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RESEARCH

Postoperative Hematocrit and Lactate Trajectory After Neonatal Cardiopulmonary Bypass: A Retrospective Cohort Study

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Abstract

Lactate clearance is a key marker of oxygen delivery after neonatal cardiac surgery. While higher hematocrit (HCT) is often targeted to optimize oxygen delivery, its relationship with postoperative lactate clearance remains unclear. We hypothesized that supraphysiologic initial HCT may decrease lactate clearance. To assess whether higher initial postoperative HCT is associated with delayed lactate clearance. We performed a retrospective cohort study of 201 neonates requiring CPB. Clinical data were extracted from the EHR using standardized PC4 and STS definitions. The primary predictor was initial HCT analyzed as a continuous variable. Outcomes were lactate at 1, 6, 12, and 24 h and lactate trajectories over 24 h. Multivariable linear regression adjusted for demographics, surgical complexity, and CPB time. Linear mixed-effects models assessed longitudinal lactate trajectories. Initial postoperative HCT (median = 51, IQR = 47–56) was not associated with post-operative lactate after adjustment. However, each 10% increase in HCT was associated with higher lactate at 1 h (adjusted β = 0.40 mmol/L, 95% CI 0.03–0.80; $p = 0.03$). In mixed-effects models, HCT was not associated with lactate trajectories in the primary model but was associated after adjustment for time-varying variables (β = 0.29, 95% CI 0.03–0.55; $p = 0.03$). Higher initial postoperative HCT was not associated with lactate, but it was associated with lactate trajectories when adjusting for time-varying variables. Further studies are needed to define optimal transfusion strategies in this population, and caution may be indicated when increasing HCT to supra-physiological levels.

Keywords Neonatal cardiology · Cardiopulmonary bypass · Microcirculation · Hematocrit · Cardiac critical care · Lactate · Cardiac surgery

Introduction

The immediate perioperative care of neonates undergoing repair of complex congenital heart disease requiring cardiopulmonary bypass demands meticulous management to optimize oxygen delivery and maintain an adequate balance between oxygen delivery and consumption. This includes administration of blood products and other fluids and titration of vasoactive medications. In this setting, lactate is widely regarded as a marker of adequate tissue oxygen delivery (DO_2) and changes in lactate often guide our therapeutic interventions. Importantly, following cardiopulmonary bypass (CPB) for neonatal congenital cardiac defect repairs, impaired lactate clearance is associated with increased morbidity and mortality [1–3]. Furthermore, it has been shown that lactic acidosis is associated with poor neurodevelopmental outcome and may be a surrogate biomarker for ischemic brain injury [4].

As the major oxygen carrier in the body, hemoglobin is frequently manipulated in the perioperative period [5] to increase oxygen content [6] and thereby oxygen delivery. While higher HCT may increase oxygen-carrying capacity, extreme levels increase blood viscosity and impair microcirculatory flow [7–9], potentially limiting effective DO_2 [10] despite higher arterial oxygen content. Additionally, stored red blood cells have decreased levels of 2,3-DPG and

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a left-shift in the oxyhemoglobin dissociation curve potentially further limiting effective DO_2 [11, 12].

Despite its clear biologic plausibility, the relationship between postoperative HCT and metabolic recovery has not been thoroughly investigated. Prior studies have examined these variables in isolation. For example, investigations of HCT have mostly centered on vascular and endothelial consequences, including the effects of hemodilution in the setting of CPB, while studies of lactate have focused on its overall association with ICU outcomes. However, the potential relationship between post-operative hematocrit levels and lactate clearance as a surrogate for adequate tissue oxygen delivery has not been investigated.

We hypothesized that higher initial HCT may impair microcirculatory blood flow through increased fluid viscosity, potentially decreasing DO_2, leading to increased lactate buildup in the initial 24-hour postoperative period. Thus, we retrospectively evaluated the association between postoperative HCT measured at the first CICU-hour and lactate dynamics in neonates undergoing CPB. In addition, we explored its longitudinal relationship with lactate trajectories across all CICU timepoints within that 24-hour window.

Methods

We performed an observational retrospective cohort study of 201 neonates requiring CPB from 2017 to 2021 at the University of California San Francisco's Benioff Children's Hospital at Mission Bay. The target population included neonates undergoing surgical repair of congenital cardiac defects at UCSF Children's Hospitals. Patients were identified through the Pediatric Cardiac Critical Care Consortium (PC4) and Society of Thoracic Surgeons (STS) registries. Patients with missing CPB time and aortic cross-clamp time were excluded from the analytic cohort. For regression analyses, complete case analysis was used for each model, and sample sizes therefore varied depending on data availability. Clinical data were extracted through manual chart review from the electronic health record (EHR) utilizing the APeX system. Baseline characteristics included age and weight prior to surgery, gestational age, STAT category, CPB duration, perioperative red blood cell (RBC) transfusion volume (over first 24 postoperative hours), urine output (over first 24 postoperative hours), perioperative intravenous fluid administration (over first 24 postoperative hours), cerebral regional oxygen saturation (rSO_2) measured by near-infrared spectroscopy (NIRS), mean arterial pressure (MAP), bicarbonate, and glucose. Additionally, time-varying factors were extracted including total bilirubin, AST, capillary refill time, creatinine, blood urea nitrogen, albumin, INR, pH, pCO_2, pO_2, systolic and diastolic blood pressures, heart rate and central venous pressure at prespecified timepoints (closest to preoperative day (+/- 1 day), closest to 1, 6, 12 and 24- hour postoperatively (+/- 3 h).

The primary exposure was initial postoperative HCT upon arrival in the CICU (at 1 h). HCT was analyzed as a continuous variable scaled per 10% increase, and we also performed analysis using HCT as a binary variable with a cut-off of 50% [7] ($\geq$ 50% vs. < 50%) and cohort-based quartiles. The cut-off of 50% was chosen based on current literature, demonstrating nonlinear increases in blood viscosity with increasing HCT and the resulting effects on microvascular resistance and blood flow [13, 14].

The primary outcomes were lactate concentrations and trends. First, we assessed absolute lactate concentrations in mmol/L at 1, 6, 12, and 24 h postoperatively to capture early and evolving metabolic changes during the immediate postoperative period. Second, we assessed lactate trends across the first 24 postoperative CICU hours using mixed effect models. Third, we defined lactate clearance as [(lactate at 1 h) – (lactate at *t*)/lactate at 1 h] x 100% [15], where $t = 6$, 12 and 24 postop hours.

The primary analytical objective was to evaluate the association between initial postoperative HCT and absolute lactate concentrations at each postoperative timepoint within the first 24 CICU hours. Multivariable linear regression models were constructed to evaluate the associations between continuous initial HCT and continuous absolute lactate concentrations at 1, 6, 12, and 24 postoperative CICU hours. Important covariates were selected a priori based on clinical relevance and a directed acyclic graph (DAG, Fig. 1) and included postnatal age at surgery (continuous, days), weight at surgery (continuous, kg), STAT category (ordinal, 1–5), and CPB time (continuous, mins). We then included total perioperative RBC transfusion volume, total intravenous fluid administration over the first 24 postoperative hours, and urine output over the first 24 postoperative hours—weight-normalized where appropriate—into an expanded multivariable model for the 24 h timepoint.

Since lactate measurements were repeatedly obtained within patients over time, we then constructed linear mixed-effects models to capture potential postoperative lactate trajectories across the 24-hour CICU period. We included patient-level random intercepts to account for within-patient correlation and allow individual baseline lactate variation. Time was modeled as a categorical variable with 1 h as the reference point. Fixed effects included initial hematocrit and baseline covariates. We also adjusted for time-varying physiologic variables including cerebral regional oxygen saturation (rSO_2, %), mean arterial pressure (MAP, mmHg), bicarbonate (mmol/L) and glucose (mg/dL).

We then assessed lactate clearance at 6, 12 and 24 postoperative hours. We evaluated the association between

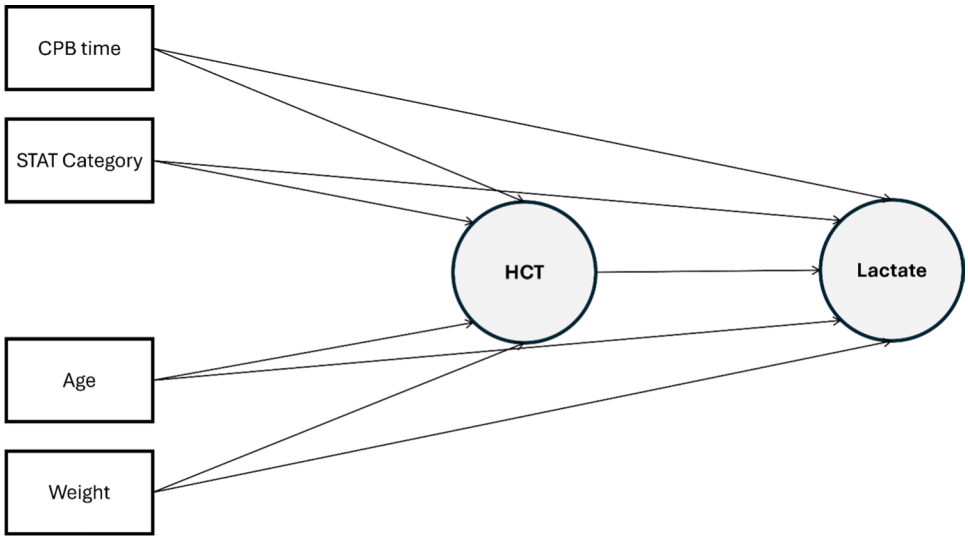

Fig. 1 Directed acyclic graph depicting the temporal structure of baseline and intraoperative variables in relation to initial postoperative HCT and subsequent lactate measurements. Age, weight, STAT category, and CPB duration occur prior to the postoperative exposure and were included as baseline covariates in primary models. *CPB = cardiopulmonary bypass; STAT = Society of Thoracic Surgeons–European Association for Cardio-Thoracic Surgery mortality category; HCT = hematocrit; CICU = cardiac intensive care unit

initial postoperative HCT and lactate clearance using separate multivariable linear regression models. Lactate clearance (% change) at each timepoint was modeled as a continuous outcome, with initial HCT per 10% increase as the primary predictor. Adjusted models included the same baseline covariates as the primary analysis, including postnatal age at surgery, weight at surgery, STAT category, and CPB duration.

Finally, we conducted analyses to evaluate potential threshold or nonlinear relationships between HCT and lactate dynamics. For this, we included categorical modeling of HCT using a predefined cutoff (≥50% vs. < 50%) and cohort-based quartiles of initial postoperative hematocrit, defined by dividing the study population into four groups based on the distribution of HCT values (25th, 50th, and 75th percentile cut points), with the lowest quartile serving as the reference group. For these analyses, we also used multivariable linear regression models with lactate as the outcome and HCT categories as the primary predictor, adjusting for the same baseline covariates as the primary analysis.

All analyses were performed using R version 4.5.0. All statistical tests were two-sided, with a prespecified α level of 0.05 considered statistically significant. The data were collected and used after the study protocol got approved by the institutional review board.

Results

A total of 201 neonates undergoing CPB surgery were included in the analysis as shown in Table 1. The median age at surgery was 6 days (IQR 2–10), and median weight at surgery was 3.26 kg (IQR 2.79–3.67). Surgical complexity was broadly distributed across STAT categories, with almost half (49.3%) of the patients in STAT categories 4–5. Median CPB time was 160 min (IQR 133–203), and median aortic cross-clamp time was 81 min (IQR 63–115). 14.4% of the patients were born preterm. There was a diverse range of congenital cardiac diagnoses, most commonly transposition of the great arteries (22.9%) and single ventricle physiology (20.4%). Early postoperative low cardiac output syndrome was documented in 6% of patients, while 3.5% required full extracorporeal membrane oxygenation.

In unadjusted univariable linear regression models, higher initial postoperative HCT at the first CICU hour was associated with higher absolute lactate at 1 h following CICU admission (β = 0.47 mmol/L per 10% increase in initial HCT, 95% CI 0.06–0.88; $p = 0.025$). (Table 2 and supplemental Fig. 1). There was no statistically significant association at 6, 12, or 24 h. After adjustment for age and weight at surgery, STAT category, and CPB time, the association between HCT and lactate at 1 h remained significant, β = 0.40 mmol/L per 10% increase in initial HCT, 95% CI 0.03–0.8; $p = 0.03$. Similar to the unadjusted models, no significant associations were observed at 6, 12, or 24 h in adjusted models (Table 2). Further adjustment for perioperative RBC transfusion (ml/kg), postoperative intravenous fluid administration (ml/kg/24 h) in the first 24 h, and urine output (ml/kg/hr) in the first 24 h, did not alter the findings at the 24th CICU hour, with no significant association between initial HCT and lactate (β = −0.04 mmol/L per 10% increase in initial HCT, 95% CI −0.4 to 0.3; $p = 0.8$).

In the linear mixed-effects model, higher initial HCT was not associated with early elevation in lactate, and not with sustained differences over time ($p > 0.05$). However, when incorporating time-varying parameters—rSO_2, mean arterial pressure (MAP), bicarbonate (HCO_3), and blood glucose for each CICU timepoint—a statistically significant association between higher initial HCT and increased

Table 1 Baseline demographic and perioperative characteristics for neonates undergoing cardiopulmonary bypass surgery for congenital heart defect repair ($N=201$)

Continuous variables	Median (IQR)	(IQR)
Age at surgery (days)	6	(2–10)
Weight at surgery (kg)	3.26	(2.79–3.67)
CPB time (min)	160	(133–203)
Aortic X clamp time (min)	81	(63–115)
Categorical variables	**n**	**Percentage %**
Gender	129	64.2%
Males		
Females	72	35.8%
Prematurity		
Yes	29	14.4%
No	171	85.1%
Unknown	1	0.5%
Diagnosis Group		
TGA	46	22.9%
Single Ventricle	41	20.4%
TAPVC	28	13.9%
TOF/PA	24	11.9%
Arch/CoA/IAA	23	11.4%
DORV	19	9.5%
Truncus	8	4%
Other	12	6%
STAT Category		
1	20	10%
2	46	36%
3	36	17.9%
4	47	23.4%
5	52	25.9%
LCOS within 2 h of CICU admission	12	6%
ECMO	7	3.5%

CPB = cardiopulmonary bypass; TAPVC = total anomalous pulmonary venous connection; TOF/PA = tetralogy of Fallot with pulmonary atresia; Arch/CoA/IAA = aortic arch hypoplasia/coarctation/interrupted aortic arch; DORV = double-outlet right ventricle; STAT = Society of Thoracic Surgeons–European Association for Cardio-Thoracic Surgery mortality category; LCOS = low cardiac output syndrome, as per PC4 database definition; ECMO = extracorporeal membrane oxygenation

Table 2 Linear regression models displaying associations between initial postoperative HCT at 1 st CICU hour and absolute lactate concentrations at 1, 6, 12, and 24 postoperative CICU hours ($n=201$)

CICU hour	Unadjusted β	*p* value	Adjusted β	*p* value
1st	0.5 [0.06, 0.9]	0.03	0.4 [0.03, 0.8]	0.03
6th	0.3 [−0.06, 0.7]	0.1	0.3 [−0.1, 0.6]	0.2
12th	0.3 [−0.04, 0.7]	0.08	0.2 [−0.2, 0.6]	0.3
24th	0.2 [−0.13, 0.5]	0.2	0.09 [−0.2, 0.4]	0.6

HCT = hematocrit %; CICU = cardiac intensive care unit; CPB = cardiopulmonary bypass; STAT, Society of Thoracic Surgeons–European Association for Cardio-Thoracic Surgery mortality category

Unadjusted and adjusted β coefficients—represented by lactate in mmol/L per 10% increase in initial HCT—are shown with 95% confidence intervals and corresponding p-values. Adjusted models include age and weight at surgery, STAT category, and CPB duration

Table 3 Primary and sensitivity linear mixed-effects models evaluating postoperative lactate trajectories over the first 24 CICU hours

Variable	Baseline model		Model including time varying covariates	
	β	*p* value	β	*p* value
Initial HCT (per 10% increase)	0.24 (−0.05, 0.53)	0.11	0.29 (0.03, 0.55)	0.03
Lactate change at 6 h vs. 1 h	−0.51 (−0.73, −0.29)	<0.001	−0.40 (−0.63, −0.17)	<0.001
Lactate change at 12 h vs. 1 h	−1.23 (−1.50, −1.00)	<0.001	−0.70 (−1.00, −0.43)	<0.001
Lactate change at 24 h vs. 1 h	−2.31 (−2.50, −2.10)	<0.001	−1.40 (−1.80, −1.10)	<0.001
Weight (per kg)	−0.39 (−0.60, −0.17)	<0.001	−0.23 (−0.43, −0.03)	0.02
Age (per day)	−0.06 (−0.10, −0.03)	<0.001	−0.06 (−0.09, −0.04)	<0.001
STAT 2 vs. STAT 1	−0.07 (−0.83, 0.70)	0.85	0.20 (−0.50, 0.90)	0.60
STAT 3 vs. STAT 1	0.16 (−0.63, 0.90)	0.70	0.28 (−0.50, 1.00)	0.50
STAT 4 vs. STAT 1	0.40 (−0.37, 1.13)	0.30	0.52 (−0.16, 1.21)	0.10
STAT 5 vs. STAT 1	0.42 (−0.36, 1.19)	0.30	0.50 (−0.26, 1.19)	0.20
CPB (per 10 min)	0.05 (0.01, 0.08)	<0.01	0.04 (0.02, 0.07)	<0.05
HCO_3 (per 10 mmol/L)	–	–	−0.20 (−0.60, 0.20)	0.30
Glucose (per 10 mg/dL)	–	–	0.07 (0.05, 0.09)	<0.001
MAP (per 10 mmHg)	–	–	−0.04 (−0.10, 0.06)	0.40
rSO_2 (per 10%)	–	–	−0.22 (−0.31, −0.12)	<0.001

The primary model includes initial postoperative hematocrit and baseline covariates (age, weight, STAT category, CPB duration). The sensitivity model additionally incorporates time-varying physiologic variables (rSO_2, MAP, bicarbonate, glucose) measured at each postoperative timepoint to reflect contemporaneous postoperative conditions. Values shown are β coefficients with 95% confidence intervals and p-values

— = Variable not included in the primary model

HCT = hematocrit %; CICU = cardiac intensive care unit; CPB = cardiopulmonary bypass; STAT = Society of Thoracic Surgeons–European Association for Cardio-Thoracic Surgery mortality category; MAP = mean arterial pressure; rSO_2 = regional oxygen saturation through near-infrared spectroscopy; HCO_3 = bicarbonate

lactate trajectory over time emerged (Table 3; Fig. 2); β = 0.29 mmol/L increase in overall 24-hour lactate trajectory per 10% increase in initial HCT (95% CI 0.03 to 0.55; $p=0.03$).

However, initial HCT was not associated with lactate clearance at 6, 12, or 24 h. In adjusted models, each 10%

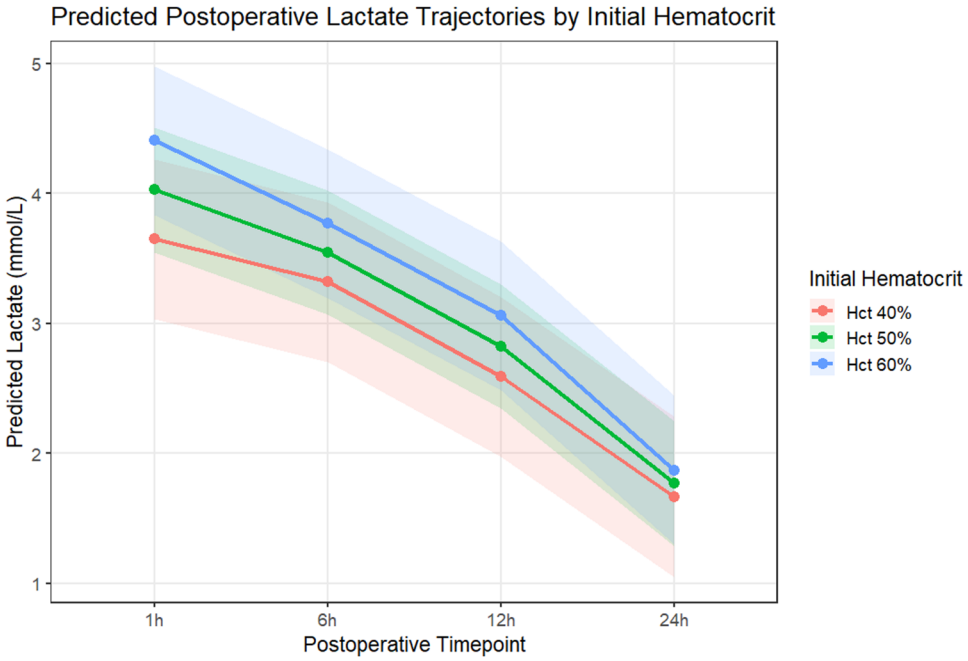

Fig. 2 Predicted postoperative lactate trajectories over the first 24 CICU hours. Linear mixed-effects model including initial postoperative HCT, patient-level random intercepts, and baseline covariates (age, weight, STAT category, and CPB duration). Curves represent predicted lactate concentrations for initial hematocrit values of 40%, 50%, and 60%, with shaded regions indicating 95% confidence intervals. *CPB = cardiopulmonary bypass; STAT = Society of Thoracic Surgeons–European Association for Cardio-Thoracic Surgery mortality category; HCT = hematocrit; CICU = cardiac intensive care unit

increase in hematocrit was associated with a 1.55% change in lactate clearance at 6 h (95% CI −6.94 to 10.03; $p = 0.72$), a 0.04% change at 12 h (95% CI −8.85 to 8.93; $p = 0.99$), and a 1.49% change at 24 h (95% CI −4.98 to 7.95; $p = 0.65$).

In addition, when HCT was analyzed as a binary or categorical variable, no association was observed between higher HCT and lactate at 24 h. Patients in the highest quartile of HCT (absolute HCT 56–67%) did not demonstrate significantly lower lactate levels compared to those in lower quartiles (supplemental Table 1).

Discussion

In this retrospective cohort study, we aimed to investigate whether high initial post–cardiopulmonary bypass hematocrit in neonates is associated with higher lactate concentrations over the first 24 h after surgery. We used a thorough statistical approach with multivariable logistic models at multiple time points, assessed lactate trends using mixed-effects models, and evaluated hematocrit as a continuous, binary, and categorical predictor. The only statistically significant finding was a very small effect observed in the mixed-effects model incorporating time-varying parameters. However, even in this model, the clinical effect size was small, with a 0.29 mmol/L increase in the overall 24-hour lactate trajectory per 10% increase in initial hematocrit, and residual confounding cannot be fully excluded in this retrospective study. Overall, our results do not support our hypothesis that high initial hematocrit is associated with higher lactate concentrations and delayed lactate clearance over the first 24 h after cardiac surgery in neonates with congenital heart disease.

The hypothesis that supraphysiologic hematocrits, often targeted following neonatal congenital heart surgery, could impair microcirculatory blood flow resulting in impaired lactate clearance is supported by a growing body of literature examining the rheologic and microvascular consequences of elevated HCT. Both experimental and clinical studies consistently demonstrate that increases in HCT lead to exponential increases in blood viscosity, particularly above thresholds of approximately 50% [16]. At lower shear rates, which are characteristic of the microcirculation, this increased viscosity promotes red blood cell aggregation and impairs capillary flow [17].

Beyond viscosity and fluid dynamics alone, elevated HCT has been shown to disrupt the structure and function of the endothelial glycocalyx leading to alterations at the blood–endothelium interface that may impair perfusion and promote inflammation [9]. In vivo imaging studies further demonstrate that higher HCT increases capillary flow heterogeneity, resulting in uneven distribution of perfusion across microvascular networks and the development of micro-regional hypoxia despite preserved or increased bulk red blood cell flux [18, 19].

A clinical study in neonates provides additional support for this potential mechanism. Polycythemic infants exhibit reduced capillary blood flow velocity and impaired microvascular perfusion, both of which improve following hemodilution or partial exchange transfusion [20]. Notably, these improvements occur without significant changes in systemic hemodynamics, suggesting that

microvascular resistance, rather than global circulatory parameters, is the primary limiting factor [21, 22].

Although higher HCT was not associated with impaired lactate clearance in our study, the lack of association between higher HCT and improved lactate clearance is also noteworthy. In fact, the observed positive association with higher early absolute lactate concentrations and lactate trajectories over-time, suggests that increasing oxygen-carrying capacity with supraphysiologic levels of hemoglobin does not always translate into improved tissue oxygenation [23] in this population. In other words, a supraphysiological HCT might not be impacting lactate clearance, but its benefits with regards to improved tissue oxygenation remains unclear.

This study has several limitations. First, its observational retrospective-cohort design limits the ability to establish true causal relationships and introduces the potential for residual confounding. Although both, multivariable and mixed effect models, along with sensitivity analyses were performed, unmeasured confounders may persist. Moreover, a single-center design minimizes the findings' overall generalizability. For example, the cardiopulmonary bypass times and the aortic cross clamp times are relatively short in our study cohort. These times reflect case mix and surgical approach and might impact the lactate burden. Second, although lactate is a widely used marker of metabolic stress, it is influenced by factors beyond tissue hypoxia, including hepatic function [24]. Although liver enzyme data were collected, they were not included in the final models due to missingness and the possibility that they may lie along the causal pathway. Third, the analysis included 7 patients who received postoperative ECMO support. ECMO may introduce confounders that may affect both hematocrit management and lactate kinetics, including transfusion exposure, hemodilution, circuit-related hemolysis and inflammation and changing lactate clearance. Fourth, the sensitivity analyses incorporating time-varying physiologic variables were limited by missingness, resulting in smaller sample sizes and reduced statistical power. Additionally, some of the physiologic variables used in the mixed-effects models might be mediators or on the causal pathway rather than confounders, this could falsely lead to an underestimation of the effect of hematocrit on lactate clearance.

In summary, higher postoperative HCT was not associated with improved metabolic recovery and was associated with higher early absolute lactate levels. When accounting for time-varying physiologic factors, higher HCT was associated with a small decrease in lactate clearance over 24 h, suggesting a dynamic and context-dependent relationship. These findings challenge the assumption that increasing HCT uniformly improves oxygen delivery and highlight the importance of considering microcirculatory function in the management of postoperative neonatal cardiac patients. Further prospective studies integrating physiologic monitoring and direct assessment of microcirculation are needed to define optimal hematocrit targets and refine transfusion strategies in this population. Moreover, caution may be indicated when increasing HCT to supra-physiological levels.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00246-026-04399-6.

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Author Contributions Matee Rehman contributed to study design, data collection, data extraction and cleaning, statistical analysis, data visualization, and manuscript drafting. Mikiko Whitehorn contributed to data collection and data extraction. Dara Torgerson contributed to the development of the statistical analysis plan. Jeffrey Fineman contributed to study conception and design, data collection, manuscript drafting, and overall supervision. Martina Steurer contributed to data collection, development of the statistical analysis plan, statistical analysis, manuscript drafting, and overall supervision. All authors reviewed and approved the final manuscript.

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Data Availability Deidentified data may be available from the corresponding author upon request and with appropriate institutional approvals.

Declarations

Conflict of interest The authors declare no competing interests.

Ethical Approval This study was approved by the Institutional Review Board at the University of California, San Francisco, and was conducted in accordance with the declaration of Helsinki.

Consent to Participate The requirement for informed consent was waived by the Institutional Review Board because of the retrospective nature of the study.

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