Application of the SCAI classification in a cohort of patients with cardiogenic shock

Benedikt Schrage MD1,2 | Salim Dabboura MD1 | Isabell Yan MD1 | Rafel Hilal MD1 | Johannes T. Neumann MD1,2 | Nils A. Sörensen MD1,2 | Alina Gößling BSc1 | Peter Moritz Becher MD1 | Hanno Grahn MD1 | Tobias Wagner MD1 | Moritz Seiffert MD1 | Stefan Kluge MD3 | Hermann Reichenspurner MD, PhD4 | Stefan Blankenberg MD1,2 | Dirk Westermann MD1,2

1Department of Interventional and General Cardiology, University Heart Centre Hamburg, Hamburg, Germany
2German Centre for Cardiovascular Research (DZHK), Partner Site Hamburg/Lübeck/Kiel, Hamburg, Germany
3Department of Intensive Care Medicine, University Clinic Hamburg-Eppendorf, Germany
4Department of Cardiothoracic Surgery, University Heart Centre Hamburg, Hamburg, Germany

Correspondence
Dirk Westermann, Department of Cardiology, University Heart and Vasculature Centre Hamburg, Martinistr. 52, 20246 Hamburg, Germany.
Email: d.westermann@uke.de

Funding information
University Heart Centre Hamburg: Abbott Diagnostics: German Research Foundation; German Heart Foundation/German Foundation of Heart Research; German Centre for Cardiovascular Research

Abstract
Background: The Society of Cardiovascular Angiography and Interventions (SCAI) have recently proposed a new classification of cardiogenic shock (CS) dividing patients into five subgroups.

Objective: Aim of this study was to apply the SCAI classification to a cohort of patients presenting with CS and to evaluate its ability to predict 30-day survival.

Methods: SCAI CS subgroups were interpreted based on the recent consensus statement and then applied to N = 1,007 consecutive patients presenting with CS or large myocardial infarction (MI) between October 2009 and October 2017. The association between SCAI classification and 30-day all-cause mortality was assessed by logistic regression analysis.

Results: Mean age in the study cohort was 67 (±15) years, 72% were male. Mean lactate at baseline was 6.05 (±5.13) mmol/l and 51% of the patients had prior cardiac arrest. Overall survival probability was 50.6% (95% confidence interval [CI] 47.5–54.0%). In view of the SCAI classification, the survival probability was 96.4% (95% CI 93.7–99.0%) in class A, 66.1% (95% CI 50.2–87.1%) in class B, 46.1% (95% CI 40.6–52.4%) in class C, 33.1% (95% CI 26.6–41.1%) in class D, and 22.6% (95% CI 17.1–30.0%) in class E. Higher SCAI classification was significantly associated with lower 30-day survival (p < .01).

Conclusion: In this large clinical cohort, the SCAI classification was significantly associated with 30-day survival. This finding supports the rationale of the SCAI CS classification and calls for a validation in a prospective trial.

KEYWORDS
cardiogenic shock, classification, SCAI
INTRODUCTION

Cardiogenic shock (CS) is the most severe form of acute heart failure and is associated with a high mortality. Albeit intense efforts, mortality has remained at a high level over the past decades. Only few randomized clinical trials showed treatment benefits for patients presenting with CS and many others have shown a neutral treatment effect.\(^1\)

The main pathophysiology of CS is end-organ hypoperfusion due to severely reduced cardiac output. Importantly, the causes of CS vary substantially within the overall patient population. Whereas in some acute myocardial infarction (AMI) is the underlying cause of CS, others are admitted with decompensated chronic heart failure or non-coronary structural heart disease leading to CS.\(^2\) These varying causes are a challenge for the design of clinical trials and the development of successful treatment strategies.

Additionally, patients with CS present to the hospital in different stages of the disease. This impacts application of different treatment options as well as outcome, as patients with beginning CS face a lower risk and demand other treatments than high-risk patients with refractory cardiac arrest. Unfortunately, staging of patients is difficult and non-reproducible in the clinical setting. This has multiple consequences for medical management and research in CS: (a) communicating the course of CS and the patient’s status between physicians is difficult and prone to misapprehension; (b) evaluating treatment options and assessing prognosis may be hampered without defining stages of disease severity; (c) enrolling patients in clinical trials is complicated without specification of distinct risk groups; and (d) especially results of clinical trials might be misinterpreted and are difficult to transfer to other populations without a “universal” language in the community.

To address this problem, the Society of Cardiovascular Angiography and Interventions (SCAI) has recently proposed a new classification of CS dividing patients into five subgroups: patients at risk of developing CS (A), patients with beginning CS (B) or classic CS (C), as well as deteriorating patients (D) or patients presenting in extremis (E).\(^3\)

However, application of this classification in the clinical setting is needed in multiple cohorts before applying it at a broader scale. The aim of this study was to test the SCAI CS classification in a broad real-world cohort of patients presenting with CS and to evaluate its association with 30-day survival.

MATERIAL AND METHODS

Study population

For the current analysis, patients aged 18 years or older with CS treated at the host institution between October 2009 and October 2017 were considered. To identify eligible patients, patient records were scanned for the ICD-10 code of CS (R.57). Hereafter, every case was manually reviewed to validate that CS was the primary diagnosis leading to hospital admission. Baseline data, comorbidities, treatment data, and in-hospital follow-up were collected in a dedicated database.

Additionally, stable patients with large AMI were selected from the prospective Biomarkers in Acute Cardiac Care study. This study enrolls patients with suspected AMI upon presentation to the emergency department and collects biomarkers samples at multiple time points. Patients are followed up during the hospital stay and after discharge.\(^4\)

This study was performed in accordance with the Declaration of Helsinki and was approved by the local ethics committee.

SCAI classification

The SCAI classes were interpreted based on the consensus statement and each patient was assigned to one of the five classes\(^3\):

SCAI class A—At risk

Hemodynamical stable patients with a large AMI, defined as highsensitive troponin I \(\geq\) 1,000 pg/ml within the first 3 hours after presentation, were selected to represent this group. Stable hemodynamics were defined as having a ratio of heart rate to systolic blood pressure below 1 without need for inotropic/vasoactive therapy, and no hypoperfusion (arterial lactate <2 mmol/L or venous lactate <2.5 mmol/L). Furthermore, patients within this group should not have signs/symptoms of CS.

SCAI class B—Beginning CS

The SCAI consensus statement defines these patients as having clinical evidence of relative hypotension or tachycardia, but without hypoperfusion. In the present study, this was interpreted as patients having signs/symptoms of CS or a ratio of heart rate to systolic blood pressure above 1, but no use of inotropic/vasoactive therapy and no hypoperfusion (definition above).

SCAI class C—Classic CS

The consensus statement describes these patients as having manifested symptoms of CS with impaired perfusion in need of therapy. In the present study, this was interpreted as patients with signs/symptoms of CS and hypoperfusion (definition above) or use of inotropic/vasoactive therapy.

SCAI class D—Deteriorating CS

These patients are described as SCAI class C patients who deteriorate despite therapy. In the present study, this was defined as SCAI class C
patients with any increase in lactate within the first 6 hours after presentation despite treatment. To avoid misclassification, patients meeting SCAI class C criteria but without a second lactate measurement were excluded from the analysis.

2.2.5 | SCAI class E—Extremis

In accordance with the SCAI consensus statement, patients with prolonged cardiac arrest and ongoing cardiopulmonary resuscitation (including extracorporeal membrane oxygenation [ECMO] assisted resuscitation) were allocated to this group.

2.3 | Statistical analysis

Continuous baseline variables are shown as mean (±SD). For binary baseline variables, absolute and relative frequencies are shown. Regular analysis of variance (ANOVA) (for continuous variables) or the χ² test/fisher’s exact test (for categorical variables) were used for comparisons between groups.

The primary endpoint of this analysis was 30-day survival, whereas patients were censored upon discharge from the hospital. Survival probability per SCAI class was estimated using the Kaplan–Meier method. Logistic regression analysis was performed to evaluate the association of SCAI class with the primary endpoint, adjusted for age, sex, prior cardiac arrest, AMI, and year of registration.

A p-value of <.05 was considered statistically significant. All analyses were performed with R statistical software version 3.5.1.

3 | RESULTS

3.1 | Overall study cohort

A total of 815 CS patients admitted with CS between October 2009 and October 2017 were identified and included in this analysis. Additionally, 192 patients with large AMI were included in this analysis, which resulted in an overall study cohort of 1,007 patients.

Mean age of the overall cohort was 67 (±15) years, 28.5% of the patients were female. AMI accounted for only 58% of the cases, which a relevant proportion being nonischemic CS. Mean lactate on admission was 6.1 (±5.1) mmol/l with a mean pH of 7.25 (±0.19). Additional baseline characteristics are shown in Table 1.

3.2 | Application of the SCAI classification

After application of the interpreted SCAI CS classification criteria, 192 patients (19%) were assigned to SCAI class A, 35 patients (4%) were assigned to SCAI class B, 369 patients (37%) were assigned to SCAI class C, 226 patients (22%) were assigned to SCAI class D, and 185 patients (18%) were assigned to SCAI class E.
Slightly more patients in SCAI class C had had a prior cardiac arrest as compared to patients in SCAI class D. Similarly, slightly more high concentrations of aspartate-aminotransferase and troponin were observed in SCAI classes C–E, reflecting the severe nature of CS. Furthermore, use of mechanical circulatory support devices, such as the Impella® (Abiomed, Danvers) or veno-arterial extracorporeal membrane oxygenation, was higher with more advanced SCAI classes.

### Table 2

Baseline characteristics per SCAI cardiogenic shock class

| SCAI | A (N = 192) | SCAI | B (N = 35) | SCAI | C (N = 369) | SCAI | D (N = 226) | SCAI | E (N = 185) | p |
|------|-------------|------|-------------|------|-------------|------|-------------|------|-------------|---|
| **Demographics** | | | | | | | | | | |
| Age (years) | 65 (±13) | 72 (±16) | 68 (±15) | 69 (±17) | 63 (±13) | <.01 |
| Female sex | 50 (26.0%) | 9 (25.7%) | 107 (29%) | 80 (35.4%) | 41 (22.2%) | .05 |
| **Cardiovascular risk factors** | | | | | | | | | |
| Smoking | 118 (61.5%) | 12 (34.3%) | 101 (27.5%) | 55 (24.4%) | 65 (35.7%) | <.01 |
| Arterial hypertension | 137 (71.4%) | 24 (68.6%) | 185 (50.4%) | 113 (50.2%) | 74 (40.7%) | <.01 |
| Hypercholesterinemia | 79 (41.1%) | 8 (22.9%) | 41 (11.2%) | 20 (8.9%) | 20 (11.0%) | <.01 |
| Diabetes | 29 (15.4%) | 12 (34.3%) | 99 (27.0%) | 63 (28.0%) | 36 (19.8%) | <.01 |
| Chronic kidney disease | 70 (36.6%) | 7 (20.0%) | 68 (18.5%) | 44 (19.6%) | 15 (8.2%) | <.01 |
| Prior myocardial infarction | 28 (14.7%) | 10 (28.6%) | 87 (23.7%) | 62 (27.6%) | 39 (21.4%) | .03 |
| Prior stroke | 8 (4.2%) | 6 (17.1%) | 35 (9.2%) | 24 (10.7%) | 9 (4.9%) | .01 |
| **Presentation** | | | | | | | | | |
| AMI complicated by cardiogenic shock | 192 (100.0%) | 24 (68.6%) | 172 (46.6%) | 94 (41.6%) | 99 (53.5%) | <.01 |
| Non-ischemic cardiogenic shock | 0 (0.0%) | 11 (31.4%) | 197 (53.4%) | 132 (58.4%) | 86 (46.5%) | <.01 |
| Prior CPR | 0 (0.0%) | 9 (25.7%) | 208 (56.5%) | 115 (50.9%) | 172 (93.0%) | <.01 |
| Duration of CPR (min) | NA | 6.2 (±5.1) | 24.2 (±26.3) | 23.2 (±19.1) | 53.9 (±43.5) | <.01 |
| Intubation | 0 (0.0%) | 9 (25.7%) | 259 (70.8%) | 137 (60.9%) | 170 (91.9%) | <.01 |
| Inotropes/vasopressors | 0 (0.0%) | 0 (0.0%) | 337 (91.3%) | 195 (86.3%) | 185 (100%) | <.01 |
| **Hemodynamics** | | | | | | | | | |
| SBP (mmHg) | 148.6 (±26.5) | 127.4 (±29.6) | 109.3 (±33.5) | 105.0 (±33.0) | 94.6 (±39.6) | <.01 |
| DBP (mmHg) | 87.4 (±14.2) | 76.3 (±21.5) | 64.3 (±22.0) | 60.9 (±21.4) | 55.9 (±26.4) | <.01 |
| Heart rate (bpm) | 80.8 (±17.0) | 90.1 (±24.5) | 90.1 (±27.4) | 95.1 (±33.8) | 80.2 (±40.6) | <.01 |
| **Laboratory** | | | | | | | | | |
| Lactate (mmol/l) | NA | 1.5 (±0.5) | 5.98 (±4.8) | 4.7 (±4.3) | 8.8 (±5.9) | <.01 |
| pH | NA | 7.37 (±0.07) | 7.25 (±0.17) | 7.30 (±0.17) | 7.16 (±0.21) | <.01 |
| eGFR (mlmin⁻¹1.73m⁻²) | 69.1 (±22.2) | 48.5 (±21.1) | 44.5 (±23.2) | 45.0 (±26.5) | 45.4 (±21.6) | <.01 |
| ASAT (U/l) | NA | 180 (±307) | 475 (±1,182) | 666 (±2,027) | 582 (±1,594) | .3 |
| CK (U/l) | 951 (±1,405) | 650 (±1,196) | 744 (±1,448) | 939 (±3,544) | 1,588 (±3,383) | <.01 |
| High-sensitive troponin I (ng/l) | 1939 (±2,479) | 1,265 (±2039) | 1,252 (±2,491) | 1,330 (±2,380) | 2015 (±3,328) | <.01 |
| **Mechanical circulatory support** | | | | | | | | | |
| Impella® | 0 (0.0%) | 3 (8.6%) | 43 (11.7%) | 26 (11.5%) | 68 (36.8%) | <.01 |
| VA-ECMO | 0 (0.0%) | 1 (2.9%) | 54 (14.6%) | 46 (20.4%) | 122 (65.9%) | <.01 |

Abbreviations: AMI, acute myocardial infarction; ASAT, aspartate-aminotransferase; CK, creatine kinase; CPR, cardiopulmonary resuscitation; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate estimated by CKD-EPI formula; SBP, systolic blood pressure; SCAI, Society of Cardiovascular Angiography and Interventions; VA-ECMO, veno-arterial extracorporeal membrane oxygenation therapy.

During a median follow-up time of 14 (interquartile range 3–30) days, 469 deaths (46.6%) occurred in the overall study cohort. Thirty-day survival probability in the overall study cohort was 50.6% (95% confidence interval [CI] 47.5–54.0%). After application of the SCAI CS classification, each SCAI class was significantly associated with a lower 30-day survival probability: 96.4% (95% confidence interval [CI] 93.7–99.0%) in SCAI-class-A, 66.1% (95% CI 50.2–87.0%) in B, [±0.17] vs. 7.30 [±0.17], p < .01 for both). Similarly, slightly more patients in SCAI class C had had a prior cardiac arrest as compared to patients in SCAI class D.
46.1% (95% CI 40.1–52.3%) in C, 33.1% (95% CI 26.6–41.1%) in D, and 22.6% (95% CI 17.1–30.0%) in E (p < .01, Figure 1).

After adjustment for multiple relevant confounders, each SCAI class was significantly associated with the primary endpoint (Figure 2).

**FIGURE 1**  Thirty-day in-hospital survival per SCAI class. CS, cardiogenic shock; SCAI, Society of Cardiovascular Angiography and Interventions

**FIGURE 2**  Association between SCAI CS classes and 30-day in-hospital survival. CI, confidence interval; SCAI, Society of Cardiovascular Angiography and Interventions [Color figure can be viewed at wileyonlinelibrary.com]

4 | DISCUSSION

In this large, unselected clinical cohort of CS patients, the SCAI classification identified patients at different stages of the disease and each class was associated with a lower 30-day survival rate. This association persisted after adjustment for several relevant confounders.

The recently published SCAI CS classification addressed the need for a universal definition of disease severity in CS, as it allows for an estimation of mortality risk. These estimates can be used as a basis for interdisciplinary communication in the clinical setting. Additionally, this classification might also have an impact on clinical research. Here, it could be used to assess the potential magnitude of benefit/utility of a therapy in a stratified scheme of CS, for example, testing therapies in more or less severe CS subpopulations.

In the present study of an unselected clinical cohort, the SCAI classification successfully identified CS patients in different risk strata. Importantly, these results have to be compared to those of a recently published study with a similar approach. Jentzer et al., applied the SCAI CS classification to an unselected cohort of patients admitted to a cardiac intensive care unit. Similar to our results, the authors could show that the SCAI CS classification was independently associated with in-hospital mortality. Of note, overall mortality in the study of Jentzer et al. was much lower as in this study. This is a direct reflection of the different patient populations used in the two studies: For this study, only patients with CS (except for SCAI class A) were considered. Hence, observed overall mortality was comparable to previous randomized clinical trials on this topic. In the study by Jentzer et al., all patients admitted to an intensive care unit were considered, with or without CS at baseline. This allowed for a broader inclusion of...
patients with different baseline risks, the majority were in SCAI classes A or B, and expectedly led to a lower overall mortality.5

Ultimately, both studies complement each other, as they could show an association of the SCAI CS classification with outcome in an at-risk population as well as in a high-risk population.

The SCAI CS classification can be applied in clinical practice as in research, and has important implications for both. At first, it highlights patients at risk of developing CS or with beginning CS (SCAI classes A and B). These patient populations have not been covered by previous randomized trials on this topic, which mostly use hypoperfusion (e.g., elevated lactate) as an inclusion criterion.6,8,9 However, preventing the development or worsening of CS in the respective patient populations is intriguing, given the paucity of effective treatments for established CS.1 Based on a previous study, it was expected that the majority of patients with CS are already admitted with CS.10 However, this study mainly analyzed patients with AMI and thus might have underestimated the at-risk population, as indicated by the high prevalence of SCAI class A patients in the study by Jentzer et al.5 As previous studies indicated improved outcomes with early treatment, the SCAI CS classification might provide a useful tool to identify patients at-risk or with beginning CS who could benefit from such therapies.11 Additionally, it might also be considered to extend the inclusion criteria of ongoing randomized controlled CS trials to cover patients with beginning CS. This seems to be justified by the high mortality risk of these patients in this study and could help with the enrollment difficulties in randomized controlled CS trials.

Secondly, the SCAI CS classification distinguishes between patients with classic CS and worsening CS. In both studies, mortality in patients with worsening CS was higher than compared to patients with classic CS. Interestingly, baseline lactate was lower and other presentation characteristics were comparable between both groups in this study. This finding is in line with the results of a recent analysis of 2,191 critically ill patients. In this study, increasing lactate within 24 hr was strongly associated with mortality, despite numerically lower baseline lactate.12 These findings support the rationale to use both, assessment of baseline lactate and early changes in lactate, to identify high-risk patients with deteriorating CS.

Thirdly, the SCAI classification is not limited to a specific cause of CS, but can be applied to a broader patient population. Previous scores/classifications for CS were developed in specific subpopulations, such as patients with AMI complicated by CS.13,14 Therefore, the generalizability of these scores to other CS subpopulations might be limited. In contrast, the SCAI classification was designed to fit to an overall CS population and has now been retrospectively used in two broad clinical cohorts, including patients with and without ischemic CS. The results from these two studies indicate that the SCAI classification provides adequate risk estimates in such broad CS populations. Barring a successful validation in a prospective trial, potentially including a comparison versus other CS scores, this classification could therefore be used to facilitate inter-disciplinary communication in the CS team and to improve awareness and early diagnosis of CS irrespective of the underlying cause.15,16

4.1 | Limitations

The major limitation of this study is its retrospective design. Not all aspects of the SCAI classification were applicable in this study. Albeit the available variables represent clinical routine and are widely used in this field, it was not possible to evaluate if additional factors such as specific biomarkers or intracardiac pressure management further improve the classification. Secondly, we cannot rule out a selection bias, as patients were identified via ICD-10 codes. This might have led to inclusion of misdiagnosed patients (e.g., patients not having CS) or failure to include patients with CS (e.g., patients having CS but being miscoded). However, it is rather unlikely that miscoding/misdiagnosis would have impacted the results, as every enrolled patient’s files were carefully reviewed for CS being the primary diagnosis and given the large sample size of the study cohort. Third, use of ICD-10 codes for patients with cardiogenic shock might have changed over time and could have impacted the results. However, as all files were manually reviewed, the impact of this should be limited to missing patients with CS and not inclusion of misdiagnosed patients. Fourth, the at-risk population only consisted of AMI patients. Hence, our findings cannot be transferred to patients at risk of CS without AMI.

5 | CONCLUSION

In a large clinical cohort of CS patients, SCAI classification was independently associated with 30-day survival. This association persisted after adjustment for several relevant confounders. These findings support the rationale of the SCAI CS classification and call for further validation in a prospective clinical trial.

DISCLOSURES
B.S. received honoraria from AstraZeneca; J.T.N. received honoraria from Siemens and Abbott Diagnostics; M.S. received honoraria from Abbott, Biotronik, Boston Scientific, Edwards Lifesciences, and Medtronic; H.R. received honoraria from Abiomed; S.B. received honoraria from Abbott, Siemens, Thermo Fisher, and Roche; D.W. received honorary from AstraZeneca, Bayer, Berlin-Chemie, and Novartis. The other authors report no conflict of interest.

ORCID
Benedikt Schrage https://orcid.org/0000-0001-9041-3922

REFERENCES
1. Thiele H, Ohman EM, de Waha-Thiele S, Zeymer U, Desch S. Management of cardiogenic shock complicating myocardial infarction: an update 2019. Eur Heart J. 2019;40(32):2671-2683.
2. Berg DD, Bohula EA, van Diepen S, et al. Epidemiology of Shock in contemporary cardiac intensive care units. Circ Cardiovasc Qual Outcomes. 2019;12(3):e005618.
3. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care
4. Neumann JT, Sorensen NA, Schwemer T, et al. Diagnosis of myocardial infarction using a high-sensitivity troponin I 1-hour algorithm. JAMA Cardiol. 2016;1(4):397-404.

5. Jentzer JC, van Diepen S, Barsness GW, et al. Cardiogenic Shock classification to predict mortality in the cardiac intensive care unit. J Am Coll Cardiol. 2019;74:2117-2128.

6. Thiele H, Zeymer U, Neumann FJ, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med. 2012;367(14):1287-1296.

7. Schrage B, Ibrahim K, Loehn T, et al. Impella support for acute myocardial infarction complicated by cardiogenic Shock. Circulation. 2019;139(10):1249-1258.

8. Udesen NJ, Moller JE, Lindholm MG, et al. Rationale and design of DanGer shock: Danish-German cardiogenic shock trial. Am Heart J. 2019;214:60-68.

9. Thiele H, Akin I, Sandri M, et al. PCI strategies in patients with acute myocardial infarction and cardiogenic shock. N Engl J Med. 2017;377(25):2419-2432.

10. Obling L, Frydland M, Hansen R, et al. Risk factors of late cardiogenic shock and mortality in ST-segment elevation myocardial infarction patients. Eur Heart J Acute Cardiovasc Care. 2018;7(1):7-15.

11. Basir MB, Schreiber TL, Grines CL, et al. Effect of early initiation of mechanical circulatory support on survival in cardiogenic shock. Am J Cardiol. 2017;119(6):845-851.

12. Masyuk M, Wernly B, Lichtauer M, et al. Prognostic relevance of serum lactate kinetics in critically ill patients. Intensive Care Med. 2019;45(1):55-61.

13. Poss J, Koster J, Fuernau G, et al. Risk stratification for patients in cardiogenic shock after acute myocardial infarction. J Am Coll Cardiol. 2017;69(15):1913-1920.

14. Schmidt M, Burrell A, Roberts L, et al. Predicting survival after ECMO for refractory cardiogenic shock: the survival after veno-arterial-ECMO (SAVE)-score. Eur Heart J. 2015;36(33):2246-2256.

15. Taleb I, Koliopoulos AG, Tandar A, et al. Shock team approach in refractory cardiogenic shock requiring short-term mechanical circulatory support: a proof of concept. Circulation. 2019;140(1):98-100.

16. Tehrani BN, Truesdell AG, Sherwood MW, et al. Standardized team-based care for cardiogenic shock. J Am Coll Cardiol. 2019;73(13):1659-1669.