products in conjunction with the billing office, perhaps at a fraction of the cost, using e-mail notification for incomplete documentation.

Finally, the strategic costs of having the hospital own the data, giving unfettered access to billing and revenue information, must be weighed carefully. Because the hospital typically owns the AIMS, the hospital, and not the anesthesiology group, owns the data. In our world today, this situation may be less than desirable.

Amr E. Abouleish, M.D., M.B.A.,* Lydia Conlay, M.D., Ph.D., M.B.A.†
*Department of Anesthesiology, University of Texas Medical Branch, Galveston, Texas. aaboulei@utmb.edu †Department of Anesthesiology, Baylor College of Medicine, Houston, Texas.

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Lipid Infusion Resuscitation for Local Anesthetic Toxicity

Proof of Clinical Efficacy

DR. Rosenblatt and colleagues at Mount Sinai Hospital in New York report in this issue of Anesthesiology the successful application of lipid emulsion infusion in the resuscitation of bupivacaine-induced cardiac arrest.[1] The patient was a 58-yr-old man with a history of coronary artery disease who presented for an elective shoulder procedure. Shortly after an interscalene block with bupivacaine (100 mg) and mepivacaine (300 mg), the patient experienced a brief seizure followed by asystole with intervals of ventricular tachycardia. These arrhythmias were refractory to multiple rounds of drugs and countershocks until a member of the staff recommended using intravenous lipid therapy. Soon after administering 100 ml of 20% Intralipid, a single heartbeat was observed, followed 20 s later by the return to a sinus mechanism with normal blood pressure. The patient was later extubated and recovered without neurologic deficit.

This remarkable case report is a watershed in the study of local anesthetic toxicity and might well mark the end of nearly 30 years of regional anesthesia practiced without a specific antidote for its most dread complication. There is a well-defined conceptual framework linking this patient to one whose extreme sensitivity to bupivacaine was reported 9 yr ago.[2] That patient experienced ventricular arrhythmias and bradycardia after receiving 22 mg bupivacaine in a subcutaneous tumescence solution. These clinicians later learned that the patient was severely carnitine deficient and investigated a possible connection between this metabolic abnormality and her sensitivity to bupivacaine. The studies in isolated mitochondria revealed that bupivacaine interferes with carnitine-dependent mitochondrial lipid transport.[3] While attempting to understand the relation of lipid metabolism to bupivacaine toxicity, the unexpected finding in rats was that pretreatment with a lipid infusion increases the bupivacaine dose required to induce asystole.[4] Similarly, administering lipid during resuscitation reliably rescued rats from otherwise fatal doses of bupivacaine. Similar observations were made in dogs where the protocol included an interval of 10 min before treatment to mimic the clinical setting where a delay in administering lipid is likely.[5] None of the six controls receiving 10 mg/kg bupivacaine recovered with cardiac massage alone, whereas all lipid-treated dogs recovered normal hemodynamic profiles. Fortunately for the patient reported in this issue, the phenomenon of lipid rescue seems to work equally well in humans.

This case report might provide the impetus to establish a uniform, coherent, and rational approach to treating severe local anesthetic toxicity. A group from Wake Forest Medical Center (Winston-Salem, North Carolina)

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recently queried academic anesthesiology departments in the United States regarding their approach to treating local anesthetic toxicity.[6] Lamentably, it was clear from the 91 responding institutions that there is little uniformity in planning for this potentially catastrophic complication. Only a small fraction of respondents would consider using lipid to treat local anesthetic toxicity.

Substantial research on lipid rescue is still needed. Intravenous lipid emulsion has a long track record of safety as hyperalimentation and in formulations of propofol, but its safety is unknown when administered in the high does used in lipid rescue. Although it is reassuring to note that the patient in the report of Rosenblatt *et al.*[1] was neurologically intact after the event, we must keep in mind that this represents a single case. Specific factors to study include defining the optimal lipid dose, rate, and duration of infusion as well as establishing a safe upper limit. It remains unanswered whether there is more benefit or harm in using epinephrine in local anesthetic cardiac toxicity, although currently, I would continue to recommend its use as part of the standard American Heart Association Advanced Cardiac Life Support protocol.

Physicians should be made aware that propofol is not a component of lipid rescue. I raise this issue because it is a common misconception that lipid rescue implies the use of propofol, which is formulated in a 10% lipid emulsion. Although small doses of propofol might be of benefit to control seizure activity in the early stages of a toxic event, propofol is contraindicated when there is any evidence of cardiac toxicity. The standard 1% formulation would require delivering gram quantities of propofol to provide the needed dose of lipid. This is unacceptable in the setting of cardiovascular collapse.

Until further studies identify an optimal regimen, lipid infusion should be used, as in this case report, only after standard resuscitative measures have proven ineffective. I believe the evidence in support of its use is now sufficient to warrant having 20% lipid emulsion available in all operating rooms, block rooms, obstetric units, and other sites where local anesthetics are used (including plastic surgery suites). It is worth noting that it has recently been found that bupivacaine delays the onset of myocardial acidosis during no-flow states, suggesting that bupivacaine may provide some degree of cardiac protection during cardiovascular collapse.[7] The point is that 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.

The mechanisms underlying lipid rescue are still incompletely understood. Recent research found in isolated rat heart that lipid infusion accelerates the decline in bupivacaine myocardial content and speeds recovery from bupivacaine-induced asystole.[8] Lipid infusion might also provide a salutary metabolic effect to the heart[9] or some other, as yet unidentified benefit. Further research to delineate the mechanisms at play might lead to even more effective therapy.

Dr. Rosenblatt and her team are to be thoroughly congratulated for saving this patient's life. While proving the clinical efficacy of lipid rescue, they have also validated a contemporary model of academic anesthesiology. There are limits to the information one can draw from a single case, but in the scenario where prospective clinical trials are impossible, we can take heart from this reported experience. A once feared complication of regional anesthesia may have just become slightly less fearsome.

Guy Weinberg, M.D., Department of Anesthesiology, University of Illinois at Chicago, Chicago, Illinois. guyw@uic.edu

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