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Letter to the Editor

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Dear Editor

We thank you for the opportunity to respond to the letter from Drs. Gomez-Nieto, Arroyo, Sears, and Viel regarding our recent publication entitled "Effects of alkalinization and rehydration on plasma potassium concentrations in neonatal calves with diarrhea".[1] The authors of the above letter have taken the first and the last sentence out of a two page discussion in which we essentially addressed their points of criticism.

In our conclusion "intravenous administration of hypertonic sodium bicarbonate solutions to neonatal calves with diarrhea and strong ion acidosis induces an immediate and sustained plasma potassium-lowering effect that appears to be caused by its efficient and rapid alkalinizing ability" we chose to use the phrasing "appears to be" on purpose, indicating that we could not say with absolute certainty. We then proceed with an in depth discussion as to why we came to this conclusion. Part of this discussion addressed the work that has been done on the treatment of hyperkalemia in humans with renal failure or experimental settings in dogs. We have previously shown that the pathogenesis of hyperkalemia in calves with neonatal diarrhea is complex as it depends on the nature of an existing acidosis but more importantly on the degree of dehydration.[2,3] It is also generally accepted that hyperkalemia in neonatal calves with diarrhea likely results from an acidemia-induced efflux of potassium ions out of the intracellular space which is likely occurring in spite of a negative potassium balance and whole body potassium depletion.[4,5] A simple experimental setting in which anesthetized dogs are infused with KCl to induce hyperkalemia[6] is therefore not a useful model that can be extrapolated to diarrheic calves. Studies comparing the effect of hypertonic versus isotonic sodium bicarbonate infusions on plasma potassium concentrations in humans with end-stage renal failure could indeed not find a profound effect for both solutions.[7,8] However, in one of those studies the utility of isotonic and hypertonic NaCl solutions were also tested under the same circumstances and there was even a tendency to increase potassium levels.[8] Again, this is not a good model to compare to diarrheic calves, as the calves in our study that received hypertonic bicarbonate solution did, without a doubt, experience a profound decrease in plasma potassium concentrations.

In our article, we also critically discussed the effects of immediate plasma volume expansion after the administration of hypertonic sodium bicarbonate solutions, the potential additive potassium-lowering effects of glucose solutions, and any problems related to the effects of different dosages of sodium bicarbonate, different osmolarities of infusion solutions and any interactions of those on the resulting change of plasma potassium concentrations. Consequently, we certainly admitted that there were some obvious weaknesses in the study which arose from the objective to provide recommendations for practitioners, who in most cases need to treat their patients on the basis of clinical signs. This study protocol was also chosen for this investigation as we were dealing with hospitalized patients, as previous studies failed to create an experimental model simulating the complex metabolic alterations occurring in diarrheic calves.[9]

The authors of the letter also stated that our conclusion "the combination of rapid alkalinisation with hypertonic sodium bicarbonate followed by a continuous infusion of larger volumes of iso- or slightly hypertonic solutions represents the best treatment strategy in dehydrated neonatal calves with diarrhea and clinical signs of hyperkalemia" could not be drawn from our findings as we did not include a control group. Again, it has to be emphasized that we did not try to find a way to treat isolated hyperkalemia in calves, but to treat "dehydrated neonatal calves with diarrhea and clinical signs of hyperkalemia". Results of our previous analyses[2,3] and the data of the present study show, that hyperkalemia in diarrheic calves is unlikely to occur in the absence of acidemia. Administration of alkalinizing fluids is considered crucial for the intravenous rehydration of depressed and dehydrated calves with diarrhea[10] and a previous study has already shown that hypertonic rehydration therapy alone (using a 5.85% saline solution in combination with an oral electrolyte solution) is not sufficient for the successful treatment of dehydrated diarrheic calves with concurrent moderate to severe acidemia.[11] We, therefore, did not deem it necessary, or ethically justifiable for the present study, to include a control group treated with solutions without alkalinizing capacity for a period of 24 hours.

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