

Case Report

# *The Use of a Hemoconcentrator for Management of Sudden Acute Hyperkalemia During Hypothermic Cardiopulmonary Bypass*

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## **Abstract**

A case is presented of a 64-year-old male undergoing saphenous vein coronary artery grafting. With application of the aortic cross-clamp and infusion of antegrade cardioplegia, unexpectedly severe aortic insufficiency caused the majority of the cardioplegic solution to enter the left ventricle and be returned to the oxygenator reservoir via the left ventricular sump. A total of 5,600 ml of cold blood cardioplegic (1,120 ml of crystalloid-K<sup>+</sup>=100 meq/L) solution was infused. Because of the inability to obtain electrical silence of the myocardium with cardioplegia, myocardial protection was obtained by total body hypothermia. The resultant serum

potassium was 7.4 meq/L at a blood temperature of 16°C. Because of the possibility of even higher serum potassium levels with rewarming, the hemoconcentrator was used to remove potassium with the ultrafiltrate. Volume was replaced with normal saline and packed red cells. Post-bypass, a cell saver was used to decrease the total volume returned to the patient, while further raising the hemoglobin concentration. The rationale for using the hemoconcentrator, and the potential consequences of untreated hyperkalemia accompanying severe hypothermia are discussed.

## **Introduction**

Adverse effects of acute hyperkalemia as a complicating factor during weaning from cardiopulmonary bypass (CPB) are well known (1). Hyperkalemia is manifest on the electrocardiogram by early repolarization, peaked, tented "T" waves, and shortening of the Q-T interval. Significant problems associated with hyperkalemia during weaning from cardiopulmonary bypass include possible atrioventricular block, disappearance of the "P" wave, progressive widening of the QRS complex to a "sine-wave," and eventual asystole (2). Hypothermia with (3) or without (4,5) CPB is usually associated with hypokalemia. The occurrence of sudden, severe hyperkalemia during extreme hypothermia is a potentially ominous event requiring immediate and precise treatment. This case report describes the use of the hemoconcentrator for rapid, controlled correction of hyperkalemia caused by an unexpected infusion of a large volume of cardioplegia

into the left ventricle and returned to the reservoir just prior to deep hypothermia.

## **Case Report**

A 64-year-old male was scheduled for a double saphenous vein coronary artery bypass grafting. Cardiac catheterization had revealed operable coronary artery disease and an ejection fraction estimated at 50%. There was no documented history of myocardial infarction. A preoperative echocardiogram revealed "mild" aortic insufficiency (AI). The patient had a history of hypertension controlled with enalapril and nifedipine. There was no history of renal disease or diabetes mellitus. Preoperative laboratory values were normal and serum potassium (K<sup>+</sup>) was 5.0 milliequivalents per liter (meq/L).

Induction of anesthesia, initial opening of the chest and institution of CPB were uneventful. The surgeon applied the aortic cross clamp and started antegrade infusion of the cold crystalloid/blood cardioplegia solution mixed in a 1:4 ratio (Table 1). It soon became evident that the AI was much worse than originally recognized. Most of the cardioplegic solution entered the left ventricle (LV) and passed into the oxygenator reservoir through the LV sump. The myocardium continued to fibrillate.

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Increasing the flow of the cardioplegic solution, and manual compression of the heart were not effective in establishing electrical silence. The patient was cooled to 16°C blood temperature. However, by the time the patient's temperature reached 16°C, some 4,700 ml of cardioplegic solution (94 meq K<sup>+</sup>) had been administered over 20 minutes. A total of 5,600 ml (112 meq K<sup>+</sup>) was administered over 45 minutes. The serum K<sup>+</sup> reached 7.4 meq/L. Of all options available, it was felt that the use of the hemoconcentrator would provide the most appropriate means of lowering the serum K<sup>+</sup>. A hemoconcentrator<sup>a</sup> was installed in the bypass circuit. Because the ultrafiltrate reflected the high K<sup>+</sup> concentration of the serum, the ultrafiltrate was replaced with normal saline (N/S) in order to lower the serum K<sup>+</sup> concentration. Over the next 15 minutes, approximately 1,500 ml of ultrafiltrate was replaced with N/S, with a concomitant decrease in serum K<sup>+</sup> to 5.6 meq/L. Ultrafiltration was continued throughout rewarming because of the continued possibility of rewarming hyperkalemia. A total of 3,500 ml of ultrafiltrate was replaced with 2,800 ml of N/S over one and one-half hours, guided by frequent monitoring of the serum K<sup>+</sup>. Just prior to weaning from CPB, the serum K<sup>+</sup> was 4.9 meq/L. The patient received two units of packed red blood cells during CPB to raise the hemoglobin concentration to acceptable levels. Following CPB, excess oxygenator volume was shunted to a "cell-saver" to provide additional concentrated red blood cells. Post-CPB, the serum K<sup>+</sup> was 4.1 meq/L and the hemoglobin was 10.8 grams/deciliter.

## Discussion

Potassium regulation in humans is a complex mechanism (6) which includes gluco-regulatory hormones, alpha- and beta-adrenergic agonists and antagonists, acid-base balance, renal function, exercise, drugs (5) and temperature (7,8).

Hypokalemia associated with hypothermia in humans may be a positive prognostic sign for survival, while normo- or hyperkalemia may represent just the opposite. The etiology of the hypokalemia is not known, but has been postulated to be due to hepatic uptake of K<sup>+</sup> (6). Hypothermic cardiopulmonary bypass (HCPB) is also generally associated with mild hyperkalemia which returns to normal following rewarming (3,8). Hyperkalemia during HCPB may be associated with renal failure, diabetes mellitus, severe acid-base alterations and infusion of cardioplegic solutions (1). Time-honored treatments for hyperkalemia include administration of sodium bicarbonate, calcium, potassium-losing diuretics, potassium exchange resin enemas and glucose-insulin combinations (2,6). All these methods are time consuming, relatively unpredictable, and, therefore, were impractical in our situation. Scavenging of the cardioplegic solution from the right heart has been shown to be effective in the prevention of hypokalemia (9,10). However, this was also impractical because the majority of the cardioplegic solution was entering the LV directly and returning to the oxygenator via the LV sump. A serum K<sup>+</sup> of 7.4 meq/L at 16°C, if left uncorrected, could be associated with extremely high serum K<sup>+</sup> concentrations on

a Model 1200, COBE Laboratories, Lakewood, CO 80215

**Table 1**

Composition of Crystalloid Cardioplegic Solution (mixed 1:4 with blood)

• KCl	100 meq/L	50 ml
• CaCl <sub>2</sub>	3.6 meq/L	2.6 ml
• MgSO <sub>4</sub>	48 meq/L	12 ml
• NaHCO <sub>3</sub>	185 meq/L	185 ml
• Regular Insulin	40 u	0.4 ml
• D10		750 ml

rewarming (8). Because of an anticipated short CPB time and the need for a rather narrow serum K<sup>+</sup> concentration range during weaning from CPB, correction needed to be accomplished in a rapid, but precise, fashion. Use of the hemoconcentrator proved to be an excellent choice for achieving those goals.

The hemoconcentrator has a pore size of 30-35 Angstroms and an average molecular weight cut-off of about 55,000 Daltons. With this molecular weight cut-off, fluids, electrolytes and some heparin and free drug fractions are in the ultrafiltrate. Cellular components, plasma proteins and protein-bound drug fractions are not filtered. Studies have not shown significant concentrations of free hemoglobin in the ultrafiltrate (11,12).

Although many reports describe the usefulness of the hemoconcentrator in controlling volume and hemoglobin during CPB (11-15), they mention its possible use in electrolyte control only in passing. However, if the hemoconcentrator is used primarily for electrolyte control, as in our case, hemoglobin concentration becomes an irrelevant end point in judging the volume of filtrate to be removed, and additional packed cells may be necessary to maintain an adequate hemoglobin level. In our case, although the filtrate volume was some 5,000 ml, only 2,800 ml of N/S was used to replace the filtrate volume, as judged by serum K<sup>+</sup> concentrations. Even with this limited crystalloid volume replacement, it was necessary to administer two units of packed cells during CPB to maintain adequate hemoglobin concentration. We felt the use of the "cell-saver" during CPB would not have afforded us the degree of control we needed for rapid and predictable regulation of the serum K<sup>+</sup>. Therefore, we elected to wait until CPB was discontinued to use the "cell-saver" to raise the hemoglobin and adjust the patient's intravascular volume.

We have found only one other report (16), and that is in the Japanese literature, in which the hemoconcentrator was used to correct cardioplegic-induced hyperkalemia. To our knowledge, this is the first English language report of such a use for the hemoconcentrator.

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