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In reply—We appreciate Dr. Sosis’ concerns about the priming principle, but disagree with many of his interpretations of our data and the literature. First, our results (Taboada et al.1) are consistent with those of O’Hara et al.2 and Engbaek et al.3 We sequentially investigated various vecuronium (VEC) priming doses, priming intervals (time interval from priming dose to intubating dose), and intubating doses. The priming dose of VEC, 10 μg/kg iv, given to awake sedated patients, allowed a faster onset from the intubating dose than no priming dose, and had minimal symptoms and side effects during the priming interval. O’Hara et al.2 established VEC dose–responses in patients during nitrous oxide–narcotic–thiopental anesthesia. It is not valid to equate the results from priming doses in awake patients with those of anesthetized patients. On the other hand, Engbaek et al.3 found decrements in train-of-four ratios from VEC, 10 μg/kg, in awake patients. This is not surprising, since train-of-four monitoring is a greater stress to the neuromuscular junction than single twitch and, thus, the former is more sensitive than the latter. The train-of-four ratio (used by Engbaek et al.3) can be decreased when single twitch tension (used by Taboada et al.1) is not. Of note, however, is the fact that VEC, 10 μg/kg iv, only caused the train-of-four ratio to decrease to 0.86 (range 0.76–0.94), which is well within previously described guidelines for a greater respiratory function.4 In addition, Engbaek et al.3 found no significant change in respiratory frequency, vital capacity, and inspiratory force from VEC, 10 μg/kg iv. Peak expiratory flow was decreased from 475 to 460 l/min in these patients. These results suggest that adequate ventilation and airway protection should be present in patients who receive VEC, 10 μg/kg iv, as a priming dose.

Furthermore, Engbaek et al.3 did not find higher incidence of side effects and symptoms from the VEC, 10 μg/kg iv, priming dose than we did. Engbaek et al.3 measured a few different side effects, such as ptosis and headlift, than we did, but with those measurements our studies have in common the results are virtually the same. For example, one of nine patients from Engbaek et al.3 had difficulty swallowing, while in our study one of 11 patients had difficult swallowing. We do not feel that the sedation in our study resulted in underestimation of side effects from the priming dose because all patients responded to our sequential evaluation of these side effects. The more important consideration to the reader is the effect of preinduction sedation with diazepam and narcotic on the rapidity of development of paralysis from the intubating dose of VEC. We do not know how important this sedation was on onset time from the intubating dose.

We do not consider blurred vision to be an important dangerous effect from priming. Blurred vision has been for years a well-accepted side effect from the widely accepted practice of administering a “defasciculating” dose of nondepolarizing muscle relaxant prior to the administration of succinylcholine. The most important implication from Dr. Sosis’ letter is the concept that there may be patients who have an exaggerated response to the administration of a priming dose of nondepolarizing muscle relaxant. We agree with this concept. However, we feel that appropriate precautions and vigilance for this consequence are the appropriate response rather than abandonment of the technique entirely. After all, we have been using the same required vigilance with the “defasciculating” dose of nondepolarizing muscle relaxant before succinylcholine for years. So, why should the priming dose of VEC be of greater concern?

In addition to the existence of patients who are very sensitive to a priming dose, there are patients who will be resistant. For example, in our study two patients had onset time from the administration of the intubating dose of VEC to 100% depression of twitch tension equal to 140 s, when the priming dose was VEC 10 μg/kg (priming interval = 4 min; intubating dose = VEC 0.1 mg/kg iv). This possibility should be anticipated, and appropriate management can be aided by information gained from a
peripheral nerve stimulator applied to the patient prior to induction of anesthesia (stimulation can be begun after induction of anesthesia).

We do not believe the previous report from Sosis et al. seriously proves the priming principle to be nonefficacious. Our interpretation of their results is that they simply chose an inferior combination of priming dose, priming interval, and intubating dose to test the technique.

Despite the priming principle, succinylcholine still has the most rapid onset of neuromuscular blockade. We were careful to point out that use of the priming principle "may be the method of choice when succinylcholine is contraindicated or undesirable." In these instances, the use of the priming principle will shorten the onset of complete neuromuscular blockade by more than a minute in most patients compared with the use of a single, large dose of nondepolarizing muscle relaxant. We feel that this fact makes the use of the priming principle an important clinical tool.

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Concentration versus Partial Pressure: Which Is Important?

To the Editor:—I wish to call attention to a possible misinterpretation of the recent article by Nakagawara et al. They present interesting findings for enflurane, halothane, and isoflurane that have potential relevance to immune defenses against infection and cancer. However, the relevance of the findings may be limited because the anesthetic partial pressures used in this study exceed those applied in clinical practice. The three anesthetics were equilibrated with a modified Hanks' solution "by bubbling each vaporized anesthetic with a carrier gas of air (4 l/min) at 4°C on a shaking plate." The solution temperature then was increased to 37°C.

This increase in temperature decreased the solubility of the anesthetic and thereby increased the partial pressure of anesthetic. The increase can be calculated from the data supplied in figure 1 of the paper. That figure indicates that 1% enflurane, halothane, and isoflurane (the lowest concentrations used) produced 0.43, 0.45, and 0.37 mg of anesthetic per liter of electrolyte solution, respectively. Each concentration can be converted to a partial pressure (given as a percentage of one atmosphere) at 37°C by the following calculations: divide by the molecular weight to give the moles; multiply by 22,400 ml/mole to give the ml of anesthetic at standard conditions; divide by 310/273 to give the ml at 37°C; divide by the electrolyte/gas partition coefficient to give the equivalent ml of anesthetic vapor per liter of air; and divide by 10 to give the percentage of one atmosphere composed by the anesthetic. These calculations indicate that at 37°C the "1%" concentrations actually were 8.0% enflurane, 7.7% halothane, and 9.3% isoflurane. Such lethal concentrations had no effect on the generation of superoxide. An effect on intracellular free calcium was produced by these concentrations of halothane and en-