Received: 6 May 2020 | Accepted: 8 May 2020
DOI: 10.1002/ccd.28990

CORE CURRICULUM

WILEY

SCAI expert consensus statement on out of hospital cardiac arrest

Amir Lotfi MD, FSCAI[1] | Lloyd W. Klein MD, MSCAI[2] | Ravi S. Hira MD, FSCAI[3] | Jaya Mallidi MD, MHS[4] | Roxana Mehran MD, MSCAI[5] | John C. Messenger MD, FSCAI[6] | Duane S. Pinto MD, MPH, FSCAI[7] | Michael R. Mooney MD, FSCAI[8] | Tanveer Rab MD, FSCAI[9] | Demetri Yannopoulos MD, FSCAI[10] | Sean van Diepen MD, MSc[11]

[1]Division of Cardiology, Baystate Medical Center, Springfield, Massachusetts

[2]Division of Cardiology, University of California, San Francisco, California

[3]Division of Cardiology, University of Washington, Seattle, Washington

[4]Santa Rosa Memorial Hospital, St. Joseph Cardiology Medical Group, Santa Rosa, California

[5]Zena and Michael A. Wiener Cardiovascular Institute, Mount Sinai School of Medicine, New York, New York

[6]Division of Cardiology, University of Colorado School of Medicine, Aurora, Colorado

[7]Division of Cardiology, Beth Israel Deaconess Medical Center, Boston, Massachusetts

[8]Minneapolis Heart Institute, Minneapolis Heart Institute Foundation, Minneapolis, Minnesota

[9]Division of Cardiology, Emory University, Atlanta, Georgia

[10]Division of Cardiology, University of Minnesota, Minneapolis, Minnesota

[11]Department of Critical Care Medicine and Division of Cardiology, University of Alberta, Edmonton, Canada

Correspondence
Amir Lotfi, Baystate Medical Center, 759 Chestnut Street, Springfield, MA 01199.
Email: amir.lotfi@bhs.org

1 | INTRODUCTION

Out of hospital cardiac arrest (OHCA) remains a significant public health problem with high mortality and morbidity. (1) The true magnitude of mortality and morbidity from OHCA is unknown due to the lack of mandatory reporting, unified national surveillance systems, difficulty in accounting for cases not attended by emergency medical services (EMS), variability in existing reporting systems and paucity of data regarding long term neurological and functional outcomes (Appendix S1).[1]

Improving survival rates with good neurological outcomes among OHCA patients requires improved response times and quality of care in the "chain of survival" from early activation of EMS and resuscitation to advanced post admission care.[2] Given the high prevalence of coronary artery disease (CAD) as the cause for cardiac arrest in patients with a presenting rhythm of ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), interventional cardiologists are often consulted to consider emergent coronary angiography (angiography) and possible percutaneous coronary intervention (PCI) in OHCA patients. While emergent angiography and PCI are indicated in selected OHCA patients when the post resuscitation electrocardiogram (ECG) shows ST-segment elevation myocardial infraction (STEMI), there are significant institutional and individual variations in performance and timing for those patients without STEMI on post resuscitation ECG. The role of the cardiac catheterization laboratory encompasses not only angiography and PCI but also hemodynamic assessment and mechanical circulatory support (MCS) device use in patients with concomitant cardiogenic shock (CS). The purpose of this document is to provide an evidence-based and patient-oriented recommendation for the management of these patients.

2 | METHODS

This document has been developed according to SCAI Publications Committee policies for writing group composition, disclosure and management of relationships with industry (RWI), internal and external review, and organizational approval.

The writing group has been organized to ensure diversity of perspectives and demographics, multistakeholder representation, and appropriate balance of RWI. Relevant author disclosures are included in Table S1. Before appointment, members of the writing group were asked to disclose all relevant financial relationships with industry (>$25,000) from the 12 months prior to their nomination. A majority of the writing group disclosed no relevant financial relationships. Disclosures were periodically reviewed during document development and updated as needed. SCAI policy requires that writing group members with a current financial interest are recused from participating in discussions or voting on relevant recommendations. The work of the writing committee was supported exclusively by SCAI, a nonprofit medical specialty society, without commercial support. Writing group members contributed to this effort on a volunteer basis and did not receive payment from SCAI.

Literature searches were performed by group members designated to lead each section and initial section drafts were authored by the section leads. Recommendations were discussed by the full writing group on a series of teleconferences until all group members agreed on the text and qualifying remarks. All recommendations are supported by a short summary of the evidence or specific rationale.

The draft manuscript was posted for public comment in February 2020 and the document was revised to address pertinent comments. The writing group unanimously approved the final version of the document. The SCAI Publications Committee and Executive Committee endorsed the document as official society guidance in May 2020.

SCAI consensus statements are primarily intended to help clinicians make decisions about treatment options. Clinicians also must consider the clinical presentation, setting, and preferences of individual patients to make judgements about optimal approaches.

2.1 | Invasive coronary angiography strategies in resuscitated OHCA patients

Table 1 summarizes the study characteristics of large studies combining all OHCA patients that compared immediate or early coronary angiography with no or delayed coronary angiography. Table 2 summarizes recently published and ongoing randomized control trials (RCTs). The literature guiding the role and timing of angiography and PCI in resuscitated OHCA patients is predominantly limited to observational studies. (Appendix S2).[3]

2.2 | Etiology of death in patients with OHCA in the critical care unit

Irrespective of initial rhythm or ECG findings, the predominant cause of death in two-thirds of patients with OHCA is due to anoxic brain injury and another one-third is due to a refractory post arrest shock and multi-organ failure.[4] The rate of survival to discharge with good neurologic function among OHCA patients is low with wide geographical variation estimated at 0.8–20%.[5] Anoxic brain injury and postcardiac arrest shock share common risk factors that are related to the timing and quality of pre hospital care.[6]

2.3 | Factors associated with unfavorable neurological outcome in OHCA patients

Among OHCA patients who are comatose after return of spontaneous circulation (ROSC), there is no single factor at the time of presentation to reliably prognosticate subsequent neurological outcome. While accurate prognostication is important to avoid pursuing futile treatments or inappropriately withdrawing treatment in patients with a chance of recovery, the quality of existing neurological prognostication studies is insufficient to make any definitive recommendations for long term neurologic outcomes.[7] Nevertheless, there are pre and intra arrest factors associated with unfavorable neurological outcomes (Table 3).[8,9] When deciding whether to offer invasive treatments, it is prudent to consider presence of co-morbidities that portend unfavorable short- and long-term prognoses, such as advanced age, severe dementia, chronic advanced respiratory failure, severe frailty or disability, end stage renal or liver disease, and advanced metastatic malignancy.[10]

2.4 | Risk stratification tools

Risk stratification scores are often used in medicine for prognostication and decision-making.[11] The Cardiac Arrest Hospital Prognosis score (CAHP),[12] CREST, and[13] C-GRA*p*H are risk stratification tools to assist in decision-making.[14] (Table 4).

In addition to risk stratification tools, imaging tools are available to provide further information regarding clinical assessment and management.[15,16] (Table 5).

3 | DECISION MAKING BASED ON SITUATIONAL AWARENESS AND ASSESSMENT ALONG THE CONTINUUM OF CARE

Given the heterogeneous nature of OHCA patients, fluidity in evolution of the clinical course, and uncertainty associated with neurological prognostication, we advocate a path of "*Situational Awareness and Assessment*"[17] taking into consideration all the clinical factors available to aid in clinical decision-making along the continuum of care of these patients (Figure 1).[17] We advocate confirming all the prehospital and hospital clinical history and data, considering carefully patient's comorbidities, patient's and family wishes if known as well as all of the favorable and unfavorable prognostic factors, synthesizing the data to provide the best possible prediction regarding the etiology of the

TABLE 1 Large observational studies comparing immediate/early angiography versus delayed or no angiography in OHCA patients

First author, year (Ref)	Design, country of recruitment	Total Patients	Patients with TTM (%)	Immediate/early angiography group							Delayed/no angiography group						Favors early angiography	Comments
				N	Time of angiography (hr)	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)	N	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)		
Nielsen N, 2009[39]	Prospective, Observational Cohort Study, Multicenter, USA and Europe	986	986 (100)	479	0	479 (100)	NA	299 (62)	303 (63)	278 (58)	507	207 (40)	NA	NA	253 (49)	3	YES HR – 1.27, 95% CI – 1.13 – 1.42)	Unwitnessed arrest, non-shockable rhythm, delayed time to achieve ROSC were predictors of poor outcomes.
Callaway CW, 2014[33]	Post analysis of Randomized T, Multicenter USA and Canada	3,981	1,566 (39)	765	0–24	591 (77)	356 (46)	705 (92)	495 (64)	413 (54)	3,216	1,028 (32)	217 (6)	NA	871 (27)	591 (18)	YES (OR:1.69–95% CI 1.06–2.7)	Patients in early angiography group were highly selected—19% of all pts - had higher rate of VT/VF; Bystander CPR, and STEMI on EKG
Reynolds JC, 2014[40]	Single Center Retrospective Cohort, UPMC, USA	1,011	559 (55)	273	0	196 (72)	145 (53)	152 (56)	NA	128 (47)	738	187 (25)	20 (3)	15 (21)	NA	155 (21)	YES OR: 1.92, (95% CI – 1.2, 3.07)	Patient who got early angiography—Selective – 27%: Higher rates of VT/VF; STEMI; with less illness severity score. In more severe strata of illness, early ANGIOGRAPHY was not beneficial
Geri G, 2015[35]	Prospective, Observational Cohort Study, France	1,722	1,222 (71)	1,094	0–6	766 (70)	295 (27)	479 (44)	419 (38)	NA	628	175 (28)	23 (4)	NA	129 (21)	NA	YES HR – 1.86, (95% CI 1.57–2.21)	Patient with early angiography—higher rate of Vt/VF, STEMI, witnessed arrest, bystander CPR and therapeutic hypothermia

TABLE 1 (Continued)

First author, year (Ref)	Design, country of recruitment	Total Patients	Patients with TTM (%)	Immediate/early angiography group: N	Time of angiography (hr)	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)	Delayed/no angiography group: N	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)	Favors early angiography	Comments
Kern KB, 2015[21]	Retrospective and prospective cohort study, USA and Europe	754	738 (98)	439	0	328 (75)	192 (44)	209 (48)	272 (62)	250 (57)	315	109 (35)	6 (2)	NA	63 (20)	49 (16)	YES HR – 1.65 (95% CI – 1.40 – 1.95)	Patients chosen for early angiography – higher rate of STEMI
Vyas A, 2015[43]	Prospective, Multicenter, Observational Cohort Registry Data, USA	4,029	2,363 (59)	1953	0–24	1953 (100)	639 (33)	1,253 (64)	1,484 (76)	1,147 (59)	2076	2076 (100)	163 (8)	NA	1,234 (59)	821 (40)	YES OR – 1.52, 95% CI – 1.28 – 1.80	All are shockable rhythms. Patients chosen for early angiography were younger, had witnessed arrest and STEMI on EKG.
Dankiewicz J, 2015[34]	Post hoc analysis of RCT, Europe and Australia	544	544 (100)	252	0–6	201 (80)	0	101 (40)	130 (52)	NA	292	207 (71)	0	25 (27)	133 (46)	NA	NO HR – 0.82, 95% CI – 0.64 – 1.03	N0 STEMI Patients. Factors associated with favorable prognosis: Younger age, initial shockable rhythm, less time to ROSC, no cardiogenic shock
Vandeboncoeur TF, 2018[42]	Prospective, Multicenter Observational Cohort, Arizona, USA	1881	655 (35)	1,007	0–24	729 (72)	450 (45)	482 (48)	728 (72)	654 (65)	874	209 (24)	49 (5)	NA	464 (53)	268 (31)	YES HR –2.10, 95% CI – 1.3 – 3.5	Propensity score analysis. Patients with early angiography had higher rates of witnessed arrest, initial shockable rhythm and TTM
Jaeger D, 2018[36]	Prospective, Multi-center Observational Cohort, Registry Data, France	7,584	2,599 (34)	2,958	0(Admission to cath lab)	932 (32)	NA	NA	1,327 (45)	1,085 (37)	4,626	858 (19)	NA	NA	1,116 (24)	856 (19)	YES OR 0.66, 95% CI 0.58,0.75)	Propensity matched analysis. Significantly higher proportion of patients in early angiography group got TTM

(Continues)

TABLE 1 (Continued)

				Immediate/early angiography group							Delayed/no angiography group							
First author, year (Ref)	Design, country of recruitment	Total Patients	Patients with TTM (%)	N	Time of angiography (hr)	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)	N	VT/VF (% of N)	STE (% of N)	PCI (% of N)	Survival (% of N)	Survival with good CPC (% of N)	Favors early angiography	Comments
Jentzer JC, 2018[37]	Prospective, 2 center Registry Data, USA	599	413 (69)	283	0–24	197 (70)	126 (45)	151 (53)	159 (56)	80 (28)	316	106 (34)	12 (4)	NA	98 (31)	35 (11)	YES OR – 2.85, 95% CI. – 2.04 – 4.0)	Patients selected for early angiography had higher percentage of VT/VF, ST elevation and bystander CPR, and lower severity illness
Staudacher II, 2018[41]	Retrospective, Single Center Cohort Study, Denmark	507	507 (100)	291	0–3	NA	181 (62)	208 (71)	207 (71)	NA	216	NA	39 (18)	49 (23)	137 (63)	NA	NO HR – 0.69, 95% CI – 0.35 – 1.37	Propensity matched analysis. Early angiography/PCI not associated with mortality benefit with or without ST elevation. Predictors of 30 day mortality—Older age, cardiogenic shock, elevated lactate and low hemoglobin at presentation. Significantly higher proportion of shock patients in early invasive group

TABLE 2 Recently published/ongoing randomized trials of immediate versus delayed angiography in OHCA patients without STEMI on ECG at presentation

Name of study	Clinical trials identifier	Design, country of recruitment	Inclusion/exclusion criteria	Total number of patients	Primary outcome	Groups	Results
COACT, 2019[22]	NTR 4973	Open Label, Multicenter, Netherlands	Inc: Comatose OHCA patients + Initial Shockable Rhythm + No STEMI of ECG Ex: Shock/obvious noncardiac cause	538	Survival at 90 days	Immediate: Upon presentation Delayed: After neurological recovery	No difference
ACCESS	NCT03119571	Open Label, Multicenter, United States	Inc: Comatose OHCA patients + Initial Shockable Rhythm + No STEMI on ECG Ex: STEMI	864	Survival to Hospital Discharge with good neurological outcome	Immediate: Admission to Cath Lab Delayed: Admission to ICU for further assessment	To be published
DISCO	NCT02309151	Open Label, Multicenter, Sweden	Inc: Witnessed OHCA with achieving ROSC >20 min Exc: STEMI/obvious noncardiac cause	1,006	Survival at 30 days	Immediate: Within 120 min Not Immediate: No angiography or after 3 days	To be published
PEARL	NCT02387398	Open Label, Multicenter, United States	Inc: Comatose OHCA patients with No STEMI Ex: STEMI/obvious noncardiac etiology	99	Safety and Efficacy of early angiography	Interventional Early angiography: Within 120 min Control group: After 6 hr	To be published
EMERGE	NCT02876458	Open Label, Multicenter, France	Inc: Comatose OHCA patients Ex: STEMI on ECG/obvious noncardiac etiology	970	Survival with no or minimal neurological sequelae at 180 days	Immediate: Upon presentation Delayed: 46–96 hr	To be published
TOMAHAWK	NCT02750462	Open Label, Multicenter, Germany	Inc: Comatose OHCA patients Ex: STEMI/ Hemodynamic Instablity	558	All cause mortality at 30 days	Immediate: Upon presentation Delayed: After 24 hr/ neurological recovery	To be published
COUPE	NCT 02641626	Open Label, Multicenter, Spain	Inc: Comatose OHCA patients Ex: STEMI/ Hemodynamic Instability	166	Survival with good neurological outcome at 30 days	Urgent: Upon presentations Deferred: After neurological Recovery	To be published

arrest and anticipated neurological and hemodynamic outcomes to help guide the role and timing of invasive strategy in these patients (Figure 1).

We also support the use of terminology such as activation of cardiac cath Lab (CCL) rather than angiography as it encompasses other invasive procedures (angiography, PCI, Right Heart Cath, MCS, and others) that may need to be performed in this patient population. We recommend use of terminology—"Definite" or "Defer" activation of CCL along a decision-making continuum at initial and subsequent encounters based on clinical history, presence of favorable/unfavorable resuscitation factors, initial rhythm, ECG and hemodynamic status (Figure 1). This terminology is subsequently used in all recommendations in this document (Table 6).

3.1 | OHCA patients with shockable rhythm and STEMI on post ROSC ECG

Among patients with an initial shockable rhythm and diagnostic ST-segment elevations on post ROSC ECG, the prevalence of acute thrombotic coronary occlusion or culprit lesion causing cardiac arrest is greater than 85%.[15] Given this observation, conscious survivors of OHCA at presentation with initial ECG showing STEMI should be treated with immediate angiography and primary PCI as is the current standard of care for STEMI patients.[18] Among comatose OHCA patients with ROSC and STEMI, there are no RCTs to support favorable neurological outcomes or survival benefit of immediate angiography. In the targeted temperature management (TTM) trial, which evaluated 33 versus 36°C after cardiac arrest, 41% of the 939 patients had acute STEMI on their initial ECG. In this study, lower time to ROSC was the highest predictor of survival with good neurological function.[19,20] In the International Cardiac Arrest (INTCAR) Registry, among 746 comatose post-arrest (79% OHCA) patients, 26.5% presented with STEMI and 73.5% without STEMI on their initial ECG; 91% of STEMI patients had immediate angiography whereas 33% patients without STEMI had an immediate angiography.[21] The survival rate was 55.1% in the STEMI group versus 41.3% in the patients without STEMI on their initial ECG. The decision to perform immediate angiography was based on operator preference rather than a predefined protocol targeting certain selected patients who might benefit from immediate angiography. Therefore, the ECG should not be the sole determinant when activating the CCL and instead should be paired with the clinical presentation and the patient's clinical findings when considering invasive assessment and potential therapies.

3.2 | OHCA patients with shockable rhythm without STEMI on post ROSC ECG

Among OHCA patients with an initial shockable rhythm without STEMI on post-ROSC ECG, the prevalence of an acute thrombotic occlusion is ~3.4–30%.[21,22] The prevalence of significant, stable or thrombotic, non-occlusive lesions on angiography ranges from 24 to 60%.[23,24] While ECG and biomarkers are used in patients with acute coronary syndrome without cardiac arrest, these lack specificity or sensitivity in predicting coronary ischemia as the cause of initial or recurrent cardiac arrest in these patients.[25] When ST elevation is not present, current noninvasive methods lack sensitivity to definitely assess ongoing coronary ischemia and tools to prognosticate neurological outcomes at presentation in comatose patients are inadequate as mentioned. As such, the decision of if and when to activate the CCL in these patients is challenging.

The existing literature on evaluating the benefit of angiography in these patients is predominantly from observational cohort studies (Table 1) with several limitations (Appendix S2). A meta-analysis of 23 observational studies showed that angiography performed within 24 hr was associated with improved survival (Risk Ratio:1.52, 95% CI: 1.32–1.74, $p < .001$) and better neurological outcomes (Risk ratio: 1.69, 95% CI: 1.40–2.04, $p < .001$) compared with angiography performed more than 24 hr later or not at all.[26]

The only published randomized trial, Coronary Angiography after Cardiac Arrest Trial (COACT), involving 538 patients showed no survival benefit at 90 days for immediate or early angiography in hemodynamically stable OHCA patients without STEMI on ECG compared with delayed angiography after neurological recovery.[22] Patients with CS unresponsive to medical therapy, obvious or suspected non-coronary cause of arrest, and STEMI on the ECG were excluded in this trial. TTM was initiated in more than 90% of patients and 87% of patients received norepinephrine. Neurological etiologies were the cause of death in more than 70% of the patients in both groups. In the delayed-angiography group, 14.4% of the patients underwent urgent coronary angiography because of CS, recurrent ventricular arrhythmia, or recurrence of ischemia. In 39.5% of these patients, an unstable lesion was detected, a percentage that was higher than that in the immediate-angiography group (13.6%). PCI was performed in 22 of these patients (57.9%), which is higher than the percentage of patients in the immediate-angiography group (33%), but the rate of survival among these patients was not lower than that in the total cohort (71.1 and 65.4%, respectively).[27]

TABLE 3 Factors associated with survival with neurological outcome in patients without of hospital cardiac arrest

Factors	Comments
Age	Each decade of life was significantly associated with a 21% decrease in the odds of a favorable neurological outcome.[92]
Witnessed arrest	In a meta- analysis of studies reporting the predictors of survival from OHCA, the survival to hospital discharge among patients with witnessed arrest was significantly higher at 13.5% compared to 6.4% in those with an unwitnessed arrest.[9]
Bystander CPR	In the CARES registry[93] bystander CPR was initiated in 46.9% of all witnessed events and survival to discharge rate was significantly higher in these patients at 13.7% compared to 7.5% in those without bystander CPR
Achievement of ROSC <30 min	A study of 227 patients with OHCA admitted to intensive care unit showed that mean time to achieve ROSC was significantly lower at 18.3 ± 15.1 min for patients who had a CPC score of 1 or 2 at discharge compared to 48.6 ± 17.9 min for those with unfavorable neurological outcomes (CPC) score of 3,4,5[94] Even among those treated with TTM, a shorter time interval from collapse to ROSC was strongly associated with improved neurological outcomes.
Shockable rhythm	In the 2017 CARES report,[91] patients with an initial VF/p VT had significantly higher rate of survival to discharge at 29.1% compared with 10.1% for patients with PEA and 2.4% for patients with asystole.
Lactate	There is strong correlation between elevated lactate level of greater than 7 mmoL/L and a corresponding pH of less than 7.2 to be strongly associated with multiorgan failure, severe anoxic brain injury, and increased mortality.[85]

3.3 | OHCA patients with non-shockable rhythms

While the prevalence of obstructive CAD in patients resuscitated from shockable rhythms including VT or VF, ranges from 25 to 60%,[28-30] the prevalence of CAD in OHCA patients with initial non-shockable rhythms, asystole, or pulseless electrical activity (PEA), is

TABLE 4 Out of hospital cardiac arrest risk scores

Study	Cohort	Variables	Outcomes	Validation
Cardiac Arrest Hospital Prognosis (CAHP)[12]	1,410 patients 41% with post ROSC STEMI of ECG 69% male	Age as continuous variable Non-shockable rhythm Time from collapse to BLS Time from BLS to ROSC Location of cardiac arrest Epinephrine dose Arterial pH	CAHP <150 86% had early invasive strategy 61% discharged alive from hospital with 95% with CPC score 1 or 2 CAHP 150–200 66% had early invasive strategy 10% discharged alive from hospital with 88% with CPC score 1 or 2 CAHP >200 47% had early invasive strategy 3% discharged alive from hospital with 86% with CPC score 1 or 2	C-statistic reached 0.93 (95% CI: 0.91 to 0.95) in the development cohort, and 0.91 (95% CI: 0.88 to 0.93) in the prospective validation cohort
CREST Model[13]	638 patients derivation 318 patients Validation No patients with STEMI 18.9% recognized identified as circulatory cause of death	History of coronary artery disease Non-shockable rhythm Ejection fraction <30% at time of admission Shock at the time of admission Ischemic time > 25 min	CREST score death due to shock 0–7.1% 1–9.5% 2–22.5% 3–32.4% 4–20% 5–50%	Area under the curve (AUC) 0.68 in the validation cohort
C-GRA*p*H[14]	122 patients derivation 344 patients validation	History of coronary artery disease Glucose ≥200 mg/dL Non-shockable rhythm Age > 45 *p*H (arterial) ≤ 7.0	C-GRA*p*H (0–1) 70% with CPC score of 1 or 2 C-GRA*p*H (4, 5) 98% with CPC score 3 to 5	AUC of 0.814 in the validation cohort with a c-statistics of 0.81

Note: ROSC return of spontaneous circulation; STEMI ST segment elevation myocardial elevation; BLS basic life support; CPC cerebral performance categories (Appendix S4); CI Confidence interval.

TABLE 5 Imaging for risk stratification of patients without of hospital cardiac arrest

Imaging modality	Evaluation
Computed topography of head	Anoxic encephalopathy Intracranial bleeding Subarachnoid hemorrhage
Magnetic resonance imaging of brain	Anoxo-ischemic brain injury
Echocardiogram	Post myocardial infarction complications • Free wall rupture • Ventricular septal defect • Mitral valve papillary/chordae dysfunction/rupturePulmonary embolus Pericardial tamponade/effusions Valvular dysfunction Regional wall motion abnormalities Hypertrophic cardiomyopathy

not well-defined. In OHCA patients presenting with non-shockable rhythms, noncardiac etiologies of OHCA often need to be considered.[31,32] The overall survival and favorable prognosis is significantly higher with initial shockable rhythm compared to a non-shockable rhythm.[21,33-44] Primary PEA and asystole are more often due to non-coronary etiologies especially in the elderly with multiple co morbidities.[45] It is also possible that some patients with initial non-shockable rhythms may develop VF/VT with administration of epinephrine during resuscitation, and they should still be treated as OHCA with non-shockable rhythm.[46]

4 | ACCESS AND PERI-PROCEDURAL ANTITHOMBOTIC THERAPY DURING PCI IN OHCA PATIENTS

Patients with OHCA are at higher risk of bleeding when undergoing PCI due to injuries sustained as a result of loss of consciousness and

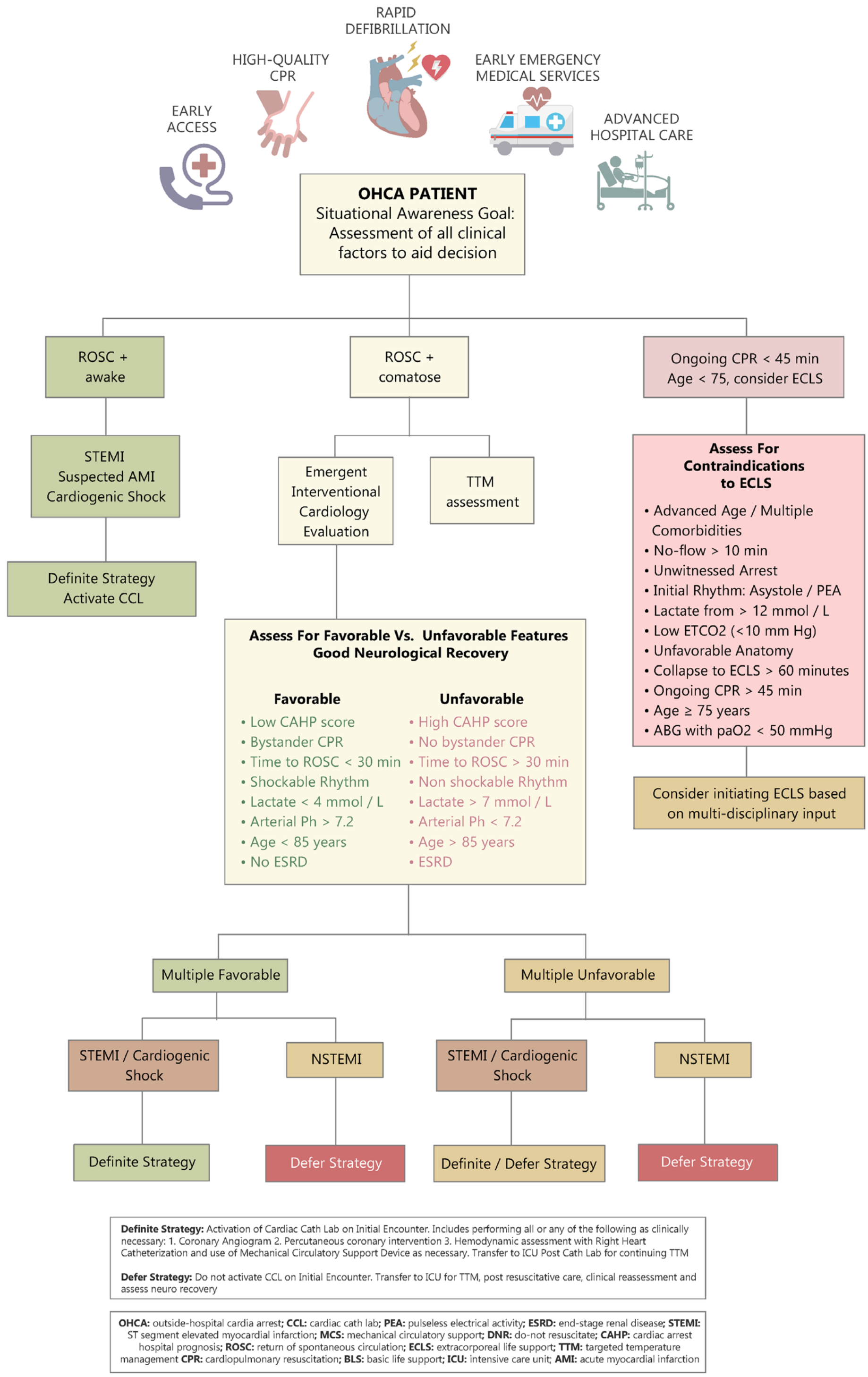

FIGURE 1 Algorithm of clinical factors available to aid in decision-making along the continuum of care of patients with out-of-hospital cardiac arrest [Color figure can be viewed at wileyonlinelibrary.com]

often there is concern for intracranial bleeding.[47] These patients are also at higher risk of thrombotic complications because of potentially delayed bioavailability of oral antiplatelet agents either due to active vomiting or reduced absorption from the gut in the setting of low cardiac output state with concomitant CS.[48] The pro-inflammatory state resulting in endogenous changes in coagulation and anticoagulation cascades as part of postresuscitative state, multiorgan dysfunction and therapeutic hypothermia increases the risk of both bleeding and thrombotic complications in OHCA patients.[49]

4.1 | Vascular access

Radial access is associated with reduced risk of bleeding complications compared with femoral access among patients undergoing PCI for ACS.[50] However, in a large RCT and a retrospective analysis, there was no difference in the primary outcome, including mortality, among patients undergoing primary PCI between radial and femoral access.[51,52] In the setting of OHCA, especially if there is concomitant shock, traditionally the femoral access site has been preferred—due to

TABLE 6 Consensus statement on out of hospital cardiac arrest recommendations

Situational awareness in patients with OHCA	In all comatose OHCA patients, we recommend dynamic clinical decision-making of "definite" or "defer" transport to CCL based on situational awareness and assessment involving all clinical factors along the entire continuum of care.
OHCA patients with non-shockable rhythms	In OHCA patients with initial non-shockable rhythm, we recommended deferring transport to CCL at initial encounter
OHCA patients with shockable rhythm and STEMI on Post ROSC ECG	In selected comatose OHCA patients with ROSC exhibiting STEMI on ECG we recommend a definite invasive strategy.
OHCA patients with shockable rhythm without STEMI on Post ROSC ECG	We recommend deferring invasive strategy at initial encounter in hemodynamically stable, comatose OHCA patients without STEMI on post ROSC ECG.
Access for intervention	In OHCA patients undergoing PCI, we recommend choosing the access site as per the operator's expertise and local standard catheterization lab protocols. For both routine femoral access and large bore access in case of hemodynamic support in patients with concomitant shock, we recommend the safe access site practices to reduce the risk of bleeding.
Antiplatelet therapy	We recommend ticagrelor or prasugrel as the preferred P2Y12 inhibitor in OHCA patients undergoing PCI.
Anticoagulation therapy	Among OHCA undergoing PCI we recommend the use of unfractionated heparin with monitoring as the peri-procedural anticoagulant given the availability of a reversal agent in cases of life threatening bleeding and reduced risk of acute stent thrombosis compared to bivalirudin.
Target temperature management (TTM)	We recommend against the use of prehospital TTM using cold intravenous crystalloids. We recommend initiating TTM inpatient as soon as possible.
Barriers and Public Reporting	SCAI advocates making OHCA exclusion based on exceptional risk from public reporting analysis of PCI outcomes. The principle to be followed is that "Public reporting of outcomes in high-risk patients, if done at all, should accurately reflect the performance of those operators and institutions." Additionally, SCAI recommends continuing to track process measures and outcomes in all patients suffering OHCA, including early access to coronary angiography and use of PCI.

Abbreviations: CCL, Cardiac catheterization laboratory; ECG, electrocardiogram; OHCA, out of hospital cardiac arrest; PCI, percutaneous coronary intervention; ROSC, return of spontaneous circulation; SCAI, society of coronary angiography and intervention; STEMI, ST-segment elevation myocardial infarction.

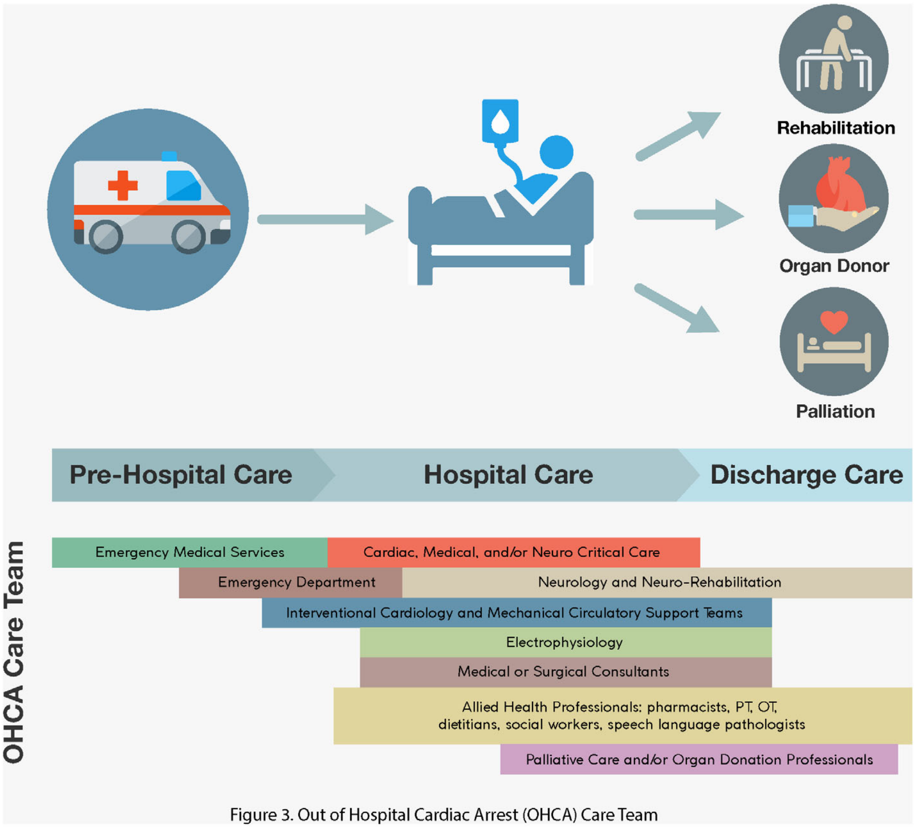

FIGURE 2 Out of hospital cardiac arrest (OHCA) care team [Color figure can be viewed at wileyonlinelibrary.com]

TABLE 7 Studies showing mild therapeutic hypothermia improves neurologic outcomes in comatose OHCA patients with shockable rhythm

First author, year (Ref)	Design, country of recruitment	Total number of patients	Inclusion/exclusion criteria	Target temperature protocol	Outcomes
Holzer M, 2002[66]	Blinded, Randomized, Multicenter Trial in 5 European Countries (Austria, Italy, Belgium, Finland, Germany)	275 Hypothermia: 136 Normothermia: 137	Inc: > 18 to <75 years; Witnessed cardiac arrest, ventricular fibrillation or non-perfusing ventricular tachycardia as the initial cardiac rhythm, presumed cardiac origin of the arrest, estimated interval of 5–15 min from the patients collapse to first attempt at resuscitation, No more than 60 min from collapse to ROSC Ex: Temperature below 30^0 C on admission, comatose state due to administration of drugs, pregnancy, response to verbal commands after ROSC, evidence of hypotension for more than 30 min after ROSC, evidence of hypoxemia for more than 15 min after ROSC, terminal illness that preceded arrest, pre-existing coagulopathy.	Target Temperature of 32 and 34°C was maintained for 24 hr from the start of cooling, followed by passive rewarming to a temperature above 36°C over a period of 8 hr	Favorable neurological outcomes was significantly higher in hypothermia group (55 vs. 39%). Mortality at six months was significantly lower in hypothermia group (41 vs. 55%)
Bernard SA, 2002[65]	Open labeled, Randomized, Multicenter Trial, Melbourne, Australia	77 Hypothermia: 43 Normothermia: 34	Inc: Ventricular fibrillation, Persistent coma after ROSC Ex: <18 years for men; < 50 years for women (because of possibility of pregnancy), cardiogenic shock, possible causes of shock other than cardiac arrest	Basic cooling initiated in ambulance. Core temperature of 33°C was maintained for at least 12 hr after arrival to hospital. Beginning at 18 hr, active rewarming was done for next 6 hr.	Favorable neurologic outcomes were significantly higher in hypothermia group (49 vs. 26%) There was no statistically significant mortality difference between hypothermia (51%) and normothermia (68%)

concerns of vasospasm, increasing procedural time to reperfusion and convenience of restricting the working field to groin area if hemodynamic support is required.

4.2 | Antiplatelet therapy

Among comatose survivors of OHCA undergoing PCI, post-resuscitative state and hypothermia may affect the absorption and metabolism of oral antiplatelets agents.[48] Some studies have shown an increased rate of stent thrombosis among post OHCA patients with hypothermia.[53] As demonstrated in patients with STEMI without cardiac arrest, the newer oral antiplatelet agents (ticagrelor and prasugrel) in addition to aspirin may be associated with reduced risk of ST even among comatose OHCA patients and are favored over clopidogrel.[54-57] In patients with a large thrombus burden, bolus doses of glycoprotein IIb/IIIa inhibitors may be considered after carefully weighing against the increased risk of bleeding.[58] Cangrelor, an intravenous P2 Y12 inhibitor may be an option to use in these patients as a bridge during delayed absorption of oral agents.[59]

4.3 | Anticoagulation therapy

Studies among patients with acute coronary syndromes have consistently showed increased risk of acute stent thrombosis with bivalirudin compared with unfractionated heparin.[60] OHCA patients undergoing PCI are at high risk for thrombotic events and bleeding complications as described above and the risk of bleeding should be weighed against thrombotic risk.

TABLE 8 Prehospital cooling

Trial reference	Number of patients	Type of cooling	Outcomes
Bernard SA[67]	234 patients Patients with VF 118 patients pre hospital cooling	Rapid infusion of 2 L of ice-cold lactated Ringer's solution	Mean core temperature decreased by 0.8°C No difference in outcomes (47.5%% prehospital cooled, 52.6%% in hospital cooled risk ratio 0.90, 95% confidence interval 0.70 to 1.17, $p = .43$) Median volume 1900 ml
Castren M[69]	200 patients 104 in control arm	Intra-arrest cooling with transnasal evaporative cooling pre hospital	There were no significant differences in rates of return of spontaneous circulation between the groups (38% pre hospital cooling vs. 43% in hospital subjects, $p = .48$), in overall survival of those admitted alive (44 vs. 31%, respectively, $p = .26$)
Bernard, SA[68]	163 patients Initial rhythm of asystole and PEA	Two liters ice-cold Hartmann's solution 40 ml/Kg Goal fluid <8°C	Decreased mean core temperature compared to control (1.4°C pre hospital cooling vs. 0.2°C in hospital cooling $p < .001$) No difference in outcomes (12% pre hospital cooled, 9% in hospital cooled $p = .5$). Median Volume received 1,500 ml
Kim, F[70]	1,359 patients 583 with VF 776 without VF Standard care 291 with VF 380 without VF	2 L of 4°C normal saline following ROSC	Decreased mean core temperature both groups (1.2°C patients with VF and 1.3°C in patients without VF) Survival to hospital discharge was similar among the intervention and control groups among patients with VF (62.7% [95% CI, 57.0–68.0%] vs. 64.3% [95% CI, 58.6–69.5%], respectively; $p = .69$) Increased rates of rearrest and pulmonary edema Patient with VF 49% received 2 L Patient without VF 44% received 2 L
Nordberg P[71]	677 patients 334 in control arm	Intra-arrest cooling with transnasal evaporative cooling pre hospital	In the intervention group, 60 of 337 patients (17.8%) were alive at 90 days versus 52 of 334 (15.6%) in the control group (difference, 2.2% [95% CI, −3.4% to 7.9%]; RR, 1.14 [95% CI, 0.81–1.57]; $p = .44$).

5 | POST RESUSCITATIVE STATE INTERDISCIPLINARY TEAM AND TARGETED TEMPERATURE MANAGAMENT

Irrespective of whether a definite versus deferred CCL strategy is employed, post resuscitative support is important to the patient's overall outcome. Although studies have not specifically evaluated optimal cardiac arrest team membership, studies evaluating OHCA care systems, hospital volumes, and receiving hospital characteristics allow us to demonstrate that a multi-disciplinary approach may improve clinical outcomes (Figure 2).[61-64] Following the publication of two seminal trials[65,66] demonstrating improved survival with the delivery of in-hospital mild hypothermia for patients with resuscitated VF or VT (Table 7), OHCA patients should be assessed for targeted temperature managament (TTM) on arrival to the hospital. Pre hospital initiation of TTM has not demonstrated improved clinical outcomes and in some situations, it is associated with possible harm (Table 8).[67-71]

6 | SHOCK IN OHCA PATIENTS

Patients with OHCA and shock have substantially higher mortality rates and worse neurological outcomes.[72] Patients with shock have an onset of systemic hypoperfusion usually in response to ischemic cardiac injury. This hypoperfusion induces a cascade of pro-inflammatory signaling, vasomotor dysregulation, vasodilation, multisystem organ dysfunction, and death.[49] The early and late manifestations of shock in OHCA patients are related to a multitude of clinical factors; many of which may be unrelated to the underlying cardiac function.[73] Decisions surrounding extent of coronary revascularization are an important aspect of care among patients with shock.[74] In the randomized study—PCI Strategies in Patient with Acute Myocardial Infarction and Cardiogenic Shock (CULPRIT SHOCK)[75]—more than 50% of the patients had resuscitation prior to randomization, and more than 60% of the deaths were related to refractory CS. In patients who underwent PCI of the culprit lesion only, the composite risk of death and

TABLE 9 Ongoing extracorporeal life support studies in cardiac arrest

Study	Inclusion criteria	Exclusion	Outcome	Location
A Comparative Study Between a Pre-hospital and an In-hospital Circulatory Support Strategy (ECMO) in Refractory Cardiac Arrest (APACAR2) (APACAR2) NCT02527031	210 Patients • Refractory cardiac arrest (defined by the failure of professionals to resuscitate at the 20th min of cardiac arrest with a minimum of 3) Automatic External Defibrillator (AED) or equivalent analyze • And Beginning of external cardiac massage within the first 5 min after cardiac arrest (no flow <5 min.) with shockable rhythm or the presence of signs of life during resuscitation (any rhythm): spontaneous movement, absence of mydriasis and/or pupillary response, spontaneous breathing movements • And medical cause of the cardiac arrest • And end-tidal CO2 (ETCO2) above 10 mmHg at the time of inclusion • And absence of major co-morbidity. And Extra-corporeal Membrane Oxygenation (ECMO) team available and on-site before the 40th min	• Children under 18 years of age ◦ Adults over 65 years of age ◦ Period of more than 5 min without cardiac massage after collapsing ◦ Known co-morbidity that compromises the prognosis for short or medium-term survival ◦ Cardiac arrest during transportation times	Survival with good neurological outcome (CPC 1 or 2) at 6 months	Paris, France
Early initiation of extracorporeal life support in refractory OHCA (INCEPTION) NCT03101787	110 Patients 1 ≥18 to ≤70 years 2 Witnessed OHCA 3 Initial rhythm of VF/VT or AED administered 4 Bystander BLS 5 No ROSC	1 ROSC 2 Terminal heart failure (NYHA III or IV) 3 Severe pulmonary disease (COPD GIII of GIV) 4 Oncological disease 5 Pregnancy 6 Bilateral femoral bypass surgery 7 Pre-arrest CPC-score of 3 or 4 8 Advanced directive 9 Multitrauma (ISS >15) 10 Start cannulation >60 min after arrest	30-day survival rate with favorable neurological status	Maastricht university medical center
ECPR for Refractory Out-Of-Hospital Cardiac Arrest (EROCA) NCT03065647	30 Patients • OHCA of presumed non-traumatic etiology requiring CPR • Predicted arrival time at ECPR-capable hospital within timeframe specified • Witnessed arrest or initial shockable rhythm (VT or VF) • Persistent cardiac arrest after initial cardiac rhythm analysis and shock (if shock is indicated)	• Sustained return of spontaneous circulation (ROSC) • Advanced directive indicating do not attempt resuscitation (DNAR) or do not intubate (DNI) • Preexisting evidence of opting out of study • Prisoner • Pregnant (obvious or known) • ECPR capable ED is not at the destination hospital as determined by EMS	1 ED arrival interval [Time Frame: Measured within one hour cardiac arrest onset] Proportion of patients with emergency department (ED) arrival less than or equal to 30 min from 911 caLL (or cardiac arrest onset if witnessed by EMS personnel). 2 ECPR initiation interval (Time Frame: Measured within 2 hr of cardiac arrest	University of Michigan

TABLE 9 (Continued)

Study	Inclusion criteria	Exclusion	Outcome	Location
		• Legally authorized representative (LAR) or family member aware of study and refuses study participation at the scene	onset) Proportion of ECPR eligible patients with ECPR flow initiated less than or equal to 30 min from ED arrival	
Hyperinvasive Approach in Cardiac Arrest NCT01511666	170 patients • minimum of 18 and maximum of 65 years • witnessed out-of-hospital cardiac arrest of presumed cardiac cause • minimum of 5 min of ACLS performed by emergency medical service team without sustained ROSC • unconsciousness (Glasgow Coma Score < 8) • ECMO team and bed-capacity in cardiac center available.	• OHCA of presumed noncardiac cause • unwitnessed collapse • pregnancy • sustained ROSC within 5 min of ACLS performed by EMS team • conscious patient • known bleeding diathesis or suspected or confirmed acute or recent intracranial bleeding • suspected or confirmed acute stroke • known severe chronic organ dysfunction or other limitations in therapy • "do not resuscitate" order or other circumstances making 180 day survival unlikely • known pre-arrest cerebral performance category CPC ≥3.	Composite endpoint of survival with good neurological outcome (CPC 1–2) 6 months	Charles University, Czech Republic
Advanced Reperfusion Strategies for Refractory Cardiac Arrest (ARREST) NCT03880565	180 patients • minimum of 18 and maximum of 75 years • witnessed out-of-hospital cardiac arrest of presumed due to ventricular fibrillation or ventricular tachycardia • No ROSC following 3 defibrillation shocks • Body morphology able to accommodate a Lund University Cardiac Arrest System (LUCAS™) automated CPR device • Estimated transfer time from the scene to the ED or CCL of <30 min	• Age < 18 years old or > 75 years old • Non-shockable initial OHCA rhythm (pulseless electrical activity [PEA] or asystole) • Valid do-not-attempt-resuscitation orders • Blunt, penetrating, or burn-related injury, drowning, electrocution or known overdose • Known prisoners • Known pregnancy • Nursing home residents • Unavailability of the cardiac catheterization laboratory • Severe concomitant illness that drastically shortens life expectancy or increases risk of the procedure • Absolute contraindications to emergent coronary angiography including known anaphylactic reaction to angiographic contrast media and/or active gastrointestinal or internal bleeding	Experimental: Early extracorporeal membrane oxygenation facilitated resuscitation Standard: Standard advanced cardiac life support (ACLS) resuscitation Primary outcome: Survival to hospital discharge	Minneapolis, Minnesota, United States

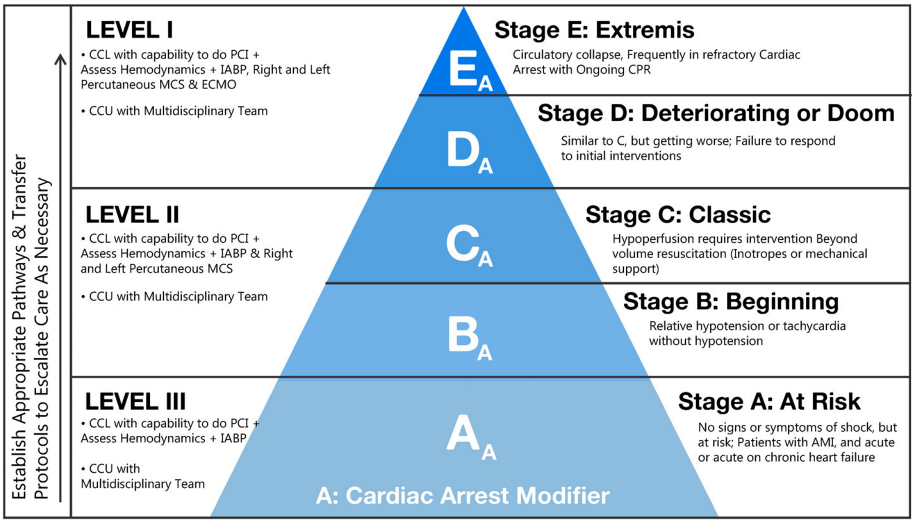

FIGURE 3 Proposed levels of care based on SCAI cardiogenic shock classification schema for patients with cardiac arrest and shock [Color figure can be viewed at wileyonlinelibrary.com]

severe renal failure was significantly lower compared with those who underwent immediate multivessel PCI.

There are no large randomized trials to date evaluating the use of MCS devices among OCHA patients with shock. The role of MCS in patients with STEMI complicated by shock, excluding comatose patients with OHCA, is being evaluated in the DanGer Shock trial whereby 360 patients will be randomized to percutaneous transvalvular microaxial flow pump (Impella CP Abiomed, Danvers, MA) versus guideline therapy.[76] In the IMPRESS in Severe Shock (IMPella vs. IABP Reduces mortality in STEMI patients treated with primary PCI in Severe cardiogenic SHOCK) trial 48 patients, (92% with cardiac arrest prior to randomization) were randomly assigned to MCS (Impella CP Abiomed, Danvers, MA) versus IABP (Intra-Aortic Balloon Pump). Treatment with Impella CP did not reduce mortality compared to IABP at 30 days.[72] The use of MCS, specifically Impella, has rapidly been increasing among patients undergoing PCI with limited evidence of efficacy and possible increase in adverse events.[77,78] Therefore, the role of MCS and extracorporeal membrane oxygenation in patients with OHCA and shock, with a focus on patient selection criteria, needs to be evaluated in randomized controlled trials, a topic that is beyond the scope of this document.

The role of extracorporeal life support (ECLS) for refractory OHCA is of research interest and in single centers with the necessary resources and regimented protocols have demonstrated benefit.[79] There are ongoing randomized trials to determine if ECLS demonstrates benefit in carefully selected patients with OHCA (Table 9). Whether this strategy can be employed outside of dedicated centers and is generalizable has not been demonstrated.[80,81] SCAI has proposed a simple classification system for rapid assessment of patients with CS.[82] A recent article retrospectively analyzed hospital survivors to a single cardiac ICU over an 8-year period and associated the SCAI shock classification with post discharge mortality in patient with acute coronary syndrome and heart failure but not cardiac arrest.[83] Institutions need to implement appropriate pathways and transfer protocols to hospitals that have the adequate resources to provide acute care for deteriorating patients (Figure 3).[84]

7 | CURRENT BARRIERS AND FUTURE DIRECTIONS OF PUBLIC REPORTING

Public reporting of procedural outcomes for PCI and coronary artery bypass grafting is expected in contemporary practice.[85] In the current public reporting formats, noncardiac mortality (ie, neurologic death) is indistinguishable from cardiac mortality and is therefore attributed as "PCI related mortality". This substantially impacts the observed clinical outcomes of both the physician and institution performing PCI. Outcomes in high risk OHCA patients cannot be accurately accounted for by current risk-adjustment models.[86-88] In addition, it is uncertain if public reporting of outcomes leads to risk averse behavior with fewer patients undergoing PCI or not.[89,90] A SCAI Position Statement on Public Reporting has previously been published, recommending exclusion of OHCA patients from public reporting of PCI outcomes and adjudication of high risk classification at local facility level as necessary.[85] In order to be able to identify OHCA as its own subset, the Chest Pain-MI Registry and the CathPCI Registry have recently undergone significant revisions (cvquality.acc.org/NCDR-Home/Data-Collection/What-Each-Registry-Collects). The data elements between these two registries have been harmonized for consistency. Cath PCI version 5 has recently added far more variables regarding cardiac arrest status (Appendix S3). The goal of collecting more comprehensive data on cardiac arrest is to develop accurate risk-adjustment models for benchmarking and public reporting. Further efforts to standardize the data elements, metrics and definitions used in various OHCA registries will enhance collaboration to improve outcomes among these patients.[91]

8 | CONCLUSION

Comatose patients with OHCA have a high mortality and morbidity. The management decisions in these patients are dynamic and interdependent, necessitating frequent clinical evaluations and multidisciplinary team-based approach along the entire continuum of care from pre hospital to post hospital care.

ORCID

Amir Lotfi https://orcid.org/0000-0001-8154-8497
Lloyd W. Klein https://orcid.org/0000-0003-0156-3094
Roxana Mehran https://orcid.org/0000-0002-5546-262X
Duane S. Pinto https://orcid.org/0000-0002-2309-8480

REFERENCES

1. Graham R, McCoy MA, Schultz AM, et al. In: Graham R, McCoy MA, Schultz AM, eds. Strategies to improve cardiac arrest survival: a time to act. Washington (DC): National Academies Press (US); 2015.
2. Hazinski MF, Nolan JP, Billi JE, et al. Part 1: Executive summary: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Circulation. 2010;122:S250-S275.
3. Welsford M, Belley-Cote EP. Emergent coronary angiography post arrest-Do observational studies provide enough evidence for strong recommendations? Resuscitation. 2018;123: A5-A6.
4. Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med. 2004;30:2126-2128.
5. Girotra S, van Diepen S, Nallamothu BK, et al. Regional variation in out-of-hospital cardiac arrest survival in the United States. Circulation. 2016;133:2159-2168.
6. Lemiale V, Dumas F, Mongardon N, et al. Intensive care unit mortality after cardiac arrest: the relative contribution of shock and brain injury in a large cohort. Intensive Care Med. 2013;39:1972-1980.
7. Geocadin RG, Callaway CW, Fink EL, et al. Standards for studies of neurological prognostication in comatose survivors of cardiac arrest: a scientific statement from the american heart association. Circulation. 2019;140:e517-e542.
8. Kaji AH, Hanif AM, Bosson N, Ostermayer D, Niemann JT. Predictors of neurologic outcome in patients resuscitated from out-of-hospital cardiac arrest using classification and regression tree analysis. Am J Cardiol. 2014;114:1024-1028.
9. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. Circ Cardiovasc Qual Outcomes. 2010;3:63-81.
10. Dumas F, Blackwood J, White L, et al. The relationship between chronic health conditions and outcome following out-of-hospital ventricular fibrillation cardiac arrest. Resuscitation. 2017;120:71-76.
11. Tangri N, Kent DM. Toward a modern era in clinical prediction: the TRIPOD statement for reporting prediction models. Am J Kidney Dis. 2015;65:530-533.
12. Bougouin W, Dumas F, Karam N, et al. Should we perform an immediate coronary angiogram in all patients after cardiac arrest?: insights from a large French registry. JACC Cardiovasc Interv. 2018;11: 249-256.
13. Bascom KE, Dziodzio J, Vasaiwala S, et al. Derivation and validation of the CREST model for very early prediction of circulatory etiology death in patients without st-segment-elevation myocardial infarction after cardiac arrest. Circulation. 2018;137:273-282.
14. Kiehl EL, Parker AM, Matar RM, et al. C-GRApH: a validated scoring system for early stratification of neurologic outcome after out-of-hospital cardiac arrest treated with targeted temperature management. J Am Heart Assoc. 2017;6.
15. Garcia-Tejada J, Jurado-Roman A, Rodriguez J, et al. Post-resuscitation electrocardiograms, acute coronary findings and in-hospital prognosis of survivors of out-of-hospital cardiac arrest. Resuscitation. 2014;85:1245-1250.
16. Kim YJ, Min SY, Lee DH, et al. the role of post-resuscitation electrocardiogram in patients with ST-segment changes in the immediate post-cardiac arrest period. JACC Cardiovasc Interv. 2017;10: 451-459.
17. Stanton N, Chambers PR, Piggott J. Situational awarness and safety. Saf Sci. 2001;39:189-204.
18. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;127:e362-e425.
19. Kjaergaard J, Nielsen N, Winther-Jensen M, et al. Impact of time to return of spontaneous circulation on neuroprotective effect of targeted temperature management at 33 or 36 degrees in comatose survivors of out-of hospital cardiac arrest. Resuscitation. 2015;96: 310-316.
20. Rab T, Kern KB, Tamis-Holland JE, et al. Cardiac Arrest: a treatment algorithm for emergent invasive cardiac procedures in the resuscitated comatose patient. J Am Coll Cardiol. 2015;66:62-73.
21. Kern KB, Lotun K, Patel N, et al. Outcomes of comatose cardiac arrest survivors with and without st-segment elevation myocardial infarction: importance of coronary angiography. JACC Cardiovasc Interv. 2015;8:1031-1040.
22. Lemkes JS, Janssens GN, van der Hoeven NW, et al. Coronary angiography after cardiac arrest without ST-segment elevation. N Engl J Med. 2019;380:1397-1407.
23. Dumas F, Cariou A, Manzo-Silberman S, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. Circ Cardiovasc Interv. 2010; 3:200-207.
24. Radsel P, Knafelj R, Kocjancic S, Noc M. Angiographic characteristics of coronary disease and postresuscitation electrocardiograms in patients with aborted cardiac arrest outside a hospital. Am J Cardiol. 2011;108:634-638.
25. Zanuttini D, Armellini I, Nucifora G, et al. Predictive value of electrocardiogram in diagnosing acute coronary artery lesions among patients with out-of-hospital-cardiac-arrest. Resuscitation. 2013;84: 1250-1254.
26. Welsford M, Bossard M, Shortt C, Pritchard J, Natarajan MK, Belley-Cote EP. Does early coronary angiography improve survival after out-of-hospital cardiac arrest? a systematic review with meta-analysis. Can J Cardiol. 2018;34:180-194.
27. Lemkes JS, Janssens GN, van Royen N. Coronary angiography after cardiac arrest without ST-Segment elevation. Reply N Engl J Med. 2019;381:189-190.
28. Davies MJ, Thomas A. Thrombosis and acute coronary-artery lesions in sudden cardiac ischemic death. N Engl J Med. 1984;310:1137-1140.
29. Garcia S, Drexel T, Bekwelem W, et al. Early access to the cardiac catheterization laboratory for patients resuscitated from cardiac arrest due to a shockable rhythm: the Minnesota resuscitation consortium twin cities unified protocol. J Am Heart Assoc. 2016;5.
30. Spaulding CM, Joly LM, Rosenberg A, et al. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. N Engl J Med. 1997;336:1629-1633.
31. Silfvast T. Cause of death in unsuccessful prehospital resuscitation. J Intern Med. 1991;229:331-335.
32. Virkkunen I, Paasio L, Ryynanen S, et al. Pulseless electrical activity and unsuccessful out-of-hospital resuscitation: what is the cause of death? Resuscitation. 2008;77:207-210.
33. Callaway CW, Schmicker RH, Brown SP, et al. Early coronary angiography and induced hypothermia are associated with survival and

functional recovery after out-of-hospital cardiac arrest. Resuscitation. 2014;85:657-663.
34. Dankiewicz J, Nielsen N, Annborn M, et al. Survival in patients without acute ST elevation after cardiac arrest and association with early coronary angiography: a post hoc analysis from the TTM trial. Intensive Care Med. 2015;41:856-864.
35. Geri G, Dumas F, Bougouin W, et al. Immediate Percutaneous Coronary Intervention Is Associated With Improved Short- and Long-Term Survival After Out-of-Hospital Cardiac Arrest. Circ Cardiovasc Interv. 2015;8(10):e002303.
36. Jaeger D, Dumas F, Escutnaire J, et al. Benefit of immediate coronary angiography after out-of-hospital cardiac arrest in France: a nationwide propensity score analysis from the ReAC Registry. Resuscitation. 2018;126:90-97.
37. Jentzer JC, Scutella M, Pike F, et al. Early coronary angiography and percutaneous coronary intervention are associated with improved outcomes after out of hospital cardiac arrest. Resuscitation. 2018; 123:15-21.
38. Ko E, Shin JK, Cha WC, et al. Coronary angiography is related to improved clinical outcome of out-of-hospital cardiac arrest with initial non-shockable rhythm. PLoS ONE. 2017;12:e0189442.
39. Nielsen N, Hovdenes J, Nilsson F, et al. Outcome, timing and adverse events in therapeutic hypothermia after out-of-hospital cardiac arrest. Acta Anaesthesiol Scand. 2009;53:926-934.
40. Reynolds JC, Rittenberger JC, Toma C, Callaway CW, Post Cardiac Arrest S. Risk-adjusted outcome prediction with initial post-cardiac arrest illness severity: implications for cardiac arrest survivors being considered for early invasive strategy. Resuscitation. 2014;85:1232-1239.
41. Staudacher II, den Uil C, Jewbali L, et al. Timing of coronary angiography in survivors of out-of-hospital cardiac arrest without obvious extracardiac causes. Resuscitation. 2018;123:98-104.
42. Vadeboncoeur TF, Chikani V, Hu C, Spaite DW, Bobrow BJ. Association between coronary angiography with or without percutaneous coronary intervention and outcomes after out-of-hospital cardiac arrest. Resuscitation. 2018;127:21-25.
43. Vyas A, Chan PS, Cram P, Nallamothu BK, McNally B, Girotra S. Early coronary angiography and survival after out-of-hospital cardiac arrest. Circ Cardiovasc Interv. 2015;8.
44. Myerburg RJ, Halperin H, Egan DA, et al. Pulseless electric activity: definition, causes, mechanisms, management, and research priorities for the next decade: report from a National Heart, Lung, and Blood Institute workshop. Circulation. 2013;128:2532-2541.
45. Weiser C, Testori C, Sterz F, et al. The effect of percutaneous coronary intervention in patients suffering from ST-segment elevation myocardial infarction complicated by out-of-hospital cardiac arrest on 30 days survival. *Resuscitation*. 2013;84:602-608.
46. Cournoyer A, Cossette S, Potter BJ, et al. Prognostic impact of the conversion to a shockable rhythm from a non-shockable rhythm for patients suffering from out-of-hospital cardiac arrest. Resuscitation. 2019;140:43-49.
47. Champigneulle B, Haruel PA, Pirracchio R, et al. Major traumatic complications after out-of-hospital cardiac arrest: Insights from the Parisian registry. Resuscitation. 2018;128:70-75.
48. Pruller F, Milke OL, Bis L, et al. Impaired aspirin-mediated platelet function inhibition in resuscitated patients with acute myocardial infarction treated with therapeutic hypothermia: a prospective, observational, non-randomized single-centre study. Ann Intensive Care. 2018;8:28.
49. Mongardon N, Dumas F, Ricome S, et al. Postcardiac arrest syndrome: from immediate resuscitation to long-term outcome. Ann Intensive Care. 2011;1:45.
50. Mason PJ, Shah B, Tamis-Holland JE, et al. An update on radial artery access and best practices for transradial coronary angiography and intervention in acute coronary syndrome: a scientific statement from the american heart association. Circ Cardiovasc Interv. 2018;11: e000035.
51. Le May M, Wells G, So D, et al. Safety and efficacy of femoral access vs radial access in st-segment elevation myocardial infarction: The SAFARI-STEMI randomized clinical trial. JAMA Cardiol. 2020;5(2): 126-134.
52. Mallidi J, Ulahannan J, Chaubey VK, et al. Comparison of clinical outcomes: bivalirudin with transfemoral access versus heparin with transradial access in patients with ST segment elevation myocardial infarction. Crit Pathw Cardiol. 2019;18:130-134.
53. Shah N, Chaudhary R, Mehta K, et al. Therapeutic hypothermia and stent thrombosis: a nationwide analysis. JACC Cardiovasc Interv. 2016;9:1801-1811.
54. Rosencher J, Gouffran G, Bougouin W, Varenne O. Optimal antiplatelet therapy in out-hospital cardiac arrest patients treated by primary percutaneous coronary intervention. Resuscitation. 2015;90: e7-e8.
55. Schupke S, Neumann FJ, Menichelli M, et al. Ticagrelor or prasugrel in patients with acute coronary syndromes. N Engl J Med. 2019;381: 1524-1534.
56. Wallentin L, Becker RC, Budaj A, et al. Ticagrelor versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2009;361: 1045-1057.
57. Wiviott SD, Braunwald E, McCabe CH, et al. Prasugrel versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2007;357:2001-2015.
58. Jimenez-Britez G, Freixa X, Flores E, et al. Safety of glycoprotein IIb/-IIIa inhibitors in patients under therapeutic hypothermia admitted for an acute coronary syndrome. Resuscitation. 2016;106:108-112.
59. Pruller F, Bis L, Milke OL, et al. Cangrelor induces more potent platelet inhibition without increasing bleeding in resuscitated patients. J Clin Med. 2018;7.
60. Bittl JA, He Y, Lang CD, Dangas GD. Factors affecting bleeding and stent thrombosis in clinical trials comparing bivalirudin with heparin during percutaneous coronary intervention. Circ Cardiovasc Interv. 2015;8:e002789.
61. Kajino K, Iwami T, Daya M, et al. Impact of transport to critical care medical centers on outcomes after out-of-hospital cardiac arrest. Resuscitation. 2010;81:549-554.
62. Soholm H, Wachtell K, Nielsen SL, et al. Tertiary centres have improved survival compared to other hospitals in the Copenhagen area after out-of-hospital cardiac arrest. Resuscitation. 2013;84: 162-167.
63. Spaite DW, Bobrow BJ, Stolz U, et al. Statewide regionalization of postarrest care for out-of-hospital cardiac arrest: association with survival and neurologic outcome. Ann Emerg Med. 2014;64:496-506.e1.
64. Wnent J, Seewald S, Heringlake M, et al. Choice of hospital after out-of-hospital cardiac arrest- -a decision with far-reaching consequences: a study in a large German city. Crit Care. 2012;16:R164.
65. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med. 2002;346:557-563.
66. Hypothermia after Cardiac Arrest Study G. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med. 2002;346:549-556.
67. Bernard SA, Smith K, Cameron P, et al. Induction of therapeutic hypothermia by paramedics after resuscitation from out-of-hospital ventricular fibrillation cardiac arrest: a randomized controlled trial. Circulation. 2010;122:737-742.
68. Bernard SA, Smith K, Cameron P, et al. Induction of prehospital therapeutic hypothermia after resuscitation from nonventricular fibrillation cardiac arrest*. Crit Care Med. 2012;40:747-753.
69. Castren M, Nordberg P, Svensson L, et al. Intra-arrest transnasal evaporative cooling: a randomized, prehospital, multicenter study

(PRINCE: Pre-ROSC IntraNasal Cooling Effectiveness). Circulation. 2010;122:729-736.
70. Kim F, Nichol G, Maynard C, et al. Effect of prehospital induction of mild hypothermia on survival and neurological status among adults with cardiac arrest: a randomized clinical trial. JAMA. 2014;311:45-52.
71. Nordberg P, Taccone FS, Truhlar A, et al. Effect of trans-nasal evaporative intra-arrest cooling on functional neurologic outcome in out-of-hospital cardiac arrest: the PRINCESS randomized clinical trial. JAMA. 2019;321:1677-1685.
72. Ouweneel DM, Eriksen E, Sjauw KD, et al. Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump in Cardiogenic Shock After Acute Myocardial Infarction. J Am Coll Cardiol. 2017;69:278-287.
73. Sionis A, Rivas-Lasarte M, Mebazaa A, et al. Current use and impact on 30-day mortality of pulmonary artery catheter in cardiogenic shock patients: results from the cardshock study. J Intensive Care Med. 2019;885066619828959.
74. Scholz KH, Maier SKG, Maier LS, et al. Impact of treatment delay on mortality in ST-segment elevation myocardial infarction (STEMI) patients presenting with and without haemodynamic instability: results from the German prospective, multicentre FITT-STEMI trial. Eur Heart J. 2018;39:1065-1074.
75. Thiele H, Akin I, Sandri M, et al. One-year outcomes after pci strategies in cardiogenic shock. N Engl J Med. 2018;379:1699-1710.
76. 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.
77. Amin AP, Spertus JA, Curtis JP, et al. The Evolving Landscape of Impella(R) Use in the United States Among Patients Undergoing Percutaneous Coronary Intervention with Mechanical Circulatory Support. Circulation. 2020;141(4):273-284.
78. Dhruva SS, Ross JS, Mortazavi BJ, et al. Association of use of an intravascular microaxial left ventricular assist device vs intra-aortic balloon pump with in-hospital mortality and major bleeding among patients with acute myocardial infarction complicated by cardiogenic shock. JAMA. 2020;323(8):734-745.
79. Yannopoulos D, Bartos JA, Raveendran G, et al. Coronary artery disease in patients with out-of-hospital refractory ventricular fibrillation cardiac arrest. J Am Coll Cardiol. 2017;70:1109-1117.
80. Bartos JA, Grunau B, Carlson C, et al. Improved survival with extracorporeal cardiopulmonary resuscitation despite progressive metabolic derangement associated with prolonged resuscitation. Circulation. 2020;141:877-886.
81. Bougouin W, Dumas F, Lamhaut L, et al. Extracorporeal cardiopulmonary resuscitation in out-of-hospital cardiac arrest: a registry study. Eur Heart J. 2019;ehz753.
82. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. Catheter Cardiovasc Interv. 2019;94:29-37.
83. 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.
84. Rab T, Ratanapo S, Kern KB, et al. Cardiac shock care centers: JACC review topic of the week. J Am Coll Cardiol. 2018;72:1972-1980.
85. Klein LW, Harjai KJ, Resnic F, et al. 2016 Revision of the SCAI position statement on public reporting. Catheter Cardiovasc Interv. 2017;89:269-279.
86. Garratt KN, Weintraub WS. Public reporting III: improving the value of public physician quality information. Circulation. 2018;137:2318-2320.
87. Henriques JP, Claessen BE, Dangas GD, et al. Performance of currently available risk models in a cohort of mechanically supported high-risk percutaneous coronary intervention- -From the PROTECT II randomized trial. Int J Cardiol. 2015;189:272-278.
88. Klein LW, Maroney J. Risk-adjusted models of 30-day mortality following coronary intervention: how can they be made more clinically relevant? JACC Cardiovasc Interv. 2013;6:623-624.
89. Resnic FS, Welt FG. The public health hazards of risk avoidance associated with public reporting of risk-adjusted outcomes in coronary intervention. J Am Coll Cardiol. 2009;53:825-830.
90. Werner RM, Asch DA. The unintended consequences of publicly reporting quality information. JAMA. 2005;293:1239-1244.
91. Cardiac Arrest Registry to Enhance Survival-Executive-Report-digital-version.pdf>. 2017:16–17.
92. Terman SW, Shields TA, Hume B, Silbergleit R. The influence of age and chronic medical conditions on neurological outcomes in out of hospital cardiac arrest. Resuscitation. 2015;89:169-176.
93. Kragholm K, Wissenberg M, Mortensen RN, et al. Bystander Efforts and 1-Year Outcomes in Out-of-Hospital Cardiac Arrest. N Engl J Med. 2017;376:1737-1747.
94. Komatsu T, Kinoshita K, Sakurai A, et al. Shorter time until return of spontaneous circulation is the only independent factor for a good neurological outcome in patients with postcardiac arrest syndrome. Emerg Med J. 2014;31:549-555.
95. Mlinaric A, Horvat M, Supak SV. Dealing with the positive publication bias: why you should really publish your negative results. Biochem Med (Zagreb). 2017;27:030201.

SUPPORTING INFORMATION

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

How to cite this article: Lotfi A, Klein LW, Hira RS, et al. SCAI expert consensus statement on out of hospital cardiac arrest. *Catheter Cardiovasc Interv*. 2020;96:844–861. https://doi.org/10.1002/ccd.28990