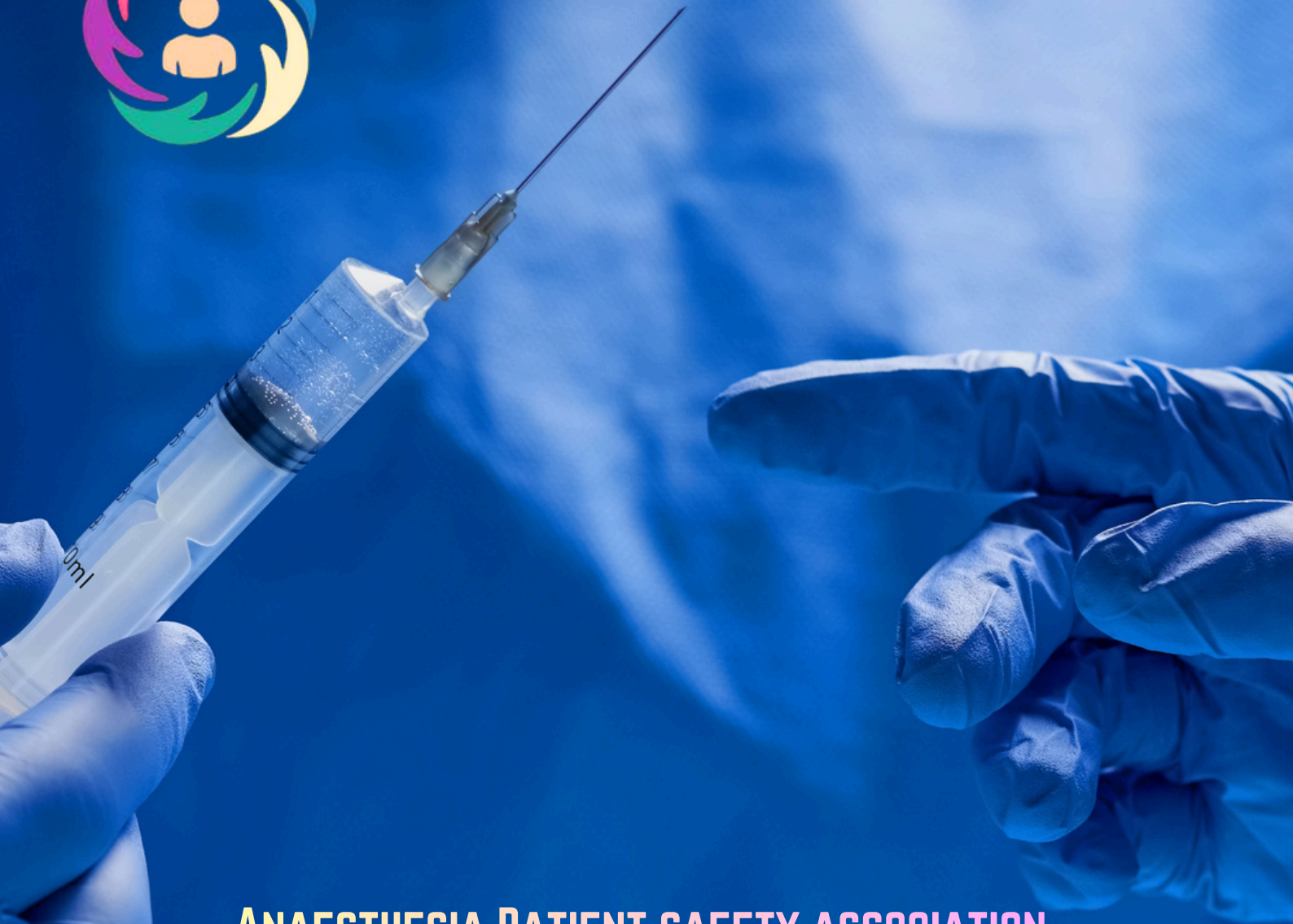




ANAESTHESIA PATIENT SAFETY ASSOCIATION

APSA

NEWSLETTER

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In this issue...

Volume 1, Issue 5

The September edition arrives with the largest collection of incidents we have published in a single issue — fifteen reports spanning the operating theatre, the ICU, and the recovery area. They cover airway emergencies, equipment failures, regional anaesthesia complications, anaphylaxis, and harm that occurred beyond the surgical field. Together, they offer something more valuable than any individual case: a pattern. And patterns, when we are willing to look at them honestly, tell us where to focus.

This month, Dr. Sudheesh Kannan, our Editorial Lead, takes us through each of these incidents in detail — grouping them by mechanism, drawing out the learning, and asking the questions that matter most. We encourage you to read his editorial carefully before moving to the individual reports.

The two articles in this edition speak directly to themes that run through several of this month's cases. The first examines perioperative fluid management — a domain where decisions made quietly in the background carry significant consequences. The second addresses perioperative anaphylaxis — its recognition, its mimics, and the critical importance of acting early and without hesitation. Both are timely, practical, and worth discussing with your team.

Every fortnight, on Thursdays at 7 PM, APSA hosts AIR Rounds — a live discussion open to all, where two incidents from the registry are examined together. Eleven episodes have been completed and are available on our YouTube channel. The link is the same every time: [Join AIR Rounds](#). We would be glad to have you in the room.

These reports remain anonymous. The commentary that follows each one is not about blame — it is about the only question that matters: what do we do differently next time?

We are grateful to every colleague who submitted a report. It takes honesty and professional courage to share what went wrong. We thank our reviewers for their careful analysis, and our editorial and creative teams for bringing this edition together.

Read it carefully. Discuss it with your team. The value of these reports lies not in the reading, but in the conversation that follows.

Dr Murali Thondebhavi
Project Lead, Anaesthesia Incident Registry



LEARNING FROM EVERY INCIDENT FROM NEAR MISSES TO SENTINEL EVENTS

Patient safety in anaesthesia is built not only on successful outcomes, but also on our willingness to examine the unexpected, the preventable, and the narrowly avoided. The incident reports from last two months provide 15 valuable learning incidents spanning airway emergencies, equipment failures, medication errors, regional and neuraxial complications, positioning injuries, cardiovascular events, anaphylaxis, vascular access problems and personnel safety. Together, they demonstrate that adverse events rarely arise from a single failure; more often, they occur when several small gaps align.



Dr Sudheesh Kannan
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AIRWAY AND RESPIRATORY EVENTS

Airway-related incidents remain among the most consequential events in anaesthetic practice. These cases reinforce a fundamental principle: airway preparedness is a system responsibility. Functional equipment, capnography, suction, emergency front-of-neck access, difficult-airway plans and appropriately trained personnel must be available before an airway crisis occurs. Difficult airway in remote locations like ER, can be a nightmare, especially when proper equipment check has not been carried out. Residual anaesthetic effects can be a cause of apnoea and PACU recovery scoring can prevent catastrophe.

EQUIPMENT AND MEDICATION SAFETY

Several reports demonstrate how apparently simple equipment or medication errors can produce major physiological consequences.

A post-CABG patient developed profound hypotension when a syringe-pump/infusion-management error resulted in excessive propofol administration. A faulty HME filter caused unexpectedly high airway pressures in another patient; changing the filter immediately corrected the problem. A central sheath procedure was briefly interrupted when an incorrectly sized needle prevented guidewire passage. Each incident was recognised before permanent harm occurred.

Medication errors were equally instructive. A retrospective case described accidental administration of suxamethonium instead of midazolam because of look-alike packaging; prompt recognition and intubation prevented harm. Likewise, inadvertent loading of two vials of rocuronium into one syringe, resulting in prolonged neuromuscular blockade and approximately three hours of ventilation.

The message is clear: medication preparation should remain under direct anaesthesiologist supervision, with vial confirmation, separation of look-alike drugs, clear labelling and a deliberate checking process. Similarly, equipment selection should never be assumed to be correct simply because the equipment is part of a standard kit.



VIGILANT 

CARDIOVASCULAR AND THROMBOEMBOLIC EVENTS

Two cardiovascular cases demonstrate the importance of vigilance beyond the operating room. Of which, one of them was of an elderly ASA III patient undergoing brachial embolectomy developed sweating, palpitations, T-wave inversion, bradycardia, hypotension and elevated troponin six hours postoperatively despite normal pre-operative cardiac investigations. Cardiology intervention and antithrombotic treatment resulted in resolution of ECG changes. The lesson is that normal pre-operative ECG and echocardiography do not eliminate perioperative cardiac risk, particularly in vascular surgery and high-risk patients. Postoperative surveillance must therefore continue well beyond recovery from anaesthesia. there is a report of sudden cardiac arrest due to failure of recognition of DVT, in a patient undergoing below knee amputation, signaling the importance of suspicion of DVT in long standing immobilization.

REGIONAL AND NEURAXIAL ANAESTHESIA

Regional anaesthesia continues to provide major benefits, but the reports remind us that it is not risk-free and emphasizes the need for continuous monitoring. Inadvertant arterial puncture and transient hoarseness due to drug spread has been reported after supraclavicular brachial plexus block. Adequate analgesia, anxiolysis and continuous visualisation of the needle tip are therefore essential.

Neuraxial complications included post-dural puncture headache (PDPH) following spinal anaesthesia for LSCS and another case in which PDPH occurred together with positioning-related sciatic neuropraxia after orthopaedic surgery. These cases remind us that even recognised complications can significantly affect patient experience. Prevention through appropriate needle selection, careful positioning, regular pressure-point assessment and early treatment remains essential.



POSITIONING AND VASCULAR ACCESS

Position-related injuries were reported in both periods. A prolonged orthopaedic/plastic surgical procedure resulted in a scalp haematoma from a head ring, while a prone spine patient developed a superficial periocular abrasion from an eye patch. Although neither resulted in permanent injury, the latter generated significant patient dissatisfaction.

These incidents demonstrate that minor physical injuries may have major implications for patient trust and medicolegal risk. Pressure points, eye protection, head supports and limb positioning should be checked systematically throughout prolonged procedures, not only at the beginning.

Vascular access produced another important warning. During labour analgesia, an IV cannula became dislodged and extravasated, with progressive swelling eventually leading to cellulitis and concern for compartment involvement. The patient's complaint had initially been dismissed. This is a powerful reminder that patient-reported pain, swelling or tightness around an IV site deserves immediate assessment.

INFECTION AND PERSONNEL SAFETY AND PERSONNEL SAFETY

A needlestick incident during minimally invasive cardiac surgery in an HCV-positive patient involved two surgical assistants—one during suturing and another while clearing the trolley. The second injury is particularly instructive because it occurred after the operative procedure itself.

Known or suspected blood-borne-virus exposure requires strict adherence to sharps safety, appropriate PPE, hands-free passing techniques and safe disposal. Importantly, postoperative instrument handling is part of the safety chain, not an activity outside it.



ANAPHYLAXIS AND TRANSFUSION REACTIONS

The multiple reports of anaphylaxis has been reported following antibiotic and platelet transfusions. Of interest is the refractory antibiotic-associated reaction occurred in a patient taking beta-blockers and required adrenaline, noradrenaline, vasopressin and prolonged resuscitation before haemodynamic recovery. This illustrates how concomitant medication can modify the response to anaphylaxis and why early recognition, immediate adrenaline and escalation strategies are critical.



Perhaps the most important message from these 31 reports is that near misses are as valuable as adverse outcomes. Wrong needle selection, inadvertent arterial puncture, block failure, absence of IV access before a nerve block, and medication preparation errors were all detected before catastrophic harm occurred. These events provide an opportunity to correct systems before the next patient is injured.

Across the reports, recurring solutions are remarkably consistent: checklists, equipment readiness, direct supervision, clear communication, adequate monitoring, early recognition, escalation protocols and respectful attention to patient concerns.

Anesthesia Incident Reporting therefore represents more than an incident-reporting mechanism. It is a collective learning system. Every equipment failure, airway difficulty, medication error, positioning injury, near miss and sentinel event can become a safety intervention when openly reported and thoughtfully analysed.

The goal is not to create a culture of blame. It is to create a culture in which clinicians can say, “This happened; this is why it happened; and this is what we must change so that it does not happen again.” In anaesthesia, where small deviations can rapidly become life-threatening, that culture of learning may be one of our most powerful patient-safety tools.



DOCKED MAKES IT SIMPLE FOR CLINICIANS TO LOG, ORGANISE, AND SHARE INCIDENT DATA

**“You Remain,
And Will
Always Remain,
Completely
Anonymous.”**

— Team Docked



When the Block Does Not Take: Anticipating and Managing Regional Anaesthesia Failure

An ASA III male scheduled for forefoot amputation received an ultrasound-guided popliteal sciatic nerve block performed by a trainee anaesthesiologist, using 25 mL of 0.25% bupivacaine diluted with lignocaine. At assessment twenty minutes after injection the block was inadequate for surgery, and the procedure was completed with supplementary sedation. No adverse event occurred and the failure was managed pragmatically. The incident was reported principally as an issue of trainee supervision, competency documentation and the need for a pre-agreed fallback plan.

Regional anaesthesia is generally reliable, yet block failure remains an important challenge, particularly during training. Failure may be complete or partial, manifesting as inadequate sensory distribution, patchy anaesthesia, premature regression, or failure to cover all nerves required for the surgical field. 1,2

Prevention begins with appropriate block selection. The operator must understand the surgical site, its nerve supply and the limitations of the chosen technique, and must weigh the anticipated duration of surgery when selecting both technique and local anaesthetic dose. 1,2

Operator factors are major determinants of outcome. Inexperience, incomplete anatomical knowledge and imprecise technical execution all contribute to failure. 1 Ultrasound improves target identification but does not abolish it: poor needle-beam alignment, loss of the needle tip, an inappropriate trajectory and failure to recognise the pattern of local anaesthetic spread are characteristic problems of the learning curve. 1

For spinal anaesthesia, patient positioning and needle placement are particularly important, and difficulty may arise from obesity, spinal deformity, pain or anxiety. 2. Correct positioning, an appropriate needle trajectory and careful maintenance of needle position during injection minimise failure. 2

Drug-related factors merit equal scrutiny. The agent, concentration, dose and volume should be verified, and drug identification and expiry checked before injection. 1,2 Dilution that lowers the effective concentration may yield a block of insufficient density for surgical anaesthesia.

Finally, the block must be given adequate time to develop and then formally tested against the requirements of the planned procedure before incision. 1,2 Patient anxiety, inadequate communication and operating-room time pressure may contribute to both perceived and actual failure. 1,2

Successful regional anaesthesia therefore depends on appropriate planning, anatomical knowledge, meticulous technique, careful drug preparation, systematic assessment and a predefined rescue plan agreed before the patient enters the operating room. 1,2

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Correct at Insertion Is Not Correct Forever: Central Venous Catheter Migration

A central venous catheter was inserted under full aseptic precautions in an ASA III patient with carcinoma of the stomach. Position was confirmed at insertion by free aspiration from all lumens and by imaging. Over the subsequent 24 hours in the intensive care unit the catheter migrated from its original central position and ultimately caused pleural puncture, with extravascular infusion of fluid and drugs into the pleural space. The patient developed hypotension and respiratory compromise; imaging demonstrated pneumohaemothorax, requiring drainage and corrective management. The complication arose from migration rather than from the insertion itself.

Central venous catheter (CVC) malposition is an important but under-recognised complication. While malposition may occur at insertion, secondary migration can follow an initially satisfactory placement, with catheter tips moving in response to changes in patient position, respiration, body habitus or catheter movement. [1,2]

Migration may result in catheter dysfunction, thrombosis, vessel erosion or perforation, arrhythmias, infusion into unintended vessels and, rarely, cardiac tamponade. [1,2] A freely flowing catheter or easy aspiration of blood does not reliably exclude malposition. [1]

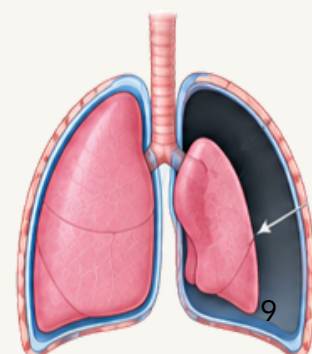
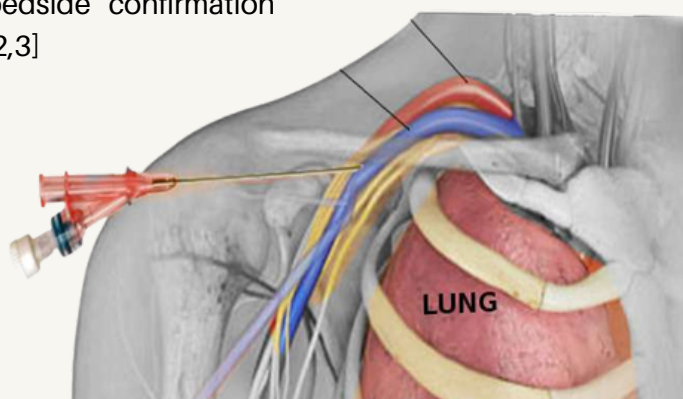
Recognition therefore demands continued vigilance. New difficulty with aspiration or infusion, a change in the external catheter length at the skin, unexplained infusion resistance, chest, neck or shoulder discomfort, arrhythmias or unexpected drug effects should all raise suspicion; the absence of symptoms does not exclude malposition. [1] Following insertion, catheter-tip position should be objectively confirmed. Chest radiography remains widely used, while ultrasound and intracavitary ECG-based techniques permit bedside confirmation where expertise is available. [2,3]

Prevention begins with appropriate site and catheter selection. Ultrasound guidance improves procedural success but does not by itself prevent malposition. [1] The right internal jugular approach generally offers a more direct route to the superior vena cava. Appropriate catheter length, controlled guidewire advancement and avoidance of excessive insertion depth are important. [1,2] Position should be reassessed after major changes in patient position and whenever catheter function changes.

The safety principle is straightforward: place, confirm, secure, reassess. A CVC is not safe merely because it was correctly positioned at insertion. Migration is a dynamic complication, and its recognition must therefore be equally dynamic.

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Adrenaline-Resistant Anaphylaxis: The Beta-Blocked Patient

A 42-year-old female with hypertensive heart disease (ejection fraction 43%) developed sudden hypotension with desaturation shortly after intravenous piperacillin-tazobactam, administered pre-procedurally at surgical request. Despite aggressive resuscitation with bilateral venous access, adrenaline, noradrenaline, vasopressin and intravenous hydrocortisone, haemodynamic recovery required 90 minutes. The delayed response was attributed to concurrent beta-blocker therapy blunting the physiological response to catecholamines. Point-of-care ultrasound and bedside echocardiography excluded primary cardiac causes. She was transferred to the surgical ICU on triple inotropes, extubated the following morning and discharged after two days.

Anaphylaxis is a severe, rapidly evolving systemic hypersensitivity reaction that may be fatal. Anaesthetic anaphylaxis involves cardiovascular collapse in 88% of cases, bronchospasm in 36%, angioedema in 24% and dermatological features in approximately half of all presentations, reflecting the massive release of basophil- and mast-cell-derived mediators into the systemic circulation. Anaphylaxis-related mortality is 3-6%, and a further 2% of patients sustain a poor neurological outcome. Adverse drug reactions are a common precipitant in otherwise healthy patients.^{1,2,3}

Beta-adrenergic antagonists are widely prescribed for arterial hypertension, cardiac arrhythmias, congestive heart failure, migraine prophylaxis and glaucoma. Because adrenaline acts through both beta-1 and beta-2 receptors, concurrent beta-blockade opposes adrenaline-induced inotropy and bronchodilation, producing clinical resistance to the single most important drug in anaphylaxis. Where adrenaline fails in a patient receiving beta-blockers, prompt administration of glucagon is indicated.^{4,5}

Resuscitation Council UK guidance supports consideration of a 1 mg intravenous dose of glucagon in adults taking beta-blockers. Vasopressin, and in refractory vasoplegia methylene blue, may also be required. Where clinically practicable, beta-blockers are best avoided in patients with idiopathic anaphylaxis, since these agents directly impair the efficacy of life-saving adrenaline. Patients highly susceptible to sudden, unpredictable anaphylaxis — those with severe food allergy, systemic mast cell disorders or recurrent idiopathic anaphylaxis — should be counselled regarding this risk and should avoid non-selective beta-blockers where feasible.^{1,5}

The perioperative corollary is equally important: antibiotics must not be administered without the anaesthesiologist's knowledge and readiness to manage anaphylaxis, and serum tryptase should be sampled within 30-60 minutes of the event for retrospective confirmation.

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The New Filter That Failed: HMEF Obstruction as a Cause of High Airway Pressure

An ASA II male posted for urgent revision vascular surgery was intubated and ventilated mechanically. Post-intubation airway pressures were unexpectedly high at 41 cmH₂O despite equal air entry, correct endotracheal tube position and adequate depth of anaesthesia. Bronchodilators were administered empirically, but pressures remained elevated even after prone positioning. Replacement of the heat and moisture exchanger filter returned pressures to 28 cmH₂O, identifying a faulty filter as the cause. Critically, the filter had been new at induction, demonstrating that even unused equipment can fail.

Heat and moisture exchanger filters (HMEFs) are used routinely in ventilatory circuits to provide humidification and microbial filtration. The most important safety concern is progressive or sudden filter obstruction, which may arise from secretions, blood, condensate or a manufacturing defect. Clinically this presents as rising airway pressure, reduced tidal volume, increasing carbon dioxide or difficulty with manual ventilation. Case reports have described potentially life-threatening airway obstruction caused by defective or blocked HME filters. Incorrect orientation of the filter within the circuit, leak around connections or sudden disconnection may also cause complications.¹

Anaesthesiologists should therefore inspect the HMEF for integrity, correct orientation, secure connections and visible contamination, and should ensure that the complete breathing circuit, including the HMEF, is incorporated into the pre-use machine and circuit check. The filter should be replaced if it becomes visibly contaminated, wet or obstructed, or if unexplained resistance develops.²

HMEFs also increase apparatus dead space, which may be clinically significant with low tidal volumes, in paediatric patients and in those with limited ventilatory reserve. Filter resistance rises with flow and may increase further with the accumulation of moisture and particulate matter.³

High airway pressure after intubation should trigger a systematic, stepwise evaluation of patient, circuit and equipment: bronchospasm and secretions; circuit kinking or condensate; and finally the filter and valves. A new filter must not be assumed to be a functional one. When sudden unexplained difficulty in ventilation occurs, HMEF-related complications belong firmly in the differential diagnosis, and the filter should be removed or replaced promptly.

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A Hoarse Voice after Supraclavicular Block: Recognising Recurrent Laryngeal Nerve Involvement

An ASA II male undergoing right distal humerus plating received an ultrasound-guided right supraclavicular brachial plexus block with 25 mL of 0.25% bupivacaine and 8 mg dexamethasone. Immediately after the block he reported hoarseness of voice, which was his only complaint. No respiratory distress, stridor or desaturation was documented. The hoarseness resolved spontaneously within 10 to 12 hours. The likely mechanism was recurrent laryngeal or vagal nerve involvement from local anaesthetic spread within the neck.

Hoarseness of voice due to recurrent laryngeal nerve palsy is a recognised complication of brachial plexus block, more frequent with the interscalene than the supraclavicular approach, and one that may occur despite ultrasound guidance.^{1,3} Although ultrasound permits visualisation of the plexus, needle and local anaesthetic spread, unintended spread to adjacent neural structures can still occur.^{1,2}

The likely mechanism is transient blockade of the recurrent laryngeal nerve (RLN), either directly or through RLN fibres travelling within the vagus nerve.^{1,2} The course of the nerve differs between sides: the right RLN lies relatively close to the subclavian artery and brachial plexus, whereas on the left the vagus nerve lies closer to the subclavian artery and the RLN is more medial.¹ Medial deposition or proximal spread of local anaesthetic may therefore produce RLN or vagal blockade,^{1,2} and large injection volumes facilitate such unintended spread.^{1,3} A tourniquet has been proposed as a further contributor to proximal spread, although this remains hypothetical.⁴

Prevention rests on continuous visualisation of the needle tip, accurate drug deposition and use of the lowest effective volume, with 15-20 mL generally sufficient for a targeted supraclavicular block.¹⁻³ Prolonged massage after injection should be avoided.³

Management is principally supportive, as reported cases have been transient. The airway and oxygenation should be assessed while pneumothorax and local anaesthetic systemic toxicity are excluded.¹⁻³ Reassurance and observation sufficed in some cases, whereas airway support was instituted where anxiety, tachypnoea or concern for airway safety developed.^{1,2} Symptoms resolve as the block regresses, generally within approximately eight hours; aspiration precautions may be warranted while vocal cord function remains impaired.^{1,2}

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Sedation without Reserve: Monitored Anaesthesia Care in the High-Risk Patient

A patient who had undergone total glossectomy and chemoradiation, with a massive oral flap and severe radiation fibrosis, presented with stridor and acute laryngeal oedema and was managed under monitored anaesthesia care for emergency tracheostomy. Multiple equipment failures compounded the crisis: a floppy bougie, a malfunctioning laryngoscope, suction difficulty and, critically, no capnograph to confirm tube placement. The anterior, left-displaced oedematous larynx was eventually intubated bronchoscopically, but placement could not be confirmed. An intensivist, uncertain of tube position, directed its removal; re-intubation was delayed and irreversible cardiac arrest and death followed.

Monitored anaesthesia care (MAC) in a high-risk patient should never be regarded as light sedation in just another patient. The category includes elderly patients and those with obstructive sleep apnoea or cardiopulmonary disease, obesity, frailty, or those receiving multiple central nervous system depressants. Such patients have limited physiological reserve, and even small doses of sedative can precipitate airway obstruction, hypoventilation or cardiovascular instability. A thorough pre-anaesthetic assessment minimises these complications.¹

Assessment should identify every vulnerability. In addition to ASA physical status and functional capacity, the airway, obesity and obstructive sleep apnoea (including STOP-Bang), pre-existing respiratory disease, cardiac status, pulmonary hypertension, renal and hepatic dysfunction and baseline oxygen requirement should be evaluated, together with a review of opioids, benzodiazepines and other depressants and any previous difficulty with anaesthesia or sedation.^{1,2}

Assessment of the procedure is equally important: its duration, the intensity of painful stimuli, patient position, draping and, critically, whether the airway will remain accessible to the anaesthesiologist. A clear plan for rescue ventilation and for conversion to general anaesthesia — including a functioning emergency front-of-neck access kit and a team trained to use it — must exist before MAC begins.²

Common pitfalls include repeated sedative boluses, excessive opioid-sedative combinations, unrecognised sleep apnoea and the assumption that a normal SpO₂ indicates adequate ventilation. Supplemental oxygen delays the detection of hypoventilation, which makes capnography particularly valuable during moderate and deep sedation, and mandatory for confirmation of tracheal tube placement.^{2,3}

Safety depends on titration and anticipation. Optimise local or regional analgesia to reduce sedative requirement, administer drugs slowly in small increments and allow time for effect. ECG, blood pressure, SpO₂ and end-tidal carbon dioxide should be monitored continuously as appropriate, and suction, oxygen, bag-mask ventilation, airway equipment and vasoactive drugs must be immediately available.^{2,3} The fundamental principle is simple: anticipate deterioration, detect it early, and be prepared to rescue the patient when MAC becomes general anaesthesia.

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Small Injuries, Real Harm: Iatrogenic Trauma During Airway Management

An ASA III male in the 71-80 year age group underwent radiofrequency ablation for atrial fibrillation under general anaesthesia with tracheal intubation and transoesophageal echocardiographic monitoring. During direct laryngoscopy the tip of the tongue was caught between the laryngoscope blade and a lower tooth, producing a minor laceration with bleeding. Intubation was completed successfully, the laceration was recognised intraoperatively and managed conservatively, and no airway compromise or significant haemorrhage resulted. Advanced age, dental changes and the requirement for transoesophageal echocardiography all increased the complexity of airway management.

Iatrogenic airway injuries are an important clinical concern, accounting for 6% of medical liability claims within the ASA Closed Claims database. Although 87% of airway-related traumas are transient, 8% of documented cases result in death. The most frequent sites of injury are the larynx (87%), pharynx (51%), oesophagus (48%), trachea (39%), temporomandibular joint (27%) and nose (13%).¹

Prevention begins with careful airway examination and recognition of anticipated difficulty. For nasal intubation, vasoconstrictor application, selection of small warmed tracheal tubes, avoidance of excessive force and passage under flexible bronchoscopic vision guard against mucosal contusion. For orotracheal intubation, tracheal injury is minimised by avoiding excessive cuff pressure, selecting an appropriate tube size and preventing traumatic contact from stylets, laryngoscopes and other adjuncts. Detailed preoperative dental assessment, counselling regarding injury risk and the application of minimal mechanical force during airway manoeuvres reduce injury further.^{1,2,3}

Such injuries typically result from direct trauma to the lips, nasal passages or airway mucosa during instrument insertion, or from the unintended introduction of air or fluid into body cavities. The central upper incisors are particularly vulnerable, with 50% of dental injuries occurring during laryngoscopy. Tracheal tube placement frequently injures the uvula, while nasotracheal intubation produces mucosal contusion of the inferior turbinate and septum in over half of cases. Post-intubation laryngeal injuries commonly present as vocal cord haematoma, supraglottic bruising and mucosal scarring, whereas supraglottic airway placement most often causes transient sore throat.^{1,2}

Two practical points deserve emphasis in elderly patients: the tongue must be displaced completely to the left before the blade is advanced, and any oral bleeding noticed after intubation should be investigated before draping, since an unrecognised laceration can bleed into the airway during a long procedure.



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Where, Not When: Deciding the Site of Laryngeal Mask Removal

A 58-year-old female underwent left total knee replacement under general anaesthesia following a failed spinal. Induction included morphine 5 mg, propofol and sevoflurane; the airway was secured with a size 3 laryngeal mask airway, and rocuronium 40 mg was given thereafter. Reversal with sugammadex 200 mg was administered once spontaneous ventilation resumed. Within five minutes of transfer to the post-anaesthesia care unit with an oropharyngeal airway in situ she became apnoeic, desaturated and unresponsive with a palpable carotid pulse. Bag-mask ventilation was commenced immediately, spontaneous ventilation resumed after five minutes and full recovery followed within ten.

Removal of a laryngeal mask airway (LMA) is a critical transition in airway management. The question is not simply whether removal should occur in the operating theatre or the post-anaesthesia care unit (PACU), but where the airway can be removed most safely for this particular patient.

Evidence comparing early removal under anaesthesia with late removal after recovery is limited. A Cochrane review found no clear overall superiority of either strategy, although airway obstruction was more frequent following early removal (15.6% versus 4.6%).^[1]

For an uncomplicated patient who is fully awake, breathing adequately, has intact protective reflexes and meets standard extubation criteria, removal in the operating theatre permits immediate recognition and management of obstruction with full anaesthetic expertise and equipment at hand.

Patients with residual sedation, airway oedema, significant obstructive sleep apnoea, a difficult airway, excessive secretions or incomplete neuromuscular recovery are at greater risk of obstruction. In these patients removal should be deferred until recovery is adequate, with quantitative neuromuscular monitoring and complete reversal where applicable.^[2,3] Residual opioid effect deserves particular attention: central apnoea may emerge only after the stimulation of transfer has ceased.

Where removal is planned in the PACU, this must not mean removing the device and then transferring the patient. The patient should remain appropriately monitored throughout, with skilled personnel and airway rescue equipment — including a self-inflating bag and suction — immediately available, and should not leave the operating theatre without return of consciousness, protective reflexes and stable oxygen saturation.

The safety principle is therefore to remove the LMA where the patient can be best monitored and the airway most effectively rescued, rather than simply where it is routine. Patient readiness, not habit, should determine both the timing and the location.

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Time Under Pressure: Positioning-Related Injury in Prolonged Surgery

An ASA II obese male (BMI 33) underwent prolonged resection of a thigh sarcoma with anterolateral thigh flap reconstruction under general anaesthesia with tracheal intubation. Postoperatively a scalp haematoma was identified at the site of the head ring used for positioning. Pressure from the head ring over an extended operative period caused localised trauma without skull fracture. The event represents a recognised but preventable complication of positioning device use during prolonged procedures.

Perioperative pressure injuries result from sustained tissue loading over bony prominences, compounded by immobility, friction, shear and impaired perfusion. Anaesthetised patients can neither reposition themselves nor report discomfort, which makes surgical duration a major risk factor.^{1,2} Supine procedures commonly threaten the sacrum, heels and occiput, while prone positioning exposes the face, chest, iliac crests, knees and feet.² Positioning devices themselves — head rings, gel supports and padding — concentrate pressure at a single point and may cause injury when left unaltered for hours.

Prevention begins with preoperative risk assessment considering age, body mass index, comorbidities, mobility, nutritional status, skin condition, planned position and anticipated duration.^{1,3} Intraoperative hypotension, hypothermia and blood loss increase susceptibility further.² Patients should be positioned in physiological alignment with adequate padding, and pressure over vulnerable areas redistributed using offloading surfaces.² Silicone foam dressings may be considered over high-risk bony prominences.² Wrinkled linen, moisture and sliding or dragging during transfers should be avoided because they increase friction and shear.¹ Procedures expected to exceed three to four hours warrant scheduled reassessment of pressure points, with repositioning or alteration of table position where feasible.³

Management depends on early recognition and prevention of progression. A complete skin assessment should be performed immediately after surgery and compared with preoperative findings, recognising that injury may become apparent only over the subsequent 72 hours.¹ New discoloration or skin change should be documented and communicated at handover, with continued postoperative surveillance.^{1,2} Pressure should be relieved from the affected area, protective dressings applied where appropriate and the wound care team involved for significant injury.² High-risk patients should receive postoperative education and follow-up.¹

Meticulous risk assessment, pressure redistribution, safe positioning and postoperative surveillance remain central to prevention, and high-risk positions and long durations should be flagged prospectively at the team brief rather than identified afterwards.

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'Running' Is Not 'Delivering': Syringe Pump Malfunction and Infusion Safety

A post-CABG patient was mechanically ventilated in the intensive care unit with a propofol infusion running at 3 mL/hr. During a nursing shift change the rate was increased to 5 mL/hr without documentation or verification. A second nurse subsequently noted that systolic blood pressure had fallen from 150 to 49 mmHg and that the entire remaining 30 mL of propofol had been infused. Vasopressors and fluid resuscitation were administered promptly and the patient was stabilised. A faulty syringe pump was implicated, but the event also exposed gaps in handover communication, infusion verification and monitoring frequency.

Syringe pumps are indispensable for the administration of propofol, opioids, vasopressors and inotropes. Malfunction may cause over-infusion, under-infusion, interruption of therapy, delayed delivery or unintended bolus administration, with potential for serious harm. Failure modes include mechanical damage, software defects, battery failure, alarm failure and user-interface problems.¹

In a review of 896 drug-error reports from the Australian Incident Monitoring Study, 234 were equipment-related; of these, 49 (20.9%) involved syringe pumps, comprising 36 instances of incorrect pump use, 11 pump failures and 2 attributable to unfamiliarity with the device.² A case report has described an electronic syringe pump delivering buprenorphine at an accelerated rate following a short circuit in the electronic control system.³

A safe pre-use check should trace the pathway from syringe to tubing to intravenous access to patient. It includes physical inspection of the pump, battery, display, alarms and mechanical components; verification of correct syringe type and seating; and confirmation of drug concentration, dose, rate and volume. Kinks, clamps, disconnections, leakage, air bubbles and problems with intravenous access should be excluded. Independent double-checking is recommended for high-risk infusions, and handover during infusion of vasoactive or sedative agents must include explicit rate verification and cross-sign-off.¹

A pump displaying 'running' does not guarantee effective drug delivery. Pump function should be correlated with the volume actually delivered from the syringe and with the patient's physiological response; any unexpected haemodynamic change should prompt prompt immediate review of all running infusions.⁴

Where malfunction is suspected, the infusion should be transferred at once to a backup pump, while the suspect device is removed from service, labelled, documented and referred for biomedical evaluation.⁴

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Deflation as a Trigger: Tourniquet Use, Thrombus Dislodgement and Fatal Embolism

An ASA III male receiving therapeutic heparin for known deep vein thrombosis was brought for below-knee amputation under neuraxial anaesthesia. Immediately following tourniquet inflation he developed cardiovascular collapse. Acute pulmonary thromboembolism was diagnosed, the tourniquet having dislodged the existing thrombus into the pulmonary circulation. Despite resuscitative measures the outcome was fatal. The event highlights a critical failure of preoperative venous thromboembolism risk stratification and surgical planning.

Pneumatic tourniquets provide a bloodless surgical field, but their use carries important thromboembolic implications. Inflation produces venous stasis and local haemostatic changes that may contribute to thrombus formation, and venous embolic material has been demonstrated shortly after tourniquet deflation.[1,2] Although clinically significant pulmonary embolism is uncommon, it can be catastrophic.

Risk assessment must precede tourniquet inflation. Particular caution is warranted in patients with recent trauma, prolonged immobilisation, previous deep vein thrombosis or pulmonary embolism, malignancy, obesity, advanced age, thrombophilia or other established risk factors.[3] Known proximal deep vein thrombosis in the operative limb is a recognised contraindication to tourniquet application, and therapeutic anticoagulation alone does not protect against mechanical dislodgement of an established thrombus. In high-risk trauma patients, limb exsanguination with an Esmarch bandage deserves particular caution, since pulmonary embolism has been reported following exsanguination and tourniquet release.[4]

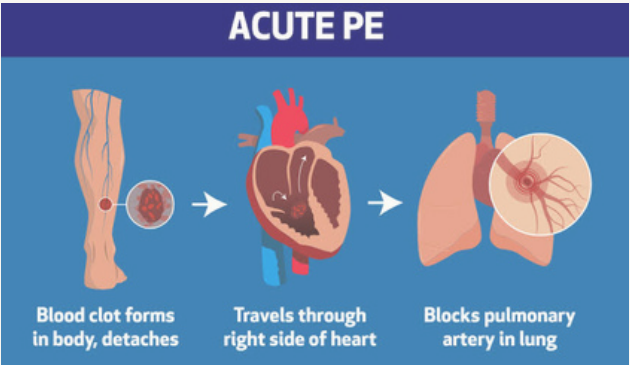
The tourniquet should be used only where necessary, at the lowest effective pressure and for the shortest practical duration.[5] Prolonged inflation should be avoided, and surgical and anaesthesia teams should communicate clearly before deflation.

Release is a critical period. Haemodynamic change should be anticipated, with close monitoring for sudden hypoxaemia, hypotension, tachycardia, arrhythmia or an unexplained fall in end-tidal carbon dioxide — features that may signal significant embolisation — and readiness to manage pulmonary embolism if clinically suspected.

Patients facing prolonged postoperative immobility require formal venous thromboembolism risk assessment with appropriate mechanical or pharmacological prophylaxis, balanced against bleeding risk.[3] Where a limb thrombus is known preoperatively, Doppler venous mapping, multidisciplinary discussion and consideration of alternatives to tourniquet use should precede surgery. The safety message is sequential: identify risk, minimise tourniquet exposure, communicate before deflation, monitor closely and maintain appropriate prophylaxis.

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Hidden Under the Drapes: Transfusion-Associated Anaphylaxis During Anaesthesia

An ASA II male undergoing wound debridement under general anaesthesia developed anaphylactic shock following intraoperative platelet transfusion. The reaction was characterised by severe hypotension, bronchospasm and angioedema, with a 3 g/dL fall in haemoglobin suggesting concurrent haemolysis. Adrenaline boluses and an infusion were commenced, with noradrenaline added for haemodynamic support. The platelet infusion was stopped and the angioedema resolved over approximately four hours. The patient was managed successfully without permanent sequelae.

Transfusion-associated anaphylaxis is uncommon and easily missed under anaesthesia. The usual warning features — urticaria, flushing and angioedema — may be absent or concealed beneath drapes, leaving sudden hypotension, bronchospasm, rising airway pressure, hypoxaemia or cardiovascular collapse as the first clues, which may be misattributed to other drugs or to surgical causes.¹ Platelet components, which contain donor plasma proteins, are a recognised though infrequent cause of severe reactions.

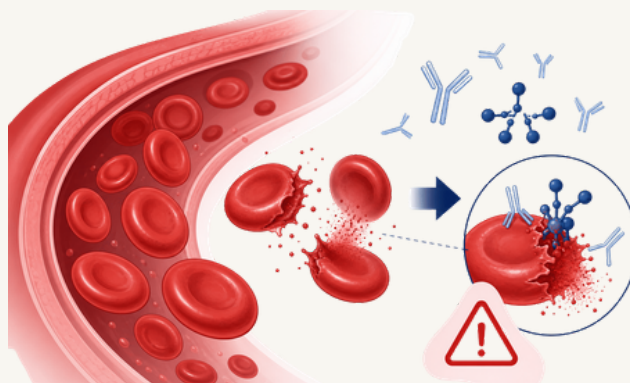
The safest approach is to consider anaphylaxis early and to treat it as a clinical diagnosis rather than await confirmation. The transfusion should be stopped immediately, help summoned and resuscitation begun. High-flow oxygen, airway and ventilatory support, rapid intravenous crystalloid for vasodilatory shock and, above all, adrenaline are the cornerstones of treatment. Adrenaline should not be delayed pending laboratory results; carefully titrated intravenous adrenaline may be required in severe perioperative reactions. Antihistamines and corticosteroids must not delay adrenaline and have little role in reversing the acute cardiovascular or airway components of severe anaphylaxis.²

In parallel, the blood bank should be informed and the implicated unit and giving set retained for investigation. Serum tryptase supports the diagnosis retrospectively but must not delay treatment. IgA and anti-IgA testing and assessment for haemolysis should be guided by clinical context; the presence of haemolysis raises the possibility of a combined or incompatibility-related reaction.^{2,3}

From a patient safety perspective, vigilance, correct patient-component identification, rapid recognition, early adrenaline, a structured team response and accurate haemovigilance reporting matter as much as pharmacological treatment. The first five to ten minutes of any transfusion warrant close observation for early allergic features, and under-reporting of transfusion reactions remains a recognised problem. After a severe reaction the patient should carry a clearly documented transfusion-reaction alert for future care.^{3,4}

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No Urine, Repeated Attempts, Frank Haematuria: Preventing Urethral Injury at Catheterisation

A 70-year-old hypertensive male underwent a six-hour endoscopic rectal procedure under general anaesthesia. Intraoperative catheterisation yielded no urine throughout. A repeat attempt after the procedure by a trained nurse failed twice, with successful passage only at the third attempt, followed by frank haematuria of approximately 200 mL. Urology was consulted; guidewire negotiation was possible but catheter passage was not. Point-of-care ultrasound demonstrated a full bladder with no Foley balloon visible and significant clot burden. Cystoscopy confirmed urethral injury with clots, and evacuation with catheter placement was performed successfully.

Urethral catheterisation is a routine perioperative procedure, yet traumatic insertion can cause bleeding, false passage, infection and later urethral stricture. Iatrogenic urethral injury is among the most common forms of urethral trauma, with catheterisation an important cause.[1]

Prevention begins before insertion. There should be a clear indication, and the smallest appropriate catheter should be selected. Adequate lubrication, aseptic technique and gentle insertion are essential.[2] In men the penis should be held upright and the catheter advanced without force. Resistance — particularly at the prostatic urethra — should never be overcome by repeated or forceful attempts, as this creates a false passage.[1]

Balloon inflation is another preventable cause of injury. The balloon should never be inflated until the catheter has been fully advanced and urine is draining freely, since inflation within the urethra causes significant trauma.[1]

Particular caution is required in patients with previous difficult or traumatic catheterisation, known urethral stricture, prostatic enlargement or previous urological surgery. Repeated failed attempts should prompt early urological assistance rather than escalating force, and guidewire-assisted or cystoscopic catheterisation may be required.[1]

Absent urine output in a catheterised patient is itself a red flag and should prompt review of catheter position long before the end of a prolonged procedure, rather than being attributed to oliguria. Point-of-care ultrasound is the appropriate first-line assessment: a full bladder with no visible Foley balloon indicates a misplaced or failed catheter and should guide immediate escalation. Other red flags include blood at the meatus, urethral bleeding, haematuria, pain, inability to drain urine, resistance during insertion and new perineal or scrotal swelling; the absence of blood at the meatus does not exclude injury.[1]

A simple sequence applies: indication, lubrication, gentle insertion, confirmation of urine, balloon inflation, securement. Safe catheterisation is not merely successful insertion — it is atraumatic insertion and the recognition of when to stop.

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Difficult Supraglottic Airway Insertion in Anticipated Difficult Airway Cases: Precautions and Best Practices

An adult male patient with American Society of Anesthesiologists physical status III and an anticipated difficult airway underwent induction for combined regional and general anaesthesia. Supraglottic airway insertion was attempted twice and failed, with bleeding noted from the oral cavity during device removal. Subsequent laryngoscopy revealed a small epiglottis with adhesions completely obscuring the glottic view, unimproved despite external laryngeal manipulation. Bougie-assisted intubation resulted in initial oesophageal placement, with successful tracheal intubation achieved on the second attempt. Critically, intermittent bag-mask ventilation was maintained throughout, preventing desaturation and averting a cannot-intubate-cannot-oxygenate crisis.

When laryngoscopy fails, second-generation supraglottic airway devices (SADs) serve as the primary rescue device, successfully re-establishing ventilation in over 90% of cases. Since failure of one airway technique increases the composite risk of failure across subsequent attempts, every step of airway management must be planned preoperatively to safeguard patient safety. Consequently, in patients with an anticipated difficult airway, meticulous planning of each stage of airway management is essential to ensure patient safety. (1,2)

Clinicians must perform a separate assessment of supraglottic device placement, as predictors of difficult laryngoscopy do not reliably correlate with difficulty in supraglottic airway (SGA) placement. This assessment can be performed using the "RODS" mnemonic, which evaluates restricted mouth opening, airway obstruction, airway disruption, and short thyromental distance. Preoperative screening should also identify deep pharyngeal obstructions and aspiration risk. (1,2)

Current AIDAA standards restrict airway management to three attempts at tracheal intubation and three attempts at SAD insertion, proceeding further only if oxygen saturation remains at or above 95%. A call for help should be initiated and Code D declared immediately upon encountering the first difficulty in airway management. If ventilation is successfully rescued with an SGA in a non-emergency case, awakening the patient is the safest course of action. If rescue fails, clinicians must immediately declare complete ventilation failure and proceed to emergency surgical cricothyroidotomy. (4)

Several precautions can help prevent complications during SGA insertion. An adequate depth of general anaesthesia should be achieved to suppress upper airway reflexes. Any cricoid force applied during rapid sequence induction should be released prior to insertion, as sustained pressure reduces the hypopharyngeal space and prevents the device tip from sliding behind the cricoid cartilage. (3,2)

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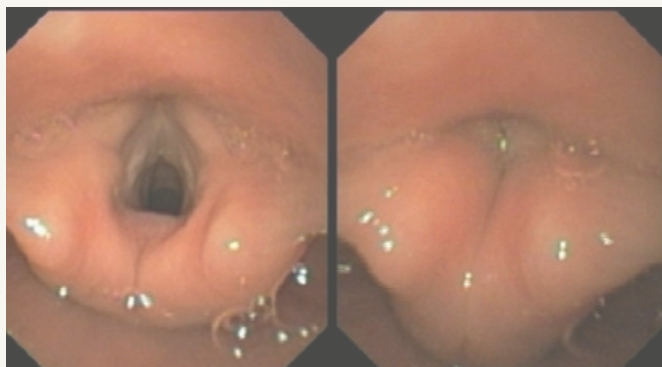


Otorhinolaryngology Surgeries: Shared Airway and Laryngospasm — Prevention and Precautions

A young adult male patient underwent septoplasty under general anaesthesia with endotracheal intubation. Following extubation, he developed breath-holding spells consistent with laryngospasm. Insertion of a laryngeal mask airway permitted brief spontaneous ventilation, but symptoms recurred upon its removal. The patient was subsequently re-intubated with a muscle relaxant, at which point pink frothy secretions were observed, consistent with negative pressure pulmonary oedema secondary to laryngospasm. He was transferred to the intensive care unit for overnight mechanical ventilation under sedation and was successfully extubated the following morning without any documented residual harm. This case underscores the importance of anticipating post-extubation airway compromise.

Airway safety takes precedence over all other considerations in otorhinolaryngological surgery, as the shared airway remains continuously vulnerable to mechanical obstruction, endotracheal tube disconnection, tube displacement, cuff or balloon rupture from surgical instruments, and accidental extubation. These procedures range from day-care interventions to highly complex reconstructions for malignancy, and in every case the surgical team operates in close proximity to, or directly within, the airway. Successful outcomes in these 'shared airway' procedures therefore demand a high degree of cooperation and communication between the anaesthetist and the surgeon. (1,2)

Any episode of airway obstruction in an anaesthetised patient may result from laryngospasm. Laryngospasm presents typically in 75% of cases, but the study by Visvanathan et al. demonstrated that 25% present atypically, manifesting as simple airway obstruction, regurgitation and vomiting, or desaturation. (3,4)



The All India Difficult Airway Guidelines 2025 emphasise risk stratification (SAFE — Stratify risk, Assemble and Anticipate, Facilitate Extubation, and Evaluate and Escalate) as an effective strategy for extubation. Any patient with an increased risk of secretions or blood near the airway should be considered AT RISK for airway compromise. Quantitative assessment of neuromuscular blockade, ensuring the patient is alert, warm, comfortable, pain-free, haemodynamically stable, and maintaining peri-extubation oxygen supplementation are all essential steps. The decision between deep or awake extubation must be individualised, and appropriate pharmacological agents that deepen the plane of anaesthesia, provide antitussive effect, or reduce airway reactivity (e.g., dexmedetomidine, propofol, lignocaine) may be selected to achieve the desired effect. (5)

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Perioperative Fluid Management: Goal-Directed Therapy vs. Restrictive Strategies

INTRODUCTION

Perioperative fluid therapy is a cornerstone of anaesthetic practice, essential for maintaining tissue perfusion, organ function, and fluid-electrolyte balance(1). However, inappropriate fluid choice or administration can lead to complications such as acute kidney injury, pulmonary oedema, coagulopathy, delayed gastrointestinal recovery, and prolonged hospital stay(2). Consequently, practice has shifted from liberal fluid administration towards more individualized approaches, including restrictive and goal-directed fluid therapy, to optimize patient outcomes.(6)

Physiology of fluid and electrolyte balance

Fluid and vasoactive support are guided by patient's physiologic status and hemodynamic stability , with the aim of maintaining adequate blood volume, perfusion pressure, cardiac output, tissue blood flow and sufficient oxygen delivery to the tissues. Fluid therapy is the cornerstone of hemodynamic support, particularly during surgical trauma and blood loss, by resytoring circulating volume and improving tissue perfusion. Optimal organ perfusion and metabolic homeostasis are achieved through individualized fluid therapy combined with vasoactive support and mechanical cardiovascular support.(8)

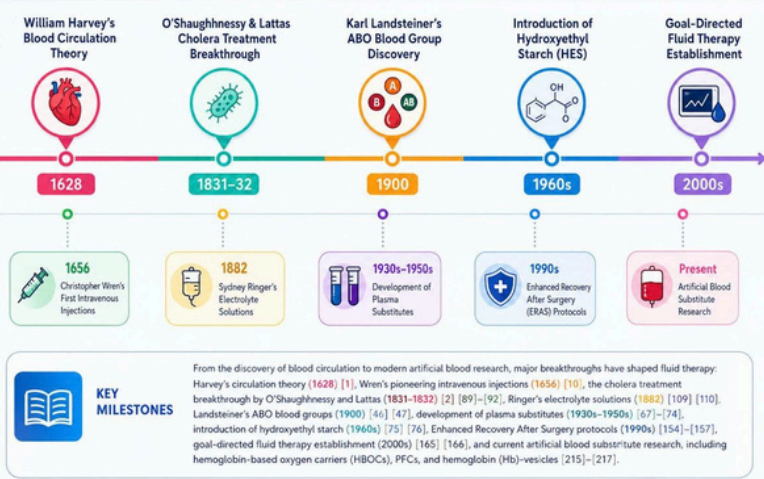


Fig 1: Key milestones in development of fluid management strategies

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The Era of Liberal Fluid Therapy

Emergence of Restrictive Fluid Strategies
Liberal fluid therapy traditional aimed to replace fasting, insensible, and presumed “third-space” losses to maintain tissue perfusion, using empirical rules such as 4-2-1 and 10-15ml/kg body weight formulas. (3,7). However, these approaches often resulted in fluid overload and subsequent pulmonary, cardiac, and gastrointestinal complications, impaired wound healing, and delayed recovery, prompting re-evaluation of liberal fluid administration.

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- Emergence of Restrictive Fluid Strategies

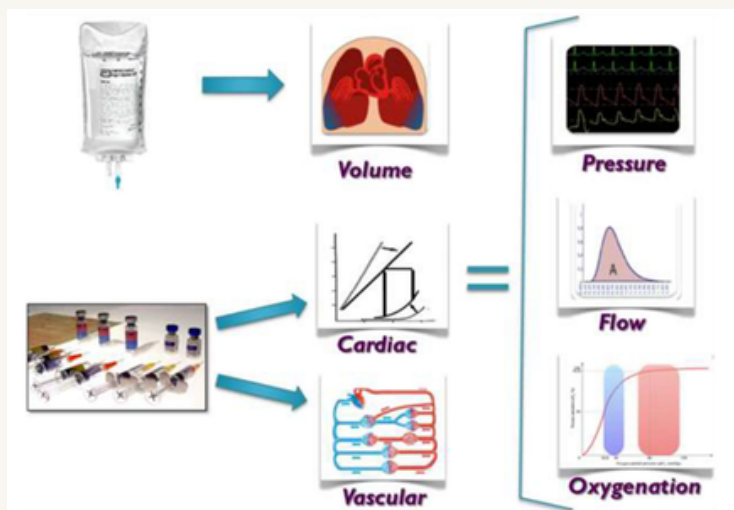


Figure 2: Evolution of restrictive fluid therapy and goal-directed monitoring

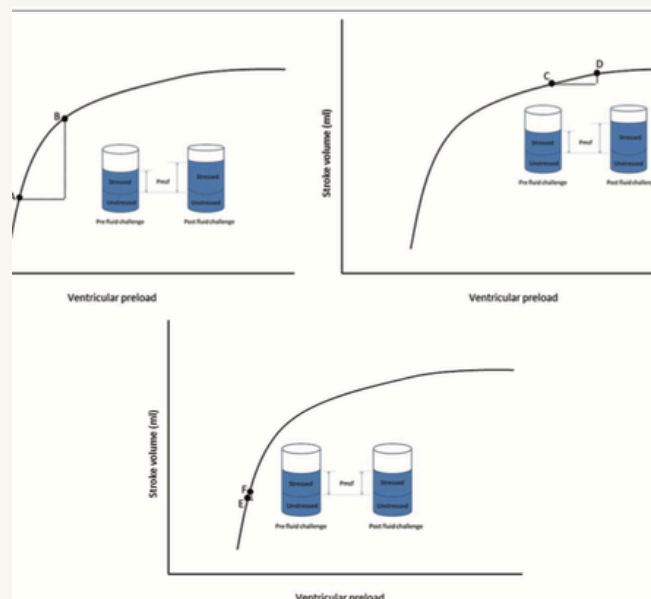


Fig 3: Frank Starling Law and fluid administration(11)

Restrictive fluid therapy evolved to minimise fluid overload, while maintaining adequate tissue perfusion and oxygen delivery. Integrated into Enhanced Recovery After Surgery (ERAS) protocols, this strategy promotes individualized fluid management using haemodynamic assessment rather than routine replacement of presumed losses(10). While it reduces cardiopulmonary complications and facilitates recovery, excessive restriction may result in hypoperfusion, acute kidney injury, and organ dysfunction.

Goal-Directed Fluid Therapy

Goal-Directed Fluid Therapy (GDFT) is an individualized approach that optimizes preload using predefined haemodynamic targets such as stroke volume, cardiac index and oxygen delivery. Dynamic parameters, including stroke volume variation and pulse pressure variation, guide fluid and vasoactive therapy.(5)

Development of Goal-Directed Therapy

Goal-Directed Fluid Therapy (GDFT) emerged as an individualised fixed-volume fluid regimen. Earlier definitions classified restrictive (<1.75 L/day), balanced (1.75–2.75 L/day), and liberal (>2.75 L/day) fluid strategies, but subsequent evidence and ERAS protocols shifted focus toward individualised therapy. Advances in minimally invasive haemodynamic monitoring and ERAS protocols have enabled precision-guided fluid administration. Current debate focuses on GDFT versus individualised restrictive (zero-balance) fluid approaches.

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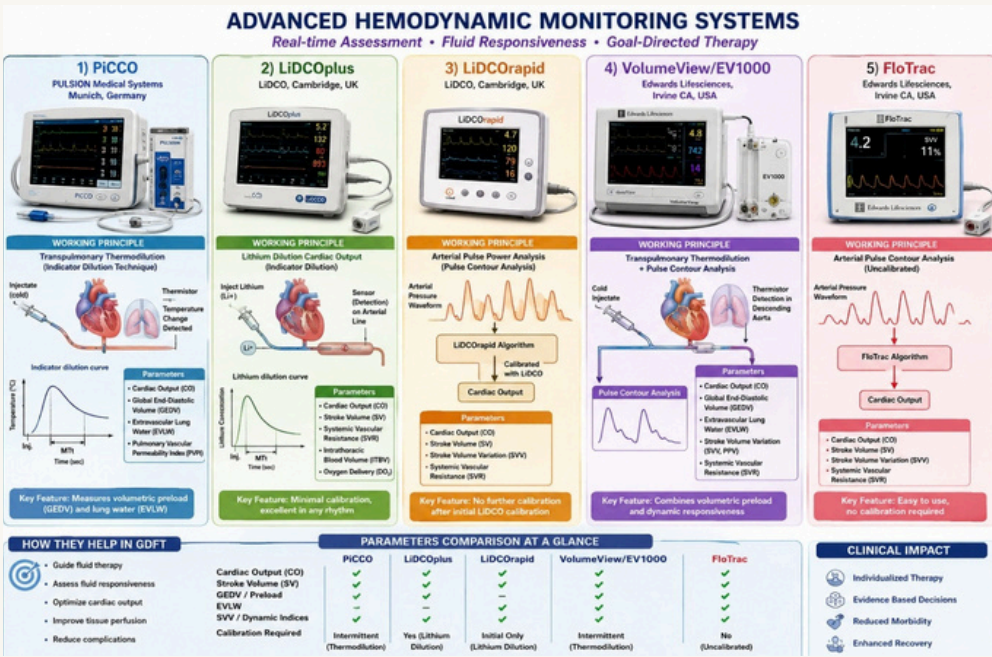


Figure 4: Advanced haemodynamic monitoring systems

Physiological Basis

GDFT is based on the Frank–Starling law principle, whereby increased preload enhances stroke volume only on the ascending limb of the Frank-Starling curve. Beyond this point, additional fluid provides little haemodynamic benefit and increases the risk of oedema.

Monitoring Parameters Used in GDFT

Cardiac output monitoring has evolved from the gold standard invasive Pulmonary Artery Catheter (PAC) to minimally pulse contour analysis, which estimates cardiac output using a computer-based algorithm. Common devices include PiCCO (Pulsion Medical Systems, Munich, Germany), LiDCoplus (LiDCO, Cambridge, UK), LiDCOrapid (LiDCO, Cambridge, UK), VolumeView/EV1000 (Edwards Lifesciences, Irvine CA, USA), and FloTrac (Edwards LifeSciences, Irvine CA, USA). All require calibration except FloTrac.

Fig 4: Advanced hemodynamic monitoring systems in GDFT (11)

Principles of GDFT

Preload Optimization

Uses dynamic indices (e.g. PPV, SVV) to identify fluid responsiveness and administer fluid boluses until stroke volume no longer improves.

Individualized Therapy

Fluid vasopressor, and inotrope therapy is tailored to the patient's physiology comorbidities and surgical procedure.

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Continuous Reassessment

Ongoing haemodynamic monitoring guides repeated assessment and adjustment of therapy throughout the perioperative period.

Advantages of GDFT

Reduced Complications

Minimizes fluid overload and hypovolaemia

Decreases the risk of pulmonary edema, surgical site infections, anastomotic leaks, postoperative ileus, and cardiovascular events.

Lowers the likelihood of acute kidney injury and organ dysfunction

Fewer postoperative complications compared with conventional fluid management strategies.

Shorter Hospital Stay

Earlier discharge from both the intensive care unit and the hospital.

Improved gastrointestinal recovery facilitates, earlier initiation of oral feeding and mobilization.

Decreased need for prolonged postoperative monitoring and additional therapeutic interventions.

Lower healthcare costs and more efficient utilization of hospital resources.

Improved Tissue Perfusion

Optimal cardiac output and oxygen delivery.

Prevent tissue hypoxia, metabolic acidosis, and progressive organ dysfunction.

Better Recovery Outcomes

Faster restoration of normal physiological function after surgery.

Earlier return of bowel function, reduced postoperative fatigue, and improved functional recovery.

Complements Enhanced Recovery After Surgery (ERAS) protocols by facilitating early mobilization, improved pain management, and faster return to normal activities.

Enhances patient satisfaction, improves quality of recovery, and contributes to better short- and long-term clinical outcomes.

Limitations of GDFT

Higher Costs

Requires specialized haemodynamic monitoring, increasing equipment and disposable costs, with limited cost-effectiveness in low-risk or resource-limited settings.

Need for Advanced Monitoring

Limited availability of advanced monitoring restricts routine use of GDFT.

Reduced Accuracy in Certain Conditions

Dynamic indices (SVV, PPV) are unreliable in arrhythmias, spontaneous breathing, low tidal volume ventilation, open-chest surgery, right ventricular dysfunction, and raised intra-abdominal pressure, requiring alternative methods to assess fluid responsiveness.

Restrictive Fluid Therapy

Definition

Restrictive Fluid Therapy (RFT) is a perioperative fluid management strategy that aims to administer the minimum amount of intravenous fluid necessary to maintain adequate intravascular volume, tissue perfusion, and organ function while avoiding fluid overload and a positive fluid balance. It emphasizes on judicious, individualized fluid administration, replacing only measured physiological losses and correcting documented hypovolemia rather than according to fixed formulas or presumed third-space losses.

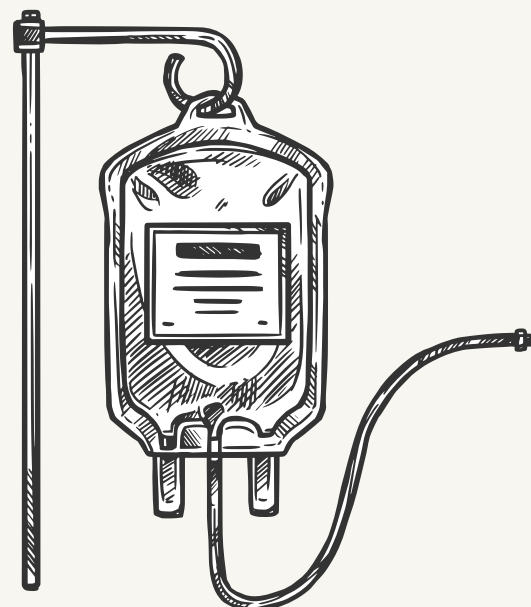
Key Features

Minimizes fluid administration while maintaining adequate perfusion.

Replaces documented blood loss and physiological losses rather than estimated "third-space" losses. Prevents positive fluid balance and tissue oedema.

Prevents positive fluid balance and tissue oedema.

Commonly incorporated into ERAS protocols and combined with GDFT in high-risk patients.



Physiological Basis

Maintains Euvolemia

Preserves cardiac output and tissue perfusion.

Endothelial Glycocalyx Preservation

Prevents glycocalyx damage from fluid overload.

Reduces Interstitial Edema

Minimizes pulmonary edema, interstitial oedema, and tissue oedema.

Optimizes Oxygen Delivery

Maintains an appropriate balance between circulating volume and oxygen transport.

Preserves Microcirculation

Preserve microvascular perfusion and capillary exchange by preventing capillary edema, improving tissue perfusion.

Protects Organ Function

Limiting unnecessary fluid administration reduces the risk of pulmonary congestion, myocardial strain, bowel wall edema, renal interstitial edema, and impaired wound healing.

Maintaining near-zero fluid balance promotes optimal function of lungs, kidneys, gastrointestinal tract, and surgical tissues.

Integration with Modern Perioperative Care

RFT complements ERAS and promotes early mobilization, faster return of bowel function, and shorter hospital stay through individualized fluid management.

Key Physiological Principle

Restrictive fluid therapy maintains euvolemia, preserves the endothelial glycocalyx and prevents interstitial oedema improving tissue perfusion, organ function, and postoperative outcomes.

Components of Restrictive Fluid Therapy

Maintenance Fluid Therapy

Low-rate maintenance balanced crystalloids to meet physiological needs; avoid routine fasting deficit replacement.

Target Euvolemia

The primary goal is to maintain euvolemia rather than achieving a positive fluid balance.

Fluids are administered only when there is clinical or hemodynamic evidence of hypovolemia.

Avoid both under-resuscitation and fluid overload.

Replacement of Measured Losses

Replace only documented losses such as blood loss, urine output (when appropriate), gastrointestinal losses, and surgical drain losses.

Third space loss replacement is avoided.

Judicious Use of Intravenous Fluids

Give fluid boluses only after assessing fluid responsiveness, with ongoing assessment.

Early Use of Vasopressors

Hypotension due to vasodilation is treated with vasopressors rather than administering fluids alone.

Appropriate Fluid Choice

Prefer balanced crystalloids; use colloids selectively only for volume expansion in some selected chosen population.

Continuous Monitoring and Reassessment

Individualize fluid therapy according to patient's evolving physiological status rather than fixed protocols.

Integration with Enhanced Recovery Protocols

Restrictive fluid therapy is a key component of ERAS pathways.

Supports enhanced recovery with earlier mobilization, oral intake, and shorter hospital stay.

Advantages of Restrictive Therapy

- 1.Reduced Tissue Edema
- 2.Improved Pulmonary Function
- 3.Faster Gastrointestinal Recovery
- 4.Enhanced Wound Healing
- 5.Lower Postoperative Complications
- 6.Shorter Hospital Stay
- 7.Preservation of Endothelial Glycocalyx
- 8.Better Integration with ERAS Protocols
- 9.Reduced Need for Blood Transfusion
- 10.Cost-Effective Perioperative Care

Limitations of Restrictive Therapy

- 1. Risk of Hypovolemia
- 2. Increased Risk of Organ Hypoperfusion
- 3. Potential for Acute Kidney Injury (AKI)
- 4. Hemodynamic Instability
- 5. Difficulty in Defining the Optimal Fluid Threshold
- 6. Need for Close Monitoring
- 7. Limited Applicability in Certain Clinical Situations
- 8. Conflicting Evidence in Some Surgical Populations
- 9. Dependence on Clinical Expertise

Comparative Table:

Goal-Directed vs. Restrictive Fluid Therapy

Final Verdict Based on Current Scientific Articles and Guidelines

Clinical Situations: GDFT vs RFT – At a Glance		
CLINICAL SITUATION	GOAL-DIRECTED FLUID THERAPY (GDFT)	RESTRICTIVE FLUID THERAPY (RFT)
 Major abdominal surgery	 Strongly recommended Improves tissue perfusion and reduces postoperative complications	 Useful but may be insufficient alone Helpful as part of ERAS protocols, but may not meet the high physiological demands
 High-risk elderly patients	 Preferred Individualized fluid optimization reduces risk of organ dysfunction and improves outcomes	 Risk of hypoperfusion May increase risk of hypotension, organ hypoperfusion, and acute kidney injury
 Low-risk short surgeries	 Usually unnecessary Standard fluid therapy is generally adequate	 Commonly adequate Avoids unnecessary fluid administration and related complications
 Septic surgical patients	 Beneficial Optimizes cardiac output and tissue oxygen delivery in the hyperdynamic state	 Often inadequate Increased circulatory demands and vasodilatation make restriction insufficient
 Cardiac dysfunction	 Useful with monitoring Advanced hemodynamic monitoring helps avoid both overload and under-resuscitation	 Requires caution Can be used cautiously; excessive restriction may reduce cardiac output
 Renal impairment	 Individualized optimization Maintains renal perfusion while avoiding fluid overload	 Excess restriction may worsen AKI Risk of renal hypoperfusion and progression to acute kidney injury
 KEY TAKE-HOME MESSAGE: GDFT is most beneficial in high-risk patients and major surgeries. RFT is appropriate for low-risk cases but should be applied with caution in physiologically compromised patients. 		

Current evidence supports restrictive baseline fluid therapy combined with goal-directed haemodynamic optimization, rather than liberal or overly restrictive fluid administration. Guidelines (ERAS, NICE, ASA) recommend individualized fluid management using balanced crystalloids, dynamic monitoring in high-risk surgeries, and avoidance of fluid overload and hypoperfusion.The future of perioperative fluid therapy lies in precision medicine tailored to patient physiology, surgical risk, and real-time haemodynamic response.

Conclusion

Perioperative fluid management has evolved significantly toward evidence-based individualized care. Goal-Directed Fluid Therapy and Restrictive Fluid Therapy each offer advantages and limitations. A hybrid approach combining restrictive baseline fluids with goal-directed optimization currently represents the most scientifically supported and patient-safe strategy.

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Perioperative Anaphylaxis: Diagnosis, Triggers and Treatment Pathways

Perioperative Anaphylaxis: When a Routine Case Turns Critical

“The blood pressure monitor alarmed, the airway pressures climbed, and within moments a routine operation had become a race against time.”

A 45-year-old female posted for laparoscopic cholecystectomy underwent an uneventful induction of anesthesia. Shortly after administration of prophylactic antibiotic Cefazolin, her blood pressure suddenly plummeted to 60/30 mmHg. Simultaneously, airway pressures increased, ventilation became difficult, and the end-tidal carbon dioxide (EtCO₂) began to fall.

There was no significant blood loss, no electrocardiographic abnormality, and no obvious surgical cause for the deterioration. Immediate fluid resuscitation was initiated, epinephrine 50 mcg was administered twice followed by infusion, hydrocortisone and diphenhydramine were given, and serum tryptase was sent for confirmation of diagnosis. The patient subsequently recovered and was referred for allergy testing.

For the anaesthesiologist, such situations are uncommon but unforgettable. Perioperative anaphylaxis remains one of the most challenging emergencies encountered in the operating room because diagnosis often has to be made within minutes, based solely on clinical suspicion and physiological changes.

Pathophysiology:

Anaphylaxis is a severe, life-threatening generalized hypersensitivity reaction. It involves activation of mast cells and basophils, followed by the release of histamine, tryptase, prostaglandins, and other inflammatory mediators.

These substances produce systemic vasodilatation and increased capillary permeability, leading to profound hypotension and cardiovascular instability.^{3,5}

Reactions may be IgE-mediated, commonly associated with rocuronium, succinylcholine, and latex. They may also occur through non-IgE-mediated pathways involving direct mast-cell activation, as seen with agents such as vancomycin.

Regardless of the mechanism, the final common pathway is mediator release resulting in vasodilatation, capillary leak, bronchospasm, and tissue edema.

Common Perioperative Triggers

A thorough understanding of perioperative triggers is essential for both prevention and management.

Neuromuscular blocking agents remain the most common cause of perioperative anaphylaxis, accounting for approximately 50–70% of reported cases. Rocuronium and succinylcholine are particularly implicated because of sensitization to quaternary ammonium ions. Cisatracurium is generally considered a safer alternative.^{1,2}

Among antibiotics, cefazolin and penicillin are important triggers because of hypersensitivity reactions involving the β-lactam ring. In patients with documented allergy, alternatives such as clindamycin or azithromycin may be considered.

Propofol has been associated with rare reactions, particularly in patients with egg or soy allergy, although true allergy remains uncommon.

Etomidate and ketamine are potential alternatives in susceptible individuals.

Opioids such as morphine and meperidine may trigger direct mast-cell degranulation. Fentanyl and sufentanil are generally considered safer options in patients with a history suggestive of allergic reactions.

True allergy to local anaesthetics is rare and is more commonly associated with ester agents such as procaine and tetracaine due to PABA metabolites. Amide local anaesthetics such as lidocaine and bupivacaine are usually well tolerated.

Gelatin-based colloids can produce severe reactions, while hydroxyethyl starch has also been implicated through non-IgE-mediated mechanisms.

Latex and chlorhexidine continue to be important non-drug causes, with chlorhexidine reactions increasingly recognized in contemporary anesthesia practice

ANESTHESIA DRUGS AND ANAPHYLAXIS RISK				
Drug category	High Risk drugs	Mechanism	Low Risk alternatives	Notes
Neuro muscular blockers	Rocuronium,Succinyl Choline	IgE mediated (Quaternary ammonium ions)	Cisatracurium	Most common cause 50 to 70% of cases
Antibiotics	Cefazolin, Penicillin	IgE mediated (B-Lactam ring)	Clindamycin, Azithromycin	-
Hypnotics/Sedatives	Propofol (egg/Soy allergy)	Non IgE (Direct Histamine release)	Etomidate , Ketamine	True allergy very rare avoid in egg/ soya allergy
Opioids	Morphine, Meperedine	Direct Mast cell degranulation	Fentanyl, Sufentanyl	Fentanyl be safest in allergy history
Local Anesthetics	Esters like Procaine and Tetracaine	PABA metabolites	Amides like Lidocaine and Bupivacaine	True allergy rare
Colloids	Gelatins(Gelofusine)	IgE mediated	Crystalloid Albumin	HES also risky (Non IgE mediated)
Others	Latex , Chlorhexidine	IgE mediated (Latex) Direct (Chlorhexidine)	Latex free environment Povidone Iodine	Chlorhexidine reactions are increasing

Clinical Presentation:

Recognizing the Early Signs

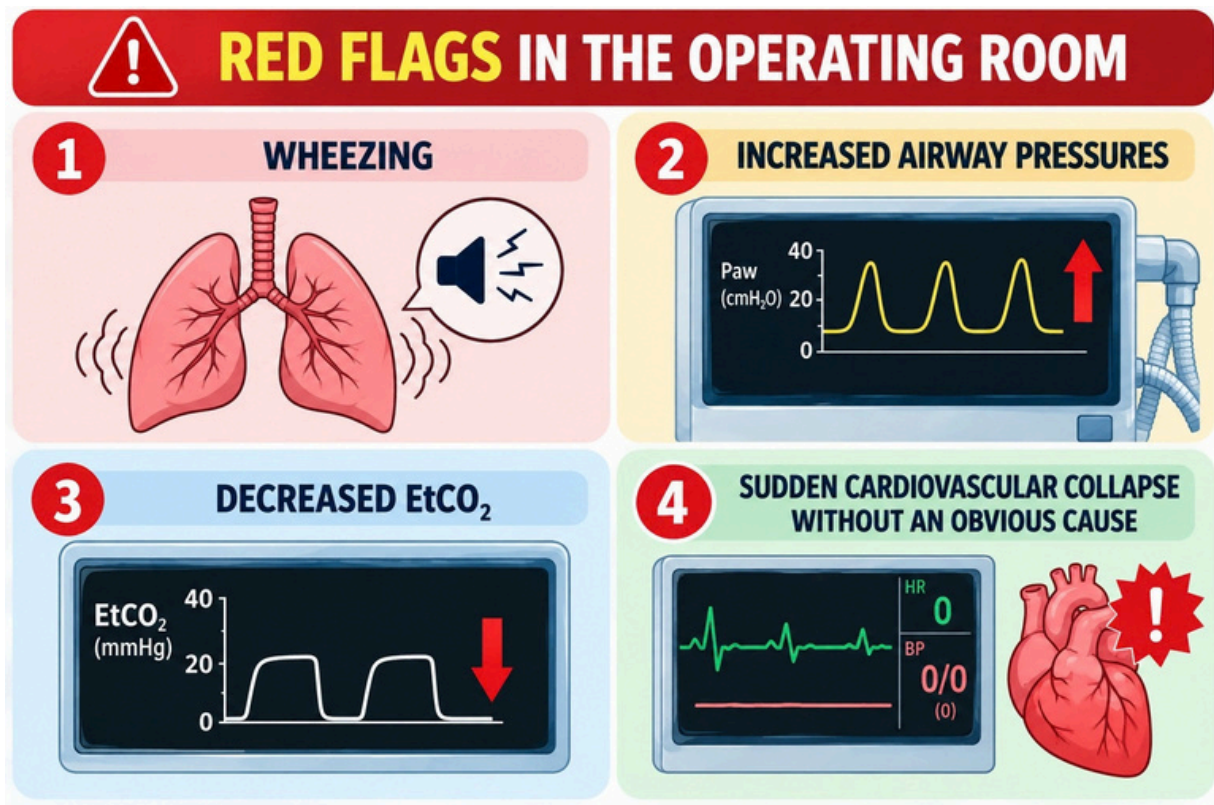
Recognition remains the cornerstone of successful management. Unfortunately, the earliest manifestations are often missed under general anesthesia.^{2,5}

Cutaneous signs such as erythema and urticaria may remain unnoticed because of surgical draping. Cardiovascular manifestations typically include hypotension due to decreased systemic vascular resistance, accompanied by tachycardia or, in severe cases, bradycardia.

Respiratory manifestations may include severe airway edema, increased peak airway pressures, bronchospasm, and a decrease in EtCO₂. A sudden fall in EtCO₂ in the absence of another explanation may be one of the earliest indicators of severe circulatory compromise.

Red Flags in the Operating Room:

1. **Wheezing**
2. **Increased airway pressures**
3. **Decreased EtCO₂**
4. **Sudden cardiovascular collapse without an obvious cause**



All these occurring immediately after administration of a drug. Early recognition of these red flags is crucial because delay in treatment remains one of the major determinants of mortality.

Differential Diagnosis

Not every episode of intraoperative hypotension and bronchospasm represents anaphylaxis. Important differential diagnoses include anaphylactoid reactions, haemorrhage, pulmonary embolism, and vasovagal reactions.^{2,5}

Anaphylaxis typically presents with hypotension, bronchospasm, visible rash when present, and a decrease in EtCO₂. Anaphylactoid reactions may appear clinically similar but are not IgE mediated. Haemorrhage is usually accompanied by tachycardia, declining hematocrit, and obvious surgical bleeding. Pulmonary embolism may present with increased central venous pressure, hypoxemia, and signs of right-heart strain on electrocardiography. Vasovagal reactions are characterized by bradycardia and transient hypotension.

DIFFERENTIAL DIAGNOSIS	
Condition	Clues
Anaphylaxis	Hypotension+ Bronchospasm+Rash (if visible)+ Decrease in ETCO2
Anaphylactoid reaction	Similar but non IgE mediated
Hemorrhage	Tachycardia, decreased Hematocrit, noticeable surgical bleeding
Pulmonary embolism	Increased CVP, decreased Spo2, right heart strain (ECG)
Vasovagal reaction	Bradycardia , Transient hypotension

Immediate Management:

Once suspected, management should be immediate and systematic. Additional help should be summoned and emergency equipment made available. Potential triggers such as neuromuscular blocking agents or antibiotics should be discontinued.^{2,3,6}

Airway and breathing must be addressed urgently because airway edema may make intubation difficult. Administration of 100% oxygen and epinephrine nebulization for bronchospasm should be considered.

- Circulatory support is critical.
- Rapid fluid boluses of 1–2 litres of crystalloid should be administered to counteract the profound vasodilatation and capillary leak.
- Intravenous epinephrine remains the first-line therapy.
- If hypotension remains refractory despite fluids and repeated epinephrine boluses, norepinephrine infusion should be commenced.
- Whenever feasible, surgery should be discontinued until the patient has been stabilized.

Epinephrine and Adjunctive Therapy

- Epinephrine remains the cornerstone of treatment.^{3,6}
- Intravenous boluses of 10–50 mcg may be repeated up to three times and followed by an infusion of 2–10 mcg/min when required.
- In the absence of intravenous access, 0.3–0.5 mg may be administered intramuscularly into the anterolateral thigh.
- Adjunctive therapy includes diphenhydramine 25–50 mg intravenously and ranitidine 50 mg intravenously, although the role of H₂ blockers remains controversial.
- Hydrocortisone 100–200 mg intravenously is frequently administered to reduce the risk of biphasic reactions.
- Persistent bronchospasm may be treated with salbutamol or albuterol nebulization.

Diagnostic Work-Up:

Following stabilization, diagnostic confirmation becomes important.

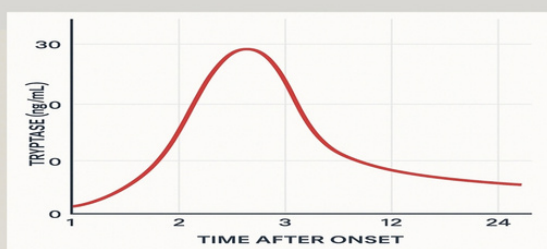
Serum tryptase remains the most valuable laboratory marker of mast-cell activation.

Levels peak approximately 1–2 hours after the reaction and serial measurements should be obtained at 0 hour, 1 hour, and 6 hours. A level greater than 11.4 ng/ml suggests mast-cell activation.^{4,5}

Histamine levels increase within 5–10 minutes but have a very short half-life, limiting their clinical utility.

Definitive identification of the culprit agent requires skin testing, including prick and intradermal tests performed approximately 4–6 weeks after the reaction. Neuromuscular blocking agents, antibiotics, and latex should be specifically evaluated whenever indicated.^{4,5}

DIAGNOSTIC WORK UP



INTRAOPERATIVE ANAPHYLAXIS

IMMEDIATE MANAGEMENT

1 SUSPECT & CALL FOR HELP



Summon additional help and ensure emergency equipment is available

2 STOP POTENTIAL TRIGGERS



Neuromuscular blocking agents



Antibiotics



Discontinue potential triggering drugs

3 AIRWAY AND BREATHING



Give 100% oxygen



Consider epinephrine nebulization for bronchospasm



Be prepared for difficult intubation due to airway edema

4 CIRCULATION



Rapid fluid bolus
1–2 litres
of crystalloid



EPINEPHRINE
(First-line therapy)

IV bolus
10–50 mcg
(repeat up to 3 times)



If hypotension persists
Start **norepinephrine infusion**

5 ADJUNCTIVE THERAPY

H1 BLOCKER



Diphenhydramine
25–50 mg IV

H2 BLOCKER



Ranitidine
50 mg IV
(role controversial)

CORTICOSTEROID



Hydrocortisone
100–200 mg IV
(to reduce risk of biphasic reactions)

6 TREAT PERSISTENT BRONCHOSPASM



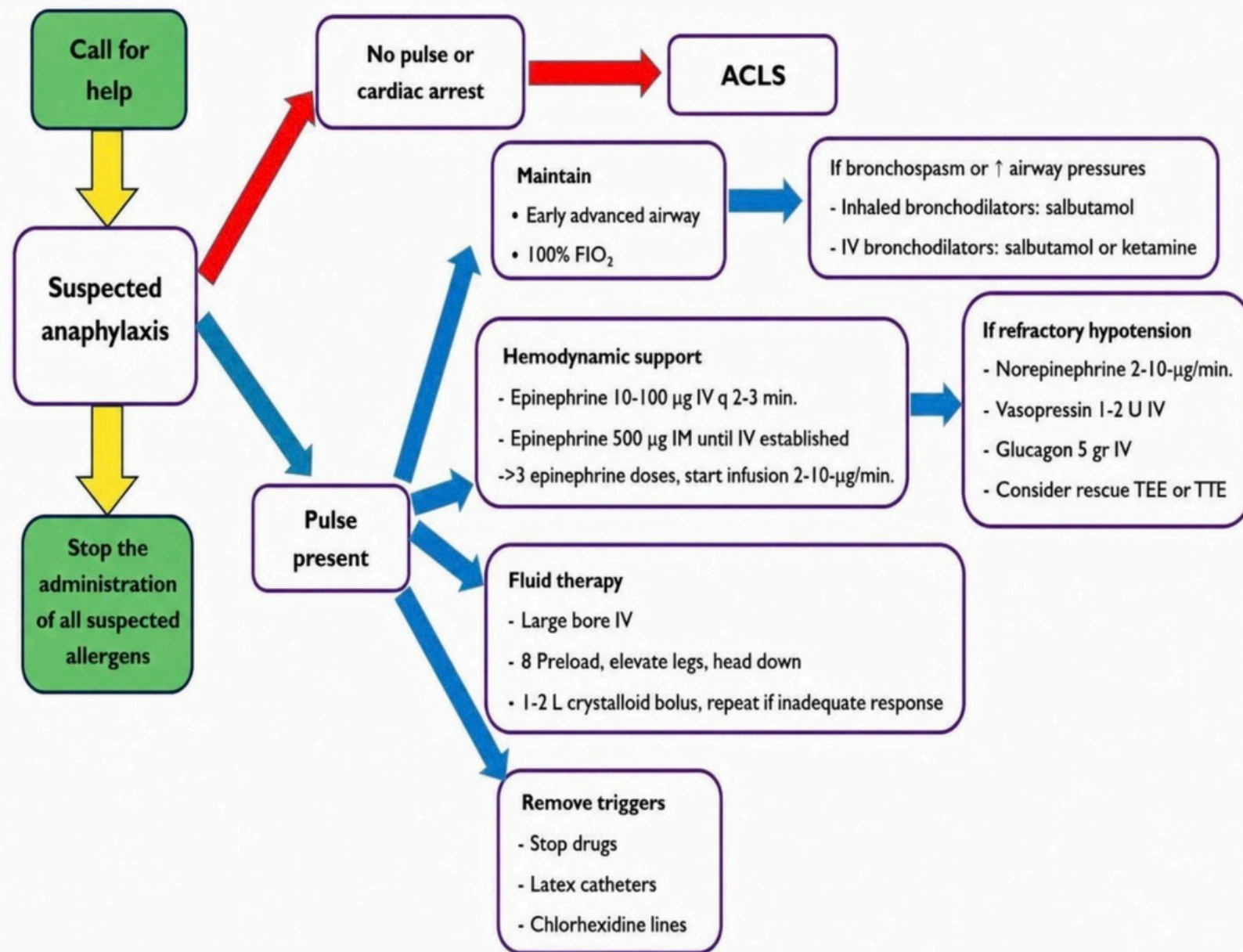
Salbutamol / Albuterol
nebulization

7 CONSIDER STOPPING SURGERY



Whenever feasible, discontinue surgery until the patient is stabilized.

Algorithm for management of Perioperative anaphylaxis



Prevention and Risk Mitigation:

Prevention remains the most effective strategy.

Every patient should undergo careful preoperative screening with particular attention to allergy history. High-risk patients should be managed in a latex-free environment whenever possible.^{2,5} Rocuronium should be avoided in patients with suspected allergy, with cisatracurium considered as an alternative. Similarly, cefazolin alternatives such as clindamycin may be used in selected patients with β -lactam allergy.

The role of prophylactic steroids and antihistamines remains controversial and is generally reserved for patients with a prior history of reactions, as evidence supporting routine preventive use remains limited.

Key Takeaways:

- Suspect anaphylaxis with sudden cardiovascular collapse, bronchospasm, and decreased EtCO₂.
- Epinephrine remains the first-line drug; IV administration is preferred, with IM administration reserved for situations where IV access is unavailable.
- Draw serum tryptase early to support diagnosis.
- Identify and test potential triggers postoperatively to prevent recurrence.

Conclusion:

Perioperative anaphylaxis may be rare, but its consequences are immediate and potentially devastating. The anaesthesiologist must maintain constant vigilance for unexplained hypotension, bronchospasm, increased airway pressures, and falling EtCO₂, particularly when these occur shortly after administration of a drug. Early recognition, prompt administration of epinephrine, aggressive fluid resuscitation, and systematic identification of the offending trigger remain the foundations of successful management.

Close monitoring, prompt response, and early intervention are critical in reducing morbidity and mortality—an essential practice every anaesthesiologist must internalize and implement.

“In perioperative anaphylaxis, survival often depends not on the drug in the syringe, but on the anaesthesiologist who recognizes the diagnosis before anyone else in the operating room.”

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On behalf of the editorial team, we extend our sincere gratitude to each reviewer for your invaluable support, insight, and commitment to advancing science through peer review.

Thank you for being an integral part of our journey.

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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.

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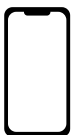
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