Anesthetic Management of Intracranial Space-Occupying Lesions: A Comprehensive Narrative Review

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ABSTRACT

Anesthetic management of patients with intracranial space-occupying lesions (ISOLs) is one of the most complex challenges in neuroanesthesia practice. These lesions—encompassing primary and metastatic brain tumors, abscesses, and vascular malformations—elevate intracranial pressure (ICP) and threaten cerebral perfusion pressure (CPP), necessitating a meticulous and individualized approach to every phase of anesthetic care. This narrative review provides a comprehensive overview of the pathophysiology of raised ICP, including the Monroe-Kellie doctrine, cerebrovascular autoregulation, and herniation syndromes, followed by a detailed discussion of preoperative assessment, anesthetic induction strategies, maintenance, intraoperative monitoring, and emergence. Particular emphasis is placed on pharmacological considerations—including the role of propofol-based total intravenous anesthesia, the limitations of volatile agents, and the management of neuromuscular blockade—as well as critical adjunctive measures such as osmotherapy, positioning, and normothermia. An understanding of these principles is essential for the safe perioperative management of patients undergoing neurosurgical procedures.

Keywords: neuroanesthesia; intracranial pressure; cerebral perfusion pressure; space-occupying lesion; total intravenous anesthesia; Monroe-Kellie doctrine

INTRODUCTION

Intracranial space-occupying lesions (ISOLs) encompass a broad spectrum of pathologies—including primary and metastatic neoplasms, brain abscesses, arteriovenous malformations, and cavernomas—that share the common feature of mass effect within the rigid calvarium. The neurosurgical and anesthetic management of these conditions requires an integration of fundamental neurophysiological principles with precise pharmacological and technical expertise [1].

The primary goal of neuroanesthesia in patients with ISOLs is to maintain adequate cerebral perfusion while avoiding further elevation of intracranial pressure (ICP). Secondary objectives include facilitating optimal surgical conditions, ensuring rapid and smooth emergence for neurological assessment, and preventing secondary brain injury from hypoxia, hypercapnia, hypotension, or hyperthermia [2]. This review synthesizes current evidence and clinical principles relevant to the anesthetic management of adult patients undergoing neurosurgical resection or intervention for intracranial space-occupying lesions.

PATHOPHYSIOLOGY

The Monroe-Kellie Doctrine

The cranial vault is a rigid, non-expansile structure containing three principal compartments: brain parenchyma (approximately 80% of intracranial volume), cerebral blood volume (approximately 10%), and cerebrospinal fluid (CSF, approximately 10%). According to the Monroe-Kellie doctrine, the total intracranial volume is fixed; any increase in one compartment must be offset by a reciprocal decrease in another to maintain normal ICP (reference range: 5–15 mmHg in adults) [1].

Intracranial space-occupying lesions constitute a fourth, pathological compartment. Initial compensation is achieved by displacement of CSF into the spinal subarachnoid space and by compression of venous sinuses. Once these compensatory mechanisms are exhausted—at a critical threshold—small incremental increases in lesion volume precipitate exponential rises in ICP. The anesthesiologist must understand this pressure-volume relationship, as any intraoperative maneuver that increases cerebral blood volume (e.g., hypercapnia, coughing, Valsalva) may provoke acute ICP crises at this vulnerable stage [1,2].

Cerebral Perfusion Pressure and Autoregulation

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure (MAP) and ICP: CPP = MAP − ICP. Adequate CPP (target: 60–70 mmHg in most adult patients) is essential to prevent cerebral ischemia. In the healthy brain, cerebrovascular autoregulation maintains constant cerebral blood flow (CBF) over a wide range of MAPs (approximately 50–150 mmHg). However, this autoregulatory capacity is frequently impaired in the vicinity of brain tumors and other ISOLs, rendering perfusion pressure-passive and making the peritumoral tissue exquisitely vulnerable to systemic hemodynamic fluctuations [3].

Cerebral metabolic rate for oxygen (CMRO2) and CBF are tightly coupled under physiological conditions. Hypercarbia increases CBF by cerebrovascular vasodilation, thereby raising ICP; hypocarbia causes vasoconstriction and reduces ICP. Controlled mild hyperventilation (target PaCO2: 35 mmHg) may be used transiently during intraoperative ICP crises, although prolonged hypocarbia risks cerebral ischemia through excessive vasoconstriction [2].

Brain Edema and Herniation Syndromes

Perilesional edema, predominantly vasogenic in origin in the context of brain tumors (reflecting disruption of the blood-brain barrier), amplifies mass effect and ICP elevation. Vasogenic edema responds well to corticosteroids, particularly dexamethasone, which should be initiated 24–48 hours preoperatively in elective cases. Cytotoxic edema, arising from cellular injury (e.g., post-ischemic), does not respond to steroids [1].

Uncompensated ICP elevation leads to herniation syndromes. Transtentorial (uncal) herniation, characterized by ipsilateral pupillary dilatation and contralateral hemiparesis, reflects uncal compression of the oculomotor nerve and cerebral peduncle. Central transtentorial herniation presents with the Cushing triad (hypertension, bradycardia, irregular respirations)—a late, ominous sign mandating emergency neurosurgical decompression. Tonsillar herniation through the foramen magnum causes sudden respiratory arrest. Recognition of these syndromes is essential for both preoperative assessment and intraoperative management [2,3].

PREOPERATIVE ASSESSMENT

Clinical and Radiological Evaluation

Preoperative assessment begins with an evaluation of ICP status. Clinical indicators of raised ICP include morning-predominant headache (reflecting nocturnal ICP elevation in the recumbent position), nausea and vomiting (particularly projectile), papilloedema on fundoscopy, altered consciousness (GCS <14), and focal neurological deficits. A history of seizures, current anticonvulsant therapy, corticosteroid use, and airway-related concerns (radiation fibrosis, cervical instability) must be elicited [2].

Neuroimaging must be reviewed systematically. Midline shift on CT or MRI of >5 mm indicates significant mass effect; >10 mm is considered critical. Perilesional T2/FLAIR hyperintensity delineates the extent of vasogenic edema. Hydrocephalus (ventricular dilatation) may necessitate preoperative ventricular drainage. Contrast enhancement indicates blood-brain barrier disruption and is typical of high-grade gliomas and abscesses. Posterior fossa lesions

carry additional risk of acute hydrocephalus from CSF pathway obstruction and proximity to brainstem structures [1].

Emergency vs. Elective Classification

The urgency of surgery fundamentally shapes the anesthetic plan. Emergency indications—including rapid neurological deterioration, herniation signs, acute hydrocephalus, or intratumoral hemorrhage—preclude extended preoperative optimization. In these settings, modified rapid sequence induction (RSI) is preferred, with ICP-attenuating measures (controlled hyperventilation, mannitol bolus) applied concurrently. Elective cases permit preoperative optimization: dexamethasone administration for at least 24–48 hours, correction of electrolyte disturbances (hyponatremia is particularly common in brain tumor patients), and review of anticonvulsant drug levels [3].

Premedication should be used cautiously. Benzodiazepines may cause hypercapnia and raise ICP; if used at all, doses should be minimized or avoided entirely. Antiemetics are routinely administered, as vomiting produces Valsalva-mediated ICP spikes. Dexamethasone is continued perioperatively [2].

ANESTHETIC INDUCTION

Pharmacological Considerations

Induction in patients with ISOLs must balance two competing priorities: preventing the ICP surge provoked by laryngoscopy and intubation, while avoiding the hypotension that reduces CPP. Propofol is the preferred induction agent, as it reduces CMRO2 and CBF, lowers ICP, preserves autoregulation, and has anticonvulsant properties. Hypotension is a recognized risk, particularly in hypovolaemic patients, and must be anticipated with adequate volume loading and vasopressor readiness [2,4].

Ketamine was historically considered contraindicated in ISOLs due to its sympathomimetic cerebrovascular effects. Contemporary evidence, however, suggests that under controlled ventilation with concurrent propofol administration, ketamine does not significantly elevate ICP and may be used in carefully selected cases, particularly where haemodynamic stability is paramount. Thiopental, though theoretically advantageous for its CMRO2 suppression, has largely been supplanted by propofol due to its prolonged emergence and cardiovascular depression. Etomidate offers haemodynamic stability but carries the risk of myoclonus (which may be confused with seizure activity) and adrenocortical suppression with repeated dosing [4].

Succinylcholine causes transient ICP elevation via muscle fasciculation-mediated increases in cerebral venous pressure. In patients with ISOLs and elevated ICP, a modified RSI using high-dose rocuronium (1.2 mg/kg) is preferred, as it achieves rapid intubating conditions without the ICP liability of succinylcholine. The addition of lidocaine (1.5 mg/kg IV) 2–3 minutes before laryngoscopy may attenuate the sympathetic response to intubation, though evidence for this practice is limited [1,2].

ANESTHETIC MAINTENANCE

Total Intravenous Anesthesia vs. Inhalational Agents

Total intravenous anesthesia (TIVA) with propofol and an opioid (typically remifentanil or fentanyl) is the preferred maintenance technique for patients with ISOLs. Propofol reduces ICP, preserves autoregulation, and facilitates rapid, titratable emergence. Remifentanil, with its context-insensitive half-life and metabolism via non-specific plasma esterases, provides excellent intraoperative analgesia without the risk of opioid accumulation, enabling smooth and predictable awakening [4].

Volatile anesthetic agents (isoflurane, sevoflurane, desflurane) cause dose-dependent cerebrovascular vasodilation, increasing CBF and ICP—effects that are particularly pronounced above 1 MAC. At sub-MAC concentrations, the CMRO2-reducing effects may partially offset the vasodilatory ICP elevation, but autoregulation is progressively impaired. Nitrous oxide is generally avoided in neurosurgery due to its propensity to expand intracranial gas spaces and increase CMRO2. If volatile agents are used, concentrations should be maintained below 1 MAC and combined with controlled hypocapnia [3,4].

Intraoperative Monitoring

Standard monitoring (ECG, pulse oximetry, capnography, invasive arterial pressure, urinary output) is supplemented in neuroanesthesia by specific modalities. Invasive arterial blood pressure monitoring via radial artery catheterization provides beat-to-beat hemodynamic surveillance and enables frequent arterial blood gas sampling for PaCO2 optimization. Target MAP is individualized to maintain CPP ≥60 mmHg, accounting for the measured or estimated ICP [2].

Bispectral index (BIS) monitoring facilitates titration of propofol in TIVA, with a target range of 40–60 providing adequate anesthetic depth while avoiding oversedation that may compromise hemodynamic stability and delay emergence. Neuromuscular blockade monitoring (train-of-four stimulation) guides rocuronium dosing and ensures complete reversal

before extubation, which is critical to prevent coughing-induced ICP elevation. In selected cases, neurophysiological monitoring (somatosensory evoked potentials, motor evoked potentials, electrocorticography) may be employed, with implications for anesthetic drug selection—TIVA is strongly preferred when such monitoring is used, as volatile agents suppress cortical potentials [1,4].

Osmotherapy and ICP Management

Mannitol (0.25–1 g/kg IV) is the most widely used osmotic agent for intraoperative ICP reduction, acting by creating an osmotic gradient that draws water from the brain parenchyma into the intravascular compartment. Its onset is rapid (15–30 minutes) and duration 2–6 hours. Serum osmolality should be monitored and maintained below 320 mOsm/kg to prevent renal toxicity. Hypertonic saline (3% or 23.4%) is an alternative with similar efficacy and a favorable profile in patients with concurrent hypovolemia [2].

Head-of-bed elevation to 15–30 degrees facilitates venous drainage from the cranium and reduces ICP without compromising CPP in most patients. Careful patient positioning to avoid jugular venous compression (neutral head position, avoidance of extreme neck flexion or rotation) is essential. Normothermia should be maintained throughout, as fever increases CMRO2 and worsens ischemic injury [3].

EMERGENCE AND EXTUBATION

Emergence from neuroanesthesia is a critical and high-risk phase. Coughing, straining, and acute hypertension during extubation provoke ICP surges that may be catastrophic in patients with residual mass effect or intracranial hemorrhage. Smooth emergence is facilitated by anticipating the transition from deep anesthesia, ensuring adequate analgesia prior to extubation, and administering lidocaine or a short-acting opioid (remifentanil) to attenuate airway reflexes [4].

Extubation criteria in neurosurgical patients are more stringent than in the general surgical population: the patient must be fully awake, following commands, protecting the airway, and breathing spontaneously with adequate tidal volumes. Any new neurological deficit—motor weakness, pupillary asymmetry, altered consciousness—detected during emergence warrants urgent neurosurgical reassessment. Delayed awakening requires systematic exclusion of metabolic, pharmacological, and structural causes [1,2].

SPECIAL CONSIDERATIONS: AWAKE CRANIOTOMY

Awake craniotomy with intraoperative language or motor mapping is an established technique for resection of lesions in or adjacent to eloquent cortex. Anesthetic management involves a sleep-awake-sleep or monitored anesthesia care paradigm, requiring careful patient selection, meticulous airway planning, and excellent communication between the anesthetic and surgical teams. The anesthesiologist must ensure patient comfort and cooperation during the awake phase while being prepared for urgent conversion to general anesthesia in the event of seizure, airway compromise, or patient distress [3].

CONCLUSION

The anesthetic management of patients with intracranial space-occupying lesions demands a thorough understanding of intracranial pressure physiology, cerebrovascular pharmacology, and the specific risks of each phase of the perioperative period. TIVA with propofol and remifentanil provides the most favorable neurophysiological profile and is the preferred technique in most clinical scenarios. Meticulous attention to ICP management, hemodynamic stability, and smooth emergence is essential to optimize both safety and postoperative neurological outcomes. Ongoing advances in intraoperative monitoring, targeted osmotherapy, and individualized anesthetic regimens continue to refine neuroanesthesia practice.

AUTHOR CONTRIBUTIONS

Concept and design: S.D. Data collection: S.D. Data analysis and interpretation: S.D. Manuscript writing: S.D. Critical revision: S.D. Final approval: S.D.

CONFLICT OF INTEREST

The author declares no conflict of interest.

REFERENCES

1. Prabhakar H, Mahajan C, Kapoor I. Essentials of Neuroanesthesia. Academic Press; 2017.
2. Cottrell JE, Patel P. Cottrell and Patel's Neuroanesthesia. 6th ed. Elsevier; 2017.
3. Brambrink AM, Kirsch JR. Anesthesia for Neurosurgery. In: Miller RD, ed. Miller's Anesthesia. 9th ed. Elsevier; 2020:2122–2171.
4. Engelhard K, Werner C. Inhalational or intravenous anesthetics for craniotomies? Pro inhalational. Curr Opin Anaesthesiol. 2006;19(5):504–508.
5. Todd MM, Warner DS, Sokoll MD, et al. A prospective, comparative trial of three anesthetics for elective supratentorial craniotomy. Anesthesiology. 1993;78(6):1005–1020.
6. Talke PO, Gelb AW. Postcraniotomy pain remains a real headache! Eur J Anaesthesiol. 2005;22(5):325–327.