Reply:

We must thank Drs. Matic, Arndt, and Lee for their insightful comments. It is heartening to hear anesthesiologists echoing what we have been teaching residents and other practitioners in airway courses and workshops for more than a decade.

Additionally, these same sentiments and concepts form the foundation of our objectives in educating anesthesia practitioners to alternative ways of thinking and acting to address just the kind of "critical airways" that Matic and Arndt described. The use of simple but practical strategies to rapidly predict and identify difficulty in all of its dimensions (bag mask ventilation, supraglottic devices, tracheal intubation and a surgical airway) in an emergency, is fundamental to our overall teaching goal.

With the on-going emphasis in airway management research and education, we are confident that patient outcome will improve.

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Bilateral foot drop: looking for the needle in the wrong haystack?

To the Editor:

We read with interest the description of a case of bilateral foot drop following spinal anesthesia.¹ We have described the etiology of such postoperative deficits (preoperative injury, co-existing neuropathy, surgical trauma, tourniquets, positioning and anesthetic techniques).² The case mentioned requires further clarification.

1. Documentation of neurovascular deficits in any limb with fractures is considered part of basic trauma (orthopedic) examination. Further, trauma to the lumbosacral spine may also result in bilateral foot drop.

2. Did the patient have other co-existing disease with subclinical neuropathy (e.g., diabetes)? Pre-existing neuropathies have been identified as risk factors in nerve palsies.³

3. Does the plural "tibial pins" suggest that these were inserted on both sides?

4. Was the femur alone operated? How were the leg fractures managed? If they were also operated upon, the foot drop could be related to the surgery.⁴

5. What was the position of the patient during surgery? Femoral traction on a hip table (supine) may stretch the sciatic nerve. In the lateral position the sciatic nerve of the dependent side may be compressed.⁵

The authors erroneously identify paralysis of foot flexors as the cause of foot drop. Neurological examination is more important to rule out neurological deficits following the trauma rather than preceding the anesthetic (as they recommend). Prior to searching for iatrogenic causes, if they had documented the presence or absence of bilateral foot drop (at admission or after the traction pin application), they may not be faulted for looking for the metaphoric needle in the wrong anesthetic haystack!

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Reply:

This is in response to the letter from Dr. Naveen Eipe and Dr. Nihar Ranjan, regarding the above mentioned article in the May 2005 issue of the Canadian Journal of Anesthesia. I thank Dr. Naveen for his comments, and would like to clarify the points raised by him.

There was no documentation of preoperative neurological deficit in the basic trauma examination. It is

quite true that trauma to the lumbosacral spine can result in bilateral foot drop, but this was ruled out in the preanesthesia visit and the magnetic resonance imaging done postoperatively. The patient did not have any other coexisting disease with subclinical neuropathy (diabetes), although pre-existing neurological disease should also be ruled out e.g., lumbar disc syndrome or spondylitis or recent herpes zoster in such cases. The tibial pins were inserted on both sides, and the femur alone was operated during the first stage. The legs were surgically reduced in the second stage, so the foot drop could easily be related to the surgery. The patient was operated in the lateral position, no femoral traction was applied (on the hip table). We agree that the sciatic nerve compression in the lateral position could again be a factor (but unilateral).

Since the patient had no neurological deficit preoperatively, a neurologist's opinion was not sought. Consultation was undertaken postoperatively, after the patient developed foot drop. There was no documentation of the presence of foot drop at admission or after the insertion of tibial pins (bilateral) so the etiology was most likely related to surgery or spinal anesthesia. Bilateral foot drop after spinal anesthesia is possible only if lumbar puncture is performed above the third lumbar segment, which is uncommon. Considering the angle of entry of the spinal needle, trauma to one side of the conus is possible.

We emphasize that this case is not meant to attribute blame, but to highlight that this complication, though rare, does occur, and has its own medicolegal implications.

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Does bilateral thoracic sympathectomy predispose to reflex bronchospasm following tracheal intubation?

To the Editor:

Thoracic endoscopic sympathectomy has become the technique of choice for treating intractable essential hyperhidrosis.¹ We report severe bronchospasm following tracheal intubation in a patient with a previous history of bilateral thoracic sympathectomy. Consent for publication was obtained from the patient.

A 31-yr-old non-smoking woman, 60 kg, without a history of allergy, or asthma, was scheduled for left knee arthroscopy. Two months previously, she had an uneventful general anesthetic for bilateral thoracoscopic sympathectomy to treat essential hyperhidrosis. Preoperative examination revealed clear lungs. Following preoxygenation, anesthesia was induced with propofol 2.5 mg·kg⁻¹ *iv*, vecuronium 0.15 mg kg⁻¹ *iv*, and fentanyl 2 µg·kg⁻¹ *iv*. Ventilation by facemask was easy. Following onset of neuromuscular block, tracheal intubation was easily performed by direct laryngoscopy. Immediately following intubation, ventilation became difficult. Chest auscultation revealed bilateral expiratory wheezing associated with decreased air entry and increased airway pressure up to 60 cm H₂O. Oxygen saturation, as monitored by pulse oximetry, decreased from 100% to 80%. Anesthesia was deepened by inhalation of sevoflurane (4–8% inspired concentration) in 100% oxygen. Six doses of 20 µg ipratropium bromide and five doses of 100 µg salbutamol were administered via the endotracheal tube. During the following five minutes, ventilation became progressively easier, oxygen saturation increased to 100%, and breath sounds normalized. Anesthesia was continued with 2% to 4% sevoflurane. Throughout this event, no cutaneous flushing was observed, blood pressure ranged between 110/90 mmHg and 130/95 mmHg, with pulse rate between 65 and 115 beats·min⁻¹. Surgery and recovery proceeded uneventfully. Twelve weeks after surgery, an atopy skin patch test was negative.

The severe bronchospasm experienced by the patient may have been secondary to an anaphylactic reaction to the induction agents. In a recent French survey of anaphylaxis,² the most common features were cardiovascular manifestations (71.5%), followed by cutaneous symptoms (69%). The least frequent feature was bronchospasm which only occurred in 44.2% of the cases, and was associated with a history of atopy or asthma. In our patient, there was no history of atopy or reactive airway disease and the bronchospasm was not associated with any cardiovascular or cutaneous manifestations. The severe bronchospasm occurred immediately following tracheal intubation, suggesting that it may have been a reflex response which was triggered by instrumentation of the airway under a light level of anesthesia. Other causes of bronchospasm such as unrecognized aspiration, carinal irritation, secretions, or chemical irritants that may have contaminated the tracheal tube were also considered and excluded.

Patients with essential hyperhidrosis have sympathetic overactivity, associated with compensatory high parasympathetic tone. Sympathectomy results in