CORRESPONDENCE

First, in humans with cervical spinal cord transection, sympathetic neural transmission is interrupted anatomically and not, as in our study, by injection of local anesthetic into the thoracic epidural space that result in substantial local anesthetic plasma concentrations. In fact, our study shows that intravenous administration of a local anesthetic resulted in a significant attenuation to inhaled acetylcholine, which was not significantly different from that observed after epidural administration, despite bronchial sympathectomy. Thus, the effect of the local anesthetic per se seems to play a dominant role and must be considered in the interpretation of the observed effects in our study. Therefore, the findings that, in humans with spinal cord transection, bronchial hyperresponsiveness was present is not unexpected, and we agree that this might be explained on the basis of unopposed parasympathetic airway innervation in this specific patient population.

Second, the extent of direct sympathetic innervation of the bronchial system varies greatly from species to species. In guinea pigs, cats, and dogs, there is a well-developed direct sympathetic innervation of the bronchial system, whereas there is only a rare sympathetic innervation of the human airway.1 Using autoradiographic techniques, an additional sympathetic innervation of the bronchial system in humans is not well described (mainly glands, vessels, and alveolar walls, but not bronchial smooth muscle) and is assumed to have no functional relevance for airway resistance regulation.1,2 Finally, Peter J. Barnes (1992) writes in his review about modulation of neurotransmission in airways that, in contrast to guinea pigs, cats, and dogs, there is no adrenergic bronchodilator response to direct nerve stimulation in humans.3 Thus, results obtained from animal models may not represent the regulations of airway resistance in the human bronchial system.

In summary, we do not believe that the arguments raised by Dicpinigaitis et al. are contradictory to our statement that “blockade of pulmonary sympathetic innervation by thoracic epidural anesthesia seems to be of no relevance for airway resistance in humans.”

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Femoral Nerve Injury May Be Related
to Abdominal Wall Retractor

To the Editor—I read the recent article by Warner et al.1 with great interest. The Mayo Clinic's early adoption of computerized medical records provides an especially large data base within which to assess the frequency of rare complications, in this case, lower extremity motor neuropathy.

Warner et al. identified only four cases of femoral neuropathy, none of which was associated with either common peroneal or sciatic neuropathy. This low incidence is in agreement with the early report by Dhnuer2 in which all four cases of perioperative lower extremity neuropathy (in 30,000 anesthetics) involved the common peroneal nerve. One potential cause (often present in patients undergoing laparotomy in the lithotomy position) of isolated femoral neuropathy is direct pressure from the metal blade of an abdominal wall retractor. Is it possible that the four patients who suffered femoral nerve injuries had undergone intraabdominal procedures, possibly distinguishing them from patients with injury either to the common peroneal or sciatic nerves?

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