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Memory by Subarachnoid Regional Anesthesia

To the Editor:—In their statistically elegant study of lower limb phantom posture during induction of spinal anesthesia, Dr. Isaacson *et al.*[1] have rejected the concept that phantoms adopt an archetypal position of orthopedic rest. Instead, they have reverted to a plastic model determined by limb posture during the onset of anesthetic blockade.

Unfortunately, in their study, the authors chose an experimental protocol of proprioceptive acuity that was unlikely to guarantee reliable phantom development within the restricted 10-min period of their observations. In our earlier study of 169 clinically successful limb blocks, we reported that phantoms only *began* to occur approximately 10 min after injection. A much longer period usually was required before subjective phantoms became sufficiently intense and stable to be reportable in three-dimensional coordinates for all joint postures of the shoulder, elbow, wrist, and hand. The following incidences or "yield" of measurable subjective phantom postures were recorded after a significantly longer maturation period of 30–45 min from the time of completing the regional anesthetic procedure[2]:

upper limb interscalene block (n = 110): 86% phantom yield
lower limb epidural (n = 50): 10% phantom yield
lower limb subarachnoid (n = 9): 55% phantom yield

Dr. Isaacson *et al.*[1] did not comment on their yield of spontaneous lower limb phantoms, if any occurred, nor on the disappointingly low yield of measurable lower limb phantoms that we reported, even after a 30- to 45-min observation period. Nevertheless, comparison of our two studies may lead to a compatible resolution. In the meantime, Isaacson *et al.*[1] have produced a scholarly mathematical presentation of changing proprioception within the limits of their short 10-min observation period. Unfortunately, those limits are poorly suited to the appreciably longer maturation period required by anesthetically deafferented phantoms before they are perceived consciously, with all phantom joints at mid point of joint range. Therefore, on the basis of our experience, we maintain that these investigators did not allow sufficient time for full maturation of a *subjective* phantom posture—a neuroanatomic–anesthetic process that remains incompletely understood, even after an interval of 25 yr after the publication of our investigation. This is an intriguing and largely speculative phenomenon worthy of further study, particularly from the point of view of sports medicine, but under more leisurely, prolonged, and stable experimental conditions than were afforded in the protocol of Isaacson *et al.*[1]

Philip R. Bromage, M.B.B.S., F.F.A.R.C.S., F.R.C.P.(C.),* **Ronald Melzack, Ph.D.** *Department of Anesthesia, McGill University, Montreal, Canada (retired)

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Phantom Limb Sensation: A Need for More Elaborated Studies

To the Editor:—I read with interest the article by Isaacson *et al.*[1] that examined phantom limb sensations during subarachnoid block. Abnormal phantom sensation has been described previously, not only during spinal[2] and epidural anesthesia[3,4] but also during brachial plexus blocks[3,4] and intravenous regional anesthesia.[4] I would like to comment on a few issues.

First, the period of clinical observation after subarachnoid injection seems to be too short to permit a full form of phantom sensations. With both epidural anesthesia and peripheral nerve blocks, phantom sensations are reported 20–30 min after the onset of anesthesia. Although the onset time of subarachnoid block is more rapid than that of these other forms of anesthesia, more information might have been gained with a longer period of observation. Second, the authors conclude that proprioceptive memory involves a dynamic neuroplastic imprinting process that is influenced by limb position before the onset of regional anesthesia, rather than the classic "fixed body" schema.[2,5] This is consistent with a previous study with spinal anesthesia in which Moriyama *et al.*[5] noted that the incidence of false answers was related to the perceived position of the lower limb before the block and was not influenced by subsequent general anesthesia. These authors argued that when input from the limb was blocked, the "short-term" memory became a more persistent "long-term" memory.

There is perhaps no absolute contradiction between the classic "body schema"[2] and the "additive neuroplastic process."[1] The reappearance of phantom limb pain after administration of a regional anesthetic supports some role for transient deafferentation produced by the block. The "flexed" position in the "body schema" concept does not represent a position of rest but is the memory of an archaic "tetrapod" schema disinhibited by anesthesia. Regardless, understanding this process remains an exciting challenge.

Marc E. Gentili, M.D., Centre Médico-chirurgical Saint-Vincent, Saint-Grégoire, France. Marc.Gentili@wanadoo.fr or mgentili@cpa-sante.com

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In Reply:—We thank Drs. Bromage, Melzack, and Gentili for their interest in our work. Both letters express concern about the relatively short duration of observation in our study.

Our study differs from that of Gentili *et al.*[1] and Bromage and Melzack[2] in two respects. (1) We exclusively studied lower extremity phantom proprioception after subarachnoid block. The difference in local anesthetic onset between subarachnoid block in the 3- to 8-min range[3] and major peripheral and epidural blocks in the 10- to 30-min range,[4] depending on local anesthetic used, or the potential for variable or incomplete anesthetic blockade with these nonsubarachnoid blocks may account for the delayed phantom phenomena emergence that these authors observed in their studies. Our observations clearly show that a phantom sensation can develop within 10 min, especially in the originally flexed limb. (2) Our subjects were forced to limit their limb position perception to either bent or straight. Limiting the subject response to a two-category variable of bent or straight may have resulted in a greater and possibly earlier yield of phantom perceptions in our study. These yields are clearly documented for each time point. In addition, it should be pointed out that the study of Drs. Bromage and Melzack and the study of Dr. Gentili involved phantom observations of affected limbs that necessitated surgery. Our study evaluated normal, healthy limbs that were not being operated on. The confounding influence of pathophysiologic pain or injury on the incidence of phantom sensations has yet to be determined and may result in different phantom yields.

We were well-aware of the work by Moriyama *et al.* and Gentili *et al.*; however, they were not cited because full manuscripts have not been published to date. In developing our study design, our protocol attempted to balance time of onset of sensory and proprioceptive block with surgical time delay concerns. In addition, a surgical procedure scheduled for 45–60 min would not be able to tolerate plain lidocaine spinal anesthesia with a research delay longer than 12–15 min. However, neuroplastic phenomena do show change with time, as clearly demonstrated by numerous experimental works on hippocampal long-term potentiation.[5] Proprioceptive phantom sensation as a manifestation of spinal cord neuroplasticity may exhibit time-dependent changes as well.

We do not claim a formal opposition between the existence of any classic body schema and the neuroplastic processes that we documented. Our observations suggest that a neuroplastic processes seems to override or dominate any existing default "position of rest" or "tetrapod" settings for the period of our observations. In this respect, a longer observation time using our protocol may yield more information either to confirm our initial observations or to reflect subsequent migration to any default settings observed at later times from the noted authors' studies.

Sheldon A. Isaacson, M.D., University of Rochester Medical Center, Rochester, New York. sheldon_isaacson@urmc.rochester.edu

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Acromegaly, the Mallampati, and Difficult Intubation

To the Editor:—I would like to congratulate Hubert Schmitt *et al.*[1] on their study of difficult intubation in acromegalic patients. Their experience is unrivaled, and it is reassuring to know that serious difficulty with the airway is not common.

I would like to make two pleas to future investigators. The first is for the abandonment of the four-grade Mallampati classification. A fourth grade was added by Samsoon and Young[2] in their retrospective study.

I have been able to discuss the reason for this change with Dr. Young, the senior author. He confirmed my suspicion that they made an entirely reasonable assumption that the four grades of glottic visibility described by Cormack and Lehane[3] would be predicted by four grades of oropharyngeal visibility. We know now that this is not the case and should cease pretending that we can describe oropharyngeal appearances with such precision.

My second plea is for investigators to report likelihood ratios because they provide an understandable estimate of risk when one is confronted by a patient with a test result.[4] The likelihood ratio is calculated by dividing the sensitivity by 1-specificity, and the result is the number of times more likely it is that a positive result will be seen in someone with the condition being sought than in one without. The likelihood ratio for a positive Mallampati test in the series of Schmitt *et al.*[1] thus would be 44/24 = 1.8. I find that this gives me a better understanding of the poor performance of the Mallampati. With the use of nomograms,[4] a knowledge of the likelihood ratio also allows estimation of the positive predictive value in populations with a different risk because of a higher or lower incidence of difficulty. In this case, I estimate that had Schmitt *et al.*[1] chosen to define difficult laryngoscopy as grade 3 after the application of external laryngeal pressure and use of an appropriate laryngoscope blade (three patients), then the positive predictive value of the Mallampati would be 3%.

The only report in which the Mallampati has performed well was of a series of patients with cervical spine disease (likelihood ratio = 14).[5] The failure of the Mallampati in the study of Schmitt *et al.*[1] tends to support our contention that the Mallampati owes much of its predictive power to the effect of cranio-cervical rigidity on mouth opening ability, malalignment of oropharyngeal axes, and oropharyngeal visibility. The Mallampati can be regarded as essentially a test of craniocervical extension.

Ian Calder, F.R.C.A., National Hospital for Neurology and Neurosurgery, London, United Kingdom. icalder@aol.com

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In Reply:—We appreciate Dr. Calder's interest and his comments and are grateful for the opportunity to reply. First, regarding original Mallampati scoring, we agree with Dr. Calder that there are some problems in assessing reliably and reproducibly pharyngeal structures. There is indeed a considerable interobserver variability in performing the modified Mallampati test.[1] We used the modified test (performed by one investigator) because this enabled us to distinguish patients as class IV who had a very large tongue, concealing most of the oral cavum, a typical acromegalic feature. Moreover, this class has been shown to have the best reproducibility.[2]

Second, regarding likelihood ratio and predictive power of the Mallampati test, first, we have to apologize for an error in the Results. The sensitivity of the Mallampati test was 76%, and the specificity was 44% (page 112, line 36; the terms are interchanged).[3] We do agree with Dr. Calder that the likelihood ratio for a positive test result is easily to interpret and therefore should be cited more frequently. Because of its limited use in the literature, we did not refer to it. The likelihood ratio for a positive Mallampati test in our study was 1.4. This is a poor value and confirms other publications reporting a poor predictive value of the Mallampati test as the only preoperative screening test. However, the Mallampati test is recommended as one of several tests (thyromental distance, head and neck mobility, jaw movement) that are useful for preoperative airway evaluation.[4,5] In contrast to this poor overall predictive value, Mallampati class IV yielded a specificity of 0.97 and a likelihood ratio of 9.6 (95% CI; 3.0–30.8) in predicting a difficult laryngoscopy in our study. This is important because a special patient with this sign has a higher risk of a difficult airway compared with one without, and we think this is helpful information to the anesthetist. Of course, this high specificity is at the expense of the sensitivity, but there are, as Dr. Calder mentioned, many factors influencing Mallampati sign and performance of intubation. We agree that head and neck mobility may influence the visibility of pharyngeal structures,[6] but from our data, an association between head and neck mobility and Mallampati class cannot be deduced. We are convinced that soft tissue, anatomical shape of the mandible, and the function of the temporomandibular joint are important, too. There are special tests that assess these factors better than the Mallampati test does.[4]

Hubert Schmitt, M.D.,* Martin Radespiel-Tröger, M.D., Harald Mang, M.D. *Friedrich-Alexander University, Erlangen, Germany. hubert.schmitt@kfa.imed.uni-erlangen.de

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CSE in Labor and Hypoglycemia

To the Editor:—We read with interest the letter by Crites and Ramanathan[1] in which they described a case of acute hypoglycemia after combined spinal–epidural analgesia in a parturient with diet-controlled diabetes mellitus.

We were surprised that the authors considered the test dose to be negative. An increase in maternal heart rate of greater than 10 beats/min (in this case, from 110 to 138 beats/min) during a 2-min period after an epinephrine-containing test dose is exactly the criteria Pietro *et al.*[2] used to define a positive result. The signs and symptoms experienced by the patient may have been caused by the inadvertent intravenous injection of lidocaine.

We agree that the onset of effective regional analgesia often is associated with decreases in heart rate and blood pressure reflecting a decrease in maternal catecholamine concentration.[3,4] The authors speculate that the sudden reductions in catecholamine and cortisol concentrations was what produced the hypoglycemia. However, they noted no change in the patient's pulse or blood pressure after combined spinal–epidural analgesia. In our unit, we have used combined spinal–epidural analgesia in more than 11,000 patients and have not yet seen a similar complication.

Finally, we suggest that it is inappropriate to use 5% dextrose to treat severe, symptomatic hypoglycemia. Dextrose at 10% can be infused peripherally, or a higher concentration can be infused centrally.[5]

Sonia R. Verma, M.B.B.S., M.D., F.R.C.A.,* Felicity Plaat, B.A., M.B.B.S., F.R.C.A. *Queen Charlotte's Hospital, London, United Kingdom. soniave@hotmail.com

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In Reply:—We appreciate the comments by Drs. Verma and Platt about our letter to the editor[1] and would like to respond to each comment separately.

First, we did not consider the test dose to be negative. The initial increase in heart rate from 100 to 138 beats/min caused concern, and we removed the epidural catheter and replaced it with a new catheter with no difficulty. Because we suspected the patient's initial symptoms, such as tachycardia and dizziness, to be positive responses to the test dose, we perhaps missed the rapidly developing hypoglycemia and did not check her blood sugar concentration at the beginning of this event.

Second, we disagree with the assessment of Drs. Verma and Platt that all symptoms and signs shown by this patient, such as severe hypotension and acute hypoglycemia, can be attributed to an inadvertent intravenous injection of a test dose of 3 ml lidocaine.

Third, to state that we noted no change in the patient's pulse or blood pressure after combined spinal–epidural analgesia is not true. We reported profound hypotension and described in detail how we treated her symptoms.

Fourth, we administered 5% dextrose infusion because this solution was readily available in our cart. Maternal blood sugar concentration increased safely and without any delay, with complete alleviation of all symptoms.

Finally, acute hypoglycemia after regional anesthesia in parturients may not be such a rare occurrence. I wish to draw the attention of Drs. Verma and Platt to a recent case report[2] in which the authors described the occurrence of profound hypoglycemia in a healthy parturient to whom epidural anesthesia was administered for labor.

Jaya Ramanathan, M.D., University of Tennessee, Memphis, Tennessee. jkr50@aol.com

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Intubating Laryngeal Mask Airway and Muscle Relaxants: Never Together?

To the Editor:—We read the article of van Vlymen *et al.*[1] about the use of neuromuscular blocking agents and the intubating laryngeal mask airway (ILMA) with considerable interest. We feel that this article highlights the failure of the ILMA to find its true role in airway management.

There is no advantage afforded by the use of an ILMA in low-risk patients: insertion of the ordinary laryngeal mask airway[2] and positive-pressure ventilation are well-tolerated both in the paralyzed[3] and in the nonparalyzed population.[4] In patients considered to have a high risk of aspiration but who are straightforward to intubate, the airway is most quickly and safely secured by tracheal intubation with direct vision. The blind technique of ILMA insertion has less success.[1]

In cases of expected difficult intubation, it is also more difficult to insert a laryngeal mask airway.[5] Awake fiberoptic intubation is well-proven to provide a safe and controlled means of intubating the airway with direct vision, with the added advantage of no loss of protection from regurgitation. When the airway is safe, then neuromuscular relaxation can be given. In contrast, a trial with the ILMA found failure to intubate the trachea in 3 of 31 patients.[6] This is unacceptably high in anesthetized, paralysed patients.

The laryngeal mask airway has found a place in the management of the unexpected difficult airway[7]: to complicate a difficult situation by then attempting blind intubation *via* the ILMA has little to commend it. Although the question that the authors posed was well-answered by their study, a more pertinent question would be this: If muscle relaxants are necessary, should the ILMA be used?

Stephen M. Edwards, M.B.Ch.B., F.R.C.A.,* Cait P. Searl, B.Sc., M.B.Ch.B., M.R.C.P., F.R.C.A. *Morriston Hospital, Swansea, United Kingdom. smedwards@doctors.org.uk

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In Reply:—Edwards and Searl have suggested that our article "highlights the failure of the ILMA [intubating laryngeal mask airway]" (*Fastrach*™; LMA North America, San Diego, CA). In our opinion, their comments reflect a misunderstanding of the potential role of the ILMA in airway management. The basis for their comment that "there is no advantage afforded by the use of an ILMA in low-risk patients" is

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unclear. In fact, it could be argued that the ILMA offers significant advantages over the "ordinary laryngeal mask airway" because it is easier to insert, offers the option of tracheal intubation—should the situation arise at a later time during the operation, and is less likely to be inadvertently discarded after the case. It is not clear what is lost by inserting an ILMA rather than an ordinary laryngeal mask airway.

Because we did not study patients with "expected difficult intubation,"[1] we cannot comment on the authors' statement that it is more difficult to insert the device in this patient population. However, we have used the ILMA successfully to intubate patients with cervical spine disorders.[2] Although it may be more difficult to insert a regular laryngeal mask airway in "expected difficult intubation," this may or may not be true with the ILMA. Further studies with the ILMA in patients with difficult airways are clearly needed.

Finally, using a small dose of a muscle relaxant to facilitate tracheal intubation with the ILMA does not necessarily negate its potential facilitating tracheal intubation.

Janet M. van Vlymen, M.D., F.R.C.P.C., Margarita Coloma, M.D., W. Kendall Tongier, M.D., Paul F. White, Ph.D., M.D., F.A.N.Z.C.A.* *University of Texas Southwestern Medical Center at Dallas, Dallas, Texas. paul.white@utsouthwestern.edu

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Lower Respiratory Tract (LRT) Infection

To the Editor:—The September 2000 issue of ANESTHESIOLOGY contains an article by Akça *et al.*[1] that is of great potential importance to critical care anesthesiologists caring for patients with the clinical manifestations of ventilator-associated pneumonia (VAP).[2] The authors[1] report lower respiratory tract infections (VAP) with multidrug resistant organisms in certain patients "early" in their intensive care course. However, these findings are not entirely straightforward for the reasons noted herein.

The use of clinical criteria, even when supplemented by quantitative culture, to diagnose VAP is overly sensitive and of limited value[3] in differentiating lower respiratory tract infection involving only the airways (colonization and purulent bronchitis for which antibiotic therapy is generally not indicated and, when used, can increase the number of multidrug resistant organisms as well as delay diagnosis of the true cause[2]) from what also involves the lung parenchyma—VAP. VAP caused by multiresistant bacteria is associated with an increased attributable mortality, and timely, accurate antibiotic therapy has been shown to improve outcome.

As noted, it is the authors'[1] finding of "early" multidrug resistant organisms in the lower respiratory tract that is of great interest. This information may allow accurate initial therapy in patients who have VAP. However, most patients to whom antibiotics are administered and in whom pulmonary infiltrates develop in the intensive care unit do not have VAP.[2,4] Therefore, until improved diagnostic and clinical strategies are shown to better differentiate lower respiratory tract colonization from VAP and allow better use of antibiotics, we must be cognizant of the ongoing potential to increase inadvertently the burden of multiply resistant organisms from excessive, inappropriate antibiotic treatment when using current standard diagnostic methods similar to those used by Akça *et al.*[1]

Kenneth E. Shepherd, M.D., Harvard Medical School, Boston, Massachusetts, and Massachusetts General Hospital, Boston, Massachusetts. KEShepherd@partners.org

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In Reply:—First, we share the concerns of Dr. Shepherd about diagnosing ventilator-associated pneumonia (VAP).[1] However, neither one of our commentaries bring a novel and accurate diagnostic approach to VAP. Dr. Shepherd was concerned whether we overdiagnosed or overtreated our patients. In the meantime, we are concerned whether the *recommended* invasive (bronchoscopic) diagnostic techniques[2] worsen the ventilatory status and management of patients by prolonging the duration of mechanical ventilation. Both of the clinical concerns are extremely important because VAP is the leading nosocomial infection in the intensive care unit,[3] and it surely increases mortality.[4]

In response to Dr. Shepherd's concern, I would like to refer to two recent studies in which the investigators tested the sensitivity and specificity of various diagnostic techniques by the help of "gold standard" histologic and microbiologic references.[5,6] Torres *et al.*[6] showed that the sensitivity (43–83%) and specificity (67–91%) ranges of both invasive and noninvasive diagnostic sampling techniques were unsatisfactory, and causative organisms were missed in a significant number (17–83%) by all techniques. Their conclusion was that all sampling techniques for detecting VAP were of limited value.

Fabregas *et al.*[5] showed that clinical criteria—which were nearly the same as those we used— had reasonable diagnostic value. Noninvasive and invasive sampling techniques had diagnostic values comparable to clinical criteria. They concluded that an algorithm guiding antibiotic treatment exclusively by microbiologic results did not increase the overall diagnostic accuracy and would have the risk of undertreatment.

In short, diagnosis of VAP is extremely important in care of the

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critically ill. Moreover, we still do not have the accurate diagnostic tools.

Ozan Akça, M.D., OUTCOMES RESEARCH™ Institute, University of Louisville, Louisville, Kentucky. ozan.akca@louisville.edu

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LTA Cannula Can Facilitate a Difficult Tracheal Intubation

To the Editor:—A Cormach and Lehane[1] grade III view of the glottus often is associated with difficulty in passing an endotracheal tube. This situation occurs infrequently; I estimate that I have encountered no more than 200 such patients during 30 yr of practice. In this letter, I describe a modification of a conventional intubation technique using a laryngotracheal spray cannula to identify the laryngeal aperture and guide the endotracheal tube into the trachea.

The LTA 360 Kit (Abbott Laboratories, North Chicago, IL) is a disposable syringe and cannula unit. The unit consists of a semiflexible, curved white cannula, 3 mm in diameter and 20 cm in length, fused to a 4-ml syringe filled with 4 ml lidocaine HCl, 4%. A black mark 10.5 cm proximal to the rounded distal tip provides a visual aid for the depth of insertion. With the black mark at the glottis, the multiple perforations in the cannula lie beneath the vocal cord and spray the trachea with topical anesthetic.

I use the LTA kit before intubating all my adult patients. If during laryngoscopy I do not see the familiar anatomical landmarks of the larynx, I try one or more of the conventional maneuvers, such as changing the position of the head, changing laryngoscope blade, or asking an assistant to apply pressure to the thyroid and cricoid cartilages. If these maneuvers fail, I plan for an alternative technique to intubate the trachea. However, if I can see the epiglottis and the posterior margin of the larynx (Cormack and Lehane[1] grade III), I will proceed with the intubation using the following technique.

With the laryngoscope in one hand and the LTA cannula in the other, I use the distal tip of the cannula to probe and identify the larynx. As I attempt to pass the cannula under the epiglottis and between the vocal cords, I try to displace the arytenoids posteriorly and to get a glimpse of the posterior commissure. I spray the larynx and trachea with the LTA lidocaine and then, while neither removing the laryngoscope from the hypopharynx nor removing the LTA cannula from between the vocal cords, I continue with the intubation sequence. With the LTA cannula resting against the right corner of the mouth, I insert the endotracheal tube between the LTA cannula (on the right) and the laryngoscope (on the left) and advance the endotracheal tube in the same direction as the cannula. When the endotracheal tube is presumed to be in the trachea, I remove the LTA cannula from the mouth, attach the tracheal tube to the anesthesia breathing circuit, and check for tracheal intubation by exhaled carbon dioxide and breath sounds.

I also have used this technique for two additional groups of patients (Cormach and Lehane[1] grade II) who may present a challenge to intubate. One group includes patients with a short epiglottis, an epiglottis that does not adequately retract with a curved laryngoscope and continually slips off the tip of a straight blade. The second group of patients includes the morbidly obese, particularly those with obstructive sleep apnea, whose pharyngeal soft tissues impinge on a clear view of the distinct landmarks of the larynx.

Other related techniques have been described. Nolan and Wilson[2] evaluated the use of a gum elastic bougie for tracheal intubation in a similar patients (Cormach and Lehane[1] grades II and III). They first placed an elastic guide into the trachea and then threaded the endotracheal tube over the guide. I have not had an opportunity to compare their technique with mine. However, in my practice, the LTA cannula is more readily available then the gum elastic bougie.

My personal series using this LTA cannula technique for 3 yr includes approximately 30 patients. For these patients, this technique has been 100% successful. I offer three possible explanations for the success of this technique. (1) The thin LTA cannula is easier to view as it passes into the glottis than is the thicker endotracheal tube. (2) The LTA cannula improves the exposure of the glottis by displacing the arytenoid cartilage posterior and lifting the epiglottis. (3) By using the movement of the LTA cannula to instruct an assistant applying posterior pressure to the larynx, I optimize the position and exposure of glottis.

I must emphasize that when one decides to use this technique and to leave the LTA cannula in the trachea, one is committed to continuing the intubation sequence. If the patient requires ventilation by mask, the LTA cannula must be removed. Initially, this technique may feel awkward, but hopefully, this communication will encourage other anesthesiologists to try it. For anesthesiologists who already use the LTA cannula as part of their routine intubation technique, this technique may facilitate a difficult intubation and obviate the need for additional equipment and the risk of additional attempts at tracheal intubation.

Alfred Feingold, M.D., Cedars Medical Center, Miami, Florida. afein@sprintmail.com

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