LACTATED FLUID USE IN REPTILES: IS THERE A BETTER SOLUTION?

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Abstract: Reptiles produce lactate as a result of anaerobic metabolism. A number of studies demonstrate that healthy reptiles accumulate relatively large amounts of lactate after undergoing various physical and physiological exertions. A reptile is normally able to reduce its lactate load by engaging in various behavioral modalities such as inactivity and thermoregulation. It is the authors' belief that many sick reptiles presented for treatment have accumulated a significant lactate load. Illness, improper husbandry, and the stress of restraint for treatment all contribute to the metabolic accumulation of what can be massive amounts of lactate. These lactate loads present impediments to a number of physiological functions. It is the premise of this paper that fluid therapy involving the use of lactated Ringer's solution adds to the lactate burden of the animal. The authors suggest that it is clinically beneficial to administer nonlactated fluid therapy to reptiles whenever possible.

Key words: reptiles, lactate, fluid therapy, anaerobiosis, lactic acid

INTRODUCTION

Treatment of ill reptiles frequently involves the use of fluid therapy to restore plasma and cellular fluid volumes and to assist in metabolite and electrolyte normalization. Lactated Ringer's solution is commonly suggested as the fluid of choice for fluid therapy in reptiles[6,7,12]. However, the authors believe that lactated Ringer's solution may actually be detrimental to the recovery of an ill reptile.

This paper presents a brief review of selected studies which demonstrate that a number of reptile species accumulate large amounts of blood and tissue lactate following a variety of activities. Additionally, fluid compartmentalization in reptiles is contrasted with that of mammals. The authors recommend a nonlactated fluid therapy protocol for reptiles. This recommendation is based on reptiles' physiological tendency toward severe lactic acidosis, compartmentalization of fluids, and typical fluid loss patterns of reptiles seen in clinical practice.

DISCUSSION

Lactate Metabolism in Reptiles

Reptiles utilize anaerobic metabolism under a variety of circumstances.[2,8,9,13,14,17] Anaerobic metabolism is an inefficient method for generating ATP when compared with aerobic metabolism, and results in the conversion of glycogen to lactate. It has been demonstrated that routine activities such as locomotion, diving, and mating, are generally within the aerobic scope for several species studied, and therefore do not result in accumulation of large lactate loads.[2,8,9,14] However, massive loads of lactate are generated by many reptile species following forced activity, bursts of voluntary activity, and, in some cases, hibernation.[2,8,9,13,14,17]

Resting reptiles have been shown to have blood lactate levels ranging from 4-20 mg/dl[2,9]. Moderate activity results in lactate accumulations of 30-80 mg/dl, and prolonged or vigorous activity quickly produces levels exceeding 100 mg/dl.[2,9,14] Reptiles with the higher concentrations of lactate are physiologically exhausted and are unable to appropriately respond to stimuli. Maximum or near maximum levels of lactate can be produced in only 30 sec of vigorous struggling in a number of lizard species.[2,9,13,14,17]

One study of the green iguana (*Iguana iguana)* has demonstrated that greater than two-thirds of an iguana's energy during activity is derived from anaerobiosis[13]. Slightly lower but similar figures have been reported for diverse and commonly seen reptiles such as *Pseudemys, Terrapene, Pogona (Amphibolorus), Alligator, Crocodylus, Physignathus, Tiliqua, Trachydosaurus, Anolis, Thamnophis, Sauromalus, Chelonia. Varanus* appear to be a general exception; these animals have a higher aerobic scope and proteinaceous blood buffers, both of which act to resist acidosis[2].

Temperature plays an integral role in several aspects of lactate metabolism in reptiles.[1,2,5,9,14,17] Although anaerobic metabolism is maximal in the first half minute of activity regardless of temperature, the ability to sustain activity through anaerobic metabolism, and to reach and tolerate maximal lactate amounts, is improved at the preferred body temperature for a number of species.[2,9,17] For example, one study has demonstrated that green anoles (*Anolis carolinensis*) at 37°C were able to produce 20% more lactate before exhaustion than anoles at 20°C[2]. The recovery from anaerobiosis is also temperature dependent, and is generally maximal near preferred body temperatures. Temperatures above and below preferred temperatures result in slower increases in tissue oxygenation, and slower removal of lactate (reconversion to glycogen). Reduction of large accumulations of blood lactate to resting levels takes hours to days even at preferred temperatures, and is severely hampered by low temperatures.[1,2,8,9,14,17]

Application to Fluid Therapy

So what is the clinical implication of all this physiological gumbo? First, massive lactate accumulation is common, quite rapid, and physiologically normal for many reptile species. Lactate, however, produces acidemia (decreased blood pH),[2,3] which causes a decrease in the ability of hemoglobin to effectively bind oxygen (known as the Bohr effect). This lactate induced acidemia/oxygen deficit can create a cycle of anaerobiosis if the animal cannot recover sufficiently[2,9]. Animals maintained above or below their preferred optimum temperature can succumb sooner to the adverse effects of lactic acidosis. Additionally, lactic acid loads may saturate hepatic abilities to eliminate lactate in the blood, and the prolonged acidemia can lead to significant electrolyte shifts through renal hydrogen ion/potassium exchanges[3,5]. Is it really that hard to imagine that the typical iguana or box turtle presented as the clinical "train wreck" of improper husbandry, poor nutrition, and infections, has already accumulated a substantial lactic acid load? In addition, even more lactic acid is produced during capture and transport, as well as during our examinations with accompanying restraint and sampling.

The Solution

Because additional lactic acid is probably the last thing a stressed or ill reptile needs, the authors suggest a 50/50 combination of Dextrose 5% in water and a nonlactated, isotonic multiple electrolyte solution (such as Normosol-R, (CEVA Laboratories, Overland Park, KS) or Plasma Lyte (Baxter Health Care, Deerfield, IL)). This combination forms a mildly hypotonic nonlactated solution that is rapidly utilized when administered subcutaneously, epicoelomically, intracoelomically or intravascularly. Descriptions of routes of fluid administration have been previously described.[6,7,10,12,15]

The authors believe that this nonlactated solution is physiologically more beneficial than lactated Ringer's solution or saline solution. Not only does this solution avoid the addition of lactate to an animal that is already likely to be overburdened with lactic acid, but it may also benefit the reptile patient because it contains buffers more readily utilized by reptiles. When lactated Ringer's solution is administered to mammals, the lactate in the solution generally acts as a buffer, binding hydrogen ions in blood and tissue. A reptile in lactic acidosis, however, will not derive any buffering benefits from additional lactate. Conversion of lactate to a bicarbonate equivalent is presumptively (as is true for all other animals studied) limited to the liver. These liver pathways are already fully saturated in a reptile in lactic acidosis. It is also possible that the lactate isomer present in lactated Ringer's is not utilizable by reptiles and therefore would be of no benefit even if the pathways were not saturated. (Dr. A.M. Merritt, 1997, pers. comm.). Acetate and gluconate, the buffers contained in Normosol-R and Plasma Lyte, may be better buffers for reptiles. Unlike lactate, conversion of acetate and gluconate can be performed in many non-hepatic cells. The effectiveness of these buffers, therefore, is not hindered by lactic acidosis[11].

The solution suggested by the authors is mildly hypotonic fluid. Use of a hypotonic fluid is warranted based on fluid compartmentalization and the common clinical forms of dehydration in reptiles. In reptiles, as in mammals, the two main fluid compartments are the extracellular fluid compartment (ECF) and the intracellular fluid compartment (ICF). In reptiles, as in mammals, the electrolyte compositions of these two compartments differ significantly - extracellular fluid is very high in sodium and very low in potassium; the opposite is true for intracellular fluid. In contrast to mammals, however, reptiles have a relatively higher total body water[16]. Of this total body water, intracellular fluid volumes are also relatively greater than that of mammals. Extracellular fluid volumes, most of which is plasma, are generally lower than most mammals[16]. The osmotic pressure of plasma in reptiles is comparable to, or higher than, most mammals[4,7]. Taking into account a reptile's relatively lower? plasma volume and higher intracellular fluid volumes, restoration of intracellular fluid volumes is at least as important as restoring circulating plasma volumes in dehydrated reptiles.

Use of a mildly hypotonic fluid is also appropriate, and more beneficial than an isotonic solution, because of the form of dehydration most commonly seen in reptiles. In general, three forms of dehydration can occur: 1) isotonic dehydration, which is generally associated with hemorrhage, severe tissue damage, emesis, diarrhea, or anorexia; 2) hypotonic dehydration, which generally results from prolonged anorexia; and 3) hypertonic dehydration, which generally results from insensible water loss or failure to drink[10]. The form of any given reptile patient's dehydration must be evaluated by the clinician based on history, clinical signs, and diagnostics. In the authors' experience, some degree of water loss is common in reptile patients. Thus, the administration of mildly hypotonic fluids (as previously described) more quickly replenishes intracellular and total body stores than an isotonic preparation.

Does the choice of fluids make a clinical difference to our reptile patients? This has not yet been objectively evaluated. Anecdotally, both authors have experienced excellent clinical responses to nonlactated, mildly hypotonic fluid treatments. Hopefully, the preceding assertions based on reptile and mammal physiology will be supported or refuted by controlled studies and clinical assessments of actual patients.

In a final note - calcium, potassium, sodium, and magnesium differences exist between lactated Ringer's, physiological saline solution, and maintenance and replacement formulations of nonlactated buffered solutions. The clinician must evaluate the need for the above ions with specific consideration of the patient's needs, particularly when using fluid therapy that is of longer duration and routes that are more direct (such as i.o. and i.v. routes). Exploration of electrolyte interactions in reptiles is beyond the scope of this paper and the authors' expertise.

CONCLUSION

Lactic acidosis is highly likely in our reptile patients. A safe, efficient nonlactated fluid formulation discussed in this paper avoids compounding acidemia and maximizes intracellular rehydration in reptile patients.

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