Article

Not peer-reviewed version

Postoperative Blood Pressure Does Not Affect Lactate Clearance in Cardiac Surgery: A Retrospective Observational Cohort Study

James Hall *, George Botros , Michael Khilkin

Posted Date: 15 September 2025

doi: 10.20944/preprints202509.1297.v1

Keywords: postoperative cardiac surgery; lactic acid; perfusion pressure

Article

Postoperative Blood Pressure Does Not Affect Lactate Clearance in Cardiac Surgery: A Retrospective Observational Cohort Study

James Hall *, George Botros and Michael Khilkin

NYU Langone Long Island Hospital, Dept. of Cardiac Surgery

* Correspondence: james.hall@nyulangone.org

Abstract

Background: Tight blood pressure control is a cornerstone of postoperative cardiac surgery patients. In addition, plasma lactate levels are frequently monitored in this setting as it is a marker for malperfusion, with early elevated levels being associated with increased morbidity and mortality. Elevations from malperfusion may be due to decreased cardiac output, hypovolemia, or persistent post bypass vasoplegic response. Here, we investigate whether lower blood pressures, significant changes from baseline, and cardiac perfusion pressures delay the clearance of lactate after cardiac surgery. **Methods:** This is a retrospective cohort observational study of patients who have undergone coronary artery bypass graft (CABG) and valve replacement or repair surgeries at NYU Langone Long Island Hospital over a 6 month period. Postoperative blood pressures and lactate levels were examined over the first 16 hours of care. The primary outcome was a statistical relationship between specific pressures and fraction of cleared lactate. Intensive Care Unit and hospital length of stay, and mortality were secondary outcomes. **Results:** A total of 81 patients met inclusion criteria. The average pre-operative mean arterial blood pressure (MAP) was 95.4 mmHg and the average MAP in the first 6 hours post-operatively was 78.4 mmHg. The average change in MAP from baseline was a decrease of 16.7%. The average cleared lactate fraction by 16 hours postoperatively was 85.9%. Lactate clearance was associated in a statistically significant way only with the need for inotropic support on postoperative day 1, p=0.03. There was a slight trend toward a delay in lactate clearance in those with lower early systolic blood pressures, p=0.14. **Conclusion:** Lactate clearance appears to occur largely independently of postoperative blood pressures in the first 16 hours after surgery but may be delayed in those requiring inotropic support through the morning or postoperative day one.

Keywords: postoperative cardiac surgery; lactic acid; perfusion pressure

1. Introduction

Optimal hemodynamic management in the immediate timeframe after cardiac surgery is key for limiting complications and ensuring rapid recovery. Often used as a marker for hemodynamic effectiveness, elevations in lactic acid are common after cardiac surgery. This retrospective analysis examines the question of whether lower blood pressures and cardiac perfusion pressures affect the clearance of lactic acid after cardiac surgery to better estimate optimal hemodynamic targets.

There are multiple causes for lactic acidosis after cardiac surgery. These include residual lactate from the globally hypoperfused and tissue hypoxic states that exist while on cardiopulmonary bypass (CPB), increased production triggered by excess catecholamines, cytokines, and cortisol, reperfusion injury after hypoperfusion and hypothermia, impaired hepatic clearance during CPB, and microvascular dysfunction related to a systemic inflammatory response [1–4]. Early postoperative elevated lactate levels are common after cardiac surgery with incidence of elevations greater than 3 mmol reported to occur in 10-20% of patients [5]. Early elevated levels, measured in the first hour postoperatively, have been shown to be associated with increased rates of complications and

 doi:10.20944/preprints202509.1297.v1

increased morbidity and mortality. By comparison, late lactate increases, occurring after the first hour, were shown to be likely benign and transient, without significant worsening of outcomes [6].

Clearance of postoperative lactate depends on adequate perfusion and intact liver function where circulating lactate is converted to pyruvate. Lactate clearance may be correlated to blood pressure targets postoperatively as pressures below the patient's baseline may result in persistent malperfusion in the setting of hypertensive vascular remodeling. Since hypertension remains the most common comorbidity for coronary artery disease, as much as 80%, the prevalence of hypertensive vascular remodeling in the cardiac surgery population is high [7]. It remains common practice in cardiac surgery, despite an absence of supporting evidence, to maintain strict postoperative blood pressure ceilings to avoid hemorrhage from anastomotic compromise or small vessel bleeding.

Beyond vascular remodeling, organ autoregulatory changes may contribute to a persistent lactate. These mechanisms that promote continuous blood flow for organ perfusion in the setting of non-homogenous vascular beds, are known to be impaired after CPB, potentially leading to local malperfusion or ischemia in susceptible tissues [8,9]. While this rationale is the source of the well-established practice of allowing and inducing higher blood pressures in the setting of acute neurologic injury, this has not become common practice in the cardiac surgery population [10]. In neurologic injury, this practice is designed to ensure adequate cerebral perfusion pressures, defined as mean arterial pressure minus intracranial pressure. Similarly, in cardiac surgery, coronary perfusion pressure (CPP) is defined as diastolic blood pressure minus left ventricular end-diastolic pressure, a pressure that can be estimated from the pulmonary diastolic pressure. The choice of relatively low blood pressure targets after cardiac surgery leads to lower diastolic pressures and consequently CPPs. This could, in theory, create additional myocardial oxygen demand and increased lactate production.

In addition, lower blood pressure targets could also perpetuate local or systemic hypoperfusion and could lead to impaired lactate clearance or persistent lactate production. To investigate this, we examined the relationship between both absolute and baseline-relative blood pressures and lactate clearance as measured by fraction of lactate cleared by the morning after surgery.

2. Materials and Methods

For this study, we performed a retrospective observational chart review study of patients undergoing CABG and valve replacement or repair surgeries at NYU Langone Long Island between January 1 and July 1, 2022 as a convenience sample. We recorded hemodynamic parameters including systemic and pulmonary blood pressures, lactate levels immediately postoperatively and on the morning of postoperative day (POD) 1, as well as ICU length of stay, hospital length of stay, and mortality. Patients with baseline pulmonary hypertension or mitral stenosis were excluded, as were patients who did not have pulmonary artery catheters in place or who were missing lactate values, see **Table 1**. Pulmonary diastolic pressures were used as a proxy from LVEDP for purposes of CPP calculation. This proxy has been previously validated in patients without pre-existing pulmonary hypertension. [11]

The primary outcome was lactate clearance as determined by the fraction above 1 mmol/L that cleared from the initial lactate to the measurement on the morning of POD1. One mmol/L was used as a cutoff for a normal value that excluded mild elevations. The calculation used to determine lactate clearance (LC) utilized initial lactate value (ILV) and first postoperative morning lactate value (PMLV).

$$\mathbf{LC = 1 - [\,(1 - PMLV)\,/\,(1 - ILV)\,]}$$

Secondary outcomes included length of stay in the ICU and hospital and in-hospital mortality. The data were analyzed using linear regression with a significance level of p=0.05. All analyses were performed using SPSS Statistics version 28 software (IBM, Armonk, NY). This study was approved by the institutional review board of NYU Langone (s22-00962).

 doi:10.20944/preprints202509.1297.v1

Table 1. Baseline Demographics.

Age	**Mean** 68.3	**Range (SD)** 44-89 (9.46)
Sex	M 61 (75.3%)	F 20 (24.7%)
Procedure:	CABG	49 (60.5%)
	Isolated Valve	14 (17.3%)
	Combined	14 (17.3%)
Pressors am POD1	22	27.20%
Multiple Pressors am POD1	1	1.20%
Inotropes am POD1	29	35.80%

3. Results

Eighty-one patients met inclusion criteria, 75.3% male and 24.7 female. 49 (60.5%) underwent isolated CABG, the remainder were divided among isolated valve replacement, combined bypass and valve and ascending aortic surgery. Twenty-three (28.4%) required pressors through the morning of post-operative day 1, and 29 (35.8%) required inotropic support. See **Table 1.**

The average pre-operative mean blood pressure was 95.4 mmHg (95% CI 72-119) The average mean arterial pressure in the first 6 hours post-operatively was 78.4 mmHg (95% CI 63-94). The mean calculated coronary perfusion pressure in the first 6 hours was 43.2 (95% CI 25-62. The average change in mean blood pressure from baseline was a decrease of 16.7% (95% CI -40.3 to 6.9). Twenty-three patients (28.4%) required pressors the morning of POD1. Twenty-nine patients (35.8%) required inotropic support the morning of POD1. Please see **Table 2** for additional detailed blood pressures.

The average ICU and hospital lengths of stay for the group were 3.6 days and 6.5 days respectively. One patient died within 30 days. 38.3% experienced an increase in lactic acid post-operatively. The average fraction of lactate cleared by 16 hours post-op was 85.9% (95% CI 37.7% - 134.1%, greater than 100% represents a morning lactate of less than 1 mmol/L).

Table 2. Blood Pressure Variables.

	Mean	Range	95% Confidence Interval
Preoperative mean arterial pressure	95.4	72-142	72-119
Average systolic pressure first four hours	119.3	91-148	95-144
Average diastolic pressure first four hours	58.0	44-80	41-75
Average mean arterial pressure first four hours	78.4	64-96	63-94
Mean arterial pressure change from baseline, first four hours	-16.7	-41.2 - +17.6	-40.3 - +6.9
Average coronary perfusion pressure first four hours	43.2	23-65	25-62
Peak systolic blood pressure, first 16 hours	138.9	108-177	107-171
Peak diastolic blood pressure, first 16 hours	63.5	39-107	40-87
Peak mean arterial pressure, first 16 hours	88.7	65-123	68-109
Mean arterial pressure, greatest difference from baseline	-5.90%	-37.9 - +37.1	-34 - +22
Peak coronary perfusion pressure, first 16 hours	43.4	12-79	19-68
Lowest systolic blood pressure, first 16 hours	104.9	81-143	79-131
Lowest diastolic blood pressure, first 16 hours	46.9	31-67	33-61
Lowest mean arterial pressure, first 16 hours	66.2	52-80	53-79
Lower coronary perfusion pressure, first 16 hours	37.3	21-55	22-53
ICU length of stay	3.6	1-35	-5 – 12
Hospital length of stay	6.5	3-35	-1.7 – 14.7
In hospital mortality	1 (1.2%)	0-1	
Fraction of lactate cleared	85.9%	0-110%	37.7-134.1

 doi:10.20944/preprints202509.1297.v1

On regression analysis, the percent of cleared lactate was negatively associated with the need for inotropic support at 16 hours post-op, p=0.03, but did not reach statistical significance with any of the blood pressure combinations or need for pressors. There was a slight trend toward delayed clearance with lower initial systolic pressures, p=0.14, **See Table 3.**

Table 3. Correlations after Regression.

Correlation to Lactate Clearance with Regression	P=value	Pearson's R
Age	**0.38**	**0.098**
Peak coronary perfusion pressure	**0.76**	**0.035**
Preoperative mean arterial pressure	**0.34**	**0.108**
Average systolic pressure first four hours	**0.14**	**0.168**
Average diastolic pressure first four hours	**0.92**	**0.012**
Average mean arterial pressure first four hours	**0.39**	**0.097**
Mean arterial pressure change from baseline, first four hours	**0.62**	**0.055**
Average coronary perfusion pressure first four hours	**0.46**	**0.083**
Peak systolic blood pressure, first 16 hours	**0.73**	**0.039**
Peak diastolic blood pressure, first 16 hours	**0.37**	**0.100**
Peak mean arterial pressure, first 16 hours	**0.39**	**0.098**
Mean arterial pressure, greatest difference from baseline	**0.75**	**0.036**
Peak coronary perfusion pressure, first 16 hours	**0.76**	**0.035**
Mean arterial pressure, least difference from baseline	**0.32**	**0.113**
Lowest coronary perfusion pressure	**0.51**	**0.075**
Pressors in the morning, postoperative day one	**0.83**	**0.024**
Inotrope in the morning, postoperative day one	**0.03**	**0.207**

Eighty-one patients met inclusion criteria, 75.3% male and 24.7 female. Forty-nine (60.5%) underwent isolated CABG, the remainder were divided among isolated valve replacement, combined bypass and valve and ascending aortic surgery. Twenty-three (28.4%) required pressors through the morning of POD 1, and 29 (35.8%) required inotropic support, see **Table 1.**

The average pre-operative mean blood pressure was 95.4 mmHg (range 72-142, SD 11.9). The average mean arterial pressure in the first 6 hours post-operatively was 78.4 mm Hg (range 64-96, SD 7.7). The mean calculated coronary perfusion pressure in the first 6 hours was 43.2 (range 23-65, SD 9.3). The average change in mean blood pressure from baseline was a decrease of 16.7% (range decrease of 41.2% to increase of 17.6%, SD 11.8%). Please see **Table 2** for additional detailed blood pressures.

A minority (38.3%) experienced an increase in lactic acid post-operatively. The average fraction of lactate cleared by 18 hours post-op was 85.9%, SD 24.1%. On regression analysis, the percent of cleared lactate was negatively associated with the need for inotropic support at 18 hours post-operatively but did not reach statistical significance with any of the blood pressure combinations including change from pre-operative baseline, or the need for pressors. A minority (38.3%) experienced an increase in lactic acid post-operatively, s**ee Table 3.** The average ICU and hospital lengths of stay for the group were 3.6 days and 6.5 days respectively. One patient died within 30 days.

4. Discussion

The data presented in this limited study did not support the experimental hypothesis that lower blood pressures or significant decreases in blood pressures would result in a statistically significant decreased fraction of lactate clearance. These data ought to be interpreted with the understanding that no value measured was a significant deviation from the normal range, and that blood pressures outside of established normal values might result in different findings. Given the variety of etiologies of increased lactate in the post-operative period and the difficulty of isolating each cause for specific testing, these data must only be considered in the aggregate, and that specific models would need to be made to isolate each component for accurate associations.

 doi:10.20944/preprints202509.1297.v1

To date, no previous studies were identified that have examined this specific topic. Multiple studies exist that show delay or failure of lactate clearance has a strong association with morbidity and mortality after cardiac surgery, but they do not include associative data on blood pressures [12,13]. There are a number of studies, however, that address the formation of lactate based on targeted MAPs while on CBP and include associated outcomes. Targeting higher MAP's while on CPB is associated with less lactate formation which in turn is associated with improved outcomes overall [14–16].

A 2024 observational study showed that as little as one minute of MAP <65 mmHg was statistically significantly associated with acute renal injury[17]. Based on the established studies of autoregulation and organ compromise in hypotension, the null result in this study suggests that lactate clearance depends much more on other factors such as liver washout or intra-operative production, than on persistent production from hypoperfusion in the post-operative period.

This study has a number of limitations. The number of patients powered this study to only appreciate a large effect size. The multiple sources of lactate render it a non-specific marker and may not help to establish blood pressure targets. The recorded point values used to capture blood pressure from chart records may not appreciate temporary or limited changes which may have a large effect. Additionally, improved perfusion from new cardiac grafting likely has a significant effect on coronary autoregulation.

Given these data, we can make no specific clinical recommendation based on this negative study. Future studies would be required to elucidate lactate production and clearance based on post-operative perfusion. Additionally, the important clinical endpoint of a relevant target post operative blood pressure, either absolute or relative to the patient's known baseline, requires further retrospective and prospective investigations.

5. Conclusions

Within the normal range, there is no significant difference in lactate clearance based on specific blood pressure values or changes from baseline after cardiac surgery.

Author Contributions: Conceptualization, JH and MK.; methodology, JH,MK, and GB.; software, JH.; validation, JH, MK, GB.; formal analysis, JH.; investigation, JH, MK, GB.; data curation, JH.; writing—original draft preparation, MK, GB.; writing—review and editing, JH.; supervision, JH.; project administration, JH.; All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board of NEW YORK UNIVERSITY (protocol s22-00962, 11 February, 2024).

Informed Consent Statement: Patient consent was waived due to retrospective data review only.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors on request.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Teixeira FC, Fernandes TEL, Leal KCDS, Ribeiro KRB, Dantas DV, Dantas RAN. Factors associated with increased lactate levels in cardiac surgeries with cardiopulmonary bypass: a scoping review. *Rev Bras Enferm*. 2024;77(1):e20230117. Author 1, A.; Author 2, B. Title of the chapter. In *Book Title*, 2nd ed.; Editor 1, A., Editor 2, B., Eds.; Publisher: Publisher Location, Country, 2007; Volume 3, pp. 154–196.

 doi:10.20944/preprints202509.1297.v1

2. Fath Al-Bab Ibrahim GSB, et al. *Hyperlactatemia and Coronary Artery Bypass Grafting*. ZUMJ. 2024;30(4):1338-1345.Author 1, A.B.; Author 2, C. Title of Unpublished Work. *Abbreviated Journal Name* year, *phrase indicating stage of publication (submitted; accepted; in press)*.
3. O'Connor E, Flack T, McDermott S, et al. *Interpretation of perioperative lactate abnormalities in adult cardiac surgery*: effects of ischemia-reperfusion during cardiopulmonary bypass and hypothermia on lactate metabolism. *Eur J Cardiothorac Surg*. 2012;42(4):607–613.Author 1, A.B.; Author 2, C.D.; Author 3, E.F. Title of Presentation. In Proceedings of the Name of the Conference, Location of Conference, Country, Date of Conference (Day Month Year).
4. Feng J, Kant S, Sellke FW. *Microvascular dysfunction following cardioplegic arrest and cardiopulmonary bypass*. *Vessel Plus*. 2021;5:30.
5. Minton J, Sidebotham DA. Hyperlactatemia and cardiac surgery. *J Extra Corpor Technol*. 2017;49(1):7-15.
6. Algarni KD, Almuzaini R, Rajab A, et al. Early hyperlactatemia after cardiac surgery: incidence, predictors, and clinical outcomes. *J Cardiothorac Surg*. 2020;15:152.
7. Moroz GZ, et al. Comorbidity in patients with chronic coronary syndromes undergoing PCI. *Wiad Lek*. 2020;73(3):215-220.
8. Kant S, Feng J, Sellke FW. Microvascular dysfunction following cardioplegic arrest and cardiopulmonary bypass. *Front Med (Lausanne)*. 2023;10:1110532.
9. Hori D, Ono M, Brady K. Optimal blood pressure during cardiopulmonary bypass: defining the limits of cerebral autoregulation. *J Thorac Cardiovasc Surg*. 2017;153(2):287-295.
10. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: blood pressure management recommendations. *Stroke*. 2019;50(1):e12-e23.
11. Lang, IM. Diastolic pulmonary artery to pulmonary capillary wedge pressure difference: a predictor of outcome after left ventricular assist implantation. *J. Am Heart Assoc.* 2020; 9(7). https://doi.org/10.1161/JAHA.120.016110
12. Patmano G, et al. Lactate clearance predicts morbidity/mortality after cardiac surgery. *Turkiye Klinikleri J Cardiovasc Sci*. 2019.
13. Marbach JA, et al. Lactate clearance and mortality in cardiogenic shock. *J Am Heart Assoc*. 2022.
14. Miao Q, et al. Target blood pressure management during cardiopulmonary bypass and postoperative lactate levels. *Crit Care*. 2021;25(1):243.
15. Inoue S, et al. Low mean arterial pressure in early cardiopulmonary bypass associated with higher postoperative lactate. *Anesth Analg*. 2017;125(2):543-549.
16. Toraman F, et al. Lactate levels after cardiac surgery and relationship with systemic hypoperfusion. *Heart Surg Forum*. 2004;7(2):E155-E159.
17. Smith A, Turoczi Z, Al-Subaie N, Zilahi G. Postoperative hypotension after cardiac surgery is associated with acute kidney injury. *J Cardiothorac Vasc Anesth*. 2024;38(8):1683-1688. doi:10.1053/j.jvca.2024.04.024.