Title : SERUM POTASSIUM LEVELS DURING PROLONGED HYPOTHERMIA

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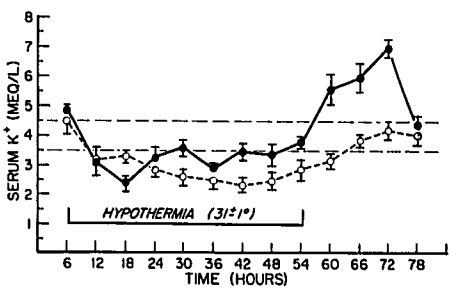
Introduction. Current practice of brain resuscitation for head-injured patients in our institution involves whole body hypothermia for about 48 hours. During this treatment, we noticed that serum potassium fell below normal in 3 patients. Correction of potassium deficit with intravenous repeated doses of KCl resulted in hyperkalemia during the rewarming period. We discontinued this form of treatment in our next 3 patients. The literature gives conflicting information on the effect of prolonged hypothermia on serum potassium.[1]

Methods. Six patients with brain trauma or hypoxia had their body temperatures lowered to 30-32° C (rectal) by surface cooling and maintained at this level for 48 hours. Intravenous fluid intake was limited to 1500 ml/day of 5% dextrose in 0.45% saline solution. Dexamethasone, 100 mg initially followed by 8 mg every 2 hours was given to all patients. Five of the six patients also received thiopental sodium 15 mg/kg initial dose followed by 4 mg/kg/hour by continuous infusion. Arterial P_{CO2} was maintained between 35 and 40 torr by controlled ventilation. Intracranial pressure, ECG and the EEG were monitored. Potassium concentrations were serially measured in serum, urine and gastric drainage. In the first 3 patients, potassium was infused to maintain serum potassium levels above 3.5 meq/l. In the next 3 patients, potassium was given only in the quantities needed to replace the losses measured in the urine and gastric juice. At the end of 48 hours, all patients were rewarmed over a 6-8 hour period to 37° rectal temperature.

Results. The results of the two forms of therapy are summarized in figure 1. In the first 3 patients rewarming resulted in elevated serum potassium levels above 5.5m Eq/L and in each case this was associated with cardiac arrhythmias (t-wave elevation, premature ventricular contractions) which responded to treatment with Kayexalate enemas, and intravenous insulin and dextrose. In the next 3 patients, we elected to withhold all potassium therapy in excess of measured losses, while closely observing the ECG. No arrhythmias were seen during the period of hypothermia. When these patients were rewarmed, serum potassium returned to normal levels and no arrhythmias were seen.

Discussion. We have observed that serum potassium falls with prolonged hypothermia. Potassium replacement in excess of potassium loss results in elevated serum potassium during rewarming to levels that can cause cardiac arrhythmias. We suggest that only the losses of potassium during hypothermia be replaced.

FIGURE 1. EFFECT OF HYPOTHERMIA AND REWARMING ON SERUM POTASSIUM LEVELS.



Serum potassium levels were measured at 6 hour intervals in 2 groups of 3 patients each during whole-body hypothermia for 48 hours, and during rewarming for 24 hours. One group (●—●) received supplemental KCl to maintain serum potassium concentration above 3.0 during the period of hypothermia. This treatment resulted in elevations of serum potassium which required treatment at 72 hours. The second group (●—●) received only sufficient KCl to replace measured urine and gastric losses. The broken lines represent the normal range of serum potassium concentration.

References.

1. Churchill-Davidson, H.C,: A practice of anesthesia, Philadelphia, W.B. Saunders, 1978. p.611.