Title
Prognostic significance of haemodynamic parameters in patients with cardiogenic shock.

Journal
European Heart Journal: Acute Cardiovascular Care, 12(10)

Authors
Berg, David
Kaur, Gurleen
Bohula, Erin
et al.

Publication Date
2023-10-25

DOI
10.1093/ehjacc/zuad095

Peer reviewed

ESC European Society of Cardiology

European Heart Journal: Acute Cardiovascular Care (2023) **12**, 651–660
https://doi.org/10.1093/ehjacc/zuad095

ORIGINAL SCIENTIFIC PAPER
Heart Failure and Cardiomyopathies

Prognostic significance of haemodynamic parameters in patients with cardiogenic shock

David D. Berg [1]*, Gurleen Kaur[2], Erin A. Bohula[1], Vivian M. Baird-Zars[1], Carlos L. Alviar[3], Christopher F. Barnett[4], Gregory W. Barsness[5], James A. Burke[6], Sunit-Preet Chaudhry[7], Meshe Chonde[8], Howard A. Cooper[9], Lori B. Daniels[10], Mark W. Dodson[11], Daniel A. Gerber[12], Shahab Ghafghazi[13], Umesh K. Gidwani[14], Michael J. Goldfarb[15], Jianping Guo[1], Dustin Hillerson[16], Benjamin B. Kenigsberg[17], Ajar Kochar [1], Michael C. Kontos[18], Younghoon Kwon[19], Mathew S. Lopes[1], Daniel B. Loriaux[20], P. Elliott Miller[21], Connor G. O'Brien[4], Alexander I. Papolos[17], Siddharth M. Patel [1], Barbara A. Pisani[22], Brian J. Potter[23], Rajnish Prasad[24], Robert O. Rowsell[25], Kevin S. Shah[26], Shashank S. Sinha[27], Timothy D. Smith[28], Michael A. Solomon [29], Jeffrey J. Teuteberg[12], Andrea D. Thompson[30], Sammy Zakaria[31], Jason N. Katz[20], Sean van Diepen [32], and David A. Morrow[1], on behalf of the CCCTN Investigators

[1]Levine Cardiac Intensive Care Unit, Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 60 Fenwood Road, Suite 7022, Boston, MA 02115, USA; [2]Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [3]Leon H Charney Division of Cardiology, Bellevue Hospital Center, New York University School of Medicine, New York, NY, USA; [4]Division of Cardiology, Department of Medicine, University of California San Francisco, San Francisco, CA, USA; [5]Department of Cardiovascular Medicine, Mayo Clinic, Rochester, MN, USA; [6]Division of Cardiology, Lehigh Valley Heart Network, Allentown, PA, USA; [7]Department of Medicine, St Vincent Heart Center, Indianapolis, IN, USA; [8]Department of Cardiology, Smidt Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, USA; [9]Westchester Medical Center, New York Medical College, Valhalla, NY, USA; [10]Division of Cardiovascular Medicine, Department of Medicine, University of California San Diego, La Jolla, CA, USA; [11]Department of Pulmonary and Critical Care Medicine, Intermountain Medical Center, Murray, UT, USA; [12]Division of Cardiovascular Medicine, Stanford University School of Medicine, Stanford, CA, USA; [13]Cardiovascular Medicine, University of Louisville, Louisville, KY, USA; [14]Division of Cardiology, Zena and Michael A. Wiener Cardiovascular Institute, Icahn School of Medicine at Mount Sinai, New York, NY, USA; [15]Division of Cardiology, Jewish General Hospital, McGill University, Montreal, QC, Canada; [16]Department of Medicine, Division of Cardiovascular Medicine, University of Wisconsin School of Medicine and Public Health, Madison, WI, USA; [17]Departments of Cardiology and Critical Care, MedStar Washington Hospital Center, Washington, DC, USA; [18]Division of Cardiology, Department of Medicine, Virginia Commonwealth University, Richmond, VA, USA; [19]Division of Cardiology, University of Washington, Seattle, WA, USA; [20]Division of Cardiology, Department of Medicine, Duke University, Durham, NC, USA; [21]Section of Cardiovascular Medicine, Yale University, New Haven, CT, USA; [22]Section of Cardiovascular Medicine, Department of Internal Medicine, Wake Forest Baptist Medical Center, Winston-Salem, NC, USA; [23]Cardiology Service, Department of Medicine, Centre Hospitalier de l'Université de Montréal (CHUM) Research Center and Cardiovascular Center, Montreal, QC, Canada; [24]Division of Cardiology, Wellstar Health System, Marietta, GA, USA; [25]Division of Cardiology, Lenox Hill Hospital, Northwell Health, Zucker School of Medicine, New York, NY, USA; [26]Division of Cardiology, Department of Medicine, University of Utah, Salt Lake City, UT, USA; [27]Inova Heart and Vascular Institute, Inova Fairfax Medical Center, Falls Church, VA, USA; [28]Lindner Center for Research and Education, The Christ Hospital, Cincinnati, OH, USA; [29]Critical Care Medicine Department, National Institutes of Health Clinical Center and Cardiovascular Branch, National Heart, Lung, and Blood Institute of the National Institutes of Health, Bethesda, MD, USA; [30]Division of Cardiovascular Medicine, Department of Internal Medicine, University of Michigan, Ann Arbor, MI, USA; [31]Division of Cardiology, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, USA; and [32]Department of Critical Care Medicine and Division of Cardiology, Department of Medicine, University of Alberta, Edmonton, Alberta, Canada

Received 10 June 2023; revised 17 July 2023; accepted 6 August 2023; online publish-ahead-of-print 28 August 2023

Aims Invasive haemodynamic assessment with a pulmonary artery catheter is often used to guide the management of patients with cardiogenic shock (CS) and may provide important prognostic information. We aimed to assess prognostic associations and relationships to end-organ dysfunction of presenting haemodynamic parameters in CS.

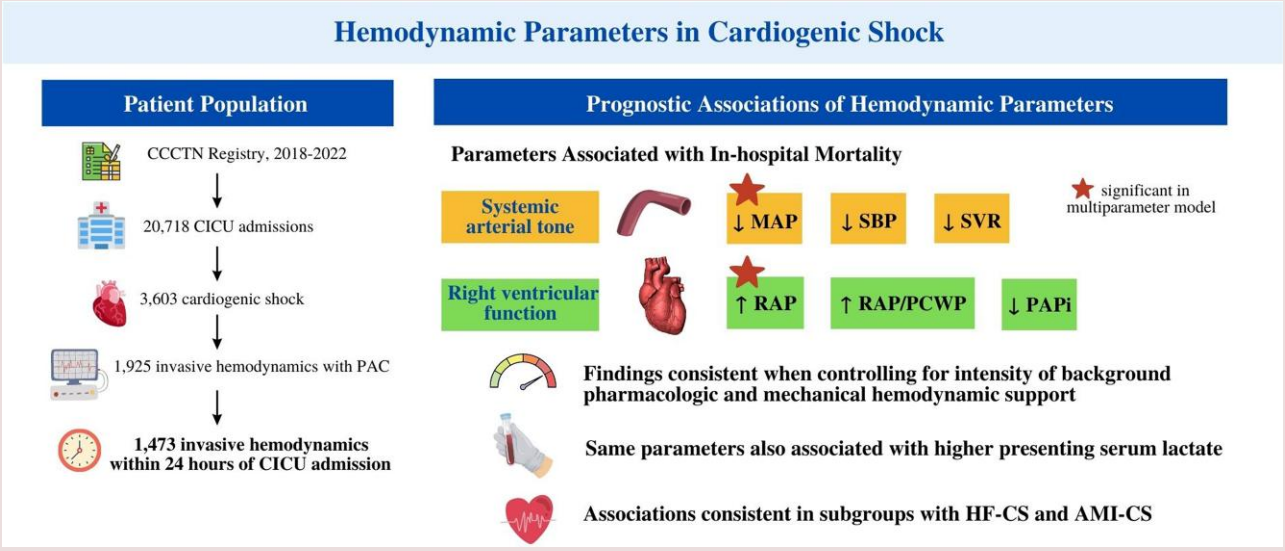
Methods and results The Critical Care Cardiology Trials Network is an investigator-initiated multicenter registry of cardiac intensive care units (CICUs) in North America coordinated by the TIMI Study Group. Patients with CS (2018–2022) who underwent invasive

* Corresponding author. Tel: +1 617 278 0145, Fax: +1 617 734 7329, Email: dberg1@bwh.harvard.edu, Twitter: @ddbergMD @TIMIStudyGroup

haemodynamic assessment within 24 h of CICU admission were included. Associations of haemodynamic parameters with in-hospital mortality were assessed using logistic regression, and associations with presenting serum lactate were assessed using least squares means regression. Sensitivity analyses were performed excluding patients on temporary mechanical circulatory support and adjusted for vasoactive-inotropic score. Among the 3603 admissions with CS, 1473 had haemodynamic data collected within 24 h of CICU admission. The median cardiac index was 1.9 (25th–75th percentile, 1.6–2.4) L/min/m^2 and mean arterial pressure (MAP) was 74 (66–86) mmHg. Parameters associated with mortality included low MAP, low systolic blood pressure, low systemic vascular resistance, elevated right atrial pressure (RAP), elevated RAP/pulmonary capillary wedge pressure ratio, and low pulmonary artery pulsatility index. These associations were generally consistent when controlling for the intensity of background pharmacologic and mechanical haemodynamic support. These parameters were also associated with higher presenting serum lactate.

Conclusion In a contemporary CS population, presenting haemodynamic parameters reflecting decreased systemic arterial tone and right ventricular dysfunction are associated with adverse outcomes and systemic hypoperfusion.

Graphical Abstract



Keywords Cardiogenic shock • Haemodynamics • Pulmonary artery catheter • Outcomes

Introduction

Cardiogenic shock (CS) is a complex clinical syndrome characterized by inadequate tissue perfusion due to ineffective cardiac output (CO).[1] Short-term mortality for patients with CS remains high (30–40% in contemporary registries and clinical trials),[2–4] underscoring the critical need for improved risk stratification and management strategies in CS.

Although there are few data to inform appropriate monitoring in patients with CS, and clinical practice patterns vary widely,[5] invasive haemodynamic assessment with a pulmonary artery catheter (PAC) is often used to guide management.[6] Invasive haemodynamic data provided by a PAC can confirm the presence and severity of CS, the pattern of ventricular involvement [i.e. left ventricular, right ventricular (RV), or biventricular], and vascular resistances in the pulmonary and systemic arterial beds. PACs also allow clinicians to monitor responses to therapeutic interventions and may provide important prognostic information. For these reasons, some experts have advocated for routine invasive haemodynamic assessment in patients with CS.[7] Notably, several observational analyses have supported a possible benefit of complete invasive haemodynamic assessment,[5,8,9] and a randomized trial to rigorously assess the impact of early PAC use in patients with CS due to decompensated heart failure (HF-CS) is now underway (NCT05485376).

Given the increased focus on invasive haemodynamic assessment in the management of CS, it is important to better understand the relationships between specific haemodynamic parameters, shock severity, and outcomes. Accordingly, we aimed to assess the prognostic importance of specific haemodynamic parameters with respect to mortality and the associations of these parameters with end-organ dysfunction in a well-characterized cohort of patients with CS from a multinational registry.

Methods

Study population

The Critical Care Cardiology Trials Network (CCCTN) is an investigator-led collaborative research network of advanced cardiac

Table 1 Clinical characteristics of cardiogenic shock patients with and without invasive haemodynamic assessment within 24 h of CICU admission

	All CS admissions (*N* = 3603)	CS admissions in primary analysis cohort (*N* = 1473)	CS admissions not in primary analysis cohort (*N* = 2130)	*P*-value
Demographics				
Age, median (25th–75th), year	65 (55–74)	63 (53–72)	66 (56–75)	<0.001
Female sex, *n* (%)	1162 (32.3%)	431 (29.3%)	731 (34.3%)	0.001
Race				
White, *n* (%)	2085 (57.9%)	897 (60.9%)	1188 (55.8%)	<0.001
Black, *n* (%)	709 (19.7%)	305 (20.7%)	404 (19.0%)	
Other, *n* (%)	809 (22.5%)	271 (18.4%)	538 (25.3%)	
BMI, median (25th–75th), kg/m^2	27.6 (23.9–32.3)	27.9 (24.3–32.6)	27.4 (23.7–32.2)	0.014
Comorbidities				
Diabetes mellitus, *n* (%)	1362 (37.8%)	561 (38.1%)	801 (37.6%)	0.770
Hypertension, *n* (%)	2114 (58.7%)	847 (57.5%)	1267 (59.5%)	0.235
Coronary artery disease, *n* (%)	1280 (35.5%)	531 (36.1%)	749 (35.2%)	0.586
Cerebrovascular disease, *n* (%)	308 (8.6%)	109 (7.4%)	199 (9.3%)	0.040
Peripheral artery disease, *n* (%)	316 (8.8%)	111 (7.5%)	205 (9.6%)	0.029
Heart failure, *n* (%)	1944 (54.0%)	836 (56.8%)	1108 (52.0%)	0.005
Atrial fibrillation, *n* (%)	1136 (31.5%)	449 (30.5%)	687 (32.3%)	0.261
Ventricular arrhythmia, *n* (%)	315 (8.7%)	154 (10.5%)	161 (7.6%)	0.003
Severe valvular disease, *n* (%)	571 (15.9%)	234 (15.9%)	337 (15.8%)	0.959
Pulmonary hypertension, *n* (%)	272 (7.6%)	107 (7.3%)	165 (7.8%)	0.590
Chronic kidney disease, *n* (%)	995 (27.6%)	391 (26.5%)	604 (28.4%)	0.232
On dialysis, *n* (%)	157 (15.8%)	55 (14.1%)	102 (16.9%)	0.233
Pulmonary disease, *n* (%)	507 (14.1%)	200 (13.6%)	307 (14.4%)	0.478
Liver disease, *n* (%)	96 (2.7%)	34 (2.3%)	62 (2.9%)	0.270
Shock type				
AMI-CS, *n* (%)	939 (26.1%)	416 (28.2%)	523 (24.6%)	<0.001
HF-CS, *n* (%)	2197 (61.0%)	963 (65.4%)	1234 (57.9%)	
De novo, *n* (%)	609 (16.9%)	247 (16.8%)	362 (17.0%)	
Acute-on-chronic, *n* (%)	1588 (44.1%)	716 (48.6%)	872 (40.9%)	
Other CS, *n* (%)	467 (13.0%)	94 (6.4%)	373 (17.5%)	
Illness severity				
SOFA score, median (25th–75th)	7 (5–10)	7 (4–10)	7 (5–10)	0.106
SCAI stage				
C, *n* (%)	661 (18.4%)	231 (15.7%)	430 (20.2%)	<0.001
D, *n* (%)	1081 (30.0%)	496 (33.7%)	585 (27.5%)	
E, *n* (%)	1082 (30.0%)	403 (27.4%)	679 (31.9%)	
Unknown, *n* (%)	779 (21.6%)	343 (23.3%)	436 (20.5%)	
Preceding cardiac arrest, *n* (%)	826 (22.9%)	298 (20.2%)	528 (24.8%)	0.001
Presenting LVEF				
<20%	640 (34.1%)	322 (39.6%)	318 (29.9%)	<0.001
20–<30%	540 (28.7%)	242 (29.7%)	298 (28.0%)	
30–<40%	276 (14.7%)	111 (13.6%)	165 (15.5%)	
40–<50%	58 (7.1%)	58 (7.1%)	93 (8.7%)	
≥50%	272 (14.5%)	81 (10.0%)	191 (17.9%)	
Laboratory values				
Lactate, median (25th–75th), mmol/L	2.6 (1.5–4.9)	2.3 (1.4–4.4)	2.8 (1.6–5.1)	<0.001

Continued

Table 1 ***Continued***

	All CS admissions (*N* = 3603)	CS admissions in primary analysis cohort (*N* = 1473)	CS admissions not in primary analysis cohort (*N* = 2130)	*P*-value
Arterial pH, median (25th–75th)	7.34 (7.25–7.42)	7.35 (7.27–7.42)	7.34 (7.23–7.42)	0.053
Total bilirubin, median (25th–75th), mg/dL	1.0 (0.6–1.8)	1.0 (0.6–1.8)	1.0 (0.6–1.9)	0.986
eGFR, median (25th–75th), mg/dL	46 (29–66)	46 (30–69)	45 (28–65)	0.049
Platelets, median (25th–75th), 1000/uL	202 (149–263)	203 (153–262)	202 (147–264)	0.603
ALT >200 U/L, *n* (%)	905 (27.2%)	391 (27.9%)	514 (26.7%)	0.445
CICU resource utilization				
LOS, median (25th–75th), days	10.6 (5.2–21.5)	12.4 (6.3–24.5)	9.5 (4.3–19.1)	<0.001
Mechanical ventilation, *n* (%)	1663 (46.2%)	733 (49.8%)	930 (43.7%)	<0.001
Acute RRT, *n* (%)	472 (13.1%)	229 (15.6%)	243 (11.4%)	<0.001
Shock management				
Vasoactive medications, median (25th–75th), #	1 (1–2)	1 (1–2)	1 (1–2)	0.369
VIS @ 4 h	4.0 (0.0–11.0)	4.0 (0.0–11.0)	4.0 (0.0–11.0)	0.432
VIS @ 24 h	2.8 (0.0–8.0)	3.8 (0.0–9.0)	2.5 (0.0–7.5)	<0.001
Mechanical circulatory support, *n* (%)	1328 (36.9%)	804 (54.6%)	524 (24.6%)	<0.001
IABP, *n* (%)	884 (24.5%)	510 (34.6%)	374 (17.6%)	<0.001
Impella, *n* (%)	374 (10.4%)	256 (17.4%)	118 (5.5%)	<0.001
TandemHeart, *n* (%)	50 (1.4%)	33 (2.2%)	17 (0.8%)	<0.001
VA-ECMO, *n* (%)	127 (4.9%)	61 (5.9%)	66 (4.2%)	0.057
Surgical (non-durable) VAD, *n* (%)	178 (4.9%)	125 (8.5%)	53 (2.5%)	<0.001
Mortality				
In-hospital mortality, *n* (%)	1067 (29.6%)	400 (27.2%)	667 (31.3%)	0.003
CICU mortality, *n* (%)	822 (22.8%)	311 (21.1%)	511 (24.0%)	0.023

ALT, alanine aminotransferase; AMI, acute myocardial infarction; BMI, body mass index; CICU, cardiac intensive care unit; CS, cardiogenic shock; eGFR, estimated glomerular filtration rate; HF, heart failure; IABP, intra-aortic balloon pump; LOS, length of stay; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; RRT, renal replacement therapy; SCAI, Society of Cardiovascular Angiography and Intervention; SOFA, sequential organ failure assessment; VIS, vasoactive-inotropic score; VA-ECMO, veno-arterial extracorporeal membrane oxygenation; VAD, ventricular assist device.

intensive care units (CICUs)[10] in the United States and Canada, with scientific and operational oversight provided by the TIMI Study Group (Boston, MA). Methods for the CCCTN Registry have been previously described.[11] From 2018 to 2022, a total of 39 participating centres contributed clinical data on all consecutive medical admissions to the CICU during annual 2-month collection periods. In addition, year-round capture of consecutive admissions was permitted. The study complies with the Declaration of Helsinki, and the research protocol and waiver of informed consent were approved by the ethics committees at each of the participating institutions. We encourage parties interested in collaboration and data sharing to contact the corresponding author directly for further discussions.

The presence of shock was assessed by site investigators and categorized by type of shock as previously described.[3] The aetiology of CS was further subdivided into CS due to acute myocardial infarction (AMI-CS), HF-CS, and secondary (non-myocardial) CS (e.g. severe valvular disease, pericardial tamponade). Starting in 2018, invasive haemodynamic data were collected as optional fields in the electronic case record form. All admissions, including clinical characteristics and haemodynamic parameters, were reviewed by the central coordinating centre via automated checks and manual reviews.

The present analysis was restricted to patients with CS at the time of CICU admission and excluded patients with post-cardiotomy shock. Clinician-assigned mixed shock patients were also excluded, because the prognostic relationships of haemodynamic parameters for certain subphenotypes within this category (e.g. sepsis in patients with underlying cardiomyopathy) may not generalize to the broader CS population. The primary analysis cohort included only those patients who underwent invasive haemodynamic assessment with a PAC within 24 h of CICU admission (including immediately before CICU admission). This time window was selected to focus on the prognostic significance of presenting haemodynamic profiles rather than on haemodynamic trajectories, and to align with other data elements collected on CICU presentation [e.g. clinical characteristics, laboratory values, initial Society of Cardiovascular Angiography and Interventions (SCAI) stage, initial Sequential Organ Failure Assessment (SOFA) score]. Presenting lactate values were those obtained in closest proximity to CICU admission, with a window between 24 h prior to CICU admission and 12 h post-CICU arrival. For analyses comparing the prognostic performance of different haemodynamic parameters, the analysis cohort was restricted further to include only those patients with complete haemodynamic information. No imputation was performed.

Table 2 Distribution of haemodynamic parameters in the full cohort and in the subgroups of patients with AMI-CS and HF-CS

	All cardiogenic shock		AMI-CS		HF-CS	
Parameter	**N**	**Median (25th–75th)**	**N**	**Median (25th–75th)**	**N**	**Median (25th–75th)**
HR (bpm)	1444	91 (75–108)	399	90 (75–106)	952	91 (77–108)
SBP (mmHg)	1444	101 (89–115)	405	99 (84–117)	946	101 (91–114)
MAP (mmHg)	1446	74 (66–86)	404	71 (63–87)	949	76 (67–86)
RAP (mmHg)	1412	15 (10–19)	394	13 (10–18)	928	15 (10–20)
PASP (mmHg)	1444	48 (38–59)	408	42 (34–51)	943	50 (40–61)
PADP (mmHg)	1451	25 (20–31)	407	22 (17–28)	941	26 (20–32)
PCWP (mmHg)	1234	24 (18–30)	345	22 (16–28)	810	25 (19–31)
CI (L/min/m^2)	1395	1.9 (1.6–2.4)	383	2.1 (1.7–2.6)	925	1.9 (1.5–2.3)
SVR (dynes*sec*/cm^5)	1326	1256 (899–1688)	356	1160 (855–1559)	886	1312 (934–1737)
PVR (dynes*sec*/cm^5)	1185	164 (86–273)	322	133 (70–213)	788	176 (92–293)
CPO (W)	1367	0.65 (0.49–0.84)	371	0.69 (0.50–0.89)	910	0.64 (0.50–0.82)
RAP/PCWP	1206	0.60 (0.44–0.80)	333	0.64 (0.47–0.80)	797	0.59 (0.43–0.80)
PAPI	1394	1.6 (1.0–2.5)	386	1.5 (0.9–2.2)	919	1.6 (1.1–2.6)
RVSWI (g-m/m^2)	1315	5.3 (3.3–7.9)	347	5.0 (3.0–7.5)	884	5.3 (3.3–7.9)
TPG (mmHg)	1228	8 (4–13)	342	7 (4–11)	807	9 (5–13)
PA Compliance	1345	1.9 (1.3–3.0)	359	2.5 (1.7–3.7)	900	1.8 (1.2–2.7)
PA Elastance	1349	1.1 (0.8–1.6)	361	0.9 (0.6–1.2)	902	1.2 (0.8–1.7)

The distributions are provided for all patients with available measurements for a particular haemodynamic parameter among patients with CS in the primary analysis cohort ($n = 1488$). AMI-CS, acute myocardial infarction-related cardiogenic shock; CI, cardiac index; CPO, cardiac power output; DPG, diastolic pressure gradient; HF-CS, heart failure-related cardiogenic shock; HR, heart rate; MAP, mean arterial pressure; MCS, mechanical circulatory support; PA, pulmonary artery; PADP, pulmonary artery diastolic pressure; PAPI, pulmonary artery pulsatility index; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RVSWI, right ventricular stroke work index; SBP, systolic blood pressure; SVR, systemic vascular resistance; TPG, transpulmonary gradient; VIS, vasoactive-inotropic score.

Haemodynamic parameters and background haemodynamic support

Standardized data elements captured in the CCCTN registry include vital signs [heart rate (HR), systolic blood pressure (SBP), mean arterial pressure (MAP)] and the following invasive haemodynamic parameters: right atrial pressure (RAP), pulmonary artery systolic pressure (PASP), pulmonary artery diastolic pressure (PADP), pulmonary capillary wedge pressure (PCWP), and cardiac index (CI). Although there are multiple methods for estimating CO, estimates based on the Fick principle using a pulmonary artery oxygen saturation were prioritized in the present analysis since this was the most common method for CO estimation among patients in our dataset. In cases where a CO estimate based on the Fick principle was not available, CO estimates using thermodilution were used (12.5%). In addition, the following haemodynamic parameters were derived (see Supplementary material online, *Methods*): cardiac power output (CPO), systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), pulmonary artery pulsatility index (PAPI), RAP/PCWP ratio, RV stroke work index (RVSWI), diastolic pressure gradient (DPG), transpulmonary gradient (TPG), pulmonary artery compliance, and pulmonary artery elastance. A patient was considered to have complete haemodynamic information if all measured and derived parameters were available.

Patients were categorized as being on pharmacologic (continuous infusions) and/or temporary mechanical circulatory support (MCS) at the time of invasive haemodynamic assessment. To quantify the intensity of background pharmacologic therapy, the vasoactive-inotropic score (VIS) was calculated (see Supplementary material online, *Methods*).[12] Based on temporal proximity to the timing of invasive haemodynamic assessment, either a 4-hour or 24-hour assessment of VIS was used.[13] Temporary MCS devices included intra-aortic balloon pump (IABP) counterpulsation, Impella percutaneous ventricular assist systems (CP, 5.0, 5.5, RP), TandemHeart percutaneous ventricular assist systems, and veno-arterial extracorporeal membrane oxygenation (VA-ECMO).[14]

Statistical analysis

Clinical characteristics were summarized for CS patients who underwent invasive haemodynamic assessment within 24 h of CICU admission and for those who did not. Categorical variables are shown as counts and percentages, and continuous variables as medians with 25th–75th percentile ranges. Differences in the clinical characteristics were evaluated using Pearson's chi-square test for categorical variables and the Wilcoxon rank sum test for continuous variables.

The distributions of each haemodynamic parameter were summarized using all available data among cases in the primary analysis cohort and for the subgroups of patients with AMI-CS and HF-CS. The associations between each haemodynamic parameter and in-hospital mortality were evaluated using univariable logistic regression. The results are presented graphically in descending order of statistical strength of association based on Wald χ^2 value from the logistic regression model, and odds ratios with 95% confidence intervals are provided. For each parameter significantly associated with in-hospital mortality in univariable analysis, the following analyses were performed to account for background therapy and clinical characteristics: (i) adjusted analyses incorporating VIS as a covariate in the logistic regression model (Model 2); (ii) sensitivity analyses excluding patients on any form of temporary MCS at the time of invasive haemodynamic assessment and adjusting for VIS (Model 3); and (iii) sensitivity analyses excluding patients on any form of temporary MCS at the time of invasive haemodynamic assessment and adjusting for VIS, age, sex, SCAI stage, and cardiac arrest before CICU admission (Model 4). VIS was not collected

in the first two annual cycles of the registry (2017–2019), so the VIS-adjusted analyses excluded approximately 22% of patients present in the primary analysis cohort. To evaluate independent prognostic associations, each parameter significantly associated with in-hospital mortality in univariable analysis was also included in a multi-parameter model and a backward selection procedure was applied. Correlations between variables in the multi-parameter model were assessed (see Supplementary material online, *Table S1*) and variance inflation factors (VIFs) examined to ensure that there were no concerns about multicollinearity.

To evaluate the associations of haemodynamic variables with systemic malperfusion, presenting serum lactate (continuous) was regressed on each haemodynamic parameter using least squares (LS) means regression. In addition, adjusted analyses incorporating VIS as a covariate in each LS means regression model and sensitivity analyses excluding patients on any form of temporary MCS at the time of invasive haemodynamic assessment and adjusting for VIS were performed. Results are presented graphically in descending order of the statistical strength of association of each haemodynamic parameter based on the absolute value of the *t* statistic for the haemodynamic parameter in the model.

To assess for differences in the prognostic significance of haemodynamic parameters in the subgroups of patients with AMI-CS and HF-CS, the associations of each haemodynamic parameter with in-hospital mortality and presenting serum lactate were evaluated using the same approach. Heterogeneity of association was formally tested by incorporating a subgroup-by-haemodynamic parameter interaction term in the logistic and LS means regression models, respectively.

Results were considered statistically significant at a two-sided *P*-value <0.05. Analyses were performed using SAS System V9.4 (SAS Institute Inc., Cary, NC).

Results

Study population and haemodynamic parameters

Of 20 718 CICU admissions between 2018 and 2022, 3603 presented to the CICU with CS. A total of 1925 (53.4%) underwent invasive haemodynamic assessment with a PAC during their CICU course. Of those cases, 1473 underwent invasive haemodynamic assessment within 24 h of CICU admission and comprised the primary cohort for this analysis. In this cohort, the median timing of assessment was 1 h after CICU admission (25th–75th percentile, 1 h before CICU admission to 6 h after CICU admission). This cohort included 325 (22.1%) admissions not receiving any pharmacologic or mechanical haemodynamic support (e.g. assessment performed before the initiation of support), 723 (49.1%) on pharmacologic support only, and 420 (28.5%) on temporary MCS at the time of invasive haemodynamic assessment. Most patients ($n = 1132$; 76.8%) in the primary analysis cohort had complete data for every haemodynamic parameter during that initial assessment.

The clinical characteristics and indices of shock severity among CS admissions in the primary analysis cohort were generally similar to those among CS cases not in the primary analysis cohort, with comparable SOFA scores (median, 7 vs. 7; $P = 0.106$) and VIS at 4 h following CICU admission [median, 4.0 (0.0–11.0) vs. 4.0 (0.0–11.0); $P = 0.432$] (*Table 1*). Serum lactate was slightly lower in the primary analysis cohort. In addition, the proportion of patients with AMI-CS (28.2% vs. 24.6%) and HF-CS (65.4% vs. 57.9%) as compared with secondary CS (6.4% vs. 17.5%) was higher in the primary analysis cohort ($P < 0.001$). A higher proportion of patients in the primary analysis cohort were ultimately managed with temporary MCS (54.6% vs. 24.6%; $P < 0.001$), and in-hospital mortality was lower (27.2% vs. 31.3%; $P = 0.007$).

Among all admissions with CS in the primary analysis cohort, including those with ongoing pharmacologic or MCS, the median CI was 1.9 (25th–75th percentile, 1.6–2.4) L/min/m^2, median MAP was 74 (66–86) mmHg, and median SVR was 1256 (899–1688) dynes*sec*/cm^5. Biventricular filling pressures were elevated with a median RAP of 15 (10–19) mmHg and median PCWP of 24 (18–30) mmHg. Comparing the subgroups of patients with AMI-CS and HF-CS, the median CPO was virtually identical, with a slightly higher median CI and a correspondingly lower median MAP among those with AMI-CS (*Table 2*).

Prognostic associations of haemodynamic parameters

Haemodynamic parameters significantly associated with in-hospital mortality included low MAP [OR per 1-SD, 0.70 (95% confidence interval, 0.60–0.80)], low SBP [OR per 1-SD, 0.78 (0.68–0.89)], low SVR [OR per 1-SD, 0.78 (0.67–0.90)], elevated RAP [OR per 1-SD, 1.27 (1.11–1.45)], elevated RAP/PCWP ratio [OR per 1-SD, 1.24 (1.09–1.41)], and low PAPI [OR per 1-SD, 0.70 (0.55–0.89); *Figure 1*]. When these six variables were considered collectively in a multi-parameter model, low MAP [aOR per 1-SD, 0.70 (0.60–0.80)] and elevated RAP [aOR per 1-SD, 1.28 (1.12–1.46)] remained significant.

After adjustment for VIS in the single parameter models (Model 2), low MAP [aOR per 1-SD, 0.74 (0.62–0.89)], low SVR (aOR per 1-SD, 0.82 (0.69–0.97)], elevated RAP (aOR per 1-SD, 1.28 (1.09–1.50)], and elevated RAP/PCWP (aOR per 1-SD, 1.21 (1.04–1.41) remained significantly associated with in-hospital mortality. By contrast, while the adjusted associations of SBP (aOR per 1-SD, 0.86 (0.72–1.01)] and PAPI (aOR per 1-SD, 0.76 (0.57–1.00)] with in-hospital mortality remained directionally consistent, they were no longer statistically significant (*Figure 2*). The same pattern was observed when patients receiving temporary MCS at the time of invasive haemodynamic assessment were excluded and analyses were iteratively adjusted for VIS (Model 3) and then VIS, age, sex, SCAI stage, and cardiac arrest before CICU admission (Model 4). Notably, CI, CPO, and PCWP

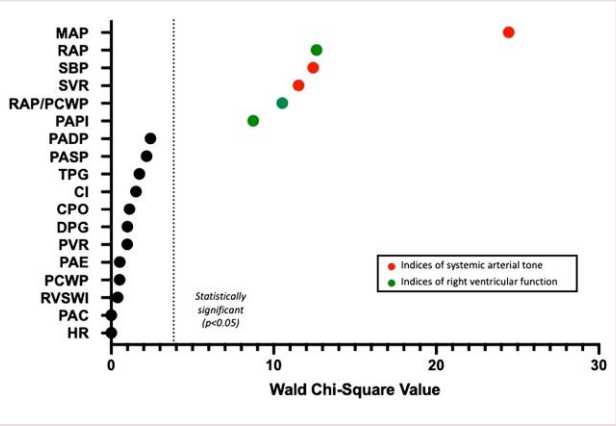


Figure 1 Strength of univariable associations between presenting haemodynamic parameters and in-hospital mortality. Haemodynamic parameters are ordered according to strength of association with in-hospital mortality based on Wald χ^2 values from univariable logistic regression models. Haemodynamic indices significantly associated with in-hospital mortality and reflecting either decreased systemic arterial tone or impaired right ventricular function are highlighted in colour. Analyses are restricted to patients with complete haemodynamic data ($n = 1132$). CI, cardiac index; CPO, cardiac power output; DPG, diastolic pressure gradient; HR, heart rate; MAP, mean arterial pressure; PAC, pulmonary artery compliance; PADP, pulmonary artery diastolic pressure; PAE, pulmonary artery elastance; PAPI, pulmonary artery pulsatility index; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; SBP, systolic blood pressure; SVR, systemic vascular resistance; TPG, transpulmonary gradient.

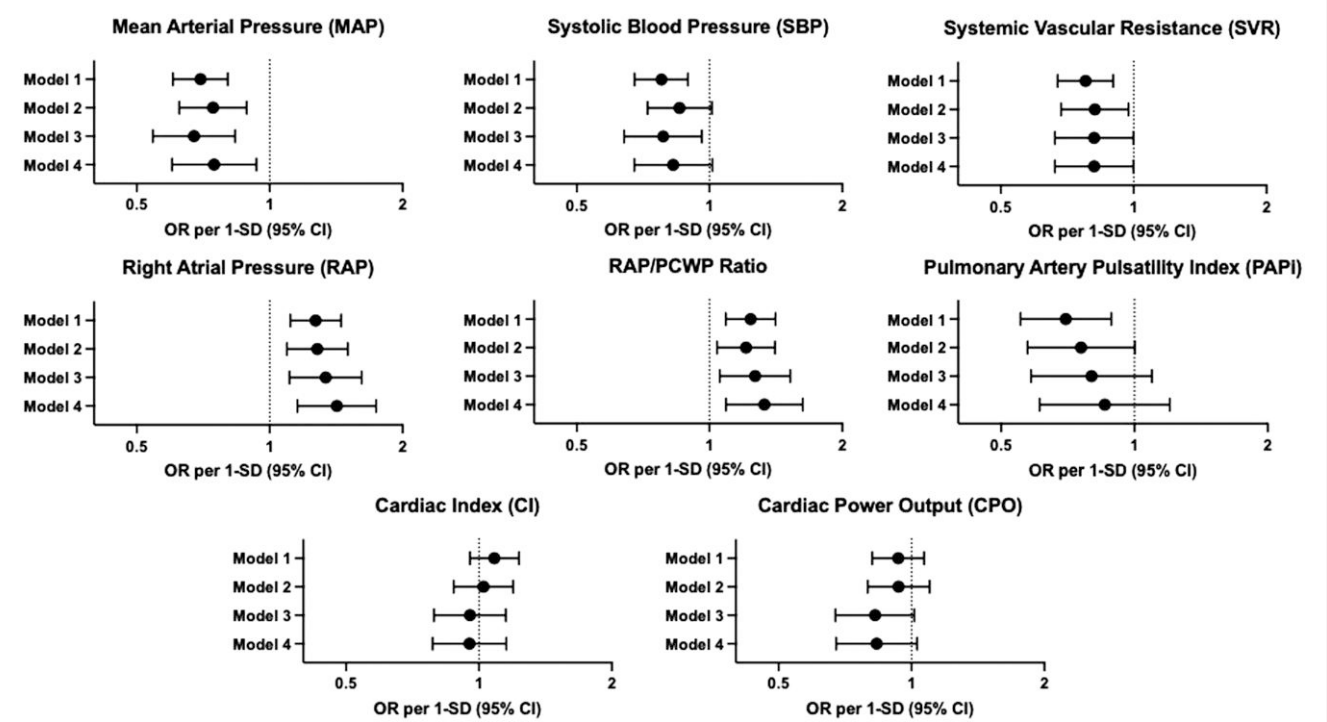


Figure 2 Adjusted associations between haemodynamic parameters and in-hospital mortality accounting for background haemodynamic support. Associations with in-hospital mortality are shown: unadjusted (Model 1); adjusted for vasoactive-inotropic score (VIS) (Model 2); excluding patients with MCS at the time of invasive haemodynamic assessment and adjusting for VIS (Model 3); and excluding patients with MCS at the time of invasive haemodynamic assessment and adjusting for VIS, age, sex, SCAI stage, and preceding cardiac arrest (Model 4). VIS was not collected in earlier annual cycles of the registry, so the VIS-adjusted analyses exclude approximately 22% of patients from the primary analysis cohort. Approximately 25% of patients in the primary analysis cohort were receiving mechanical circulatory support at the time of haemodynamic assessment. MCS, mechanical circulatory support; OR, odds ratio; SD, standard deviation; VIS, vasoactive-inotropic score.

were not associated with in-hospital mortality in unadjusted or adjusted analyses.

All six haemodynamic parameters significantly associated with in-hospital mortality were also associated with higher presenting serum lactate (*Figure 3*). Sensitivity analyses excluding admissions receiving temporary MCS at the time of invasive haemodynamic assessment and adjusting for VIS are presented in Supplementary material online, *Figure S1*.

Subgroup analyses

The unadjusted mortality associations of each haemodynamic parameter were generally consistent in patients with HF-CS and AMI-CS (*Figure 4*). Subgroup sensitivity analyses excluding patients on any form of temporary MCS at the time of invasive haemodynamic assessment and adjusting for VIS are presented in Supplementary material online, *Figure S2*.

Discussion

In this analysis of CS admissions from a large, contemporary, multicentre registry of CICUs in North America, we assessed the prognostic significance and relationship to end-organ dysfunction of specific haemodynamic parameters. We found that haemodynamic indices reflecting decreased systemic arterial tone and impaired RV function were most strongly associated with in-hospital mortality and elevation in presenting serum lactate. By contrast, haemodynamic indices reflecting low CO and elevated left ventricular filling pressures were not associated with either in-hospital mortality or presenting serum lactate. These findings were consistent even when controlling for intensity of background pharmacologic and mechanical haemodynamic support (*Graphical Abstract*). Taken together, these results highlight that: (i) the haemodynamic parameters most strongly associated with mortality are also the parameters most strongly associated with shock severity as manifested by systemic malperfusion; and (ii) that inappropriate systemic vasodilation and RV involvement in CS are particularly prognostically important in a contemporary CS population. More broadly, these data support the value of comprehensive haemodynamic profiling of CS patients and suggest potential therapeutic targets for future investigation.

Assessment of systemic arterial tone

In our analysis, the strongest haemodynamic indicator of both in-hospital mortality and systemic malperfusion was MAP, which is the mathematical product of CO and SVR. The primacy of MAP over its components suggests that both flow and systemic arterial tone matter; however, our results demonstrate that systemic arterial tone is the more prognostically important component of the two. Previous studies, dating back to the SHOCK trial and registry, have highlighted the distinct clinical phenotype of vasodilatory CS.[1,15,16] Challenging the prevailing paradigm of CS as a syndrome that invariably results in compensatory systemic vasoconstriction in response to impaired CO, the SHOCK trial investigators demonstrated that SVR varies widely in CS and is often near normal despite the use of vasopressors. More recent

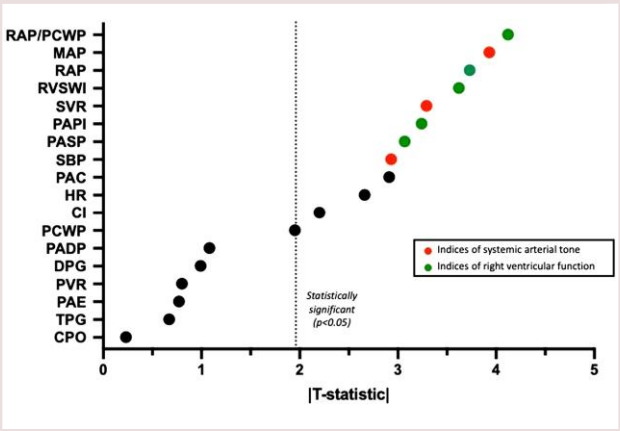


Figure 3 Strength of univariable associations between presenting haemodynamic parameters and presenting serum lactate. Haemodynamic parameters are ordered according to strength of association with serum lactate based on the absolute value of the *t* statistic in the univariable least squares means regression models. Haemodynamic indices significantly associated with presenting serum lactate and reflecting either decreased systemic arterial tone or impaired right ventricular function are highlighted in colour. Analyses are restricted to patients with complete haemodynamic data ($n = 1132$). CI, cardiac index; CPO, cardiac power output; DPG, diastolic pressure gradient; HR, heart rate; MAP, mean arterial pressure; PAC, pulmonary artery compliance; PADP, pulmonary artery diastolic pressure; PAE, pulmonary artery elastance; PAPI, pulmonary artery pulsatility index; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; SBP, systolic blood pressure; SVR, systemic vascular resistance; TPG, transpulmonary gradient.

work by our group and others has demonstrated that the vasodilatory CS profile is associated with poorer outcomes.[3] The prognostic importance of MAP and SVR in this analysis thus aligns with the current model of CS progression, in which an initial cardiac insult often leads to a systemic inflammatory response and vasodilation, which further exacerbates systemic malperfusion.[1] What is less clear is whether the variation in systemic arterial tone observed in CS patients represents fundamentally different phenotypes with distinct pathobiological features, a gradient of severity within the same phenotype, or simply distinct temporal assessments of the same phenotype. Although all haemodynamic assessments in our analysis were performed within 24 h of CICU admission, we are not able to account for differences in CS duration before CICU admission.

Notably, there was a consistent relationship between indicators of systemic arterial tone and mortality in models accounting for and not accounting for background pharmacologic and mechanical support. This highlights that while the intensity of required haemodynamic support is prognostically important,[13,17,18] so too is achieved MAP. These data should not be interpreted as indicating that a higher MAP target is necessarily better in CS since this association is inherently confounded. However, they reinforce the importance of systematically evaluating optimal MAP targets in CS in prospective clinical trials (e.g. NCT05168462).

Assessment of right ventricular dysfunction

Historical perspectives on the prognostic significance of RV dysfunction in CS have been framed by CS due to RV myocardial infarction (RVMI). In a seminal analysis from the SHOCK trial registry, patients with RV dominant AMI-CS were shown to have similarly poor outcomes as patients with LV dominant AMI-CS,[19] challenging the traditional view at the time that RV shock was associated with a favourable long-term prognosis.[20] More recently, using contemporary data from a mixed cohort of patients with AMI-CS and HF-CS, investigators from the Cardiogenic Shock Working Group demonstrated that an RV congestive profile (i.e. RAP ≥12 mmHg), whether in the context of a left ventricular congestive profile (i.e. PCWP ≥18 mmHg) or not, was associated with higher mortality than a left ventricular congestive profile alone.[21]

In the present analysis, which also includes a broad contemporary CS cohort, elevated RAP again emerged as an important indicator of adverse prognosis along with elevated RAP/PCWP ratio and low PAPI. These parameters were also highly associated with elevated presenting serum lactate, a marker of systemic malperfusion. Interestingly, RAP emerged from our multivariable analyses with a stronger association with mortality than PAPI or RAP/PCWP ratio. This finding is a reminder of the usefulness of an assessment of central venous pressure along with MAP when data from a PAC are not yet available or cannot be obtained. Whereas an elevated RAP may have multiple contributors, including stressed blood volume, increased RV afterload, and diminished RV contractility,[22] the collective associations of each of these RV parameters with shock severity and outcomes suggest that impaired RV contractile function is the principal clinical observation of prognostic significance. Further supporting this conclusion, neither pulmonary arterial tone [i.e. PVR and pulmonary artery elastance (PAE)] nor left ventricular filling pressures (i.e. PCWP) were significantly associated with either in-hospital mortality or lactate.

Several factors may account for the apparent greater prognostic relevance of RV dysfunction in contemporary CS populations as compared with older studies. First, HF-CS is now the most common cause of CS in contemporary practice,[3] whereas historical CS cohorts exclusively focused on AMI-CS. Along these lines, a prior study suggested that RV dysfunction may be more prognostically relevant in HF-CS than AMI-CS.[23] Nevertheless, in our study, there was no meaningful heterogeneity in the prognostic significance of these parameters between CS subtypes in our dataset. Second, our cohort reflects contemporary clinical practice, in which left-sided MCS devices are commonly used in the management of CS. Indeed, one of the potential explanations for the fact that CI and CPO were not associated with in-hospital mortality in the overall CS cohort, contrasting with prior work from the SHOCK registry which demonstrated the prognostic importance of these variables,[24] is that clinicians are able to recognize and promptly initiate mechanical support in the setting of severely diminished flow. By contrast, clinicians may be less attuned to RV dysfunction and thus less likely to initiate RV-directed therapies (both pharmacologic and mechanical) in the management of CS. Although it is not possible to fully disentangle the complex relationships linking RV dysfunction to poor outcomes in this type of observational analysis, our observations spotlight the RV as a potentially important therapeutic target for improving outcomes in CS. Moreover, our finding with respect to CPO is consistent with findings from another contemporary population.[8]

Strengths and limitations

A key strength of this analysis is the use of a contemporary cohort of well-phenotyped CS patients with comprehensive clinical characteristics, complete baseline haemodynamic profiling, and detailed characterization of background pharmacologic and mechanical therapies. Nevertheless, several limitations should be acknowledged. First, not all sites in the registry provided invasive haemodynamic data and not all CS patients underwent invasive haemodynamic assessment. In a recent analysis from CCCTN, the range of PAC utilization across centres was 8.3–73.2% of all shock admissions, and the primary drivers of PAC use included MCS, site-related variation, and a primary diagnosis of HF.[5]

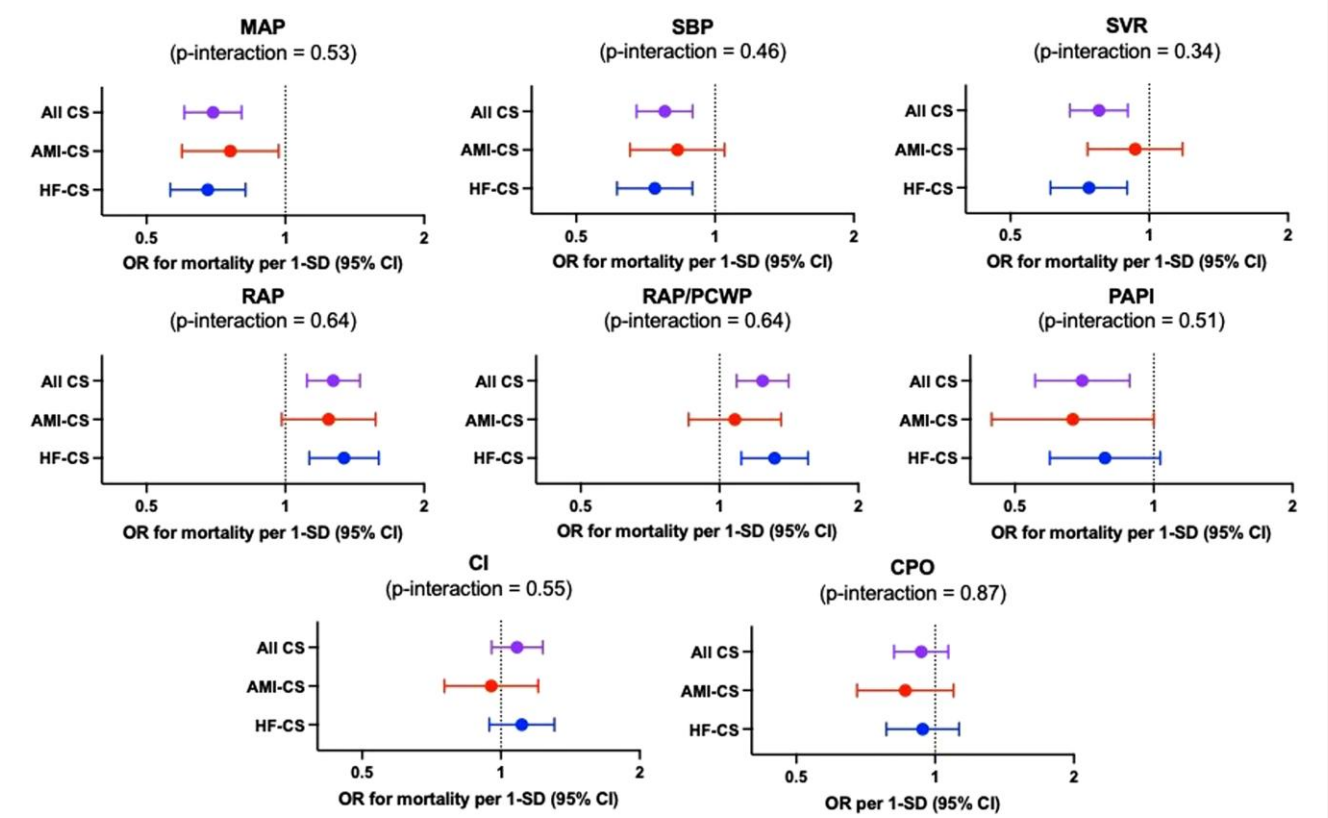


Figure 4 Association of haemodynamic parameters with in-hospital mortality in patients with acute myocardial infarction-related cardiogenic shock vs. decompensated heart failure-related cardiogenic shock. Analyses are restricted to patients with complete haemodynamic data ($n = 1132$). CI, cardiac index; CPO, cardiac power output; MAP, mean arterial pressure; PAPI, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; SBP, systolic blood pressure; SVR, systemic vascular resistance.

Thus, there is potential for selection bias in our study population. Mitigating this risk, indices of critical illness and shock severity were similar in patients in the primary analysis cohort and those not in the primary analysis cohort, suggesting that the primary analysis cohort was reasonably representative of the broader CS population in our registry of North American CICUs. Moreover, such selection pressure might impact the generalizability but would not reduce the validity of our findings. Second, although all numeric haemodynamic data were manually reviewed by the coordinating centre, the assessment of haemodynamic tracings was not centrally adjudicated. Third, approximately 14% of patients did not have an available presenting serum lactate and approximately 22% of patients did not have an available VIS (since VIS was added later to the registry), thus diminishing the power of those analyses and increasing the risk of type II error. Fourth, although we did not find any statistical heterogeneity between the subgroups of patients with AMI-CS and HF-CS, it is possible that these analyses were underpowered. Future analyses should continue to explore potential differences in the prognostic relevance of haemodynamic parameters in these CS subtypes, particularly since there may be important pathophysiological differences. Fifth, serial haemodynamic assessments were not available for this analysis; therefore, we were not able to assess the prognostic significance of haemodynamic trajectories. Sixth, although in-hospital mortality is a critically important outcome, it is agnostic to individual goals of care and patient candidacy for advanced therapies, which are relevant when interpreting CS outcomes. Finally, these data are observational in nature and therefore subject to confounding. As a result, we are not able to determine whether haemodynamic parameters reflecting decreased systemic arterial tone and RV dysfunction are causally related to in-hospital mortality and systemic malperfusion or simply adverse prognostic indicators.

Conclusions

In this analysis of CS admissions from a large, contemporary, multicentre registry, presenting haemodynamic variables reflecting decreased systemic arterial tone and indicators of impaired RV function were the parameters most strongly associated with presenting lactate and adverse outcomes, even when controlling for intensity of background pharmacologic and mechanical haemodynamic support. These findings demonstrate the value of invasive haemodynamic measurements in risk assessment and highlight several potentially important haemodynamic targets in CS that warrant further investigation in prospective clinical trials.

Supplementary material

Supplementary material is available at *European Heart Journal: Acute Cardiovascular Care* online.

Conflict of interest: D.D.B., E.A.B., V.M.B-Z., J.G., S.M.P., and D.A.M. are members of the TIMI Study Group, which has received institutional research grant support through Brigham and Women's Hospital from Abbott, Abiomed, Amgen, Anthos Therapeutics, ARCA Biopharma, Inc., AstraZeneca, Bayer HealthCare Pharmaceuticals, Inc., Daiichi-Sankyo, Eisai, Intarcia, Ionis Pharmaceuticals, Inc., Janssen Research and Development, LLC, MedImmune, Merck, Novartis, Pfizer, Quark Pharmaceuticals, Regeneron Pharmaceuticals, Inc., Roche, Siemens Healthcare Diagnostics, Inc., Softcell Medical Limited, The Medicines Company, Zora Biosciences. M.A.S. receives research support from the National Institutes of Health Clinical Center intramural research funds. A.D.T. is supported by NIH-NHLBI (K08HL163328).

Data availability

We encourage parties interested in collaboration and data sharing to contact the corresponding author directly for further discussions.

References

1. van Diepen S, Katz JN, Albert NM, Henry TD, Jacobs AK, Kapur NK, *et al.* Contemporary management of cardiogenic shock: A scientific statement from the American heart association. *Circulation* 2017;**136**:e232–e268.
2. Berg DD, Bohula EA, Morrow DA. Epidemiology and causes of cardiogenic shock. *Curr Opin Crit Care* 2021;**27**:401–408.
3. Berg DD, Bohula EA, van Diepen S, Katz JN, Alviar CL, Baird-Zars VM, *et al.* Epidemiology of shock in contemporary cardiac intensive care units. *Circ Cardiovasc Qual Outcomes* 2019;**12**:e005618.
4. Harjola VP, Lassus J, Sionis A, Køber L, Tarvasmäki T, Spinar J, *et al.* Clinical picture and risk prediction of short-term mortality in cardiogenic shock. *Eur J Heart Fail* 2015;**17**: 501–509.
5. Kadosh BS, Berg DD, Bohula EA, Park J-G, Baird-Zars VM, Alviar C, *et al.* Pulmonary artery catheter use and mortality in the cardiac intensive care unit. *JACC Heart Fail* 2023; **11**:903–914.
6. Chatterjee K. The swan-ganz catheters: past, present, and future. A viewpoint. *Circulation* 2009;**119**:147–152.
7. Tehrani BN, Truesdell AG, Psotka MA, Rosner C, Singh R, Sinha SS, *et al.* A standardized and comprehensive approach to the management of cardiogenic shock. *JACC Heart Fail* 2020;**8**:879–891.
8. Garan AR, Kanwar M, Thayer KL, Whitehead E, Zweck E, Hernandez-Montfort J, *et al.* Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower in-hospital mortality. *JACC Heart Fail* 2020;**8**:903–913.
9. Hernandez GA, Lemor A, Blumer V, Rueda CA, Zalawadiya S, Stevenson LW, *et al.* Trends in utilization and outcomes of pulmonary artery catheterization in heart failure with and without cardiogenic shock. *J Card Fail* 2019;**25**:364–371.
10. Morrow DA, Fang JC, Fintel DJ, Granger CB, Katz JN, Kushner FG, *et al.* Evolution of critical care cardiology: transformation of the cardiovascular intensive care unit and the emerging need for new medical staffing and training models: a scientific statement from the American heart association. *Circulation* 2012;**126**:1408–1428.
11. Bohula EA, Katz JN, van Diepen S, Alviar CL, Baird-Zars VM, Park J-G, *et al.* Demographics, care patterns, and outcomes of patients admitted to cardiac intensive care units: the critical care cardiology trials network prospective North American multicenter registry of cardiac critical illness. *JAMA Cardiol* 2019;**4**:928–935.
12. Gaies MG, Gurney JG, Yen AH, Napoli ML, Gajarski RJ, Ohye RG, *et al.* Vasoactive-inotropic score as a predictor of morbidity and mortality in infants after cardiopulmonary bypass. *Pediatr Crit Care Med* 2010;**11**:234–238.
13. Patel SM, Berg DD, Bohula EA, Baird-Zars VM, Barnett CF, Barsness GW, *et al.* Clinician and algorithmic application of the 2019 and 2022 society of cardiovascular angiography and intervention shock stages in the critical care cardiology trials network registry. *Circ Heart Fail* 2022;**16**:e009714.
14. Berg DD, Barnett CF, Kenigsberg BB, Papolos A, Alviar CL, Baird-Zars VM, *et al.* Clinical practice patterns in temporary mechanical circulatory support for shock in the critical care cardiology trials network (CCCTN) registry. *Circ Heart Fail* 2019;**12**:e006635.
15. Menon V, Slater JN, White HD, Sleeper LA, Cocke T, Hochman JS. Acute myocardial infarction complicated by systemic hypoperfusion without hypotension: report of the SHOCK trial registry. *Am J Med* 2000;**108**:374–380.
16. Hochman JS. Cardiogenic shock complicating acute myocardial infarction: expanding the paradigm. *Circulation* 2003;**107**:2998–3002.
17. Naidu SS, Baran DA, Jentzer JC, Hollenberg SM, van Diepen S, Basir MB, *et al.* SCAI SHOCK stage classification expert consensus update: A review and incorporation of validation studies: this statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021. *J Am Coll Cardiol* 2022;**79**:933–946.
18. Jentzer JC. Understanding cardiogenic shock severity and mortality risk assessment. *Circ Heart Fail* 2020;**13**:e007568.
19. Jacobs AK, Leopold JA, Bates E, Mendes LA, Sleeper LA, White H, *et al.* Cardiogenic shock caused by right ventricular infarction: a report from the SHOCK registry. *J Am Coll Cardiol* 2003;**41**:1273–1279.
20. Zehender M, Kasper W, Kauder E, Schonthaler M, Geibel A, Olschewski M, *et al.* Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. *N Engl J Med* 1993;**328**:981–988.
21. Thayer KL, Zweck E, Ayouty M, Garan AR, Hernandez-Montfort J, Mahr C, *et al.* Invasive hemodynamic assessment and classification of in-hospital mortality risk among patients with cardiogenic shock. *Circ Heart Fail* 2020;**13**:e007099.
22. Haddad F, Doyle R, Murphy DJ, Hunt SA. Right ventricular function in cardiovascular disease, part II: pathophysiology, clinical importance, and management of right ventricular failure. *Circulation* 2008;**117**:1717–1731.
23. Jain P, Thayer KL, Abraham J, Everett KD, Pahuja M, Whitehead EH, *et al.* Right ventricular dysfunction is common and identifies patients at risk of dying in cardiogenic shock. *J Card Fail* 2021;**27**:1061–1072.
24. Fincke R, Hochman JS, Lowe AM, Menon V, Slater JN, Webb JG, *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**:340–348.