Polytrauma Considerations

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KEYWORDS

• Decompressive craniectomy • Polytrauma • Severe traumatic brain injury • Multiple system trauma • Secondary injury • Raised intracranial pressure • Cranial defect

KEY POINTS

- The polytrauma patient with severe traumatic brain injury is particularly vulnerable to secondary injury.
- Appreciation of basic principles of trauma resuscitation and their interplay with evolving secondary injury is essential to direct care that is both neuroprotective and neuroproactive.
- Decompressive craniectomy (DC) may be performed early—with emergent intervention—or later, to manage refractory raised intracranial pressure (ICP); polytrauma may necessitate modification of this clinical paradigm.
- Decompressive craniectomy produces several measurable changes to the physiology and metabolism of the injured brain.
- Judicious use of DC in the setting of polytrauma requires ongoing individualized assessment, with the ultimate goals of mitigating secondary injury and optimizing outcomes.

INTRODUCTION

Decompressive craniectomy (DC) is an essential tool in the armamentarium of the neurosurgeon managing malignant cerebral edema with resultant refractory raised intracranial pressure (ICP) in a variety of settings–from trauma to vascular calamity to tumor to infection. The indications for and timing of DC are detailed in David J. Caldwell and colleagues' article, "Primary and Secondary Decompressive Craniectomy in Traumatic Brain Injury: Indications and Timing"; and Michael J. Gigliotti and Francis J. Jareczek's article, "Indications and Timing in Ischemic Infarction and Other Disorders," in this issue. The timing of this intervention is dictated by the underlying pathology and its progression, as well as patient-specific factors such as age, additional injuries, medical comorbidities, hemodynamic stability, blood dyscrasias, and severe metabolic derangement. This article will explore the narrow topic of how comorbid polytrauma impacts these considerations.

EPIDEMIOLOGY OF POLYTRAUMA WITH COMORBID TRAUMATIC BRAIN INJURY

Understanding the epidemiology of polytrauma allows the clinician to anticipate likely combinations of injury and their relative severity. The terms *polytrauma*, *multiple system trauma*, and *major* trauma often are used interchangeably. Yet, the term *polytrauma* lacks a validated definition. *Polytrauma* was initially used to denote 2 or more comorbid injuries.[1] Tscherne and colleagues[2] advanced a definition of greater than or equal to 2 severe injuries, whereby one—or the sum total of all—injuries was considered life-threatening. Others have tried to define polytrauma in terms of Injury Severity Score (ISS) or Abbreviated Injury Score, though there is no consensus regarding threshold values.[3,4] Any discussion of outcomes among patients with polytrauma, therefore, is limited by heterogeneity of injury classification. Still, a few generalizations can be made. The primary determinant of mortality in the polytrauma patient is

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Abbreviations

ABP	arterial blood pressure
ARDS	acute respiratory distress syndrome
CBF	cerebral blood flow
CPP	cerebral perfusion pressure
DC	decompressive craniectomy
DECRA	Decompressive Craniectomy in Diffuse Traumatic Brain Injury
ICP	intracranial pressure
ISS	Injury Severity Score
PRx	pressure reactivity
RESCUEicp	Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure
TBI	traumatic brain injury

often the severity of the head injury.[5] The presence of head injury may confer an up to 3-fold increase in mortality.

A large European registry cataloging 26,541 trauma cases offers some perspective on the impact of polytrauma in general and more specifically, the impact of traumatic brain injury (TBI) in this setting.[6] Fifty percent of registry cases included a diagnosis of head or thoracic trauma. Thoracic trauma was seen principally in conjunction with polytrauma, whereas two-thirds of head injury patients presented with significant TBI alone. Polytrauma with abdominal injury was associated with the highest rate of mortality across all age groups (33.1% ages 16–65 years, 64.1% age > 65 years); comorbid head injury demonstrated the next highest mortality rate (29.3% ages 16–65, 56.2% age > 65 years). Isolated head injury accounted for 12.9% of deaths among patients 16 to 65 years of age and 35.4% of those older than 65 years. The authors noted that mortality was greater for *polytrauma* than for the sum of the individual injury components. Though only 16% of patients in the registry met criteria for polytrauma, this subgroup accounted for nearly half of the recorded deaths (43%). The remainder was due primarily to a combination of isolated TBI and limb fractures in elderly patients. While the incidence of polytrauma among elderly patients was lower, such patients demonstrated mortality rates twice that of younger adults. A more recent prospective series of polytrauma patients in the Netherlands revealed an overall decrease in deaths (18%, with <1% because of exsanguination), with a concomitant significant increase in deaths attributable to TBI (66% of those recorded), more than the 10-year study period.[7]

Even moderate TBI doubles predicted mortality when associated with extracranial injury.[8,9] Regel and colleagues[10] analyzed their experience in the management of 3406 polytrauma patients more than a 20-year period in Germany. Sixty-nine percent of patients presented with TBI. The most frequent combination was head and extremity injury (63%). Increasing total injury severity correlated with increased head injury severity. Prognosis was worse with injuries involving the brain, thorax, and extremities. Mortality was significantly higher in the presence of head injury (30.5% with TBI vs 24.4% without).

SECONDARY INJURY AND IMPLICATIONS FOR THE POLYTRAUMA PATIENT WITH SEVERE TRAUMATIC BRAIN INJURY

As the initial resuscitation effort unfolds, the clinician must consider that the polytrauma patient with TBI is particularly susceptible to the effects of secondary injury. Secondary injury unfolds over the minutes, hours, and days after the primary insult. While radiographic intracranial hemorrhage is certain to generate a request for neurosurgical consultation, secondary injury is not visible on the initial CT head, and therefore may not be foremost in the minds of the frontline trauma team treating the patient. The primary brain injury—with disruption of the blood-brain barrier—exposes neuronal cells to circulating immune cells and inflammatory mediators, setting into motion a series of inflammatory cascades. The release of cytokines, chemokines, and complement anaphylotoxins may drive leukocytes across the abnormally permeable blood-brain barrier, leading to further inflammation and injury of neural tissue.[11,12] Additional insults—hypoxemia, hypotension, ischemia, cerebral edema, and raised ICP—all threaten to exacerbate this inflammatory response. There is also evidence to suggest that polytrauma patients presenting with concomitant musculoskeletal injury are subject to the activation of multiple inflammatory cascades in parallel.[13,14]

Challenges unique to the military experience—such as the distances involved in and physiologic stress posed by air evacuation—offer valuable insight regarding the risk of secondary injury and its triggers. In one series of 438 patients with moderate-to-severe TBI—259 with polytrauma (59%) and 179 without—conditions associated with an increased risk of secondary injury included temperature (T) greater than 99.5°F (61%), serum sodium (Na) less than 145 mmol/L (46%), partial pressure of oxygen (P_{O_2}) less than 80 mm Hg (19%), partial pressure of carbon dioxide (P_{CO_2}) greater than 40 mm Hg (49%), and systolic blood pressure less than 110 mm Hg (44%).[15] In a secondary analysis of the same cohort, those with

polytrauma who were subjected to early (within 1 day) evacuation out of theater were likely to experience at least one episode of hypotension *en route* and had a greater likelihood of receiving blood products.[16] Those with polytrauma tended to be evacuated later and endured a more prolonged hospital course. However, no difference in mortality was observed between groups. The authors concluded that patients with polytrauma may warrant more extensive resuscitation/stabilization measures before transport.

Indeed, the clinical manifestations of secondary injury are insidious and can be lethal. The Trauma Coma Data Bank study demonstrated that a single episode of hypotension between injury and arrival to the trauma center doubled mortality, increased morbidity, and was associated with poorer outcomes.[17,18] Similarly, hypoxia—which may be present at the scene in up to 60% of cases—has been associated with higher mortality and worse neurologic outcomes.[19,20] Stocchetti and colleagues observed hypotension in nearly one-quarter (24%) and hypoxemia in greater than half (57%) of patients with TBI (mean GCS 7) in the field. While 80% of those with preserved blood pressure and saturation in the field went on to favorable outcomes, the prognosis for those with compromise of both was uniformly poor. Chi and colleagues[21] reported that nearly 40% of isolated TBI patients sustain a secondary insult (hypotension, hypoxia, or both) in the field and that the presence of hypoxia resulted in a significant increase in the odds of mortality (odds ratio [OR] 2.66, $P<.05$). Therefore, care must be taken to avoid hypotension and hypoxia.

Jeremitsky and colleagues[22] examined physiologic factors that might impact outcomes over the first 24h after injury, finding that hypotension, hyperglycemia, and hypothermia were associated with increased mortality, whereas hypocapnia, acidosis, and hypoxemia were associated with significantly longer intensive care unit length of stay. Cerebral blood flow (CBF) has been shown to be at its nadir in the first 24 hours after TBI, and low CBF at any point postinjury has been associated with worse neurologic outcomes, underscoring the critical need to support blood pressure and oxygenation.[23] Using xenon CT, Marion and colleagues[24] demonstrated that low CBF in patients—without mass lesions—in the first few hours after injury was followed by a 24-hour period of hyperemia. Low CBF in the first 24 hours was associated with (but not statistically significant for) poor outcome. Data derived from the Trauma Coma Data Bank patients suggested that avoidance of the shock state would have provided an estimated 9.3% reduction in unfavorable outcomes, prompting Piek and colleagues[17] to recommend vigilance for the recognition of hypotension through the period of triage and transition to the intensive care unit setting, coupled with prompt and aggressive treatment to restore normal perfusion.

INITIAL CLINICAL DECISION-MAKING IN POLYTRAUMA WITH SEVERE TRAUMATIC BRAIN INJURY

The patient presenting with multisystem trauma should be managed in accordance with ATLS guidelines.[25] Initial clinical decision making is driven principally by hemodynamic stability. If hemodynamically stable, the central nervous system injury may take precedence. However, for the hypotensive trauma patient, hypovolemia because of ongoing hemorrhage is the presumption; this should trigger aggressive resuscitation and if indicated, emergent damage control intervention on the part of the trauma team. ATLS guidelines recommend emergent laparotomy for control of bleeding in the setting of a positive Focused Assessment with Sonography for Trauma scan with hemorrhagic shock. Interestingly, intra-abdominal injury requiring emergent operative intervention is relatively less common among patients with severe TBI; yet, undiagnosed abdominal injury is the primary cause of death in patients presenting with both abdominal injury and TBI.[26] A recent study by Bazmi and colleagues[27] investigated the incidence of comorbid abdominal and pelvic injuries among patients with TBI (approximately 15%). In this retrospective series, the need for DC was independently associated with an increased likelihood of requiring abdominopelvic surgery, and those who underwent such surgeries demonstrated an increased rate of in-hospital and 6-month mortality, as well as worse outcomes at 6 months.

Identification of severe TBI during the initial trauma systems survey should prompt a series of calculations on the part of the consulting neurosurgeon. If a patient with depressed level of consciousness demonstrates lateralizing findings, a space-occupying process—with the possibility of raised ICP—should be presumed. The neurosurgeon should be given the opportunity to assess the patient to determine whether surgical intervention is indicated, reasonable, and feasible. If the patient is hemodynamically unstable, attention to resuscitation—with a goal of restoring mean arterial pressure and preserving cerebral perfusion pressure (CPP)—becomes the immediate end point. Hypoxia should be avoided; ventilation parameters should encourage oxygenation and

discourage hypercarbia. While hyperosmolar therapy may be considered in the absence of ICP monitoring for patients with acute neurologic decline and/or signs of herniation, bolus-dose mannitol may exacerbate hypotension and potentially contribute to expansion of an extra-axial hematoma, if present.[28]

Diagnostic CT imaging should be obtained as soon as clinical stability permits to guide further therapy. The neurosurgical consultant should engage the trauma team and other surgical subspecialists to *inventory* the involved organ systems, including the need for extracranial interventions. Remember that actions taken to support one organ system may have physiologic consequences for the injured brain. Appreciation of basic principles of trauma resuscitation and their interplay with evolving secondary brain injury is essential to direct care that is both neuroprotective and neuro-proactive. Effective communication among care providers and ongoing, individualized assessment of risks and benefits for a given procedure is invaluable in navigating the optimal management of the polytrauma patient.

THE ROLE OF DECOMPRESSIVE CRANIECTOMY IN POLYTRAUMA

The most recent guidance from the Brain Trauma Foundation offers a Level IIA recommendation for secondary DC to address *late* (but not *early*) refractory raised ICP to improve mortality and favorable outcomes in the setting of severe TBI.[29] This update to the 4th edition document followed publication of the 12-month outcome data for the Decompressive Craniectomy in Diffuse Traumatic Brain Injury (DECRA) trial, as well as release of the Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure (RESCUEicp) study. Yet, there is little—if any—evidence-based guidance in the literature around the topic of DC in the specific setting of polytrauma. In a recent retrospective cohort study of patients undergoing operative intervention for acute traumatic subdural hematoma, craniectomy was not significantly associated with poor outcomes in univariate or multivariate analysis, but both hematoma thickness ($\geq$20 mm) and the presence of polytrauma were significantly associated with poor outcome in multivariate analysis.[30] Application of a Classification and Regression Tree algorithm using these two parameters, coupled with age (threshold 54 years old), yielded 3 subgroups of patients, among which those with polytrauma had the worst and overwhelmingly poor prognosis (91% unfavorable).

It is curious that none of the major published DC trials from the past 15 years—DECRA, RESCUEicp, or Randomized Evaluation of Surgery with Craniectomy for Patients Undergoing Evacuation of Acute Subdural Hematoma—specifically address the influence of comorbid polytrauma on outcomes for patients presenting with severe TBI and an indication for craniectomy.[31–33] None of these trials specified polytrauma among inclusion or exclusion criteria or stratified outcomes through the lens of multisystem injury or comorbidities. Only DECRA reported the ISS (median 33 for DC, 32 standard care) and Trauma Score—ISS (median 0.74 for DC, 0.72 standard care). Historically, an ISS greater than or equal to 16 has been accepted as the threshold for *major trauma* (with a predicted mortality of 10%) and an increased likelihood of associated multisystem injury.[34] Per personal communication from the senior author for the RESCUEicp trial, there was a trend toward higher mortality in polytrauma DC patients (34%) as compared with DC patients with isolated TBI (28%) (Hutchinson PJ, Kolias AG, personal communication, 2025). However, a lack of formal, stratified secondary outcome data in the context of these studies may leave the reader questioning the extent to which multisystem injury contributes to the observed primary outcomes for these patients.

Of course, these studies also differ with respect to the timing of the surgical intervention relative to the primary injury. DC may be performed early—in conjunction with an emergent procedure indicated for evacuation of a mass lesion—when, in the opinion of the operating surgeon, reimplantation of the bone flap would risk contributing to ongoing ICP elevation. DC may also be performed later in the patient's course as a definitive measure for the management of medically refractory raised ICP. The author would assert that in cases of polytrauma, it is sometimes necessary to consider decompression outside of this traditional clinical paradigm in order to accommodate the many moving parts of a multisystem insult. There is sometimes a subjective *therapeutic window* whereby *earlier* decompression might be considered—between initial resuscitation and the point at which the natural history of any comorbid injuries might induce progressive, overwhelming systemic physiologic distress. Examples of such distress may include acute respiratory distress syndrome (ARDS), hemodynamic instability, significant metabolic derangement, febrile episodes, and/or abnormal hematologic parameters. Recognizing this opportunity requires astute clinical judgment to weigh the current ICP burden and its likelihood of progression against the impact that worsening cardiopulmonary and/or renal function may have secondarily on the intracranial process.

Once this window closes, there may be a period during which the patient is simply too medically ill

to tolerate a trip to the operating room under general anesthesia and with potential significant blood loss. What typically follows is an arms race of escalating measures to control ICP and maintain adequate CPP via medical means. It has been the author's experience that the general trauma team may respond to the clinical *sign* of raised ICP with increasingly aggressive interventions—some of which may have unintended, deleterious consequences for the injured brain (such as secondary hypotension and hypoperfusion)—when a more targeted approach to treat the underlying systemic process (whether it be ARDS or fever or metabolic derangement) would facilitate achieving the intended goal. The consulting neurosurgeon must maintain awareness of the impact that treatment decisions to support one organ system may have on another. Ongoing, multidisciplinary communication is key. In such cases, DC may be considered once the patient is deemed medically stable to tolerate the stress of operation and after reassessment of the patient's neurologic status at that time.

CONSEQUENCES OF DECOMPRESSIVE CRANIECTOMY IN THE POLYTRAUMA PATIENT WITH SEVERE TRAUMATIC BRAIN INJURY

DC produces several measurable effects on the injured brain, aside from those that are radiographically evident: volume expansion, improvement in midline shift, and opening of basal cisterns. The DECRA trial confirmed that ICP will decrease in the postcraniectomy state. Other studies have corroborated this finding, with the additional caveats that ICP reaches a nadir soon after craniectomy, before gradually increasing over the first 24 to 48 hours postprocedure, and ultimately stabilizing at a new set point.[35–37] However, compliance (Δ volume/Δ pressure) may be a more sensitive indicator of the evolving relationship between pressure and volume within the intracranial compartment. The compliance of an *open skull* (postcraniectomy) system is necessarily different than that of a closed system. Abdullah and colleagues[38] explored the impact of DC on invasively measured brain compliance (Spiegelberg GmbH & Co.), as well as pressure volume index (PVI = the volume that when added to the cerebrospinal fluid space would produce a 10-fold rise in ICP), jugular bulb oximetry, brain tissue oxygen ($PbtO_2$), and CBF. Preoperative compliance—abnormal in all but 1 of 17 patients—quickly increased to the normal range in 12 of those patients following decompression. Similarly, the PVI—abnormal in all patients preoperatively—normalized in 12 of 17 patients. Precraniectomy and postcraniectomy values for the other indices demonstrated no significant change.

The response of $PbtO_2$ to craniectomy has also been investigated. Stiefel and colleagues[39] demonstrated a 114.8% increase in $PbtO_2$ (21.2 ± 13.8 mm Hg to 45.5 ± 25.4 mm Hg) following decompression; they also observed an immediate and sustained decrease in ICP, coupled with an increase in CPP. Interestingly, there was not a robust relationship between the change in $PbtO_2$ and change in ICP postprocedure ($R^2 = 0.3$), suggesting other factors might be at play. Lubillo and colleagues[40] published a retrospective, observational series (n = 42) exploring whether postcraniectomy changes in $PbtO_2$ might predict outcome. While a general increase in $PbtO_2$ was observed postoperatively, patients who ultimately achieved favorable outcomes experienced a more pronounced improvement over the first 24 hours after craniectomy; these patients also tended to have a higher admission $PbtO_2$ and a lower percentage of time at a subthreshold value preoperatively. Unilateral craniectomy was associated with a significantly greater increase in mean $PbtO_2$ values than bifrontal craniectomy at the 12-hour and 24-hour time points postprocedure; the reason for this observed difference was uncertain.

Cerebral hemodynamic parameters are also affected by decompression of the injured brain. Wang and colleagues[41] tracked the cerebrovascular pressure reactivity (PRx) of patients undergoing DC for severe TBI. While the initial mean postprocedure PRx remained positive—consistent with ongoing microvascular dysfunction—subsequent measurements trended toward a more negative value, indicating progressive improvement in function. For patients managed conservatively, no significant change in PRx was observed across time. Timofeev and colleagues,[37] likewise, noted an initial increase in PRx but also observed that adequate CPP could be achieved at significantly lower levels of arterial blood pressure (ABP) postdecompression. Xenon-enhanced CT has been used to investigate quantitative global and regional CBF predecompression and postdecompression. In one small retrospective series of patients (n = 27) undergoing primary DC for TBI, the majority (81%) demonstrated normal to hyperemic global CBF postdecompression, as well as preserved CBF under the defect (70.4%); areas of hypoperfusion postcraniectomy were felt to reflect contusion and/or devitalized brain.[42] Those in the early mortality group tended to manifest a mismatch between ICP (normal) and $PbtO_2$ (low), as well as regional cerebral hypoperfusion superimposed upon global hypoperfusion, postdecompression.

This effect has also been demonstrated in the context of stroke, whereby perfusion CT performed 6 hours before surgery and 12 hours postprocedure demonstrated a significant increase in CBF to all measured territories, as well as an increase in cerebral blood volume within the penumbra.[43] Reduced CBF and cerebral blood volume preoperatively was posited to reflect reduced CPP and impaired autoregulation. Mean transit time and time-to-peak residue function (Tmax) were not affected by craniectomy.

Taken together, these data underscore the importance of continuing multimodality monitoring postdecompression in order to detect persistent physiologic dysfunction and respond to changes that might occur because of the intersection of the brain injury with the effects of injury to other organ systems (in particular, the lungs). While monitoring and targeting ICP within normal limits is perhaps most familiar to clinicians, these data also suggest that there may be some benefit to evaluating cerebrovascular autoregulatory capacity in the postoperative period.[44] For the patient with impaired autoregulation and cerebrovascular PRx, there may be value in maintaining ABP and CPP (50–60 mm Hg) at a relatively lower level, whereas mitigating the potential harms of hyperemia and edema. A patient with intact autoregulation, on the other hand, may tolerate higher levels of CPP (~70 mm Hg), if indicated.

In addition to the physiologic repercussions of decompression, the physical cranial defect may present its own set of challenges. Care must be taken to keep pressure off the defect area, whereas maintaining neutral head and neck alignment (even more problematic if a cervical collar remains in place). This requires vigilance on the part of the clinician. A well-positioned, moldable foam pillow beneath the occiput will raise the head and prevent pressure necrosis along the incision line. Be mindful of the fact that a ventilator circuit under any tension may inadvertently pull the head toward the side of the defect; consciously direct the endotracheal tube to the side contralateral to the defect to prevent this occurrence. While there is no explicit contraindication to prone positioning to assist management of ARDS following craniectomy, caution must be exercised to avoid dislodgment of indwelling invasive monitors as well as to avoid any pressure at the site of the cranial defect.[45] Recognize that dependent shifting of intracranial contents toward the defect may occur, potentially perturbing function of an intraventricular catheter or exacerbating tension along a suture line. The head of bed may be elevated in the prone position, and appropriate sedation, as well as measures to maintain adequate ABP and CPP, should be continued.

Indeed, the presence of the cranial defect must be taken into account during routine, scheduled positioning changes in bed, as well as if any additional operative interventions are anticipated. Nonsupine positioning for neurosurgical procedures may limit or require creativity with respect to skull pin placement. Ongoing communication with critical care staff and other surgical subspecialists is essential to maintain awareness of potential orthopedic interventions that might require lateral decubitus positioning or plastics interventions, whereby an approach to the face or forehead area might inadvertently result in pressure applied to the defect area through the sterile drapes. A helmet should be procured for use during transfers and when the patient is eventually mobilized.

Finally, the presence of a cranial defect has implications with respect to the diagnostic testing that accompanies declaration of death by neurologic criteria. It should be acknowledged that the mere presence of a skull defect does not provide an indication for ancillary testing.[46] Though cerebral angiography demonstrating the absence of intracranial CBF is considered the gold standard reference, caution must be exercised in the interpretation of this study for the postcraniectomy patient. A selective power injection of the common carotid and vertebral arteries may seem to *fill* the candelabra when, in fact, no meaningful spontaneous blood flow is present, resulting in a false-negative.[47] A similar phenomenon has been observed with computed tomography (CT) Angiography (though this modality is not recommended as a confirmatory test because of insufficient evidence). In fact, 1 prospective observational study demonstrated apparent CBF by perfusion CT in 2 postcraniectomy patients for whom death by neurologic criteria had been confirmed by clinical examination as well as electroencephalogram (EEG) and evoked potential findings.[48] The observed *flow* in these cases may reflect increased compliance of an *open skull* system or decreased intracranial pressure (providing diminished resistance to blood flow) within the intracranial compartment. There are also case reports of false-negative CBF detected by transcranial Doppler in postcraniectomy patients with documented electrocerebral silence by EEG.[49] Brain perfusion scintigraphy, by contrast, seems to be less sensitive to the presence of a cranial defect. One retrospective study demonstrated no difference in the observed scintigraphy result—with or without craniectomy—among patients with clinical evidence of brain death, perhaps suggesting a role for preferential selection of brain perfusion scintigraphy as a confirmatory test when a craniectomy has been performed.[50]

SUMMARY

The patient presenting with severe TBI in the setting of polytrauma demands a systematic approach to care that emphasizes multidisciplinary involvement and effective communication among team members. Clinical decision-making regarding the indications for and timing of DC, in this context, may not conform to traditional paradigms. Nimble clinical judgment is necessary to seize the *therapeutic window* for decompression–between the initial resuscitation and point at which the natural history of comorbid injuries may produce overwhelming systemic physiologic distress; once that interval closes, the patient may be too medically ill to tolerate a major invasive intervention for an extended period. DC, itself, induces several measurable effects on the physiology and metabolism of the injured brain; continued multimodality monitoring in the postoperative period is essential to detect ongoing derangement of pressure, cerebrovascular, and brain tissue oxygen indices, as well as potential impacts related to dysfunction of other organ systems. The presence of a cranial defect creates physical challenges to positioning and has repercussions with respect to the diagnostic testing performed in conjunction with assessment for death by neurologic criteria. Judicious use of DC in the setting of polytrauma requires individualized assessment, balancing the risks and benefits of invasive intervention to relieve raised intracranial pressure relative to the physiologic impact of injuries affecting other organ systems, with the goals of mitigating secondary injury and optimizing outcomes.

CLINICS CARE POINTS

- The primary determinant of mortality in the polytrauma patient is the severity of the traumatic brain injury (TBI).
- Polytrauma patients are particularly susceptible to secondary injury in the setting of TBI.
- It is essential to maintain awareness of the impact injury to other organ systems may have on the injured brain, as well as the management of raised intracranial pressure (ICP). Optimize the medical management of underlying injuries to mitigate secondary injury to the brain.
- The timing of DC in the polytrauma patient with TBI may not conform to traditional clinical paradigms and may be performed *earlier,* in anticipation of worsening systemic effects of multisystem injury or *later,* in cases whereby those effects predominate earlier in the clinical course.
- DC induces several measurable changes to the physiology and metabolism of the injured brain, underscoring the importance of continuing multimodality monitoring postdecompression.
- The presence of a cranial defect–while not, in and of itself, an indication for ancillary testing during brain death assessment–may affect interpretation of the results of such studies, resulting in false-negatives. Cerebral scintigraphy may be less sensitive to the altered compliance of an open skull system.

DISCLOSURE

The author has nothing to disclose.

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