not extend into the postoperative period. We do not believe that any problems existed because our surgical colleagues have never been reticent about alerting us to any problems that might possibly be the fault of anesthesia, nor did our postoperative visits or QA reviews turn up any such problems. Much greater resources are necessary, though, to exhaustively study patients throughout the perioperative process. In the near future, the use of information systems will make longitudinal studies less resource-intensive and easier to do. We agree that this would be a better approach.

Another issue brought up by Drs. Fisher and Kelley is the fact that surgeons are under pressure to reduce costs by becoming more efficient. That is, unfortunately, not true in enough institutions. Although that may be true in particular markets where managed care has achieved significant market penetration, most of the country still operates under a misguided approach that allows surgeons incredible latitude when it comes to efficiency. As for their assertion that the increase in average case duration balanced the savings in anesthetic drugs, their figures are, I believe, erroneous. The case duration, calculated by dividing the cost/case by the cost/h in figure 1 of my original paper, was 2.7 - 2.8 h at each time point.

Finally, Drs. Fisher and Kelley’s comment about being unconvinced until a randomized prospective trial is done is unrealistic. Observational data for management and resource utilization purposes are a common methodology. I encourage Drs. Fisher and Kelly to read the JAMA editorial by Berwick referenced in my original paper or Dr. Duncan’s more recent editorial in Anesthesi vsia & Analgesia.

In conclusion, despite the acerbic nature of some of my comments, I am thrilled that this paper has generated such careful reading and so many incisive comments by so many anesthesiologists. In their critique, the correspondents are adhering strongly to the intended use of this article. The article as a whole was meant to stimulate discussion. Its detailed methodology was meant to encourage a careful analysis of whether its results are likely to be duplicated in a practice setting different from Duke University. The degree to which each physician agrees with what has been done in this experiment is irrelevant. It is of much greater importance that this paper has energized the discussion of how to best use limited societal resources in pursuit of uncompromised patient care.

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References
5. Duncan PG: That was then, this is now! The value of observing change (editorial). Anesth Analg 1998; 86:225-7

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Small Doses of Sufentanil Will Produce Violent Coughing in Young Children

To the Editor — The article by Bennett et al. (Anesthesiology 1997; 87:1070-4) was interesting and enlightening. I have observed during my personal practice and working with residents that small doses of sufentanil (1 μg/kg or less) will, commonly and unfortunately, produce violent coughing in young children and even in adolescents. Premedication with midazolam has a modest sparing effect. These children were clearly in distress, and some will, if one is not careful, become hypoxic without bradypnea or apnea. When I first noted this reproducible pattern, some of my colleagues, experts in opioid anesthesia, proposed that such coughing may merely reflect reduced chest wall compliance. Dr. Bennett has shed some light on this issue by demonstrating vocal cord closure in adults receiving sufentanil. The authors make no mention that boluses of sufentanil produced paroxysms of coughing in the patients they studied. Possibly this is just one more age-related difference. This phenomenon also may be of interest as an increasing number of patients, children and adults, are sedated with narcotics, given by non-anesthesiologists outside of the operating room. Encouraged by the work in this study, it would certainly seem appropriate to study this phenomenon in other patient age groups and with other, natural occurring and synthetic, narcotics. I am certainly curious how medication commonly believed to be cough-suppressing may have such a paradoxical effect on the vocal cords.

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In Reply.—We thank Dr. Yemen for his interest in our publication. Our adult patients have not experienced the episodes of “coughing” associated with the administration of sufentanil that he describes in children. The literature on opioid-induced difficult ventilation in neonates and children is at best confused and consists mostly of isolated case reports.

Baraka described post-extubation laryngospasm after opioid-based anesthesia in a 4-year-old child. Naloxone terminated the laryngospasm. MacGregor et al. described difficult ventilation after initiation of a fentanyl infusion in an intubated neonate. They extubated the child, fearing an endotracheal tube obstruction. They could not ventilate the extubated child. A cardiorespiratory arrest resulted. Naloxone was administered and restored the ability to ventilate. They ascribed the difficult ventilation to chest wall rigidity.

Perhaps the chest wall component plays a larger role in causing difficult ventilation in infants and children than in adults. The only way to clarify the issue is to conduct a prospective study in children similar to that done in adults.3

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References

1. Baraka A. Fentanyl-induced laryngospasm following tracheal extubation in a child. Anesthesiology 1995; 83:775
3. Bennett JA, Abrams JT, Van Riper DF, Horow JC: Difficult or impossible ventilation after sufentanil induction of anesthesia is caused primarily by vocal cord closure. Anesthesiology 1997; 87:1070-4

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Is Phenylephrine or Sodium Bisulfite Neurotoxic?

To the Editor.—We have read with interest the article by Sakura et al., in which they attributed the transient neurologic deficit seen in patients after spinal anesthesia to phenylephrine added to tetracaine solution. Regrettably the authors failed to acknowledge that the commercially available 0.5% phenylephrine solution (Kowa, Nagoya, Japan) contained 0.1% sodium bisulfite, well known for its neurotoxicity when administered neuraxially.2,3 The amount of sodium bisulfite given in their patients ranged from 0.5 to 0.75 mg, approximately half the dose that caused permanent hind-limb paralysis in rabbits, and approximately one tenth the concentration that caused irreversible spinal monosynaptic reflex in rats.1 Unless preservative-free phenylephrine solution is used in combination with tetracaine, phenylephrine, per se, cannot be regarded as an etiology of the reported transient neurologic sequelae.

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