

Systematic Assessment of Shock Severity in Postoperative Cardiac Surgery Patients



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ABSTRACT

BACKGROUND The Society for Cardiovascular Angiography and Interventions (SCAI) shock classification has been shown to provide robust mortality risk stratification in a variety of cardiovascular patients.

OBJECTIVES This study sought to evaluate the SCAI shock classification in postoperative cardiac surgery intensive care unit (CSICU) patients.

METHODS This study retrospectively analyzed 26,792 postoperative CSICU admissions at a heart center between 2012 and 2022. Patients were classified into SCAI shock stages A to E using electronic health record data. Moreover, the impact of late deterioration (LD) as an additional risk modifier was investigated.

RESULTS The proportions of patients in SCAI shock stages A to E were 24.4%, 18.8%, 8.4%, 35.5%, and 12.9%, and crude hospital mortality rates were 0.4%, 0.6%, 3.3%, 4.9%, and 30.2%, respectively. Similarly, the prevalence of postoperative complications and organ dysfunction increased across SCAI shock stages. After multivariable adjustment, each higher SCAI shock stage was associated with increased hospital mortality (adjusted OR: 1.26-16.59) compared with SCAI shock stage A, as was LD (adjusted OR: 8.2). The SCAI shock classification demonstrated a strong diagnostic performance for hospital mortality (area under the receiver operating characteristic: 0.84), which noticeably increased when LD was incorporated into the model (area under the receiver operating characteristic: 0.90).

CONCLUSIONS The SCAI shock classification effectively risk-stratifies postoperative CSICU patients for mortality, postoperative complications, and organ dysfunction. Its application could, therefore, be extended to the field of cardiac surgery as a triage tool in postoperative care and as a selection criterion in research.

(J Am Coll Cardiol 2023;82:1691-1706) © 2023 by the American College of Cardiology Foundation.

Despite considerable advancements in patient care over recent years, cardiogenic shock (CS) is still associated with mortality rates up to 50%.¹ CS encompasses a broad variety of clinical manifestations, degrees of severity, and therapeutic options. Therefore, its treatment requires a well-coordinated, multidisciplinary approach. However, concise communication of patients' disease



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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 [Author Center](#).

Manuscript received April 27, 2023; revised manuscript received July 5, 2023, accepted August 15, 2023.

ISSN 0735-1097/\$36.00

<https://doi.org/10.1016/j.jacc.2023.08.031>

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ABBREVIATIONS AND ACRONYMS

AUROC = area under the receiver operating characteristic

CA = cardiac arrest

CICU = cardiac intensive care unit

CS = cardiogenic shock

CSICU = cardiac surgery intensive care unit

DMV = duration of mechanical ventilation

ESRD = end-stage renal disease

ICU = intensive care unit

LD = late deterioration

LOS = length of stay

SCAI = Society for Cardiovascular Angiography and Interventions

SOFA = Sequential Organ Failure Assessment

tMCS = temporary mechanical circulatory support

VAD = ventricular assist device

progression, therapeutic interventions, and risk status is a major challenge for treating physicians. The marked heterogeneity of disease severity has also presumably contributed to failures in attempts of prospective CS trials to demonstrate a survival benefit of advanced treatment modalities, such as temporary mechanical circulatory support (tMCS).¹⁻³

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In 2019, the Society for Cardiovascular Angiography and Interventions (SCAI) released an expert consensus statement, establishing a CS classification scheme that offered a common framework for CS staging and risk prediction. The classification was endorsed by numerous medical societies in the cardiovascular field.¹ In the following years, numerous retrospective and few prospective studies successfully evaluated the SCAI shock classification in different cardiovascular patient cohorts, including unselected cardiac intensive care unit patients,⁴ patients with documented CS,⁵ patients with mixed cardiogenic-septic shock,⁶ patients with acute heart failure,⁷ and patients with out-of-

hospital cardiac arrest (CA).⁸ The results from the validation studies led to some modifications of the classification, which were announced in the SCAI Expert Consensus Update in 2022.⁹ One major point of criticism was the varying interpretation of SCAI shock stage definitions potentially limiting the generalizability of the respective study results.

Despite its wide application in cardiology, the SCAI shock classification has to this date not been evaluated in cardiac surgery patients, a cohort at significant risk of CS.¹⁰

In this study, therefore, we set out to evaluate the diagnostic ability of the SCAI shock classification regarding all-cause hospital and intensive care unit (ICU) mortality in postoperative cardiac surgery intensive care unit (CSICU) patients. Additionally, we investigated the association between SCAI shock stages and common postoperative complications and organ dysfunction.

METHODS

ETHICS AND REPORTING GUIDELINE. This study was approved by the research ethics committee of Charité-Universitätsmedizin Berlin (EA4/068/23). The approval included the collection of data on implied consent owing to the retrospective and observational nature of the study.

STUDY POPULATION. The study included all postoperative ICU admissions of adult cardiac surgery patients treated at the Department of Cardiovascular and Thoracic Surgery of the Deutsches Herzzentrum Berlin ("German Heart Center Berlin"; since January 1, 2023: "Deutsches Herzzentrum der Charité") between November 1, 2012, and June 30, 2022. Postanesthesia care unit admissions were also included under the collective term of "ICU admission" for this study. Patients were observed from initial ICU admission until ICU discharge or death. The study flow chart is presented in [Figure 1](#).

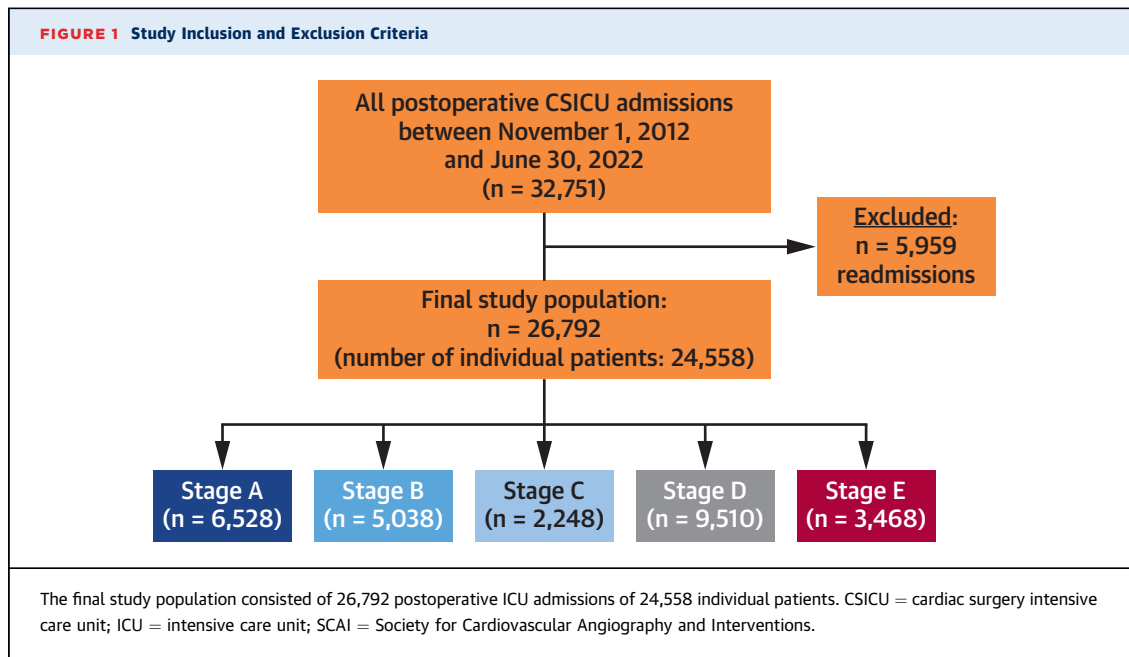
Our institution is a quaternary care center for cardiovascular disease. The Department of Cardiovascular and Thoracic Surgery offers the full spectrum of cardiothoracic surgery procedures ranging from minimally invasive surgery and transcatheter procedures to a transplantation program and a special expertise in tMCS and ventricular assist device (VAD) therapy. Our institution's CSICU is a closed, 2-ward, 44-bed unit serving critically ill cardiac surgery patients.

DATA COLLECTION. Patient demographics, surgical data, vital signs, laboratory values, outcome data, International Classification of Diseases-10th Revision admission diagnoses, as well as interventions performed during the initial CSICU admission were extracted from the electronic patient data management system. The patient data management system's implementation was completed, as far as was necessary for this study, by November 1, 2012, thereby marking the beginning of the study period. Clinical scores (eg, EuroSCORE [European System for Cardiac Operative Risk Evaluation] II, APACHE [Acute Physiology And Chronic Health Evaluation]-IV) as well as data from invasive hemodynamic monitoring were not sufficiently available for the entire study period and thereby excluded from the analysis. Sequential Organ Failure Assessment (SOFA) scores were calculated retrospectively as described in the [Supplemental Material](#).

Procedural data were analyzed for the first surgical procedure preceding the initial CSICU admission. Surgery types encompassed the main surgery type of the index procedure as specified by the operating surgeon.

Data preprocessing, formulas for the vasoactive-inotropic score and norepinephrine-equivalent vasopressor doses, as well as detailed definitions of secondary outcomes and ICU interventions are reported in the [Supplemental Material](#).

DEFINITION OF SHOCK STAGES. To meet the requirements of consistent and uniform SCAI shock



criteria, we largely adhered to the SCAI shock stage definitions established by Jentzer et al⁴ in a retrospective validation study on a large cohort of unselected cardiology ICU patients with thoughtfully selected clinical variables and clearly defined, reproducible cutoff values for SCAI shock stages.

Shock stages were defined using data from the first 24 hours of the observational period according to the criteria established by Jentzer et al,⁴ with 2 notable exceptions: 1) in accordance with the recent modification of lactate thresholds by the SCAI SHOCK consensus workgroup,⁹ and due to our in-house experience with postoperative CSICU patients, lactate levels must increase to >2 mEq/L to justify reclassification; and 2) we extended the definition of stage E's tMCS-criterion by also including venoarterial extracorporeal membrane oxygenation and Impella (Abiomed) because this was more in line with the original SCAI SHOCK classification scheme¹ and better suited our surgical patient cohort. **Table 1** summarizes the criteria for SCAI shock stages used in our analysis.

Late deterioration (LD) was defined in accordance with Jentzer et al⁴ as increasing vasopressor requirements in the form of increasing norepinephrine-equivalent vasopressor doses after the initial 24 hours.

STATISTICAL ANALYSIS. The primary outcomes were all-cause hospital mortality and all-cause CSICU mortality. Secondary outcomes included postoperative complications, duration of mechanical

ventilation (DMV), and length of initial intensive care unit stay (LOS). Furthermore, the association between SCAI shock stages and laboratory parameters indicative of hemodynamic deterioration, systemic inflammation, and end-organ dysfunction was analyzed. Hospital mortality was analyzed separately before and as of January 1, 2015, due to a change of medical leadership at our institution, new guidelines for tMCS,¹¹ and enhancement of comparability with previous validation studies.

Continuous variables are reported as mean ± SD. Categorical variables are reported as number and percentage. Pearson chi-square test was used to compare groups. Trends across SCAI shock stages were determined using linear regression for continuous variables and the Cochran-Armitage test for trend for binary variables.

Logistic regression was used to determine the association between SCAI shock stages and hospital mortality with and without adjustment for LD and additional clinically relevant confounders.

Discrimination was assessed using the area under the receiver operating characteristic (AUROC) and DeLong test was used to compare AUROCs. Calibration was assessed with calibration curves and Brier scores. AUROCs and Brier scores were reported with 95% CIs computed with 2,000 stratified bootstrap replicates. Values of $P < 0.05$ were considered statistically significant. P values for trends are reported with Holm-Sidak correction. Statistical analyses were carried out with Python 3.9 and R 4.0.3.

TABLE 1 Study Definitions of SCAI Shock Stage Criteria

Term	Definition
Stage B (hypotension/tachycardia)	Presence of any of the following criteria: Admission SBP <90 mm Hg Minimum SBP <90 mm Hg during first 1 h Admission MAP <60 mm Hg Minimum MAP <60 mm Hg during first 1 h Admission HR >100 beats/min Maximum HR >100 beats/min during first 1 h Admission HR > admission SBP Mean HR > mean SBP during first 1 h
Stage C (hypoperfusion)	Presence of any of the following criteria: Admission lactate >2 mEq/L Urine output <720 mL during first 24 h Creatinine increased by ≥0.3 mg/dL during first 24 h
Stage D (deterioration)	Presence of any of the following criteria: (Maximum lactate > admission lactate) and (maximum lactate >2 mEq/L) during first 24 h ^a Number of vasoactives during first 24 h > number of vasoactives during first 1 h Maximum VIS during first 24 h > VIS during first 1 h Maximum NEE during first 24 h > NEE during first 1 h
Stage E (refractory shock)	Presence of any of the following criteria: Mean SBP during first 1 h <80 mm Hg and on vasoactives Mean MAP during first 1 h <50 mm Hg and on vasoactives Number of vasoactives during first 1 h >2 Number of vasoactives during first 1 h >1 and tMCS (including IABP, Impella [Abiomed], and VA-ECMO) within first 24 h ^a Admission lactate ≥10 mEq/L

SCAI shock stages were defined according to the criteria established by Jentzer et al 2019.⁴ ^aDeviations from the original criteria. Reproduced with permission from Elsevier.
HR = heart rate; IABP = intra-aortic balloon pump; MAP = mean arterial pressure; NEE = norepinephrine-equivalent vasopressor dose; SBP = systolic blood pressure; tMCS = temporary mechanical circulatory support; VA-ECMO = venoarterial extracorporeal membrane oxygenation; VIS = vasoactive-inotropic-score.

RESULTS

STUDY POPULATION. We included 26,792 postoperative CSICU admissions (24,558 individual patients) of adult, postoperative patients in our study. The study population had a mean age of 67.2 ± 13.1 years and 15.1% of participants were aged older than 80 years. The study included 8,564 (32.0%) female patients. Our study population received a broad variety of cardiac surgery types with coronary bypass surgery (24.5%), valve surgery (22.7%), aortic surgery (15.1%), and transcatheter aortic valve replacement (13.3%) being the most frequent. A third of index procedures were nonelective (Table 2). A total of 21,929 (81.9%) patients received vasoactives during the CSICU stay, including vasopressors in 21,855 (81.6%) and inotropes in 10,279 (38.4%) cases. Among patients receiving vasoactives, 9,207 (42.0%) received >1 vasoactive drug concomitantly. In total, 12,605 (49.3%) patients had peak arterial lactate levels >2 mEq/L, 786 (2.9%) patients received intra-aortic balloon pump therapy, 313 (1.2%) patients were treated with Impella, 874 (3.3%) patients had

venoarterial extracorporeal membrane oxygenation support, and 1,725 (6.4%) patients had VADs. Overall, 14,792 (55.2%), 8,743 (32.6%), 12,375 (46.2%), and 3,468 (12.9%) patients fulfilled at least 1 of the criteria for stages B, C, D, and E, respectively. Relative frequencies of SCAI shock criteria and associated hospital mortality rates are portrayed in Supplemental Figures 1 and 2.

The proportion of patients with SCAI shock stages A to E were 24.4%, 18.8%, 8.4%, 35.5%, and 12.9%, respectively, in the overall cohort (Central Illustration). However, this distribution differed substantially in particular surgery types (Figure 2).

Patients in higher SCAI shock stages on average had more extensive surgical procedures, less frequently elective procedures, and more frequently admission diagnoses of CA and end-stage renal disease (ESRD). In addition, patients in higher SCAI shock stages had increasing vital sign and laboratory abnormalities at admission (Table 2).

As SCAI shock stages increased, patients more frequently required supportive ICU interventions (eg, renal replacement therapy), advanced treatment modalities (eg, pulmonary vasodilators), and more anti-infectives and higher dosages of vasoactives (Table 3).

CSICU MORTALITY, HOSPITAL MORTALITY, AND POSTOPERATIVE COMPLICATIONS AS A FUNCTION OF SCAI SHOCK STAGE.

A stepwise increase in crude CSICU mortality and hospital mortality with each higher SCAI shock was observed. Hospital mortality increased from 0.4% in SCAI shock stage A to 30.2% in SCAI shock stage E ($P < 0.001$). Both CSICU and hospital mortality rates were below average (ie, 5.6% and 6.2%) in SCAI shock stages A to D, respectively (Central Illustration). A similar stepwise increase in hospital mortality was seen in relevant subgroups (Figure 3).

Compared with SCAI shock stage A, the unadjusted OR values for hospital mortality in SCAI shock stages B to E were 1.44, 8.01, 12.02, and 100.67, respectively (Table 4).

The SCAI shock classification had an AUROC of 0.842 (95% CI: 0.833-0.850) for hospital mortality overall and AUROCs were consistently high for every surgery type, except transplant surgery, VAD implantation, and tMCS surgery (Supplemental Table 3).

In general, the prevalence of postoperative complications steadily increased with increasing SCAI shock stages (Figure 4), as did Day 1 SOFA scores, LOS, DMV, and rate of unplanned ICU readmission. Likewise, significant trends in laboratory values

TABLE 2 Baseline Characteristics, Surgical Data, and Admission Data

	With Data, %	Overall	Stage A (n = 6,528)	Stage B (n = 5,038)	Stage C (n = 2,248)	Stage D (n = 9,510)	Stage E (n = 3,468)	P Value
Age, y	100	67.2 ± 13.1	69.9 ± 12.9	66.8 ± 13.1	66.8 ± 14.3	67.0 ± 12.3	63.1 ± 13.3	<0.001
Female	100	8,564 (32.0)	2,440 (37.4)	1,631 (32.4)	684 (30.4)	2,941 (30.9)	868 (25.0)	<0.001
Body mass index, kg/m ²	100	27.4 ± 5.1	27.3 ± 5.0	27.1 ± 4.8	27.6 ± 5.4	27.4 ± 5.2	27.5 ± 5.2	<0.01
Cardiac arrest	100	102 (0.4)	7 (0.1)	6 (0.1)	8 (0.4)	25 (0.3)	56 (1.6)	<0.001
End-stage renal disease	100	397 (1.5)	17 (0.3)	9 (0.2)	151 (6.7)	151 (1.6)	69 (2.0)	<0.001
Surgery duration, min	100	200.3 ± 129.7	138.8 ± 87.6	168.0 ± 88.8	156.9 ± 136.1	237.8 ± 118.0	288.7 ± 179.7	<0.001
Cardiopulmonary bypass	100	15,766 (58.8)	2,470 (37.8)	2,630 (52.2)	808 (35.9)	7,221 (75.9)	2,637 (76.0)	<0.001
Cardiopulmonary bypass time, min	100	75.4 ± 87.6	37.4 ± 54.4	52.5 ± 58.9	50.6 ± 85.0	99.6 ± 88.5	129.7 ± 118.6	<0.001
Aortic cross-clamp time, min	100	41.7 ± 48.4	23.8 ± 36.3	32.9 ± 37.9	25.9 ± 43.2	57.2 ± 47.3	55.7 ± 66.7	<0.001
Elective procedure	97.6	17,448 (66.7)	5,024 (78.7)	3,787 (77.2)	1,347 (61.6)	6,209 (66.9)	1,081 (31.8)	<0.001
Surgery type	99.9							
CBS	-	6,567 (24.5)	1,476 (22.6)	1,736 (34.5)	227 (10.1)	2,663 (28.0)	465 (13.4)	-
Valve surgery	-	6,065 (22.7)	1,260 (19.3)	1,174 (23.3)	322 (14.3)	2,634 (27.7)	675 (19.5)	-
Aortic surgery	-	4,045 (15.1)	911 (14.0)	608 (12.1)	424 (18.9)	1,725 (18.1)	377 (10.9)	-
TAVR	-	3,549 (13.3)	1,903 (29.2)	690 (13.7)	471 (21.0)	418 (4.4)	67 (1.9)	-
Combined valve surgery and CBS	-	2,018 (7.5)	180 (2.8)	235 (4.7)	85 (3.8)	1,102 (11.6)	416 (12.0)	-
VAD implantation	-	927 (3.5)	3 (0.0)	20 (0.4)	33 (1.5)	125 (1.3)	746 (21.5)	-
Pericardial surgery	-	789 (2.9)	175 (2.7)	164 (3.3)	69 (3.1)	251 (2.6)	130 (3.8)	-
Cardiac arrhythmia surgery	-	778 (2.9)	241 (11.9)	126 (2.5)	305 (13.6)	79 (0.8)	27 (0.8)	-
Vascular surgery	-	666 (2.5)	183 (2.8)	115 (2.3)	101 (4.5)	150 (1.6)	117 (3.4)	-
Transplant surgery	-	376 (1.4)	8 (0.1)	5 (0.1)	30 (1.3)	154 (1.6)	179 (5.2)	-
Thoracic surgery	-	359 (1.3)	99 (1.5)	59 (1.2)	68 (3.0)	84 (0.9)	49 (1.4)	-
Other	-	352 (1.3)	57 (0.9)	81 (1.6)	97 (4.3)	71 (0.7)	46 (1.3)	-
tMCS surgery	-	194 (0.7)	1 (0.0)	1 (0.0)	10 (0.4)	22 (0.2)	160 (4.6)	-
Congenital heart surgery	-	87 (0.3)	24 (0.4)	22 (0.4)	3 (0.1)	28 (0.3)	10 (0.3)	-
Admission vital signs								
SBP, mm Hg	98.3	113.1 ± 27.0	129.4 ± 24.9	106.4 ± 25.9	120.2 ± 27.1	110.5 ± 23.7	95.6 ± 23.7	<0.001
MAP, mm Hg	98.3	76.0 ± 15.4	83.8 ± 14.2	72.3 ± 16.2	79.4 ± 15.8	73.9 ± 14.2	70.4 ± 13.2	<0.001
HR, beats/min	99.4	82.5 ± 20.6	73.4 ± 13.4	80.4 ± 19.0	78.6 ± 18.5	84.2 ± 16.7	100.7 ± 30.3	<0.001
Core temperature, °C	79.4	36.4 ± 0.8	36.3 ± 0.7	36.4 ± 0.7	36.3 ± 0.9	36.4 ± 0.8	36.4 ± 1.0	<0.001
Shock index	98.2	0.78 ± 0.3	0.59 ± 0.16	0.79 ± 0.25	0.69 ± 0.25	0.8 ± 0.24	1.12 ± 0.44	<0.001
Urinary output during first 24 h, L	100	3.58 ± 1.97	3.79 ± 1.96	3.89 ± 1.88	2.17 ± 2.18	3.79 ± 1.7	3.04 ± 2.16	<0.001
Admission laboratory data								
Creatine kinase, U/L	99.9	208.0 ± 1,288.9	131.4 ± 285.0	141.8 ± 255.8	162.3 ± 776.9	190.3 ± 1,248.5	526.5 ± 2,792.6	<0.001
CK-MB, U/L	99.3	41.9 ± 89.4	29.0 ± 37.3	31.6 ± 36.8	29.5 ± 61.6	43.2 ± 67.0	85.6 ± 199.4	<0.001
LDH, U/L	98.9	327.9 ± 626.7	235.2 ± 114.4	241.5 ± 168.7	292.5 ± 406.7	315.9 ± 417.9	681.5 ± 1,488.5	<0.001
Creatinine, mg/dL	99.9	1.2 ± 0.8	1.1 ± 0.5	1.0 ± 0.5	1.6 ± 1.5	1.2 ± 0.8	1.6 ± 1.1	<0.001
Serum urea, mg/dL	99.8	46.3 ± 28.0	42.8 ± 23.0	40.3 ± 22.1	51.8 ± 31.2	44.5 ± 25.6	63.1 ± 39.4	<0.001
Hemoglobin, g/dL	99.9	12.6 ± 2.2	12.7 ± 2.1	12.9 ± 2.1	12.3 ± 2.3	12.8 ± 2.2	12.0 ± 2.4	<0.001
Leukocyte count, 1,000/μL	99.9	9.0 ± 4.8	8.2 ± 4.0	8.4 ± 4.5	9.2 ± 4.3	9.0 ± 4.3	11.2 ± 7.1	<0.001
C-reactive protein, mg/dL	75.8	2.7 ± 5.1	2.0 ± 4.3	2.1 ± 4.5	3.1 ± 5.4	2.4 ± 4.7	4.8 ± 6.7	<0.001
Lactate, mEq/L	91.4	2.2 ± 2.9	0.9 ± 0.3	0.9 ± 0.3	2.1 ± 1.8	2.0 ± 1.7	6.5 ± 5.1	<0.001
Arterial pH	97.2	7.38 ± 0.08	7.39 ± 0.06	7.39 ± 0.07	7.38 ± 0.08	7.38 ± 0.08	7.34 ± 0.09	<0.001
Bicarbonate, mEq/L	97.0	22.6 ± 3.1	23.5 ± 2.8	23.2 ± 2.8	23.0 ± 3.2	22.5 ± 2.9	20.5 ± 3.5	<0.001
Base excess, mEq/L	96.9	-2.0 ± 3.4	-1.1 ± 2.7	-1.4 ± 2.8	-1.7 ± 3.4	-2.1 ± 3.3	-4.5 ± 4.1	<0.001

Values are mean ± SD or n (%), unless otherwise indicated. P values are reported for trend across SCAI shock stages A to E.

CBS = coronary bypass surgery; CK-MB = creatine kinase muscle-brain type; LDH = lactate dehydrogenase; Shock index = ratio of heart rate to systolic blood pressure; TAVR = transcatheter aortic valve replacement; VAD = ventricular assist device; other abbreviations as in Table 1.

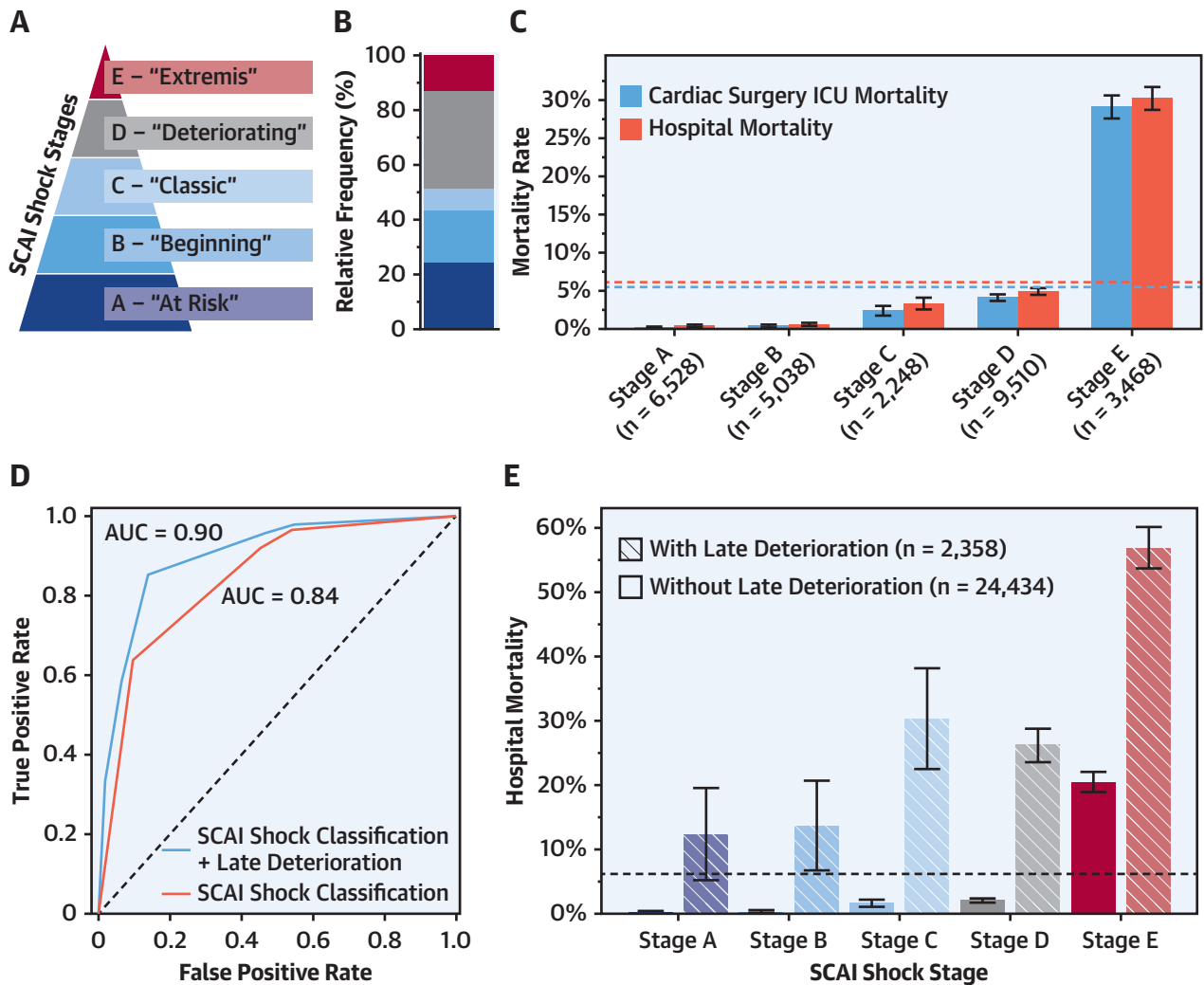
representing overall organ dysfunction were evident across SCAI shock stages (Table 3).

LATE DETERIORATION. Overall, LD occurred in 2,358 (8.8%) patients. LD was observed in 81 (1.2%) patients with stage A, in 95 (1.9%) patients with stage B, in 132

(5.9%) patients with stage C, in 1,118 (11.8%) patients with stage D, and in 932 (26.9%) patients with stage E.

LD was associated with a substantially higher hospital mortality overall (37.6% vs 3.1%; P < 0.001), and within every SCAI shock stage (all P < 0.001) (Central Illustration, Table 4). Patients with LD

CENTRAL ILLUSTRATION The Society for Cardiovascular Angiography and Interventions Shock Classification in Cardiac Surgery



Roeschl T, et al. J Am Coll Cardiol. 2023;82(17):1691-1706.

The SCAI shock classification stratified patients into 5 stages A to E (A) of respective relative proportions (B). Cardiac surgery ICU and hospital mortality increased as a function of higher SCAI shock stage (C). The incorporation of LD, defined as increasing vasopressor requirements after 24 h, noticeably increased the classification's diagnostic performance (D) and further stratified patients according to hospital mortality risk (E). The dashed lines represent average mortality rates and error bars represent 95% CIs. Subfigure A was reproduced with permission from SCAI (source: Naidu et al 2022⁹). ICU = intensive care unit; LD = late deterioration; SCAI = Society for Cardiovascular Angiography and Interventions.

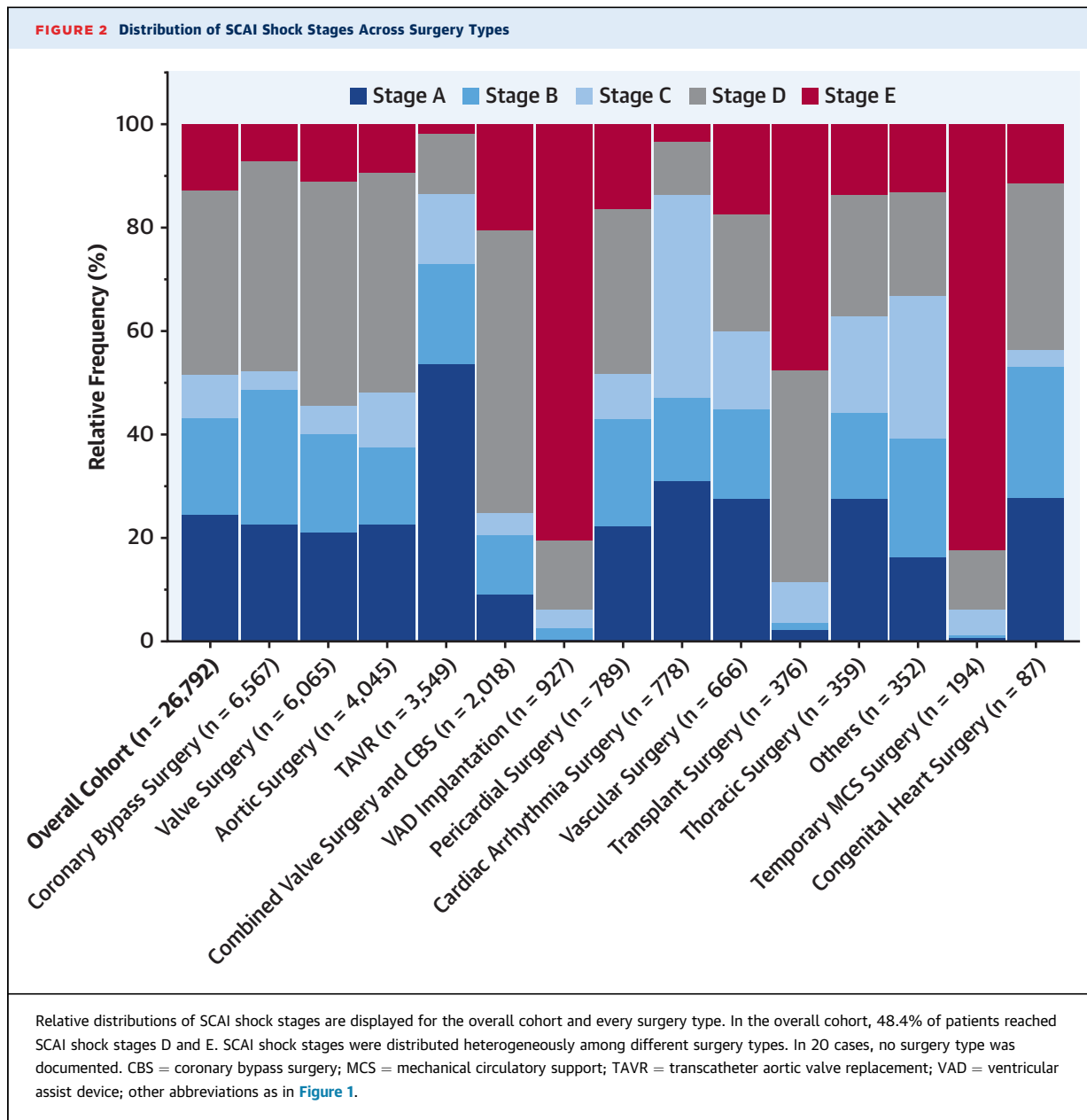
received inotropes in 83.6% and tMCS in 29.1% of cases.

When LD was added as the only additional predictor variable besides the SCAI shock classification to a logistic regression model, AUROCs significantly increased to 0.90 and 0.925 for hospital mortality and CSICU mortality (each $P < 0.001$), respectively. The adjusted ORs for hospital mortality were 1.37, 6.22,

7.24, 52.6, and 9.51 for SCAI shock stages B to E and LD, respectively.

SCAI shock stages C to E and LD remained independent predictors of hospital mortality, when adjusted for the Day 1 SOFA score (Table 4).

Overall, each higher SCAI shock stage was associated with increased hospital mortality compared with SCAI shock stage A (not significant for stage B; all



other $P < 0.001$ in every logistic regression model used in our study (Figure 5). Moreover, LD, CA, age, female sex, ESRD, and surgery duration could be identified, among others, as significant predictors of hospital mortality with adjusted ORs of 8.18, 3.37, 1.27 (per 10-y increase), 1.22, 2.31, and 1.12 (per 1-h increase), respectively. Regression results are summarized in Table 4. The full models' AUROCs were 0.926 and 0.95 for hospital mortality and CSICU mortality, respectively (Table 5).

The logistic regression models used in our study showed good model calibration with Brier scores ranging from 0.04 to 0.05 (Supplemental Figure 3).

DISCUSSION

This study evaluated the prognostic performance of the SCAI shock classification in a large cohort of unselected, postoperative CSICU patients according to

TABLE 3 ICU Interventions, Severity of Organ Dysfunction, and Outcomes as a Function of SCAI Shock Stage

	With Data, %	Overall	Stage A (n = 6,528)	Stage B (n = 5,038)	Stage C (n = 2,248)	Stage D (n = 9,510)	Stage E (n = 3,468)	P Value
Vasoactive therapy during first 1 h	99.5	21,365 (80.1)	4,053 (62.6)	3,790 (75.2)	1,432 (64.3)	8,637 (91.3)	3,453 (99.9)	<0.001
Vasoactives during first 1 h, n	99.6	1.2 ± 0.9	0.7 ± 0.6	0.9 ± 0.6	0.9 ± 0.7	1.3 ± 0.6	2.7 ± 0.8	<0.001
Peak VIS first 1 h	99.5	10.6 ± 15.0	4.2 ± 5.4	6.0 ± 6.6	6.7 ± 9.3	10.3 ± 10.0	32.7 ± 26.2	<0.001
Peak VIS	100	16.2 ± 28.7	4.4 ± 6.0	6.4 ± 8.6	8.8 ± 18.6	18.2 ± 26.4	52.3 ± 48.1	<0.001
Peak NEE first 1 h	99.5	0.11 ± 0.15	0.04 ± 0.05	0.06 ± 0.07	0.07 ± 0.09	0.1 ± 0.1	0.33 ± 0.27	<0.001
Peak NEE	100	0.16 ± 0.29	0.04 ± 0.06	0.06 ± 0.09	0.09 ± 0.19	0.18 ± 0.27	0.54 ± 0.5	<0.001
Vasopressor therapy	100	21,855 (81.6)	4,086 (62.6)	3,797 (75.4)	1,449 (64.5)	9,062 (95.3)	3,461 (99.8)	<0.001
Inotropic therapy	100	10,279 (38.4)	661 (10.1)	761 (15.1)	743 (33.1)	4,728 (49.7)	3,386 (97.6)	<0.001
Inhaled nitric oxide	100	2,820 (10.5)	36 (0.6)	38 (0.8)	97 (4.3)	801 (8.4)	1,848 (53.3)	<0.001
Inhaled iloprost	100	2,040 (7.6)	13 (0.2)	22 (0.4)	57 (2.5)	563 (5.9)	1,385 (39.9)	<0.001
Methylene blue	100	105 (0.4)	1 (0.0)	2 (0.0)	1 (0.0)	23 (0.2)	78 (2.2)	<0.001
Corticosteroid therapy	100	1,537 (5.7)	52 (0.8)	67 (1.3)	70 (3.1)	661 (7.0)	687 (19.8)	<0.001
Anti-infective agents, n	100	0.7 ± 1.5	0.1 ± 0.6	0.2 ± 0.7	0.5 ± 1.3	0.8 ± 1.6	2.1 ± 2.3	<0.001
Invasive ventilation during first 24 h	97.9	25,095 (95.7)	5,607 (90.2)	4,758 (96.0)	1,899 (90.5)	9,387 (98.9)	3,444 (99.5)	<0.001
Hemodialysis	100	825 (3.1)	12 (0.2)	7 (0.1)	132 (5.9)	318 (3.3)	356 (10.3)	<0.001
CRRT	100	1,602 (6.0)	15 (0.2)	19 (0.4)	74 (3.3)	482 (5.1)	1,012 (29.2)	<0.001
PAC	100	3,906 (14.6)	145 (2.2)	174 (3.5)	180 (8.0)	1,352 (14.2)	2,055 (59.3)	<0.001
PCI	100	653 (2.4)	138 (2.1)	76 (1.5)	66 (2.9)	191 (2.0)	182 (5.2)	<0.001
Impella (Abiomed)	100	313 (1.2)	1 (0.0)	4 (0.1)	2 (0.1)	19 (0.2)	287 (8.3)	<0.001
VA-ECMO	100	874 (3.3)	5 (0.1)	4 (0.1)	26 (1.2)	141 (1.5)	698 (20.1)	<0.001
IABP	100	786 (2.9)	1 (0.0)	11 (0.2)	14 (0.6)	109 (1.1)	651 (18.8)	<0.001
tMCS during first 24 h	100	1,411 (5.3)	3 (0.0)	12 (0.2)	23 (1.0)	120 (1.3)	1,253 (36.1)	<0.001
VAD during first 24 h	100	1,480 (5.5)	50 (0.8)	127 (2.5)	161 (7.2)	229 (2.4)	913 (26.3)	<0.001
Day 1 SOFA score	100	9.2 ± 3.0	7.3 ± 2.6	7.9 ± 2.5	9.7 ± 2.7	10.0 ± 2.4	12.2 ± 2.6	<0.001

Continued on the next page

the criteria and cutoff values established by Jentzer et al.⁴

Our study confirmed that the SCAI shock classification can successfully categorize postoperative CSICU patients into risk groups A-E with increasing CSICU mortality, hospital mortality, LOS, DMV, severity of organ dysfunction, and prevalence of postoperative complications. Strong mortality gradients across SCAI shock stages were also observed in relevant subgroups, thereby further highlighting its application in various cardio-surgical settings.

In our postoperative CSICU cohort, the diagnostic performance of the SCAI shock classification in predicting hospital mortality was superior to that previously reported in other cardiovascular patient cohorts, such as patients with established cardiogenic shock (AUROC = 0.65),¹² in cardiac intensive care unit (CICU) patients with sepsis and mixed septic-cardiogenic shock (AUROC = 0.68; 95% CI: 0.64-0.72),⁶ and in unselected CICU patients (AUROC = 0.765).⁴ Furthermore, the diagnostic performance was consistently high across patients with the most frequent surgery types. However, this did not apply to patients who received durable VAD

implantation, transplant surgery, or tMCS surgery before CSICU admission. In these small subcohorts, the SCAI shock classification showed poor diagnostic performance. This can be attributed to the fact that these surgery types on the one hand entail the treatment of CS per se and on the other hand are associated with extreme hemodynamic alterations that could not be adequately captured by the SCAI shock criteria used in our study.

The observed hospital mortality rates, ranging from 0.4% to 30.2%, were substantially lower compared with previous validation studies, in which reported hospital mortality rates spanned from, eg, 0.6% to 90.6%⁷ or 3.0% to 67.0%⁴ from SCAI shock stage A to E, respectively. Similarly, stages A to D were associated with below average hospital mortality rates in our study and our patient cohort exhibited the lowest stage E short-term mortality rate reported so far.⁹

In contrast to unselected CICU patients in the study by Jentzer et al,⁴ our CSICU patients more frequently reached SCAI shock stages D (35.5% vs 7.3%) and E (12.9% vs 1.0%), despite lower overall hospital mortality rates (6.2% vs 9.1%). Only one-half

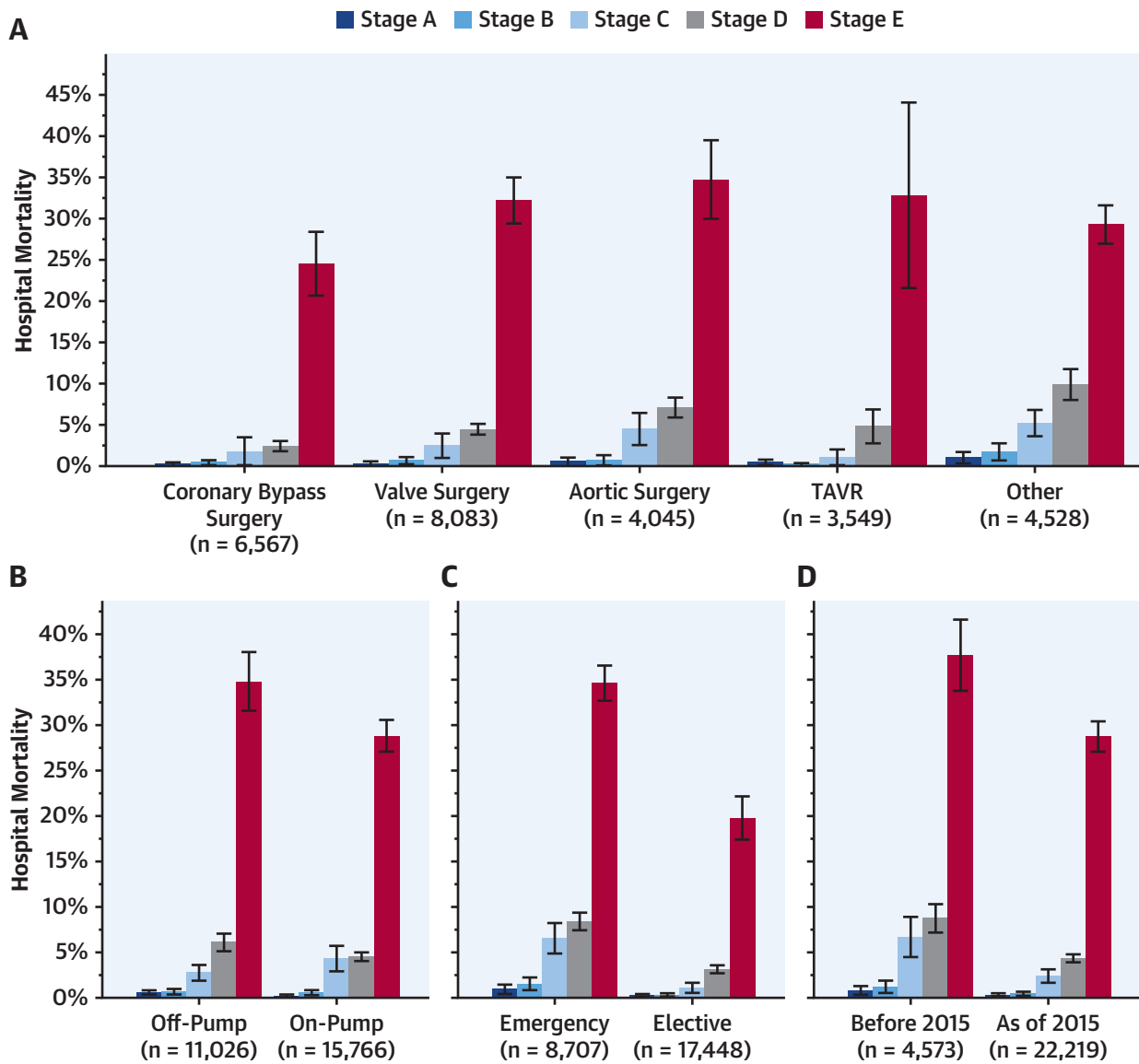
TABLE 3 Continued

	With Data, %	Overall	Stage A (n = 6,528)	Stage B (n = 5,038)	Stage C (n = 2,248)	Stage D (n = 9,510)	Stage E (n = 3,468)	P Value
Laboratory parameters indicative of hemodynamic deterioration, systemic inflammation, and end-organ dysfunction								
Peak arterial lactate, mEq/L	95.3	3.6 ± 4.2	1.3 ± 0.7	1.4 ± 0.9	2.4 ± 2.2	4.4 ± 3.4	9.4 ± 6.5	<0.001
Minimum arterial pH	98.2	7.31 ± 0.07	7.34 ± 0.05	7.33 ± 0.05	7.33 ± 0.07	7.29 ± 0.07	7.26 ± 0.09	<0.001
Minimum arterial base excess, mEq/L	98.1	-4.0 ± 3.7	-2.2 ± 2.8	-2.8 ± 2.8	-2.9 ± 3.4	-4.8 ± 3.5	-7.3 ± 4.3	<0.001
Minimum hemoglobin, g/dL	98.3	9.3 ± 1.9	10.3 ± 1.7	9.9 ± 1.6	9.8 ± 2.0	8.8 ± 1.7	7.9 ± 1.6	<0.001
Minimum PaO ₂ , mm Hg	98.3	76.8 ± 31.2	88.7 ± 36.1	82.0 ± 30.6	88.5 ± 42.6	68.0 ± 19.4	65.2 ± 29.0	<0.001
Minimum SaO ₂ , %	98.0	91.1 ± 10.2	94.0 ± 7.6	93.2 ± 8.0	92.6 ± 9.4	89.2 ± 11.1	86.8 ± 12.2	<0.001
Minimum ScvO ₂ , %	81.9	63.3 ± 10.5	66.9 ± 9.0	66.2 ± 8.9	64.5 ± 11.3	61.3 ± 10.4	60.7 ± 11.7	<0.001
Peak creatinine, mg/dL	96.0	1.3 ± 1.0	1.0 ± 0.5	0.9 ± 0.4	1.8 ± 1.6	1.3 ± 0.9	2.0 ± 1.3	<0.001
Peak serum urea, mg/dL	96.0	51.0 ± 33.6	39.2 ± 20.5	37.4 ± 19.6	60.6 ± 38.0	52.1 ± 31.7	83.0 ± 45.3	<0.001
Peak leukocyte count, 1,000/μL	96.7	14.0 ± 7.1	11.0 ± 5.5	12.0 ± 5.6	12.8 ± 6.2	15.2 ± 6.9	19.2 ± 8.5	<0.001
Peak CRP, mg/dL	77.7	12.7 ± 10.2	7.4 ± 7.4	8.2 ± 7.7	13.5 ± 10.0	14.2 ± 10.0	19.4 ± 10.8	<0.001
Peak CK-MB, U/L	96.7	82.6 ± 153.5	42.2 ± 51.5	49.0 ± 51.3	61.1 ± 136.0	91.6 ± 154.4	190.6 ± 276.5	<0.001
Peak creatine kinase, U/L	96.6	1,039.5 ± 4,886.1	428.1 ± 723.4	509.7 ± 549.7	998.5 ± 6,460.4	1,178.0 ± 5,878.4	2,537.5 ± 7,535.2	<0.001
Peak LDH, U/L	96.2	569.7 ± 1,304.8	276.3 ± 199.8	295.1 ± 194.7	425.9 ± 635.1	547.1 ± 1,003.2	1,628.1 ± 2,881.2	<0.001
Peak AST, U/L	96.2	251.3 ± 1,148.8	52.8 ± 118.8	61.1 ± 117.8	138.6 ± 530.4	215.2 ± 853.7	1,040.1 ± 2,640.5	<0.001
Peak GGT, U/L	95.6	71.7 ± 121.3	52.0 ± 87.6	49.8 ± 80.2	81.0 ± 140.9	74.9 ± 119.9	123.4 ± 181.4	<0.001
Peak total bilirubin, mg/dL	95.2	1.4 ± 1.9	0.9 ± 0.7	0.9 ± 0.7	1.3 ± 1.8	1.5 ± 1.5	3.1 ± 3.8	<0.001
Minimum albumin, g/dL	62.9	2.2 ± 0.6	2.5 ± 0.5	2.4 ± 0.5	2.2 ± 0.6	2.1 ± 0.5	1.8 ± 0.5	<0.001
Peak INR	96.5	1.6 ± 0.7	1.4 ± 0.3	1.4 ± 0.3	1.5 ± 0.6	1.6 ± 0.6	2.2 ± 1.3	<0.001
Peak lipase, U/L	61.4	84.4 ± 206.6	47.0 ± 176.9	41.7 ± 86.9	83.9 ± 200.1	78.7 ± 168.6	162.4 ± 324.4	<0.001
Minimum platelet count, 1,000/μL	96.7	142.2 ± 66.1	164.2 ± 62.6	159.2 ± 63.9	159.0 ± 74.0	131.6 ± 59.8	98.1 ± 58.7	<0.001
Secondary outcomes								
Acute kidney injury	89.1	2,298 (9.6)	21 (0.4)	26 (0.6)	178 (11.0)	798 (8.9)	1,275 (38.8)	<0.001
Delirium	86.9	3,092 (13.3)	219 (4.5)	198 (4.6)	183 (12.9)	1,627 (17.4)	865 (25.3)	<0.001
Rethoracotomy	100	977 (3.6)	12 (0.2)	41 (0.8)	26 (1.2)	513 (5.4)	385 (11.1)	<0.001
Symptomatic anemia requiring transfusion	100	6,999 (26.1)	398 (6.1)	486 (9.6)	362 (16.1)	3,403 (35.8)	2,350 (67.8)	<0.001
Acute hypoxemic respiratory failure	86.7	3,826 (16.5)	409 (8.6)	405 (9.1)	254 (16.0)	1,765 (19.3)	993 (30.2)	<0.001
Respiratory failure requiring reintubation	100	324 (1.2)	25 (0.4)	17 (0.3)	16 (0.7)	170 (1.8)	96 (2.8)	<0.001
Prolonged pulmonary weaning requiring tracheotomy	100	1,157 (4.3)	31 (0.5)	40 (0.8)	73 (3.2)	478 (5.0)	535 (15.4)	<0.001
Duration of mechanical ventilation, h	100	61.5 ± 182.8	10.4 ± 57.6	14.9 ± 72.1	45.0 ± 180.1	70.0 ± 176.3	213.0 ± 326.9	<0.001
Length of ICU stay, h	100	105.2 ± 243.8	29.4 ± 81.0	38.3 ± 105.8	78.6 ± 230.4	121.3 ± 222.6	318.2 ± 443.3	<0.001
Unplanned ICU readmission	80.7	972 (4.5)	168 (2.8)	168 (3.6)	85 (4.5)	420 (5.6)	131 (8.3)	<0.001
Primary outcomes								
ICU mortality	100	1,491 (5.6)	14 (0.2)	20 (0.4)	54 (2.4)	393 (4.1)	1,010 (29.1)	<0.001
Hospital mortality	100	1,651 (6.2)	28 (0.4)	31 (0.6)	75 (3.3)	468 (4.9)	1,049 (30.2)	<0.001

Values are n (%) or mean ± SD, unless otherwise indicated. P values are reported for trend across SCAI shock stages A to E.

AST = aspartate aminotransferase; CRP = C-reactive protein; CRRT = continuous renal-replacement therapy; GGT = gamma-glutamyl transferase; ICU = intensive care unit; INR = international normalized ratio; PAC = pulmonary artery catheter; PaO₂ = arterial partial pressure of oxygen; PCI = percutaneous coronary intervention; RBC = red blood cell; SaO₂ = arterial oxygen saturation; ScvO₂ = central-venous oxygen saturation; other abbreviations as in Tables 1 and 2.

FIGURE 3 Hospital Mortality as a Function of SCAI Shock Stage in Relevant Subgroups



Mean hospital mortality with 95% CIs as a function of SCAI shock stage and surgery type (A), off-pump vs on-pump surgery (B), surgical urgency (C), and time epoch (D). Surgery type and surgical urgency were missing in 20 and 637 cases, respectively. In general, hospital mortality rate increased steadily as a function of SCAI shock stage in relevant subgroups. Hospital mortality rate was lower among patients treated as of January 1, 2015, in every SCAI shock stage (not significant for stage A, $P < 0.05$ for stage B, all others $P < 0.001$). Abbreviations as in Figures 1 and 2.

as many postoperative CSICU patients remained in SCAI shock stage A during the first 24 hours compared with the Jentzer et al⁴ findings on CICU patients (24.4% vs 46.0%). The overall distribution of SCAI shock stages in our unselected, surgical patient cohort (Figure 2) was more similar to the distribution

seen in preselected CICU patients with established CS¹³ or after out-of-hospital CA,⁸ despite these cohorts showing much higher short-term mortality rates of 38.7% and 53.3%, respectively. This disparity once more highlights patients' pronounced dependency on hemodynamic support and intensive care measures

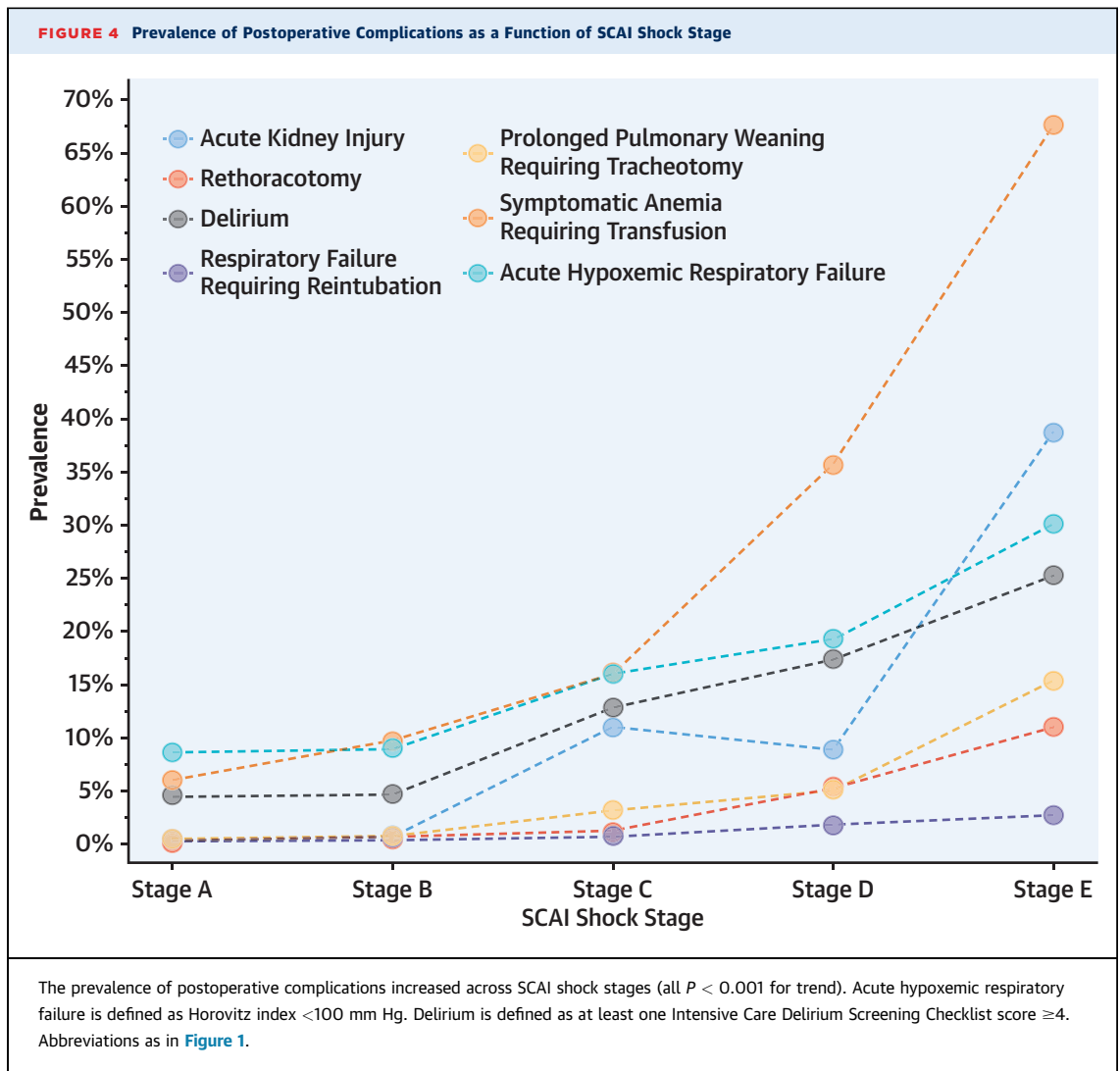
TABLE 4 Hospital Mortality as a Function of SCAI Shock Stage, LD, and Additional Confounders

	Crude Hospital Mortality	Hospital Mortality Stratified by LD (-/+)	Unadjusted OR ^a	Adjusted OR ^b	Adjusted OR ^c	Adjusted OR ^d
Stage A (reference stage)	0.43%	0.28% 12.35%	1.00	1.00	1.00	1.00
Stage B	0.62%	0.36% 13.68%	1.44 (0.86-2.40) <i>P</i> = 0.17	1.37 (0.82-2.29) <i>P</i> = 0.23	1.17 (0.70-1.96) <i>P</i> = 0.55	1.26 (0.75-2.11) <i>P</i> = 0.38
Stage C	3.34%	1.65% 30.30%	8.01 (5.18-12.40) <i>P</i> < 0.001	6.22 (4.00-9.66) <i>P</i> < 0.001	2.48 (1.57-3.90) <i>P</i> < 0.001	4.28 (2.72-6.72) <i>P</i> < 0.001
Stage D	4.92%	2.09% 26.21%	12.02 (8.20-17.62) <i>P</i> < 0.001	7.24 (4.92-10.66) <i>P</i> < 0.001	2.92 (1.97-4.33) <i>P</i> < 0.001	4.71 (3.16-7.02) <i>P</i> < 0.001
Stage E	30.25%	20.47% 56.87%	100.67 (68.97-146.94) <i>P</i> < 0.001	52.6 (35.89-77.08) <i>P</i> < 0.001	12.07 (8.12-17.92) <i>P</i> < 0.001	16.59 (10.98-25.07) <i>P</i> < 0.001
LD	-	3.13% 37.57%	18.62 (16.68-20.79) <i>P</i> < 0.001	9.51 (8.41-10.76) <i>P</i> < 0.001	8.01 (7.04-9.11) <i>P</i> < 0.001	8.18 (7.19-9.3) <i>P</i> < 0.001
Day 1 SOFA score (per 2-point increase)	-	-	-	-	2.13 (2.01-2.25) <i>P</i> < 0.001	-
Surgery duration (per 1-h increase)	-	-	-	-	-	1.12 (1.08-1.16) <i>P</i> < 0.001
Cardiopulmonary bypass time (per 1-h increase)	-	-	-	-	-	1.03 (0.97-1.10) <i>P</i> = 0.3
Aortic cross-clamp time (per 10-min increase)	-	-	-	-	-	0.99 (0.97-1.00) <i>P</i> = 0.11
Female sex	-	-	-	-	-	1.22 (1.07-1.40) <i>P</i> < 0.01
Age (per 10-y increase)	-	-	-	-	-	1.27 (1.20-1.34) <i>P</i> < 0.001
Admission diagnosis of cardiac arrest	-	-	-	-	-	3.37 (1.99-5.69) <i>P</i> < 0.001
End-stage renal disease	-	-	-	-	-	2.31 (1.63-3.28) <i>P</i> < 0.001
Elective procedure	-	-	-	-	-	0.47 (0.41-0.54) <i>P</i> < 0.001
Vasoactive therapy during the first 24 h	-	-	-	-	-	1.47 (1.02-2.14) <i>P</i> < 0.05
Invasive ventilation during the first 24 h	-	-	-	-	-	2.58 (1.30-5.10) <i>P</i> < 0.01
tMCS during the first 24 h	-	-	-	-	-	3.84 (3.23-4.55) <i>P</i> < 0.001
VAD during the first 24 h	-	-	-	-	-	1.08 (0.88-1.34) <i>P</i> = 0.46

Crude hospital mortality and hospital mortality stratified by LD are reported next to ORs for hospital mortality derived by ^athe SCAI classification or LD alone, ^bSCAI and LD, ^cSCAI, LD, and Day 1 SOFA score, and ^dSCAI, LD and additional confounders. ORs for hospital mortality are reported with 95% CIs and *P* values. LD was defined as increasing vasopressor requirements after the first 24 h.
LD = late deterioration; SCAI = Society for Cardiovascular Angiography and Interventions; SOFA = Sequential Organ Failure Assessment; other abbreviations as in Tables 1 to 3.

after cardiac surgery. Accordingly, the distributions of SCAI shock stages in our cohort of unselected CSICU patients and CICU patients⁴ were most similar for less-invasive surgery types including procedures frequently conducted within the cardiology field, such as transcatheter aortic valve replacement or cardiac arrhythmia surgery (eg, pacemaker implantation). Distributions deviated more strongly in surgical procedures associated with pronounced hemodynamic alterations, such as VAD implantation, tMCS surgery, or transplant surgery.

LD IS A CRUCIAL RISK MODIFIER. LD was first introduced into the broader context of the SCAI shock classification by Jentzer et al to describe shock progression beyond the first 24 hours and was addressed as a risk modifier in the SCAI SHOCK Stage Classification Expert Consensus Update.^{4,9} In our study cohort, the prevalence of LD increased steadily across SCAI shock stages, reaching its peak of 26.9% in stage E. The presence of LD further and distinctively separated patients across all SCAI shock stages into high-risk and low-risk subgroups. LD substantially



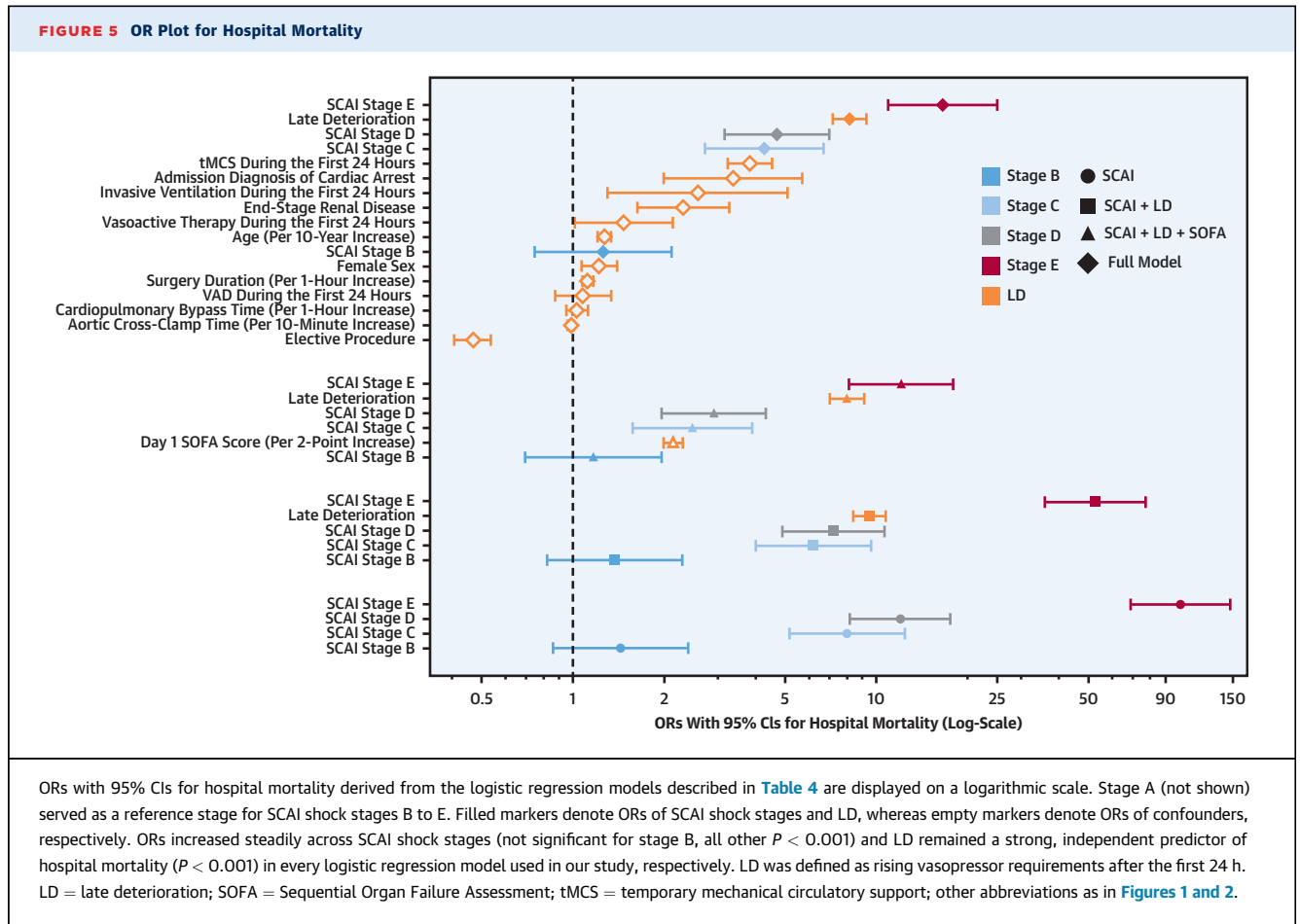
increased the diagnostic ability for hospital mortality over a broad risk range of 0.28% (stage A without LD) to 56.9% (stage E with LD) when added as a covariate besides SCAI shock stages to a logistic regression model ([Central Illustration](#)).

In addition, LD remained a strong predictor of hospital mortality, when adjusted for Day 1 SOFA scores and when additional confounders were added to the model. This implies that LD, even when applied outside of the traditional SCAI shock classification, is a worrisome phenomenon requiring adequate attention among treating physicians.

Besides LD and SCAI shock stages C to E, we could identify advanced age and an admission diagnosis of CA as independent risk factors, thus confirming the results of previous SCAI shock validation studies.^{4,5,14} Moreover, female sex was identified as an

independent risk factor for hospital mortality in our patient cohort. Similar sex disparities in short-term mortality were noted in previous studies including patients with acute myocardial infarction-CS¹⁵ and after cardiac surgery.¹⁶ Surgery duration, surgical urgency, ESRD, and tMCS, vasoactives, and invasive ventilation during the first 24 hours were also independently associated with hospital mortality.

A FULLY AUTOMATED DECISION SUPPORT TOOL FOR POSTOPERATIVE CARE. Due to the strong diagnostic performance of the SCAI shock classification in our study cohort, especially with the incorporation of LD, we propose to use the SCAI shock classification as a decision support tool in the clinical care of postoperative CSICU patients. In this sense, patients classified into SCAI shock stages A and B may be considered for early CSICU discharge. These



patients had a more than 10-fold lower crude hospital mortality rate compared with average hospital mortality rate and comparatively rarely had postoperative complications, LD, or unplanned ICU readmissions. On the other hand, patients classified as SCAI shock stage E or patients with stage C upward and concomitant LD displayed markedly high hospital mortality rates and, therefore, should be systematically assessed for shock type (eg, via invasive hemodynamic monitoring) and evaluated regarding eligibility for advanced treatment modalities. Advanced treatment modalities might exemplarily include tMCS, corticosteroids, anti-infectives, or methylene blue depending on the underlying shock etiology. Regarding the eligibility criteria for the initiation or escalation of tMCS, we propose to include echocardiographic findings and absolute levels of arterial lactate and vasoactive-inotropic score in the assessment as previously published¹⁷ in addition to shock severity expressed by SCAI shock stage and LD.

Last, the SCAI shock classification identified patients at increased risk of postoperative

complications as well as organ dysfunction, thus potentially enabling physicians to anticipate these events and intervene early.

The definitions of SCAI shock stages according to Jentzer et al are based on variables commonly obtained in postoperative CSICU patients and generally stored in a machine-readable format. Therefore, real-time, automated classification of patients into SCAI shock stages during the first 24 hours and detection of LD beyond 24 hours could be achieved through an electronic health record-integrated application in postoperative CSICU patients, similarly to the suggestion by Jentzer et al for CICU patients.⁴ In this way the SCAI shock classification could be readily implemented in clinical practice without adding significantly to the documentation expenditure of healthcare providers.

RESEARCH IMPLICATIONS FOR INTENSIVE CARE AFTER CARDIAC SURGERY. When the SCAI shock classification was introduced in 2019, one of the main research objectives was to stratify patients by CS severity because it was assumed that treatment

TABLE 5 Areas Under the Receiver Operating Characteristic

	CSICU Mortality	Hospital Mortality
SCAI shock classification	0.859 (0.850-0.867)	0.842 (0.833-0.850)
SCAI shock classification + LD	0.925 (0.918-0.930)	0.90 (0.893-0.907)
SCAI shock classification + LD + Day 1 SOFA score	0.949 (0.944-0.954)	0.924 (0.916-0.930)
SCAI shock classification + LD + confounders ("full model")	0.950 (0.945-0.954)	0.926 (0.919-0.932)

Areas under the receiver operating characteristic are reported with 95% CIs.
CSICU = cardiac surgery intensive care unit; other abbreviations as in Tables 2 to 4.

efficiency of, for example, advanced treatment modalities varied considerably in patient subsets at different mortality risks. In cardiology patients, this was best seen by the fact that numerous prospective randomized trials failed to demonstrate the expected therapeutic benefit of tMCS in CS presumably due to considerable heterogeneities in disease severity, etiology, and comorbidities in patients enrolled.^{1,2,18} Regarding the postoperative CSICU patient in shock, the list of advanced treatment modalities subject to inconclusive evidence of effectiveness extends beyond tMCS,¹⁹ exemplarily including methylene blue therapy,²⁰ corticosteroid therapy,²¹ liberal red blood cell transfusion strategies,²² colloidal volume replacement,²³ inhaled pulmonary vasodilators,²⁴ heart rate modulation via epicardial pacing in the absence of overt bradycardia,²⁵ rhythm control in patients with postoperative atrial fibrillation,²⁶ and liberal antimicrobial therapy.²⁷ In this sense, and given the observed trends of, for example, decreasing serum hemoglobin, serum albumin, and arterial pH levels across SCAI shock stages, respectively, while inflammatory parameters increased accordingly in our study, one might argue that additional therapeutic target areas in postoperative care could emerge from these insights and could be the subject of future prospective studies.

The observation that hospital mortality rates decreased among all SCAI shock stages in patients treated as of compared with before January 1, 2015 (Figure 3) points toward an overall improvement in shock management over the recent years, which should be built on further.

STUDY LIMITATIONS. Due to the retrospective nature of our study, prospective validation of our study results, preferably in a multicenter trial, is indispensable.

As a quaternary care clinic, our institution receives critically ill patients in CS from external clinics and highly morbid patients deemed inoperable at other institutions, contributing to the high proportion of

nonelective cases, advanced age, and comparatively high mortality rates observed in our study, potentially limiting the generalizability of our results.

Only a marginal proportion of patients had CA before hospital admission according to available International Classification of Diseases-10th Revision codes. Data regarding neurologic outcomes after CA was absent. Therefore, we refrained from incorporating the recently proposed “A” modifier into our interpretation of the SCAI shock classification,⁹ which we think is justifiable in postoperative CSICU patients compared with CICU patients, where a notable proportion of patients had an admission diagnosis of CA.^{4,28} Similarly, data regarding limitations of therapy were not available.

Patient assessment scores, such as EuroSCORE, were not sufficiently available therefore not included as covariates in the logistic regression models used in our study despite potentially being meaningful.

Due to an unselected patient cohort, and because invasive hemodynamic measurement data were not ubiquitously available, we cannot quantify the proportion of patients truly or solely in CS compared with other shock etiologies or mixed-shock states. Nevertheless, the considerably high proportion of patients with LD and in stage E who received inotropes and tMCS, respectively, is at least indicative of a significant contribution of cardiac dysfunction to shock development in our patient cohort (eg, due to preoperative acute myocardial infarction complicated CS or postoperative low cardiac output syndrome). Furthermore, the SCAI shock classification has recently been applied to other mixed cohorts, displaying noteworthy diagnostic performances.^{4,6}

CONCLUSIONS

In the largest yet conducted SCAI shock classification validation study, we demonstrated the practical utility of the SCAI shock classification in a so far untested cohort of postoperative CSICU patients. The SCAI shock classification provided robust risk stratification overall and in relevant subgroups. The diagnostic performance of the SCAI shock classification in our cohort of unselected patients exceeded that of comparable studies and further increased when LD was integrated into the model. In addition, the SCAI shock classification also identified patients at increased risk of postoperative complications and organ dysfunction.

We conclude that the SCAI shock classification has the potential to provide a common vocabulary for postoperative patient status and risk prediction in the field of cardiac surgery. Due to its simple definitions,

completely built on machine-readable data, the SCAI shock classification could be readily applied as a triage tool in postoperative care through a fully automated, electronic health record-integrated application, easily available to treating physicians. We propose to include the SCAI shock stages as a surrogate parameter of disease severity in future clinical trials regarding intensive care and the treatment of shock, to ensure consistent patient selection and precise conclusions on treatment effectiveness.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Falk has received educational grants (including travel support), fees for lectures and speeches, fees for professional consultation, and research and study funds from Medtronic GmbH, Biotronik SE & Co, Abbott GmbH & Co KG, Boston Scientific, Edwards Lifesciences, Berlin Heart, Novartis Pharma GmbH, JO TEC GmbH, and Zurich Heart. Dr O'Brien has received research funding from the British Heart Foundation and the National Institute for Health Science Research; and has served as a consultant for Teleflex. Dr Schoenrath has received institutional grants from Novartis and Abbott; has received nonfinancial support from Medtronic; and has received institutional fees (speaker honoraria) from Orion Pharma outside of the submitted work. Dr Potapov has received educational grants (including travel support), fees for lectures and speeches, fees for professional consultation, and research and study funds from Medtronic GmbH, Abbott GmbH & Co KG, and Abiomed. Dr Ott has received research and study funds from Novartis Pharma GmbH; and has received consulting fees, fees for lectures, and travel grants from Abiomed Europe GmbH. Dr Balzer has received funding from Medtronic; has received grants from the German Federal Ministry of Education and Research, the German Federal Ministry of Health, the Berlin Institute of Health Hans Böckler Foundation; Einstein Foundation, and the Berlin University Alliance outside the submitted

work; has received personal fees from Elsevier Publishing; and has received other from Robert Koch Institute. Dr Meyer has received lecturing fees from Bayer and Medtronic; has received consulting fees from Medtronic and Pfizer; and is founder and managing director of x-cardiac GmbH. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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PERSPECTIVES

COMPETENCY IN PATIENT CARE AND PROCEDURAL

SKILLS: The SCAI shock classification effectively risk stratifies patients for postoperative complications, organ dysfunction, and mortality. Patients in SCAI shock stages A and B may be considered for early discharge from the ICU, whereas those in SCAI shock stage E or in stages C and D with late deterioration are at high mortality risk and may benefit from early initiation of advanced treatment modalities such as temporary mechanical circulatory support.

TRANSLATIONAL OUTLOOK: Prospective studies with precise differentiation of shock types are needed to evaluate the efficacy of advanced treatment modalities in relation to disease severity.

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KEY WORDS cardiac surgery, cardiogenic shock, cardiovascular, critical care, mortality

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