Anesthesiology 2007; 106:631

The CobraPLA Basic Design Has Been Modified to Aid Insertion

To the Editor:—We read with interest the study by van Zundert *et al.*[1] regarding performance characteristics of three disposable extraglottic airways. van Zundert found that the CobraPLA (Engineered Medical Systems, Indianapolis, IN) was more difficult to insert and caused more mucosal trauma than either the *LMA-Unique*™ (LMA North America Inc., San Diego, CA) or the Soft Seal laryngeal mask (Portex Ltd., Hythe, United Kingdom), noting that this finding is in contrast to previous studies conducted by Gaitini *et al.*[2] and Akca *et al.*[3] He attributes this difference, in part, to the fact that the patients in Akca's study were paralyzed. However, Akca's patients were not relaxed before insertion of the devices studied. As an alternative, we suggest that van Zundert's more precisely defined ease of insertion (3/2/1/0 *vs.* difficult/not difficult), along with a greater number of patients studied (105 patients *vs.* 40 patients studied each by Gaitini and Akca)[2,3] might have allowed a statistical difference to emerge. Although the CobraPLA has a flexible tip to aid insertion at the back of the throat, we believe that CobraPLA's straight breathing tube might have contributed to the insertion difficulty (fig. 1A).

As a result of our own experience using the CobraPLA in several hundred patients and at our suggestion, the manufacturer (Engineered Medical Systems) has modified the basic design of the device to incorporate a distal bend in the breathing tube on both the standard CobraPLA and the newly introduced CobraPLUS (figs. 1B and C) while leaving the other features of the airway (*e.g.*, flexible tip, circumferential cuff) unchanged. The decision to accomplish this design change was driven in part by discussions with Dr. van Zundert while his study was in progress (although actual results were not known) regarding how the device might logically be improved to aid insertion. As a result of this modification, the specific product studied by van Zundert is no longer being manufactured. We believe the curved distal end greatly facilitates insertion and minimizes trauma because it now conforms to the shape of the anatomy it must traverse, and initial reports with its use have been encouraging (Xavier Marquez, M.D., Instituto Urologico, Caracas, Venezuela, personal communication, April 2006).

The fact that the basic design of the CobraPLA studied by van Zundert is no longer being manufactured in no way diminishes the importance of his study. Rather, we believe it validates how respected researchers can help to drive product design for improved patient safety. The improved performance characteristics of the newly changed CobraPLA, require validation by additional research. We have sent the newly designed CobraPLA to Dr. van Zundert and would be interested to learn his initial impressions.

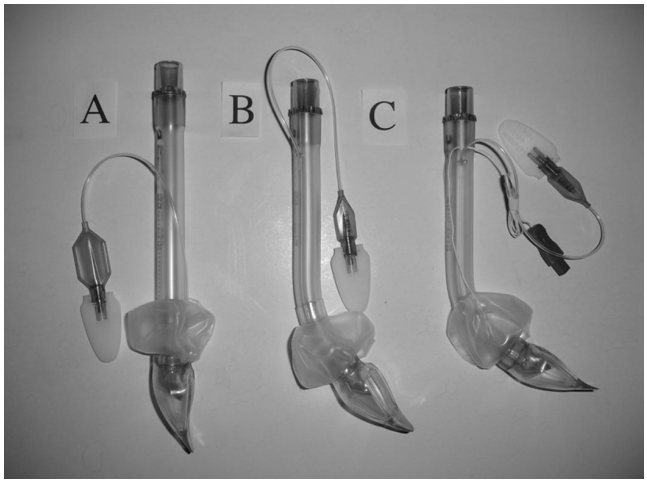

Fig. 1. (*A*) CobraPLA size 3—note straight breathing tube. (*B*) Currently produced CobraPLA size 3 with distal curve in breathing tube to aid insertion. (*C*) CobraPLUS (with temperature-monitoring thermistor on the cuff) size 3 with similar distal curve in breathing tube.

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Dr. Alfery is the inventor of the CobraPLA and the CobraPLUS (Engineered Medical Systems, Indianapolis, Indiana) and receives royalties on sales.

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In Reply:—We thank Drs. Alfery and Szmuk for their positive comments about our article.[1] In many countries, the use of extraglottic devices equals that of tracheal intubation. It is imperative that all new extraglottic devices undergo carefully conducted clinical trials to determine their safety and efficacy *versus* the current standard, the laryngeal mask airway, which has been extensively used and studied.[2] Data that has been collected about one extraglottic device need not necessarily apply to another. Our study was probably one of many factors that resulted in the decision to redesign the CobraPLA (Engineered Medical Systems Inc., Indianapolis, IN). The new version, the CobraPLUS, seems to be easier to insert and the integral temperature probe is a nice feature, but this requires confirmation. That industry responded to our article is encouraging.

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Hyperoxia to Reduce Surgical Site Infection?

To the Editor:—I read with interest the review article of Dr. Mauermann and Dr. Nemergut[1] about the anesthesiologist's role in preventing surgical site infection (SSI).

The authors deal with hypothermia and write, "The incidence of SSI was 5.8% in the normothermic group and 18.8% in the hypothermic group. The patients who developed SSIs required hospital stays nearly 1 week longer than those who did not develop a SSI, indicating that these were *clinically significant complications*" (italics added).

They also review the role of hyperoxia to reduce SSI and write, "Both of these studies found statistically significant reductions in the rates of SSIs in the 0.8 fraction of inspired oxygen (FIO_2) group *versus* the 0.3 FIO_2 group." They correctly cite the two studies[2,3] that found that 80% oxygen could halve surgical site infection *versus* 30% oxygen; they also cite another study[4] that found that 80% oxygen increased surgical site infection *versus* 35%. But surprisingly, they do not report whether the impressive reduction in SSI using 80% oxygen found in those two studies reduced clinically significant complications.

Greif *et al.*[2] report a 54% relative risk reduction of SSI using 80% *versus* 30% oxygen. However, patients who received 80% oxygen had 12.2 days of hospitalization *versus* 11.9 days among those who received 30% oxygen. Moreover, no difference was found for time to first solid food intake or staples removed.

Belda *et al.*[3] report a 39% relative risk reduction of SSI using 80% *versus* 30% oxygen. Again, consistently with the lack of clinically significant benefit, there was no difference in days of hospitalization, time to solid food intake, or staples removed.

The authors conclude that ". . . high inspired oxygen levels in the perioperative period confers some benefit in reducing the incidence of SSIs." They do not report, however, the lack of clinically significant benefit.

In contrast with these results, Pryor *et al.*[4] did find clinically significant harm among patients who received 80% oxygen (longer hospital stay, higher reoperation rate). This study, the lack of clinical benefit in the other two studies, and the inexistence of data evaluating more moderate oxygen concentrations (45–60%)[5] should prevent anesthesiologists from accepting 80% as the ideal perioperative oxygen concentration to improve surgery outcomes.

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David C. Warltier, M.D., Ph.D., served as Handling Editor for this exchange.

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In Reply:—We appreciate the interest of Dr. Tornero-Campello in the anesthesiologist's role in the prevention of surgical site infections (SSIs). Further, we note his repeated contention that the duration of hospitalization is a more appropriate outcome measure than the incidence of infection is been published in letters to multiple journals.[1–3]

Our objectives in preparing this article[4] were to produce an evidence-based, timely, concise, and clinically relevant document to educate practicing anesthesiologists regarding the best approaches to reducing surgical site infections. We believe that there is sufficient evidence to suggest that normobaric hyperoxia reduces the incidence of SSI in colorectal surgery.[5,6] Nevertheless, Dr. Tornero-Campello's focus on hospital duration of stay as an outcome measure allows us to reexamine these data in greater detail.

Dr. Tornero-Campello notes that one study[7] that included a heterogenous population of patients undergoing major abdominal surgery did not show a benefit of hyperoxia and may have been associated with an increase in morbidity and in the duration of hospitalization. It is extremely important to note that criticism of this study has been significant. For example, variables such as anesthetic technique, fluid management, and pain management were not controlled.[8,9] Information on blood glucose control, strongly associated with the incidence of SSI, was not included.[10] The small sample size and statistical analysis has not held up to rigorous *post hoc* examination.[11] Although patients were prospectively randomized to their perioperative oxygen group, the presence of SSI was determined by retrospective chart review,[8,9] as we note in our article. It is imperative that we consider these methodologic flaws when we weigh the results of this study. Last, it would seem intuitive that if hyperoxia did confer an increase risk of "clinically significant harm," some hint of this risk would have been evident in the two larger studies by Grief *et al.* [5] and Belda *et al.*[6]

Faults with the above study aside, we concede that the use of hyperoxia was not associated with a decrease in the duration of hospitalization in either of the two studies cited in our review.[5,6] Nevertheless, we reject Dr. Tornero-Campello's assertion that the prevention of SSI is only significant if it results in a tenable "difference in

days of hospitalization, time to solid food intake, or staples removed." Indeed, it is difficult for us to imagine any SSI that is not clinically significant: Even an infection that may be easily treated in the outpatient setting results in the use of antibiotics, which may further increase the prevalence of antibiotic-resistant organisms and leads to an increased cost of care.[4,12]

Although we may debate the clinical significance of hyperoxia in the prevention of SSIs, we hope Dr. Tornero-Campello will agree that the prevention of any infection, even if it not associated with an increase in duration of hospitalization, is a clinically relevant outcome and a substantial improvement in patient care.

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Use of Continuous Positive Airway Pressure in Anesthetized Infants

To the Editor:—The suggestion has been made that "The application of CPAP [continuous positive airway pressure] . . . may be crucial to help maintain airway patency in anesthetized infants."[1] Before this suggestion can be applied, there is additional crucial information that must be obtained. The effect of CPAP on respiratory control in the awake state may be quite different than that in the anesthetized state. As the authors have pointed out, infants are dependent on neural input for airway maintenance. However, no data have been presented about the effect of loss of neural input (*i.e.*, the anesthetized state) and its effect on control of respiration in the presence of CPAP. End-tidal carbon dioxide increased from 41 to 46 mmHg in the study with increasing depth of propofol anesthesia, with "no further change resulting from the application of CPAP." If ventilation is depressed and there is an increase in dead space ventilation (V_D/V_T), due to decreased tidal volume, end-tidal carbon dioxide may not change when in fact arterial carbon dioxide tension is increasing. One can see this unchanged or decreased end-tidal carbon dioxide when CPAP is applied to a spontaneously breathing patient anesthetized with an inhalation agent. In the exhaled breaths after the release of CPAP applied for only a minute or so, there is a large outpouring of carbon dioxide. Studies of CPAP in spontaneously breathing infants should include data on arterial carbon dioxide tension.

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In Reply:—We thank Dr. Rothstein for his interesting comments regarding our article.[1] As he correctly points out, the effect of added elastic and/or resistive load on respiratory drive may differ in the awake and anesthetized states. In the awake state, an increase in elastic and/or resistive load is compensated by an increase in neuronal inspiratory drive. This compensatory neuronal drive may be absent or diminished during general anesthesia,[2] and this may be particularly so in infants who are highly susceptible to anesthesia-induced attenuation of neuronal input. Continuous positive airway pressure (CPAP) may itself reduce inspiratory drive through the Herring-Breuer inflation reflex.[3] The resulting hypercapnia will depend on the reduction in minute alveolar ventilation. In previous studies in anesthetized children, the impact of CPAP on minute ventilation has been clinically insignificant.[4] Keidan *et al.* studied the effect of CPAP (6 cm H_2O) on work of breathing and respiratory indices in healthy spontaneously breathing children (median age, 1.0 yr) during halothane–nitrous oxide anesthesia.[4] Application of CPAP *via* a facemask significantly decreased the work of breathing but had no significant effect on inspiratory tidal volume, inspiratory minute volume, or end-tidal carbon dioxide tension. To the extent that CPAP relieves existing upper airway narrowing, CPAP can also improve gas exchange, resulting in a reduction of hypercapnia and improved oxygenation. It was the purpose of our study to determine the interaction of propofol anesthesia and CPAP on upper airway caliber and configuration in infants. We

considered it neither relevant to the study hypothesis nor ethically justifiable to perform arterial puncture for blood gas analysis in our infant subjects. Given the relatively brief duration of CPAP application, any increase in arterial carbon dioxide tension was likely small; indeed, after removal of CPAP, we observed no outpouring of carbon dioxide as determined by end-tidal measurement.

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Lipid Emulsion for Bupivacaine Toxicity: Too Soon to Celebrate?

To the Editor:—I read with great interest the case report by Rosenblatt *et al.*[1] and the accompanying editorial by Weinberg,[2] which appeared in the July 2006 issue of ANESTHESIOLOGY. The authors, especially Dr. Weinberg, exhibit considerable enthusiasm about the use of lipid emulsion during resuscitation of a patient with assumed bupivacaine-induced toxicity. Indeed, the patient recovered fully after a series of pulseless rhythms, no small triumph considering the difficulties surrounding this untoward occurrence in the operating room. Dr. Weinberg's laboratory research supports the continued clinical investigation of lipid emulsion therapy in this setting. However, I do have reservations about the proof of its efficacy presented in this case, and would like to offer an alternative interpretation below. In addition, the anesthesia team could have avoided pitfalls that may have increased risk to the patient.

The anesthesia preoperative assessment established that the patient had ischemic heart disease, possibly unstable, an abnormal electrocardiogram, and no recent cardiac studies. I am curious as to why it was decided to proceed without obtaining a more informative evaluation including a pharmacologic stress test to determine the adequacy of left ventricular function and the absence of regional wall motion abnormalities. Depending on the results of the testing, this elective surgery could have been postponed pending further medical management of the patient or cancelled because of unacceptably high risk. The patient's refusal of further study and the cardiologist's oversimplified consultation should not have compromised good medical care.

The amount of local anesthetic administered to the patient for interscalene block, although acceptable according to the patient's weight, was probably high considering his heart disease. As the authors discuss, he may have been more sensitive to bupivacaine's direct toxic effects on the heart. The mechanism is not completely understood, but inhibition of adenosine triphosphate–sensitive inwardly rectifying potassium channels has been demonstrated in myocardial cell cultures.[3] I would have chosen 0.25% bupivacaine alone or mixed with 1.5% mepivacaine, which should provide satisfactory postoperative analgesia. Although controversial because of the presence of significant heart disease in the patient, the addition of a small amount of epinephrine to the local anesthetic, whatever the choice, may have allowed for earlier detection of intravascular injection and reduced toxicity effects from lower peak bupivacaine blood levels. An argument could be made to use a higher concentration of bupivacaine to anesthetize the anterior shoulder if regional anesthesia only is considered, but the authors do not reveal whether the latter was intended or general anesthesia was also planned.

The onset of central nervous system excitability observed in this patient is usually an early sign of systemic local anesthetic toxicity, and may herald cardiovascular collapse secondary to effects on cardiorespiratory control processes in the brainstem.[4] Propofol was administered on two occasions to suppress seizures in this patient, and I believe this added further negative inotropic, chronotropic, and dromotropic injuries. It has been associated with episodes of bradycardia, second-degree atrioventricular block, and asystole resulting from muscarinic receptor stimulation.[5] Propofol has been shown to inhibit L-type calcium channels in ventricular sarcolemmal preparations, which may explain its negative inotropic effect.[6] Bupivacaine and propofol likely had additive if not synergistic myocardial depressant effects in this case. Avoidance of this combination, especially in a patient with heart disease, seems warranted. Additional midazolam and possibly lipid emulsion may be better choices for treating local anesthetic toxicity in this scenario.

The return of normal sinus rhythm occurred after more than 20 min of cardiopulmonary resuscitation and the administration of multiple antidysrhythmic drugs before lipid emulsion therapy was considered. It is possible that the effects from these other drugs and the spontaneous elimination of local anesthetic from central nervous system and cardiac tissues contributed appreciably to the patient's recovery. Administration of lipid emulsion in the presence of measurable blood levels of local anesthetic would have given more credence to the conclusions drawn. While cautiously optimistic, I am undecided about the effectiveness of lipid emulsion for the treatment of systemic bupivacaine toxicity based on the results of this one case.

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Advanced Cardiac Life Support for Presumed Bupivacaine-related Cardiac Arrest

To the Editor:—I read with interest the case report of Dr. Rosenblatt *et al.*[1] about a presumed bupivacaine-related cardiac arrest after the injection of 40 ml local anesthetic solution for interscalene brachial plexus blockade.

The authors write that "The electrocardiogram showed asystole . . ."; subsequently, tracheal intubation was performed, and during 20 min of cardiac life support, 3 mg epinephrine, 2 mg atropine, 300 mg amiodarone, and 40 U arginine vasopressin were administered. They also used monophasic defibrillation at escalating energy levels of 200, 300, 360, and 360 J. According to the text, "Cardiac rhythms included ventricular tachycardia with a pulse, pulseless ventricular tachycardia that momentarily became ventricular fibrillation, and eventually asystole. The arrhythmias observed during most of the resuscitation period were pulseless ventricular tachycardia and asystole."

Current guidelines for the use of monophasic defibrillation recommend the use of 360 J for the initial and subsequent shocks, because of the lower efficacy of this waveform[2] (if compared with biphasic defibrillation); the authors used 200 J.

The arrhythmias most often observed were pulseless ventricular tachycardia and asystole, the latter being a "nonshockable rhythm." Moreover, current guidelines explicitly recommend not to defibrillate if there is doubt about whether the rhythm is asystole or fine ventricular fibrillation.[3] It is also recommended to use a single shock strategy followed by immediate resumption of chest compressions.[4] However, the authors report having used repeated attempts to defibrillate the patient, even after 20 min of cardiac arrest; did they attempt to defibrillate asystole?

The cardiac rhythm fortunately returned to sinus after a lipid emulsion was given intravenously, and in the end, the patient had no neurologic sequelae, but was the advanced cardiac support optimum?

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Anesthesiology 2007; 106:635–6

Lipid Infusion for Cardiotoxicity: Promise? Yes—Panacea? Not

To the Editor:—Dr. Rosenblatt and her Mount Sinai colleagues,[1] along with Doctor Weinberg and his companion editorial,[2] deserve the specialty's heartfelt gratitude for reporting a definitive treatment of the dreaded enigma of resuscitation from (racemic) bupivacaine–induced cardiotoxicity. It is a vexing problem that, heretofore, called for prolonged resuscitation at best and heroic measures up to, and including, cardiopulmonary bypass at worst. None of these measures ever could assure resumption of spontaneous heartbeat—let alone restoration of normal brain function. All of that may have become a nightmare of the past, now that the experimentally promising infusion of lipid emulsion[3] has borne fruit in reversing clinical cardiac asystole promptly and completely.[1] As the editorial[2] states, lipid infusion well may prove to be a watershed antidote for this fearsome local anesthetic complication.

The bone I have to pick here is the unfortunate inference that the undoubted success of lipid infusion in reversing cardiac arrest imputes its corresponding effectiveness in treating any and all local anesthetic–induced toxicity, starting with the editorial's seductive title (". . . Resuscitation for *Local Anesthetic Toxicity*" [italics mine]) and ending with the suggestion that lipid emulsion be stored routinely wherever peripheral nerve blocks are performed. The editorial's title, regrettably, restates the original report's misleading overgeneralization (". . . Resuscitation for *Local Anesthetic Toxicity*" [italics mine]). My comments are by no means intended to belittle the authors' contribution, but rather to constrain lipid infusion to its proven limits as a treatment option for racemic bupivacaine–induced cardiac asystole.

Lipid infusion well may prove to be the silver bullet for treating racemic bupivacaine–induced asystole, but it by no means has been demonstrated (as yet) to be effective in treating local anesthetic–induced cardiotoxicity in general, and even less so in treating local anesthetic cerebrotoxicity. Rather, lipid infusion has been shown to be effective only in reversing racemic bupivacaine–induced asystole.[3] That is to say, unintended cardiotoxicity caused by any local anesthetic other than racemic bupivacaine probably is better treated by conventional advanced cardiac life support resuscitation. Witness, for example, recent reports of conventional methods in restoring cardiac function after cardiotoxicity from closely related local anesthetics such as levobupivacaine[4] or ropivacaine.[5] More to the point yet: Lipid emulsion infusion hardly is a panacea for treating the noncardiac (and far more common) manifestations of local anesthetic toxicity such as convulsions from cerebrotoxicity. Moreover, restoration of normal sinus rhythm does not necessarily imply restoration of normal cerebral function. Quite the contrary, permanent brain damage (underreported because of court-imposed constraints) may be the all-too-common heartbreaking outcome.

To take the authors' words truly to heart, turn around their conclusion instead, and question whether racemic bupivacaine (and its lipid emulsion antidote) still warrant a place in ". . . areas in which peripheral nerve blocks are being performed." Rather, one might just as well consider a moratorium on the use of bupivacaine and avoid this dreadful complication (and its unconventional treatment) altogether—using more heart-kind, equally long-lasting local anesthetics such as levobupivacaine or ropivacaine.

With safer monomeric alternatives to racemic bupivacaine (now irreverently dubbed "retro-bupivacaine") available, hasn't the time arrived to retire this notoriously hazardous local anesthetic that by now has accumulated more than 30 yr of evidence of disproportionate cardiotoxicity? All that racemic bupivacaine has going for it, in truth, is the low price of a generic drug. No question but that drug cost is a substantial practice expense—still, patient safety always must be the overriding consideration.

In the flush of exciting news, it is all too convenient to ignore the dark side of this case report. The stark truth remains that, even in skilled hands, racemic bupivacaine is an unpredictably cardiotoxic local anesthetic that—perhaps—should be retired from practice altogether. It is a bit like promoting a surefire cure for lung cancer, rather than snuffing out cigarette smoking in the first instance, or not belting up because your car has the latest inflatable device.

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Lipid Rescue from Bupivacaine Cardiac Arrest: A Result of Failure to Ventilate and Maintain Cardiac Perfusion?

To the Editor:—Weinberg[1] is correct: ". . . lipid infusion should be used, as in this case report,[2] only after standard resuscitative measures have proven ineffective." In that case, effective ventilation with 100% oxygen and maintenance of cardiac perfusion did not occur for at least 4½ min, *i.e.*, "90s" from first seizure to second seizure, and a conservative estimate of 3 min from it to the establishment of ventilation *via* intubation of the trachea.* "Oxygen . . . delivered by a facemask attached to a self-inflating resuscitation bag "[2] will not reverse the severe respiratory acidosis, which occurs within 30 s after tonic–clonic seizures.[3,4]

Almost a half century ago (1960),[5] we reported 112 "Severe Systemic Reactions (Respiratory Arrest, Convulsions, Cardiovascular Collapse)" in 36,113 patients from local anesthetics (amino-esters and -amides) without mortality or morbidity. From the study, we postulated† that (1) with the onset of tonic–clonic seizures, severe respiratory acidosis occurred simultaneously, *i.e.*, within seconds; and (2) effective oxygen therapy and maintenance of cardiac perfusion was the "antidote" to avoid severe, permanent complications from local anesthetics. This "antidote,"[5] when effectively executed, has avoided the "antidote" stated by Weinberg.[1]

In 1978,[6] the administration of bupivacaine in 11,080 patients from its first phase III (clinical) study for the US Food and Drug Administration[7] was reported. Twelve of the patients had tonic–clonic seizures. Using the previously postulated treatment, none resulted in morbidity or mortality.

In 1980 and 1982, we clinically verified the postulate.[3,4] And, in 1983, it reversed two cardiac arrests (one in a parturient in labor) from bupivacaine without complications.[8]

To conclude, paraphrasing Weinberg[1]: Lamentably, it is clear from 91 responding academic anesthesiology departments that there is little uniformity in planning for this potentially catastrophic complication. Perhaps the protocol when administering a regional block that follows and has avoided morbidity and mortality from seizures might help to solve this problem.[3–8]

First, before executing a regional block, monitoring is the same as if intravenous or inhalation anesthesia is being administered.

Second, drugs for resuscitation are in syringes. And, they and equipment (anesthesia machine, endotracheal tubes, *etc.*) are immediately available (within arm's reach), not in drawers, on shelves, or down the hall.

Third, immediately when seizures are imminent (patient become incoherent, loses consciousness) or start, ventilation with 100% oxygen is begun. When they start, whether intubation should occur is debatable. If ventilation *via* an oral airway is unobstructed, attempting to do so could interrupt ventilation for a significant period of time and precipitate cardiac arrest.

Fourth, when the heart rate decreases to 30 beats/min in the nonathlete, 1:1,000 epinephrine in 0.3- to 0.5-ml increments is administered to increase heart rate to 60 or more beats/min. When the rate does not respond or decreases to 25 or fewer beats/min, cardiac compression is started.

Last, when cardiac arrest occurs, Advanced Cardiac Life Support as noted by Rosenblatt *et al.*[2] is performed. And, henceforth because of their reported case, ". . . lipid rescue should be considered before ceasing resuscitative efforts even if its use is contemplated after a significant delay in the setting of prolonged cardiac arrest."[1]

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* This assumes that "standard monitors"[2] included the electrocardiographs were attached before executing the block and that drugs (excluding the lipid) and equipment were prepared for immediate use.

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In Reply:—I am gratified by the response to the recent case report[1] and accompanying editorial[2] on the successful use of lipid in treating local anesthetic cardiac toxicity. Two of the letters are by authors whose work each occupies substantial space on my bookshelf. I appreciate the support for the lipid method that Dr. Moore showed by adding it to his proposed protocol for treating local anesthetic systemic toxicity.

I agree with Dr. Shupak that propofol is not a good choice for treating patients with local anesthetic–induced toxicity. This was not always my opinion, and a review I wrote on the topic[3] might have lead Rosenblatt's team to use propofol for seizure suppression—*mea culpa*. However, given (1) the potential for rapid and unpredictable progression to cardiac depression shortly after central nervous system symptoms, (2) overwhelming evidence of the cardiac suppressive effects of propofol, and (3) the commonly held, but incorrect, belief that propofol and 20% lipid are interchangeable, I believe that propofol should be removed from any such protocol and considered *contraindicated* in treatment of local anesthetic toxicity.

I caution Dr. Shupak against writing statements that begin with "I would have chosen . . ." as it rarely looks good to question what someone did *in extremis*. Dr. Rosenblatt and associates did not have the advantage of hindsight but performed admirably in saving the patient's life. Writing honestly about such an experience vicariously enriches our collective clinical wisdom.

Dr. Shupak points out that the patient's recovery at the same time as the lipid infusion might not have been causally related, although he remains "cautiously optimistic." However, I have a distinct advantage. My "considerable enthusiasm" is based on having performed many dozens of experiments over several years in several animal models of bupivacaine toxicity in which lipid failed to resuscitate only twice (arterial pressure and electrocardiographic traces of typical experiments can be viewed at www.lipidrescue.org). Dr. Shupak also wrote that he is "undecided about the effectiveness of lipid . . ." Given the second case report by Litz *et al.*, I consider it unwise to withhold lipid infusion for a patient unresponsive to standard resuscitative measures. Saying "there's not enough evidence" seems too harsh a sentence for the suffering patient.

Dr. de Jong's question of general applicability is partially answered by Dr. Rosenblatt's case (asystole after combined bupivacaine *and* mepivacaine) and the recent report by Litz *et al.*[4,5] of the successful use of lipid in resuscitating a patient in ropivacaine-induced asystole. It is not known whether lipid infusion could benefit a patient with purely central nervous system symptoms of overdose; we wait for laboratory studies or, possibly, case reports.

Dr. de Jong also cites case reports of successful resuscitation with standard methods for cardiac arrest after ropivacaine and levobupivacaine as support for an argument to avoid bupivacaine in favor of the other agents. I believe these cases serve as reminders that use of all local anesthetics carries risk. Knowing they exist, I wonder how often such cases go unreported when the resuscitation is not successful. Accurate numerators and denominators for critical events during regional anesthesia with specific local anesthetics are not available. However, a recent survey by Corcoran *et al.*[6] shows that bupivacaine surpasses all other local anesthetics as the preferred agent when prolonged anesthesia is required. Lacking epidemiologic evidence supporting a clear safety advantage of alternatives, I think it is unlikely that bupivacaine will be retired from use.

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Anesthesiology 2007; 106:637–8

In Reply:—We appreciate the interest that our case report[1] and the accompanying editorial[2] have generated. Before we address the points raised in the letters to the editor, we would call to the readers' attention a case report by Litz *et al.*[3] that was published 1 month after ours. They describe a patient with ropivacaine-induced asystole after an axillary block who was successfully resuscitated after the infusion of 20% lipid. Their patient was an 84-yr-old, 50-kg woman who received 40 ml ropivacaine, 1%, secondary to a miscommunication. After a tonic–clonic seizure that was treated with thiopental, she experienced ventricular extrasystoles, followed by bradycardia and asystole. While cardiopulmonary resuscitation was being performed, she was given 20% intralipid at 2 ml/kg, followed by a continuous infusion of intralipid at a rate of 10 ml/min. After she had received a total intralipid dose of 4 ml/kg, wide complex tachyarrhythmia was observed, and her blood pressure was restored. This is the first report of intralipid reversing the toxic effects of a monomeric local anesthetic.

In response to Dr. Shupak's letter, although our patient had docu-

mented ischemic heart disease and previous coronary bypass graft surgery, he was on maximal medical therapy and had refused further diagnostic and surgical interventions. His shoulder was causing him considerable discomfort. We considered that the planned shoulder arthroscopy presented a low risk for cardiac events and that his informed refusal to subject himself to further workup should not be a contraindication. After the event, our patient did consent to a cardiac catheterization. This revealed no bypassable disease, normal left ventricular end-diastolic pressure, and moderate left ventricular dysfunction. Like our patient, the patient reported by Litz *et al.* had underlying cardiac disease that included a mild form of Morgagni-Adams-Stokes syndrome, left bundle-branch block, and grade II mitral and tricuspid regurgitation. We concur with Dr. Shupak that our patient's underlying cardiac disease may have made him more susceptible to the cardiotoxic effects of bupivacaine, but our intention was to avoid general anesthesia. Bupivacaine, 0.5%, was chosen because it provides superior surgical anesthesia and longer postoperative analgesia.

Dr. Tornero-Campello and Dr. Moore raised concerns about the sequence and efficacy of events during the cardiopulmonary resuscitation. This case occurred only days before the November 28, 2005, on-line publication of the updated advanced cardiac life support guidelines that subsequently were published in the December 13, 2006, supplement to *Circulation*.[4] We therefore were in compliance with then applicable guidelines in the use of defibrillation energies. The patient had been receiving supplemental oxygen at 3 l/min before the commencement of the block. As soon as seizure activity was detected, oxygen was delivered from a facemask connected to a self-inflating resuscitation bag. This was continued through the subsequent seizures, until the trachea had been intubated. We therefore disagree with Dr. Moore's suggestion that hypoxia contributed significantly to the prolongation of the seizure activity. We reiterate that after this entire episode, our patient sustained no permanent neurologic sequelae.

We concur with Dr. de Jong that lipid emulsion is not a panacea for treating the common and noncardiac manifestations of local anesthetic toxicity, but we respectfully disagree with his assertions that bupivacaine is an antiquated agent. Introduced into clinical practice in 1963, bupivacaine has been used to provide superb-quality analgesia and analgesia countless times, and without event. It was not until 16 yr after its introduction that attention was called to its potential cardiotoxicity.[5] Ropivacaine is a substantially more expensive agent that is also cardiotoxic[6,7] and is still in the infancy of its use. Questions about its superiority to bupivacaine when used in equipotent doses remain unanswered. Rather than abandoning bupivacaine altogether, as Dr. de Jong suggests, we propose that all practitioners of regional anesthesia become familiar with the use of 20% lipid. To this end, we encourage physicians to visit a relatively new Web site dedicated to providing this potentially life-saving information.*

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Anesthesiology 2007; 106:638

War Produces Anesthesiologists

To the Editor:—The main message and demonstration of the excellent article by Martin *et al.*[1] is that World War II created the need for, and the rapid production of, anesthesiologists. From my perspective, as someone who became an anesthesiologist because of the Vietnam War, the same is true of the Vietnam War both in concept and in the process details that were so well described by Martin *et al.* At the height of the Vietnam War in 1968–1969, there were great numbers of soldiers with multiple fragment wounds due to rocket-propelled grenades, and there was a need for medical doctors to staff the anesthesia and orthopedic departments of forward-placed MASH units. The US Army offered graduating interns, who for one reason or another were not going straight into a residency (called a Berry deferment), a 2-yr on-the-job training (OJT) assignment in anesthesiology or orthopedics rather than just routinely becoming a general medical officer. The anesthesiology OJT tour of duty started out with being assigned to a stateside Army hospital and being taught/trained by a board-eligible or -certified anesthesiologist for approximately 3 months (a short, intense 90-day course in anesthesiology). At this juncture, many/most OJTs were assigned to positions in forward-placed MASH units in Vietnam for 1 yr, and these OJTs practiced almost exclusively emergency/trauma anesthesia. The remainder of the 2-yr obligation (6 months) was fulfilled by practicing anesthesia once again in a stateside Army hospital. It is my impression that many/most of the medical doctors who went through the OJT experience in anesthesiology in 1968–1969 then went on to take formal residencies in anesthesiology, and presumably many became board-certified anesthesiologists. Therefore, with respect to the anesthesiology "Short Course," the teachers of the course, and the eventful professional outcome (board eligibility/certification) for the doctors who went through the program, I think the Vietnam War OJT anesthesia program was quite similar to the World War II anesthesia program.

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Anesthesiology 2007; 106:639

World War II Short Course: A Personal View

To the Editor:—I read with interest the article by my colleague on the World War II short course on anesthesiology and its impact on the specialty of anesthesiology.[1] The authors documented far-reaching effects of 6-month anesthesiology short courses set up for the military during World War II. One short course graduate was my father, Frank Leo Faust, M.D. He had been called from his surgical residency to active duty in the Navy the day after the bombing of Pearl Harbor in 1941. Three years later, additional volunteers were sought from military physicians for the short courses in anesthesiology. It was anticipated that a great number of wounded would need surgery during the planned invasion on the beaches of Japan. His short course took place at Lahey Clinic during late 1944.

Although it would not be expected that a 6-month trainee might make "academic" contributions, he published an article in ANESTHESIOLOGY on a series of repeated sympathetic blocks he performed on 40 veterans at the US Naval Hospital in New Orleans, Louisiana, after the war.[2] In contrast to modern publication styles, one figure in that article is a pen and ink anatomical "cartoon" drawing signed by the author himself.

After discharge from the Navy, he entered private practice in New Orleans. In the early years, he actually carried his anesthesia machine in his trunk when called to do cases at some hospitals. He introduced regional anesthesia techniques he had learned in Boston into anesthesia practice in New Orleans. Obtaining American Board of Anesthesiology certification in 1950, he practiced in New Orleans for 34 yr and resides there now at age 91.

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In Reply:—We thank Drs. Benumof and Faust for sharing their stories in regard to our article.[1] These stories demonstrate the important impact that war has had on the specialty of anesthesiology. It is important to record these observations before they are lost to history. The repeat of the short course strategy in Vietnam proves that the idea was well conceived and answered the Armed Forces' need.

David P. Martin, M.D., Ph.D.,* Christopher M. Burkle, M.D., Brian P. McGlinch, M.D., Mary E. Warner, M.D., Alan D. Sessler, M.D., Douglas R. Bacon, M.D., M.A. *Mayo Clinic College of Medicine, Rochester, Minnesota. martin.david@mayo.edu

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Cable Trapped Under Dräger Fabius Automatic Pressure Limiting Valve Causes Inability to Ventilate

To the Editor:—I would like to report a problem with the Dräger Fabius anesthesia machine (Telford, PA) that caused the inability to ventilate. After the inhalational induction of a 2,100-g infant presenting for abdominal surgery, a muscle relaxant was given to facilitate intubation. As paralysis developed, a large circuit leak was discovered, making manual ventilation impossible. The machine had passed its preoperative checkout, and a further rapid check of the circuit did not uncover any disconnections, breaks in the circuit, or obvious explanation for the inability to generate positive pressure. During this check, the automatic pressure limiting (APL) knob was rotated back and forth through its range several times, but this did not correct the inability to ventilate. A self-inflating ventilation bag was used to ventilate the patient while we continued to troubleshoot the system. It was discovered that the temperature monitoring cable had become trapped between the knob and the base of the APL Valve (fig. 1A).

During normal operation, the APL dial of the Dräger Fabius anesthesia machine is lifted 4 mm from the base into the "open" position, releasing any positive pressure within the circuit. Closure of the APL Valve requires

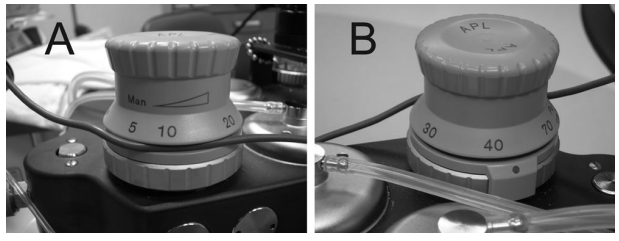

Fig. 1. (*A*) Rear view, recreated. Temperature cable trapped between knob and base of the automatic pressure limiting valve. (*B*) Front view, recreated. Automatic pressure limiting knob shields view of cable trapped beneath knob. Elevation of knob is subtle and easily missed.

turning the control knob in a clockwise direction and descent of the knob onto its base, to generate positive pressure within the circuit. If the knob is manually lifted off its base, or prevented from descending by a foreign object, the APL Valve reverts to the "open" position and positive pressure cannot be generated, regardless of rotation of the knob.

Patient monitor cables are often run behind the carbon dioxide absorber arm to keep them free of the breathing circuit, placing them to the rear of the APL. In this position, the APL screens the cables from the view of the anesthetist (fig. 1B). Elevation of the knob is subtle and easily missed during a cursory inspection of the APL Valve. Merely rotating the APL Valve is not sufficient to free a cable trapped beneath.

This cause of APL Valve failure could easily be corrected by adding a skirt or lip to the APL knob extending over the base of the valve to prevent foreign objects from becoming wedged between the knob and the base. Anesthetists who work with the Dräger Fabius anesthesia machine should be aware of this potential problem and closely examine the APL Valve in the event of inability to generate positive pressure.

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In Reply:—The automatic pressure liming (APL) valve used on the Fabius GS Anesthesia Machine (Dräger Medical, Inc., Telford, PA) has two functions: (1) to limit the maximum pressure during manual ventilation and (2) to exhaust excess gas into the scavenger system during manual and spontaneous ventilation. Pulling up on the knob quickly releases pressure when in the manual ventilation mode.

A line, cable, or other material caught underneath the APL Valve adjustment knob could, depending on the thickness of the object, interfere with the proper functioning of the valve and may affect manual ventilation. It will not affect spontaneous or automatic ventilation. The Fabius GS will not pass the preuse leak test if an object is caught underneath the APL Valve adjustment knob. The author of this correspondence states that the device had passed the preoperative checkout. This indicates that the temperature-monitoring cable became caught underneath the APL Valve adjustment knob at some point between the preoperative check and induction.

The reported incident emphasizes the challenges surrounding the increased number of cables and hoses associated with patient monitoring in the operating room environment. The Fabius GS Operator's Instruction Manual contains a warning to route all lines and cables away from the APL Valve to prevent interference with the APL Valve adjustment knob. The breathing system mounting arm provides an area underneath the arm for routing lines and cables connecting to the breathing system. Dräger Medical, Inc. also offers a boom arm as an accessory that can assist the user in cable management. Device users should perform the daily and preuse checklist to confirm the proper functioning of the device before use and follow all warnings and cautions outlined in the Operator's Instruction Manual, before and during use.

Dräger Medical, Inc. is committed to providing reliable, quality products and appreciates this matter being brought to our attention. This type of information is valuable when evaluating potential enhancements to our products.

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Improving Intubation Success Using the CTrach Laryngeal Mask Airway™

To the Editor:—The CTrach Laryngeal Mask Airway™ (LMA North America, Inc., San Diego, CA) allows for visualization of the glottis before intubation, as well as concurrent patient ventilation.[1] However, we have found that even after the administration of glycopyrrolate, the use of antifog liquid, or placing the unit in warm water, the view port either fogs or is obscured by either oropharyngeal secretions or the lubricating gel in the endotracheal tube, should the first intubation attempt be unsuccessful. A simple solution to this problem is to use a hemostat or similar device to advance a disposable sponge swab moistened with warm normal saline (*e.g.*, Item 6075; Sage Products, Inc., Cary, IL) through the CTrach™ to clean the viewing port. We have now used this technique in more than 10 instances, with uniformly excellent results (fig. 1).

Marco A. Maurtua, M.D.,* Delia B. Maurtua, M.D., Andrew Zura, M.D., D. John Doyle, M.D., Ph.D. *Cleveland Clinic, Cleveland, Ohio. maurtum@ccf.org

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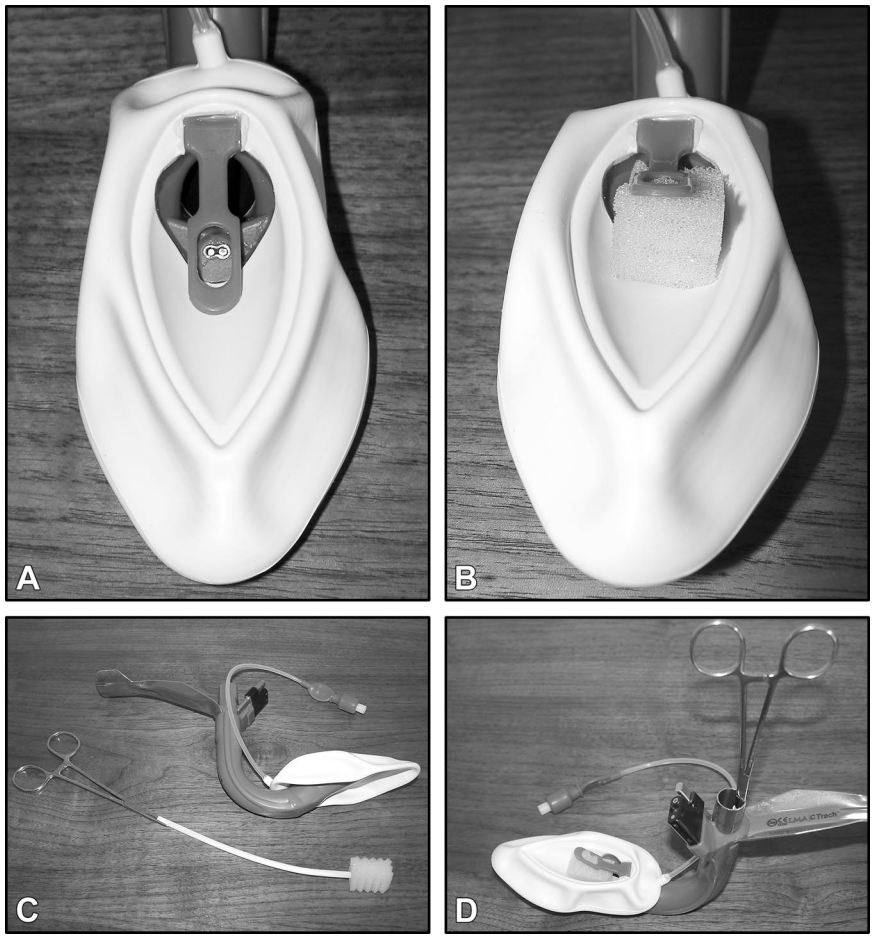

Fig. 1. ***A*** **and** ***B*** **show the CTrach Laryngeal Mask Airway™ with and without optic lens protective sponge swab.** ***C*** **shows the hemostat and protective sponge swab and side view of the CTrach Laryngeal Mask Airway™.** ***D*** **shows the protective sponge swab inserted in the CTrach Laryngeal Mask Airway™.**