mortality for patients with ECMO was lower than the non-ECMO cohort (30.1% vs

Table 1
Summary of observational studies of intra-aortic balloon pump in acute myocardial infarction and cardiogenic shock

Author, Year Published	Number of Patients	Population	Outcomes
Moulopoulos et al,[9] 1986	N = 52 34 IABP	AMICS	10/34 patients survived longer than a month. 15 patients in whom IABP could not be placed, none survived
Bengtson et al,[10] 1992	N = 200 99 IABP	AMICS	In-hospital mortality 53% Patency of infarct-related vessel was a predictor of survival. No difference between IABP and no IABP arms
Waksman et al,[11] 1993	N = 85 20 IABP	AMICS	In-hospital and 1-y survival was significantly higher in the IABP arm (46% and 38% vs 19% and 10%, respectively; $P<.001$).
Stomel et al,[12] 1994	N = 64 13 thrombolytics 29 IABP 22 thrombolytics + IABP	AMICS	Survival improved in thrombolytics + IABP group compared with thrombolytics or IABP alone (68% vs 23% or 28%, respectively; $P = .0049$).
Anderson et al,[13] 1997	N = 310 68 IABP	AMICS	Despite more adverse events and moderate bleeding, the IABP cohort showed a trend toward lower 30-d and 1-y mortality rates.
Kovack et al,[14] 1997	N = 46 patients 27 IABP	AMICS who received thrombolytics	Patients in the IABP arm had significantly higher hospital survival (93% vs 37%, respectively; $P = .0002$).
Brodie et al,[15] 1999	N = 1490	AMI with and without CS	Pre-PCI IABP was associated with lower cardiac events in CS (n = 119) (14.5% vs 35.1%, respectively; $P = .009$), in CHF or low ejection fraction (n = 119) (0% vs 14.6%, respectively; $P = .10$), and in high-risk patients (n = 238) (11.5% vs 21.9%, respectively; $P = .05$).
Kumbasar et al,[16] 1999	N = 45 25 IABP	Anterior AMI who received thrombolytics	IABP had significantly higher rates of thrombolysis in myocardial infarction grade 3 flow (n: 11%; 44% vs n: 1%, respectively; 5%; $P<.05$). There was a

(*continued on next page*)

Table 1 (*continued*)

Author, Year Published	Number of Patients	Population	Outcomes
			trend toward a lower in-hospital mortality in the IABP group (n: 0 [0%] vs n: 3; [15%]; $P = .08$).
Sanborn et al,[17] 2000	N = 856 279 IABP, 160 IABP + thrombolytics 132 thrombolytics only	AMICS	Thrombolytic group had a lower in-hospital mortality compared with no-thrombolytics (54% vs 64%, respectively; $P = .005$). The IABP group had a lower in-hospital mortality compared with no-IABP (50% vs 72%, respectively; $P<.0001$).
Barron et al,[18] 2001	N = 23,180 7268 IABP	AMICS	IABP was associated with significantly lower mortality in the thrombolytic group (67% vs 49%, respectively) but not in PCI group (45% vs 47%, respectively).
Zeymer et al,[19] 2011	N = 653 163 IABP	AMICS	In-hospital mortality, with and without IABP, was 56.9% and 36.1%, respectively. In the multivariate analysis the use of IABP was not associated with improved survival (OR 1.47; 95% CI, 0.97–2.21; $P = .07$).
Sjauw et al,[20] 2012	N = 292 199 IABP	STEMI with CS treated with PCI	30-d mortality in IABP vs no-IABP was 47% vs 28%, respectively; OR 1.67 (95% CI, 1.16–2.39), no difference after propensity stratification 3-d mortality in pre-PCI IABP vs post-PCI was 64% vs 40%, respectively; OR of 1.56 (95% CI, 1.18–2.08), no difference after propensity stratification
Zeymer et al,[21] 2013	N = 1913 487 IABP	AMICS	In-hospital mortality with and without IABP was 43.5% and 37.4% respectively. In multivariate analysis, IABP was associated with increased mortality (OR 1.45; 95% CI, 1.15–1.84).

Table 2
Summary of randomized clinical trials of intra-aortic balloon pump in acute myocardial infarction and cardiogenic shock

Author, Year Published	Number of Patients	Population	Outcomes
Ohman et al,[22] 2005	57 patients	AMICS who received thrombolytics	No difference in 6-mo mortality (34% for IABP + thrombolytics vs 43% for thrombolytics alone [n = 27]; adjusted *P* = .23)
Prondzinsky et al,[24] 2010	45 patients	AMICS status post-PCI	No difference in in-hospital mortality, Acute Physiology and Chronic Health Evaluation II scores, interleukin-6 levels, and CI at 4 d.
Thiele et al,[25] 2012	598 patients	AMICS	No difference in 30-d mortality, the time to hemodynamic stabilization, the length of stay in the intensive care unit, serum lactate levels, the dose and duration of catecholamine therapy, renal function, major bleeding, peripheral ischemic complications, and stroke
Thiele et al,[26] 2018	591 patients	AMICS	No difference in 6-y mortality, recurrent myocardial infarction, stroke, repeat revascularization, or rehospitalization for cardiac reasons

41.7%, respectively; *P* = .034). A subgroup analysis of patients in profound CS found a significant difference in mortality between groups (39.1% in ECMO vs 72% in non-ECMO; *P* = .008); however, in patients without profound shock, there was no significant difference in 30-day mortality between the groups (26.1% vs 21.9%, respectively; *P* = .39). Esper and colleagues[36] studied 18 patients who underwent VA-ECMO in the catheterization laboratory for AMICS and found an in-hospital survival rate of 67% and 6-month survival of 55%. More than one-third of patients had an IABP placed and were on vasopressors or inotropes. Similarly, Negi and colleagues[37] studied 15 patients with AMICS (one-third presenting with cardiac arrest) and showed a 47% survival rate. More than 90% of patients were on 1 to 2 inotropes at the time of ECMO, 60% had an IABP, and the vascular complication rate was greater than 50%. Lastly, a recent observational study by Vallabhajosyula and colleagues[38] using the National Inpatient Sample database evaluated 2962 patients in a period of 14 years and demonstrated a survival rate of 40.8%. There was a significant trend to improved survival over time and 12% of patients were bridged to LV assist device (LVAD) or heart transplantation.[38]

There are no RCTs to date evaluating the use of ECMO in AMICS. Two European studies, EURO SHOCK and ECLS-SHOCK, currently are enrolling patients. EURO-SHOCK will randomize 428 patients to ECMO or standard therapy and will evaluate 30-day mortality as the primary outcome; their expected study completion date is February 2024.[39] Similarly, ECLS-SHOCK will enroll 420 patients with AMICS undergoing revascularization and randomize to ECMO or medical therapy alone. The primary outcome is 30-day mortality and the estimated study completion date is August 2023.[40]

TandemHeart

TandemHeart (LivaNova, London, UK) used a percutaneous centrifugal pump to provide flows up to 3 L/min to 5 L/min using cannulas similar to VA-ECMO. There are few studies assessing the

Table 3
Major observational studies of venoarterial–extracorporeal membrane oxygenation in acute myocardial infarction and cardiogenic shock

Observational Studies				
Author/Trial (Year)	**Sample Size**	**Device (s)**	**Results**	**Notes**
Esper et al,[36] 2015	18	VA-ECMO	67% survival rate, very high bleeding rates (>90%)	Single-center experience, peripheral ECMO, average length of ECMO was 3.2 d ± 2.5 d
Negi et al,[37] 2016	15	VA-ECMO	47% survival rates, 53% vascular complication rates	Small sample, single center, 33% with cardiac arrest, 60% with STEMI
Sheu et al,[35] 2010	219	VA-ECMO	60.9% survival in ECMO vs 28% survival in the non-ECMO cohort	All patients prior to ECMO had a IABP and were on dobutamine
Takayama et al, 2013	90	VA-ECMO	49% survival.	Combined AMI and CHF patients in shock; 23 patients underwent permanent LVAD and 9 heart transplantation.
Vallabhajosyula et al, 2019	2962	ECMO	40.8% survival	Survival improved from 0% in 2000– to 54.9% in 2014. Potential bias due to administrative database. Multicenter, large sample study

hemodynamic and clinical outcomes of TandemHeart in patients with AMICS (Table 4). Kar and colleagues[41] studied 80 patients with AMICS and found that TandemHeart led to a rapid improvement several hemodynamic measures, including CI, systolic blood pressure, urine output, and lactic acid levels. The mortality rates were 40.2% and 45.3% at 30 days and 6 months, respectively, for AMICS patients. Smith and colleagues[42] analyzed 56 patients, 16 (29%) of whom had AMICS, and found improved hemodynamics with the use of TandemHeart. They also found that survival was significantly influenced by the indication of the TandemHeart (23.8% in bridge to recovery vs 51% in bridge to LVAD or surgery [P = .04]), and patients who did not receive definitive therapy had poor outcomes (13.8% survived to hospital discharge). Further observational data are being collected in the TandemHeart Experiences and MEthods (THEME Registry); an ongoing multicenter study (ClinicalTrials.gov Identifier: NCT02326402).

Two underpowered RCTs have been conducted with the use of TandemHeart. Thiele and colleagues[43] randomized 20 patients to IABP and 21 patients to TandemHeart. They found cardiac power index and other hemodynamics measures improved more effectively with TandemHeart; however, complications, including severe bleeding and limb ischemia, were more frequent. The investigators also found no difference in 30-day mortality between groups; however, the study was underpowered to detect these differences.[43] Burkhoff and colleagues[44] randomized 33 patients with AMICS to treatment with IABP or TandemHeart. They similarly found improved hemodynamics with higher CI and lower pulmonary capillary wedge pressure with the use of TandemHeart; however, there was no difference in 30-day mortality between the groups.[44]

Table 4
Major studies of TandemHeart in acute myocardial infarction and cardiogenic shock

Study, Publication Year	Number of Patients	Study Type	Outcomes
Thiele et al, 2005	41	RCT: IABP vs TandemHeart	No difference in 30-d mortality. TandemHeart led to improvement in hemodynamics but was associated with more complications, including bleeding and limb ischemia.
Burkhoff et al,[44] 2006	33	RCT: IABP vs TandemHeart	No difference in 30-d mortality. TandemHeart led to improvement in hemodynamics.
Kar et al,[41] 2011	117 total, 80 with AMI, 37 with NICM	Observational: TandemHeart in refractory shock	30-d and 6-mo mortality rates were 40.2% and 45.3%, respectively, in AMI, vs 32% and 35%, respectively, in NICM. TandemHeart led to improvement in hemodynamics.
Smith et al,[42] 2018	56 total, 16 (29%) AMI	Observational, CS due to advanced HF and AMI	Survival was significantly influenced by indication (23.8% in bridge to recovery vs 51% in bridge to LVAD or surgery; $P = .04$). TandemHeart led to significant improvements in CI and PCWP.
Schwartz et al, 2012	76, 19 received TandemHeart, 58% AMI	Observational	30-d mortality 63%

Abbreviations: NICM, nonischemic cardiomyopathy; PCWP, pulmonary capillary wedge pressure.

IMPELLA

Impella (Abiomed, Danvers, Massachusetts) is continuous nonpulsatile micro-axial pump that has an inlet area that aspirates blood from the left ventricle and ejects it through the outlet into the ascending aorta, at a rate up to 5.5 L/min. Observational studies assessing the use of Impella in CS have compared it with either medical therapy, IABP, or ECMO (Table 5). The Impella-EUROSHOCK registry was an observational-single arm study that evaluated 120 patients with AMICS supported with an Impella 2.5. The feasibility study demonstrated a 64% 30-day mortality; however, it showed feasibility of device placement and improvement in lactate levels.[45] Karatolios and colleagues[46] compared Impella to medical therapy in 90 patients with cardiac arrest (27 patients were treated with Impella) and demonstrated 65% survival in the Impella cohort compared with 20% in the medical therapy cohort. Schrage and colleagues[47] matched patients from the IABP-SHOCK II trial to patients supported with an Impella device in Europe. They demonstrated no significant difference in 30-day all-cause mortality (48.5% vs 46.4%, respectively; $P = .64$) but did show higher rates of severe bleeding and vascular complications in the Impella group. The main limitation of this study was that the degree of CS was not taken into account when matching patients. Lemor and colleagues[48] analyzed AMICS patients from the National Inpatient Sample from 2015 to 2017 who underwent PCI and had either Impella or ECMO support. Propensity-matched analysis showed significantly lower mortality in the Impella cohort (26.7% vs 43.3%, respectively; $P = .02$) as well as lower ischemic stroke and vascular complication rates. This study, however, also was limited by the inability to match patients according to the degree of shock. Loehn and colleagues[49] showed improved survival with the use of Impella before PCI (50% pre-PCI Impella vs 23.1% post-PCI Impella). Helgestad and colleagues[50] demonstrated lower 30-day mortality in patients receiving Impella compared with a matched control group that underwent IABP placement (40% vs 77.5%, respectively; P log rank <0.001).

Table 5
Summary of randomized controlled trials and observational studies for Impella

Randomized Controlled Trials

Author/ Trial (Year)	Sample Size	Comparison	Results	Notes
ISAR-SHOCK[51] (2008)	12 vs 13	Impella 2.5 vs IABP	Similar 30-d mortality in both groups (46% for both)	Improved CI with Impella device
IMPRESS[52] (2017)	24 vs 24	Impella CP vs IABP	Similar 30-d (46% vs 50%, respectively; $P = .92$) and 6 mo mortality (50% for both; $P = .9$)	>90% of patients with cardiac arrest prior device placement

Observational studies

Author/Trial (Year)	Sample Size	Device (s)	Results	Notes
INOVA[60] (2019)	82	IABP, Impella, ECMO	30-survival was 63.4% for all patients (62/82 supported with Impella)	A multidisciplinary team–based approach can improve outcomes.
NCSI[59] (2019)	171	Impella CP	72% survival with best practices (early RHC, MCS, and PCI)	Lactate <4 and cardiac power output >0.6 are good predictors of survival. Multicenter study
Utah Cardiac Recovery Shock Team[64] (2019)	123	IABP, Impella, ECMO	54.5% survival (for the entire cohort—IABP, Impella, ECMO)	33.3% of patients supported with Impella. AMICS in 61%
Schrage et al,[49] 2019	237 matched patients from IABP-SHOCK trial	Impella CP vs IABP	No difference in survival (48.5% vs 46.4%, respectively; $P = .64$)	Selection bias and unable to compare degree of shock between patients
EUROSHOCK[45] (2013)	120	Impella 2.5	64% 30-d mortality	Impella is feasible and reduced lactate levels
Karatolios et al,[46] 2018	90	Impella CP vs medical therapy	65% survival in Impella cohort vs 20% with medical therapy (27/90 with Impella support)	All patients had cardiac arrest. Single-center study

Lemor et al,[48] 2020	5730 vs 560 (450 propensity matched)	Impella CP vs ECMO	Propensity matched: in-hospital mortality rates were 26.7% vs 43.3%, respectively.	Potential bias due to administrative database. Multicenter, large sample study
Loehn et al,[49] 2020	73	Impella CP	50% survival for Impella Pre-PCI vs 23.1% for Impella post-PCI	More patients in the Impella post-PCI group had cardiac arrest, although younger patients in the Impella pre-PCI group with higher percentage of left main disease.
Helgestad et al,[50] 2020	903 (279 with MCS)	Impella CP vs IABP	Lower 30-d mortality compared with matched control group (40% vs 77.5%, respectively; *P* log rank <0.001).	Matched cohort included 40 patients in each group.

Abbreviation: RHC, right heart cath.

Two underpowered RCTs have been conducted evaluating Impella in AMICS. Both compared Impella versus IABP in a small sample of patients. The ISAR-SHOCK trial randomized 25 patients to either Impella 2.5 or IABP and demonstrated safety and feasibility to use Impella 2.5 in AMICS. Patients treated with Impella had similar 30-day mortality when compared with IABP (46%); however, Impella did provide better hemodynamic support.[51] The IMPella versus IABP Reduces mortality in STEMI patients treated with primary PCI in Severe cardiogenic SHOCK (IMPRESS) trial was a randomized, prospective, open-label, multicenter trial that enrolled 48 patients with AMICS and randomized patients to an Impella CP or IABP.[52] The investigators aimed to enroll more than 100 patients but the trial was prematurely stopped due to poor enrollment. Overall, the results showed similar mortality rates for both cohorts (46% for Impella and 50% for IABP; $P = .9$).

RIGHT VENTRICULAR FAILURE

Acute right coronary artery occlusion proximal to the right ventricular (RV) branches, or less commonly left circumflex artery occlusion, often results in RV ischemia. RV ischemia can lead to depressed RV systolic function decreasing transpulmonary flow and left ventricular filling. This can result in diminished preload and cardiac output. The severity of the hemodynamic compromise in patients with RV failure is related to the extent of RV ischemia, left ventricular function, and ventricular interdependence.[53] Patients with RV dysfunction are prone to bradyarrhythmias, which can further decrease cardiac output. Hemodynamic compromise from RV failure, therefore, should be treated first with volume resuscitation, restoration of physiologic rhythm or pacing, and inotropic agents. Patients with persistent RV failure can be considered for RV MCS devices.

In patients with left ventricular dysfunction, increased left ventricular pressures and pulmonary venous pressures lead to increased RV afterload, which further decreases RV output. Lala and colleagues[54] analyzed patients from the SHOCK trial, which recruited primarily patients with left ventricular failure and found that the prevalence of RV failure (ie, biventricular failure) was 38%. They defined RV failure using hemodynamic parameters: central venous pressure greater than 10 mm Hg, central venous pressure/pulmonary capillary wedge pressure greater than 0.63, pulmonary artery pulsatility index less than 2, and RV stroke work index less than 450 g*m/m^2. Using similar definitions, Basir and colleagues demonstrated similar findings in the National Cardiogenic Shock Initiative (NCSI) and identified these patients as having increased mortality compared with those with isolated left ventricular failure.

VA-ECMO is a powerful RV assist device (RVAD), and, in the setting of concomitant left sided failure, may be the preferred modality of MCS, because it provides biventricular support. Unfortunately, data on its use specifically for RV failure in the setting of AMICS are limited.

The Impella RP is a percutaneous microaxial pump designed to support the RV. There are few data demonstrating the impact of Impella RP on outcomes in patients with RV dysfunction (**Table 6**). Cheung and colleagues[55] studied 18 patients, 39% of whom had AMI and found that Impella RP led to improvements in hemodynamic measures and reported a 30-day survival rate of 72% and a 1-year survival rate of 50%. The RECOVER RIGHT study included 30 patients with RV failure refractory to medical therapy. The investigators found that patients had improvement in hemodynamics with the use of an Impella RP. Overall, 73.3% of patients survived to 30 days.[56]

TandemHeart–RVADs (TH-RVADs) use an extracorporeal centrifugal flow pump and 2 venous cannulas to deliver blood from the right atrium (RA) to the main PA via bilateral femoral venous cannulation. A 21F inflow cannula is placed in the RA and a second 21F outflow cannula is inserted into the main PA. Usually, the outflow cannula is placed in the main PA via the right femoral vein, and the inflow cannula is placed in the RA via the left femoral vein. If the distance from femoral vein to fifth intercostal space exceeds 58 cm or femoral access cannot be used, the internal jugular venous access can be utilized. There also is a ProtekDuo (LivaNova, London, UK) dual-lumen cannula, which can be placed in the right internal jugular vein. It contains 2 lumens within one 29F or 31F cannula, taking blood from RA to the extracorporeal pump then delivering it to the PA. There are few data on the use of TH-RVAD on outcomes (see **Table 5**). Kapur and colleagues[57] retrospectively studied outcomes in 46 patients with RV failure who received a TH-RVAD, of whom 21 patients were cannulated percutaneously. TH-RVAD implantation was associated with a significant decrease in RA pressure and a significant increase in CI. In-hospital mortality was 33% in patients with AMI. In another study by Kapur and colleagues,[58] 9 patients, 6 of whom had AMI, had improved hemodynamics when

Table 6
Major studies assessing acute mechanical circulatory support in right ventricular dysfunction

Study, Year Published	Device	Number of Patients	Population	Outcomes
Cheung et al,[55] 2014	Impella RP	18	39% AMI, other etiologies include post-transplant, myocarditis	30-d survival 72% 1-y survival 50% Hemodynamic effects: increased CI, decreased RA pressure
Anderson et al,[56] 2015	Impella RP	30	40% AMI, others include post-LVAD	30-d survival 73.3% Hemodynamic effects: increased CI, decreased RA pressure
Kapur et al,[57] 2013	TH-RVAD	46	25% AMI, others include post–cardiac surgery, transplant, myocarditis	In-hospital mortality 57% Hemodynamic effects: increased CI, MAP and PA, decreased RA pressure
Kapur et al,[58] 2011	TH-RVAD	9	66.7% AMI, others include post–cardiac surgery	In-hospital mortality 44% Hemodynamic effects: increased RV stroke volume, MAP and PA, decreased RA pressure
Truby et al,[65] 2015	VA-ECMO	179	26% AMI, others include post–cardiac surgery	In-hospital mortality 38.6% Hemodynamic effects: decreased RA and mean PA pressure

Abbreviations: MAP, mean arterial pressure; PA, pulmonary artery.

treated with TH-RVAD, with an in-hospital mortality rate of 44%.

SHOCK PROTOCOLS AND TEAMS

Shock protocols allow for a uniform treatment strategy in an effort to provide patients, nurses, and clinicians a systematic pathway of care,[59] although shock teams provide a diverse set of options that can be catered to the individual patient, taking into account operator and institutional expertise.[60] This concept is best exemplified in the work of the NCSI. Investigators involved in the study began by reviewing outcomes data in AMICS and forming best practices, which were put together into a shock protocol. The study was limited to evaluating outcomes in patients with AMICS and not other shock phenotypes. The study also used inclusion and exclusion criteria similar to previous RCTs in an effort to compare with prior work.

The shock protocol was piloted in metro Detroit and named the Detroit Cardiogenic Shock Initiative.[61] A 41-patient pilot study found

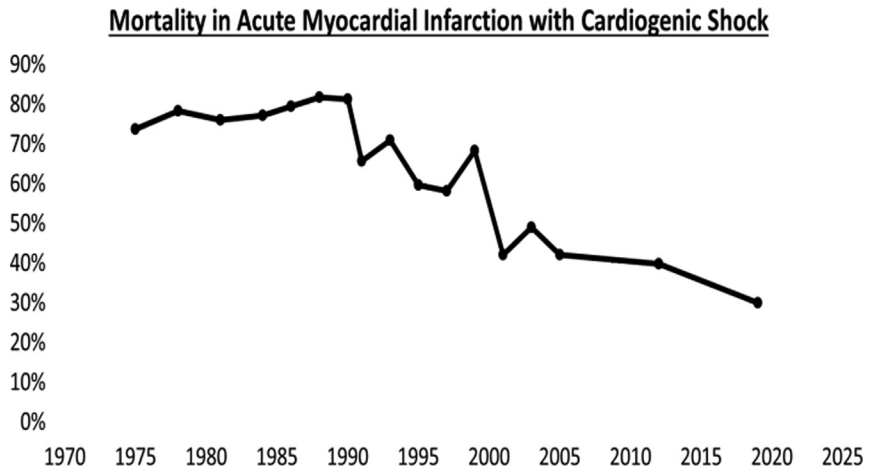

Fig. 1. The 50-year mortality trend in AMI complicated by CS. Over 50 years, mortality in AMI with CS has increased steadily from approximately 80% to close to 30%. (*Adapted from* Goldberg RJ, Spencer FA, Gore JM, Lessard D, Yarzebski J. Thirty-year trends (1975 to 2005) in the magnitude of, management of, and hospital death rates associated with cardiogenic shock in patients with acute myocardial infarction: a population-based perspective. Circulation. 2009;119(9):1211-1219; with permission.)

the protocol could be used across selected centers and was associated with high survival compared with historical studies and local outcomes. The study then was expanded and renamed the NCSI. The goal was to see if the shock protocol could be reproduced in centers across the United States. In total, greater than 60 sites were recruited with a goal of enrolling 400 patients. The NCSI is the first contemporary study to evaluate outcomes of a shock protocol. The best practices included in the protocol are[1] to identify AMICS early and treat patients in the catheterization laboratory (early is defined as <90 minutes to 120 minutes of diagnosis and prior to escalating use of inotropes)[2]; placement of Impella prior to PCI, because PCI can result in reperfusion injury, distal embolization, and transient cessation of coronary perfusion with balloon inflations and stents, which are better tolerated with MCS; and[3] use of pulmonary artery catheters to assess patients underlying hemodynamic state and to guide further therapy, including escalation of MCS, identification of RV failure, and weaning. The study has

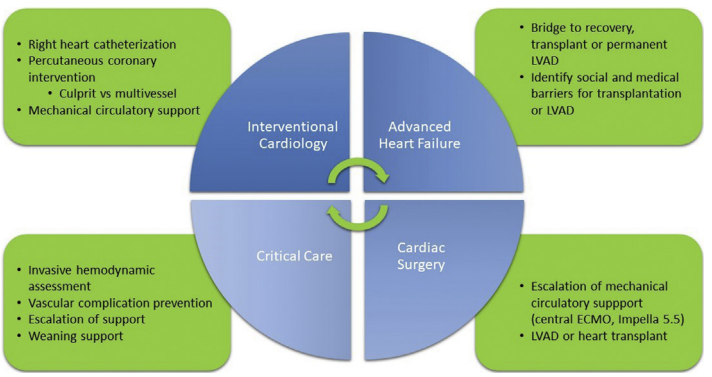

Fig. 2. Key components of a CS team. Using a shock team and protocol has been associated with improved outcomes in numerous observational studies. Early triage, prompt identification, and rapid delivery of MCS based on a patient's physiologic state are steps important in CS management. A multidisciplinary team–based approach, which includes interventional cardiology, advanced heart failure, cardiac surgery, and critical care, has proved efficient in improving outcomes without delaying care and it is highly recommended in clinical practice. Early identification of shock starts in the emergency department and the decision to send a patient to the catheterization laboratory should not be delayed, which highlights the importance of good communication between the emergency department and the cardiology team. Escalation for additional left ventricle or RV support as well as transfer to a tertiary care center (if needed) should be discussed early by the multidisciplinary team.

enrolled more than 300 patients with AMICS and has demonstrated survival to hospital discharge greater than 70%.[62,63]

SUMMARY

AMI complicated by CS is a deadly condition associated with significant morbidity and mortality (Fig. 1). Despite 20 years of medical advancements, early revascularization remains the sole therapy proved to improve outcomes. MCS devices provide a physiologically plausible mechanism of improving outcomes by offering hemodynamic stability for revascularization and improving end-organ perfusion. Results from well-powered RCTs, however, are not yet available. RCTs have been difficult to conduct in this patient population; until such trials are performed, implementing shock teams and protocols has been associated with improved outcomes in observational studies and may be considered (Fig. 2). Technological advancements will lead to continued development of more mobile, smaller-caliber, and more powerful MCS devices. Understanding the mechanism of action and physiologic effects of these devices, therefore, is critically important.

DISCLOSURE

M.B. Basir is a consultant for Abbott Vascular, Abiomed, Cardiovascular Systems, Chiesi, Procyrion and Zoll. A. Lemor and L. Ya'qoub report no conflicts of interest.

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