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Decompressive Craniectomy and Traumatic Brain Injury: A Review

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ABSTRACT

Intracranial hypertension is the largest cause of death in young patients with severe traumatic brain injury. Decompressive craniectomy is part of the second level measures for the management of increased intracranial pressure refractory to medical management as moderate hypothermia and barbiturate coma. The literature lack of concepts is their indications. We present a review on the state of the art.

Keywords: Decompressive craniectomy; Brain trauma injury; Intracranial hypertension

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Introduction

Currently morbidity and mortality due to traumatic injuries are a well-recognized major public health problem. Similarly the traumatic brain injury (TBI) is a major public health concern worldwide, according to the predictions, neurotrauma will account an increasing number of deaths worldwide by 2020 [1]. Unfortunately, overall trauma ranks among the leading causes of death and occurs in all regions, affecting people in all age and income groups [2].

TBI is defined as an injury to the head arising from blunt or penetrating trauma or from acceleration/deceleration forces associated with one or more of the following: decreased level of consciousness, amnesia, objective neurologic or neuropsychological abnormality, skull fracture(s), diagnosed intracranial lesion(s), or head injury listed as a cause of death in the death certificate [3,4]. It is the most common cause of death and disability in children and young adults [5]. In the United States, TBI generate 235.000 hospitalizations, 50000 deaths, and permanent disability in 99000 [6]. The economic burden for TBI alone in the United States in 2000 was estimated in $110 billion derived from direct (e.g. medical) and indirect (e.g. lost productivity) costs [7].

One of the main characteristic of TBI is that patients without a severe TBI, can experience subsequent mental and/or medical problems [8,9]. The acute consequences of TBI are just only a half of the complete problem, the long-term repercussions of TBI are substantial especially among adolescents and young adults, whose brains continue to mature and develop [10]. To make matters worse, people who sustain TBI during or before adolescence can have a limited return to pre-trauma academic or work activities that aggravate the economic and physical consequences derived from the treatment and the rehabilitation. Furthermore, they may have diminished attainment of personal milestone [11-13]. Among the many problems that arise as

a result TBI, intracranial hypertension (IHT) is a major cause of complications and death. Thus, it is comprehensible that neurosurgeons perform considerable effort to controlling intracranial pressure (ICP) in patients with TBI. Decompressive craniectomy (DC) has been advocated as one strategy for managing ICP [14].

The aim of this work is to review the history, indications, technical aspects, complications, cost-effectiveness, and to discuss what the current medical evidence is telling us about DC, also are discussed some future trends of this neurosurgical technique.

History

As a procedure, DC was first described by Annandale in 1894 [15,16], and throughout the second half of the XIX century almost all neurosurgery pioneers had been performed craniectomies as palliative measure for patients with intractable tumors, but Kocher in 1901, was the first one to propose the palliative decompressive craniotomy for patients with raised intracranial pressure following traumatic brain injury (TBI) [17-19]. From the close collaboration of Kocher with Harvey Cushing DC was proposed for the treatment of other brain disorders [20,21]. Cushing in 1908 [22] published the subtemporal decompressive operations for the intracranial complications associated with bursting fractures of the skull.

Due to poor clinical outcomes DC quickly fall into discredit [15,16], and was almost abandoned when experimental evidence [23] suggested that decompression worsen cerebral oedema. However in 1968, Clark et al, reported 2 cases with 100% of lethality [24]. In 1971, Kjellberg *et al.*, [25] reported 73 cases, using large bifrontal craniectomy with 18% of surveillance. Venes and Collins, in 1975, reported in a retrospective analysis of 13 patients who had bifrontal decompressive craniectomy for the management of posttraumatic cerebral edema, a significant decrease in expected mortality, but severe morbidity in the survivors, and only one patient returned to the pretrauma level of neurological function [26]. So, throughout the 1980's its popularity returned. Pereira et al in 1977, present the results observed with large bifrontal decompressive craniotomy performed on 12 patients with severe cerebral edema, a 50% surveillance and 41.6% of excellent neurological and mental improvement [6], also in 1980, Gerl and Tavan reported that extensive bilateral craniectomy with opening of the dura offers the possibility of rapid reduction of intracranial pressure, they observed a 70% of mortality, and a 20% of the cases with full recovery [27]. In 1990, Gaab *et al.*, [28] with a prospective study design with 37 patients <40 years old, they performed 19 bifrontal craniectomies and 18 hemicranietomies, and report 5 deaths, all others achieved full social rehabilitation or remained moderately disabled; they established as best predictor of a favourable outcome an initial posttraumatic Glasgow coma scale (GCS) ≥7.

During the last lustrum DC has become very popular again, published papers have had a strikingly increase; unfortunately they are mainly retrospective reviews with limited number of cases. DC has been used, as in the past, for many neurosurgical conditions including intracerebral haematomas [29] and brain infarction [30]. All this evidence makes us to ask ourselves as Tagliaferri *et al.*, [31] stated: have we found a "panacea" for all neurosurgical diseases?

The Rationale Of DC

As previously mentioned, among problems secondary to TBI, brain edema, and as consequence, ICH, are the meanly of them, more dramatically, TBI is the most common cause of intracranial hypertension [32], and even more dramtically ICH is the most frequent cause of death and disability following severe TBI [33-35].

Brain edema formation is a secondary injury caused by a cascade of mechanisms initiated at the moment of injury [36]. ICH is a frequent complication of severe TBI [37-39], near to 70% of brain injured patients will present ICH [40-43]. Life threatening episodes of raised ICP are usually associated with conditions that afflict wide areas of the brain such as global cerebral swelling after a trauma [44]. ICH results in alteration of physiologic parameters like the cerebral perfusion pressure (CPP) and then brain oxygenation [45]. A CPP less than 60-70 mmHg is associated with diminished oxygenation and altered metabolism in brain parenchyma [46]. ICH (defined as ICP ≥20 mmHg) is a known independent risk factor for poor neurological outcomes [47], that the reason because in the pathophysiology of the primary and secondary lesions in TBI, both brain edema and ICH, are the pillar targets to prevent and wane the progression of brain damage.

As stated by the Monroe-Kelly doctrine [29,48,49], "the sum of the intracranial volumes of blood, brain, CSF and other components is constant and that an in increase in any one of these must be offset by an equal decrease in another" [50], so the skull is a rigid structure, unexpansible, in order to maintain a constant blood pressure, the volumes inside the cranium should be constant. The raised ICP results in "spatial compensation", i.e., extrusion of CSF and blood (mainly venous) from the intracranial cavity. CSF has a key role in spatial compensation because it can be expelled to the spinal theca, the reservoir [51,52]. However should be remembered that CSF

shift is time- and age-dependent variable. Older people can accommodate more of the expanding new volume due to the additional space created by cerebral atrophy; conversely, young people get symptomatically faster, due to the lack of space.

Is a well-known fact that TBI patients with refractory ICH have worst outcomes, and are more likely to develop herniation syndromes [53,54]. The management of refractory ICH is a crude challenge for neurosurgeons. Methods used such as hyperventilation, barbiturate therapy and therapeutic hypothermia, are ineffective in some patients, and is also the reason because DC is an option when other methods of controlling ICP are not effective [55].

It is scarcely logical to comprehend that to achieve good quality survival is required that much of the brain dysfunction associated with these conditions be reversible. If contrary, probably the intervention is saving lives of very poor quality [44].

Indications and Contraindications

Overall, there are no widely accepted indications for craniectomy [13]. Some indications for decompressive hemicraniectomy are the unilateral lesions, such as unilateral swelling, contusions, extradural or subdural hemorrhage, midline shift [56], generally is required bifrontal decompression for diffuse cerebral edema with no obvious midline shift (Table 1).

However, regard to TBI, according to the European Brain Injury Consortium and Brain Trauma Foundation guidelines for severe TBIs, DC should be incorporated to the second-tier therapeutic arsenal in patients with refractory ICH to first-tier therapeutic measures [57-60], i.e., when appropriate targeted surgery and medical treatment fails, DC is the option [31].

The best time to decompress a patient is still under discussion [56], but early DC (within 24 h after injury) is recommended for severely head injured patients without brain stem dysfunction requiring neurosurgery for removing intracranial collections [61]. Also, data suggested that complications of TBI may be reduced following early DC [62-65].

This decision can be made intraoperatively based on the patient's mechanism of injury; age; degree of underlying cerebral swelling, atrophy, or both; and the surgeon's estimation of the likelihood that the patient will develop severe ICH [13]. Should be noted that in places where multimodal monitoring, DC can be the choice treatment to prevent brain herniation [59]

Otherwise, the outcome of the patients who undergo late DC (after 24 h) is more encouraging [31].

As reviewed by Lubillo *et al.*, [59] contraindications for DC are:

- Patients with GCS 3 post-resuscitation, with dilated and fixed pupils
- Patient >65 years old
- Devastating trauma that won't allow patient survive more than 24 h.
- Irreversible systemic disease in the short term
- Uncontrollable ICH during more than 12h besides all energetic therapeutic measures
- O_2 arterio-venous difference <3,2vol%, measured in the side of hemicraniectomy or a $PtiO_2$ <10mmHg in the apparently health area since patient admission.

Technical Aspects

At the time of initial surgery, as well as during the following cranioplasty, appropriate closure techniques address concerns regarding infection, adhesion, and injury.

Dural grafts and anti-adhesion barriers are important in minimizing these concerns and facilitating the follow-up dissection for cranioplasty [66].

The ideal technique implies the removal of bone in the entire supratentorial hemicranium. One of the most important landmarks for this procedure is the root of the zygoma, it allows the identification the floor of the temporal fossa. Also are important landmarks: the asterion (confluence of the lamboid, occipitomastoid, and temporoparietal sutures, indicates the area of transition between the transverse and sigmoid sinuses), the keyhole (identifies the pterion and indicates the location of the frontal, temporal, and orbital cavities), the inion, the glabella, and the midline (delineates the course of the superior sagittal sinus). When the patient's head is placed in the head-holder, it is ideal that the sagittal plane of the head be 0-15° horizontal to the floor [67]. Fronto-subtempo-parieto-occipital DC with dural opening and enlargement with duraplasty is the most used decompressive technique, being the only technique that avoids brain herniation through DC hole, and prevents venous infarctions that power brain swelling [59]. Skin incisions for decompressive hemicraniectomy include the large reverse question mark frontotemporoparietal incision and the L.G. Kempe modified incision or midline sagittal incision with "T-bar"; skin incisions for bilateral decompressive craniectomies include perform two hemicraniectomies or to perform the Kjellberg type DC (standard bicoronal incision). Regard dural opening can be used different ways of opening that includes fish-mouth incision, stellate incision, C-shaped fashion incision and cruciate incision [30,68].

The choice of materials for grafting and dural substitution depends on the surgical goal, but reducing the potential for dural adhesion is critical.

Table 1. Decompressive Craniectomy complications: recommendations and comments[a].

Complications	%	Recommendation	Comment
Herniation through the craniectomy defect	27.8%	Performing a sufficiently large craniectomy	DC plus augmentative duraplasty would achieve a similar decompressive effect, compared with leaving the dura open.
Subdural effusion (Hygromas)	21.3%;	After removal of part of the cranium, augmentative duraplasty should be performed	This complication may need more aggressive treatment because of its tendency to cause midline shift
Post-traumatic hydrocephalus	9.3%	Employ surgical intervention as soon as possible after the diagnosis of hydrocephalus and the exclusion of contraindications.	DC with a superior limit closer than 25 mm to the midline might predispose to the development of hydrocephalus.
Syndrome of the trephined	13%	Perform early cranial repair, before the skin flap sinks; within 8 weeks after craniectomy	Remember that in a patient with TBi, early cranioplasty may increase the risk of infection, and thus is not recommended. Is the most common DC complication after 1 month
Contralateral haematoma after decompressive craniectomy	7.4%	operation and early detection and intervention are the keys in the management	Reduction in ICP after craniotomy is considered an important reason for the formation of an intracranial hematoma remote from the site of operation

[a] Adapted from [55]

Extensive adhesions can often cause increased operative time, risk of dural violation, brain injury, and surgeon frustration during the cranioplasty [66]. A surgeon can also expect to face greater difficulty with dissection of adhesions when the length between the first and second surgery is increased [66]. It involves removing a large piece of the skull and opening the underlying dura to allow the brain to expand. In this way the brain swelling that causes raised intracranial pressure can be accommodated by increasing volume instead [44]. Inappropriate techniques for DC, e.g., do not smooth the bony edges; do not try at maximum to do bone removal as large as possible performing wrong approaches like only subtemporal decompression, or only frontotemporal decompression, can generate iatrogenic brain lesion, and even generate brain herniation trough the craniectomy.

Complications

DC is no exempt of complications; these can appear following a time-dependent pattern, first things can happen are expansion of hemorrhagic contusion, followed by appearance of new subdural hematoma on contralateral side, seizures, leakage of CSF, and brain herniation [69]. Some complications can be directly fatal, such as intracranial infection and contralateral intracranial haematoma, while others can adversely affect the patient's neurological and intellectual recovery [55].

Complications are more frequent in patients with low GCS and those over 60yo [69]. Nearly 50% of patients had at least one complication [70]. In the Aarabi *et al.*, [71] study up to 50% of patients developed subgaleal and subdural hygromas, although these situations resolve spontaneously without posterior surgical intervention.

Risk factors that increase the rate of infection and require implant removal included orbital extension of the craniectomy defect, proximity to facial sinuses, and large contour abnormalities with corresponding large dead spaces [72]. Staging reconstruction of high-risk cranial defects followed by definitive cranial defect reconstruction improved the likelihood of implant retention and successful cranioplasty outcome [72].

- Herniation through the craniectomy defect, happening in up to 27.8% of patients; a though to counteract this phenomenon is to perform a DC, as large as possible. DC combined with augmentative duraplasty would achieve a similar decompressive effect, compared with leaving the dura open, and it would be helpful in preventing herniation through the cranial defect by limitation of cephalocoele [55].
- Subdural effusion happens in near 21.3%, probably due to CSF disturbances derived from the DC itself. Contralateral subdural effusion caused by DC has rarely been reported [73]. Probably they are likely caused by deranged patterns of CSF flow in the presence of low ICP [13]. This complication may relate with postoperative neurological deterioration or raised ICP [74-76]. Risk factors of subdural effusion after head trauma include

subarachnoid hemorrhage, shrinkage of the brain due to intraoperative retraction, and significant shifting of the brain tissue [55]. This complication may need more aggressive treatment because of its tendency to cause midline shift [76].

• Post-traumatic hydrocephalus, it occurence range in up to 9.3%; probably because severely injured patients cannot show clinical symptom, CT scan can be extremely useful for the detection of patients developing dilatations or ventricular shifts [55]. It was recently suggested that communicating hydrocephalus is an almost universal finding after hemicraniectomy and that early cranioplasty may prevent the need for permanent cerebrospinal fluid diversion in these patients, but according to findings by Rahme *et al.*, [77] hydrocephalus does not frequently occur after DC, thus is not an independent risk factor for communicating hydrocephalus in patients with raised ICP. After DC, in particular, several factors have been associated with the development of communicating hydrocephalus, such as older age, subarachnoid hemorrhage, CSF infection, lower GCS and wide craniectomic flap [78]. Craniectomy with a superior limit closer than 25 mm to the midline might predispose to the development of hydrocephalus as well [79]. Shunting is clearly necessary when the lumbar CSF pressure is consistently >180 mmH_2O or the typical symptoms of normal pressure hydrocephalus are present, unless there are surgical contraindications [55].

• Syndrome of the trephined, clinical manifestations includes headaches, dizziness, irritability, epilepsy, discomfort and psychiatric symptoms, those especially to be related to large cranial defects [55]. Perform early cranial repair, before the skin flap sinks; within 8 weeks after craniectomy. However, in a patient with TBI, early cranioplasty may increase the risk of infection, and thus is not recommended [55].

• Contralateral hematoma after DC, happens in up to 7.4%; Uncommon phenomenon and is associated with significant morbidity [69]. Reduction in ICP after DC is considered an important reason for the formation of an intracranial, thus hematoma remote from the site of operation. They have an early apparition, thus early detection; intervention and operation are the keys in the management.

Cost-Effectiveness of DC in TBI

Although in medicine, treatments for saving lives should not be based on economic issues, these kind of analysis are important, because allows to us be realistic to the truth, and not be blinded by what can offer this neurosurgical panacea. Malmivaara *et al.*, [80] established that cost of neurosurgical treatment for one quality adjusted life year (QALY) in 2400 □. Ho *et al.*, [81] had demonstrated that hospital costs increase with severity of TBI and peaked when the predicted risk of an unfavorable outcome was about 80%. The average cost per life-year gained (US$671,000 per life-year) and QALY (US$682,000 per QALY) increased substantially and became much more than the usual acceptable cost-effective limit (US$100,000 per QALY) when the predicted risk of an unfavorable outcome was >80%. Thus, as a lifesaving procedure, DC is not cost-effective for patients with extremely severe TBI.

However, Whitmore *et al.*, [82] have also demonstrated that aggressive care remains significantly better at all ages. When all costs are considered, aggressive care is also significantly less costly than routine care ($1,264,000 ± $118,000 *vs.* $1,361,000 ± $107,000) for an average 20-year-old. They also observed that aggressive care remains significantly less costly until age 80, at which age it costs more than routine care. However, even in the 80-year-old, aggressive care is likely the more cost-effective approach. Comfort care is associated with poorer outcomes at all ages and with higher costs for all groups except 80-year-olds. In conclusion, when all the costs of severe TBI are considered, aggressive treatment is a cost-effective option, even for older patients.

Evidence

As previously mentioned, in the last 5 years many papers have been published about the benefits, limitations and complications of DC. Despite methodological limitations, there are some randomized trials of interest in TBI. One small prospective single-centre randomized trial was published in 2001 [83]. This trial, performed in children (27 cases), showed promising results in favor of DC. However, the surgical procedure used (bitemporal decompression without opening of the dura) is not currently regarded as the standard approach. In 2011 a second randomized larger trial, the DECRA study, has been published [84]. The main lesson from the DECRA study is that surgical reduction of intracranial pressure does not necessarily result in better outcome for patients, and indeed appears to worsen them in at least some circumstances [85].

More currently, Tagliaferri *et al.*, [31] studied 155 adults with severe diffuse TBI and first-tier refractory ICH (ICP >20 mmHg for >15min) that were randomly assigned to undergo either early bifrontotemporoparietal DC (indications:

as a last-tier therapy or in association with a hematoma evacuation) or standard care medical treatment. Those patients treated with DC had lower intracranial pressure and shorter stay in the Intensive Care Unit. However, they also had worse scores on the Extended Glasgow Outcome scale and greater risk of unfavorable outcome than those receiving standard care, although the mortality rate at 6 months was similar. Is ongoing another prospective, randomized-controlled TBI trial, the RESCUEicp (http://www.rescueicp.com/). This study has enrolled 309 patients as of June 2011 with a target of 400 cases. Patients are randomized when maximal medical therapy fails to control ICP with a threshold of 25 mmHg for more than 1 to 12 h at any time post-injury; previous evacuation of hematoma is allowed only before randomization.

Other factors related to DC, have been evaluated, Jiang *et al.* study suggested that large fronto-temporoparietal DC (standard trauma craniectomy) significantly improved the outcome in severe TBI patients with refractory ICH, compared with routine temporoparietal craniectomy, and had a better effect in terms of decreasing ICP [70].

Prognosis

TBI age is an important prognostic factor per sec [86]. Unfortunately, most retrospective [71,87] and prospective studies [84] have an age limit at 60-65 years [31]. Williams *et al.*, [88] in their study including patients with wider age range (15-90 years), the mean age of patients with a good outcome was significantly younger. Tagliaferri *et al.*, [31] in their case series found as most important prognostic factor by far the age. Only 7% of patients over 65 years old had a good outcome, the 6-month mortality of 72% was similar to the 77% 1-year mortality reported by De Bonis *et al.*, [89] for a population of 44 patients with traumatic brain injury older than 65 years treated with DC. As mentioned by Tagliaferri *et al.*, [31] some papers on DC have not reported a correlation between age and outcome [71,87].

Adequate bone flap size is another factor related to survival, Tagliaferri *et al.*, [31] also found that large bone flap (larger than 12 cm) was related to survival only in patients younger than 65 years. Although not statistically significant, those patients with a large bone flap also had better outcome. Overall one to two thirds of the surviving patients have been reported to have a favorable outcome and the mortality has been reported as less than 20% [61,70,90-101].

Ethical Considerations

Whilst a significant number of patients survive following surgery and go on to make a good functional recovery, a significant number remain severely disabled, the ethical problem has been the high number of seriously disabled survivors following decompressive craniectomy [44]. To what degree that outcome is acceptable to those individuals is difficult to determine, there has to come a point where the primary brain injury is so severe that if a patient survives the most likely long term outcome is one of severe neurological disability [102]. Some authors consider that long-term results justify DC after severe TBI [103]. Of main importance is the fact that the perception of the use of DC in clinical practice by neurosurgeons all over the world has gone well beyond the evidence published [104], thus it is critical to assess the patient treatment under the light of the strict evidence information, to avoid at maximum invaluable mistakes that can affect patients life or give infructuous hopes to their families

Conclusions

Decompressive craniectomy is an efficient technique to reduce intracranial hypertension. The use of proper surgical technique can be the key to a good surgical outcome. In the future will be the conduct of clinical trials to standardize the correct technique, surgical timing and makes a better choice of patients suitable for this technique.

Conflict of Interest: None declared.

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