

volume of 70 ml and a potassium level of 31.8 mEq/l—the higher of two measured prearrest values), the calculated potassium infusion rate is 0.25 mEq · kg⁻¹ · h⁻¹.

In the studies of animals receiving potassium infusions to which the authors refer, ponies tolerated 1.78–1.99 mEq · kg⁻¹ · h⁻¹ for slightly over 1 h on the average before developing terminal dysrhythmias,⁴ and dogs received 2.0 mEq · kg⁻¹ · h⁻¹ for 3 h prior to exhibiting “impending” ventricular fibrillation.^{5,6} Also, Hiatt and Hiatt⁵ found that 0.3 mM/kg rapid potassium injection produced ventricular fibrillation in dogs, and not 0.1 mM/kg, as noted in the case report.¹ Hence, we conclude that this patient’s hyperkalemia resulted from a potassium load that was significantly less than stated in the case report and that was not given “in rates that far exceeded rates shown to cause cardiac toxicity in humans and animals.”

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In Reply:—Hyperkalemic cardiac arrest occurs when the potassium (K⁺) concentration gradient between interstitial fluid and the intercellular fluid of the myocardial cell decreases until cell membrane repolarization cannot occur. *In vivo* measurements of interstitial K⁺ concentration during hyperkalemic arrest are not available. Thus, we used whole blood sample K⁺ concentrations to reflect this gradient. Other studies of hyperkalemic arrest due to blood transfusions used blood infusion rates to reflect K⁺ load.^{1–4} The K⁺ determined expresses milliequivalents per liter of fluid tested. University of Wisconsin Hospital uses ADSOL[®]-preserved packed red blood cells. The absolute volume of plasma in each unit is unknown. The absolute milliequivalent infusion can only be estimated. The K⁺ in the packed red blood cell infusion significantly elevated the serum K⁺ (measured). This resulted in increased interstitial fluid K⁺ and cardiac arrest. The important point of the case report is that transfusion rates can exceed the ability of the body to equilibrate and normalize serum K⁺. The result in this case was cardiac arrest and death. In addition, the higher the K⁺ in these units, the greater the risk may be. Since I know of no other means to estimate the K⁺ load during transfusion than transfusion rate and unit K⁺ concentration, I continue to advocate vigilance when transfusion rates exceed 120 ml/min.⁴

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Antagonism of Vecuronium-induced Neuromuscular Block

To the Editor:—The study by Magorian *et al.*¹ “addressed the clinical problem of antagonizing a profound neuromuscular blockade” induced

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by vecuronium. The authors showed statistically that recovery time was the same regardless of whether neostigmine was given during deep