Letters

Dear Dr. Cross:

Thank you for your case report using the ultrafiltrator as a tool for treatment of hyperkalemia (The Use of a Hemocencentrator for Management of Sudden Acute Hyperkalemia During Hypothermic Cardiopulmonary Bypass. J Extra-Corpor Technol. 1992; 24(1): 33-35).

I will admit that I was somewhat surprised that you could find no other papers dealing with the technique of dilutional ultrafiltration, a two-part process first dealing with dilution of an unwanted plasma substance followed by removal (in the same concentration as present in the plasma) of the diluted substance, using ultrafiltration. The technique as far as I know is as old as the ultrafiltrator itself. I have heard it referred to as a "bastard" dialysis, and it is a standard portion of many hyperkalemia protocols. I frequently performed this procedure myself as a student at the Medical University of South Carolina.

I have several questions pertaining to the management of this case:

First, was suction utilized to facilitate the removal of excess plasma fluid following dilution? If so, what millimeters of mercury range was the suction regulator set at?

The choice of a diluent was normal saline. Did you have other electrolyte/pH imbalances with the patient? Normal saline is quite acidic, and is notorious for depleting patient bicarb levels and other wanted ions. Magnesium and ionized calcium levels can be drastically reduced with large infusions of normal saline, which may have a deleterious effect on the electroconductivity of the compromised myocardium. What electrolyte replacement did the patient require prior to removal of the cross clamp?

Finally, your report mentioned that the patient required two units of blood to "raise the hemoglobin concentration to acceptable levels." Were these transfusions related to a truly low volume, low hemoglobin state, or were they related to a low hemoglobin state secondary to excessive hemodilution. In the past in emergency situations, I have used multiple hemoconcentrators (2 or 3) from the same circuit with suction. This technique greatly increased the quantity of ultrafiltrate produced which allowed for a much greater quantity of diluent without large swings in the hemoglobin concentration.

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Dear Ms. Palmer:

I read with interest the case report by David A. Cross, titled "The Use of a Hemocencentrator for Management of Sudden Acute Hyperkalemia During Hypothermic Cardiopulmonary Bypass" (J Extra-Corpor Technol. 1992; 24(1): 33-35). However, I can not understand all the involvement and excitement over the excretion of approximately 20 mEq of potassium. The highest K measured was 7.4 mEq/L and this would have been a transient reading. The K level at the end of bypass was 4.9 mEq/L. The ultrafiltrate produced by a hemoconcentrator, regardless of the transmembrane pressure gradient, can only reflect the extracellular fluid present at the time of filtration. In this case, 3.5 liters of ultrafiltrate would contain, at most, 3.5 X 5.5 = 19.25 mEq K. If I have not read the article correctly and the total volume of ultrafiltrate was 5 liters, then the K filtered was approximately 27 mEq. Mannitol induced urine produced on bypass, in the presence of even reasonable renal function, will contain on average 12 - 20 mEq/L of K. Passage of one liter of urine on bypass can be accomplished easily, especially if perfusion pressures are kept a little high in the rewarm phase.

Administration of over 100 mEq of K occurs easily with the use of warm continuous retrograde cardioplegia for a double valve repair in our institution and we regularly come off bypass in those cases with K levels of 6 and 6.5 mEq/L. Within 30 minutes and no special intervention, the levels have fallen to close to normal.

I suspect that if Dr. Cross had done nothing other than administer some furosemide, there would have been no problem.

Looking forward to hearing from you. Thank you and regards.

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In response:

Thank you for the opportunity to respond to Dr. Bianchi's and Mr. Merrill's comments regarding my case report (1). Their comments are well thought-out and timely.

Mr. Merrill's letter contains numerous questions in detail about the case, many of which are not really pertinent to the main thrust of the paper. As any perfusionist knows, the details of management may vary tremendously and can be adjusted to provide the rate of filtrate formation desired within wide limits. Generally, we rarely use the hemoconcentrator. Although I personally believe we should make better use of it, our surgeons prefer the cell saver for hemoconcentration post-bypass. When we do use the hemoconcentrator, it is even more rare that we use it for electrolyte concentration problems. Consequently, I have not done the in-depth studies to which Mr. Merrill refers. However, I will address some of his more general questions.

First, the hemofilter in our case was connected via the blood cardioplegia line before mixing, and was returned to the cardiotomy reservoir. Transmembrane suction was applied at about 100 mmHg, but transmembrane pressure was not monitored. Second, although normal saline is acidotic, our cardioplegia solution contains 185 mEq/L of bicarbonate, and additional bicarbonate, Mg^{++} and Ca^{++} were added as needed. We used an alpha-stat CO_2 management, and the uncorrected blood pH was consistently 7.35 - 7.45 during CPB. Third, we did not take the time to determine what component of the red cell volume replacement was purely due to hemodilution, although dilution was a significant factor. The hemoglobin level quoted was following use of the cell saver in addition to administration of the two bank units. Use of the cell saver did not influence the serum potassium significantly. Fourth, in spite of a mannitol load with the pump prime, the urine output during severe hypothermia was 180 ml. The urine output increased significantly only after rewarming to a total output during CPB of about 500 ml. I will discuss some pertinent aspects relevant to urine output during hypothermic conditions in response to Dr. Bianchi's questions.

First, when using warm continuous cardioplegia, one is in a WYSIWYG (what you see is what you get) situation with regard to serum potassium. I would ask Dr. Bianchi to remember that the patient was cooled rapidly to 16°C. WYSIWYG does not apply in such a case. The 7.4 mEq/L was measured at that temperature, after the total administration of 112 mEq of potassium, and was maintained for about 45 minutes. I can account for only an additional 10 - 15 mEq at that serum level. My calculations of potassium removal via the hemoconcentrator were 30 - 35 mEq. However, it is well known that potassium is sequestered at very cold temperatures and released into the central circulation during rewarming (see references in my original paper). Moreover, it has been known since the 1960s that both glomerular filtration and potassium elimination during hypothermia is impaired significantly (2). This additional 30 - 35 mEq, in addition to an unknown quantity which could potentially be released into the circulation during rewarming, could be problematic. Until the hemofiltration was begun, the serum potassium did not change during the period hypothermia. In spite of the mannitol load via the pump prime, it was only after hemoconcentration was begun that serum potassium began to decrease significantly. The remaining serum potassium was further diluted by the addition of the normal saline. If 5000 ml of filtrate lowers the serum potassium by 30 mEq, and an additional 2800 cc of saline maintained a serum level of near 6.0 during rewarming, and 4.9 on termination of bypass and after resumption of urine output, there is an implication that a rather significant quantity of potassium was released into the system during rewarming. Part of the increase in serum potassium was undoubtedly handled via the increased urine output during and after rewarming. I must assume that the remainder of the potassium apparently re-equilibrated with the extracellular fluid compartment during the combined warm and cold phases. Also, it is not our custom to regularly discontinue bypass with serum potassium of 6 - 6.5 mEq/L. Finally, one should remember that I was less concerned with urine potassium concentration that serum potassium concentration, and was working with patient care in mind, rather than running a research project. had I planned in advance to do the case as we did, I would have certainly monitored all parameters more thoroughly. I am not suggesting that use of the hemoconcentrator is the only method which one may employ to correct an acute hyperkalemia, but is a useful adjunct when extremely low central temperatures may make other methods less predictable. It was a surprise to me that more information regarding the use of the hemoconcentrator for this purpose was not available.

References

1. Cross DA. The use of a hemoconcentrator for management of sudden acute hyperkalemia during hypothermic cardiopulmonary bypass. J Extra-Corpor Technol. 1992; 24: 33-35.
2. Boylan JW, Hong SK. Regulation of renal function in hypothermia. Amer J Physiol. 1966; 211: 1371-1378.

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