THE AMERICAN ACADEMY
OF
CARDIOVASCULAR PERFUSION
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The Academy Newsletter

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45th Annual Seminar of The American Academy of Cardiovascular Perfusion

AACP Annual Seminar Photos


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ABIOMED

DEVELOPING ACTIVE LEARNING ACTIVITIES FOR DIDACTIC PERFUSION COURSES

Catherine Kim

Milwaukee School of Engineering
Perfusion Program
Milwaukee, WI

In the world of academia, traditional lecturing has been the most common pedagogical approach for centuries. However, it creates an environment for students to be passive learners in the classroom. Alternatively, active learning is a pedagogical approach intended to encourage students to engage with content in manners which have been shown to improve exam performance, clinical reasoning skills, critical thinking skills, and the transfer of knowledge to new environments. Given that perfusion is inherently a profession where didactic information must be applied in practice, the implementation of active learning strategies is worth considering.

Five active learning activities have been specifically designed for didactic perfusion courses to assist perfusion students in understanding basic perfusion concepts such as coronary anatomy, pressure monitoring, and circuit parameters. These activities have been designed based on research conducted on the science of learning, active learning worksheets, the Learning Cycle, and process-orientated guided inquiry learning (POGIL). Key concepts were identified, learning objectives were specified, and models (figures) were created as targets of engagement and analysis. Following each model, questions and prompts are given to guide students through identifying, understanding, and exploring the concepts incorporated in the activity as well as opportunities to apply the newly gained knowledge and understanding.

The goal of these activities is to bridge the gap between the information in the classroom and the application in the operating room. With discretion from instructors, it is recommended that new perfusion students complete these activities to improve their understanding of basic perfusion concepts and their ability to understand these concepts in a clinically significant manner. Please reach out to Catherine Kim at catkatkim@gmail.com if you are interested in these activities.

The full manuscript of this article has been submitted to the journal Perfusion for possible publication.

Nicole Elvezio

Cardiovascular Perfusion Program

Quinnipiac University
Hamden, CT

The Effects of Cardiopulmonary Bypass on Lactic Acid

Lactate, the conjugate base of lactic acid, is the end-product of glycolysis, commonly recognized as a biomarker for inadequate tissue perfusion. An elevated blood lactate level is known as Hyperlactatemia (HL), which can be detrimental to patient outcomes after cardiac surgery. HL can be further classified into Type A and Type B lactic acidosis which may develop from both hypoxic and non-hypoxic mechanisms. During cardiopulmonary bypass (CPB), it has been hypothesized that microcirculatory dysfunction and impaired tissue oxygenation, secondary to the pro-inflammatory effects, contribute to the early onset of HL. HL during CPB has been associated with higher postoperative intra-aortic balloon pump (IABP) usage, longer duration of intensive care unit (ICU) stays, and prolonged mechanical ventilation.

Lactate is produced from the metabolization of one molecule of glucose and two molecules of pyruvate by means of glycolysis. The normal blood lactate level in the human body is 0-2 mmol/L; a value above 3-5 mmol/L is generally accepted to define HL parameters. The way lactate is used as a biomarker for tissue hypoperfusion, HL is essentially a biomarker of circulatory failure. Elevated levels of lactate indicate the anaerobic glycolysis present in circulation, which leads to inadequate tissue perfusion. Type A HL is attributed with tissue hypoxia, whereas Type B HL is associated with no tissue hypoxia. HL occurrence can be further classified into early and late onset, where early onset develops in the operating room (OR) or in the early arrival of the ICU associated with increased morbidity and mortality, and late onset develops within 6-12 hours of ICU arrival and is a benign, self-limiting condition that usually resolves within 24 hours. There is a 14.9% mortality in patients with a lactate > 3mmol/L compared to 1.5% mortality in patients with a lactate < 3 mmol/L (Minton & Sidebotham, 2017). Lactate levels provide a generally adequate idea of how well the patient is perfusing and whether the patient needs to be fluid resuscitated or inotropic therapy needs to be initiated and/or altered accordingly.

Through complement activation, the inflammatory proteins produced during CPB, may have harmful effects on microcirculation including vasoconstriction, altered red blood cell (RBC) deformity and activation, or destruction, of platelets with initiation of the coagulation cascade. The main factors that can potentially lead to organ dysoxia during CPB involve the degree of hemodilution and low peripheral oxygen delivery, known as DO2. Additional studies have shown that increased lactate production during CPB has been associated with DO2 < 280 mL/min/m^2 , the double-edged sword of decreased hematocrit (HCT) versus blood product administration, inadequate flow, utilization of hemofiltration to hemoconcentrate and Epinephrine induced through accelerated aerobic metabolism.

Utilization of hemofiltration on CPB is indicated for patients starting with a low hematocrit, hypervolemia or known chronic progressed kidney disease with anticipation of altered electrolytes and impending arrythmias developing. In a 2009-2023 study performed in the Prince Sultan Cardiac Center in Saudi Arabia, there were 637 enrolled adult patients with inclusion criteria being cardiac surgery on CPB, and an ejection fraction (EF) > 35%. In this study, results were compared between the hemofiltration utilization group and the control group. The group with hemofiltration lead to hemoconcentration, decreased hemodilution, increased HCT, and decreased blood transfusion however resulted in hypotension related to hypovolemia, therefore causing impaired tissue perfusion, decrease oxygen supply (DO2) and as a result increased the serum lactate level. The SVO2 and urine output decreased significantly in this group as well. The control group (Group C) without hemofiltration during CPB, maintained adequate hemodilution which improved tissue perfusion, oxygenation, and decreased levels of serum lactate. This study also showed that the use of hemofiltration cleared only 3% of lactate production, and therefore is more closely associated with lactic acidosis. In the hemofiltration group, Phenylephrine pushes on pump were required to maintain a mean arterial pressure (MAP) greater than 65.

Phenylephrine induces vasoconstriction of microcirculation, which then decreases tissue blood flow, decreases oxygen supply and therefore potentiating the anaerobic metabolism and increased production of lactic acid. Additionally, Phenylephrine has been studied to find that its utilization on CPB results in impairment of microcirculatory blood flow, peripheral arteriovenous shunting, and deterioration in the microvascular flow pattern of erythrocytes within the capillaries, leading to a rise in lactate levels, despite apparent adequate oxygen supply. Another study showed the use of catecholamines, such as Norepinephrine, which is another medication commonly used on pump, is responsible for splanchnic vasoconstriction which reduces perfusion to the gastrointestinal tract during and after CPB leading to elevated lactate levels. With this study, it has been recommended as more beneficial to add crystalloids to the circuit and increase pump flow to maintain adequate circulating volume and adequate oxygen supply/demand than to administer Phenylephrine; however, in perfusion, each case is unique and is one entire balancing act that must always be taken into consideration.

With all that has been studied and documented thus far in the limited research regarding the effects of CPB directly on elevated lactate levels, there are few special circumstances where elevated lactate levels are more commonly and consistently seen on pump. This includes longer pump runs, achieving hypothermia especially deep hypothermic circulatory arrest (DHCA) and hyperglycemia. Often, medical professionals in the OR, will look to Perfusion when the lactate levels are elevated during and after CPB, however, it is significant to remember when rewarming from DCHA, hyperglycemia is essentially guaranteed. The increase in glucose production, related to the release of stress hormones and cytokine release on pump leading to insulin resistance, doesn't allow for the abundance of glucose to enter the oxidative pathway and is therefore

Potential causes of hyperlactatemia in cardiac surgical patients.

Cause	Mechanism(s)	Onset
Inadequate oxygen delivery during CPB	Tissue hypoxia (Type-A)	Early
Low cardiac output	Tissue hypoxia (Type-A)	Early or late
Severe anemia/hemodilution	Tissue hypoxia (Type-A)	Early or late
	Accelerated glycolysis (Type-B)	Early
Systemic inflammatory response syndrome	Impaired tissue perfusion due to microcirculatory failure (Type-A) (e.g., due to prolonged CPB, massive transfusion)	Early
Exogenous catecholamines (epinephrine, salbutamol, isoproterenol)	Accelerated glycolysis (Type-B)	Early or late
Hepatic ischemia	Tissue hypoxia (Type-A)	Early or late
	Reduced lactate clearance (Type-B)	Early or late
Limb ischemia	Tissue hypoxia (Type-A) (e.g., IABP)	Early
Mesenteric ischemia	Tissue hypoxia (Type-A) (e.g., due to NOMI or arterial embolus)	Late
Septic shock	As per systemic inflammatory response syndrome	Late

Cause	Mechanism(s)	Onset
Administration of lactated Ringer's solution	Increased lactate load (sodium lactate) (Type-B)	Early or late
Lactate-buffered renal replacement fluid	Increased lactate load (sodium lactate) (Type-B)	Late
Grand mal seizure	Accelerated aerobic glycolysis (Type-B)	Late
Renal failure	Reduced lactate clearance (Type-B) Tissue hypoxia (Type-A)	Late
Pancreatitis	As per systemic inflammatory response syndrome	Late
Drug related	Propofol syndrome (Type-B) Sodium nitroprusside (Type-A)	Late

(Minton & Sidebotham, 2017)

degraded to lactate via the glycolytic pathway. Adequate communication and control of blood sugars with Anesthesia can prevent hyperglycemia during and after CPB, especially when anticipated under certain circumstances. While there are no bulletproof methods of preventing the buildup of lactic acid on CPB or methods to efficiently clear it prior to weaning, there are established potential causes that can be further examined in future studies in order to develop prevention methodology.

References

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Thank You

We would like to thank the Members of The Academy for the recognition received at the meeting in Nashville. We sincerely appreciate the plaque, the meaningful booklet containing Past President messages and the beautiful crystal heart vase.

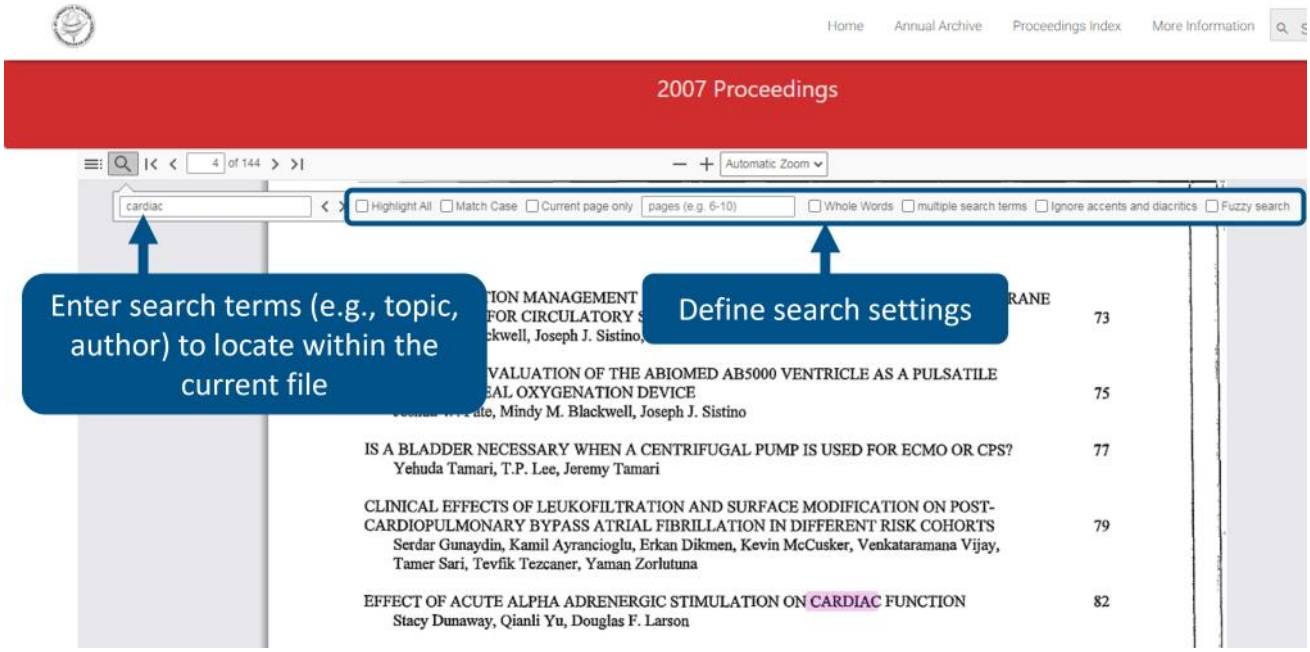
To all of our Members, we would like to extend our thanks for all of your encouragement, unwavering support and willingness to help over the past 20+ years. We have made so many memories and lifelong friends over the years.

Thanks again for everything!
Jill and David Palanzo

Proceedings Archival Site

All twenty-nine issues of the ***Proceedings of The American Academy of Cardiovascular Perfusion*** will be available in searchable PDF format through our new Proceedings Archival Site.

On the site's home page, type your search in the box in the upper right. That will locate the volumes containing the key word. Then click on the magnifying glass in the upper left and follow the instructions below.



Perfusion Journal to Become Totally Digital

Starting in January 2025, the *Perfusion Journal* will become totally digital. You will no longer receive printed issues. The journal will be accessed through the Academy website (Members' Only Page) and will allow you to access all issues of *Perfusion* not just from 2000 to the present.

You will still receive printed copies for this current year, but you can access the journal online through our website.

Hotel Lights

It was very nice for the Loews Vanderbilt Hotel in Nashville to install hall lights that resemble convoluted disks for our meeting.

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Important Academy Dates

The ACADEMY ANNUAL MEETING DEADLINES

ABSTRACT DEADLINE	**October 15, 2024**
MEMBERSHIP DEADLINE	**December 3, 2024**
PRE-REGISTRATION	**January 11, 2025**
HOTEL REGISTRATION	**January 11, 2025**
2024 ANNUAL MEETING	**February 5-8, 2025**

Welcome to New Members

The American Academy of Cardiovascular Perfusion would like to welcome the following individuals whom were voted into membership at the Closing Business Meeting of our annual meeting in Nashville, Tennessee.

Fellow Member
Laura Dell'Aiera

Members
Ryan Acker
Michael Audette
Carolyn Cavallo
Brian Cress
Michael Cronin
William Dauch
Joy Evangelin
Shelby Evans
Ashley Gartner
Erin Harris
Hannah Hedtke
Derek Howell
Anna Iulianelli
Sydney Jaramillo
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Saarah Hussain
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Emma Nahas
Alexander Nartey
Asia Nguyen
Anne Nguyen
Lacey Ordini
Olivia Osuch
Kylee Overton
Nicholas Ownbey
Elder Palacios
Patricia E. Palma
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Joseph M. Timpa
Erin Tobin
Michael Venegas-Anderson
Taylor Viana
Brittney Waters
Cara Weigel
Arissa Wickstrom
Cozette Wilkins
Lauren Yudin

Awards Committee Selects Winning Student Paper Presentations

Madison Lynch

Four students received awards for their paper presentations at the Annual Seminar in Nashville.

2024 – Jeffrey B. Riley Best Student Paper Presentation Award ($1000)
Madison Lynch - Putting The AI In Training: Dynamic Perfusion Scenario Tool

Ashley Mathews

2024 – Richard Adams Student Paper Presentation Award ($500)
Ashley Mathews - Alpha-Gal Syndrome: A Hidden Risk In Cardiac Surgery

Joseph M. Timpa

2024 – Aaron G. Hill Student Paper Presentation Award ($500)
Joseph M. Timpa - Bivalirudin: An Anticoagulation Alternative For Cardiopulmonary Bypass

Lauren Gawlinski

2024 - Lawrence Award ($500)
Lauren Gawlinski - Expansion Of The Heart Transplant Donor Pool With Donation After Circulatory Death Utilizing Normothermic Regional Perfusion

2025 Annual Meeting

Denver, Colorado
February 5-8, 2025

Our Host Hotel
Embassy Suites Denver Downtown Convention Center
1420 STOUT STREET, DENVER, CO 80202

Reservations: 1-800-HILTONS

Single/Double Occupancy: $219.00

Remember to mention that you will be attending the Annual Conference of The American Academy of Cardiovascular Perfusion (AACP).

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