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Complete Hemodynamic Profiling With Pulmonary Artery Catheters in Cardiogenic Shock Is Associated With Lower In-Hospital Mortality

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ABSTRACT

OBJECTIVES The purpose of this study was to investigate the association between obtaining hemodynamic data from early pulmonary artery catheter (PAC) placement and outcomes in cardiogenic shock (CS).

BACKGROUND Although PACs are used to guide CS management decisions, evidence supporting their optimal use in CS is lacking.

METHODS The Cardiogenic Shock Working Group (CSWG) collected retrospective data in CS patients from 8 tertiary care institutions from 2016 to 2019. Patients were divided by Society for Cardiovascular Angiography and Interventions (SCAI) stages and outcomes analyzed by the PAC-use group (no PAC data, incomplete PAC data, complete PAC data) prior to initiating mechanical circulatory support (MCS).

RESULTS Of 1,414 patients with CS analyzed, 1,025 (72.5%) were male, and 494 (34.9%) presented with myocardial infarction; 758 (53.6%) were in SCAI Stage D shock, and 263 (18.6%) were in Stage C shock. Temporary MCS devices were used in 1,190 (84%) of those in advanced CS stages. PAC data were not obtained in 216 patients (18%) prior to MCS, whereas 598 patients (42%) had complete hemodynamic data. Mortality differed significantly between PAC-use groups within the overall cohort ($p < 0.001$), and each SCAI Stage subcohort (Stage C: $p = 0.03$; Stage D: $p = 0.05$; Stage E: $p = 0.02$). The complete PAC assessment group had the lowest in-hospital mortality than the other groups across all SCAI stages. Having no PAC assessment was associated with higher in-hospital mortality than complete PAC assessment in the overall cohort (adjusted odds ratio: 1.57; 95% confidence interval: 1.06 to 2.33).

CONCLUSIONS The CSWG is a large multicenter registry representing real-world patients with CS in the contemporary MCS era. Use of complete PAC-derived hemodynamic data prior to MCS initiation is associated with improved survival from CS. (J Am Coll Cardiol HF 2020;8:903–13) © 2020 by the American College of Cardiology Foundation.

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ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

CO = cardiac output

CS = cardiogenic shock

DHF = decompensated heart failure

ECMO = extracorporeal membrane oxygenation

IABP = intra-aortic balloon pump

MCS = mechanical circulatory support

PAC = pulmonary artery catheter

PCWP = pulmonary capillary wedge pressure

RAP = right atrial pressure

SCAI = Society for Cardiovascular Angiography and Interventions

Cardiogenic shock (CS) is a condition of low cardiac output (CO) with persistent hypotension, hypoperfusion, and life-threatening multiorgan failure attributable to impaired ventricular function (left or right, or both) (1–2). Pulmonary artery catheters (PACs) directly measure pulmonary and cardiac pressures and oxygen saturation and are used to calculate an array of hemodynamic parameters including CO and vascular resistances. Such monitoring facilitates triage and management of patients presenting with acute hemodynamic decompensation. Specifically, PACs allow operators to assess the relative contributions of right and left ventricular failure to guide medical therapy with vasoactive medications, inotropes, and mechanical circulatory support (MCS) device(s) for CS (3). However, no definitive randomized controlled trial has tested the utility of PACs for CS.

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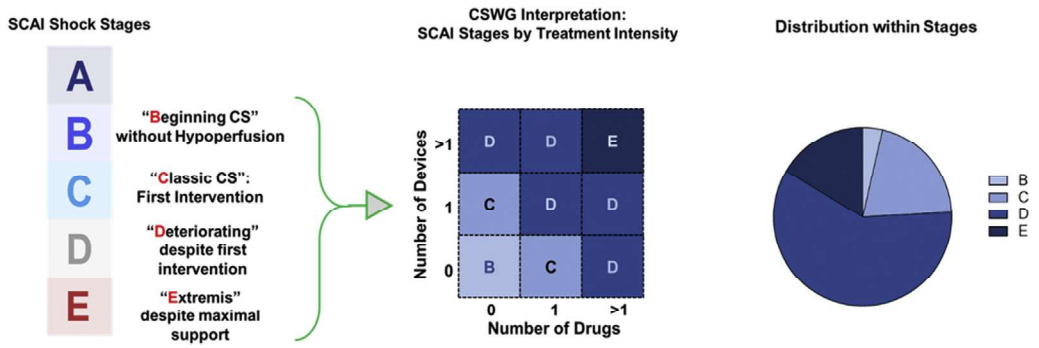
Retrospective and prospective studies have shown no benefit from PAC-guided treatment in patients with decompensated heart failure (DHF) (4–6). However, patients with CS are conventionally excluded from clinical trials assessing the use of PACs. Recently, a 5-stage CS classification scheme was proposed by a multidisciplinary group of experts from the Society for Cardiovascular Angiography and Interventions (SCAI) to differentiate among patient subsets based on severity (7). Recent widespread availability of short-term percutaneous MCS devices for CS has led to proposed management algorithms guided by PAC-derived invasive hemodynamic data (8,9). Similarly, studies using hemodynamically-guided decision making have shown improved outcomes with the use of PAC in CS (3,10,11). A recent white paper dealing with PAC use in CS recommended that PACs be used in all patients undergoing MCS to monitor effectiveness, optimize device settings, assess the need for escalation, and guide timing and rate of weaning (3). However, no studies have explored whether complete hemodynamic profiling using PAC is associated with clinical outcomes in CS. This study sought to investigate the association between the use of PAC prior to initiation of MCS and clinical outcomes in CS by using data from the Cardiogenic Shock Working Group (CSWG) registry. Hypothetically, the timely use of complete PAC measurements would be associated with outcomes in patients with advanced shock, as defined by the SCAI CS staging system.

METHODS

DATA SOURCE. The CSWG is an academic research consortium with a national registry begun in 2016. A group of 16 clinical sites across the United States contributed CS patient data (Supplemental Table 1). Those sites included community and university hospitals with registry inclusion dependent on a minimum of 100 patients with CS per year. For this analysis, patients with CS at the first 8 sites contributing registry data between 2016 and 2019 were included, regardless of CS cause. The registry details a standardized set of data elements which were predefined by principal investigators and collected retrospectively. Elements included patient, procedural, and hospital characteristics. Patient demographics, laboratory, and hemodynamic data were collected at a single time point as close to admission as possible, prior to short-term MCS (i.e., use of an intra-aortic balloon pump [IABP], the Impella (Abiomed, Danvers, Massachusetts), venoarterial extra corporeal membrane oxygenation [VA-ECMO], or extracorporeal centrifugal flow pumps) initiation. CS diagnosis was physician-adjudicated at each site and defined as follows: a sustained episode of systolic blood pressure <90 mm Hg for at least 30 mins or use of vasoactive agents and/or a cardiac index value of <2.2 l/min/m^2 determined to be secondary to cardiac dysfunction, in the absence of hypovolemia; or use of an MCS device for clinically suspected CS. Treatments for CS were left to the discretion of the clinicians at each center and were not guided by a prescribed algorithm. Both the use of vasopressors/inotropes and of MCS at any time throughout a patient's hospitalization was used to assign SCAI stages in the study cohort as described in an earlier report from this registry (12). Quality assurance was achieved through adjudication at each site by the respective clinical coordinators and principal investigator. Values were centrally audited and screened by the CSWG research team for any discrepancies or major outliers and resolved with the submitting site. This study was approved by the Tufts Health Sciences Institutional Review Board, and all sites contributing data (Supplemental Table 1) received approval to include data in this registry from their respective Institutional Review Board.

STUDY POPULATION. Between 2016 and 2019, data from 1,565 patients with CS were collected. The cause(s) of CS were reported by each site as acute myocardial infarction (AMI), acute DHF, or other. AMI

FIGURE 1 Stages of Cardiogenic Shock



Adapted from Baran et al. (7). CSWG = Cardiogenic Shock Working Group; SCAI = Society for Cardiovascular Angiography and Intervention.

was defined as any primary diagnosis of either non-ST-segment elevation or ST-segment elevation AMI. Acute DHF was defined as any primary diagnosis of acute or chronic HF not otherwise related to AMI. Other causes included postcardiotomy, myocarditis, or CS not otherwise specified. Patients younger than 18 years of age (n = 10.06%) and those with unknown mortality status at hospital discharge (n = 150; 9.6%) were excluded, leaving a study population of 1,414 patients with CS from 8 hospitals for analysis. Then a recently published SCAI CS staging system was applied to stratify this cohort by SCAI stage, as previously described, allowing evaluation of PAC utility across the spectrum of CS severity (12).

According to the SCAI stages, clinical deterioration based on persistent hypotension and hypoperfusion is the main determinant of the SCAI Stage and is associated with a need for intensification of treatment (7). Therefore, treatment escalation for CS was used as a proxy for persistent hypotension and hypoperfusion to retrospectively define maximum deterioration as hemometabolic parameters were only assessed at admission. A CSWG-adapted definition of SCAI stages was applied to this study cohort based on total use of vasopressors, inotropes, and MCS across a patient's hospital course (**Figure 1**) (12). SCAI Stage A patients are those at risk for CS and therefore were not captured in this study population. Stage B patients are those exhibiting early symptoms not including hypoperfusion and therefore did not require pharmacological or MCS. Stage C patients include those with hypotension and hypoperfusion requiring intervention beyond volume resuscitation including those requiring either 1 vasopressor/inotrope or 1 MCS device. Stage D patients are those whose conditions deteriorate despite initial intervention, defined in this dataset by the need for multiple drugs or MCS devices. Finally, Stage E patients are those who deteriorate further and require maximal support, defined in this dataset as requiring at least 2 MCS devices and 2 drugs during their hospitalization. Patients requiring cardiopulmonary resuscitation on admission were included in Stage E.

CLASSIFICATION OF PAC USAGE. Patients were divided into 3 categories: those with a complete PAC assessment, those with an incomplete assessment, and those who did not receive a PAC catheter prior to MCS initiation. PAC usage was defined in the population by the presence of invasive hemodynamic parameters including right atrial pressure (RAP), pulmonary artery systolic pressure, pulmonary artery diastolic pressure (PADP), pulmonary capillary wedge pressure (PCWP), pulmonary artery (PA) oxygen saturation, and CO. Complete hemodynamic profiling with PAC required documentation of 5 measurements: RAP, pulmonary artery systolic pressure, PADP, PCWP, and PA saturation. Measured values were chosen instead of derived values to ensure accuracy of groupings (e.g., although there are other methods for estimating CO, there is no way to estimate PA saturation). Calculated hemodynamic parameters derived from the above-mentioned measurements (cardiac index, mean pulmonary artery pressure, systemic vascular resistance, cardiac power output, pulmonary artery pulsatility index, and so forth) were noted but not essential for designations as a complete profile. If any of these 5 hemodynamic values were not reported (e.g., only RAP or oxygen saturation documented from a central venous catheter, or pulmonary artery pressure documented in the absence of PCWP and so forth),

TABLE 1 Baseline Characteristics of Cardiogenic Shock Patients Across 3 Subgroups

		PAC Usage			
	All (N = 1,414)	None (n = 260)	Incomplete Assessment (n = 556)	Complete Assessment (n = 598)	p Value
Males	1,025 (72.5)	196 (73.4)	413 (75.2)	416 (69.6)	0.085
Race					<0.001
White	647 (45.8)	138 (51.7)	158 (28.8)	351 (58.7)	
Hispanic/Latino	31 (2.2)	11 (4.1)	10 (1.8)	10 (1.7)	
African-American	28 (2.0)	10 (3.7)	5 (0.9)	13 (2.2)	
Asian	31 (2.2)	13 (4.9)	10 (1.8)	8 (1.3)	
Other	82 (5.8)	5 (1.9)	6 (1.1)	71 (11.9)	
Medical history					
Hypertension	681 (48.2)	145 (54.3)	262 (47.7)	274 (45.8)	<0.001
Diabetes mellitus	489 (34.6)	79 (29.6)	212 (38.6)	198 (33.1)	0.030
Atrial fibrillation	296 (20.9)	20 (7.5)	90 (16.4)	186 (31.1)	<0.001
Prior MI	374 (26.4)	54 (20.2)	168 (30.6)	152 (25.4)	<0.001
Prior HF	768 (54.3)	87 (32.6)	286 (52.1)	395 (66.1)	<0.001
Chronic kidney disease	323 (22.8)	43 (16.1)	110 (20.0)	170 (28.4)	0.004
Peripheral vascular disease	60 (4.2)	13 (4.9)	13 (2.4)	34 (5.7)	0.722
COPD	101 (7.1)	13 (4.9)	32 (5.8)	56 (9.4)	0.108
Stroke/TIA	159 (11.2)	20 (7.5)	54 (9.8)	85 (14.2)	0.116
Valvular disease	214 (15.1)	20 (7.5)	56 (10.2)	138 (23.1)	<0.001
PCI	293 (20.7)	63 (23.6)	118 (21.5)	112 (18.7)	<0.001
CABG	114 (8.1)	18 (6.7)	46 (8.4)	50 (8.4)	0.056
Ventricular tachycardia	216 (15.3)	22 (8.2)	54 (9.8)	140 (23.4)	<0.001
ICD	329 (23.3)	25 (9.4)	84 (15.3)	220 (36.8)	<0.001
CRT	97 (6.9)	4 (1.5)	37 (6.7)	56 (9.4)	<0.001
Low institutional PAC placement	313 (22.14)	140 (52.43)	112 (20.40)	61 (10.20)	<0.001
Demographics					
Age	1,412 (59.91 ± 14.78)	265 (62.41 ± 15.38)	549 (60.28 ± 13.32)	598 (58.47 ± 15.60)	0.011
Weight, kg	1,138 (85.25 ± 22.62)	219 (87.37 ± 21.37)	323 (84.63 ± 25.53)	596 (84.82 ± 21.35)	0.305
Metabolic panel					
AST	788 (459.41 ± 1,492.57)	115 (329.06 ± 1,044.31)	236 (574.09 ± 1,413.83)	437 (431.78 ± 1,626.74)	0.299
BUN	1,026 (32.38 ± 20.47)	165 (26.32 ± 16.46)	321 (32.02 ± 20.64)	540 (34.44 ± 21.12)	<0.001
Lactate	676 (4.37 ± 4.21)	99 (6.09 ± 4.82)	328 (3.97 ± 3.95)	249 (4.21 ± 4.14)	<0.001
Bicarbonate	836 (22.12 ± 5.45)	119 (19.97 ± 5.20)	245 (20.74 ± 5.16)	472 (23.38 ± 5.31)	<0.001
Serum creatinine	1,295 (1.76 ± 1.14)	182 (1.7 ± 1.29)	520 (1.86 ± 1.16)	593 (1.70 ± 1.07)	0.041
pH	576 (7.31 ± 0.15)	110 (7.25 ± 0.17)	255 (7.32 ± 0.14)	211 (7.32 ± 0.13)	<0.001
Echocardiography					
Admission EF	771 (24.94 ± 15.53)	112 (31.79 ± 17.27)	348 (23.74 ± 15.14)	311 (23.81 ± 14.68)	<0.001
LVEDD	996 (5.89 ± 1.37)	108 (5.01 ± 1.05)	397 (6.01 ± 1.4)	491 (5.99 ± 1.35)	<0.001
Heart rate	1,248 (92.02 ± 22.72)	195 (89.99 ± 27.5)	470 (94.8 ± 23.07)	583 (90.45 ± 20.38)	0.003
SBP	1,245 (98.17 ± 20.02)	192 (101.26 ± 26.3)	472 (97.52 ± 18.36)	581 (97.68 ± 18.82)	0.07
DBP	1,241 (61.98 ± 13.95)	191 (61.88 ± 15.98)	470 (61.73 ± 13.94)	580 (62.22 ± 13.25)	0.85
Hemodynamics					
RAP	1,030 (14.2 ± 6.95)	-	432 (14.87 ± 7.12)	598 (13.71 ± 6.79)	0.008
PCWP	848 (24.5 ± 8.9)	-	250 (26.12 ± 9.21)	598 (23.82 ± 8.68)	<0.001
MAP	1,117 (74.26 ± 13.95)	-	527 (74.28 ± 14.46)	590 (74.25 ± 13.5)	0.97
mPAP	1,074 (32.73 ± 9.86)	-	476 (33.16 ± 10.18)	598 (32.39 ± 9.59)	0.20
PASP	1,080 (47.67 ± 15.1)	-	482 (48.5 ± 15.76)	598 (47.00 ± 14.53)	0.10
PADP	1,074 (25.3 ± 8.25)	-	476 (25.57 ± 8.48)	598 (25.09 ± 8.07)	0.34
PA sat	755 (52.02 ± 13.55)	-	157 (53.48 ± 17.25)	598 (51.64 ± 12.39)	0.13
CPO	988 (0.63 ± 0.41)	-	432 (0.63 ± 0.42)	556 (0.63 ± 0.41)	0.98
Cardiac index	1,057 (1.86 ± 0.59)	-	474 (1.85 ± 0.63)	583 (1.86 ± 0.56)	0.78
Cardiac output	1,050 (3.84 ± 2.43)	-	464 (3.86 ± 2.65)	586 (3.83 ± 2.23)	0.83

Values are n (%) or n (mean ± SD).

AST = aspartate aminotransferase; BUN = blood urea nitrogen; CABG = coronary artery bypass graft; COPD = chronic obstructive pulmonary disease; CPO = cardiac power output; CRT = cardiac resynchronization therapy; DBP = diastolic blood pressure; EF = ejection fraction; HF = heart failure; ICD = implantable cardioverter-defibrillator; LVEDD = left ventricular end diastolic diameter; MAP = mean arterial pressure; MI = myocardial infarction; mPAP = mean pulmonary artery pressure; PA sat = pulmonary artery oxygen saturation; PAC = pulmonary artery catheter; PADP = pulmonary artery diastolic pressure; PASP = pulmonary artery systolic pressure; PCI = percutaneous coronary intervention; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure; SBP = systolic blood pressure; TIA = transient ischemic attack.

TABLE 2 Distribution of Hemodynamic Data Across Complete and Incomplete Groups

Hemodynamic Parameters Collected	All (N = 1,414)	Number of PAC Parameters Collected					
		0 (n = 260)	1 (n = 40)	2 (n = 51)	3 (n = 198)	4 (n = 267)	5 (n = 598)
PASP	1,080 (76.38)	0 (0.00)	1 (2.50)	19 (37.25)	195 (98.48)	267 (100.00)	598 (100.00)
PADP	1,074 (75.95)	0 (0.00)	0 (0.00)	17 (33.33)	192 (96.97)	267 (100.00)	598 (100.00)
PCWP	848 (59.97)	0 (0.00)	4 (10.00)	30 (58.82)	24 (12.12)	192 (71.91)	598 (100.00)
PA Sat	755 (53.39)	0 (0.00)	28 (70.00)	32 (62.75)	9 (4.55)	88 (32.96)	598 (100.00)
RAP	1,037 (73.34)	0 (0.00)	7 (17.50)	4 (7.84)	174 (87.88)	254 (95.13)	598 (100.00)

Values are n (%).

PA = pulmonary artery; PA Sat = pulmonary artery oxygen saturation; PAC = pulmonary artery catheter; PADP = pulmonary artery diastolic pressure; PASP = pulmonary artery systolic pressure; PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure.

the PAC assessment was considered incomplete. If a PAC was placed after MCS initiation, the patient was considered in the "no-PAC" category for the purpose of this analysis. Institutions were defined as low-PAC users if they were in the lowest tertile of PAC use among the contributing centers, and this variable was used in a multivariate model to control for variability in center practice.

HEMODYNAMIC ASSESSMENT. To assess the importance of hemodynamic assessment on clinical outcomes, the association between in-hospital mortality and hemodynamic values obtained from PACs was analyzed. Additional vital sign parameters (blood pressure and heart rate) were also included. Hemodynamic parameters were analyzed as categorical variables split into quartiles within the overall study cohort and in subcohorts based on CS cause (AMI-CS, acute DHF-CS).

STATISTICAL ANALYSIS. Descriptive statistics were analyzed among the 3 PAC subgroups to determine differences in baseline characteristics. Categorical variables were reported as frequencies and percentages and compared using Pearson chi-squared tests. Continuous variables were reported as mean ± SD and compared using independent *t*-tests. Differences in mortality among groups were assessed using chi-squared tests among the entire study cohort and among subcohorts of varying CS severity, as defined by previously validated maximum SCAI stages reached across hospitalization. The association between PAC use and mortality was further analyzed in a univariate logistic regression model and then adjusted for other significant univariate predictors of mortality including use of PAC at the study site, comorbidities (hypertension, diabetes mellitus, ventricular tachycardia, and implantable cardioverter-defibrillator); and cause of shock and age in a multivariate model. These models were run in the overall cohort as well as within each SCAI stage. Results from logistic regression models were reported as odds ratios (ORs) with 95% confidence intervals (CIs) Additionally, the frequency of in-hospital mortality was compared across quartiles of each hemodynamic parameter in the AMI-CS and acute DHF-CS cohorts using the Pearson chi-squared test. An alpha level of 0.05 was used to determine significance for all statistical analyses.

RESULTS

Data from 1,414 patients at 8 clinical sites were analyzed. Baseline characteristics are summarized in Table 1. Of the study cohort, 1,025 patients (72.5%) were male, and 712 (50.4%) presented with acute DHF-CS. CS was treated with vasoactive and/or pressor agents in 1,043 patients (73.8%). MCS devices included IABPs in 770 (54.5%), an Impella device in 410 (29%), and ECMO in 333 patients (23.6%). A number of patients (n = 99; 7.0%) received multiple MCS devices during their hospitalization. The majority of patients (n = 758, 53.6%) were in SCAI Stage D, with 263 (18.6%) in Stage C and 212 (15%) in Stage E shock. A total of 260 patients (18%) had no documented use of PAC prior to MCS, whereas 598 (42%) had a complete set of hemodynamic data recorded. Of those with an incomplete assessment (40%), PADP and PCWP were most likely to be missing, followed by PA saturation and RAP (Table 2).

Patients with acute DHF-CS were more likely to have a complete PAC assessment than those with AMI-CS (52.8% vs. 32.2%, respectively; $p < 0.001$) (Table 3). Patients with CS not receiving MCS (n = 224) were more likely to have a complete PAC assessment (79.9%); those with a complete assessment were more likely to not escalate past SCAI Stage B (0.4% vs. 0.4% vs. 7.2%, respectively; $p < 0.001$). Those treated with ECMO (n = 333) had a complete assessment

TABLE 3 Assessment of PAC Usage by SCAI Stages, Causes of CS, and Device Usage

		PAC Usage			
	All (N = 1,414)	None (n = 260)	Incomplete Assessment (n = 556)	Complete Assessment (n = 598)	p Value
Acute mechanical support treatment					
No MCS	224 (15.8)	30 (13.4)	15 (6.7)	179 (79.9)	<0.001
IABP	770 (54.5)	130 (16.9)	338 (43.9)	302 (39.2)	<0.001
Impella	410 (29.0)	80 (19.5)	169 (41.2)	161 (39.3)	0.33
ECMO	333 (23.6)	95 (28.5)	161 (48.4)	77 (23.1)	<0.001
Multiple MCS	308 (21.8)	62 (20.1)	124 (40.3)	122 (39.6)	0.55
Medical Therapy	1,043 (73.8)	109 (10.5)	445 (42.7)	489 (46.9)	<0.001
Shock cause					
MI	494 (34.9)	123 (24.9)	212 (42.9)	159 (32.2)	<0.001
HF	712 (50.4)	57 (8.0)	286 (40.2)	369 (51.8)	<0.001
Other	178 (12.6)	59 (33.2)	50 (28.1)	69 (38.8)	<0.001
SCAI Stage					
B	46 (3.3)	1 (2.2)	2 (4.4)	43 (93.5)	<0.001
C	263 (18.6)	68 (25.9)	63 (24.0)	132 (50.2)	<0.001
D	758 (53.6)	66 (8.7)	361 (47.6)	331 (43.7)	<0.001
E	212 (15.0)	39 (18.4)	90 (42.5)	83 (39.2)	<0.001

Values are n (%).

ECMO = extra-corporeal membrane oxygenation; HF = heart failure; IABP = intra-aortic balloon pump; MCS = mechanical circulatory support; MI = myocardial infarction; SCAI = Society of Cardiovascular Angiography and Interventions.

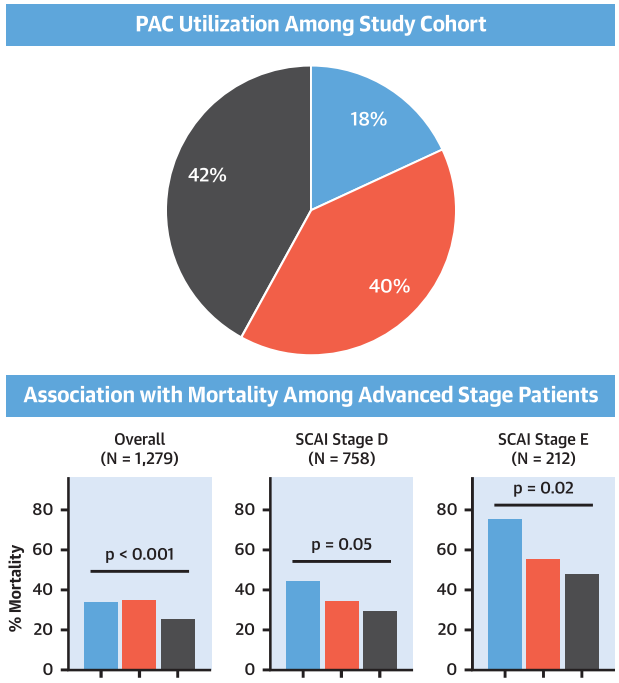
performed prior to therapy in 23% of cases. Patients who did not have invasive hemodynamics assessed prior to treatment at the study site were more likely to have a history of hypertension (54.3% vs. 47.7% vs. 45.8%, respectively; $p < 0.001$) whereas those with incomplete assessment were more likely to be diabetic (29.6% vs. 38.6% vs. 33.1%, respectively; $p < 0.001$).

PAC USAGE ASSOCIATION WITH MORTALITY. Crude mortality among patients according to SCAI stages were as follows: Stage B, 0%; Stage C, 10.65%; Stage D, 32.98%; and Stage E, 55.19% ($p < 0.001$). Patients whose CS had different causes experienced different rates of mortality in aggregate (AMI-CS, 39.47%; acute DHF-CS, 25.28%; and other 24.16%; $p < 0.001$). Mortality differed significantly among PAC groups within the overall cohort ($p < 0.001$) and each SCAI Stage subcohort (Stage C; $p = 0.03$; Stage D; $p = 0.05$; Stage E; $p = 0.02$) (Central Illustration). The complete PAC assessment group had the lowest in-hospital mortality compared to the other groups across all SCAI stages. Similarly, in both the acute DHF-CS and the AMI-CS cohorts, the complete PAC group had the lowest mortality ($p < 0.001$ in acute DHF-CS, and $p = 0.07$ in the AMI-CS group). After adjustments were made for comorbidities, cause of shock, and PAC usage per site, as mentioned above, having no PAC assessment was associated with significantly higher odds of mortality than having full PAC assessment in the overall cohort (adjusted OR: 1.57; 95% CI: 1.06 to 2.33) (Figure 2). Moreover, incomplete PAC assessment was associated with higher odds of mortality than complete PAC assessment in the overall cohort (adjusted OR: 1.71; 95% CI: 1.29 to 2.25). There were no significant differences between the odds of in-hospital mortality in no-PAC and incomplete PAC assessments.

HEMODYNAMIC PARAMETERS ASSOCIATED WITH IN-HOSPITAL MORTALITY. Hemodynamic parameters were available for analysis in 1,279 patients. Differences in hemodynamic measurements among the 3 groups are shown in Table 1, and the association between individual parameters with in-hospital mortality is shown in Figure 3. RAP and PCWP differed between those in the incomplete and complete PAC groups, but other hemodynamic parameters did not. Mean arterial pressure (MAP) and RAP differed significantly across quartiles in the overall, AMI-CS, and acute DHF-CS cohorts. Their associations appear to be linear, with decreased MAP and increased RAP significantly associated with higher mortality. Elevated heart rate was also associated with higher mortality, although the trend does not appear linear; PCWP, cardiac power output, and cardiac index did not appear to impact mortality consistently across the cohorts.

CENTRAL ILLUSTRATION **Frequency of Mortality Among PAC Use Overall and by SCAI Stage**

PAC Utilization	Study Definition
None	Presence of **NONE** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation • Right Atrial Pressure
Incomplete Assessment	Presence of **1-4** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation
Complete Assessment	Presence of **ALL** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation **AND** presence of Right Atrial Pressure



Garan, A.R. et al. J Am Coll Cardiol HF. 2020;8(11):903-13.

PAC = pulmonary artery catheter; SCAI = Society of Cardiovascular Angiography and Interventions.

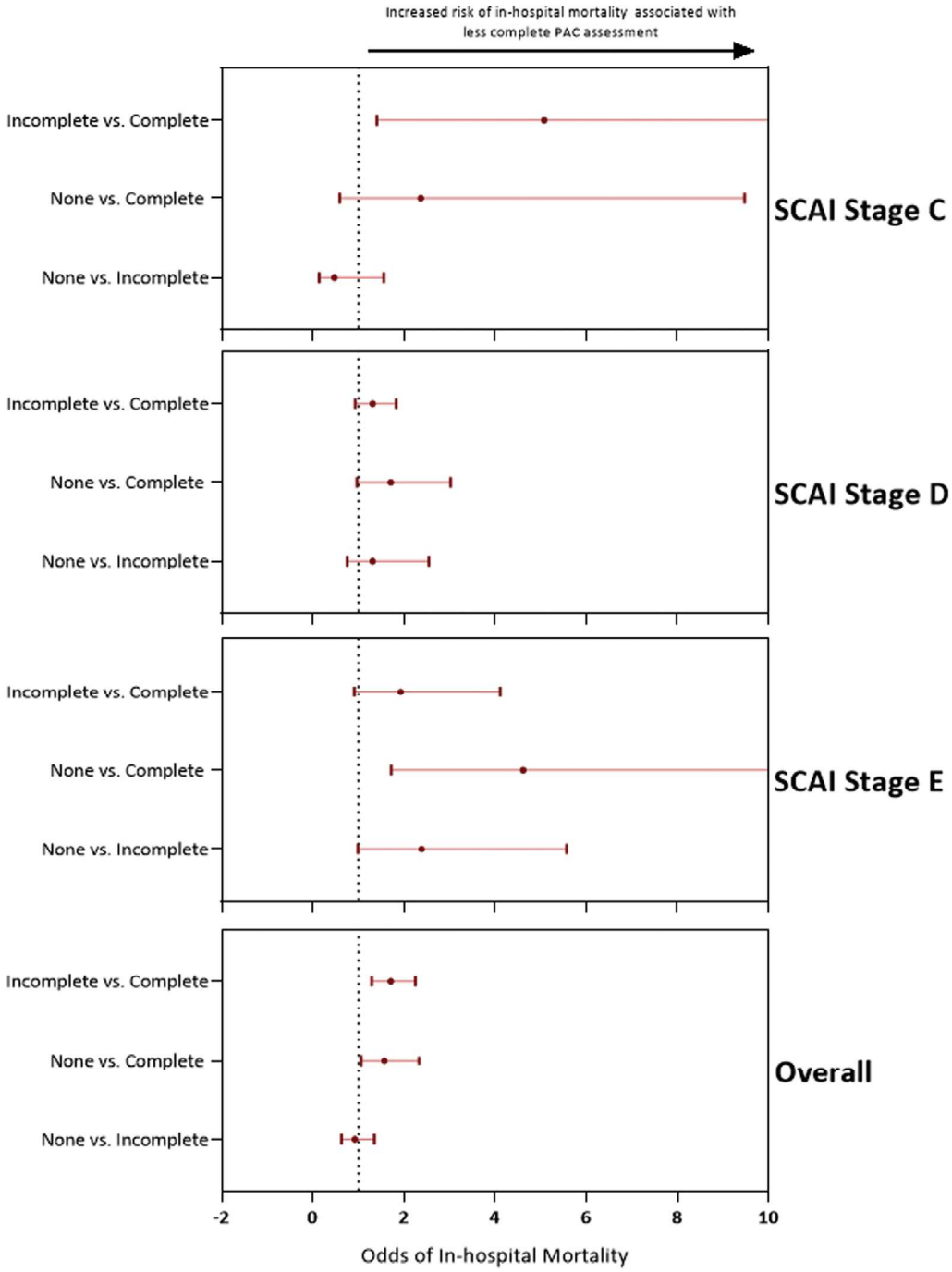
DISCUSSION

This study describes the association between clinical outcomes and use of PAC in one of the largest multicenter registries representing real-world patients with CS in the contemporary acute MCS era. It was observed that CS patients with complete PAC data obtained prior to MCS initiation had improved survival compared to those who did not, even after accounting for potentially confounding factors. This difference was more pronounced in the sickest cohort of patients (SCAI Stages D and E patients). Having an incomplete hemodynamic dataset was equivalent to having no PAC data with regard to in-hospital mortality. These data represent one of the largest multicenter "real-world" experiences with hemodynamic assessment for CS across multiple tertiary care centers.

The optimal use of hemodynamic monitoring with PACs in hospitalized patients with HF remains controversial. PACs became a ubiquitous feature of intensive care unit management in the 1980s and 1990s until several large trials showed no benefit to their use in broad populations of critically ill patients (13). Their use further declined after the ESCAPE (Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness) trial showed no benefit to routine use of PACs in decompensated patients with HF, but notably that trial excluded patients with CS (5). The 2013 American College of Cardiology/American Heart Association HF guidelines recommend the use of invasive hemodynamic monitoring using a PAC to guide therapy in patients with respiratory distress or evidence of impaired perfusion in whom the adequacy or excess of intracardiac filling pressures cannot be determined from clinical assessment (Class I, Level of Evidence: C) (14).

There have been major advances in acute HF management recently and especially in CS with temporary MCS therapy widely available (2). Early recognition and triage of patients with CS using specific therapeutic algorithms is increasingly common, including identification of the shock subtype and an understanding of the expected impact of a devices on parameters such as CO, PCWP, RAP, and MAP (3). Knowledge of these parameters allows the practitioner to choose the device or combination of devices

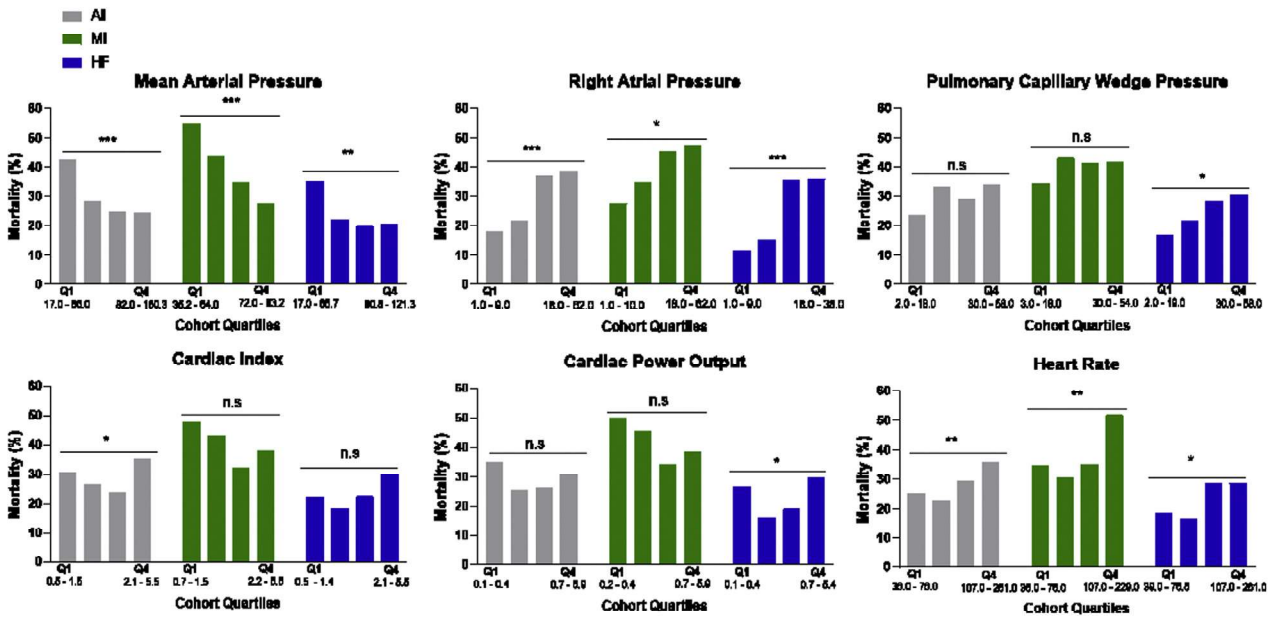
FIGURE 2 Adjusted Odds of Mortality Associated With Less Complete PAC Assessment



Odds ratios were adjusted for PAC use by institution, hypertension, cause of cardiogenic shock, diabetes, age, ventricular tachycardia, and implantable cardioverter-defibrillator. PAC = pulmonary artery catheter; SCAI = Society of Cardiovascular Angiography and Interventions.

that best match the patient's needs. Additional benefits from acquisition of complete PAC data include early identification of patients with significant hemodynamic compromise requiring immediate MCS, in order to avoid irreversible end-organ dysfunction resulting from treatment delays. Additionally, PAC data facilitate early recognition of a biventricular shock state which is often underappreciated and may

FIGURE 3 Frequency of Mortality by Hemodynamic Parameter Quartiles Assessed by Pac Stratified by Cause of Cardiogenic Shock



*p ≤ 0.05; **p ≤ 0.01; ***p ≤ 0.001. NS = not significant; PAC = pulmonary artery catheter.

require consideration of biventricular support (1). Finally, the continuous feedback obtained from the PAC facilitates optimization of volume status, adjustment of vasoactive medications in a more targeted fashion, and recognition of when patients can be weaned from such devices. Single-center reports have demonstrated that the use of PAC in patients with CS has been associated with lower short- and long-term mortality rates (15). Similarly, some registries have demonstrated an association between use of PAC and improved outcomes, although these datasets have lacked the granularity necessary to further understand this association (10,11). Not surprisingly, there is increasing focus on use of invasive hemodynamic monitoring for the management of CS (3).

The present analysis provides data from a large, contemporary registry including patients supported by multiple MCS device platforms to support the timely use of PACs in reducing mortality in CS. It has been previously established that patients with refractory shock (SCAI shock Stage E) had >20-fold higher crude in-hospital mortality than hemodynamically stable patients without shock (SCAI shock Stage A) (1). Not surprisingly, the present data promote the hypothesis that treatment decisions guided by early and complete hemodynamic profiling in patients with greater degrees of hemodynamic compromise lead to improved outcomes. This is likely because PAC-derived hemodynamic data not only confirm the severity of CS but also enable clinicians to monitor responses to therapeutic interventions. Much in the same way that "routine" use of PAC in decompensated HF did not prove beneficial, the benefit of obtaining PAC data in patients with less severe shock was not as evident in the present dataset. This is also reiterated in the American Heart Association scientific statement on management of CS, which emphasizes PAC use in patients with moderate to severe CS who are unresponsive to initial therapy (2).

Patients with incomplete characterization of the hemodynamic profile had worse outcomes than those with a complete evaluation in the present analysis. The authors speculate that this could be partly due to a compromise in underestimating the degree of right heart failure resulting in end-organ damage caused by hypoperfusion and congestion. Hemodynamic monitoring is always meant to complement other markers of end-organ perfusion in CS (2). However, estimation of hemodynamics based on the physical examination can be highly inaccurate, even for the experienced clinician, with exaggerated rates of misrecognition of the most high-risk patients (16). Using surrogates of a complete hemodynamic assessment or obtaining limited information from a PAC misses the

opportunity to fully define the patient's hemodynamic profile to guide therapeutic decision making.

Various hemodynamic indices have been shown to be prognostic indicators of outcomes in CS. In the present analysis, MAP, RAP, and heart rate were associated with mortality regardless of CS cause. Other parameters demonstrated prognostic importance in subsets of the registry. For example, PCWP correlated with mortality among patients with acute DHF-CS but not those with AMI-CS. These findings are consistent with those in other reports where intracardiac filling pressures correlated with outcomes in acute DHF patients (17). Interestingly, cardiac power output, which was shown to be a powerful prognostic measurement in AMI-CS, did not demonstrate similar value in this dataset (18). This may be due, in part, to the inclusion of multiple causes of cardiogenic shock, where this parameter is not known to have prognostic value. In addition, this difference may be explained, in part, by widespread use of MCS to improve this hemodynamic index in this registry population. Indeed, measurements of indices such as cardiac power outlet with PACs often informs decisions regarding MCS application.

STUDY LIMITATIONS. The present data are retrospective in nature and include inherent limitations. Patients in whom a PAC was placed after initiation of MCS were scored as having no PAC data in the present analysis. Furthermore, because registry data were derived from documentation in electronic health records, "incomplete" PAC data may have included instances where clinicians had complete PAC data at the bedside but did not subsequently document a complete set of hemodynamic values. It is important to note that, although an association was observed between PAC use in CS and reduced mortality, one cannot necessarily conclude causality. PAC-derived hemodynamics are diagnostic data and do nothing to improve the patient's condition, unless those data are interpreted accurately and are coupled with expeditious use of a treatment strategy aimed at improving outcomes. In addition, lack of PAC use may be related to the acuity of the patient; often, the severity of hemodynamic collapse can preclude the need or opportunity for diagnostic studies until patients are stabilized. In the present data, those requiring ECMO were least likely to have had a PAC placed prior to initiation of therapy, although it is also important to note that there was a relative underrepresentation of SCAI Stage D and E patients in the no-PAC group. Additionally, patients are often transferred to tertiary care hospitals after initiation of pressor/inotrope therapies or temporary MCS. Centers may use "hybrid" methodologies to combine minimally invasive and noninvasive methods to hemodynamically monitor CS patients without a PAC.

However, real-world, multicenter registry reports of more than 1,400 patients with CS help address the pressing need for additional evidence of PAC use in CS prior to initiation of MCS, which was emphasized in a recently published white paper on the subject (3). Future analyses will include prospectively collected data with PAC and MCS timing captured in order to examine how PAC data guide stepwise escalation of MCS therapies and explore the impact of PAC use on hospital costs and length of stay. Potentially important variables were not included in this multivariate model because they were missing in many cases (e.g., lactate concentrations) or missing disproportionately in the study subsets (e.g., MAP), although we anticipate prospective data collection will dramatically improve this limitation. Other key variables including recent cardiac arrest and hospital transfer, for example, which were not collected in this report are being captured in this evolving dataset. Although present registry data support PAC use to manage patients with CS requiring MCS, definitive randomized controlled trials are necessary to confirm this observation.

CONCLUSIONS

This study presents data from one of the largest multicenter registries representing real-world patients with CS in the contemporary acute MCS era. Use of complete hemodynamic data obtained by timely placement of PACs prior to MCS initiation was associated with lower mortality in patients with advanced Stages of CS.

AUTHOR RELATIONSHIP WITH INDUSTRY

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: There is considerable variability in PAC usage among tertiary care centers for patients with CS. Complete hemodynamic profiling of CS patients with PACs prior to MCS initiation was associated with improved outcomes in a large, multicenter registry of CS patients supported with multiple MCS device platforms. This association was particularly evident in patients with the greatest degree of hemodynamic compromise.

TRANSLATIONAL OUTLOOK: A randomized, controlled trial evaluating PAC use in patients with CS being considered for MCS is necessary because prior trials have largely excluded patients with CS, and none have been conducted in the era of widespread MCS use.

REFERENCES

1. Jentzer JC, van Diepen S, Barsness GW, et al. Cardiogenic shock classification to predict mortality in the cardiac intensive care unit. J Am Coll Cardiol 2019;74:2117–28.

2. van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. Circulation 2017;136(16): e232–68.

3. Saxena A, Garan AR, Kapur NK, et al. Value of hemodynamic monitoring in patients with cardiogenic shock undergoing mechanical circulatory support. Circulation 2020;141: 1184–97.

4. Richard C, Warszawski J, Anguel N, et al. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2003;290:2713–20.

5. Binanay C, Califf RM, Hasselblad V, et al. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial. JAMA 2005;294: 1625–33.

6. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. JAMA 2005;294:1664–70.

7. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. Catheter Cardiovasc Interv 2019;94:29–37.

8. Basir MB, Kapur NK, Patel K, et al. Improved outcomes associated with the use of shock protocols: updates from the national cardiogenic shock initiative. Catheter Cardiovasc Interv 2019; 93:1173–83.

9. Tehrani BN, Truesdell AG, Sherwood MW, et al. Standardized team-based care for cardiogenic shock. J Am Coll Cardiol 2019;73:1659–69.

10. O'Neill WW, Grines C, Schreiber T, et al. Analysis of outcomes for 15,259 US patients with acute myocardial infarction cardiogenic shock (AMICS) supported with the Impella device. Am Heart J 2018;202:33–8.

11. Hernandez GA, Lemor A, Blumer V, et al. Trends in utilization and outcomes of pulmonary artery catheterization in heart failure with and without cardiogenic shock. J Cardiac Fail 2019;25: 364–71.

12. Thayer KL, Zweck E, Ayouty M, et al. Invasive hemodynamic assessment and classification of in-hospital mortality risk among patients with cardiogenic shock. Circ Heart Fail 2020;13: e007099.

13. Marik PE. Obituary: pulmonary artery catheter 1970 to 2013. Ann Intensive Care 2013;3:38.

14. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;62:e147–239.

15. Rossello X, Vila M, Rivas-Lasarte M, et al. Impact of pulmonary artery catheter use on short- and long-term mortality in patients with cardiogenic shock. Cardiology 2017;136:61–9.

16. Narang N, Chung B, Nguyen A, et al. Discordance between clinical assessment and invasive hemodynamics in patients with advanced heart failure. J Cardiac Fail 2020;26:128–35.

17. Cooper LB, Mentz RJ, Stevens SR, et al. Hemodynamic predictors of heart failure morbidity and mortality: fluid or flow? J Cardiac Fail 2016; 22:182–9.

18. Fincke R, Hochman JS, Lowe AM, et al. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. J Am Coll Cardiol 2004;44: 340–8.

KEY WORDS cardiogenic shock, hemodynamics, mechanical circulatory support, pulmonary artery catheter

APPENDIX For supplemental tables, please see the online version of this paper.

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EDITORIAL COMMENT

Can a Pulmonary Artery Catheter Improve Outcomes in Cardiogenic Shock?*

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Mortality rates among patients presenting with cardiogenic shock (CS) remain unacceptably high, despite advances in reperfusion therapy and temporary mechanical circulatory support (MCS). Recent efforts in managing CS have included the development of specific definitions, management guidelines, and treatment algorithms. However, a paucity of high-quality evidence support current clinical practices. Consequently, there is much debate about the most appropriate course of action to take when faced with a patient in CS. One of these issues is the value of placing a pulmonary artery catheter (PAC), whether debated from a diagnostic and/or a therapeutic point of view. Intuitively, it makes sense that an invasive hemodynamic assessment may provide diagnostic clarity (e.g., right heart failure) in these highly varied clinical scenarios as well as provide information to guide a rational therapeutic response (e.g., temporary MCS). In fact, the PAC was once common in critical care management and touted by advanced heart failure centers as a routine practice.

However, routine hemodynamic monitoring with a PAC fell out of favor in the early 2000s after randomized clinical trials showed no outcome benefit with its use for chronic heart failure management or in critical care (1). In contrast, recent studies have suggested that, in select CS situations, hemodynamically guided protocols in concert with temporary MCS could improve outcomes (2). Moreover, it has become increasingly apparent that CS is not a homogeneous disorder and extrapolation of prior experiences in related disorders, such as heart failure and critical care, may not be appropriate. It is in this context that a report in this issue of *JACC: Heart Failure* attempts to address whether obtaining invasive hemodynamic data from a PAC in CS improves outcomes.

SEE PAGE 903

In one of the first reports from the Cardiogenic Shock Working Group (CSWG), Garan et al. (3) examined the relationship between hemodynamic information obtained by PAC in patients presenting with CS relative and in-hospital mortality. The analysis was performed using a retrospective dataset from 8 tertiary care hospitals (i.e., >100 CS cases each) and included all patients presenting with CS, regardless of cause. Use of PAC was categorized as "no PAC," "incomplete PAC," or "complete PAC" and had to be placed prior to MCS initiation. Patients were further divided into retrospectively assigned Society for Cardiovascular Angiography and Interventions (SCAI) CS classification (4), and mortality was assessed by the use of PAC. Mortality was significantly lower in the complete PAC group in the overall cohort, as well as in those patients presenting with severe CS (defined as SCAI CS Stage D or E). When stratified by CS cause (e.g., acute myocardial infarction [AMI] vs. acute heart failure [AHF]), patients with complete PAC hemodynamics also had the lowest mortality. Patients with AHF were also more likely to have a complete PAC assessment compared to those presenting with AMI (52.8% vs. 32.2%, respectively; $p < 0.001$). The authors hypothesized that having complete PAC hemodynamic data aided in earlier identification of CS

*Editorials published in *JACC: Heart Failure* reflect the views of the authors and do not necessarily represent the views of *JACC: Heart Failure* or the American College of Cardiology.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Heart Failure* author instructions page.

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and provided treatment guidance, which translated into reduced mortality.

An important observation made in that report was that most patients who underwent complete PAC hemodynamic assessment prior to therapy escalation were those presenting with AHF; 66% of patients with complete PAC assessment had a history of HF. Patients in the overall cohort with different CS causes notably experienced significant differences in mortality (AMI, 39.47%; ADHF, 25.28%; Other, 24.16%; $p < 0.001$). This finding was similar to that in a previous study showing patients with acute CS, compared to acute decompensation of chronic HF were more likely to have in-hospital mortality (5). Although complete PAC hemodynamics were associated with the lowest mortality among all groups in the analysis, the cause of CS is an important factor contributing to heterogeneity, and a PAC may help to refine such CS subsets. Acute and chronic HF would appear to be pathophysiologically distinct from the acute hemodynamic collapse of an acute coronary syndrome. Going forward, parsing CS analyses and therapeutic strategies by cause would appear to be prudent.

Other noteworthy aspects of the analysis included the multiple centers contributing data, size of the cohort, use of the recently developed SCAI CS classification, and the separation of chronic HF patients (with an acute exacerbation) from the acute myocardial infarction population. In addition, the authors were careful to not overstate the conclusions and were forthcoming about limitations, including the retrospective data collection, the associative nature of their findings, and the lack of detail regarding therapeutic decisions based upon a diagnostic tool.

These are important real-world data, but there are reasons to be cautious. Foremost, which the authors acknowledge, is that patients presenting with the highest acuity may be less likely to have invasive hemodynamic assessment performed prior to death or initiation of MCS. The rapid deterioration and hemodynamic collapse of such patients would provide a negative survival bias to the "no-PAC" group. In fact, patients in the "no-PAC" group had significantly higher lactate levels and lower pH, both measurements of CS severity (4). Furthermore, patients requiring extracorporeal membrane oxygenation (ECMO) and, arguably the most severe case of CS, were least likely to have a PAC placed prior to MCS initiation. The report also lacks other details. For example, specific SCAI stages at the time of PAC placement and time to PAC placement or time to death from hospital presentation were not reported, which would help to address timeliness of PAC use. Also, treatment decisions based on the hemodynamic data and resulting complications were not catalogued. Finally, many patients were transferred to the tertiary care investigator sites in the registry, and the generalizability of the findings to the first encounter hospital is unclear.

Despite the aforementioned limitations, the authors are to be commended for providing more observational evidence for a debated practice and linking the approach to CS outcomes. Given the relatively low risk and modest invasive nature of PAC placement, the potential for the resource-intensive circulatory support and the overall high mortality of CS, one might conclude that the results of this analysis are enough to influence practice patterns in favor of early invasive hemodynamic assessment. Critics of this ideology may argue that CS is as much a clinical diagnosis as it is a hemodynamic one, and there may be risks to hemodynamically driven treatment decisions that lead to overly aggressive escalation of care when benefits are unknown. It is important to emphasize that a PAC is a diagnostic tool, not a therapeutic one. The CardioMEMS experience has illustrated the importance of matching diagnostic power with well thought out therapeutic responses. Any trial of PAC use in CS should have PAC-based therapeutic decisions that are clearly laid out.

Meantime, while we await more data and future trials, should a PAC be placed, if possible, in CS? We would advocate such a decision be made by specialized teams where the collective experience of diverse providers can be brought to bear. If we accept the complexity of CS and the limitations of current evidence, the best decisions for our patients are likely made by a group of coordinated providers to improve outcomes (6). In our opinion, the PAC does provide important information to define the nature of CS, but its ultimate utility will dependent upon the therapeutic responses that ensue. We should remember that we have been here before; the ESCAPE (Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness) trial was an important lesson that only randomized evidence will settle the PAC debate.

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REFERENCES

1. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. JAMA 2005;294:1664–70.

2. Basir MB, Kapur NK, Patel K, et al. Improved outcomes associated with the use of shock protocols: updates from the national cardiogenic shock initiative. Catheter Cardiovasc Interv 2019;93:1173–83.

3. Garan AR, Kanwar M, Thayer KL, et al. Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower in-hospital mortality. J Am Coll Cardiol HF 2020;8:903–13.

4. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. Catheter Cardiovasc Interv 2019;94:29–37.

5. Jones TL, Tan MC, Nguyen V, et al. Outcome differences in acute vs. acute on chronic heart failure and cardiogenic shock. ESC Heart Fail 2020;7:1118–24.

6. Taleb I, Koliopoulou AG, Tandar A, et al. Shock team approach in refractory cardiogenic shock requiring short-term mechanical circulatory support: a proof of concept. Circulation 2019;140:98–100.

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