ANAESTHESIA PATIENT SAFETY ASSOCIATION

NEWSLETTER

AIR REPORT: THE SECOND

LAST: SPOT STOP.

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APSA

In this issue...

Some situations in anaesthesia demand everything from you at once. When local anaesthetic enters the bloodstream, or when something goes wrong in the neuraxial space, there is no time to look things up. The margin is narrow, the signs can deceive, and the window to act closes quickly. This edition of the APSA Newsletter takes a close look at both. The articles on Local Anaesthetic Systemic Toxicity and neuraxial complications are not just clinical reviews — they are an invitation to be better prepared than you were yesterday.

The incident reports are harder to read. That is precisely the point.

A gauze piece left behind during surgery. The time-out exists for this very reason — not as a bureaucratic checkbox, but as the last line of defence when the mind is occupied and the hands are moving. Two separate incidents of wrong drug injected intrathecally — two — because the intrathecal space does not forgive, and what goes in cannot come back. Look-alike ampoules, unlabelled syringes, and a single moment of distraction are, together, a dangerous combination. A machine malfunction traced to a scavenging system fault — a quiet but firm question to all of us: do we truly complete our pre-use checks, or do we simply go through the motions? And a positioning injury, one of the most underreported categories of perioperative harm, quietly attributed to the surgical condition long after the damage is done.

Each incident is shared anonymously. The commentary that follows is not about blame — it is about the only question that matters: what do we do differently next time?

We are grateful to the clinicians who reported these incidents. It takes honesty, and no small amount of courage, to share what went wrong. We thank our reviewers for their careful analysis, and our editorial and creative team for bringing this newsletter together with such dedication.

Read it carefully. Discuss it with your team. And the next time you are in that operating theatre — remember that safety is not a habit you develop once. It is a decision you make, every single time.

Dr Murali Thondebhavi
Project Lead, Anaesthesia Incident Registry

ONE WRONG AMPOULE AWAY — EVOKES THE THIN MARGIN OF ERROR

During an emergency lower segment caesarean section performed at night, neostigmine was inadvertently prepared in place of bupivacaine for spinal anaesthesia and administered without pre-injection verification. Absence of the expected spinal block prompted review, at which point the incorrect ampoule was identified. Unsupervised nursing drug preparation and the absence of an independent double-check at the point of administration were the principal contributing factors.

An error is a failure to execute an intended action correctly. A medication error is any error in the medication process, whether or not it results in patient harm. James Reason classifies human errors into slips, lapses, and mistakes — each reflecting a distinct failure in cognitive processing. In anaesthesia practice, drug errors can be life-threatening. Man, machine, medicine, and modus operandi are the principal contributory factors. The Institute of Medicine (IOM) reported that 44,000–98,000 patients die annually as a result of medical errors, a substantial proportion being medication-related. (1) These figures represent only the tip of the iceberg, as many incidents go unreported owing to fear of punitive action, absence of structured reporting systems, and underrecognition of near-miss events.

Jensen et al. described several strategies to prevent drug errors in the operating room. All syringes must be labelled immediately upon preparation. The label of every drug ampule or syringe should be carefully verified before drawing up and again before administration. Look-alike ampules and high-alert medications must be physically separated on the anaesthesia trolley. The anaesthesia provider who administers the drug should be the one who draws it up and labels the syringe.(2)

The five rights framework — right drug, right patient, right dose, right time, and right route — remains the foundational standard for safe drug administration and must be verified at every point of delivery.(3) Universal adoption of these practices is imperative to reduce preventable medication harm across all anaesthetic settings.

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LOOK ALIKE / SOUND ALIKE

IDENTICAL PACKAGING, FATAL CONSEQUENCES — HIGHLIGHTS THE SYSTEMIC FLAW

During spinal anaesthesia for an obstetric procedure, a wrong drug was administered intrathecally. Ampule similarity was identified as a primary contributing factor, with both preparation and administration steps lacking independent verification. Serious cardioneurological consequences followed. The absence of mandatory double-checking before neuraxial injection and the near-identical appearance of drug containers on the spinal trolley were the principal system failures identified.

Inadvertent intrathecal injection of an unintended drug is among the most serious complications in anaesthetic practice. Reported cases involving tranexamic acid, atracurium, potassium chloride, neostigmine, and various antibiotics illustrate the potentially catastrophic cardioneurological consequences — including refractory seizures, cardiovascular collapse, and death. No standardised management guidelines currently exist, reflecting both the rarity and the heterogeneity of these events.(1)

Root cause analyses consistently identify two principal contributing factors: human error at the point of preparation or administration, and physical similarity between drug containers. Drugs housed in near-identical ampules or vials stored in proximity on the neuraxial tray create conditions for inadvertent substitution — a phenomenon termed syringe swap when pre-drawn syringes are involved. The psychological toll on practitioners involved in such incidents should not be overlooked; in addition to potential regulatory consequences, these events can cause lasting professional and personal distress.(2,3)

Preventive strategies are well-described and readily implementable. Prefilled, single-blister-packed spinal drug units and dual-sterile-envelope packaging for local anaesthetics have been proposed to reduce handling errors.(1) At the institutional level, strict physical separation of look-alike containers, standardised and clearly labelled neuraxial trays, mandatory independent double-checking before any intrathecal injection, and systematic verbal verification as part of the spinal checklist are all effective, low-cost measures that merit universal adoption.(4)

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4. Kane MM, Sambou P, Ntab SO, Barboza D. Accidental Intrathecal Injection of Tranexamic Acid during Emergency Cesarean Section. EAS J Anaesthesiol Crit Care. 2025;7:258–261.

STOP AND
DOUBLE-CHECK

COUNTED OUT, LEFT IN: RETAINED SURGICAL ITEMS

During a total gastrectomy performed under general anaesthesia with a combined neuraxial technique, a gauze piece was retained intraabdominally. Abdominal closure was completed before the surgical gauze count was finalised, and the WHO Surgical Safety Checklist Sign-Out was not completed prior to closure.

Retained surgical items (RSIs), particularly abdominal packs (gossypiboma), are preventable 'never events' associated with significant morbidity, reoperation, and medicolegal consequences.[1] Although traditionally regarded as a surgical responsibility, the anaesthesiologist plays a vital complementary role in prevention through sustained vigilance, effective communication, and disciplined adherence to safety protocols.

The risk of RSIs is heightened during emergency surgeries, unplanned procedural changes, obesity, and major haemorrhage.[1][2] As a constant intraoperative presence, the anaesthesiologist is uniquely positioned to recognise these high-risk scenarios and reinforce the importance of accurate surgical counts. Active participation in the WHO Surgical Safety Checklist — including the Time Out and Sign Out stages — ensures that sponge and instrument counts are audibly confirmed before wound closure.[3]

Anaesthesiologists also directly manage countable items such as throat packs, which must follow strict insertion and removal tracking protocols.[5] In damage-control surgeries where packs may be intentionally retained, meticulous documentation, unambiguous communication, and structured handover during transitions of care are essential safeguards. Communication failures remain a leading contributor to adverse surgical events, underscoring the anaesthesiologist's critical role in maintaining team situational awareness.[6]

When a count discrepancy arises, the anaesthesiologist must ensure haemodynamic stability during radiographic evaluation and advocate for thorough investigation before closure proceeds. Prevention of retained surgical items is ultimately a shared team responsibility, with the anaesthesiologist serving as an indispensable safeguard through vigilance, communication, and collaborative

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2. Steelman VM, Shaw C, Shine L, Hardy-Fairbanks AJ. Retained surgical sponges: a systematic review and meta-analysis of risk factors. J Healthc Qual. 2019;41(4):214–222.
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4. Joint Commission. Sentinel Event Alert Issue 51: Preventing unintended retained foreign objects. Oakbrook Terrace, IL: Joint Commission; 2013.
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HEAD DOWN AND BREATHLESS: THE HIDDEN RISK OF STEEP TILT

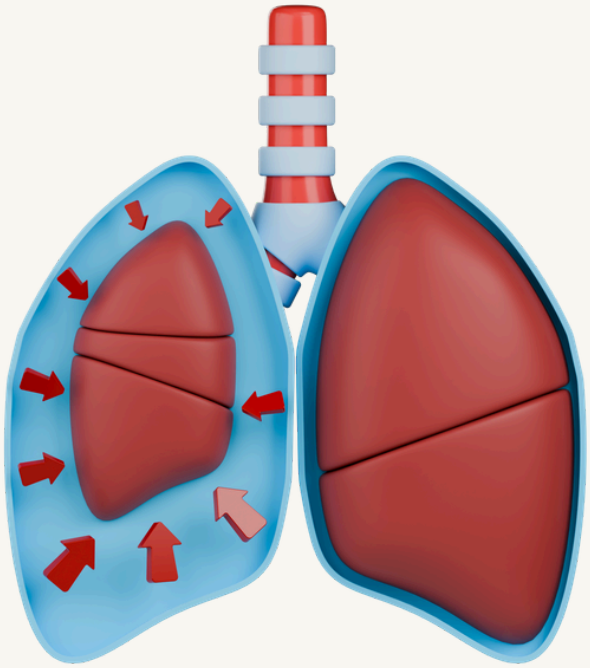
During laparoscopic surgery with steep Trendelenburg positioning, pneumothorax developed intraoperatively. Rising airway pressures were initially attributed to the effects of positioning and pneumoperitoneum, which delayed recognition. Haemodynamic compromise subsequently developed. Diagnostic anchoring to a positional aetiology and delayed exclusion of barotrauma were identified as key contributing factors.

Pneumothorax during Trendelenburg positioning is a rare but potentially life-threatening complication, encountered primarily during laparoscopic and robotic surgeries.[1] The combination of steep head-down tilt and carbon dioxide (CO_2) pneumoperitoneum imposes significant physiological stress — the diaphragm is displaced cephalad, functional residual capacity is reduced, and peak airway pressures required for adequate ventilation are substantially elevated.[2,3]

Two principal pathways contribute to this complication:
Barotrauma due to elevated inspiratory pressures can cause alveolar rupture, with air tracking along the bronchovascular sheath into the mediastinum and subsequently the pleural space.[2,4] Capnothorax: CO_2 may enter the pleural cavity through congenital diaphragmatic defects, such as those at the oesophageal hiatus, or through iatrogenic breaches during surgery.[1,2]

The Trendelenburg position, frequently adopted for internal jugular vein cannulation, may additionally bring the lung cupola closer to the needle trajectory, increasing the risk of direct pleural puncture.[2]

Early recognition is critical, heralded by a sudden rise in airway pressures, unexplained hypoxia, or haemodynamic instability.[1,3] Key preventive strategies include, lung-protective ventilation: Low tidal volumes and limited peak inspiratory pressures to mitigate barotrauma, [3,4] Maintaining the minimum effective pneumoperitoneum pressure and reducing the degree of head-down tilt wherever clinically feasible[1,5] and real-time imaging for all central venous access to minimise the risk of accidental pleural injury.[2]



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2. Damas AM, et al. Capnothorax During Laparoscopy in Trendelenburg Position. Acta Med Port. 2020;33(3):202-203.
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UP TOO SOON, DOWN TOO FAST: THE HIDDEN RISKS OF EARLY MOBILISATION

Following a prolonged head and neck surgery with free flap reconstruction, an acute cerebral infarction was identified on postoperative day 1. The intraoperative course was documented as uneventful, with no recorded episodes of hypotension. No intraoperative precipitant was identified.

Early postoperative mobilisation is a cornerstone of enhanced recovery after surgery (ERAS) protocols, with well-established benefits including reduced venous thromboembolism, fewer pulmonary complications, decreased incidence of delirium, and shorter hospital stay. However, the risks associated with early ambulation remain underappreciated and demand structured perioperative planning. Potential complications include patient falls, orthostatic hypotension, haemodynamic instability, and inadvertent dislodgement of intravenous lines, urinary catheters, surgical drains, or monitoring devices.

Several factors may compromise the safety of early ambulation. Residual effects of sedatives, opioids, and neuromuscular blocking agents impair both motor coordination and cognitive function in the immediate postoperative period. Fluid shifts, surgical blood loss, and concurrent vasodilatory medications contribute to postural hypotension. Postoperative delirium, particularly prevalent in elderly patients, further amplifies fall risk. The presence of multiple indwelling devices restricts mobility and heightens the likelihood of accidental dislodgement during initial mobilisation attempts.

The anaesthesiologist's contribution to safe mobilisation extends across the entire perioperative continuum. Preoperative assessment should incorporate validated frailty scoring, nutritional evaluation, cognitive screening, and baseline functional capacity. Intraoperatively, avoidance of long-acting sedatives, complete reversal of neuromuscular blockade confirmed by quantitative monitoring, opioid-sparing regional techniques, multimodal analgesia, and goal-directed fluid therapy collectively facilitate earlier and safer functional recovery. Safe early mobilisation ultimately depends on a coordinated, multidisciplinary approach involving anaesthesiologists, surgeons, nursing staff, and physiotherapists, underpinned by clear communication and structured handover across all points of care transition.

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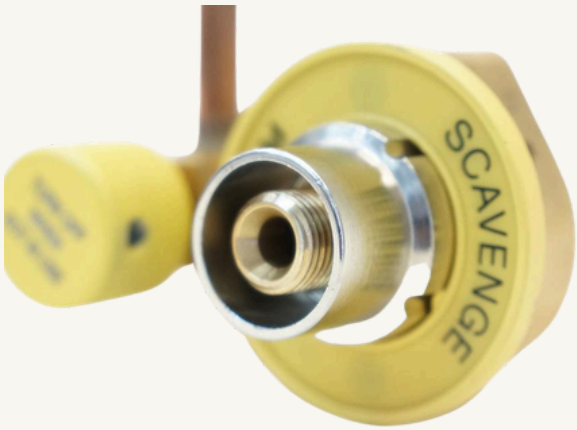
OPEN VALVE, SILENT FAILURE: HOW A SMALL OVERSIGHT COMPROMISES VENTILATION

During elective orthopaedic surgery, the anaesthesia machine was replaced intraoperatively as the airway pressure was not receding below 10 cmH_2O with APL valve set at minimum. The scavenging system was subsequently identified as faulty and corrected promptly, averting ventilation compromise.

The adjustable pressure limiting (APL) valve is a critical safety component of the anaesthesia breathing circuit, regulating circuit pressure during manual and spontaneous ventilation. Despite widespread adoption of automated pre-anaesthesia machine checks, APL valve dysfunction continues to be reported and carries the potential for unrecognised ventilation failure with serious patient consequences.

Several distinct failure mechanisms have been described. Extrinsic obstruction — including entrapment of capnography sampling lines, temperature probe cables, or monitoring leads between the APL control knob and valve base — can prevent complete valve closure during manual ventilation. Physical deterioration of internal valve components, such as cracks in the valve body or degradation of O-rings and valve seats, may cause uncontrolled pressure loss and failure to sustain circuit pressures. The APL valve may also be inadvertently left fully open or incorrectly configured owing to unfamiliarity with a particular machine design, leading to complete manual ventilation failure.

Any unexpected difficulty in transitioning from mechanical to manual ventilation should prompt immediate inspection of the APL valve alongside a systematic circuit leak assessment. Monitoring lines and cables must be routinely routed clear of the APL valve assembly. Automated machine checks must never be regarded as a substitute for hands-on functional verification. The traditional pre-use check — occluding the Y-piece, closing the APL valve, compressing the reservoir bag, and confirming both a pressure rise and an audible release on valve opening — remains a simple, reliable, and indispensable safeguard that must precede every anaesthetic. The check should include the scavenging system too.



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FORGOTTEN BUT NOT FORGIVEN: THE TOURNIQUET THAT STAYED BEHIND

A tourniquet applied for peripheral intravenous cannulation in the operating theatre was not removed following successful venous access. The cannulated limb was subsequently positioned beneath surgical drapes, obscuring the tourniquet from view. The error was identified during intraoperative assessment of the covered limb, and timely recognition averted ischaemic sequelae.

A tourniquet inadvertently left in place following intravenous cannulation is a preventable procedural error with potentially serious consequences. Prolonged venous occlusion causes progressive distal congestion, manifesting initially as pain, oedema, and vascular engorgement. If constriction persists, arterial inflow may be compromised, progressing to nerve injury, compartment syndrome, venous thrombosis, tissue necrosis, and potentially irreversible limb damage.[1] Systemic complications, including rhabdomyolysis and acute kidney injury, have been reported. Retained tourniquets are formally classified as 'never events,' reflecting a fundamental lapse in procedural vigilance.

Several contributing factors predispose to this error: poor tourniquet visibility due to colour, size, or concealment beneath surgical drapes; clinician distraction during high-workload phases; communication gaps between team members; cannulation at non-standard access sites; use of improvised tourniquet substitutes; and inadequate ambient lighting.[2] In the operating theatre, the risk is substantially heightened when a cannulated limb is positioned and draped before the tourniquet has been removed.

Prevention demands both individual accountability and robust system-level safeguards. The tourniquet must be released immediately upon successful venous access and before the cannula is secured. Use of brightly coloured, visually conspicuous tourniquets improves detection under drapes and dim lighting. Incorporating a mandatory 'tourniquet removed' confirmation step within standardised IV insertion checklists provides a dependable structural safeguard. Verbal closed-loop confirmation between team members and active engagement of conscious patients to report persistent tightness or discomfort offer additional layers of protection.[3] Consistent application of these measures can effectively eliminate this category of preventable harm.

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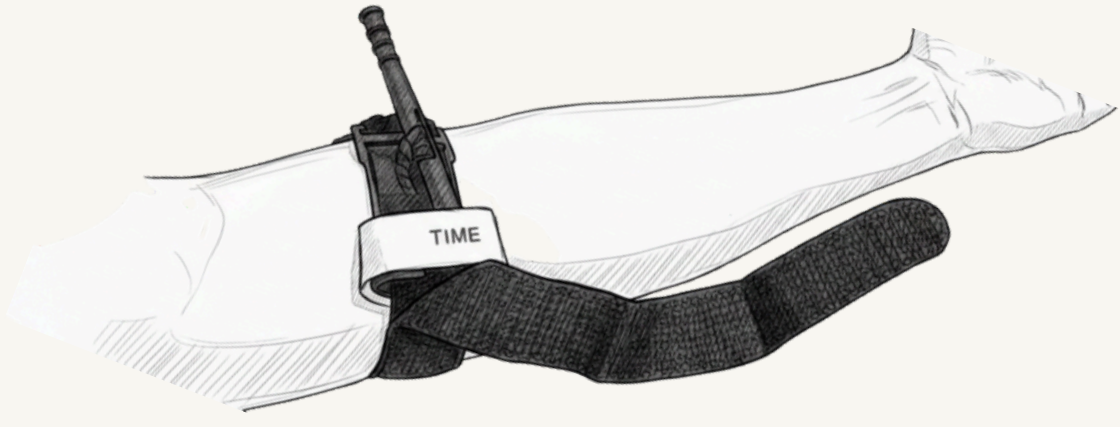
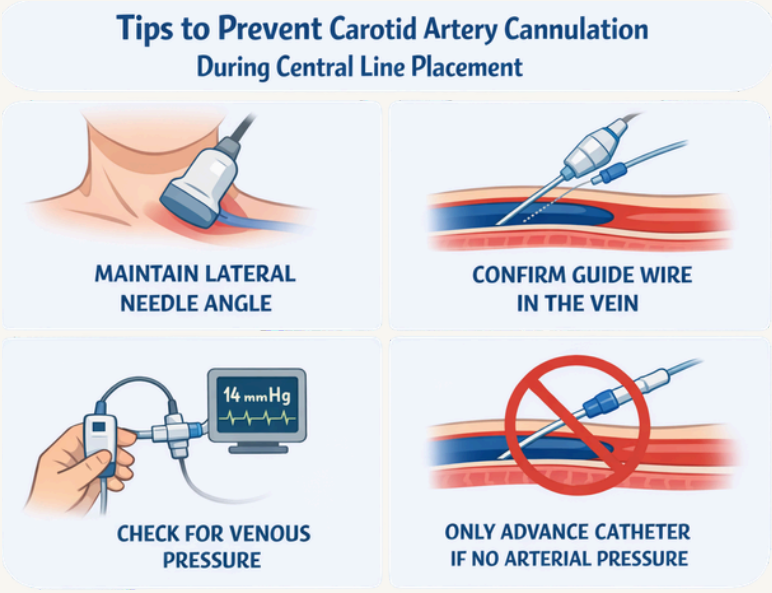


SEEING THE VEIN, HITTING THE ARTERY: WHEN ULTRASOUND GUIDANCE FALLS SHORT

During central venous catheterisation of the left internal jugular vein in a renal transplant recipient, the carotid artery was inadvertently punctured by a senior resident under ultrasound guidance. The needle is believed to have shifted from the vein to the carotid artery during guide wire placement. A dilator and catheter were advanced before arterial placement was recognised, and a large haematoma developed on removal. Medial needle angulation and guide wire advancement without arterial pressure confirmation were identified as the key technical contributors.

Ultrasound guidance for central venous catheterisation has markedly improved procedural safety, reducing the number of insertion attempts, time to cannulation, and associated complications. However, ultrasound does not eliminate the risk of inadvertent arterial injury. Carotid artery puncture remains a recognised complication even under direct real-time imaging, and advancement of a dilator or large-bore catheter into the carotid can result in catastrophic consequences — including stroke, major haemorrhage, pseudoaneurysm, and arteriovenous fistula. [1]



Two principal ultrasound approaches are employed. The short-axis (out-of-plane) view permits simultaneous visualisation of the carotid artery and internal jugular vein, aiding assessment of their anatomical relationship, but provides limited continuous tracking of the advancing needle tip. The long-axis (in-plane) view enables continuous visualisation of the full needle shaft and tip, enhancing procedural precision; however, simultaneous arterial and venous imaging is not possible, necessitating accurate pre-cannulation vessel identification.

Evidence suggests the long-axis approach is associated with fewer needle redirections, shorter cannulation time, and a lower incidence of posterior wall punctures.[2] Regardless of the approach chosen, continuous needle tip visualisation must be maintained throughout the procedure. Medial needle angulation during cannulation or guide wire advancement is a well-recognised risk factor for carotid injury and must be actively avoided — the needle should be directed perpendicular to, or laterally away from, the artery. Ultrasound guidance must complement, never replace, sound anatomical knowledge and disciplined procedural technique. [3]

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2. Vogel JA, et al. Is long-axis view superior to short-axis view in ultrasound-guided central venous catheterization? Crit Care Med. 2015;43(4):832-839.
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NEURAXIAL COMPLICATIONS REVIEW

Neuraxial anaesthesia encompassing spinal, epidural and combined spinal epidural (CSE) remains an important method in the practice of anaesthesiology due to its various advantages, including minimal equipment requirements, simplicity of drug administration, expedited postoperative recovery, and applications in perioperative and labour analgesia.

Neuraxial complications following neuraxial anaesthesia are extremely rare, but when they occur, they lead to life-altering consequences. That is why it is important to focus on this topic for safe anaesthesia practice. Incidence of neuraxial injury associated with regional anaesthetic techniques varies widely- so much so that it's difficult to cite a meaningful overall risk for injury. Out of all neuraxial complications described in the literature, spinal-epidural hematoma can lead to permanent paraplegia due to the expanding nature of the hematoma. In the same manner, an epidural abscess also leads to serious and devastating consequences. The incidence of spinal epidural hematoma is 1:2,00000 in obstetric epidural, 1:22,000 in hip fracture surgeries, and 1:3,600 in patients undergoing knee arthroplasty. The incidence of spinal epidural hematoma increases with advanced age, coagulation abnormalities, female gender, and type of surgery.[1,2,3] A nuanced understanding of these risks, along with adherence to evolving guidelines and evidence-based practice, is essential for enhancing patient safety and outcomes.

This article provides a structured overview of neuraxial anaesthesia complications along with a recent literature review and practical strategies for prevention and management.

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NEURAXIAL COMPLICATIONS LEAD TO LIFE-ALTERING CONSEQUENCES

1. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, et al. The second American Society of Regional Anesthesia and Pain Medicine practice advisory on neurologic complications associated with regional anesthesia and pain medicine. Reg Anesth Pain Med. 2015;40(5):401–30.

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Neuraxial anaesthesia-related complications may be classified according to their clinical nature as follows:

1. Hemodynamic and physiological complications: hypotension, bradycardia, high or total spinal anaesthesia, and rarely cardiac arrest.

2. Technical or procedural complications: failed spinal anaesthesia, inadequate or patchy block, traumatic needle placement, and accidental dural puncture.

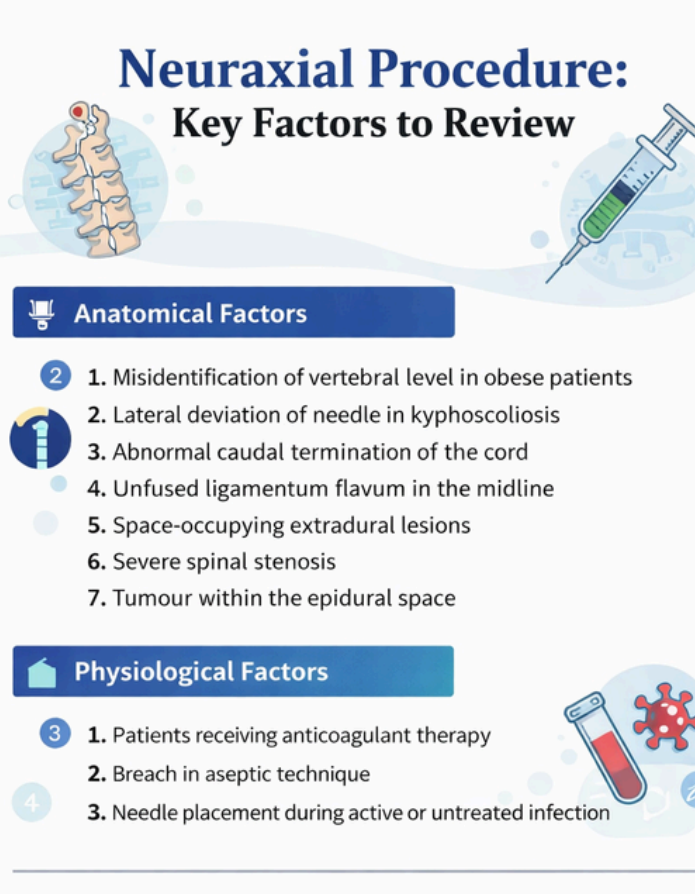
3. Neurological complications: post-dural puncture headache, transient neurological symptoms, nerve root injury, cauda equina syndrome, spinal cord injury, and persistent neurological deficit.

4. Hemorrhagic/compressive complications: spinal epidural hematoma and other neuraxial hematomas causing neural compression.

5. Infectious complications: epidural abscess and meningitis.

6. Drug-related complications: local anaesthetic systemic toxicity.

Clinically, these complications may also be considered according to timing of presentation as immediate, early, or delayed. Immediate complications, such as hypotension, bradycardia, high spinal block, or local anaesthetic systemic toxicity, occur within minutes and require prompt recognition and treatment. Early or delayed complications, particularly new neurological deficits, severe back pain, radicular symptoms, fever, or bladder/bowel dysfunction, should prompt urgent evaluation to exclude compressive or infective neuraxial pathology.[1,4]



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Neurological Complications Following Neuraxial Anaesthesia: Case-based Clinical Insights

Case Heading	Description	Lesson
Delayed lethal CNS toxicity after low dose intrathecal bupivacaine administration (Frontiers in Anaesthesiology 2023)	55-year-old male received 10 mg intrathecal bupivacaine for lower limb surgery. Developed nausea, agitation, paraesthesia, myoclonus, progressing to loss of consciousness. CT/MRI normal; EEG showed spike activity (C3, C4, P3, P4, T5). Managed with sedation and valproate, with complete recovery.	CNS toxicity to local anaesthetic though very rare should be vigilantly cared for in clinical settings it co relates directly with the potency and lipid solubility of the local anaesthetic. Drugs such as bupivacaine with higher potency and lipid solubility have higher risk of causing CNS toxicity.
CES after Spinal Anaesthesia in Spinal Stenosis (2015, RAPM)	Two patients with occult spinal stenosis developed neurologic deterioration after spinal anesthesia; one recovered, the other required decompression	Spinal stenosis is an under-recognized risk factor; careful selection is essential in symptomatic patients.
Reversible CES after Combined Spinal–Epidural (2011, Can J Anaesth)	CES developed after CSE but resolved completely over time.	Early recognition and supportive care can lead to full recovery.
Persistent CES in Obstetric Patient (2015, J Clin Anesth)	Post-caesarean patient developed persistent neurological deficit after CSE	Severe complications can occur even in young, healthy patients
CES in Patient with Pre-existing Neurological Disease (2017)	Post-polio patient developed CES after neuraxial anesthesia without structural cause	Absence of intraoperative events does not exclude serious complications
CES after Epidural in Orthopedic Surgery (2018)	Two cases developed CES after epidural anesthesia; imaging suggested arachnoiditis/structural changes.	Degenerative spine and prior pathology increase risk.
CES after Difficult Spinal with Epinephrine	CES occurred after multiple puncture attempts and use of vasoconstrictor.	Difficult spinal and vasoconstrictors may reduce neural perfusion.
Spinal Cord Infarction after Epidural + GA (2017, JA Clinical Reports)	Patient developed paraplegia with MRI-confirmed cord infarction without compression.	Hypoperfusion alone can cause severe neurological injury.
Anterior Spinal Artery Syndrome after Spinal (2015, J Clin Anesth)	Post-spinal hypotension led to anterior cord syndrome in obstetric patient.	Maintaining hemodynamics is critical to prevent ischemic injury.

Case Heading	Description	Lesson
CES with Tarlov Cyst (2023, BMC Anesthesiology)	CES occurred after CSE; imaging revealed previously undiagnosed Tarlov cyst.	Occult spinal lesions significantly increase vulnerability
CES with Arachnoiditis (2018, Medicine)	Delayed neurological deficit with cauda equina root enhancement on MRI.	Inflammatory/chemical injury should be considered when no compression is seen.
Acute CES after Spinal Anaesthesia (2025, A&A Practice)	Obstetric patient developed acute CES with incomplete recovery despite treatment.	Early MRI and multidisciplinary management are essential but may not reverse injury.
Spinal Cord Infarction due to Hyperlordosis (BJA)	Neurologic deficit developed after prolonged surgery in hyperlordotic position.	Positioning can critically reduce spinal cord perfusion
Paraplegia in Undiagnosed OPLL/Stenosis (2024)	Severe neurological deficit after spinal anesthesia revealed occult canal compromise.	Undiagnosed stenosis can convert minor insult into major injury.
Accidental epidural injection (2025, A&A Practice)	Accidental epidural chlorhexidine administration led to acute neurological symptoms with incomplete recovery.	Safety systems reduce but do not eliminate wrong-drug errors
Fatal arachnoiditis following accidental intradural injection of chlorhexidine (2026, RAPM)	A recent fatal case reported accidental intradural injection of chlorhexidine, leading to rapidly progressive arachnoiditis and neurological deterioration, culminating in death.	Intradural exposure causes catastrophic neurotoxicity with high mortality

SPINAL STENOSIS

Spinal stenosis may result from narrowing of the spinal canal due to hypertrophy of the ligamentum flavum, bony overgrowth, or vertebral degeneration, particularly in osteoporotic females, as well as herniation of the nucleus pulposus. Patients with spinal stenosis may harbour clinical or subclinical neurological deficits that can be unmasked or exacerbated during or following neuraxial intervention.[1,3]

SPINAL CORD BLOOD FLOW

Based on previous case reports, prolonged hypotension and hypoperfusion may lead to spinal cord ischaemia or infarction. The risk of ischaemia increases when hypotension is associated with additional factors such as vascular stenosis, embolism, or extreme positioning (e.g., hyperlordosis). Recent studies recommend the prophylactic use of vasopressor infusions to prevent hypotension and maintain adequate spinal cord perfusion.[1,2,3]

SPINAL/ EPIDURAL HEMATOMA

Spinal haematoma is a neurological emergency that, if not promptly recognised and treated, may result in permanent neurological deficit. It is strongly associated with anticoagulant therapy and underlying coagulopathy. Consensus recommendations emphasise strict adherence to the timing of neuraxial procedures in relation to anticoagulant dosing. Early diagnosis with imaging and prompt surgical decompression within 6–12 hours are crucial for favourable neurological outcomes.[5,6]

CAUDA EQUINA SYNDROME

Cauda equina syndrome manifests as bladder and bowel dysfunction with bilateral lower leg motor and sensory deficit. It can be due to neural element compression, maldistribution of local anaesthetic, or exposure of neural elements to higher local anaesthetic doses.

LOCAL ANAESTHETIC NEUROTOXICITY

Spinal anaesthesia is commonly administered by intrathecal administration of local anaesthetic into the intrathecal space. It is not common for local anaesthetic systemic toxicity affecting the central nervous system and cardiovascular system to occur in this scenario, but it is rarely encountered in spinal anaesthesia.

ARACHNOIDITIS

Inflammatory condition of arachnoid matter that can lead to radiculopathy, pain, sphincter dysfunction and paralysis. It can occur due to infection, surgery, chemical insult and even with blood patches.[7] Iatrogenic causes have been documented, most notably following oil-based myelography or blood patch, and sometimes after simple puncture. However, accidental injection of antiseptic agents, such as chlorhexidine, into the epidural or dural spaces remains exceedingly rare and under-reported, despite its devastating consequences.

5. *Pozza DH, Tavares I, Cruz CD, Fonseca S. Spinal cord injury and complications related to neuraxial anaesthesia procedures: a systematic review. Int J Mol Sci. 2023;24(5):4665.*

6. *Peterson W, Martin R, Arnold D. Delphi consensus recommendations for neuraxial anesthesia in adults with platelet disorders and coagulation defects: communication from the ISTH SSC Subcommittee on von Willebrand Factor. J Thromb Haemost. 2025;23:2039–49.*

7. *Doat-Sarfati V, Mattei S, De Crouy AC, Leclercq D, Weiss N, Psimaras D, et al. Fatal arachnoiditis following accidental intradural injection of chlorhexidine: a case report and literature review. Reg Anesth Pain Med. 2026;rapm-2026-107636.*

TRANSIENT NEUROLOGICAL SYMPTOMS (TENS)

Transient neurological symptoms (TNS) are defined as symmetrical bilateral pain in the back or buttocks, or pain radiating to the lower extremities, following recovery from spinal anaesthesia.[8] Symptoms typically appear within 24 hours of recovery and resolve completely within 2 days without any sequelae. TNS has traditionally been associated with lignocaine, but has also been observed with mepivacaine, hyperbaric bupivacaine, and others. Recent literature suggests that the occurrence of TNS is additionally influenced by intraoperative patient positioning. Non-steroidal anti-inflammatory agents such as ketorolac, as well as intravenous corticosteroids such as dexamethasone — commonly used for the prevention of postoperative nausea and vomiting — have been found helpful in reducing the incidence of TNS. The choice of local anaesthetic also plays a role in prevention; ropivacaine, levobupivacaine, bupivacaine, and prilocaine are associated with a lower incidence of TNS compared to lignocaine.

POST-DURAL PUNCTURE HEADACHE

Post-dural puncture headache (PDPH) is a type of headache that occurs within 5 days after a lumbar puncture due to leakage of cerebrospinal fluid from the dural puncture site. It is often associated with neck stiffness or hearing-related symptoms and usually resolves on its own within 2 weeks or after treatment with an epidural blood patch.

Recent literature highlights the importance of patient positioning during spinal anaesthesia in relation to post-dural puncture headache (PDPH). The lateral decubitus position is more effective than the sitting position in reducing the incidence of PDPH. Needle size and type also significantly influence the likelihood of PDPH; smaller-gauge needles are associated with a lower incidence. Additionally, the use of non-cutting (pencil-point) needles further reduces the risk. The number of attempts is directly correlated with the incidence of PDPH, with a higher number of attempts associated with a greater likelihood of its occurrence. This is usually self-limiting and resolves without any sequelae.Conservative management includes hydration, caffeine, and analgesics. Epidural blood patch is a definitive treatment in the management of unintentional dural puncture and refractory PDPH. Epidural infusion of hydroxyl ethyl starch was also found to be equally efficacious as prophylactic blood patch but however, some of the studies showed that epidural blood patch is superior in reducing the duration of hospital stay. Novel application of fibrin glue showed promising results in reducing the duration of symptoms due to PDPH. Trials demonstrated that sphenopalantine ganglion block (SPG) and greater occipital nerve block (GONB) are also helpful in reducing the symptoms and are safer and less invasive than epidural blood patch.[9,10]

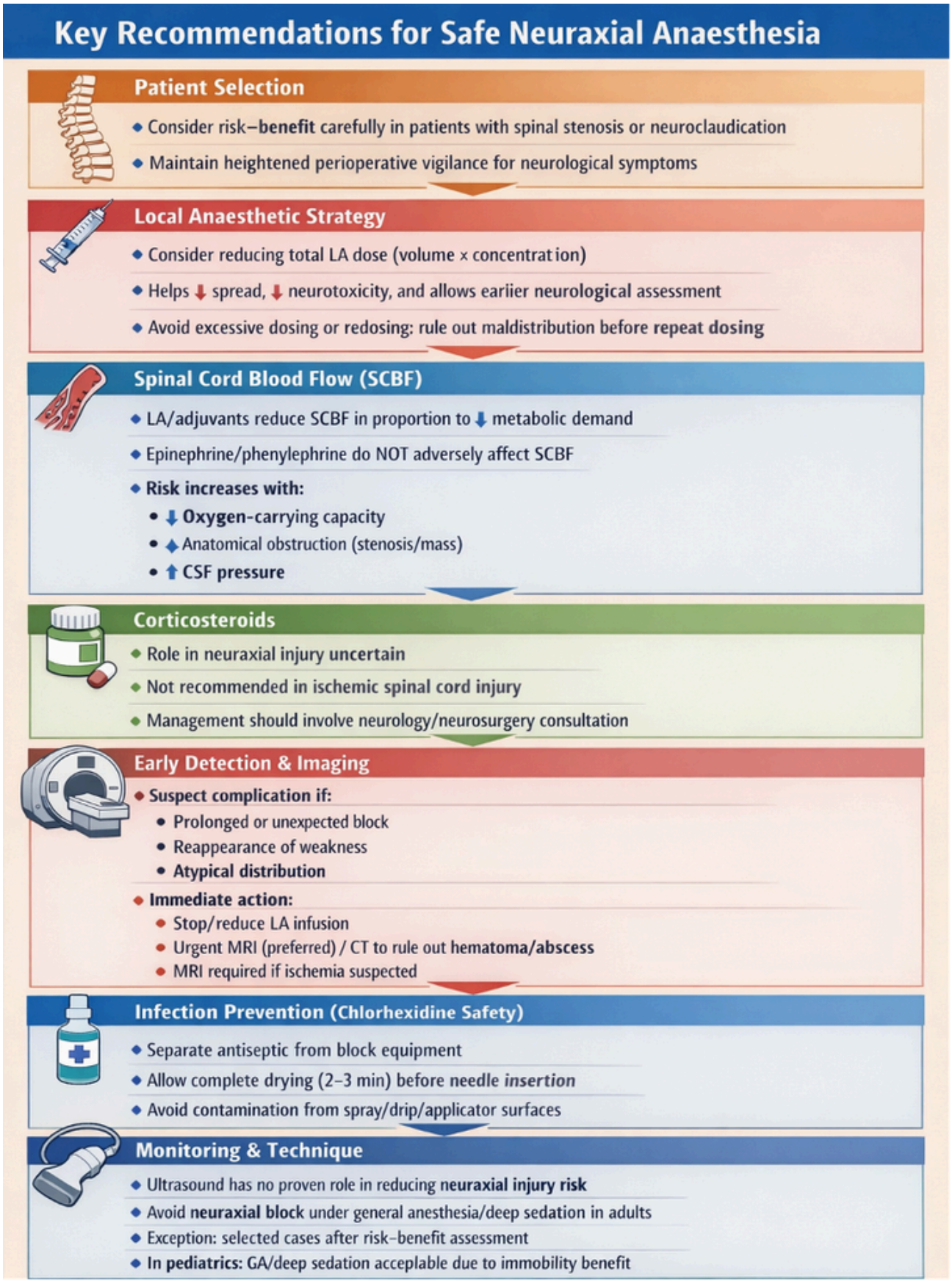
8. *Forget P, Borovac JA, Thackeray EM, Pace NL. Transient neurological symptoms (TNS) following spinal anaesthesia with lidocaine versus other local anaesthetics in adult surgical patients: a network meta-analysis. Cochrane Database Syst Rev. 2019;12(12):CD003006.*

9. *Uppal V, Russell R, Sondekoppam R, et al. Consensus practice guidelines on postdural puncture headache from a multisociety, international working group: a summary report. JAMA Netw Open. 2023;6(8):e2325387.*

10. *Alatni RI, Alsamani R, Alqefari A. Treatment and prevention of post-dural puncture headaches: a systematic review. Cureus. 2024;16(1).*

Neuraxial anaesthesia remains an indispensable tool in the armamentarium of modern anaesthetic practice and is considered to be the chosen option for opioid sparing anaesthesia practice. Neuraxial complications are rare in modern routine anaesthesia practice. Though uncommon, they can cause significant morbidity and even mortality in the short and long run. Recent literature suggests that the complications are preventable. Early recognition is critical in prevention and treatment of complications. Adherence to guidelines on various aspects like anticoagulants usage and dose should be strictly adhered to in order to improve the outcomes and safety.

IN NEURAXIAL ANAESTHESIA, SAFETY LIES NOT MERELY IN TECHNIQUE, BUT IN ANTICIPATION, VIGILANCE AND TIMELY INTERVENTION.



FUTURE DIRECTIONS

1. Ultrasound guidance for improved accuracy
2. Artificial intelligence and simulation training
3. Refinement of drug regimens to minimise neurotoxicity
4. Development of international registries for complication tracking.

LOCAL ANAESTHETIC SYSTEMIC TOXICITY (LAST): RECOGNITION AND MANAGEMENT

Local Anaesthetic Systemic Toxicity (LAST) refers to the effects, signs, and symptoms that systemic absorption of Local Anaesthetics (LAs) has on the body or inadvertent intravascular injections. LA's are administered in clinical practice by a wide range of providers, including anaesthesiologists, surgeons, emergency medicine physicians, dentists, and others. Despite their widespread use, awareness of LAST and knowledge of its management remains inadequate [1,2]. Prompt recognition and appropriate management of LAST is essential to minimize the risks associated with regional anaesthesia. However, due to its rare occurrence, the diagnosis may be missed and treatment might be delayed.

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PROMPT RECOGNITION AND APPROPRIATE MANAGEMENT OF LAST IS ESSENTIAL

1. Collins J. Awareness of local anaesthetic toxicity issues among hospital staff. Anaesthesia 2010; 65:960.

2. Sagir A, Goyal R. An assessment of the awareness of LA systemic toxicity among multi-specialty postgraduate residents. J Anesth 2015; 29:299.

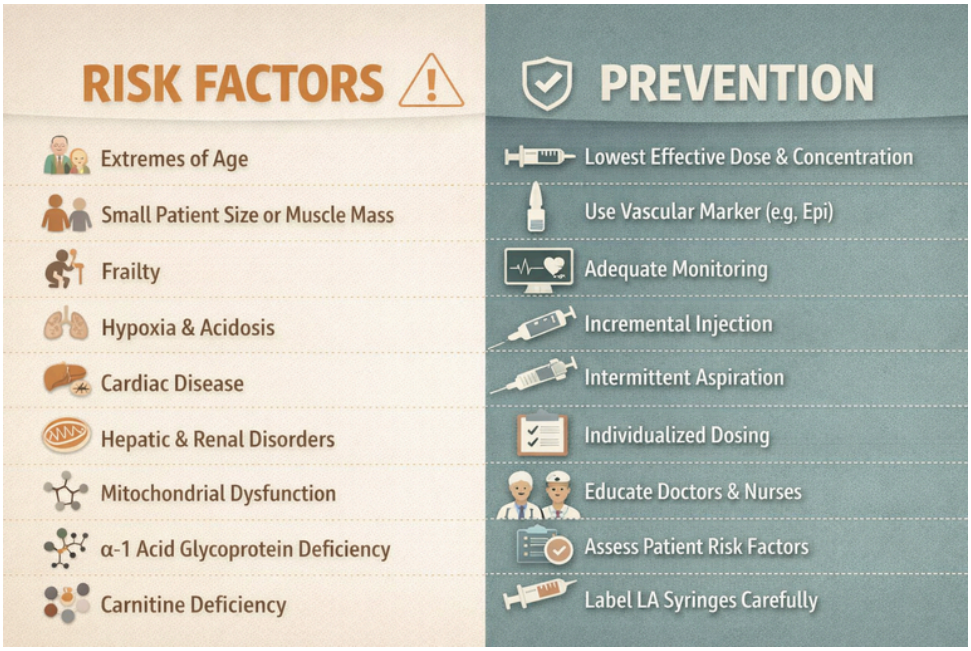
Based on recent literature, the incidence of LAST varies but is relatively low, ranging from 2 to 2.8 per 10,000 patients [3]. Individuals with organ disease or those at the extremes of age are more vulnerable. Increased cardiac output and decreased alpha-1 acid glycoprotein content in pregnancy increase the risk of LAST due to increased systemic abruption and peak serum concentration [4]. While LAST is less common in children than in adults overall, it affects about 8 out of every 100,000 injections in paediatric patients [5], with children under three years old being the most impacted. Local anaesthetic clearance is slowed in older individuals due to reduced organ perfusion and impaired hepatic function, which raises the risk even more. Additionally, end-organ dysfunction raises the risk of LAST in all age groups. But even this may understate the actual incidence due to the possibility of underreporting and misdiagnosis.

RISK OF LAST: CONTRIBUTING FACTORS AND THEIR MITIGATION

The clinical severity and the risk of LAST depends on several factors: the type of LA used, it's plasma concentration, and the patient's physiological condition and co-morbidities. The peak plasma concentration is influenced by the total dose administered, the rate of injection, the injection site, the administration technique, and patient-specific factors affecting the pharmacokinetics of the drug—namely its distribution, metabolism, and excretion.[6]

Additionally, the pharmacodynamics of LAs vary among individuals. Certain groups are more susceptible to increased toxicity at similar blood or tissue concentrations. These at-risk groups include patients with pre-existing heart disease, frailty, low muscle mass, very young or elderly age, impaired liver perfusion and function, decreased plasma α-1 acid glycoprotein levels, or acidosis. Acidosis, in particular, shifts the binding equilibrium unfavorably, increasing the concentration of free LA in plasma.

Because of these multiple interacting factors, no single preventive measure is sufficient. Table 1 summarizes risk factors and potential strategies to reduce the risk of LAST [6,7].



4. Mock ND, Griggs KM, Mileto LA. LA Systemic Toxicity during Labor, Birth, and Immediate Postpartum: Clinical Review. 2021 Nov-Dec 01MCN Am J Matern Child Nurs. 46(6):330-338.

5.Lee SH, Kim S, Sohn JT. Lipid emulsion treatment for LA systemic toxicity in pediatric patients: A systematic review. Medicine (Baltimore). 2024 Mar 15;103(11):e37534.

6. Macfarlane AJR, Gitman M, Bornstein KJ, El-Boghdadly K, Weinberg G. Updates in our understanding of local anaesthetic systemic toxicity: a narrative review. Anaesthesia. 2021 Jan;76 Suppl 1:27-39. doi: 10.1111/anae.15 282. PMID: 33426662.

7. Rupnik, Barbara, Nitish Aggarwal, Michael R. Fettiplace and Marina Gitman. "LA Systemic Toxicity (LAST) Revisited: A Paradigm in Evolution." (2020).

CLINICAL PRESENTATION

LAST is frequently overlooked and like many unfavorable consequences, underreported. Recent evidence shows that "atypical LAST," which includes modest indications such as cardiovascular dysfunction alone. LAST causes a wide range of symptoms and signs of CNS and CV toxicity (Table 2). These can be mild to severe, and they can occur alone or in combination. Exclusive CNS symptoms occur in almost half of documented cases, whereas mixed CNS and CV symptoms in about one-third, and isolated CV symptoms in the rest [8]. Many of these events occurred during general anaesthesia or deep sedation, making CNS toxicity difficult to identify. Seizures were the most prevalent early manifestation overall, accounting for almost 50% of cases. Minor CNS features or "prodromes" such as tinnitus, metallic taste, hallucinations, slurred speech, limb twitching, extremity paraesthesia, intention tremor, facial sensorimotor, and eye movement abnormalities were noted in only about 16% of patients by DiGregorio et al. [11], but about 30% in combined data from Vasques et al. and Gitman et al. [9,10]; this is consistent with an increase in LAST due to absorption or gradual onset during infusion. Arrhythmias (including bradycardia, tachycardia, and VT/VF), conduction abnormalities (bundle branch block, AV conduction block, widened QRS), hypotension, and cardiac arrest (including non-shockable rhythms- PEA, and asystole) were the most prevalent presenting signs of CV toxicity. Severe LAST is characterized by progressive toxicity (particularly hypotension and bradycardia) that deteriorates rapidly over minutes. It is challenging to predict which individuals will progress. However, early treatment might delay or prevent progression; hence, it is essential to be prepared to act early in any patient receiving local anaesthetic who exhibits signs or symptoms consistent with LAST.

PRESENTING SYMPTOMS AND SIGNS

Prodrome	Major CNS	Major CV
Tinnitus	Agitation/confusion	Bradycardia/heart block
Perioral numbness	Obtundation	Hypotension
Metallic taste	Seizure	VT/VF
Hallucination	Coma	Asystole
Limb twitching		
Hypertension		
Tachycardia		

LOCAL ANAESTHETIC SYSTEMIC TOXICITY: SUBTLE PRODROMES, RAPID DETERIORATION—EARLY RECOGNITION SAVES LIVES.

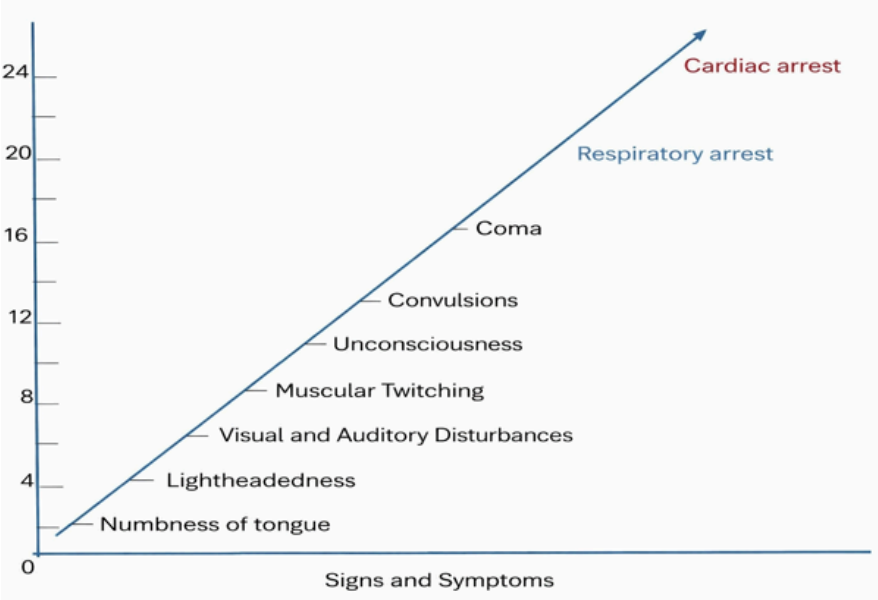
8. Neal JM, Barrington MJ, Fettiplace MR, et al. The Third American Society of Regional Anaesthesia and Pain Medicine Practice Advisory on LA systemic toxicity: executive summary 2017. Reg Anesth Pain Med 2018;43:113-23.

9.Gitman M, Barrington MJ. LA systemic toxicity: a review of recent case reports and registries. Reg Anesth Pain Med 2018;43:124-30. 12.

10. Vasques F, Behr AU, Weinberg G, Ori C, Di Gregorio G. A review of LA systemic toxicity cases since publication of the American Society of Regional Anaesthesia recommendations: to whom it may concern. Reg Anesth Pain Med 2015; 40:698-705.

11. Di Gregorio G, Neal JM, Rosenquist RW, Weinberg GL. Clinical presentation of LA systemic toxicity: a review of published cases, 1979 to 2009. Reg Anesth Pain Med 2010;35: 181-7.

The signs and symptoms of LAST correlate with the serum concentration of LA. This has been well documented for lidocaine, as shown in the following graph



TIME BETWEEN LA INJECTION AND SYMPTOM ONSET:

It's crucial to note that these symptoms don't always develop in the order listed and the timing of onset of symptoms can reveal important information about the mechanism of toxicity. Direct intravascular injection or partial intravascular injection is usually indicated by symptoms that appear within five minutes [13], whereas systemic absorption is suggested by symptoms that appear within twenty to thirty minutes. After injection, symptoms appear in about 26% of cases within the first minute, 22% within 1–5 minutes, 10% within 6–10 minutes, 20% within 11–30 minutes, within 31 to 60 minutes 12% and >60 minutes around 10% (pie chart-Fig2). However, variations in onset time have been observed, with studies reporting that 22% of cases develop symptoms within 30 minutes and 23% within 60 minutes [10]. The most recent American Society of Regional Anaesthesia and Pain Medicine (ASRA-PM) practice advisory on LAST has observed a continued shift of LAST toward delayed presentation, with a significant number of cases presenting between 11 and 60 minutes after injection and, in some cases, over an hour [8].

Local Anaesthetic Agents: Dosing & Maximum Dosage [12]

Drug	Description	Relative Potency	Onset (min)	Duration (hrs) Without Epinephrine	Duration (hrs) With Epinephrine	Max Dose (mg/kg) Without Epinephrine	Max Dose (mg/kg) With Epinephrine
Lidocaine	Amide	2	5–10	1–2	2–4	3	7
Prilocaine	Amide	2	5–10	1–2	2–4	6	9
Bupivacaine	Amide	8	10–15	3–12	4–12	2	2.5
L-Bupivacaine	Amide	8	10–15	3–12	4–12	2.5	3
Ropivacaine	Amide	6	10–15	3–12	4–12	3	4

12. *World Federation of Societies of Anaesthesiologists. https://resources.wfsahq.org/atotw/pharmacology-for-regional-anaesthesia/.*

PREVENTION:

Prevention is a multifactorial process. Ultrasound-guided nerve blocks reduce the risk of LAST by 60%–65% as compared to PNS alone. Increased accuracy of delivery permits reduction in volume, and thus dose of LA; reduced incidence of vascular puncture; visual detection of intravascular injection allows termination of injection before a significant dose is delivered. However, it does not impact the risk of LAST resulting from systemic absorption of LA. It is advisable to perform fractionated injection of LA in aliquots of <5 mL, pausing for 30–45 seconds between injections, with gentle aspiration before injection. Other measures include – vascular markers (epinephrine), clear labelling and meticulous handling of LA-containing syringes. The transition from Luer connectors to standard small-bore connectors to reduce the risk of wrong route injection should be considered.

MANAGEMENT:

Considering the atypical or delayed presentations and cases of LAST after even simple tissue infiltration of LA, all patients receiving LA should have oxygen, standard monitoring, and intravenous access applied. Monitoring should continue for at least 30 minutes after completion of the injection, as delayed presentations are increasingly occurring. Immediate access to a LAST Management Checklist and "LAST Rescue Kit" - all medications and resuscitation equipment required should be immediately available.

Immediate management consists of stopping the LA injection and calling for help. The key priorities are prompt airway and resuscitative management and early infusion of lipid emulsion, aiming to prevent the negative spiral of hypoxia, hypercarbia and acidosis, which all potentiate LAST, while also simultaneously reversing the toxic tissue levels of local anaesthesia. Interrupting this cycle at an early stage will attenuate or altogether avoid pharmacotoxicity progression and possible cardiovascular collapse.

The mainstay of LAST treatment is intravenous lipid emulsion (ILE), a mixture of triglycerides and egg phospholipid. The lipid droplets contain a strongnegatively charged phospholipid "shell" with a vigorous attractive force for positively charged drugs. Positively charged, fat-soluble LA binds to negatively charged lipid particles. Intravenous lipid emulsion therapy may shuttle any LA agent from high blood flow organs – such as the heart or brain – to storage or detoxification organs such as muscles or the liver, improve the cardiac output and blood pressure (hence further facilitating the shuttling effect), and postconditioning myocardial protection may also occur. Lipid therapy may also have a direct role in reversing mitochondrial dysfunction, sodium channel blockade and nitric oxide-mediated vasodilatation. An initial bolus of 100 mL should be administered over 2–3 minutes (1.5 mL·kg–1 if the lean body weight is <70 kg), followed by a 20% lipid emulsion infusion of 200–250 mL over 15–20 minutes (0.25 mL·kg–1·min–1 if the lean body weight is <70 kg). If circulatory stability is not attained, boluses of up to a maximum of three can be administered, or alternatively, increasing the infusion to 0.5 mL·kg–1·min–1 is suggested. The maximum recommended dose of 20% lipid emulsion is 12 mL·kg–1.

13. On'Gele MO, Weintraub S, Qi V, Kim J. LAs, LA Systemic Toxicity (LAST), and Liposomal Bupivacaine. Anesthesiol Clin. 2024 Jun;42(2):303-315.

Seizure activity may exacerbate metabolic acidosis, and prompt prevention and termination is crucial. Seizures should preferably be controlled with benzodiazepines, given the cardio-depressant effects of thiopentone and propofol. If seizures persist despite all efforts, low-dose neuromuscular blockade can be considered to reduce metabolic acidosis and hypoxia from ongoing muscular contraction. Conventional therapies should be used to treat hypotension, bradycardia or arrhythmias, and standard cardiopulmonary resuscitation should be commenced if circulatory arrest occurs. The combination of central nervous system and cardiac dysfunction as the major signs of LAST points strongly to mitochondrial metabolism as the most important clinical target of LAST. The heart and brain are highly intolerant of anaerobic metabolism and inhibition of oxidative phosphorylation, and the resulting depletion of tissue adenosine triphosphate (ATP) could be the most important underlying mechanism in severe LAST, as it might explain why traditional haemodynamic treatments (e.g. vasopressors and inotropes) are only marginally effective.

The best treatment is to address the underlying toxicity directly by reducing tissue local anaesthetic content. If epinephrine is used, small initial doses of ≤1 μg·kg−1 are preferred to avoid impaired pulmonary gas exchange and increased afterload. Vasopressin is not recommended. In the absence of rapid recovery following advanced life support measures and intravenous lipid emulsion therapy, early consideration should be given to cardiopulmonary bypass for circulatory support. If cardiac output is maintained but there are deleterious CVS effects – such as arrhythmias, conduction block, progressive hypotension, and bradycardia – standard Advanced Cardiac Life Support algorithms should be followed, omitting LA, such as lidocaine, for the treatment of arrhythmia. Amiodarone is the first-line antiarrhythmic agent for ventricular dysrhythmia.
Figure 3 shows a holistic approach to prevention, detection and management of LAST. [6]

Post-event management Following an episode of LAST with CVS features, patients should be monitored for at least 6 hours, while isolated and rapidly terminating CNS features require patient monitoring for a minimum of 2 hours.

Figure 3: LAST Management : Holistic Approach

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Every movement needs its energy, and ours comes from a remarkable team of early-career anaesthesiologists spread across the length and breadth of India.

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Our APSA Ambassadors are rooted in their own institutions and communities, yet united by a shared purpose — making anaesthesia safer for every patient. They will engage with peers through academic forums, social media, awareness campaigns and community outreach. They will be writing, presenting, debating, and most importantly, having real conversations about why safety culture matters in our specialty.

They are not just representatives — they are change-makers. And they are already making a difference simply by choosing to be here.

We are proud of this team. Not just for what they will do, but for who they already are — thoughtful, driven young doctors who understand that great anaesthesia is not only about skill, but about systems, awareness, and accountability.

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Dr Hiral Virani
Dr Jagdeep Nayak
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