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Clinical Investigation

Effects of Escalating Temporary Mechanical Circulatory Support in Patients With Worsening Cardiogenic Shock

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Background: *Cardiogenic shock–related mortality is substantial, and temporary mechanical circulatory support (MCS) devices are frequently used. The authors aimed to describe patient characteristics and outcomes in patients with worsening cardiogenic shock requiring escalation of temporary MCS devices.*

Methods: *Worsening cardiogenic shock was defined as persistent hypotension, increasing doses of vasopressors/inotropes, worsening hypoperfusion, or worsening invasive hemodynamics. Escalation of temporary MCS devices was defined as adding or exchanging an existing MCS device. Variables were evaluated by logistic regression models and receiver operating characteristic curves.*

Results: *From July 1, 2016, to July 1, 2018, a total of 81 consecutive patients experienced worsening cardiogenic shock requiring temporary MCS escalation. The etiology of cardiogenic shock was heterogeneous (33.3% acute myocardial infarction and 61.7% decompensated heart failure). Younger age (<62 years), lower body mass index (<28.7 kg/m^2), lower preescalation lactate levels (<3.1 mmol/L), higher postescalation blood pressure (>85 mm Hg), and lower postescalation lactate levels (<2.9 mmol/L) were associated with greater odds of survival. The presence of a pulmonary artery catheter at the time of escalation was associated with greater odds of survival (P = .05). Escalation of temporary MCS in Society for Cardiovascular Angiography and Interventions stage E shock was associated with 100% mortality (P = .05). The rate of overall survival to discharge was 32%.*

Conclusion: *Patients requiring temporary MCS escalation represent a high-risk cohort. Further work is needed to improve outcomes in this patient population.* ***(Tex Heart Inst J. 2022;49(6):e217615)***

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Cardiogenic shock (CS) is associated with substantial morbidity and mortality and is increasing in incidence.[1,2] Of patients presenting with acute myocardial infarction (AMI), 5% to 10% experience CS, with a historical mortality rate of 40% to 50%.[3] Considerably less is known about long-term outcomes in patients presenting with non-AMI CS. Revascularization in AMI is the only therapy proven to improve survival in CS. Technologic advancements have led to the availability of multiple temporary mechanical circulatory support (tMCS) devices. To date, there have been no adequately powered randomized control trials demonstrating improved survival with the use of these devices. Mechanical circulatory support devices incur substantial cost and resource use.[4] Despite growing experience, many factors related to appropriateness and timing of tMCS use and escalation remain unknown. The need for tMCS escalation is likely a marker of poor outcomes in this high-risk cohort, but this patient population has not been studied to confirm this. Further risk stratification to identify patients who might benefit from tMCS escalation, or in whom tMCS may be futile, is needed to guide clinical decision-making. The authors aimed to describe the characteristics and clinical outcomes of patients presenting with worsening CS requiring tMCS escalation.

Patients and Methods

Study Population

Between July 1, 2016, and July 1, 2018, a total of 446 patients had tMCS devices placed in the study cardiac catheterization laboratory. The use of tMCS was for CS, high-risk percutaneous coronary intervention (PCI), and other indications. The electronic medical records of these patients were reviewed, and 81 patients who underwent escalation of existing tMCS in the setting of worsening CS were identified.

Escalation of tMCS was defined as exchanging a tMCS device with a tMCS device that provides greater flow or adding a tMCS device to augment an existing tMCS device. Worsening CS was defined as any 1 of the following despite the presence of an initial tMCS device: persistent hypotension (systolic blood pressure [SBP] <90 mm Hg), increasing doses of vasopressors/inotropes, worsening end-organ perfusion parameters (persistent elevation in creatinine/blood urea nitrogen, aspartate transaminase/alanine transaminase, or lactic acid [LA]), or worsening invasive hemodynamics (cardiac output [CO], cardiac index, cardiac power output [CPO], or pulmonary artery pulsatility index [PAPi]). Clinical outcomes and patient characteristics of survivors vs nonsurvivors were compared. The study was approved by the institutional review board of Henry Ford Health System.

Baseline and Admission Characteristics

Demographic data were extracted from electronic medical records via chart review and included birth date, sex, and race. Data on height, weight, and body mass index (BMI) closest to the index date (defined as the date of tMCS escalation) were similarly extracted. Baseline comorbidity information as known before the index hospitalization was ascertained in a similar fashion.

Admission and hospitalization characteristics were obtained for all patients. Hemometabolic parameters were obtained pre– and post–tMCS escalation; preescalation was defined as the most recent measurements before tMCS escalation, and postescalation was defined as 24 hours postescalation. The Society for Cardiovascular Angiography and Interventions (SCAI) CS Classification System at the time of escalation was applied.[5] Appropriate candidates were evaluated for advanced heart failure therapies (durable left ventricular assist device [LVAD] and transplant) during the index hospitalization.

Escalation Characteristics

Patients with CS at the institution studied are routinely discussed in an informal shock care team that includes clinicians from interventional cardiology, advanced heart failure and transplant cardiology, and cardiac surgery. Acute myocardial infarction CS is typically treated using the National Cardiogenic Shock Initiative protocol.[6] Given the heterogeneity of patients with CS, differing etiologies of shock, and other individual patient characteristics, there is no protocolized escalation pathway in routine use. Routine hemometabolic parameters, including blood pressure, vasopressor/inotrope requirements, and end-organ perfusion parameters, were used to guide therapy in all patients requiring tMCS escalation. For patients with a pulmonary artery catheter (PAC) in place at the time of escalation, additive invasive hemodynamic parameters were also used to guide escalation of therapy, including cardiac index, CPO, PAPi, and cardiac output deficit (COD).[6-8] Cardiac output deficit was defined as (target cardiac output [CO] – actual CO), where "target CO" is calculated using target cardiac index and equals (2.2 × body surface area) and "actual CO" is obtained via Fick measurement.

Devices used included intra-aortic balloon pump (IABP), Impella 2.5, Impella CP, Impella 5.0, Impella RP (Abiomed), TandemHeart (LVAD, LivaNova), and ProtekDuo (right ventricular assist device, LivaNova) as well as extracorporeal membrane oxygenation (ECMO). Intraprocedural escalation was defined as tMCS escalation occurring before leaving the cardiac catheterization laboratory during the index procedure. Postprocedural escalation was defined as tMCS escalation occurring after the patients has left the cardiac catheterization laboratory following initial tMCS placement. On hemometabolic stabilization, weaning of tMCS was attempted. Weaning was individualized without a predefined protocol and typically guided by invasive hemodynamics, echocardiography, and metabolic perfusion parameters. Large-bore tMCS devices are typically removed in the cardiac catheterization laboratory. Complication rates assessed were those associated with tMCS devices and included stroke (ischemic or hemorrhagic), substantial bleeding (defined as need for blood transfusion), limb ischemia, and the use of antibiotics in the setting of suspected infection with or without the presence of an infectious source.

Statistical Analysis

Group comparisons were performed using χ^2 tests for nonsparse categorical variables, Fisher exact tests for sparse categorical variables, 2-sample *t* tests for normally distributed numerical variables, and Wilcoxon rank sum tests for nonnormally distributed numerical variables, and a paired *t* test was used to compare hemodynamics pre– and post–tMCS escalation. Univariate logistic regression models (with 95% CIs) were used to assess variables associated with in-hospital survival in patients with tMCS escalation. In addition, receiver operating characteristic curves were created for variables associated

with in-hospital survival. A 2-sided *P* value <.05 was deemed statistically significant. Statistical analyses were carried out using SAS v 9.1 (SAS Institute).

Results

Between July 1, 2016, and July 31, 2018, 81 consecutive patients were included in this study (61 [14.2] years, 72.8% male) (Fig. 1). Baseline characteristics listed in Table I. Survivors were younger and had a lower BMI than nonsurvivors. The proportion of women was similar in both groups.

Admission and hospitalization characteristics are listed in Table II. The etiology of shock was heterogeneous (33.3% AMI and 61.7% acute decompensated heart failure). Further, 61.7% of patients were transferred from outside hospitals to the institution under study, and 42.0% had their initial tMCS device placed before transfer. In addition, 77.5% of patients were on vasopressors or inotropes preescalation. Shock on admission (at the time of initial medical contact compared with the development of shock later during index hospitalization) was present in 63.0% of patients, with more nonsurvivors presenting with shock than did survivors (72.7% vs 42.3%, respectively; *P* = .01). Ninety-one percent of patients with AMI underwent revascularization. Regarding SCAI CS classification, 7.4% of patients had SCAI stage C shock at the time of escalation and typically experienced escalation because of worsening invasive hemodynamic parameters (CPO and PAPi). The majority of the study cohort comprised patients with SCAI stage D shock (81.5%) at the time of escalation. Further, 11.1% had SCAI stage E shock, and most of those patients experienced intermittent or persistent cardiac arrest at the time of escalation. The percentage of survivors decreased progressively with each SCAI shock stage (Fig. 2).

Seven patients received advanced heart failure therapies (durable LVAD, n = 3; cardiac transplant, n = 1; and durable LVAD followed by cardiac transplant, n = 3), 5 of whom survived. All durable LVADs were placed

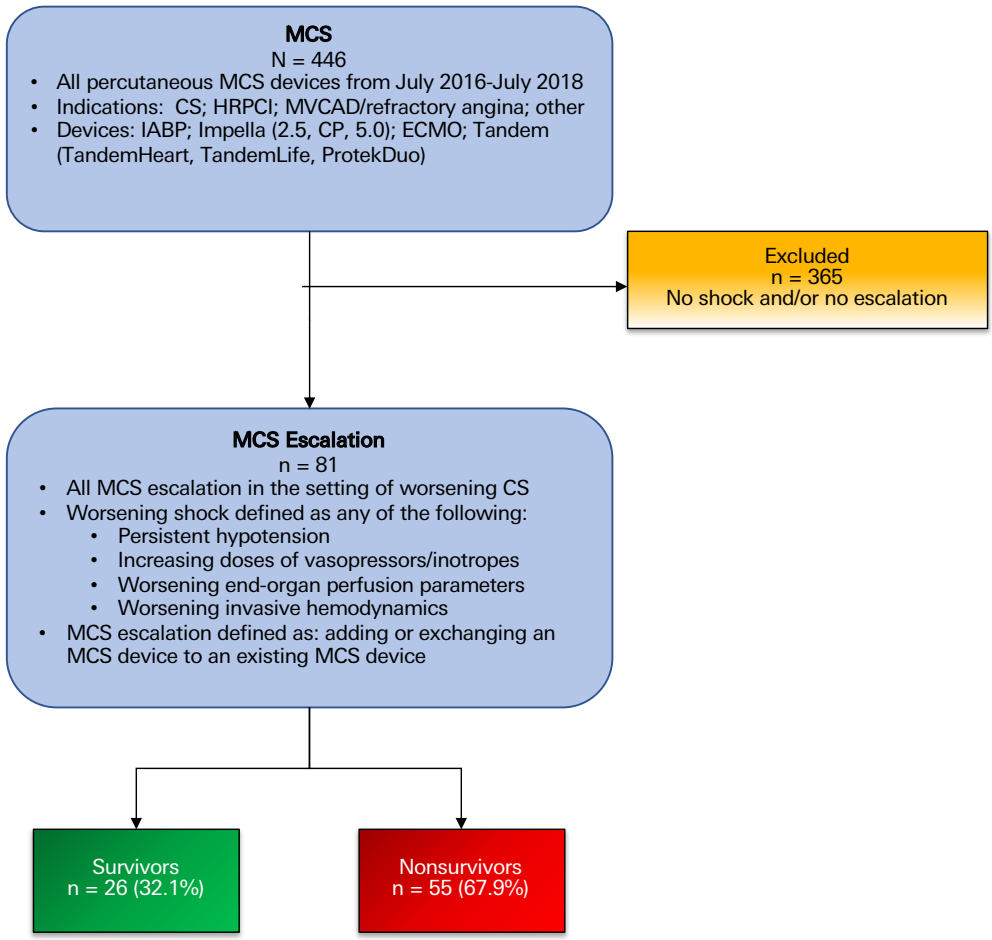

Fig. 1 *Flowchart shows selection of patients included in the study.*

CP, cardiac power; CS, cardiogenic shock; ECMO, extracorporeal membrane oxygenation; HRPCI, high-risk percutaneous coronary intervention; IABP, intra-aortic balloon pump; MCS, mechanical circulatory support; MVCAD, multivessel coronary artery disease.

TABLE I. Baseline Characteristics

	All (N = 81)	Survivors (n = 26)	Nonsurvivors (n = 55)	*P* value[a]
Age, mean (SD), y	61 (14.2)	55 (13.8)	65 (13.5)	<.01
Male, No. (%)	59 (72.8)	20 (76.9)	39 (70.9)	.79
BMI, mean (SD), kg/m^2	32.0 (8.8)	29.1 (9.1)	33.6 (8.4)	.03
Race, No. (%)				
White	63 (77.8)	18 (69.2)	45 (81.8)	.35
Black	14 (17.3)	6 (23.1)	8 (14.5)	.36
Other/unknown	4 (4.9)	2 (7.7)	2 (3.6)	.24
Diabetes mellitus, No. (%)	34 (42.0)	9 (34.6)	25 (45.5)	.47
Hypertension, No. (%)	53 (65.4)	15 (57.7)	38 (69.1)	.33
Renal insufficiency, No. (%)	11 (13.6)	2 (7.7)	9 (16.4)	.49
Dialysis, No. (%)	2 (2.5)	0 (0.0)	2 (3.6)	.99
CVD, No. (%)	9 (11.1)	0 (0.0)	9 (16.4)	.05
Moderate valvular disease, No. (%)	11 (13.6)	3 (11.5)	8 (14.5)	.99
AVD, No. (%)	5 (6.2)	1 (3.8)	4 (7.3)	.99
MVD, No. (%)	6 (7.4)	2 (7.7)	4 (7.3)	.99
CAD, No. (%)	55 (67.9)	15 (57.7)	40 (72.7)	.21
Prior PCI, No. (%)	41 (63.0)	11 (42.3)	30 (54.5)	.35
Prior CABG, No. (%)	11 (13.6)	3 (11.5)	8 (14.5)	.99
CHF, No. (%)	24 (29.6)	10 (38.5)	14 (25.5)	.32

AVD, aortic valvular disease; BMI, body mass index; CABG, coronary artery bypass graft; CAD, coronary artery disease; CHF, congestive heart failure; CVD, cerebrovascular disease; MVD, mitral valvular disease; PCI, percutaneous coronary intervention.

[a] $P < .05$ was deemed statistically significant.

during the index hospitalizations, and all cardiac transplants occurred in subsequent hospitalizations.

Escalation characteristics are listed in Table III. The majority of tMCS devices were placed percutaneously (95.1%), and 14.8% of patients received more than 1 escalation. Twenty-one percent of patients underwent intraprocedural escalation before leaving the cardiac catheterization laboratory based on immediate postprocedure assessment of hemodynamics. In all, 30.9% of patients underwent escalation within 24 hours and 56.8% within 48 hours. The presence of a PAC preescalation was associated with improved survival compared with the absence of a PAC (80.8% vs 58.2%, respectively; $P = .05$). The safety profiles related to tMCS devices are detailed in Table III. Overall, 60.5% of patients had 1 or more complications. The devices used are listed in Table IV; the 2 most common tMCS devices initially implanted were IABP (n = 32) and Impella CP (n = 32); in total, they constituted 79.0% (n = 64) of patients requiring escalation. Their escalation patterns are shown in Figure 3.

Hemodynamics pre– and post–tMCS escalation are detailed in Figure 4. In survivors, tMCS escalation resulted in a significant increase in CO (from 4.4 to 5.9 L/min; $P = .01$) and CPO (from 0.78 to 1.07 watts, $P = .01$). Nonsurvivors also had an increase in CO (from 3.9 to 7.1 L/min, $P < .001$), cardiac index (from 1.9 to 3.4 L/min/m^2, $P < .001$), and CPO (from 0.63 to 1.07 watts, $P < .001$) postescalation along with a significant decrease in central venous pressure (CVP; from 17.0 to 11.4 mm Hg, $P < .001$). However, nonsurvivors had persistent hypotension with tMCS escalation (SBP, 96.9 to 85.5 mm Hg, $P = 0.01$; mean arterial pressure [MAP], 77.1 to 66.8 mm Hg, $P < .01$) and continued to have elevated LA (from 6.9 to 4.4 mmol/L, $P = .06$) postescalation. Both groups' COD had negative means postescalation, indicating that both survivors (0.1 to –1.2 L/min, $P = .04$) and nonsurvivors (1.1 to –2.2 L/min, $P < .001$) experienced closed COD postescalation.

Hemometabolic parameters of survivors in comparison to nonsurvivors are detailed in Table V. Preescalation, survivors had lower LA (3.3 vs 6.9 mmol/L, $P = .02$), higher CPO (0.78 vs 0.63 watts, $P = .02$), and lower COD (0.1 vs 1.1 L/min, $P = .04$) than those of nonsurvivors, whereas no significant differences were found between the 2 groups in SBP, MAP, CVP, systemic vascular resistance, CO, cardiac index, and PAPi. Younger age (<62 years), lower BMI (<28.7 kg/m^2), and lower preescalation LA (<3.1 mmol/L) were associated with a higher odds ratio of survival, as detailed in Figure 5. Receiver operating characteristic curves demonstrated prognostic thresholds associated with survival and mor-

TABLE II. Hospitalization Characteristics

	All (N = 81), No. (%)	Survivors (n = 26), No. (%)	Nonsurvivors (n = 55), No. (%)	*P* value[a]
Etiology of shock				
AMI	27 (33.3)	6 (23.1)	21 (38.2)	.21
STEMI	18 (22.2)	5 (19.2)	13 (23.6)	.78
NSTEMI	9 (11.1)	1 (3.8)	8 (14.5)	.26
ADHF	50 (61.7)	18 (69.2)	32 (58.2)	.46
ICM	27 (33.3)	8 (30.8)	19 (34.5)	.99
NICM	23 (28.4)	10 (38.5)	13 (23.6)	.10
Other/unknown	4 (4.9)	2 (7.7)	2 (3.6)	.59
Shock on admission[b]	51 (63.0)	11 (42.3)	40 (72.7)	.01
Transfer	50 (61.7)	13 (50.0)	37 (67.3)	.15
In-network	13 (16.0)	2 (7.7)	11 (20.0)	.21
Out-of-network	37 (45.7)	11 (42.3)	26 (47.3)	.81
With MCS	34 (42.0)	8 (30.8)	26 (47.3)	.23
Cardiac arrest[c]	29 (35.8)	7 (26.9)	22 (40.0)	.32
Vasopressor/inotrope preescalation (n = 71, S = 22, NS = 49)	55 (77.5)	18 (81.8)	37 (75.5)	.99
1	22 (31.0)	8 (36.4)	14 (28.6)	.61
≥2	33 (46.5)	10 (45.5)	23 (46.9)	.81
Revascularization (AMI n = 35)	32 (39.5)	7 (26.9)	25 (45.5)	.55
PCI	31 (38.3)	7 (26.9)	24 (43.6)	.99
CABG	1 (1.2)	0 (0.0)	1 (1.8)	.99
AHF therapy	7 (8.8)	5 (19.2)	2 (3.7)	.03
LVAD[d]	3 (3.8)	1 (3.8)	2 (3.7)	
Transplant[e]	1 (1.3)	1 (3.8)	0 (0.0)	
LVAD + transplant	3 (3.8)	3 (11.5)	0 (0.0)	

ADHF, non-AMI acute decompensated heart failure; AHF, advanced heart failure; AMI, acute myocardial infarction; CABG, coronary artery bypass graft; ICM, ischemic cardiomyopathy; LVAD, left ventricular assist device; MCS, mechanical circulatory support; NICM, nonischemic cardiomyopathy; NS, nonsurvivors; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; S, survivors; STEMI, ST-segment elevation myocardial infarction.

[a] $P < .05$ was deemed statistically significant.

[b] Shock at time of initial medical contact compared with development of shock later on during index hospitalization.

[c] Includes in-hospital and out-of-hospital cardiac arrest.

[d] All LVADs occurred during index hospitalization.

[e] All transplants occurred subsequent hospitalizations.

tality. The areas under the curve (AUCs) for age, preescalation lactate, and postescalation blood pressure were greater than 0.7, denoting an acceptable discrimination for predictor of survival. The AUCs for BMI and preescalation CPO were less than 0.7, suggesting unknown discrimination for predictor of survival. Age greater than 73 years demonstrated a strong association with mortality (specificity, 92%; sensitivity, 29%), whereas age younger than 53 years was a strong predictor of survival (specificity, 80%; sensitivity, 46%). A preescalation lactate level greater than 6.8 mmol/L had specificity of 91% and sensitivity of 41% for predicting mortality. The AUC was greatest in postescalation blood pressure, indicating that persistence of hypotension postescalation (SBP <85 mm Hg) and (MAP <67 mm Hg) was associated with a poor prognosis (specificity, 92%; sensitivity, 58% and specificity, 92%; sensitivity, 39%, respectively).

Discussion

Despite improvements in door-to-reperfusion metrics and heart failure therapies, the incidence of CS is increasing.[1,2] Patients presenting with CS are increasingly

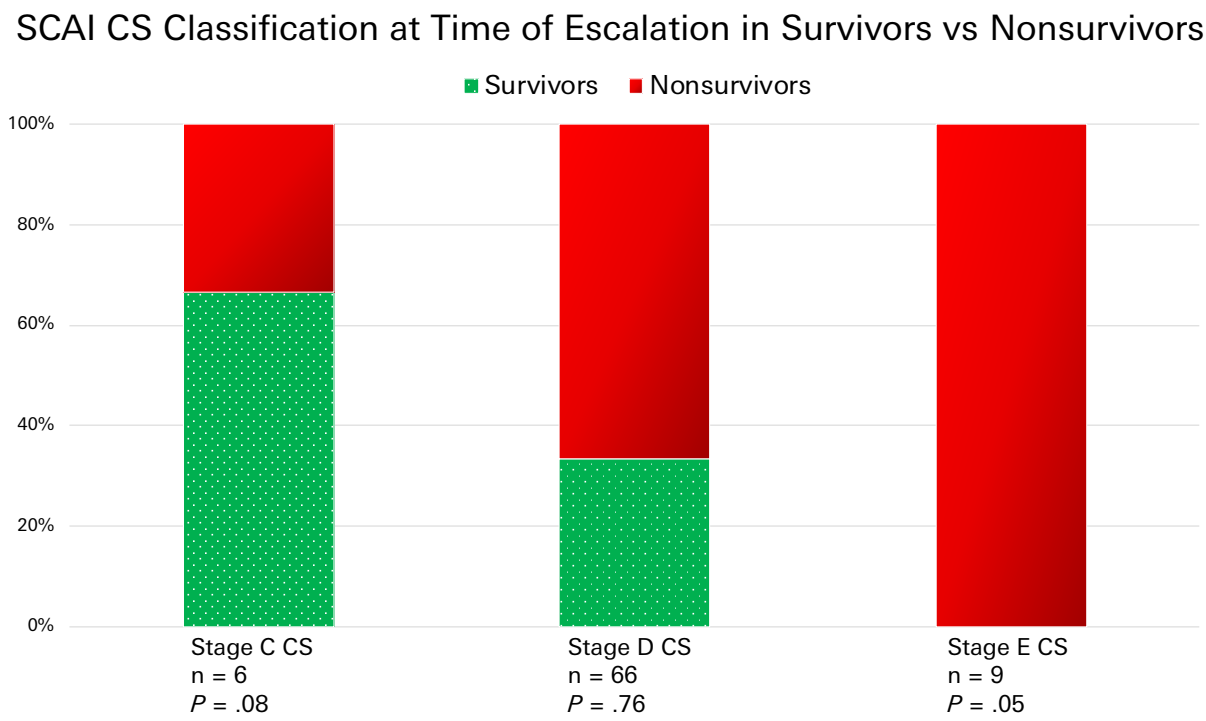

Fig. 2 *Graph reports the CS stage in survivors and nonsurvivors, according to SCAI classifications, at the time of escalation.* P < .05 *was considered statistically significant.*

CS, cardiogenic shock; SCAI, Society of Coronary Angiography and Intervention.

older, have more complex cases, and have more comorbid conditions, leading to higher rates of multiorgan failure.[9,10] A contemporary review of patients with AMI-CS from the Nationwide Inpatient Sample from 2000 to 2014 demonstrated a substantial rise in the incidence of multiorgan failure, which was associated with increased lengths of stay and higher in-hospital mortality.[11] In an effort to improve the hemodynamic profile of patients with CS, there has been substantial growth in the use of nondurable tMCSs over the past decade, with the aim of providing systemic perfusion and preventing multiorgan dysfunction syndrome.[10] Cardiogenic shock requiring tMCS is a fatal condition with high rates of morbidity and mortality. Historically, the survival rate in CS has been reported as 47% (SHOCK: AMI-CS; PCI ± IABP),[12] 60% (IABP-SHOCK II: AMI-CS; IABP),[13] 49% (refractory CS of all causes; ECMO),[14] 51% (CULPRIT-SHOCK: AMI-CS; PCI ± IABP),[15] and 39% (refractory CS of all causes; ECMO and Impella).[16] Further escalation of tMCS likely incurs an even poorer prognosis and worse outcome; however, this has not been assessed. The present study found that patients requiring escalation of tMCS because of worsening CS of all causes had a survival-to-discharge rate of 32%. Identifying patients who might benefit from tMCS escalation, or in whom tMCS may be futile, is warranted to aid further risk stratification in this patient population.

This study demonstrates key observations in patients with refractory CS. At the time of escalation, SCAI stage E shock incurred a mortality rate of 100% (P = .05), raising the question of futility of tMCS escalation in stage E shock (Fig. 2). The SCAI CS classification is a simple clinical tool that can assist with rapid risk stratification in CS.[5] Jentzer et al[17] found that patients presenting with stage E shock had an in-hospital mortality rate of 67%, and Schrage et al[18] similarly found a mortality rate of 68% in a cohort with AMI-CS presenting with stage E shock. Thus, the benefit of tMCS escalation in this high-risk cohort when initial tMCS is already failing is unclear. In addition, the use of a PAC immediately preescalation was associated with increased survival (P = .05). Despite controversy in its routine use for heart failure, the PAC is an important tool in the management of CS,[7,8,19,20] especially when an initial tMCS device is failing. Hemodynamic data provided by a PAC can help identify univentricular vs biventricular failure, thus guiding appropriate tMCS selection. In this study, the majority of patients (90%) were initially treated with isolated left ventricular support, whereas most escalation included biventricular support (58% of patients), highlighting the importance of hemodynamic guidance and early identification of biventricular failure (Table IV). Hemodynamic reassessment in the cardiac catheterization laboratory to assess the effects of initial

TABLE III. Temporary MCS Escalation Characteristics

	All (N = 81), No. (%)	Survivors (n = 26), No. (%)	Nonsurvivors (n = 55), No. (%)	*P* value[a]
Method of escalation				.09
Percutaneous	77 (95.1)	23 (88.5)	54 (98.2)	
Central/surgical	4 (4.9)	3 (11.5)	1 (1.8)	
No. of escalations				.99
1	69 (85.2)	22 (84.6)	47 (85.5)	
>1	12 (14.8)	4 (15.4)	8 (14.5)	
Timing of escalation				.39
Intraprocedural	17 (21.0)	7 (26.9)	10 (18.2)	
Postprocedural	64 (80.2)	19 (73.1)	45 (81.8)	
Duration until escalation from first MCS device (n = 64), h				
<24	25 (30.9)	5 (19.2)	20 (24.7)	.16
≤48	21 (25.9)	6 (23.1)	15 (27.3)	.99
>48	13 (16.0)	7 (26.9)	6 (10.9)	.06
Unknown	5 (6.2)	1 (3.8)	4 (7.3)	.99
Preescalation PAC	53 (65.4)	21 (80.8)	32 (58.2)	.05
MCS complications	49 (60.5)	18 (69.2)	31 (56.4)	.33
Stroke[b]	9 (11.1)	2 (7.7)	7 (12.7)	.71
Limb ischemia	6 (7.4)	2 (7.7)	4 (7.3)	.99
Blood transfusion	26 (32.1)	9 (34.6)	17 (30.9)	.80
Device malfunction	2 (2.5)	0 (0.0)	2 (3.6)	.99
Antibiotic use[c]	31 (38.3)	15 (57.7)	16 (29.1)	.02

AMI, acute myocardial infarction; MCS, mechanical circulatory support; PAC, pulmonary artery catheter.

[a] $P < .05$ was deemed statistically significant.

[b] Includes ischemic and hemorrhagic stroke.

[c] Empiric antibiotics for suspected for infection with/without an identified source.

tMCS allows for early alteration of device selection. In this study, 17 patients underwent tMCS escalation during their index procedure (intraprocedural escalation) and had a numerically but not statistically significantly higher survival rate than that for those who underwent escalation after their index procedure (41.2% vs 29.7%, respectively; $P = .39$).

Results highlight the significance of transitioning from hemodynamic shock to hemometabolic shock and multiorgan dysfunction. Potential benefits of early tMCS use are prevention of multiorgan failure and improved outcomes.[16,21] However, if implemented late and after the onset of hemometabolic shock, escalation carries an uncertain benefit. Despite an adequate level of support and similar postescalation CO, cardiac index, CPO, and COD between survivors and nonsurvivors, nonsurvivors had persistently elevated LA and hypotension that were worse than in survivors postescalation. In addition, nonsurvivors had no improvement in markers of kidney and liver injury postescalation (nonsurvivors' creatinine levels: preescalation 2.3 to postescalation 2.8 mg/dL, $P = .09$; nonsurvivors' aspartate transaminase: preescalation 1,932.3 to postescalation 2,099.5 units/L, $P = .85$).

Similar to previous work, this study reiterates potential prognostic findings in patients with refractory CS (Fig. 5). Advanced age is well known to be associated with poor outcomes.[22] Patients younger than 62 years were more likely to survive, with an odds ratio of 4.3 (95% CI, 1.6-11.6; $P < .01$). A preescalation LA less than 3.1 mmol/L was associated with increased survival, with an OR of 6.6 (95% CI, 1.9-22.8, $P < .01$), and a preescalation LA greater than 6.8 mmol/L had 90% specificity for mortality. CPO has previously been shown to be a prognostic marker in CS and, in the present study population, a preescalation CPO greater than 0.9 watts was associated with survival, whereas a CPO less than 0.5 watts was associated with mortality.[7]

The primary drawbacks of tMCS, aside from cost and resource use, are the associated complications. The impact of infection and systemic inflammatory response in CS is substantial.[23] In the present study, 38.3% of pa-

TABLE IV. Initial and Escalation Temporary MCS Devices

	All (N = 81), No. (%)	Survivors (n = 26), No. (%)	Nonsurvivors (n = 55), No. (%)	*P* value[a]
Initial MCS				
Biventricular support	3 (3.7)	1 (3.8)	2 (3.6)	.99
VA ECMO	2 (2.5)	1 (3.8)	1 (1.8)	.54
ProtekDuo + Impella CP	1 (1.2)	0 (0.0)	1 (1.8)	.99
Left-sided support only	73 (90.1)	23 (88.5)	50 (90.1)	.71
IABP	32 (39.5)	11 (42.3)	21 (38.2)	.81
Impella 2.5	4 (4.9)	1 (3.8)	3 (54.5)	.99
Impella CP	32 (39.5)	10 (38.5)	22 (40.0)	.99
Impella 5.0	2 (2.5)	0 (0.0)	2 (3.6)	.99
TandemHeart[b]	3 (3.7)	1 (3.8)	2 (3.6)	.99
Right-sided support only				
ProtekDuo	3 (3.7)	1 (3.8)	2 (3.6)	.99
Noncardiac support				
VV ECMO	2 (2.5)	1 (3.8)	1 (1.8)	.54
Escalation MCS				
Biventricular support	47 (58.0)	16 (61.5)	31 (56.4)	.81
VA ECMO	7 (8.6)	3 (11.5)	4 (9.1)	.67
VA ECMO + IABP	1 (1.2)	1 (3.8)	0 (0.0)	.32
VA ECMO + Impella 2.5	2 (2.5)	0 (0.0)	2 (3.6)	.99
VA ECMO + Impella CP	17 (21.0)	6 (23.1)	11 20.0)	.78
BA ECMO	1 (1.2)	0 (0.0)	1 (1.8)	.99
ProtekDuo + IABP	3 (3.7)	2 (7.7)	1 (1.8)	.24
ProtekDuo + Impella CP	5 (6.2)	2 (7.7)	3 (5.5)	.65
ProtekDuo + Impella 5.0	3 (3.7)	0 (0.0)	3 (5.5)	.55
ProtekDuo + TandemHeart[b]	2 (2.5)	0 (0.0)	2 (3.6)	.99
Impella RP + TandemHeart[b]	1 1.2)	0 (0.0)	1 (1.2)	.99
Impella RP + Impella CP	2 (2.5)	0 (0.0)	2 (3.6)	.99
Central VA ECMO	1 1(.2)	1 (3.8)	0 (0.0)	.32
Central VA ECMO + Impella CP	1 (1.2)	0 (0.0)	1 (1.2)	.99
VA ECMO + central LV "vent"	1 (1.2)	1 (3.8)	0 (0.0)	.32
Left-sided support only	34 (40.7)	10 (34.6)	24 (43.6)	.99
Impella 2.5	1 (1.2)	0 (0.0)	1 (1.2)	.99
Impella CP	16 (19.8)	4 (15.4)	12 (21.8)	.57
Impella 5.0	10 (12.3)	3 (11.5)	7 (12.7)	.99
TandemHeart[b]	6 (7.4)	2 (7.7)	4 (7.3)	.99
LVAD	1 (1.2)	1 (3.8)	0 (0.0)	.32

BA ECMO, biatrial extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LVAD, durable left ventricular assist device; MCS, mechanical circulatory support; VA ECMO, venous-arterial extracorporeal membrane oxygenation; VV ECMO, venous-venous extracorporeal membrane oxygenation.

[a] $P < .05$ was deemed statistically significant.

[b] TandemHeart with/without oxygenator.

tients were on empiric antibiotics for clinical suspicion of infection, regardless of the presence of an identified infectious nidus. The rate of limb ischemia in this study was lower (7.4%) than those in older ECMO registries that reached up to 20%, but it was similar to rates more contemporary studies (~9%); this likely indicates a better overall safety profile because of improved protocols and frequent use of reperfusion catheters.[16,24,25]

Limitations of this study are as follows. First, similar to other observational studies, this study could not

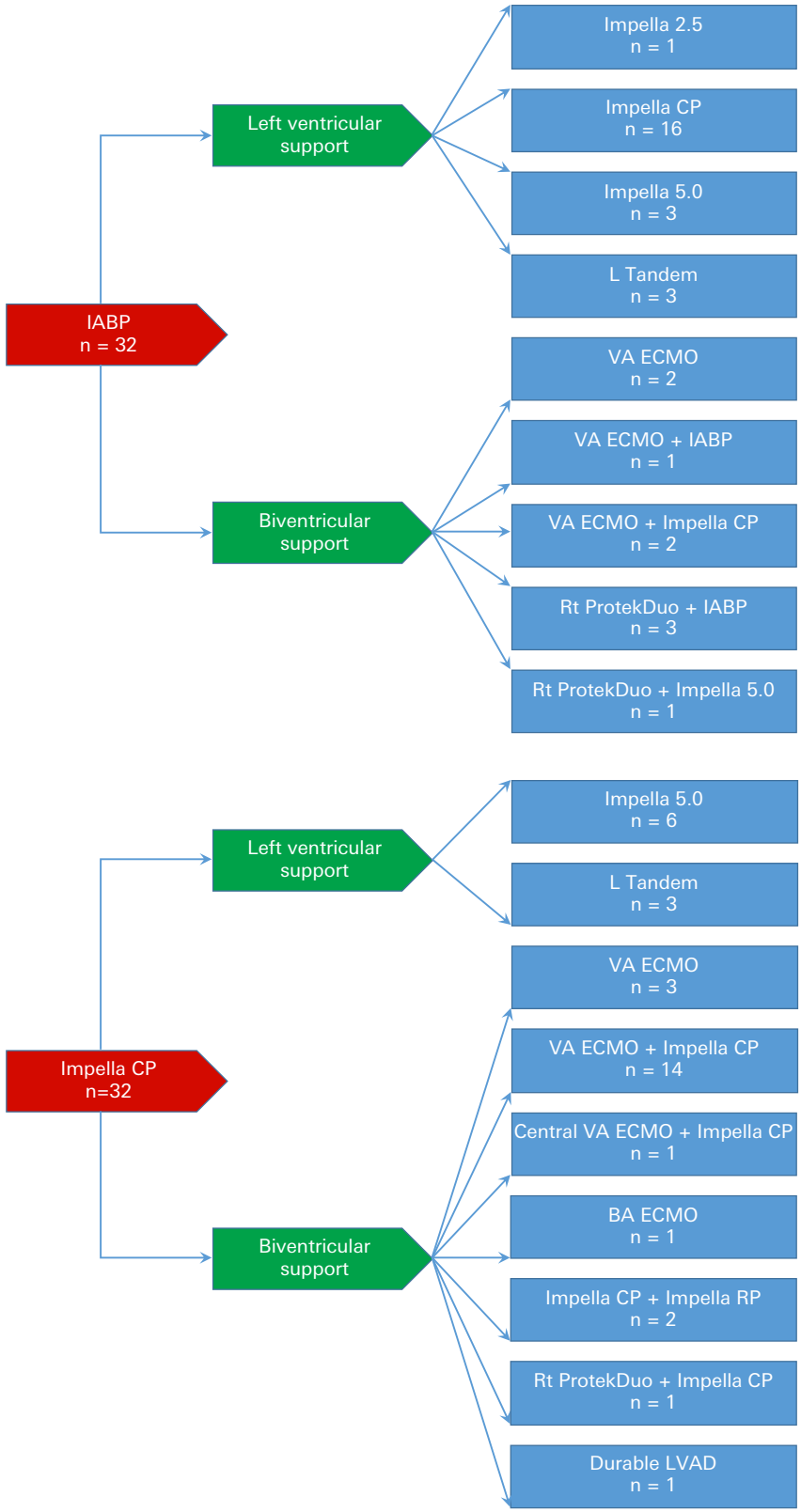

Fig. 3 *Chart shows escalation patterns after initial mechanical circulatory support with an intra-aortic balloon pump or an Impella CP.*

BA ECMO, biatrial extracorporeal membrane oxygenation; CP, cardiac power; IABP, intra-aortic balloon pump; L tandem, left ventricular assistance with TandemHeart; LVAD, left ventricular assist device; Rt ProtekDuo, right ventricular assistance with ProtekDuo; VA ECMO, venous-arterial extracorporeal membrane oxygenation.

TABLE V. Hemometabolic Parameters in Survivors Compared With Nonsurvivors Pre- and Post-MCS Escalation[a]

	Survivors (n = 26)	Nonsurvivors (n = 55)	*P* value[b]
Preescalation[c]			
Presence of PAC, No. (%)	21 (80.8)	32 (58.5)	.05
SBP, mm Hg	106.5 (25) (n = 21)	96.9 (23.6) (n = 49)	.13
MAP, mm Hg	82.6 (17.3) (n = 24)	77.1 (17.6) (n = 50)	.19
Heart rate, beats/min	98.3 (28.1) (n = 23)	90.5 (19.4) (n = 49)	.26
Lactic acid levels, mmol/L	3.3 (4.0) (n = 21)	6.9 (6.4) (n = 46)	.02
Creatinine levels, mg/dL	2.1 (1.37) (n = 21)	2.3 (1.3) (n = 50)	.56
BUN levels, mg/dL	34.5 (20.3) (n = 21)	39.0 (19.4) (n = 50)	.38
AST levels, U/L	911.7 (4529.5) (n = 19)	1,932.3 (4,286.0) (n = 43)	.40
ALT levels, U/L	597.6 (2,847.5) (n = 19)	1,237.0 (2,695.0) (n = 43)	.40
Vasopressor/inotrope	1.8 (0.8) (n = 18)	2.1 (1.1) (n = 37)	.31
CVP, mm Hg	13.8 (6.3) (n = 16)	17.0 (7.8) (n = 33)	.16
PASP, mm Hg	43.8 (15.4) (n = 17)	47.7 (15.2) (n = 30)	.40
PADP, mm Hg	22.5 (7.7) (n = 17)	26.0 (9.0) (n = 30)	.18
SVR, dynes/s/cm^{-5}	1,585 (715) (n = 14)	1,378 (733) (n = 27)	.39
CO (Fick), L/min	4.4 (1.9) (n = 18)	3.9 (1.2) (n = 28)	.28
CI (Fick), L/min/m^2	2.1 (0.9) (n = 18)	1.9 (0.6) (n = 28)	.39
CPO, watts	0.78 (0.3) (n = 17)	0.63 (0.22) (n = 30)	.02
PAPi	1.8 (1.1) (n = 16)	1.6 (1.1) (n = 30)	.56
COD, L/min	0.1 (1.8) (n = 18)	1.1 (1.3) (n = 28)	.04
Postescalation (24 h)			
Presence of PAC, No. (%)	21 (80.8)	35 (63.6)	.13
SBP, mm Hg	102.7 (16.0) (n = 24)	85.5 (20.0) (n = 48)	<.001
MAP, mm Hg	80.1 (14.3) (n = 26)	66.8 (14.3) (n = 49)	<.001
Heart rate, beats/min	90.1 (28.6) (n = 25)	98.1 (23.0) (n = 50)	.19
Lactic acid levels, mmol/L	2.0 (1.4) (n = 23)	4.4 (5.3) (n = 36)	.04
Creatinine levels, mg/dL	1.94 (1.28) (n = 23)	2.8 (1.4) (n = 35)	.02
BUN levels, mg/dL	31.7 (23.1) (n = 23)	42.5 (22.2) (n = 35)	.07
AST levels, U/L	797.6 (3,016.0) (n = 21)	2,099.5 (3,303) (n = 32)	.15
ALT levels, U/L	469.4 (1,361.8) (n = 21)	1,181.3 (1,857.0) (n = 32)	.14
Vasopressors/inotropes, No.	2.2 (1.1) (n = 13)	2.2 (1.0) (n = 30)	.99
CVP, mm Hg	10.9 (3.9) (n = 21)	11.4 (4.0) (n = 37)	.65
PASP, mm Hg	34.6 (9.6) (n = 20)	34.8 (11.2) (n = 34)	.95
PADP, mm Hg	20.7 (9.5) (n = 20)	21.3 (7.1) (n = 34)	.79
SVR, dynes/s/cm^{-5}	1,026 (329) (n = 16)	858 (315) (n = 28)	.10
CO (Fick), L/min	5.9 (1.5) (n = 16)	7.1 (4.1) (n = 30)	.27
CI (Fick), L/min/m^2	2.7 (0.76) (n = 16)	3.4 (1.8) (n = 30)	.15
CPO, watts	1.07 (0.58) (n = 16)	1.07 (0.58) (n = 28)	.99
PAPi	1.4 (0.6) (n = 20)	1.5 (1.5) (n = 34)	.78
COD, L/min	–1.2 (1.6) (n = 16)	–2.2 (4.0) (n = 30)	.34

ALT, alanine transaminase; AST, aspartate transaminase; BUN, blood urea nitrogen; CI, cardiac index; CO, cardiac output; COD, cardiac output deficit; CPO, cardiac power output; CVP, central venous pressure; MAP, mean arterial pressure; MCS, mechanical circulatory support; PAC, pulmonary artery catheter; PADP, pulmonary artery diastolic pressure; PAPi, pulmonary artery pulsatility index; PASP, pulmonary artery systolic pressure; SBP, systolic blood pressure; SVR, systemic vascular resistance.

[a] All data are shown as mean (SD), unless otherwise indicated.

[b] $P < .05$ was deemed statistically significant.

[c] Most recent before escalation.

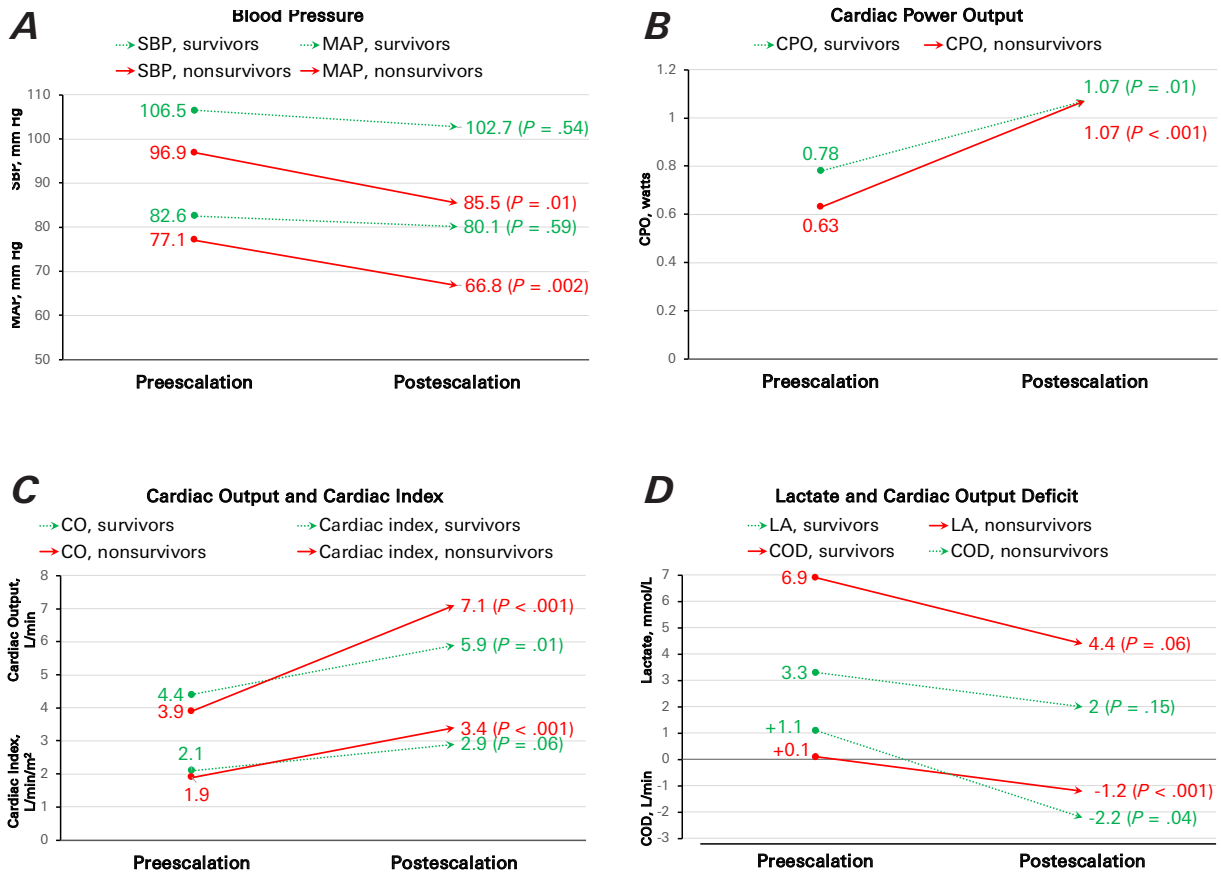

Fig. 4 *Graphs compare the effects of mechanical circulatory support on hemometabolic measurements after escalation based on survival, including* ***A****) blood pressure,* ***B****) CPO,* ***C****) CO and cardiac index, and* ***D****) lactate and CO deficit.* P < .05 *was considered statistically significant.*

CPO, cardiac power output; CO, cardiac output; COD, cardiac output deficit; LA, lactate levels; MAP, mean arterial pressure; SBP, systolic blood pressure.

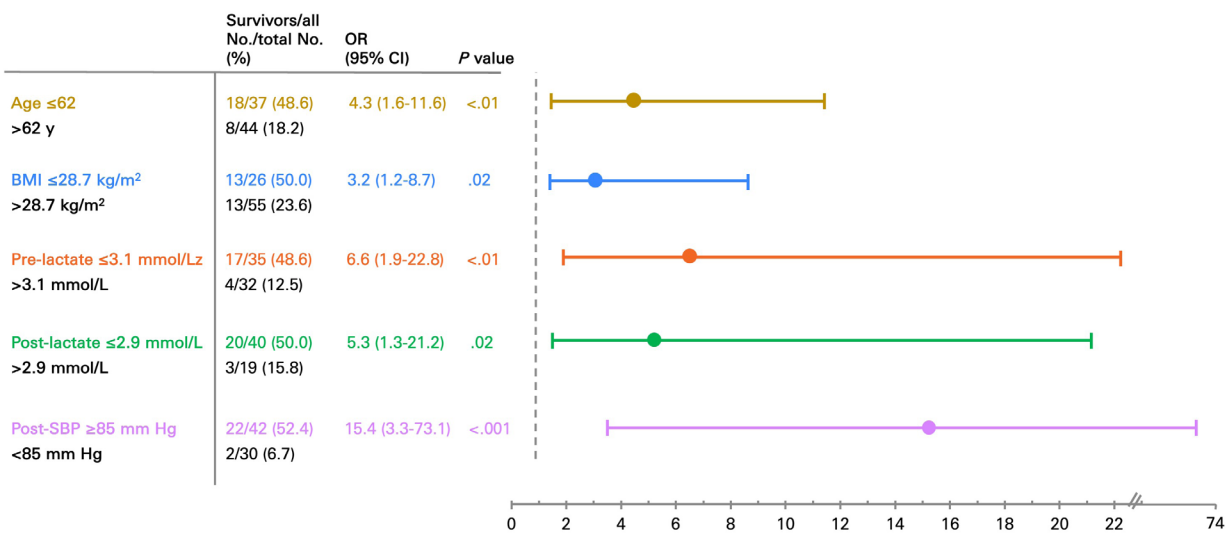

Fig. 5 *Graph shows the hemometabolic variables significantly associated with survival based on optimal cut points. Pre-lactate measurements were the most recently obtained before escalation. Post-lactate and post-SBP measurements were obtained 24 hours after escalation.* P < .05 *was considered statistically significant.*

BMI, body mass index; OR, odds ratio; SBP, systolic blood pressure.

establish causality or exclude the potential effect of selection bias; thus, the results are hypothesis-generating and may help guide future studies. Second, this study cohort is heterogenous and included multiple etiologies of CS as opposed to a single shock phenotype, limiting the researchers' ability to draw any definitive conclusions about "best practices" in CS management. Third, a multivariate model was not performed because of the variation in missing available data for each patient and the small cohort. Finally, this is a single-center study with a modest sample size, which led to wide CIs, limits the external validity of the study, and makes the conclusions less certain.

Conclusion

This paper describes clinical characteristics and outcomes of patients who underwent escalation of mechanical circulatory support because of worsening CS with an overall in-hospital survival of 32%. The presence of a PAC at the time of escalation was associated with improved survival. Escalation of tMCS in patients with SCAI stage E shock was associated with high mortality. Additional work is needed to improve outcomes for this high-risk cohort.

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