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The Role of Decompressive Craniectomy in the Management of Patients Suffering Severe Closed Head Injuries

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1. Introduction

Severe Traumatic Brain Injury (S-TBI) is the major cause of mortality, morbidity, and disability among people younger than 45 years old. It constitutes a major problem in developed countries, not only for the affected patients and their families but also for the society, with serious socio-economic ramifications. It cannot be overemphasized the society's burden from S-TBIs, which may exhaust even the most developed health care systems. During the last 30 years, advances in pre-hospital treatment, novel imaging modalities, intensive care monitoring improvements, rehabilitation advances, as well as better understanding of the S-TBI pathophysiology have decreased the overall mortality rate from 70-80% in 1970's to 30 % nowadays. Severe TBI is still associated with unfavorable outcome (death or severe disability) in up to 60%. Continuous efforts of the neurosurgical community focus not only on decreasing the S-TBI associated mortality, but also on improving the quality of life and the functional outcome of patients suffering S-TBIs [Danish, et al., 2009; Honeybul, et al., 2011].

It has been demonstrated by Marmarou and his colleagues [Marmarou et al., 1991] that increased intracranial pressure (ICP) is strongly associated with poor outcome in patients suffering S-TBIs, making thus intracranial hypertension the most frequent cause of death and disability. Moreover, it has been postulated that this association between increased ICP and poor outcome is linear. Frequently, the greatest challenge for a neurosurgeon treating a patient suffering a S-TBI is the management of increased ICP, which in a large number of cases overwhelms brain's ability to regulate cerebral blood flow (CBF), resulting thus to cerebral ischemia and consequently to severe disability and/or death. Elevated ICP is usually defined as an ICP above a threshold of 20 mmHg, measured within any intracranial space (subdural, intraventricular, extradural or intraparenchymal compartments). The cause of increased ICP in patients with S-TBIs is the result of an increase in brain parenchyma volume at the expense of one or more of the other two intracranial components (cerebral

blood volume, cerebral spinal fluid). An increase in brain water content (edema), increased cerebral blood volume, and /or the presence of hematomas contribute significantly to increases of ICP in patients with S-TBI.

It has been proposed that the employment of DC may drastically lower the increased ICP by allowing expansion of the edematous cerebral hemispheres. Furthermore, it has been postulated that DC interrupts the vicious cycle of intracranial hypertension via the impairment of the cerebral perfusion pressure (CPP), which inevitably results into further ICP increasing and may eventually lead to cellular injury and death. In this chapter the historical evolution of the surgical technique of DC is presented along with a brief description of the various surgical types and techniques, which are currently utilized in clinical practice. Moreover, the current concepts and controversies, the ongoing clinical trials, and the procedure associated complications are presented and discussed.

2. Decompressive craniectomy: Historical landmarks

Decompressive craniectomy has been proposed for many years as a valid treatment option for severe medically refractory intracranial hypertension, caused by various pathological conditions such as, S-TBIs, extensive cerebral infarction, massive subarachnoid hemorrhage, large intraparenchymal hemorrhage, severe intracranial infections, and extensive venous sinus thrombosis.

Hippocrates was the first who clearly described the indications for trephination in severe head injuries. Indeed, trephination remained for centuries the major surgical intervention for managing patients with severe closed head injuries. The concept of decompression (removal of a variable amount of calvaria) was introduced in the late 1890 by Annandale. Kocher in 1901 proposed the opening of the skull for relieving increased intracranial pressure, while Harvey Cushing [Cushing, 1905] performed a subtemporal decompressive craniectomy for treating moribund edema caused by an intracranial neoplastic disorder.

The concept of large cranial and dural decompression along with the removal of any underlying masses was initially described by Miyazaki in 1966, while Kjellberr and Prieto refined this surgical technique in 1971. For decades, DC was known as an occasionally life saving procedure, associated however with numerous serious complications. Therefore, the vast majority of neurosurgeons were not very eager in incorporating DC in the trauma neurosurgical armamentarium. Characteristically, Clarke in 1968 stated that the only reason for reporting his experience from performing DCs in S-TBI patients was for warning other neurosurgeons to avoid performing similar surgery.

Decompressive craniectomy in its current form was recently re-introduced by Guerra and his coworkers [Guerra et al., 1999], who reported favorable outcome in more than 50% of their cases, undergoing DC after suffering S-TBIs. Since then, many non-randomized, usually retrospective, and small size clinical studies have suggested that DC may be a valuable treatment option, when maximal medical treatment has failed to control increased ICP. It has to be pointed out that the number of the published articles in the medical literature regarding the role of DC in the management of patients with S-TBI has been geometrically increased during the last decade (Fig. 1). However, the pertinent literature demonstrates a wide variation in clinical outcomes, and ill-defined indications for performing DC in patients with S-TBIs [Aarabi et al., 2006; Howard et al., 2008; Jagannathan, et al., 2007; Morgalla et al., 2008; Münch et al., 2000].

Fig. 1. Diagrammatic representation of the number of PubMed listed articles regarding the role of DC in S-TBI patients, during the decade 2000-2010.

2.1 Types of surgical decompression and surgical procedures

Historically, DC is defined as the removal of different parts and portions of the skull, with or without opening of the underlying dura, and augmentative duroplasty. A variety of operations have been proposed and different surgical techniques have been developed for brain decompression. These include: a) the classic and widely used fronto-temporo-parietal craniectomy (either unilateral or bilateral), b) the bifrontal (bicoronal) craniectomy, c) the subtemporal decompression or the recently modified temporal craniectomy, and d) the hinge (door-like) craniotomy.

The fronto-temporo-parietal craniectomy (hemicraniectomy), theoretically consists of extensive bone resection, exposing practically almost the whole underlying cerebral hemisphere. The patient is placed in supine position, with his/her head turned towards the opposite direction. An extended reverse questionmark skin incision is performed starting one cm in front of the tragus, extending above and behind the ipsilateral ear (approximately to the posterior mastoid line) and then curving forward one or two cm laterally from the midline, ending at or just behind the frontal hair line. Key points of the procedure are the extension of the decompression to the floor of the middle cranial fossa (all the way to the zygomatic arch with preservation of the superficial temporal artery and the branches of the facial nerve), (Fig. 2), and adequate size decompression with at least 12cm in its largest diameter (Fig. 3). Usually, the temporalis muscle is dissected in one plane (osteoplastic flap), by using monopolar cautery. According to another technique, the temporalis muscle may be mobilized separately, and its fascia may be dissected and harvested for the duraplasty. The pterion and the temporal bones have to be adequately exposed. Burr holes are placed to the pterion, temporal bone, posterior parietal and frontal regions, as close as possible to the scalp incision, taking advantage of the whole skin flap. Then, the underlying bulging dura is carefully stripped off the bone, in all the burr holes with the use of a fine dissector. The burr holes are connected by using a high-speed craniotome, and then the inferior rim of the temporal bone is carefully removed in pieces, by a large rongeur exposing thus the floor of the middle cranial fossa. At this point, the ipsilateral sphenoid wing can be

drilled off, by using a diamond burr, if the patient's condition allows such a time-consuming maneuver. The bone flap may be preserved by implanting it in an abdominal subcutaneous pocket, or it can be frozen and appropriately stored. The underlying dura is incised and opened in a cruciate fashion (Fig. 4). Augmentative duraplasty is performed then by using either galea aponeurotica, or temporal fascia, or commercially available dural substitutes. The importance of performing meticulous hemostasis cannot be overemphasized. The wound is closed in anatomical layers, avoiding any tension at the skin margins. The surgeon judges according to brain edema and ICP measurements, whether the temporal fascia is sutured back or just a few approximating sutures are placed to the temporalis muscle [Apuzzo, 1993; Huang & Wen, 2010; Valadka & Robertson, 2007].

Fig. 2. Early postoperative CT scan showing adequate decompression of the middle cranial fossa in a patient undergoing decompressive craniectomy.

Fig. 3. Early postoperative CT scan demonstrating a large size decompressive craniectomy.

Fig. 4. Intraoperative picture showing cruciate opening of the dura. Please note the bulging, edematous brain, and the engorged cortical veins.

In bilateral hemicraniectomy, a bone ridge of approximately 3-4 cm in width is preserved over the superior sagittal sinus (SSS) (Fig 5). In bifrontal DC, a bicoronal skin flap is

performed and bilateral frontal bones including the bone over the SSS are removed. A key point of this procedure is the careful elevation of the bone flap, which requires careful dissection of the underlying SSS.

Fig. 5. Delayed CT scan of a patient undergoing bilateral hemicraniectomy. Please note the preservation of the bone over the superior sagittal sinus.

In the hinge craniotomy, the bone flap is repositioned in its place with the use of three titanium miniplates. A Y-shaped miniplate is placed just posterior to the coronal suture, a simple straight two-hole miniplate is placed at the sphenoid wing, and another miniplate at the posterior temporal region. It has to be emphasized, that only the Y-shaped miniplate is secured to the surrounding skull, while the other two miniplates are secured only at one side acting as buttress plates, preventing this way future bone flap settling, while allowing temporary expansion of the underlying edematous brain [Kenning, et al., 2009; Schmidt, et al., 2007].

In selected cases, DC can be combined with ipsilateral temporal lobectomy (anterior temporal lobectomy and uncusectomy), preventing thus the risk of transtentorial brain herniation. In this procedure the head of the patient should be turned 45° towards the contralateral side, and a fronto-temporo-parietal craniectomy is performed. After the dural incision and under microscopic magnification, an anterior temporal lobectomy is performed, sparing the superior temporal gyrus. A sub-pial aspiration/resection technique is used for removing the anterior 4-5 cm of the inferior and middle temporal gyri, as well as the fusiform gyrus, the uncus, the parahippocampal gyrus, and potentially the mesial temporal structures [Chibbaro, et al., 2008]. A decompressive craniectomy may be performed either for preventing or for treating severe brain swelling. Prophylactic or primary DC is defined as the surgical decompression performed primarily for evacuation of an underlying mass of any type, whenever the surgeon decides that removal of the bone flap along with the overlying bone flap will benefit the patient. According to the Congress of Neurological Surgeons' guidelines, a prophylactic DC may be performed in: a) comatose patients with epidural hematoma, b) in patients with acute subdural hematoma with thickness greater than 10 mm, or midline shift greater than 5 mm, c) in patients with admitting GCS score <8 and traumatic parenchymal lesions greater than 50 cm³ in volume, or greater than 20 cm³ with midline shift of at least 5 mm and/or cisternal compression, and d) in patients with open (compound) depressed cranial fractures, greater in thickness than that of the adjacent cranium, or with underlying hematoma, dural penetration, pneumocephalus, infection, or frontal sinus involvement [Bullock, et al., 2006; Sahuquillo & Arikan, 2006]. Secondary DC or therapeutic decompression is defined as the surgical decompression performed in patients with massive unilateral or bilateral brain edema in order to control high ICP refractory to maximal medical therapy.

2.2 Current concepts and controversies

Decompressive craniectomy has recently become a valid, widely-performed treatment option for managing patients with medically refractory intracranial hypertension. The Brain Trauma Foundation (BTF) and the Brain Injury Consortium (BIC) consider DC as a second-tier therapy for medically intractable intracranial hypertension. The algorithm of treating increased ICP of traumatic origin is based on a set of therapeutic maneuvers and first-line measures as head elevation, maintenance of adequate oxygen tension, maintenance of normovolemia and normal osmosis, normothermia, appropriate sedation and analgesia, avoiding pyrexia and seizures, adequate CSF draining via an external ventriculostomy, mild to moderate hypocapnia, administration of mannitol and hypertonic solutions, and neuromuscular blockade. When these first-level measures fail, only a few therapeutic options are available. These second-tier therapies are the administration of high dose barbiturates, induction of hypothermia, and DC. The BIC state that DC may be considered in exceptional situations, while Bullock et al suggest that DC may be the procedure of choice in patients with post-traumatic edema, hemispheric swelling, or diffuse injury given the appropriate clinical context. This context however, remains to be defined [Brain Trauma Foundation [BTF], 2007; Bullock, et al., 2006; Maas, et al., 1997].

Numerous experimental models have demonstrated that DC reduces secondary brain injury. These effects are thought to be the result of an increase in collateral cerebral circulation, reduction in tissue edema, and improvement in oxygenation and energy metabolism [Stiver, 2009; Weiner et al., 2010]. Furthermore, postoperative radiological evaluation in cases of DC shows amelioration of midline shift, and improvement of the preoperative compression of the basal cisterns [Laalo, et al., 2009].

Despite the constantly increasing clinical employment of DC in the management of patients with S-TBIs, there are still several points of controversy, regarding its exact role in the treatment of these patients. The most important controversial points may be summarized to the following:

- Lack of clear indications and guidelines, regarding the selection of candidates for DC. Cochrane data base analysis in 2007 [Sahuquillo & Arikan, 2006] concluded that there was no evidence to support the routine use of secondary DC to reduce unfavorable outcome in adults suffering S-TBIs and refractory intracranial hypertension. Contrariwise, it seems that there is more solid evidence in pediatric trauma patients, in whom DC seems to reduce the risk of death and unfavorable outcome [Sahuquillo & Arikan, 2006].
- The exact role of ICP measurements and the ICP waveform type in selecting patients for DC. Many investigators suggest that a single episode of ICP > 20 mmHg lasting at least for 5 minutes is an indication for performing DC, while others suggest a higher ICP threshold of 25 to 30 mmHg.
- Patient's age. Most neurosurgeons are very reluctant to perform DC in patients over 60 years old. Indeed, outcome seems to be worse in elderly patients. However, this issue remains to be addressed.
- The presence of comorbidity. It appears that multi systemic trauma patients have worse outcome. The presence of cardiological and other systemic underlying pathology, as well as the preoperative use of anti-platelet or anticoagulant medication should be seriously assessed before deciding to perform a DC.
- Ideal timing for performing DC. The question of early versus late intervention remains still unanswered. It is apparent that surgical decompression needs to be performed

before irreversible brain stem compression and/or herniation occur [Ruf, et al., 2003; Timofeev, et al., 2008].

- Ideal size of decompression. It was previously mentioned that adequate decompression of the floor of the middle cranial fossa is essential for achieving optimal relaxation of the perimesencephalic cisterns. It has been also proven that small DCs and small dural openings may cause swollen brain tissue to herniate through the bony defect, causing strangulation, infarction and worsening of the brain swelling. The current trend is to perform large size DCs with a diameter larger than 12 cm. However, the issue of the appropriate size DC has to be addressed in a prospective, randomized study.
- The exact role of other parameters of neuromonitoring, such as brain tissue oxygen, markers of anaerobic metabolism (microdialysis), transcranial Doppler ultrasonography measurements, and electroencephalographic monitoring may provide further information, making the selection of ideal surgical candidate for DC more accurate [Bor-Seng-Shu, et al., 2006; Weiner, et al., 2010].
- The effect of DC in the functional outcome of patients with S-TBIs remains to be proven. Does the performance of DC provide better functional outcome? Several clinical studies have suggested that DC reduces ICP but the overall functional outcome remains essentially unchanged [Danish, et al., 2009; Howard, et al., 2008; Morgalla, et al., 2008].

During the last decade a systematic attempt was made to prospectively assess the role of DC in the management of patients suffering S-TBIs and/or presenting medically refractory intracranial hypertension [Ban, et al., 2010; Morgalla et al., 2008; Valadka & Robertson, 2007]. Two independent, parallel, multi-centric, prospective clinical studies (the DECRA and the Rescue ICP trials) were designed and are underway for evaluating the exact role of DC in the management of patients with S-TBIs. These studies are supposed to address the issue of the efficacy of DC but also may clarify other DC-associated controversial points.

2.3 The DECRA clinical trial

The early DEcompressive CRAniectomy (DECRA) in patients with severe traumatic brain injury is a multi-centric, prospective, randomized trial, coordinated by The National Trauma Research Institute, the National Health and Medical Research Council of Australia, the Victorian Trauma Foundation, the ANZICS Foundation, and the Western Australian Institute for Medical Research. The primary objective of the trial is to determine whether early decompressive craniectomy compared to conventional management strategies in patients with severe diffuse traumatic brain injury and early refractory intracranial hypertension improves neurological outcomes, at six months post injury. The inclusion criteria are: severe diffuse traumatic brain injury defined as GCS<9 and CT-scan with evidence of brain swelling (Marshall score grade D2-4), or GCS score > 8 before intubation and Marshall score D3 or D4 on the obtained brain CT scan, age 15-60 years, ICP monitor insertion, decompression within 72 hours from the injury, and medically refractory ICP (defined as ICP> 20mmHg for more than 15 minutes continuously or cumulative during one hour) [Cooper, et al., 2008].

The exclusion criteria are: intracranial hemorrhage>3cm in diameter, intracranial mixed hemorrhagic contusion>5cm in long axis, previous craniectomy, presence of epi-dural and/or sub-dural hematoma > 0.5 cm in thickness, co-existent spinal cord injury, penetrating brain injury, arrest at the scene, unreactive pupils >4mm in diameter, GCS score =3, general contraindications for neurosurgical intervention, or no change of survival after careful consideration of the obtained brain CT and the patient's clinical examination.

The surgical technique used is the bifrontal decompressive craniectomy (as described by Polin and his coworkers) with a single fronto-temporal bone flap extending across the midline. The underlying dura could be opened either by making a bilateral cruciate incision or by employing a large L-shaped incision, with the lower corner of the L facing laterally. The dural openings are covered with dural or fascial patches [Polin, et al., 1997].

DECRA trial investigators presented their initial results in April 2011. They reported on 155 randomly assigned cases (73 patients underwent DC while 82 had standard care). According to their results the investigators concluded that in adults with severe diffuse traumatic brain injury and refractory intracranial hypertension, early bifronto-temporo-parietal decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU. However, they found that patients undergoing craniectomy had worse scores on the Extended Glasgow Outcome Scale than those receiving standard care (odds ratio for a worse score in the craniectomy group: 1.84). Similarly, patients undergoing DC demonstrated a greater risk of an unfavorable outcome (odds ratio: 2.21). The observed death rates at six months were similar in the craniectomy group (19%) and the standard-care group (18%) [Cooper, et al., 2011].

It has to be emphasized however, that the DECRA study carries significant weaknesses and biases. It is of great interest that out of 3700 patients, who were potential candidates for participating in this study, only 155 were finally recruited. This may be a significant selection bias. In addition, the small number of study participants significantly decreases the statistical strength of the DECRA study. Furthermore, the utilized ICP threshold of 20 mmHg for assigning patients for DC may be considered too low for patients with S-TBIs.

2.4 The rescue ICP trial

The Randomized Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intra-Cranial Pressure, is a multi-centric 48 (centers from 19 different countries) clinical study, organized as a collaborative research project between the university of Cambridge (Departments of Neurosurgery and Neurointensive Care), and the European Brain Injury Consortium. It is a randomized controlled trial comparing the efficacy of DC versus optimal medical management for the treatment of refractory intracranial hypertension following brain trauma. The inclusion criteria are: patients with S-TBI, age 10-65 y.o, with an abnormal CT-scan requiring ICP monitoring, and raised ICP > 25 mmHg for > 1 hour to 12 hours. Patients may have immediate operation for a mass lesion but not a decompressive craniectomy. Patients with immunological, hepatic, or renal compromise may be included in this study as long as there is a description of the type and the extent of their impairment. Contrariwise, patients with bilateral fixed and dilated pupils, bleeding diathesis, a devastating injury, inability to follow them, inability to monitor ICP, primary decompression, brainstem injury, or patients that were treated according to the Lund protocol, or have received barbiturates before their randomization, should be excluded from the study. The surgical treatment comprises of a large unilateral fronto-temporo-parietal craniectomy for unilateral brain edema or of large bilateral fronto-temporo-parietal craniectomies for diffuse bilateral hemisphere swelling. The craniectomy extends from the frontal sinus anteriorly to the coronal suture posteriorly, and to the pterion laterally. It is accompanied by a large dural opening (dural flap pedicles are based on the superior sagittal sinus medially, and also a division of the falx anteriorly is required).

The primary endpoint of the Rescue-ICP study will be the outcome assessment at discharge (GOS score), and then at six months after injury (Extended GOS score), while secondary

endpoints will be: a) the assessment of outcome using the SF-36 and the SF-10 questionnaires, b) assessment of ICP control, c) length of stay in the ICU, d) hospital length of stay, and e) a detailed health-economic analysis of the collected data [Hutchinson, et al., 2006]. The results of the Rescue-ICP study are expected with great interest, and may greatly influence the future of DC and the overall management of patients suffering S-TBIs.

3. Decompressive craniectomy complications

Decompressive craniectomy has been associated with high mortality rates, mainly due to the severity of the underlying trauma, and also with numerous, and occasionally severe complications. Several factors have been identified as predisposing to the development of DC-associated complications. These include low GCS score upon admission, patient's old age, the presence of comorbidity, and the systematic preoperative anti-coagulant administration. Yang et al [Yang, et al., 2008] reported that the frequency of DC associated complications was 62% for admitting GCS scores 3-5, 39% for GCS scores 6-9, and 36 % for GCS scores >9. In addition, older patients (> 60 y.o) tend to have higher complications rates and prolonged ICU stay. Likewise, preoperative administration of anti-coagulant or antiplatelet medication increases the risk of intraoperative and/or postoperative bleeding. The alterations in cerebral compliance, CBF autoregulation, and altered CSF dynamics associated with S-TBIs, are the main reason for developing DC-associated complications. Several theories suggesting that the sudden change from prolonged severe compression to a state of maximal vasodilation and hyperemia following DC may cause loss of autoregulation, and may be responsible for the occurrence of DC associated complications [Aarabi, et al., 2009; Yang, et al., 2008]. The most commonly occurred complications may be divided to:

1. Perioperative complications

- a. Blossoming of pre-existing cerebral contusions. Expansion of pre-existing hemorrhagic contusions, as demonstrated on serial CT scans, has been reported as frequently as 40% of the cases, and may be considered inherent to the injury evolving process.
- b. Evolution and expansion of contralateral masses. The decompression provided by DC allows the expansion of contralateral masses (hematomas), which that were preoperatively tamponated by the swollen brain.
- c. External cerebral herniation. Expansion of the edematous brain through the bony defect may occur, especially during the first two postoperative weeks. The compression of the cortical veins of the herniated cerebral tissue leads to infarction, and unfortunately to further swelling. In small size decompressions, a mushroom-like herniation of the swollen parenchyma may occur. Therefore, most surgeons propose large craniectomies and wide dural openings for protecting the underlying cortical veins and for minimizing the risk of external cerebral herniation.

2. Early postoperative complications.

- a. Subdural effusions and hygromas are very often developed in cases of DC (Fig 6). Aarabi and his coworkers [Aarabi, et al., 2009] have reported subdural effusions and hygromas in 50% of their cases. Alteration of CSF circulation after a DC may incite the development of hygromas, which may progressively expand in volume. However, the hygromas very rarely demand surgical evacuation, and they usually resolve spontaneously.

Fig. 6. Postoperative CT scan demonstrating the development of a subdural hygroma.

- b. Impaired wound healing and infection may complicate a DC, and prolong patient's hospitalization. The performance of an extensive DC may compromise the skin flap blood supply (especially in the occipital area), and thus may increase the possibility of postoperative wound healing problems. Therefore, it is of paramount importance to maintain adequate skin flap blood supply by preserving the superficial temporal artery, and by avoiding tight skin suturing during closure. The observed high infection rate in patients undergoing DC may also be partially explained by the fact that the vast majority of these patients require prolonged ICU treatment and multiple interventions.
- c. Post-DC hydrocephalus incidence has been reported to be as high as 15 %, and is associated with poorer clinical outcome (Fig 7). The treatment of hydrocephalus with CSF shunting remains controversial, and the timing of intervention before or after cranioplasty is still disputable.

Fig. 7. Delayed postoperative CT scan demonstrating severe ventriculomegaly in a patient undergoing bilateral hemicraniectomy.

- d. The syndrome of the trephined is a frequent delayed complication and its diagnosis is often overlooked. Occasionally it may be presented with delayed onset of focal neurological deficits.
- e. Paradoxical herniation with brain stem compression and neurological deterioration is a very rare complication and may be precipitated by a lumbar puncture in a craniectomized patient.

3.1 Cranioplasty

One of the drawbacks of DC is the fact that a second surgical intervention is required to repair the bone defect. Autologous bone grafts may be used, which are preserved subcutaneously, or stored frozen under specific conditions in storage bone-banks. Tailor-made heterologous or synthetic bone grafts may also be used. The previous belief of delayed cranioplasty for minimizing the risk of infection, is seriously questioned nowadays, and an

increasing number of surgeons prefer early surgical skull reconstruction. Several surgeons support the idea of immediate cranioplasty even during the initial hospitalisation, as early as 2-4 weeks after trauma, when there is no suspicion of infection. The possibility of a new head injury with the exposed brain unprotected must be taken into consideration. Moreover, the observation of better functional outcome after cranioplasty, may further increase the number of early cranioplasty cases [Beauchamp, et al., 2010; Morina, et al., 2011].

4. Conclusions

The management of refractory post-traumatic intracranial hypertension remains a challenge for neurosurgeons, anesthesiologists, and neuro-intensivists. Cerebral ischemia leading to severe disability or death is unfortunately the only result expected. In order to deal with this dramatic situation only few treatment options exist. The ultimate measure to relieve uncontrollable ICP is an extensive decompressive craniectomy. It is proven that DC increases the volumetric compensatory capacity and reduces ICP. However, the well-documented risks and drastically complications of DC have to be seriously considered before performing a DC. In carefully selected cases these risks may be outweighed by the expected benefit. The results of the two ongoing prospective, randomized, controlled trials are expected to enlighten us on the exact role of DC in the management of patients with S-TBIs, and its effect on their long-term functional outcome.

5. References

- Aarabi, B., Hesdorffer, DC., Ahn, ES., Aresco, C., Scalea, TM., & Eisenberg, HM. (2006). Outcome following decompressive craniectomy for malignant swelling due to severe head injury. *J Neurosurg* Vol.104, No.4, (April 2006), pp. 469-79, ISSN 0022-3085
- Aarabi, B., Chesler, D., Maulucci, C., Blacklock, T., & Alexander, M. (2009). Dynamics of subdural hygroma following decompressive craniectomy: a comparative study. *Neurosurg Focus* Vol.26, No.6, (June 2009), pp.E8, ISSN 1092-0684
- Apuzzo, MLJ. (1993). Complication Avoidance and Management, In: *Brain Surgery*, Volume 2, part 4, pp.1283-1296, Churchill Livingstone Inc., ISBN 0-443-08709-1, New York
- Ban, SP., Son, YJ., Yang, HJ., Chung, YS., Lee, SH., & Han, DH. (2010). Analysis of complications following decompressive craniectomy for traumatic brain injury. *J Korean Neurosurg Soc* Vol.48, No.3, (September 2010), pp.244-50, ISSN 1225-8245
- Beauchamp, KM., Kashuk, J., Moore, EE., Bolles, G., Rabb, C., Seinfeld, J., Szentirmai, O., & Sauaia, A. (2010). Cranioplasty after post injury decompressive craniectomy: is timing of the essence? *J Trauma* Vol. 69, No.2, (August 2010), pp.270-4, ISSN 0022-5282
- Bor-Seng-Shu, E., Hirsch, R., Teixeira, MJ., De Andrade, AF., & Marino, R Jr. (2006). Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with posttraumatic brain swelling treated by surgical decompression. *J Neurosurg* Vol.104, No.1, (January 2006), pp.93-100, ISSN 0022-3085
- Brain Trauma Foundation BTF. (2007). Guidelines for the management of severe traumatic brain injury, 3rd edition. *J Neurotrauma* Vol. 24, No. 1, (2007), ISSN 0897-7151
- Bullock, MR., Chesnut, R., Ghajar, J., Gordon, D., Hartl, R., Newell, DW., Servadei, F., Walters, BC., & Wilberger, JE. (2006). Guidelines for the Surgical Management of

- Traumatic Brain Injury. *Neurosurgery* Vol. 58, No. 3, (March 2006), pp. S2-vi, ISSN 0148-396X
- Chibbaro, S., Marsella, M., Romano, A., Ippolito, S., & Benericetti, E. (2008). Combined internal uncrusectomy and decompressive craniectomy for the treatment of severe closed head injury: experience with 80 cases. *J Neurosurg* Vol.108, No.1, (January 2008), pp. 74-9, ISSN 0022-3085
- Cooper, DJ., Rosenfeld, JV., Murray, L., Wolfe, R., Ponsford, J., Davies, A., D'Urso, P., Pellegrino, V., Malham, G., & Kossmann, T. (2008). Early decompressive craniectomy for patients with severe traumatic brain injury and refractory intracranial hypertension--a pilot randomized trial. *J Crit Care* Vol.23, No.3, (September 2008), pp. 387-93, ISSN 1541-6933
- Cooper, DJ., Rosenfeld, JV., Murray, L., Arabi, YM., Davies, AR., D'Urso, P., Kossmann, T., Ponsford, J., Seppelt, I., Reilly, P., & Wolfe, R.; DECRA Trial Investigators; Australian and New Zealand Intensive Care Society Clinical Trials Group. (2011). Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med* Vol.364, No.16, (2011 April 21), pp.1493-502, ISSN 0028-4793
- Guerra, WK., Gaab, MR., Dietz, H., Mueller, JU., Piek, J., & Fritsch, MJ. (1999). Surgical decompression for traumatic brain swelling: indications and results. *J Neurosurg* Vol.90, No.2, (February 1999), pp.187-96, ISSN 0022-3085
- Cushing H. (1905). The establishment of cerebral hernia as a decompressive measure for inaccessible brain tumors; with the description of intramuscular methods of making the bone defect in temporal and occipital regions. *Surg Gynecol Obstet* Vol.1, pp.297-314, ISSN 0039-6087
- Danish, SF., Barone, D., Lega, BC., & Stein, SC. (2009). Quality of life after hemicraniectomy for traumatic brain injury in adults. A review of the literature. *Neurosurg Focus* Vol.26, No.6, (June 2009), pp.E2, ISSN 1092-0684
- Honeybul, S., Ho, KM., Lind, CR., & Gillett, GR. (2011). Surgical intervention for severe head injury: ethical considerations when performing life-saving but non-restorative surgery. *Acta Neurochir (Wien)* Vol. 153, No.5, (May 2011), pp. 1105-10, ISSN 0001-6268
- Howard, JL., Cipolle, MD., Anderson, M., Sabella, V., Shollenberger, D., Li, PM., & Pasquale, MD. (2008). Outcome after decompressive craniectomy for the treatment of severe traumatic brain injury. *J Trauma* Vol.65, No.2, (August 2008), pp.380-6, ISSN 0022-5282
- Huang, X., & Wen, L. (2010). Technical considerations in decompressive craniectomy in the treatment of traumatic brain injury. *Int J Med Sci* Vol.7, No.6, (November 8), pp. 385-90, ISSN 1449-1907
- Hutchinson, PJ., Menon, DK., & Kirkpatrick, PJ. (2005). Decompressive craniectomy in traumatic brain injury--time for randomised trials? *Acta Neurochir (Wien)* Vol.147, No.1, (January 2005), pp. 1-3, ISSN 0001-6268
- Hutchinson, PJ., Corteen, E., Czosnyka, M., Mendelow, AD., Menon, DK., Mitchell, P., Murray, G., Pickard, JD., Rickels, E., Sahuquillo, J., Servadei, F., Teasdale, GM., Timofeev, I., Unterberg, A., & Kirkpatrick, PJ. (2006). Decompressive craniectomy in traumatic brain injury: the randomized multicenter RESCUE icp study (www.RESCUEicp.com). *Acta Neurochir Suppl* Vol.96, pp.17-20, ISSN 0001-6268

- Jagannathan, J., Okonkwo, DO., Dumont, AS., Ahmed, H., Bahari, A., Prevedello, DM., Jane, JA Sr., & Jane, JA Jr. (2007). Outcome following decompressive craniectomy in children with severe traumatic brain injury: a 10-year single-center experience with long-term follow up. *J Neurosurg* Vol.106, No.4, (April 2007), pp.268-75, ISSN 0022-3085
- Kenning, TJ., Gandhi, RH., & German, JW. (2009). A comparison of hinge craniotomy and decompressive craniectomy for the treatment of malignant intracranial hypertension: early clinical and radiographic analysis. *Neurosurg Focus* Vol.26, No.6, (June 2009),pp. E6, ISSN 1092-0684
- Laalo, JP., Kurki, TJ., Sonninen, PH., & Tenovuo, OS. (2009). Reliability of diagnosis of traumatic brain injury by computed tomography in the acute phase. *J Neurotrauma* Vol. 26, No.12, (December 2009), pp. 2169-78, ISSN 1557-9042
- Maas, AI., Dearden, M., Teasdale, GM., Braakman, R., Cohadon, F., Iannotti, F., Karimi, A., Lapierre, F., Murray, G., Ohman, J., Persson, L., Servadei, F., Stocchetti, N., & Unterberg, A. (1997). EBIC-guidelines for management of severe head injury in adults. European Brain Injury Consortium. *Acta Neurochir (Wien)* Vol.139, No.4, pp. 286-94, ISSN 0001-6268
- Marmarou, A., Anderson, RL., Ward, JD., Choi, SC., Young, HF., Eisenberg, HM., Foulkes, MA., Marshall, LF., & Jane, JA. (1991). Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg* Vol. 75, (November 1991),pp. S59-66, ISSN 0022-3085
- Morgalla, MH., Will, BE., Roser, F., & Tatagiba, M. (2008). Do long-term results justify decompressive craniectomy after severe traumatic brain injury? *J Neurosurg* Vol.109, No.4, (October 2008), pp.685-90, ISSN 0022-3085
- Morina, A., Kelmendi, F., Morina, Q., Dragusha, S., Ahmeti, F., Morina, D., & Gashi, K. (2011). Cranioplasty with subcutaneously preserved autologous bone grafts in abdominal wall-Experience with 75 cases in a post-war country Kosova. *Surg Neurol Int* Vol.2, (May 28), pp. 72. ISSN 2152-7806
- Münch, E. Horn, P., Schürer, L., Piepgras, A., Paul, T., & Schmiedek, P. (2000). Management of Severe Traumatic Brain Injury by Decompressive Craniectomy. *Neurosurgery* Vol. 47, No. 2, (August 2000), pp. 315-323, ISSN 0148-396X
- Polin, RS., Shaffrey, ME., Bogaev, CA., Tisdale, N., Germanson, T., Bocchicchio, B, & Jane, JA. Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. (1997). *Neurosurgery* Vol.41, No.1, (July 1997), pp.84-92, ISSN 0148-396X
- Ruf, B., Heckmann, M., Schroth, I., Hügens-Penzel, M., Reiss, I., Borkhardt, A., Gortner, L., & Jödicke, A. (2003). Early decompressive craniectomy and duraplasty for refractory intracranial hypertension in children: results of a pilot study. *Crit Care* Vol. 7, No.6, (December 2003), pp. R133-8, ISSN 1364-8535
- Sahuquillo, J., & Arikan, F. (2006). Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. *Cochrane Database Syst Rev* Vol, 25, No.1, pp.CD003983
- Schmidt JH, 3rd., Reyes, BJ., Fischer, R., & Flaherty, SK. (2007). Use of hinge craniotomy for cerebral decompression. Technical note. *J Neurosurg* Vol.107, No.3, (September 2007), pp.678-82, ISSN0022-3085

- Stiver, SI. (2009). Complications of decompressive craniectomy for traumatic brain injury. *Neurosurg Focus* Vol.26, No.6, (June 2009), pp. E7, ISSN 1092-0684
- Timofeev, I., Czosnyka, M., Nortje, J., Smielewski, P., Kirkpatrick, P., Gupta, A., & Hutchinson, P. (2008). Effect of decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury. *J Neurosurg* Vol.108, No.1, (January 2008), pp.66-73, ISSN 0022-3085
- Valadka, AB., & Robertson, CS. (2007). Surgery of cerebral trauma and associated critical care. *Neurosurgery* Vol.61, No.1, (July 2007), pp.203-20, ISSN 1220-8841
- Weiner, GM., Lacey, MR., Mackenzie, L., Shah, DP., Frangos, SG., Grady, MS., Kofke, A., Levine, J., Schuster, J., & Le Roux, PD. (2010). Decompressive craniectomy for elevated intracranial pressure and its effect on the cumulative ischemic burden and therapeutic intensity levels after severe traumatic brain injury. *Neurosurgery* Vol. 66, No.6, (June 2010), pp. 1111-8, ISSN 0148-396X
- Yang, XF., Wen, L., Shen, F., Li, G., Lou, R., Liu, WG., & Zhan, RY. (2008). Surgical complications secondary to decompressive craniectomy in patients with a head injury: a series of 108 consecutive cases. *Acta Neurochir (Wien)* Vol.150, No.12, (December 2008), pp, 1241-7, ISSN 0001-6268

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The present two volume book "Brain Injury" is distinctive in its presentation and includes a wealth of updated information on many aspects in the field of brain injury. The Book is devoted to the pathogenesis of brain injury, concepts in cerebral blood flow and metabolism, investigative approaches and monitoring of brain injured, different protective mechanisms and recovery and management approach to these individuals, functional and endocrine aspects of brain injuries, approaches to rehabilitation of brain injured and preventive aspects of traumatic brain injuries. The collective contribution from experts in brain injury research area would be successfully conveyed to the readers and readers will find this book to be a valuable guide to further develop their understanding about brain injury.

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