Hyperkalemia from Nonelectrolyte Solutions

To the Editor—A "regulatory volume decrease" through a shift of potassium from cells was suggested by Hirose et al. to explain the increase of the serum potassium concentration from nonelectrolyte solutions, such as mannitol and glycine. Studies made at this institution provide additional information about the nature of this hyperkalemia.

The increase in potassium is three times greater following intravenous infusion of isosmotic (2.2%) glycine compared to isosmotic (5%) mannitol solution in male volunteers, despite similar degrees of hyponatremia. This suggests that increased serum potassium is not related to hyponatremia per se and, hence, cannot be explained by dilution acidosis.

Glycine solution does not cause hemolysis or impair urinary excretion of potassium, and therefore, a shift of intracellular potassium remains the only possible cause of this hyperkalemia.

The intensity of the hyperkalemic response can be obtained by measuring serum potassium at the end of a short (20-min) intravenous infusion of nonelectrolyte solution. Results from such experiments in young male volunteers show that increase in serum potassium was 0.91 ± 0.35 mg/dL from a 2.2% solution and 0.36 ± 0.26 mg/dL from a 1.5% solution of glycine. The effect of toxicity would promote a more pronounced cell swelling from the hypo-osmotic solution, and this also is evidenced by a smaller dilution of the serum sodium during infusion of 1.5% glycine. However, if cell swelling triggers the hyperkalemia, which is implied by Hirose et al., one would expect the serum potassium concentration to correlate inversely with the glycine concentration of the infusion; but this is not the case.

Our data suggest that hyperkalemia is related directly to how much nonelectrolyte solution is transported into the cells rather than to the degree of cell swelling that triggers the "regulatory volume decrease." This hypothesis is consistent with the referenced changes in serum potassium. As the distribution volume of glycine at the end of glycine infusion is double the size of the extracellular space, one can assume that more glycine enters the cells during infusion of 2.2% glycine, and therefore, hyperkalemia is more pronounced. This hypothesis is supported further by experiments in sheep. The distribution volumes at the end of infusions of 1.8 L 2.2% glycine solution and 4.0 L 1.5% glycine were only 15–19 L and about 16 L, respectively. These volumes are similar in size to the expected extracellular space in these animals, which indicates that a surplus amount of glycine is transported into the cells much more slowly than in humans. There was no increase in serum potassium with the glycine, which is consistent with the view that hyperkalemia depends on the rate of intracellular accumulation of the nonelectrolyte solution. On the other hand, if hyperkalemia was triggered by cell

References


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swelling, one would expect increased serum potassium when 1.5% glycine was given, as the low osmolality of this fluid promotes diffusion of water into the cells.

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Potency Versus Onset of Neuromuscular Blocking Agents

To the Editor:—In the search for the ideal neuromuscular relaxant with rapid onset and short duration of action, the molecular design of such agents through elucidation of the kinetic mechanisms of receptor binding are being pursued and are of great importance.

The recent article by Min et al. has offered an elegant approach...