

## CORRESPONDENCE

outpatient elective surgery and were otherwise completely healthy. The anesthetic technique in these infants consisted of an inhalation induction with oxygen, nitrous oxide, and halothane. None of the three received a barbiturate or an opioid.<sup>1-3</sup>

As mentioned in our discussion,<sup>4</sup> respiratory drive, even in the full-term infant, undergoes a maturational process, with normal "adult" breathing patterns and carbon dioxide response curves not achieved until 42-43 weeks postconceptional age. Before this age, "abnormalities" in respiratory drive are of similar type but lesser in magnitude and frequency when compared with the premature infant of a younger postconceptional age.<sup>5,6</sup> Postanesthetic apnea in the former preterm infant is well documented, and most pediatric anesthesiologists admit such patients for 12-24 h postoperatively for apnea monitoring up to a postconceptional age varying between 46 and 60 weeks.<sup>7,8</sup> As Hannallah *et al.* are aware, attention to the problem of postanesthetic apnea in expremature infants was focused originally by case reports<sup>9</sup> and a retrospective study,<sup>10</sup> and similar recommendations for inpatient admission and careful postoperative monitoring to ours were made at the time, before careful prospective studies were performed. Then, when careful studies using polysomnography by Welborn *et al.*<sup>7</sup> and Kurth *et al.*<sup>8</sup> were published, recommendations based on more objective data could be made. It is clear from those careful studies that many of the apneic spells were clinically unrecognized, detected only by the apnea monitor.

Although we do not think that postanesthetic apnea in full-term infants is nearly as frequent as in the expremature infant, nevertheless, the problem exists. Clearly, a large-scale prospective study of young, healthy full-term infants with polysomnography and preferably standardized anesthetic techniques as well as similar surgeries should be performed before more objective guidelines can be established. Until that time, we still recommend consideration of short-term postanesthetic apnea monitoring for young full-term infants, even if healthy and undergoing uncomplicated anesthetics.

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## References

1. Tetzlaff JE, Annand DW, Pudimat MA, Nicodemus HF: Postoperative apnea in a full-term infant. *ANESTHESIOLOGY* 69:426-428, 1988
2. Coté CJ, Kelley DH: Postoperative apnea in a full-term infant with a demonstrable respiratory pattern abnormality. *ANESTHESIOLOGY* 72:559-561, 1990
3. Karayan J, LaCoste L, Fuscuardi J: Postoperative apnea in a full-term infant. *ANESTHESIOLOGY* 75:375, 1991
4. Andropoulos DB, Heard MB, Johnson KL, Clarke JT, Rowe RW: Postanesthetic apnea in full-term infants after pyloromyotomy. *ANESTHESIOLOGY* 80:216-219, 1994
5. Albani M, Bentele KHP, Budde C, Schulte FJ: Infant sleep apnea profile: Preterm vs. term infants. *Eur J Pediatr* 143:261-268, 1985
6. Stebbins VA, Poets CF, Alexander JA, Arrowsmith WA, Southall DP: Oxygen saturation and breathing patterns in infancy: I. Full term infants in the second month of life. *Arch Dis Child* 66:569-573, 1991
7. Welborn LG, Ramirez N, Oh TH, Ruttimann UE, Fink R, Guzzeta P, Epstein BS: Postanesthetic apnea and periodic breathing in infants. *ANESTHESIOLOGY* 65:658-661, 1986
8. Kurth CD, Spitzer AR, Broennle AM, Downes JJ: Postoperative apnea in preterm infants. *ANESTHESIOLOGY* 66:483-488, 1987
9. Gregory GA: Outpatient anesthesia, *Anesthesia*. 1st edition. Edited by Miller RD. New York, Churchill Livingstone, 1981, p 1329.
10. Steward DJ: Preterm infants are more prone to complications following minor surgery than are term infants. *ANESTHESIOLOGY* 56:304-306, 1982

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## Sympathetically Maintained Pain after Surgery May Be Prevented by Regional Anesthesia

**To the Editor:**—Rocco<sup>1</sup> described two patients who had recurrence of their sympathetically maintained pain (SMP) after surgery under general anesthesia. The author then postulated that general anesthesia may cause a recurrence of SMP and suggested that regional anesthesia rather than general anesthesia be used in patients who had suffered previously from reflex sympathetic dystrophy. We strongly agree with Rocco and share his opinion concerning the possible role of general anesthesia, not only in "releasing a dormant pain," but also in favoring the appearance of SMP after surgery.

In our experience with patients presenting with SMP after surgery, we are confronted by the striking fact that most of them had surgery under general anesthesia. Two recent cases emphasize this point. The first patient was a 26-yr-old man who had severe multiple injuries of the right lower limb and developed SMP after surgery performed under general anesthesia. SMP was relieved with intravenous guanethidine blocks.<sup>2</sup> Two subsequent surgical procedures were performed using epidural anesthesia, with no relapse of SMP. Eighteen months later, this patient underwent a short-duration general anes-

## CORRESPONDENCE

thetia for plates and screws removal that was followed by the recurrence of SMP, again successfully treated with intravenous guanethidine blocks. The second patient was a 51-yr-old man who had three consecutive operations after a severe trauma of the right wrist and hand, with no subsequent SMP, despite a long-term immobilization of the limb. All three procedures were performed under brachial plexus block. Twelve months after the initial trauma, the second patient received general anesthesia for plates removal. One week later, he experienced severe reflex sympathetic dystrophy, which was treated successfully with intravenous regional blocks and stellate ganglion blocks.

If we consider that the syndrome of sympathetically maintained pain often is related to a dysfunction of the sympathetic nervous system,<sup>3</sup> we could postulate that regional anesthesia, by allowing the preoperative onset of a sympathetic blockade, could prevent its development. A preemptive effect of regional anesthesia has been advocated for other types of neuropathic pain, especially the occurrence of phantom limb pain after amputation.<sup>4</sup> Obviously, large prospective studies are required to confirm the possible preemptive role of regional anesthesia.

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## References

1. Rocco AG: Sympathetically maintained pain may be rekindled by surgery under general anesthesia. *ANESTHESIOLOGY* 79:865, 1993
2. Bonica JJ: Causalgia and other reflex sympathetic dystrophy, *The Management of Pain*. Edited by Bonica JJ. Philadelphia, Lea & Febiger, 1990, pp 250-256
3. Raja SN: Reflex sympathetic dystrophy: Pathophysiology and logical basis for therapy. *Pain Digest* 2:274-280, 1992
4. Bach S, Noreng MF, Tjelliden NU: Phantom limb pain in amputees during the first 12 months following limb amputation after preoperative lumbar blockade. *Pain* 33:297-301, 1988

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## Reevaluation of Physiologic Curvature of the Thoracolumbar Spinal Column in the Supine Position

**To the Editor:**—The physiologic curvature of the spinal column has a significant influence on the spread of local anesthetics in the subarachnoid space. With respect to the supine position, it is widely accepted that the highest point of the lumbar curvature is at L3 and the lowest point of the thoracic curvature is at T5-T6.<sup>1,2</sup> However, few data are available to support this concept, especially in living humans. We have reviewed sagittal magnetic resonance images of the spine in 10 patients obtained because of back pain, which were reported as normal by a radiologist. In none of the ten patients were the highest and lowest points of the spinal canal at L3 and T5-6, respectively. It is, however, common practice in those patients to place a pillow under the patient's knees for comfort. This probably influences interpretation of the magnetic resonance images. Therefore, we have examined sagittal magnetic resonance images of spine in 10 healthy volunteers to determine the highest and lowest points of the spinal canal. The volunteers comprised seven men and three

women, aged 16-59 yr (median 31 yr). They were kept supine, and the knees were extended. Again, we found that in none of the subjects were the highest and lowest points of the spine at L3 and T5-T6, respectively. Of the 10 subjects examined, the highest point of the lumbar spinal canal is at L4 in nine and at L3-L4 in one. The lowest point of the thoracic spinal canal was at T8 in five, T9 in two, T8-T9 in two, and T7-T8 in one. Typical sagittal magnetic resonance images of the thoracolumbar spinal canal are seen in figure 1. T1-weighted sagittal scans were obtained by a surface coil and fast spin echo. Although there may be ethnic differences in curvature of the spinal column, the highest and lowest points of the spinal canal, the level of termination of the spinal cord, and the level of termination of the dural sac observed in our volunteers were similar to those depicted in text books of magnetic resonance imaging.<sup>3-5</sup> However, because of the small number of subjects, the question concerning ethnic differences remains unsolved.