SCAI cardiogenic shock classification after out of hospital cardiac arrest and association with outcome

Nilesh Pareek MA MBBS MRCP1,2 | Rafal Dworakowski MA MBBS MRCP1,2 | Ian Webb MA PhD FRCP1,2 | Jemma Barash Bsc MBBS1 | Gift Emezu MBBS1 | Narbeh Melikian Bsc MD FRCP1,2 | Jonathan Hill MA MBBS FRCP1,2 | Ajay Shah MD FRCP1,2 | Philip MacCarthy Bsc PhD FRCP1,2 | Jonathan Byrne Bsc PhD FRCP1,2

1Department of Cardiology, King’s College Hospital NHS Foundation Trust, London, UK
2School of Cardiovascular Medicine and Sciences, BHF Centre of Excellence, King’s College London, London, UK

Correspondence
Nilesh Pareek, School of Cardiovascular Medicine and Sciences, BHF Centre of Excellence, London, UK.
Email: nileshpareek@nhs.net

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Abstract
Objectives: We aimed to validate the Society for Cardiovascular Angiography and Interventions (SCAI) classification to evaluate association with outcome in a real-world population and effect of invasive therapies.

Background: Cardiogenic shock is common after Out of Hospital Cardiac Arrest (OOHCA) but is often multifactorial and challenging to stratify.

Methods: The SCAI shock grade was applied to an observational registry of OOHCA patients on admission to our center between 2012 and 2017. The primary end-point was 30-day mortality and secondary end-points were mode of death and 12-month mortality. Provision of early CAG and mechanical circulatory support (MCS) was evaluated by SCAI shock grade using logistic regression.

Results: Three hundred and ninety-three patients (median age 64.3 years (24.9% females) were included. One hundred and seven patients (27.2%) were in Grade A, 94 (23.9%) in Grade B, 66 (16.8%) in Grade C, 91 (23.2%) in Grade D, and 35 (8.9%) in Grade E. There was a step-wise significant increase in 30-day mortality with increasing shock grade (A 28.9% vs. B 33.0% vs. C 54.5% vs. D 59.3% vs. E 82.9%; p < .0001). With worsening shock grade, requirement for renal replacement therapy and mortality from multiorgan dysfunction syndrome and cardiogenic causes increased. Early CAG was performed equally in all groups but was significantly associated with reduced mortality in SCAI grade D only (OR 0.26 [CI 0.08–0.91], p = .036).

Conclusions: Increasing SCAI shock grade after OOHCA is associated with 30-day mortality, requirement for renal replacement therapy and mortality attributed to multiorgan dysfunction syndrome and cardiac etiology death.

Keywords
cardiac arrest, cardiogenic shock, SCAI classification

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1 | INTRODUCTION

Out of Hospital Cardiac Arrest (OOHCA) occurs in over a quarter of a million patients a year and presents a major public health challenge.\(^6\) Hemodynamic instability is present in approximately 50% of patients after OOHCA and several studies have shown a relationship between the development of shock and poor outcome after OOHCA, including neurological outcome.\(^2,3\) Clinical trials of cardiogenic shock (CS) have been composed of significant proportions of patients with OOHCA,\(^4,5\) but the pathophysiology of shock in these patients may represent a distinct clinical entity due to the presence of concomitant post-arrest myocardial stunning (PAMS) and systemic inflammatory response syndrome (SIRS). This is reflected in a heterogeneity of outcomes after OOHCA with mortality attributed to multi-organ dysfunction syndrome and cardiac etiology death in ~40% of patients only. This indicates a requirement for improved early classification of CS in patients with OOHCA which might support emergency selection of invasive therapies on arrival to a heart attack center, particularly revascularization and use of mechanical circulatory support (MCS) devices.\(^6\)

CS has classically been defined as a systolic blood pressure < 90 mmHg for >30 min without vasopressor support or requirement of these agents.\(^7\) The Society for Cardiovascular Angiography and Interventions (SCAI) have recently developed a novel CS classification to reflect a spectrum of shock which has been endorsed by multiple societies.\(^6\) In a recent validation of this classification in patients admitted to cardiac intensive care units, there was a signal that OOHCA significantly increases mortality across the spectrum of shock grades. However, in this study, the classification was applied a later time-point which would not guide early use of invasive therapies and also did not evaluate the relationship of SCAI grade with mode of death nor the relative effect of these invasive therapies on outcome.

Accordingly, the purpose of this study was to validate the SCAI shock classification in a cohort of primary cardiac etiology OOHCA particularly on admission to a Heart Attack Centre—atime-point that enables initiation of emergency therapies—and to evaluate the association with 30-day and 12-month mortality. In addition, we sought to characterize requirement for renal replacement therapy and mode of death by shock grade.

2 | METHODS

2.1 | Study setting

This study is a retrospective analysis of a prospectively collected and maintained registry of patients admitted to King’s College Hospital, London between May 1, 2012 to May 1, 2017 and were recruited into the King’s Out of Hospital Cardiac Arrest Registry (KOCAR). Across London, patients who experience OOHCA in the community are served by the London Ambulance Service (LAS), which is the largest emergency medical service in the United Kingdom. A standardized systematic protocol has been established whereby patients with OOHCA and return of spontaneous circulation (ROSC), along with ST segment elevation on the post-arrest ECG elevation, are taken directly to a cardiac center cardiac catheterization laboratory and all other OOHCA survivors are conveyed to the closest hospital emergency department. King’s College Hospital is the main cardiac arrest center in South East London, treating a population of over 1 million. The decision to perform coronary angiography (CAG) was made by the admitting interventional cardiologist and following initial treatment in the cardiac catheter laboratory, patients are then transferred to the Intensive Care Unit for ongoing supportive measures. The study was performed according the principles of Declaration of Helsinki and was approved by the local Research Ethics Committee.

2.2 | Study population

We included all patients over the age of 18 years who presented with OOHCA and had sustained ROSC in the field from May 1, 2012 to December 31, 2017. Inclusion criteria for the registry was all patients with ST elevation on ECG and for patients without ST elevation, if there was absence of a clear noncardiac etiology. We excluded patients with recognition of death before or on arrival to our center, evidence of an obvious noncardiac cause of arrest (suicide, trauma, drowning, substance overdose), patients with suspected or imaging confirmed intra-cerebral bleeding, known neurological disability (Cerebral Performance Category (CPC) 3 or 4 or any known survival limiting disease to 6 months preceding the cardiac arrest.

2.3 | Data collection

Data was collected using a dedicated database based on the Utstein style recommendations.\(^8,9\) Prehospital data was collected including initial rhythm, use of bystander cardiopulmonary resuscitation (CPR) and time of return of spontaneous circulation (ROSC). Zero-flow time was defined as time from the cardiac arrest to commencement of CPR and the low-flow time as duration of CPR until ROSC. Hospital data including arterial blood gas results such as pH and lactate and routine blood tests, which were all collected on admission were recorded. In addition, we recorded baseline cardiovascular investigations including electrocardiography, left ventricular ejection fraction and results of CAG where appropriate. Significant coronary artery disease was defined as a lesion of 70% severity in a single angiographic plane. A culprit lesion was classified as an atherothrombotic occlusion with presence of thrombus and/or easy passage of the coronary guidewire and any lesion over 70% in a single angiographic plane that the clinician treated as a culprit or with evidence of less than TIMI III flow. These definitions are in accordance with previously published articles in this area.\(^10,11\) MCS devices were implanted at clinicians’ discretion.
2.4 | SCAI shock classification

The SCAI grade is a consensus-based classification based on hemodynamic, metabolic, clinical, and physical examination parameters. Briefly, this classification is a pragmatic tool which classifies CS from A–E with “A" modifier signifying the occurrence of cardiac arrest—namely—At risk. Beginning Shock, Classic Shock, Deteriorating Shock and Extremis. In accordance with the classification, we defined the SCAI shock grades using objective markers of hemodynamics including blood pressure and heart rate, vasopressor requirements and presence of renal failure (study definition shown in Table 1). Patients were ascribed the SCAI shock classification retrospectively by an independent clinician who was blinded to patient outcome and mode of death and this was defined as the worst grade of shock within the first 1 hr of arrival to the cardiac catheterization laboratory.

2.5 | Outcome

The prespecified primary end-point was mortality at 30 days. The principal secondary end-point was mortality at 12 months. Additional secondary end-points were poor neurological outcome (defined as principal secondary end-point was mortality at 12 months. Additional end-points were poor neurological outcome (defined as neurological grade,13 multiorgan dysfunction syndrome,14 or cardiac etiology (refractory arrhythmia or refractory CS) at 12 months in accordance with previous studies. Follow-up was tracked using the medical records and by telephone contact and patients with incomplete data were excluded from the final analysis.

### TABLE 1

Study definitions of SCAI shock classifications

| SCAI stage | Study definition |
|------------|-----------------|
| Stage A “At risk” | OOHCA with no evidence of hemodynamic instability |
|            | Systolic BP > 90 mmHg and HR < 100 bpm with no vasopressor requirement |
| Stage B “Beginning” | OOHCA with relative hypotension or tachycardia without hypoperfusion |
|            | Systolic BP > 90 mmHg with HR > 100 bpm or low dose bolus vasopressor to maintain |
|            | BP > 90 mmHg with GFR > 60 mmHg |
| Stage C “Classic” | Patients with OOHCA and CS requiring intervention |
|            | Low dose bolus vasopressor to maintain |
|            | BP > 90 mmHg with GFR < 60 mmHg or one vasopressor infusion to maintain systolic |
|            | BP > 90 mmHg |
| Stage D “Deteriorating” | Patients with OOHCA and CS but deteriorating and failure to respond to initial interventions |
|            | Two vasopressor infusions to maintain systolic |
|            | BP > 90 mmHg |
| Stage E “Extremis” | Patients with overt or impending circulatory collapse |
|            | Failure to maintain systolic BP > 90 mmHg with two or more vasopressors |

Abbreviations: BP, blood pressure; HR, heart rate; OOHCA, Out of Hospital Cardiac Arrest; SCAI, Society for Cardiovascular Angiography and Interventions.

2.6 | Statistical analysis

Normally distributed data are represented as mean ± standard deviation and non-normally distributed data are presented as medians with ranges or interquartile range (IQR) as appropriate. Comparisons were performed with Student’s t test or ANOVA for continuous, normally distributed variables; Chi-square for proportions and Mann–Whitney for non-normally distributed variables. After stratification by baseline SCAI class, survival curves were constructed using the Kaplan–Meier method, and compared using a log rank test. Event times were measured from time of index cardiac arrest until the end-point. We constructed a multivariate logistic regression model with forced inclusion of variables independently associated with mortality in this population from existing literature (age, initial cardiac arrest rhythm, and lactate) as well as early coronary angiography based on SCAI grade. Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). A p < .05 was considered statistically significant. All analyses were undertaken using SPSS version 25.

3 | RESULTS

3.1 | Baseline characteristics

Between May 1, 2012 and December 31, 2017, 1,015 patients suffered OOHCA in the South London area, of whom, 251 failed to regain ROSC with emergency medical services. Of this cohort of survivors, 129 died before reaching KCH. Six hundred and thirty-five patients reached KCH with ROSC of whom 236 patients were deemed to not have a primary cardiac etiology cause of the cardiac arrest. After excluding 6 patients with incomplete data, 393 patients were included in the registry (Figure 1).

Baseline patient demographics are presented in Table 2. The median age was 64.3 years (IQR 21.6), the majority of patients were male (75.1%). Most patients had cardiac arrest at residence (n = 225, 57.3%), 283 patients (72.0%) were shockable rhythms and bystander CPR was performed in 288 (73.0%). Two hundred and thirty-three patients (59.2%) had ST elevation/LBBB on post-ROSC ECG. Early CAG was performed in 305 patients (77.6%) and percutaneous coronary intervention in 196/305 (64.2) patients. 30-day survival for the entire cohort was 183/393 (46.5%).

3.2 | Rates of cardiogenic shock by the SCAI classification

On arrival to our cardiac arrest center, 107 patients (27.2%) were in SCAI A, 94 (23.9%) in SCAI B, 66 (16.8%) in SCAI C, 91 (23.2%) in SCAI D and 35 (8.9%) in SCAI E. The classifications were well matched in terms on age and gender. However, patients with sicker SCAI grade had significantly lower rates of shockable rhythms, lower LVEF and pH and higher low flow times. Patients with higher shock grades also had higher lactate, creatinine and worse hemodynamics,
characterized by lower systolic and diastolic blood pressure and heart rate (Table 3).

### 3.3 SCAI shock classification and primary and secondary end-points

During a follow-up at 30 days, we observed a statistically significant difference between the event rate for mortality for the five SCAI shock grades (A 29.0% vs. B 33.0% vs. C 54.5% vs. D 59.3% vs. E 82.9%; \( p < .0001 \); Figure 2a). In a univariate logistic regression analysis, compared with SCAI grade A, 30-day mortality was significantly higher in SCAI grades C-E (SCAI C: OR 2.94, 95% CI 1.55–5.58; SCAI D: OR 3.58, 95% CI 1.98 to 6.46; SCAI E: OR 11.85, 95% CI 4.48–31.36 (all < 0.0001) but there was no difference between the SCAI grades A and B (OR 1.21, 95% CI 0.66–2.20).

During a follow-up of 12 months, we observed a similar trend with a statistically significant difference between the event rate for mortality for the five SCAI shock grades (A 30.8% vs. B 35.1% vs. C 59.1% vs. D 61.5% vs. E 85.7%; \( p < .0001 \)) and poor neurological outcome (CPC 3–5) (A 43.0% vs. B 42.6% vs. C 63.6% vs. D 69.6% vs. E 85.7%; \( p < .0001 \)) (Table 4 and Figure 2).

### 3.4 Association with mode of death and requirement for renal replacement therapy

While absolute mortality rate increased across the shock grades, we also observed a significant difference in relative mode of death between the different classes (Figure 3). Neurological etiology death was a relatively more prevalent mode of death in patients with less severe than in those with more severe shock (A 87.1% vs. B 75.0% vs. C 59.1% vs. D 61.5% vs. E 85.7%; \( p < .0001 \)) and poor neurological outcome (CPC 3–5) (A 43.0% vs. B 42.6% vs. C 63.6% vs. D 69.6% vs. E 85.7%; \( p < .0001 \)). Rates of cardiogenic etiology death also rose as shock grade increased (A 0%, B 6.2%, C 5.6%, D 11.1%)

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**TABLE 2** Baseline characteristics

| Variables                        | Total (n = 393) |
|----------------------------------|----------------|
| Age (yr)                         | 64.3 (21.6)    |
| Male—no./total no. (%)           | 296/393 (75.1) |
| CAD risk factors—no./total no. (%) |                |
| DM                               | 71/393 (18.1)  |
| Smoking                          | 234/393 (59.5) |
| ECG—no./total no. (%)            |                |
| ST elevation/LBBB                | 233 (58.8)     |
| ST depression                    | 41 (10.4)      |
| RBBB                             | 45 (11.4)      |
| Normal                           | 76 (19.2)      |
| LVEF (%)                         | 40 (20)        |
| CAG—no./total no. (%)            | 305/393 (77.6) |
| PCI—no./total no. (%)            | 196/305 (64.2) |
| Mechanical circulatory support—no./total no. (%) | 110/393 (28.0) |
| IABP                             | 102/110 (92.7) |
| Impella                          | 6/110 (5.4)    |
| ECMO                             | 2/110 (1.8)    |
| Residence—no./total no. (%)      | 225/393 (57.3) |
| Zero flow time (min)             | 4.1 (6.6)      |
| Low flow time (min)              | 27.2 (20.3)    |
| Witnessed—no./total no. (%)      | 315/393 (80.2) |
| Bystander CPR—no./total no. (%)  | 288/393 (73.3) |
| Shockable rhythm—no./total no. (%) | 283/393 (72.0) |
| Adrenaline—no./total no. (%)     | 265/392 (67.3) |
| Admission pH                     | 7.18 (0.20)    |
| Lactate (mmol/L)                 | 6.05 (4.6)     |
| Creatinine (micromol/L)          | 120.9 (80.1)   |

Abbreviations: CAD, coronary artery disease; DM, diabetes mellitus; ECMO, extra-corporeal membranous oxygenation; IABP, intra-aortic balloon pump; LBBB, left bundle branch block; LVEF, left ventricular ejection fraction; RBBB, right bundle branch block.
There was a significant relative increase in the use of advanced MCS (Impella and/or ECMO) with increasing SCAI severity (A 0% vs. B 3.4% vs. C 0% vs. D 6.5% vs. E 29.4%; \( p < .0001 \)). The use of MCS was not associated with improved survival at 30 days (53.6% vs. 53.8%; \( p = .531 \)) or at 12 months (51.8% vs. 51.0%; \( p = .490 \)) and no interaction between type of device, SCAI grade and the primary endpoint was observed.

3.6 | Clinical presentation and prognostic impact of early CAG based on SCAI classification

There was no significant difference in the rates of ST elevation (A 63.6% vs. B 59.6% vs. C 62.1% vs. D 46.7% vs. E 68.6%; \( p = .133 \)) by SCAI shock classification but there was increased provision of early CAG in patients with lower SCAI grade, particularly grades A – C (A 81.3% vs. B 81.9% vs. C 89.4% vs. D 63.8% vs. E 68.6%; \( p = .001 \)). However, patients with SCAI class E had higher rates of CAD (A 75.8% vs. B 77.9% vs. C 61.0% vs. D 75.9% vs. E 91.7%; \( p = .04 \)) but not multi-vessel CAD (A 33.3% vs. B 27.2% vs. C 30.5% vs. D 50.8% vs. E 54.2%; \( p = .592 \)). Rates of PCI and multi-vessel PCI also did not differ between the different SCAI shock grades (Table 5).

A multivariate regression analysis was performed for each SCAI classification after adjustment for age, initial shockable rhythm and lactate. In this analysis, early CAG was associated with improved 30-day mortality only in SCAI grade D (OR 0.26 [CI 0.08–0.91], \( p = .036 \)) (Table 5). By contrast, early CAG was not associated with improved survival in the other SCAI grades (A (OR 0.54 [CI 0.16–1.86], \( p = 0.331 \)); B (OR 1.31 [CI 0.33–5.31], \( p = 0.703 \)); C (OR 0.78 [CI 0.06–9.99], \( p = 0.30 \)); E (OR 0.397 [CI 0.29–5.40], \( p = 0.488 \)) (Table 6). We did find a significant interaction between performance of early CAG and initial rhythm in Grades A–C but not in Grades D–E (Table S1).

4 | DISCUSSION

In this study, we validated the SCAI shock classification in OOHCA patients on arrival to a specialist center and our principal findings are that, when applied at this early time-point, this grade is associated with mortality at 30 days and at 12 months. Higher SCAI shock grades were also associated with mortality attributed to multi-organ dysfunction syndrome and cardiac etiology death and requirement for renal replacement therapy. Finally, in a selected group of patients with CS and OOHCA, we found that early CAG might potentially be associated with improved outcome in SCAI grade D.

CS is observed in 40–70% of OOHCA patients and is associated with increased mortality.\(^4\,^5\) The classical definition of CS, which is used both in clinical trials is a systolic blood pressure < 90 mmHg or vasopressor requirement, associated with signs of impaired organ perfusion and a cardiac index <2.2 L/min/m\(^2\).\(^7\) Current guidelines...
recommend use of this simplistic definition for defining CS but this does not reflect the broad clinical spectrum of the condition after OOHCA, nor the complex interplay of contributory pathophysiological processes, such as the presence of PAMS and SIRS. For example, in our study, low-flow time and LVEF were significantly worse with a higher SCAI class—suggesting that PAMS might be a critical causal factor in the development of hemodynamic instability after OOHCA. Several shock grades have been developed but with only limited application in OOHCA and not with a robust evaluation of appropriate end-points such as mode of death. These include the INTERMACS grade, presence of heart failure, invasive hemodynamics (Diamond and Forrester classification), "warm or cold and dry and wet" or right

**TABLE 3** Baseline characteristics by SCAI shock classification

|                | Grade A | Grade B | Grade C | Grade D | Grade E | p    |
|----------------|---------|---------|---------|---------|---------|------|
| Male (%)       | 81 (75.7) | 75 (79.8) | 53 (80.3) | 59 (64.8) | 27 (77.1) | .122 |
| Age            | 61.0 (18.0) | 61.1 (24.0) | 68.5 (18.0) | 65.4 (19.7) | 68.8 (23.8) |      |
| Shockable      | 88 (82.2) | 73 (77.7) | 49 (74.2) | 54 (59.3) | 19 (54.3) | <.0001 |
| Witnessed      | 89 (83.2) | 76 (80.9) | 53 (80.3) | 72 (79.1) | 25 (71.4) | .67  |
| Zero flow      | 3.44 (4.8) | 2.88 (5.0) | 4.94 (5.8) | 4.86 (8.5) | 5.93 (9.8) | .332 |
| Low flow       | 23.0 (17.1) | 22.1 (18.3) | 28.0 (16.0) | 33.7 (23.3) | 41.1 (30.7) | <.0001 |
| ST elevation   | 68/107 (63.6) | 56 (59.6) | 41 (62.1) | 42 (66.2) | 24 (68.6) | .133 |
| LVEF (%)       | 43.8 (9.7) | 42.8 (9.7) | 40.2 (11.1) | 40.3 (11.1) | 36.2 (11.5) | .013 |
| Systolic BP (mmHg) | 122.2 (21.8) | 118.3 (28.2) | 100.9 (23.7) | 105.0 (32.6) | 102.7 (33.0) | <.0001 |
| Diastolic BP (mmHg) | 72.8 (16.5) | 70.4 (19.7) | 57.8 (17.1) | 62.1 (21.9) | 60.1 (21.5) | <.0001 |
| Heart rate (bpm) | 80.3 (14.4) | 96.9 (22.6) | 82.5 (18.7) | 102.0 (127.8) | 85.3 (23.6) | .044 |
| pH             | 7.24 (0.13) | 7.25 (0.13) | 7.19 (0.15) | 7.10 (0.25) | 7.02* (0.20) | <.0001 |
| Lactate        | 4.53 (3.11) | 4.62 (3.35) | 5.7 (3.76) | 7.39 (5.44) | 10.6 (5.97) | <.0001 |
| Creatinine     | 106.9 (56.7) | 94.7 (36.6) | 142.4 (54.3) | 147.5 (130.2) | 143.4 (86.2) | <.0001 |
| Early CAG      | 87/107 (81.3) | 77/94 (81.9) | 59/66 (89.4) | 58/91 (63.8) | 24/35 (68.6) | .01  |
| CAD            | 66/87 (75.8) | 60/77 (77.9) | 36/59 (61.0) | 44/58 (75.9) | 22/24 (91.7) | .04  |
| MV-CAD         | 29/87 (33.3) | 21/77 (27.2) | 18/59 (30.5) | 30/59 (50.8) | 13/24 (54.2) | .592 |
| Culprit        | 59/87 (67.8) | 62/94 (65.9) | 34/66 (51.5) | 38/58 (65.5) | 20/24 (83.3) | .287 |
| PCI            | 59/87 (67.8) | 51/77 (64.2) | 29/59 (49.1) | 35/58 (60.3) | 21/24 (87.5) | .001 |
| Multi-vessel PCI | 6/87 (6.9) | 7/77 (9.1) | 7/59 (11.9) | 7/58 (12.0) | 3/24 (12.5) | .12  |
| MCS            | 16/107 (15.0) | 27/94 (28.7) | 19/66 (28.7) | 31/91 (34.0) | 17/35 (48.6) | .001 |
| IABP           | 16/16 (100) | 26/27 (96.2) | 19/19 (100.0) | 29/31 (93.8) | 12/17 (70.6) |      |
| Impella        | 0/16 (0) | 0/27 (0) | 0/19 (0) | 2/31 (6.5) | 4/17 (23.5) |      |
| ECMO           | 0/16 (0) | 1/27 (3.4) | 0/19 (0) | 0/31 (0) | 1/17 (5.9) |      |

Abbreviations: BP, blood pressure; bpm, beats per minute; CAG, coronary angiography; CAD, coronary artery disease; ECMO, extra-corporeal membranous oxygenation; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; PCI, percutaneous coronary intervention.

**FIGURE 3** Relative mode of death by SCAI classification. There is a step-wise increase in deaths attributed to MODS and cardiac causes as SCAI shock increases. MODS, multiorgan dysfunction syndrome.
or left ventricular predominance. These classifications individually confer specific advantages but, to date, no classification has been developed specifically for patients with CS after OOHCA and the majority have significant limitations for applicability in an acute setting. While it is known that hemodynamic instability after OOHCA classically deteriorates over a 72 hr period after the event, we were most interested in application of the grade on arrival since, it is at this point, that a decision on an early invasive approach with MCS can be made.

The SCAI classification of shock is a consensus-based classification which was designed in order to be a pragmatic and practical tool that could be easily and specifically applied on arrival to center, partly to support selection of therapies. While developed by experts in the field and with endorsement of several societies, the classification is empirical and was not previously validated in a clinical cohort in an acute setting. A recent study by Jentzer et al retrospectively validated the SCAI classification in a large cohort of patients in the first 24 hr after admission to cardiac intensive care units and found a similar association with hospital mortality as our study. There was a signal from this study that the prognosis after OOHCA was particularly poor but the proportion of these patients in this registry was low, there was significant heterogeneity of etiologies and with no data on mode of death, which is an important consideration in this group of patients.

In our study, we found that the SCAI classification is easily applied on arrival to a specialist center with accurate reflection of the spectrum of CS after OOHCA, with association with early and late mortality as well as requirement for renal replacement therapy. The variation in 30-day mortality from SCAI A (28.9%) to SCAI E (82.9%) suggests that this classification is an accurate discriminator for different grades of shock. The findings that higher SCAI classes were associated with greater mortality from multi-organ dysfunction syndrome and cardiac etiology death after OOHCA gives an early indication that the SCAI classification might be used to for early selection of patients for targeted advanced MCS. Since IABP was the predominant modality of MCS in this study and known to not be of benefit in this situation, these findings might still legitimately support the use of the SCAI classification to guide provision of more advanced MCS in certain scenarios such as severe cardiogenic shock, although this hypothesis would certainly need validation in further studies.

The provision of early CAG and MCS in patients with OOHCA remains controversial. Current guidelines recommend that patients with ST elevation are conveyed directly to a cardiac catheterization laboratory but patients without ST elevation are only transferred for angiography after excluding noncardiac causes. There are limited randomized data to support this strategy, and the recently reported COACT trial showed no benefit for early CAG in patients without ST elevation though, in this study, patients were excluded if they had evidence of hemodynamic instability and rates of culprit coronary lesions were low (−13%). There was a signal from this study that the prognosis after OOHCA was particularly poor but the proportion of these patients in this registry was low, there was significant heterogeneity of etiologies and with no data on mode of death, which is an important consideration in this group of patients. In our study, we found that the SCAI classification is easily applied on arrival to a specialist center with accurate reflection of the spectrum of CS after OOHCA, with association with early and late mortality as well as requirement for renal replacement therapy. The variation in

### Table 4: Outcomes by shock classification

| Group | Mortality at 30 days | Mortality at 12 months | Poor neurological outcome at 12 months | p |
|-------|----------------------|-----------------------|---------------------------------------|---|
| A     | 31/107 (29.8)        | 33/107 (30.8)         | 46/107 (43.0%)                       | .0001 |
| B     | 31/94 (33.0)         | 33/94 (35.1)          | 40/94 (42.6%)                        | .0001 |
| C     | 36/66 (54.5)         | 39/66 (59.1)          | 42/66 (63.6%)                        | .0001 |
| D     | 54/91 (59.3)         | 56/91 (61.5)          | 64/91 (69.6%)                        | .0001 |
| E     | 29/35 (82.9)         | 30/35 (85.7)          | 30/35 (85.7%)                        | .0001 |

### Table 5: Outcomes of SCAI grades with and without early CAG

| Group | Early CAG | 30 day mortality | p |
|-------|-----------|------------------|---|
| A     | 87/107 (81%) | 20/87 (23%) | <.0001 |
| B     | 77/94 (78%) | 22/77 (29%) | <.0001 |
| C     | 59/66 (88%) | 30/59 (51%) | <.0001 |
| D     | 58/91 (64%) | 26/58 (45%) | <.0001 |
| E     | 24/35 (69%) | 19/24 (79%) | <.0001 |

Abbreviations: CAG, coronary angiography; MODS, multiorgan dysfunction syndrome; RRT, renal replacement therapy.
from early CAG because they exhibited features of more severe hemodynamic shock than Grades A–C—putting them at higher risk of MODS or cardiac etiology death—but without reaching the point of futility observed in patients in SCAI Grade E.

Using the currently accepted definition of CS, previous studies have recruited high numbers of patients with OOHCA who have high rates of neurological death, which may have attenuated benefit in patients receiving advanced MCS.24,25 In this registry, there were high rates of use of IABP across all shock grades but without any impact on outcome. While the rates of advanced MCS were generally low, which reflects a lack of consensus regarding appropriateness of use in this population, their use was significantly higher with more severe shock. With further robust validation, the SCAI classification might be incorporated into research studies and help to refine stratification of patients, finesse indications for treatment and to guide selection of therapies, including appropriate use of early CAG and advanced MCS in the future.

### 4.1 Limitations

This was a single center, retrospective study and while this has enabled collection of a rich dataset and a standardized protocol of care, it also introduces the possibility of bias. For example, the decision, or otherwise, to perform CAG and PCI were ultimately based on clinical discretion. Therefore, it cannot be excluded that any association of improved outcome with early CAG might be attributed to an element of patient selection and requires further validation. Second, the SCAI shock classification is practical and consensus-based and an individual patient might at one time possess specific characteristics of several grades. Hence, we sought to objectively define the shock grade using hemodynamic and vasopressor requirements only and the use of these variables did appear to correlate well with shock severity. Third, the rates of usage of advanced MCS (Impella and ECMO) were relatively low, which reflects absence of guideline support and current practice for OOHCA across many centers. These findings might not therefore reflect outcome where VA-ECMO and E-CPR for OOHCA is performed routinely.

Fourth, the numbers in this study were relatively small which reduced the power, so the findings should be viewed as hypothesis generating and require robust validation in larger prospective studies. Finally, the patients were selected on arrival to specialist heart attack center with access to 24/7 emergency angiography and MCS so the observed outcomes may not be applicable outside of this setting.

### 4.2 Conclusions

The SCAI shock classification can be applied on admission to patients with primary cardiac etiology OOHCA. Higher SCAI shock grades were associated with mortality, requirement for renal replacement therapy and mortality attributed to multi-organ dysfunction syndrome and cardiac etiology death. Patients in SCAI grade D demonstrated

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**TABLE 6** Multivariable logistic regression analysis for the primary end-point

| SCAI A | SCAI B | SCAI C | SCAI D | SCAI E |
|--------|--------|--------|--------|--------|
| OR (95% CI) | OR (95% CI) | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| p | p | p | p | p |
| Age (year) | 1.04 (1.00–1.08) | 1.06 (1.02–1.10) | 1.05 (0.99–1.11) | 1.03 (0.99–1.11) | 1.06 (0.99–1.11) |
| Initial nonshockable rhythm | 1.37 (1.19–1.56) | 1.39 (1.21–1.61) | 1.41 (1.23–1.61) | 1.43 (1.25–1.63) | 1.45 (1.27–1.65) |
| Lactate (mmol/L) | 0.96 (0.92–1.01) | 0.97 (0.93–1.02) | 0.98 (0.94–1.03) | 0.99 (0.95–1.03) | 1.00 (0.96–1.04) |
| Early invasive approach | 0.54 (0.34–0.85) | 0.53 (0.33–0.84) | 0.52 (0.32–0.82) | 0.51 (0.31–0.81) | 0.50 (0.30–0.80) |
benefit with early CAG and revascularization, data which are hypothesis-generating and merit further validation in a larger randomized study.

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**ORCID**

Nilesh Pareek  https://orcid.org/0000-0002-2720-2134

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SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section at the end of this article.

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