a leak. We describe here a simple method to identify a possible leak. The technique consists of disconnecting the carbon dioxide/anesthetic agent sampling tube (Datex Capnomac Ultima; Helsinki, Finland) from the patient breathing system and using the sampling tube to instead "sniff" around the vaporizer. We recently have had four different vaporizers of the Dräger Vapor 2000 (Dräger Medical AG & CO, Lübeck, Germany) variety leak. The "sniff" correctly identified the leak in all cases. No vapor concentrations were identified when the vaporizer is not leaking. We believe the problem with these vaporizers is the damage caused to the vaporizer during refilling. When filling these vaporizers, the lever need not be flush with the front of the vaporizer. The lever should only be pushed to achieve slight resistance. Closing the lever all the way causes a leak at the filling port. We bring this problem and a simple method to identify an external vaporizer leak to the attention of your readership.

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DOI: 10.1213/01.ANE.0000158999.42505.AC

Lidocaine Toxicity in Volunteer Subjects Undergoing Awake Fiberoptic Intubation

To the Editor:

The paper by Martin et al. (1) raises serious concerns. Anesthesiologists acting as subjects on a training course in awake intubation were used to validate a training manikin. Martin et al. failed to set an upper dose limit for topical lidocaine administration and observed side effects indicating lidocaine toxicity.

Death as a result of lidocaine toxicity has occurred in a healthy 19-yr-old college student volunteer after bronchoscopy for research purposes (2). That research protocol also failed to specify an upper dose limit for lidocaine. The safety of volunteers and subjects should be paramount. It is essential to specify an upper dose limit when using toxic drugs for research or when providing this type of training on volunteers.

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DOI: 10.1213/01.ANE.0000159000.93358.AC

In Response:

We appreciate the reiteration by Woodall et al. of our concerns regarding lidocaine toxicity in endoscopy volunteers in an educational setting. It is, however, incorrect to suggest that our study protocol did not include an upper dose limit. Our topical anesthesia procedure was discontinued if the lidocaine dose approached 15 mg/kg or if local anesthetic effect diminished before adequate topical anesthesia. This upper limit was based on two published studies (1,2) with ranges for total applied lidocaine of 5.5 to 16 mg/kg (average, 9.3 mg/kg) and 5.0 to 15 mg/kg (average, 8.2 mg/kg) in sedated patients. Mean plasma concentrations in these studies were 2.9 μg/mL and 1.29 μg/mL. Plasma concentrations exceeding 5 μg/mL were noted in two patients (1). No signs or symptoms of lidocaine toxicity were reported in either study.

The dose range of applied lidocaine in our study was 7.1 to 14.7 mg/kg with a median dose of 9.6 mg/kg. This is consistent with the previously published work and reflects the dose distribution in a study population of 39 nonsedated subjects. We reported a high incidence of subjective cerebral side effects. Similar symptoms are alluded to in the published description of the training course in local anesthesia of the airway conducted by Woodall et al. themselves (3).

The "acceptable range" for topically applied lidocaine is suggested from studies where the drug has been used successfully and without complications (1,2). This does not mean that the margins of the dose range are completely safe or effective. One-sided 95% upper confidence intervals can be calculated when the occurrence of an event (e.g., clinical lidocaine toxicity) is reported as zero, using the formula 3/n where n is the number of patients (4). In the series of Efthimiou et al. and Langmack et al., the risks of clinical lidocaine toxicity were less than or equal to 7.3% (3 of 41) and 5.9% (3 of 51), respectively. We employed a similar dose range in unsedated subjects and 7.7% (3 of 39) of participants showed objective signs of early lidocaine toxicity. No major adverse events occurred.

The 2001 British Thoracic Society guidelines (5) recommend limiting the topical dose of lidocaine to 8.2 mg/kg. This is based on the mean dose reported in the study of Langmack et al. (2). As many as half of the patients in this study required doses of lidocaine higher than the subsequent recommendations of the British Thoracic Society to achieve adequate topical anesthesia. Similarly, the upper limit used by Woodall et al.'s group of 9 mg/kg (3) is referenced to the work of Efthimiou et al. presumably based on their average dose of 9.3 mg/kg (1).

Although the imperative for successful topical anesthesia in the clinical setting may justify doses of lidocaine in the upper level of the acceptable range, especially when followed by general anesthesia, the same cannot be said when the procedure is performed on volunteers for educational purposes. It is not possible to predict which people in a healthy population will develop toxicity from doses in the upper range.

Successful application of topical anesthetic to the airway is not a safe endpoint (6). Despite the problems associated with nominating a maximum dose of lidocaine, it is nevertheless important if the risk of toxicity is to be minimized. We have subsequently adopted the published average dose (1) of approximately 9 mg/kg as our maximum dose for awake volunteers. This will not guarantee adequate topical anesthesia in all subjects nor will it guarantee plasma concentrations limited to the therapeutic range in all subjects. However, it should eliminate the risk of significant clinical toxicity.

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Overall, we found that adding a very small dose of intrathecal morphine (0.05 mg) to spinal bupivacaine and fentanyl, together with local anesthetic infiltration and regular nonsteroidal antiinflammatory drugs, produced improved postoperative analgesia and higher patient satisfaction despite a higher incidence of opioid-related side effects. We continue to recommend that clinicians consider using a small dose of neuraxial morphine for analgesia after postpartum tubal ligation in women who will be inpatients for 24 h after the operation.

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Awareness During Anesthesia

To the Editor:

The report by Sebel et al. (1) is worrisome if the conclusions are valid. Can the authors provide more documentation for their results? In which cases did personnel in the operating room confirm the recalled events, and what were the details? In cases that lacked such confirmation, how were the investigators convinced of awareness? Is it possible that what the authors interpreted as recollections of intubation were actually recollections of extubation?

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DOI: 10.1213/01.ANE.0000159009.93168.02

In Response:

We thank Dr. Atwood for his interest in our study. Assessment of awareness during anesthesia depends to a greater or lesser extent on self-reporting. Thus, it is impossible to independently confirm descriptions of pain and paralysis. It is, of course, possible that some of the awareness descriptions were related to extubation. Obvious patient reports of extubation were not included in the 25 patients in Table 4 in our original article. Full details of the individual patient reports are included so that the reader can evaluate the cases for him or herself.

We agree that our report is "worrisome." This is a complication that has often been ignored or disregarded by the profession. However, recently, the Joint Commission of Healthcare Organizations has published a sentinel alert on awareness during anesthesia (1) and the ASA has appointed a task force to look into the issue of monitoring and awareness. We applaud the president elect of the ASA, Dr. Guidry, who addressed the issue in the January 2005 ASA newsletter (2). It thus appears that the profession is taking this "worrisome" complication seriously.

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Ultrasound Evidence of Intraneural Injection

To the Editor:

Schafhalter-Zoppoth et al. (1) reported an interesting case of delayed sensory recovery purportedly as a result of inadvertent femoral nerve impalement and subtotal intraneural injection visualized by ultrasound. A short video clip they provided showing withdrawal movement of the block needle that has initially traversed the femoral nerve is presumably evidence of nerve impalement. A second video clip shows presumably a subtotal intraneural injection represented by a hypoechoic fluid collection underneath the hyperechoic nerve shadow. These ultrasound images and videos invite some comments.

The elliptical hyperechoic density shown in the article (Fig. 1A) is indeed a typical ultrasonographic image of the femoral nerve in cross-section. Generally, nerve fascicles are not readily seen in the femoral nerve location, and it is difficult to tell if the hyperechoic density represents a single nerve structure. One may visualize divisions of the femoral nerve at the time of local anesthetic injection when the hyperechoic nerve structure is split into two, highlighted by surrounding hypoechoic fluid collection within an expanding femoral sheath compartment. Without knowledge of prolonged anesthesia in this case, I would have considered the image in Figure 1B a picture of an extraneural injection within the sheath compartment.

The authors suggest that an intraneural injection diminishes nerve diameter as the result of compression and increased intraneural pressure (Fig. 1). To the contrary, I suggest a true intraneural injection will show on ultrasound an obvious expansion of the hyperechoic nerve structure as the nerve increases in size. Our preliminary study in a pig model shows that a purposeful direct intraneural injection into the brachial plexus with a 22-gauge long bevel needle and 10 mL of saline dramatically increases nerve dimension on gross examination and the ultrasonographic appearance of the hyperechoic nerve structure (Fig. 1). We speculate that ultrasound imaging may add safety to the practice of peripheral nerve block by showing early evidence of an inadvertent intraneural injection, but further imaging studies are warranted.

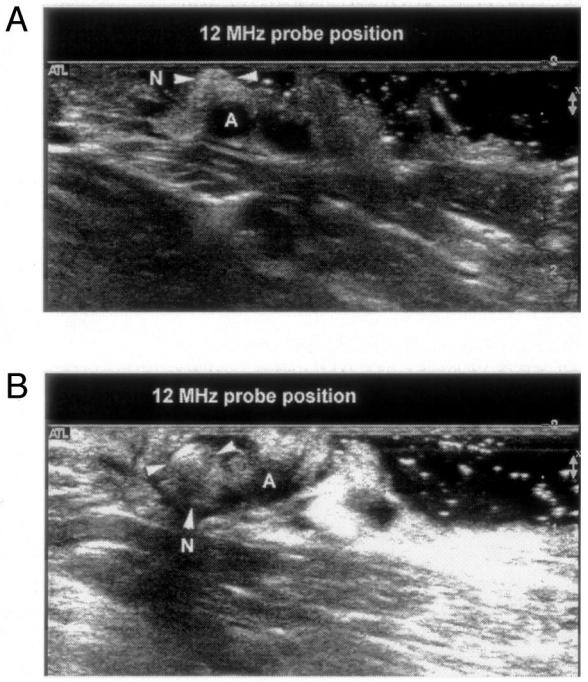

Figure 1. A, Transverse sonogram of the brachial plexus of a pig using a 12 MHz probe before direct intraneural saline injection. A = axillary artery, *n* = brachial plexus, arrows indicate size of nerve. B, Transverse sonogram of the brachial plexus after an intraneural 10-mL saline injection. A = axillary artery, *n* = brachial plexus, arrows indicate an increase in nerve size.

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DOI: 10.1213/01.ANE.0000159011.02691.23

In Response:

We thank Dr. Chan for his interest in our report and his insightful commentary. Although intraneural injection has been the focus of recent investigations (1), sonographic evidence has been limited. One case of ultrasound imaging obtained in follow-up after presumed intraneural injection has been published (2). Since acceptance of our manuscript a case series of ultrasound guided injections into neuromas has been described (3). In contrast, our letter reports injection into the normal fasciculated tissue of the femoral nerve (4).

We emphasize that most of the 35-mL injection is extraneural, with only approximately 0.1 mL being considered intraneural (assuming limited longitudinal distribution with equal spread in all directions within the nerve). We agree with Dr. Chan and other authors on the basic principle that intraneural injection acutely expands nerve structure (5–8). Our transverse sonograms show fasciculated femoral nerve architecture (9) on both sides of the block needle. Even without a surgical pathology specimen, our ultrasound scans and clinical course make a highly compelling case for intraneural injection. In clinical practice we have appreciated anatomic divisions of the femoral nerve only rarely during these procedures (approximately 2 in 73 cases of femoral nerve blocks with ultrasound guidance) and do not include our case as one such example.

Dr. Chan presents new experimental data demonstrating high-volume (10 mL) injections into porcine brachial plexus nerves. However, one potentially important issue is that the consequences of intraneural injection in a dividing nerve may be quite different than injection in a discrete nerve without local branching. These new data raise many exciting questions regarding characteristic internal signs of nerve injection injury that can be answered with real time high-resolution ultrasound imaging. More importantly, a major safety goal with imaging is to develop techniques that improve needle (10) and nerve visibility to reduce block-related complications.

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Existential Distress and Palliative Sedation

To the Editor:

I read with interest the article by Perry Fine (1). Dr. Fine justly addressed existential distress and palliative sedation (he prefers to label it as total sedation), given that existential suffering can be just as consequential and debilitating as physical suffering. However, he did not discuss a valuable subset of palliative sedation that can be used for existential distress, respite sedation (2,3). Respite sedation is a form of palliative sedation in which patients are deeply sedated for a predetermined amount of time (usually 24 to 48 h), and then reawakened to assess the extent of symptomatic improvement and the need for further sedation. Because many dying patients are afflicted with existential turmoil that engenders fear, fatigue, and insomnia, respite sedation may break a cycle of sleep deprivation and existential distress and allow such patients the opportunity to regain psychological strength and assuage the existential issues that precipitated the need for palliative sedation. Respite sedation also allows second-guessing and reassessment by health care providers, patients, and family members, negating the sense of overwhelming finality and guilt that may occur with continuous deep sedation.

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Dr. Fine does not wish to respond.