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Neuroprotection at the Bedside: A Sequenced, Tier-Based Operational Strategy for Severe Acute Brain Injury

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ABSTRACT: Guidance for severe acute brain injury is distributed across a dozen documents written for different purposes and updated on different cycles. The clinician at the bedside at 03:00 needs one sequence, in order, with numbers.

This review assembles current evidence into a single operational strategy organised the way care is actually delivered: what to do in the first hour, what runs continuously for every patient regardless of intracranial pressure, what to monitor and when to place it, what the targets are, how to escalate through tiers when those targets fail, how to manage the parallel problems that arrive alongside intracranial hypertension, where non-traumatic injury demands a different answer, how to come back down, and when to stop.

Recommendations are drawn from the Brain Trauma Foundation guidelines (fourth edition with the 2020 decompressive craniectomy update; a fifth edition remains in preparation and unpublished), the Seattle International Severe Traumatic Brain Injury

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Consensus Conference tiered algorithms for management with intracranial pressure monitoring and with combined pressure and brain oxygen monitoring, the CREVICE protocol for settings without invasive monitoring, the 2026 Brain Trauma Foundation penetrating injury guidelines, and current pain, sedation, seizure, transfusion, and ventilation evidence. Every threshold is given with its evidentiary strength, and areas of genuine uncertainty — transfusion thresholds, brain tissue oxygen targets, decompressive craniectomy selection, autonomic prophylaxis — are marked as contested rather than resolved

1. SCOPE, AUDIENCE, AND HOW THIS DOCUMENT WAS DEVELOPED

Scope. Adults with severe acute brain injury requiring intensive care. The evidence base is predominantly traumatic; Section 11 states explicitly where non-traumatic injury diverges and where the recommendations here should not be transferred. Paediatric practice, spinal cord injury, and post-cardiac-arrest brain injury are outside scope.

Audience. Intensivists, neurologists, neurosurgeons, emergency physicians, and senior nursing staff delivering care in units with and without invasive neuromonitoring.

Development. This is a narrative synthesis, not a systematic review. Source guidelines and primary trials were identified by targeted searching of PubMed and of the websites of the issuing guideline bodies, supplemented by screening of reference lists; the literature was current to August 2026. Sources were selected for relevance to bedside decision-making rather than against predefined inclusion criteria, and no formal risk-of-bias appraisal was undertaken. The draft was circulated to all authors, who reviewed the clinical content, checked the cited thresholds against the primary sources, and approved the final version. No primary patient data were collected, and no protocol described in this document has been prospectively implemented or evaluated at the participating institution.

Three rules govern everything below.

Sequence beats intensity. Most preventable secondary injury comes from doing the right things in the wrong order — escalating sedation before treating pain, draining cerebrospinal fluid before checking head position, hyperventilating before checking whether the endotracheal tube tie is obstructing jugular outflow. Work down the list.

The floor is not optional. Section 4 applies to every patient with severe acute brain injury, at every hour, whether or not intracranial pressure is being monitored. Failures here account for more harm than failures at the top of the escalation ladder, and they are invisible because nothing alarms.

Every escalation needs an exit. Sections 7 and 10 pair. Nothing in Tier 2 or Tier 3 should be started without a stated condition for stopping it.

2. HOUR ZERO: THE FIRST SIXTY MINUTES

Table 1. Immediate priorities

#	Action	Target	Note
1	Airway and oxygenation	SpO_2 ≥94%; PaO_2 80–120 mmHg	A single episode of SpO_2 <90% is independently associated with worse outcome. Avoid sustained hyperoxia.[1]
2	Ventilation	$PaCO_2$ 35–40 mmHg	**No prophylactic hyperventilation**, particularly in the first 24 h. One of the few strong negative recommendations in the field.[1]
3	Blood pressure	SBP ≥110 mmHg (≥100 if aged 50–69)	Hypotension is the most damaging single physiological insult. Correct before anything else. **Trauma-specific — see Section 11 for intracerebral haemorrhage, where the target is the opposite.**[1]

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#	Action	Target	Note
4	Rapid neurological exam	GCS motor score, pupils, focal deficit	Perform before sedation and paralysis if at all possible — this exam anchors every subsequent decision.
5	Non-contrast CT head	Within 30 min of arrival for GCS ≤8	Repeat at 6 h, or immediately on any deterioration.
6	Neurosurgical referral	Immediate for any mass lesion	Evacuation of a surgical lesion outranks every medical measure in this document.
7	Reverse anticoagulation	Per agent and per local protocol	Do not wait for a repeat scan to start reversal.
8	Tranexamic acid	1 g over 10 min, then 1 g over 8 h, **within 3 h of injury**	Benefit concentrated in mild-to-moderate injury; no demonstrated benefit where GCS is 3 or both pupils are unreactive.[9]
9	Seizure prophylaxis	7 days	Reduces *early* post-traumatic seizures only. No effect on late seizures or outcome. Levetiracetam is widely used, with comparable efficacy to phenytoin and fewer interactions.[1,30] Not indicated in non-traumatic injury — see Section 11.
10	Temperature and glucose	Start correcting immediately	See Section 4.

What not to do in hour zero: corticosteroids (associated with increased mortality — an absolute contraindication[10]), prophylactic hyperventilation, prophylactic hypothermia, hypotonic fluids of any kind, and delaying reversal or surgery for further imaging.

3. PENETRATING INJURY: WHERE THE ANSWER DIFFERS

The second-edition penetrating traumatic brain injury guidelines were published in 2026 and are the first substantive update in more than two decades.[6] Two points are worth carrying to the bedside. First, the systematic literature search underlying them closed in August 2022, so they do not incorporate the transfusion or brain-oxygen literature discussed below; read them alongside, not instead of, the current trial evidence. Second, the recommendations on antibiotic prophylaxis, dural repair, and the management of retained fragments are specific to penetrating mechanism and are not inferable from blunt-injury practice. Where mechanism is penetrating, consult the source document directly.

4. THE FLOOR: CONTINUOUS MEASURES FOR EVERY PATIENT

These run from admission until the patient no longer has a brain at risk. They are the highest-yield and most frequently omitted part of neuroprotection.

Table 2. Mechanical and positional

Measure	Specification	Why
Head of bed	30°	Optimises the balance between venous drainage and cerebral perfusion pressure
Neck	Midline, neutral	Rotation obstructs jugular venous outflow and raises ICP within seconds
Cervical collar	Loosen or clear as early as safely possible	A tight collar is a common, entirely reversible cause of unexplained ICP elevation

Measure	Specification	Why
Endotracheal tube ties	Never circumferentially tight	Same mechanism. Check this before escalating any therapy
Hip flexion	<90°	Raises intra-abdominal and thoracic pressure
Suctioning	Pre-oxygenate; brief; consider lidocaine or an analgesic bolus first	A predictable ICP trigger

Table 3. Systemic physiological targets

Variable	Target	Strength
Temperature	36.0–37.5 °C; treat fever promptly and investigate its cause	Strong observational; fever independently worsens outcome
Glucose	140–180 mg/dL (7.8–10 mmol/L)	Avoid both hypoglycaemia and values >200 mg/dL
Sodium	135–145 mmol/L; **isotonic fluids only**	Hypotonic fluid is directly harmful; never use 5% dextrose or 0.45% saline for maintenance
Osmolality	Monitor if osmotherapy used; ceiling ≈320 mOsm/kg for mannitol; where hypertonic saline is the agent, the operative ceiling is serum sodium 155–160 mmol/L	Guides safe repeat dosing. The osmolality ceiling is conventional rather than trial-derived; an osmolar gap above 20 mOsm/kg is the more reliable signal of mannitol accumulation
Haemoglobin	Contested — see below	Two 2024 trials point the same way with differing statistical strength[17,18]
Nutrition	Enteral feeding started by 24–48 h; full caloric replacement by day 5–7	Guideline-supported mortality benefit[1]
VTE prophylaxis	Mechanical from admission; low-molecular-weight heparin from 24–72 h once the CT is stable	Delay beyond 72 h substantially raises thrombotic risk without clear haemorrhagic benefit
Bowel and bladder	Avoid retention and constipation	Both raise intra-abdominal pressure and act as PSH triggers

Contested: transfusion threshold. Two randomised trials reported in 2024. TRAIN (n = 850, acute brain injury of mixed aetiology; liberal threshold 9 g/dL versus restrictive 7 g/dL) found fewer unfavourable neurological outcomes at 180 days with the liberal strategy.[17] HEMOTION (n = 742, traumatic brain injury; liberal threshold 10 g/dL) found a difference in the same direction that did not reach statistical significance for its primary outcome (68.4% versus 73.5% unfavourable).[18] SAHaRA, in aneurysmal subarachnoid haemorrhage, was likewise neutral.[19] The honest reading is not that the trials disagree but that all three point one way and only one is statistically decisive. A defensible position: transfuse below 7 g/dL in all patients, and use a threshold near 9 g/dL where there is documented brain tissue hypoxia, ongoing intracranial hypertension, or established vasospasm risk. This is a reasonable extrapolation, not a guideline recommendation.

Table 4. Analgesia and sedation — assess before you escalate

Step	Action
1	Assess pain with a validated behavioural scale (CPOT or BPS) at fixed intervals — not only when the patient looks distressed[8]
2	Treat pain first. Short-acting opioid bolus (fentanyl, or remifentanil where available)

Step	Action
3	Reassess ICP and agitation after analgesia before adding or increasing any sedative
4	Sedate to a defined target, not to stillness. Propofol and/or dexmedetomidine; midazolam where haemodynamics do not tolerate propofol
5	Cap propofol below 4 mg/kg/h and limit duration; monitor lactate, creatine kinase, triglycerides and ECG for propofol infusion syndrome
6	Ketamine does not raise ICP in ventilated, sedated patients and is a reasonable adjunct where blood pressure is fragile
7	Neuromuscular blockade is a therapy, not a sedation strategy — it abolishes the neurological exam and mandates continuous EEG if used beyond a test dose

The single most useful habit in this table: when ICP rises, ask "is this pain?" before asking "is this sedation depth?" The answer is pain more often than most units assume, and the treatment is cheaper and safer.

5. MONITORING: WHAT TO PLACE, AND WHEN

Table 5. Indications

Device	Place when	Adds
ICP monitor	GCS 3–8 after resuscitation **with** an abnormal CT; or normal CT plus two of: age >40, motor posturing, SBP <90 mmHg	The core variable[1]
External ventricular drain	Preferred over parenchymal bolt when hydrocephalus, intraventricular haemorrhage, or a need for therapeutic drainage exists	Monitors **and** treats. Cannot measure and drain simultaneously[24]
Brain tissue oxygen ($PbtO_2$)	Where available and expertise exists, in severe TBI with ICP monitoring	Detects hypoxia occurring at normal ICP. Phase II data support a reduced hypoxic burden;[15] the phase III comparative-effectiveness trial is ongoing and should be consulted for its published result before this is treated as established practice[16]
Continuous EEG	Unexplained depressed consciousness, any clinical seizure, fluctuating examination, neuromuscular blockade, refractory ICP	Non-convulsive status is common and invisible without it
Autoregulation index (PRx) and optimal CPP	Where the platform exists	Individualises the CPP target rather than applying a population number. Feasibility and safety demonstrated; outcome benefit not yet[20]
Automated pupillometry	Serial, all patients	Objective, trendable, detects change earlier than manual assessment[31]
Transcranial Doppler	Adjunct	Vasospasm; non-invasive estimate where invasive monitoring is unavailable or delayed

Where invasive monitoring is unavailable. A substantial share of severe brain injury worldwide is managed without an ICP monitor, and the absence of a monitor is not a reason to abandon protocolised care. The CREVICE consensus protocol

provides a structured imaging- and examination-based approach for exactly this setting and should be adopted explicitly rather than improvised.[5]

Practical note on the EVD. A drain that is open is not measuring. Decide explicitly, and document, whether this patient's drain is primarily a monitor (closed, intermittently opened) or primarily a therapy (open at a set height, with intermittent closure to read). Ambiguity here is a common source of both unrecognised intracranial hypertension and over-drainage.

6. TARGETS

Table 6. Numbers to treat to

Variable	Target	Comment
ICP	<22 mmHg	Treat sustained elevation — conventionally >22 mmHg for more than 5 minutes — rather than transient spikes with an obvious trigger[1]
CPP	60–70 mmHg	Do not drive above 70 with aggressive fluid and vasopressor — this causes respiratory failure without neurological benefit[1]
CPP (individualised)	Optimal CPP derived from PRx, where available	Feasible and safe in a randomised phase II trial; an outcome-powered trial has not been completed. Preferred to the population range only where the platform and the expertise both exist[20]
$PbtO_2$	>20 mmHg	Treat hypoxia even when ICP is normal. The combined ICP/$PbtO_2$ escalation algorithm is set out in SIBICC-2[4]
$PaCO_2$	35–40 mmHg baseline	Lower only as a deliberate, time-limited tier
PaO_2	80–120 mmHg	Avoid hypoxia and sustained hyperoxia
Temperature	36.0–37.5 °C	
Sodium	135–145 mmol/L, or a higher deliberate target during osmotherapy, with a stated ceiling	Define the target explicitly; do not let it drift

ICP dose matters more than ICP peaks. The variable that tracks outcome is cumulative time-above-threshold, not the highest number on the chart. Where the monitoring platform allows, chart area-under-curve above 20 mmHg. Where it does not, count and record the minutes.

7. TIERED ESCALATION FOR INTRACRANIAL HYPERTENSION

Aligned to the SIBICC consensus algorithms for management with ICP monitoring[3] and with combined ICP and brain oxygen monitoring.[4] Tiers are a sequence, not a rigid ladder: move up promptly when a patient is deteriorating, and move down as soon as the trigger resolves.

Table 7. Tier 0 — before you treat the number

Check	Time to complete
Head position, neck alignment, collar, tube ties	30 seconds
Pain — assess and treat	2 minutes
Ventilator dyssynchrony, coughing, breath stacking	1 minute
Fever	Immediate
Bladder distension, abdominal distension	1 minute
Seizure — clinical or suspected non-convulsive	Immediate EEG if suspected
Transducer level, waveform quality, drain status	30 seconds

A substantial proportion of ICP elevations resolve within Tier 0. Skipping it converts a positioning problem into an osmotherapy problem.

Table 8. Tier 1

Intervention	Dose / specification
Deepen analgesia and sedation	Bolus, then reassess before increasing infusion
CSF drainage via EVD	Bounded volume per episode, over minutes rather than seconds; reassess between episodes. Intermittent and continuous strategies have both been described; the comparative evidence is limited[2,4]
Osmotherapy — mannitol	0.25–1 g/kg IV bolus; monitor osmolality, osmolar gap, sodium, renal function, volume status
Osmotherapy — hypertonic saline	3% bolus, or 23.4% via central access for herniation; monitor sodium closely against a stated ceiling
CPP optimisation	Raise mean arterial pressure toward the CPP target with vasopressor rather than volume loading
$PaCO_2$	Maintain at the low end of normal (35–38 mmHg)
Neuromuscular blockade	Test dose to determine whether ICP is being driven by muscular activity or dyssynchrony

Table 9. Tier 2

Intervention	Specification	Caution
Mild hyperventilation	$PaCO_2$ 32–35 mmHg	Time-limited. Requires brain oxygen or jugular venous saturation monitoring if sustained. See Section 8
Neuromuscular blockade infusion	Continuous	Mandates continuous EEG; abolishes the exam
Higher-dose or repeated osmotherapy	Guided by osmolality and sodium ceilings	Watch renal function and volume status
MAP challenge	Controlled MAP increase with monitoring of the ICP response	Distinguishes intact from impaired autoregulation and directs subsequent CPP strategy
Repeat CT and surgical reassessment	Low threshold	A new or expanding lesion changes the entire plan

Table 10. Tier 3 — salvage

Intervention	Evidence position
Barbiturate coma (pentobarbital or thiopental), titrated to EEG burst suppression	Lowers ICP reliably; causes hypotension, immunosuppression, ileus, prolonged sedation. No demonstrated outcome benefit. Not for prophylaxis[1]

Intervention	Evidence position
Secondary decompressive craniectomy	Reduces mortality in refractory intracranial hypertension, with a shift toward survival in states of severe disability and vegetative state.[12] **Early** craniectomy for diffuse injury produced worse outcomes and should not be performed for that indication.[11] This is a goals-of-care conversation as much as a surgical decision
Mild hypothermia 35–36 °C	Lowers ICP. Trials of prophylactic and early therapeutic hypothermia have not improved outcome and one was stopped for harm.[13,14] Salvage use only

8. HYPERVENTILATION: THE ONE TIER THAT REQUIRES ITS OWN RULES

Hypocapnia is the fastest ICP-lowering intervention available and the easiest to leave running. It has two independent time courses that must be tracked separately.

Efficacy decays. Cerebrospinal fluid pH normalises over roughly 6–24 hours through bicarbonate transport and renal compensation, and the vasoconstrictive effect wanes with it. Hyperventilation cannot function as maintenance therapy — beyond the first day the patient carries the ischaemic cost without the pressure benefit. A second consequence follows: once adaptation has occurred, rapid restoration of normocapnia produces relative hypercapnia at the vessel wall, with rebound vasodilation.

Ischaemic exposure accumulates from the first minute, concentrated in watershed territories already perfusing at margin.

Table 11. Operating rules

Rule	Specification
Indication	Acute ICP crisis or impending herniation only. Never prophylactic, never routine
Floor	$PaCO_2$ never below 30 mmHg without $PbtO_2$ or jugular venous oxygen saturation monitoring
Exit plan	Written at the moment of initiation, naming the definitive therapy being started alongside — osmotherapy, CSF diversion, or surgery
Charted variable	Cumulative minutes below $PaCO_2$ 35 mmHg, totalled and handed over
Return to normocapnia	Stepwise: 1–2 breaths/min at a time, with continuous ICP observation. Rebound risk is a function of the rate of correction
Review trigger	Any cumulative exposure beyond a few hours prompts senior review and reassessment of goals of care

The cumulative-exposure counter proposed here is a reasoning-derived charting convention, not a validated metric. It is offered because the alternative — a variable that no one totals — reliably under-represents the dose delivered. It has not been prospectively evaluated.

9. THE PARALLEL PROBLEMS

Intracranial hypertension rarely arrives alone.

Seizures and non-convulsive status. Prophylaxis for 7 days reduces early seizures only; do not continue beyond a week without an indication. Any unexplained failure to wake, fluctuating examination, or subtle motor phenomenon warrants continuous EEG. Non-convulsive status is a treatable cause of refractory intracranial hypertension that is invisible on examination.

Sodium. Distinguish the syndrome of inappropriate antidiuresis from cerebral salt wasting by volume status, not by sodium alone — the sodium is identical and the treatments are opposite. Correct chronic or unknown-duration hyponatraemia at no more than 8 mmol/L in 24 hours, with a hard limit of 10–12 mmol/L, to avoid osmotic demyelination.[27] Diabetes insipidus after severe injury signals brainstem compromise and demands both treatment and reassessment of prognosis.

Fever. Distinguish infection from central fever from paroxysmal sympathetic hyperactivity. Central fever is a diagnosis of exclusion and typically shows a high, sustained, non-swinging temperature with relative bradycardia for the temperature and a negative work-up. Treat all three; each degree of fever raises cerebral metabolic demand.

Paroxysmal sympathetic hyperactivity. Episodic tachycardia, hypertension, tachypnoea, hyperthermia, diaphoresis and posturing occurring together, without another cause; the consensus diagnostic criteria should be used rather than clinical impression.[23] Onset is typically around the end of the first week, not the first 48 hours.[22]

Treatment of established PSH is symptom-directed: propranolol, gabapentin, clonidine, and opioids are all used, with a small and largely observational evidence base.[22] Prophylaxis is a newer and much weaker proposition. A single-centre randomised trial of 90 patients with moderate-to-severe injury reported PSH in 60% of controls, 33% with propranolol 40 mg twelve-hourly, and 10% with propranolol plus gabapentin; propranolol alone shortened ventilation and ICU stay, while the combination reduced incidence further but was associated with longer ventilation. Mortality and GCS improvement did not differ.[21] This is a plausible and welcome signal, but it is one small unblinded single-centre trial with a surrogate primary endpoint and no functional-outcome benefit, and it should not be read as establishing prophylaxis as standard care. Units choosing to adopt it should reproduce the trial's population and exclusions, define a CPP floor below which the drug is withheld — beta-blockade lowers the pressure cerebral perfusion depends on — and treat the practice as local protocol pending replication.

Ventilation. Lung-protective tidal volumes are compatible with neuroprotection and should be used; high tidal volumes are associated with lung injury after brain injury.[25] Where permissive hypercapnia and ICP control conflict, ICP wins in the acute phase — but the conflict should trigger reassessment rather than acceptance.[26]

Mobilisation. Begin early, coordinated with the ICP plan, with a defined time-out threshold at which activity stops.

10. COMING BACK DOWN

Escalation protocols are written; de-escalation protocols usually are not. This is where iatrogenic harm accumulates.

Table 12. Weaning sequence

Order	Step	Condition
1	Wean hyperventilation first	Stepwise, hours, watching for rebound
2	Reduce neuromuscular blockade	Restores the examination
3	Reduce osmotherapy	Watch sodium, osmolality, rebound intracranial pressure
4	Lighten sedation and attempt a neurological wake-up assessment	Only once ICP has been stable for a defined interval
5	EVD weaning trial	Raise the drain, then clamp, with defined ICP and clinical stop criteria; image before removal
6	Reassess seizure prophylaxis, autonomic agents, and every other standing order	Most are continued by inertia

The general rule: reverse the order of escalation, change one variable at a time, and define in advance what would make you stop.

11. WHERE NON-TRAUMATIC INJURY DIVERGES

Most of the evidence above is traumatic. The physiology of secondary injury is shared, but four divergences are large enough that transferring the trauma answer causes harm.

Intracerebral haemorrhage. The blood-pressure recommendation inverts. Where trauma demands SBP ≥110 mmHg, spontaneous intracerebral haemorrhage in the first hours calls for early, controlled *lowering* toward approximately 140 mmHg, avoiding large or abrupt reductions.[32,33] Current guidance supports early intensive lowering as safe and probably beneficial for

functional outcome.[34] Seizure prophylaxis is not indicated. Applying the trauma target to an intracerebral haemorrhage is one of the more common and consequential errors in mixed units.

Aneurysmal subarachnoid haemorrhage. Oral nimodipine 60 mg six-hourly for 21 days is a firm recommendation and improves outcome; it is not vasospasm therapy in the mechanical sense and should be started irrespective of transcranial Doppler findings.[35,36] Delayed cerebral ischaemia is a distinct syndrome from the intracranial hypertension modelled above, peaks days 4–14, and is managed by induced hypertension and endovascular rescue rather than by the tiers in Section 7. Blood pressure before aneurysm securing is managed differently again. Prophylactic anticonvulsants are not routinely recommended.

Malignant middle cerebral artery infarction. Decompressive hemicraniectomy differs from the trauma indication in both timing and selection: pooled randomised data support surgery within 48 hours in patients under 60, with a large absolute mortality reduction and a shift in surviving disability that must be discussed explicitly with the family before operating.[37] The DECRA caution against early craniectomy for diffuse traumatic injury does not transfer here.

Anoxic, metabolic, and inflammatory encephalopathies. Where the injury is diffuse and non-structural, ICP monitoring is rarely indicated, prognostication timelines differ, and the primary intervention is treatment of the underlying cause. The floor in Section 4 still applies in full.

12. PROGNOSTICATION AND GOALS OF CARE

Defer definitive prognostication for at least 72 hours after injury, and longer where sedation, hypothermia, renal failure, or hepatic dysfunction confound the examination. Early prognostication is unreliable and carries a well-documented risk of self-fulfilling withdrawal.[28]

Prognostic models estimate outcome for populations, not for the patient in front of you. Use them to structure a conversation, not to end one.

During the uncertain window, run a **time-limited trial**: full physiological optimisation with an explicit, documented agreement on what will be reassessed and when. This is the structure that permits aggressive support and honest uncertainty to coexist. Involve palliative care concurrently rather than terminally — for prognostic communication and family support, not only for withdrawal.[29]

Surrogates in this setting are simultaneously informants, advocates, negotiators, and people in acute grief. Prognostic uncertainty, not bad news, is the dominant source of their distress. Structure the conversations: early, repeated, with the same clinician where possible, and with the uncertainty named rather than smoothed.[29]

Every intervention in this document can be delivered to a patient who will not benefit from it. Attach each escalation to a stated goal, and revisit the goal when the trajectory changes.

13. THE DAILY NEUROPROTECTION CHECKLIST

For rounds and for every handover. If a unit adopts one thing from this document, adopt this.

- ICP target, current value, and **minutes above threshold in the last 24 h**
- CPP target — population range or individualised — and time below it
- $PaCO_2$ target and **cumulative minutes below 35 mmHg**
- EVD: monitor or therapy? Height, volume drained in 24 h, status during transport
- Pain score, assessment compliance, and sedation target with current depth
- Temperature, sodium, glucose, haemoglobin — with the target beside each value
- Seizure prophylaxis: day number and stop date
- PSH: prodrome present? Drug started? Time from admission to first dose
- VTE prophylaxis: started or, if not, the documented reason

- Nutrition: route and percentage of goal
- Aetiology-specific item: nimodipine day number (aSAH), BP target direction (ICH), craniectomy window (malignant MCA)
- What tier is this patient on, and what is the condition for coming down?
- Family communication: last conversation, next scheduled, goals documented

14. LIMITATIONS AND EVIDENCE STATUS

This is a practical synthesis, not a systematic review, and it should be used as an operational scaffold rather than as a substitute for the source guidelines.

The adult severe traumatic brain injury guidance underpinning much of this document is the fourth edition of the Brain Trauma Foundation guidelines with its 2020 decompressive craniectomy update; a fifth edition is in preparation and, at the time of writing, unpublished. Readers should check for its release. Recommendations are drawn predominantly from traumatic brain injury evidence; Section 11 marks the principal points at which extrapolation to other forms of severe acute brain injury fails, but the extrapolation elsewhere is reasonable rather than empirically established.

Several thresholds here reflect physiological reasoning rather than trial evidence and are labelled as such in the text, including the cumulative hypocapnia counter in Section 8 and the bounded CSF drainage volume in Table 8. Areas of active uncertainty include transfusion thresholds, brain tissue oxygen target values and their outcome benefit, patient selection for decompressive craniectomy, autonomic prophylaxis, and the optimal method of individualising cerebral perfusion pressure.

The authors report no primary data. No protocol described here has been prospectively implemented or evaluated at any participating institution, and no statement in this document should be read as a report of local practice or local outcomes.

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