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Case Report

# Clinical Use of Thiopental in Perioperative Seizure in Subdural Hemorrhage and Space Occupying Lesion (SOL) Patient : A Case Report

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**ABSTRACT**

Subdural hematoma (SDH) is a blood clot between the dura mater and the arachnoid membrane surrounding the brain. Trauma is the leading cause of subdural hematoma. The perioperative management provided by the anesthetist for trauma brain injury is to prevent secondary injuries that occur and things that can be avoided, such as hypotension, hypoxemia, hyperglycemia, or hypercarbia. A 30-year-old man came with complaints of headaches after hitting a drainage canal. When the patient had a post-seizure, the patient was fully conscious. A CT-scan examination revealed subdural bleeding; the patient was diagnosed with subdural hematoma with epilepsy and a history of space-occupying lesions. The patient was planned to undergo a craniotomy. Management of TBI in the presence of a history of SOL is very complex and requires good perioperative management to prevent mortality and morbidity. This case report will explain the principles of neuroanesthesia used to prevent secondary brain injury with the facilities and infrastructure available at the hospital.

## Introduction

Acute subdural hematoma (SDH) is a frequently encountered neurosurgical emergency, most commonly resulting from traumatic brain injury, and often necessitates prompt surgical evacuation to mitigate the risk of irreversible neurological damage or mortality [1]. While the management of isolated SDH is well established, the presence of concurrent neurological comorbidities such as epilepsy or a history of space-occupying lesions (SOLs) significantly complicates the perioperative course. These patients pose unique anesthetic challenges due to the heightened risk of intraoperative seizures, elevated intracranial pressure (ICP), impaired cerebral autoregulation, and reduced cerebral perfusion pressure (CPP), all of which require a meticulous and individualized anesthetic strategy grounded in neuroprotective principles [2-3].

The clinical scenario becomes even more complex when SDH is superimposed on a previously treated SOL, particularly in patients with a functioning ventriculoperitoneal (VP) shunt and in whom breakthrough seizures occur despite prophylactic antiepileptic therapy. Such a constellation of pathologies, although rare, is clinically significant and poses a substantial challenge to anesthetic and surgical teams. Balancing ICP control, seizure suppression, maintenance of CPP, and adequate surgical access in this context demands a high level of expertise and interdisciplinary coordination. The consideration between general anesthesia techniques of total intravenous vs. balanced

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anesthesia and the interaction of anesthesia drugs with antiseizure drugs/anti-epilepsy drugs (AEDs) consumed by patients before surgery is of particular concern in perioperative case management. Maintaining homeostatic, cerebral, and systemic autoregulation; preventing secondary brain trauma; and providing optimal brain relaxation are the main goals to be able to produce good outcomes for patients [2,4-5].

In this report, we present a rare and intricate case involving SDH in a patient with a prior SOL and established VP shunt who developed intraoperative seizures. Through this case, we aim to emphasize the complex interplay between structural brain pathology, seizure disorders, and anesthetic neuroprotection. Furthermore, we highlight critical perioperative decision-making steps that may serve as a reference in similarly high-risk neurosurgical cases, where evidence-based guidance remains limited.

## Case Report

A 30-year-old man was found unconscious on the edge of a water canal. The patient was said to have had seizures shortly before falling into the water canal due to epilepsy he had suffered from since his teenage years. We did not get data on the type of anti-epilepsy drugs (AEDs) that he routinely consumed, but through a family statement it was known that the patient did not take the drug the day before the incident. A month earlier, the patient underwent VP shunt surgery due to generalized seizures, and from the results of the head CT at that time, it was found that there was a space-occupying lesion (SOL) located dominantly in the left ventricle, which was suspected to be a choroid plexus papilloma and caused obstructive hydrocephalus.

Upon arrival at the emergency room (ED), the patient was in a compos mentis state and complained of a general headache and pain in the laceration on the forehead. Physical and neurological examination within normal limits. A head non-contrast CT (NCHCT) showed an acute subdural hematoma (SDH) in the left frontotemporoparietal region, subarachnoid hemorrhage, and a solid lesion compressing the lateral ventricle, leading to bilateral dilation suggestive of hydrocephalus (Figure 1). The patient was diagnosed with subdural hematoma complicated by epilepsy and a space-occupying lesion undergoing craniotomy decompression evacuation SH. Patient presenting with an ASA 2, with a recommendation for fasting for six hours prior to surgery.

Prior to surgery, informed consent was obtained from the patient's family regarding the planned procedure. The patient was premedicated with phenytoin 100 mg orally, ranitidine 50 mg iv, and a loading dose of mannitol 200 cc, followed by a maintenance regimen of 120 cc every 8 hours. A 6-hour fasting period was observed before the procedure, and two units of packed red blood cells (PRBCs) were prepared for potential intraoperative transfusion. An intravenous (IV) line was established with 0.9% NaCl crystalloid solution, and postoperative admission backup to the intensive care unit (ICU) was planned for continuous monitoring. Standard preoperative anesthetic preparation included setting up the anesthesia workstation, airway management equipment, anesthetic agents, and emergency medications.

Upon arrival in the operating room, the patient's vital parameters were as follows: Consciousness level of Compos mentis (Glasgow Coma Scale: E4M6V5), blood pressure 120/80 mmHg, heart rate 80 beats per minute, respiratory rate 22 breaths per minute, oxygen saturation 98% on room air. General anesthesia was induced using the following agents: Thiopental 5 mg/kg, Fentanyl 1-2 mcg/kg, Lidocaine 1-1.5 mg/kg, and Rocuronium 0.5 mg/kg. Following induction, endotracheal intubation was performed using a 7.0-mm endotracheal tube secured at 21 cm at the lip.

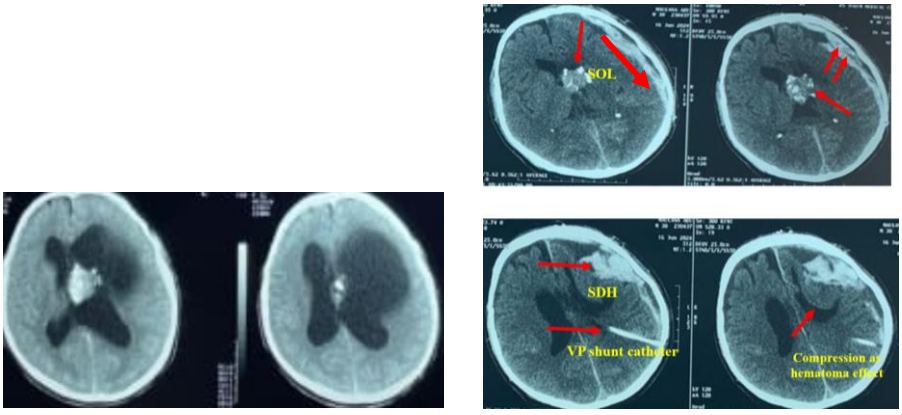


**Figure 1- Head CT scan. A. Before VP shunt. B. After VP shunt: showed mass tumor and more recovery from hydrocephalus. C: Subdural hemorrhage (SDH).**

Maintenance of anesthesia was achieved with oxygen, thiopental (1–3 mg/kg/hour), dexmedetomidine (0.3–0.5 mcg/kg/hour), and intermittent rocuronium administration.

The surgical procedure lasted 3 hours (Figure 2), during which the patient's hemodynamic parameters remained stable: Systolic blood pressure: 115–118 mmHg, diastolic blood pressure: 80–83 mmHg, end-tidal $CO_2$ ($EtCO_2$): 25–32 mmHg, Oxygen saturation: 99–100% Approximately midway through the procedure, the patient exhibited generalized intraoperative tremors involving both upper and lower extremities, suggestive of seizure activity, before the hematoma was fully evacuated. This was managed by deepening anesthesia by increasing the thiopental infusion to 4 mg/kg/hour and adding an extra rocuronium half-induction dose. Intraoperative fluid management included 0.9% NaCl: 1000 mL, Gelafusal: 500 mL, and packed red blood cells: 2 units. Fluid output comprised 100 mL of urine and an estimated 1400 mL of blood loss.

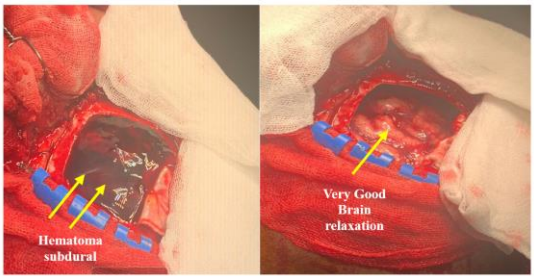


**Figure 2- Intraoperative brain condition, before hematoma evacuation and brain relaxation score very relaxed (BRS 1) after hematoma evacuation.**

Given the patient's neurological status and intraoperative course, extubation was deferred, and the patient was transferred to the ICU for postoperative monitoring and ventilatory support. Upon ICU admission, the patient remained intubated and sedated. Head elevation at 30° was maintained to optimize intracranial pressure management.

Postoperative pharmacologic management included Parecoxib 2x40 mg iv. for analgesia, Dexmedetomidine (0.2–0.7 mcg/kg/hour) for sedation, Tranexamic acid 500 mg every 8 hours, Nimodipine 60 mg every 6 hours orally, and Phenytoin 100 mg every 12 hours.

At 24 hours postoperatively, the patient remained hemodynamically stable, with arterial blood gas (ABG) values within normal limits. Ventilator weaning was initiated, followed by successful extubation.

On postoperative day 2, the patient experienced a generalized seizure lasting approximately 30 seconds, which was promptly terminated with intravenous diazepam 10 mg. To prevent further seizure recurrence, valproic acid 500 mg orally twice daily was introduced.

By postoperative day 3, the patient had regained full consciousness, remained hemodynamically stable, and had been seizure-free for 24 hours. Consequently, he was transferred to the general ward for further monitoring and continued recovery.

## Discussion

The perioperative management of patients with concurrent SDH, epilepsy, and intracranial mass lesions poses a unique challenge. The primary goals include ICP control, prevention of secondary brain injury, seizure control, and maintenance of cerebral perfusion pressure.

In this case, based on imaging, the intracranial mass was suspected to be a choroid plexus papilloma, a rare benign tumor known to arise in the ventricular system. Choroid plexus papillomas can cause excessive cerebrospinal fluid (CSF) production and obstructive hydrocephalus, both of which contribute to elevated intracranial pressure (ICP). The mass effect, hydrocephalus, and perilesional irritation can increase cortical excitability, thereby predisposing the patient to seizure activity [6-7]. The cause of the first seizure experienced in our case was due to increased ICP due to hydrocephalus and perilesional irritation that stimulated seizure activity in the cortex.

Seizures in epilepsy are caused by abnormal electrical activity in the brain, often due to a disruption in the normal communication between nerve cells. This disruption can be triggered by various factors, including genetics, brain injury, stroke, tumors, or infections. In some cases, the cause of seizures in epilepsy remains unknown [8-9]. Multiple mechanisms of pathophysiology of epilepsy-related brain tumors, such as mechanical compression, imbalance of vascularization and oxygen demand of the tumor, inflammatory processes, and neurotransmitter dysbalance, play the major roles in epileptogenesis in brain tumor patients. Peritumoral tissue is a part of the "epileptogenic zone," where the level of balance of neurotransmitters (GABA-ergic vs glutamatergic) at this zone leads to epileptogenicity of the tumor itself [10]. Anti-epileptic drugs (AEDs) effect in brain tumors should be able to control both the progression of the tumor and the frequency of seizures. Levetiracetam (LEV) and valproic acid (VA) are common drugs used in the control of epileptic seizures, both of which are monotherapy drugs in seizures due to tumors. Both work by not inducing the enzyme isocitrate-dehydrogenase (IDH 1/2), which is believed to play a role in the high incidence of seizures in tumors. A consensus states that non-enzyme drugs should be chosen in the treatment of seizures in brain tumors to avoid interference of AEDs with antineoplastic drugs and other drugs such as dexamethasone [10-11]. In our case, one of the considerations for choosing anesthesia techniques is to consider the effect of AEDs on anesthesia drugs as well as the effects of anesthesia itself on epilepsy or seizure activity. For example, the effect of LEV, which

reduces the duration of muscle paralysis (MR) and nociceptive thresholds, inhaled drugs (sevoflurane and $N_2O$), opioids (meperidine and morphine), and muscle relaxants (laudanosin and succinylcholine) can provoke the seizures [3,12-13]. Because seizure awakening is a primary trigger in increasing ICP, raising CMRO2, lowering CPP, and activating secondary injuries. Antiepileptic drugs are generally continued throughout the perioperative period unless mapping of seizure foci is anticipated. Antiepileptic drugs can induce hepatic enzymes and increase the metabolism of neuromuscular blockers, opioids, and dexmedetomidine, potentially necessitating higher doses [14-15]. Unfortunately in our current case, we failed to find information regarding the AEDs that patients consumed.

The selection of thiopental in facilitating general anesthesia in our case departs from the above considerations. Thiopental has been established for use in the therapy of seizures of epileptic status, to refractory seizures in critical care [16-17]. Thiopental, a short-acting barbiturate, has demonstrated considerable efficacy in the management of refractory seizures and elevated intracranial pressure (ICP), particularly in cases unresponsive to first-line therapies. By inducing burst suppression on electroencephalography (EEG), thiopental effectively reduces cerebral metabolic demand and cerebral blood flow, leading to significant reductions in ICP [18]. Comparative studies have shown that thiopental achieves better control of refractory intracranial hypertension than pentobarbital, with a success rate of 50% versus 28% [19-20]. In refractory status epilepticus, thiopental has been reported to terminate both clinical and electrographic seizures reliably, with favorable hemodynamic tolerance. Although thiopental may induce hypotension due to reduced mean arterial pressure, the consequent drop in cerebral perfusion pressure (CPP) generally remains within target thresholds and does not reach critical levels [21-22].

Beyond its role in seizure and ICP control, thiopental exhibits neuroprotective properties through multiple mechanisms. Experimental studies in reversible cerebral ischemia models have shown that intra-arterial administration of thiopental significantly reduces neuronal and glial apoptosis and improves neurological scores compared to control groups [23]. These protective effects are believed to be mediated by membrane stabilization, attenuation of oxidative stress, and preservation of cellular ion homeostasis [23-24]. In vitro studies further support thiopental's ability to delay hypoxia-induced depolarization and enhance post-hypoxic protein synthesis, contributing to neuronal survival [24-25]. Moreover, thiopental acts as a potent free radical scavenger, reducing hydroxyl radical generation and lipid peroxidation [26-28]. Clinical data also suggest its intraoperative use during aneurysm clipping is associated with a reduction in postoperative neurological complications, potentially due to decreased cerebral oxygen consumption and redistribution of cerebral blood flow to ischemic zones, a phenomenon known as "inverse steal" [29].

With the various advantages above, the choice of anesthesia falls for thiopental, considering that our case has more than one risk factor for experiencing perioperative seizures. The incidence of epilepsy in brain tumors (epilepsy-related brain tumor—ERBT) depends on the histology of the tumor, being highest in low-grade glioma (>80%), glioblastoma, and meningioma. Location of lesion on frontal, parietal, and temporal lobes is reported to be associated with a higher risk of seizures [10]. Meanwhile, the incidence of post-traumatic seizures of SDH is 5%-25%, with studies reporting an incidence of seizures in acute SDH of 24%-30%. The highest incidence of early post-traumatic seizures (ePTS) in aSDH is seen within 24 hours of injury [30]. A systematic study shows the risk factors for seizures in aSDH with various mechanisms that can underlie the occurrence of seizures, including inflammation, tissue damage or presence of scar tissue, occult cortical injury, or hematoma-related factors like mass effect or hemorrhage, hemoglobin degradation products, and changes in blood flow [31-32].

The incidence of intraoperative seizures is also not uncommon in craniotomy, even under general anesthesia. Although the incidence varies (0.5%-50%), a study showed that risk factors for intraoperative seizures with GA include a history of previous seizures, diagnosed brain tumors, and temporal craniotomy [33-34]. To date, there are no established guidelines for the optimal management of intraoperative seizures, but intravenous benzodiazepines, barbiturates, and anticonvulsant boluses have been used to treat intraoperative seizures in the past [35]. Rapid assessment and management of airway, breathing, and circulation should be prioritized, followed by the administration of anticonvulsant therapy. In this case, the patient underwent successful induction; however, intraoperatively, the patient experienced seizures. An increase in the dosage of thiopental was implemented to achieve burst suppression and terminate the ongoing seizures. We increased the thiopental dose we use to manage intraoperative seizures in this case. The important monitoring tool in the use of thiopental both in the maintenance of intraoperative anesthesia and induction of barbiturates in intensive care is electroencephalography (EEG) to monitor burst suppression. However, what is interesting is that there are case reports that indicate that intraoperative seizures persist even though burst suppression appears from EEG with propofol as an anesthesia maintenance agent [33, 36]. In another case, intraoperative seizures with maintenance using propofol were also reported [37]. The limitation of our case is that we do not have EEG as a

monitoring because of the limitations of the hospital where we work on this case.

The management of status epilepticus (Figure 3) begins with the stabilization phase (0-5 minutes). Benzodiazepines, specifically intravenous midazolam, lorazepam, or diazepam, are recommended as the initial therapeutic choice. The second phase of therapy should commence when the duration of seizures reaches 20 minutes. Agents utilized during this phase include intravenous fosphenytoin, valproic acid, levetiracetam, or phenobarbital. The third phase of therapy should be initiated when the seizure duration extends to 40 minutes. Suppose the second-line therapy fails to terminate the seizures. In that case, consideration should be given to either repeating the second-line treatment or employing anesthetic doses of thiopental, midazolam, pentobarbital, or propofol, with continuous EEG monitoring [17]. In our case, multiple factors strongly contribute to the occurrence of intraoperative seizures, such as the location of the hematoma and craniotomy (fronto-temporo-parietal), ERBT, and craniotomy, which are the risk factors for recurrent seizures. Therefore, the prophylactic use of AEDs in our case is highly recommended where we use phenytoin injection and mannitol administration. Mannitol (hyperosmolar therapy) is one of the non-surgical techniques to reduce ICP in first-tier therapy strategy, where the objective result of intraoperative ICP reduction in the form of a brain relaxation score (BRS) can be directly evaluated; the recommended dose in a systematic study of Bayesian analysis is 5 ml/kg [5]. Another ICP-lowering technique used in this case is the installation of a VP shunt, where a VP shunt is one of the surgical techniques that is generally used to lower ICP in emergency conditions. Especially in brain tumors, VP shunts are generally installed in the early stages before the tumor is evacuated where hydrocephalus occurs, a sign of intracranial hypertension, even as a non-invasive ICP monitor [38-40]. The management of acute subdural hematoma (SDH) (Figure 4) is guided by the disease progression, radiological findings, and the patient's prognosis. Despite advancements in diagnostic and therapeutic options over the years, traumatic acute subdural hematoma in adults remains a life-threatening condition generally associated with underlying injuries to the brain's structure. In addition to managing hematoma evacuation when indicated, medical management of cerebral edema and intracranial pressure is equally important for maximizing patient outcomes following severe injury [41]. The selection of neuromuscular blocking agents for emergency intubation in patients undergoing neurosurgical procedures remains a contentious issue.

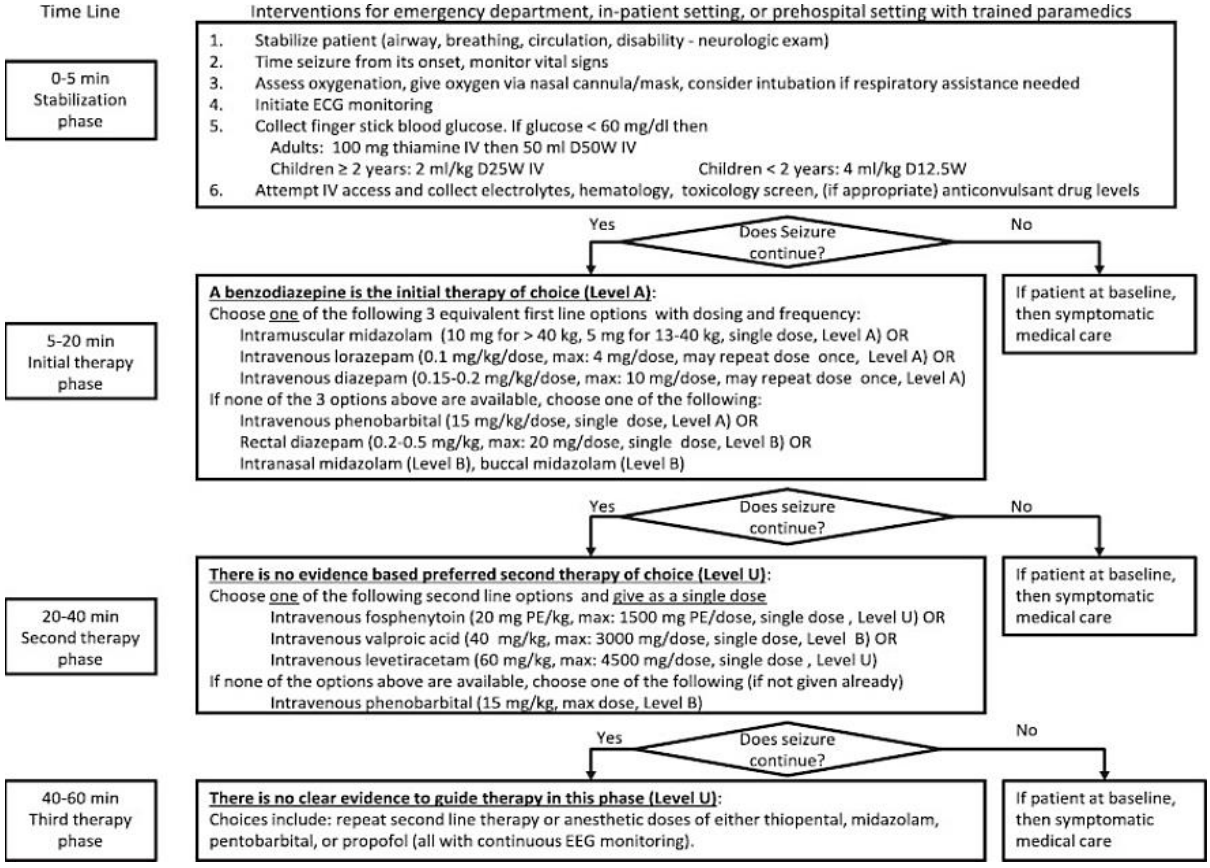


**Figure 3- Epilepsy algorithm. Adapted from Gauser et al.**

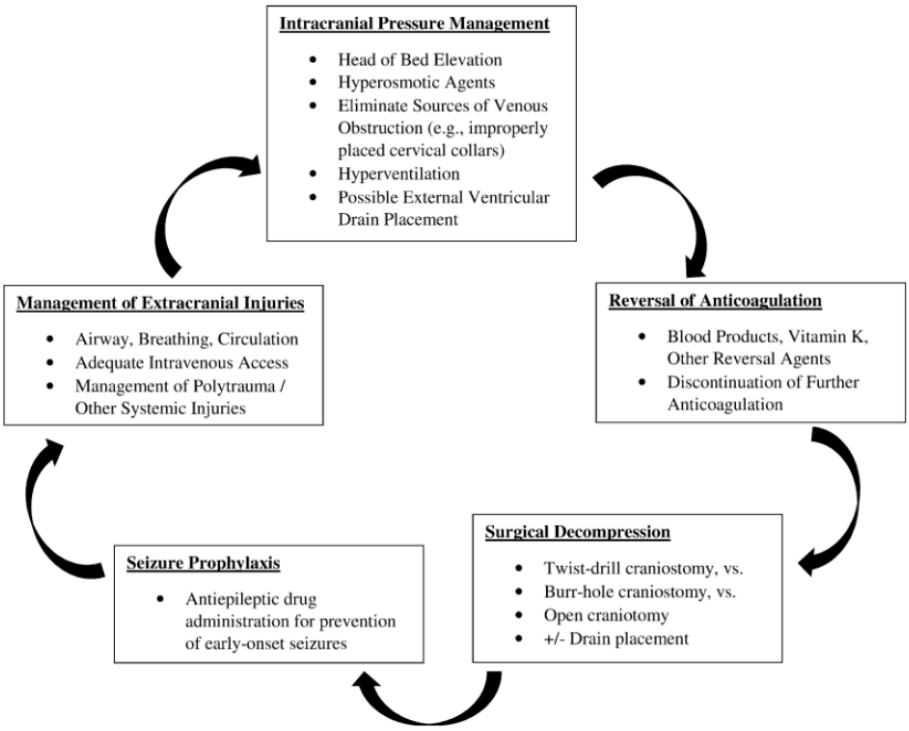


**Figure 4- Subdural Hematoma Management. Adapted from Karibbe et al.**

Succinylcholine can elevate intracranial pressure (ICP), although the clinical significance of this effect is not definitively established. However, succinylcholine's rapid onset and elimination minimize the duration of potential ICP elevation. Pretreatment with a small dose of a non-depolarizing neuromuscular blocker may mitigate fasciculations, reducing the likelihood of increased ICP. Ventilation should be carefully managed to ensure adequate oxygenation ($PaO_2$ > 60 mmHg) and normocarbia ($PaCO_2$ 35-45 mmHg). Hyperventilation should be reserved for short-term ICP control, as excessive and prolonged hyperventilation can induce cerebral vasoconstriction, potentially leading to cerebral ischemia [42]. In this case, fentanyl 200 mcg, thiopental 200 mg, lidocaine 60 mg, and rocuronium 30 mg were used as induction agents, followed by intubation. Anesthetic maintenance during craniotomy should aim to (1) ensure patient unconsciousness, (2) promote brain relaxation to enhance surgical exposure and reduce retractor-induced injury, and (3) provide neuroprotection to minimize the risk of secondary systemic injury [42].

Postoperative patients may be admitted to a general ward or an intensive care unit (ICU). The decision regarding ICU admission generally depends on the preoperative Glasgow Coma Scale (GCS) score, the extent and location of the surgery, and the presence of midline shift [43]. Management of head trauma in the ICU is aimed at preventing secondary injuries, maintaining optimal cerebral perfusion pressure by reducing intracranial pressure (ICP), monitoring neurological damage, and providing supportive care [44].

Options for managing increased ICP include:

- Adequate Anesthesia and Analgesia: Inadequate sedation and analgesia can elevate ICP due to increased cerebral blood flow (CBF) associated with heightened metabolic activity in the brain [45].
- Positioning: Careful positioning is crucial, including avoiding obstruction of cerebral venous drainage by preventing excessive neck flexion and/or rotation [45].
- Ventilation Control: Prudent use of hyperventilation while maintaining hemodynamic stability is important; excessive and prolonged hyperventilation can exacerbate cerebral ischemia and should be avoided [45].
- Hyperosmolar Therapy: Both mannitol and hypertonic saline are effective treatments for intracranial hypertension resulting from traumatic brain injury (TBI). Mannitol (0.25-1 g/kg) reduces ICP through reflex vasoconstriction due to decreased viscosity, which leads to increased microvascular perfusion and osmotic diuresis [45].
- Cerebrospinal Fluid Drainage: An external ventricular drain (EVD) allows for the monitoring and management of ICP through cerebrospinal fluid drainage [45].

## Conclusion

Traumatic brain injury (TBI) is associated with poor outcomes, including high mortality and disability rates. Current management strategies for TBI focus on preventing primary injuries and avoiding secondary damage. Careful and structured perioperative preparation is crucial, encompassing preoperative patient assessment, medication and equipment readiness, anesthetic planning, and postoperative care. The perioperative goals include facilitating early brain decompression, providing balanced anesthesia for surgery, maintaining adequate cerebral perfusion while optimizing intracranial hemodynamics, and aggressively preventing secondary injuries such as hypoxemia and abnormal carbon dioxide levels. Intraoperative seizure management presents a challenge in neurosurgery; thus, the selection of appropriate anesthetic agents is vital for achieving favorable patient outcomes. By meticulously considering perioperative factors, healthcare providers can optimize the management of patients with head injuries and enhance outcomes following surgical interventions. Collaborative efforts among multidisciplinary teams, including neurosurgeons, anesthesiologists, intensivists, and nursing staff, are essential for delivering comprehensive care tailored to each patient's needs.

### Data Availability

Data availability is available upon request to the author.

### Consent

The author has received consent from the patient to be included in this case report.

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