

RVC, a congenital defect of female and male Jersey cattle, is characterized by stenosis of the vulvo-vestibular portion of the female reproductive tract and stenosis of the anal sphincter in both sexes. Parturition is abnormal due to inadequate relaxation of the vestibular ring and vestibulovulvar area. Dystocia in primigravida heifers can be relieved only by episiotomy or Caesarian section. Furthermore, udder edema in many RVC heifers or cows is so severe and persistent that it results in mastitis and loss of quarters. Rectovaginal constriction is a genetic defect most likely stemming from homozygosity of a simple autosomal recessive gene.

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Intravenous High Potassium Therapy for Diarrheic Calves

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Summary

During the course of diarrhea in neonatal calves, a number of intracellular and extracellular fluid and electrolyte changes occur. Effective therapy should be directed towards restoration of whole body balance, not treatment of changes only in the vascular compartment. Although hyperkalemia is present during acute diarrhea, there is loss of potassium from cells and a whole-body potassium deficit. Intracellular-extracellular potassium imbalance causes detrimental changes in the membrane's potential in many tissues. Restoration of a more normal membrane potential requires replacing the intracellular potassium deficient and correcting the extracellular hyperkalemia. This is accomplished by initiating the movement of potassium in the cells and addition of potassium to the extracellular pool via the replacement therapy.

In dehydrate-diarrheic-acidotic calves with elevated plasma potassium concentrations 8.5 ± 0.7 mEq/l, the administration of 1 liter of fluid containing 23 mEq/l of potassium in 60 minutes resulted in a decrease in plasma potassium concentration to 6.7 ± 0.2 mEq/l, while intracellular potassium concentration increased from a diarrheic concentration of 147 ± 4 to 153 ± 4 following therapy. This occurs because of the inclusion of both glucose and bicarbonate in the therapy: glucose augments potassium flux into the intracellular pool and bicarbonate causes a transmembrane potassium-hydrogen ion exchange. Potassium continues to decrease towards normal as long as the extracellular glucose concentration is elevated. Bicarbonate increased both intracellular and extracellular pH during and following therapy. The immediate change was 0.1 pH units in both pools.

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