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Metabolic Dynamics During Cardiopulmonary Bypass: Oxygen Delivery, Carbon Dioxide Production, and Lactate Response Despite Preserved Global Perfusion

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Abstract

Objective

To analyze intraoperative metabolic parameters from carbon dioxide during cardiopulmonary bypass (CPB) and their association with postoperative hyperlactatemia in elective cardiac surgery.

Methods

This study observed 26 adult patients undergoing cardiac surgery with CPB. Blood gases were collected at specific times: after cardioplegia and during rewarming at 32°C. It calculated oxygen delivery (DO_2), consumption (VO_2), carbon dioxide production (VCO_2), respiratory quotient (RQ), and the DO_2/VCO_2 ratio. Lactate levels were measured during and up to 24 hours post-surgery. Paired t-tests compared intraoperative data, and repeated-measures ANOVA analyzed lactate and hemoglobin over time.

Results

Indexed DO_2 stayed above critical thresholds during CPB (265 ± 60 vs. 272 ± 52 mL/min/m², $p = 0.48$). VO_2 increased during rewarming (36 ± 14 to 52 ± 16 mL/min/m², $p < 0.001$), while VCO_2 did not change significantly ($p = 0.62$). RQ decreased from 2.00 ± 0.80 to 1.50 ± 0.50 ($p = 0.012$). The DO_2/VCO_2 ratio showed a non-significant downward trend ($p = 0.18$). Lactate levels rose over time, peaking 30 minutes after protamine at 3.9 ± 1.2 mmol/L and remained high at 24 hours.

Conclusions

Hyperlactatemia occurred despite maintained oxygen delivery during CPB. During rewarming, oxygen consumption rose, and metabolic reactivation was indicated by changes in RQ. While CO_2 variables reflected these changes, they couldn't reliably predict increases in lactate. A multimodal monitoring approach combining oxygen transport and metabolic markers is needed, rather than relying on a single perfusion threshold.

INTRODUCTION

Cardiopulmonary bypass (CPB) enables extracorporeal circulation during cardiac surgery but causes significant changes in systemic hemodynamics, oxygen transport, and cellular functions metabolism.[1] Despite advances in pump technology and membrane oxygenation, CPB remains associated with hemodilution, hypothermia, nonpulsatile flow, systemic inflammatory activation, and microvascular perfusion heterogeneity, all of which may impair effective tissue oxygen utilization.[2,3]

Postoperative hyperlactatemia is frequently observed after cardiac surgery with CPB and is associated with increased morbidity and mortality, including acute kidney injury, prolonged mechanical ventilation, and extended intensive care unit stay.[4–6] Elevated lactate levels have traditionally been interpreted as markers of inadequate oxygen delivery (DO_2) leading to anaerobic metabolism. Consequently, goal-directed perfusion (GDP) strategies have emphasized maintenance of indexed DO_2 (iDO_2) above critical thresholds—commonly 260–280 mL/min/m²—to reduce postoperative organ dysfunction.[7–9] Recent systematic reviews confirm that lower intraoperative DO_2 during CPB is consistently associated with higher rates of acute kidney injury and lactate elevation.[9]

However, lactate kinetics during and after CPB are multifactorial. Besides the oxygen supply–demand imbalance, factors leading to hyperlactatemia include catecholamine-driven glycolysis, inflammatory responses, mitochondrial dysfunction, reperfusion physiology, and impaired lactate clearance.[5,10]Contemporary understanding recognizes that lactate elevation can occur even when overall oxygen delivery is maintained, reducing its specificity as a sole marker of intraoperative hypoxia.[10]

Variables derived from carbon dioxide offer a supporting physiological perspective. These include the veno-arterial carbon dioxide gradient ($Pv–aCO_2$), carbon dioxide production (VCO_2), respiratory quotient ($RQ = VCO_2/VO_2$), and ratios that combine oxygen delivery with CO_2 production (such as DO_2/VCO_2). These markers have been suggested to indicate tissue hypoperfusion and metabolic stress in critical care settings.[11–13] In the context of CPB, recent studies have examined the relationship between CO_2-derived indices and hyperlactatemia, indicating potential usefulness in detecting early metabolic changes imbalance.[14] Nonetheless, emerging data suggest that these parameters may have limited ability to predict major postoperative complications after cardiac surgery.[15]

Furthermore, the metabolic transitions occurring during rewarming—a phase characterized by restoration of enzymatic activity and temperature-dependent increases in oxygen consumption—have not been systematically examined in relation to intraoperative CO_2 kinetics and postoperative lactate dynamics. A more integrated evaluation of oxygen- and CO_2-derived variables during CPB may therefore improve the interpretation of metabolic alterations observed in the perioperative period.

Study Hypotheses

1. CO_2-derived variables (VCO_2, RQ, DO_2/VCO_2) change significantly during CPB, particularly during rewarming.
2. Alterations in these indices are temporally associated with postoperative lactate elevation.
3. CO_2-derived parameters provide complementary information to indexed DO_2 in detecting intraoperative metabolic imbalance.

METHODS

Study design and ethical approval:

This prospective observational study was conducted within the Department of Cardiothoracic and Vascular Surgery at a tertiary care teaching hospital. In compliance with the Declaration of Helsinki and with authorization from the Institutional Human Ethics Committee (Approval No.; CSP-□/24/APR/04/153), the study was executed. Participants were informed in the preoperative area, and written consent was secured from all involved.

Patient Population:

Adult patients scheduled for elective cardiac surgery with cardiopulmonary bypass were screened over a three-month period. A total of twenty-six patients were included.

Inclusion Criteria:

- Age ≥20 years
- Elective coronary artery bypass grafting (CABG), valve surgery, or combined procedures
- Requirement for CPB

Exclusion Criteria:

- Emergency surgery
- Preoperative lactate >2 mmol/L
- Hemoglobin <8 g/dL
- Known renal or hepatic dysfunction
- Pre-existing metabolic disorders affecting lactate metabolism
- Preoperative mechanical circulatory support

These criteria were used to reduce confounding factors that might affect oxygen transport or lactate kinetics.

Anaesthetic management:

The administration of general anesthesia was conducted in accordance with institutional protocols. Following induction and tracheal intubation via laryngoscopy, patients were mechanically ventilated using volume-controlled ventilation to sustain normocapnia. Standard monitoring comprised electrocardiography, invasive arterial pressure measurement, pulse oximetry, nasopharyngeal temperature assessment, and urine output monitoring. Systemic anticoagulation was accomplished through the administration of intravenous heparin at a dose of 400 IU/kg to maintain an activated clotting time exceeding 480 seconds prior to the initiation of cardiopulmonary bypass.

CPB management:

CPB was established using:

- Non-pulsatile roller pump
- Hollow-fibre membrane oxygenator
- Crystalloid-primed circuit
- Standard arterial line filter

Arterial cannulation was performed in the ascending aorta. Venous drainage was achieved via right atrial or bicaval cannulation, depending on the procedure

Perfusion targets

- Indexed pump flow: 2.4 L/min/m²
- Mean arterial pressure: 60–80 mmHg
- Hematocrit during CPB: 22–25%
- Systemic temperature: 28–32°C (moderate hypothermia)

Packed red blood cells were administered as required to maintain oxygen-carrying capacity. Hemofiltration was used at the perfusionist's discretion for volume management.

Myocardial protection was achieved with intermittent cold-blood cardioplegia. Rewarming was performed gradually to normothermia prior to separation from CPB.

Protamine sulfate (1–1.3 mg per 100 IU heparin) was administered following CPB discontinuation.

Data collection and sampling protocol

Arterial and central venous blood samples were obtained at four predefined time points:

1. 10 minutes after administration of cardioplegia
2. During rewarming at core temperature 32°C
3. 30 minutes after protamine administration
4. 24 hours postoperatively

Arterial blood samples were obtained from the indwelling arterial catheter, and venous samples were obtained from the central venous line. Blood gas analysis was performed using a calibrated blood gas analyzer.

Calculation of metabolic variables

Arterial and venous oxygen contents were calculated using standard equations:

$CaO_2 = (Hb \times 1.36 \times SaO_2) + (0.0031 \times PaO_2)$
$CvO_2 = (Hb \times 1.36 \times SvO_2) + (0.0031 \times PvO_2)$

DO_2 = Pump flow × CaO_2 × 10 / Body surface area

VO_2 = Pump flow × (CaO_2 − CvO_2) × 10 / Body surface area

Carbon dioxide production (VCO_2) was estimated using the veno-arterial pCO_2 gradient:

VCO_2 = Pump flow × ($PvCO_2$ − $PaCO_2$)

Given the near-linear relationship between CO_2 partial pressure and content within physiological ranges, this method was used as a surrogate for CO_2 production during CPB. VCO_2 was estimated indirectly and does not represent direct volumetric carbon dioxide elimination.

Respiratory Quotient (RQ):

RQ was calculated as VCO_2/VO_2. The DO_2/VCO_2 ratio was calculated as an index of oxygen supply relative to metabolic CO_2 generation.

Outcome measures:

Primary outcome:

Postoperative hyperlactatemia is defined as a blood lactate concentration > 2 mmol/L.

Secondary outcomes

- Intraoperative changes in DO_2, VO_2, VCO_2
- Respiratory quotient
- DO_2/VCO_2 ratio
- Serial lactate kinetics

Statistical analysis:

Statistical analysis was performed using SPSS software (27.0).

Continuous variables are presented as mean ± standard deviation. Normality was assessed using the Shapiro–Wilk test.

- Paired t-tests were used to compare intraoperative variables between cardioplegia and rewarming phases.
- Repeated-measures analysis of variance (ANOVA) assessed temporal trends in lactate and hemoglobin.
- Bonferroni correction was applied for post hoc comparisons when appropriate.

A p-value <0.05 was considered statistically significant.

Methodological rigor:

To minimize variability between patients and procedures, anesthesia, surgical procedures, and cardiopulmonary bypass (CPB) management were standardized based on institutional protocols. The anesthesia approach—covering induction agents, maintenance, and vasoactive drugs—temperature management, transfusion limits, pump flow rates, and mean arterial pressure targets were predetermined and uniformly implemented.

CPB employed a consistent circuit setup and oxygenator system, with pump flows adjusted for body surface area. Hematocrit levels were kept within a predefined range during bypass. Acid–base regulation followed a consistent method (α-stat/pH-stat, as outlined in the full methods).

Blood gas samples from arterial and venous blood were collected at specific CPB stages (initiation, cooling, stable hypothermia, rewarming, and pre-weaning) to ensure consistent timing. All samples were analyzed with the same calibrated blood gas analyzer. Key parameters, including oxygen delivery (DO_2), carbon dioxide production (VCO_2), respiratory quotient (RQ), and the DO_2/VCO_2 ratio, were calculated using standardized equations and adjusted for body surface area when needed. By combining directly measured gas exchange data with calculated metabolic indices, this study aimed to comprehensively evaluate oxygen transport and carbon dioxide removal during CPB.

RESULTS

Study Population

Twenty-six adult patients scheduled for elective cardiac surgery with cardiopulmonary bypass were analyzed. The average age was 52 ± 15 years, with 54% being male. The mean duration of CPB was 92 ± 24 minutes. Hematocrit levels during CPB were kept at 23.1 ± 2.4%. Baseline demographic and operative details are summarized in Table 1.

Table 1

Baseline Demographic and Operative Characteristics (n = 26)

Variable	Value
Age (years), mean ± SD	52 ± 15
Sex (Male/Female)	14 / 12
Body Mass Index (kg/m²)	25.8 ± 3.9
Diabetes mellitus, n (%)	7 (27%)
Hypertension, n (%)	11 (42%)
Preoperative hemoglobin (g/dL)	12.6 ± 1.4
Preoperative LVEF (%)	54 ± 8
CABG	10 (38%)
Valve surgery	9 (35%)
Combined procedures	7(27%)
Cardiopulmonary bypass time (min)	92 ± 24
Aortic cross-clamp time (min)	64 ± 18
Lowest temperature on CPB (°C)	30.8 ± 1.2
Hematocrit during CPB (%)	23.1 ± 2.4

Intraoperative Oxygen Transport and Metabolic Variables

Indexed oxygen delivery (DO_2) stayed above standard critical levels throughout CPB. No notable difference in DO_2 was observed between the cardioplegia and rewarming phases ($p = 0.48$).

During rewarming, oxygen consumption (VO_2) rose markedly ($p < 0.001$), indicating metabolic reactivation. In contrast, carbon dioxide production (VCO_2) remained largely unchanged ($p = 0.62$). The respiratory quotient (RQ) declined significantly ($p = 0.012$). The DO_2/VCO_2 ratio showed a downward trend but was not statistically significant ($p = 0.18$).

Notably, the rise in VO_2 during rewarming showed a substantial effect size (Cohen's $d = 0.83$), reflecting a significant metabolic shift. **(**Table 2**)** The elevated calculated RQ values likely reflect limitations of surrogate VCO_2 estimation during CPB rather than true physiological respiratory quotient values.

Table 2
Oxygen Transport and CO_2-Derived Variables During CPB (n = 26)

Variable	10 min after Cardioplegia	Rewarming (32°C)	p-value
DO_2 (mL/min/m²)	265 ± 60	272 ± 52	0.48
VO_2 (mL/min/m²)	36 ± 14	52 ± 16	< 0.001*
VCO_2 (mL/min/m²)	72 ± 25	75 ± 21	0.62
Respiratory Quotient (RQ)	2.00 ± 0.80	1.50 ± 0.50	0.012*
DO_2 / VCO_2 ratio	3.7 ± 1.5	3.3 ± 1.2	0.18
*Statistically significant ($p < 0.05$)			

Lactate Kinetics

Lactate levels increased progressively during CPB and peaked 30 minutes after protamine administration. Repeated-measures ANOVA showed a significant time effect ($F = 18.6$, $p < 0.001$). (Table 3)

Table 3
Serial lactate levels

Time Point	Lactate (mmol/L), Mean ± SD
10 min post-Cardioplegia	1.90 ± 0.88
Rewarming (32°C)	2.40 ± 0.93
30 min Post-Protamine (Peak)	3.78 ± 3.43
24 Hours Postoperative	2.08 ± 0.96
Lactate peaked after separation from CPB and partially normalized by 24 hours.	

Correlation Between Metabolic Reactivation and Hyperlactatemia

No significant correlation was found between the rise in oxygen consumption during rewarming (ΔVO_2) and peak postoperative lactate levels ($r = -0.002$, $p = 0.993$). Table 4.

Table 4
Correlation Analysis

Variable Pair	Pearson r	p-value
ΔVO_2 vs Peak Lactate	−0.002	0.993

This indicates that the level of metabolic reactivation was not a direct factor in the rise of postoperative lactate levels.

Predictive Performance of DO_2/VCO_2 Ratio

Receiver operating characteristic (ROC) analysis was conducted to evaluate the capacity of the DO_2/VCO_2 ratio at the time of rewarming in predicting clinically significant hyperlactatemia (lactate > 3 mmol/L). Table 5.

Table 5
ROC Analysis for Lactate > 3 mmol/L

Predictor	AUC	Interpretation
DO_2/VCO_2 (Rewarming)	0.568	Poor discrimination

The AUC was 0.568, which suggests limited ability to distinguish between groups performance.

Hemoglobin Trends

Hemoglobin levels showed significant variation over time ($F = 4.92$, $p = 0.006$), indicating effects of hemodilution and postoperative recovery. (Table 6)

Table 6
Hemoglobin Levels at Different Study Intervals

Time Point	Hemoglobin (g/dL), Mean ± SD
10 min Post-Cardioplegia	8.0 ± 1.8
Rewarming	8.3 ± 1.6
30 min Post-Protamine	10.2 ± 2.0
24 Hours Postoperative	10.1 ± 1.4

DISCUSSION

This prospective physiological study of adult patients undergoing cardiopulmonary bypass (CPB) found that indexed oxygen delivery (DO_2) consistently remained above critical thresholds throughout the procedure. Rewarming was associated with a notable, clinically meaningful increase in oxygen consumption (VO_2), yet postoperative lactate levels also increased. Importantly, neither VO_2 nor the DO_2/VCO_2 ratio predicted significant hyperlactatemia. These results suggest that the rise in lactate after modern CPB is more likely due to complex metabolic and inflammatory responses rather than an inadequate global oxygen supply.

Inadequate oxygen delivery during CPB has been associated with organ dysfunction and hyperlactatemia. Ranucci et al. showed that lower DO_2 levels during bypass are linked to acute renal failure and worse outcomes. More recent research indicates that goal-directed perfusion strategies

aiming for higher DO_2 levels can help reduce postoperative kidney injury.[7,8] A recent systematic review by Dias et al. also emphasizes the importance of ensuring sufficient oxygen delivery during CPB.[9]

In our cohort, DO_2 remained within or above levels generally considered safe. Despite this, lactate levels rose after surgery. This disconnect suggests that mechanisms beyond traditional oxygen debt may be at work.

CPB induces a systemic inflammatory response marked by cytokine activation, endothelial dysfunction, and metabolic changes.[2,3] These responses might lead to altered substrate use and heightened glycolytic activity, occurring independently of overall oxygen delivery.

Hyperlactatemia after cardiac surgery is common and associated with adverse outcomes.[4–6] However, elevated lactate levels do not necessarily imply tissue hypoxia. Garcia-Alvarez et al. demonstrated that hyperlactatemia may occur despite adequate oxygen delivery due to metabolic and mitochondrial factors.[10]

Our findings are consistent with this paradigm. The absence of correlation between the increases in VO_2 associated with rewarming and peak lactate suggests that lactate elevation may reflect metabolic activation rather than oxygen supply failure.

Hypothermia during CPB reduces metabolic rate and oxygen consumption. As temperature normalizes, VO_2 increases accordingly. Leone et al. demonstrated that temperature modulation influences oxygen delivery dynamics during CPB.

In the present study, VO_2 increased significantly during rewarming, with a large effect size (Cohen's d = 0.83), confirming substantial metabolic reactivation. However, this physiologic transition did not translate into proportional changes in lactate kinetics, suggesting that lactate elevation is not simply a consequence of oxygen demand during rewarming.

CO_2-derived parameters have been proposed as surrogates of microcirculatory dysfunction and anaerobic metabolism.[12,13] Recent cardiac surgery studies have explored their predictive utility during CPB.[14,15]

In our cohort, ROC analysis demonstrated limited discriminatory performance of DO_2/VCO_2 for predicting lactate > 3 mmol/L (AUC 0.568). This finding aligns with recent data suggesting that CO_2-derived indices may not reliably predict major postoperative complications in cardiac surgery populations.[15]

Therefore, although physiologically appealing, CO_2-derived metrics may provide limited standalone utility during elective cardiopulmonary bypass procedures where overall perfusion is sustained.

Clinical Implications

Our data suggest a nuanced view of lactate during CPB. When DO_2 stays within accepted thresholds, moderate postoperative lactate rises may not always signal insufficient oxygen delivery. These results highlight the need for interpreting lactate levels in context, rather than automatically increasing pump flow or transfusing based solely on lactate values.

Limitations

This was a single-center observational study with a small sample size. Measurements of microcirculatory and mitochondrial functions were not conducted. Inflammatory mediators and catecholamine levels were also not quantified, so mechanistic explanations are only speculative.

CONCLUSIONS

During modern CPB with maintained oxygen delivery, rewarming triggers notable metabolic activation. Elevated lactate levels after surgery happen regardless of whether global oxygen delivery (DO_2) is adequate or if VO_2 increases during rewarming. CO_2-based indices are of limited use in predicting these changes. These results indicate that hyperlactatemia during CPB results from complex metabolic and inflammatory processes rather than just insufficient oxygen supply.

What This Study Adds

This study offers three key insights:

1. Keeping DO_2 above established thresholds does not prevent postoperative lactate levels from rising.

2. Rewarming is a metabolically active and potentially fragile phase of CPB.

3. CO_2-based indices capture physiological changes but may not solely predict lactate kinetics during controlled perfusion.

Focusing on intraoperative metabolic dynamics rather than just static thresholds emphasizes the need to consider phase-specific physiology in CPB management.

Declarations

Ethical approval and consent to participate:

The study was conducted in accordance with the Declaration of Helsinki and the ethical guidelines of the Indian Council of Medical Research (ICMR). Ethical approval was obtained from the Institutional Human Ethics Committee (Approval No.; CSP-□/24/APR/04/153). Written informed consent was obtained from all participants prior to enrolment.

Consent for publication:

Not applicable.

Availability of data and materials

The datasets collected during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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This study received no funding.

Authors' contributions

DJ, JAJ and SS conceived the study. ST & RE contributed to data acquisition. DJ supervised. AJ, JAJ & DJ performed statistical analysis. AJ, SS & RE drafted the manuscript. All authors reviewed and approved the final manuscript.

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