sympathetic tone. They assumed that a calf-minus-toe index of 0°C indicated the presence of significant baseline sympathetic tone, whereas we required a gradient of 4°C. We maintain that in this group of patients we would have measured some temperature changes had a significant sympathectomy been induced by intrathecal sufentanil.

Davidson and Ginosar also point out that 6 of 12 subjects in Matsukawa’s study did not develop a complete sympathectomy. This is probably not a reflection of methodologic problems with temperature changes failing to demonstrate the presence of a sympathectomy. We doubt that a sympathectomy actually occurred in these subjects and would not have expected temperature changes to occur in all patients. Rather this demonstrates that an epidural block induced with 1% lidocaine to T10 does not consistently block the transmission of all sympathetic stimuli to the lower extremities. Blocks may have been segmental, patchy, or some of the sympathetic control of the lower extremities may derive from above T10.

The above argument is refuted by Davidson and Ginosar, who refer to a paper by Stevens et al., stating that there is no difference between the sympathetic block induced by a spinal and an epidural anesthetic. However, Stevens paper deals with a different phenomenon than the one we are discussing. The authors specifically attempted to induce a complete sympathectomy that would ablate adrenal and cardiovascular responses to a cold pressor stimulus applied in the upper extremity. In contrast, Matsukawa et al. and our group were looking at changes in sympathetic tone in the dermatomal levels affected by light, analgesic blocks.

Davidson and Ginosar also point out that core hypothermia in the bupivacaine group was not necessarily caused by redistribution. They claim that it could have been caused by losses to the environment (intravenous fluid, exposure in the operating room, and so on). We believe this is incorrect for several reasons:

1. The core temperature cooled, whereas the periphery warmed up. Had there not been a sympatholysis that resulted in redistribution, then the periphery should also have cooled.
2. The spinal anesthetic was induced after fluid loading. Any changes in core body temperature resulting from the fluid loading had already occurred before induction of spinal anesthesia.
3. The changes in core and peripheral temperatures began immediately after spinal anesthesia induction and were well established before significant environmental losses could have occurred.

Davidson and Ginosar also criticize our choice of a control group (cesarean section patients), claiming they were dissimilar from our study group (laboring women). We appreciated that these groups are different, although we wanted a control group that we were sure would develop a sympatholysis so as to avoid equivocal results. Therefore we chose cesarean section patients as our control group. None of our results can be explained logically by baseline differences between the groups. We maintain that differences between the groups did not affect our results or conclusions.

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To the Editor:—I read with interest the article by Benumof et al. examining the development of critical desaturation after paralysis with 1 mg/kg of succinylcholine. I am concerned that some of the authors’ interpretations, as well as the commentary in “This Month in Anesthesiology,” may not be entirely justified and may, in some cases, lead to premature or potentially hazardous intervention. Benumof et al. conclude that “before significant desaturation. . . a rescue option should be instituted aggressively and early.” The commentary warns that “potentially fatal hypoxia may occur long before recovery.” These conclusions are based largely on a mathematical model of hemoglobin desaturation during apnea, duration of succinylcholine effect culled from other articles, and an arbitrary assumption of what constitutes functional neuromuscular recovery to ensure adequate ventilation.

I am primarily concerned about the reasonably healthy adult patient, who comprises the majority of most anesthesiologists’ cases. First I am not sure the authors’ determination of the duration of succinylcholine effect is accurate. How the mean time to 50% recovery after succinylcholine was determined is not clear; one of the references cited used 0.8 mg/kg; another used 40 mg/m² BSA, which using their data is > 1.1 mg/kg; and a third reported 10%, 25%, 75%, and 90%, but not 50% twitch height recovery. Further, one reference reports the time to 50% recovery began at the time of injection and that 80 ± 24 s elapsed before paralysis occurred.
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another reports time to recovery began at the first indication of a reduction in twitch, not at the cessation of ventilation. In the other references it appears, but is not clearly stated, that the beginning time was at the time of injection, not at the time ventilation stopped. Based on my reading of the cited references, the mean time from cessation of ventilation caused by paralysis until recovery of 50% twitch could be shortened by 30–90 s.

Second the requirement of a return to 50% peripheral twitch height may not be necessary for some minimal level of ventilation that would prevent critical hemoglobin desaturation. One of the references states that, during general anesthesia, after paralysis with succinylcholine, “in most cases respiration had begun prior to any demonstrable activity at the thumb.” A second study, also during general anesthesia, measured the actual apneic period and found it to be approximately 2 min less than the time required for 50% twitch recovery. A third reference reported diaphragmatic recovery paralleling adductor pollicis recovery but preceding it by 2 min at all degrees of recovery. Finally a study of unanesthetized volunteers receiving succinylcholine by continuous infusion showed that airway patency was maintained, and vital capacity and inspiratory pressure were greater than 65% of control when peripheral strength was 20% of control. Even during general anesthesia, 50% peripheral twitch recovery may not be the absolute lower limit for sufficient ventilation with 100% oxygen to maintain hemoglobin saturation.

Benumof et al. perform their analysis and base their recommendation on the use of a 1 mg/kg dose of succinylcholine. However, at least one of their references and several major textbooks of anesthesia recommend 0.5–1.0 mg/kg for an intubation dose in healthy adults and note that the ED95 for intravenous succinylcholine is 0.20–0.25 mg/kg. Further, although the duration of paralysis is directly related to dose, once recovery begins it proceeds at approximately 25% per min. Regardless of dose (within the clinical range of 0.2–4.0 mg/kg). After an intravenous dose of 0.5 mg/kg succinylcholine, the time to 50% twitch height recovery is about 5.4 min, at which time the apnea model predicts an oxyhemoglobin saturation of 97–98% for a healthy adult.

Bearing in mind that no subjects were studied, it seems to me that, in the instance of a healthy adult, Benumof et al. have overstated their case. If, using the information above, the actual duration of apnea is decreased by 45–60 s, then the hemoglobin saturation will only have decreased to between 94 and 92%. Or if the dose of succinylcholine is reduced to 0.6–0.8 mg/kg, then the duration of apnea would be in the 6–7 min range, and saturation would have decreased only to 98–96%.

I cannot dispute the data for small children or obese or ill adults, although more definitive data than presented would be preferable. My recommendation would be to minimize the need for aggressive intervention by always ensuring the best possible preoxygenation and by using the lowest effective dose of succinylcholine. Like any anesthesiologist, I would always be prepared for aggressive intervention, but I prefer prevention to intervention.

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