Review article

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Decompressive craniectomy in head injury

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Abstract: Decompressive craniectomy has been used to treat severe intracranial hypertension secondary to various causes like trauma, cerebral infarction, subarachnoid hemorrhage, and spontaneous hemorrhage, refractory to medical treatment. There are many different approaches grouped under the term 'decompressive craniectomy' with all of them aiming at reduction of raised intracranial pressure. We have reviewed the literature and tried to describe the mechanism, various types, indications and complications of this procedure.

Keywords: craniectomy, decompressive, trauma

HISTORICAL BACKGROUND

According to Seydel[1], even Hippocrates gave indications for trephination in head injury. Kocher[2] wrote the following in his book on brain concussion, intracranial pressure (ICP), and surgical treatment of diseases of the brain, published in 1901: "If there is no CSF pressure, but brain pressure does exist, pressure relief must be achieved by opening the skull. Relief of pressure by trephination is indicated in all cases of brain pressure." The modern concept of decompression for traumatic brain injury (TBI) was introduced by Harvey Cushing before World War I[3,4]. He performed a subtemporal decompressive craniectomy for elevated ICP related to neoplastic growth as early as 1905[3], and later reported the application of this operation to wartime trauma[4]. Decompressive craniectomy(DC) represents a large cranial and dural decompression, often associated with removal of mass lesions such as subdural hematoma or traumatic intracerebral hematoma. Decompressive craniotomy was initially described by Miyazaki in 1966, popularized by Kjellberg and Prieto in 1971[5].

The skull in an average adult encloses a total volume of 1475 ml, including 1300 ml of brain, 65 ml of cerebrospinal fluid (CSF), and 110 ml of blood[6]. The Monroe-Kellie doctrine states that the sum of the intracranial volumes of brain, blood, CSF, and other components is constant, and that an increase in any one of these must be offset by an equal decrease in another, or else pressure increases. An increase in intracranial pressure caused by an expanding intracranial volume is distributed evenly throughout the intracranial cavity[7,8].

Normal values of intra cranial pressure (ICP) vary with age, being 10 to 15 mm Hg in an adult, 1.5 to 6 mm Hg in a term-infant, and can be subatmospheric in newborns[9]. ICP values of 20 to 30 represent mild intracranial hypertension, while sustained ICP values exceeding 40 mm Hg indicate severe, life-threatening intracranial hypertension that requires immediate treatment[10,11]. Malignant brain edema is a state of severe, progressive and diffuse cerebral edema that causes rapid clinical deterioration unresponsive to aggressive treatment. It is usually seen in patients with Type III severe head injury, aneurysmal subarachnoid haemorrhage (SAH), and massive brain infarction[12,13]. These patients present clinically as severe hemispheric syndrome including hemiplegia, forced eye and head deviation and progressive deterioration of consciousness within first 48 hours. On computed tomography (CT), it is visible as compression of ventricles, obilitration of the basal cisterns, loss of normal gyral pattern, and poor white-grey matter differentiation[13]. Death occurs by herniation when the ICP is raised and the capacity for adjustment by fluid shifts from the CSF and vascular compartments is already maximized[14,15].

A secondary increase in ICP is often observed 3 to 10 days after the trauma principally as a result of a delayed hematoma formation, such as epidural hematomas, acute subdural hematomas, and traumatic hemorrhagic contusions with surrounding edema, which may require evacuation[16]. Other contributing factors are cerebral vasospasm[17], hypoventilation, and hyponatremia.

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Severe traumatic brain injury (TBI, GCS < 8) occurs in 60% of polytraumatised children after car accidents or child abuse, and is associated with high morbidity and mortality[18,19]. Diffuse brain swelling and multiple cerebral contusions are the most common cause of death after severe head injury in pediatric patients[20]. The primary therapeutic aim is to maintain an adequate cerebral blood flow (estimated from cerebral perfusion pressure, CPP = MAP-ICP, where MAP = 1/3 systolic BP+2/3 diastolic BP)[21,22]. The surgical removal of a part of the calvaria to create a window in the cranial vault is the most radical intervention for intracranial hypertension, negating the Monroe-kellie doctrine of fixed intracranial volume, and allowing for herniation of swollen brain through the bone window to relieve pressure. Decompressive craniectomy (DC) has been used to treat severe intra cranial hypertension refractory to medical treatment secondary to various causes like trauma, cerebral infarction, subarachnoid hemorrhage, and spontaneous hemorrhage[21]. Posttraumatic brain edema leading to refractory intracranial hypertension is the main prognostic factor in brain injured patients, despite progress in the diagnosis and therapy[23]. The pathophysiology of this brain edema is not known, vasogenic and cytotoxic edema and quite possibly cerebral vasocongestion contribute to this phenomenon[24]. Brain swelling and intracranial hypertension in the early posttraumatic period has been proposed to be induced by cerebral hyperemia, especially in children[25,26]. The second tier options for intracranial hypertension are barbiturate coma, hypothermia, hyperventilation, or decompressive craniectomy. Decompressive craniectomy should be the first option among the second tier options rather than the last one because it leads to the fastest relief by immediate reduction of intracranial hypertension, has the lowest rate of complications, is simple and safe[23]. The mechanisms by which DC provides relief in raised intracranial pressure are:

1. It lowers the ICP immediately and permanantly.
2. It adds a vector of expansion to the cerebral hemispheres which relieves brain herniation.
3. Allows exploration of the subdural space.
4. In addition it provides quick tapering of the medical treatment such as; hypothermia, barbiturates, osmotic diuretics, ventriculostomy, prolonged hyperventilation, and hypertonic saline, in order to avoid its potential complications[27,28,29,30].

TYPES OF DECOMPRESSIVE CRANIECTOMY

There are many different approaches grouped under the term 'craniectomy'. Unilateral or bilateral, frontal and subtemporal decompression and circumferential hemicraniectomy[31,32,33,34,35]. Subtemporal decompressions are unilateral or bilateral bony decompressions designed to take the pressure off the temporal lobes to prevent uncal herniation, and have been used in other cranial conditions such as pseudotumor cerebri. Cerebellar decompression for mass lesion is a standard neurosurgical response to any process in the posterior fossa (hemorrhage, tumor, infection, or stroke) that threatens cerebellar tonsillar herniation, and is not controversial[15]. Hemispheric decompressive craniectomy is widely performed in trauma[35]. Bifrontal decompressive craniectomy is an aggressive approach described by Kjellberg and Prieto[5] before the era of modern neuroimaging with computed tomography. Venes and Collins[36] described this strategy as well. This approach is particularly useful in the pediatric population, in which diffuse injury without mass lesions and with ICP elevation is relatively common. Bifrontal craniectomy is performed with the patients in supine position, mild degree of reverse Trendelenberg's position, a bicoronal skin incision made and temporalis muscles reflected inferiorly[37].

Bitemporal decompression aim is to provide relief in compression on brain stem bilaterally. In bilateral hemicraniectomy, only a rim of bone remains on top of the superior sagital sinus to avoid ligating the falx and sinus. There after the dura is opened, and starting at its temporal base, the dura is enlarged with the temporal fascia in a dovetail manner, followed by water-tight closure of the dura and a fascial graft. The bone flap is stored in sterile condition at -80C and re-implantation (cranioplasty) is performed after 6 weeks to 3 months[23]. Larger the removed bone flap is, the more is the reduction in ICP[38].

The indication guidelines of DC are[23];

1. Age < 50 years
2. Brain swelling on CT scan, unilateral or bilateral with correlating clinical deterioration.
3. No fatal primary brain injury with irreversible brainstem signs or herniation with neurological pons signs

4. Refractory intracranial hypertension (> 30 mm Hg)
5. Intracranial hypertension with deterioration in clinical status (GCS score of 4 or higher, decerebrate posturing, mydriasis), and increase in pulsatility index with decrease in diastolic flow on transcranial Doppler ultrasonography (TCD).
6. Surgical intervention before irreversible brain stem damage or generalized ischemic brain damage monitoring of ICP, and B wave, AEPs (auditory evoked potentials) and SEPs.

Patients with primary fatal brainstem injury, an initial and persisting GCS score of 3 and / or bilaterally dilated and fixed pupils, are not candidates for the surgery[23].

The American association of neurological surgeons (AANS)[13] has recommended decompressive craniotomy for patients with traumatic brain injury (TBI) and refractory intracranial hypertension (IH) if some or all of the following criteria were met:

1. Diffuse cerebral swelling on cranial CT imaging.
2. Within 48 hrs of injury.
3. No episodes of sustained intracranial hypertension (ICP) > 40 mm Hg before surgery.
4. GCS > 3 at some point subsequent to injury.
5. Secondary clinical deterioration, and
6. Evolving cerebral herniation syndrome.

The **complications** of DC are;

1. Infections like meningitis or cerebral abscess[39].
2. Bone flap resorption by aseptic necrosis[22,13].
3. Contralateral subdural effusion (6.5%), occurs on an average 14 days after DC[40].
4. Communicating hydrocephalus[41,42], most probably due to mechanical blockade or inflammation of arachnoid granulations by post surgical debris[42]
5. Ipsilateral hemorrhagic swelling[41]
6. Subdural hygroma is the most common complication (26%)[23,41], most often which respond to puncture only[23]
7. Paradoxical herniation after lumber puncture in a case of DC[43]
8. Hypotension in children during emergent craniotomy for trauma[44]
9. Postoperative seizures[23]

An increase in cerebral blood flow after DC causes congestion in the decompressed brain and does not, as hypothesized by Cooper et al[45], parallels the development of acute brain edema. The focal increase in blood flow in decompressed brain may protect the brain from secondary ischemic cell damage (lactate and potassium clearance)[23].

A number of prognostic factors determine the outcome in TBI after DC. Age more than 50 years is associated with poorer outcome after the surgery[46,47], as is a low admission or post resuscitation GCS, presence of cranial fracture, absence of pupillary response / brainstem reflexes, respiratory insufficiency, refractory rise in ICP and the status of the basal cisterns or third ventricle on CT scan[47]. Other factors which significantly correlate with the outcome, are the location of the lesion, ICH (intracerebral hemorrhage) volume, GCS at the time of follow-up, lowest recorded GCS, severity of surrounding edema, timing of surgery, occurrence of preoperative neurological deterioration, and presence of acute hemispheric swelling or concomitant subdural hematoma[47]. The outcome of BDC varies from report to report. The percentage of good outcome has ranged from 7 to 70%, and mortality from 13.5 to 90%[5,48,49,28,50]. The amount of primary brain injury, timing of surgery, level of ICP, and GCS score before the surgery are considered as predictive of outcome after BDC[5,48,49,28,5137,52]. Following criteria have been proposed for good outcome after BDC: 1. Initial GCS >5, 2. small pupils on admission, 3. observed clinical deterioration, 4. rapid surgical intervention, and 5. absence of brain infarction in the pre-operative scans[13].

Although decompression does not reverse the primary brain injury associated with traumatic injury, it can ameliorate secondary damage caused by elevation of ICP. For this reason DC should be performed within 48 hours of the trauma, before the period of maximal cerebral swelling (ICP elevation should not exceed 40 torr)[37].

Eisenberg et al[53], reported that midline shift is very strong predictor of abnormal ICP, and the risk of death is greater if the midline shift is large. Conversely the investigations of Miller et al[54], and Tabaddor et al[55], demonstrated no or only a poor correlation between the midline shift and ICP.

Marshal[56] demonstrated that CT defined injury type was a highly significant independent predictor of mortality, even when age and GCS motor score were

included in the predictive model. Bullock et al[57], prospectively studied 85 patients with ICH whose initial need for craniotomy was uncertain, and found the peek ICP to be the strongest predictor of outcome. CT and clinical predictors included cisternal status, edema severity, and admission GCS. The authors found that the weight each predictor depended on the location of the ICH. For temporoperietal lesions hematoma size, degree of edema, GCS, basal cistern status, and ICP data correlated with outcome. However for frontal lesions peak ICP alone was predictive of outcome. These findings expand on those reported by Gallbraith and Teasdale[58], who found that all patients with intradural lesions and sustained ICP in excess of 30 mm Hg, and only one patient with ICP less than 20 mm Hg, required operative intervention. Mathiesen et al[59], reviewed data collected prospectively for the head injury trial -2 (nimodipine trial) on 218 TBI patients not obeying commands within 24 hrs of injury, and found that the outcome from the craniotomy was adversely affected by neurological deterioration (defined as a fall in GCS by 2 points or from 4 to 3, or the development of pupillary dilatation) before surgery, suggesting that patients with factors strongly associated with neurological deterioration (initial CT characters of presence of SAH, focal lesion with volume > 40 cc, and compressed or absent cisterns) should be considered for early surgery.

Sahuquillo and Arikan[60,61], while reviewing the literature found only one randomized clinical trial in 27 children who had traumatic brain injury (TBI)[30]. This trial showed a reduced risk ratio for death of 0.54 (95% CI 0.17- 1.72) and a risk ratio of 0.54 for death, vegetative status or severe disability 6 to 12 months after injury (95% CI 0.29- 1.07)[62]. All the available studies in adults are either case series or cohorts with historical controls. These reports suggest that DC effectively reduces ICP in most (85%) patients with ICH refractory to conventional medical treatment[41,37]. Choksey et al[63] retrospectively reviewed 202 patients with traumatic ICH and showed that craniotomy significantly improved the probability of good outcome. Hatashita and Hoff[64], showed that decompressive frontoperietal craniectomy in cats led to significant reduction in ICP, reduction in cortical grey and white matter tissue pressure, increased pressure volume index, and increase4d tissue compliance. The authors found that the craniectomy significantly increased the volumetric compensatory capacity of the intracranial cavity, a finding consistant with that of Hase et al[65], who documented a dramatic increase in intracranial compliance after the decompression. Yoo et al[66] studied intra operative ventricular pressure in a cohort of 20 patients with refractory intra cranial hypertension after both traumatic and non traumatic insult who underwent bilateral

Table 1 : Review of the literature on decompressive craniectomy in severe closed head injury[23]

	Author	Year	Total	Alive	Dead
1.	Cushing	1905	_	_	_
2.	Kerr	1968	2	_	2
3.	Kjellberg and Prieto	1971	50	11	39
4.	Ransohoff et al	1971	35	14	21
5.	Venes and Collins	1975	13	9	4
6.	Cooper et al	1976	50	5	45
7.	Pereira et al	1977	12	6	6
8.	Shigemori et al	1979	15	5	10
9.	Yamaura et al	1979	154	109	45
10.	Gerl and Tavan	1980	30	7	23
11.	Krone and Kelly	1985	7	6	1
12.	Alexander et al	1987	15	13	2
13.	Karlen and Stula	1987	7	2	5
14.	Gower et al	1988	10	6	4
15.	Hatashita et al	1993	3	_	3
16.	Whitfield and Guazzo	1995	1		1 _
17.	Dam Hieu et al	1996	2	2	0
18.	Polin et al	1997	35	27	8
19.	Waltraud Kleist et al	1999	57	46	11

frontotemporoperietal DC with dural expansion and grafting. They found a 50.2 ±16.6 reduction of initial ICP after craniectomy, and a further reduction to 15.7 ± 10.7% of initial ICP after dural opening. Polin et al[37] showed a significant decrease in ICP after bifrontal DC, as well as a significant difference in postoperative ICP, when compared with ICP measured 48-72 hrs after injury in a cohort of historically matched controls. Gower et al[67] found that 7 of 10 patients who underwent subtemporal craniectomy for medically refractory intracranial hypertension had an average decrease in ICP of 34%. Kunze et al[62] found a reduction in mean ICP from 41.7 to 20.6 mm hg in 28 patients after in unilateral or bilateral decompressive craniectomy for post traumatic edema refractory to maximal medical therapy. Whitfield et al[68] demonstrated similar outcome ($p = 0.003$) in 26 patients who underwent bifrontal DC for refractory intracranial hypertension. Munch et al[69], however failed to demonstrate postoperative reduction in ICP after unilateral hemispheric decompression. They however showed a significant outcome difference between patients undergoing rapid DC and those undergoing delayed DC. Guerra et al[27] prospectively performed DC following a standardized protocol, for posttraumatic diffuse brain edema in 57 severe TBI patients (GCS 4 to 6). They found that 58% of the primary DC group, and 65% of the secondary DC group (for persistently raised ICP following evacuation of a surgical mass lesion), experienced good outcome or moderate disability at one year. Gower et al[67], in a retrospective study found that sub temporal decompression offered significantly lower mortality as compared with barbiturate coma. Lee et al[70] documented a significant improvement in outcome with the addition of temporal lobectomy to sub temporal decompression and debridement of contused brain. Mortality decreased from 56% to 8%, with a concomitant increase in average GCS from 2.2 to 4. The study by Polin et al[37] demonstrated that surgery performed within 48 hrs after injury was significantly associated with favorable outcome when compared with surgery performed longer than 48 hrs after injury (46% versus 0% respectively).

Two prospective randomized controlled trials are currently run in an attempt to provide class 1 evidence on the role of surgical decompression in the treatment of raised ICT after severe head injury; The RESCUE ICP study[41], is an international multricentre trial, co-ordinated by the university of Cambridge Academic Neurosurgy Unit and the European Brain Injury Consortium (EBIC) and the DECRA trial[71], run and co-ordinated by the Austalian centers.

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