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RESEARCH PAPER

Evaluation of serial venous and arterial lactate concentrations in healthy anesthetized sheep undergoing ovariectomy

Lindsey A Mathews*, Michele Barletta†, Daniel C Almeida*, Lynelle F Graham* & Jane E Quandt‡
*Department of Veterinary Clinical Sciences, College of Veterinary Medicine, University of Minnesota, Saint Paul, MN, USA
†Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, GA, USA
‡Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, University of Georgia, Athens, GA, USA

Correspondence: Michele Barletta, Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, 501 DW Brooks Drive, Athens, GA 30602, USA. E-mail: mbarlett@uga.edu

Abstract

Objective To determine if lactate concentrations in jugular venous and auricular arterial blood differ in anesthetized sheep.

Study design Prospective, controlled experimental study.

Animals Twelve healthy adult ewes, 4–7 years and weighing 62–77 kg.

Methods Jugular venous blood was collected before anesthesia ($PreO_v$) for measurement of lactate concentration, packed cell volume and total protein. Ewes were administered a standard anesthesia protocol. Jugular venous ($IntraO_v$) and auricular arterial ($IntraO_a$) blood samples were obtained 40 minutes after induction of anesthesia, and again in recovery ($PostO_v$ and $PostO_a$). An additional blood sample was drawn 6 weeks post-operatively from non-fasted sheep (NF_Lact). Lactate concentrations were compared among $PreO_v$, $IntraO_v$ and $IntraO_a$, $PostO_v$ and $PostO_a$, and between $PreO_v$ and NF_Lact with paired t-test and repeated measure analyses of variance (ANOVA) with $PreO_v$ as a covariate ($p \leq 0.05$).

Results $IntraO_v$ lactate concentration had decreased from $PreO_v$ There were significant differences between arterial and venous IntraO and PostO lactate concentrations. There was no significant difference between IntraO and PostO, or $PreO_v$ and NF_Lact.

Conclusions and clinical relevance Lactate concentrations were significantly lower in anesthetized sheep compared to non-anesthetized sheep. Lactate concentrations in venous blood were higher than in arterial blood. Therefore, anesthetic status and sampling site should be considered when interpreting lactate concentrations, and the sampling site should be consistent for repeated measurements.

Keywords arterial, lactate, sample site, serial, sheep, venous.

Introduction

The importance of measurement of blood lactate concentration during anesthesia and critical care has been made clear in both human and veterinary medicine (Karagiannis et al. 2006; Allen & Holm 2008; Tennent-Brown et al. 2010; Zacher et al. 2010; Green et al. 2011). With the introduction of point-of-care monitors, serial measurements of blood lactate concentrations are easily performed. Knowledge of blood lactate concentration has proven beneficial in the indirect assessment of tissue perfusion and tissue oxygenation (Lagutchik et al. 1998; Hughes et al. 1999; Pang & Boysen 2007; Green

et al. 2011). Lactate is now considered a crucial diagnostic measurement for detection of sepsis and tissue necrosis, and can serve as a prognostic indicator in veterinary patients (Pang & Boysen 2007; Green et al. 2011). Elevations in mammalian lactate are generally the result of anaerobic glucose metabolism needed to produce adenosine triphosphate (ATP) in the face of insufficient tissue oxygenation. This process produces an amount of lactate that is easily cleared by hepatic and renal filtration mechanisms. However, during disease these mechanisms can become overwhelmed. The body begins to accumulate lactate and lactic acidosis can occur (Allen & Holm 2008).

Two types of lactic acidosis are termed type A hyperlactatemia (hypoxic form) and type B hyperlactatemia (non-hypoxic form) depending on the cause of increased lactate in the blood (Karagiannis et al. 2006). A critically ill patient with poor perfusion generally forms a type A hyperlactatemia. Prolonged cellular hypoxia and anaerobic metabolism produce lactate as a by-product that overwhelms metabolic processes. Type A hyperlactatemia should be considered whenever a patient has decreased tissue perfusion or decreased oxygen delivering capabilities, such as anemia or hypoxemia (Karagiannis et al. 2006). Type B hyperlactatemia occurs in the absence of hypoxia but as a result of metabolic defects or decreased clearance of lactate such as with severe liver disease, diabetes mellitus, neoplasia, toxin ingestion, or certain drugs (Karagiannis et al. 2006).

Cellular processes produce L-lactate whereas D-lactate is produced by bacterial metabolism (Allen & Holm 2008). Since the method of measurement in this study was using a point-of-care device, only L-lactate was measured. Elevations in lactate concentrations can occur during anaerobic glycolysis and lactate is produced mostly by skeletal muscle, brain, gastrointestinal tract, red blood cells and also by the skin. In healthy animals, excess lactate is changed back to pyruvate in the liver and kidneys where it can be utilized again in metabolic processes and energy production following the Kreb's cycle, gluconeogenesis and further glycolysis (Pang & Boysen 2007; Allen & Holm 2008). Mechanisms that decrease oxygen delivery to tissues, such as shock, and mechanisms that increase oxygen demand, such as exercise, hyperthermia, and inflammation, can cause a relative oxygen deficit at the tissue level. The end result in both situations forces the cells to undergo anaerobic glycolysis to maintain the production of ATP, while increasing lactate production. This elevation in cellular lactate ultimately causes elevations in blood lactate concentration until the body is able to either metabolize or excrete the excess via the liver and the kidney, respectively (Pang & Boysen 2007; Allen & Holm 2008).

While a single blood lactate measurement may be useful in certain situations, the value of only one reading has been called into question (Nel et al. 2004; Stevenson et al. 2007a; Zacher et al. 2010; Green et al. 2011). More emphasis is now being placed on sequential blood lactate measurements to determine a patient's response to therapy (Nel et al. 2004; Stevenson et al. 2007a; Zacher et al. 2010; Green et al. 2011). Lack of improvement in serial blood lactate concentrations may indicate the need for more aggressive therapy, surgical intervention, or a poor prognosis (Nel et al. 2004; Pang & Boysen 2007; Stevenson et al. 2007a; Zacher et al. 2010; Green et al. 2011). For example, a significant elevation in lactate immediately after cardiac arrest is not necessarily indicative of a poor prognosis. If the blood lactate concentration in this patient does not decrease over time, it signifies the presence of one or more abnormalities that must be corrected.

The appropriate site for collection of blood for lactate measurement has been argued in both human and veterinary medicine. The assumption has been that arterial blood lactate would reflect a more uniform concentration since the travel time of arterial circulation is low and is not influenced by regional metabolic processes (Gallagher et al. 1997; Hughes et al. 1999; Evron et al. 2007). Clinically, however, the difference in arterial concentrations in comparison to central venous or mixed venous concentrations seems to be insignificant in humans (Gallagher et al. 1997).

To the best of the authors' knowledge, serial blood lactate measurements comparing arterial to venous blood samples have not been evaluated in anesthetized sheep. The purpose of this study was to determine if there is a difference in lactate concentrations between jugular venous blood and auricular arterial blood in sheep. We hypothesized that 1) venous lactate concentrations would be higher than arterial lactate concentrations, 2) intraoperative blood lactate concentrations would decrease from pre-anesthesia baseline concentrations, and 3) non-fasting, 6 week post-anesthetic concentrations would be similar to fasted, pre-anesthetic concentrations.