

ISSN Print: 1813-176x
ISSN Online: 1994-4624

International Journal of Molecular Medicine and Advance Sciences
https://ijmmas.com/

Research Article

Impact of Anesthetic Management and Timing of Decompressive Craniectomy on Perioperative Outcomes, Survival, and recovery in Patients With Severe brain trauma: A Prospective Cohort Study.

Dr. Neeta Devi[1*], Dr. Ali Akram Khan[2], Dr. Hafsa Khan[3], Dr. Aisha[4], Dr. Areeba Almas Siddique[5], Dr. Muhammad Ibrahim[6].

[1]MBBS FCPS (Anaesthesiology) Liaquat University of Medical and Health Sciences, Jamshoro (MBBS) Dow University of Health Sciences (FCPS; CPSP) Senior Registrar (Anaesthesiology), Gambat Medical College, PAQSJIMS.
[2]MBBS FCPS (Anaesthesiology) Senior Registrar, Dow University of Health Sciences
[3]MBBS Sir Syed College of Medical Sciences for Girls, Karachi
[4]MBBS FCPS (Anaesthesiology) Dow University of Health Sciences
[5]MBBS Jinnah Sindh Medical University
[6]MBBS FCPS (Anaesthesiology) Shaheed Mohtarma Benazir Bhutto Medical University, Larkana (MBBS) Senior Registrar, Karachi Metropolitan University.

Correspondence:
Dr. Neeta Devi.

Received: 30-10-2026
Revised: 11-11-2026
Accepted: 23-11-2026
Published: 24-11-2026

DOI: 10.61336/ijmmas.22-04-36

Abstract: ***Background: Objective:*** To assess associations of anesthetic management and decision-to-incision timing with perioperative outcomes, 180-day survival and functional recovery after secondary decompressive craniectomy for severe traumatic brain injury. ***Study design:*** Longitudinal cohort analysis. ***Place and duration:*** The regional context was tertiary neurotrauma care in Sindh. The study period was 20 November 2025 to 20 February 2026, with 180-day follow-up through 20 August 2026. ***Methodology:*** The analysis included 240 adult records with ages ranging from 18 to 65 years. Decision-to-incision timing was classified as ≤6 hours or >6–12 hours. Intravenous and volatile maintenance techniques were evaluated using descriptive statistics and multivariable logistic regression for 180-day mortality. Adjustment included age, Glasgow Coma Scale score, pupil response and preoperative hypotension. ***Results:*** The earlier and later timing groups contained 125 and 115 records. Mortality at 180 days was 32/125 (25.6%) versus 37/115 (32.2%). The adjusted odds ratio for later timing was 1.42 (95% CI 0.75–2.69). The odds ratio for intravenous versus volatile maintenance was 1.35 (95% CI 0.70–2.60). Favorable functional outcome occurred in 88/233 (37.8%) records with an available assessment. ***Conclusion:*** The analysis did not establish a difference in 180-day mortality by maintenance technique or timing category. Wide confidence intervals preclude equivalence claims. Survival and disability should be assessed together when evaluating perioperative care.

Keywords: Traumatic brain injury; Decompressive craniectomy; Anesthesia; Hypotension; Survival; Glasgow Outcome Scale; Sindh.

Copyright @ 2026: This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

INTRODUCTION

Severe traumatic brain injury requires protection of the injured brain while the team treats intracranial hypertension and systemic instability. Induction, ventilation, fluid administration and blood pressure control can alter cerebral perfusion during an operation. Established guidance therefore places physiological control alongside intracranial pressure management [1]. Decompressive craniectomy creates space for a swollen brain but its ability to lower pressure does not ensure an acceptable functional outcome. The updated surgical recommendations distinguish the clinical circumstances of early pressure elevation from those of more resistant intracranial hypertension [2]. These distinctions matter when studying the timing of surgery in an observational cohort.

The major trials answer different surgical questions. Rescue decompression for sustained refractory intracranial hypertension reduced mortality relative to continued medical care but also increased survival with severe disability [3]. In contrast, a trial of decompression at an earlier stage of refractory pressure elevation did not show a survival advantage and found worse six-month functional outcomes [4]. Neither comparison establishes that a patient should wait after an urgent surgical decision. An injury-to-operation interval, a duration of treatment-resistant pressure and an administrative delay after a decision represent different processes. Combining them into a single early-versus-late variable can misidentify both the exposure and the clinical implication.

Anesthetic management is similarly broader than the maintenance agent. A short period of hypotension, bleeding around decompression or inadequate ventilation may change the physiological risk independently of whether a volatile or intravenous technique is used. Perioperative reviews emphasize preventing secondary injury and maintaining cerebral perfusion [5]. A randomised study of ketofol versus propofol found differences in haemodynamic stability without a corresponding difference in its measured functional outcomes [6]. Thus an intervention can improve a physiological endpoint without establishing better survival or recovery. A cohort needs to capture these outcomes separately and record the circumstances that influenced treatment selection.

The question is relevant to Sindh, where neurotrauma services receive patients with varying referral histories and access to specialist care. A prospective Karachi cohort documented both mortality and functional outcomes after decompression [7]. The published national experience informs regional study design but cannot supply contemporary performance estimates for other hospitals. The present analysis examined anesthetic technique and decision-to-incision timing in a cohort restricted to secondary decompression. Its objectives were to describe perioperative outcomes, compare survival and functional recovery across timing categories and assess whether associations persisted after adjustment for baseline severity.

MATERIALS AND METHODS

Study design and setting

The analysis used a longitudinal postoperative cohort structure. The regional clinical context was tertiary neurosurgery and intensive care in Sindh. Relevant services include the Shaheed Mohtarma Benazir Bhutto Institute of Trauma in Karachi and Liaquat University Hospital in Hyderabad/Jamshoro [8] [9]. The study period was 20 November 2025 to 20 February 2026. Follow-up extended to 180 days after the index operation and the completion date was 20 August 2026. The postoperative observation window separated early mortality from later survival and functional recovery.

Eligibility and time origin

Eligibility specifications were adults aged 18–65 years with blunt severe traumatic brain injury, a post-resuscitation Glasgow Coma Scale (GCS) score of 3–8 and secondary decompression for intracranial hypertension refractory to staged medical management. Intracranial pressure exceeded 25 mmHg after treatment escalation. Primary decompression during evacuation of an acute mass lesion was outside scope. Penetrating injury, children and people already declared brain dead were excluded from the target population. The analytical frame comprised 240 postoperative records. It did not include a nonsurgical comparator. Consequently, the analysis addressed prognosis after surgery rather than the comparative effectiveness of surgery and continued medical treatment.

The timing exposure was the interval from the decision for secondary decompression to skin incision. Earlier timing was defined as ≤6 hours and later timing as >6–12 hours. The six-hour boundary was an analytical classification and not a clinical recommendation. Decision time referred to the point at which surgical eligibility and the intention to operate were established. It was distinct from injury time, hospital arrival, theatre arrival and availability of an ICU bed. Survival time began at incision because the cohort was restricted to records reaching surgery. This time origin supports a postoperative prognosis analysis but does not estimate the effect of a timing policy beginning at eligibility.

Anesthetic exposures and physiology

Maintenance technique was classified as propofol-based total intravenous anesthesia or volatile-based anesthesia. Technique was an exposure category rather than a randomised assignment. Intracranial pressure and preoperative instability were considered when interpreting differences between categories. A specific volatile agent, concentration, induction dose or adjunct was not evaluated. Intraoperative hypotension was defined as systolic blood pressure below 90 mmHg for at least five consecutive minutes between induction and operating-room departure. Preoperative hypotension used the same pressure boundary before induction. This fixed definition supported consistent classification. It does not replace age-specific blood pressure goals or individualized cerebral perfusion targets.

Perioperative fields distinguished intracranial pressure before surgery from pressure at the designated 24-hour assessment. Blood loss was expressed in millilitres. Transfusion indicated receipt of at least one red-cell unit during surgery or the first 24 hours. Pneumonia was classified within the index admission. These variables were secondary descriptive outcomes. Detailed pressure traces, end-tidal carbon dioxide, arterial oxygenation, vasopressor dose and cumulative anesthetic exposure were outside the available variable set. These factors remain relevant to the interpretation of technique selection and should be included in a comprehensive perioperative assessment.

Outcomes and functional assessment

The primary endpoint was all-cause mortality by 180 postoperative days. Hospital mortality and mortality by day 30 were reported separately. Functional recovery used the eight-category Glasgow Outcome Scale–Extended (GOSE), for which structured assessment provides a reproducible approach [10]. Categories 1–4 were classified as unfavorable and 5–8 as favorable. Death was category 1 and was retained in functional analyses. The complete outcome distribution was also retained because a binary classification conceals differences between death, severe disability and greater independence. Known survival status did not imply availability of a complete functional assessment.

Seven survivors had a missing 180-day functional assessment. Analyses of function therefore used 233 records, including all deaths. Mortality analyses used all 240 records. Best-case and worst-case bounds assigned the seven missing functional

assessments to favorable or unfavorable outcome, respectively. ICU duration was summarized for the index admission with death ending the stay. It was treated descriptively because early mortality can shorten admission. Survival was administratively censored at day 180 with no earlier loss of vital status. Missing function did not censor a record from the mortality analysis.

Data management and statistical analysis

Baseline variables comprised age, sex, post-resuscitation GCS, pupil response, preoperative hypotension and intracranial pressure. Abnormal pupils meant at least one nonreactive pupil. Exposure fields covered decision-to-incision timing, maintenance technique and intraoperative hypotension. Outcome fields distinguished hospital death, day-30 death, day-180 death and functional recovery. Data checks assessed agreement between vital status, the postoperative day of death and GOSE category. Deaths were retained in every applicable outcome denominator. Functional missingness was handled separately from vital status. Reporting followed the structure of the STROBE statement for cohort studies [11].

Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Categorical outcomes were reported as counts with explicit denominators. Baseline-adjusted logistic regression for 180-day mortality included age per ten-year increase, GCS per point, abnormal pupils, preoperative hypotension, later timing and intravenous maintenance. A second exploratory regression added intraoperative hypotension. This exposure could lie on a pathway between anesthetic management and outcome, so its inclusion did not provide a better estimate of the total treatment effect. Odds ratios were reported with 95% Wald confidence intervals and two-sided p-values. Standard errors used a sandwich covariance estimate with a small-sample degrees-of-freedom multiplier.

Python with NumPy and SciPy was used for maximum-likelihood estimation. Covariates were specified before estimation rather than selected through univariable significance testing. Group contrasts were descriptive. Confidence intervals were used to assess precision and compatibility with a range of associations. No proportional-hazards regression, interaction test, external prediction validation or centre effect was fitted. The survival display included numbers alive at selected postoperative times. The maintenance-technique comparison was adjusted only for the listed baseline variables and could not resolve selection by unmeasured injury pattern, clinician judgement or treatment intensity. A confidence interval crossing the null was not interpreted as evidence of equivalence.

RESULTS

The cohort comprised 240 records, with 125 in the earlier timing group and 115 in the later group (Figure 1). Mean age was 35.1 ± 10.0 years and 175/240 (72.9%) were male. Median GCS was 6.0 (IQR 5.0–7.0). Abnormal pupils were present in 57/240 (23.8%) records and preoperative hypotension in 37/240 (15.4%). Table 1 presents baseline characteristics. Similarity in selected variables did not establish exchangeability between the exposure groups.

Median decision-to-incision time was 2.8 hours (IQR 1.8–4.3) in the earlier group and 8.4 hours (IQR 7.5–9.9) in the later group. Preoperative hypotension was more frequent in the later group: 25/115 (21.7%) versus 12/125 (9.6%). Intravenous maintenance was classified in 135/240 (56.3%) records. Intraoperative hypotension occurred in 17/125 (13.6%) earlier records and 25/115 (21.7%) later records. The 24-hour intracranial pressure summary was restricted to records surviving beyond postoperative day 1 (Table 2).

Hospital mortality was 57/240 (23.8%). Mortality increased to 58/240 (24.2%) by day 30 and 69/240 (28.8%) by day 180. At 180 days mortality was 32/125 (25.6%) in the earlier group and 37/115 (32.2%) in the later group. Figure 2 presents postoperative survival and the functional distribution. Of 233 records with a GOSE assessment, 145 (62.2%) had an unfavorable outcome and 88 (37.8%) had a favorable outcome. Favorable outcomes were 49/121 (40.5%) versus 39/112 (34.8%).

In baseline-adjusted logistic regression, later timing had an odds ratio of 1.42 (95% CI 0.75–2.69; p=0.283). Intravenous versus volatile maintenance had an odds ratio of 1.35 (95% CI 0.70–2.60; p=0.368). Older age and lower GCS were associated with greater mortality. The age odds ratio per ten years was 2.13 (95% CI 1.44–3.14) and the GCS odds ratio per point was 0.72 (95% CI 0.59–0.89). Table 3 presents both regression specifications. Adding intraoperative hypotension did not materially change the timing or maintenance estimates.

All deaths were included in the GOSE denominator. Functional missingness affected four earlier-group survivors and three later-group survivors. Across the full cohort the unfavorable assignment of all missing assessments yielded 88/240 (36.7%) favorable outcomes. Assigning all seven as favorable yielded 95/240 (39.6%). These bounds are not a confidence interval. Blood loss, transfusion, pneumonia and ICU duration remained secondary descriptive outcomes (Table 2).

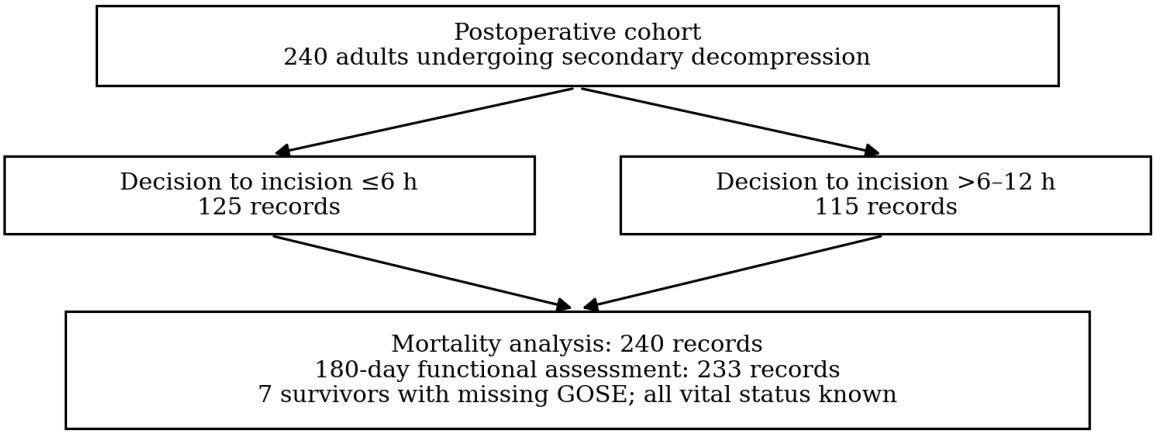


Figure 1. Cohort structure and outcome denominators

Table 1. Baseline characteristics and exposures

Characteristic	All records (n=240)	≤6 hours (n=125)	>6–12 hours (n=115)
Age, years	35.1 ± 10.0	35.6 ± 9.7	34.5 ± 10.4
Male sex	175/240 (72.9%)	96/125 (76.8%)	79/115 (68.7%)
GCS score	6.0 (5.0–7.0)	6.0 (5.0–7.0)	6.0 (5.0–7.0)
Abnormal pupil response	57/240 (23.8%)	29/125 (23.2%)	28/115 (24.3%)
Preoperative hypotension	37/240 (15.4%)	12/125 (9.6%)	25/115 (21.7%)
Preoperative ICP, mmHg	32.3 ± 4.8	31.8 ± 4.8	32.8 ± 4.8
Decision-to-incision time, h	5.7 (2.7–8.4)	2.8 (1.8–4.3)	8.4 (7.5–9.9)
Intravenous maintenance	135/240 (56.3%)	70/125 (56.0%)	65/115 (56.5%)

Entries are mean ± SD, median (IQR) or n/N (%). GCS: Glasgow Coma Scale. ICP: intracranial pressure. Timing is measured from a decision for secondary decompression, not from injury.

Table 2. Perioperative and follow-up outcomes

Outcome	All records	≤6 hours	>6–12 hours
Intraoperative hypotension	42/240 (17.5%)	17/125 (13.6%)	25/115 (21.7%)
Blood loss, mL	701.0 ± 244.2	682.8 ± 233.9	720.7 ± 254.5
Red-cell transfusion	58/240 (24.2%)	33/125 (26.4%)	25/115 (21.7%)
Postoperative ICP, mmHg	17.3 ± 4.0; n=214	17.6 ± 4.1; n=114	17.0 ± 3.8; n=100
Pneumonia during admission	55/240 (22.9%)	32/125 (25.6%)	23/115 (20.0%)
ICU duration, days	12.0 (7.0–17.2)	13.0 (7.0–18.0)	12.0 (6.0–17.0)
Hospital mortality	57/240 (23.8%)	28/125 (22.4%)	29/115 (25.2%)
30-day mortality	58/240 (24.2%)	28/125 (22.4%)	30/115 (26.1%)
180-day mortality	69/240 (28.8%)	32/125 (25.6%)	37/115 (32.2%)
Favorable GOSE (5–8)	88/233 (37.8%)	49/121 (40.5%)	39/112 (34.8%)
Unfavorable GOSE (1–4)	145/233 (62.2%)	72/121 (59.5%)	73/112 (65.2%)
Missing functional assessment	7	4	3

Death is GOSE 1 and is included in unfavorable outcomes. Functional percentages use records with an assessment. ICP at 24 hours is restricted to records surviving beyond day 1; n denotes its available denominator. ICU duration includes fatal admissions. A shorter stay is not interpreted as a favorable outcome.

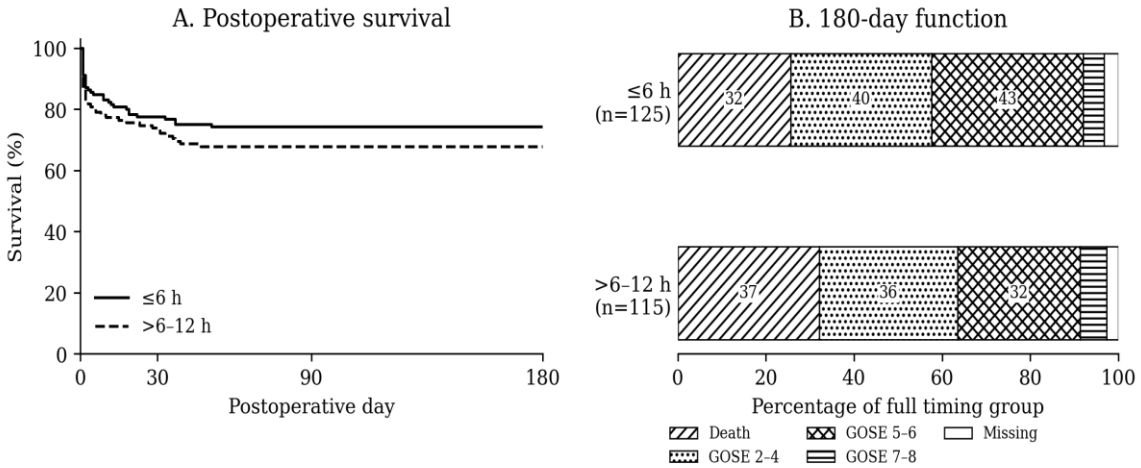


Figure 2. Survival and functional outcomes by timing

The survival panel starts at incision and has only day-180 administrative censoring. Numbers alive at days 0, 30, 90 and 180 are shown below. Functional bars include missing assessments in the full group denominator. Bar labels show selected counts. Numbers alive at days 0 / 30 / 90 / 180: ≤6 h: 125 / 97 / 93 / 93; >6–12 h: 115 / 85 / 78 / 78.

Table 3. Adjusted associations with 180-day mortality

Predictor	Baseline-adjusted OR (95% CI)	p-value	Extended OR (95% CI)	p-value
Age, per 10 years	2.13 (1.44–3.14)	<0.001	2.13 (1.45–3.14)	<0.001
GCS, per point	0.72 (0.59–0.89)	0.002	0.72 (0.59–0.89)	0.002
Abnormal pupils vs reactive	1.60 (0.76–3.34)	0.213	1.60 (0.76–3.37)	0.212
Preoperative hypotension	1.97 (0.76–5.11)	0.161	2.01 (0.76–5.33)	0.161
Timing >6–12 h vs ≤6 h	1.42 (0.75–2.69)	0.283	1.44 (0.75–2.73)	0.270
Intravenous vs volatile	1.35 (0.70–2.60)	0.368	1.36 (0.70–2.62)	0.364
Intraoperative hypotension	Not included	—	0.89 (0.37–2.10)	0.783

OR: odds ratio. CI: confidence interval. Both regressions use all 240 records and 69 deaths. The extended regression adds a potentially intermediate exposure and is exploratory.

DISCUSSION

The analysis assessed anesthetic management, decision-to-incision timing and outcomes after secondary decompression. Crude six-month mortality was higher in the later group but the adjusted interval included both little difference and a clinically important adverse association. The maintenance-technique interval was also imprecise. These findings do not establish equivalence between strategies. A selected postoperative cohort can describe prognosis and identify imbalance in measured physiology. It cannot establish a safe waiting period or identify a superior anesthetic technique without addressing treatment selection and the clinical processes preceding surgery.

National evidence provides the most relevant starting point for regional interpretation. The published Karachi prospective series reported 18 hospital deaths among 72 decompressed patients and favorable outcomes in 51.4% at three months [7]. Hospital mortality in the current analysis was similar in magnitude but the favorable functional proportion was lower at six months. Differences in outcome scale, follow-up, eligibility and primary versus secondary decompression prevent direct benchmarking. A more recent institutional report from Jinnah Postgraduate Medical Centre concerned decompression in patients with traumatic mass lesions [12]. That experience informs the regional context but its operation addresses a different decision from secondary decompression after medical treatment fails.

International trial evidence explains why timing needs a precise origin. The rescue-decompression trial compared surgery with continued medical management for persistent refractory intracranial hypertension. Six-month surgical mortality was 26.9% versus 48.9% with medical management [3]. The associated disability distribution makes survival alone an incomplete description of benefit. The early-decompression trial studied a different pressure threshold and earlier treatment stage. Its twelve-month report did not establish improvement in survival or neurological outcome [13]. These trials do not validate a six-hour decision-to-incision boundary. The landmark-trial literature instead supports distinguishing indication and treatment stage before comparing outcomes [14].

The distinction between primary and secondary decompression also limits transfer of evidence. A large randomised comparison during evacuation of acute subdural haematoma found similar disability and quality-of-life outcomes with craniotomy and craniectomy, with different subsequent surgical and wound burdens [15]. It evaluated whether the bone flap should be replaced during a mass-lesion operation. The present analysis was restricted to secondary decompression. Combining these groups in a registry may associate timing with outcome because rapid mass-lesion evacuation and later rescue surgery represent different injury trajectories. Eligibility should therefore be classified before assigning either a timing category or an outcome comparison.

Anesthetic technique requires the same attention to indication and comparator. The ketofol trial showed better haemodynamic stability than propofol alone but similar measured GOSE outcomes. A newer trial of ketamine and lidocaine adjuncts to propofol-based anesthesia found reduced propofol requirements with ketamine and no detectable difference in its functional outcome measure [16]. These are regimen-specific physiological findings. They do not establish that intravenous maintenance is superior to volatile maintenance in all severe brain trauma. In the current analysis the adjusted interval remained wide. Doses, adjuncts and physiological responses are therefore essential to interpreting anesthetic exposure beyond the maintenance label.

Perioperative hypotension remains clinically relevant despite the imprecision of its additional regression coefficient. An observational study of postintubation hypotension in severe injury examined the association between an airway-related physiological insult and survival [17]. A scoping review of decompression identified heterogeneous blood pressure variability measures and limited evidence linking them to long-term outcomes [18]. A small fluid-management trial reported physiological differences without a clear difference in ICU or hospital duration [19]. These reports support detailed measurement of the haemodynamic course. A threshold event alone cannot summarize the duration, depth and causes of perfusion compromise.

Intracranial pressure is an important intermediate endpoint but cannot replace assessment of recovery. A trial of continuous hypertonic saline did not demonstrate better six-month neurological outcome with that strategy [20]. A trial adding brain tissue oxygen monitoring to pressure monitoring similarly did not establish superiority for its neurological endpoint [21]. These findings concern defined interventions and do not suggest that monitoring or treatment of dangerous physiology is unnecessary. A lower postoperative pressure cannot by itself establish that a timing category or anesthetic strategy improves functional survival. Physiological and patient-centred outcomes should therefore remain separate.

Blood loss and transfusion are potential co-interventions. Red-cell exposure was described separately because it occurs after the timing and anesthetic decisions. A traumatic brain injury transfusion trial did not establish a significant reduction in its unfavorable-outcome endpoint with a liberal strategy [22]. A separate acute-brain-injury trial found fewer unfavorable outcomes with liberal transfusion but included diagnoses beyond traumatic injury and used a different GOSE boundary [23]. The population, threshold and endpoint determine how those results should be interpreted. The reason and timing of transfusion are needed to distinguish haemorrhage-related risk from the effect of the transfusion decision.

Long-term function requires a plan extending beyond discharge. Continued improvement after six months was documented in follow-up of the rescue-decompression trial [24]. Structured interviews and questionnaires can also differ in their capture of global functional status [25]. For Sindh services, feasible follow-up could combine clinic assessment with a standardised telephone interview for people unable to travel. The informant and assistance required at home, work and in the community should be documented. The missing-outcome bounds in this analysis show why assessed survivors alone cannot represent overall recovery. Death and missing assessments need explicit placement in the outcome distribution.

Age, consciousness and pupil response are established components of prognostic assessment. External validation shows that tools based on these characteristics require evaluation in their intended population [26]. Contemporary neuroprognostication guidance also emphasizes uncertainty and the limitations of isolated predictors [27]. Regression coefficients from this analysis cannot determine an individual prognosis or treatment eligibility. Regional assessment requires reliable baseline examinations, imaging, extracranial injury severity, treatment-limitation decisions and access to rehabilitation. A multidisciplinary record should connect emergency stabilization, anesthesia, neurosurgery and ICU care with a documented functional follow-up plan.

Recent reviews emphasize heterogeneity in functional outcome assessment [28]. Surrogate perceptions add a dimension beyond the scale score [29]. GOS-based and GOSE-based trial estimates need not be interchangeable [30]. A regional cohort should therefore retain the full outcome distribution, prespecify its favorable boundary and distinguish the patient's report from a surrogate assessment.

Several limitations affect interpretation. The analysis was restricted to adults reaching secondary decompression and excluded those dying before surgery or remaining on medical management. Starting survival at incision cannot resolve selection and guarantee-time bias from the surgical decision. The timing boundary was an analytical cut-point and exposure categories were simplified. Imaging severity, extracranial injury burden, treatment withdrawal, detailed anesthetic dosing and centre-level resources were outside the covariate set. Seven functional assessments were missing and follow-up ended at 180 days. The

confidence intervals were wide for both principal exposures. These limitations preclude causal claims, equivalence conclusions and direct comparison of institutional performance.

CONCLUSION

Anesthetic maintenance technique and decision-to-incision category did not show precise adjusted associations with 180-day mortality. The estimates do not support equivalence or a permissible surgical waiting interval. Assessment of secondary decompression should distinguish survival from functional recovery and primary mass-lesion surgery from rescue surgery. Future Sindh cohorts should use clearly defined indications and time origins, detailed physiological records and structured follow-up to support more reliable treatment comparisons.

Declarations

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Data availability: Summary data and analytical definitions are presented in the manuscript.

REFERENCES

1. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ, et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery 2017;80(1):6-15. doi: https://doi.org/10.1227/NEU.0000000000001432. PMID: 27654000. Available from: https://pubmed.ncbi.nlm.nih.gov/27654000/
2. Hawryluk GWJ, Rubiano AM, Totten AM, O'Reilly C, Ullman JS, Bratton SL, et al. Guidelines for the Management of Severe Traumatic Brain Injury: 2020 Update of the Decompressive Craniectomy Recommendations. Neurosurgery 2020;87(3):427-434. doi: https://doi.org/10.1093/neuros/nyaa278. PMID: 32761068. Available from: https://pubmed.ncbi.nlm.nih.gov/32761068/
3. Hutchinson PJ, Kolias AG, Timofeev IS, Corteen EA, Czosnyka M, Timothy J, et al. Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension. N Engl J Med 2016;375(12):1119-30. doi: https://doi.org/10.1056/NEJMoa1605215. PMID: 27602507. Available from: https://pubmed.ncbi.nlm.nih.gov/27602507/
4. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 2011;364(16):1493-502. doi: https://doi.org/10.1056/NEJMoa1102077. PMID: 21434843. Available from: https://pubmed.ncbi.nlm.nih.gov/21434843/
5. Kim H. Anesthetic management of the traumatic brain injury patients undergoing non-neurosurgery. Anesth Pain Med (Seoul) 2023;18(2):104-113. doi: https://doi.org/10.17085/apm.23017. PMID: 37183278. Available from: https://pubmed.ncbi.nlm.nih.gov/37183278/
6. Maheswari N, Panda NB, Mahajan S, Luthra A, Pattnaik S, Bhatia N, et al. Ketofol as an Anesthetic Agent in Patients With Isolated Moderate to Severe Traumatic Brain Injury: A Prospective, Randomized Double-blind Controlled Trial. J Neurosurg Anesthesiol 2023;35(1):49-55. doi: https://doi.org/10.1097/ANA.0000000000000774. PMID: 36745167. Available from: https://pubmed.ncbi.nlm.nih.gov/36745167/
7. Laghari AA, Bari ME, Waqas M, Ahmed SI, Nathani KR, Moazzam W. Outcome of Decompressive Craniectomy in Traumatic Closed Head Injury. Asian J Neurosurg 2018;13(4):1053-1056. doi: https://doi.org/10.4103/ajns.AJNS_195_17. PMID: 30459866. Available from: https://pubmed.ncbi.nlm.nih.gov/30459866/
8. Shaheed Mohtarma Benazir Bhutto Institute of Trauma. Department of Neurosurgery. Karachi: SMBBIT; cited 20 November 2026. Available from: https://smbbit.gos.pk/DepartmentDetails?id=14
9. Liaquat University Hospital. Department of Neurosurgery. Hyderabad/Jamshoro: LUH; cited 20 November 2026. Available from: https://luh.gos.pk/view_depart/43
10. Wilson JT, Pettigrew LE, Teasdale GM. Structured interviews for the Glasgow Outcome Scale and the extended Glasgow Outcome Scale: guidelines for their use. J Neurotrauma 1998;15(8):573-85. doi: https://doi.org/10.1089/neu.1998.15.573. PMID: 9726257. Available from: https://pubmed.ncbi.nlm.nih.gov/9726257/
11. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Prev Med 2007;45(4):247-51. doi: https://doi.org/10.1016/j.ypmed.2007.08.012. PMID: 17950122. Available from: https://pubmed.ncbi.nlm.nih.gov/17950122/
12. Kalhoro A, Hassan S, Bokhari I, Brohi GM, Ahmed F, Ahmed T. The role of decompressive craniectomy in traumatic brain injury: an institutional experience in a tertiary care hospital. Natl J Health Sci 2024;9(4):260-264. doi: https://doi.org/10.21089/njhs.94.0260.
13. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, Ponsford J, et al. Patient Outcomes at Twelve Months after Early Decompressive Craniectomy for Diffuse Traumatic Brain Injury in the Randomized DECRA Clinical Trial. J Neurotrauma 2020;37(5):810-816. doi: https://doi.org/10.1089/neu.2019.6869. PMID: 32027212. Available from: https://pubmed.ncbi.nlm.nih.gov/32027212/

14. Patel PP, Egodage T, Martin MJ. Decompressive craniectomy for traumatic brain injury: a review of recent landmark trials. Trauma Surg Acute Care Open 2025;10(Suppl 1):e001784. doi: https://doi.org/10.1136/tsaco-2025-001784. PMID: 40260232. Available from: https://pubmed.ncbi.nlm.nih.gov/40260232/
15. Hutchinson PJ, Adams H, Mohan M, Devi BI, Uff C, Hasan S, et al. Decompressive Craniectomy versus Craniotomy for Acute Subdural Hematoma. N Engl J Med 2023;388(24):2219-2229. doi: https://doi.org/10.1056/NEJMoa2214172. PMID: 37092792. Available from: https://pubmed.ncbi.nlm.nih.gov/37092792/
16. Ramadurai R, Swaminathan S, Bidkar PU, Seshan JR, Anbazhagan S, Chinnarasan V. Comparison of subanaesthetic ketamine and lidocaine as adjuvants to propofol-based total intravenous anesthesia on propofol requirement during emergency decompressive craniectomy for traumatic brain injury: a prospective, double-blind, three-arm randomized controlled trial. J Clin Neurosci 2026;154:112284. doi: https://doi.org/10.1016/j.jocn.2026.112284. PMID: 42710328. Available from: https://pubmed.ncbi.nlm.nih.gov/42710328/
17. Price J, Lachowycz K, Major R, McLachlan S, Keeliher C, Finbow B, et al. Prehospital Postintubation Hypotension and Survival in Severe Traumatic Brain Injury. JAMA Netw Open 2025;8(11):e2544057. doi: https://doi.org/10.1001/jamanetworkopen.2025.44057. PMID: 41264271. Available from: https://pubmed.ncbi.nlm.nih.gov/41264271/
18. Bhatt O, Rizk AA, Kumar N, Goyal K, Bindra A, Singh GP, et al. Hemodynamic variability in patients undergoing decompressive craniotomy or craniectomy: A scoping review. J Clin Neurosci 2026;148:111981. doi: https://doi.org/10.1016/j.jocn.2026.111981. PMID: 41819737. Available from: https://pubmed.ncbi.nlm.nih.gov/41819737/
19. Jonna D, Kaloria N, Khurana BJK, Hazarika A, Jain K, Bhatia N, et al. Comparison of Intraoperative Fluid by Goal-Directed and Conventional Fluid Administration in Patients Undergoing Early Decompressive Craniectomy: A Prospective Randomized Controlled Trial. Asian J Neurosurg 2026;21(3):484-492. doi: https://doi.org/10.1055/s-0046-1817812. PMID: 42535245. Available from: https://pubmed.ncbi.nlm.nih.gov/42535245/
20. Roquilly A, Moyer JD, Huet O, Lasocki S, Cohen B, Dahyot-Fizelier C, et al. Effect of Continuous Infusion of Hypertonic Saline vs Standard Care on 6-Month Neurological Outcomes in Patients With Traumatic Brain Injury: The COBI Randomized Clinical Trial. JAMA 2021;325(20):2056-2066. doi: https://doi.org/10.1001/jama.2021.5561. PMID: 34032829. Available from: https://pubmed.ncbi.nlm.nih.gov/34032829/
21. Payen JF, Launey Y, Chabanne R, Gay S, Francony G, Gergele L, et al. Intracranial pressure monitoring with and without brain tissue oxygen pressure monitoring for severe traumatic brain injury in France (OXY-TC): an open-label, randomised controlled superiority trial. Lancet Neurol 2023;22(11):1005-1014. doi: https://doi.org/10.1016/S1474-4422(23)00290-9. PMID: 37863590. Available from: https://pubmed.ncbi.nlm.nih.gov/37863590/
22. Turgeon AF, Fergusson DA, Clayton L, Patton MP, Neveu X, Walsh TS, et al. Liberal or Restrictive Transfusion Strategy in Patients with Traumatic Brain Injury. N Engl J Med 2024;391(8):722-735. doi: https://doi.org/10.1056/NEJMoa2404360. PMID: 38869931. Available from: https://pubmed.ncbi.nlm.nih.gov/38869931/
23. Taccone FS, Rynkowski CB, Møller K, Lormans P, Quintana-Díaz M, Caricato A, et al. Restrictive vs Liberal Transfusion Strategy in Patients With Acute Brain Injury: The TRAIN Randomized Clinical Trial. JAMA 2024;332(19):1623-1633. doi: https://doi.org/10.1001/jama.2024.20424. PMID: 39382241. Available from: https://pubmed.ncbi.nlm.nih.gov/39382241/
24. Kolias AG, Adams H, Timofeev IS, Corteen EA, Hossain I, Czosnyka M, et al. Evaluation of Outcomes Among Patients With Traumatic Intracranial Hypertension Treated With Decompressive Craniectomy vs Standard Medical Care at 24 Months: A Secondary Analysis of the RESCUEicp Randomized Clinical Trial. JAMA Neurol 2022;79(7):664-671. doi: https://doi.org/10.1001/jamaneurol.2022.1070. PMID: 35666526. Available from: https://pubmed.ncbi.nlm.nih.gov/35666526/
25. Horton L, Rhodes J, Menon DK, Maas AIR, Wilson L, Collaborative European NeuroTrauma Effectiveness Research in TBI (CENTER-TBI) Participants and Investigators. Questionnaires vs Interviews for the Assessment of Global Functional Outcomes After Traumatic Brain Injury. JAMA Netw Open 2021;4(11):e2134121. doi: https://doi.org/10.1001/jamanetworkopen.2021.34121. PMID: 34762111. Available from: https://pubmed.ncbi.nlm.nih.gov/34762111/
26. Dijkland SA, Helmrich IRAR, Nieboer D, van der Jagt M, Dippel DWJ, Menon DK, et al. Outcome Prediction after Moderate and Severe Traumatic Brain Injury: External Validation of Two Established Prognostic Models in 1742 European Patients. J Neurotrauma 2021;38(10):1377-1388. doi: https://doi.org/10.1089/neu.2020.7300. PMID: 33161840. Available from: https://pubmed.ncbi.nlm.nih.gov/33161840/
27. Muehlschlegel S, Rajajee V, Wartenberg KE, Alexander SA, Busl KM, Creutzfeldt CJ, et al. Guidelines for Neuroprognostication in Critically Ill Adults with Moderate-Severe Traumatic Brain Injury. Neurocrit Care 2024;40(2):448-476. doi: https://doi.org/10.1007/s12028-023-01902-2. PMID: 38366277. Available from: https://pubmed.ncbi.nlm.nih.gov/38366277/
28. Iaquaniello C, Scordo E, Robba C. Prediction of functional outcome after traumatic brain injury: a narrative review. Curr Opin Crit Care 2025;31(5):591-598. doi: https://doi.org/10.1097/MCC.0000000000001290. PMID: 40522960. Available from: https://pubmed.ncbi.nlm.nih.gov/40522960/

29. Maiga AW, Cook M, Nordness MF, Gao Y, Rakhit S, Rivera EL, et al. Surrogate Perception of Disability after Hospitalization for Traumatic Brain Injury. J Am Coll Surg 2024;238(4):589-597. doi: https://doi.org/10.1097/XCS.0000000000000960. PMID: 38214447. Available from: https://pubmed.ncbi.nlm.nih.gov/38214447/
30. Raccagni NG, Dotti M, Lightfoot YA, Loda R, Baker MS, Heihre JM, et al. A Meta-epidemiologic Analysis of Differences in Treatment-Effect Estimates According to Outcome Scale (GOS vs. GOSE) in Moderate-to-Severe Traumatic Brain Injury Trials. Neurocrit Care 2026. doi: https://doi.org/10.1007/s12028-026-02634-9. PMID: 42625005. Available from: https://pubmed.ncbi.nlm.nih.gov/42625005/.