Title : POTASSIUM CHANGES FOLLOWING VASCULAR CLAMP RELEASE DURING RENAL TRANSPLANTATION

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Introduction. Cardiac arrests have been reported to occur following release of the vascular clamps in patients undergoing renal transplantation.[1,2] In each case the donor kidney was perfused with Collins' solution, a solution whose potassium concentration approximates that of intracellular fluid (115 meq/L). This solution has been shown to protect cadaver kidneys during storage. Increases in blood potassium have been shown to occur following release of the vascular clamps during renal transplantation. The source of this potassium is not clear. The potassium could be released from the cadaver kidney or from restoration of the circulation to the leg whose circulation had been interrupted during renal vascular anastomosis or, from both. A study was undertaken to determine the source of the potassium and the extent of the rise in potassium occurring during renal transplantation by comparing blood potassium changes immediately prior to and following release of the vascular clamps in patients receiving Collins' perfused kidneys and patients receiving kidneys from living donors, containing no preservative solution.

Method. Twenty-four consenting adult patients undergoing surgery for renal transplantation were included in the study. Eighteen patients received kidneys perfused with Collins' solution (Group 1) and six received kidneys (from living donors), which did not contain Collins' solution (Group 2). The two groups were similar with respect to age and ASA physical status. Six of the eighteen patients in Group 1 (33%) and 1 of 6 (17%) in Group 2 were insulin-dependent diabetics.

Anesthesia was induced with thiopental. The use of muscle relaxants, depolarizing or nondepolarizing, to facilitate intubation was left to the discretion of the anesthesiologist. The proportion of patients receiving succinylcholine was similar in the two groups. Anesthesia was maintained with oxygen, nitrous oxide, halothane and nondepolarizing muscle relaxants when necessary. Following induction, a 5-1/4", 16 gauge Angiocath catheter was inserted into the right internal jugular vein. Blood potassium was measured from a peripheral vein prior to induction and immediately thereafter. Blood potassium was measured from the central line immediately prior to and 15 seconds after release of each vascular clamp, as well as at 45 minute intervals throughout the case. Blood gases were also measured in most patients immediately prior to and following release of the vascular clamps as well as at hourly intervals. Blood glucose was measured in diabetic patients.

Efforts were made to drain the kidney of its preservative solution prior to restoration of its circulation and vascular clamps were released slowly. Patients who had demonstrated metabolic acidosis during surgery received appropriate amounts of sodium bicarbonate and those patients who demonstrated a high or a rising potassium with or without EKG changes received intravenous insulin, glucose, and calcium in an effort to lower the serum potassium prior to release of the vascular clamps.

Blood potassium was measured on an Orion Model 30 sodium/potassium analyzer.

Blood potassium values before and after release of the renal vascular clamps were compared for statistically significant differences using a paired t-test.

Results. In patients receiving cadaver kidneys perfused with Collins' solution, blood potassium increased from 4.66 ± 0.15 meq/L (mean + SEM) to 5.04 ± 0.17 following release of vascular clamp ($p < .01$). In patients receiving kidneys from live donors (not perfused with Collins' solution), blood potassium changed from 4.62 ± .12 meq/L to 4.78 ± .16 meq/L following release of clamps. (P N.S.)

The maximum rise in potassium was 1.1 meq/L.

Discussion. These results indicate that the Collins' perfused kidney is the major source of the potassium rise following release of the vascular clamps during renal transplantation despite drainage of the kidney prior to anastomosis and slow release of the clamps. Although some patients had potassium increases of up to 1.1 meq/L, the potassium values following clamp release were all in the normal range in our patients. Because a higher peak level is reached if the initial level is greater, elevated blood potassiums were actively lowered using glucose and insulin prior to clamp release in our patients.

In conclusion, potassium infusion from a Collins' perfused kidney produced a small increase in blood potassium, which may be clinically significant in patients with pre-existing hyperkalemia.

References.

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2. Hirshman CA, Edelstein G: Intraoperative Hyperkalemia and Cardiac Arrests During Renal Transplantation in an Insulin Dependent Diabetic. Anesthesiology, in press.