To the Editor:—We read with interest the case report by Albertin et al., who reported a bilateral subdural hematoma (SDH) in a parturient after epidural anesthesia. In a previous report, we reviewed 47 cases of cerebral hemorrhage after spinal (26 cases) and/or epidural (21 cases) anesthesia (table 1). Almost half of the patients developed bilateral subdural hematoma after dural puncture by an epidural needle. In contrast, subdural hematoma after spinal anesthesia occurred most frequently on the left side of the brain. It is possible that the increased incidence of bilateral subdural hematoma after epidural (11 of 21 vs 4 of 26) can be attributed to the greater loss of cerebrospinal fluid volume after inadvertent dural puncture with a large-bore epidural needle. Cerebrospinal fluid leak through a large dural hole might lead to marked cerebral hypotension associated with excessive traction on the bridging veins on both sides of the brain, resulting in bilateral subdural hematoma formation.

References

In Reply:—In our case report, we state that the magnetic resonance imaging scanning was performed without contrast because the patient was breast-feeding. As Camann and Stuebe wrote, current recommendations suggest that the use of radiographic contrast media is compatible with breast-feeding.

However, the informative sheet of all three contrast brands in use at our hospital state that breast-feeding is a relative contraindication to contrast medium use. One contrast medium producer recommends a 24-h suspension of breast-feeding for lactating women in case of contrast medium administration. Kubik-Huch et al. have demonstrated that small amounts of radiographic contrast medium are excreted into human breast milk during lactation, but the amount transferred to a nursing infant orally is so small that the authors state that the recommendation of a 24-h suspension of breast-feeding for lactating women should be reconsidered.

The clinical relevance of contrast medium excretion into human breast milk is not known. But because magnetic resonance imaging scanning without contrast gave us a satisfactory diagnostic certainty, we decided not to expose the patient to an avoidable risk.

Andrea Albertin, M.D.,* Chiara Marchetti, M.D., Daniela Mamo, M.D., Davide Poli, M.D., Elsa Dedola, M.D. *Vita-Salute University of Milan, IRCCS San Raffaele, Milan, Italy. albertin.andrea@hsr.it

References

To the Editor:—We read with interest the case report by Mashour et al., who reported a bilateral subdural hematoma (SDH) in a parturient after epidural anesthesia. In a previous report, we reviewed 47 cases of cerebral hemorrhage after spinal (26 cases) and/or epidural (21 cases) anesthesia (table 1). Almost half of the patients developed bilateral subdural hematoma after dural puncture by an epidural needle. In contrast, subdural hematoma after spinal anesthesia occurred most frequently on the left side of the brain. It is possible that the increased incidence of bilateral subdural hematoma after epidural (11 of 21 vs 4 of 26) can be attributed to the greater loss of cerebrospinal fluid volume after inadvertent dural puncture with a large-bore epidural needle. Cerebrospinal fluid leak through a large dural hole might lead to marked cerebral hypotension associated with excessive traction on the bridging veins on both sides of the brain, resulting in bilateral subdural hematoma formation.

Table 1. Cerebral Subdural Hematoma after Spinal versus Epidural Anesthesia

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<th></th>
<th>Spinal Group</th>
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<tr>
<td>Number of patients</td>
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<tr>
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<td>11</td>
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<td>Right subdural hematoma</td>
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<td>Intracerebral hematoma</td>
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<td>Pregnant patients</td>
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<td>Blood patch</td>
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Further analysis of the 47 cases of SDH showed that half of the published cases were in obstetric patients (22 of 47 cases); 6 cases were reported after spinal anesthesia, and 17 cases followed epidural

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Further analysis of the 47 cases of SDH showed that half of the published cases were in obstetric patients (22 of 47 cases); 6 cases were reported after spinal anesthesia, and 17 cases followed epidural
anesthesia. It is possible that pregnant patients are more liable to develop post-dural puncture subdural hematoma. However, this observation might be attributed, in part, to the frequent use of epidural analgesia during labor.

We agree with Mashour et al.\(^1\) that SDH has been found to develop after inadvertent dural puncture even when an epidural blood patch was placed. Analysis of the published cases of SDH (table 1) showed that epidural blood patch was used in 8 of 21 cases to treat post-dural puncture headache, but this treatment did not prevent the formation of SDH. However, SDH was not ruled out before applying the blood patch.

References

1. Mashour GA, Schwamm LH, Leffert L: Intracranial subdural hematomas and cerebral herniation after labor epidural with no evidence of dural puncture. \textit{Anesthesiology} 2006; 104:610–2


(Accepted for publication August 17, 2006.)

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To Sleep, Perchance to Decode?

To the Editor—Plourde et al.\(^3\) recently reported the effects of propofol on the brain’s response to different forms of auditory stimuli during sedation and anesthesia. This holistic approach is the only rational option to understand such phenomena as learning during anesthesia and perioperative dreaming.

One of the most interesting findings from this study is the extreme diminution of cortical response to auditory stimuli during the first episode of “sedation” before anesthesia. The cortical response to auditory stimuli during the period of “sedation” before anesthesia was much less than at baseline or at an equivalent blood level of sedation during recovery. The authors offer the explanation of acute tolerance to propofol in the interpretation of these results, which I would suggest is unnecessary.

We too have noted an increase in brain activation during propofol sedation, most evident in the statistical maps used to define brain activity in neuroimaging studies. During propofol or thiopental sedation, a very visible increase in statistical activation to auditory stimulation is present, although in actuality a small decrease in regional cerebral blood flow occurs.\(^2\) Plourde \textit{et al.} also document a similar discrepancy between blood oxygenation level-dependent signal magnitudes, which remain similar in many regions to baseline, and the increase in statistical significance, from roughly \(t = 14\) at baseline to 19 in recovery, clearly visible in their brain map figures. There is no doubt that the subjects in the study of Plourde \textit{et al.} were awake and sedated during recovery, because they remembered some 60% of the stimuli presented to them. Therefore, the phenomenon of increased statistical significance seems to be a marker of the effect of a sedative drug on auditory stimulation in an awake cortex. The new, paradoxical finding in the study of Plourde \textit{et al.}, however, is that this situation is absent in the initial “sedation” condition, where both blood oxygenation level-dependent signal and significance are similar to the anesthesia condition. The only behavioral measure prospectively collected in this study is memory performance, and it too is noticeably absent in the initial “sedation” condition, being the same as during anesthesia. However, at recovery, the memory performance is at the expected 60% of control. As noted by Plourde \textit{et al.}, these sedative concentrations of propofol should produce performance of approximately 50% of baseline/control, as we have documented in our studies.\(^5\)

The most parsimonious explanation of the paradoxical findings during the initial “sedation” condition is that the subjects were indeed asleep, whereas they were awake and truly sedated in the recovery condition. The contention that sedation was equivalent at the begin-

References

1. Zeidan A, Chaaban M, Farhat O, Baraka A: Cerebral rebleeding by spinal anesthesia in a patient with undiagnosed chronic subdural hematoma. \textit{Anesthesiology} 2006; 104:613–4


(Accepted for publication August 17, 2006.)

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ning and at recovery is based solely on measured concentrations of propofol, not on behavioral state. Clear interpretation of the imaging findings is difficult because volunteers were performing no active behavioral task (e.g., button press to hearing a word) to measure their degree of sedation and responsiveness while inside the scanner. Although the authors state that “it is difficult to fall asleep in the cramped and noisy scanner environment,” our experience has been that, even without drug, it is actually easy to fall asleep in a 3-T magnetic resonance scanner in the absence of an active task. Other investigators at our institution have had similar experiences; being confined in a constant position with the arms tucked in under warm blankets, particularly if the eyes are closed, is a situation similar to swaddling a baby to induce sleep. The “close monitoring” of the subjects is not defined, and an “impression” of whether the subject is awake or asleep is difficult to assess while in the scanner, particularly in retrospect. Any subjects sleeping after initial sedation would wake up when moved out of the scanner in preparation for induction of anesthesia.

On another point, some caution must be exercised regarding which auditory processes are affected most by propofol. The authors state that higher level processing for speech and voice is abolished during anesthesia, whereas the response to nonspecific auditory stimuli is not. However, the cortical response to nonspecific auditory stimuli was determined using all measured data versus a rest condition, whereas response to speech and voice were measured using only a subset of the data collected, with the comparison being between two active conditions. The effect of this change in data handling is evident in baseline. Whereas maximal t values for the nonspecific auditory stimulation are on the order of 14, those of the speech- and voice-related activation are only approximately 3.5 (the significance threshold is 3.2). Consequently, blood oxygenation level-dependent signal magnitudes at baseline for nonspecific auditory stimuli are approximately 30, whereas for speech and voice they are approximately 5. Therefore, the presence of decreased but still present brain activity in response to voice or speech during anesthesia would not likely be detected in this study.

Plourde et al. have presented a more refined evaluation of auditory processing during sedation and anesthesia than has heretofore been published. The interesting questions raised by these findings clearly elucidate paths for further study. However, the conclusions drawn from the current study are limited by lack of behavioral data and by issues of statistical significance. I would suggest that the evidence for acute tolerance to propofol is confounded by sleeping subjects. The issue of inhibition of higher order but not general auditory processes by propofol is still an open one. It seems likely that decoding of auditory stimuli during anesthesia is still functioning at some level, because numerous studies have demonstrated at least an implicit form of memory for these stimuli, albeit in much larger sample sizes (e.g., Deeprose et al.1).

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References


(Accepted for publication August 17, 2006)

Anesthesiology 2006; 105:1279–80

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In Reply:—We thank Dr. Veselis for his insightful comments on our article.1 His first point is that the lack of specific auditory activation during the light sedation stage was most probably caused by physiologic sleep. The interactions we had with the subjects during the experiment and the informal debriefing between runs make us think this is unlikely: Subjects after the light sedation run seemed more "chatty" than sleepy. In addition, as we point out in the article, normal sleep does not abolish responses to complex sounds. Hence, even if the subjects had fallen asleep, this would not per se explain the lack of auditory responses. Despite these arguments and as indicated in our article, we agree that we have no way to unambiguously demonstrate that subjects were not sleeping during the light sedation recordings. In future experiments, we will have to monitor subjects’ level of arousal more closely, although even asking subjects to perform an active task might not be sufficient. Lack of response would not necessarily indicate sleep; it could also indicate attentional, motivational, motor, or memory alterations due to the anesthesia, for example. The most objective means is probably monitoring of an ongoing electroencephalograph, a complicated task in a magnetic resonance imaging suite.

Irrespective of the sleep issue, we still believe the evidence of increased neural excitability (or disinhibition) after anesthesia with propofol because the level of activation during recovery (propofol plasma concentration: 0.64 μg/ml) significantly exceeded baseline levels. The critical question is, Does the development of this hyperexcitability require previous exposure to a higher concentration of propofol? Veselis believes that the answer is no, based on a study2 that revealed a visible increase in statistical activation to auditory stimulation during sedation (not preceded by exposure to higher concentration) with propofol or thiopental. The statistical significance of that effect was not reported. We proposed acute tolerance as the most likely explanation for the increased excitability seen during recovery in our study because we have observed an analogous phenomenon with the 40-Hz auditory steady state response: The amplitude during recovery from propofol anesthesia significantly exceeded baseline amplitude.3 This phenomenon was not observed during propofol sedation not preceded by exposure to a higher concentration4 or during prolonged recordings in nonanesthetized subjects (unpublished observations, Gilles Plourde, M.D., M.Sc., Professor, Department of Anesthesia, Montreal Neurologic Hospital, Montreal, Quebec, Canada, 2002). We believe that this topic deserves further inquiry.

Dr. Veselis’ second point is that we were not able to demonstrate higher level processing during anesthesia because of insufficient power. We did discuss in the article the factors that could have led to a relative loss of signal during the anesthesia and hence affected our ability to pick up a signal. However, we disagree that the lack of response during anesthesia for higher level processing is explained on the basis of a lack of power:

First, we did have sufficient power to detect speech- and voice-related responses during baseline (and recovery). During anesthesia, we showed, using the same functional magnetic resonance imaging sampling protocol, that these responses were absent with mean signal amplitudes very close to zero and one-sample t tests yielding P > 0.2. Naturally, neither we nor anyone else can prove the null hypothesis of no activation, regardless of how large a sample is studied or how many data points are acquired. However, as we also point out in the article,
our conclusion is consistent with electrophysiologic data from higher order visual cortex in monkeys.

Second, and perhaps more important, we successfully demonstrated significant negative activation during anesthesia in the words-versus-scrambled words contrast. This suggests that the protocol had adequate sensitivity, because with the same number of trials, we picked up an atypical response difference between the two stimulus classes. If the lack of normal positive response to words or voice stimuli were simply due to low signal and/or high noise, we would not have detected the unusual negative response either.

Third, the reduced level of significance for the words versus scrambled words and vocal versus nonvocal stimuli was expected because we were comparing activations produced by different classes of stimuli (instead of a stimulus vs. silent baseline). This is therefore not an indication of inadequate signal acquisition.

We look forward to additional research on this topic from Dr. Veselis’ group and others, because much clearly remains to be learned from the study of neural responses during anesthesia.

In Reply.—Reutershan et al.1 reported on the finding of reduced endotoxin-induced acute lung injury in mice pretreated with isoflurane. In their investigation of the underlying cause of this phenomenon, the authors focused on neutrophil recruitment and chemokine concentrations. They found a reduction of neutrophil recruitment and CXCL1 and CXCL2/5 chemokine concentrations in the lungs of isoflurane-treated mice. Previous studies have not established a causal link between chemokine levels or the associated neutrophil accumulation and endotoxin-induced lung injury. There is, however, evidence that the cytokines tumor necrosis factor and interleukin 1 are involved in a reduction of alveolar ion and the associated fluid transport and hence pulmonary edema clearance.2,5 Particularly tumor necrosis factor has been strongly implicated in the pathogenesis of pulmonary edema, and its effect is mediated by nitric oxide.2,4 Tumor necrosis factor–induced lung injury has been strongly implicated in the pathogenesis of pulmonary edema, and tumor necrosis factor has been shown to reduce plasma interleukin 1–dependent formation of pulmonary edema might be an inhibition of nuclear transcription factor KB.6 The reduction in chemokine gene network. Recent Prog Horm Res 2003; 58:95–130

References


(Received for publication August 17, 2006.)

Role of a Reduction of Cytokine Levels in Isoflurane-mediated Protection from Endotoxin-induced Lung Injury

To the Editor.—Reutershan et al.1 reported on the finding of reduced endotoxin-induced acute lung injury in mice pretreated with isoflurane. In their investigation of the underlying cause of this phenomenon, the authors focused on neutrophil recruitment and chemokine concentrations. They found a reduction of neutrophil recruitment and CXCL1 and CXCL2/5 chemokine concentrations in the lungs of isoflurane-treated mice. Previous studies have not established a causal link between chemokine levels or the associated neutrophil accumulation and endotoxin-induced lung injury. There is, however, evidence that the cytokines tumor necrosis factor and interleukin 1 are involved in a reduction of alveolar ion and the associated fluid transport and hence pulmonary edema clearance.2,5 Particularly tumor necrosis factor has been strongly implicated in the pathogenesis of pulmonary edema, and its effect is mediated by nitric oxide.2,4 Tumor necrosis factor–induced lung injury has been strongly implicated in the pathogenesis of pulmonary edema, and tumor necrosis factor has been shown to reduce plasma interleukin 1–dependent formation of pulmonary edema might be an inhibition of nuclear transcription factor KB.6 The reduction in chemokine gene network. Recent Prog Horm Res 2003; 58:95–130

References


(Received for publication August 17, 2006.)

In our study,1 we investigated the role of isoflurane on recruitment of neutrophils (polymorphonuclear leucocytes [PMNs]) in a model of lipopolysaccharide-induced lung injury. We found that pretreatment with isoflurane reduced PMN recruitment and protected from lung damage when administered within certain time windows. We revealed that chemotaxatants CXCL1 and CXCL2/5 in the bronchoalveolar lavage fluid were reduced when mice inhaled isoflurane 1 h before lipopolysaccharide exposure and suggested that isoflurane might affect the release of these chemokines.

In his letter, Dr. Eisenhut suggests that tumor necrosis factor α- and interleukin 1–dependent formation of pulmonary edema might be primarily responsible for lipopolysaccharide-induced lung damage and that nuclear transcription factor KB might be the main target for the antinflammatory effects of isoflurane observed in our study. Although increased vascular permeability and PMN recruitment are two key factors in lung injury, there is evidence that regulation of both is different and one might occur without the other.2 For example, vascular protein leakage is dependent on inducible nitric oxide syn-
these from alveolar macrophages; however, PMN recruitment is not. In our model, lipopolysaccharide inhalation results in a rapid PMN accumulation in the vascular bed within 1 h, whereas vascular protein leakage is not seen until 6 h after lipopolysaccharide exposure. Lung injury in our model is completely dependent on PMN recruitment as demonstrated in CXCR2-deficient mice, suggesting that vascular leakage might be a secondary event.

Chemokactants are essential in acute lung injury. Functional blocking of chemokines or their receptors curbs lung damage. In our model, CXCL1 and CXCL2/3 account for approximately 70% of the chemotactic activity in lipopolysaccharide-exposed bronchoalveolar fluid. Tumor necrosis factor α and interleukin 1 may also contribute, although tumor necrosis factor α- and interleukin 1-independent production of CXCL1 and CXCL2/3 has been reported. Although type II cells are involved, alveolar macrophages seem to be the major source of chemokines in the lung as shown by depletion studies. Our data do not allow distinguishing between effects of isoflurane on either cell type. The concept of cytokine-dependent fluid transport across alveolar epithelial cells is interesting and may well promote lung damage. This pathway could involve nuclear transcription factor-KB activation and might be particularly relevant for the delayed, CXCL1- and CXCL2/3-independent protection observed in our study.

We agree that future studies should further characterize the mechanisms underlying the antiinflammatory effects of isoflurane in lung injury.

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References


(Accepted for publication August 16, 2006.
We agree with the author that the type of HRV analysis may crucially influence the results. Latson et al. investigated absolute values, whereas we analyzed the different frequency bands of the power spectrum as normalized units.

Currently, HRV-derived data are processed and presented in different ways. First, absolute HRV values are frequently analyzed and, as the author stated, may have advantages in the analysis of autonomic reflex integrity. Nevertheless, total power of HRV and its different parts decrease with increasing age, underlying cardiovascular disease, and impaired autonomic regulation. Thus, an absolute HF of, for example, 1,000 ms^2/Hz may be considered as below normal in young and healthy patients, normal in older healthy patients, and even above normal range in patients with coronary artery disease. Therefore, absolute values do not reflect the specific impact of the parasympathetic or the sympathetic activity of the autonomic regulation. Second, HRV data are normalized to total power. If the total power is high (e.g., 10,000 ms^2/Hz), an HF of 1,000 ms^2/Hz is only 10%, reflecting a low parasympathetic activity in the individual. If the total power is only 2,000 ms^2/Hz, the same absolute HF value would reflect a considerably higher parasympathetic activity. The aim of our studies was to identify a general predictor of postspinal hypotension; therefore, we investigated the different frequencies in normalized units and cannot comment on absolute HF values in our patients. Absolute values were analyzed as well, but demonstrated only a trend toward higher HF in normotensive patients compared with hypotensive patients. Therefore, we cannot recommend the analysis of absolute values for prediction of postspinal hypotension based on our data.

The guidelines of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology do not favor any one of the mathematical techniques, and each may have advantages and disadvantages.

However, the ratio of LF to HF was demonstrated to be the most valuable parameter of HRV in terms of prediction of postspinal hypotension. This parameter reflects the balance of the autonomic nervous system and is independent of relative or absolute values because the ratio of absolute and normalized values is mathematically identical. Risk stratification based on the analysis of this autonomic balance was demonstrated to successfully guide prophylactic therapy.

We conclude that in a homogenous group of patients, analysis of absolute values of HRV parameters is probably the best method to determine reflex integrity or “gain.” If the trial’s patient population demonstrates a large variation with respect to age, sex, and underlying diseases, relative values may be more appropriate. LF to HF ratio may be particularly useful for HRV analysis because it is independent of this difference.

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References

(Accepted for publication August 18, 2006.)

To the Editor.—I read with great interest the article by Hanss et al. in which the authors elegantly demonstrate a relation among heart rate variability, the risk of hypotension after spinal anesthesia for cesarean delivery, and the effectiveness of prophylactic measures such as prehydration and vasopressor use in terms of heart rate variability as well as clinical outcome.

However, I am puzzled by the fact that there was no control for the time of day when the experiments were performed. The authors did their baseline assessments between 18:00 and 20:00, which might be totally different from the values obtained during the experiments. Chassard and Bruguerolle recently reviewed the effects of time of day as a synchronizer for different processes and bodily functions such as drug metabolism, cortisol levels, and hepatic blood flow. Therefore, it should not be surprising to see different results depending on the time of day when the experiments were performed. A clinical observation is that early morning elective cesarean deliveries tend to have a higher incidence of hypotension than afternoon deliveries. This may represent a multifactorial problem, however, after the results of Hanss et al. Heart rate variability might be a good representation of one of the main factors influencing postspinal hypotension: sympathetic relative overactivity, which might be synchronized with the time of day.
In Reply:—We thank Dr. Lacassie for and appreciate his valuable comment on our study.¹ We agree with his statement that if prophylactic treatment of postspinal hypotension is based on measurement of heart rate variability (HRV), timing of the analysis is crucial. Measurement of HRV is performed to analyze autonomic nervous system regulation. It was clearly demonstrated by Chassard and Bruguerolle² as well as Takase et al.³ that autonomic nervous system activity demonstrates circadian rhythm. Therefore, timing of the measurement is important and should be comparable in all patients. In our previous studies, baseline analysis of HRV on the day of surgery before volume preload and anesthesia was demonstrated to identify patients at risk of hypotension in the course of subarachnoid block,⁴,⁵ whereas the analysis on the day before surgery did not correlate with postspinal hypotension.

Events of the current study were defined based on these findings. Baseline analysis identifying patients at risk was the analysis on the day of surgery before volume preload and anesthesia. Because cesarean deliveries were performed at 8:00 AM, all analyses were performed at the same time. The measurement on the day before surgery was performed according to our previous studies and confirmed that there was no correlation between HRV parameters at that event and postspinal hypotension. We conclude that HRV changes during a 24-h period. Therefore, under study conditions, analyses should be performed at the same time to secure comparability of results, as was done in our study.

References

2. Chassard D, Bruguerolle B. Chronobiology and anesthesia. Anesthesiology 2004; 100:413–27

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To the Editor:—The ProSeal™ laryngeal mask airway (PSLMA; Laryngeal Mask Company Limited, Mahé, Seychelles) is not designed as an intubation device. However, we may need to intubate the trachea with a PSLMA in place, using it as an intubation conduit with fiberoptic guidance. This situation may occur in an unanticipated difficult intubation when a PSLMA is used to ventilate the patient. The PSLMA might then be preferred to the LMA-Classic™ when a potentially full stomach is a concern.¹-³

On the LMA North America Web site,* which includes PSLMA specifications, it is mentioned that it is possible to introduce a 4.5-mm-ID uncuffed endotracheal tube into a size 1.5 or 2 PSLMA. We tested this information with a 4.5 uncuffed Mallinckrodt (MMJ S.A. de C.V., a Mallinckrodt Company, Juarez, Mexico) tube, and we realized that this tube does not fit into a size 1.5 PSLMA. Moreover, a size 2 PSLMA is also too small to accept easily a 4.5 uncuffed tube. However, it is possible to introduce a 4.0 uncuffed tube into a 1.5 or 2 PSLMA.

Support was provided solely from institutional and/or departmental sources. After numerous failed attempts to acquire a manufacturer reply to this letter, it is being published without a response. —Michael M. Todd, M.D., Editor-in-Chief

We believe that the problem may be related to variations in the outer diameter of endotracheal tubes from one company to another, inner diameter being the same.

In our opinion, this misinformation on the LMA North America Web site should be corrected because many anesthesiologists transcribe it on their difficult airway chart for emergency airway management.

References


(Received for publication June 28, 2006.)