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 device (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.

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

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.

References


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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.
To the Editor—I read with interest the review article of Dr. Mauermann and Dr. Nemergut 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 (FiO2) group versus the 0.3 FiO2 group.” They correctly cite the two studies that found that 80% oxygen could halve surgical site infection versus 30% oxygen; they also cite another study 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. 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. 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. 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%) should prevent anesthesiologists from accepting 80% as the ideal perioperative oxygen concentration to improve surgery outcomes.

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References

1. Mauermann WJ, Nemergut EC: The anesthesiologist’s role in the prevention of surgical site infection. ANESTHESIOLOGY 2006; 105:413–21

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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 hypoxemia 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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References

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,2 with “no further change resulting from the application of CPAP.” If ventilation is depressed and there is an increase in dead space ventilation (Vd/Vt), 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.

Peter Rothstein, M.D., Columbia University, New York. ptr1@columbia.edu

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,2 with “no further change resulting from the application of CPAP.” If ventilation is depressed and there is an increase in dead space ventilation (Vd/Vt), 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 H2O) 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

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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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References

(Accepted for publication November 15, 2006.)
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 waveform2 (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 infusion to its proven limits as a treatment option for racemic bupivacaine–induced asystole, but it by no means has been belittle the authors’ contribution, but rather to constrain lipid infusion hardly is a panacea for treating the noncardiac (and far more common) manifestations of local anesthetic toxicity such as convulsions from cerebrotoxicity. Rather, lipid infusion has been shown to improve outcomes in cases of severe local anesthetic–induced cardiotoxicity in general, and even less so in treating local anesthetic–induced cardiac arrest.5 It is also recommended to use a single shock strategy followed by immediate resumption of chest compressions.6 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 infusion was given intravenously, and in the end, the patient had no neurologic sequelae, but was the advanced cardiac support optimum?

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References


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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 heart-beat—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 emulsion3 has borne fruit in reversing racemic bupivacaine–induced 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.4 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 levobupivacaine4 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 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.”

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References


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References


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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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References


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

To the Editor.—Weinberg¹ is correct: “. . . lipid infusion should be used, as in this case report, 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 . . . will not reverse the severe respiratory acidosis, which occurs within 30 s after tonic-clonic seizures.”²,³

Almost a half century ago (1960),¹ 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,”⁴ when effectively executed, has avoided the “antidote” stated by Weinberg.¹

In 1978,⁵ the administration of bupivacaine in 11,080 patients from its first phase III (clinical) study for the US Food and Drug Administration 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.²,³ And, in 1983, it reversed two cardiac arrests (one in a parturient in labor) from bupivacaine without complications.⁶

To conclude, paraphrasing Weinberg¹: 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.⁵–⁸

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 tube, 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 non-athlete, 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 ceased resuscitative efforts even if its use is contemplated after a significant delay in the setting of prolonged cardiac arrest.”⁴

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References


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In Reply—I am gratified by the response to the recent case report and accompanying editorial 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. Rosenblatt 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 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 a patient after a presumed bupivacaine-related cardiac arrest. ANESTHESIOLOGY 2006; 105:217-8

In Reply—We appreciate the interest that our case report and the accompanying editorial 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. 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-

References

5. Weinberg GL: In defence of lipid resuscitation. Anesthesiology 2006; 105:807-8

Correspondence


(Accepted for publication November 15, 2006.)

Guy L. Weinberg, M.D., University of Illinois at Chicago, Chicago, Illinois. guyw@uic.edu
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. Torneros-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.3 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 cardiotoxic6,7 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.8

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References
5. Albright GA. Cardiac arrest following regional anesthesia with etidocaine or bupivacaine. Anesthesiology 1979; 51:285–7


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To the Editor—The main message and demonstration of the excellent article by Martin et al. 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 tours 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 OJT practices 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 anesthesia, and presumably many became board-certified anesthesiologists. Therefore, with respect to the anesthesiology “Short Course,” the teachers of the course, and the eventual 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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Reference

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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. 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. In contrast to modern publication styles, one figure in that article is a pen and ink anatomical “cartoon” drawing signed by the author himself.

To the Editor— I would like to report a problem with the Draeger 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 beneath 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...
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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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. 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).

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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™.