duced postoperative fatigue, heightened feeling of well-being, and better maintenance of homeostasis.[6–8] Moreover, clinically significant carbon dioxide embolism is rare (0.001%) during laparoscopic procedures[9,10] unlike PFO whose incidence is relatively high. Therefore, we believe that PFO cannot be a ground for eliminating laparoscopic surgery from possible surgical treatments.

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(Accepted for publication October 28, 2009.)

Anesthetic Effects and Lipid Resuscitation Protocols

To the Editor:

Hicks *et al.*[1] studied the effect of lipid emulsion, epinephrine, and vasopressin on survival rate after bupivacaine-induced cardiac arrest in a porcine model. The results of the authors demonstrated a completely different and unexpected outcome when compared with previous studies that used rodent and canine models. Although species difference may partially explain the different outcomes, one must acknowledge that the anesthetics used in these studies were also markedly different. It is possible to study conscious animals in a canine model because dogs are easily trained. This closely mimics the human clinical scenario when bupivacaine is inadvertently injected intravenously during an attempted regional anesthetic with minimal sedation. Conversely, swine are more difficult to handle without heavy sedation or general anesthesia. Governmental regulations may sometimes disallow animal experimentation in the conscious state. Hicks *et al.* used ketamine, xylazine, and α-chloralose to induce general anesthesia. These drugs are known to work well in large animals such as swine. In a similar porcine study, Mayr *et al.*[2] used azaperone, atropine, ketamine, and piritramid followed by isoflurane after intubation. These anesthetic regimens produce hemodynamic and cardiac electrophysiologic effects, which may explain the failure of lipid rescue protocols in these studies.

Azaperone is a butyrophenone that, like droperidol, may have detrimental electrophysiologic effects at the high doses used in animals.[2] Azaperone also blocks α-adrenergic receptors, producing hypotension, impaired thermoregulation,[3,4] and probably causing the extreme hypotension in the absence of epinephrine in the study of Mayr *et al.*[2] Hicks *et al.*[1] used α-chloralose, an anesthetic that was historically used as a rodenticide.[5] α-Chloralose decreases cardiac conduction velocity in the cardiac muscle and atrioventricular node, prolongs the QTc interval, delays atrioventricular conduction, increases the ventricular refractory period, and exacerbates atrioventricular block caused by verapamil.[6] Drugs that decrease cardiac conduction velocity will enhance bupivacaine arrhythmias,[7] and α-chloralose has also been shown to be proarrhythmic toward the ischemic porcine heart.[8] One can speculate that even if lipid rescue could partially reverse the effects of lipophilic drugs such as bupivacaine, one would not expect this for hydrophilic drugs such as α-chloralose. Through multiple hemodynamic and electrophysiologic effects, the anesthetics, as used in these porcine studies of bupivacaine-induced cardiac arrest, may have contributed to the failure of lipid rescue. For animal studies to optimally contribute to our understanding of resuscitation from inadvertent bupivacaine toxicity, studies should incorporate anesthetic and sedative techniques as that used in humans.

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(Accepted for publication October 28, 2009.)

In Reply:

We thank Drs. Woehlck and El-Orbany for their interest in our recently published article that examined lipid emulsion using a porcine model of bupivacaine-induced cardiac arrest.[1] Their letter raises several important issues. In the Discussion section of our article, we explained some of the major differences between the various animal models that have been used to evaluate lipid treatment for local anesthetic toxicity.

We recognize the potential drug interactions between the anesthetics agents and the experimental protocol. The anesthetic agents, such as xylazine, ketamine, and α-chloralose, were chosen to preserve hemodynamic and electrophysiologic stability at the doses used in our study. Propofol was avoided because of the confounding effect of lipid pretreatment, as found in other animal studies of this nature. Despite this limitation, we were able to achieve a stable hemodynamic profile in all animals before the induction of cardiac arrest with the bupivacaine injection. After examining the electrocardiographic data (mean $\pm$ SD), we did not observe any occurrences of prolonged PR (120.6 $\pm$ 13.7), QRS (59.1 $\pm$ 14.6), or QTc intervals (347.4 $\pm$ 26.4), during the baseline period before the induction of cardiac arrest.[2] Our electrophysiologic data are perhaps different from the results mentioned in the letter of Woehlck and El-Orbany because much higher doses of α-chloralose were used in other studies (75 mg/kg in one study[3] and 100 mg/kg in another[4]) in contrast to a moderate dose of 40 mg/kg used in our study.

We agree with Drs. Woehlck and El-Orbany that it would be useful to consider further experiments that expand our understanding of the potential therapeutic benefits of lipid emulsion in the setting of cardiac arrest induced by toxic doses of local anesthetic, especially at a time when various national and international organizations are in the process of developing recommendations incorporating lipid treatment.

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(Accepted for publication October 28, 2009.)

Insertion of the i-gel™ Airway Obstructed by the Tongue

To the Editor:

In the July 2009 issue of ANESTHESIOLOGY, Theiler *et al.*[1] published an article in which they compared the *Laryngeal Mask Supreme*™ (Laryngeal Mask Company, Henley-on-Thames, United Kingdom) with the i-gel™ (Intersurgical Ltd., Wokingham, Berkshire, United Kingdom) airway. The authors commented that the bulky design of the i-gel™ made insertion time longer, and that tongue size may have an influence on insertion. We noticed a similar problem during the insertion of i-gel™ a few times. During insertion, the cuff carried the tongue along with it posteriorly, making further motion of the i-gel™ impossible. All the patients were in "sniffing the morning air" position as advised by the manufacturer.[2] The device was adequately lubricated. Jaw thrust and insertion with deep rotation were tried[2] when difficulty occurred, but these maneuvers did not solve the problem. Hence, we had to remove and then reinsert the i-gel™ after pulling out and stabilizing the tongue.

The i-gel™ has a noninflatable cuff made of styrene ethylene butadiene styrene. This cuff fits snugly onto the perilaryngeal framework.[2] Unfortunately, the texture and design of the cuff entraps the tongue during insertion. The manufacturers recommend insertion of the device without introducing the fingers,[2] but we feel, in difficult circumstances