TITLE: POTASSIUM SUPPLEMENTATION IN B2-AGONIST INDUCED HYPOKALEMIA

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Introduction. Acute hypokalemia occurs routinely during the infusion of B2-agonists used for tocolysis. There are conflicting reports in the literature concerning the need for supplemental potassium (K+) in these patients. This study was undertaken to determine the efficacy of supplemental K+ in animals receiving ritodrine.

Methods. Ten mongrel dogs in four groups were induced with thiopental and maintained with halothane and oxygen. Ventilation was adjusted to maintain a PaCO2 of 35-45 torr. Lactated Ringer's solution (LR) was infused at 5 ml/kg/hr. Group I dogs received no medication added to this infusion. Group II received potassium chloride (KCl) 0.5 mg/kg/hr, Group III 2.8 mcg/kg/min ritodrine, and Group IV 0.5 mcg/kg/hr KCl and 2.8 mcg/kg/min ritodrine. After three hours all medications were stopped and infusion of LR was continued for two more hours. Arterial blood gases, pH and serum K+ were measured at baseline and every 30 minutes. Urinary K+ was measured hourly. Serum lactate was measured at baseline, three and five hours. Statistical analyses were done using Scheffé's test for multiple comparisons.

Results. In all groups arterial blood gas tensions remained at control levels throughout the experiment. K+ alone (Group II) caused a significant (P<0.01) rise in serum K+ during the infusion. The urinary excretion of K+ also increased (P<0.01) in this group. In contrast, ritodrine (Group III) caused a significant (P<0.01) fall in serum K+ but no change in urinary excretion as compared to Group I. Ritodrine + KCl (Group IV) caused no significant change from Group I in serum or urine K+. Serum pH levels fell during infusion to a significant (P<0.01) level in Groups III (ritodrine) and IV (ritodrine + KCl). The decreased pH in Group IV appeared slightly greater but was not statistically different from Group III. Serum lactates rose to significant (P<0.01) levels in Group III and IV as compared to Groups I and II, and there was no statistical difference between Groups III and IV.

Discussion. A decrease in serum K+ with no increase in urinary excretion of K+ occurred with ritodrine infusion. This acute hypokalemia, which occurs with ritodrine as with other B2-agonists, is probably due to a shift of extracellular K+ into the cell secondary to the action of the glucose-insulin pump. The stable serum and urine K+ levels seen in Group IV (ritodrine + KCl) as compared to Group II (ritodrine alone) must be due to the intracellular shift of this exogenous K+. Recommendations to restore serum K+ to normal by the administration of K+ have been made. Our data show that infusion of KCl with ritodrine will prevent the hypokalemia which has been seen with ritodrine infusion alone. No adverse effects could be found with the infusion of the supplemental K+. We noticed a downward trend in serum pH though not significant when K+ was added to the ritodrine infusion. Although no deleterious effects were found with vigorous K+ supplement in our study, supplemental K+ is probably unnecessary since there is no total body K+ deficit but rather only a compartmental shift of K+. Our recommendation is further substantiated by the lack of cardiovascular changes due to the acutely decreased K+.