

Lactic acidosis during Anesthesia

Essay

**Submitted for partial fulfillment of the Master degree in
Anesthesia**

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2015**

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

صدق الله العظيم
سورة البقرة آية (٣٢)



Acknowledgement

*First and foremost, thanks to **Allah** for giving me the will and the patience to finish this work,*

*In a few grateful words, I would like to express my deepest gratitude and appreciation to **Prof. Dr. Gamal Fouad Saleh Zaki**, Professor of Anesthesia, Intensive Care and Pain Management Faculty of Medicine Ain Shams University, for his great concern and generous help. Without his generous help, this work would not have been accomplished in its present picture.*

*I am sincerely grateful to, **Dr. Rami Mounir Wahba**, Lecturer of Anesthesia, Intensive Care and Pain Management Faculty of Medicine Ain Shams University for his kind help and constructive suggestions to achieve this work,*

*I would also like to express my deep appreciation to **Dr. Ghada Mohamed Samir** Lecturer of Anesthesia, Intensive Care and Pain Management Faculty of Medicine Ain Shams University for his great kindness, constant assistance and guidance.*

*Lastly, there are no words to express my gratitude to my **wife ,dr. Rafeak and to my family** who charged me with love and encouragement .*



Mohamed Youssef Mohamed

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List of Abbreviations

- AIDS** : Acquired immune deficiency syndrome
- ATP** : Adenosine triphosphate
- ARDS** : Acute respiratory distress syndrome
- cAMP** : Cyclic adenosine monophosphate
- cLDH** : Cytoplasmic lactate dehydrogenase
- CO₂** : Carbon dioxide
- COX** : Cytochrome c oxidase
- CPB** : Cardiopulmonary bypass
- DCA** : Dichloroacetate
- DKA** : Diabetic Ketoacidosis
- DNA** : Deoxyribonucleic acid
- ETC** : Electron Transport Chain.
- FFA** : Free fatty acid
- GPR81** : G-protein couple receptor

List of Abbreviations

H : Hydrogen

HCO₃ : Bicarbonate

H₂CO₃ : Carbonic acid

Hg : Mercury

HIF-1 : Hypoxia-inducible factor 1

HIV : Human immunodeficiency virus

ICU : Intensive care unit

IVC : Inferior vena cava

Kcal : Kilocalories

KSS : Kearns–Sayre syndrome

LDH : Lactate dehydrogenase

MCT : Monocarboxylate transport protein

MERRF: Myoclonic Epilepsy with Ragged Red Fibers

List of Abbreviations

MELAS : Mitochondrial Encephalopathy, Lactic Acidosis,
and Stroke-like episodes

MILS : Maternally Inherited Leigh's Syndrome

Na : Sodium

NADH : Nicotinic acid dehydrogenase (reduced form)

NAD⁺ : Nicotinic acid dehydrogenase

NaHCO₃ : Sodium bicarbonate

NH₂ : Amine

NRTI : Nucleoside analogue reverse transcriptase inhibitor

O₂ : Oxygen

PEO : Progressive External Ophthalmoplegia

List of Abbreviations

PGC1- α : Peroxisome proliferator activated receptor

gamma coactivator 1-alpha

pH : $-\log_{10}$ for Hydrogen ion

PLDH : Peroxisomal lactate dehydrogenase

PvCO₂ : Central venous carbon dioxide tension

PS : Pearson's Syndrome

RRT : Renal replacement therapy

TCA : Tricarboxylic acid cycle.

THAM : Tris-hydroxymethyl aminomethane

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Introduction

Lactic acidosis results from the accumulation of lactate and protons in the body fluids and is often associated with poor clinical outcomes. Mortality is increased when lactic acidosis accompanies low-flow states or sepsis, and the higher the lactate level, the worse the outcome. Although hyperlactatemia is often attributed to tissue hypoxia, it can result from other mechanisms. Control of the triggering conditions is the only effective means of treatment. (*Jeffrey et al., 2014*)

The cellular dysfunction in hyperlactatemia is complex. Tissue hypoxia, if present, is a major factor. If the cellular milieu is also severely acidic, cellular dysfunction is likely to be exacerbated. The later factor alone can decrease cardiac contractility, cardiac output, blood pressure, and can attenuate the cardiovascular responsiveness to catecholamines. The major causes of lactic acidosis have been divided into disorders associated with tissue hypoxia (type A) and disorders in which tissue hypoxia is absent (type B). Cardiogenic or hypovolemic shock, severe heart failure, severe trauma, and sepsis are

Introduction and Aim of the Work

the most common causes of lactic acidosis, accounting for the vast majority of cases. (*Kraut et al., 2012*)

Treatment is directed towards correcting the underlying cause of lactic acidosis and optimizing tissue oxygen delivery by cardiopulmonary support. Evidence so far indicates that alkali therapy is not beneficial; it may, in fact, cause harm by worsening intracellular acidosis. Furthermore, bicarbonate therapy may lead to electrolyte disturbances. Although patients may be tachypneic initially, ventilatory muscle fatigue may ensue rapidly and may require mechanical assistance. (*Puskarich et al., 2013*)

Aim of the Work

To review the current medical literature as regards the pathophysiology, etiology, presentations and management of lactic acidosis during anesthesia.

Chapter (1)

*Lactate shuttle, pathophysiology of
lactic acidosis and hyperlactatemia
during anesthesia*

Lactate Shuttle, Pathophysiology of Lactic Acidosis and Hyperlactatemia During Anesthesia

Glucose Metabolism:

Glucose is the only fuel in biology. It is used as an energy source in living cells; from bacteria to humans. Use of glucose may be by either aerobic respiration, anaerobic respiration, or fermentation. Glucose is the human body's key source of energy, through aerobic respiration, providing about 3.75 kilocalories (16 kilojoules) of food energy per gram. Breakdown of carbohydrates (e.g. starch) yields mono- and disaccharides, most of which is glucose. (*Fairclough et al., 2004*)

Anaerobic and Aerobic Metabolism of Glucose:

1- Glycolysis and Krebs's Cycle:

Glucose is stored mainly in the liver and muscles as glycogen. It is distributed and used in tissues as free glucose. Through glycolysis and later in the reactions of the