

Hyperlactatemia and Patient Outcomes After Pediatric Cardiac Surgery

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Elevated serum lactate level, or hyperlactatemia, is often associated with alterations in tissue perfusion, increased risk for complications in the postoperative period, and patient mortality. Measuring lactate levels is a relatively simple and noninvasive method of obtaining useful data about an impending clinical deterioration in a seemingly hemodynamically stable patient. This article evaluates the current practice of measuring lactate levels in pediatric patients after cardiac surgery and the association between these levels and patient outcomes. The article addresses periods of increased risk for decreased perfusion, the critical postoperative period, use of lactate measurements in conjunction with a risk scoring system for pre- and postoperative congenital heart disease patients, and the implications of elevated lactate levels in nursing practice. (*Critical Care Nurse*. 2018;38[5]:e1-e6)

During cardiopulmonary bypass surgery and the critical postoperative period, patients are at risk for decreased perfusion. The resulting elevated serum lactate levels, or hyperlactatemia, may be associated with an increased risk for complications in the postoperative period. Therefore, clinicians may use serum lactate levels to predict patient morbidity and mortality. This article examines the practice of measuring lactate levels in pediatric patients after cardiac surgery. A literature review was conducted to determine whether a single value or a trend in lactate values is a useful predictor of postoperative outcomes, with a focus on implications for the critical care nurse caring for the pediatric patient in the cardiac intensive care unit (CICU).

A 4-day-old female infant with hypoplastic left heart syndrome was moved to the CICU after a stage 1 palliative procedure in the operating room. The patient had been intubated and sedated, with an arterial catheter, a central venous catheter, a peripheral intravenous catheter, a mediastinal chest tube, and atrial and ventricular pacer wires (which had subsequently been removed). Epinephrine at 0.02 µg/kg per minute and milrinone at 0.5 µg/kg per minute had been initiated in the operating room. The anesthesia report indicated that

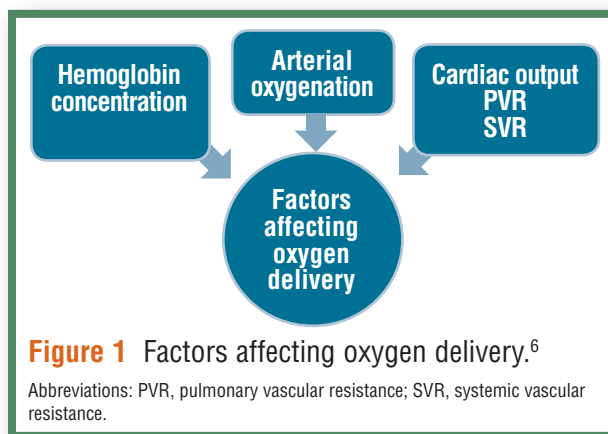
the patient had been easily intubated with a 3.5 endotracheal tube on the first attempt, secured at 10 cm at the lip, and there were no problems with ventilating the patient. The lactate level was 6.0 mmol/L after the bypass operation. Normal lactate levels are approximately 1.5 mmol/L in healthy individuals and less than 2.0 mmol/L in critically ill patients.¹ On arrival at the CICU, the patient's lactate level was 7.1 mmol/L. Is this hyperlactatemia related to tissue hypoxia, or is it an expected finding after cardiac bypass in the operating room?

Pathophysiology

Lactate is produced in all tissues of the human body, with the highest production occurring in the muscles. During carbohydrate metabolism, both aerobic and anaerobic processes occur, which differ in the amount of lactate produced. In aerobic metabolism, pyruvate enters the Krebs cycle, resulting in a small amount of lactate production. In anaerobic metabolism, pyruvate is converted into lactic acid by lactate dehydrogenase; this lactic acid is considered a by-product of glycolysis and is used during the Cori cycle in the liver.² Although the primary organ responsible for lactate clearance is the liver, the kidneys also provide clearance when lactate levels are increased in the body. Two forms of lactic acid exist: D-lactate and L-lactate. The focus of this discussion is L-lactate, which is the product of glycolysis and is a marker used to evaluate hypoperfusion and tissue hypoxia.

Definitions

What does a lactate level of 7.1 mmol/L mean in terms of the patient in the case study? When an arterial blood sample is sent to the laboratory, the lactate level measured is the difference between the amount of lactate produced in the body and the body's ability to consume or clear the lactate. In healthy individuals, lactate production can occur in any tissues, but those with the



most production are the skin, brain, intestines, skeletal muscles, and red blood cells.³ These tissues produce an average daily lactate volume of 1400 mmol.^{3,4}

Postoperative lactate values greater than 4.4 mmol/L have been directly correlated to worse postoperative outcomes in surgical patients.⁴ Type A, or secondary, hyperlactatemia is directly related to poor tissue perfusion and is the most common type in patients after cardiac surgery. Other major causes of type A hyperlactatemia are sepsis, anemia, and hypoperfusion.⁵ Type B, or primary, hyperlactatemia is often associated with a disease process such as liver disease, thiamine deficiency, drugs or toxins, or inborn errors of metabolism. Type B hyperlactatemia is not associated with hypoperfusion.

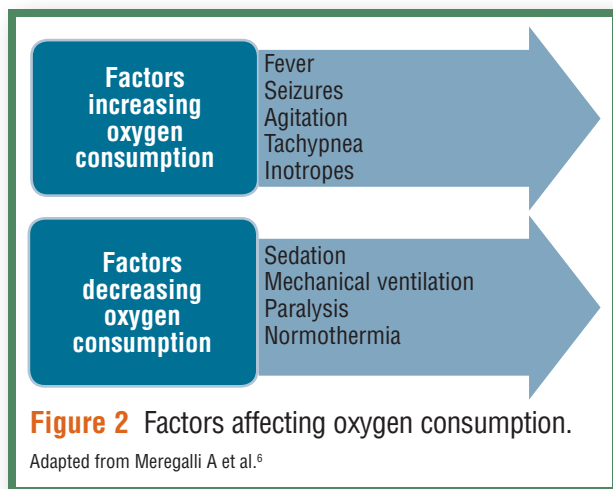
Increased lactate production may be a result of impaired or altered oxygen delivery or an overall imbalance between oxygen delivery and tissue needs (Figure 1).⁶ Therefore, prompt identification and correction of impaired tissue perfusion can help decrease morbidity and mortality in postoperative cardiac patients. The goals of treatment would include a decrease in the imbalance between oxygen delivery and tissue needs (Figure 2).⁶

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Lactate clearance provides valuable information about the body's ability to remove the lactate and the effectiveness of the treatment modalities. Lactate clearance occurs largely during the first 2 hours after insult, and levels begin to normalize between 2 and 4 hours.⁷

Intraoperative Lactate Levels and Outcomes

The Norwood procedure is associated with the highest mortality risk in the treatment of congenital heart disease, with death rates ranging from 6% to 25%.⁸ Risk factors include aortic atresia, small ascending aorta, presence of cardiac and/or noncardiac anomalies, chromosomal abnormalities, lower preoperative pH, palliation after 14 or 30 days, longer circulatory arrest or cardiopulmonary bypass times, and the presence of tricuspid regurgitation.⁸

Patient management after a Norwood procedure focuses on balancing the systemic and pulmonary circulations. The identification of the patient in the case study as high risk preoperatively, as evidenced by the small ascending aorta and longer bypass times, could allow for a proactive approach during the postoperative period. Evidence suggests that lactate level can be used as an accurate predictor of mortality following pediatric cardiac surgery.⁹ Mortality modeling demonstrated that an inability to clear blood lactate levels to less than 6.76 mmol/L within the first 24 hours after surgery was highly predictive of death within the first 30 days.⁹ Abnormal lactate levels are considered an early predictor of low cardiac output or an impending clinical change. Prompt identification of patients with an elevated lactate level can contribute to the decision to

perform elective extracorporeal membrane oxygenation cannulation.

A study by Alves et al⁴ involving low- and moderate-risk patients found a similar association between elevated lactate levels intraoperatively and an increased risk for complications in the postoperative period. Prolonged use of vasopressors was required by patients because of decreased cardiac output, which also resulted in alterations in kidney function. In addition, lactate levels greater than 4.2 mmol/L on admission to the ICU were associated with lower survival rates in the postoperative period.¹⁰ Higher lactate levels on admission also correlated with increased number of days receiving ventilation and prolonged use of vasopressors.

The Risk Adjustment for Congenital Heart Surgery (RACHS-1) tool rates the risk associated with surgical procedures using 6 predetermined categories based on the congenital heart defect. Risk category 1 has the lowest mortality risk, and risk category 6 has the highest mortality risk. If the patient has more than 1 defect, such as a ventricular septal defect and transposition of the great arteries, he or she should undergo the highest-risk procedure.¹¹ The postoperative Norwood procedure used in the case study was classified as risk category 6.

Patients with lower RACHS-1 scores preoperatively have been found to have lower lactate levels in the postoperative period. Hazan et al⁵ demonstrated a correlation between an increased preoperative RACHS-1 score and decreased survival of those patients postoperatively. Patients with RACHS-1 scores greater than 4 are expected to have compromised cardiac output in the postoperative period.¹² The study also found

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a positive correlation between lower lactate levels and survival in the postoperative period. By using a scoring system preoperatively and detecting trends in lactate levels postoperatively, providers helped identify patients at risk for complications in the postoperative period.

Lactate Trends

“Lactime,” or the time it takes for the lactate level to normalize, is supported by several studies as a more effective indicator of mortality or complications than the actual lactate level.¹³ Prolonged lactime has been correlated

with increased postoperative complications and poor outcomes. A study by Mak et al¹⁴ indicated that an elevated lactate level with a peak after postoperative hour 30 was linked to increased mortality.

Postoperative Lactate Levels

Cardiopulmonary bypass affects major organ systems through a decrease in perfusion to vital organs, which results in activation of anaerobic metabolism and subsequent elevation in lactate levels. Additionally, multiple blood transfusions and the contact between the

Goal-directed therapy includes intensive monitoring and aggressive management of hemodynamics in patients with a high risk of morbidity and mortality.

synthetic circuit and the patient's blood result in the activation of

inflammatory mediators. Hyperlactatemia in the immediate postoperative period is directly affected by intraoperative events such as cardiopulmonary bypass flow, oxygen delivery, circulatory arrest times, temperature, hematocrit levels, and inflammation. Alteration in the delivery of oxygen to the tissues or increased consumption of oxygen, even when oxygen delivery to the tissues is normal, may occur. Hyperlactatemia after cardiopulmonary bypass is caused by excessive lactate production intraoperatively or decreased liver clearance due to hypoperfusion.

A lactate level greater than 2 mmol/L at 48 hours after a surgical procedure has an 86% sensitivity in predicting mortality.¹⁵ Clinicians found that elevated lactate levels were more sensitive indicators for compromised circulation than were clinical measures, such as urine output, capillary refill time, and core versus peripheral temperature.¹⁶ Clinical measures are late signs of an alteration in perfusion, and decisions about patient care based on these signs do not represent timely intervention. In complex congenital heart disease of the neonate, serial lactate measurements obtained during the first 3 days after surgery directly reflected patient outcomes.¹⁷

Occult Hypoperfusion

Clinical indicators such as blood pressure, heart rate, and urine output are often normal while the lactate level remains elevated in critically ill patients. A study by Meregalli et al⁶ measured blood lactate levels in 44 high-risk, hemodynamically stable surgical patients and

noted that at 12, 24, and 48 hours postoperatively, lactate levels were higher in nonsurvivors than in survivors. Heart rate, mean arterial blood pressure, and oxygen saturation levels were noted to be similar in the nonsurvivor and survivor groups.

In critically ill patients, a thorough patient assessment as well as laboratory data, such as lactate levels, should be used in conjunction to determine end-organ perfusion. The inability to clear lactate during resuscitation can be directly correlated with patient outcomes. For adults, goal-directed protocols to manage hyperlactatemia have been established and published. Goal-directed protocols for elevated lactate levels also are included in sepsis guidelines. However, no specific guidelines exist for management of elevated lactate level in pediatric cardiac surgical patients. The pediatric data support use of lactate levels as an endpoint for therapies and as a guideline for management.

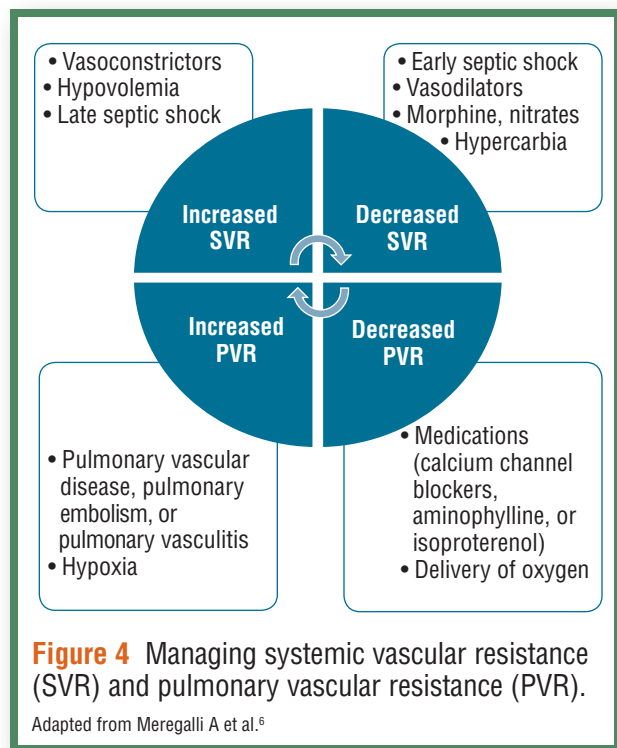
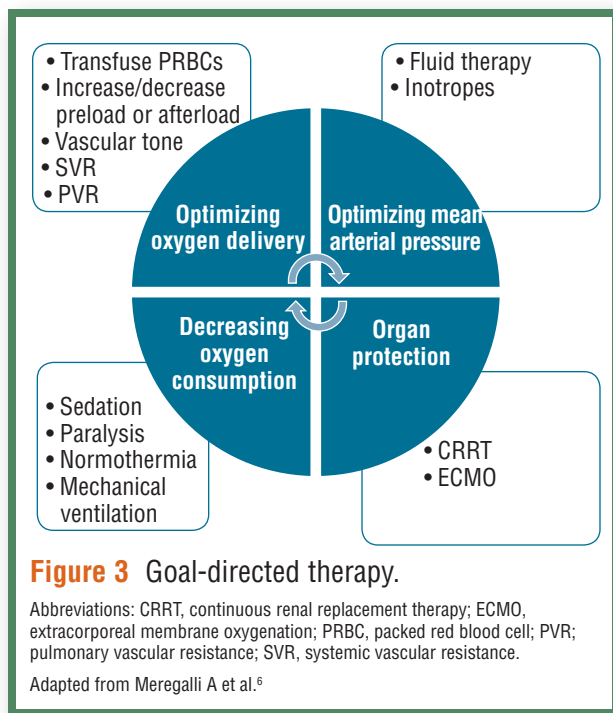
Treatment

Treatment methods include early monitoring of lactate levels, but this alone is not enough. Goal-directed therapy, a technique used in critical care medicine, includes intensive monitoring and aggressive management of hemodynamics in patients with a high risk of morbidity and mortality.¹⁸ An example of a successful goal-directed therapy is the Surviving Sepsis Campaign, which aims at reducing lactate levels using fluid therapy and antibiotic therapy within 3 hours. A goal of decreasing lactate levels by 20% within 2 hours using aggressive fluid and inotropic management led to shortened ICU stay of patients with a lactate level at or above 3 mmol/L on admission.¹⁹

In addition, vasopressor therapy and reassessment should occur within 6 hours of initial presentation. Hyperlactatemia after cardiac surgery is universally related to unmatched oxygen delivery and tissue needs, as evidenced by decreased perfusion, oxygen debt, or altered end-organ perfusion. Critical goals of therapy in postoperative cardiac patients include reducing oxygen consumption and/or increasing oxygen delivery, fluid resuscitation, vasopressor support, and preserved end-organ function (Figure 3).¹⁹

Nursing Implications for Practice

As stated previously, the Norwood procedure is classified as risk category 6, the highest risk level for postoperative mortality. RACHS-1 is used to identify high-risk patients in the preoperative period. The information



from the operating room team that is vital to postoperative management includes bypass times, arrhythmias, blood product administration, vasopressors, peak lactate level intraoperatively and before leaving the operating room, and complications. The case briefly presented here is an example of a high-risk patient based on structural heart disease, postoperative management, and the various risk factors discussed.

Medical management should focus on balancing pulmonary and systemic perfusion, while maintaining adequate cardiac output. Key nursing interventions include providing sedatives and/or analgesics and avoiding hyperventilation, which can increase pulmonary blood flow, which in turn reduces systemic output (Figure 4).⁶ Close monitoring of vital signs, hemodynamic waveforms, and presence of acidosis (either respiratory or metabolic) is crucial in postoperative management. Optimizing hematocrit levels to greater than 40% and blood product replacement to correct coagulopathy are also important in postoperative management. Temperature regulation aids in decreasing metabolic demands and risk for arrhythmias.

Elevated lactate levels can occur for many different reasons, and the significance of the elevation should be correlated with the clinical picture. Although lactate level is useful in conjunction with an overall assessment of the patient, a single value is not used in diagnosing or

predicting outcomes, as it usually does not explain the reason for the accumulation of lactate in the body. Elevated lactate level is related to either an increase in anaerobic metabolism or altered clearance related to end-organ dysfunction. Increased duration of hyperlactatemia is correlated to prolonged hypoperfusion, and an elevation in creatinine level is often an indication of kidney dysfunction. Serial lactate measurements may be used in determining risk for morbidity and mortality. Data from the literature on adults and sepsis guidelines have been introduced into the pediatric arena, but more research is needed in the congenital cardiac surgery population.

Conclusion

Lactate levels are valuable indicators in the period after pediatric cardiac surgery. The critical care nurse should be aware of the implications of rising lactate levels and alert to impending decompensation or end-organ injury in this vulnerable population. Available data suggest that serial lactate measurements can be a useful adjunct to clinical assessment and consideration of case-specific risk factors in guiding proactive management to prevent adverse events. **CCN**

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See also

To learn more about pediatric cardiac care, read "Maternal Stress and Anxiety in the Pediatric Cardiac Intensive Care Unit" by Lisanti et al in the *American Journal of Critical Care*, March 2017;26:118-125.

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