

Use of the Cook Airway Exchange Catheter® to Facilitate Fiberoptic Intubation: Are We Trying to Solve a Problem That We Created?

To the Editor:—In their report, Ayoub *et al.*¹ found that advancing the endotracheal tube (ETT) into the trachea over the fiberoptic bronchoscope (FOB) fails in about one third of patients. They also found that by inserting a Cook Airway Exchange Catheter® (Cook Critical Care, Bloomington, IN) (CAEC) alongside the FOB, tracheal intubation was successfully accomplished in these patients. They concluded that the presence of the FOB and the CAEC together inside the lumen of the ETT minimized the size of the cleft along the ETT bevel (created by the OD of the FOB and the internal diameter of the ETT), therefore decreasing the likelihood of impingement on the arytenoids cartilages and enhancing the chances of passing the ETT into the trachea.

Before this technique becomes an accepted practice, we would like to forward the following comments.

First, the authors' reported 32% failure rate of advancing the ETT over the FOB is unusually high. Had the authors used an appropriate fiberoptic intubation technique, their failure rate would have been extremely low. They used a FOB with an OD of 3.8 mm and ETTs with internal diameters of 7.5 and 8.0 mm. We believe that the great disparity in these diameters contributed to their high incidence of failure in passing the ETT over the FOB.

Second, we feel that the authors were biased in their comparison of the success rate using the FOB alone (despite its small diameter) *versus* the combined use of the FOB and the CAEC. They compared one attempt to advance the ETT over the FOB with up to three attempts when the CAEC was used. If only the first attempts of both techniques were compared, the success rate of using the FOB alone would have been higher (68%) than using both the FOB and the CAEC (9 of 16, or 56%).

Third, it is not clear how the authors directed the CAEC through the cords into the trachea. Because the FOB was already in the trachea, it could not have been used to visualize and direct the CAEC toward the cords. For the CAEC to pass through the cords under vision, its tip should be distal to the tip of the FOB. Withdrawing the FOB from the

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In Reply:—Thank you for referring to me the letter of Drs. El-Orbany, Klimas-Osolkowski, and Salem. We agree with Dr. El-Orbany *et al.* that the difference between the OD of the fiberoptic bronchoscope (FOB)(3.8 mm) and the ID of the endotracheal tube (ETT) (7.5–8 mm) contributed to the high incidence of failure in advancing the ETT over the FOB. However, in our department and in many other institutions, the 3.8 mm FOB may be the only available size. In order to solve this problem, multiple maneuvers have been suggested to facilitate the advancement of the ETT, such as 90-degree anticlockwise rotation and designing different tube tips.¹ Moreover, we have designed a removable conical polyvinyl chloride sleeve to sheath the insertion cord of the FOB, which increases its size from 3.8 mm to 5.5 mm; the use of this sleeve technique increases the incidence of successful advancement from the first attempt to 96%.²

Our report shows that failure from the first attempt when using the FOB alone amounts to 32%. It is only in these failed attempts that we introduced the Cook Airway Exchange Catheter® (Cook Critical Care, Bloomington, IN), which increased the success rate up to 9 of 16 from the first attempt. Also, as mentioned in our manuscript, when the ETT tube failed to pass over the FOB to the trachea, the FOB was not withdrawn, and the tip of the ETT tube was kept in close proximity to

trachea until its tip lies in the pharynx and aligning the CAEC alongside the FOB to introduce both as a unit through the cords is time consuming, carries no guarantee for a successful attempt, and leaves the airway unprotected in the process. Blind insertion of the CAEC with the expectation that it will find its way through the cords can cause, literally, what the authors were trying to avoid, that is, trauma making further attempts at ETT placement more difficult.

Fourth, from our experience and the experience of others, difficulty in passing the ETT over the FOB is rarely encountered.² The use of a proper technique is the best prophylaxis against failure of ETT advancement over a FOB. Using the largest FOB that fits easily inside an appropriate size ETT, using the jaw thrust maneuver (to decrease the posterior pharyngolaryngeal angle), applying generous lubrication, and placing the ETT in warm water to make it more pliable can ensure almost no failure in ETT advancement.² In the rare situation when the ETT cannot be advanced, a gentle 90-degree counterclockwise rotation can be successfully utilized.³ Introducing another device adjacent to the FOB during intubation can be time consuming, can cause trauma to the airway, and most importantly, should not be an alternative to the use of an appropriate fiberoptic intubation technique.

Mohammad I. El-Orbany, M.D.,* Katarzyna Klimas-Osolkowski, M.D., M. Ramez Salem, M.D. Advocate Illinois Masonic Medical Center, Chicago, Illinois. ninos-joseph@advocatehealth.com

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the glottis, which facilitated the introduction of the Cook Airway Exchange Catheter® from the first attempt.

In conclusion, we agree with Dr. El-Orbany *et al.* that the use of a proper technique is the best prophylaxis against the failure of ETT advancement over the FOB. However, we can still encounter failure to advance the tube over the FOB despite the use of a larger FOB or a smaller tube size, the application the jaw thrust maneuver, generous lubrication, and 90-degree counterclockwise rotation. It is this situation that the introduction of Cook Airway Exchange Catheter® can centralize the tube in front of the glottis and facilitate advancement.

Chakib M. Ayoub, M.D.,* Antoine M. Lteif, M.D., Marwan S. Rizk, M.D., Naji M. Abu Jalad, M.D., Ussama Hadi, M.D., Anis S. Baraka, M.D., F.R.C.A. American University of Beirut, Beirut, Lebanon. chakib.ayoub@aub.edu.lb

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Nerve Root "Irritation" or Inflammation Diagnosed by Magnetic Resonance Imaging

To the Editor:—In their case report, Avidan *et al.*,¹ suggested that transient nerve root irritation may be evident in magnetic resonance imaging as an enhancement of the affected nerve roots. Fortunately, their case was apparently transient, although it was not followed with a second magnetic resonance imaging weeks or months later. In figure 1 of Avidan *et al.*,¹ enhancement of nerve roots implies inflammation and edema² as the contrast media gadolinium is extruded into the extraneural vascular space (endoneurium), probably in the early phase of arachnoiditis.³ From this point on, some cases may evolve into the proliferative phase of arachnoiditis with infiltration of fibroblasts and progressively denser collagen forming adhesions, fibrosis, and scarring.⁴ Why some cases advance and others do not is not yet clear, but Myers and Sommer³ noted that neurotoxic injury to the cauda equina may be patchy because the neurotoxic agent distribution may be uneven, which is also a characteristic distribution of pain and dysesthesia in arachnoiditis.⁵

Enhanced but not abnormally distributed nerve roots, noted in figure 1 of Avidan *et al.*,¹ are seen in the inflammatory stage of arachnoiditis. In contrast, clumped nerve roots, usually abnormally distributed in the thecal sac, shown in figure 2, seen 3 to 7 months after the injurious event,⁶ may indicate that fibrinous bands and thicker collagen are beginning to develop, forming adhesive and sometimes constrictive arachnoiditis. Matsui *et al.*⁷ showed in serial magnetic resonance imaging studies performed every 7 days after laminectomy up to the 49th day that only 20% of the cases with evidence of nerve root enhancement progressed to arachnoiditis.

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In Reply:— We would like to thank Dr. Aldrete for his remarks, which enlighten our clinical and magnetic resonance imaging (MRI) findings in a patient with transient neurologic symptoms after spinal injection of lidocaine.

As Dr. Aldrete commented, there is more to be learned about this phenomenon and about MRI findings at different stages not only after intrathecal, but also after epidural anesthesia.

Although epidural infection is a very rare complication, it has become an important clinical problem with the increased use of epidural catheters for prolonged analgesia. At present, MRI is the diagnostic procedure of choice. Although the characteristic appearance of epidural and spinal infection is well documented by MRI, it has not been

Obviously, we need to learn more about this phenomenon. Nevertheless, it is important to identify these cases at this point because the duration of the inflammatory period has not yet been defined precisely and because early treatment with nonsteroidal antiinflammatory drugs and corticosteroids may prevent its evolution into the proliferative phase.

J. Antonio Aldrete, M.D., M.S. University of South Florida School of Medicine, Tampa, Florida. taldrete@arachnoiditis.com

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compared with MRI findings in asymptomatic patients after epidural or spinal injections (with or without catheter). This lack of knowledge may cause a problem in diagnosis when evaluating MRI scans of patients with suspected epidural or spinal infections.

We are therefore planning a controlled prospective study of serial MRI after spinal and epidural injections.

Alexander Avidan, M.D.,* Elyad Davidson, M.D., Moshe Gomori, M.D. *Hadassah University Hospital, Ein Kerem, Jerusalem, Israel. avidan3@netvision.net.il

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Postoperative Cognitive Dysfunction: Overinterpretation of Data?

To the Editor:—In their multicenter, cohort study of patients undergoing major surgery, Johnson *et al.*¹ detected a fivefold increase in early postoperative cognitive dysfunction (POCD) in middle-aged patients undergoing major surgery with general anesthesia when compared with their age-matched relatives. In addition, *post hoc* univariate and multivariate logistic regression analyses suggested that alcohol cessation, allocation to test centers, and the additional administration of epidural analgesia were factors that significantly contributed to the risk of POCD.

The observation that epidural analgesia contributed to POCD is a surprising finding as it is in contrast to a separate publication in which

investigators of the same ISPOCD2 multicenter group did not find a significant difference in POCD between general anesthesia and regional anesthesia in 525 randomized patients at 1 week and 3 months after surgery.² Similarly, Williams-Russo *et al.*³ demonstrated in a prospective, randomized, controlled study of 262 older adults that epidural anesthesia does not increase the incidence of POCD at 1 week and 6 months even when continued as postoperative epidural analgesia. It is thus difficult to understand why Johnson *et al.*¹ put such emphasis on an outcome that is derived from a *post hoc* and underpowered data analysis of a subgroup of 92 nonrandomized patients who probably underwent more invasive and extensive surgery than

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the rest of the observation group. Despite these limitations, the authors invoke a speculative hypothesis of local anesthetic toxicity to explain the association of epidural analgesia with POCD.

The risk factor analysis in this study is further weakened by the fact that those patients presenting to different centers had a highly significant variation in risk for POCD, which is shown in table 5.¹ Study centers could correct for variations "with regard to patient population, data collection variations, and anesthetic and surgical practices."¹ We are left to speculate as to the many reasons why patient outcome is dissimilar at the various contributing centers. This confounder could invalidate the entire study by masking the identification of other risk factors and calls for further explanation.

Hence, the authors' summary statement that epidural analgesia is a risk factor for POCD is at best a tenuous speculation based on a *post hoc* analysis of a small, nonrandomized subgroup of patients. This questionable conclusion could result in a neuroaxial technique being withheld despite compelling level 1 evidence for improved cardiovascular and pulmonary outcomes and a reduction in perioperative mortality.⁴ Perhaps a more rational message based on the presented data should be not to caution against the practice of epidural analgesia, but rather to warn patients against having operations at the particular centers that exhibit a significantly higher incidence of POCD.

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In Reply:—Thank you for the opportunity to reply to Rosemeier *et al.*. The studies comparing general with regional anesthesia both involve only elderly patients.^{1,2} Our own original study, ISPOCD³ confirmed that age was the predominant determinant of postoperative cognitive dysfunction (POCD) in the elderly. The current study on middle-aged patients allows for the possibility that other factors such as subtle drug side effects that would be obscured in an elderly population may be revealed.

The high incidence of POCD in the patients receiving epidural analgesia was contrary to our expectations. It was part of our primary data, and we were obliged to report it. We did not conclude that there was a causal association between epidural analgesia and POCD; rather, we suggested a hypothesis to explain our findings (hypotheses are by definition speculative). We clearly acknowledged that there may be explanations other than a direct causal relation between epidural analgesia and POCD to account for our findings (*e.g.*, more major surgery) even though we took into account duration of surgery and blood loss.

We agree that there is strong evidence to support the use of epidural analgesia after major surgery especially in high-risk patients. We certainly did not caution against it but recommend keeping an open mind.

Rosemeier *et al.* should not be alarmed that a multicenter study such as ours revealed significant differences in outcomes between participating centers. Patient selection and characteristics, hospital environment, policies, and protocols inevitably vary, and all may be relevant in the etiology of POCD. Differences in language, culture, testing personnel, and test timing may influence its assessment. The center statistic was included to compensate for these unknown factors in the analysis.

Frank Rosemeier, M.D., M.R.C.P.,* Michael Avidan, M.B., Ch.B., F.C.A., Andrea Kurz, M.D., Ph.D., Menelaos Kranikolas, M.D., Alex Evers, M.D. *Washington University School of Medicine, St. Louis, Missouri. frank_rosemeier@yahoo.com

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In the multivariate analysis, we were looking for factors in addition to the differences between the centers. The analysis may be underpowered for some effects, but our observations were, nevertheless, statistically significant at the conventional level.

Larger and more detailed studies are required to explore all of the risk factors determining POCD that may or may not include epidural analgesia.

Tim Johnson, F.R.C.A.,* Terri Monk, M.D., Lars S. Rasmussen, M.D., Kari Korttila, M.D., Volkert D. Siersma, M.T.D., Jaime Canet, M.D Hope Hospital, Salford, United Kingdom. tim.johnson@srht.nhs.uk

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Neurally-mediated Cardiotoxicity of Local Anesthetics: Direct Effect of Seizures or of Local Anesthetics?

To the Editor:—Ladd *et al.*¹ showed in conscious sheep that the administration of incremental doses of local anesthetics in the central nervous system bloodstream induces minimal systemic recirculation,

dose-dependent central nervous system excitatory behavior, and electroencephalographic changes. These authors also observed that bupivacaine is more neurotoxic than levobupivacaine, which is also more

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neurotoxic than ropivacaine. Finally, they stated that there are no differences among the drugs concerning their arrhythmogenicity.

It is well established that large doses of local anesthetics slow cardiac conduction velocity in a dose- and use-dependent manner by inhibiting the fast-inward sodium current.² The slowing of ventricular conduction velocity is responsible for a proportional QRS widening^{3,4} and facilitates the occurrence of reentrant ventricular arrhythmias around arcs of conduction block.^{5,6} In other words, the increase in heart rate by enhancing the slowing of ventricular conduction is one of the main mechanisms facilitating the occurrence of serious reentrant ventricular arrhythmias in the case of high plasma concentration. Moreover, Mazoit *et al.*⁷ and our team⁸ reported that bupivacaine is more use-dependent than levobupivacaine and ropivacaine. This fact explains at least in part the differences in direct cardiotoxicity in these three local anesthetics. As demonstrated by Bernards and Artru^{9,10} and our team,¹¹ an overdose of local anesthetics induces an increase in the sympathetic outflow by their direct neurotoxicity and therefore an increase in heart rate, facilitating the occurrence of serious reentrant arrhythmias when toxic cardiac concentrations are reached.

In the elegant study published by Ladd *et al.*,¹ RR and PR intervals are obviously shortened. In contrast and surprisingly, QRS is shortened by the administration of any local anesthetics at any dose. One might argue that the baseline values are somewhat widened and that the method of measurement, including the paper speed, is not specified in the method section. Nevertheless, because QRS is not widened, one could specially argue that there is no direct cardiotoxicity and that the observed arrhythmias are only due to the pre- and convulsant effects of local anesthetics as any seizures can provoke. In conclusion, the sentence in the summary that "no differences were found in their arrhythmogenic potential"¹ may be confusing because the analyzed arrhythmogenicity in the present study is only in terms of indirect and neurally mediated cardiotoxicity and not in terms of direct cardiotoxicity.

Jean E. de La Coussaye, M.D., Ph.D.,* A.Guy M. Aya, M.D., Ph.D., Jean-Jacques Eledjam, M.D. *University-Hospital of Nîmes, France. jean.emmanuel.delacoussaye@chu-nîmes.fr

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In Reply:—We thank Dr. de La Coussaye *et al.* for their interest in our paper.¹ We are very mindful of their major contributions to the investigation of local anesthetic intoxication, and we are pleased to respond to their constructive comments.

Our paper describes studies with an experimental preparation in which drug effects produced by the infusion of local anesthetics directed to the brain (and some other parts of the head) in conscious animals are limited to those effects caused by their actions on the brain. We measured the recirculating drug concentrations and believe they were too low to cause significant effects anywhere in the body other than in the brain. In their letter, de La Coussaye *et al.* have given an eloquent description of modification by local anesthetics of electrical events in the heart. Over many years in our various experiments on conscious sheep we have repeatedly observed that the central neurotoxicity of these drugs increases sympathetic drive on the heart, and we agree that this would be the principal cause of the arrhythmias seen *in conscious subjects*. However, because previous experiments using intravenous doses in conscious subjects have produced toxic drug concentrations in the heart and brain concurrently, it has not been previously possible to observe the effects of local anesthetic-induced central nervous system sympathetic stimulation alone. Our ongoing studies are attempting to examine the issue of concurrent heart and brain intoxication in the absence and presence of such sympathetic stimulation, by performing crossover studies in anesthetized *versus* conscious subjects.

In our paper, shortened PR and RR intervals are certainly consistent with sympathetic stimulation, but it is not clear why QRS intervals are

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shortened. The QRS shortening follows a similar flat dose-response trend to that of RR interval, but we point out that this change in QRS is comparatively minor and, moreover, is much shorter lived, suggesting a different cause. In response to the question about our data acquisition system, the analogue signal is amplified and converted to 16-bit digital data at 256 Hz; therefore, our aliasing time is a little less than 8 ms. This sampling format is sufficient to find any obvious changes in timings and also to discern the peaks of electrical derivatives of left ventricular pressure (LV-dP/dt) acquired concurrently on the same system.

In conclusion, the lack of widening of QRS does indeed suggest a lack of direct cardiotoxicity. This preparation was set up to answer the specific question of indirect cardiotoxicity and to separate the components of direct and indirect toxicity. It is therefore best read with its companion paper,² which deals with direct effects in a conscious preparation in which the heart is specially targeted with graded, toxic doses of the same local anesthetics without pharmacologically significant drug recirculation to brain. Perhaps we were remiss in stating that "no differences were found in their arrhythmogenic potential" without adding the qualifying phrase "in terms of their central nervous system mediated (indirect) cardiac toxicity." We thank Dr. de La Coussaye *et al.* for pointing this out. We had intended that our paper convey this message.

Leigh A. Ladd, B.VSc., Susan E. Copeland, M.VetClinStud, Laurence E. Mather, Ph.D.* University of Sydney at Royal North Shore Hospital, St. Leonards, Australia. lmather@med.usyd.edu.au

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Efficacy of Acute Normovolemic Hemodilution in Cardiac Surgery

To the Editor:—We read with interest the recent article published by Casati *et al.*¹ evaluating the use of acute normovolemic hemodilution (ANH) in adult cardiac surgery. In this study, the authors failed to demonstrate any beneficial effect of ANH on allogeneic blood requirements. However, this study evaluated the contribution of ANH as part of a blood conservation strategy rather than the efficacy of ANH itself. Indeed, the efficacy of ANH as a blood-saving technique is essentially related to a reduction of the net erythrocyte loss. Therefore, as shown by different mathematical analyses, the efficacy of the technique depends on the initial erythrocyte mass (which is related to the preoperative hematocrit and the circulating blood volume), the transfusion trigger (the hematocrit at which erythrocytes will be transfused), and most importantly, the perioperative blood losses.^{2,3}

In the study from Casati *et al.*,¹ the baseline blood conservation approach aimed to markedly decrease perioperative blood losses by the use of cell-saving techniques and administration of tranexamic acid. In addition, the amount of blood harvested by ANH was quite low (500 ml) and returned to the patients before leaving the operating room. As a result, hematocrit at intensive care unit arrival was similar in the control and ANH groups. As postoperative blood losses were comparable in the ANH and the control groups, the net postoperative erythrocyte loss was similar in both groups. It is therefore not surprising that the use of limited ANH in such a blood conservation approach did not result in a decreased allogeneic blood requirement.

The demonstration of the efficacy of ANH as a blood conservation technique requires protocols that either avoid the concomitant use of other techniques decreasing perioperative blood loss or compare these techniques with ANH. Clearly, the adoption of a well defined blood

conservation strategy results in a significant reduction in allogeneic blood requirements in cardiac surgery.^{4,5} The effectiveness of any blood conservation strategy depends on a careful cost-benefit analysis of the different techniques to be used in relation to the patient's limitations, the type of surgical procedures and techniques, and the health care environment.

Philippe J. Van der Linden, M.D., Ph.D.,* Stefan G. De Hert, M.D., Ph.D.† *Dept. of Cardiac Anesthesia, CHU Charleroi, Belgium; †Dept. of Anesthesiology, UZ Antwerpen, Belgium. philippe.vanderlinden@chu-charleroi.be

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In Reply:—I thank Van der Linden and De Hert for their comments regarding our article.¹

I want to underline again that the aim of our work was not to demonstrate the efficacy *per se* of intraoperative acute normovolemic hemodilution (ANH) in adult open-heart surgery, but whether the introduction of low volume ANH in our standardized intraoperative blood-sparing protocol, constituted by the association of the infusion of tranexamic acid and the use of a cell-saving technique, could produce some additive advantage in terms of reduction of allogeneic transfusions. Furthermore, in the discussion of the article we justified our choice to perform low volume ANH with the intention to enroll also in the study patients with low body mass index and patients with relative low values of hemoglobin (12 g/dl).

The negative results of our study clinically confirm the theoretical conclusions of mathematical analysis on ANH,² which state that only high volume ANH in presence of significant intraoperative bleeding significantly reduces the need for allogeneic transfusions.^{3,4}

Furthermore, our study may be considered an indirect demonstration of the efficacy of our intraoperative blood-sparing protocol to prevent excessive blood loss, rendering unnecessary the addition of low volume ANH in this type of patient.

I totally agree with the conclusions of Van der Linden and De Hert: each blood conservation strategy must be studied considering the patient's limitations, the type of surgical procedures, and the health

care environment. Because my opinion is that the addition of an adequate amount of ANH to our actual effective blood conservation approach⁵ may determine, particularly in patients with an increased risk for excessive bleeding, further improvements to our research of a cardiovascular surgery with the minimum number of patients necessitating donor blood; at the moment, various studies considering high volume ANH are in progress at our institution.

Valter Casati, M.D. Policlinico di Monza, Italy. valter.casati@policlinicodimonza.it

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Comparison of External Heat Exchange Systems

To the Editor:—The finding by Kabbara *et al.*¹ that “standard hospital blankets heated to 38°C forced air were equally as effective (in maintaining core temperature) as commercial blankets heated with forced air to 43°C” does not mean that the heat exchange capabilities of these two systems are the same. Core temperature (Tc) cannot discriminate between external heating systems such as these, because it is insulated from any heat exchange at the skin by the prevailing thermal conductance, the heat exchange rate between core and skin. Thermal conductance is the reason that in any heat exchange cascade, Tc is the last temperature to change.

Accurate discrimination requires measurement of the *mean skin temperature* (Tmsk), the *area-weighted average temperature* of multiple skin sites representative of the body surface area. Tmsk is the relevant temperature because the skin is the heat exchange site of the systems being compared and because Tmsk, together with thermal conductance, determines the rate of change in Tc. Being “equally as effective” at maintaining Tc does not mean that the two systems are “equally as effective” as heat exchangers at the skin. Without measuring Tmsk, one cannot determine whether these systems maintained Tc by design or by default, and 29% of all patients still became hypothermic.

In whole body rewarming of hypothermic, postoperative patients, Ereth *et al.*² also found no difference in Tc between one group warmed with a Bair Hugger (Augustine Medical Inc., Eden Prairie, MN) at 43°C and another with blankets warmed to 40°C and changed every 15 min. Tmsk was significantly higher in the Bair Hugger group but, despite this active warming, Tc did not increase more rapidly, Ereth argued, because of “thermoregulatory vasoconstriction” (*i.e.*, low thermal conductance).² In fact, because Tmsk < Tc, direct heating of Tc would have been impossible. In Ereth’s study, the Bair Hugger was rendered ineffective because it was insulated from the patient by an intervening cotton blanket. While Kabbara does not say whether his Bair Hugger was insulated in this way, this practice has become common and could explain the lack of a significant difference in this study.

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In Reply:—The purpose of our study was to evaluate an experimental forced air warming technique in surgical patients during general anesthesia.¹ Distal esophageal temperature was chosen as the primary outcome measure to quantify the degree of intraoperative core hypothermia, because most thermoregulatory research incorporates core temperature measures and because nearly all the major adverse effects of mild hypothermia (*e.g.*, decreased resistance to wound infections, morbid cardiac outcomes) have been described in terms of core temperature.^{2,3} Compared with the standard commercial forced air warming system, the experimental technique resulted in similar mean core temperatures (> 36°C) between groups at the end of surgery. We did not measure thermal conductance of the heat exchange systems used, and as Dr. English points out, accurate discrimination of thermal conductance between external heating methods requires measurement of area-weighted average temperature of multiple skin sites. Therefore, our study was able to quantify an important and relevant outcome measure, core temperature, but was not able to quantify the amount of heat exchange at the skin.

Although the laboratory investigation of Dr. Kempen⁴ (which inspired us to perform this study) showed that forced air warming with hospital bed sheets was able to heat standardized thermal bodies twice as effectively as commercial blankets using identically warmed 38°C forced air, the efficacy of forced air warming systems in maintaining

Commercial blankets are designed to provide a low resistance airflow path to distribute heat consistently over the skin *area* under the cover. Such a consistent heated area cannot be guaranteed by blowing air between blankets, because of the variable airflow resistance of such an *ad hoc* arrangement. It is a basic law of physics, which this paper will not change, that all heat exchange is a function of area. The authors rely heavily on a model by Kempen,³ which was biased in favor of the noncommercial system because it measured only in the low resistance airflow path and ignored the effect of airflow distribution. A model whose centerpiece was a “standard thermal body” from which “heat transfer” was estimated. But this estimate was irrelevant, because the standard thermal body has no clinical counterpart; inappropriate, because the standard thermal body was *unperfused* and, therefore, depended on temperature, not the heat transfer rate; and inaccurate, because the standard thermal body was *unstirred*.

Tc is one important temperature in thermoregulation. But in any “further evaluation” of their suggested method, I trust that the authors will address not only the physiology of thermoregulation but also the physics of heat exchange. It would then be possible to attribute a plausible cause to a perceived effect.

Michael J. English, M.B., B.S., F.R.C.A. Montreal General Hospital, Montreal, Quebec, Canada. englishm@sympatico.ca

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intraoperative normothermia in anesthetized patients depends not only on heat exchange area, but also on the total energy flux exiting the warming hose, insulating effect of sheets or blankets covering the patient, degree of thermoregulatory vasoconstriction, airflow resistance beneath the blankets, thickness of the convective thermal boundary layer, and velocity distribution within and outside the convective thermal boundary layer. Moreover, metabolic heat production in combination with effective heat conservation may contribute substantially to the maintenance of perioperative normothermia regardless of heat transfer at the skin from external warming devices.⁵ Indeed, the relatively high incidence of core temperature < 36°C in both groups at the end of surgery (27-31%) in our study¹ was likely the result of limited surface area available for forced air warming, combined with infusion of ~ 2 l of room temperature IV fluids. Infusing unwarmed (or inadequately warmed) IV fluids has been shown to increase the incidence and severity of perioperative hypothermia.^{6,7} We agree with Dr. English that any further evaluation of our experimental forced air warming technique should address not only safety and efficacy, but also thermal balance and body heat content.

Abdallah Kabbara, M.D., Charles E. Smith, M.D., F.R.C.P.C.* Alfred C. Pinchak, P.E., Ph.D., M.D. MetroHealth Medical Center, Cleveland, Ohio. csmith@metrohealth.org

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Why Not Use Amsorb Alone as the CO₂ Absorbent and Avoid Any Risk of CO Production?

To the Editor:—I read with interest the recent article by Knolle *et al.* regarding the use of Amsorb (Armstrong, Coleraine, Northern Ireland) to detect the dehydration of conventional carbon dioxide (CO₂) absorbents that contain strong bases.¹ Amsorb is a new CO₂ absorbent, which does *not* contain strong bases such as sodium or potassium hydroxide.² The authors correctly note that the dehydration of strong base containing CO₂ absorbents cannot be detected in clinical practice, and this places patients undergoing general anesthesia at risk for potentially lethal carbon monoxide (CO) poisoning due to the degradation of volatile inhaled anesthetics by such dry absorbents.

Knolle *et al.* found that by placing a layer of Amsorb either above or below a conventional CO₂ absorbent, depending on the direction of gas flow, a color change in the Amsorb can be used to detect the desiccation of the conventional absorbent. This could alert a clinician to change the conventional CO₂ absorbent to prevent CO formation when volatile inhaled anesthetics are employed.

However, the authors note that Amsorb itself has been shown to be an effective CO₂ absorbent, which does not produce CO when used with volatile inhaled anesthetics even when completely dry.^{2,3} Therefore, patients face no risk of CO poisoning when Amsorb alone is employed as the CO₂ absorbent.

My question to the authors is: why not simply use Amsorb alone as the CO₂ absorbent and avoid the risk of CO production at all? This would also avoid the need to specially prepare a conventional strong base containing CO₂ absorbent with a layer of Amsorb in the appropriate area and watch for a color change there.

Mitchel B. Sosis, M.D., Ph.D. Holy Redeemer Hospital and Medical Center, Meadowbrook, Pennsylvania. mitchelsosis@hotmail.com

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In Reply:—We thank Dr. Sosis for his comment in which he wonders why we propose the combination of strong base free Amsorb with strong base containing CO₂ absorbents and not the simpler alternative of Amsorb alone.

As Dr. Sosis points out, Amsorb (Armstrong, Coleraine, Northern Ireland) has the advantage of not producing CO when used with volatile inhaled anesthetics. That makes it a most attractive CO₂ absorbent. However, we see a second issue here, and that is that Amsorb has been shown to have only half the CO₂ absorbing capacity of strong base containing absorbents.¹ We actually confirmed that finding in one² of the papers cited by Dr. Sosis in reference to Amsorb's advantage as a CO₂ absorbent. In clinical anesthesia, this characteristic of Amsorb results in higher consumption and consequently in higher costs.³ For these reasons, many anesthesiologists may hesitate to use Amsorb. We see our proposed combination, which takes advantage of an unexpected property of Amsorb, allowing both avoidance of CO

production and optimum CO₂ absorption, as widening the range of choices available to anesthesiologists at the present time.

Erich Knolle, M.D.,* Hermann Gilly, Ph.D. *University of Vienna, Vienna, Austria. erich.knolle@univie.ac.at

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The Influence of the Duration of Anesthesia on Neuromuscular Potency

To the Editor:—I would like to congratulate Drs. Plaud *et al.*¹ on their recent study of the influence of the duration of anesthesia on neuromuscular potency. Their data nicely explain the frequent observation that after the administration of doses of muscle relaxants, which are twice the reported ED₉₅, complete twitch suppression at the adductor pollicis may not occur when the blocking drug is given shortly after the induction of anesthesia. However, I do not necessarily agree with their statement that “it is reasonable to assume that the results of this study apply to [the] laryngeal muscles,”¹ nor to the conclusion that their observations explain why doses of 2–3 × the ED₉₅ are necessary to obtain excellent conditions for intubation.

They correctly note in their discussion that apparent neuromuscular potency may be increased by enhancing drug delivery to the effect site by increases in muscle blood flow. Skin temperature at the hand may increase by 5°C in the first few minutes of general anesthesia.^{2,3} This increase in temperature presumably reflects an increase in muscle perfusion and skin blood flow. If this is so, then drug delivery to the muscles of the hand will be enhanced and should result in higher peak drug levels at the myoneural junction. As a consequence, a dose that produces a 95% twitch depression at the adductor pollicis after 15 min of general anesthesia (when most dose–response studies are actually performed) will result in a lesser degree of block (at the hand) when administered immediately after induction of anesthesia.

Comparable changes in muscle temperature and hence muscle perfusion of muscles more relevant to ease of intubation than the adductor pollicis (the masseter, the laryngeal adductors, the diaphragm) are unlikely. It is generally recognized that the indirectly evoked muscular

response at the hand is not a very useful measure for evaluating readiness for tracheal intubation. A brisk (but diminished) response of the adductor pollicis does not preclude excellent conditions for laryngoscopy.

The term “intubation dose” is not necessarily synonymous with a dose of relaxant $\geq 2 \times$ the ED₉₅. This convention dates back to the mid 1980s when available neuromuscular blockers had relatively slow onset profiles. For drugs with a faster onset of action, lower multiples of conventionally cited ED₉₅s are entirely practical.^{4,5}

Aaron F. Kopman, M.D. Saint Vincent's Hospital and Medical Center, New York, New York. akopman@rcn.com

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In Reply:—We thank Dr. Kopman for his remarks on our article and would like to take this opportunity to interpret our findings in a different way. We measured the onset characteristics of mivacurium-induced neuromuscular blockade at two muscles, the adductor pollicis and the corrugator supercilii muscles, to rule out the possibility of a local effect (temperature or blood flow) at one muscle.¹ The duration of anesthesia before mivacurium injection had a major influence on the level of paralysis at both muscles, suggesting that local effects at the adductor pollicis were not a major factor. We proposed that these results might apply to laryngeal muscles because we have previously demonstrated that after rocuronium injection the pharmacodynamic profile of the corrugator supercilii is the same than at the adductor laryngeal muscles.²

It should be noted that muscle blood flow was not measured in our study and that its role in enhancing neuromuscular blockade is only a hypothesis. Other factors, such as the broader plasma concentration profile that occurs after a bolus injection when cardiac output is decreased (a front end kinetics phenomenon³) could explain the greater neuromuscular blockade seen after 15 min of anesthesia. Likewise, our results are validated by the fact that we observed the same trend in maximum neuromuscular blockade at two different sites of measurement (*i.e.*, the adductor pollicis and the corrugator supercilii).

We agree with Dr. Kopman that the adductor pollicis is not a valuable guide for evaluating the time of tracheal intubation. This statement has been demonstrated repeatedly since the original study performed by Bencini and Newton.⁴

Finally, Dr. Kopman emphasizes that the exact intubating dose of muscle relaxant is controversial: some experts recommend twice the

ED₉₅ measured at the adductor pollicis,⁵ others suggest a lesser dose,⁶ and others report good results with no muscle relaxant.⁷ The debate continues because to date, no data are available regarding the exact minimal dose of the three major components of anesthesia (*i.e.*, hypnotic, the opioid, and the muscle relaxant) that will provide good or excellent conditions in > 95% of cases, without major side effects (*i.e.*, hypotension with a large dose of hypnotic, pharyngolaryngeal injury when no muscle relaxant is injected, major hypertension, or awareness with underdosage of opioid or hypnotic). All our study provides is an explanation for the occasional failure of muscle relaxants to provide excellent conditions in all cases, even at relatively large doses (2 × ED₉₅).

Benoit Plaud, M.D., Ph.D., Francois Donati, Ph.D., M.D., F.R.C.P.C.* *Hopital Maisonneuve Rosemont, Montreal, Quebec, Canada. francois.donati@umontreal.ca

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Central Block or Not Central Block under Ticlopidine Therapy?

To the Editor:—I wish to comment on the case report by Maier *et al.* about severe hematomas after lumbar sympathetic blockades in patients under irreversible platelet inhibitors.¹ I would like to highlight, respectfully, that inappropriate MEDLINE searches were performed. The authors stated, "There are no studies regarding the safety of epidural or spinal anesthesia in patients during or immediately after discontinuation of antiplatelet drug treatment. Only one case report describes a subarachnoid hematoma following a very difficult lumbar puncture in a patient receiving ticlopidine." Horlocker *et al.*² prospectively studied 1,000 procedures in 924 patients given spinal or epidural anesthesia to determine whether preoperative antiplatelet medications were related to the development of hemorrhagic complications. Antiplatelet therapy was defined as aspirin or any other nonsteroidal antiinflammatory drug. There were no documented spinal hematomas. A literature search on the National Library of Medicine's MEDLINE system identified three reports of spinal hematoma associated with ticlopidine therapy and central block.³⁻⁵ Mayumi and Dohi³ described a case of a 70-yr-old woman admitted for amputation of the first toe during spinal anesthesia. Ticlopidine administration was discontinued. After several unsuccessful attempts at lumbar puncture using a 23-gauge spinal needle, the operation was performed during general anesthesia. On the second operative day, urinary retention developed. Six days after surgery, muscle weakness developed and on the eighth postoperative day, a myelogram demonstrated an extramedullary hematoma. At laminectomy, a hematoma was found in the subarachnoid space. The neurosurgeon noted no vascular abnormalities or tumors. This was the first spinal hematoma reported in association with central

nervous block in a patient without a vascular tumor who was taking ticlopidine. Kawaguchi and Tokutomi⁴ described a 72-yr-old woman admitted for choledocholithotomy during general anesthesia combined with epidural block. Ticlopidine administration was stopped 12 days before the operation. An epidural catheter was inserted at T9-T10 interspace. Just after the operation, motor paralysis occurred. After 2 days, magnetic resonance imaging revealed epidural hematoma extending from T7 to L1.

Maurizio Fattorutto, M.D. Hospital Erasme, Brussels, Belgium.
m.fattorutto@swing.be

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In Reply:—We thank Dr. Fattorutto for his comments and for providing two additional references to our topic.^{1,2} One of these case reports has already been included in the review of Urmey and Rowlingson.³ However, these case reports and this review did not influence European and US guidelines at all. Dr. Fattorutto cites the study of Horlocker *et al.*,⁴ which is no counterargument because the patients received aspirin-like drugs, but none of them received an irreversible antiplatelet aggregation inhibitor like ticlopidine. The authors were aware of the small power of their retrospective trial, which lead them to the conclusion that "the finding of zero events does not imply the risk is zero in the whole population."⁴ Life-threatening bleeding is not a statistical problem; any bleeding is too much bleeding. Therefore, we appreciate Dr. Fattorutto's comments. To prevent bleeding, our recommendation should not be neglected.⁵ Any medication with irreversible antiplatelet aggregation inhibitors should be discontinued for at least 7 days before any invasive techniques like neuroaxial blockade, in which hemorrhage cannot be controlled by external compression.

Christoph Maier, M.D., Ph.D.,* Thomas Weiss, M.D., Michael Zenz, M.D., Ph.D. *Berufsgenossenschaftliche Kliniken Bergmannsheil, Bochum, Germany. christoph.maier@ruhr-uni-bochum.de

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Overdose, Coronary Artery Disease, or Both

To the Editor:—I read with interest the case report by Mueller *et al.*, which appeared in the September issue of ANESTHESIOLOGY.¹ I believe that the precipitating cause of the arrest in this case may have been an overdose of sodium thiopental followed by additional use of a β -blocker and not exclusively the undetected coronary artery disease.

We know the recommended induction dose of pentothal for “healthy” patients is 2.5 to 4.5 mg/kg.² This 47-year-old patient had received a dose of more than 6 mg/kg of thiopental together with 100 mcg of fentanyl and 60 mg of lidocaine for induction of general anesthesia along with isoflurane. We know that pentothal produces venodilation, decreasing myocardial contractility, and a decrease in sympathetic output. Even though the coronary angiogram was negative in this patient, he had severe risks to suspect the contrary: hypertension, history of angina, claudication, hyperlipidemia, and a smoking history of 20 pack-year.

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In Reply:—Unfortunately, Dr. Via-Reque appears to have missed the main point of our case report,¹ which was to alert anesthesiologists to the possibility that, in some rare cases, coronary angiography may grossly underestimate the true degree of coronary artery stenosis. As revealed at the autopsy, severe triple vessel atherosclerosis was unequivocally present in the patient described in this report and was the cause of his death.

To specifically address the points raised by Dr. Via-Reque, the dose of thiopental was similar to that used for a previous, uneventful anesthetic. In both instances, the drug was given in fractionated doses. The blood pressure and heart rate after tracheal intubation (176/105 mmHg, 101 bpm) do not support Dr. Via-Reque’s theory of exaggerated venodilation or decreased sympathetic output due to excessive induction agent. Indeed, the hyperdynamic response to intubation could be easily used to support the contention that *more* thiopental-

This combination of a relative anesthetic overdose, combined with coronary artery disease, might easily have resulted in severe myocardial ischemia, leading to circulatory collapse and ventricular fibrillation. It is possible that a more judicious induction might have avoided these problems.

Enrique Via-Reque, M.D. Central DuPage Hospital, Winfield, Illinois. viareque@aol.com

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fentanyl-lidocaine should have been used. As a final point, had the cardiac arrest been solely due to an anesthetic overdose, one would be hard pressed to explain the multiple subsequent episodes of cardiac arrest in the intensive care unit in the absence of any anesthetic agent having been administered.

Rashmi N. Mueller, M.D.* Mark H. Zornow, M.D. *University of Texas Medical Branch at Galveston, Galveston, Texas. ramuelle@utmb.edu

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Lidocaine Administration, Seizures, and Causality

To the Editor:—The case report by DeToledo *et al.*¹ contained a typographical error in the second sentence: “Serum concentrations of lidocaine below 5 mg/ml ...treat status epilepticus.” (I think 5 μ g/ml was intended—5 mg/ml must be avoided!). I further question whether this case report supports his conclusions or implies that local anesthesia be contraindicated in patients with seizures, particularly because all details were not included for his patients.

Patient no. 1 was 36 years old, diabetic, and subtherapeutic on phenytoin when she experienced a typical seizure. She was given a dose of 1,500 mg phenytoin (bound extensively to plasma proteins and with a very long half life = 22 h), which increased her serum concentration 15.5 μ g/ml. She was given a calculated total dose of 1,524.6 mg lidocaine during a period of 6 h *via* infusion in addition to 150 mg initial bolus dosing (total cumulative dose of 21.7 mg/kg). Lidocaine

redistributes rapidly, with typical terminal elimination of 90–120 min, yet it raised the blood concentration to 21 μ g/ml (sampling or administration error?). The authors also discussed active metabolites of lidocaine as possibly contributory to the grand mal seizure, but metabolism would seem very limited with these relations. Of note is the work of Knight *et al.*,² where 21 mg/kg of lidocaine was infused intravenously up to onset of cardiac bypass in less time (accompanied by 0.6 mg/kg of diazepam and no seizures) with peak levels of only 9.47 ± 1.35 μ g/ml occurring at sternotomy and decreasing to 8.4 before bypass. While diabetics are known to experience hypoglycemic seizures, glucose concentrations were not presented by these investigators. Moreover, slowly increasing concentrations of lidocaine typical of therapeutic infusions present with CNS depression, rather than the seizures associated with rapidly increasing (bolus) concentrations.³

The second patient with a history of focal and secondarily generalized seizures received 20 mg lidocaine *via* the right internal carotid during cerebral angiography (any sedation?) and developed a right hemisphere seizure, which did not generalize—what luck! I would

After numerous failed attempts to acquire a Reply to this Letter, it is being published without a response. —Michael M. Todd, Editor-in-Chief

ask if Dr. DeToledo continues to support his recent assertion, "A history of partial seizures is not a major risk factor for the precipitation of seizures in patients treated with IV lidocaine." Might this be generalized to include regional anesthetics, while perhaps adding that concomitant benzodiazepines administration greatly increases safety?

Paul Martin Kempen, M.D. Department of Anesthesiology, University of Pittsburgh, Presbyterian University Hospital, Pittsburgh, Pennsylvania. pkempe@pol.net

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Airway Fire during Tracheostomy: Should We Extubate?

To the Editor:—Airway fire is an uncommon but potentially damaging complication during tracheostomy. Although the airway fire management protocol calls for immediate removal of the endotracheal tube (ETT),¹ we may be faced with a situation of irreversible loss of the airway with removal of the ETT or further thermal or chemical damage if it is left in the trachea.²

A 54-yr-old woman with obesity (120 kg), congenital heart disease (two previous aortic valve replacements), amiodarone-induced interstitial lung disease, pulmonary hypertension, Cushing syndrome, and recurrent congestive heart failure underwent elective tracheostomy. Anesthesia was induced with 160 mg propofol, 100 µg fentanyl, 8 mg vecuronium, and air-oxygen-desflurane. She underwent ventilation *via* the existing polyvinyl chloride ETT. Oxygen, 100%, was utilized 5 min before incision of the trachea. The trachea was incised with a scalpel, and hemostasis was achieved with diathermy. Ventilation was then discontinued, the cuff of the ETT deflated, and the ETT was withdrawn until its tip was above the tracheal opening. Further diathermy to a bleeding vessel in the subcutaneous fat resulted in a 10-cm flame jetting from the incision, which was extinguished with placement of moist gauze directly over it. After ensuring that there were no further flames, the endotracheostomy tube was inserted into the trachea without incident, and there was no apparent distal airway burn injury on fiberoptic bronchoscopy. The patient had superficial burns to the skin around the tracheostomy site. After the airway was secured with the endotracheostomy tube, the extracted ETT was inspected and found to be undamaged.

This case illustrates two points regarding tracheostomies. First, use of diathermy in the proximity of a 100% oxygen gaseous environment is contraindicated. Both surgeon and anesthesiologist must be aware of this, communicate with one another, and be vigilant to prevent its

occurrence. Second, immediate extubation is not always the best response to tracheostomy fires in every circumstance. When the fire is immediately extinguished, when there is concern about potential loss of the airway, and when there is reason to believe that the ETT is not involved in the fire, then the risk-benefit analysis may favor securement of the airway as a priority, superseding removal of the ETT.

The ETT was not removed at the time because it had already been withdrawn beyond the tracheal incision before the use of electrocautery, and the site of the flame was believed to be the trachea and subcutaneous tissue. The deep tracheostomy wound in this obese patient may have contributed to formation of the fire in the fatty tissue.³ If the ETT had been on fire or if there were any doubt that the fire had not been completely extinguished, we would advocate removal of the ETT without delay. In this patient, the benefits of leaving the ETT outweighed the risk of losing the airway if the surgeon had been unable to insert the endotracheostomy tube because of extensive burn damage to the tracheal incision site. Immediate extubation during any airway fire is appropriate as a general guideline, but the importance of an individualized risk-benefit judgment in patients with potentially difficult airways is emphasized.²

Ju-Mei Ng, F.A.N.Z.C.A.,* Philip M. Hartigan, M.D. *Brigham and Women's Hospital, Boston, Massachusetts. jumei@myrealbox.com

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Failure of Paging Shortcuts to Facilitate Stat Paging in Medical Emergencies

To the Editor:—I wish to report several failures of paging shortcuts to facilitate arrival of operating room personnel in the event of unscheduled operating room emergencies. In each case, the paging shortcuts were intended to save the time required to call back and receive verbal information regarding the location and nature of the emergency. In two cases at two separate hospitals, a 4-digit code was used instead of

a call-back number to indicate that the anesthesiology staff and/or residents should report immediately to the operating room. Because not all anesthesiology personnel were aware of the adoption of the paging shortcuts, attempts were made to call the number as if it were a standard 4-digit telephone extension. When the number was discovered to be unassigned, the hospital operator was queried, who was also unaware of the significance of the 4-digit number. In a third case, the operating room personnel paged the anesthesiologists using the 4-digit

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call-back extension followed by the digits 911 to indicate an emergency. Although this is a widely understood shortcut in the United States, unintended processing by the digital pager resulted in confusion. The pages were received by a Motorola Advisor Elite pager (Motorola, Inc. Boynton Beach, Florida). When a 7-digit numerical message is entered by touchtone telephone and received by this pager, a hyphen is automatically entered between the third and fourth digits to facilitate reading of a standard 7-digit US telephone number. This made the 4-digit call-back extension and the 911 "emergency code" appear to be a standard 7-digit telephone number originating outside the hospital. The identity of the 4-digit extension was further obscured by the location of the hyphen between the digits of the intended call-back number. In contrast, manually inserting a hyphen *via* telephone with the asterisk key would properly place the hyphen between the fourth and fifth digits, eliminating the confusion. The anesthesiology personnel attempted to call the number as if it were a standard "outside line" and arrived only after a second page directly to the operating room telephone number. Although this pager can also receive alphanumeric messages sent through the Internet, these Internet-based pages may have significant delays compared with telephone pages and are therefore rarely used in emergencies.

In each case, circumventing the normal emergency response procedure

of paging to a call-back telephone number by substituting or adding informally adopted paging shortcuts resulted in failure of the intended result, which was to provide a more rapid response. One should not assume that all personnel will understand locally adopted paging shortcuts when used in emergency notification procedures unless the intended recipients are properly informed. In addition, when using one-way communication, as when using paging shortcuts, verification of information transfer does not occur as it does when a call-back number is used. Anesthesiologists may have responsibilities in several areas of a hospital. Paging shortcuts adopted by one nursing unit may not be consistent with similar shortcuts informally adopted by other units and may become a source of confusion. In addition to performing regular tests of the paging system, it may be beneficial to involve hospital-wide telecommunications personnel in paging shortcuts created by individual nursing units or adopted otherwise on an informal basis. Standardization of emergency response protocols within a hospital may reduce miscommunications.

Harvey J. Woehlck, M.D. Medical College of Wisconsin, Milwaukee, Wisconsin. hwoehlck@mcw.edu

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Alternative Methods of Orbitotracheal Intubation

To the Editor:—Sander *et al.*¹ describe fiberoptic transorbital intubation, a valuable alternative to tracheotomy in an unusual group of patients. We would like to mention that additional options exist that may be suitable for many patients in this group. On several occasions, we have intubated similar patients through the orbit and have found success using awake, direct laryngoscopy with topical anesthesia. A long, straight laryngoscope blade, such as a Miller 3 or longer blade, can be used in conjunction with an endotracheal tube with a predominantly straight stylet. Anatomic requirements for the use of direct transorbital laryngoscopy include the previous removal of the orbital floor and maxillary bones in such a way to create a straight pathway for visualization of the vocal cords. To perform direct laryngoscopy, the laryngoscope blade is first passed through the empty orbit. The laryngoscope blade is then used to sequentially elevate the soft palate, tongue, and finally the epiglottis to allow direct visualization of the

vocal cords. Although the fiberoptic technique may be successful in most situations, direct laryngoscopy as mentioned here may be advantageous when fiberoptic laryngoscopy is rendered difficult by blood or secretions or when a fiberoptic scope cannot be immediately obtained. A lighted stylet may also be useful as an alternative to fiberoptic transorbital intubation when situations render fiberoptic visualization difficult.

Harvey J. Woehlck, M.D.,* Lois A. Connolly, M.D. *Medical College of Wisconsin, Milwaukee, Wisconsin. hwoehlck@mcw.edu

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Egg Allergy and Blood Products: You Say Albumen, I Say Albumin. . .

To the Editor:—Severe hypotension developed during the administration of albumin 5% (human) after cardiac surgery in a patient known to have an allergy to eggs. Blood bank personnel questioned the advisability of giving albumin to any egg-allergic patient, and the patient's medical record was labeled with "albumin allergy." We were thus prompted to study the validity of the presumed association.

The proteins from the whites of eggs are collectively referred to as albumen.¹ Albumins are any water-soluble proteins that, like albumen, form a white coagulum when heated. Ovalbumin (45 kd), the principal

protein of egg albumen, is a different protein from human serum albumin (67 kd). A distinct lactalbumin occurs in milk, and other albumins are found in muscles and plants. A person allergic to all albumins would be hard pressed to find sustenance.

A person allergic to pure human serum albumin would also have a difficult situation. Albumin is the most abundant of the plasma proteins. However, 5% albumin is not a pure entity, and adverse reactions are reported.² Prekallikrein activator is sometimes a deleterious component, though seldom a concern in modern preparations.³ Bacterial contamination can occur, but cultures of the contaminated bottle were sterile.

Withholding serum albumin-containing products from egg-allergic patients is tantamount to withholding propofol from penicillin-allergic

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patients because of the letter “p.” Fleming, by the way, named penicillin on the hunch that the antibiotic might prove to be a *protein*. The suffix “in” is therefore carried by most antibiotics today. Thankfully, no adverse reactions coincided with administrations of vancomycin, nitroglycerin, or digoxin to our patient.

Samuel J. Smith, M.D., M.P.H.,* Dwight Geha, M.D., Theodore A. Alston, M.D., Ph.D. *Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts. sjsmith@partners.org

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