Current management of traumatic intracranial hypertension: What you need to know

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ABSTRACT: Intracranial hypertension (ICH) has long been recognized as a major challenge in the management of patients with severe traumatic brain injury. Although it has become widely accepted that patients with unremarkable brain computed tomography scans may not require invasive intracranial pressure (ICP) monitoring, the threshold for using such monitoring in other severe traumatic brain injury patients should be low. Intraparenchymal monitors and external ventricular drains are both effective monitoring techniques. No specific prophylactic interventions are available to prevent ICH. Instead, initial management focuses on maintaining physiological parameters in the normal range and intervening promptly when deviations occur. Management of elevated intracranial pressure proceeds along a series of interventions that begins with the least invasive and least complex measures and progressively escalates to those with greater scope and risk. However, the sequence of these steps is not rigorously defined, and considerable latitude may be exercised according to a specific patient's individual situation. Choosing the appropriate therapies for different patients requires the clinician to have a deep understanding of the pathophysiology of ICH. (*J Trauma Acute Care Surg.* 2026;00: 00–00. Copyright © 2026 Wolters Kluwer Health, Inc. All rights reserved.)

KEY WORDS: Intracranial hypertension; intracranial pressure; management; traumatic brain injury.

"One who has an oozing cutting wound in his head, which has penetrated to the bone and smashed in his cranium, who suffers from stiffness in his neck: [This is] a medical condition you will not be able to treat."[1] This passage from the Edwin Smith Papyrus may well describe a critically ill patient with intracranial hypertension (ICH).[2]

Although medical care has advanced far beyond anything that ancient physicians could have imagined, modern-day practitioners who treat these same patients often achieve no better outcomes than those of our Egyptian predecessors 3,500 years ago. Elevated intracranial pressure (ICP) continues to be a much-feared cause of poor outcomes after severe traumatic brain injury (sTBI).[3] This article is a practical management-oriented review for bedside clinicians who are entrusted with the care of these complex and critically ill patients. Although the vast majority of published literature in this area comes from studies of closed TBI, the same management principles have been recommended for patients with penetrating TBI.[4]

WHEN TO MONITOR ICP

The pioneering studies of the Traumatic Coma Data Bank helped to quantify the poor outcomes associated with elevated ICP and hypotension in patients with sTBI. This work led to the widespread practice of inserting ICP monitors in any patient with sTBI, which is often defined as a Glasgow Coma Scale[5] (GCS) score of ≤8 or as failure of a patient to obey simple commands, which corresponds to a motor score on the GCS of ≤5. Failure to obey commands can be a good working definition of coma.

Subsequently, as the clinical community gained more experience with ICP monitoring and as the speed and quality of computed tomography (CT) scanning improved, some began to question the practice of uniformly monitoring ICP in every sTBI patient. In particular, sTBI patients with relatively unimpressive cranial CT scans may have diffuse axonal injury or other pathologies that are rarely associated with elevated ICP. Intoxication with alcohol and other drugs may also be the primary reason for a depressed neurological examination. Specific variables have been proposed to help clinicians decide which patients are more likely to require invasive ICP monitoring.[6]

In late 2012, publication of the BEST TRIP (Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure) trial caused further reevaluation of the need for invasive ICP monitoring in all sTBI patients.[7] This randomized, controlled, prospective, single-blind study conducted in Bolivia and Ecuador compared patients with parenchymal ICP monitors with those in whom elevated ICP was inferred from clinical and CT findings. The primary outcome did not differ significantly between the two groups. A subsequent consensus-based interpretation emphasized that, instead of comparing monitored to unmonitored patients, the trial actually compared two sTBI management protocols, one using invasive ICP monitoring and another using serial CT scanning and neurological examinations.[8] Other important points were that generalizability of these results to other practice settings may be limited and that management of ICH was not a focus of the trial because many enrolled patients did not develop ICH.

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A prospective cohort study published in 2023 used the database of the CREACTIVE (Collaborative REsearch on ACute Traumatic brain Injury in intensiVe care medicine in Europe) Consortium to study outcomes in almost 1,500 TBI patients in dozens of intensive care units (ICUs) in Italy and Hungary. These investigators identified an association between ICP monitoring and worse 6-month outcomes and also with greater frequency of medical interventions and respiratory and infectious complications. However, causality could not be determined. The authors point out that "clinical studies on ICP monitoring reflect only the context in which ICP data are used and do not question the value of knowing ICP values," emphasizing instead the need for better understanding of how ICP data can be used to improve patient outcome.[9] The fourth edition of the Brain Trauma Foundation (BTF) sTBI guidelines concludes, "Management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality."[10] Other authors have emphasized the need for improved understanding of which patients are most likely to benefit from invasive monitoring.[11]

The most prudent course is to consider ICP monitoring in sTBI patients with a GCS score of ≤8 and/or motor score of ≤5 when CT reveals the presence of brain swelling or other acute injury that could account for the poor neurological examination (Figs. 1 and 2). Toxicology screen and basic blood laboratory studies are helpful for identifying metabolic derangements that can worsen a patient's neurological examination. If ICP monitoring is deferred, hourly neurological examinations and repeat CT scanning after a short interval, such as 4 to 6 hours, are recommended. Deterioration of a patient's neurological status should prompt the neurosurgeon to reassess the decision not to monitor ICP.

HOW TO MONITOR ICP

Intraparenchymal monitors are commonly used to measure ICP. Although the devices sold by various vendors may use different technologies, they share certain common features. Ease of insertion is a major advantage. They can be inserted at almost any location on the cranial vault, usually to a depth of

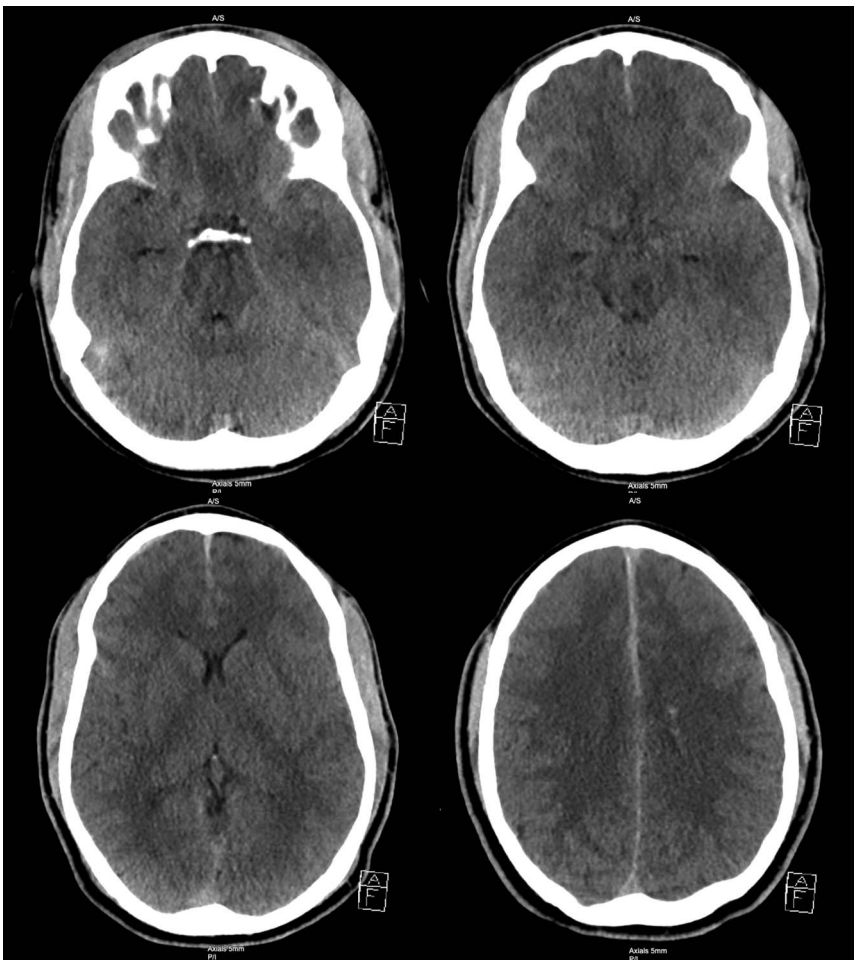


Figure 1. Initial head CT in a 35-year-old TBI patient with Glasgow Coma Scale score of 6. Imaging reveals minimal subdural hemorrhage along the anterior falx, scattered minimal subarachnoid and intraparenchymal hemorrhage, and hypodensities consistent with edema in the temporal and inferior frontal lobes. Distinct sulci and gyri are not visualized, suggesting mild edema of the cerebral hemispheres. However, the grey-white junction is preserved. Intracranial pressure (ICP) monitoring was initiated, and episodic increases in ICP during the first few days after injury responded to treatment with osmolar agents. The patient went on to a good recovery.

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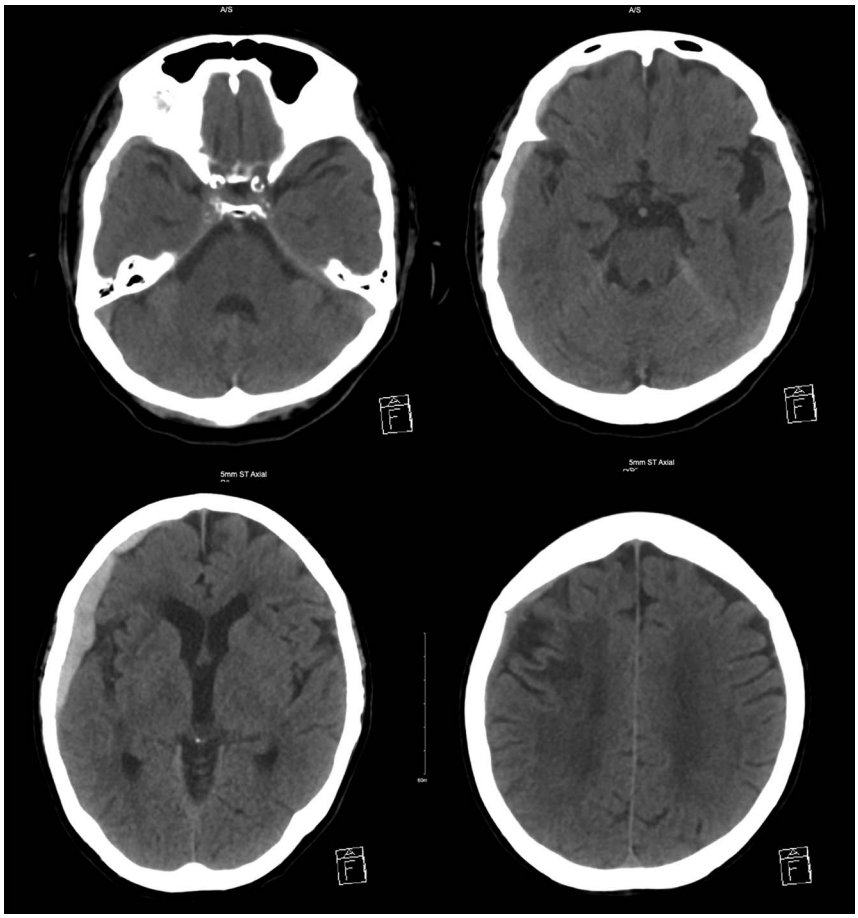


Figure 2. Acute subdural hematoma in an elderly patient on oral anticoagulation. Some compression of underlying gyri is present, but the overall degree of mass effect is minimal. Compare to mild diffuse edema without a significant mass lesion in Figure 1.

several centimeters into the brain parenchyma. ICP is measured continuously and is displayed on a bedside monitor. These monitors are calibrated prior to insertion but cannot be recalibrated after insertion.

In contrast, an external ventricular drain (EVD) is usually inserted at Kocher's point in the posterior frontal region, 2 to 3 cm lateral to the midline and 1 to 2 cm anterior to the coronal suture. Because precise targeting is needed for successful cannulation of the cerebral ventricles, compression or shift of the ventricles may make insertion difficult or even impossible. An EVD is a hollow tube connected to an external transducer, which permits re-zeroing as often as needed. More importantly, it can be used as a therapeutic device to drain cerebrospinal fluid (CSF) if ICP increases. This benefit of EVDs must be weighed against a higher risk of infection than seen with parenchymal monitors.

It is not possible to monitor ICP accurately while an EVD is open to drainage; monitoring or drainage is best thought of as an "either/or" decision. However, some commercially available EVD catheters have been designed to permit measurement of ICP while the catheter is open and draining CSF.[12] Simultaneous use of both a parenchymal monitor, which permits continuous ICP monitoring, and an EVD, which is left open for continuous drainage of CSF, has also been proposed and evaluated, although this is not common practice.[13,14]

The EVD is zeroed at the level of the foramen of Monro, which is approximated at the tragus.[15] The top of the drip chamber can be set at any height, ranging from several centimeters below the zero point to many centimeters above it. At many centers, the default practice is to set the top of the drip chamber at a height of 10 cm. However, raising the drip chamber above zero cm might reduce the amount of CSF that can be drained after opening of the stopcock, potentially making it more difficult to control ICP. The proposed rationale for keeping the drip chamber above 0 cm is to prevent overdrainage of CSF, which is feared to cause collapse (marked decrease in size) of the cerebral ventricles and thereby make further CSF drainage impossible, and/or to prevent the cerebral cortex from falling inward from the dura, potentially causing formation of a subdural hematoma. These concerns are unfounded in most sTBI patients. Ventricular collapse in this population is more likely due to cerebral edema than to overdrainage. Likewise, edema within the recently injured brain usually prevents the cortex from falling inward. One potential strategy is to start with the drip chamber at 10 cm and then lower it to facilitate CSF drainage if elevated ICP becomes difficult to control.

Institutions vary in their preferences for leaving the EVD closed or open. Leaving it open to continuous drainage may result in an overall lower ICP, but the intermittent nature of ICP

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measurements (often performed only hourly) creates the possibility that ICP elevations may go undetected and untreated until the next hourly measurement. Leaving the EVD closed permits continuous monitoring of ICP, with opening of the stopcock and drainage of CSF whenever ICP is sustained above the threshold level. The exact way in which these nursing orders are written varies among institutions. One example is "drain CSF for ICP sustained above 20 mm Hg for 5 minutes." Some institutions specify drainage for a certain amount of time (such as 5 minutes) before rechecking ICP and, if it is still elevated, to repeat as often as needed. Others specify drainage of a certain volume of CSF, such as 5 mL, and then recheck and repeat as needed. Some institutions limit the frequency with which CSF can be drained, such as no more than three times an hour, whereas others have no such constraints. These restrictions are designed to prevent overdrainage of CSF, but again, overdrainage is less of a concern in patients with acute severe TBI than in patients with other neurological diseases.

We favor keeping the EVD closed to monitor ICP continuously. Intermittent CSF drainage is performed as needed for increases in ICP. Failure of CSF drainage to control ICP suggests that a different plan is needed, such as raising the ICP goal or trying different interventions. As mentioned previously, use of EVDs that can simultaneously measure ICP and drain CSF can eliminate the need to choose between continuous drainage or continuous monitoring.

External ventricular drains and parenchymal monitors each have their specific advantages and disadvantages. These are summarized in Table 1. In many institutions, parenchymal monitors are preferred if the index of suspicion for ICH is low, whereas EVDs are used if hydrocephalus, intraventricular hemorrhage, or presumed ICH is present. In the big picture, the decision to monitor ICP and to act on the data provided is more important than the specific type of monitoring device that is used.

THRESHOLD FOR TREATING ICP

The physiologic definition of elevated ICP remains a matter of debate, partly because "normal" values are poorly defined, since healthy or normal patients do not undergo invasive ICP monitoring. The Traumatic Coma Data Bank identified 20 mm Hg as the value above which persistent elevations of ICP are associated with worse outcomes.[16] This has been the traditionally accepted threshold for treatment, but the fourth edition of the BTF Guidelines for the Management of Severe Traumatic Brain Injury recommends a value of 22 mm Hg.[10] That number comes largely from a single retrospective study of ICP and autoregulation that used a single summary value for ICP across the entire monitoring period.[17] The guideline's authors rated this as Class 2 data, but the analytic process and the decision to change the recommended treatment threshold from 20 to 22 mm Hg have been criticized.[18]

TABLE 1. Comparison: EVD Versus IP Monitors of Intracranial Pressure

Feature/Question	EVD	IP Monitor
Primary function	ICP monitoring plus CSF drainage (therapeutic)	ICP monitoring only (no CSF drainage)
Ability to treat ICP directly	Yes — can drain CSF to lower ICP immediately	No — monitoring only; requires other therapies to reduce ICP
Accuracy and calibration	Measures ventricular pressure referenced to external transducer; can be re-zeroed/recalibrated at bedside	High initial accuracy; cannot be re-zeroed after insertion (drift possible over time)
Use when ventricles are small/effaced	Placement may be difficult or impossible with severe cerebral edema or slit ventricles	Easier placement even with effaced ventricles or midline shift
Insertion complexity and logistics	More technically involved (burr hole + catheter into ventricle)	Relatively simpler insertion (bolt or tunneled catheter)
Risk of infection (ventriculitis/meningitis)	Higher infection risk vs. IP	Lower infection/ventriculitis risk overall than EVD
Hemorrhage/insertion bleeding risk	Risk of catheter tract hemorrhage; some series report higher hemorrhage rates vs. IP	Lower bleeding rates reported in some series
Catheter malfunction/blockage	Prone to blockage or occlusion (clot, debris) — may require replacement	Less prone to gross blockage; electronic drift or sensor failure possible
Continuous waveforms and data fidelity	Good waveform fidelity but can be affected by drainage/leveling/air column	Good continuous waveform; less subject to leveling issues with patient position
Mobility/nursing care	Requires leveled drainage system, careful height adjustments and strict nursing protocols	Easier patient handling and mobilization (no external drainage system)
Monitoring during CSF drainage	ICP values may be unreliable while actively draining (need to clamp to measure true ICP)	ICP readings are continuous
Impact on outcomes (mortality/functional outcome)	Comparative studies show no consistent superiority of one device type for long-term outcome; management and clinical context matter	Same — choice often driven by clinical scenario, institutional practice, and anticipated need for CSF drainage
Typical indications favoring use	Acute hydrocephalus, intraventricular hemorrhage, need for therapeutic CSF drainage	Routine ICP monitoring when ventricles small, high bleeding risk, or drainage not expected to be necessary
Cost/resource use	Lower cost but potentially higher nursing/maintenance burden (leveling, ICU care) and replacement procedures	Higher cost but lower maintenance logistics; costs increase if replacement needed because of sensor failure or drift

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EVD, external ventricular drain; IP, intraparenchymal.

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More important than the exact definition of elevated ICP is the length of time that an increase can be tolerated before intervention becomes necessary. Many practitioners do not treat elevated ICP until the increase has persisted for a defined period of time, such as 5 minutes. Transient elevations are often caused by patient movement, suctioning, coughing, and other stimuli. These increases in ICP resolve spontaneously.

Instead of blind adherence to a single number to define high ICP, clinicians should recognize the dynamic nature of ICP and cerebral perfusion pressure (CPP) (defined as mean arterial pressure [MAP] minus ICP) and their management in sTBI patients.[10,19] In many cases, such as persistent ICPs in the low 20s, it may be best to raise the treatment threshold to 25 mm Hg. Doing so spares the patient potentially avoidable interventions and their possible adverse effects. Attention should be paid to maintaining the CPP at an appropriate level, such as 50 to 70 mm Hg, with adjustments to this target based on the patient's clinical condition and other data.[20]

Monitoring of brain tissue oxygen tension ($PbtO_2$) or other cerebral physiologic parameters can help inform decisions about ICP management and appropriate thresholds for ICP and CPP.[21] Ongoing large trials such as BOOST-3 (Brain Oxygen Optimization in Severe TBI, Phase 3) will be critical to clarify the role of $PbtO_2$ monitoring in routine practice.[22] The pressure reactivity index or autoregulation-guided "optimal CPP" can also be used to individualize thresholds instead of applying the same rigid cutoff value to all patients.[23]

Some investigators emphasize the concept of "total ICP dose," or time burden of ICP, which includes both intensity (or magnitude) of ICP and its duration.[24] In such a conceptualization, a brief ICP spike to 25 mm Hg may be benign, but a sustained ICP of 18 to 20 mm Hg for many hours could be deleterious.

Age significantly modifies intracranial compliance and tolerance of elevated ICP. Pediatric patients may maintain adequate cerebral perfusion at lower CPP targets, whereas older adults with cerebral atrophy may exhibit minimal ICP elevation despite substantial mass effect.[25,26] Individualizing management according to age-related pathophysiology may optimize both safety and efficacy of therapy.[27]

PROPHYLAXIS AGAINST ELEVATED ICP

Numerous interventions are available to reduce elevated ICP. However, none have been shown to improve outcomes when used prophylactically (Table 2). In many cases, prophylactic administration makes things worse by causing complications or increasing length of stay. To use a sports analogy, we cannot play offense and attempt to control the game. The best we can do is be vigilant and intervene promptly when ICP increases—in other words, to play very good defense.

Hypernatremia

Prophylactic hypernatremia is used in many ICUs that treat sTBI patients. However, this practice is not supported by high-quality studies or recommended in clinical management guidelines.[10,19,28] Furthermore, the brain rapidly adapts via intracellular entry of electrolytes that balance the extracellular hypertonicity, followed soon thereafter by accumulation of organic osmolytes within cells,[19] such as taurine, glutamine, sorbitol, myoinositol, and others. Thus, any benefit from induced hypertonicity is short-lived. In the authors' experience, injured brain tissue that is destined to swell will likely do so even if the blood osmolality has been artificially elevated.

TABLE 2. Partial List of Failed Prophylactic Treatments in sTBI Patients

Barbiturates
CPP elevation
Decompressive Craniectomy
Dehydration
Hypernatremia
Hyperventilation
Hypothermia
Numerous pharmacological treatments
Pharmacologic paralysis
Steroids

Unfortunately, if prophylactic induction of hypernatremia has already raised blood osmolality and/or serum sodium concentration to prohibitively high levels before refractory ICH declares itself, the clinician finds that he or she is now prevented from using osmolar therapy just when it is needed most. To use another analogy, this is equivalent to an archer unnecessarily shooting all the arrows in his quiver, with the result that he now has no arrows to use when they are really needed.

When ICH begins to resolve in a patient who remains hypernatremic, the clinician should resist the urge to treat the hypernatremia aggressively. Sudden decreases in serum sodium concentration may cause ICP to increase as free water enters the still-hypertonic brain tissue. The best strategy may be to accept the higher sodium concentration temporarily, until cerebral edema has had more time to resolve. Even then, careful reduction of serum osmolality via frequent small boluses of water through a feeding tube is preferred to intravenous administration of hypotonic fluids.

Lumbar Drainage

A dogma-challenging proposal is to use controlled lumbar drainage in selected sTBI patients to prevent or blunt ICP elevations.[29] Criteria to determine potential appropriateness of this intervention include presence of an EVD or parenchymal ICP monitor, absence of cisternal effacement, and absence of temporal contusions.[30] Small volumes of CSF are drained at regular intervals. Although lumbar drainage is well established in patients with aneurysmal subarachnoid hemorrhage,[31] its use in sTBI patients is still a new concept that is under investigation. At this time, those who plan to use lumbar drainage in sTBI patients are advised to do so carefully, with an ICU team that is thoroughly familiar with applicable treatment protocols.

Prophylactic use of hypothermia and decompressive craniectomy (DC) are discussed in the section on management of elevated ICP.

INITIAL MANAGEMENT OF STBI PATIENTS

Surgeons, intensivists, and other acute care providers must avoid the trap of giving in to nihilism when they first encounter sTBI patients. With appropriate management in the ambulance,

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the emergency department, and the ICU, many of these patients go on to make excellent recoveries.[32]

The starting point for management of sTBI patients is to maintain normal homeostasis and to focus on the well-established concept of avoiding secondary insults.[33] As mentioned previously, no neuroprotective drugs are currently available to prevent ICH or improve outcome from sTBI. However, maintaining normal homeostatic conditions may prevent or minimize physiologic derangements that can provoke increases in ICP.

Basic measures that may help prevent ICP spikes include elevation of the head of bed to 30°, loosening of a cervical collar (if present) to avoid compression of the jugular veins, and prophylactic administration of anticonvulsants for the first 7 days after injury, such as levetiracetam as a 1,000 mg bolus followed by 500 mg every 12 hours for 7 days.[10] Commonly recommended physiologic targets include ICP <20 to 22 mm Hg and CPP of 60 to 70 mm Hg. Aggressive attempts to enforce upper limits on CPP must be balanced against the potential need to raise MAP to ensure adequate perfusion to the brain, especially because many sTBI patients demonstrate low cerebral blood flow or even ischemia in the immediate postinjury period, and particularly during the first few hours after injury.[34] If $PbtO_2$ monitors are in place, they can help assess the adequacy of cerebral metabolism.

Other targets include a hemoglobin concentration at or above 7 g/dL and sodium concentration in the normal range of 135 to 145 mEq/L.[35] Care must be taken to prevent hyponatremia. A common PaO_2 target is 80 to 100 mm Hg. Supraphysiologic arterial oxygen tension has not been shown to improve outcomes after sTBI, and observational and retrospective studies associate prolonged hyperoxia with worse outcomes.[36–38] We therefore avoid high FiO_2 and very high PaO_2 values while strictly preventing hypoxia. Our $PaCO_2$ goal is the normal range of 35 to 45 mm Hg.[35] Transient hyperventilation may be needed for acute herniation or sudden refractory increases in ICP but only as a bridge until definitive therapy can be initiated.[39] Sustained hyperventilation produces temporary vasoconstriction that can cause or exacerbate cerebral ischemia in an injured brain.[40,41]

A common goal for core temperature is normothermia. Fevers are often treated aggressively based on numerous reports linking elevated temperatures to adverse physiologic events[42] and poor outcomes in patients with TBI and other forms of acute brain injury.[43] Many practitioners initiate targeted temperature control to keep a patient's temperature in the desired range, such as 36.0°C to 37.5°C.[44] Various techniques may be used, including commercially available temperature control devices or other methods. Interestingly, however, studies that have examined the effect of fever prevention efforts on outcome have so far failed to demonstrate any benefit, doubtless due at least in part to unwillingness of providers to participate in randomized prospective studies that would leave some patients with untreated fevers. In this sense, temperature control is somewhat similar to ICP monitoring. Both still await demonstration of efficacy for improving outcomes.

Basic management practices include analgesia and sedation. Many sTBI patients have painful injuries that can contribute to agitation, as can direct effects of the brain injury and/or the discomfort of having an endotracheal tube in place. Minimizing pain and agitation is a compassionate intervention. Pain and agitation can also raise ICP if left untreated. Initially, short-acting agents are preferred because they need to be paused only briefly before their effects diminish, thus allowing frequent neurological assessments that are not confounded by the effects of medications. Fentanyl and propofol are frequently used for this purpose.[45] Dexmedetomidine has been growing in use in recent years,[46] as has ketamine.[45] Contrary to earlier teaching, more recent studies demonstrate that ketamine does not raise ICP and can preserve cerebral perfusion in hypotensive patients, supporting its selective use in combination with other sedatives.[47]

Continuous electroencephalography has become common in many neuro ICUs.[48] In TBI patients, common indications include failure of a patient to improve as much as expected and unexplained deterioration in neurological status. The goal is to identify abnormalities that may respond to treatment with anticonvulsants or other measures.

STEPWISE MANAGEMENT OF ELEVATED ICP

Tiers of Care

The concept of grouping treatments for ICH in sTBI patients into "tiers" of care has become quite popular. This grouping is based on the simple concept of starting with the least invasive and least dangerous interventions and then adding or substituting other treatments as needed.[49] However, this is only a very general approach. At the clinician's discretion, more advanced therapies can be administered very early in a patient's course, bypassing less aggressive measures.[19]

This same philosophy underlies the sequencing of the interventions listed hereinafter and in Figure 3. The authors have followed this basic algorithm for decades, but they are not locked into it and will go "off script" if doing so seems to be best for a particular patient.

Repeat CT Scan

If a brain CT scan has not been obtained recently, and especially if the patient is still within the first or second day of injury, the onset of ICH may prompt repeat CT scanning to rule out an expanding or delayed intracranial hematoma. Other events that might prompt CT scanning for worsening ICH include recent intracranial surgery or insertion of an intracranial monitor, decrease or loss of CSF flow from an EVD, and worsening of clinical neurological examination. On the other hand, if a patient has been in the ICU for several days, has not had any recent procedures, and has had a recent CT scan, the yield from obtaining yet another scan is lower, especially when multiple CT scans are obtained on the same day.

First Steps: Sedation, Analgesia, and Paralysis

If a patient seems to be agitated or experiencing pain, a good initial step is to administer a bolus of an analgesic such as fentanyl or a sedative such as propofol. The need for frequent boluses suggests that the rate of continuous infusion of these medications may need to be increased.

If ICP remains elevated despite adequate sedation and analgesia, some patients may benefit from pharmacologic paralysis. This intervention had fallen out of favor because of concerns about increased risks of critical illness polyneuropathy[50] and other complications. However, the pendulum has somewhat

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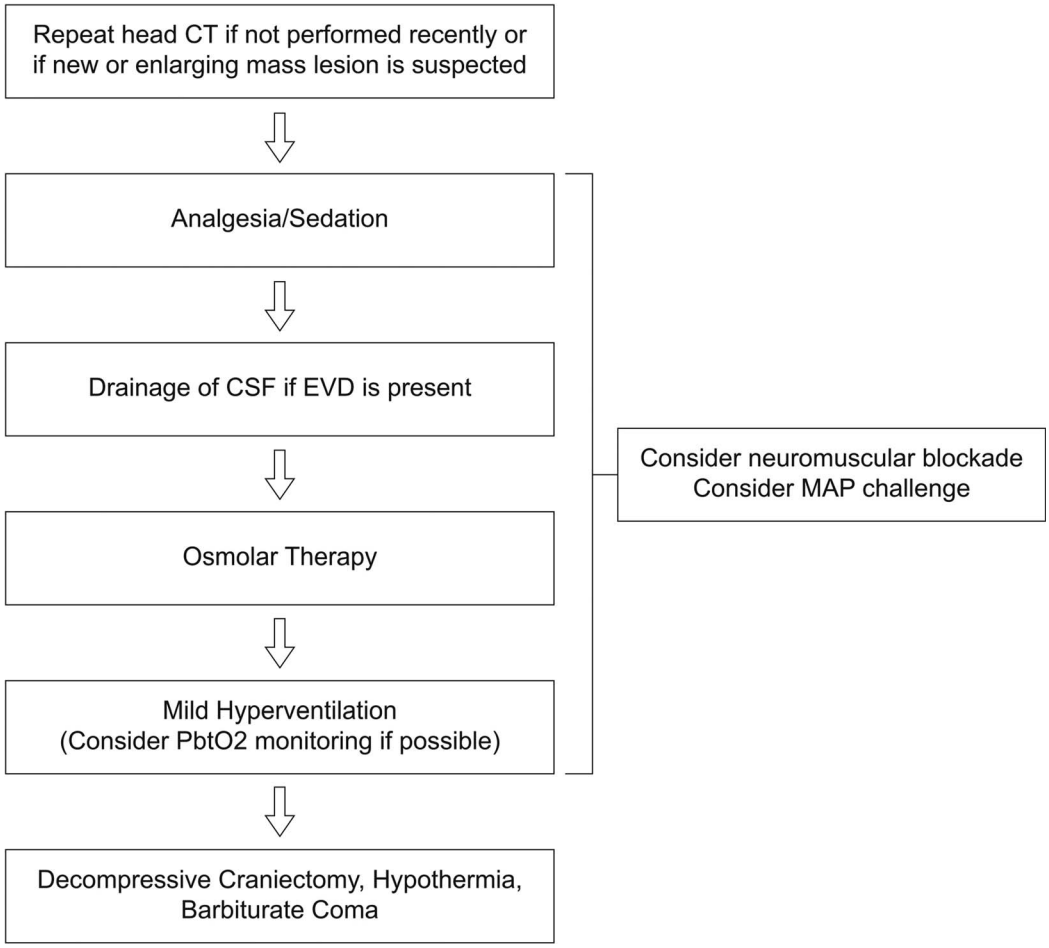


Figure 3. Algorithm for stepwise management of elevated ICP.

swung back in the other direction after the advent of improved monitoring techniques and protocolization of care to optimize safety when this treatment becomes necessary.[51] A reasonable approach is to trial this intervention for a short period while both the adequacy of paralysis and the ICP response are monitored.[19] Reduction in ICP suggests that the neuromuscular blocking agent should be continued.

Second Step: CSF Drainage

The next step to treat persistent ICH is CSF drainage (if available). This can also be performed in concert with escalation of sedation or pharmacologic paralysis. The need for frequent opening of the EVD is not necessarily a problem if the ICP continues to respond well. Potentially concerning signs include increasingly frequent need to drain CSF and/or progressively less response of ICH to drainage.

Third Step: Osmolar Therapy

If analgesia/sedation and CSF drainage fail to control ICP, a common next step is osmolar therapy. A frequent question is which medication to use. Although strong opinions exist on both sides, the available data suggest that both mannitol and hypertonic saline are effective for lowering ICP.[19] Hypotensive patients perhaps should not be given mannitol because the subsequent osmotic diuresis may exacerbate hypotension. On the other hand, patients with congestive heart failure or fluid overload may benefit from the diuretic effect of mannitol. However, in most cases, either form of osmolar treatment is appropriate. Administration should be via intravenous bolus on an as-needed basis. Continuous infusion is not recommended, and neither is scheduled bolus dosing, such as every 4 or 6 hours.[19]

If a patient's need for aggressive osmolar therapy continues for a sufficiently long period of time, it is almost inevitable that stopping points will be reached, such as serum osmolality >320 mOsm/L or osmolar gap >20 mOsm/L (possibly increasing this to 55 mOsm/L for persistent ICH).[52] An understudied treatment strategy is to use the minimal effective dose of osmotic agent so that osmolar treatment can be continued for the longest possible duration before reaching a stopping point, which would necessitate escalation to additional therapies.

For example, if a bolus of 23.4% saline is desired, administration of only 10 or 20 mL may be just as efficacious as giving larger doses. If a bolus of mannitol is administered, many practitioners often follow with a bolus of normal saline in a volume that is proportional to the dose of mannitol, such as 25 g of mannitol followed by 250 mL of saline or 100 g of mannitol followed by 1 L of saline. This practice is thought to reduce the risk of hypovolemia and hypotension from the osmotic diuresis that follows repeated boluses.

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Ideally, cerebral edema will peak and then begin to recede before osmolar therapy must be stopped. One way to conceptualize this question is to think of a race to see which of two possible endpoints occurs first. Will ICP elevations respond to sedation, CSF drainage, and osmolar therapy for enough days to allow cerebral edema to subside and ICP to normalize? This is the preferred outcome. Or will stopping points defined by serum osmolality or osmolar gap be reached while refractory ICH persists, necessitating progression to the next level of therapy? Further study is needed to clarify the effect of judicious dosing of osmolar treatments on delaying the arrival of osmolality-based stopping points and, more importantly, on patient outcome.

Fourth Step: Mild Hyperventilation

The traditional next step in the management pathway is consideration of mild hyperventilation, often to a $PaCO_2$ in the low 30s (when measured in mm Hg). Lowering of $PaCO_2$ results in constriction of the cerebral vasculature. The resulting decrease in cerebral intravascular volume is relatively slight but often sufficient to lower ICP.

However, caution is needed when deploying this intervention. Excessive cerebral vasoconstriction may adversely affect outcome, presumably from reduction of cerebral blood flow to levels that are inadequate for an acutely injured brain, which is already highly vulnerable to secondary insults like hypoxia or hypotension.[53] These situations may be ideal for using $PbtO_2$ monitoring to prevent excessive hyperventilation.

Another problem with hyperventilation for prolonged management of ICH is that its effects are transient (Fig. 4). Even when $PaCO_2$ is maintained at the same low level, the cerebral vasculature immediately begins to return to its baseline diameter, a process that is complete in less than 24 hours.[40] However, sudden restoration of normal $PaCO_2$ after many hours of hyperventilation causes the brain's vasculature to dilate well beyond its baseline diameter. A common scenario is for a well-intentioned but misguided clinician to adjust ventilator settings for a sTBI patient in whom $PaCO_2$ has been low for many hours. His or her goal is to bring $PaCO_2$ back up to the normal range. However, almost immediately after this change in ventilation, ICP begins to increase dramatically because the cerebral vasculature quickly dilates. This sudden spike in ICP frequently results in emergency trips to the CT scanner and initiation of aggressive therapies to lower the ICP. It would have been better if the ventilator settings had been left alone or, best of all, if hyperventilation had not occurred in the first place.

Current thinking is that hyperventilation should be used only as a temporizing measure in appropriate patients until definitive treatment can be provided.[39] For example, a comatose patient with sTBI who presents with a fixed and dilated pupil and contralateral weakness can be assumed to harbor an intracranial mass lesion, and hyperventilation may be initiated to buy sufficient time to obtain a CT scan and take the patient to the operating room.

For Use at Any Time: MAP Challenge

In normal cerebral pressure autoregulation, the cerebral arteries dilate or constrict over a broad range of CPPs. This activity maintains cerebral blood flow at a constant level even when blood pressure varies widely. In the past, it was often stated that cerebral autoregulation was absent in sTBI patients. In fact, total absence of autoregulation is not seen in most cases, but it is true that autoregulation may be impaired.

Pressure autoregulation can potentially be harnessed to help control ICP. This concept has been recognized and used by many practitioners over the years and has been formalized as a MAP challenge.[19,54] In brief, baseline values for MAP, ICP, $PbtO_2$, and other cerebral physiologic parameters of interest are recorded, and then infusion of a pressor is carefully initiated to raise MAP by 10 mm Hg. After a few minutes at the higher MAP, the same parameters are again recorded. In a pressure-passive state with absent or severely impaired autoregulation, the increased MAP is transmitted directly to the cerebral vasculature, which dilates, causing increased ICP. In such cases, MAP should be returned to its original value.

However, if autoregulation is even partially intact, the cerebral vessels will constrict in response to the elevated MAP. This reduction may lower ICP, and it may be advantageous to keep the MAP at the higher level. Although the current generation of neurointensivists and neurosurgeons has been taught to control MAP and systolic blood pressure closely in order to prevent development or expansion of traumatic cerebral hemorrhagic lesions, the risk/benefit analysis in these cases often favors a slight increase of blood pressure.

Final Interventions

When ICH persists despite all the above interventions, clinicians have traditionally resorted to three options.

Barbiturate Coma

Barbiturate-induced coma may be achieved with phenobarbital or pentobarbital. Because of the very long half-life of the former, pentobarbital became a more common choice, but even this drug requires a considerable amount of time to be metabolized before a reliable neurological examination can be performed.[55] Pressors should be immediately available because the myocardial depressant effects of pentobarbital may cause hypotension. In general, barbiturate coma is not used as frequently as it once was because its effects are prolonged and its prophylactic or therapeutic use has not been shown to improve outcomes.[56]

Hypothermia

When used prophylactically, hypothermia has not been associated with improved outcomes in clinical trials, even when initiated in the prehospital setting.[57–59] Possible explanations for these negative results are numerous, including problems with achieving target temperature in a timely manner,[57,60] prohibitively long time intervals between injury and induction of hypothermia in even the most aggressive clinical settings,[57,60] inherent differences between humans and experimental animals in body size and brain structure,[60] de facto hypocapnia caused by maintenance of "normal" arterial pH during induced hypothermia,[60] and failure to focus this treatment only on the subpopulations most likely to benefit.[61]

As a therapeutic tool, induced hypothermia can lower ICP in some sTBI patients. However, as with barbiturate coma, enthusiasm for its use has waned in part because of the failure of clinical trials to demonstrate improved outcomes, mirroring the lack of effect of prophylactic hypothermia in these patients.[57–59,62,63] Suggestion of benefit in specific subgroups may serve as the impetus

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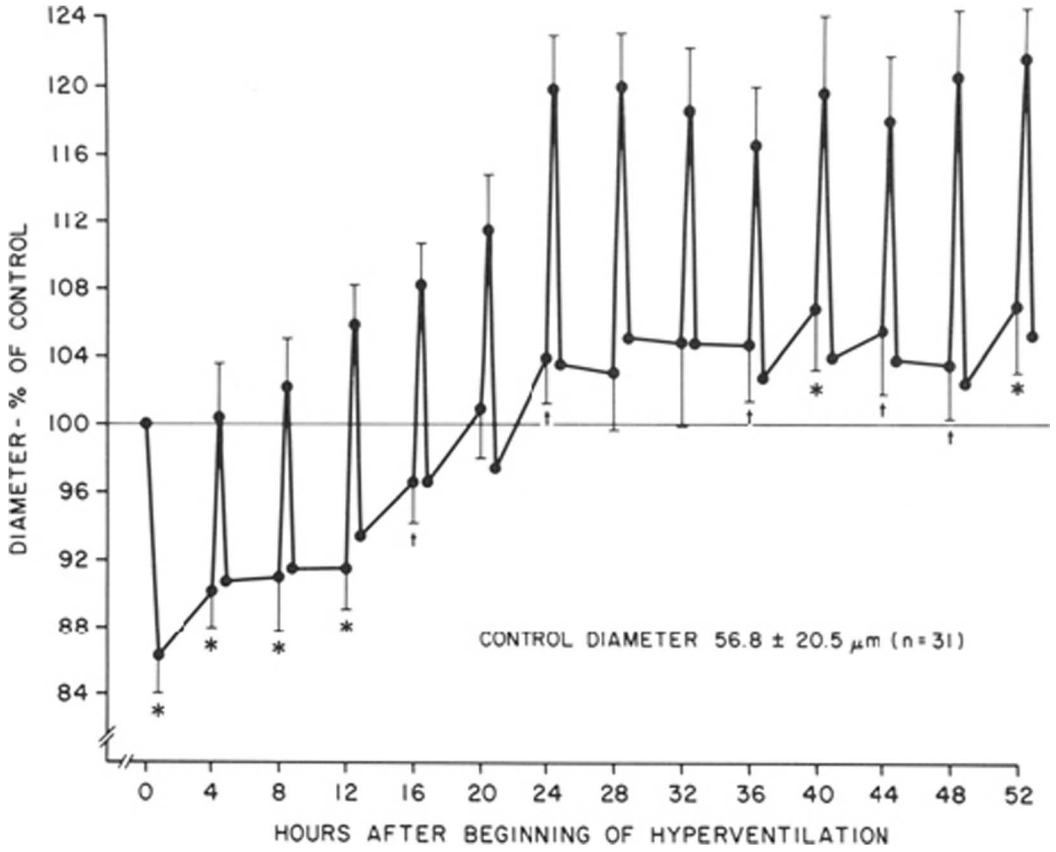


Figure 4. Graph showing the diameter of pial arterioles in rabbits at 30 minutes and at 4-hour intervals after the beginning of hyperventilation. The percentages of control values are shown (mean ± SEM). Lower values were obtained at a $PaCO_2$ of 25 mm Hg, and peak values were taken during brief periods of normoventilation to a $PaCO_2$ of 38 mm Hg. Values marked with an asterisk are significantly different from control at $p < 0.05$, and values marked with a dagger at $p < 0.1$. Reproduced with permission from Ref. [40]. An Open Access or Creative Commons publishing model conveys no rights to use this material in any format without written permission from the JNS Publishing Group.

for future trials in more focused patient populations,[61,64] but for the time being, temperature modulation in sTBI patients focuses on maintaining normothermia and avoiding fevers.[44]

Decompressive Craniectomy

Several decades ago, performance of DC became increasingly popular for TBI patients in whom nonsurgical management had failed to control refractory ICH. However, the efficacy of this intervention was debated.[65] The Decompressive Craniectomy (DECRA) trial examined the role of early DC in sTBI patients who demonstrated 15 minutes of ICP >20 mm Hg within the first 48 hours after injury.[66] The very early surgery in patients with minimally elevated ICP suggests that this study can almost be viewed as a trial of prophylactic DC. Prespecified analysis demonstrated worse outcomes in the surgery group, but importantly, the surgery group had a statistically significant percentage of patients with nonreactive (but not necessarily dilated) pupils. When this discrepancy was accounted for in post hoc analyses, outcomes between the two groups did not differ.[66] Critics point out that many clinicians would have tried more aggressive nonsurgical interventions before going to surgery so quickly,[67] and/or they would have accepted a higher ICP.[68] Also, an operation other than the bifrontotemporoparietal decompression that was part of the study protocol would have been preferred by many. The BTF's 2020 update of the recommendations for DC states that secondary DC for early refractory ICP elevation is not recommended to improve outcomes.[69]

As a therapeutic intervention, DC is commonly used to treat refractory ICH. Evidentiary support can be found in the prospective, randomized, outcome-blinded RESCUEicp (Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure) trial, in which sTBI patients were treated by a standardized protocol for controlling ICP.[70] If ICH persisted despite other interventions, patients were randomized to DC or best medical management. Outcomes were assessed at 6 months.

The authors concluded that there was benefit to performing DC as a salvage procedure in appropriate patients. However, their definition of favorable outcome merits discussion. Traditionally, when the eight-category Glasgow Outcome Scale—Extended[71] is used to assess outcomes, the bottom four categories (death, vegetative state, lower severe disability, and upper severe disability) have been considered unfavorable outcomes, whereas the top four categories (lower moderate disability, upper moderate disability, lower good recovery, and upper good recovery) were favorable outcomes. However, the RESCUEicp investigators

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specified a priori that they would consider upper severe disability to be a good outcome. Using this classification scheme, DC was found to improve outcomes. However, if the traditional favorable/unfavorable distinction is used, then it is difficult to demonstrate a beneficial effect of DC as a salvage therapy in sTBI patients with refractory ICH.

A prespecified 24-month analysis of RESCUEicp confirmed that the survival benefit of DC persists but continues to be offset by higher rates of vegetative state, severe disability, and moderate disability, reinforcing the need for careful shared decision making with families when considering surgery.[72]

The RESCUE ASDH (Randomized Evaluation of Surgery with Craniectomy for Patients Undergoing Evacuation of Acute Subdural Haematoma) trial focused on patients undergoing surgical evacuation of a traumatic SDH. They were randomized to have the bone flap either replaced or left off.[73] Neurological outcomes between the two groups did not differ. However, as could perhaps be predicted, the patients in whom the bone flap was replaced had a higher rate of return to the operating room for subsequent removal of the bone flap, whereas those in the craniectomy group had a higher rate of wound-related complications, partly because of additional surgery to replace the bone in a delayed manner.

Of course, clinicians must judge the applicability of any clinical trial results to the specific patient at whose bedside they are standing. Overall, DC can often reduce ICP and prevent death in sTBI patients with refractory ICH, but these patients are often left with significant and life-changing neurological deficits. Because intensivists and surgeons are predisposed to aggressive action, some of the hardest decisions to make are those in which DC is deemed inappropriate because of a patient's poor neurological examination, CT scan findings, underlying medical status, and/or other factors. Not surprisingly, families are often devastated by their loved one's injury and desperate for any intervention, but a clinician who thinks that DC is inappropriate in a given patient should not accede to pressure from eager but misguided family members.

Another important consideration is that DC is not a benign operation. Complication rates as high as 30% to 50% have been reported, including wound infection or breakdown, subdural CSF collections, ventriculomegaly, hydrocephalus, syndrome of the trephined, headaches, cosmetic defects, and infection of the cranial prosthesis after cranioplasty.[74]

POTENTIAL FUTURE DIRECTIONS

Some tools for lowering ICP or guiding management of ICH that have been discussed previously, such as prophylactic lumbar drainage or $PbtO_2$ monitoring, may see expanded use in the future. Serial measurement of biomarker concentrations in CSF and/or peripheral blood may someday be used to monitor the trajectory of brain injury and thus inform patient management.[75] Cerebral microdialysis is another monitoring modality that can shed light on the status of cerebral metabolism,[76] but the requisite equipment and training of personnel have so far limited its diffusion into general practice. Noninvasive monitoring technologies may acquire a more defined role in clinical decision making, especially when used for serial assessments. Potential examples include optic nerve sheath diameter ultrasound, transcranial Doppler pulsatility indices, and quantitative pupillometry.[77–79] Integration with teleneurocritical platforms could extend these capabilities to remote or resource-limited settings.[80] Sophisticated bedside integration of multiple streams of data from different monitoring devices may offer new insights into cerebral pathophysiology and its management,[81] just as artificial intelligence–based analytics may uncover previously unrecognized avenues for management of ICH.

In the near future, the optimal strategy for management of ICH may be increasingly guided by the context of the injury and the environment in which care is delivered. For example, severe blast-related TBI in combat casualties who will require prolonged transport to sites of definitive care may best be managed by aggressive cranial decompression during their initial neurosurgical treatment.[82] Additionally, a role may become better defined for decompressive craniotomies in which the bone flap is allowed to "hinge" or "float" by loosely securing it on only one side.[83] More broadly, individualization of care may become more widespread, and thresholds for intervention and targeted therapeutics may vary according to patient demographic and clinical factors, the status of cerebral pressure autoregulation, and integrated data from multimodal monitoring.

CONCLUSION

Even after decades of investigation, management of ICP and ICH in sTBI remains grounded in simple but durable principles: maintenance of physiologic homeostasis, prevention of secondary injury, and judicious escalation of therapies. Prophylactic use of ICP-lowering interventions offers no proven benefit, while vigilant monitoring and timely, stepwise treatment remain essential. Future progress will likely depend on identification of individualized treatment thresholds informed by multimodal monitoring, appreciation of ICP burden rather than static cutoffs, and integration of ICP control into a broader strategy that prioritizes both survival and functional recovery. A thorough understanding of the pathophysiology of ICH remains the cornerstone for improving outcomes in these critically ill patients.

AUTHORSHIP

A.B.V. contributed in the conception and design. A.B.V., B.B., L.A.D., and A.S. contributed in the literature review. A.B.V., B.B., L.A.D., R.A.A., and A.S. contributed in the drafting of the manuscript. A.B.V., B.B., L.A.D., R.A.A., and A.S. contributed in the critical revision.

DISCLOSURE

Conflicts of Interest: Author Disclosure forms have been supplied and are provided as Supplemental Digital Content (http://links.lww.com/TA/F143).

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