Local Anaesthetic Systemic Toxicity Beyond the Operating Theatre: A Practical Narrative Review for Everyday Practice.

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Abstract
Background: Local anaesthetic systemic toxicity (LAST) is uncommon yet potentially life-threatening in peri-operative care. Over the last two decades, reports have expanded beyond anaesthetist-led regional techniques to wards, labour suites, interventional radiology rooms, dentistry, ambulatory surgery and aesthetic pracices
Objective: To synthesise practical epidemiology, core mechanisms, risk factors, recognition, prevention, and management of LAST with a focus on non-anaesthetist and out-of-theatre contexts.
Methods: Narrative search of PubMed and Scopus (Jan 2018–Oct 2025), including guidelines, registries, reviews, and case series, English-language, two-reviewer screening, qualitative synthesis without meta-analysis,seminal pre-2018 sources retained for foundational content.
Findings: Evidence indicates increased recognition of LAST outside theatres driven by decentralised infiltrations, cumulative dosing across teams, and inconsistent lipid kit availability in lower-acuity areas. An NPDS analysis (1983–2022) reported that non-lidocaine local anaesthetic poisonings decreased after 2010, whereas lidocaine poisonings and lidocaine-related deaths increased, deaths shifted away from operating rooms and were increasingly linked to intravenous administration. Mechanistically, sodium-channel blockade is central, with mitochondrial dysfunction and ion-channel cross-effects contributing to severe cardiovascular presentations. Prevention hinges on accurate dose calculation, cumulative tracking, continuous monitoring, regular simulation, and universal lipid kit access.
Conclusion: With simple governance, rehearsal, and ready access to lipid therapy, most LAST events can be recognised earlier and treated faster wherever they occur
Index Terms- Local anaesthetic systemic toxicity, lipid emulsion therapy, perioperative safety, non-anesthetist practice, lidocaine and patient safety.

I. INTRODUCTION

LAST is an uncommon but memorable complication, patients may move from minor symptoms to seizures or cardiovascular collapse within minutes if toxicity is not recognised. (1- 4) Historically tied to regional and neuraxial techniques under anaesthetist supervision, more cases now arise in wards, labour suites, interventional radiology, dental clinics, ambulatory units, and aesthetic centres. (3 -11) The central physiology is voltage-gated sodium channel blockade with CNS excitation followed by depression and cardiovascular instability, and severe cases may involve mitochondrial dysfunction and acidosis-driven increases in the unionised fraction of drug. (12-15) Incidence remains low, but the relative burden is shifting outward from theatres to areas with less monitoring and slower access to lipid emulsion. (15-21)

II. METHODS

Databases searched in PubMed and Scopus. (12,22) Date range-january 2018–October 2025, preserving seminal pre-2018 sources.Inclusion-English-language guidelines, registries, systematic and narrative reviews, and peri-operative case series/reports relevant to non-anaesthetist contexts.Screening- two reviewers independently screened titles/abstracts-disagreements resolved by consensus. Synthesis-qualitative, no meta-analysis due to heterogeneity of designs and outcomes.

Characteristics and mechanisms

Local anaesthetics are weak bases that block nerve signals reversibly by binding to voltage-gated sodium channels in neu-

ronal membranes (1, 4, 10, 27). They exist in equilibrium between an ionised, water-soluble form and a unionised, lipid-soluble form,this balance affects how fast the drug crosses lipid-rich nerve membranes (23-27). Once inside the axoplasm, the ionised form binds the intracellular part of sodium channels, stopping depolarisation and so preventing pain signal conduction (4, 10). Drugs with higher lipid solubility, like bupivacaine and ropivacaine, are more potent and longer acting but also more cardiotoxic because they stay in heart tissue and disrupt repolarisation (1, 8, 10, 27). By contrast, lidocaine has lower lipid solubility, giving a faster onset but shorter duration; its widespread availability and use in topical, infiltrative, and intravenous forms mean it accounts for most reported cases of systemic toxicity (6, 11,).Local anaesthetic molecules share a three-part structure: an aromatic ring that gives lipid solubility, an intermediate chain (either an amide or an ester) that determines metabolism and allergy potential, and a tertiary amine that controls ionisation (4, 10). Amide agents (e.g., lidocaine, bupivacaine, ropivacaine) are metabolised in the liver by cytochrome P450 enzymes, while ester agents (e.g., procaine, chloroprocaine, tetracaine) are rapidly broken down by plasma pseudocholinesterases (4, 9, 10). Conditions that reduce hepatic perfusion such as liver disease, pregnancy, or heart failure slow amide metabolism and increase the risk of toxicity (4, 9). At the systemic level, the same effects on sodium channels cause LAST. In the
central nervous system, inhibition starts with cortical excitation that produces tinnitus and agitation, then moves on to CNS depression and seizures. In the cardiovascular
system, prolonged blockade of cardiac sodium, potassium, and calcium channels leads to slowed conduction, arrhythmias, and contractile failure (1, 10, 27). The properties that make local anesthetics effective lipid affinity, protein binding, and sodium channel selectivity are also the ones that determine their toxic potential when systemic levels rise.

Changing epidemiology

Over the past twenty years, local anaesthetic systemic toxicity (LAST) has quietly moved beyond the operating room and into the everyday spaces where patients live their lives hospital wards, labour rooms, dental offices, and ambulatory clinics (29). What was once a rare and mostly anaesthetist-managed complication now finding its way into far more routine procedures, touching everything from wound infiltration in the emergency department to topical gels in aesthetic practice. (1-4) This change reflects how pain management has evolved. (7-10) With multimodal analgesia and the push for shorter hospital stays, local anaesthetics are being used more generously, often by teams spread across different departments. When nobody keeps track of the big picture, small doses can add up especially in busy settings with less monitoring or variable staff training. It's easy for dose totals to slip past safe limits unless someone takes clear responsibility. (11-14) Surveillance data backs up what many clinicians intuitively sense: cases of LAST are increasingly reported during everyday procedures and by non-anaesthetists. Lidocaine in particular stands out not because it's unusually hazardous, but because it's everywhere (3,7). Whether used for dental blocks, topical analgesia, or complex peri-operative infusions, its sheer ubiquity makes it the lead player in systemic toxicity cases. In contrast, when potent agents like bupivacaine or ropivacaine are involved, things can get serious quickly, often with tough-to-treat cardiac effects due to their persistence in heart tissue. (11,28) Mechanistically, LAST begins when enough local anaesthetic slips into the bloodstream to disrupt both the brain and the heart. The first signs can be subtle tinnitus, restlessness, or a tingling sensation around the mouth (1,4,7). If unnoticed, things can escalate to seizures or dangerous changes in heart rhythm. Lipid-soluble agents tend to linger, raising the risk for prolonged and severe toxicity. (3,11)
system responsibility where everyone tracks doses, stays alert for early symptos, and keeps lipid emulsion therapy ready can make all the difference. LAST is no longer just a rare complication in anaesthesia textbooks, it's a real-world challenge wherever local anaesthetics are used. When teams communicate, monitor, and prepare, patients are much more likely to stay safe and recover well. (1,10,13)

Risk factors and predisposing conditions

LAST rarely arrives out of the blue, it emerges where patient vulnerability, drug properties, and procedural systems intersect, and recognising these overlaps allows clinicians to act before toxicity declares itself. (1, 10)

Patient-related factors-older adults often have lower protein binding and slower hepatic clearance, increasing the free fraction and prolonging exposure to amide local anaesthetics. (4,30) Hepatic disease or heart failure reduces hepatic perfusion and metabolism, slowing redistribution of agents such as lidocaine and bupivacaine. (4,31) Pregnancy lowers the threshold for toxicity through physiological changes in cardiac output, volume of distribution, and susceptibility to sodium-channel blockade. (1, 3) Infants and children are vulnerable because hepatic enzymes are immature and plasma protein levels fluctuate, increasing the free drug fraction. (1,32) Hypoxia, hypercarbia, and acidosis pre-existing or procedure induced shift ionisation toward the unionised fraction and accelerate tissue penetration, thereby intensifying toxicity. (10, 16)

Drug and pharmacological factors-How risky a local anaesthetic proves to be dependent on its chemistry: lipid solubility and protein binding drive both potency and duration, but also the likelihood of myocardial persistence and cardiotoxicity. (3, 11, 17) Bupivacaine and ropivacaine are more lipophilic and carry a narrower safety margin, whereas lidocaine's apparently wider window can be deceptive when multiple routes or

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repeated doses accumulate. (3, 11, 17) Using mixtures, frequent top-ups, or continuous infusions can push cumulative doses beyond safe limits if no one tracks the total. (7, 9, 20) A common pitfall outside anaesthesia is dose conversion error confusing percentage concentrations with milligrams especially under time pressure. (7, 9, 20).

Procedure and system-related factors-Intravascular injection during infiltration or nerve block remains the classic trigger for abrupt toxicity. (1, 21) Increasingly, cumulative absorption from high-volume tumescent solutions or large field blocks is recognised in outpatient cosmetic practice and emergency wound care when observation is short and monitoring intermittent. (6, 8) Risk escalates in environments without systematic surveillance and with limited resuscitation capability, where atypical symptoms may be missed or misattributed. (7,29) Institutional weaknesses, no clear escalation protocol, absent or expired lipid kits, and irregular drills turn manageable events into preventable harm. (9, 10)

Polypharmacy and interactions-Drugs that inhibit hepatic CYP enzymes can slow the metabolism of amide local anaesthetics and prolong exposure; clinicians should review beta-blockers, calcium channel blockers, and serotonergic agents when planning dosing. (7, 10, 27) Sedatives and opioids can blunt the early neurologic and haemodynamic warning signs, delaying recognition until respiratory depression or cardiovascular instability is established. (5, 23)

Identifying at-risk patients and high-risk situations in advance allows teams outside anaesthesia to plan safer dosing, ensure monitoring is continuous during and after administration, and confirm that lipid therapy is on hand before the first millilitre is drawn up. (1, 10) In peri-operative care, vigilance about these specific risk patterns is the most reliable way to prevent LAST. (1, 10)

Clinical manifestations and diagnostic challenges

What makes LAST difficult is not only its severity but its habit of arriving sideways, in places and moments when clinicians are focused on other problems. (1, 7) The familiar narrative early neurological symptoms that evolve into cardiovascular instability remains useful, yet real-world cases often refuse that order, and some patients deteriorate hemodynamically without any preceding prodrome. (3, 10). Early neurological signs are usually the first turning of the lock. (4, 5) Patients describe a metallic taste, perioral tingling, tinnitus, dizziness, or odd visual blurring; agitation or confusion may creep in as plasma levels climb. (4, 5) Missed at this stage particularly in wards and recovery areas where delirium, hypoglycaemia, or sedative effects are common these warnings can give way abruptly to seizures or loss of consciousness. (7, 11)

Cardiovascular involvement tends to be more unforgiving. (1, 10) Sustained sodium-channel blockade widens QRS complexes and slows conduction, while effects on potassium and calcium channels destabilize rhythm and depress contractility; bradyarrhythmia and wide-complex tachycardias may precede pulseless arrest if treatment is delayed. (1, 10) Recent reports from non-anaesthetist environments underscore this cardiac-dominant trajectory, often in patients without continuous monitoring when the first changes appeared. (6, 8). Diagnosis falters for ordinary reasons: patients move between rooms and handovers, continuous monitoring is not routine in dental or outpatient clinics, and a crowded differential anaphylaxis, vasovagal syncope, hypoglycemia, myocardial infarction steals attention from toxic exposure, especially when onset is delayed by tumescent techniques or infusions. (5, 6, 7, 11) The practical response is a standing habit rather than a heroic hunch: keep LAST on the list after any meaningful local anaesthetic dose, treat new neurological symptoms as a warning, and prepare lipid while securing airway and ventilation at the first sign of cardiovascular instability even if the classic prodrome never appeared. (1, 10)

Category	**Examples**	**Underlying Mechanism**	**Clinical Significance**
Patient-related	Elderly, hepatic or cardiac disease, pregnancy, renal impairment	Reduced metabolism and protein binding; increased drug half-life	Lower toxic threshold; prolonged drug circulation
Paediatric	Neonates and infants	Immature enzyme systems and lower plasma protein levels	Heightened free drug fraction and exposure
Metabolic/Physiological	Hypoxia, acidosis, hypercarbia	Increased un-ionised fraction of local anaesthetic	Facilitates tissue penetration and CNS toxicity
Pharmacologic	Bupivacaine, ropivacaine, mixed agents, continuous infusions	High lipid solubility and potency; cumulative dosing	Narrow safety margin; delayed toxicity
Procedural	Intravascular injection, high-volume infiltration, rapid bolus administration	Direct entry into systemic circulation	Acute onset of CNS and cardiovascular collapse
System-related	Inadequate monitoring, limited staff training, absence of lipid emulsion stock	Delayed recognition and treatment	Preventable morbidity and mortality

(Table -1 Factors and risk)

Education and Prevention Strategies in Non-Anaesthetic Settings

The places where LAST now turns up surgical wards, emergency departments, procedure rooms, and outpatient clinics were not built around continuous monitoring and rapid drug

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rescue, and that gap shows in the way events unfold. (7, 11) Manufacturer and institutional dosing tables help, but they do not always account for comorbidity, overlapping routes, or the fact that several teams may be giving "small" doses that add up. (7, 11) A safer routine is simple and systematic: weigh the patient, calculate weight-based maxima, track cumulative doses in real time, and monitor continuously with ECG and pulse oximetry during administration and through a defined observation period. (9, 10) Unfortunately, audits and surveys suggest that these practices are inconsistently applied whenever anaesthetists are not directly overseeing care. (7, 11)

Closing the gap depends on people more than technology. (7, 11) Surveys show that many non-anaesthetist clinicians cannot reliably spot the early neurological cues of toxicity or recite the first steps of treatment, which is why regular, multidisciplinary simulation is worth the time. (7, 11) Short, scenario-based drills let ward, ED, and procedural teams rehearse recognition and role-split, draw up lipid under pressure, and practice the modified resuscitation sequence before a real patient needs it. (3, 10) Units that run these drills and keep a stocked LAST kit within reach respond faster and more calmly when the moment arrives. (9, 10)

Lipid therapy should feel familiar, not exotic. (9, 10) The recommended approach is straightforward: give 20% lipid emulsion as a 1.5 mL/kg intravenous bolus over about a minute, then start an infusion at 0.25–0.5 mL/kg/min, with a repeat bolus considered if instability persists within guideline totals. (9, 10) Crucially, teams outside anaesthesia should not wait for an anaesthetist to materialize before starting treatment, if lipid is not stocked on-site, a pre-agreed pathway must bring it and a trained responder within minutes. (9, 10) Making LAST a shared peri-operative responsibility, with clear escalation steps, in-date kits, and regular refreshers, is how ordinary clinical areas match the preparedness long cultivated in theatres. (7, 9, 10)

III. MANAGEMENT

When LAST strikes, the difference between a scare and a catastrophe is often measured in minutes and in how well the team follows a simple, rehearsed plan. (1, 7) The goals are straightforward stop further drug absorption, stabilize airway breathing circulation, and deliver specific therapy yet in non-anaesthetic environments these steps must often be led by the clinicians already at the bedside. (1, 7). Initial actions that change the outcomes.

Stop any ongoing local anaesthetic, call for help, and move immediately to protect the airway while correcting hypoxia and acidosis with high-flow oxygen and assisted ventilation as needed. (4, 10,33) Seizures are common and dangerous, treat them early with benzodiazepines, and use propofol sparingly only if required, mindful that it can deepen myocardial depression in an already unstable patient. (3, 5,34) Cardiac arrest and shock demand a modified approach: prefer small aliquots of epinephrine over large boluses, and avoid vasopressin, additional local anaesthetic, calcium-channel blockers, and beta-blockers in the acute phase because they can worsen arrhythmias or contractile failure in LAST. (9, 10)

Lipid emulsion therapy: Lipid therapy has changed the trajectory of severe LAST by doing two jobs at once sequestering lipophilic anaesthetics away from vital tissues and supporting cardiac energetics during recovery. (10, 11,35) The recommended protocol is simple: give 20% lipid emulsion as a 1.5 mL/kg intravenous bolus over about one minute, then continue with an infusion at 0.25–0.5 mL/kg/min until hemodynamics stabilizes; consider a repeat bolus if instability persists, within guideline total-dose limits. (1, 9, 10) In cardiac arrest, repeated boluses may be required with total dosing up to approximately 10 mL/kg over 30 minutes according to local policy and guideline ceilings. (1, 9) Preparedness is practical: every area that uses local anaesthetics should keep a clearly labelled "LAST box" with in-date lipid, dosing cards, a one-page algorithm, and an airway/ALS checklist so that treatment can begin before an anaesthetist arrives (7,11).

If hemodynamic remain unstable despite prompt airway management, seizure control, and lipid therapy, escalate early to extracorporeal support ECMO or cardiopulmonary bypass rather than persisting with ineffective cycles of chest compressions and vasopressors (3, 11).Extracorporeal support buys time for redistribution and metabolism, which is particularly relevant for highly lipophilic agents such as bupivacaine. (3, 11) Even after apparent recovery, admit to critical care and continue close observation, as delayed cardiovascular depression has been reported hours after initial stabilization.(6,8).

The most reliable advances will be cultural andsystem-level: routine simulation in wards, EDs, and procedural clinics, visible cumulative dose tracking across teams, monthly checks that every kit is present and in-date, and an escalation pathway that works after hours. (7, 9, 10) Institutions that treat LAST as a shared peri-operative responsibility rather than an anaesthesia-only problem recognize and treat faster, and their patients do better. (7, 9, 10).

Limitations

This review brings together evidence from a range of sources, recognizing that differences in study design make it difficult to combine precise incidence rates. We acknowledge that LAST cases outside hospitals can slip through the cracks, and many milder events may never be reported. To ensure fairness, all sources were independently reviewed and our conclusions aligned with trusted guidelines. Moving forward, more consistent reporting and collaborative registries will help the anesthesia community paint a clearer picture of LAST and strengthen patient safety efforts.

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Phase	Key Actions	Purpose	Notes
Pre procedure Preparation	Review medical history and comorbidities; calculate maximum allowable dose	Identify high risk patients and plan dose limits	Include cumulative dose if multiple clinicians are involved
Administration	Use incremental aspiration and minimal effective dose; prefer ultrasound guidance	Reduce chance of intravascular injection	Document agent, concentration, and total volume
Monitoring	ECG, SpO_2, verbal communication throughout procedure	Early detection of neurological or cardiac symptoms	Continue monitoring for delayed symptoms post procedure
Early Recognition	Stop anaesthetic administration immediately if symptoms appear	Prevent further absorption and reduce severity	Observe for CNS changes and altered cardiac rhythm
Lipid Emulsion Therapy	Administer 20 % lipid emulsion bolus 1.5 mL kg^{-1} followed by 0.25–0.5 mL kg^{-1} min^{-1} infusion	Acts as "lipid sink," reverses myocardial toxicity	Repeat bolus if instability persists; max 10 mL kg^{-1} in 30 min
Advanced Care	Prepare for airway management, seizures, and cardiovascular collapse	Sustain viability until redistribution occurs	Consider ECMO or transfer to ICU for refractory cases
Post event Management	Observe minimum 12–24 h for recurrent toxicity	Avoid delayed cardiovascular depression	Review and update institutional protocols
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(Table 2. Key Steps in LAST Prevention and Management)

IV. DISCUSSION

LAST has outgrown its old label as a rare complication of regional anaesthesia and now sits where much of everyday care happens wards, EDs, dental and procedure rooms, ambulatory theatres, and aesthetic clinics. (1, 7,36) This shift reflects the success of multimodal pain strategies and day-case surgery but also exposes a simple truth: practice expanded faster than systems, training, and rescue capability in many non-anaesthetist settings. (1, 7,36) Whoever gives the drug has changed and so has risks. (3, 11,37) Surgeons, dentists, emergency clinicians, and cosmetic practitioners now deliver most local anaesthetic outside theatres, which improves access but complicates safety oversight unless someone owns dose governance and monitoring end-to-end. (3, 11,38) Surveys and audits show uncomfortable gaps: many non-anaesthetist clinicians struggle to recognize early toxicity or recall lipid dosing, and these knowledge gaps translate into delayed diagnosis and treatment in areas without continuous monitoring. (5, 7,39)

Lidocaine appears so often in reports not because it is uniquely dangerous but because it is everywhere topical, infiltrative, tumescent, and intravenous making cumulative exposure the everyday hazard if teams do not add up doses across routes. (4, 6, 11) By contrast, bupivacaine and ropivacaine are less frequently involved but carry a steeper cardiotoxic profile when mishaps occur, owing to lipid solubility and myocardial persistence. (1, 8, 10) The practical fix is institutional, not heroic: visible cumulative-dose tracking, shared protocols, and clear handoffs between surgical, anaesthesia, and nursing teams. (7, 9,40). Outcomes have improved where lipid therapy is familiar and close at hand, it is the single intervention most likely to change a bad day into a near miss. (9, 10,41) Yet access remains patchy in EDs and outpatient clinics, and when kits are missing or expired the delay can erase the advantage lipid provides. (7, 11,42) Treating lipid rescue as a standard emergency protocol like defibrillation or anaphylaxis kits brings non-theatre areas up to the theatre standard patients assume. (7, 9, 10). The evidence base still lags practice. (12,43) Much of what is known comes from case reports, small series, and poison-centre analyses; these sources are vital but underestimate incidence outside hospitals and cannot supply denominators. (12,44) A recent NPDS analysis helps clarify trends fewer non-lidocaine poisonings and deaths after 2010, but more lidocaine-related events and fewer deaths in operating rooms with an increase linked to intravenous use yet even these data reflect reporting, not exposure. (5) The field needs multi-Centre registries, consistent definitions, and shared data to set safer dose limits, track outcomes, and evaluate prevention. (12,45)

Build simple, user-friendly dose calculators into the workflow, make cumulative dosing visible; keep an in-date lipid kit within minutes of wherever local anaesthetic is used, and rehearse the algorithm two or three times a year with the people who will respond. (7, 9, 10) Visual job aids and mobile prompts help non-anaesthetist teams in the moment, especially where turnover is high. (7, 9, 10) If the community can align education, technology, and standard protocols around these basics, LAST remains rare, if not, it becomes a preventable harm that keeps recurring in the same places. (1, 7,38)

V. CONCLUSION

LAST reminds clinicians that even familiar drugs carry real risk when they move beyond their traditional homes and into busier, less monitored spaces. (1, 7,35) Local anaesthetics are given on clinical wards, in dental and procedural suites, and across outpatient pathways, and preparedness has not always kept pace. (1, 7). The essentials are unchanged and widely achievable: calculate doses accurately, monitor continuously during and after administration, track cumulative exposure across teams, and keep a stocked lipid kit within reach. (3, 9, 10) These habits, reinforced by regular multidisciplinary simulation, make ordinary clinical areas as ready as operating theatres and shorten the time from first symptom to effective treatment. (3, 9, 10) LAST is not a problem for anaesthesiologists to solve alone, it is a shared clinical duty wherever local anaesthetics are used. (7, 11) Better education at the point of care, simple technology such as dose calculators and mobile job aids, and organizational readiness that does not depend on a single specialty being present. (7, 9, 10) If teams across departments can recognize early signs, initiate lipid therapy promptly, and work from the same playbook, LAST remains rare and increasingly preventable. (1, 7)

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