

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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Anesthesiology
74:1169, 1991

In Reply:—Hyperkalemic cardiac arrest occurs when the potassium (K⁺) concentration gradient between interstitial fluid and the intracellular 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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Anesthesiology
74:1169–1170, 1991

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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(Accepted for publication March 13, 1991.)

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(Accepted for publication March 13, 1991.)

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

or light neuromuscular blockade and that a repeat dose of neostigmine did not improve the situation. We have several concerns about this study.

First, we wonder if one or possibly two patients skew the results of this study. We do recognize that patient response is quite variable, but we are concerned about the range of values reported for recovery to 10% of twitch ($t(10)$) in the control patients (22–48 min) compared to the patients receiving a placebo and subsequently neostigmine (17–87 min). If the patient in the latter group who had the prolonged recovery of 87 min is eliminated, do the statistics change? If so, we feel this is clinically relevant and should be reported. On the one hand, manipulating and selecting data to draw conclusions is scientifically invalid; on the other, one patient should not make or break a study.

Even if the statistics do not change, we clinically do not agree with the authors' conclusions "that there were no differences in recovery among the three groups of patients who received neostigmine." In our practice, reversal of a vecuronium-induced neuromuscular blockade in 55 min rather than 75 min is a significant difference. The additional 20 min of paralysis can be costly indeed to the patient, hospital, and anesthesiologist, depending on the circumstances and on what needs to be done to manage the problem. Furthermore, the study showed that reversal of neuromuscular blockade with no visible twitch present results in a prolonged period of partial paralysis ($t(10-90)$) exactly when one does not need it—*i.e.*, during emergence of anesthesia. Is

not ventilation and sedation until reversal is rapid and predictable preferable? In short, we believe that this study demonstrates that reversal of vecuronium-induced neuromuscular blockade should not be attempted until an adequate twitch is present.

Last, we wonder why some of the $t(10)$ and $t(90)$ results represented in figure 4 of the study do not agree with the data presented in table 2.

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(Accepted for publication March 13, 1991.)

Anesthesiology
74:1170, 1991

In Reply:—We would like to thank Dr. Bentley and Dr. Lahet for their careful reading of our paper and for their comments on it. Their observation that the data in table 2 and figure 4 do not correlate exactly is correct. We apologize for this, as it is a result of an error in the plotting of the figure. The data in table 2 are correct with the exception of a typographic error in the range for $t(10)$ in the placebo/placebo group. The range should be 22–63 min and not 22–48 min, as printed. The concern expressed by Bentley and Lahet about the apparent difference in variability between the $t(10)$ values for the placebo/placebo group and placebo/neostigmine was, we think, partly the result of the error in the published data.

The results of our statistical analysis were not dependent on the results from any one patient. Our results were based on a prospective power analysis to determine the numbers of patients we should study, and the patients were assigned to their group in a randomized manner. Therefore, we are confident in the statistical validity of our results. However, to address the particular concern of Bentley and Lahet, we reanalyzed our data with the patient to which they refer excluded. Our results were unchanged; *i.e.*, there were no differences in time to 90% twitch height recovery or to the attainment of a train-of-four ratio of 75% among the three groups who received neostigmine, regardless of whether neostigmine administration was early, late, or repeated.

Because the neostigmine groups were not statistically different, we must correct the contention of Bentley and Lahet that they were clinically different. They contend that the difference between a mean of 55 and 75 min is clinically significant. However, these particular mean values represent only our sample and are only estimates of the population mean. What the statistics tell us is that the values for the population means are not likely to be different. Therefore, they are un-

justified in claiming a clinical difference in recovery times, because there is in fact no difference.

Also, they comment that the early administration of neostigmine "results in a prolonged period of partial paralysis ($t(10-90)$) exactly when one does not need it", *i.e.*, during emergence of anesthesia. We do not understand the point of this comment. The alternative to early administration of neostigmine and partial paralysis in this situation is not to administer neostigmine until later, when a twitch response is present. Consequently, during emergence from anesthesia, the patient will be paralyzed completely—surely an equally undesirable situation and one that requires continued endotracheal intubation and ventilatory support.

They imply by their comments that we recommended the early administration of neostigmine at a time when there is no twitch response. Although we inherently agree that neostigmine should not be given until a twitch response is present, we were unable to prove any harm in giving neostigmine when no twitch response was present.

We are pleased that our paper and conclusion stimulated concern about the important issue of antagonism of neuromuscular blockade.

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(Accepted for publication March 13, 1991.)