Review

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Effects of anesthesia on tumor perfusion and infiltration during brain tumor neurosurgery

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Abstract

Brain tumor surgeries are among the most challenging procedures in neurosurgery, necessitating careful anesthetic management to achieve maximal tumor resection while preserving neurological function. Anesthesia affects key physiological parameters, including cerebral blood flow, intracranial pressure, and blood-brain barrier permeability, which are crucial in determining surgical outcomes. This mini-review examines the effects of various anesthetic agents, such as propofol, isoflurane, and sevoflurane, on tumor perfusion and infiltration, highlighting their potential to modulate tumor biology through mechanisms involving immune response, angiogenesis, and molecular pathways. The mini-review identifies critical gaps in the literature, particularly concerning the long-term impacts of anesthetic agents on tumor biology and patient survival, and calls for well-conducted randomized controlled trials to address these issues. Furthermore, it explores innovative strategies, including targeted anesthesia delivery and the integration of genomic and proteomic data, to enhance personalized patient care. By synthesizing current knowledge and identifying areas for future research, this mini-review aims to provide a foundation for optimizing anesthetic protocols in brain tumor surgeries to improve both short- and long-term patient outcomes.

Keywords: anesthesia, brain tumor surgery, neuroanesthesia, neurosurgery, tumor infiltration, tumor perfusion

Introduction

Brain tumor surgeries are among the most complex and critical procedures in neurosurgery due to the intricate anatomy of the brain and the need to balance maximal tumor resection with the preservation of neurological function. Brain tumors account for a significant proportion of intracranial neoplasms, and optimizing surgical outcomes is vital to enhancing patient survival and quality of life[1]. Anesthesia plays a crucial role in these surgeries, influencing not only sedation and pain management but also critical aspects of tumor physiology, such as perfusion and infiltration, which are essential determinants of surgical success and long-term patient prognosis[2].

HIGHLIGHTS

- Propofol lowers CBF, ICP, and supports BBB integrity during brain tumor surgery.
- Volatile agents raise CBF, promote angiogenesis, and disrupt the tumor microenvironment.
- Propofol preserves immunity; volatile agents suppress NK and T-cell activity.
- Propofol linked to better recovery, less cognitive dysfunction post-surgery.
- Tailored anesthesia based on tumor and patient profile improves outcomes.

The impact of anesthesia on brain tumor surgery is mediated through several mechanisms, including alterations in cerebral blood flow (CBF), intracranial pressure (ICP), and blood-brain barrier (BBB) permeability. Different anesthetic agents, such as propofol, isoflurane, and sevoflurane, have varying effects on these parameters, which can consequently influence tumor perfusion and infiltration[3]. For instance, inhalational anesthetics like isoflurane may increase CBF due to vasodilation, while intravenous agents such as propofol tend to decrease cerebral metabolism, leading to reduced CBF and potentially limiting tumor perfusion[4]. Additionally, anesthetics can modify the tumor microenvironment and alter immune responses, further affecting tumor behavior during and after surgery[5].

Despite advances in neuroanesthesia and the use of sophisticated intraoperative monitoring techniques, the influence of anesthesia on brain tumor perfusion and infiltration remains an area of active research. Several studies have suggested that certain anesthetic agents could potentially promote tumor recurrence or metastasis by modulating factors such as hypoxia,

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angiogenesis, and immune surveillance[6]. However, evidence in this area remains mixed, and there is a pressing need for well-conducted clinical trials to better elucidate these relationships. While some research indicates that volatile anesthetics may enhance tumor growth through various signaling pathways, others have shown their potential protective effects against tumor spread, illustrating the complex interplay between anesthesia, tumor biology, and surgical outcomes[7].

The objective of this mini-review is to explore the mechanisms by which anesthesia affects tumor perfusion and infiltration during brain tumor surgery and to analyze the clinical implications of these effects. This mini-review aims to address critical gaps in the current understanding of anesthesia's role in modulating tumor behavior, thereby providing a foundation for refining anesthetic protocols to optimize surgical outcomes. The novelty of this study lies in its comprehensive examination of both the direct and indirect effects of anesthetic agents on brain tumor physiology and its potential to inform clinical practices and guidelines in neuro-oncological surgery. By integrating recent findings and emerging insights, this mini-review will contribute to a clearer understanding of how anesthetic choices can be tailored to improve both short- and long-term patient outcomes.

Overview of anesthesia in brain tumor surgery

In brain tumor surgeries, the primary types of anesthesia utilized are general, regional, and local anesthesia, with a predominant reliance on general anesthesia. General anesthesia, typically administered through inhalation agents (such as sevoflurane and isoflurane) or intravenous agents (like propofol), is favored for its ability to provide deep sedation, pain control, and muscle relaxation, essential for the controlled surgical environment required in craniotomies and tumor resections[8]. While regional anesthesia, such as scalp nerve blocks, may be used adjunctively to manage perioperative pain, local anesthesia is rarely employed as a primary method due to the invasive and complex nature of these surgeries[9].

The most commonly used anesthetic agents in brain tumor surgeries include propofol, sevoflurane, and isoflurane. Propofol, an intravenous anesthetic, is known for its rapid onset, short duration, and reduced postoperative nausea and vomiting. It also has immunomodulatory and anti-inflammatory properties, which are considered beneficial in reducing cancer recurrence and metastasis[9]. Conversely, inhalation agents like sevoflurane and isoflurane are widely used for their ease of administration and potent anesthetic effects, but they may increase CBF and ICP, which could complicate surgeries involving brain tumors[10]. Studies have suggested that sevoflurane and isoflurane might promote tumor cell growth through pathways involving hypoxia-inducible factors, unlike propofol, which is associated with suppressing these pathways[11].

The choice of anesthesia in brain tumor surgeries is influenced by several factors, including the tumor type, location, and patient-specific considerations such as age and comorbidities. For example, tumors located near critical brain structures or those associated with elevated ICP may necessitate the use of agents like propofol that minimize CBF and pressure changes[8]. Additionally, patient factors such as age, cardiovascular stability, and existing neurological conditions might dictate the preference for specific anesthetic regimens. For instance, in patients with Parkinson's disease undergoing brain surgery, the choice between propofol and sevoflurane can impact postoperative outcomes like delirium, necessitating a tailored anesthetic approach[10].

Mechanisms of anesthesia affecting tumor perfusion

Hemodynamic effects of anesthesia

Anesthesia significantly impacts CBF through mechanisms involving both vasodilation and vasoconstriction. Volatile anesthetic agents such as isoflurane and sevoflurane are known to cause cerebral vasodilation, which can increase CBF. This effect occurs primarily due to the direct action of these agents on vascular smooth muscle cells, resulting in the relaxation of cerebral vessels. The degree of vasodilation and resulting changes in CBF vary depending on the concentration and duration of exposure to the anesthetic agent. On the other hand, propofol, an intravenous anesthetic, tends to decrease CBF by reducing cerebral metabolism and subsequently lowering the demand for blood flow, a phenomenon known as flow-metabolism coupling[8,12]. The relative changes in CBF and ICP under propofol and volatile agents are illustrated conceptually in Fig. 1. As evidenced by previous studies, propofol is known to reduce CBF. However, in most cases, this reduction does not significantly impact cerebral metabolism. This is largely due to the brain's intrinsic mechanisms, such as cerebral autoregulation and CO_2 reactivity, which play a crucial role in preserving metabolic stability despite changes in perfusion[13,14].

Changes in ICP also play a critical role in influencing tumor perfusion during brain surgery. Elevated ICP can compress cerebral vessels, reducing perfusion pressure and potentially leading to cerebral ischemia. Volatile anesthetics such as sevoflurane and isoflurane may increase ICP by causing cerebral vasodilation and an increase in cerebral blood volume, whereas propofol is more likely to maintain a stable or reduced ICP, thereby potentially offering a more controlled perfusion environment[15]. Effective management of ICP is crucial during brain tumor surgeries to ensure adequate perfusion to the surrounding brain tissue while avoiding exacerbation of cerebral edema or hemorrhage[16].

Brain tumors are commonly associated with increased ICP, which can lead to cerebral edema and further compromise cerebral perfusion[17]. In this context, the choice of anesthetic agents plays a critical role in managing ICP. Volatile anesthetics, such as sevoflurane and isoflurane, are known to cause cerebral vasodilation, which can further elevate ICP and exacerbate cerebral edema[17,18]. In contrast, propofol has been shown to reduce CBF and ICP, making it a preferred option in neurosurgical procedures where intracranial hypertension is a concern[3,19].

Anesthetic agents can also modify the permeability of the BBB, which is pivotal in regulating drug delivery to the tumor site. The BBB serves as a critical barrier that restricts the entry of most systemic drugs into the brain. Volatile anesthetics like isoflurane and sevoflurane have been shown to alter the expression and integrity of tight junction proteins, such as claudins and occludins, in the endothelial cells of the BBB, potentially leading to increased permeability. This disruption of the BBB can result in vasogenic edema, which may complicate surgical outcomes by increasing the risk of brain swelling and intracranial

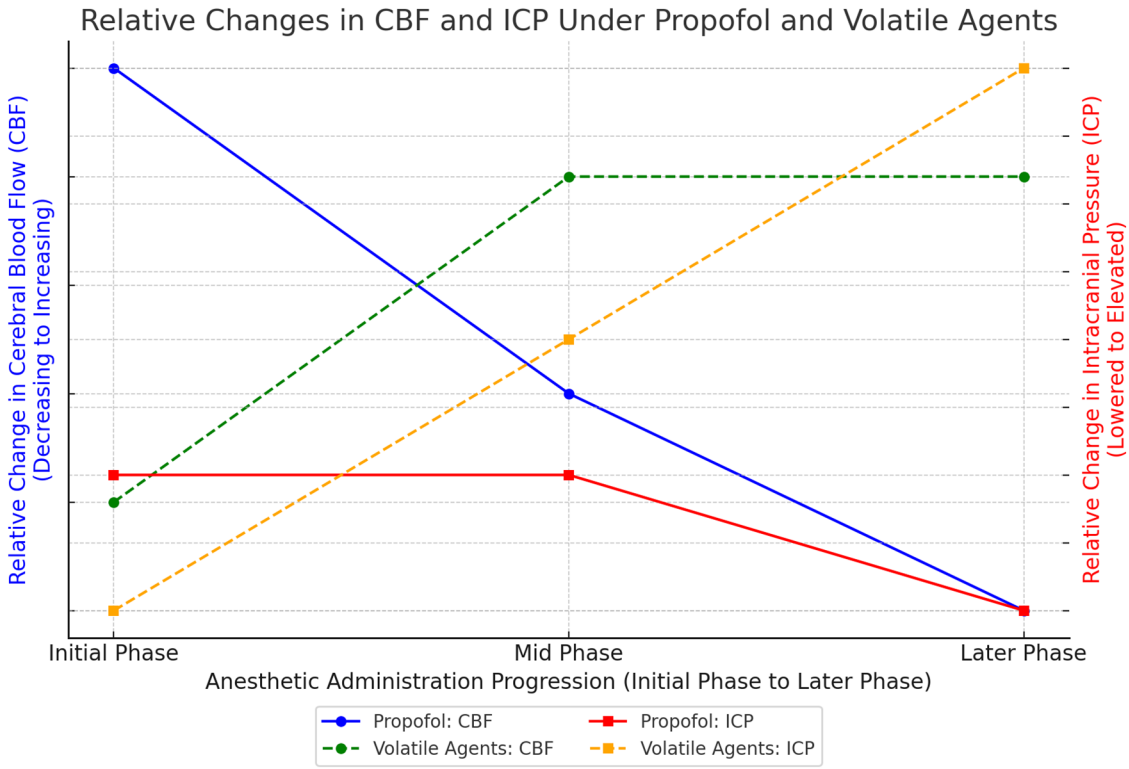

Figure 1. Relative changes in CBF and ICP under propofol and volatile agents.

hypertension[12]. Conversely, agents like propofol have demonstrated protective effects on the BBB, potentially maintaining its integrity better than volatile anesthetics. This characteristic may enhance drug delivery to the tumor site while minimizing the risk of edema and related complications[8,15].

Thus, understanding the hemodynamic effects of anesthesia, including its impact on CBF, ICP, and BBB permeability, is crucial for optimizing brain tumor surgery outcomes. The choice of anesthetic agent should consider the tumor's location, patient-specific factors, and the overall goals of the surgical procedure to balance adequate perfusion and minimize the risk of adverse effects.

The mechanisms by which different anesthetic agents modulate CBF, ICP, and tumor perfusion are summarized in Fig. 2.

Anesthetic agents and tumor microenvironment

Different anesthetic agents can significantly influence the tumor microenvironment, particularly by affecting tumor vasculature. Volatile anesthetics, such as isoflurane and sevoflurane, are known to promote vasodilation, which can increase blood flow within the tumor microenvironment. This increase in blood flow potentially enhances the delivery of oxygen and nutrients to tumor cells, supporting their growth and proliferation. Additionally, volatile anesthetics may elevate the expression of pro-angiogenic factors like vascular endothelial growth factor (VEGF), which is crucial in promoting the formation of new blood vessels within the tumor, facilitating further tumor growth and metastasis. This effect is particularly relevant in established tumors rather than in the initial, preneoplastic phase[20,21]. In contrast, intravenous anesthetics such as propofol exhibit anti-inflammatory and anti-angiogenic properties. Propofol has been shown to downregulate VEGF expression, thereby inhibiting angiogenesis and reducing blood vessel formation in the tumor microenvironment[22]. Most studies suggest that propofol-based anesthesia may be more beneficial than inhalational agents in reducing cancer recurrence. Retrospective research has associated volatile anesthetics with lower recurrence-free survival in cancer patients undergoing elective surgery[23,24].

The choice of anesthetic agent can differentially affect tumor perfusion. Volatile anesthetics may enhance tumor perfusion and angiogenesis, potentially worsening outcomes by supporting tumor growth, whereas intravenous agents like propofol may limit these processes through their anti-angiogenic effects, thus potentially offering a protective role against tumor progression[25].

Systemic vs. local effects

Anesthesia affects both systemic and localized physiological processes. Systemically, anesthetic agents can cause cardiovascular changes such as hypotension and reduced cardiac output, which may indirectly impact tumor perfusion by decreasing perfusion pressure and oxygen delivery to the tumor[22]. Locally, the effects of anesthesia on cerebral vasculature include alterations in CBF and metabolism. Volatile anesthetics typically increase CBF due to vasodilation, which may enhance perfusion in brain tumors, potentially promoting tumor progression[24]. To counteract anesthesia-induced hypotension and diminished cardiac output, anesthesiologists may administer vasopressors and inotropes to induce vasoconstriction and

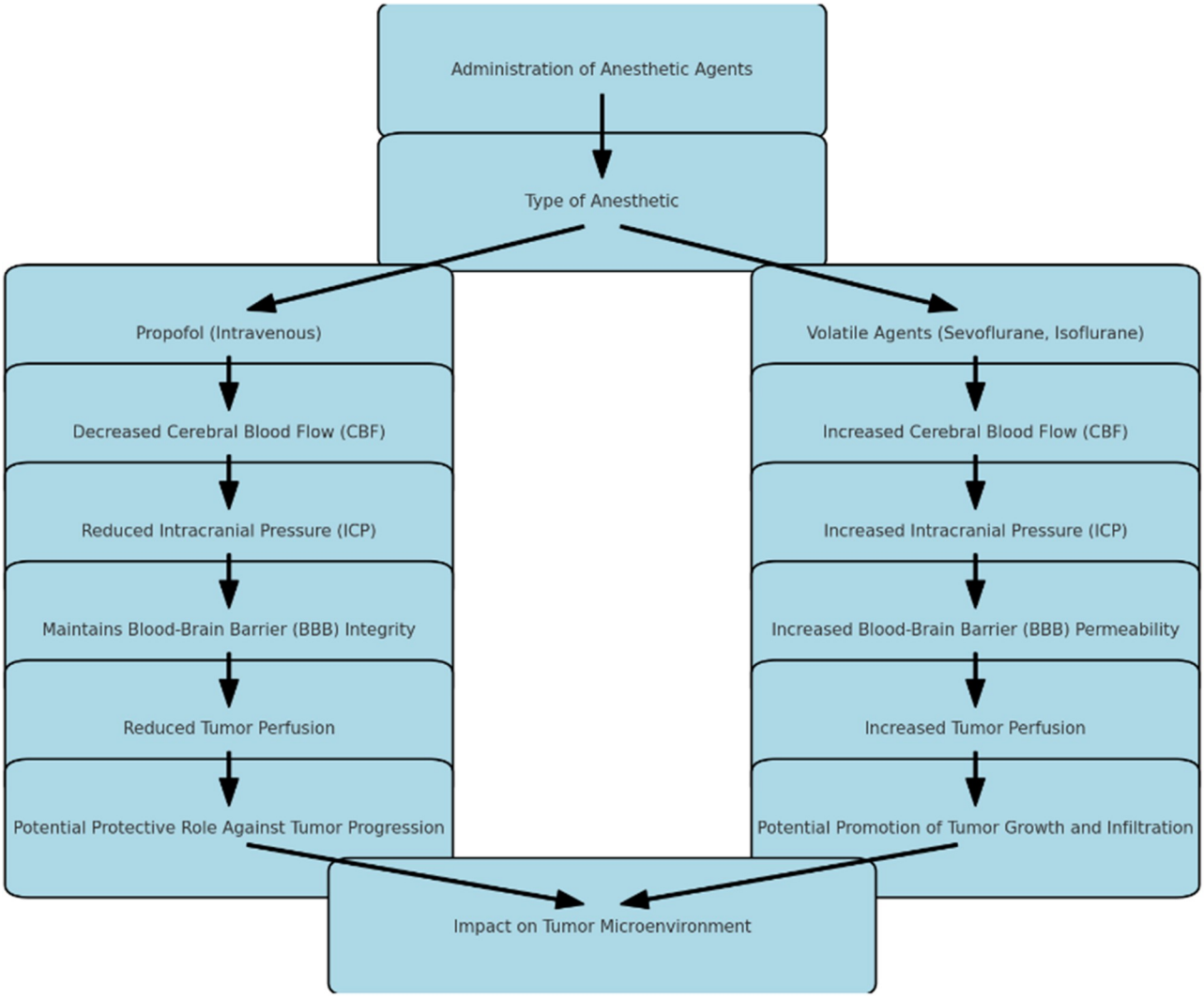

Figure 2. Mechanisms of anesthesia affecting tumor perfusion.

improve cardiac output[25,26]. Nevertheless, it is important to keep in mind that excessive vasoconstriction may impair perfusion to vital organs, and inotropes can increase myocardial oxygen demand, potentially leading to ischemia and arrhythmias with prolonged use[25].

Hypoxia, hypercapnia, and metabolic alterations induced by anesthesia also significantly impact tumor perfusion. Anesthetics that increase CBF, such as isoflurane and sevoflurane, may exacerbate tumor hypoxia by increasing oxygen consumption within the tumor microenvironment[25]. Conversely, propofol may reduce metabolic demands and oxygen consumption, thereby mitigating tumor hypoxia and potentially limiting tumor growth and metastasis[24].

In this context, anesthesiologists can modify ventilation settings to correct hypoxia during anesthesia. Strategies include increasing the fraction of inspired oxygen (FiO_2), optimizing tidal volume and respiratory rate, and applying positive end-expiratory pressure (PEEP) to enhance oxygenation and prevent hypoxia[26]. However, excessive PEEP may reduce venous return and lower cardiac output, potentially worsening hypotension[27].

By understanding these differential effects, clinicians can tailor anesthetic strategies to optimize both surgical and oncologic outcomes, taking into account individual tumor characteristics and patient health status.

Mechanisms of anesthesia affecting tumor infiltration

Impact on tumor cell migration

Anesthetic agents can significantly influence molecular pathways related to tumor cell adhesion, motility, and invasion. Volatile anesthetics, such as isoflurane and sevoflurane, have been reported to affect the activity of matrix metalloproteinases (MMPs) and integrins, which are crucial in facilitating tumor cell invasion. MMPs degrade the extracellular matrix (ECM), a process that allows tumor cells to penetrate surrounding tissues and metastasize. Volatile anesthetics may upregulate MMP expression, thereby enhancing tumor cell invasiveness[25]. Conversely, intravenous anesthetics like propofol appear to

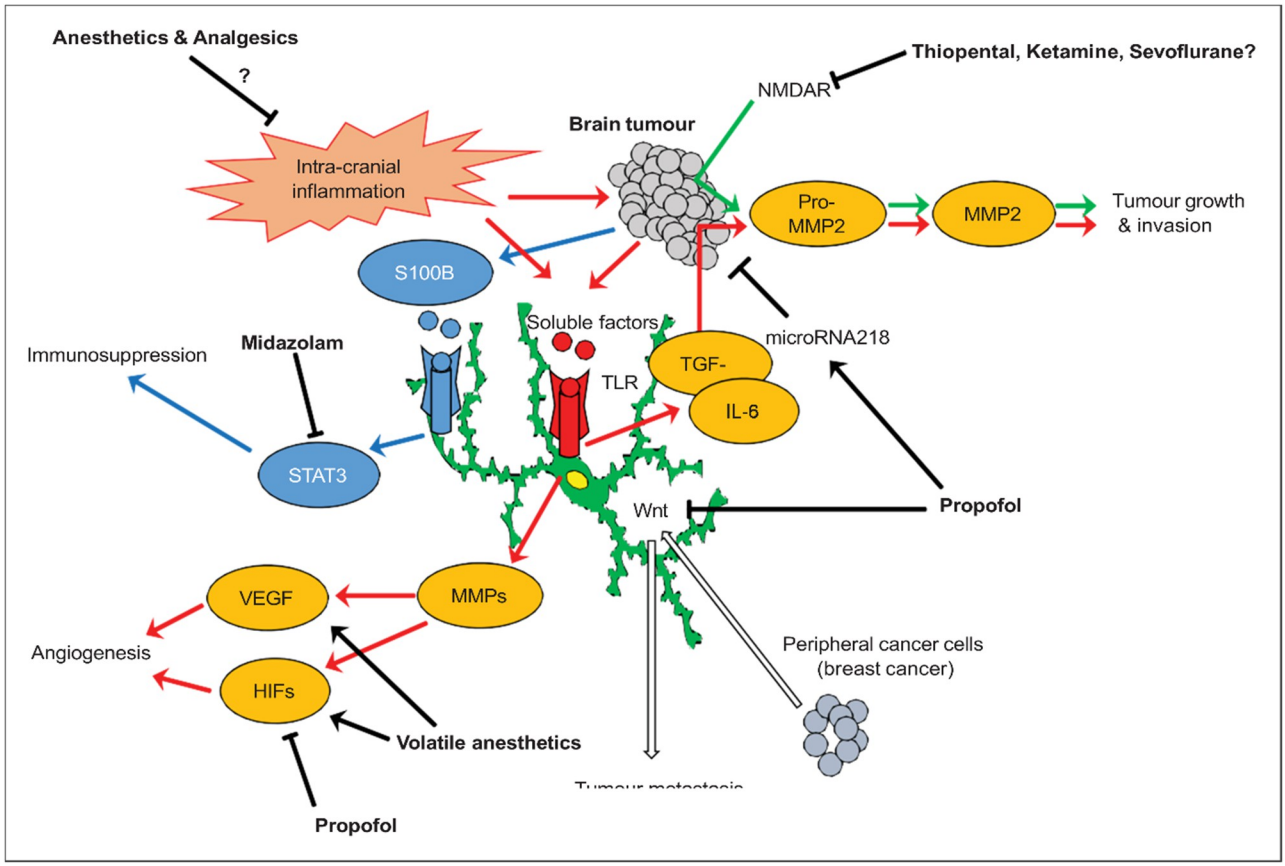

Figure 3. Molecular interactions influenced by anesthetic agents[30].

inhibit MMP activity and downregulate integrin expression, reducing tumor cell adhesion and motility[28].

Anesthetics also modify the ECM around the tumor. The ECM is a structural network that provides mechanical support to cells; its alteration can significantly impact tumor infiltration. Volatile anesthetics may contribute to ECM breakdown by increasing collagen degradation, facilitating tumor cell movement. In contrast, intravenous agents such as propofol might maintain ECM integrity by limiting degradation processes, potentially reducing the risk of metastasis[28].

Anesthesia-induced modulation of the immune response

Different anesthetic agents can modulate immune cell function and tumor immune evasion. Volatile anesthetics, such as sevoflurane, have been shown to suppress the function of natural killer (NK) cells and cytotoxic T-cells, both of which play a crucial role in detecting and eliminating tumor cells[29]. This suppression may facilitate tumor immune evasion, allowing for unchecked growth and spread. In contrast, propofol has been demonstrated to preserve or enhance NK cell function and T-cell activity, thereby promoting an effective immune response against tumor cells[28].

Furthermore, anesthesia affects inflammation and cytokine release, influencing tumor progression and infiltration. Volatile anesthetics tend to promote an anti-inflammatory environment by reducing the release of pro-inflammatory cytokines such as interleukins (IL-6, IL-8) and tumor necrosis factor-alpha. The effects of anesthetic agents, including their modulation of molecular pathways such as TLR, MMPs, STAT3, and IL-6, and their role in influencing immune suppression, tumor invasion, and angiogenesis, are summarized in Fig. 3[30]. While this can minimize surgical stress, it may also weaken the anti-tumor immune response. On the other hand, propofol helps maintain a balanced immune environment by modulating cytokine release, preserving inflammatory responses necessary for effective tumor immunosurveillance[30].

The impact of anesthesia on immune modulation is particularly relevant in the context of chimeric antigen receptor (CAR) T cell therapy, which relies on functional cytotoxic T-cells to target tumor cells[31]. Volatile anesthetics like sevoflurane may suppress T-cell activity, potentially diminishing CAR-T cell efficacy[29], whereas propofol has been shown to preserve immune function and may be a more favorable option for patients undergoing immunotherapy. Further research is needed to optimize anesthetic strategies that support immune-based cancer treatments while minimizing tumor progression risks[28].

Interactions with surgical techniques

Anesthetic management directly affects the precision and safety of surgical resections, which impacts tumor infiltration and local recurrence. Deep anesthesia induced by volatile agents may impair the surgeon's ability to achieve precise tumor margins due to changes in brain tissue consistency and perfusion,

potentially increasing the risk of leaving residual tumor cells and, consequently, the rate of recurrence[29]. In contrast, intravenous anesthetics like propofol, with their neuroprotective effects and stable hemodynamic profile, may facilitate better surgical precision, thus reducing the likelihood of local recurrence[28,30].

Anesthetics also affect intraoperative imaging techniques, such as magnetic resonance imaging (MRI) and ultrasound, that guide tumor resection[33]. Volatile anesthetics can alter brain tissue signal intensity on MRI scans, making it more challenging to delineate tumor margins[34]. In contrast, intravenous agents like propofol may offer more consistent imaging conditions, allowing for more accurate tumor resection[31].

Clinical implications and considerations

Selection of anesthetic protocols

Selecting the appropriate anesthetic protocol for brain tumor surgeries is crucial and should be individualized based on tumor characteristics, patient conditions, and anticipated intraoperative challenges. For tumors located in eloquent brain areas or regions with high vascularity, intravenous anesthetics like propofol may be preferable due to their ability to reduce CBF and ICP, thereby minimizing the risk of bleeding and facilitating precise resection[35]. For patients with pre-existing cardiovascular conditions, volatile anesthetics such as sevoflurane, which provide stable hemodynamics, may be a better choice; however, these agents can potentially increase the risk of tumor recurrence by promoting angiogenesis and tumor cell migration. The choice of anesthesia directly influences both short- and long-term outcomes, such as neurocognitive function and overall survival, underscoring the need for a personalized approach[31].

Implications for postoperative outcomes

Anesthetic management significantly impacts postoperative recovery, including neurocognitive function, morbidity, and quality of life. Volatile anesthetics have been associated with an increased risk of postoperative cognitive dysfunction (POCD) due to their effects on cerebral perfusion and neuroinflammation[36]. In contrast, propofol has neuroprotective properties that may reduce the incidence of POCD and improve overall recovery[37]. The comparative effects of these anesthetics on cognitive function and postoperative complications are summarized conceptually in Fig. 4. Furthermore, the type of anesthesia used can influence the risk of tumor recurrence or metastasis. Evidence suggests that intravenous anesthetics like propofol may inhibit cancer cell

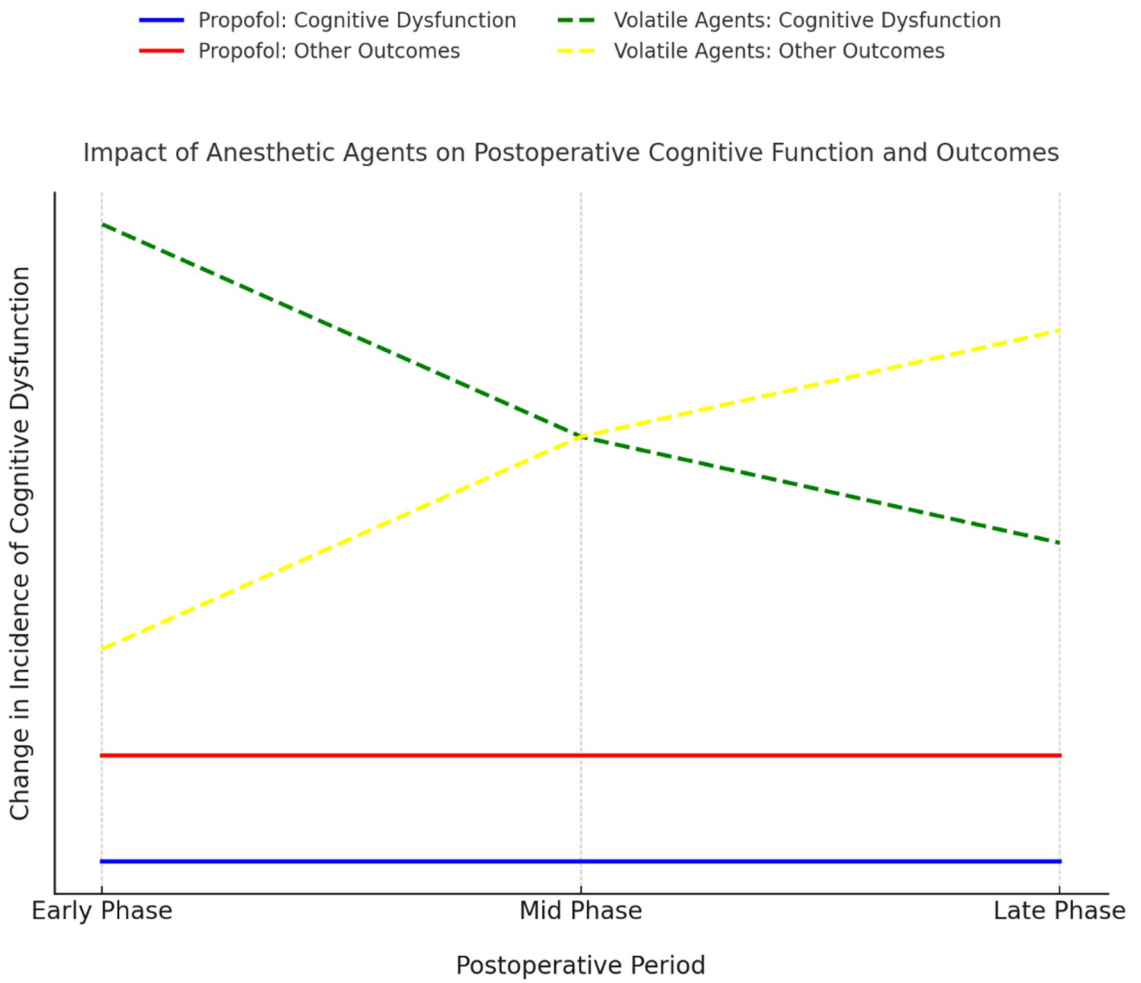

Figure 4. Impact of anesthetic agents on postoperative cognitive function and outcomes.[37]

proliferation and reduce metastatic potential by modulating immune responses and inflammatory pathways, whereas volatile anesthetics may promote these processes.[31]

Guidelines for future practice

To optimize surgical and oncological outcomes in brain tumor surgeries, best practices in anesthesia management should emphasize individualized patient care. This includes thorough preoperative assessments to identify patient-specific risks, choosing anesthetic agents that minimize adverse effects, and considering intraoperative challenges such as tumor location and surgical technique[37]. Integrating current research findings, such as the potential benefits of propofol in reducing tumor spread and preserving cognitive function, into clinical protocols can enhance patient outcomes. Developing standardized guidelines based on tumor type, patient condition, and expected surgical difficulties will support anesthesiologists and surgeons in making informed decisions that optimize both immediate and long-term results[33].

Current research gaps and future directions

Identify gaps in existing literature

Despite extensive research on the role of anesthesia in cancer surgeries, significant gaps remain in understanding the long-term effects of different anesthetic agents on tumor biology, particularly concerning tumor perfusion and infiltration. Most existing studies are limited to short-term outcomes or retrospective analyses, lacking comprehensive data from prospective, randomized controlled trials (RCTs) that can determine how various anesthetics influence long-term cancer recurrence and survival[31]. Moreover, while preclinical studies indicate that anesthetic agents may either promote or inhibit tumor growth and metastasis, robust clinical evidence to translate these findings into practice remains insufficient[38].

Future research needs

To address these gaps, future research should prioritize conducting large-scale, multicenter RCTs to evaluate the long-term oncologic outcomes of patients receiving different types of anesthetics during surgery. These studies should examine endpoints such as tumor recurrence rates, overall survival, and disease-free survival, while also controlling for confounding factors such as tumor type, stage, and patient comorbidities[31]. Furthermore, integrating clinical trials with molecular studies investigating the biological effects of anesthetics on cancer cells and the tumor microenvironment could provide a more comprehensive understanding of their impacts[39].

Innovative approaches

Emerging technologies and novel anesthetic strategies provide new insights into the management of brain tumor surgeries. For example, targeted anesthesia delivery methods, such as localized drug delivery systems or advanced monitoring of anesthetic depth, could enable more precise control over the effects of anesthesia on tumor physiology[38]. Additionally, integrating genomic and proteomic data into anesthetic management could help identify biomarkers that predict patient response to specific anesthetic agents[40], allowing for a more personalized approach to anesthesia during cancer surgeries. Leveraging these innovations in future research could significantly enhance the optimization of anesthetic protocols in brain tumor surgeries.[41]

Conclusion

This mini-review underscores the critical role of anesthesia in brain tumor surgeries, emphasizing its impact on both surgical success and long-term patient outcomes. Anesthetic agents such as propofol, isoflurane, and sevoflurane exert distinct effects on cerebral physiology, including CBF, ICP, and BBB permeability, which can significantly influence tumor perfusion, infiltration, and recurrence rates. While volatile anesthetics may promote tumor growth through mechanisms such as increased blood flow and pro-angiogenic signaling, intravenous agents like propofol could offer protective benefits by reducing inflammation and maintaining BBB integrity. Given the complexities of tumor biology and patient-specific variables, personalized anesthesia management is crucial. Future research should focus on RCTs to explore the long-term effects of anesthetic choices on tumor progression and patient survival. Additionally, emerging technologies, such as targeted anesthesia delivery and genomic-based approaches, could provide new insights into optimizing anesthesia protocols. By integrating current evidence with innovative strategies, clinicians can better tailor anesthesia management to enhance both immediate and long-term outcomes in brain tumor surgeries.

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Consent

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A.Dh., S.S.B., D.C.C., A.E.M., R.F.M.A., I.M., and A.D. contributed to the conceptualization, literature search, and drafting of the manuscript. A.Dh., S.S.B., and A.D. were involved in critical review and editing of the manuscript. A.D. served as the corresponding author, coordinated the manuscript development process, and ensured the final version met submission standards. All authors have read and approved the final version of the manuscript.

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