

POTASSIUM BALANCE AND β_2 -ADRENERGIC STIMULATION IN THE CONSCIOUS DOG

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It has been recognised for a number of years that catecholamines can induce changes in serum potassium concentration (1). Hypokalaemia appears to be mediated by way of β receptors (2). It is possible that both β_1 and β_2 receptors are involved. Salbutamol is a β_2 agonist used in the management of asthma and parturition and, potentially, for afterload reduction, each circumstance one where potassium status is of clinical importance.

The effect of salbutamol infusion has been assessed in two groups of five conscious dogs. A 100 μ g bolus of salbutamol was given followed by an infusion of 3 μ g/min. In both groups, arterial plasma K^+ (CA K^+) fell significantly by 0.9 mmol/l during infusion over 60 minutes. Urinary electrolyte excretions, measured in one of the groups, indicated that the hypokalaemia was not due to urinary potassium loss. It was deduced that potassium had moved intracellularly. No change in H^+ status occurred to account for this. A pronounced rise in plasma insulin immunoreactivity from a basal level of 87 ± 12 pmol/l to a peak of 1583 ± 378 pmol/l suggested that insulin might be responsible for the potassium shifts.

Other studies on cardiac pacemaker cells and on avian erythrocytes suggest that sympathomimetic agents may directly affect potassium transport by cell membranes. Salbutamol, with little β_1 agonist activity, might not be expected to affect directly membrane transport of K^+ in the heart. Measurement of arterial-coronary sinus potassium differences (CA-CS K^+) revealed no significant potassium loss from the heart with established hypokalaemia (CA-CS $K^+ = 0.1 \pm 0.1$ mmol/l when CA K^+ had fallen from 4.4 to 3.5 mmol/l). Although an early prolongation of QT (standard ECG lead II) occurred with salbutamol (5' after commencement of infusion) this may have been due to a low extracellular potassium.

This study shows that β_2 stimulation in the dog produces hypokalaemia by movement of potassium from the extracellular to the intracellular compartment, although the myocardium may not share significantly in this movement.

References:

1. D'Silva, J.L. J. Physiol. (1934) 82:393
2. Todd, E.P., and Vick, R.L. Amer. J. Physiol. (1971) 220:1964