

CASE REPORT

Thiopental-Based Neuroprotective Anesthesia for Emergency Craniotomy in an Adolescent with Multifocal Epidural Hematoma and Impending Herniation: A Case Report

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ABSTRACT

Background. Multifocal traumatic epidural hematoma with pneumocephalus and impending herniation is a neurosurgical emergency in which anesthetic management can limit or add to secondary brain injury. Reports in adolescents are few.

Objective. To describe the anesthetic management and physiological course of an adolescent who underwent emergency decompressive craniotomy and facial reconstruction under a thiopental-based protocol.

Methods. A 17-year-old male (50 kg, Glasgow Coma Scale 12, ASA 4E) was admitted after a high-velocity injury. Computed tomography showed epidural hematomas (66 and 43 mL), pneumocephalus, a 15-mm midline shift and early transtentorial herniation. Anesthesia was induced with fentanyl 2 mcg/kg, thiopental 4 mg/kg and atracurium 0.5 mg/kg and maintained with sevoflurane without nitrous oxide, with invasive arterial monitoring and targets of mean arterial pressure (MAP) 85–95 mmHg and end-tidal CO₂ 30–35 mmHg. Intracranial pressure was estimated, not measured.

Results. MAP fell from 104 to 86 mmHg (–17.3%) at induction and stayed at 85–95 mmHg thereafter; the response to intubation was blunted. Estimated cerebral perfusion pressure was 58–80 mmHg. Blood loss was 600 mL, replaced with 1,200 mL of crystalloid, without transfusion. The patient was extubated on postoperative day 2 and left the intensive care unit on day 5 with a Glasgow Coma Scale score of 15 and a Glasgow Outcome Scale score of 5.

Conclusion. A titrated thiopental-based induction with strict pressure and ventilation targets and no nitrous oxide was followed by stable hemodynamics and a good early recovery. One case cannot show that thiopental protected the brain.

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1. Introduction

Traumatic brain injury is a leading cause of death and disability in adolescents and young adults, particularly in low- and middle-income regions.¹ Injury evolves in two phases: the mechanical insult, and a secondary phase of excitotoxicity, energy failure, edema and ischemia that perioperative care can limit or aggravate.^{2,3} Epidural hematoma is among the most time-critical extra-axial lesions. It usually follows a temporoparietal fracture near the pterion that tears the middle meningeal artery.^{4–6} In a pediatric trauma-center series about half of the children with epidural hematoma needed craniotomy,⁷ and in modern series timely surgery gives a high probability of a favorable outcome.⁸ Because the bleeding is arterial, the intracranial compensatory reserve can be exhausted within hours, as described by the Monroe-Kellie doctrine.^{9,10} Once the reserve is used up, small added volumes cause large rises in intracranial pressure (ICP),¹¹

and brain tissue herniates beneath the falx and through the tentorial incisura; brainstem compression can end in Duret hemorrhage and death (Figure 1).¹²

Evacuation by craniotomy is the definitive treatment, and surgical guidelines recommend evacuation of epidural hematomas larger than 30 mL.¹³ The anesthetic still influences the result. Autoregulation is often impaired after severe injury, so cerebral perfusion pressure (CPP = mean arterial pressure [MAP] – ICP) follows systemic pressure; hypotension and hypoxia deepen ischemia, whereas hypertensive surges during laryngoscopy can raise ICP and bleeding.^{14–17} Ventilation matters as well: hyperventilation lowers ICP and relaxes the brain but reduces cerebral blood flow,¹⁸ and extreme derangements of PaCO₂ are associated with higher mortality.¹⁹ Facial fractures add airway risk,²⁰ and intracranial air makes nitrous oxide unsafe because the gas expands closed air spaces and can produce tension pneumocephalus.^{21–24}

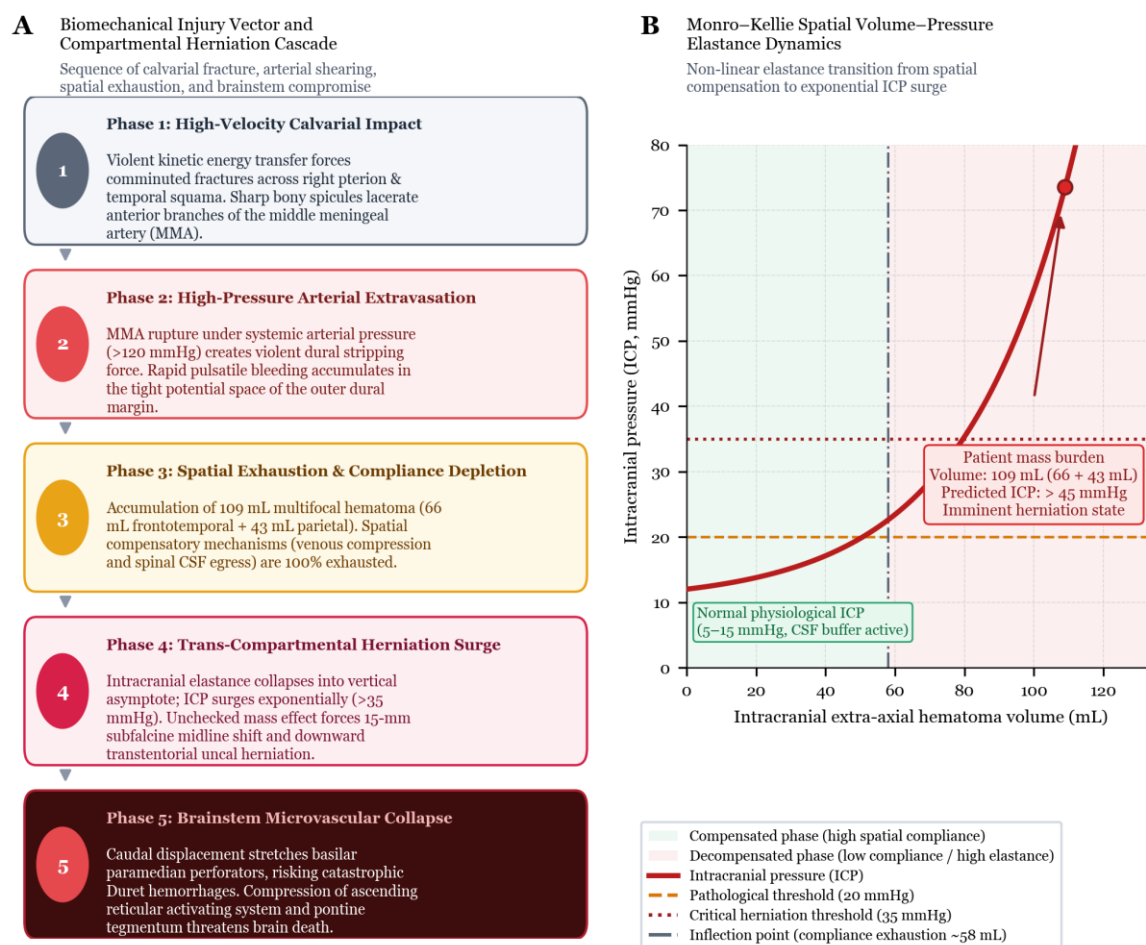


Figure 1. Schematic of the progression from epidural hematoma to brainstem herniation. (A) Five-phase sequence from calvarial impact and middle meningeal artery laceration to brainstem compromise. (B) Illustrative nonlinear volume–pressure (Monro–Kellie) curve. The curve, the inflection point at about 58 mL and the predicted ICP above 45 mmHg are model values, not measurements. ICP, intracranial pressure; MMA, middle meningeal artery; CSF, cerebrospinal fluid.

Thiopental is an ultra-short-acting barbiturate that acts on GABA-A receptors and has prolonged elimination kinetics.²⁵ It lowers cerebral metabolism and, through flow–metabolism coupling, cerebral blood volume and ICP. Experimental and clinical reports suggest neuroprotective effects,^{26–28} and it is used as a last-tier treatment of intracranial hypertension.²⁹ Barbiturate coma lowered ICP in children with refractory intracranial hypertension after traumatic brain injury,^{30,31} yet guidelines place high-dose barbiturates in the second tier or second line, and the evidence that they improve outcome is weak.^{16,32,33} Propofol, ketamine and volatile agents each have advantages and drawbacks in neurotrauma.^{3,34,35} Published anesthetic reports of adolescents and young patients with traumatic brain injury are few and mostly single cases.^{36–38}

This report describes the anesthetic management and physiological course of an adolescent with multifocal epidural hematomas, pneumocephalus and impending herniation who underwent emergency decompressive craniotomy and facial reconstruction under a thiopental-based protocol, and it states what one case can and cannot show.

2. Methods

2.1. Report design and reporting guideline

This is a single case report prepared according to the CARE guideline.³⁹

2.2. Setting

The patient was managed at Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia, a tertiary referral and teaching hospital of Universitas Brawijaya. Care took place in the emergency department, the operating theater and the intensive care unit (ICU).

2.3. Patient information and case selection

The patient was a 17-year-old male, 50 kg and 160 cm (body mass index 19.53 kg/m²), who was brought to the emergency department after a high-velocity injury. His last oral intake was dinner on the preceding evening, and he had had no previous general anesthetic. The case is reported because multifocal epidural hematomas, pneumocephalus, impending herniation and facial trauma required simultaneous neurosurgical and reconstructive surgery under one anesthetic.

2.4. Case timeline

Time is given relative to the first observation (T0, awake in the emergency department) and is shown in Table 1. Ten milestones (T0–T9) span 150 minutes, followed by five days in the ICU. Percentage change ($\Delta\%$) from T0 was calculated as $[(T_n - T_0) / T_0] \times 100$. Rate-pressure product (RPP) was calculated as heart rate $\times$ systolic pressure, and CPP as MAP – ICP.

Table 1. Clinical timeline of the case from admission to day 5, using relative time.

Time point	Clinical event	Investigation and result	Intervention
Admission (day 0)	High-velocity injury; GCS 12; otorrhagia; facial laceration	RR 28/min; SpO ₂ 98%; BP 129/91 (MAP 103.67 mmHg); HR 74; pupils 3 mm, isocoric; CT: EDH 66 + 43 mL, pneumocephalus, 15-mm shift, transtentorial herniation; WBC $15.9 \times 10^3/\mu\text{L}$	Two IV lines; 2 U PRBC and 1 U whole blood cross-matched
T1 (5 min)	Pre-oxygenation	HR 73; MAP 102 mmHg; SpO ₂ 100%	FiO ₂ 1.0 by face mask
T2 (10 min)	Induction	HR 68; MAP 86 mmHg (−17.3%); est. ICP 28; CPP 58 mmHg	Fentanyl 100 mcg; thiopental 200 mg; atracurium 25 mg
T3 (15 min)	Intubation at first attempt	HR 76; MAP 91 mmHg; RPP 8,740 bpm·mmHg; ETCO ₂ 36 mmHg	7.5 cuffed tube; sevoflurane 0.8–1.0 MAC in O ₂ –air
T4–T5 (25–40 min)	Skin incision; dural opening	MAP 95 → 90 mmHg; est. ICP 29 → 22; CPP 66 → 68 mmHg	Right frontotemporoparietal craniotomy
T6 (65 min)	Hematoma evacuation	MAP 85 mmHg; est. ICP 14; CPP 71 mmHg	66 + 43 mL evacuated; MMA branches secured
T7–T8 (110–140 min)	Facial reconstruction; closure	MAP 88–92 mmHg; CPP 76–80 mmHg	Debridement, rotation flap; dural hitching, bone flap and skin closure
T9 (150 min)	Transfer to ICU	MAP 90 mmHg; CPP 77 mmHg; ETCO ₂ 38 mmHg	Intubated and sedated; no inotrope
Days 1–2	Sedation weaned (12 h); extubated (24 h)	GCS 10 → 11; PaO ₂ /FiO ₂ 440–460; WBC 13,800 → 11,500/ μL	Weaning; nasal cannula
Days 3–5	GCS 14 → 15; walking independently	WBC 9,400 → 6,800/ μL ; GOS 5	Room air; left the ICU on day 5

Notes: CT, computed tomography; EDH, epidural hematoma; FiO₂, inspired oxygen fraction; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; HR, heart rate (beats/min); ICP, intracranial pressure (estimated); MAC, minimum alveolar concentration; MAP, mean arterial pressure; MMA, middle meningeal artery; PRBC, packed red blood cells; RPP, rate-pressure product; RR, respiratory rate; WBC, white blood cell count; CPP = MAP – estimated ICP.

2.5. Clinical findings and diagnostic assessment

Assessment comprised neurological triage (Glasgow Coma Scale [GCS], pupils), laboratory tests and emergency non-contrast computed tomography (CT) of the head with 3D craniofacial reconstruction. ICP was not measured. Before decompression it was estimated from dural tension and radiological midline shift, and after decompression from the surgeon's grading of brain relaxation.

2.6. Therapeutic intervention

Two peripheral lines (18G and 20G) were placed and 0.9% sodium chloride was started at 100 mL/h. Two units of packed red blood cells and one unit of whole blood were cross-matched. After pre-oxygenation with an inspired oxygen fraction (FiO₂) of 1.0, fentanyl 2 mcg/kg (100 mcg), thiopental 4 mg/kg (200 mg, over 30–45 seconds) and atracurium 0.5 mg/kg (25 mg) were given. A 7.5 cuffed tube was passed by direct laryngoscopy.

Anesthesia was maintained with sevoflurane 0.8–1.0 minimum alveolar concentration (MAC) in oxygen–air (FiO₂ 0.50); nitrous oxide was avoided because of pneumocephalus. Ventilation was volume-targeted (tidal volume 350 mL, 7 mL/kg; 16 breaths/min; positive end-expiratory pressure 5 cmH₂O) to an end-tidal CO₂ (ETCO₂) of 30–35 mmHg. A 20G radial arterial catheter gave continuous pressure, with a MAP target of 85–95 mmHg. Core temperature was kept at 36.5–37.0°C with an esophageal probe and convective warming.

The neurosurgeons performed a right frontotemporoparietal decompressive craniotomy, evacuated both hematomas, secured the torn middle meningeal arterial branches and hitched the dura. During the same anesthetic, plastic surgeons debrided the wounds and closed the nasal and philtrum lacerations with a rotation flap. The patient went to the ICU intubated and sedated.

2.7. Outcome measures and follow-up

Outcomes were heart rate, MAP, oxygen saturation (SpO₂), ETCO₂, estimated ICP, CPP and RPP at the ten milestones, and in the ICU the GCS, pupils, MAP, PaO₂/FiO₂ ratio, leukocyte count and ventilation status. The Glasgow Outcome Scale (GOS) was recorded at ICU discharge. Follow-up ended at ICU discharge on day 5. No inferential statistics were calculated, because $n = 1$.

2.8. Ethics and consent

The Central Medical & Health Committee (CMHC) Ethics Committee, Indonesia, approved the report under reference number 2025/4109. Written informed consent was obtained from the patient's legal guardians (parents) for the analysis and publication of anonymized records, physiological data and images, in line with the Declaration of Helsinki.⁴⁰

3. Results

3.1. Presentation and baseline findings

On arrival the patient had a GCS of 12 (E3 V4 M5), a patent airway and spontaneous breathing at 28 breaths/min with SpO₂ 98% on room air. Blood pressure was 129/91 mmHg (MAP 103.67 mmHg) and heart rate 74 beats/min. Both ears were bleeding, and there was facial edema with a deep nasal and philtrum laceration. The pupils were 3.0 mm, isocoric and reactive to light, and the heart rate was 74 beats/min, so neither anisocoria nor a Cushing response was present at triage despite the CT findings below. A GCS of 12 classifies the injury as moderate.

The leukocyte count was $15.9 \times 10^3/\mu\text{L}$, platelet count $283 \times 10^3/\mu\text{L}$, prothrombin time 10.7 s (control 11.6 s), activated partial thromboplastin time 28.9 s (control 26.3 s) and random glucose 120 mg/dL. Urea nitrogen and creatinine were reported as normal. Findings are summarized in Table 2, and the timeline in Table 1.

Table 2. Admission findings.

Finding	Value	Reference interval
Weight; height; body mass index	50 kg; 160 cm; 19.53 kg/m ²	18.50–24.90 kg/m ²
Respiratory rate; SpO ₂ (room air)	28/min; 98%	12–20/min; ≥ 95%
Blood pressure; MAP	129/91 mmHg; 103.67 mmHg	90–120/60–80; 70–100 mmHg
Heart rate	74 beats/min	60–100 beats/min
Glasgow Coma Scale	12 (E3 V4 M5)	15 (E4 V5 M6)
Pupils	3.0 mm, isocoric, reactive	2.0–4.0 mm, isocoric
Leukocytes	15.9 × 10 ³ /μL	4.5–11.0 × 10 ³ /μL
Platelets	283 × 10 ³ /μL	150–450 × 10 ³ /μL
Prothrombin time (control)	10.7 s (11.6 s)	9.8–12.5 s
Activated partial thromboplastin time (control)	28.9 s (26.3 s)	25.0–35.0 s
Random glucose	120 mg/dL	70–140 mg/dL
Urea nitrogen; creatinine	Reported as normal	7–20 mg/dL; 0.6–1.2 mg/dL
CT: epidural hematoma; midline shift	66 + 43 mL (109 mL); 15 mm leftward	None; 0 mm

Notes: BP, blood pressure; CT, computed tomography; MAP, mean arterial pressure; SpO₂, peripheral oxygen saturation. Reference intervals as given in the submitted manuscript.

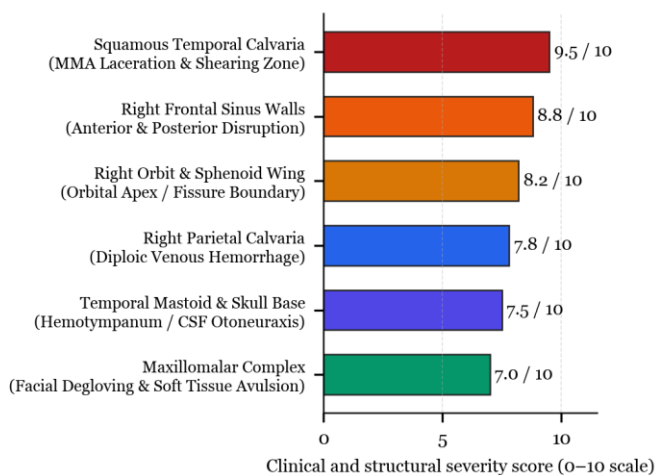
3.2. Diagnostic findings

Head CT showed epidural hematomas of 66 mL in the right frontotemporal region and 43 mL in the right parietal region (total 109 mL), pneumocephalus along the frontal convexity and skull base, and 15 mm of leftward subfalcine herniation with distortion of the lateral ventricles and effacement of the ipsilateral basal cisterns. There was

downward transtentorial herniation at pontine level with partial obliteration of the ambient and prepontine cisterns. Fractures involved the right squamous temporal bone through the groove of the middle meningeal artery, the right frontal sinus walls, superior orbital fissure, parietal bone, mastoid and occipital bone, as well as the maxillary and sphenoid sinuses and frontozygomatic sutures (Figure 2).

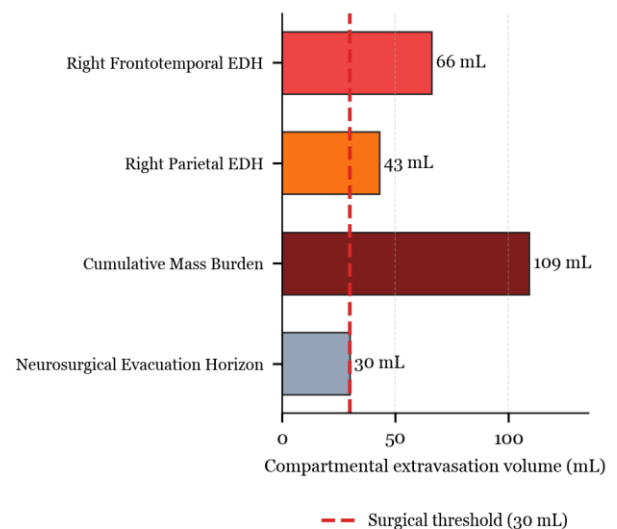
A Craniofacial Skeletal Fracture Topography & Disruption

Multiregional fracture severity indexing across skull base and calvaria



B Compartmental Hematoma Volumetrics & Herniation Vectors

Cumulative hematoma volume (109 mL) exceeding surgical threshold by 263%



Computed tomography herniation parameters

- 1.5 cm (15 mm) leftward subfalcine shift (high ACA stroke risk)
- Transtentorial uncal descent with pontine cistern obliteration
- Pneumocephalus from skull base breach

Figure 2. Fractures and hematoma volumes on computed tomography. (A) Severity scores (0–10) assigned to six fracture regions. (B) Hematoma volumes (66 mL frontotemporal, 43 mL parietal, 109 mL combined) against the 30 mL surgical threshold; the combined volume is 263% above the threshold. EDH, epidural hematoma; MMA, middle meningeal artery; ACA, anterior cerebral artery; CSF, cerebrospinal fluid.

3.3. Treatment course and outcome

The elapsed time from T0 to ICU transfer was 150 minutes. Induction (T2, 10 min) lowered MAP from 104 to 86 mmHg (–17.3%) and heart rate from 74 to 68 beats/min (–8.1%). Intubation (T3) succeeded at the first attempt without desaturation; MAP was 91 mmHg, heart rate 76 beats/min

and RPP 8,740 bpm·mmHg. From incision to ICU transfer MAP stayed at 85–95 mmHg and SpO₂ at 100% (Table 3, Figure 3). ETCO₂ was 31–38 mmHg and exceeded the 30–35 mmHg target at T3, T7 and T8 (36 mmHg) and T9 (38 mmHg). Estimated ICP fell from 38 mmHg at baseline to 28 mmHg after induction and to 12–14 mmHg after hematoma evacuation. Calculated CPP was therefore 66 mmHg at

baseline, 58 mmHg after induction (the lowest value) and 61–80 mmHg thereafter (Figure 4). RPP remained below 9,600 bpm·mmHg at all milestones.

Table 3. Intraoperative hemodynamic and ventilatory record at ten milestones.

Milestone	Time (min)	HR	MAP	SpO ₂	ETCO ₂	Est. ICP	CPP	RPP	MAP Δ%	CPP Δ%
T0 baseline	0	74	104	98	—	38	66	9,546	0.0	0.0
T1 pre-oxygenation	5	73	102	100	—	38	64	9,198	-1.9	-3.0
T2 induction	10	68	86	100	33	28	58	7,344	-17.3	-12.1
T3 intubation	15	76	91	100	36	30	61	8,740	-12.5	-7.6
T4 incision	25	78	95	100	34	29	66	9,360	-8.7	0.0
T5 dural opening	40	72	90	100	31	22	68	8,208	-13.5	+3.0
T6 evacuation	65	70	85	100	33	14	71	7,560	-18.3	+7.6
T7 reconstruction	110	74	88	100	36	12	76	8,288	-15.4	+15.2
T8 closure	140	75	92	100	36	12	80	8,700	-11.5	+21.2
T9 ICU transfer	150	76	90	100	38	13	77	8,664	-13.5	+16.7

Notes: HR, beats/min; MAP and estimated ICP, mmHg; SpO₂, %; ETCO₂, mmHg; CPP = MAP – estimated ICP, mmHg; RPP = HR × systolic pressure, bpm·mmHg; Δ%, change from T0 = [(Tn – T0) / T0] × 100. ICP was estimated (see Methods). Systolic pressures are not tabulated.

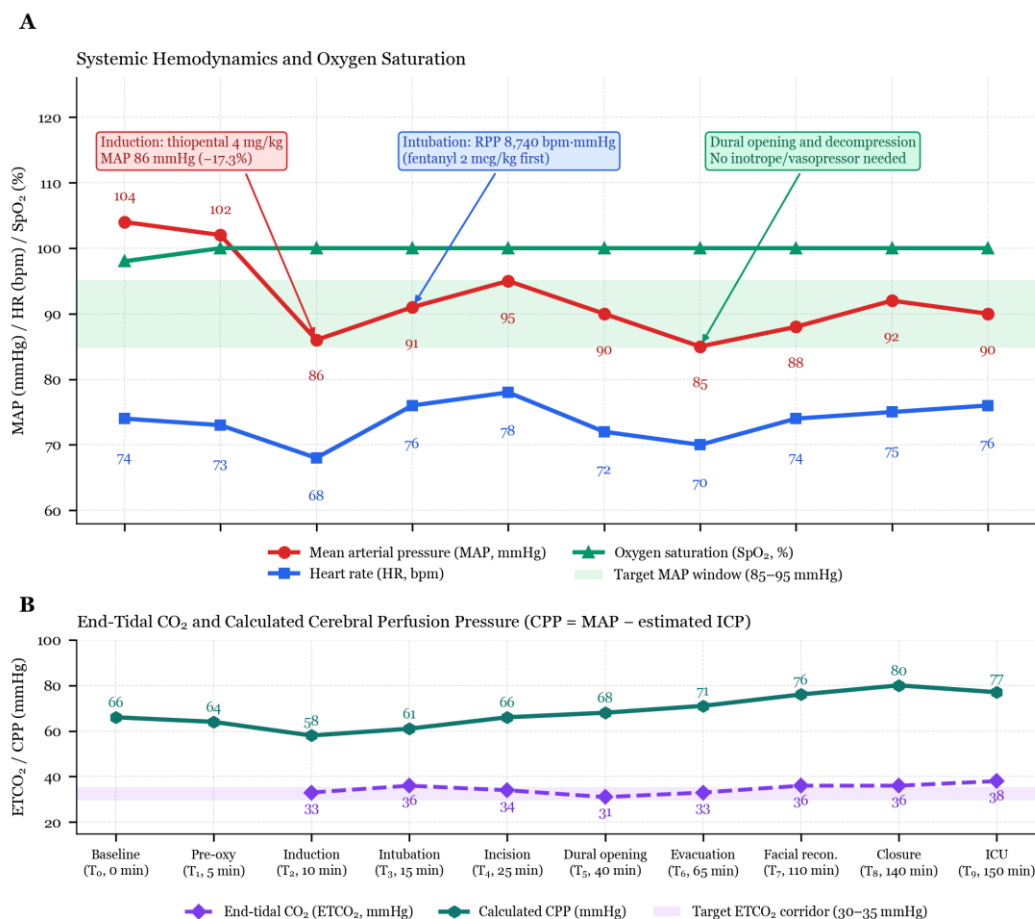


Figure 3. Intraoperative course at ten milestones. (A) Mean arterial pressure (MAP), heart rate (HR) and oxygen saturation (SpO₂); the shaded band is the MAP target of 85–95 mmHg. (B) End-tidal CO₂ (ETCO₂; target 30–35 mmHg, shaded) and calculated cerebral perfusion pressure (CPP = MAP – estimated ICP). Values are those of Table 3. ICP, intracranial pressure; RPP, rate-pressure product.

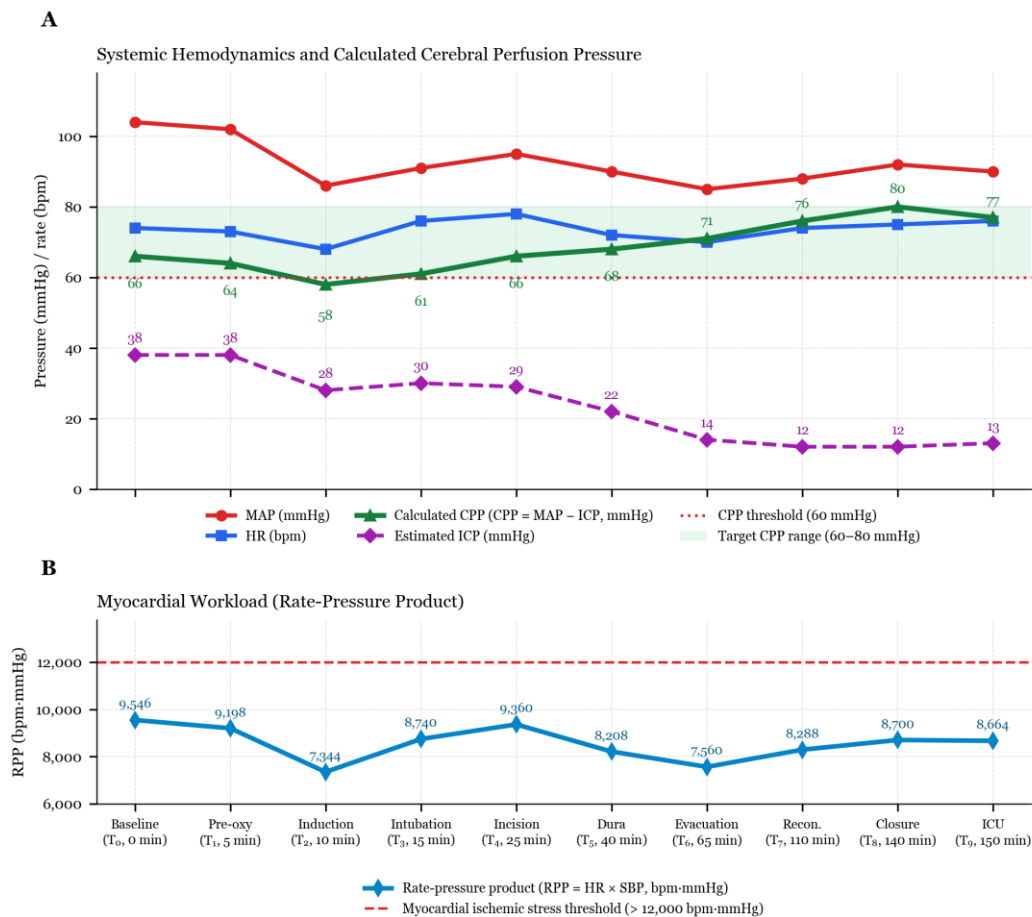


Figure 4. Calculated cerebral perfusion pressure and myocardial workload. (A) MAP, HR, estimated ICP and calculated CPP across the ten milestones; the dotted line marks 60 mmHg. (B) Rate-pressure product (RPP = HR × systolic pressure); the dashed line marks 12,000 bpm·mmHg. ICP values are estimates.

Blood loss was about 600 mL and 1,200 mL of balanced crystalloid was given; no blood product and no inotrope was needed. The brain was relaxed after the dura was opened, and the facial wounds were closed with a rotation flap. The patient reached the ICU intubated and sedated at 150 minutes.

On ICU arrival he was ventilated in synchronized intermittent mandatory mode with a PaO₂/FiO₂ ratio of 420 (FiO₂ 0.40) and MAP of 88 ± 4 mmHg (Table 4).

Sedation was weaned after 12 hours (GCS 10: E3, intubated, M6; eyes opening to voice and obeying commands). He was extubated to a nasal cannula at 24 hours (GCS 11: E4, intubated, M6) with clear speech and intact cranial nerves. The GCS was 14 at 48 hours and 15 at 72 hours, the leukocyte count fell from 15,200 to 6,800/μL (−55.3%), and the rotation flap remained viable without infection. On day 5 he left the ICU with a GCS of 15, a GOS score of 5 and no focal deficit.

Table 4. Intensive care course and neurological recovery.

Time	GCS (E/V/M)	MAP (mmHg)	PaO ₂ /FiO ₂	WBC (/ μ L)	Ventilation	Neurological and clinical milestones
ICU arrival (day 1, hour 0)	Sedated (E1 T M1)	88 ± 4	420 (FiO ₂ 0.40)	15,200	Mechanical ventilation (SIMV)	Head elevated 30°; pupils isocoric 3 mm, brisk
Hour 12	10 (E3 T M6)	86 ± 3	440 (FiO ₂ 0.35)	13,800	Weaning; sedation interrupted	Eyes opening to voice; obeying commands
Hour 24 (day 2)	11 (E4 T M6)	85 ± 4	460 (FiO ₂ 0.30)	11,500	Extubated to nasal cannula	Clear speech; alert; no cranial nerve deficit
Hour 48 (day 3)	14 (E4 V4 M6)	84 ± 3	480 (room air)	9,400	Room air (SpO ₂ 99%)	Oriented to person and place; mild retrograde amnesia
Hour 72 (day 4)	15 (E4 V5 M6)	82 ± 4	490 (room air)	8,100	Room air (SpO ₂ 99%)	Fully oriented; walking independently; flap viable
Day 5	15 (E4 V5 M6)	80 ± 3	495 (room air)	6,800	Room air (SpO ₂ 99%)	Discharged from the ICU; GOS 5

Notes: E, eye; V, verbal; M, motor; T, intubated (no verbal score); GCS, Glasgow Coma Scale; MAP, mean arterial pressure; PaO₂/FiO₂, arterial oxygen tension to inspired oxygen fraction ratio (mmHg); WBC, white blood cell count; SIMV, synchronized intermittent mandatory ventilation; GOS, Glasgow Outcome Scale.

4. Discussion

4.1. Principal lesson of the case

In an adolescent with multifocal epidural hematomas, pneumocephalus and impending herniation, a slowly titrated thiopental–fentanyl–atracurium induction, sevoflurane without nitrous oxide, invasive pressure monitoring and a MAP window of 85–95 mmHg were followed by stable hemodynamics and a good early recovery. Evacuation of the hematoma, not the anesthetic, relieved the mass effect. The report documents a feasible approach; it cannot show that thiopental protected the brain.

4.2. Comparison with the published literature

Anesthetic reports in young patients with traumatic brain injury are mostly single cases: neuroanesthesia for a child with a gunshot wound,³⁶ thiopental with dexmedetomidine for brain relaxation during craniotomy,³⁷ and a pediatric epidural hematoma caused by contrecoup injury.³⁸ They give little quantitative physiology, so a point-by-point comparison is not possible. The main goals of care in this case (avoiding hypotension and hypoxia, controlling

ventilation, avoiding nitrous oxide when intracranial air is present) follow current reviews and guidelines.^{3,14,23,41}

Table 5 sets this case beside published series chosen for relevance to the clinical problem, with the numbers as the authors reported them. Outcomes after surgery for epidural hematoma are generally good: in-hospital mortality was 3.1% among 131 children in Pakistan,⁴² 6.8% among 131 surgically treated patients in a level-1 trauma center,⁸ 13.9% in Korean patients with a GCS of 8 or less⁴³ and 18.6% after burr-hole surgery in Tanzania.⁴⁴ A full recovery in our patient, who had a GCS of 12 and herniation on CT, is consistent with these reports. Data on thiopental are heterogeneous. Barbiturate coma lowered ICP in children with refractory intracranial hypertension with preserved CPP,^{30,31} thiopental during aneurysm clipping was associated with fewer neurological complications,²⁶ brain relaxation did not differ between thiopental and propofol in a randomized study in which thiopental gave higher MAP at induction,⁴⁵ and in elective tumor craniotomy thiopental infusion delayed extubation compared with propofol.⁴⁶ None of these studies concerned a single induction dose in traumatic epidural hematoma.

Table 5. Published reports used for comparison.

Report	Design and sample	Population or intervention	Main findings as reported
This case	Case report, n = 1	17-year-old, GCS 12; epidural hematomas 66 + 43 mL; thiopental–fentanyl–atracurium induction; craniotomy and facial reconstruction	MAP –17.3% at induction, then 85–95 mmHg; extubated day 2; GOS 5 at ICU discharge (day 5)
Khan et al. 2026 ⁴²	Retrospective cohort, 289 patients (131 pediatric), Pakistan	Surgical evacuation of traumatic epidural hematoma	In-hospital mortality 3.1% in children vs 14% in adults; pediatric volume ≥ 33.5 mL predicted mortality; earlier surgery lowered risk in children
Gutowski et al. 2018 ⁸	Retrospective, 268 patients, 131 operated, level-1 trauma center (1997–2012)	Surgically treated epidural hematoma	Mortality 6.8%; midline shift, other intracranial injuries and injury severity predicted outcome
Choi et al. 2022 ⁴³	Registry analysis (Korean Neuro-Trauma Data Bank)	Epidural hematoma with GCS ≤ 8 who underwent surgery	Mortality 13.9%; good outcome 60.8% at 6 months; age and bilateral unreactive pupils independent risk factors
Cremonini et al. 2020 ⁷	Retrospective, 106 children with epidural hematoma, level-I trauma center (2008–2018)	Pediatric epidural hematoma	Craniotomy in 50%; mortality 2%; 13% discharged with cognitive or motor deficits; 40% presented with GCS 15
Koipapi et al. 2024 ⁴⁴	Cross-sectional, 156 patients, Tanzania	Burr-hole surgery for acute extra-axial hematoma (70% epidural)	Mortality 18.6%; good outcome 75.6%; 19.2% had herniation syndromes
Graziano et al. 2025 ⁵¹	CENTER-TBI secondary analysis, 3,661 patients (227 pediatric, 135 adolescents)	Management and outcome of pediatric versus adult traumatic brain injury	ICP monitored in 39.4% of pediatric ICU patients; age < 18 years associated with a lower risk of poor 6-month outcome (adolescents OR 0.31)
Velle et al. 2020 ³⁰	Retrospective cohort, 21 children (median age 14 years)	Barbiturate coma for refractory intracranial hypertension after TBI	Lower ICP and no major side effects; median GOS 5 at 1 year
Velle et al. 2021 ³¹	Cohort, 17 children (median age 15 years)	Barbiturate coma, high-resolution monitoring	Median ICP 22 → 16 mmHg at 5 h; CPP 65 → 63 mmHg (not significant); favorable outcome in 82%
Kim et al. 2021 ²⁶	Retrospective, 491 patients, propensity-matched	Thiopental during clipping of unruptured aneurysm	Postoperative neurological complications 5.5% vs 17.1% (p = 0.001)
Rozec et al. 2014 ⁴⁶	Randomized trial, 80 patients	Elective supratentorial tumor craniotomy; propofol vs thiopental infusion	Median time to extubation 149 vs 453 min (propofol vs thiopental); hemodynamics similar
Valentino et al. 2025 ⁴⁵	Randomized experimental study, Indonesia	Elective craniotomy for supratentorial tumor; propofol vs thiopental	Good brain relaxation in 90% (thiopental) vs 70% (propofol), p = 0.118; higher diastolic pressure and MAP at induction with thiopental

Notes: Values are those reported by the authors; designs and populations differ, and no pooled or head-to-head comparison is intended. CT, computed tomography; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; ICP, intracranial pressure; CPP, cerebral perfusion pressure; MAP, mean arterial pressure; OR, odds ratio; TBI, traumatic brain injury.

Current guidelines and a Cochrane review give high-dose barbiturates only a second-line role in refractory intracranial hypertension, because ICP falls without a clear gain in outcome and hypotension is common.^{16,32,33} Ketamine, long avoided for fear of raising ICP, did not increase ICP significantly in a recent meta-analysis, although hypotension episodes were more frequent,³⁵ and propofol remains a common alternative; this case did not compare agents. Pediatric guidelines propose a lower CPP

target (40–50 mmHg, depending on age and autoregulation) than the adult value of 60 mmHg,⁴⁷ and perfusion-pressure targets after traumatic brain injury remain under reappraisal.¹⁵ The CPP of 58 mmHg after induction in our patient lay just under the adult goal, and all CPP values are estimates.

4.3. Pathophysiological or mechanistic interpretation

Thiopental lowers the cerebral metabolic rate and, through coupled vasoconstriction, cerebral blood volume,^{26,28} which may have contributed to brain relaxation. None of this was measured here: there was no EEG, no measurement of cerebral blood flow or metabolism, and no

ICP monitor. The fall in estimated ICP from 28 to 12–14 mmHg followed decompression and cannot be attributed to the drug. At induction MAP fell by 17.3% and estimated CPP to 58 mmHg, so thiopental was not hemodynamically neutral even at 4 mg/kg after fentanyl. Figure 5 sets out the proposed cascade as a conceptual schematic and not as patient data.

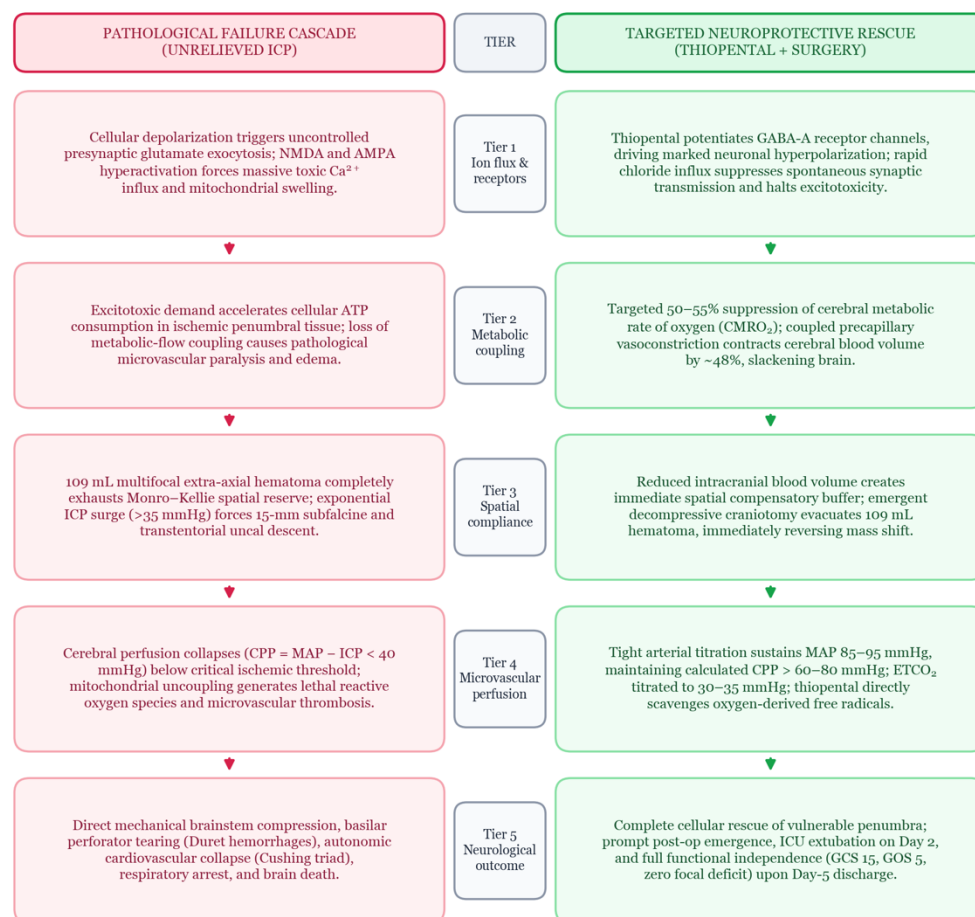


Figure 5. Conceptual comparison of the failure cascade of unrelieved intracranial hypertension and the proposed neuroprotective rescue sequence. ICP, intracranial pressure; CMRO₂, cerebral metabolic rate of oxygen; CPP, cerebral perfusion pressure; GOS, Glasgow Outcome Scale.

4.4. Implications for practice

The elements that can be transferred to similar cases are a slow, weight-based induction after an opioid, invasive pressure monitoring, explicit MAP and ETCo₂ targets, exclusion of nitrous oxide when intracranial air is present, normothermia and euolemia,⁴⁸ and one anesthetic for neurosurgical and reconstructive procedures in a stable patient, which avoided a second anesthetic. The choice of induction agent should follow the hemodynamic state of the patient and local experience.

4.5. Strengths

The report gives a complete timed record at ten milestones, weight-based doses, clear reasoning for avoiding nitrous oxide, and a day-by-day account of recovery.

4.6. Limitations

This is one patient. ICP and CPP were estimated; invasive monitoring is the reference, and a reliable non-invasive continuous method is not yet available.⁴⁹ Transcranial Doppler estimates⁵⁰ were not used, no depth-of-anesthesia or cerebral monitoring was applied, and decompressive surgery was performed at the same time, so the

contribution of thiopental cannot be separated. No postoperative imaging, hemoglobin or osmotherapy data are given, follow-up ended at ICU discharge on day 5, and the GOS is normally assigned months after injury.

4.7. Recommendations for future work

Comparing induction agents in traumatic epidural hematoma needs a prospective registry or randomized design with ICP or non-invasive ICP monitoring, processed EEG, defined CPP targets and outcome assessed at 3 to 6 months. Cases such as this one are best reported with relative time and the CARE checklist.

5. Conclusion

In an adolescent with multifocal epidural hematomas, pneumocephalus and impending herniation, a titrated thiopental-based induction with sevoflurane, arterial pressure and ETCo₂ targets and no nitrous oxide was followed by stable hemodynamics, an estimated CPP of 58–80 mmHg and a good recovery by day 5. The report supports the feasibility of this approach. One case cannot show that thiopental protected the brain, because ICP, cerebral metabolism and long-term outcome were not

measured and the hematoma was evacuated at the same time.

6. Declarations

Ethical approval and consent to participate. This case report was approved by CMHC Research Center Ethics Committee, Indonesia, under reference number 2025/4109.

Consent for publication. Written informed consent for publication of this case and the accompanying data was obtained from the patient's legal guardians (parents).

Availability of data and materials. The datasets and physiological records generated during this study are available from the corresponding author on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. S.M.M.S.: Conceptualization; Investigation; Data curation; Writing – original draft. B.H.L.: Methodology; Supervision; Writing – review & editing. E.N.: Methodology; Supervision; Writing – review & editing. D.A.S.: Investigation; Formal analysis; Writing – review & editing. All authors approved the final manuscript.

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Conflict of interest (competing interests) statement. The authors declare that they have no competing interests.

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