e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

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

Neuroprotective Anesthetic Management Using Thiopental in a 17-Year-Old with Multifocal Epidural Hematoma and Impending Brain Herniation: A Case Report

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ARTICLE INFO

Keywords:
Decompressive craniotomy
Epidural hematoma
Neuroprotection
Thiopental
Traumatic brain injury

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

https://doi.org/10.37275/jacr.v7i1.862

A B S T R A C T

Introduction: Epidural hematoma resulting from severe traumatic brain injury demands immediate neuroanesthetic intervention. Multifocal lesions accompanied by pneumocephalus and impending brain herniation present profound perioperative challenges requiring targeted cerebral perfusion management. **Case presentation:** A 17-year-old male weighing 50 kg sustained severe polytrauma, presenting with a Glasgow Coma Scale of 12 and active auditory canal bleeding. Imaging revealed multifocal epidural hematomas in the right frontotemporal (66 cc) and right parietal (43 cc) regions, alongside pneumocephalus, a 1.5 cm subfalcine herniation, and downward transtentorial herniation. The patient, classified as ASA physical status 4E, required an emergent decompressive craniotomy and concurrent facial reconstruction. A neuroprotective anesthetic strategy was deployed utilizing thiopental, fentanyl, and atracurium to minimize the cerebral metabolic rate and control intracranial pressure. Anesthesia was maintained with sevoflurane. Hemodynamics were strictly titrated to ensure optimal cerebral perfusion pressure. Following successful surgical hematoma evacuation, the patient was admitted to the intensive care unit and demonstrated an excellent neurological recovery after a five-day admission. **Conclusion:** Thiopental serves as a highly effective neuroprotective induction agent for severe traumatic brain injury with intracranial hypertension. Meticulous hemodynamic control and targeted reduction of cerebral metabolism are critical in preventing secondary ischemic cascades and improving functional outcomes in polytrauma patients.

1. Introduction

Traumatic brain injury represents a profound and devastating public health challenge, persistently remaining a leading cause of global morbidity and mortality. This severe epidemiological burden is particularly pronounced among the adolescent and young adult populations, demographics that are disproportionately subjected to high-velocity mechanical forces from motor vehicle collisions, catastrophic falls, and severe sports-related injuries.[1] The adolescent brain presents unique physiological vulnerabilities alongside distinct resilience factors. While possessing a higher degree of neuroplasticity compared to the adult or geriatric population, the developing brain remains highly susceptible to the devastating effects of hypoxic-ischemic events and massive pathological shifts in intracranial pressure. The overarching socioeconomic impact of such injuries

is immense, encompassing not only acute, intensive medical costs but also prolonged cognitive rehabilitation, lifelong physical disability, and the permanent loss of societal productivity. The pathophysiology of traumatic brain injury is classically divided into primary and secondary injury phases. The primary injury occurs at the exact moment of impact, involving direct mechanical damage to the neural parenchyma, irreversible axonal shearing, and immediate microvascular disruption. Secondary injury develops insidiously over the subsequent hours and days, driven by a complex, compounding cascade of cellular ischemia, severe neuroinflammation, biochemical excitotoxicity, and profound cerebral edema.[2]

Within the incredibly diverse spectrum of acute craniocerebral trauma, the epidural hematoma emerges as a critical, life-threatening subtype of traumatic brain injury. This specific intracranial pathology is characterized by the acute, high-pressure accumulation of blood within the potential anatomical space strictly located between the inner table of the skull calvaria and the periosteal layer of the dura mater.[3] The underlying etiology of an epidural hematoma is predominantly arterial in nature. It most frequently results from high-impact, direct-force trauma to the temporal or parietal bones, yielding linear or depressed skull fractures that cause a traumatic laceration, shearing, or frank avulsion of the middle meningeal artery. This artery, coursing superficially along the inner aspect of the temporal bone, is highly vulnerable to shearing forces when the structurally fragile squamous portion of the temporal bone fractures. Upon vascular rupture, arterial blood extravasates under high systemic pressure, aggressively and forcefully stripping the tightly adherent dura mater away from the internal calvarial surface. Although epidural hematomas account for a relatively small percentage of all traumatic brain injury cases, their presence is strongly correlated with exceptionally high morbidity and mortality rates if they are not rapidly diagnosed and aggressively managed.[4] Unlike subdural hemorrhages, which may evolve slowly via venous oozing, the high-pressure arterial bleeding associated with an epidural hematoma drives rapid volumetric expansion, demanding immediate clinical recognition and intervention.

The classic clinical trajectory of an isolated epidural hematoma often involves a highly distinct, biphasic neurological presentation. Following the initial traumatic head injury, patients may experience a transient loss of consciousness directly attributable to the primary concussive force, which is then followed by a spontaneous neurological recovery and a period of apparent clinical normalcy. This transient return of consciousness is widely recognized in clinical neurology as the lucid interval. This temporary neurological baseline can be exceedingly clinically deceptive. During this specific interval, the patient may converse appropriately, ambulate independently, and appear entirely stable, all while the arterial hemorrhage continues to expand silently and aggressively within the fixed confines of the cranial vault. This deceptive clinical presentation potentially leads to critical, catastrophic delays in definitive radiological diagnosis and necessary surgical intervention if the evaluating healthcare provider is not highly vigilant and thoroughly suspicious of the underlying mechanism of injury.[5]

The rigid anatomy of the human skull dictates that its total internal volume is strictly constant, encompassing three distinct compartments: the brain parenchyma, the cerebrospinal fluid, and the total intracranial blood volume.[6] As the epidural hematoma rapidly expands, it progressively and systematically exhausts the intrinsic physiological compensatory mechanisms of the brain. These initial compensatory mechanisms involve the rapid displacement of cerebrospinal fluid into the spinal subarachnoid space and the forced expulsion of venous blood from the cerebral dural sinuses. Once this spatial compliance is completely depleted, the mathematical relationship between intracranial volume and intracranial pressure shifts from a linear progression to a steep, exponential curve. At this critical juncture, a virtually imperceptible addition of hematoma volume causes a catastrophic, life-threatening spike in intracranial pressure.

Without prompt neurosurgical decompression, this drastically increased intracranial pressure creates a severe internal pressure gradient. This pressure

gradient forces the physical displacement of functional brain tissue across rigid dural structures, directly precipitating fatal brain herniation syndromes. These syndromes, including subfalcine herniation where the cingulate gyrus is pushed laterally under the falx cerebri, and downward transtentorial herniation where the medial temporal lobe is forced forcefully through the tentorial notch, directly compress and compromise vital brainstem structures.[7] This severe mechanical brainstem compression leads inexorably to an irreversible cessation of respiratory drive, catastrophic autonomic dysregulation manifesting as the Cushing reflex, and ultimately, absolute brain death. Consequently, emergent surgical evacuation via a decompressive craniotomy remains the absolute, undisputed gold standard treatment for a rapidly expanding epidural hematoma, allowing for complete hematoma removal, direct visualization and cauterization of the bleeding arterial source, and the immediate, life-saving relief of intracranial hypertension.

Anesthetic management for an urgent decompressive craniotomy in the setting of severe traumatic brain injury requires highly specialized, meticulously targeted neuroanesthetic strategies. The neuroanesthesiologist assumes a uniquely critical role in determining the ultimate functional and neurological outcome of the patient. The practitioner must master a profoundly delicate physiological balance throughout the entire perioperative period. The primary clinical objective centers strictly on maintaining robust cerebral perfusion pressure. Cerebral perfusion pressure is fundamentally defined as the mathematical difference between the systemic mean arterial pressure and the intracranial pressure. In the setting of severe traumatic brain injury, intrinsic cerebral autoregulation is frequently abolished, rendering cerebral blood flow entirely and passively dependent on the systemic mean arterial pressure.[8] Consequently, any episode of systemic hypotension immediately translates to irreversible cerebral ischemia, while uncontrolled hypertension can exacerbate vasogenic edema and accelerate the expansion of the intracranial hemorrhage. Thus, optimizing systemic oxygenation, strictly maintaining normocapnia to regulate cerebral vascular resistance, and deploying targeted pharmacological agents that actively lower intracranial pressure while mitigating secondary ischemic brain injury are absolute imperatives.

Furthermore, the concurrent presence of multifocal epidural hematomas alongside complex facial fractures presents fiercely competing surgical and airway management priorities, drastically elevating the inherent complexity of the perioperative care required. Severe maxillofacial trauma significantly complicates the process of securing a definitive airway, necessitating highly advanced intubation techniques that meticulously avoid exacerbating potential cervical spine injuries or triggering dangerous sympathetic surges in intracranial pressure. Additionally, the radiological presence of intracranial air, diagnosed as pneumocephalus, introduces absolute contraindications for specific inhalational anesthetic gases, mandating a highly customized, rigorous approach to the induction and maintenance of general anesthesia.[9]

Central to this highly customized neuroanesthetic approach is the strategic utilization of specific induction agents. Barbiturates, prominently thiopental, have long been recognized for their extraordinarily potent neuroprotective capabilities in the face of acute intracranial catastrophes. Thiopental induces a profound, dose-dependent reduction in the cerebral metabolic rate for oxygen. Because cerebral blood flow is intricately coupled to cerebral metabolism, this profound metabolic suppression induces a highly beneficial secondary cerebral vasoconstriction. This targeted vasoconstriction effectively decreases the total intracranial blood volume, leading to an immediate and dramatic reduction in overall intracranial pressure. Moreover, thiopental provides critical cellular-level protection by actively attenuating glutamate-mediated excitotoxicity and preventing free radical lipid peroxidation, blocking the biochemical mechanisms that are entirely central to the devastating cellular cascade of secondary brain injury.[10]

The aim of this study is to delineate the highly targeted, neuroprotective anesthetic management of a high-risk pediatric polytrauma patient presenting with catastrophic intracranial pathology. The novelty of this

case report lies in the successful, simultaneous management of severe multifocal epidural hematoma, pneumocephalus, and impending transtentorial herniation utilizing a specifically titrated thiopental-based anesthetic regimen to facilitate both emergent neurosurgical decompression and extensive plastic reconstructive surgery.

2. Case Presentation

Written informed consent was explicitly obtained from the legal guardians of the pediatric patient prior to the drafting and submission of this manuscript, in strict accordance with the ethical principles outlined in the Declaration of Helsinki. The patient's family received a comprehensive explanation regarding the academic and scientific objectives of this case report. They provided voluntary, documented consent for the publication of all associated clinical data, operative details, and anonymized radiological and intraoperative photographs. To ensure strict patient confidentiality and privacy, all personally identifiable information has been meticulously redacted or obfuscated from both the textual narrative and the supplementary media. The guardians were fully informed that the publication of this material is intended exclusively for educational and scientific dissemination within the global medical community, and that no financial compensation would be provided. Furthermore, the family formally acknowledged that while the information will be permanently distributed within the public domain and peer-reviewed medical literature, the absolute anonymity of the patient is guaranteed to the highest possible extent permitted by law.

Patient history and initial assessment

A 17-year-old male was admitted to the hospital emergency department following a severe traumatic accident that occurred during the morning hours in the Bandulan area. The precise chronology and exact kinematic mechanism of the traumatic event were unknown at the time the patient arrived at the emergency installation. A relative transported the patient to the hospital, noting that his last oral intake was dinner the previous evening. Upon arrival, the patient exhibited a marked decrease in his level of consciousness and was actively bleeding from both auditory canals. Due to his significantly altered mental status, the patient was entirely unable to recall any details surrounding the traumatic incident. His past medical history was unremarkable, with no established history of diabetes mellitus, hypertension, or cardiovascular disease. Anthropometric measurements recorded a body weight of 50 kg and a total height of 160 cm (Table 1a).

Physical and neurological examination

The initial primary survey and physical examination revealed critical clinical findings. The patient's airway was definitively patent without any signs of mechanical obstruction, and he was breathing spontaneously. The respiratory rate was recorded at 28 breaths per minute, maintaining a peripheral oxygen saturation of 98% on ambient room air. Pulmonary auscultation demonstrated clear, bilateral vesicular breath sounds without any abnormal adventitious findings. Hemodynamically, the patient exhibited a blood pressure of 129/91 mmHg with a strong, easily palpable radial pulse. The heart rate was stable at 74 beats per minute, featuring a regular rhythm without detectable murmurs or gallops.

A detailed neurological assessment yielded a Glasgow Coma Scale score of 12 (specifically E3, V4, M5), indicating a state of mild confusion while the patient remained conscious. Pupillary examination showed isocoric pupils measuring exactly 3 mm bilaterally, briskly reactive to direct light, without any overt signs of lateralization or focal neurological deficits at the time of triage. Abdominal examination revealed no distension or ascites, accompanied by normal bowel sounds. There were no signs of peripheral cyanosis or edema. However, the patient sustained significant maxillofacial trauma, prominently displaying severe facial swelling, widespread abrasions, and a deep laceration located over the nasal region and philtrum.

Table 1a. Summary of Clinical Findings of Patient on Admission (Part 1)

Clinical Parameter	Detailed Finding
1. GENERAL PATIENT CHARACTERISTICS	
Demographics	17-year-old male.
Anthropometrics	Body weight: 50 kg. Height: 160 cm.
Preoperative Status	Last oral intake was dinner on the previous evening. No history of diabetes mellitus, hypertension, or heart disease.
2. VITAL SIGNS & HEMODYNAMICS	
Airway & Respiration	Patent airway with spontaneous breathing. Respiratory rate: 28 breaths/min. SpO2: 98% on room air. Clear vesicular breath sounds.
Cardiovascular	Blood pressure: 129/91 mmHg. Heart rate: 74 beats/min with regular rhythm. Strong palpable radial pulse. No murmurs or gallops.
3. NEUROLOGICAL ASSESSMENT	
Consciousness Level	**Decreased level of consciousness**. Glasgow Coma Scale (GCS) score: 12 (E3 V4 M5), indicating mild confusion but conscious.
Pupillary Reflex	Isocoric pupils (3 mm) bilaterally. Briskly reactive to light.
Motor & Deficits	No laterality signs or focal neurological deficits at triage.

Diagnostic investigations

Initial laboratory investigations conducted on May 28th, 2025, demonstrated a marked leukocytosis of 15.9 x 10^3/µL, indicating a robust systemic inflammatory response secondary to severe trauma, while the platelet count remained within normal limits at 283,000/µL. Renal function parameters, including ureum and creatinine, as well as hepatic enzyme levels, were strictly within normal physiological limits. The random blood glucose concentration was 120 mg/dL, definitively confirming the absence of stress-induced hyperglycemia. The coagulation profile revealed a slightly prolonged prothrombin time of 10.7/11.6 seconds and an activated partial thromboplastin time of 28.9/26.3 seconds. A portable chest radiograph performed concurrently showed no abnormalities in the cardiopulmonary silhouette and confirmed the complete absence of any thoracic skeletal fractures.

An urgent non-contrast computed tomography scan of the head and maxillofacial structures revealed critical, life-threatening intracranial pathology. The imaging explicitly identified massive multifocal epidural hematomas: a large 66 cc hematoma located in the right frontotemporal region and a 43 cc hematoma situated in the right parietal region. Furthermore, the scan demonstrated significant pneumocephalus. The profound mass effect generated by the multifocal hematomas and subsequent cerebral edema resulted in a severe 1.5 cm leftward subfalcine herniation and distinct early signs of downward transtentorial herniation at the anatomical level of the pons (Table 1b). Three-dimensional bone window reconstructions confirmed extensive craniofacial fractures. These included severe fractures of the right frontal bone extending to the anterior and posterior walls of the right frontal sinus, the medial and lateral walls of the right orbit, the right parietal bone, the right temporal mastoid bone, and the right occipital bone. Additional maxillofacial fractures deeply involved the maxillary sinus, sphenoid sinus, right frontozygomatic suture, and sphenozygomatic structures.

Table 1b. Summary of Clinical Findings of Patient on Admission (Part 2)

Clinical Parameter	Detailed Finding
4. PHYSICAL TRAUMA & ASSOCIATED INJURIES	
Head & ENT	**Active bleeding from both ears**.
Maxillofacial	Severe facial swelling and abrasions. Deep lacerations over the nasal region and philtrum.
Abdomen & Extremities	No distension or ascites; normal bowel sounds. No peripheral cyanosis or edema.
5. LABORATORY INVESTIGATIONS	
Hematology	Leukocytosis: 15.9 x 10^3/μL. Platelets: 283,000/μL.
Coagulation Profile	Prothrombin Time (PT): 10.7/11.6 seconds. Activated Partial Thromboplastin Time (APTT): 28.9/26.3 seconds.
Biochemistry	Renal (ureum/creatinine) and hepatic function within normal limits. Random blood glucose: 120 mg/dL.
6. RADIOLOGICAL FINDINGS (CT & X-RAY)	
Intracranial Hemorrhage	**Multifocal Epidural Hematoma (EDH)**: Right frontotemporal region (66 cc) and right parietal region (43 cc).
Mass Effect & Herniation	Severe cerebral edema with **1.5 cm leftward subfalcine herniation**. Early downward transtentorial herniation at the pons level. Pneumocephalus present.
Skeletal Fractures	Fractures of right frontal bone, orbit walls, right parietal, right temporal mastoid, and right occipital bone. Maxillofacial fractures involving maxillary and sphenoid sinuses, and frontozygomatic areas.
Thorax	Chest X-ray showed no cardiopulmonary abnormalities or thoracic fractures.

Anesthetic management and surgical intervention

Given the extreme severity of the traumatic brain injury, the presence of multiple cranial fractures, and the life-threatening signs of impending brain herniation, the patient was officially classified as American Society of Anesthesiologists physical status 4E. An emergent, multidisciplinary surgical intervention plan was formulated. This involved the neurosurgical team performing a decompressive craniotomy to immediately evacuate the epidural hematomas and lower the intracranial pressure, while the plastic surgery team would concurrently perform extensive facial wound debridement and a complex rotation flap reconstruction. Comprehensive informed consent was obtained directly from the patient's family following a detailed explanation of the profound operative risks and potential benefits.

Preoperative preparation: The patient was fasted according to standard preoperative protocols based on the timeline of his last meal. Intravenous access was rapidly secured using 18G and 20G peripheral catheters to facilitate the immediate administration of resuscitation fluids and pharmacological agents. Intravenous hydration was initiated with a 0.9% sodium chloride solution at a continuous rate of 100 cc per hour to rigorously maintain euvolemia. Two units of packed red cells and one unit of whole blood were cross-matched and prepared in the operating theater to anticipate any significant intraoperative hemorrhagic events.

Induction and airway management: A targeted neuroprotective general anesthetic strategy was initiated, centering on endotracheal intubation and the active prevention of secondary neurological injury. Following pre-oxygenation, intravenous fentanyl was administered at a dose of 2 mcg/kg to profoundly blunt the sympathetic nervous system response to direct laryngoscopy, thereby preventing dangerous secondary spikes in intracranial pressure. Anesthetic induction was carefully achieved using thiopental at a dose of 4 mg/kg. Thiopental was specifically selected for its well-documented neuroprotective properties, its potent ability to induce cerebral vasoconstriction, and its robust suppression of the cerebral metabolic rate. Neuromuscular blockade was secured with atracurium at a dose of 0.5 mg/kg. Despite the presence of severe facial trauma, direct laryngoscopy was executed smoothly, and the trachea was successfully intubated.

Maintenance and intraoperative hemodynamics: General anesthesia was maintained utilizing sevoflurane at 0.8 to 1.0 minimum alveolar concentration in a highly controlled oxygen and air mixture. Nitrous oxide was strictly avoided due to the confirmed presence of pneumocephalus on the preoperative imaging. Mechanical ventilation was precisely managed using a volume-controlled mode with a tidal volume of 350 mL and a respiratory rate of 16 breaths per minute. This ventilatory strategy was designed to maintain the end-tidal carbon dioxide strictly between 30 and 35 mmHg, inducing mild cerebral vasoconstriction to assist in intracranial pressure control.

Continuous, beat-to-beat hemodynamic monitoring was facilitated via a radial arterial line placed immediately post-induction. The mean arterial pressure was tightly controlled and maintained between 85 and 95 mmHg throughout the surgical duration to ensure that the cerebral perfusion pressure remained consistently adequate, fully compensating for the severely elevated intracranial pressure prior to surgical decompression.

Figure 1 delineates the continuous intraoperative hemodynamic and respiratory trends recorded during the emergent decompressive craniotomy and concurrent maxillofacial reconstruction. The graphical timeline illustrates the stringent physiological control achieved through a targeted neuroprotective anesthetic strategy. Following the establishment of baseline awake parameters, the administration of fentanyl effectively blunted the sympathetic nervous system response, preventing any deleterious tachycardic or hypertensive spikes during direct laryngoscopy and endotracheal intubation. Subsequently, anesthetic induction utilizing thiopental produced a smooth, controlled reduction in the mean arterial pressure (MAP). Throughout the surgical duration, including the highly critical phases of dural opening and hematoma evacuation, the MAP was meticulously maintained within a narrow therapeutic window of 85 to 95 mmHg. This precise hemodynamic management ensured optimal cerebral perfusion pressure against the backdrop of severe intracranial hypertension. Concurrently, mechanical ventilation was strictly titrated to maintain end-tidal carbon dioxide ($ETCO_2$) concentrations between 30 and 35 mmHg, purposefully inducing mild cerebral vasoconstriction to further alleviate intracranial pressure prior to surgical decompression. Systemic oxygenation (SpO_2) remained consistently optimal at 100%. Ultimately, these highly coordinated multiparametric trends reflect a successful, pathophysiology-driven anesthetic approach that actively prevented secondary ischemic injury and maintained profound intraoperative stability in a high-risk polytrauma patient.

Surgical course: The neurosurgical team successfully executed a right-sided decompressive craniotomy. Upon exposing the dura mater, the massive epidural hematomas were entirely evacuated, immediately alleviating the localized mass effect and visibly reducing the tension on the underlying brain parenchyma. Concurrently, the plastic surgery team performed meticulous debridement of the contaminated and necrotic facial tissues, successfully closing the complex nasal and philtrum lacerations utilizing a rotation flap. The estimated intraoperative blood loss was approximately 600 mL. The patient received 1200 mL of crystalloid fluid resuscitation and remained highly hemodynamically stable without the need for the preemptively prepared blood products.

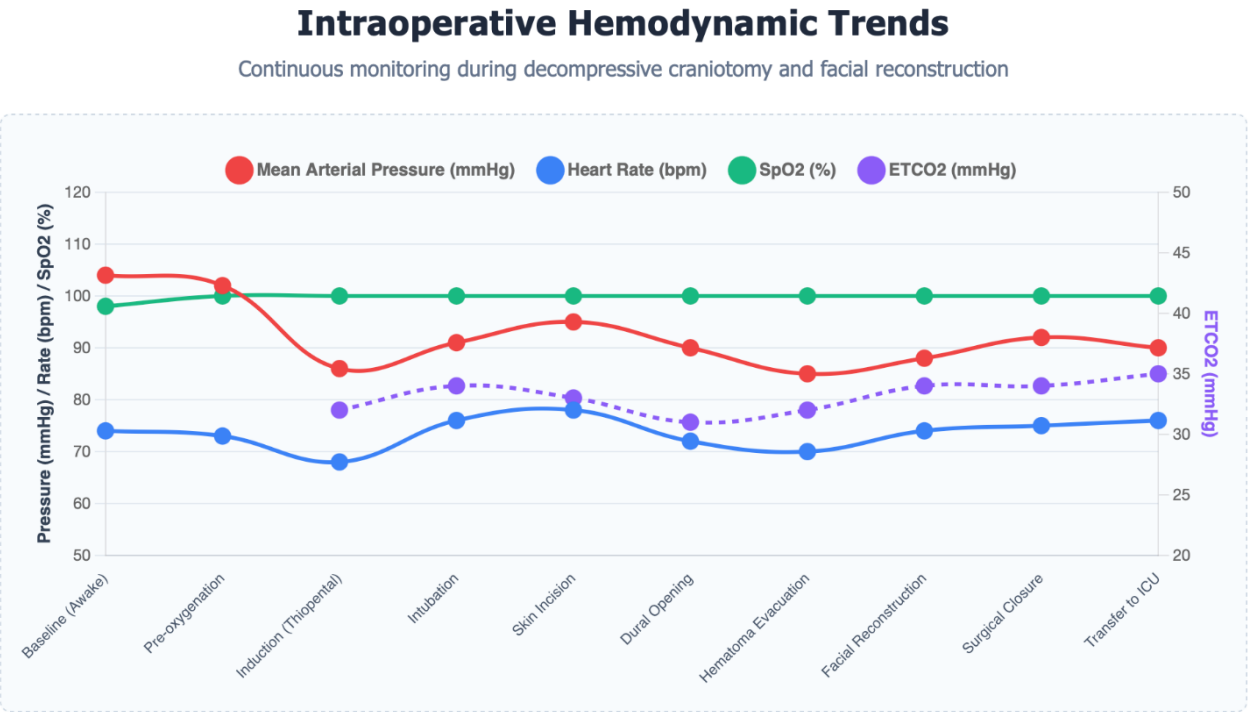

Figure 1. Intraoperative hemodynamic trends.

Postoperative care

Following the definitive evacuation of the hematomas and the completion of the facial reconstruction, the patient was transferred to the intensive care unit. Postoperative intensive care management focused on the strict, continuous monitoring of intracranial pressure, systemic oxygenation, and overall hemodynamic stability to ensure that any potential delayed complications could be immediately addressed. After receiving dedicated intensive care therapy for a duration of 5 days, the patient's clinical condition improved remarkably, and he demonstrated an excellent recovery trajectory completely devoid of any residual neurological deficits (Table 2).

3. Discussion

The perioperative anesthetic management of a multifocal epidural hematoma in a pediatric polytrauma patient presents an incredibly intricate matrix of physiological challenges. This clinical case highlights the paramount importance of thoroughly understanding cerebrovascular pathophysiology and tailoring anesthetic pharmacology to actively mitigate secondary brain injury.[11]

Epidural hematomas typically arise from direct mechanical trauma to the middle meningeal artery, precipitating the rapid, high-pressure accumulation of arterial blood within the restrictive epidural space. The fundamental principles governing intracranial dynamics are best described by the Monro-Kellie hypothesis, which dictates that the rigid cranial vault encompasses a fixed total volume containing brain parenchyma, cerebrospinal fluid, and intracranial blood.[12] As an acute epidural hematoma expands rapidly, the initial physiological compensatory mechanisms—specifically the displacement of cerebrospinal fluid into the spinal subarachnoid space and the expulsion of venous blood from the cerebral sinuses—are quickly and completely exhausted. In the presented patient, the massive combined volume of the multifocal hematomas (66 cc and 43 cc) caused an absolute and catastrophic failure of these intracranial compensatory reserves (Figure 2).

Table 2. Diagnosis, Treatment, Follow up and Outcome	
Phase	**Clinical Details & Interventions**
1. DIAGNOSIS	
Primary Neurological	• Multiple epidural hematomas situated in the right frontotemporal and right parietal regions. • Intracranial air accumulation indicating pneumocephalus. • Radiological evidence of subfalcine and downward transtentorial brain herniation.
Skeletal & Facial	• Extensive cranial fractures involving the right temporal, frontozygomatic, and sphenozygomatic structures. • Complex lacerations over the philtrum and nasal area, accompanied by severe facial abrasions.
Surgical Risk Classification	Designated as ASA physical status 4E secondary to massive polytrauma and critical intracranial hypertension.
2. TREATMENT & MANAGEMENT	
Preoperative Preparation	• Peripheral intravenous lines established using 18-gauge and 20-gauge cannulas. • Fluid resuscitation commenced with normal saline (0.9% NaCl) at a rate of 100 cc per hour. • Cross-matching completed for two units of packed red blood cells and one unit of whole blood.
Anesthetic Strategy	• Administration of general anesthesia with definitive airway management via endotracheal tube. • Deployment of strict neuroprotective protocols to limit cerebral metabolic demand. • Induction phase managed with a targeted combination of thiopental, fentanyl, and atracurium.
Surgical Interventions	• **Neurosurgery:** Emergent neurosurgical decompressive craniotomy for hematoma evacuation and pressure relief. • **Plastic Surgery:** Surgical debridement of necrotic tissue and reconstruction of nasal and philtrum defects using a rotation flap. • Thorough cleansing and debridement of superficial facial wounds.
3. FOLLOW UP AND OUTCOME	
Postoperative Care	Admitted directly to the intensive care unit for continuous invasive monitoring of hemodynamics and neurological status.
Length of Stay	The intensive care management and observation phase lasted for a total of 5 days.
Final Clinical Outcome	• The patient demonstrated a highly favorable recovery trajectory. • Successfully discharged from the hospital without any measurable residual neurological deficits.

This rapid decompensation manifested radiologically and clinically as a severe 1.5 cm subfalcine herniation alongside downward transtentorial herniation. Subfalcine herniation involves the displacement of the cingulate gyrus beneath the falx cerebri, severely compromising the anterior cerebral artery and placing the patient at imminent risk of an ischemic stroke in the affected vascular territory. Transtentorial herniation is an even more critical neurosurgical emergency; it forces the uncus and medial temporal lobe structures downward through the tentorial incisura.[13] This displacement directly compresses the ipsilateral oculomotor nerve, the posterior cerebral artery, and the vital structures of the midbrain and cerebral peduncles, ultimately leading to profound autonomic dysregulation, respiratory arrest, and irreversible brainstem death. While prompt surgical evacuation is the definitive treatment, the precise anesthetic management during the highly precarious preoperative and pre-dural opening phases strictly dictates the patient's ultimate neurological outcome.

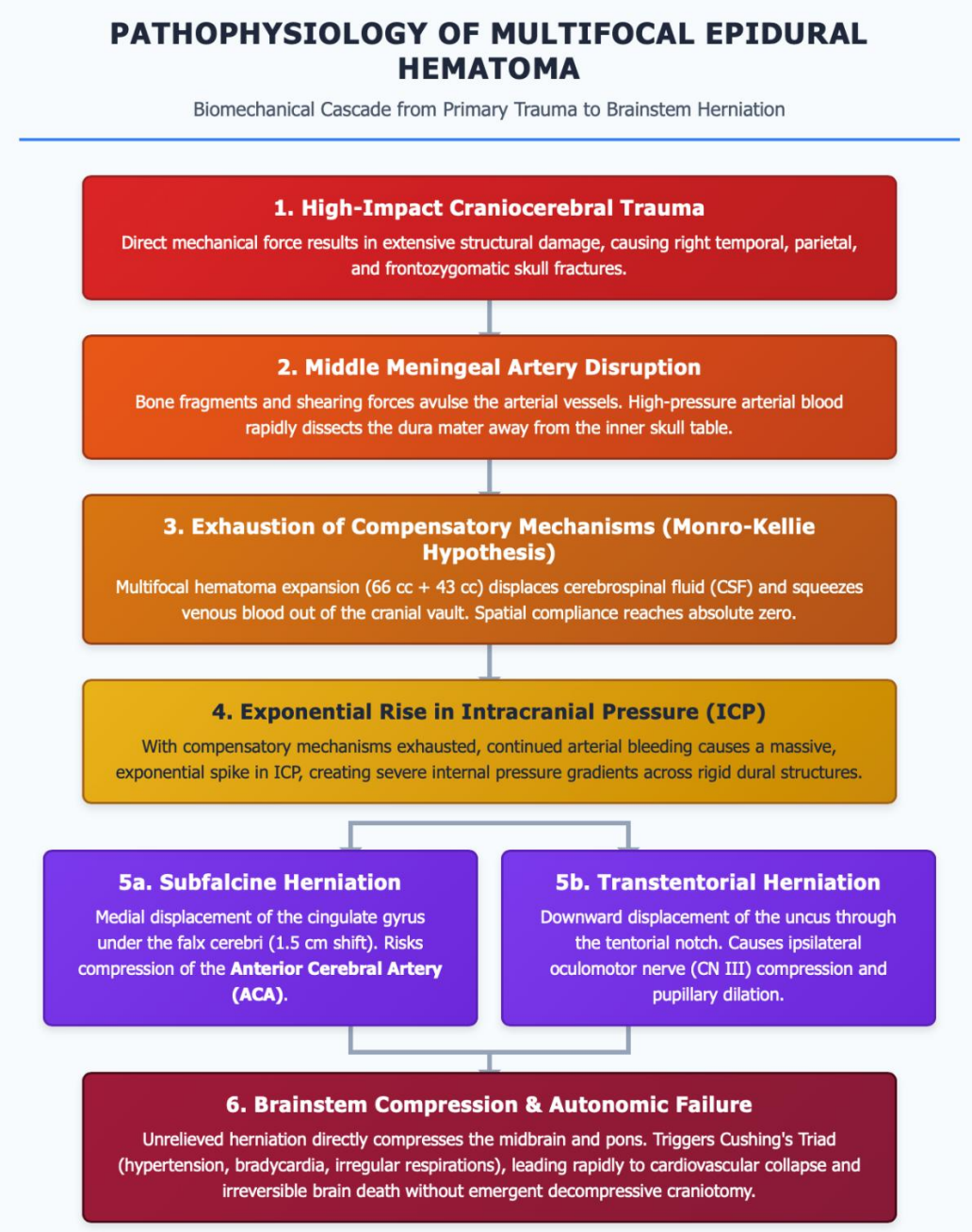

Figure 2. Pathophysiology of multifocal epidural hematoma and herniation.

The absolute cornerstone of advanced anesthetic management in severe traumatic brain injury is the rigorous maintenance of an optimal cerebral perfusion pressure.[14] Cerebral perfusion pressure is mathematically defined as the systemic mean arterial pressure minus the intracranial pressure. As repeatedly demonstrated in contemporary neuroanesthesia literature, maintaining a robust and continuous cerebral perfusion pressure is completely vital to preventing the fatal exacerbation of cerebral edema and halting the dangerous expansion of the secondary ischemic penumbra. In the management of this specific case, thiopental was strategically chosen as the primary agent for anesthetic induction. Thiopental, a highly potent barbiturate, confers massive neuroprotection through several advanced and distinct pharmacological mechanisms. Primarily, thiopental induces a profound, highly dose-dependent reduction in the cerebral metabolic rate of oxygen. Because cerebral blood flow is metabolically coupled to the cerebral metabolic rate of oxygen, this massive metabolic suppression causes secondary cerebral vasoconstriction. This targeted vasoconstriction actively decreases the total intracranial cerebral blood volume, leading to an immediate, dramatic, and life-saving reduction in the overall intracranial pressure.[15]

Furthermore, thiopental possesses unique cellular protective properties. It actively attenuates glutamate-mediated excitotoxicity and prevents free radical lipid peroxidation, both of which are central biochemical drivers of massive cellular apoptosis and necrosis in the secondary phase of traumatic brain injury.[16] This specific physiological profile makes thiopental exceptionally useful for the rapid induction of anesthesia in patients presenting with a confirmed high risk of massive intracranial hypertension or impending cerebral ischemia.

Recent highly specialized comparative studies heavily validate this pharmacological approach. Hashimoto and colleagues meticulously described a highly comparable pediatric case wherein thiopental completely superseded propofol for anesthetic induction in the setting of an epidural hematoma, explicitly noting thiopental's superior clinical efficacy in rapidly lowering intracranial pressure during hyperacute cranial emergencies.[17] Similarly, Acharya and colleagues confirmed the high efficacy of utilizing a thiopental and fentanyl-based induction triad in heavily stabilizing hemodynamics and securing highly favorable long-term neurological recovery in patients undergoing emergency decompressive craniotomies. Sevoflurane was subsequently utilized for the maintenance of anesthesia in our patient. At carefully titrated concentrations below 1.0 minimum alveolar concentration, sevoflurane exerts a remarkably minimal uncoupling effect on intrinsic cerebral autoregulation, thereby facilitating excellent hemodynamic stability and allowing for rapid, early postoperative neurological assessments.

Hemodynamic management in severe traumatic brain injury must carefully navigate a highly narrow therapeutic window. The anesthesiologist must completely avoid systemic hypotension, which causes immediate, devastating cerebral ischemia, while simultaneously preventing extreme systemic hypertension, which directly exacerbates cerebral edema and accelerates arterial hematoma expansion.[18] In our patient, a highly balanced administration of fentanyl effectively and completely blunted the massive sympathetic reflex typically triggered by direct laryngoscopy, completely preventing dangerous surges in both the mean arterial pressure and the intracranial pressure. Furthermore, the confirmed radiological presence of traumatic pneumocephalus strictly and absolutely contraindicated the use of nitrous oxide during the maintenance of anesthesia. Nitrous oxide diffuses rapidly into closed, air-filled physiological spaces significantly faster than nitrogen can exit. The administration of nitrous oxide in this scenario would rapidly convert a simple pneumocephalus into a massive tension pneumocephalus, directly precipitating acute and fatal brainstem compression on the operating table.[19]

A primary limitation of this specific study is its inherent reliance on a single patient case report narrative, which naturally restricts the broad statistical generalizability of the specific clinical findings to the entire global polytrauma population. Furthermore, while the neuroprotective mechanisms of thiopental are

exceptionally well-documented and proven in this case, modern neuroanesthesia is gradually exploring total intravenous anesthesia techniques utilizing targeted propofol infusions due to distinct differences in pharmacokinetic clearance profiles.[20]

Future clinical research must be heavily focused on establishing large-scale, multi-center randomized controlled trials directly comparing the long-term neurocognitive and functional outcomes of thiopental-based inductions versus propofol-based inductions specifically in the setting of severe pediatric traumatic brain injury. Additionally, as recently explored by Ryan and colleagues, the careful integration of highly targeted regional anesthesia techniques—such as advanced scalp blocks or specialized caudal blocks in specific pediatric trauma populations—holds massive clinical promise for significantly reducing systemic opioid requirements and greatly accelerating postoperative neurological recovery. However, these regional techniques require incredibly rigorous research to completely ensure their absolute safety in polytrauma patients who frequently present with highly active traumatic coagulopathies or massive active hemorrhage. Future neurosurgical paradigms must also meticulously optimize the exact chronological timing of these surgical interventions and fully integrate ultra-advanced non-invasive continuous neuromonitoring technologies to perfectly guide targeted, individualized hemodynamic therapies.

4. Conclusion

The perioperative management of a massive multifocal epidural hematoma complicated by severe, impending brain herniation unequivocally mandates a rapid, highly coordinated multidisciplinary approach strictly centered on the rigorous control of intracranial pressure and the absolute optimization of cerebral perfusion. This comprehensive case report clearly demonstrates that thiopental remains a highly effective, incredibly reliable neuroprotective induction agent for the highest-risk neurotrauma patients. By profoundly depressing both the cerebral metabolic rate and the total intracranial vascular volume, thiopental brilliantly facilitated a highly stable intraoperative physiological course during a massive concurrent decompressive craniotomy and complex facial reconstruction. Individualized, pathophysiology-driven anesthetic strategies are absolutely paramount in successfully minimizing secondary brain injury and ensuring the highest possible level of optimal neurological recovery in severe craniocerebral polytrauma.

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