JSCAI
The official journal of the Society for Cardiovascular Angiography & Interventions

Standards and Guidelines

SCAI Door to Lactate Clearance (SCAI DLC) Cardiogenic Shock Initiative: Definition, Hypothesis, and Call to Action

Srihari S. Naidu, MD, MSCAI [a,*], Sandeep Nathan, MD, MSc, FSCAI [b], Mir B. Basir, DO, FSCAI [c], David A. Baran, MD, FSCAI [d], Jeffrey A. Marbach, MBBS, MS, FSCAI [e], Cindy L. Grines, MD, MSCAI [f]

[a] *Department of Cardiology, Westchester Medical Center, Valhalla, New York;* [b] *Section of Cardiology, University of Chicago Medicine, Chicago, Illinois;* [c] *Center for Structural Heart Disease, Division of Cardiology Henry Ford Health System, Detroit, Michigan;* [d] *Cleveland Clinic Florida, Weston, Florida;* [e] *Knight Cardiovascular Institute, Oregon Health and Science University, Portland, Oregon;* [f] *Department of Cardiology, Northside Hospital Cardiovascular Institute, Atlanta, Georgia*

Introduction

The field of cardiogenic shock (CS) has advanced rapidly in recent years, in part due to the creation and widespread adoption of the Society for Cardiovascular Angiography & Interventions (SCAI) SHOCK Classification in 2019, subsequently refined in 2022.[1,2] As investigators adopted the classification and expanded research into the field, some themes have emerged. First, diagnosing and staging CS has become the cornerstone of management, inclusive of using the SCAI SHOCK stage trajectory. Second, CS teams are vital to the consistency of management, providing both experience and expertise, as well as checks and balances on individual patients and system-based care. Increasingly, CS is recognized to be a very dynamic condition with the transition of SCAI stages clearly associated with outcome, even more than the initial stratification of shock severity. Lastly, biochemical and hemodynamic parameters are powerful prognostic markers associated with survival.

As CS care becomes more comprehensive, with a multitude of support devices, biochemical markers, hemodynamic profiles, etiologies, phenotypes, and baseline characteristics playing a role in outcome, it has become clear that focusing on tissue perfusion is the best early and ensuing marker of survival. Previously, hemodynamic parameters have been the main surrogate for adequate perfusion, but hemodynamic thresholds that would raise clinical concern are based on the underlying shock phenotype, and hemodynamics, per se, do not consistently relate to tissue perfusion. The SCAI SHOCK criteria are intended to be used repeatedly to allow the trajectory of shock evolution to be evaluated. Worsening SCAI SHOCK stage is clearly associated with worse clinical outcomes.

Several investigations in various disease states including cardiogenic shock have focused on serum lactate as an early marker of poor perfusion: a laboratory test that is readily available at the bedside and rises and falls rapidly in response to changes in physiology or management. This document focuses on lactate as a potential recommended marker of CS trajectory and provides an initial framework and call to action for further research and consideration of its use.

Pathophysiology of lactate in CS

Lactic acid (lactate, L-enantiomer), conventionally regarded as a byproduct of anaerobic metabolism, is widely produced at low levels under basal conditions by muscle and other tissue beds including gut, brain, skin, and red blood cells, with greater production observed in response to hypoxemia or tissue hypoperfusion.[3,4] Under hypoxic conditions, glycolysis of glucose generates pyruvate, which is then reduced by L-lactate dehydrogenase to lactate.[4] Recent investigations have implicated accelerated glycolysis induced by adrenergic stress as an additional mechanism for lactate generation.[3] Lactate clearance (LC) occurs primarily through the liver—accounting for approximately 70% to 75%—via gluconeogenesis and oxidation.[4] The remaining 25% to 30% is cleared by the kidneys through similar mechanisms, with urinary excretion serving only a minor role in clearance (<10%).[4] The historic classification of lactic acidosis divides the condition into 2 categories: type A, associated with inadequate tissue perfusion or oxygenation and resulting in excess lactate production; and type B, occurring in the presence of adequate perfusion but driven by increased production, reduced clearance, or both.[5,6] A revised 3-part schema has been recently proposed, dividing

Abbreviations: AMI, acute myocardial infarction; CS, cardiogenic shock; DLC, door to lactate clearance.

Keywords: Cardiogenic shock; serum lactate.

* Corresponding author: Srihari.naidu@wmchealth.org (S.S. Naidu).

https://doi.org/10.1016/j.jscai.2025.103996
Available online 18 September 2025

conditions into increased pyruvate production secondary to stimulation of glycolysis, decreased use of pyruvate due to hypoxia, or impaired LC secondary to severe hepatic or renal injury.[7] Finally, beyond its role as a metabolic byproduct, lactate has been increasingly recognized as an important myocardial energy substrate, particularly during periods of increased metabolic demand—including heart failure and CS—underscoring the complexity of lactic acidosis in shock states.[4]

Lactate elevation and clearance as marker of shock trajectory

In critical illness, associations between peak lactate elevation, persistent lactic acidosis and mortality have been recognized for well over 5 decades, with even mildly elevated lactate portending worse outcomes.[8] An elevated baseline lactate level is a potent predictor of mortality in patients with AMI and heart failure.[9,10] While a single value, such as a baseline lactate, is associated with outcomes,[11] serial lactate levels have been demonstrated to have superior predictability.[12] The prognostic implications of lactate elevation and dynamics have been summarized in Table 1.[13–23] A secondary analysis of the CardShock registry found that in 217 patients with AMI-CS, baseline lactate was a strong predictor of 30-day mortality, as were lactate levels measured at 6, 12, and 24 hours.[3] A 50% relative reduction in lactate 24 hours after intensive care unit (ICU) admission was also predictive of improved survival.

In a post hoc analysis of the Intra-Aortic Balloon Pump in CS II (IABP-SHOCK II) trial, the 8-hour lactate level was the strongest predictor of mortality among all variables analyzed, with an optimal cutoff value of 3.1 mmol/L.[12] This measure along with LC (calculated as the difference in 2 lactate levels divided by the time elapsed) of <3.45%/h were independently predictive of time to death.

In the National Cardiogenic Shock Initiative, a lactate level at 12 to 24 hours after the use of mechanical circulatory support was independently associated with in-hospital mortality.[15] In the DanGer Shock trial, which randomly assigned selected noncomatose patients with AMI-CS to microaxial flow pump (mAFP) and compared with those assigned to standard medical therapy, there was no difference in lactate levels between groups at randomization. However, on arrival to the ICU, the mAFP group had significantly lower lactate levels than the standard-care group, a difference that persisted throughout the first 24 hours of observation.[22] The mAFP group achieved lactate normalization (defined as a lactate <2 mmol/L) 12 hours (95% CI, 5-18 hours) before the standard-care group. Similarly, in patients treated with venoarterial extracorporeal life support, LC has been associated with survival across a variety of disease states including cardiac arrest, sepsis, and CS.[18–20]

In a post hoc analysis of prospectively collected lactate data in the Dobutamine Compared to Milrinone in the Treatment of CS (DOREMI) trial, complete clearance of lactate, percentage LC, and percentage LC per hour were independent predictors of survival, with complete LC serving as the strongest predictor of survival.[17]

In a retrospective cohort analysis of 1884 patients with CS, each 1-mmol/L increase in lactate raised mortality 9%, while another retrospective cohort analysis of 43 patients on mAFP or venoarterial extracorporeal life support found 24-hour LC to be a strong predictor of survival.[13,14] Lastly, in a meta-analysis of 12 studies with over 1500 patients, LC within 24 hours was a strong prognostic marker for survival.[16]

In addition to the developing data in CS, a large body of literature supports elevated lactate as a marker of mortality, and its clearance with improved survival, in a variety of other clinical subsets from septic shock to hemorrhagic shock and burn-related injury.[24–27] Importantly, in a meta-analysis of trials that randomly assigned 1301 patients with sepsis to receive either LC-directed therapy, with q2 hour lactate sampling over the first 8 hours, vs central venous oxygen saturation–guided therapy, those guided by early LC had significant improvement in-hospital mortality, shorter mechanical ventilation times, and shorter ICU stays.[25] The authors concluded that LC was superior and recommended measuring lactate every 2 hours in patients with septic shock.

Hence, an initiative focusing on door to lactate clearance (DLC), irrespective of the etiology of CS, its starting value, and agnostic to the treatment modality(ies) chosen can potentially serve as an important goal for hospitals similar to what was historically proposed and subsequently achieved with the introduction to door to balloon times in ST-segment elevation myocardial infarction care.

Practicalities of use: individualized and systems based

The DLC as a marker of improving trajectory in CS, herein defined as a fall in lactate to <2 mmol/L within the first 24 hours of diagnosis, raises a number of issues which should be considered. First, it is important to note that the availability of lactic acid measurements varies across care settings and hospitals. Point-of-care lactic acid measurements have long been available via commercial devices with capillary blood samples but are not routinely utilized. Furthermore, the value of lactic acid depends on the sample site (venous, arterial, capillary) as well as the analyzer; however, in clinical practice the differences between these sampling locations are of little clinical significance and repeated measurements using the same location allow care teams to reliably establish trends in value.

As already discussed, there is increasing evidence that LC is associated with better outcomes, regardless of time / day of presentation or diagnosis, which also makes intuitive sense.[17,21,23,28] The best way to hasten LC and thereby facilitate resolution of shock will be variable based upon shock etiology, phenotype, SCAI SHOCK stage, and hemodynamics. While the DanGer Shock trial demonstrated the superiority of mAFP pumps in selected noncomatose AMI-CS patients, the trial results cannot be extrapolated to patients with prolonged cardiac arrest, HF-CS and right ventricular failure. Furthermore, hospitals without access to advanced circulatory support devices especially need ways to identify CS earlier and vodus on reversing the CS cascade.[29] While there is no universally accepted approach to the management of CS there is increasing emphasis on the use and development of CS teams. An important value of the DLC initiative is that protocolized reassessment of the metabolic state of the patient at regular intervals, along with invasive hemodynamic assessments, provides the local/shock team objective measures for frequent reassessment, and overall strengthens the use of such teams regardless of system resources.

It is important to emphasize that DLC is equally accessible to both regional referral centers (RRCs) as well as tertiary advanced care centers (TACs). The RRC without the capability to escalate care to advanced levels needs to be particularly cognizant of the failure of LC as an indication to rapidly re-evaluate the patient, escalate care or consider transfer to TACs. This rapid assessment, initial management, and reassessment should be completed in the early hours after diagnosis to facilitate efficient and safe transfer when warranted, or consider alternate treatments, as delays could affect the DLC and ultimately the patient outcome. And for the TAC, earlier transfer of appropriate patients allows for additional escalation options and improved survival. Organized and efficient systems of CS care are feasible and protocolized planning optimizes outcomes.[30,31]

After initial staging of CS with the SCAI SHOCK classification, we propose that lactate be measured at initial diagnosis and then subsequently every 2 to 3 hours to evaluate the clinical response to the initial management strategy. During this time, additional data should be obtained, including a more extensive physical

Table 1. Prognostic implications of lactate elevation and dynamics.

Study/author, year	Design	Population	Sample size (n)	Outcome	Lactate measurement associated with outcomes		
					Baseline lactate	Serial lactate measurements	LC
Fuernau et al,[12] 2020 (IABP-SHOCK II)	Post hoc analysis of RCT and registry	AMI-CS	666	30-d mortality	Baseline lactate level—NS: 5.8 mmol/L (IQR, 2.1-9.8 mmol/L) vs S: 3.2 mmol/L (IQR, 1.7-5.8 mmmol/L); $P < .001$ Baseline lactate, ≥5 mmol/L; multivariable cox regression HR, 1.35; 95% CI, 0.99-1.83; $P = .06$	8-h lactate—NS: 5.1 mmol/L (IQR, 2.3-11.0 mmol/L) vs S: 1.7 mmol/L (IQR, 1.2-3.2 mmol/L); $P < .001$ 8-h lactate, ≥3.1 mmol/L; multivariable cox regression HR, 2.89; 95% CI, 2.10-3.97; $P < .001$	$\text{LC } (\%/h) = \frac{(L1 - L2)}{(L1 \times \Delta t(L1, L2)} \times 100$ LC—NS: −0.4%/h (IQR, −5.7 to 5.8%/h) vs S: 1.4%/h (IQR, −2.2 to 6.5%/h); $P < .001$ LC, ≥3.45%/h; multivariable cox regression HR, 0.53; 95% CI, 0.40-0.70; $P < .001$
Lindholmet al,[3] 2020 (Cardshock)	Prospective, multicenter registry	AMI-CS; HF-CS	217	30-d mortality	Cox proportional hazards for 30-d mortality Baseline lactate HR, 1.20 mmol/L; 95% CI, 1.14-1.27; $P < .0001$	Cox proportional hazards for 30-d mortality: - 6-h lactate: HR, 1.14; 95% CI, 1.06-1.24; $P < .0001$ - 12-h lactate: HR, 1.10; 95% CI, 1.04-1.17; $P < .01$ - 24-h lactate: HR, 1.19; 95% CI, 1.07-1.32; $P < .01$	Cox proportional hazards for 30-d mortality - 50% reduction in lactate within 6 h of admission: HR, 0.82; 95% CI, 0.72-0.94; $P < .01$ - 50% reduction in lactate within 12 h of admission: HR, 0.87; 95% CI, 0.76-0.98; $P < .05$ - 50% reduction in lactate within 24 h of admission: HR, 0.74; 95% CI, 0.60-0.91; $P < .01$
Scolari et al,[13] 2020	Retrospective cohort	CS patients requiring Impella CP or VA-ECMO	43	30-d mortality	Baseline lactate—S: 4.0 mmol/L (IQR, 2.62-6.3 mmol/L) vs NS: 7.5 mmol/L (IQR, 2.8-12.0 mmol/L)	6-h lactate—S: 2.4 mmol/L (IQR, 1.7-6.2 mmol/L) vs NS: 5.9 mmol/L (IQR, 2.6-15.0 mmol/L); $P = .02$ 12-h lactate—S: 1.8 mmol/L (IQR, 1.3-2.6 mmol/L) vs NS: 4.0 mmol/L (IQR, 1.6-14.3 mmol/L); $P = .02$ 24-h lactate—S: 1.3 mmol/L (IQR, 1.1-2.3 mmol/L) vs NS: 3.5 mmol/L (IQR, 1.6-13.3 mmol/L); $P = .001$	24-h LC—S: 60.3% (IQR, 42.5%-72.8%) vs NS: 18.9% (IQR, −50% to 68.2%); $P = .04$ Logistic regression for 30-d mortality - 6-h LC: OR, 0.98; 95% CI, 0.97-0.99; $P = .01$ - 12-h LC: OR, 0.98; 95% CI, 0.96-0.99; $P = .006$ - 24-h LC: OR, 0.95; 95% CI, 0.91-0.98; $P < .001$
Jentzer and Monroe,[14] 2022	Retrospective cohort	All comer CS	1884	30-d survival	Cox proportional hazard analysis for 30-d mortality - Per 1-mmol/L increase in lactate HR, 1.09; 95% CI, 1.05-1.12 - Lactate, ≥5 mmol/L: HR, 1.96; 95 CI, 1.48-2.59	–	–
Basir et al,[15] 2023 (NCSI)	Prospective, multicenter cohort	AMI-CS	406	In-hospital mortality	Baseline lactate—S: 4.29 mmol/L vs NS: 5.95 mmol/L (P = not significant)	12-h lactate—S: 2.73 mmol/L vs NS: 6.29 mmol/L ($P < .01$) 24-h lactate—S: 2.08 mmol/L vs NS: 4.06 mmol/L ($P < .01$)	–
Marbach et al,[16] 2021	Systematic review and meta-analysis	AMI-CS; HF-CS	1500 (12 studies)	LC as a prognostic factor in CS	–	–	Median 6- to 8-h LC—S: 21.9% (IQR, 14.6%-42.1%) vs NS: 0.6% (IQR, 3.7%-14.6%). Pooled mean difference, 17.3%; 95% CI, 11.6-23.1; $P < .001$ Median 24-h LC—S: 60.7% (IQR, 58.1%-76.3%) vs NS: 40.3% (IQR, 30.2%-55.8%). Pooled mean difference, 27.9%; 95% CI, 14.1-41.7; $P < .001$

(*continued on next page*)

Table 1 (*continued*)

Study/author, year	Design	Population	Sample size (n)	Outcome	Lactate measurement associated with outcomes		
					Baseline lactate	Serial lactate measurements	LC
Marbach et al,[17] 2022 (DOREMI)	Post hoc analysis of RCT	AMI-CS; HF-CS	192	In-hospital mortality	Baseline lactate—S: 3.75 mmol/L (IQR, 2.63-5.40 mmol/L) vs NS: 3.70 mmol/L (IQR, 2.68-5.98 mmol/L); $P = .58$	4-h lactate—S: 2.50 mmol/L (IQR, 1.80-3.33 mmol/L) vs NS: 2.95 mmol/L (IQR, 2.10-4.97 mmol/L); $P = .08$	Multivariate logistic regression for mortality: - 6-h LC: OR, 2.46; 95% CI, 1.09-5.55; $P = .03$ - 12-h LC: OR, 3.98; 95% CI, 1.76-8.99; $P < .01$ - 18-h LC: OR, 3.68; 95% CI, 1.62-8.38; $P < .01$ - 24-h LC: OR, 5.44; 95% CI, 2.14-13.8; $P < .01$
Fadel et al,[18] 2024	Retrospective cohort	All comer peripheral VA-ECMO (35% AMI-CS, 30% HF-CS)	244	Death on VA-ECMO or within 24 h of decannulation	Baseline lactate—S: 2.5 mmol/L (IQR, 1.6-6.0 mmol/L) vs NS 6.6 mmol/L (IQR, 2.8-12.0); $P < .001$) Multivariate regression: elevate baseline lactate independently associated with VA-ECMO death (OR, 1.13 per-mmol/L increase; 95% CI, 1.04-1.23; $P = .003$)	8-h after VA-ECMO—S: 2.7 mmol/L (IQR, 1.4-4.9 mmol/L) vs NS: 8.4 mmol/L (IQR, 3.3-14.9 mmol/L); $P < .001$ 24-h after VA-ECMO—S: 1.3 mmol/L (IQR, 1.0-2.2 mmol/L) vs NS 3.9 mmol/L (IQR, 2.0-8.8 mmol/L); $P < .001$	8-h LC—S: 33.3% (SD, 7.8%-49.3%) vs NS: 13.5% (SD, −7.5% to 36.2%); $P = .002$ Multivariate regression 8-h LC (%) independently associated with VA-ECMO death (OR, 0.99; 95% CI, 0.98-0.99; $P < .001$)
Laimoud et al,[19] 2024	Retrospective cohort	Postcardiotomy VA-ECMO	152	In-hospital mortality	Baseline lactate—S: 5.8 mmol/L (IQR, 4.8-8.3 mmol/L) vs NS: 9.75 mmol/L (IQR, 6.55-13.4 mmol/L); $P < .001$	12-h lactate—S: 4.1 mmol/L (IQR, 2.8-6.4 mmol/L) vs NS: 11.25 mmol/L (IQR, 7.3-18.9 mmol/L); $P < .001$ 24-h lactate—S: 1.9 mmol/L (IQR, 1.4-3.1 mmol/L) vs NS 6.55 mmol/L (IQR, 4.05-20 mmol/L); $P < .001$	12-h LC—S: 39% (IQR, 7.25%-52.2%) vs NS: −24% (IQR, −59% to 15%); $P < .001$ 24-h LC—S: 66% (IQR, 53%-77%) vs NS: 20% (IQR, −72 to 53%); $P < .001$ Cox proportional hazards regression for mortality - 12-hour LC < 21.94% 2.73; 95% CI, 1.64-5.762; $P < .001$) - 24 h LC < 40.3% (HR, 1.98; 95% CI, 1.46-4.17; $P < .001$)
Sugimoto et al,[20] 2024 (SAVE-J II)	Retrospective registry	Out-of-hospital cardiac arrest requiring VA-ECMO	796	30-d survival	Baseline lactate associated with survival (OR, 0.857; 95% CI, 0.735-0.999; $P = .049$)	–	6-h LC associated with survival (OR, 2.804; 95% CI, 1.61-4.89; $P < .001$) 24-h LC associated with survival (OR, 1.010; 95% CI, 1.006-1.014; $P < .001$)
Khalife et al,[21] 2024 (CSWG)	Prospective registry	All comer CS	1381	In-hospital mortality	Baseline lactate S 3.2 mmol/L ± 2.8 vs NS 4.7 mmol/L ± 4.6 ($P < .01$)	–	–
Udesen et al,[22] 2025 (DanGer Shock)	Post hoc analysis of RCT	AMI-CS	324	72-h death from all causes, escalation of MCS, and discharge alive from the ICU	No difference in baseline lactate between standard of care and MAFP groups	ICU arrival lactate MAFP, 3.5 mmol/L (95% CI, 3.2-3.9) vs standard-care, 4.8 mmol/L (95% CI, 4.3-5.4). Mean difference, 1.3 mmol/L (95% CI, 0.7-1.0)	MAFP achieved lactate normalization (<2 mmol/L) 12 h (95% CI, 5-18 h) earlier than the standard of care group
Ikeda et al,[23] 2025	Retrospective cohort	CS requiring MAFP	501	30-d all-cause mortality or unplanned MCS reintroduction following removal	Pre-MAFP lactate ≥ 2.0 mmol/L—S: 76% vs NS: 90% ($P = .0002$) Cox proportional hazards for 30-d events - Pre-MAFP lactate ≥ 2.0 mmol/L; HR, 2.4; 95% CI, 1.5-3.9; $P = .0004$	24 h after MAFP lactate ≥2.0 mmol/L—S: 38% vs NS: 63% ($P < .0001$) Cox proportional hazards for 30-d events - 24-h after MAFP lactate ≥ 2.0 mmol/L; HR, 2.3; 95% CI, 1.7-3.1; $P < .0001$	–

AMI-CS, acute myocardial infarction complicated by cardiogenic shock; CSWG, Cardiogenic Shock Working Group; HF-CS, heart failure complicated by cardiogenic shock; HR, hazard ratio; LC, lactate clearance; MAFP, microaxial flow pump; MCS, mechanical circulatory support; NS, nonsurvivor; OR, odds ratio; RCT, randomized, controlled trial; S, survivor; VA-ECMO, venoarterial extracorporeal membrane oxygenation.

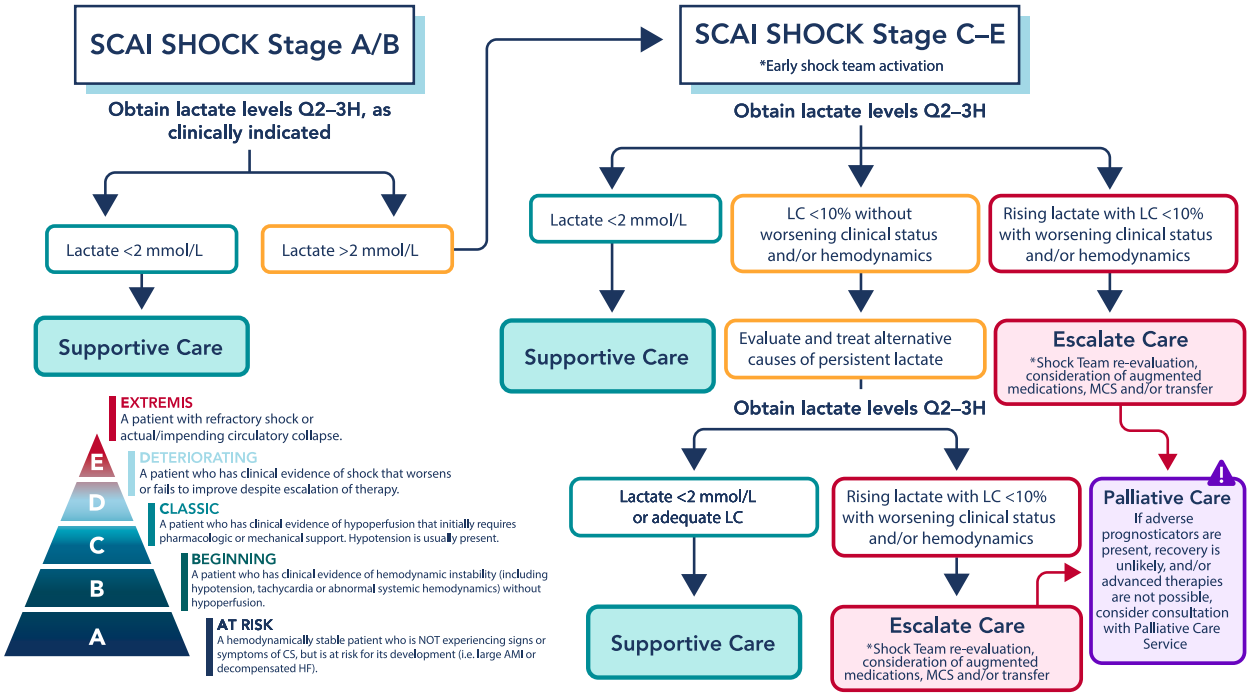

Figure 1.
Flowchart for how door to lactate clearance may be incorporated into cardiogenic shock care. Escalation options: reevaluation through additional imaging or invasive hemodynamics, vasopressors, inotropes, MCS, cardiac substrate intervention, or consider initiating palliative care. CS, cardiogenic shock; LC, lactate clearance; MCS, mechanical circulatory support; SCAI, Society for Cardiovascular Angiography & Interventions.

examination, echocardiography and invasive hemodynamic measures either using a pulmonary artery catheter or by obtaining central access which will allow clinicians to obtain a mixed venous blood gas and central venous pressure. This initial assessment will guide clinicians to establish a shock profile, phenotype and cardiac output and index. Within the first 4 to 6 hours (2-3 serial lactate measurements), a decision should be made to either escalate or modify current management, including as needed transfer to a TAC (if lactate is increasing or failing to clear) or continuing with the current management (if lactate is clearing). If the patient continues to manifest adverse prognosticators (eg, persistent acidosis, evidence of multiorgan failure, signs of significant neurologic injury, etc), recovery is deemed unlikely, and/or the patient is ineligible for advanced heart failure therapies, involvement of palliative care services as part of an integrated approach to CS is warranted. The higher the initial lactate, the more important it is to make a rapid assessment of trajectory, and the waiting time before therapy escalation or transfer discussions should be minimized in these cases. The clinician should be cognizant of factors that result in a sustained elevated lactate level despite adequate systemic perfusion, as well as those that result in LC in the absence of adequate perfusion (Figure 1, Table 2).

Ideally, health systems should provide the ability to measure lactate in real-time in all clinical settings where shock is evaluated and managed. This would include the emergency response services, emergency departments, cardiac catheterization laboratories, operating rooms and intensive care units. When transfer is being considered, communication between hospitals should prioritize the initial and subsequent lactate levels, time elapsed from the initial reading and expedite transfer to handoff the remaining time left on the DLC to the receiving facility. If shown in future work to be clearly associated with outcomes, prioritization of DLC of 24 hours within health systems, and across regions at both local and state levels may eventually be an important focus, similar to the door to balloon times for ST-segment elevation myocardial infarction (Figure 1, Table 3).[32–36]

Recent studies have demonstrated improved clinical outcomes in patients with CS by using such shock networks (Table 3).[36] Moreover, early transfer of selected patients to a TAC has been

Table 2. Therapies and conditions that potentially alter lactate dynamics.

Therapy	Observed effect	Appropriate or inappropriate
Renal replacement therapies	Decreases lactate (increased clearance)	Inappropriately decreases lactate, may no longer represent adequate perfusion
Intra-aortic balloon pump (IABP)	Decreases lactate	Appropriately decreases lactate
Venoarterial extracorporeal membrane oxygenation (VA-ECMO)	Decreases lactate	Appropriately decreases lactate
Microaxial flow pumps (mAFP)	Decreases lactate	Appropriately decreases lactate
Focal end-organ ischemia (gut, extremity)	Increases lactate (increased local production)	Inappropriately increases lactate out of proportion to systemic perfusion, but must be addressed
Liver failure	Increases lactate (decreased clearance)	Inappropriately increases lactate, may no longer be accurate marker, should improve as liver failure resolves

Table 3. Clinical outcomes in CS patients by using regionalized shock networks.

Study site	Design	Sample size	Study arms	Shock phenotypes	Key outcomes
National Cardiogenic Shock Initiative[32]	Multicenter prospective observational	406	Single-arm: early CS identification using invasive hemodynamics and pVAD before PCI	AMI-CS	Survival to discharge: 71% RHC in 91% MCS before PCI in 70% Mortality predictors: age, DM, CVA/TIA, pre-MCS (HR, SBP, lactate, and door-to-support times).
Inova Heart and Vascular Institute[31]	Single-center prospective and retrospective	204	Shock team vs historical control	AMI-CS, HF-CS	30-d survival improved: 47% (2016) → 77% (2018), $P < .01$ Mortality predictors: age ≥ 71 y, DM, dialysis, vasopressors ≥ 36 h, lactate ≥ 3.0 mmol/L, CPO <0.6 W, PAPi <1.0 at 24 h
University of Utah[33]	Single-center prospective and retrospective	244	Shock team vs historical control	AMI-CS, HF-CS	In-hospital survival improvement after implementing shock team: 61% vs 48%; $P = .041$ 30-d all-cause mortality reduction: HR, 0.61; 95% CI, 0.41-0.93 No difference in shock-to-support times or mean length of MCS support preshock and postshock team Risk factors for 30-d mortality at time of MCS implant: AMI-CS, lactate, and AKI
University of Ottawa[34]	Single-center prospective and retrospective	100	Shock team vs historical control	AMI-CS and HF-CS	No significant difference in discharge or 30-d survival No difference in MCS use or median length of stay Improved 240-d survival: HR, 0.53; 95% CI,: 0.28-0.99; $P = .03$ More HF specialist follow-up: 75% vs 50%; $P = .03$
Critical Care Cardiology Trials Network[35]	Multicenter prospective observational	1242	CICUs with vs without shock teams	AMI-CS and HF-CS	More use of invasive hemodynamics: 60% vs 49%; $P < .001$ Less overall use of MCS: 35% vs 43%; $P = .016$ More advanced MCS: 53% vs 43%; $P = .005$ Lower CICU mortality: 23% vs 29%; $P = .016$

Table adapted from Gattani et al.[36]
AKI, acute kidney injury; AMI-CS, acute myocardial infarction complicated by cardiogenic shock; CICU, cardiac intensive care unit; CPO, cardiac power output; CS, cardiogenic shock; DM, diabetes mellitus; HF-CS, heart failure complicated by cardiogenic shock; HR, hazard ratio; MSC, mechanical circulatory support; NS, nonsignificant; PCI, percutaneous coronary intervention; PAPi, pulmonary arterial pulsatility index; pVAD, percutaneous ventricular assist device; RHC, right heart catheterization; W, watts.

associated with improved survival despite greater comorbidities.[37–39] We hope that using the DLC in such networks would expedite transfers and optimize management by providing an objective criterion and a time course deadline by which to optimize care. It is also hoped that the use of a simple maker such as LC may prompt RRCs to augment their armamentarium to allow more patients with CS to be treated fully in the local environment, and thereby saving capacity for transfers to those who are most likely to benefit.

Conclusions and next steps

After suspecting or diagnosing CS using the SCAI SHOCK classification, assessment of tissue hypoperfusion using serial lactates in a timely fashion may be an important tool. We call for studying serially measured lactate levels in 2- to 3-hour intervals to help establish the diagnosis and trajectory of CS and thereby rapidly assess the effectiveness of early management. In using such an approach, changes in perfusion, either exacerbation or improvement, can be rapidly determined, allowing real-time modifications to management by the CS team that may improve survival. It is anticipated that this may prevent both inappropriate escalation of care with its inherent risks and allow for timely and optimal escalation and deescalation, including transfer to a TAC, thereby improving both survival and hospital duration. If subsequent research validates this approach, hospitals will organize into RRC and TAC models, and potentially prioritize time to transfer and DLC within 24 hours. While there are pitfalls to linking lactate solely to systemic perfusion, where lactate levels may instead partially represent enhanced clearance unrelated to perfusion (eg, early initiation of dialysis), production from a more discrete etiology (eg, ischemic gut or compartment syndrome), or lack of clearance (eg, shock liver), these should be accounted for when LC is not consistent with the global picture of perfusion.

Clinicians and health care systems may choose now to make rapid point-of-care lactate testing ubiquitous in their facilities and expedite therapy escalation as well as transfers in a timely manner for patients failing to clear lactate in the early hours of CS care. If DLC is incorporated into care algorithms, it would be utilized regardless of time of day or week to measure and trend lactate serially toward the clinical recognition of shock and shock trajectory in earlier time frames. Accumulation of real-world registry evidence will allow evaluation of this approach to validate the hypothesis. Furthermore, we call on clinical investigators and registry organizers to incorporate and evaluate serial lactate and DLC prospectively, to observe whether this rather simple intervention translates to improved survival, including the proposed sampling interval and 24-hour goal regardless of management strategy, thereby facilitating multiple protocols for CS management across a spectrum of resource availability.

Peer review statement

Associate Editors, Sandeep Nathan and Cindy L. Grines had no involvement in the peer review of this article and have no access to information regarding its peer review.

Declaration of competing interest

Srihari Naidu discloses serving as an advisor and speaker for Bristol Meyers Squibb and Zoll TherOx and as an advisor to Cytokinetics.

Sandeep Nathan discloses serving as a consultant for Zoll, Merit Medical, and Magenta Medical. Mir B. Basir discloses serving as a consultant for Abiomed, Boston Scientific, Chiesi, and Zoll. David A. Baran discloses serving as a consultant and owner of stock options for Nirsense. Cindy L. Grines discloses serving on the advisory board of Abiomed. Jeffrey A. Marbach reported no relevant relationships with industry.

Funding sources

This work was not supported by funding agencies in the public, commercial, or not-for-profit sectors.

Supplementary material

To access the supplementary material accompanying this article, visit the online version of the *Journal of the Society for Cardiovascular Angiography & Interventions* at 10.1016/j.jscai.2025.103996.

References

1. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock. *Catheter Cardiovasc Interv*. 2019;94(1):29–37. https://doi.org/10.1002/ccd.28329
2. Naidu SS, Baran DA, Jentzer JC, et al. SCAI SHOCK stage classification expert consensus update: a review and incorporation of validation studies. *J Soc Cardiovasc Angiogr Interv*. 2022;1(1):100008. https://doi.org/10.1016/j.jscai.2021.100008
3. Lindholm MG, Hongisto M, Lassus J, et al. Serum lactate and a relative change in lactate as predictors of mortality in patients with cardiogenic shock—results from the Cardshock Study. *Shock*. 2020;53(1):43–49. https://doi.org/10.1097/shk.0000000000001353
4. Yuzefpolskaya M, Schwartz S, Ladanyi A, et al. The role of lactate metabolism in heart failure and cardiogenic shock: clinical insights and therapeutic implications. *J Card Fail*. Published online January 29, 2025. https://doi.org/10.1016/j.cardfail.2025.01.011
5. Lazzeri C, Valente S, Chiostri M, Gensini GF. Clinical significance of lactate in acute cardiac patients. *World J Cardiol*. 2015;7(8):483. https://doi.org/10.4330/wjc.v7.i8.483
6. Cohen RD, Woods HF. Lactic acidosis revisited. *Diabetes*. 1983;32(2):181–191. https://doi.org/10.2337/diab.32.2.181
7. Müller J, Radej J, Horak J, et al. Lactate: the fallacy of oversimplification. *Biomedicines*. 2023;11(12):3192. https://doi.org/10.3390/biomedicines11123192
8. Vincent JL, Dufaye P, Berré J, Leeman M, Degaute JP, Kahn RJ. Serial lactate determinations during circulatory shock. *Crit Care Med*. 1983;11(6):449–451. https://doi.org/10.1097/00003246-198306000-00012
9. Henning RJ, Weil MH, Weiner F. Blood lactate as prognostic indicator of survival in patients with acute myocardial infarction. *Circ Shock*. 1982;9(3):307–315.
10. Ander DS, Jaggi M, Rivers E, et al. Undetected cardiogenic shock in patients with congestive heart failure presenting to the emergency department. *Am J Cardiol*. 1998;82(7):888–891. https://doi.org/10.1016/s0002-9149(98)00497-4
11. Davodian LW, Larsen JKP, Povlsen AL, et al. Timing and causes of death in acute myocardial infarction complicated by cardiogenic shock (from the RETROSHOCK Cohort). *Am J Cardiol*. 2022;171:15–22. https://doi.org/10.1016/j.amjcard.2022.01.050
12. Fuernau G, Desch S, De Waha-Thiele S, et al. Arterial lactate in cardiogenic shock. *JACC Cardiovasc Interv*. 2020;13(19):2208–2216. https://doi.org/10.1016/j.jcin.2020.06.037
13. Scolari FL, Schneider D, Fogazzi DV, et al. Association between serum lactate levels and mortality in patients with cardiogenic shock receiving mechanical circulatory support: a multicenter retrospective cohort study. *BMC Cardiovasc Disord*. 2020;20(1):496. https://doi.org/10.1186/s12872-020-01785-7
14. Jentzer JC, Monroe RE. Acute myocardial infarction cardiogenic shock in younger adults: a patient's experience. *J Card Fail*. 2023;29(1):30–32. https://doi.org/10.1016/j.cardfail.2022.12.001
15. Basir MB, Lemor A, Gorgis S, et al. Early utilization of mechanical circulatory support in acute myocardial infarction complicated by cardiogenic shock: the National Cardiogenic Shock Initiative. *J Am Heart Assoc*. 2023;12(23). https://doi.org/10.1161/jaha.123.031401
16. Marbach JA, Stone S, Schwartz B, et al. Lactate clearance is associated with improved survival in cardiogenic shock: a systematic review and meta-analysis of prognostic factor studies. *J Card Fail*. 2021;27(10):1082–1089. https://doi.org/10.1016/j.cardfail.2021.08.012
17. Marbach JA, Di Santo P, Kapur NK, et al. Lactate clearance as a surrogate for mortality in cardiogenic shock: insights from the DOREMI trial. *J Am Heart Assoc*. 2022;11(6):e023322. https://doi.org/10.1161/jaha.121.023322
18. Fadel RA, Almajed MR, Parsons A, et al. Feasibility and outcomes of a cardiovascular medicine inclusive extracorporeal membrane oxygenation (ECMO) service. *J Soc Cardiovasc Angiogr Interv*. 2024;3(6):101359. https://doi.org/10.1016/j.jscai.2024.101359
19. Laimoud M, Machado P, Lo MG, Maghirang MJ, Hakami E, Qureshi R. The absolute lactate levels versus clearance for prognostication of post-cardiotomy patients on veno-arterial ECMO. *ESC Heart Fail*. 2024;11(6):3511–3522. https://doi.org/10.1002/ehf2.14910
20. Sugimoto M, Takayama W, Inoue A, et al. Impact of lactate clearance on clinical and neurological outcomes of patients with out-of-hospital cardiac arrest treated with extracorporeal cardiopulmonary resuscitation: a secondary data analysis. *Crit Care Med*. 2024;52(7):e341–e350. https://doi.org/10.1097/ccm.0000000000006245
21. Khalife W, Kanwar MK, Abraham J, et al. Association of hemometabolic trajectory and mortality: insights from the Cardiogenic Shock Working Group Registry. *J Card Fail*. 2024;30(10):1196–1207. https://doi.org/10.1016/j.cardfail.2024.06.019
22. Udesen NLJ, Beske RP, Hassager C, et al. Microaxial flow pump hemodynamic and metabolic effects in infarct-related cardiogenic shock: a substudy of the DanGer Shock randomized clinical trial. *JAMA Cardiol*. 2025;10(1):9. https://doi.org/10.1001/jamacardio.2024.4197
23. Ikeda Y, Saku K, Nakata J, et al. Phase-specific hemodynamic criteria and outcomes in patients with cardiogenic shock receiving percutaneous ventricular assist devices. *J Am Heart Assoc*. 2025;14(13). https://doi.org/10.1161/jaha.125.042249
24. Kamolz LP, Andel H, Schramm W, Meissl G, Herndon DN, Frey M. Lactate: early predictor of morbidity and mortality in patients with severe burns. *Burns*. 2005;31(8):986–990. https://doi.org/10.1016/j.burns.2005.06.019
25. Pan J, Peng M, Liao C, Hu X, Wang A, Li X. Relative efficacy and safety of early lactate clearance-guided therapy resuscitation in patients with sepsis: a meta-analysis. *Medicine (Baltimore)*. 2019;98(8):e14453. https://doi.org/10.1097/MD.0000000000014453
26. He S, Wang Y, Wei S, Yang S. Correlation between lactate/albumin ratio and 28-day mortality in sepsis-associated acute kidney injury patients. *Front Med*. 2025;12, 1546112. https://doi.org/10.3389/fmed.2025.1546112
27. Jansen TC, Van Bommel J, Schoonderbeek FJ, et al. Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial. *Am J Respir Crit Care Med*. 2010;182(6):752–761. https://doi.org/10.1164/rccm.200912-1918OC
28. Dettling A, Kellner C, Sundermeyer J, et al. Incidence and predictors of weaning failure from veno-arterial extracorporeal membrane oxygenation therapy in patients with cardiogenic shock. *Eur J Heart Fail*. 2025;27(5):832–841. https://doi.org/10.1002/ejhf.3583
29. Møller JE, Engstrøm T, Jensen LO, et al. Microaxial flow pump or standard care in infarct-related cardiogenic shock. *N Engl J Med*. 2024;390(15):1382–1393. https://doi.org/10.1056/nejmoa2312572
30. Senman B, Jentzer JC, Barnett CF, et al. Need for a cardiogenic shock team collaborative—promoting a team-based model of care to improve outcomes and identify best practices. *J Am Heart Assoc*. 2024;13(6):e031979. https://doi.org/10.1161/jaha.123.031979
31. Tehrani BN, Truesdell AG, Psotka MA, et al. A standardized and comprehensive approach to the management of cardiogenic shock. *JACC Heart Fail*. 2020;8(11):879–891. https://doi.org/10.1016/j.jchf.2020.09.005
32. Basir MB, Kapur NK, Patel K, et al. Improved outcomes associated with the use of shock protocols: updates from the National Cardiogenic Shock Initiative. *Catheter Cardiovasc Interv*. 2019;93(7):1173–1183. https://doi.org/10.1002/ccd.28307
33. Taleb I, Koliopoulou AG, Tandar A, et al. Shock team approach in refractory cardiogenic shock requiring short-term mechanical circulatory support: a proof of concept. *Circulation*. 2019;140(1):98–100. https://doi.org/10.1161/CIRCULATIONAHA.119.040654
34. Lee F, Hutson JH, Boodhwani M, et al. Multidisciplinary code shock team in cardiogenic shock: a Canadian centre experience. *CJC Open*. 2020;2(4):249–257. https://doi.org/10.1016/j.cjco.2020.03.009
35. Papolos AI, Kenigsberg BB, Berg DD, et al. Management and outcomes of cardiogenic shock in cardiac ICUs with versus without shock teams. *J Am Coll Cardiol*. 2021;78(13):1309–1317. https://doi.org/10.1016/j.jacc.2021.07.044
36. Gattani R, Rosner C, Tehrani B. Systems of care approach to the management of cardiogenic shock. *Card Interv Today*. 2023;17(3):63–68.
37. Garan AR, Kataria R, Li B, et al. Outcomes of patients transferred to tertiary care centers for treatment of cardiogenic shock: a Cardiogenic Shock Working Group analysis. *J Card Fail*. 2024;30(4):564–575. https://doi.org/10.1016/j.cardfail.2023.09.003
38. Pawar S, Bansal K, Abbott JD, et al. Transfer to hub hospitals and outcomes in cardiogenic shock. *Circ Heart Fail*. 2025;18(4), e012477. https://doi.org/10.1161/circheartfailure.124.012477
39. Asemota DE, Sharma A, Miller M, et al. Characteristics and outcomes of cardiogenic shock patients undergoing interhospital transfers in a metropolitan setting. *J Am Coll Cardiol*. 2024;83(13):457. https://doi.org/10.1016/s0735-1097(24)02447-1