CME

The Surgical Approach to the Management of Increased Intracranial Pressure After Traumatic Brain Injury

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Increased intracranial pressure occurring after severe traumatic brain injury is a common and potentially devastating phenomenon. It has been clearly demonstrated that increased intracranial pressure that is refractory to initial medical measures is a poor prognostic sign. Current optimal management is based on a sequential, target-driven approach combining both medical and surgical treatment strategies. The surgical measures in current common practice include external ventricular drain insertion and decompressive craniectomy. There is evidence that both of these measures reduce intracranial pressure but the effect on outcome, particularly in the long term, is equivocal. Current Brain Trauma Foundation guidelines recommend timely evacuation of mass lesions and there is clear guidance regarding the indications for intracranial pressure monitoring; however, decompressive craniectomy is only cautiously recommended as a possible option for selected patients. In this review, we highlight the ongoing debate about the use of decompressive craniectomy to control intracranial pressure after traumatic brain injury; included is a summary of review of the most recent literature on the effect of decompressive craniectomy on increased intracranial pressure after traumatic brain injury and associated long-term outcome. The RESCUEicp and DECRA studies are discussed in detail. It is hoped that these 2 randomized controlled trials, which are evaluating the short- and longer-term outcomes of decompressive craniectomy, will provide conclusive evidence regarding the role of decompressive craniectomy in managing increased intracranial pressure after trauma. (Anesth Analg 2010;111:736–48)

Head injury is extremely common, resulting in approximately 1 million United Kingdom emergency department attendances and 20,000 inpatient consultations annually.[1] Although representing only a small proportion (approximately 4%) of all traumatic brain injury (TBI) cases, severe TBI is responsible for considerable morbidity and mortality.[2] An uncontrolled increase in intracranial pressure (ICP) is a poor prognostic factor in closed head injuries. Studies have consistently reported a decrease in both survival and proportion of good outcomes in those patients whose increase in ICP could not be managed.[3–7] A poor prognosis is especially associated with an increase in ICP within the first 24 hours after injury[3] and secondary (3–10 days posttrauma) increases in ICP.[8,9] The causes of such ICP increases are numerous and not solely associated with the nature of the original injury (Table 1).[10,11]

Herein, we discuss the use, benefits, and complications of the common surgical interventions for managing increased ICP after TBI. As part of this discussion, a review of the recent literature on the effects of decompressive craniectomy (DC) on ICP control and long-term outcome has been conducted. The details of the ongoing RESCUEicp and DECRA trials, which are 2 multicenter investigations into the use of DC as a second-tier therapy for control of ICP post-TBI, will also be presented.

LITERATURE SEARCH METHODS

PubMed and the Cochrane Library were searched using the search string "Decompressive craniectomy AND traumatic brain injury." An original search with MeSH terms "decompressive craniectomy" AND "traumatic brain injury" produced very few results and this strategy was deemed to be not adequately inclusive. A total of 194 articles were found. Studies involving either adults or children, using DC of any type (bilateral/unilateral) and that assessed the effect of DC both on ICP *and* outcome, either mortality or functional, were included. Of these, studies in which DC was performed for primary mass lesion, which did not assess both ICP and a functional outcome, and those not in English were excluded. One study examining the effect of bilateral DC on prognosis after the development of bilateral/contralateral mass lesion after an initial unilateral DC was also excluded.

ICP MANAGEMENT AFTER TBI

An optimal approach to managing TBI patients is to anticipate the onset of increased ICP. Neurosurgical services should be involved early in both assessment and

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Table 1. Causes of Post-TBI Increased ICP[10,11]

- Cerebral edema
- Hyperemia
- Mass lesion: epidural hematoma; subdural hematoma; hemorrhagic contusions; depressed skull fracture; foreign body
- Cerebral vasodilation
- Systemic hypertension
- Hydrocephalus
- Venous sinus thrombosis or any other obstruction
- Posttraumatic seizure activity (status epilepticus, subclinical seizures)
- Increased intrathoracic or intraabdominal pressure, caused by mechanical ventilation, agitation, or abnormal motor posturing
- Hyperthermia or febrile states
- Lightening from coma with inadequate sedation

TBI = traumatic brain injury; ICP = intracranial pressure.

Table 2. Indications for Intracranial ICP Monitoring[17]

- Severe head injury (defined as GCS score ≤8 after cardiopulmonary resuscitation) *plus*
  (a) Abnormal admitting head CT scan *or*
  (b) Normal CT scan *plus* ≥2 of: age >40 years, systolic blood pressure >90 mm Hg, decerebrate or decorticate position
- Sedated patient; patient in induced coma after severe TBI
- Multisystem injury with altered level of consciousness
- Patient receiving treatment that increases risk of increased ICP, e.g., high-volume IV fluids
- Postoperatively after removal of intracranial mass
- Abnormal values in noninvasive ICP monitoring, increased dynamics of simulated values, or abnormal shapes in transcranial Doppler blood flow velocity waveform (increased pulsatility) with exclusion of arterial hypotension and hypocapnia

ICP = intracranial pressure; GCS = Glasgow Coma Scale; CT = computed tomographic; TBI = traumatic brain injury.

treatment planning. Better mortality rates are observed in patients with head injuries who are treated in neurosurgical centers, even when their head injury does not necessitate neurosurgical intervention.[12] Patients deemed to be at high risk of developing increased ICP should be nursed in a regional neurosurgical unit with the option for care in a specialized neurological intensive care unit (ICU). Not only does this allow for adequate observation and management of any drains and monitors placed but the protocol-drive therapy by a specialist multidisciplinary team provided in these units results in better outcomes, especially for those patients presenting with evidence of increased ICP.[13]

Anticipating and Monitoring Increased ICP

In patients initially presenting with mild or moderate TBI, the symptoms of increasing ICP may be vague and nonspecific, such as confusion, headache, and drowsiness. The most fundamental clinical variable to determine in TBI patients is the Glasgow Coma Scale (GCS) score. A decreasing value, especially of the motor component, is a potential indicator of increasing ICP. The GCS score also determines how a patient's neurological status may be monitored; sedation or a low GCS score requiring tracheal intubation will preclude clinical assessment of ICP, for example, vomiting, ocular palsies, or headache, and will thus require ICP monitoring. This is currently invasive, by intraparenchymal monitors or ventricular catheterization. Noninvasive approaches include ultrasound sonography techniques, which are not yet fully validated.[14–16]

ICP monitoring is not necessary in the awake patient, in whom clinical assessment of neurological status is possible, and is contraindicated in the patient with a bleeding diathesis. In the latter case, all effort should be made to correct this if ICP monitoring is required. Brain Trauma Foundation (BTF) guidelines suggest that ICP monitoring is primarily used when there is difficulty in clinical assessment of the patient or if there is a high risk of increased ICP (Table 2).[17] However, fewer than half of the patients on the United States National Trauma Data Bank from 1994 to 2001 who met these criteria actually underwent ICP monitoring.[18] Moreover, the same literature review found a decrease in survival associated with monitoring, even after controlling for overall injury severity, TBI severity, craniotomy, associated injuries, comorbidities, and complications. ICP monitoring is beneficial if the monitored values are included in a formal and reasonable management protocol.

The unenhanced computed tomographic (CT) head scan is the primary investigation that can show clinically nonobvious abnormalities associated with increased risk of developing increased ICP as well as those amenable to surgical treatment. These include extraparenchymal and intraparenchymal hemorrhages, basal skull fractures, hydrocephalus, and cerebral edema. The CT may also show evidence of cerebral mass effect, such as midline shift, effacement of sulci, and compression of basal cisterns and ventricles; although there are no definitive CT features that indicate increased ICP, these features have been associated with increased risk of developing increased ICP.[19] Furthermore, an abnormal CT scan increases the risk of subsequent intracranial hypertension; 60% of patients with a closed head injury who have an abnormal CT scan developed increased ICP compared with only 13% of those with similar injuries but a normal presenting CT scan.[20] In these patients with a normal presenting CT scan, 3 clinical features were strongly associated with subsequent development of increased ICP: the age (>40 years), systolic blood pressure (<90 mm Hg), and evidence of decerebrate or decorticate posturing. Indeed, the combination of ≥2 of these 3 clinical features with normal CT head scan or GCS score <8 with abnormal CT head scan meets the BTF guideline criteria for ICP monitoring.[17]

Thus, careful and continuous clinical assessment of the patient must always have an important part in determining the most appropriate course of action and it should be reviewed regularly during the course of any implemented treatment plan.

Management of Mass Lesions

Most patients presenting with extraparenchymal mass lesions that are causing brain compression or midline shift should undergo timely surgical evacuation because the lesion will contribute to the development of increased ICP and secondary brain injury (Table 3).[21] However, there still remains some debate about the value of surgical evacuation in intraparenchymal lesions, such as contusions. Current practice reflects the contrast between readiness to surgically evacuate extraparenchymal hematomas and a more conservative approach adopted for managing intraparenchymal lesions.[22] There is some retrospectively acquired

Table 3. Mass Lesions Requiring Expedient Surgical Removal[21]

- Acute extradural hematoma: volume >30 cm^3 as measured on CT scan
- Acute subdural hematoma: thickness >10 mm *or* midline shift >5 mm as measured on CT scan
- Acute subdural hematoma: thickness <10 mm or midline shift <5 mm *but* GCS score <9, which decreased by ≥2 points between injury and admission *and/or* presenting with fixed dilated pupils *and/or* ICP >20 mm Hg
- Intraparenchymal lesion: CT evidence of mass effect *or* increased ICP refractory to medical treatment *or* progressive neurological deterioration referable to lesion
- Frontal/temporal contusion: volume >50 cm^3 as measured on CT scan *or* GCS score 6–8 and volume >20 cm^3 and midline shift >5 mm/compression of cisterns
- Posterior fossa lesion: mass effect on CT *or* neurological deterioration *or* deterioration referable to lesion
- Lesions not fulfilling these criteria may be conservatively managed along with serial imaging and close monitoring

CT = computed tomographic; GCS = Glasgow Coma Scale; ICP = intracranial pressure.

Table 4. Studies, Using Different ICP Thresholds, Investigating Difference in Outcome Between Patient Groups in Which ICP Was Controlled and in Which There Was Failure to Control ICP

Reference	ICP threshold studied (mm Hg)	Outcome
Miller et al.,[3] 1981 (prospective case series)	20	Mortality 18% if ICP < threshold 92% if ICP > threshold "Good outcome" 74% if ICP < threshold 3% if ICP > threshold
Marshall et al.,[4] 1979 (retrospective)	15	"Favorable outcome": GOS score 4–5 77% if ICP < threshold 43% if ICP > threshold
Balestreri et al.,[26] 2006 (retrospective)	20–30 "Critical ICP" threshold may be variable and should be individualized	Mortality 17% if ICP < threshold 47% if ICP > threshold
Saul and Ducker,[27] 1982 (prospective case series)	25	Mortality 15% if ICP < threshold 69% if ICP > threshold

GOS = Glasgow Outcome Scale; ICP = intracranial pressure.

evidence that patients with traumatic intracerebral hemorrhage have better outcomes if they undergo surgery[23,24]; however, these have all been single-center studies. The newly established STICH (Trauma) multicenter randomized controlled trial (RCT) will be able to more definitely evaluate the role of surgery for traumatic intracerebral hemorrhage.[25] An ICP monitoring device may be inserted at the same time as evacuation and, in any case, ICP monitoring should continue after evacuation to detect any secondary increases in ICP.[17]

The Target ICP

Higher mortality and morbidity rates were observed in those patients whose ICP was persistently >20 mm Hg[5]; various studies using different ICP thresholds have all demonstrated the poorer outcomes associated with the failure to control ICP (Table 4).[3,4,26,27] It has been suggested that early initiation of treatment, before the ICP reaches 20 mm Hg, results in better ICP control but this is from older literature.[27] The duration of intracranial hypertension also contributes to outcome.[7] The tolerated threshold may also be related to the nature of the injury, with the results of an early study suggesting that those patients with mass lesions may tolerate higher ICP increases (<40 mm Hg) and still obtain good outcomes, whereas those patients with diffuse brain injury may have poor outcomes with smaller increases in ICP (<10 mm Hg).[28] Nevertheless, most centers initiate treatment when the ICP is more than 20 to 25 mm Hg, and the BTF guidelines support the initiation of treatment when ICP is ≥20 mm Hg.[29]

The poor outcome associated with uncontrolled increased ICP may not entirely be a result of the effect on cerebral perfusion pressure (CPP) and there is conflicting evidence regarding the relative importance of ICP and CPP in final outcome. Juul et al.[30] observed that increased ICP ≥20 mm Hg in those patients whose CPP remains >60 mm Hg is an independent prognostic factor for poor outcome. However, in a small case series of 9 patients with TBI, the 4 patients in whom CPP was aggressively maintained >60 mm Hg had good outcomes (Glasgow Outcome Scale score of 4) despite having experienced prolonged episodes of ICP >40 mm Hg.[31] Of note, however, are the other 5 patients in this case series who did not survive their injuries; they all experienced episodes of ICP >75 mm Hg. These 5 patients did not undergo the same aggressive CPP-targeted therapy so it is not clear from this series if there is indeed an ICP limit that renders CPP targets futile. Nevertheless, despite potential differences in management protocols and the small number of subjects in this case series, both groups studied CPP ≥60 mm Hg, which suggests that the 4 patients in the second study may be genuine exceptions to the conclusions drawn by Juul et al. The relative importance of treating ICP and CPP after TBI is also a matter of continuing debate.

The Lund protocol is a volume-targeted approach with the dual aim of reducing or preventing increased ICP and improving perfusion around contusions. Its methods endeavor to optimize outcome by reducing mean arterial blood pressure to reduce intracerebral volume, and thus ICP, while maintaining microcirculation with drugs such as clonidine. For example, in contrast with CPP-guided approaches, the Lund protocol will accept periods of lower CPP levels (<50 mm Hg) to reduce ICP. Clinical trials using its therapeutic strategies with or without additional measures, such as low-dose prostacyclins,[32] have shown low mortality rates.[33,34]

A prospective RCT examining the effects of ICP-driven management versus CPP-driven management of severe TBI found no ultimate difference in neurological outcome between the 2 treatment groups, even though jugular venous desaturations were more frequent when ICP was the target for treatment.[35] The authors attribute this finding to the increased frequency of systemic complications, notably adult respiratory distress syndrome, observed when the CPP was the treatment target. It should be noted that the CPP target in this study was ≥70 mm Hg, whereas the

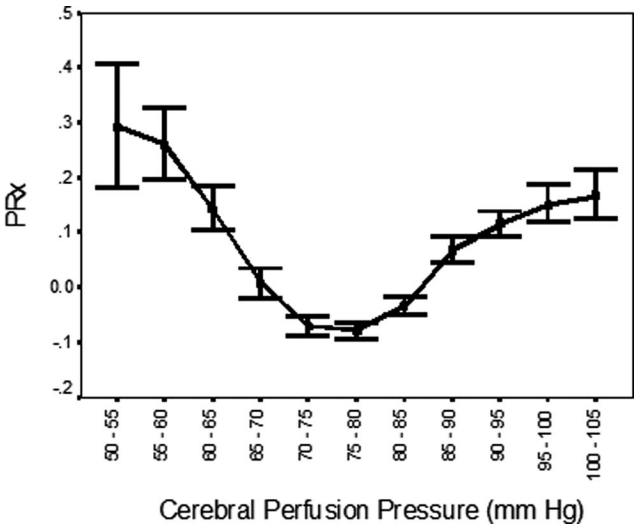

Figure 1. Plot of pressure reactivity index (PRx) (a greater value indicates poorer cerebrovascular reactivity) versus cerebral perfusion pressure in nearly 100 patients with traumatic brain injury (TBI). (Cambridge data: reproduced from Steiner et al.,[39] with permission.)

more recent BTF guidelines suggest a CPP target of 50 to 70 mm Hg but no more than 70 mm Hg.[36] Furthermore, there is the suggestion that CPP measurements should be combined with secondary monitoring modalities including cerebral oximetry or biochemistry to define the appropriate target on an individualized basis.[37,38]

More recently, the concept of an "optimal CPP" (CPP_{OPT}) has emerged, which 1 study has proposed as being the CPP value that is associated with the best possible index of cerebrovascular pressure reactivity (PRx), not necessarily the highest achieved value.[39] Calculated by computational methods, the PRx is the correlation coefficient between 40 consecutive averaged data points from 8- to 10-second windows of ICP and arterial blood pressure; a positive value indicates that ICP increases as arterial blood pressure increases, thus there is loss of normal cerebrovascular reactivity to pressure changes, consistent with poor autoregulation.[40,41] This study demonstrated that the greater the absolute difference between the patient's mean CPP and their CPP_{OPT}, the greater the likelihood of poor outcome on the Glasgow Outcome Scale.[39] The relationship of PRx and CPP has a U shape; values of CPP that are either too low or too high may result in detrimental PRx values, which are not helpful in avoiding ischemic brain insults after TBI[39,40] (Fig. 1).

Nevertheless, ICP management cannot be independent of CPP management, as is reflected by current ICP management protocols, which usually have both ICP and CPP targets. This topic has been extensively reviewed elsewhere.[38,42] The current British Trauma Foundation recommendations are to maintain ICP <20 mm Hg and CPP 50 to 70 mm Hg.[36]

SURGICAL INTERVENTIONS IN ICP PROTOCOLS

The mainstay of ICP management is medical. Our center operates a protocol that includes head elevation, adequate oxygenation, fluid resuscitation, sedation and muscle relaxation, mild hyperventilation ($Paco_2$ approximately 4.5 kPa) to reduce cerebral blood volume, and cooling (Fig. 2).[43] Although the exact nature of increased ICP management protocols may vary among centers, the most common strategy is a step-wise increase in intensity of treatment to reach the targets in ICP and CPP. The alternative "longitudinal" approach directs several treatments simultaneously as a likely cause of the increased ICP. Surgical intervention has a role in diagnosis, monitoring, and especially as a second-tier treatment for those patients whose increased ICP is refractory to maximal medical management. In our center, surgical decompression for increased ICP after TBI is performed as part of the RESCUEicp trial, in which patients whose ICP is refractory to first-line medical therapy (including sedation, paralysis, and mild hypothermia) are randomized to receive either DC or continue with maximal medical therapy that includes the use of barbiturates. Decompression for mass lesions is performed outside the remits of the trial and according to BTF guidelines.[21]

VENTRICULAR CATHETERIZATION

Use for ICP Monitoring

ICP can be monitored either through intraparenchymal systems, such as monitors placed through cranial access bolts,[44] or ventricular catheterization. On the whole, initial readings from intraparenchymal and ventricular systems have been reported as having comparable accuracy.[45] The relative advantage of ventricular catheterization with placement of the external ventricular drain (EVD) is in the potential for simultaneous ICP monitoring and cerebrospinal fluid (CSF) drainage. However, care should be taken during the interpretation of monitored ICP values obtained when the EVD is open; in the majority of such arrangements, the monitored ICP value is drastically different from intraparenchymal ICP.[46] Mass lesions sustained during TBI and defective CSF cycling after TBI may result in pressure gradients and thus differences between intraparenchymal and ventricular values.[47–49]

Use for CSF Drainage

Where ICP is monitored using intraparenchymal systems rather than EVD insertion, the use of subsequent ventriculostomy for CSF drainage is suitable as a second-tier therapy for controlling increased ICP refractory to initial measures. Neurosurgical assessment of ventricular size before insertion is important because CSF drainage from EVDs may fail to reduce ICP if there is insufficient volume of CSF within the ventricles, for example, from mass effect. Because the aim of this intervention is ICP control, the extent of drainage should be guided by the effect on ICP. A reasonable initial drainage rate is 10 to 15 mL per hour. Removal is indicated when ICP has been normal for 48 to 72 hours after withdrawal of ICP therapy; before removal, the EVD should be clamped for 12 to 24 hours and the patient's neurological status monitored carefully to ensure that removal is appropriate.

Ventricular catheterization as a method for ICP control has been investigated in a small number of prospective clinical studies.[50–52] All of them found that CSF drainage from ventriculostomy was effective at producing an immediate decrease in ICP to below pathological levels. In 1 study, which followed the ICP over 72 hours, >50% of the patients retained a stable low-level ICP over this time period (Fig. 3).[50] In this study, the patients whose ICP

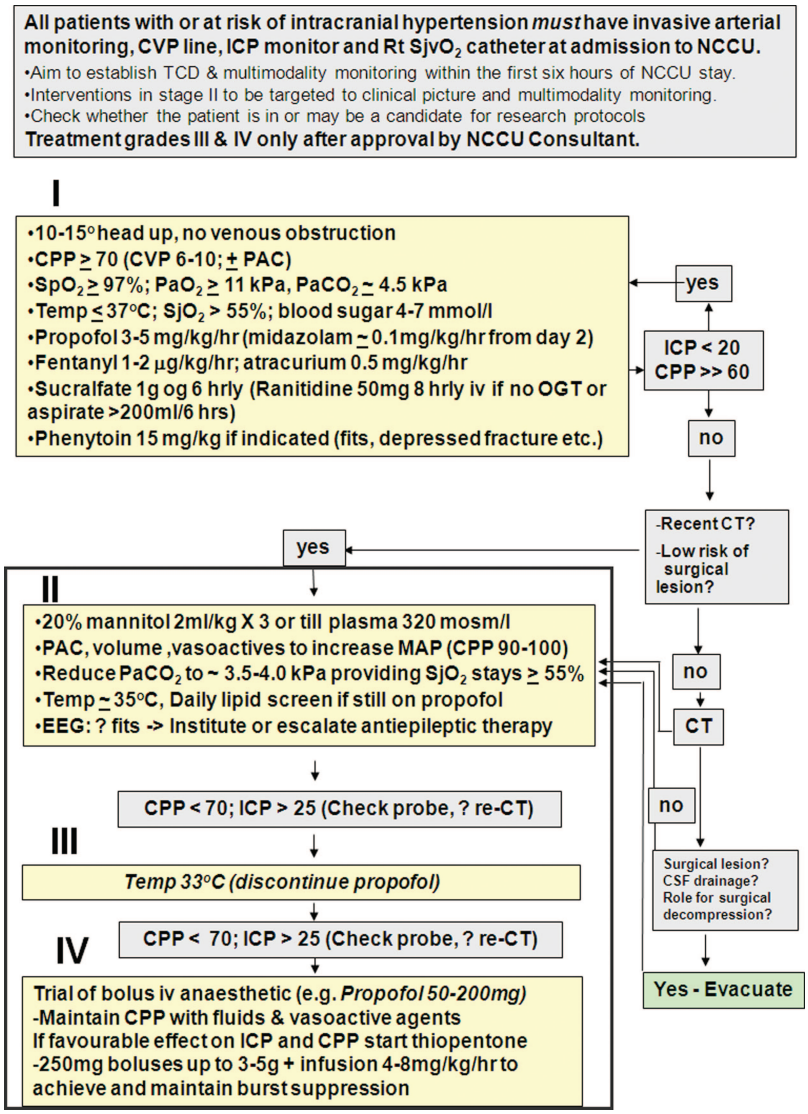

Figure 2. The intracranial pressure (ICP)/cerebral perfusion pressure (CPP) management algorithm for the Neuro Critical Care Center in Addenbrooke's Hospital, Cambridge, UK. CT = computed tomography; CVP = central venous pressure; EEG = electroencephalogram; MAP = mean arterial pressure; NCC/NCCU = neurological intensive care unit; OGT = oral-gastric tube; PAC = pulmonary artery catheter; (Rt) SjO_2 = (right) jugular bulb oxygen saturation; TCD = transcranial doppler; SpO_2 = oxygen saturation as measured by pulse oximetry. (Reproduced from Menon,[43] with permission.)

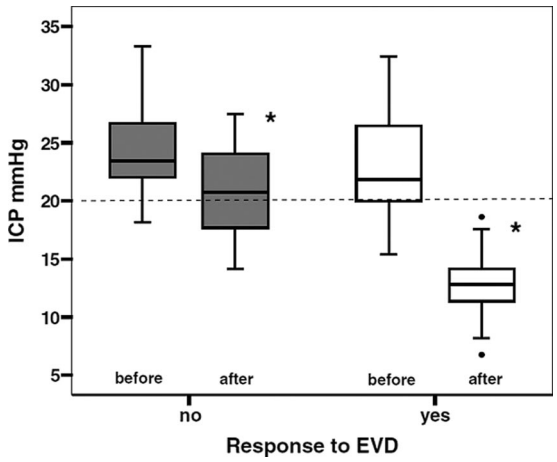

Figure 3. Mean intracranial pressure (ICP) values before and 72 hours after insertion of external ventricular drain (EVD) in 2 groups whose ICP either remained low ("Yes") or whose ICP had exceeded 20 mm Hg by the end of 24 hours after insertion ("No"). Both groups showed initial decrease in ICP to <20 mm Hg and 13 of the 24 patients ("Yes") maintained ICP levels <20 mm Hg. *Statistical significance ($P < 0.001$). (Reproduced from Timofeev et al.,[50] with permission.)

showed a sustained response to EVD insertion were able to have reduced intensity of ICP treatment and the reduced ICP was also associated with significant improvement in cerebral oxygenation, CPP in the presence of lower mean arterial blood pressure, and pressure-volume compensatory reserve,[50] but these changes have not been observed in all such studies.[52] The pressure-volume compensatory reserve is assessable by the RAP index, which is the correlation coefficient of the mean pulse amplitude of ICP and the mean ICP; a value of 0 indicates no correlation and thus good reserve whereas a value of 1 indicates poor reserve and a negative value occurring at high ICP levels indicates deranged cerebrovascular reactivity.[40] Furthermore, there is evidence to suggest that using an EVD for ICP control is also associated with favorable outcome at postdischarge follow-up.[53] Thus, given the relatively safe, albeit invasive, nature of this procedure, an EVD would be a reasonable second-tier ICP management option if practically possible. Indeed, many centers use early ventricular catheterization to enable concurrent CSF drainage and ICP monitoring.

Surgical Technique

EVD placement can take place in an operating room or in the ICU. Neuro navigation equipment may be used if there is radiographic evidence of small ventricles or any other factor that might affect the usual trajectory of EVD placement. EVDs are usually placed via a bur hole into the frontal horn of the lateral ventricle, usually on the right-hand side (nondominant); intraventricular hemorrhage in the right would direct the EVD placement to the left ventricle. With the patient lying at 30°, the external auditory meatus is approximately on the same horizontal plane as the foramen of Monroe, thus this is the landmark frequently used as 0 for the monitor scale for EVDs monitoring ICP via an external transducer. The height of the drain may be adjusted to either increase or decrease the drainage.

Complications

Complications include infection, hemorrhage, and malfunction, for example, a nondraining EVD from a blocked drain. Failure to drain CSF may also be associated with the injury itself; in the presence of a subarachnoid hemorrhage, failure to drain CSF was 4.7 times more likely.[52] Nevertheless, assessment of a nondraining EVD should first exclude modifiable problems such as the drain height. Patency may be assessed by injecting 1 mL normal saline into the EVD and assessing backflow. The ICP monitoring function may also be assessed by observing for natural respiratory variations in the ICP waveform or lowering the head of the bed to horizontal and observing an increase in ICP. A persistently nonfunctioning EVD should be removed, with or without the view to placing another one, so that infection does not occur. If a functioning EVD fails to reduce ICP adequately and control is not achieved with further medical measures, other surgical measures, such as DC, may be considered.

DECOMPRESSIVE CRANIECTOMY

Use and Benefits

Among the first modern proponents of surgical decompression, Kocher, in 1901, stated that "if there is no CSF but brain pressure exists, then pressure relief must be achieved by opening the skull"; since then, several variations of this procedure have been, and continue to be, performed.[54] Surgical decompression is theoretically appealing. The inelastic nature of the skull results in the nonlinearity of the relationship between volume and pressure observed within the cranium, thus it seems logical to remove a portion of the calvarium to move the system back to a more favorable portion of the pressure-volume curve (Fig. 4).[55] However, since its introduction more than a century ago, the practice of using DC as an ICP control measure has repeatedly come into, then fallen out of, favor.

Currently, there is wide variability in the indications for DC among centers. Many centers use DC predominantly as a last-tier treatment option for managing severe refractory increased ICP post-TBI when medical management, which may include hypothermia and paralysis, has failed whereas other centers use early decompression. There are few clear guidelines on its use. The BTF guidelines[21] state only that it is an intervention treatment option, given the appropriate clinical context, that may be used after head injury to manage medically refractory cerebral edema and resultant

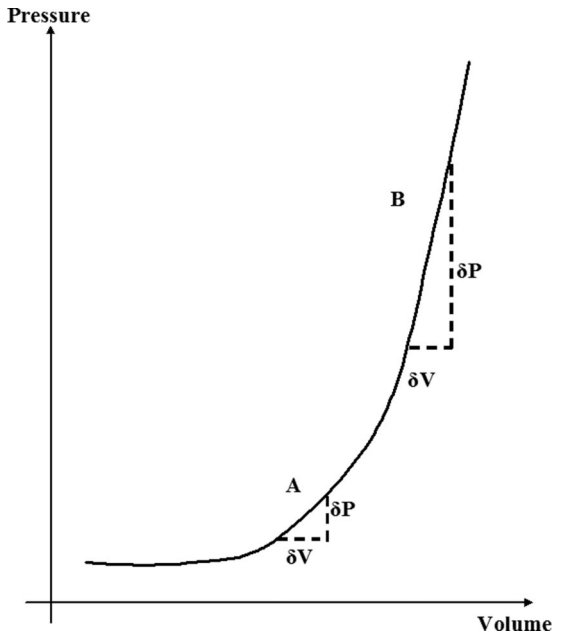

Figure 4. Graph depicting relationship between pressure and volume within the skull.[55] There is a larger change in pressure (δP) for any given change in volume (δV) at point B than at point A. The injured brain at risk of intracranial hypertension is at point B and surgical decompression aims to move it back to point A.

intracranial hypertension. This uncertainty largely stems from the yet unanswered questions regarding its effect on final neurological outcome, rate and significance of complications, as well as appropriate surgical technique.

In assessing the clinical value of using DC for controlling ICP, numerous outcome measures may be used, including ICP control, mortality, and functional outcome. The latter is especially important to assess because a major argument against the routine use of DC for ICP control is that this procedure decreases mortality without a concomitant increase in good long-term functional outcome, leaving many survivors who remain in a persistent vegetative state or are otherwise unable to regain independence in daily life. A review of clinical outcomes after DC in TBI from studies conducted between 1975 and 2006 demonstrates a wide range of outcomes,[56] with mortality rates as high as 90% and as low as 11% and functional outcomes, dichotomized into "favorable" or "unfavorable" based on their Glasgow Outcome Scale score, similarly variable. Table 5 summarizes the numerous studies published since 2006 that have investigated not only the effect of DC on ICP but also its outcome, both mortality and functional outcomes. It also includes a summary of the only RCT performed to examine this matter.[57–69]

The general trend seems to be toward a reduction in mortality with DC. However, improved prehospital TBI care and medical ICP management in the ICU may be a major contributing factor. Furthermore, although DC seems to be an effective method of reducing ICP levels, its correlation to actual clinical outcome is uncertain. Most of the studies summarized in Table 5 demonstrated that DC produces a significant decrease in ICP, although not always to <20 mm Hg, but this effect is not associated with a majority of patients having favorable outcome in all studies, that is, success in

Table 5. Summary of Studies Published Since 2006 Assessing Effect of DC on ICP, Mortality, and Functional Outcome and a Summary of the Only RCT (2001) Assessing Outcomes After DC

		Outcomes			
Study	**Method details**	**ICP control**	**Mortality**	**Functional**	**Additional comments**
Taylor et al.,[57] 2001	27 pediatric patients randomized to receive DC or maximal medical alone, including barbiturate coma; 6-mo assessment of GOS score and quality of life (Health State Utility Index)	Mean ICP was lower in the post-DC group compared with the control group over 48 h, but not significantly so	Reduced risk of death 0.54 (95% CI, 0.17–1.72)	Reduced risk of death, vegetative status, severe disability at 6 mo postinjury 0.54 (95% CI, 0.29–1.07)	Prospective RCT; this is the *only* prospective RCT on DC to date. The DC technique was bone decompression only, which is not common practice for adults
Kan et al.,[58] 2006	51 pediatric patients with TBI had DC; 45 had DC + removal of mass lesion; follow-up at 18 mo	69.4% had ICP normalized immediately postoperatively	31.4% mortality, includes 5 of 6 who had DC for control of ICP only	Mean KOSCHI = 4.5 (4 = moderate disability; 5 = good recovery)	Retrospective. Complications reported: 40% hydrocephalus, 20% epilepsy requiring medication control
Skoglund et al.,[59] 2006	19 of 150 TBI patients underwent DC; bone flap size calculated from postoperative CT scan; phone assessment of GOS score between 1 and 6 y postinjury. "Good outcome" = score 4–5	Mean ICP significantly reduced immediately and 24 h postoperatively *and* <20 mm Hg. Calculated from 9 patients only, who had preoperative ICP monitoring	11%	68% good outcome	Retrospective. Complication rate 47% (mostly associated with reinsertion of bone flap). There is a significant positive correlation between bone flap size and ICP reduction
Aarabi et al.,[60] 2006	967 TBI (closed injury) patients; 50 underwent DC only, no removal of mass lesions. GOS score assessment of survivors at 3 mo. "Good outcome" = score 4–5	85% had ICP reduced to <20 mm Hg immediately postoperatively	28%	40% good outcome; 14% in PVS; 9% severely disabled	Prospective nonrandomized. Good outcomes associated with higher postresuscitation GCS score
Jagannathan et al.,[61] 2007	23 pediatric TBI patients underwent DC in a 10-y period (mean age, 11.9 y); assessed GOS score at 2 y	83% had their elevated ICP controlled immediately postoperatively	30%	Mean GOS score at 2 y = 4.5; 81% of survivors returned to school; 18% dependent on caregivers	Retrospective. Mortality most closely associated with postoperative ICP >20 mm Hg
Olivecrona et al.,[62] 2007	21 of 92 patients with severe TBI had DC for control of increased ICP; GOS score "during recovery period"	Significant ($P < 0.001$) mean ICP reduction: 36.4 to 12.6 mm Hg (leveled to 25 mm Hg at 72 h post-DC)	14% at 3 mo; same mortality rate in both groups	71% favorable (GOS score 4–5) in DC; 61% favorable in non-DC controls	Retrospective. Did not show difference in mortality or outcome compared with control group
Howard et al.,[63] 2008	40 severe TBI patients underwent DC in a 3-y period (16 primarily for increased ICP); GOSE assessed at a mean follow-up period of 11 mo	All 16 patients who had DC primarily for increased ICP had significantly reduced ICP (35.0 ± 13.5 mm Hg to 14.6 ± 8.7 mm Hg, $P <$ 0.005)	55% (of all 40 patients)	30% good outcome overall; 67% good outcome in survivors; GOSE 5–8 = good outcome	Retrospective. Good outcome associated with higher GCS score and smaller pupil size on admission
Timofeev et al.,[64] 2008	27 with moderate to severe TBI underwent DC; ICP monitored postoperatively for 72 h; GOS score assessment at 6 mo ("favorable outcome" = score 4–5)	Median postoperative ICP values significantly lower and <20 mm Hg, for 72 h. RAP index significantly reduced; median CPP not significantly increased; PRx significantly increased	15%	48% favorable; 37% unfavorable; median postoperative PRx significantly higher in poor outcome group	Retrospective. Lower postoperative ICP levels associated with older age; *not* associated with type of craniectomy

(*Continued*)

Table 5. (*Continued*)

Study	Method details	Outcomes			Additional comments
		ICP control	Mortality	Functional	
Figaji et al.,[65] 2008	18 children <15 y old had DC after TBI for initial malignant brain swelling (8 patients) or increased ICP refractory to medical treatment (10 patients); 6 of these 10 patients also had brain tissue O_2 tension measured; continued with other medical, including barbiturates, after surgery; GOS score follow-up between 3 and 84 mo ("favorable" = 4–5)	Pooled ICP (10 patients) statistically lower after DC ($P = 0.0051$); brain O_2 tension (6 patients) significantly higher after DC ($P = 0.027$)	5.6% (1 of 18)	78% favorable; 22% unfavorable	Retrospective analysis of data. In comparison to Taylor et al., this group used a large craniectomy with durotomy. Noted that there was a high incidence of low median preoperative GCS score and papillary abnormalities even in the favorable outcomes group
Qiu et al.,[66] 2009	74 patients randomized into 2 groups to treat post-TBI unilateral brain swelling. One group underwent unilateral DC and the other (control) underwent routine unilateral temporoparietal craniectomy. GOS score at 1 y (good outcome = 4–5)	DC group had lower mean ICP values after 24, 28, 72, and 96 h than control group	27%; control group 57% ($P < 0.05$)	56.8% good outcome; 32.4% in control ($P < 0.05$)	Prospective. Comparison with another surgical technique, not medical treatment
Williams et al.,[67] 2009	172 severe TBI patients (AIS 4–5) treated with DC for increased ICP; GOSE at 1–6 y post-TBI ("good" = 5–8; "poor" = 1–4)	Greater ICP decrease post-DC observed in good outcome group (23 vs 10 mm Hg, $P <$ 0.0001); greater ICP decrease post-DC observed in survivors (22 vs 10 mm Hg, $P = 0.0003$); NB: reported changes in ICP according to survival/ outcome, not actual values.	32%; NB: 22% "head-related." NB: 30% mortality in all patients with head AIS 4–5, whether treated with DC or not	"Good outcome" 56%; "poor outcome" 44%	Retrospective. Control group had maximal medical treatment, including sedation and hypertonic saline. Proposed predictors for good outcome post-DC: younger age, pre-DC ICP (higher in those not surviving), greater decrease in ICP post-DC
Daboussi et al.,[68] 2009	26 severe TBI patients undergoing DC; GOS at 6 mo; also assessed cerebral blood flow diastolic velocity and pulsatility index	Significant decrease in mean ICP ($P \leq 0.0003$); 37 ± 17 mm Hg to 20 ± 13 mm Hg. Diastolic velocity also increased significantly and the pulsatility index decreased significantly, deemed to be "normalisation of global cerebral hemodynamics"	8%	Good outcome or moderate disability in 42%; PVS in 27%	Prospective
Bao et al.,[69] 2010	37 TBI patients with malignant diffuse brain swelling underwent bilateral DC; GOS score at 6 mo	Significant reduction in, but not normal, mean ICP after DC ($P < 0.05$): 37.7 ± 6.4 mm Hg to 27.4 ± 7.2 mm Hg. CPP also showed significant increase after DC ($P <$ 0.05): 57.6 ± 7.5 mm Hg to 63.3 ± 8.4 mm Hg	18.9%	54.1% "favorable outcome" (GOS score 4–5); 10.8% PVS; 16.2% severe deficits	Retrospective. Noted that the most frequently seen complication (18.8% of patients) was hydrocephalus

AIS = Abbreviated Injury Score; CT = computed tomographic; DC = decompressive craniectomy; GOS = Glasgow Outcome Scale; GOSE = Glasgow Outcome Scale Extended; GCS = Glasgow Coma Scale; ICP = intracranial pressure; CI = confidence interval; KOSCHI = Kings Outcome Score for Childhood Head Injury; PVS = persistent vegetative state; PRx = pressure reactivity index; RCT = randomized controlled trial; RAP = index of compensatory reserve; TBI = traumatic brain injury; CPP = cerebral perfusion pressure; NB = *nota bene*.

controlling ICP using DC does not always translate into a favorable long-term clinical outcome.[60,63,68]

Additionally, 1 study demonstrated that a reduction in ICP post-DC is not necessarily accompanied by a statistically significant increase in CPP, possibly because the CPP was already being well maintained before decompression.[64] Furthermore, this study also found that the PRx, a measure of cerebrovascular pressure reactivity, was significantly increased to positive values, suggesting poor reactivity, and the results suggest a correlation with poor outcome. Conclusions are especially hard to draw because these studies do not have control groups with which to compare outcomes. In fact, the 1 study that did report outcomes in a nonmatched control group found no statistically significant difference in mortality and clinical outcome between the group of patients who underwent DC and those who did not.[62] It is both the inconsistency in the outcomes of these studies and their demonstration of uncertain correlation between extent of ICP reduction and favorable outcome that fuel the debate about DC as a post-TBI ICP control measure. It may be that DC does not increase the likelihood of favorable outcome in patients with an initial good prognosis and that it merely shifts the outcome from death to persistent vegetative state/severe disability in patients with an initially poor prognosis.[56]

The current literature on the use of DC for ICP control is largely retrospective and nonrandomized. A Cochrane review was only able to find 1 prospective RCT, which was performed in a pediatric population, with the results supporting the use of DCs for ICP management in the pediatric population.[70] A majority of favorable outcomes was also reported in a more recent, retrospective study in a pediatric population.[65] However, researchers of another, albeit retrospective, study reported a relatively high mortality and complication rate; population characteristics may have contributed, with some patients undergoing DC for increased ICP in conjunction with mass lesions and nearly 25% of patients presenting after nonaccidental injury.[58]

Given the highly invasive nature of DC, along with its associated complications, the results from prospective RCTs are anticipated. Researchers have endeavored to investigate the clinical outcomes associated with DC and maximal medical management compared with medical management alone[57,71]; however, conclusions are difficult to apply because of the small number of participants in these studies. Patient selection is also an issue that remains contentious. High mortality rates in some studies may be a reflection of the injury or some specific patient characteristics that makes patients more likely to become refractory to medical ICP management and hence undergo DC, rather than a reflection of the nature of the procedure. The results of some studies suggest patient factors that may be considered when selecting patients for DC, including preoperative GCS score,[60,63] smaller pupils on admission,[63] and age.[64] Exclusion criteria, which include indicators of severe irreversible damage, such as bilateral fixed and dilated pupils or brainstem involvement, have also been proposed.[72] Tests proposed to be useful in guiding patient selection include assessment of arterio-jugular difference in oxygen content and transcranial Doppler ultrasonography assessment of cerebral blood flow. A high arterio-jugular difference in oxygen content may indicate an alive, and thus salvageable brain that is extracting oxygen[73] whereas the presence of systolic peaks only on transcranial Doppler ultrasonography may indicate irreversible damage.[74] However, these tests are not used routinely to inform for the decision of surgical intervention. As such, there is still no hard evidence to specify the patient groups in which DC would be most beneficial and there are no tests currently that definitively determine suitability of DC. Results of prospective RCTs may enable relevant patient factors and clinical indicators to be further defined.

Surgical Technique

DC has myriad incarnations. Factors to be considered include location (frontal, temporal, parietal, occipital), hemisphere (unilateral or bilateral), size of decompression, and dural technique (scalp closure only, duraplasty with autologous or synthetic patch). The location and hemisphere will largely be determined from the CT scan; unilateral lesions such as contusions, extradural or subdural hemorrhage, or unilateral swelling or midline shift would all be indications for unilateral decompression. Decompression for diffuse cerebral edema with no obvious midline shift on CT scan usually requires a bifrontal decompression, such as has been previously described.[75]

Given the lack of consensus about the efficacy of DC in controlling ICP, it is not surprising that there is little consensus about certain aspects of its technique. A small study of the effect of DC on ICP control in 27 post-TBI patients used both bilateral and unilateral craniectomies and found no significant association between type of craniectomy used and subsequent ICP control.[64] More importantly, the craniectomy must be of a sufficient size to allow decompression without brain herniation and damage at the bony edges. One study showed a significantly positive relationship between size of craniectomy and reduction in ICP.[59] However, a small study of the factors associated with the development of posttraumatic hydrocephalus found that an extended DC potentially predisposes to the condition.[76] Studies that have investigated the difference in outcome between patients who had early versus late decompressions have not always found comparable results. Studies have found favorable outcomes to be associated with late surgery (<24 hours),[77] early decompression (<16 hours),[78] or not associated with timing at all.[79] An important factor that may confound the results of studies into early versus late decompression is injury severity; those patients who have more severe injuries are more likely to require emergency surgery, and subsequent poor prognosis may be more associated with injury severity than the DC performed.[74] The question of optimal timing for a DC merits further investigation in a larger patient population.

Recently, the first comparative study of DC versus hinge craniectomy, in which the bone flap is not completely removed, suggested that hinge craniectomies could perform at least as well as the traditional DC in ICP control without a significantly increased risk of necessitating reoperation.[80] Although this technique is attractive because it removes the need for cranioplasty, which carries complications of its own, the results of this retrospective study should be confirmed in further trials.

Table 6. Potential Complications Arising from Decompressive Craniectomy and Subsequent Cranioplasty[81,82]

Complications arising from decompressive craniectomy
Subdural hygroma (16%–50%)
Progression of hemorrhage/contusion (5%–58%)
Intracranial infection (2%–6%)
Contralateral SDH/EDH (6%–28%)
Hydrocephalus (2%–29%)
Herniation through skull defect (26% in 1 case study defining herniation as brain tissue in the center of the defect >1.5 cm above plane of normal outer table of skull[83])
Syndrome of the trephined: a late complication consisting of headaches, confusion, dizziness, memory difficulties, mood disturbances, and sometimes motor disturbances, consisting of progressive contralateral upper limb weakness not previously affected by injury (10 of 38 patients in 1 case series[84]). In many cases, this syndrome can be reversed by cranioplasty; all patients developing motor symptoms in the case series experienced full and rapid motor recovery within days of their cranioplasty
Paradoxical herniation: has been reported as occurring as a result of lumbar puncture after large decompressive craniectomy[85,86]
Complications of subsequent cranioplasty
Bone flap resorption/sinking after cranioplasty (1.6%–12%)
Infection (11.3%)
Status epilepticus (1.6%): note that seizures after neurosurgical procedures are a well-recognized occurrence

SDH = subdural hematoma; EDH = extradural hematoma.
Percentages (where available) are quoted from those reported from several individual studies.

Complications of DC

Notwithstanding the debate about outcome after DC, both the procedure and the subsequent cranioplasty repair constitute major surgery and are thus associated with complications that must be balanced against the potential benefits of the procedure. There are a wide range of possible complications, none of which are minor or negligible in frequency (Table 6). A recent review of the numerous studies on complications showed a 30% overall complication rate after DC and subsequent cranioplasty[81] and a study of specific complications after bone cranioplasty showed a similar complication rate with 25% of those patients requiring reoperation.[82]

One of the most common complications is development of a subdural hygroma, with or without conversion to hemorrhage or complication by infection.[81] A retrospective comparison of patient and management features between those who did and did not develop hygromas showed that those who had developed subdural hygromas were significantly more likely to have been involved in motor vehicle (high dynamic) accidents and to have diffuse brain injury shown by CT scan.[87] Management features, including surgical management, were not significantly different between the 2 patient groups in this study. However, a study of a different complication, posttraumatic hydrocephalus, found that patients developing this condition were significantly more likely to have had an extended craniectomy (frontotemporoparietal bone flap plus contralateral frontal bone flap with duraplasty) instead of a standard craniectomy (12 × 15 cm bone flap removal with duraplasty) and significantly more likely to have had multiple operations.[76] Furthermore, those patients not developing hydrocephalus had better clinical outcome, although this was not statistically significant, possibly as a result of the small sample size. Other factors observed to increase the complication rate of DC are older age (>60 years old) and lower GCS score.[83] Early cranioplasty has been reported to result in a higher rate of infection.[81] Thus, both patient factors and surgical technique may contribute to the development of certain complications, which should be considered in any protocol written regarding the use of DC in ICP management.

Table 7. Summary of the 2 Multicenter Ongoing RCTs Investigating the Role of DC in TBI

	The RESCUEicp study[88] (www.rescueicp.com)	The DECRA trial[89]
Principal investigators (steering center)	P.J. Hutchinson; P.J. Kirkpatrick (Univ. of Cambridge Academic Dept. of Neurosurgery; European Brain Injury Consortium)	D.J. Cooper; J. Rosenfeld (Alfred Hospital, Melbourne, Australia)
Trial type	International multicenter, prospective RCT	Multicenter (Australia/New Zealand), prospective RCT
Trial aim	To compare neurological outcome with DC vs maximal medical management of increased ICP in TBI	To evaluate the effect of early (within 72 h of injury) DC on neurological function in patients with TBI
Size	610 patients; recruitment ongoing	210 patients; recruitment ongoing
Patient inclusion criteria	10–65 y old; abnormal CT scan; ICP refractory to protocol-based maximal medical ICP management (>25 mm Hg >1–12 h). *Exclusion*: brainstem involvement; devastating injury, patient not expected to survive 24 h; fixed dilated pupils; unable to monitor ICP; patients treated on Lund protocol; primary DC for mass lesion; barbiturates given prerandomization; bleeding diathesis; follow-up not possible	15–60 y old; severe diffuse brain injury; within 72 h postinjury; ICP refractory to optimal conventional ICU management (>20 mm Hg >15 min). *Exclusion*: penetrating injury; mass lesion; spinal cord injury; arrest at scene; no chance of survival; severe coagulopathy; fixed dilated pupils with GCS score of 3
Assessment of main outcome measures	Dichotomized GOSE (favorable = 4–8 vs unfavorable = 1–3); SF-36 (>16 y old)/SF-10 (10–16 y old) Quality of Life Questionnaire. This will be used for health economic analysis of acute and long-term care. At 6 mo, 12 mo, and 2 y postinjury	Dichotomized GOSE (favorable = 4–8 vs unfavorable = 1–3). At 6 mo and 12 mo postinjury
Other outcomes assessed	ICP and other physiological variables; days in ICU and in hospital	Mean and maximal hourly ICP; days in ICU and in hospital; brain metabolites using microdialysis (1 center only)

CT = computed tomographic; DC = decompressive craniectomy; GOSE = Glasgow Outcome Scale Extended; ICP = intracranial pressure; ICU = intensive care unit; SF = short-form health survey with 36 question items (SF-36) or 10 question items (SF-10); TBI = traumatic brain injury; RCT = randomized controlled trial; GCS = Glasgow Coma Scale.

The Future for Decompressive Craniectomies

Much of the uncertainty about the place of DC for ICP control after TBI is a result of the dearth of prospective RCTs available. There are currently 2 ongoing prospective multicenter RCTs (Table 7). The rationale behind both studies is that TBI is both common and has severe physical, psychological, social, and economic consequences; that there is currently no Class I evidence in the form of RCTs for the use of DC to control increased ICP in post-TBI patients; and that such trials will also help to establish the complication rates from this procedure.

Although the primary objective of RESCUEicp is to test the hypothesis that DC results in improved outcome as assessed by the extended Glasgow Outcome Scale, it will also include the Medical Outcomes Questionnaire with 36 items (SF-36) as an important secondary outcome measure, which will additionally assess the impact of using DC on health care systems. The SF-36 Quality of Life Questionnaire covers 8 universally valued dimensions of health and assesses individual perception of health and how current health affects quality of life.[90] No TBI-specific quality of life questionnaire is in current widespread use but the SF-36 has been validated for use in TBI patients.[91] The responses from the SF-36 are converted into 6 dimensions, each with a numbered scale, which are used to define a health state. These are, in turn, used to generate Quality Adjusted Life Years and allow for a health economics analysis of the use of ICP for control of post-TBI ICP.[92]

The results from these 2 studies will have important implications for both deciding the role of DC for control of post-TBI increased ICP and the indications for its use. This will contribute to the development of guidelines that can more clearly advise on the use of DC based on its clinical and socioeconomic (in RESCUEicp) outcomes. Furthermore, the results of DECRA may help in determining the most appropriate timing for DC. Specifically, the large recruitment numbers in both trials minimizes the impact of confounding factors caused by inherent wide patient heterogeneity in the TBI patient population.

CONCLUSIONS

Uncontrolled intracranial hypertension is associated with higher mortality, morbidity, and worse long-term outcomes after TBI. Management of increased ICP should start with the anticipation of its development and follow with a target-driven, tiered treatment protocol encompassing simple nursing measures, medical intervention, as well as surgical techniques. The importance of CPP as a treatment target should not be neglected in treatment. Two surgical interventions, in addition to primary evacuation of mass lesions, are principally used: ventricular catheterization and DC. Whereas there is a general consensus about using ventricular catheterization for monitoring as well as a second-tier intervention, this is not the case for DC and there are few clear guidelines on the matter. The current literature on this topic comprises many retrospective studies and inconclusive, or even contradictory, findings. This situation leaves the debates about the efficacy of DC unresolved. It is hoped that the findings of the RESCUEicp and DECRA RCTs will clarify the role of this operation in patients with TBI.

AUTHOR CONTRIBUTIONS

All authors helped write the manuscript. All authors approved the final manuscript.

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