Local anesthetic systemic toxicity (LAST), although rare, can be a fatal complication after regional anesthesia. The use of intravenous (IV) fat emulsion as a treatment of LAST is controversial among clinicians worldwide. This literature review aims to identify existing data supporting the use of IV lipids in the management of LAST and current best evidence-based practice by anesthesia providers during a LAST event. More than 120 articles resulted from a systematic literature search that was conducted using major search engines. Of those articles, 25 were included in this literature review. The safe use of IV lipids for the treatment of LAST is supported in the literature. Yet, there is still lack of awareness on lipid rescue therapy despite the significant evidence of its positive outcome. In the setting of local-anesthetic toxicity, lipid rescue should be considered first-line treatment. Intravenous lipids must be readily available in all institutions performing regional anesthesia. Education on their use, storage, and dosage is vital among anesthesia departments in the United States and worldwide.

Keywords: Intravenous fat emulsion, lipid emulsion, lipid rescue, lipid sink, local anesthetic toxicity.
Clinical Presentation and Risk Factors for Local Anesthetic Systemic Toxicity. Early recognition of the signs and symptoms of LAST is paramount during the management of this potentially life-threatening event. Toxicity usually occurs within minutes of intravascular injection of the LA, and CNS signs precede those of the cardiovascular system.\(^6,7\) The acute onset of any neurologic symptom after regional anesthesia should alert practitioners to the presence of LAST.\(^6\) Seizures, agitation, drowsiness, visual disturbances, metallic taste, loss of consciousness, coma, and respiratory arrest are among the signs and symptoms of CNS toxicity.\(^6-8\) These signs and symptoms are followed by changes in the cardiovascular system ranging from tachycardia, hypertension, and ventricular arrhythmia (due to sympathetic activation) to bradycardia, hypotension, loss of peripheral vasomotor tone, and asystole.\(^6,9\)

Risk factors contributing to the development of LAST include age, renal dysfunction, cardiac disease, pregnancy, hepatic dysfunction, and block site and technique.\(^6\) Elderly patients, for example, have a reduced clearance of LAs, and infants have an immature hepatic system, making them susceptible to LA toxicity.\(^6\) During pregnancy, the increased cardiac output results in an increased absorption of LA and, consequently, increased plasma concentration.\(^6,7\) Drug interactions, surface area vascularity, acidosis, hypoxia, and hypercarbia are other factors affecting LA toxicity.\(^7\) These risk factors are implicated in the development of LAST and should be considered when one is making a diagnosis.

Lipid Sink and Other Theories. Intralipid is a soy-based lipid emulsion composed mostly of long-chain triglycerides and represents the most commonly used emulsion in case reports and animal studies.\(^9\) Multiple theoretical mechanisms have been proposed to explain the antidote effect of lipid emulsion.\(^10\) The “lipid sink” is currently the most accepted theory explaining the mechanism of action of IV lipids.\(^10\) The lipid sink theory assumes the lipophilic LA molecule is sequestered by lipid emulsion in the blood.\(^11,12\) The lipid emulsion creates a lipid-rich compartment in the intravascular space that differs from the plasma aqueous phase and has the ability to entrap lipophilic drugs.\(^13\) To validate the lipid sink effect, Papadopoulo et al\(^13\) substituted the LA with 2 lipophilic dyes and visually demonstrated the dye sequestration occurring when both dyes were mixed with lipid emulsion to simulate “lipid rescue” treatment. After adding lipid emulsion to the dyes, a considerable amount of dye was sequestered by the lipid compartment, and the color intensity in the aqueous phase was reduced. The use of dye surrogates to illustrate LA drug sequestration by lipid emulsion demonstrated the lipid sink effect. Papadopoulo and colleagues confirmed the property of lipid emulsion to entrap lipophilic molecules with this study.

Another mechanism thought to explain the antidote effect of lipid emulsion is the “lipid flux” theory, whereby lipid emulsion increases the influx of acylcarnitine into the mitochondria in the cardiac muscle, thus increasing the energy necessary for muscle contraction.\(^10,14\) This theory is specific to the heart; therefore, the effect of IV lipids in the CNS cannot be explained as it is by the lipid sink theory.\(^10\) The stimulation of cardiac mitochondrial respiration through fatty acid oxidation, reduction of sodium channel inhibition by free fatty acids, and the stimulation of cardiac inotropy by activation of calcium channels are other suggested but unconfirmed mechanisms.\(^12\)

Studies of Lipid Emulsion for Treatment of Local Anesthetic Systemic Toxicity. The use of lipid emulsion for the treatment of LAST is a subject of much interest in the scientific community. Most of the current evidence of this novel treatment comes from animal studies. In humans, due to the possible lethal outcomes, a prospective study on this topic becomes unethical and unfeasible.\(^15\) However, numerous case reports discussed in the literature are evidence of the usefulness of IV lipids for the treatment of LAST. Cave et al\(^16\) conducted a systematic literature review to identify all peer-reviewed cases and clinical studies reporting the use of IV lipid emulsion as an antidote in humans. The search revealed 42 individual case reports of IV lipid emulsion application in humans: 19 cases receiving the IV lipid emulsion for the treatment of LA toxicity and 23 cases for treatment of non-LA toxins. Whiteman and Kushins\(^17\) reported the successful resuscitation with 20% IV lipids (Intralipid) of a 32-year-old woman who experienced seizures and cardiac arrest after the accidental overdose of bupivacaine (Marcaine) during an abdominoplasty and mastopexy. The patient became confused and agitated, experiencing generalized tonic-clonic seizures before going into cardiac arrest. Cardiopulmonary resuscitation was initiated, followed by a rescue protocol of a 1.5 mL/kg bolus of Intralipid 20% and a maintenance infusion of 0.25 mL/kg for 60 minutes. After 45 minutes of CPR, several seizure episodes, and the use of a cardiac defibrillator twice, the patient’s heart rhythm returned to normal. A similar case was reported by Tierney et al\(^18\) describing the use of 20% lipid infusion during the resuscitation of a 35-year-old patient who underwent cardiovascular collapse after the administration of a toxic dose of lidocaine for the incision and drainage of an abscess. The patient in this case received a total of 1,000 mg of 2% lidocaine without epinephrine (3 times more than the maximum recommended dose) via a ring block technique for the incision and drainage of a labial abscess. Approximately 20 minutes after the procedure, the patient experienced seizures followed by a pulseless electrical activity arrest. Resuscitation was initiated, and 3 minutes later, a 100-mL bolus of 20% IV lipids (Intralipid) was administered. One minute after the institution of IV lipids, a sinus tachycardia with a blood pressure of 100/60 mm Hg was noted. The afore-
mentioned published cases build and expand on the documented evidence of the successful use of IV lipids in the treatment of cardiac arrest caused by LA toxicity. Therefore, clinicians are highly encouraged to continue reporting these events.

- **Intravenous Lipids and ACLS During Local Anesthetic Systemic Toxicity.** The evidence in the literature supports the use of IV lipids for the treatment of LAST. However, how to use it promptly continues to be a controversial subject. According to the 2012 American Society of Regional Anesthesia and Pain Medicine (ASRA) checklist for the management of LAST (Figure), cardiovascular collapse can be prevented with the early infusion of lipids when LAST is suspected.\(^1\) The ASRA recommends an initial 20% lipid emulsion bolus of 1.5 mL/kg followed by a continued infusion of 0.25 mL/kg/min. The bolus can be repeated once or twice if cardiovascular collapse persists, and the infusion rate can be doubled to 0.5 mL/kg/min if blood pressure remains low. The infusion should continue for at least 10 minutes after the restoration of circulation, with a suggested upper limit of 10 mL/kg of lipid emulsion over the first 30 minutes. The ASRA also recommends avoiding vasopressin, calcium channel blockers, β-blockers, or LA during advanced cardiac life support (ACLS) and decreasing epinephrine doses to less than 1 μg/kg. The latter recommendation is based on laboratory evidence revealing that high doses of epinephrine can impair resuscitation and decrease the efficacy of lipid emulsion during LAST.\(^1\)

Li et al,\(^2\) with the intent to investigate the effect of epinephrine as a co-treatment with IV lipids in the management of LAST, conducted an experimental study with 32 rats randomly assigned to 4 groups of 8 rats each. Asystole was induced with bupivacaine, and 10 minutes of CPR was performed before they administered saline, epinephrine alone, 20% lipid emulsion bolus with epinephrine, or 20% lipid emulsion without epinephrine. The rats treated with epinephrine and lipid emulsion showed a noticeable improvement at 25 minutes compared with those treated with lipid alone. The coronary perfusion pressure was higher immediately after the administration of lipids in the epinephrine/lipid group compared with the rats treated with lipid only, and the myocardial bupivacaine content was lower in this group than the other groups. The rats that survived after the treatment with only lipids had less severe acidosis, higher PO₂, and better hypoxemia in relation to those treated with epinephrine and lipids.

In contrast to the work of Li and colleagues, Mauch et al\(^3\) studied the response of 3 groups of piglets (7 piglets each) to epinephrine, 3 μg/kg (group 1); 20% IV lipids (Intralipid 20%), 2 mL/kg (group 2); and 20% IV lipids (Intralipid 20%), 4 mL/kg (group 3), for resuscitation after severe hemodynamic compromise from bupivacaine. All animals in group 1 survived, and only 4 animals in groups 2 and 3 survived. Furthermore, hemodynamic stability occurred fastest (within 1 minute) in group 1. Mauch and colleagues concluded that epinephrine was more effective than IV lipid for the management of severe hemodynamic compromise secondary to bupivacaine toxicity in piglets.

Other studies conducted in rodents have shown the use of IV lipid emulsion results in superior hemodynamic and metabolic recovery compared with epinephrine alone or in conjunction with another vasopressor during the resuscitative efforts of bupivacaine-induced asystole.\(^4\) Controversy in the efficacy of lipid emulsion in LAST lies primarily concerning other LAs, which are less lipid soluble than bupivacaine, which may not respond to the same degree to IV lipids.\(^2\) Ozcan and Weinberg\(^2\) reported the presence of 10 peer-reviewed cases as of 2011 in which lipid emulsion was used successfully to reverse bupivacaine toxicity and 7 peer-review case reports relating the use of lipid emulsion for nonbupivacaine-related LA toxicity. Of the 7 cases of nonbupivacaine-related toxicity, only one case described persistent CNS symptoms despite the infusion of 2 boluses of 20% lipid emulsion. The unsuccessful case was related to the injection of a lidocaine/ropivacaine mixture in a patient receiving carbamazepine therapy, which could have caused a synergistic
effect, thus enhancing the CNS toxicity of the LAs.

Ozcan and Weinberg21 found evidence obtained by case reports suggesting that the infusion of lipid emulsion is useful in the reversal of CNS and cardiovascular symptoms caused by LAST related to bupivacaine and any other LA (less lipophilic than bupivacaine). Additionally, the authors supported the use of lipid emulsion at the first sign of LAST, stating no rationale for withholding this treatment until the resuscitative efforts with standard ACLS were unsuccessful.

Burch and colleagues8 conducted a literature search on the use of lipid emulsion for treatment of LA toxicity and found that in all the cases reported, most patients did not respond to standard resuscitative measures until lipid emulsion therapy was instituted, resulting in complete recovery. In the cases reviewed, an initial bolus of 1.5 mL/kg of lipid emulsion was administered, followed by an infusion of 10 mL/min.

The literature shows experimental and clinical evidence suggesting the efficacious combination of lipid emulsion and CPR during LAST in restoring spontaneous circulation. Therefore, IV lipids should be readily available to give in conjunction with ACLS protocols at the earliest signs and symptoms of toxicity. A summary of the studies reporting the use of IV lipids and ACLS during LAST is shown in Table 1. The findings from seminal studies on LAST are shown in Table 2.

- **Adverse Effects of Intravenous Lipid.** Adverse effects from IV lipid emulsion are seldom reported.8,12,16 Nonetheless, it is important to recognize and be aware of the potential risks associated with the use of IV lipid emulsion. Allergy (to soy), hyperthermia, hypercoagulability, pancreatitis, elevated liver enzyme levels, and fat embolism in infants are some of the possible adverse effects. Thrombophlebitis during peripheral IV administration can occur, and pulmonary hypertension is likely if lipid emulsion is administered at rates higher than 100 mg/kg/h. However, in the setting of LAST, potential complications from the use of IV lipids are outweighed by its benefits.8,9

**Discussion**

- **Adoption of Lipid Rescue During Local Anesthetic Systemic Toxicity: Revision of Current Practice Worldwide.** Since the first successful use of lipid therapy for the treatment of bupivacaine-induced cardiac toxicity was reported in 2006,5 the management of LAST has been revolutionized. Lipid emulsion is recognized as a potent antidote for LA toxicity, and there is evidence in the literature supporting its use as a first-line treatment of LAST. To evaluate existing practices among academic anesthesiology departments in the United States regarding the management of LA cardiac toxicity, Corcoran et al22 conducted a survey in 135 academic anesthesiology departments. The study showed a wide-ranging variety of treatment approaches for handling severe LA toxicity. When asked about the choice of usage and availability of lipid emulsion, 67 (74%) of the 91 centers completing the survey responded they would not use lipids to treat bupivacaine toxicity, and 24 (26%) would contemplate its use. Centers with a higher volume of regional anesthesia cases were 3.9 times more likely to use lipids than were low-volume centers (44% vs 17%), and those considering its use reported different storage location for the lipids, including the operating room pharmacy (39%), the hospital pharmacy (35%), the code cart (22%), or a drug dispensing machine (4%). In 59% of the centers, lipid was accessible in less than 30 minutes, 10 to 30 minutes for 26%, and more than 30 minutes for 15% of the centers. Corcoran and colleagues’ survey took place the same year the first successful use of lipid emulsion in the treatment of LA toxicity was documented. Afterward, the continuous report of comparable cases has increased the acceptance and recognition of lipid therapy as a valuable treatment of LAST.

Toledo et al23 developed a similar survey to address the availability of lipid emulsion in US obstetrics units. The survey was sent to all obstetric anesthesia directors in the country, and a 69% response rate was achieved. Lipid emulsion was readily available (stocked in the unit) in 88% of the obstetric units, with the remainder claiming to have it elsewhere in the hospital. The availability of lipid emulsion was estimated to be less than 10 minutes in 70 hospitals and 10 to 30 minutes in another 2 hospitals, with 95% of respondents having it available in less than 30 minutes. Toledo et al23 reported storage locations were as follows: a drug dispensing machine (50%), regional anesthesia cart (33%), anesthesia workroom (17%), code cart (10%), and operating room (10%). Compared with the survey led by Corcoran and colleagues in 2006, the availability and implementation of lipid emulsion for the management of LAST were considerably higher in this study by Toledo and colleagues in 2013.

The use of lipid rescue therapy for the treatment of LAST has been highly supported by the ASRA, the Association of Anesthetists of Great Britain and Ireland (AAGBI), the American Heart Association, and the American College of Medical Toxicology (ACMT).24 Aiming to assess the current position of China’s anesthesiologists in regard to the adoption of lipid therapy, Xu and associates25 conducted a survey in 41 academic anesthesiology departments listed by the orthopedic anesthesia group of the Chinese Society of Anesthesiology. Thirty-six institutions responded to the survey (88%). Of the 36 responding institutions, 22 (61%) were aware that lipid emulsion was a treatment of LAST, 13 (36%) had heard about it but were unaware of the details of the regimen, 8 (22%) had knowledge of the 2007 and 2010 AAGBI guidelines for the management of LAST, 19 (53%) recognized the 2010 ASRA guidelines, and 2 (6%) were mindful of the ACMT guidelines. Eight (22%) of the
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<td>Li et al, 2012</td>
<td>To explore the effect of epinephrine on delayed lipid-based treatment of bupivacaine-induced cardiac arrest in rats</td>
<td>Retrospective cohort study</td>
<td>32 rats (8 per group)</td>
<td>Rats received bupivacaine to induce asystole. Basic life support was performed for 10 min before the rats received saline, epinephrine alone, or 20% lipid emulsion bolus with or without epinephrine pretreatment.</td>
<td>In the rats treated with epinephrine plus lipid emulsion, there was a marked improvement in hemodynamic parameters at 25 min vs rats treated with lipid alone. However, the rats treated with lipid alone that survived had higher PO&lt;sub&gt;2&lt;/sub&gt;, less severe acidosis, and better hypoxemia relative to surviving rats given epinephrine plus lipid.</td>
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<tr>
<td>Mauch et al, 2011</td>
<td>To compare effectiveness of epinephrine and IV lipids for the treatment of severe hemodynamic compromise due to bupivacaine intoxication</td>
<td>Retrospective cohort study</td>
<td>21 piglets (7 per group)</td>
<td>Bupivacaine was infused at a rate of 1 mg/kg/min until mean arterial pressure (MAP) dropped to 50% of the initial value. Bupivacaine infusion was then stopped, and epinephrine, 3 µg/kg/min (group 1); 20% IV lipids (Intralipid 20%), 2 mL/kg (group 2); or Intralipid 20%, 4 mL/kg (group 3), was immediately administered. Survival, hemodynamic course, and ETCO&lt;sub&gt;2&lt;/sub&gt; were recorded.</td>
<td>All animals in group 1 (100%) but only 4 of 7 (57%) piglets in group 2 and group 3, respectively, survived. Normalization of hemodynamic parameters (HR, MAP) and ETCO&lt;sub&gt;2&lt;/sub&gt; was fastest in group 1 with all piglets achieving HR and MAP values at or above baseline within 1 min.</td>
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<td>Ozcan &amp; Weinberg, 2011</td>
<td>To give a background information about the status of the use of lipid emulsions in LAST</td>
<td>Literature review</td>
<td>Ten peer-reviewed cases as of 2011 reported the successful use of lipid emulsion to reverse bupivacaine toxicity and 7 peer-reviewed cases, for nonbupivacaine-related toxicity.</td>
<td>Results suggest that the infusion of lipid emulsion is useful in the reversal of CNS and CV symptoms caused by LAST related to bupivacaine and any other LA (less lipophilic than bupivacaine). Additionally, the authors supported the use of lipid emulsion at the first sign of LAST, stating no rationale for withholding this treatment until resuscitative efforts with standard ACLS were unsuccessful.</td>
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<td>Burch et al, 2011</td>
<td>To discuss the use of lipid emulsion to treat LA toxicity</td>
<td>Literature review</td>
<td>A literature search identified 7 case reports of LA toxicity in which lipid emulsion was used.</td>
<td>Lipid emulsion was found to be successful in the treatment of LA toxicity associated with various regional anesthetic techniques and multiple LAs. Most patients were unresponsive to initial management of LA toxicity with standard resuscitative measures, but all recovered completely after receiving lipid emulsion therapy.</td>
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Table 1. Studies Reporting Use of Intralipids and ACLS During Local Anesthetic Systemic Toxicity (LAST)  
Abbreviations: ACLS, Advanced Cardiac Life Support; CNS, central nervous system; CV, cardiovascular; ETCO<sub>2</sub>, end-tidal carbon dioxide; HR, heart rate; IV, intravenous; LA, local anesthetic.
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<td>Weinberg et al.² 1997</td>
<td>To associate the occurrence of severe ventricular arrhythmias with bupivacaine toxicity in a patient with isovaleric acidemia (IVA)</td>
<td>Case report</td>
<td>A 16-year-old, 60-kg female teenager with a history of IVA scheduled for bilateral axillary liposuction</td>
<td>Modified Klein’s tumescent solution (300 mL), a mixture of 0.0075% bupivacaine and epinephrine, 1:1,000,000, in lactated Ringer’s solution, was injected subcutaneously into the right axilla</td>
<td>Four minutes later, the patient’s ECG changed from a normal sinus rhythm at 80/min to a sinus rhythm of 40/min, to a junctional bradycardia, and then to a wide complex ventricular dysrhythmia at 20/min, and BP decreased from 130/70 mm Hg to 60/40 mm Hg</td>
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<td>Weinberg et al.³ 1998</td>
<td>To confirm that IV lipid treatment increases the dose of bupivacaine required to produce asystole in rats</td>
<td>Retrospective cohort study</td>
<td>Anesthetized Sprague-Dawley rats were used in pretreatment (protocol 1) and resuscitation (protocol 2)</td>
<td>Protocol 1 had 4 groups (6 for all groups)</td>
<td>Protocol 2 had 2 groups (6 for all groups)</td>
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<td>Rosenblatt et al.⁵ 2006</td>
<td>To report the first successful use of a 20% lipid infusion to resuscitate a patient from a prolonged cardiac arrest after the placement of an interscalene block with bupivacaine and mepivacaine</td>
<td>Case report</td>
<td>A 58-year-old, 82-kg, 170-cm man presenting for arthroscopic repair of a torn rotator cuff in the right shoulder</td>
<td>The brachial plexus was identified, and 40 mL local anesthetic solution (20 mL bupivacaine, 0.5%, and 20 mL of mepivacaine, 1.5%) was injected slowly (over approximately 2.5 min) in 5-mL increments with gentle aspiration between doses.</td>
<td>Thirty seconds after removal of the block needle, a tonic-clonic seizure occurred. Approximately 90 s Later, the ECG showed asystole, and no pulse, by carotid or femoral palpation, or blood pressure was detectable. Then ACLS was started. After 20 min of unsuccessful CPR efforts, 20% IV lipid was given, and sinus rhythm returned in 15 s.</td>
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Table 2. Findings From Seminal Studies on Local Anesthetic Systemic Toxicity

Abbreviations: ACLS, advanced cardiac life support; BP, blood pressure; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; IV, intravenous.
36 institutions reported to have lipid emulsion readily available and stocked according to the AAGBI and ASRA guidelines since 2008. The remaining 28 departments did not stock lipid emulsion, and only 7 (25%) had plans to stock it. The survey showed more resources should be allocated to increase the awareness of the importance of lipid rescue among anesthesiologists in China.

Similarly, Heinonen et al conducted a survey to evaluate the incidence of LAST and its treatment in Finland between the years 2011 and 2013. A questionnaire was sent to the anesthesia department of all Finnish public hospitals (N = 45), with a 100% response rate achieved. A total of 211,700 regional anesthesia cases (not including spinal anesthesia) were performed during the survey period, and 15 cases of LAST were reported. Of those cases, 14 developed CNS toxicity and only 1 progressed to cardiovascular toxicity. The patient with cardiovascular toxicity were treated with lipid emulsion, for a complete recovery after the use of lipid emulsion. However, the risk of unfavorable events with the use of IV lipids is outweighed by the established benefits of the treatment. Among the proposed theories to explain the mechanism of action of IV lipids, the lipid sink theory has the most recognition among scientists who have successfully simulated it in the laboratory.

The ASRA recommends the use of lipid emulsion at the first sign of LA toxicity, to prevent cardiovascular collapse. Multiple anesthesiology departments in the United States and around the globe have followed these recommendations. Yet, there is still a controversy in the use of lipid emulsion for the treatment of LA toxicity. The authors recommend the reporting of clinical cases of LAST managed with lipid emulsion; the easy availability of IV lipids in all institutions performing regional anesthesia; the education of anesthesia personnel regarding IV lipid use, storage, and dosage; and the development of an international protocol for the management of LAST.


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DISCLOSURES
The authors have declared no financial relationships with any commercial entity related to the content of this article. The authors did not discuss off-label use within the article.