

POINT COUNTERPOINT

Measuring Door-to-Lactate Clearance in Cardiogenic Shock

Two perspectives on whether door-to-lactate clearance is ready to serve as a standardized metric for contemporary cardiogenic shock care.

With Chirag Mehta, MD; Howard A. Cooper, MD; Srihari S. Naidu, MD; and Bernard S. Kadosh, MD

POINT: DOOR-TO-LACTATE CLEARANCE SHOULD BE INCORPORATED

By Chirag Mehta, MD; Howard A. Cooper, MD; and Srihari S. Naidu, MD

Once regarded as a binary syndrome, cardiogenic shock (CS) is now recognized as a dynamic continuum that requires serial physiologic reassessment. This conceptual shift has been reinforced by evidence demonstrating that progression to higher Society for Cardiovascular Angiography and Interventions (SCAI) SHOCK stages is associated with incrementally worse in-hospital and 1-year mortality, even among patients who survive the initial episode of shock.^{1,2} Consequently, contemporary CS management should prioritize biomarkers that reflect physiologic response to therapy rather than the delivery of interventions. Among these, serial lactate measurements have emerged as particularly informative—initially as a marker of tissue hypoperfusion defining SCAI SHOCK stage C, and subsequently as a dynamic indicator of improving or worsening systemic perfusion.

Multiple contemporary studies have established the prognostic importance of lactate clearance.³⁻¹⁰ In the DOREMI trial, rapid lactate clearance independently predicted early survival, while both the National Cardiogenic Shock Initiative (NCSI) and the Brown University Health registry demonstrated that failure to achieve lactate

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COUNTERPOINT: DOOR-TO-LACTATE CLEARANCE IS A FLAWED METRIC

By Bernard S. Kadosh, MD

Cardiogenic shock (CS) remains one of the most lethal cardiovascular emergencies despite remarkable advances in hemodynamic monitoring, temporary mechanical circulatory support (MCS), and regional systems of care. As health care systems increasingly seek measurable quality indicators, door-to-lactate clearance (DLC) has emerged as a potential performance metric, and its appeal is obvious. Lactate is accessible; a point-of-care gas returns a number within minutes that integrates the net effect of cardiac output, vascular tone, oxygen delivery, and metabolic stress. In sum, lactate is a reasonable proxy for global hypoperfusion in a syndrome that is otherwise difficult to quantify at the bedside. It is inexpensive, rapidly available, reproducible, and universally measured in virtually every emergency department, community hospital, and tertiary referral center.

Given its widespread availability and association with mortality, its attractiveness as a quality metric is understandable. However, while lactate undoubtedly has clinical value, elevating DLC to the status of a quality metric is premature and risks incentivizing the wrong behaviors. In CS, lactate should inform clinical judgment rather than define the outcome.

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clearance was associated with worse outcomes despite timely conventional management.^{6,7,10} Collectively, these observations provide the rationale for door-to-lactate clearance (DLC) as a dynamic and early resuscitation endpoint linking early shock recognition to successful and timely restoration of tissue perfusion.¹¹ Building upon the SCAI paradigm of shock recognition and expeditious management, the first 24 hours after progression to SCAI SHOCK stage C—the “golden day”—has emerged as a critical therapeutic window during which normalization of lactate may serve as a meaningful indicator of successful resuscitation and improved long-term survival.¹² We present our top 10 reasons why DLC should be incorporated into contemporary CS management algorithms.

1. DLC IS ASSOCIATED WITH IMPROVED SURVIVAL

Across contemporary observational CS cohorts, achievement of lactate clearance has been associated with an approximately 20% absolute reduction in in-hospital mortality irrespective of admission SCAI SHOCK stage, representing one of the strongest prognostic signals currently available during early resuscitation.³⁻¹⁰ In the NCSI, survival improved to roughly 80% regardless of the severity of initial presentation when DLC within 24 hours was achieved. Although these observational data do not establish that lactate clearance itself causes improved survival, they suggest that restoring tissue perfusion—as reflected by DLC—within the early period is strongly associated with meaningful improvements in patient outcomes.

2. DLC PRIORITIZES PHYSIOLOGIC RECOVERY TO PREVENT END-ORGAN DYSFUNCTION

Although clinical, laboratory, and hemodynamic parameters guide transitions between SCAI SHOCK stages, contemporary management algorithms remain predominantly defined by procedure-based metrics, including door-to-balloon time, time to mechanical circulatory support (MCS), and early invasive hemodynamic assessment.^{1,13-16} Although these metrics quantify the timely delivery of care, they do not confirm restoration of tissue perfusion. DLC shifts the focus from intervention to resuscitation by providing an objective measure of physiologic response to treatment. Accordingly, serial lactate kinetics offer greater prognostic value than isolated measurements, with persistent or worsening lactatemia indicating ongoing hypoperfusion, whereas declining lactate concentrations reflect improving tissue perfusion and effective resuscita-

tion.^{5,7,8,17} Although interpretation should be individualized in the setting of impaired lactate clearance (eg, advanced hepatic dysfunction) or disproportionate lactate production (eg, isolated regional ischemia), serial lactate measurements obtained every 2 to 3 hours remain a practical guide for tracking resuscitation and avoiding end-organ dysfunction. Importantly, DLC does not introduce a novel metric but rather repurposes a universally obtained laboratory measurement into a standardized, therapy-agnostic endpoint of physiologic recovery.

3. BRIDGING MACRO- AND MICROCIRCULATORY RESUSCITATION

Achieving conventional macrocirculatory targets through hemodynamic surrogates such as mean arterial pressure and cardiac output reflects an increased likelihood of improved tissue perfusion but does not confirm its restoration.¹⁸ In this scenario, hemodynamic targets such as cardiac output and mean arterial pressure may fall within acceptable population-based thresholds yet still fail to provide adequate tissue perfusion for the individual patient. This limitation is exemplified in normotensive CS, in which compensatory vasoconstriction preserves arterial blood pressure despite persistent tissue hypoperfusion.¹⁹⁻²¹ Accordingly, seemingly satisfactory macrocirculatory parameters may not reflect true physiologic perfusion requirements, underscoring the value of DLC as an integrative marker of successful resuscitation across both the macro- and microcirculation. In turn, serial DLC provides an opportunity to dynamically adjust macrocirculatory support in real time to ensure restoration of tissue perfusion.

4. EARLIER SHOCK IDENTIFICATION AND TEAM ACTIVATION

By measuring lactate levels on all patients along the continuum of SCAI SHOCK stages, DLC can complement bedside assessment by identifying occult hypoperfusion despite apparent clinical stabilization, thereby supporting timely subspecialty consultation, intrahospital care escalation for higher-acuity monitoring (ie, intensive care unit [ICU]), and early multidisciplinary shock team activation. DLC therefore reduces therapeutic inertia and shock misclassification, consistent with validated shock algorithms that emphasize rapid diagnosis and multidisciplinary team mobilization.²²⁻²⁶ Specifically, DLC will identify patients with occult hypoperfusion (SCAI SHOCK stage B) who in actuality are SCAI SHOCK stage C, triggering appropriate shock team management and the 24-hour DLC goal.

5. EXPEDITED TRANSFERS FROM SPOKE LOCATIONS

Most patients with CS initially present to referring (spoke) hospitals, making early recognition of ongoing hypoperfusion essential.^{22,23} Failure to achieve lactate clearance despite initial resuscitative efforts (at 2 hours post-SCAI SHOCK C or greater diagnosis) should prompt activation of a multidisciplinary shock team to optimize therapy before the next lactate assessment (at 4-6 hours) or expedite transfer to the regional shock center (at 6-8 hours) when local resources have been exhausted.^{22,27-30} By providing a standardized, objective measure of treatment response, DLC strengthens regional hub-and-spoke systems of care by facilitating timely therapeutic escalation at referring hospitals, informing earlier transfer decisions, and minimizing delays that may otherwise result in patients becoming “no-option” candidates for advanced therapies upon arrival at the hub center. This therapeutic window also provides an opportunity to prevent irreversible end-organ injury and mitigate complications that accrue during prolonged shock, including acute kidney injury, acute cardiogenic liver injury, mesenteric ischemia, limb ischemia, and secondary infections.

6. DLC OPTIMIZES THERAPEUTIC ESCALATION AND DE-ESCALATION

The optimal timing of MCS remains uncertain beyond the general consensus that intervention should occur before irreversible end-organ injury develops.^{24,31} When integrated with baseline SCAI SHOCK stage, invasive hemodynamics, and ventricular phenotype, DLC can help define this therapeutic window to inform the optimal timing and intensity of MCS. For instance, failure to achieve lactate clearance despite escalating pharmacologic support after the second or third lactate assessment may prompt earlier escalation of MCS. Conversely, favorable serial lactate kinetics can guide earlier de-escalation by supporting timely removal of temporary MCS, reducing device-related complications and ICU length of stay.³²⁻³⁴ At the opposite end of the spectrum, persistent or worsening lactatemia despite escalating support may identify patients unlikely to benefit from further advanced therapies, protecting against futile interventions.

7. DLC PROMOTES EQUITABLE CS CARE

DLC is independent of patient characteristics such as sex, race, ethnicity, age, socioeconomic status, or other factors that may inadvertently influence clinical decision-making through implicit bias.³⁵ By anchoring shock diagnosis, escalation, and de-escalation of therapy to a standardized measure of tissue perfusion, DLC can reduce unwarranted variation in care and promote more consistent application of evidence-based therapies across diverse patient populations.

8. ADDITIONAL OR RECURRENT CAUSES OF TISSUE HYPOPERFUSION

DLC may serve as an early indicator that the clinical syndrome has evolved beyond isolated CS. Failure of lactate clearance disproportionate to the conferred pharmacomechanical circulatory support, or recurrent lactatemia following an initially favorable trajectory, should prompt reassessment for evolving mixed cardiogenic-vasodilatory shock and noncardiac complications including acute mesenteric ischemia, hemorrhage, or limb ischemia related to large-bore arterial access.³⁶⁻⁴⁰ In this context, DLC functions as a dynamic physiologic surveillance tool, both at the local and cardiovascular system level.

9. UNIVERSAL ACCESSIBILITY

Lactate can be measured rapidly using widely available point-of-care testing as demonstrated in non-CS resuscitation bundles.^{41,42} By providing a standardized physiologic target, DLC can be seamlessly handed off from emergency medical services to the emergency department, catheterization laboratory, and ICU, with each team in part contributing to lactate clearance within the first 24 hours of CS diagnosis. Importantly, because DLC relies on a widely available biomarker, requires minimal infrastructure, and is therapy agnostic, it represents a highly scalable resuscitation endpoint that can be implemented across diverse health care systems, including resource-limited settings, where therapeutic capabilities may vary but the goal of restoring tissue perfusion remains central by whatever means necessary and available.

10. EMPOWERING TRAINEES AND NURSES DURING THE “GOLDEN DAY” OF CS

The first 24 hours of CS represent the “golden day” of resuscitation, during which timely and calibrated escalation and de-escalation of care have the greatest potential to influence outcomes.^{12,19} Because this critical period spans overnight coverage when staffing may be leaner, often relying on trainees and/or bedside nurses, serial lactate assessment provides these team members with an objective framework to determine whether tissue perfusion is truly improving. In this setting, DLC functions as an educational and clinical tool, enabling early recognition of inadequate resuscitation and timely reassessment of management strategy, including early contact with covering attendings and the shock team when lactate is not clearing.

CONCLUSION

No single measure is without limitations. However, few measures are as widely available, inexpensive, rapidly obtainable, and reproducible as lactate. As an objective marker that tracks tissue perfusion, correlates with both

short- and long-term outcomes, and can be reassessed every 2 to 3 hours to guide timely therapeutic adjustments, DLC offers a practical and scalable framework for contemporary CS management. As CS management continues to evolve toward precision, protocolized care, DLC has the potential to become a unifying resuscitation target that complements existing therapies on the ultimate goal of restoring tissue perfusion. ■

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THE CASE AGAINST DLC

The appeal of lactate is also its weakness. It provides a broad assessment of systemic metabolism but is agnostic to etiology. Elevated lactate is often interpreted as evidence of worsening CS, but numerous competing processes can produce identical results. Sepsis, hemorrhage, hypovolemia, limb ischemia, mesenteric ischemia, increased work of breathing, seizures, hepatic dysfunction, and medications can all drive up the lactate levels independent of cardiac output.¹ These conditions frequently coexist in critically ill patients, sometimes as a consequence of our interventions.

This etiologic ambiguity has downstream consequences. Targeting lactate clearance may drive decision-making toward inappropriate interventions such as premature initiation of temporary MCS, unnecessary renal replacement therapy, or device escalation without an understanding of the patient's hemodynamic profile. The diagnostic work in CS involves understanding the physiologic perturbations specific to the patient in front of you, which should inform decisions regarding the appropriate medications, device selection, timing, and configuration of MCS. Lactate should complement that assessment but should not supersede it.^{2,3}

More fundamentally, lactate is a marker of risk, not a validated mediator of outcomes. The literature linking lactate clearance to survival in shock is almost entirely observational.⁴ These studies consistently show that lactate clears in patients who survive, not that clearing lactate causes survival. Lactate appears to normalize in recovering patients as their underlying shock resolves, either because of or despite the interventions employed. Conversely, a severely elevated lactate in some patients may instead reflect a degree of advanced multisystem injury from which recovery is not realistic, making it a marker of futility rather than a target for intervention. No randomized prospective trial has demonstrated that actively targeting lactate clearance improves outcomes.

Quality metrics should satisfy several fundamental characteristics: They should (1) represent outcomes that matter to patients, (2) be valid and reliable, (3) reflect a disease-specific process, (4) be modifiable through improvements in care, and (5) permit meaningful risk adjustment across all levels of illness and complexity. DLC struggles on each of these dimensions. Lactate trajectories are dynamic, nonlinear, and influenced by numerous factors throughout a cardiac intensive care unit admission. It is a marker that reflects the complexity of critical illness rather than the adequacy of shock management. Moreover, no robust risk-adjustment model exists for CS outside of the SCAI SHOCK staging system, which itself incorporates lactate. Using lactate both

to classify illness severity and judge hospital performance introduces a circularity that complicates fair comparisons. In addition, reliably capturing and reporting serial lactate measurements imposes yet another administrative burden on resource-constrained health systems already beholden to extensive regulatory metrics.

Finally, outcome metrics cannot be equitably applied without accounting for differences in hospital capability. Rural and spoke hospitals often encounter patients at vastly different points along their shock trajectory. Many lack immediate access to advanced imaging, invasive hemodynamics, MCS, or multidisciplinary shock teams. Even among tertiary and quaternary referral centers, CS management remains remarkably heterogeneous and is guided more by institutional patterns than high-quality randomized evidence.⁵ Penalizing institutions based on lactate clearance risks measuring differences in available resources rather than differences in quality of care.

LOOKING AHEAD

Despite many advances in the last decade, CS care remains in a formative stage. We still lack robust tools to recognize shock early, match interventions precisely to hemodynamic phenotype, know when and how to de-escalate, and minimize the iatrogenic complications that so often undermine survival and quality of life. Our focus should be on generating quality evidence that addresses these questions, including whether lactate-driven care changes outcomes, before we hold institutions accountable to it. While the proverbial road to hell is often paved by good intentions, it is almost always lit by bad incentives. ■

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