Prospective Comparison of a Percutaneous Ventricular Assist Device and Venoarterial Extracorporeal Membrane Oxygenation for Patients With Cardiogenic Shock Following Acute Myocardial Infarction

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Background—Cardiogenic shock (CS) following acute myocardial infarction (AMI) portends a poor prognosis. Both venoarterial extracorporeal membrane oxygenation (VA-ECMO) and a percutaneous ventricular assist device (pVAD) provide hemodynamic support for patients with CS, but little is known about the best device for this population. We sought to compare outcomes of AMI patients treated with these devices.

Methods and Results—Consecutive patients with CS following AMI from April 2015 to March 2017 were enrolled prospectively if they received either device for AMI-related CS. If patients received both devices, they were analyzed according to the first used. The primary outcome was all-cause mortality. In total, 51 patients received VA-ECMO or pVAD following AMI; 20 received VA-ECMO, and 31 received pVAD. The mean age was 62.1 ± 10.1 years, and 39 (76.5%) were men. Twenty-four (47.1%) patients were ultimately supported by both devices simultaneously (20 pVAD-first, 4 VA-ECMO-first). Patients treated with pVAD or VA-ECMO were similar in baseline characteristics at initial device insertion except that the latter were on more vasopressors and were more likely to have an intra-aortic balloon pump. Seventeen (33.3%) had recent cardiopulmonary resuscitation, mean lactate was 4.86 ± 3.96 mmol/L, and mean cardiac index was 1.70 ± 0.42 L/min per m². Of the 28 (54.9%) patients surviving to discharge, 11 had received VA-ECMO first and 17 had pVAD first (P = 0.99). Survival at 1 and 2 years did not differ significantly between device groups (P = 0.42).

Conclusions—Following AMI-related CS, pVAD- and VA-ECMO-treated patients had similar outcomes. The use of both devices simultaneously was common, with almost half of patients in persistent CS after first device deployment. (J Am Heart Assoc. 2019;8:e012171. DOI: 10.1161/JAHA.119.012171.)

Key Words: acute myocardial infarction • cardiogenic shock • extracorporeal membrane oxygenation • hemodynamics • Impella • percutaneous ventricular assist device

Cardiogenic shock (CS) remains the leading cause of early mortality following acute myocardial infarction (AMI). The rapidity with which this condition can develop makes it particularly challenging to treat effectively. Some small randomized trials have demonstrated superior hemodynamic support provided by more invasive devices when compared with the intra-aortic balloon pump (IABP), although these trials have not been powered to detect differences in patient outcomes. As a result, interest in the role of more invasive and more powerful devices has been increasing, given persistently poor outcomes for this patient population. Despite recent growth in the use of percutaneous mechanical circulatory support devices (MCSD) to support patients with CS, few randomized trials have examined the role of the different devices at the clinician’s disposal. Although this trend in increasing use has been hypothesized to be associated with improved outcomes for this condition, no device has been shown to provide clear survival benefit.

Both venoarterial extracorporeal membrane oxygenation (VA-ECMO) and the percutaneous ventricular assist device (pVAD; eg, Impella [Abiomed] and TandemHeart [TandemLife]) are more invasive means of providing greater circulatory...
support in CS. The effects on hemodynamics and cardiac work differ substantially between these 2 types of device.7 Prospective data comparing the efficacy of these more invasive devices for patients with AMI-related CS are lacking. As such, we sought to prospectively evaluate the efficacy of these device types in supporting patients with this condition, assessing overall outcomes and outcomes between devices as used through our institutional practice algorithm.

Methods
Enrollment
The data that support the findings of this study are available from the corresponding author on reasonable request. This study was approved by the Columbia University Medical Center institutional review board. Participants or their surrogates provided written informed consent. A waiver of consent was granted for those without a surrogate who were too critically ill to provide informed consent before death. All patients aged ≥18 years who were treated at our institution for CS following AMI with either pVAD (Impella) or VA-ECMO between April 1, 2015, and March 31, 2017, were prospectively approached for enrollment. The choice of initial support device was left to the discretion of the clinical team, although our institutional CS algorithm was used to guide the choice of initial device and to determine the need for a second device when necessary (Figure 1). When a second device was used, the clinical team was asked to specify the reason for this (eg, hypoxemic respiratory failure, inadequate hemodynamic support, inadequate left ventricular unloading). Those enrolled in the study were followed prospectively.

Outcomes
Data collected included demographic variables and hemodynamic data whenever available. In addition, AMI details were collected including angiographic results and cardiac biomarkers (eg, CK-MB [creatine phosphokinase–MB]). The primary outcomes were survival to hospital discharge and survival without the need for heart replacement therapy (HRT; either durable left ventricular assist device [LVAD] or heart transplant). Secondary outcomes were the need for placement of a second support device and survival at 1 and 2 years.

Statistical Analysis
Categorical data are presented as percentages, and continuous data are presented as mean±SD or median with interquartile range, as appropriate. Normality was tested using the Shapiro–Wilk test. The Pearson $\chi^2$ test was used to compute the significance of the difference between groups for categorical variables. The Student $t$ test and the Wilcoxon rank sum test were used to compare groups for continuous variables, as appropriate. Logistic regression was used to study effect size and statistical significance of potential predictors of binary outcomes of interest. Variables with $P<0.1$ in univariable analysis and those felt to be clinically important with respect to the outcome of interest were included in a multivariable model. For time-to-event analyses, Kaplan–Meier estimates of event-free survival were created for groups of interest, and the log-rank test was used to compare survivor functions. $P<0.05$ was considered statistically significant. Data were analyzed using Stata (StataCorp).

Results
Patients and Initial Therapies
A total of 53 patients received either a pVAD or VA-ECMO for CS following AMI; 2 patients declined enrollment in this trial, and of the remaining 51 patients, 31 (60.8%) received a pVAD as the initial device, whereas 20 (39.2%) received VA-ECMO first. The mean age was 62.1±10.1 years, and 39 (76.5%) patients were men. Nine patients (17.7%) were undergoing active cardiopulmonary resuscitation at the time of device initiation, and another 17 (33.3%) had undergone recent cardiopulmonary resuscitation with successful resuscitation. Thirty-five patients (68.6%) were mechanically ventilated at the time of device insertion, and 23 (45.1%) had an IABP.
before either pVAD or VA-ECMO initiation. Twenty-six patients (51.0%) had been transferred to our institution after initial treatment (including percutaneous coronary intervention and MCSD initiation) for the AMI. Of these, 2 (7.7%) were transported on medications alone, 8 (30.8%) were transported on an IABP, 4 (15.4%) were transported on VA-ECMO (2 of these also with IABP), 5 (19.2%) were transported on pVAD, and 7 (26.9%) were transported on both VA-ECMO and pVAD. The initial MCSD did not differ between those presenting to our institution and those transferred to our institution from another ($P=0.65$). In all instances of pVAD use, patients were treated with the Impella CP device except for 3 who received the Impella RP device as the first device deployed for right ventricular infarction.

A comparison of baseline characteristics of patients who received pVAD versus VA-ECMO as the first support device is displayed in Tables 1 and 2. Patients who initially received VA-ECMO tended to have a higher prevalence of cardiovascular comorbidities including diabetes mellitus, hypertension, and prior cerebrovascular accident, although these differences were not statistically significant. At the time of initial device insertion, patients who received VA-ECMO were on a higher number of vasopressors than those who received pVAD first ($1.9 \pm 0.9$ versus $1.4 \pm 0.8$, $P=0.03$). VA-ECMO patients were also more likely to have an IABP at the time of support initiation ($75.0\%$ versus $25.8\%$, $P=0.001$).

All patients underwent coronary angiography. Six patients (11.8%) presented with a non–ST-segment–elevation myocardial infarction, whereas the remaining 45 (88.2%) presented with an ST-segment–elevation myocardial infarction. The left main or left anterior descending artery was the infarct vessel in 34 patients (66.7%). Patients had, on average, 2.2 (SD 0.8) epicardial coronary vessels diseased (defined as a stenosis >50%). TIMI (Thrombolysis in Myocardial Infarction) grade 3 flow was achieved in 35 (70.0%) infarct vessels, whereas 15 (30.0%) had TIMI grade <3 flow despite attempted revascularization. Two patients (6.5%) treated with pVAD and 6 (30.0%) treated with VA-ECMO initially underwent device

Figure 1. Institutional cardiogenic shock algorithm. CO indicates cardiac output; IABP, intra-aortic balloon pump; INTERMACS, Interagency for Mechanically Assisted Circulatory Support; LV, left ventricle; LVAD, left ventricular assist device; OR, operating room; pVAD, percutaneous ventricular assist device; RV, right ventricle; RVAD, right ventricular assist device; VA-ECMO, venoarterial extracorporeal membrane oxygenation.
placement after unsuccessful percutaneous coronary intervention. Of these, 4 (50.0%) were transferred to our institution on the support device after this attempt at revascularization.

Thirty-nine patients (76.4%) underwent an invasive hemodynamic assessment with pulmonary artery catheterization before the first device initiation. Among this subset of our cohort, the mean right atrial, pulmonary artery systolic and diastolic, and pulmonary capillary wedge pressures were similar between the pVAD and VA-ECMO groups (Table 2). Before MCSD support, the mean cardiac indexes for those receiving VA-ECMO and pVAD as the initial device were 1.84 ± 0.50 and 1.61 ± 0.34 L/min per m², respectively (P=0.11). The mean cardiac power output and index for the study cohort were 0.47 ± 0.18 W and 0.24 ± 0.09 W/m² and were nearly identical between these groups, whereas patients were receiving, on average, 2.5 ± 1.1 inotropic or vasopressor medication infusions, in addition to those previously mentioned who were already supported by IABP.

**Secondary Therapies**

Of those receiving pVAD first, 20 (64.5%) subsequently received VA-ECMO (while remaining with pVAD), and of those receiving VA-ECMO first, 4 (20.0%) subsequently received pVAD (Figure 2) based on clinical deterioration and as per criteria from our institutional algorithm. Two patients receiving pVAD had immediate insertion of VA-ECMO following pVAD. The hemodynamic profiles of the remaining 22 patients who had a second device inserted with a separate procedure are presented in Table 3. In 1 patient, VA-ECMO was added after pVAD because of hypoxemic respiratory failure, but in the remaining patients, it was added because of the need for a greater degree of hemodynamic support. Following pVAD insertion, these patients had mean arterial blood pressure of 66.3 mm Hg (56.7–73.7), cardiac index of 1.87 L/min per m² (1.59–2.35), and a cardiac power index of 0.28 W/m² (0.21–0.36) while receiving 3 (2.5–3.5) vasoactive and inotropic medications. The median doses of each vasopressor and inotropic infusion among this cohort of patients receiving a second device are listed in Table 3. Of those patients receiving VA-ECMO first and then a pVAD, all 4 (100%) received the second device because of inadequate unloading of the left ventricle.

Using logistic regression to analyze patients who were treated with VA-ECMO after initial treatment with pVAD, mean arterial blood pressure, intra-cardiac filling pressures, cardiac

**Table 1. Patient Demographics**

| Variable                              | All (N=51) | pVAD First (n=31) | ECMO First (n=20) | P Value |
|---------------------------------------|------------|-------------------|-------------------|--------|
| Age, y                                | 62.1±10.1  | 60.8±10.6         | 64.3±9.1          | 0.23   |
| Male sex                              | 39 (76.5)  | 23 (74.2)         | 16 (80.0)         | 0.63   |
| Hypertension                          | 30 (58.8)  | 16 (51.6)         | 14 (70.0)         | 0.19   |
| Diabetes mellitus                     | 23 (45.1)  | 12 (38.7)         | 11 (55.0)         | 0.25   |
| CVA                                   | 9 (17.7)   | 4 (12.9)          | 5 (25.0)          | 0.27   |
| Recent CPR                            | 17 (33.3)  | 9 (29.0)          | 8 (40.0)          | 0.42   |
| Active CPR                            | 9 (17.7)   | 5 (16.1)          | 4 (20.0)          | 0.72   |
| Mechanically ventilated               | 35 (68.6)  | 19 (61.3)         | 16 (80.0)         | 0.16   |
| Creatinine, mg/dL                     | 1.44±56    | 1.34±0.51         | 1.60±0.61         | 0.11   |
| Bicarbonate, mmol/L                   | 20.5±4.1   | 20.7±4.4          | 20.0±3.6          | 0.54   |
| Hemoglobin, g/dL                      | 12.4±2.4   | 12.7±1.9          | 11.8±2.9          | 0.20   |
| Platelet, ×10³/µL                     | 203.0±75.7 | 216.4±71.1        | 181.3±79.8        | 0.11   |
| Lactate, mmol/L                       | 4.86±3.96  | 4.40±3.03         | 5.49±4.97         | 0.37   |
| Aspartate aminotransferase, U/L       | 391.3±433.8| 357.3±387.8       | 444.9±504.5       | 0.50   |
| pH                                    | 7.30±0.14  | 7.30±0.12         | 7.29±0.18         | 0.66   |
| International normalized ratio        | 1.46±0.65  | 1.54±0.81         | 1.32±0.20         | 0.23   |
| IABP                                  | 23 (45.1)  | 8 (25.8)          | 15 (75.0)         | 0.001  |
| LM/LAD infarct vessel                 | 34 (66.7)  | 18 (58.1)         | 16 (80.0)         | 0.11   |
| Number of coronary vessels stenosed   | 2.2±0.8    | 2.3±0.8           | 2.0±0.8           | 0.26   |

Data are shown as mean±SD or n (%). CPR indicates cardiopulmonary resuscitation; CVA, cerebrovascular accident; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LM/LAD, left main or left anterior descending; pVAD, percutaneous ventricular assist device.

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index, and cardiac power index did not predict the addition of VA-ECMO. Instead, recent cardiac arrest and left main or left anterior descending artery infarct vessel were significant predictors of this addition. In a multivariable model controlling for age and serum lactate, left main or left anterior descending artery infarct vessel remained the lone independent predictor of the addition of VA-ECMO after pVAD treatment (odds ratio: 12.9; 95% CI, 1.2-134.1; P=0.03).

### Short- and Long-Term Outcomes
Overall, 28 patients (54.9%) survived to discharge, and 7 (13.7%) required HRT in the form of durable LVAD. Patient outcomes by device type used are displayed in Figure 3. Of those who received pVAD first, 17 (54.8%) survived to discharge and 4 (12.9%) required HRT, whereas among those who received VA-ECMO first, 11 (55.0%) survived to discharge and 3 (15.0%) required HRT. Of those who

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**Table 2. Patient Hemodynamic Profiles**

| Variable                  | All (N=51)       | pVAD First (n=31) | ECMO First (n=20) | P Value |
|---------------------------|------------------|-------------------|-------------------|---------|
| Pulse, beats/min          | 92.8±20.0        | 89.9±20.5         | 97.5±18.8         | 0.19    |
| Systolic blood pressure, mm Hg | 88.4±14.3       | 88.2±14.3         | 88.7±14.7         | 0.92    |
| Diastolic blood pressure, mm Hg | 57.8±11.9       | 58.7±11.9         | 56.3±7.6          | 0.43    |
| Mean arterial pressure, mm Hg | 68.0±10.0       | 68.6±11.1         | 67.1±7.9          | 0.62    |
| Right atrial pressure, mm Hg | 14.1±6.0        | 13.3±6.7          | 15.0±5.2          | 0.39    |
| Systolic PA pressure, mm Hg | 42.6±11.4       | 42.9±12.9         | 42.2±9.4          | 0.87    |
| Diastolic PA pressure, mm Hg | 23.9±7.5        | 23.8±9.0          | 24.1±5.0          | 0.91    |
| Pulmonary wedge pressure, mm Hg | 24.2±9.3        | 24.5±9.5          | 23.7±9.3          | 0.82    |
| PA pulsatility index      | 1.8±1.6          | 2.2±2.1           | 1.4±0.6           | 0.13    |
| Cardiac output, L/min     | 3.32±0.88        | 3.11±0.69         | 3.62±1.06         | 0.10    |
| Cardiac index, L/min/m²   | 1.70±0.42        | 1.61±0.34         | 1.84±0.50         | 0.11    |
| Cardiac power output, W   | 0.47±0.18        | 0.48±0.12         | 0.47±0.24         | 0.87    |
| Cardiac power index, W/m² | 0.24±0.09        | 0.25±0.06         | 0.24±0.12         | 0.79    |
| Number of inotropes       | 1.0±0.6          | 1.0±0.7           | 1.0±0.6           | 0.92    |
| Number of vasopressors    | 1.6±0.9          | 1.4±0.8           | 1.9±0.9           | 0.03    |

Data are shown as mean±SD. ECMO indicates extracorporeal membrane oxygenation; PA, pulmonary artery; pVAD, percutaneous ventricular assist device.

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**Figure 2.** Patient outcomes. Survival to discharge is displayed by devices employed. pVAD, percutaneous ventricular assist device; VA-ECMO, venoarterial extracorporeal membrane oxygenation.
received only pVAD, 7 (63.6%) survived to discharge and none required HRT. Of those who received only VA-ECMO, 8 (50.0%) survived to discharge and 2 (12.5%) required HRT. Finally, of those who were treated with both devices, 13 (54.2%) survived to discharge and 5 (20.8%) required HRT. Rates of survival to discharge did not differ significantly by ultimate device-treatment strategy (50.0% for VA-ECMO alone, 63.6% for pVAD alone, and 54.2% for dual-device support; \( P=0.78 \)).

The 1- and 2-year Kaplan–Meier survival estimates did not differ between those who received pVAD or VA-ECMO as the initial support device (Figure 4). One- and 2-year survival for those surviving to discharge was 92.2% and 87.8%, respectively; following discharge, 3 patients died (3 VA-ECMO first, no pVAD first). Median follow-up for those surviving to discharge was 687 days (interquartile range: 300–899) and did not differ between those treated with VA-ECMO first (median: 687 days; interquartile range: 225–924) and pVAD first (median: 686 days; interquartile range: 409–896; \( P=0.93 \)). All patients who survived to discharge without requiring HRT were free from requiring durable LVAD or heart transplant at latest follow-up.

Among those patients (24, 47.1%) with recent or active cardiopulmonary resuscitation at the time of initial device deployment, there was no difference in the rate of survival to discharge between those receiving VA-ECMO or pVAD first (54.5% versus 46.2%, \( P=0.68 \)). Similarly, among those with an IABP present before the first use of VA-ECMO or pVAD, there was no difference in survival to discharge between the 2 groups (60.0% versus 50.0%, \( P=0.69 \)). Among those patients initially presenting at our institution, there was no difference in survival depending on which device was used first (\( P=0.57 \)). Last, among those initially presenting to another institution and then subsequently transferred to ours, there was similarly no difference in survival depending on which device was used first (\( P=0.38 \)).

### Complications

A total of 9 patients (17.6%) had a stroke during their hospital course; 4 (12.9%) were treated with pVAD first and 5 (25.0%) with VA-ECMO first (\( P=0.27 \)). In addition, 4 patients (12.9%) receiving VA-ECMO first and none (0%) receiving pVAD first were treated for bacteremia. During

### Table 3. Patient Hemodynamic at Time of Addition of Second Device

| Variable                        | All (N=22)          | pVAD First (n=18) | ECMO First (n=4) |
|---------------------------------|---------------------|-------------------|------------------|
| Pulse, beats/min                | 95 (77.5–108.5; 20) | 94.5 (77–115; 16) | 98 (86–101; 4)   |
| Systolic blood pressure, mm Hg  | 88 (70–96; 21)      | 79 (70–92; 17)    | 105 (80.5–109.5; 4) |
| Diastolic blood pressure, mm Hg | 57 (50–67; 21)      | 58 (54–67; 17)    | 51 (50–62; 4)    |
| Mean arterial pressure, mm Hg  | 67 (56.7–76; 21)    | 66.3 (56.7–76; 17)| 68.8 (60–77.8; 4) |
| Right atrial pressure, mm Hg   | 13 (10–17; 14)      | 12 (10–20; 10)    | 13.5 (10–15.5; 4) |
| Systolic PA pressure, mm Hg     | 35 (33–48; 14)      | 34 (28–48; 10)    | 34.5 (33.5–42; 4) |
| Diastolic PA pressure (mmHg)    | 20 (17–24; 14)      | 20 (17–24; 10)    | 20.5 (17–24.5; 4) |
| Pulmonary wedge pressure, mm Hg | 20 (17–33; 7)       | 19 (17–24; 6)     | 33 (33–33; 1)    |
| Cardiac output, L/min           | 4.20 (3.67–4.90; 13)| 3.84 (3.23–4.30; 10)| 4.90 (4.4–7.44; 3) |
| Cardiac index, L/min/m²         | 2.00 (1.65–2.41; 13)| 1.87 (1.59–2.35; 10)| 2.43 (2.14–3.56; 3) |
| Cardiac power output, W         | 0.58 (0.45–0.66; 13)| 0.56 (0.43–0.63; 10)| 0.66 (0.58–1.17; 3) |
| Cardiac power index, W/m²       | 0.29 (0.22–0.36; 13)| 0.28 (0.21–0.36; 10)| 0.32 (0.29–0.56; 3) |
| Number of vasoactive infusions  | 3 (2.5–3.5; 20)     | 3 (2.5–3.5; 16)   | 3.0 (2–3.5; 4)   |
| Norepinephrine dose, µg/min     | 15.0 (6–30; 14)     | 15.0 (4–30; 11)   | 30.0 (6–32; 3)   |
| Vasopressin dose, U/h           | 2.4 (2.4–3.4; 12)   | 2.4 (2.4–2.4; 9)  | 6.0 (6–6; 3)     |
| Phenytoin dose, µg/min          | 200.0 (200–200; 5)  | 200.0 (200–200; 5)| NA (0)          |
| Dobutamine dose, µg/kg/min      | 2.5 (2.5–3; 9)      | 2.5 (2.5–3; 7)    | 3.8 (2.5–5; 2)   |
| Dopamine dose, µg/kg/min        | 10.0 (7–20; 11)     | 10 (8–20; 9)      | 6.5 (3–10; 2)    |
| Milrinone dose, µg/kg/min       | 0.25 (0.13–0.38; 3) | 0.19 (0.13–0.25; 2)| 0.38 (0.38–0.38; 1)|
| Epoprostenol dose, µg/min       | 3.0 (1–10; 7)       | 3.0 (1–10; 7)     | NA (0)          |

Data are shown as median (interquartile range; n). ECMO indicates extracorporeal membrane oxygenation; NA, not assessed; PA, pulmonary artery; pVAD, percutaneous ventricular assist device.
the hospital course, blood transfusions were given to 41 patients (80.4%). Patients treated with pVAD first received 5 U of packed red blood cells, whereas those with VA-ECMO first received 8 U (interquartile range: 2.5–11.5; \( P = 0.56 \)). Ten patients (50.0%) treated with VA-ECMO had a distal perfusion cannula inserted to perfuse the ipsilateral extremity. Six patients (11.8%) required intervention for vascular injury or limb ischemia; 4 (12.9%) had received pVAD first, and 2 (20.0%) had received VA-ECMO first. Two patients had fasciotomies after developing a compartment syndrome, 2 had primary repair of a vascular injury related to the support device(s), 1 had thrombectomy, and 1 had a below-knee amputation.

Of those patients who did not survive to discharge, 10 (43.5%) died of multiorgan failure, 8 (34.8%) died of cardiovascular causes, 2 (8.7%) died of anoxic brain injury, and 3 died of sepsis (13.0%). The causes of death did not differ between initial device support received (\( P = 0.61 \)).

**Discussion**

To our knowledge, this article presents the first prospective study comparing the efficacy of VA-ECMO and a pVAD for patients with CS following AMI. This cohort of patients carried a high degree of acuity, with more than half having suffered a recent cardiac arrest or undergoing active cardiopulmonary resuscitation, the majority mechanically ventilated, and the mean cardiac power index significantly lower than that observed in the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock) trial registry.\(^8\) Our principal findings are as follows. First, despite the use of mechanical circulatory support devices capable of providing a high degree of hemodynamic support including pVAD, VA-ECMO, or both in combination, short-term mortality remained high for this critically ill cohort of patients who developed CS following AMI. Second, of those surviving, a quarter required a bridge to HRT, whereas three quarters had sufficient ventricular recovery to be weaned from MCSD with good long-term

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**Figure 3.** Patient outcomes by device support type. **A,** Survival to discharge is displayed according to the initial device used. **B,** Survival to discharge free of durable LVAD is displayed according to the initial device used. **C,** Survival to discharge is displayed according to the ultimate device configuration used. **D,** Survival to discharge free of durable LVAD is displayed according to the ultimate device configuration used. LVAD indicates left ventricular assist device; pVAD, percutaneous ventricular assist device; ECMO, extracorporeal membrane oxygenation.
found similarly poor outcomes for each.5 However, interest in and the IABP for patients with advanced CS following AMI and Complicated by Cardiogenic Shock) trial compared a pVAD Intra-Aortic Balloon Pump in Acute Myocardial Infarction Most recently, the randomized IMPRESS (Impella CP Versus – ECMO has been increasing, given persistently poor outcomes for this patient population.9 Although the use of VA-ECMO and pVAD have increased for AMI-related CS, data comparing their efficacy are sparse and limited to retrospective analysis.10–15 In addition, such reports have included multiple etiologies of CS, whereas it has been increasingly recognized that different etiologies of CS have different prognoses.16 Our data are novel in that they represent a prospective analysis with only patients experiencing CS following AMI. The data demonstrate that the devices are associated with similar short- and long-term outcomes when used to support patients with AMI-related CS. Without randomization, selection bias influenced the choice of one device or the other as the initial support for patients, guided by our institutional algorithm for CS. In comparing patients who received VA-ECMO or pVAD as the initial device, a few differences are important to note that further inform the interpretation of our results. Notably, although invasive hemodynamics were similar between the 2 cohorts, patients receiving VA-ECMO first were on more vasoactive agents to maintain these hemodynamics and were more likely to have an IABP before initiation of one of the devices in this study. Seemingly, the cohort treated with VA-ECMO initially had a greater degree of compromise based on these findings.

Perhaps the most striking finding in these data is the prevalence of device use in combination, with almost half of the cohort being supported by both devices concomitantly. The use of multiple high-dose vasoactive medications is associated with poor outcomes.17,18 This may represent an association between greater degrees of hemodynamic compromise and increased mortality, but also it may be related causally to the deleterious effects of these substances on myocardial oxygen demand. Therefore, we aim to achieve a strategy of MCSD support that allows us to minimize these medications. An inability to decrease dosages or a continued rise in dosages is regarded as an indication that the current level of mechanical support is inadequate. Close examination of the hemodynamic profiles of our patients after the first device deployment demonstrated that a significant proportion remained in frank CS, dependent on numerous high-dose vasopressors or inotropes. Consequently, a second device was deployed frequently. In the most advanced cases of CS, either device alone may be insufficient to correct the hemodynamic insults following AMI. The utility of device use in combination for patients with severe CS has been recognized recently.19,20 Furthermore, the dual-device approach allows for a stepwise de-escalation of support as the patients recovers. Based on these data, our own center has developed a heightened awareness of the need for a second device and maintains close hemodynamic monitoring in the early period following initial device deployment to minimize time to escalation if a second device is necessary. Another notable finding from our data is the use of HRT, such as fully implantable LVADs. Durable LVAD implantation has emerged as a means of supporting patients whose end-
organ function and overall clinical status are not severely affected by the AMI but who sustain significant myocardial injury to prevent weaning from MCSD.21–23 Such patients, stabilized on a short-term MCSD like pVAD or VA-ECMO, may be bridged to a durable device with bridge to transplant, bridge to recovery, or destination therapy intention. This strategy may increase the proportion of patients surviving to hospital discharge after AMI-related CS. However, ventricular recovery—specifically, early improvement in ventricular function sufficient to allow for weaning from MCSD without the need for HRT—remains the preferred option, given the potential complications associated with HRT. The majority of survivors in our patient cohort did not require HRT. More important, long-term outcomes for survivors were excellent, without the need for HRT following discharge. This approach is in striking contrast to the outcomes of patients with chronic systolic heart failure who require VA-ECMO support for an episode of CS, for which the vast majority of survivors do require HRT.24

The most notable limitation for this study was that patients were not randomized to one device or the other, resulting in selection bias in the choice of device employed first. Furthermore, a number of patients were transferred to our institution after initiation of support with one of these devices at the transferring institution. Although the survival rates are similar to those of other reports of CS patients, ours is notable for the patient acuity, with the cardiac power index roughly two thirds of that reported in the SHOCK registry.6 In addition, our sample size is limited, and ours is a single-center experience with potential bias in our practice pattern. A significant impediment to CS trials of patients following AMI has been poor enrollment. Despite the lack of a clear benefit to pVAD or VA-ECMO use in CS, randomized trials have been particularly challenging to conduct with this patient population.22 In the largest CS trial to date, the cross-over rate to IABP use for patients not randomized to the device arm was relatively high.6 Therefore, to compare a device-based strategy with one without device support may not represent a real-world experience. Instead, we chose to conduct a non randomized prospective study comparing commonly used devices capable of providing a greater degree of hemodynamic support than the IABP. Although definitive conclusions about one device’s performance over another cannot be drawn from our data, the lack of an appreciably superior device among these provides clear justification for a randomized controlled trial comparing them.

Conclusions
In a prospective trial of patients with AMI-related CS, pVAD and VA-ECMO were associated with similar short- and long-term outcomes, although those receiving the latter device as initial treatment may have had worse hemodynamic compromise. A quarter of survivors required durable LVAD, and those surviving to discharge had good long-term outcomes. However, despite the use of these devices, a substantial proportion of patients remained in CS on numerous high-dose vasopressor or inotropic medications, resulting in the frequent use of both devices concomitantly. For the most advanced cases of AMI-related CS, a single device may be insufficient to correct the hemodynamic compromise. Without a clear benefit of one device over another, a randomized trial comparing these devices is justified.

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