e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

A Neuroprotective Anesthetic Strategy: Ultrasound-Guided Dual Plexus Blockade for Clavicle Fixation Following Decompressive Craniectomy

Muhammad Husni Thamrin[1], Bara Adithya[1], Muhammad Dony Hermawan[2]*

[1]Lecturer Staff, Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

[2]Specialized Residency Training Student, Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

ARTICLE INFO

Keywords:
Craniotomy
Interscalene block
Neuroanesthesia
Regional anesthesia
Traumatic brain injury

***Corresponding author:**
Muhammad Dony Hermawan

E-mail address:
donyeyeshield@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v6i2.812

A B S T R A C T

Introduction: Anesthetic management for non-neurosurgical procedures in patients with recent severe traumatic brain injury (TBI) presents a formidable challenge. General anesthesia carries inherent risks of hemodynamic instability and increased intracranial pressure (ICP), which can precipitate devastating secondary brain injury. Regional anesthesia offers a neuroprotective alternative, though its application in this specific high-risk population is not extensively documented. **Case presentation:** A 24-year-old male, ASA status III-E, required open reduction and internal fixation of a clavicle fracture six days after an emergency decompressive craniectomy for an acute epidural hematoma. To mitigate neurological risk, a definitive anesthetic plan consisting of an ultrasound-guided dual plexus blockade was implemented. This involved a combination of an interscalene brachial plexus block (15 mL of 0.375% levobupivacaine) and a superficial cervical plexus block (10 mL of 0.375% levobupivacaine), supplemented with light, non-opioid sedation using dexmedetomidine. The 150-minute surgery was completed with exceptional hemodynamic stability, no requirement for airway manipulation, and no anesthetic or surgical complications. The patient remained comfortable and neurologically intact throughout. **Conclusion:** This case demonstrates that an ultrasound-guided dual plexus blockade is a safe, effective, and neurologically protective primary anesthetic technique for clavicle surgery in the post-craniotomy patient. By providing dense surgical anesthesia while preserving stable cerebral perfusion pressure, this approach represents a superior alternative to general anesthesia in this fragile patient population. We advocate for its consideration in similar clinical scenarios.

1. Introduction

Clavicle fractures are among the most frequent injuries in orthopedics, resulting from falls, sports injuries, or motor vehicle collisions.[1] While many can be managed non-operatively, displaced fractures often necessitate surgical intervention to restore anatomical alignment, preserve shoulder function, and facilitate a quicker return to activity. The standard surgical approach is open reduction and internal fixation (ORIF), a procedure that involves exposing the fractured bone, realigning the fragments, and securing them with a metal plate and screws.[2]

Historically and traditionally, the anesthetic plan for such a procedure has been general anesthesia (GA). This approach, while undeniably effective at ensuring patient immobility, unconsciousness, and lack of pain,

represents a significant physiological trespass.[3] GA is not a gentle sleep but a controlled, reversible coma, induced and maintained by potent pharmacological agents that systematically depress the central nervous system and disrupt the body's homeostatic mechanisms.[4] This disruption, while generally well-tolerated by healthy individuals, comes with a host of inherent challenges and risks. The journey of GA is often a hemodynamic roller coaster. The induction phase, where potent intravenous agents like propofol are administered, frequently causes a sharp decrease in blood pressure due to widespread vasodilation and a reduction in cardiac output. This is immediately followed by one of the most stimulating and stressful moments in anesthesia: laryngoscopy and tracheal intubation. The manipulation of the airway triggers a powerful sympathetic nervous system response, a catecholamine surge that can cause a dramatic spike in heart rate and blood pressure. The anesthesiologist must then carefully titrate anesthetic gases and other medications to maintain a stable intraoperative course, only to face another potential period of instability upon emergence, as the patient awakens and the protective reflexes return. Beyond these hemodynamic fluctuations, the management of the airway itself presents a significant challenge. A "difficult airway," where intubation is not straightforward, is a constant concern, carrying risks that range from minor dental trauma to the catastrophic inability to secure the airway, leading to hypoxic brain injury. Even after a successful procedure, the legacy of GA persists. Postoperative nausea and vomiting (PONV) is a common and distressing side effect, driven by the effects of anesthetic agents on the brain's chemoreceptor trigger zone. Furthermore, the surgical pain that emerges as the anesthesia wears off necessitates the use of potent opioid analgesics. While effective, these medications carry their own spectrum of adverse effects, including respiratory depression, profound sedation that can delay recovery, constipation, urinary retention, and the long-term risks of tolerance and dependence.[5,6]

In recent years, a paradigm shift has occurred in the philosophy of anesthetic care, championing a move away from the systemic trespass of GA towards the targeted precision of regional anesthesia (RA). This approach is built on the principle of "numbing the part, not the whole," using carefully placed injections of local anesthetic to block the specific nerves that supply the surgical site. For clavicular surgery, this represents a particularly elegant solution. The lateral two-thirds of the bone, including the critical acromioclavicular joint, receive their sensory supply from the suprascapular nerve. This nerve is a key branch of the brachial plexus, the intricate network of nerves originating from the C5-T1 spinal roots that controls the entire upper limb. The medial third of the clavicle, along with the entire expanse of skin overlying the surgical field, is innervated by the supraclavicular nerves. These nerves are branches of the superficial cervical plexus, a separate network originating from the C1-C4 spinal roots that supplies sensation to the neck and upper shoulder region. Consequently, a successful regional anesthetic for clavicle ORIF requires a combined approach: an interscalene brachial plexus block (ISB) to target the suprascapular nerve at the level of the brachial plexus roots (C5-C6), and a superficial cervical plexus block (SCPB) to anesthetize the supraclavicular nerves as they emerge from behind the sternocleidomastoid muscle. When performed under ultrasound guidance, this dual plexus blockade can provide dense, comprehensive surgical anesthesia for the entire operative field. The benefits of this sophisticated technique are well-documented and profound. It provides superior perioperative analgesia, with the nerve block often lasting for 12 to 24 hours, effectively covering both the intraoperative period and the most painful phase of postoperative recovery.[7] This dense, long-lasting analgesia dramatically reduces, and in many cases eliminates, the need for intraoperative and postoperative opioids, thereby avoiding their entire spectrum of adverse effects. Patients are more awake, alert, and comfortable immediately after surgery. The incidence of PONV is drastically reduced, and because the systemic effects are minimal, patients can often recover faster and be discharged sooner.

This clear superiority of RA over GA for routine clavicle surgery sets the stage for the ultimate clinical challenge: the anesthetic management of the same orthopedic injury in a patient with an acutely

compromised brain. The anesthetic calculus, the careful weighing of risk and benefit, becomes profoundly more complex and the stakes infinitely higher.[8] The management of a patient requiring surgery just days after a decompressive craniectomy for a TBI is one of the most demanding scenarios in clinical anesthesiology. Here, the primary mandate of the anesthesiologist undergoes a critical shift. The goal is no longer simply to facilitate the orthopedic repair, but to do so while actively protecting the injured brain from any further harm. Every action, every medication, and every physiological change must be viewed through the lens of its potential impact on the intracranial environment.[9]

The foundational principle governing this environment is the Monro-Kellie doctrine. This doctrine posits that the skull is a rigid, non-compliant vault with a fixed volume, containing three components: the brain parenchyma, blood, and cerebrospinal fluid (CSF). In a healthy state, a small increase in the volume of one component (like a temporary increase in blood flow) can be compensated for by the displacement of another (like CSF). However, after a severe TBI, the brain is often swollen and edematous, consuming all available compensatory reserve. In this state of high intracranial compliance, even a minuscule increase in intracranial volume can cause a precipitous and catastrophic rise in intracranial pressure (ICP). This elevated ICP directly threatens the brain's blood supply. The driving force for blood flow to the brain is the cerebral perfusion pressure (CPP), which is defined by the critical relationship: CPP = Mean Arterial Pressure (MAP) - Intracranial Pressure (ICP). Following a TBI, the brain's ability to autoregulate its blood flow is often impaired or lost. The cerebral vasculature becomes a passive system, and blood flow becomes directly dependent on the CPP. The anesthesiologist must therefore fight a battle on two fronts: preventing any further rise in the already elevated ICP while simultaneously maintaining a stable MAP to ensure an adequate CPP.[10]

It is in this context that the physiological trespasses of general anesthesia, which are manageable in a healthy patient, become direct and potent threats to neurological survival. The potential for hypotension upon induction could drop the MAP, causing the CPP to plummet and inducing a secondary ischemic injury. The intense hypertensive surge during laryngoscopy could be transmitted directly to the fragile cerebral vasculature, acutely increasing cerebral blood volume, spiking the ICP, and potentially causing further bleeding or herniation. The increased intrathoracic pressure from positive pressure ventilation can impede venous return from the brain, increasing cerebral venous congestion and raising the ICP. Each of these events, a standard part of the GA journey, represents a potential secondary insult to the already injured brain. Therefore, avoiding general anesthesia in a post-craniotomy patient is not merely a preference; it is a paramount clinical objective, a neuroprotective imperative.

While the theoretical benefits of RA in this setting are clear, there is a paucity of literature describing the application of an advanced dual plexus blockade as the sole anesthetic technique for clavicle surgery in this specific, high-risk patient demographic. The novelty of this report lies in its detailed demonstration of how a meticulously planned and executed RA strategy can serve as a definitive, neurologically protective alternative to GA. The aim of this case report is to fill this critical gap by presenting the successful management of a patient who underwent clavicle ORIF six days post-craniectomy, utilizing an ultrasound-guided combined ISB and SCPB, thereby ensuring patient safety while facilitating essential orthopedic repair.

2. Case Presentation

A 24-year-old, 70 kg male was scheduled for ORIF of a displaced right mid-shaft clavicle fracture. His case was classified as American Society of Anesthesiologists (ASA) physical status III-E due to a critical recent medical history. Six days prior, the patient was involved in a high-speed motor vehicle collision and sustained a severe TBI. Upon initial presentation to the emergency department, his Glasgow Coma Scale (GCS) score was 9 (E2, V2, M5). An emergency non-contrast computed tomography (CT) scan of the head revealed a large (approximately 70 cc) right-sided temporoparietal acute epidural hematoma with a significant mass effect,

evidenced by a 12 mm midline shift to the left and effacement of the ipsilateral ventricle.

The patient underwent an emergent right-sided decompressive craniectomy and complete evacuation of the hematoma. Postoperatively, he was mechanically ventilated for 48 hours in the intensive care unit (ICU) and was successfully extubated on postoperative day three. His neurological status steadily improved, and by the time of orthopedic consultation on postoperative day five, his GCS was 15. He was hemodynamically stable, breathing spontaneously on ambient air, and had no focal neurological deficits, although he reported a mild headache and significant pain over his right clavicle. A plain radiograph confirmed a displaced, comminuted fracture of the middle third of the right clavicle, for which surgical fixation was recommended to facilitate mobilization and rehabilitation.

On the morning of the scheduled surgery (post-craniectomy day six), a comprehensive pre-anesthetic evaluation was conducted. The patient was alert, oriented, and cooperative. His vital signs were: blood pressure 128/76 mmHg, heart rate 82 beats/min, respiratory rate 16 breaths/min, and SpO_2 98% on room air. The craniectomy site was clean, dry, and without signs of infection or cerebrospinal fluid leakage. Airway examination revealed a Mallampati class II view, adequate thyromental distance (>6 cm), and a full range of cervical spine motion without pain. A repeat non-contrast head CT scan performed that morning confirmed the expected post-surgical changes with decreased mass effect and resolution of the midline shift. Preoperative laboratory investigations were within normal limits. After a multidisciplinary discussion with the neurosurgery and orthopedic surgery teams, a consensus was reached to proceed with the clavicle ORIF under regional anesthesia to eliminate the risks associated with GA. After a detailed explanation of the proposed regional anesthetic plan, its benefits, and potential risks (including block failure, nerve injury, local anesthetic systemic toxicity, and pneumothorax), the patient provided written informed consent to proceed.

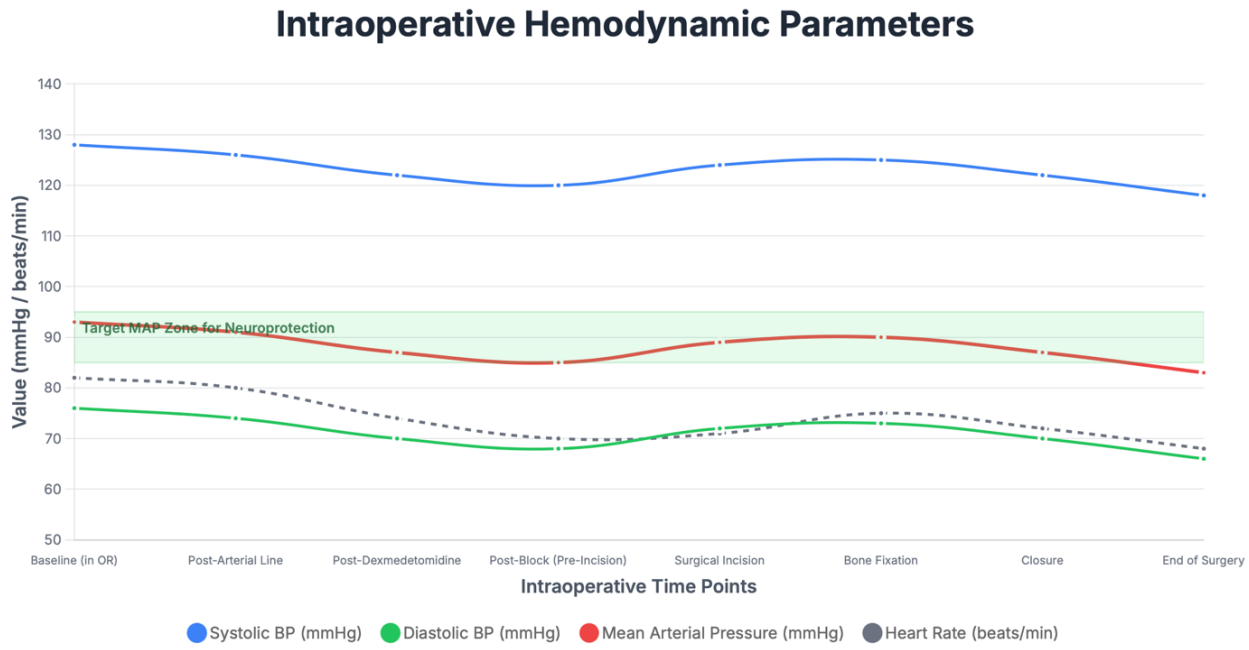
The patient was brought to the operating room, where standard ASA monitoring was applied. To allow for immediate detection and management of any hemodynamic perturbations that could compromise cerebral perfusion pressure, a 20-gauge arterial catheter was placed in the left radial artery under local anesthesia for continuous, beat-to-beat blood pressure monitoring. The patient was positioned semi-recumbent at a 30-degree head-up angle to optimize cerebral venous drainage.

For patient comfort, light sedation was achieved with a dexmedetomidine infusion, initiated at a rate of 0.4 mcg/kg/hr without a loading dose. This rate was determined to be sufficient to maintain a target Ramsay Sedation Scale score of 2-3 (patient calm, cooperative, and easily rousable) and was maintained at a fixed rate throughout the procedure. This strategy provided anxiolysis while preserving spontaneous ventilation and hemodynamic stability.

The regional anesthesia procedure was performed under sterile conditions using a high-frequency linear ultrasound transducer (12-5 MHz). With the probe in the transverse axial plane at the cricoid cartilage level, the C5, C6, and C7 nerve roots were identified as hypoechoic structures in the classic "traffic light" orientation between the anterior and middle scalene muscles. After skin infiltration, a 22-gauge, 50 mm insulated nerve block needle was inserted using an in-plane, lateral-to-medial approach. The needle tip was carefully positioned in the interscalene groove adjacent to the C5 and C6 roots. Following negative aspiration, 15 mL of 0.375% levobupivacaine was injected in increments, with ultrasound confirming circumferential spread of the local anesthetic. The ultrasound probe was used to identify the posterior border of the sternocleidomastoid muscle (SCM). The needle was inserted in-plane from posterior to anterior, advancing the tip to the fascial plane just deep to the SCM. After negative aspiration, 10 mL of 0.375% levobupivacaine was injected, visualizing the hydrodissection and linear spread of the anesthetic along this sub-fascial plane. Twenty-five minutes after block placement, a detailed sensory and motor assessment was performed. Sensory block was confirmed by a complete loss of sensation to cold stimulus (alcohol swab) in the dermatomes from C3 to T1, covering the entire surgical field. Motor block was confirmed by the inability to abduct the shoulder

(deltoid, C5-C6), flex the elbow (biceps, C5-C6), and extend the wrist (C6-C7).

The total surgical time was 150 minutes. Throughout the procedure, the patient remained hemodynamically stable with a complete lack of a surgical stress response. His intraoperative vital signs are detailed in figure 1. The dexmedetomidine infusion provided adequate sedation (Ramsay score 2-3) without respiratory compromise. Estimated blood loss was minimal (75 mL), and 1000 mL of lactated Ringer's solution was administered. A final ultrasound check confirmed the absence of diaphragmatic paralysis.



Data Interpretation:

The shaded green area represents the target Mean Arterial Pressure (MAP) range (85-95 mmHg) for optimal cerebral perfusion. The patient's actual MAP (solid red line) remained consistently within or very close to this neuroprotective zone, demonstrating the effectiveness of the anesthetic technique in avoiding both hypotension and hypertension. The stability of the heart rate and blood pressure reflects a profound block with minimal surgical stress response.

Figure 1. Intraoperative hemodynamic parameters.

Upon completion of surgery, the patient was fully awake, comfortable (Numeric Rating Scale pain score of 0/10), and neurologically stable. Given this stability and lack of sedation hangover, he met the institutional criteria for bypassing the Post-Anesthesia Care Unit (PACU) and was transferred directly to the orthopedic ward. His postoperative analgesic regimen consisted of scheduled intravenous paracetamol and oral ibuprofen. He did not require any rescue opioid analgesia in the first 24 hours and was discharged home on the second postoperative day.

3. Discussion

The management of a patient with a recent, severe traumatic brain injury (TBI) for subsequent, non-neurosurgical procedures represents a pinnacle of clinical complexity in anesthesiology. It is a scenario where the anesthesiologist's role transcends the provision of surgical anesthesia and analgesia, evolving into that of an active guardian of the central nervous system. The core principle guiding every clinical decision is the mitigation of secondary brain injury—a delayed and potentially preventable cascade of devastating cellular and physiological events that

follows the initial mechanical trauma.[11] This discussion will provide an in-depth exploration of the neuroprotective rationale that dictates anesthetic choice in this fragile patient population, followed by a granular analysis of the anatomical, technical, and pharmacological strategies that enable a successful, neurologically-focused outcome.

The paramount goal in managing a patient with recent severe TBI is the prevention of secondary brain injury. The initial, or primary, brain injury is the direct result of mechanical forces at the moment of impact—contusions, lacerations, and shearing of axons.[12] This damage is immediate and largely irreversible. However, the subsequent clinical course is profoundly influenced by the secondary injury cascade, a complex and evolving process of cellular dysfunction, inflammation, and metabolic failure that is driven not by the initial trauma, but by subsequent physiological insults. These insults include systemic hypoxia (lack of oxygen), hypercarbia (excess carbon dioxide), and, most critically, derangements in cerebral perfusion.

The intracranial space is governed by the Monro-Kellie doctrine, which conceptualizes the skull as a rigid, non-compliant box containing three components: brain parenchyma (~80%), blood (~10%), and cerebrospinal fluid (CSF) (~10%). Because the total volume is fixed, any increase in the volume of one component must be compensated for by a decrease in the volume of another. When this compensatory mechanism is exhausted, intracranial pressure (ICP) rises exponentially. In the context of TBI, cerebral edema can rapidly increase the volume of the brain parenchyma, leaving little room for compensation and causing a precipitous rise in ICP.[13]

The driving force for blood flow to the brain is the Cerebral Perfusion Pressure (CPP), defined by the critical relationship: where MAP is the mean arterial pressure. This simple equation is the cornerstone of neuroanesthesia and neurocritical care. In a healthy brain, the elegant process of cerebral autoregulation maintains constant cerebral blood flow (CBF) across a wide range of MAPs, typically between 60 and 160 mmHg. This is achieved through the active vasodilation and vasoconstriction of cerebral arterioles in response to changes in perfusion pressure (myogenic response) and metabolic signals (like $PaCO_2$). However, following severe TBI, this delicate autoregulatory capacity is often impaired or completely lost due to endothelial damage, inflammatory mediator release, and loss of vascular smooth muscle reactivity. The cerebral vasculature becomes a passive, pressure-dependent conduit. In this fragile state, CBF becomes directly proportional to CPP. Any fall in MAP or rise in ICP can critically reduce CPP, leading to cerebral ischemia and infarction. Conversely, an uncontrolled surge in MAP can lead to a breakthrough of the blood-brain barrier, exacerbating cerebral edema and increasing ICP.

In this precarious physiological state, the events surrounding the induction and maintenance of general anesthesia (GA) can be catastrophic. Each standard step of GA introduces a specific threat to intracranial homeostasis: (1) Induction and Hypotension: The induction of anesthesia, particularly with intravenous agents like propofol, frequently causes significant hypotension. This is mediated by a combination of direct myocardial depression and profound systemic vasodilation. A sudden drop in MAP in a patient with an already elevated ICP can cause CPP to fall below the critical threshold for ischemia, precipitating a secondary ischemic stroke and worsening the primary injury; (2) Laryngoscopy and Hypertension: Conversely, the intense sympathetic stimulation of laryngoscopy and tracheal intubation can provoke a severe hypertensive and tachycardic response. This catecholamine surge can increase MAP by 40-50% or more. In a brain with failed autoregulation, this sharp rise in pressure is transmitted directly to the cerebral vasculature, causing a rapid increase in cerebral blood volume. This expansion within the rigid skull acutely increases ICP, which can lead to cerebral edema, trigger intracranial hemorrhage from injured vessels, and even precipitate brain herniation; (3) Positive Pressure Ventilation and Venous Congestion: Once intubated, the use of positive pressure ventilation can further elevate ICP by increasing intrathoracic pressure. This impedes cerebral venous outflow from the head via the internal jugular veins, effectively creating a "damming" effect. The resulting increase in cerebral venous blood volume contributes directly to higher ICP; (4) Volatile Anesthetics and Vasodilation: Many potent volatile

anesthetic agents (such as sevoflurane and desflurane) are direct cerebral vasodilators. Their use can increase cerebral blood volume and ICP, an effect that is particularly pronounced in patients with compromised intracranial compliance.[14]

The regional anesthesia technique employed in this case elegantly and comprehensively sidestepped every one of these hazards. As the intraoperative data demonstrated, a state of hemodynamic equipoise was maintained throughout the procedure. By completely avoiding instrumentation of the airway, the potent and dangerous hypertensive response to laryngoscopy was eliminated. By utilizing a technique that provides dense surgical anesthesia without causing systemic vasodilation or myocardial depression, the MAP was kept exceptionally stable, ensuring a consistent and adequate CPP.[15] Furthermore, the patient's ability to maintain spontaneous ventilation at normal tidal volumes prevented any significant increase in intrathoracic pressure, thus optimizing cerebral venous drainage and minimizing ICP. This meticulous preservation of stable cerebral hemodynamics is the very essence of neuroprotection and represents the most profound and undeniable advantage of a well-executed regional anesthetic over general anesthesia in this clinical context.

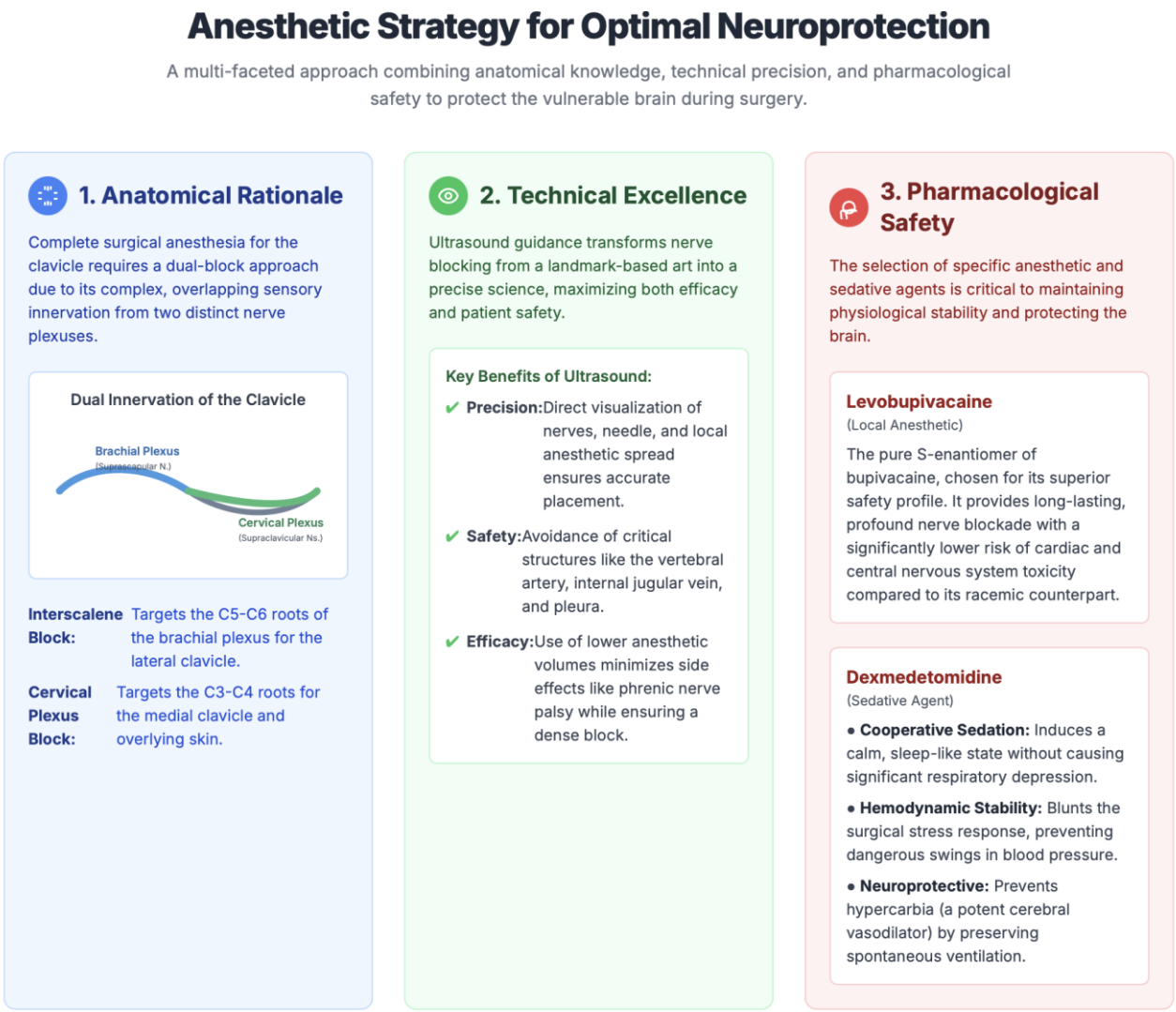


Figure 2. Anatomical, technical, and pharmacological strategy for optimal neuroprotection.

The successful implementation of this neuroprotective strategy was contingent upon a sophisticated understanding of anatomy, a high level of technical skill facilitated by modern technology, and a judicious pharmacological approach. The choice of a dual plexus blockade was dictated by the complex sensory innervation of the clavicle.[16] For complete surgical anesthesia, both the brachial and cervical plexuses must be reliably blocked. The suprascapular nerve, which arises from the superior trunk of the brachial plexus (formed by the C5 and C6 nerve roots), innervates the acromioclavicular joint and the majority of the bone and periosteum of the lateral two-thirds of the clavicle. The supraclavicular nerves (medial, intermediate, and lateral) arise from the superficial cervical plexus (C3 and C4 roots) and provide cutaneous innervation to the skin overlying the entire clavicle and the medial aspect of the bone itself. An interscalene block alone is therefore often insufficient, leading to intraoperative pain during manipulation of the medial clavicular segment Figure 2.[17]

The use of ultrasound guidance has transformed this procedure from a landmark-based estimation into a precise, anatomically-driven intervention. It is an essential component for ensuring both efficacy and safety. Direct visualization allows the practitioner to identify the brachial plexus roots nestled between the anterior and middle scalene muscles and to guide the needle tip precisely into the interscalene groove. It enables real-time confirmation of local anesthetic spread, ensuring the nerve targets are circumferentially bathed in the solution. Critically, ultrasound allows for the identification and avoidance of key vascular structures, such as the vertebral artery, transverse cervical artery, and internal jugular vein.

A crucial safety consideration in any interscalene block is the risk of iatrogenic phrenic nerve palsy. The phrenic nerve, which provides the sole motor innervation to the hemidiaphragm, is formed from the C3, C4, and C5 nerve roots and courses down the anterior surface of the anterior scalene muscle. In traditional, large-volume blocks, the local anesthetic inevitably spreads anteriorly to anesthetize the phrenic nerve, leading to hemidiaphragmatic paralysis in nearly 100% of cases. While often well-tolerated in healthy patients, this could be detrimental in a patient with any degree of respiratory compromise. Ultrasound allows for the use of lower, targeted volumes of local anesthetic, deposited precisely around the brachial plexus roots. This minimizes the anterior spread and can significantly reduce the incidence and severity of this complication.[18] The confirmed absence of diaphragmatic paralysis in our case highlights the precision and safety afforded by modern, ultrasound-guided techniques.

The choice of anesthetic agents was as critical as the technique itself. Levobupivacaine was selected as the local anesthetic due to its favorable safety profile. Local anesthetics function by blocking voltage-gated sodium channels. Bupivacaine is a chiral molecule, existing as two mirror-image enantiomers: the S(-) and R(+) forms. Racemic bupivacaine is a 50:50 mixture of both. It is the R(+) enantiomer that is predominantly responsible for the severe cardiotoxicity (ventricular arrhythmias, myocardial depression) and central nervous system toxicity seen with accidental intravascular injection. Levobupivacaine is the pure S(-) enantiomer, which has a significantly lower affinity for cardiac sodium channels and a wider margin of safety, making it a superior choice in high-risk patients or when large volumes of local anesthetic are required.[19]

The use of dexmedetomidine as the sole sedative agent was a cornerstone of the anesthetic plan, epitomizing the neuroprotective pharmacological approach. Dexmedetomidine is a highly selective alpha-2 adrenergic agonist. Its sedative and anxiolytic effects are mediated through its action on alpha-2 receptors in the locus coeruleus of the brainstem, which induces a state of "cooperative sedation" that closely mimics natural non-REM sleep. Unlike traditional sedatives like benzodiazepines or propofol, which are global CNS depressants acting on GABA receptors, dexmedetomidine does not significantly depress the respiratory drive. This unique property is invaluable in neuroanesthesia. It allows for a calm, comfortable, and cooperative patient who can still protect their own airway and maintain normal ventilation, thereby preventing hypercarbia—a potent cerebral vasodilator that can dangerously increase ICP. Furthermore, its sympatholytic properties blunt the systemic stress

response to surgery, contributing to hemodynamic stability. This ideal sedative profile, characterized by arousable sedation without respiratory compromise, allowed the patient to remain comfortable throughout a lengthy procedure while being immediately available for neurological assessment if needed. The remarkable stability and rapid recovery profile it afforded were the key factors that facilitated the patient's safe bypass of the Post-Anesthesia Care Unit (PACU), a notable benefit in terms of healthcare resource utilization and expedited patient flow, a decision not taken lightly in a patient with such a high-risk profile.[20]

While this case demonstrates a successful outcome, it is essential to acknowledge the inherent limitations of a single case report. First, the findings cannot be generalized to the broader population of post-craniotomy patients. The positive outcome may have been influenced by patient-specific factors, such as his young age and lack of other comorbidities. Second, this study design cannot establish causality. We can report a strong association between the chosen anesthetic technique and a stable clinical course, but it is impossible to conclude that this method is definitively superior to GA or that GA would have led to a worse outcome in this specific patient. Third, the procedure was performed by clinicians highly experienced in advanced ultrasound-guided regional anesthesia. The safety and efficacy reported here may not be reproducible by practitioners with less experience, representing a significant operator-dependent variable. Finally, while phrenic nerve palsy was avoided, it remains a significant potential risk of the interscalene block, even with modern techniques, and its occurrence could have been detrimental in this neurologically vulnerable patient.

4. Conclusion

The anesthetic management of a patient with a recent, severe traumatic brain injury for subsequent non-neurosurgical procedures demands a meticulous approach centered on the principle of avoiding secondary neurological insults. This case report provides compelling evidence that an ultrasound-guided dual plexus blockade is a highly effective and neurologically protective anesthetic strategy for clavicle fracture fixation in a post-craniotomy patient. This technique successfully provides dense surgical anesthesia while completely avoiding the hemodynamic volatility and airway manipulation inherent to general anesthesia. By ensuring stable cerebral perfusion pressure and minimizing intracranial pressure fluctuations, this regional anesthetic approach should be considered the primary and superior option for high-risk patients with compromised intracranial compliance, offering a pathway to essential orthopedic care without jeopardizing neurological recovery.

5. References

1. Pinto LDC, Silva CHR da, Gonçalves PMS e., Valadares RJ. Supraclavicular nerve and superior trunk block for surgical treatment of clavicle fracture in a patient with Steinert's disease – Case report. Braz J Anesthesiol. 2019; 69(1): 99–103.
2. Kishnani P, Joshi D, Sharma TH, Chauhan DK. Comparison of postoperative analgesic efficacy of 0.25% ropivacaine with dexmedetomidine versus dexamethasone as an adjuvant in bilateral superficial cervical plexus block for midline neck surgery under general anaesthesia: a randomised clinical study. J Clin Diagn Res. 2025.
3. Chen W, Xie K, Jiang Y, Zhang H, Wu D. Effect of bilateral superficial cervical plexus block on postoperative pain in thyroid and parathyroid surgery: a systematic review and meta-analysis. Med Sci Monit. 2025; 31: e949684.
4. Elsherbiny NM, Elebieby MGM, Badran AE, Mohammed MN. Superficial and deep cervical plexus blocks versus patient-controlled analgesia in total laryngectomy; a randomized trial. Pain Manag. 2025; 1–9.
5. Su L, Du Z. Enhancing the long-term benefits of ultrasound-guided modified intercostal nerve block in mastectomy: a call for comprehensive assessment. Asian J Surg. 2025; 48(9): 5470.
6. Tanigawa M, Kobayashi K, Naraghi L. A case series: Ultrasound-guided superficial cervical

plexus nerve block for musculoskeletal neck pain. J Emerg Med. 2025; 76: 1–6.
7. Gungor H, Ince A, Ciftci B, Emre Gölboyu B, Asici M, Karaaslan P, et al. Ultrasound-guided modified thoracoabdominal nerve block through perichondrial approach for postoperative analgesia management in living liver donors: a randomized, prospective, controlled study. Clin Transplant. 2025; 39(9): e70224.
8. Carda S, Grana E, Deltombe T, Reebye R. Ultrasound-guided multi-branch rectus femoris nerve block for spasticity assessment. Toxins (Basel). 2025; 17(9): 437.
9. Vu C, Willens SH, Alaniz L, Humphrey JG, Shah N, Vyas RM, et al. Ultrasound-guided maxillary nerve block in nasal surgery. Plast Reconstr Surg. 2025; 156(3): 362e–5e.
10. Sato T, Takakura M, Ozeki K, Akiyama K. A novel method of ultrasound-guided zygomaticotemporal nerve block for awake craniotomy. Korean J Anesthesiol. 2025.
11. Ikuta Y, Nakasa T, Fujii T, Yamasaki N, Ishibashi S, Sakurai S, et al. Arthroscopic ankle surgery under ultrasound-guided peripheral nerve block in patients with haemophilic ankle arthropathy-A report of five cases. Haemophilia. 2025; (hae.70132).
12. Wang X-X, Xu W-C, Fang T-F, Zhao Z-H, Lin K-N, Chen H. Safety and efficacy of ultrasound-guided distal forearm median nerve block for pediatric trigger thumb release: a retrospective observational study in a southern Chinese cohort. Front Med (Lausanne). 2025; 12(1648300): 1648300.
13. Zhang L, Liu Z, Guo J, Jin H, Zhang Z. Effectiveness of ultrasound-guided ilioinguinal and iliohypogastric nerve block in pediatric inguinal hernia surgery: a systematic review and meta-analysis of randomized controlled trials. Med Ultrason. 2025; 27(3): 323–30.
14. Pondeenana S, Wanjerdkit K, Nonsri C, Kositanurit I, Jongkongkawutthi R. Comparison of acute postoperative pain between preemptive ultrasound-guided pectoral nerve block and intraoperative pectoral nerve block in patients undergoing mastectomy. Asian Health SciTechRep. 2025; 33(3): 4118.
15. Driver L, Perice L, Brown JR, Nagdev A, Goldsmith A. Ultrasound-guided nerve blocks: expert opinion statement on patient monitoring in the emergency department setting. Intern Emerg Med. 2025.
16. Bao M, Zhang D, Liu L, Lin J, Zhao L, Li C. Ultrasound-guided nerve block anesthesia improves perioperative pain in patients undergoing free flap reconstruction for head-and-neck tumor: a randomized, controlled trial. Trials. 2025; 26(1): 345.
17. Cakmak G, Ciftci B, Vahapoglu A, Tunay A, Erden V. Comparison of ultrasound-guided pericapsular nerve group block versus intravenous ketamine for positional pain management before spinal anesthesia in patients who underwent hip fracture surgery: a randomized controlled trial. Med Sci Monit. 2025; 31.
18. Rao DYH. Efficacy of variable concentrations of ropivacaine in ultrasound-guided anterior sciatic nerve block for patients undergoing lower limb surgeries. Int J Environ Sci. 2025; 2555–60.
19. McKeeman J, Smith B, Malige A, Bates R, Pellegrino AN, Lachman J. Efficacy of liposomal bupivacaine in ultrasound-guided peripheral nerve blocks for postoperative pain management in foot and ankle surgery. Foot Ankle Int. 2025; (10711007251359634): 10711007251359634.
20. Kuntoadi D, Utama SP, Yuliana F, Hermawan A. Peripheral nerve block as a safer alternative than general anesthesia in high-risk patients with multiple comorbidities: a case report. Solo J Anesth Pain Crit Care. 2025; 5(1).