neurocritical care society Neurocrit Care (2008) 8:456–470
DOI 10.1007/s12028-008-9082-y

REVIEW ARTICLE

Decompressive Craniectomy

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Published online: 8 April 2008
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Abstract Decompressive Craniectomy (DC) is used to treat elevated intracranial pressure that is unresponsive to conventional treatment modalities. The underlying cause of intracranial hypertension may vary and consequently there is a broad range of literature on the uses of this procedure. Traumatic brain injury (TBI), middle cerebral artery (MCA) infarction, and aneurysmal subarachnoid hemorrhage (SAH) are three conditions for which DC has been predominantly used in the past. Despite an increasing number of reports supportive of DC, the controversy over the suitability of the procedure and criteria for patient selection remains unresolved. Although the majority of published studies is retrospective, the recent publication of several randomized prospective studies prompts a reevaluation of the utility of DC. We review the literature concerning the use of DC in TBI, MCA infarction, and SAH and address the evidence regarding common questions pertaining to the timing of and laterality of the procedure. We conclude that at the time of this review, there still remains insufficient data to support the routine use of DC in TBI, stroke or SAH. There is evidence that early and aggressive use of DC in good-grade patients may improve outcome, but the notion that DC is indicated in these patients is contentious. At this point, the indication for DC should be individualized and its potential implications on long-term outcomes should be comprehensively discussed with the caregivers.

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Keywords Decompressive craniectomy · Intracranial hypertension · Cerebral edema · Surgical · Subarachnoid hemorrhage · Traumatic brain injury · Stroke · Infarction · Review

Introduction

Decompressive Craniectomy (DC) describes the temporary removal of a portion of the skull for the relief of high intracranial pressure. This can be achieved by removal of the fronto-temporal-occipital bone over one or both cranial hemispheres or can involve a bi-lateral removal [1, 2]. High intracranial pressure within the fixed-volume skull, resulting from cerebral edema, intracranial hemorrhage, or a space-occupying hematoma can quickly lead to secondary brain damage, herniation, permanent neurological damage, or death. DC effectively increases the volume that the brain can occupy under the scalp and may minimize ischemic damage by allowing increased cerebral blood flow and tissue oxygenation [3–5]. Common indications for DC are traumatic brain injury (TBI), malignant middle cerebral artery (MCA) infarction, and subarachnoid hemorrhage (SAH). DC has been described in many studies as a life-saving intervention, which consistently decreases mortality and can often improve outcomes, especially when performed early in the course of the disease [6–9]. Despite growing evidence that better outcomes are associated with timely surgery, DC is still used mainly as a salvage procedure after all other options of ICP management have been exhausted. The perception of DC as a high-risk invasive treatment, historical preference for medical and non-surgical therapies, and a lack of conclusive data on the subject have limited widespread advocacy of the procedure and sparked debate concerning its benefits and real risks. In this article, we explore the

historical significance of DC, summarize recent literature on the procedure for the three main indications, and discuss some of the common issues surrounding its application.

Historical Uses of DC

The surgical removal of a portion of the skull, either for medical or superstitious reasons is known in the anthropological context as "trepanation." This commonly involved the drilling or scraping of a hole into the skull. Evidence of the most primitive craniectomy have been found in skeletons up to 6,000 years old, with well documented archaeological findings spread from pre-Columbian Peru to bronze-age Europe and Neolithic Africa [10]. In the late 1800's, the French physician and surgeon Paul Broca became intensely interested in the subject of primitive trepanation; he controversially theorized that this peculiar ancient practice was the earliest evidence of a surgical treatment for the build up of intracranial pressure [11]. Modern use of the early trepanation technique is still seen in isolated tribal societies of the South Pacific and Africa, mainly for the treatment of epilepsy and headache [12]. There is a broad speculation as to the reasons that ancient practitioners removed skull portions on living subjects, but the logic in the observed modern cases argues that opening a hole in the skull creates a way of escape for the demons or spirits possessing an ailing person [12].

Surgeons in ancient Greece and Rome that had been trained in the Hippocratic school of medicine, that emphasized the balance of certain humors of the body, used a variety of procedures that relieved pressures and resulting disease due to a buildup of a humor, including trepanation. Galen, in a more practical vein, advocated the use of skull removal for closed head injuries and splintered skull fractures in his surgical treatise. The medieval Arab physician Al-Zahrawi, known in Western literature as Abulcasis, developed the use of "non-sinking" drills designed to avoid piercing the dura during the decompression procedure [13].

In 1901, Emil Theodore Kocher became the first in the modern literature to describe the technique of surgical decompression and its value in relieving intracranial pressure [14]. Harvey Cushing later described a bilateral subtemporal craniectomy in order to relieve pressure on the brain from an inaccessible tumor [15].

Surgical Technique

DC does not describe a single standardized procedure but rather the manner of achieving adequate and lasting decompression of the brain in a given patient with the least invasive method available. Therefore, a variety of methods and variations exist with their advantages and disadvantages in a given situation. Nevertheless, there are some common points to consider.

Unilateral DC may be indicated in patients with unilateral hemispheric swelling and midline shift after TBI, ischemic stroke, or SAH and is performed utilizing a large question-mark shaped incision and craniectomy. Appropriate sizing of the craniectomy is of utmost importance since a small craniectomy may cause trans-craniectomy-defect brain herniation and venous compression along the bony margin with consequent venous infarction and exacerbated edema.

Processes that result in diffuse brain edema can be decompressed using a bilateral approach [1] or by performing a decompression of the non-dominant hemisphere. Bilateral DC may be performed by either decompressing each hemisphere through separate unilateral DC or through a bifrontal craniectomy extending from the floor of the anterior fossa to the coronal suture and the pterion bilaterally.

In our opinion, adequate decompression can only be achieved by opening the dura in a wide and stellate fashion. In the past, some surgeons chose to leave the dura intact [16], but durotomy with dural expansion has been shown to lower ICP to 30% of the pre-surgical levels as opposed to 85% when the dura left intact [5, 17]. If ICP levels remain above 30 mmHg postoperatively with the addition of medical treatment, anterior temporal lobectomy may be performed to further increase the space available for the swollen brain to expand into [6, 18].

The removed bone flap may be stored in a sub-zero degrees Fahrenheit (−20°C) temperature freezer or embedded through a separate incision in the subcutaneous layer of the patient's abdominal wall in anticipation of the subsequent cranioplasty needed to reconstruct the skull contour and offer long-term physical protection. The requirement for this second surgery to replace the bone flap is an added risk, which must be considered in the initial surgical decision. There are also complications that are specific to the cranioplasty surgery, including bone resorption [19, 20], osteomyelitis [21], and hypovascular bone necrosis [22]. Recent reports have suggested that earlier replacement of the bone flap, at 5–8 weeks instead of the more conventional 2–3 months, can reverse some of the common recovery symptoms and even create a better overall functional outcome [23, 24].

Clinical Indications

The majority of the available literature describes DC in reference to a particular primary event, most commonly TBI, SAH, or hemispheric MCA infarction (Figs. 1–3). Less frequent uses of DC with positive results for the treatment of refractory ICP have been reported in other

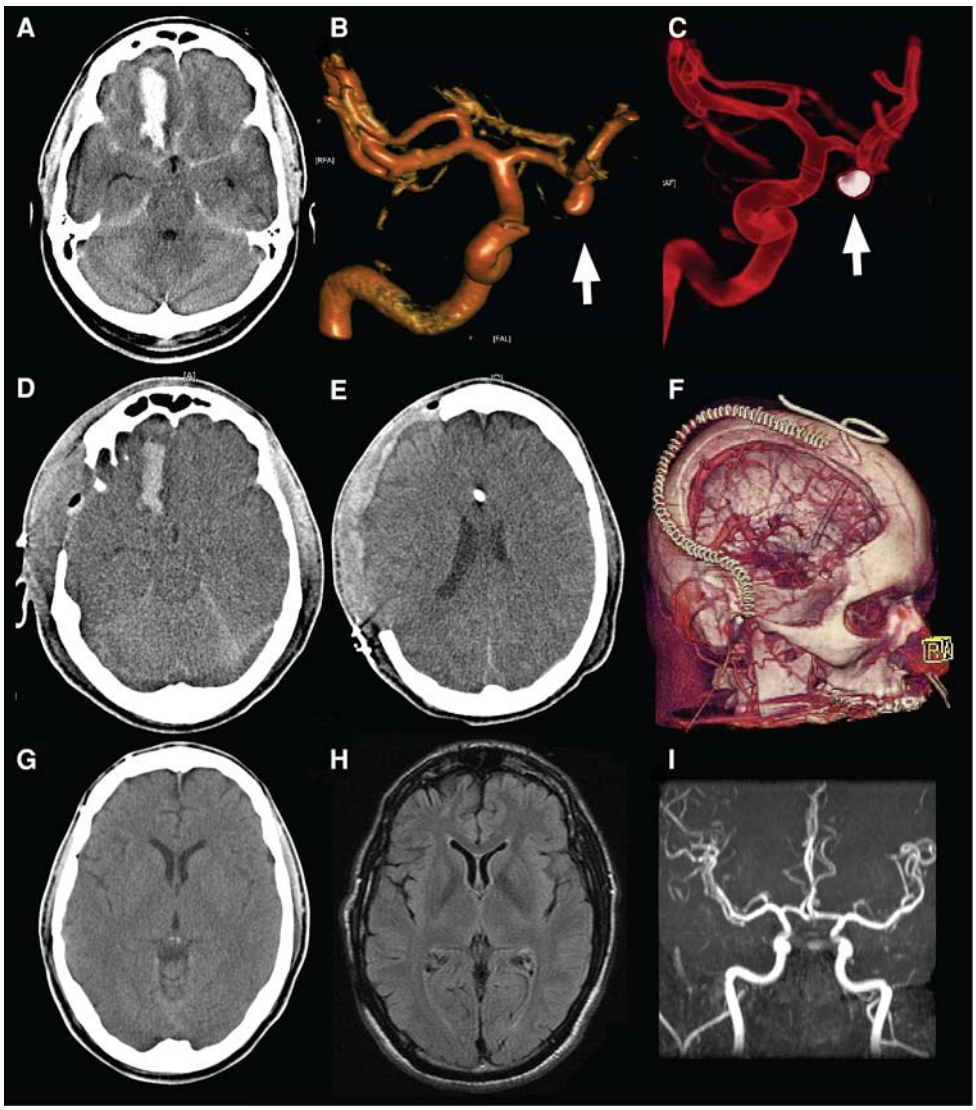


Fig. 1 Case 1: A 37 year old man presented with sudden-onset headache and rapidly deteriorated, suffered from cardiac arrest and was successfully resuscitated. Non-contrast enhanced computertomographic (CT) images of the head revealed subarachnoid hemorrhage and small gyrus rectus hemorrhage at the base of the right frontal lobe (**A**). A berry aneurysm of the anterior communicating artery was seen on CT angiography and subsequently confirmed on digital subtraction angiography (**B**, arrow). The patient underwent successful endovascular coil embolization of the aneurysm (**C**, arrow points to the coil mass). The patient suffered from persistently elevated intracranial pressures (ICP) and a second cardiac arrest. 12 h after admission the patient underwent a right frontotemporal craniectomy (**D**–**F**) and had an excellent clinical outcome (mRS of 0) without sequelae. Cranioplasty was performed 7 weeks later (**G**), and magnetic resonance (MR) fluid attenuated inversion recovery images show no strokes (**H**) and no residual filling on MR angiography (**I**) and catheter angiography at follow-up after 21 months

conditions such as subdural empyema [25], encephalitis [26], toxoplasmosis [27], and encephalopathy related to Reye's syndrome [28]. In addition to the causes listed above, intracerebral hemorrhage (ICH) can also result from hypertension, arteriovenous malformations, cavernous malformations, tumors, and dural arteriovenous fistulas. Persistently elevated ICP is a common feature in all these clinical situations, leading to secondary brain injury. In the case of ICH no studies have been conducted to assess the value of DC, likely because the controversy here is centered around the issue of removing the clot [29]. There are few articles discussing the use of DC in the management of elevated ICP not limited to a particular condition [17, 30].

Malignant MCA Infarction

There are a large number of published reports that demonstrate a consistent reduction of mortality after complete MCA infarction from around 80% with maximum medical treatment alone to as low as 8% with very early DC [6, 7, 18, 31–40]. Animal models of DC for MCA infarction have also shown not only lower ICP and decreased mortality, but also a reduction in infarction volume as well [41–43]. Unlike other etiologies of intractable ICP, use of DC as a treatment for malignant MCA infarction is described by a number of prospective studies.

Randomized Trials

The recent publication of data from three European randomized trials studying the use of DC in MCA infarction [44–47] was an important addition, see also Table 1. The French DECIMAL (www.clincaltrials.gov identifier: NCT00190203), Dutch HAMLET (ISRCTN94237756), and German DESTINY (ISRCTN01258591) trials were similarly designed multi-center, randomized control

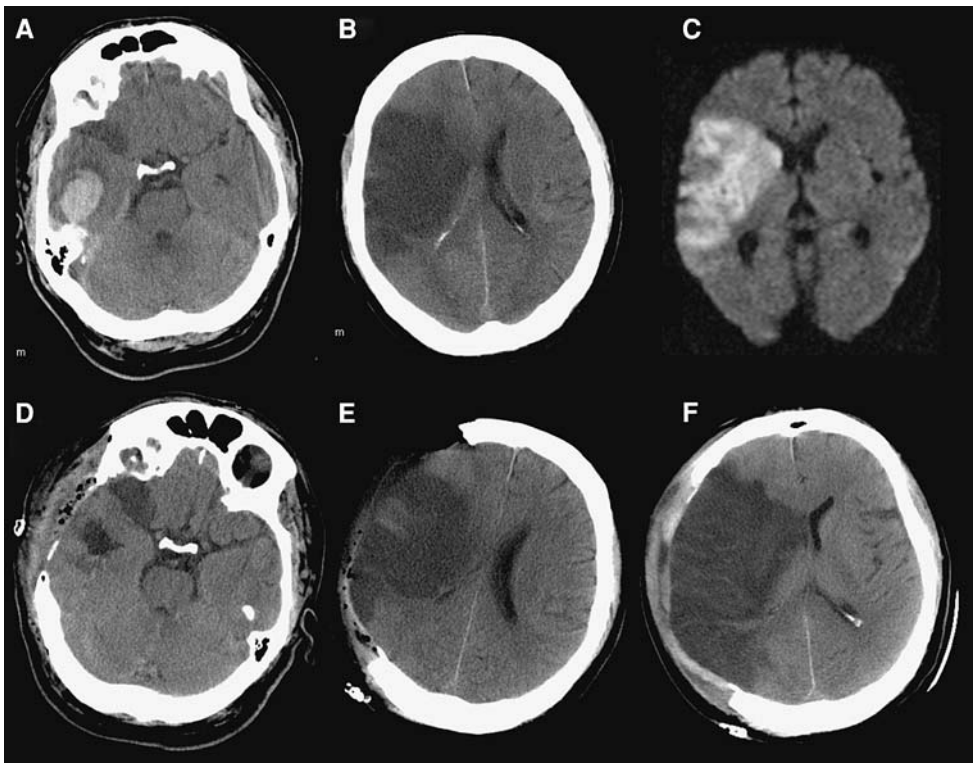


Fig. 2 Case 2: A 63 year-old man returned from an international flight when he noted slurred speech which he initially ignored. He developed gait unsteadiness, fell and was admitted to a hospital. Non-contrast enhanced computer tomographic images of the head showed hemorrhagic conversion of a portion of his right ischemic stroke on day 2 after the insult (**A**) and a large hypodense area in the right middle cerebral artery distribution (**B**). Diffusion-weighted magnetic resonance images supported the diagnosis of ischemia (**C**) and the patient underwent evacuation of the hemorrhage and a right frontotemporal craniectomy (**D**, **E**, and one week later **F**). Cranioplasty was performed 6 months later and at 3.5 year follow up the patient had a residual spastic left hemiparesis

studies. Somewhat unconventionally, the decision to pool data was made to avoid further unnecessary, and possibly unethical, patient randomization at the individual centers, and also to speed up the release of data of a sufficient size for meaningful analysis [47]. Of the three trials two interrupted recruitment early in 2006 after preliminary results became available: the DECIMAL trial because of slow recruitment and a significant difference in mortality between the treatment groups favoring surgery; and the DESTINY trial after a predefined sequential analysis showed a significant benefit of surgery on mortality. Each trial was halted after approximately half of the originally desired number of patients had been enrolled [47].

The final result was similar in both trials with a 52.8% reduction in mortality after early DC, 6–30 h after the onset of symptoms, for the 38 patients included in the DECIMAL trial [45]. In the DESTINY study that included 32 patients, 88% of the patients randomized to DC survived, compared to only 47% of the patients treated conservatively. After 12 months the group that had undergone DC showed a trend toward better outcomes [44].

HAMLET is still ongoing [46]. The paper reporting the combined results from the three studies included a total of 93 patients with 51 randomized to DC and 42 to conservative treatment. All patients were under 60 years of age and DC was performed within 48 h of infarction. One-year mortality was 22% (11/51) in the DC group, and far worse in conservative group with 71% (30/42). Modified Rankin Scale (mRS) scores showed that favorable outcomes (mRS < 5) were obtained in 75% of DC patients as opposed to just 24% in conservatively treated patients. This study provides strong evidence of the survival benefit of DC, but the authors point out that the probability of surviving in a dependant state (mRS = 4) is increased greater than ten times, from 2% (1/42) to 31% (16/52) after DC. Probability of severe disability (mRS = 5), however, was not increased by DC (5% (2/42) conservative group and 4% (2/51) DC).

There are two additional randomized trials for DC use in malignant MCA stroke, HeMMI in the Philippines and HeaDDfirst in the United States at the University of Chicago. HeaDDfirst was stopped after 26 patients, less than half of the proposed sample size, because the difference in 21-day mortality between surgical cases and medical cases was so pronounced in favor of surgery (26.7% surgical versus 45.5% medical) [48].

Non-Randomized Trials

Non-randomized studies have also demonstrated an increased survival associated with DC. Schwab et al. carried out a prospective study of 63 DC patients that demonstrated the benefit of early surgical intervention within 24 h. The patients were dichotomized into late- and early-DC cohorts by a surgical treatment time of 24 h from ictus [7]. DC was initiated at an average of 39 and 21 h

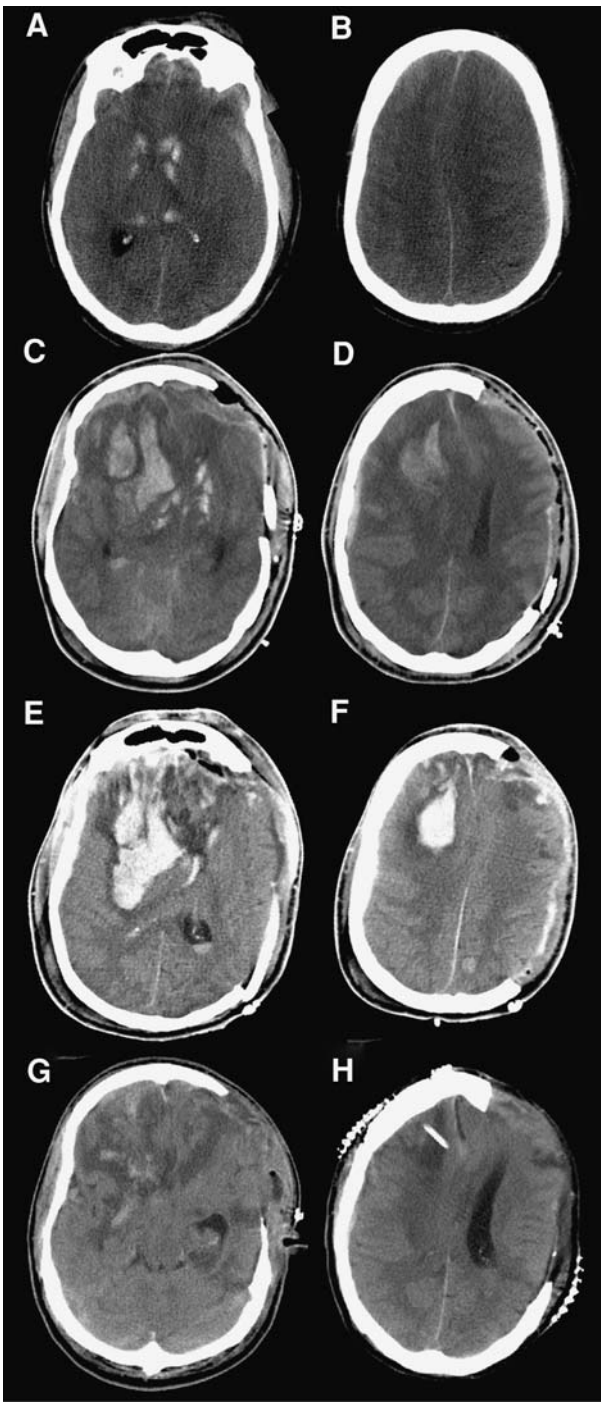


Fig. 3 Case 3: A 49 year-old gas station attendant was hit by a car at high speed and brought to a trauma center. His initial non-contrast enhanced computed tomogram of the head showed a left subdural hematoma with diffuse small intraparenchymal hemorrhages (**A**) and midline shift to the right (**B**). Evacuation of the subdural hematoma and left craniectomy was performed (**C**, **D**) with reversal of the midline shift (**D**) at the end of the procedure and progression of the hemorrhage into right frontal contusions (**E**, **F**). Evacuation of the right hemorrhages and placement of a ventriculostomy was performed (**G**, **H**). The patient improved to open his eyes to stimulation; however, in light of his overall poor prognosis, the family elected to withdraw support

from infarction, respectively. The mortality rates were 34.4% (11/32) in the late group and 16% (5/31) in the early group. These numbers were compared to a historical control which had a mortality of 78% (43/55) with conservative medical treatment alone. The early-DC group had an average intensive care stay of 7.4 days and a mean Barthel Index (BI) score of 68, as opposed to 13.3 ICU days and 62.8 mean BI for the late-DC group. The selection criteria of uncal herniation signs and midline shift were revised in the middle of the study, which led to an earlier surgical decision for the patients seen later in the duration of the study. Thus, the patients seen at the beginning of the study were those placed in the "late" DC group. In this group, 24 of 32 patients (75%) showed clinical signs of uncal herniation prior to surgery and all of the patients had midline shift evident on pre-surgery CT scan with an average of 10 mm shift at the septum pellucidum. The early-DC group had only 4 patients with signs of uncal herniation and 6 patients showing midline shift of an average 3 mm [7]. These selection criteria were the only difference between the cohorts that led to earlier DC and a mortality rate lowered from 34.4% to 16% [7].

Mori et al. retrospectively examined 71 patients with infarct volumes of greater than 200 cc and associated edema. The patients were separated into 3 study cohorts: 2 DC treatment groups based on the presence or absence of uncal herniation, called late and early groups, respectively, and a third group that received medical treatment alone. 1- and 6-month mortality was evaluated as well as 6-month Glasgow Outcome Scale (GOS) and Barthel Index scores. Mortality was significantly reduced in the late surgery group versus the conservative group (17.2% 6 month and 27.9% 1 year, compared to 61.9% and 71.4%, respectively, for the conservative group). There was a further reduction in mortality with the early-DC group to 4.8% at 6 months and 19.1% at 1 year but this was not a statistically significant difference compared with the late group. GOS and BI scores post-surgery were both significantly improved for the early surgery group compared to the late group (mean of 52.9 vs. 26.9) and the late group showed very little difference in BI score compared to the conservative group, with averages of 26.9 and 28.3, respectively. Midline shift seen on CT was significantly lower pre-surgery for the early-DC group compared to the late-DC group (mean of 6.8 mm versus 12.8 mm; $P < 0.05$) and also showed significance after surgery (mean 4.4 mm vs. 10.2 mm; $P < 0.05$). Glasgow Coma Score (GCS) before surgery was significantly higher for the early surgery group as well (11.2 vs. 6.6). All three groups in this study were older than most other studies with exclusion criteria of 80 years old, which produced group averages of 72 years for the conservative group, 65 years for the late surgery group and 64 years for the early surgery group. Patients under

Table 1 Randomized controlled trials on DC for malignant MCA infarction

Study	Studytype	N	Age/Sex	Inclusion criteria	Fu length	Measures	Outcome
DECIMAL, 2007 [45]	Multicenter RCT	38 (Target number 60)	43 ± 9, 47% Male	Malignant MCA stroke by radiological criteria within 24 h of stroke	12 months (stopped)	mRS < 4 at 6 months	50% with mRS 0–3 after DC versus 22% (NS), 53% absolute reduction in death after DC (S)
DESTINY, 2007 [44]	Multicenter RCT	32 (Target number 60)	45 ± 9, 47% Male	Malignant MCA stroke by radiological criteria within 24 h of stroke	12 months (stopped)	mRS < 4 at 6 months	47% with mRS 0–3 after DC versus 27% (NS)
HAMLET, 2006 [46]	Multicenter RCT	23 (Target number 112)	48 ± 10, 52% Male	Malignant MCA stroke by clinical and radiological criteria within 96 h of stroke	12 months (stopped)	mRS < 4 at 12 months	29% with mRS 0–3 after DC versus 11% (NS)
Pooled Analysis, 2007 [47]	Multicenter RCT	93		Enrollment in DECIMAL, DESTINY or HAMLET, recruited in first 48 h after stroke onset	12 months	Favorable (mRS 0–4) versus unfavorable (mRS 5–6) at 12 months	43% with mRS 0–3 after DC versus 21% (S), absolute risk reduction 51% (S), NNT for survival 2, for mRS 0–3 outcome 4
HeMMI	Single center RCT	Target number 56	18–65 years	MCA stroke, clinical deterioration within 72 h	6 months	mRS and Barthel Index at discharge, 2 weeks, 1,3 and 6 months	Ongoing
HeaDDfirst	Multicenter RCT	Target number 75	18–75 years	Presence of stroke, deterioration after 96 h after onset (clinical or radiological)	6 months	Mortality, functional and subjective outcome and usage of healthcare resources at 3 weeks, 3 and 6 months	Halted with 26 patients, publication pending

60 years showed the best outcomes with an average BI score of 80.0. The investigators also included many more dominant hemisphere strokes than any other study with a majority seen in each group, but did not find statistical significance in the outcomes of these patients (Ratios of dominant to non-dominant patients were 12:9 for the conservative group, 20:9 for the late group and 18:3 for the early group) [49].

Infarction Volume Threshold

Corroborated by another study [50] the DECIMAL study confirmed that an infarct volume of at least 145 cc serves as a useful cutoff, of 8 patients with a smaller infarct none died, and conversely no patient with an infarct volume greater than 210 cc survived without a DC [45]. In both the separate studies and in the pooled analysis no difference in outcome was found in patients that underwent DC in less than or after 24 h. The value of late DC performed beyond 48 h however cannot be assessed since these patients were excluded from these studies.

Age as a Factor in the Indication

A weakness of the randomized trials published to date [44, 45, 47] is the lack of data on older patients. Patients older than 60 years were excluded from these studies. In a systematic review of published DC cases younger age (less than 50 years) was the only preoperative determinant of survival with good clinical outcome [51]. In the DECIMAL trial, age was a stronger predictor of outcome than infarct volume [45]. The findings of these trials cannot readily be extrapolated to older patients, which make up a significant portion of the patient population with strokes. Also, the CT and MRI findings were not discussed because of systematic differences in the timing and modalities used among the three trials [47].

Prognostic Factors

In a recently published report, Chen et al. investigated the prognostic factors associated with DC treatment of 60 malignant MCA infarction patients. 12-month mortality was 26.6% (44/60), and 65.9% (29/44) of surviving patients had a favorable outcome (BI > 60). DC performed prior to clinical signs of herniation in patients younger than 60 years of age was significantly associated with favorable outcome. 94% of patients under 60 years old had a BI greater then 60% and 63.6% (7/11) of patients who presented with uncal signs had BI scores less than 60 indicating a poor outcome. Furthermore, higher mortality was not only associated with age and pre-surgical uncal signs, but was linked to the involvement of more than one vascular territory and a time to surgery later than 24 h [18].

The long-term benefit of DC remains unclear, especially in light of the lack of consistent follow-up information and seemingly conflicting results in the literature. Skeptics of the procedure point out that many of the studies touting the benefits of DC concentrate too much on mortality rate reduction and do not sufficiently address the functional outcome and quality of life issues for surviving patients [52–54].

The underlying reason for this debate is that quality of life after DC is not consistently measured by investigators, nor is there a universal test or scale that everyone agrees should be applied. Some of the common tools are the Barthel Index, the Glasgow Outcomes Scale, and the Rankin Scale. Foerch et al. argued that BI scores tend to underestimate the health burden of DC for MCA infarction victims because the test lacks detail in critical areas regarding communication, hand function, and orientation, all of which contribute greatly to the true functional outcome of stroke victims [54]. They found a discrepancy between the literature and their own case series which used multiple scales and a subjective questionnaire. In a prospective study which incorporated neuropsychological testing into the outcomes assessment, they found high rates of depression, and all surviving patients showed low attention span and poor visuo-spatial skills which contributed to a lower quality of life than otherwise would have been reported [53]. This study focused exclusively on patients with right-sided hemicraniectomy but the authors extrapolated the results to include an even more pessimistic outlook for left-sided DC in right-handed patients.

Moreover, the time to follow up varies highly between studies, ranging from 3 months to 1 or 2 years. Uhl and coworkers retrospectively studied 188 malignant MCA infarction patients with follow up at 3, 6, and 12 months and showed that mortality increased from 8% to 38%, then to 44% for these respective times [31].

Some studies report positive results in quantitative measures but the authors make clear that these results did not correlate to a beneficial functional outcome for the patient, which may be correct but may also represent an analysis bias of the authors [3, 38, 55]. For example, a number of studies have demonstrated that decompression led to effective relief of midline shift and cisternal visibility on post-DC CT scan, but this fact did not correlate to a better patient outcome [30, 34]. Jaeger et al. also demonstrated how decompression immediately decreased ICP and increased the PtiO2 of three patients with cerebral hypertension secondary to aSAH. The functional result was death for one patient and persistent vegetative state for the

other two [3]. At least two studies argued that the reduced mortality for elderly MCA stroke patients did not coincide with any long-term beneficial outcomes [38, 53].

Timing of DC for MCA Infarction

In an earlier publication by the same group, the timing of DC was explored in a study of 52 malignant MCA infarction patients separated into 3 cohorts: Ultra-early DC (within 6 h of symptom onset), DC after 6 h, and no DC at all [6]. The Ultra-early group had a mortality of 8.3% (1/12), compared to 36.7% (11/30) for the later DC group and 80% (8/10) for the medically treated group. The Ultra-early group also showed statistically significant reduction in ICU stay duration of 12 days compared to 18 days in the other DC group, and better Barthel Index functional outcome scores (mean of 70 vs. 53) [6]. Excluding a patient with fatal outcome, all members of the Ultra-early group regained consciousness by the 7th postoperative day. Only 55% of the later DC group and none of the members of the non-surgical group achieved consciousness by this time [6].

DC for Dominant Hemisphere Infarction

Despite consistent positive results with regard to improved MCA stroke survival rates, questions remain concerning the functional benefits and real outcomes associated with DC after malignant MCA infarction. This is illustrated by the debate surrounding the question of whether to operate on dominant versus non-dominant sided infarctions. Patients with dominant-side hemicraniectomy are underrepresented in the literature despite a lack of evidence linking dominant-side decompression to worse functional outcome [56]. The literature reflects a traditional unwillingness to operate on the dominant side of the brain, as it is assumed that decompression after a left-sided insult would inevitably lead to a poor quality of life with increased dependence [36, 53, 57]. Some studies have refuted this notion and there is encouraging data that left sided DC is not only safe, but can achieve good outcomes on par with right-sided DC.

The issue of DC on the dominant hemisphere was explicitly addressed by a prespecified subset analysis in the DECIMAL trial that did not show a significant difference in the modified Rankin scores of survivors with dominant side DC compared to nondominant side DC after 1-year follow-up [45]. In the pooled analysis of the three European randomized trials, the benefit of surgery in preventing bad outcomes was independent of the presence of aphasia at baseline [47]. In the DESTINY trial all survivors that had undergone surgery agreed with the decision to perform surgery in retrospect, even if they were still experiencing aphasia [44]. These results become even more important in light of a study which showed that significant improvement occurred after a mean period of 470 days in 13 of 14 patients with dominant-hemisphere strokes that were treated with DC in various aspects of their aphasia, and that younger age and early DC were predictors of improved recovery [58].

In two of the large prospective studies discussed above, Schwab et al. [7] and Chen et al. [18] with a combined 141 patients, both groups found no difference in quality of life outcomes for patients receiving dominant versus non-dominant hemicraniectomy. Schwab et al. operated on 11 dominant-side patients, but only included those patients with incomplete aphasia before deterioration. None of these patients progressed to global aphasia and only 3 had minor aphasia and were able to return to their previous occupations [7]. Chen et al. operated on patients with complete aphasia and found that 82.3% (14/17) of the dominant sided patients achieved BI scores >60, and were classified as good outcomes [18]. In a prospective study by Killincer et al. of 32 patients, 6 out of the 7 patients with good outcome after 6 months (modified Rankin Score 0–3) were all patients with dominant side infarctions [59]. One explanation proposed for this trend is that left-sided infarctions causing total aphasia may have lowered the patient's initial Glasgow Coma Score compared to similar right-sided infarctions. Subsequently, this lowered GCS score led to an earlier surgical decision, which minimized the ischemic damage and improved patient outcome.

Walz et al., in a series of 18 MCA infarction patients, operated on 5 dominant-side infarctions. All 5 survived, 4 with mild aphasia and one with total aphasia. The authors also used multiple outcome testing scales (ALQI and Zung depression scale) and found the quality of life assessment for left- and right-sided patients to be similar [40]. Severe depression was more common with left-side infarction. The authors argue, however, that the same symptoms are seen to similar degree in conservatively treated MCA infarction patients, and should not be a reason for restricting DC surgery to the right side [40].

In another prospective study, DC was compared directly with mild hypothermia for the treatment of massive MCA infarction by creating two treatment groups based on the side of infarction. Non-dominant infarction victims were treated with DC and hypothermia, while dominant-side infarctions received mild hypothermia alone. DC was shown to have a mortality rate of 12% compared to 47% for those patients treated with mild hypothermia alone [55]. Their results indicated that DC had a lower mortality and morbidity, and the authors concluded that DC should be the treatment of choice even in dominant-side infarction [55]. In this and another study DC was associated with reduced

catecholamine requirements compared to the control groups [55, 60]. Other reports demonstrated that ICP can be controlled by DC alone, allowing hyperventilation, barbiturates, and osmotherapy to be stopped, reducing their attendant risk to the patient [4, 47].

Conclusions for Malignant MCA Infarction

DC for elevated ICP secondary to MCA infarction is by now a well-known procedure, however, the identification of the individual patient that will benefit from this intervention remains less clear. The randomized studies discussed above allow us to draw the conclusion that the young patient aged around 45 years is more likely to survive when undergoing DC early within 48 h, at the cost of a higher likelihood of moderate to moderately severe disability. The indication to perform DC is, and remains, individualized and should involve extensive discussion with the family.

Traumatic Brain Injury

Unlike malignant MCA infarction, there are no published randomized studies of DC for TBI in adults. One randomized control study from Australia focused on pediatric TBI cases, included 27 patients, but did not include opening of the dura during the surgery, see also Table 2. Currently, there are two ongoing multicenter randomized control trials of DC for the treatment of refractory ICP elevation associated with TBI: RESCUEicp in the UK (ISRCTN66202560) and DECRA in Australia (ISRCTN61037228) with goals of recruiting 600 and 210 patients, respectively [48]. These studies are underway, and their results could help define many of the prognostic factors that will influence the use of DC in TBI in the future.

Much of the debate surrounding DC's role in treatment of elevated ICP involves the selection of patients who will benefit from the procedure and the exclusion of those for whom DC may prolong suffering with no real hope of improvement. This debate is ongoing for all uses of DC, but the literature with regard to TBI patients is particularly illustrative of the issues, which require further investigation. The optimum time frame of the procedure, relevant prognostic factors, and the age of the candidate are central factors in the discussion of patient selection. Like malignant MCA infarction, there are several studies that report encouraging use of the procedure to reduce mortality and improve outcomes, but these results have not been consistent.

In 1999, Guerra et al. published a prospective study comprising a total of 57 patients with TBI with two study cohorts: 38 patients who underwent primary DC for brain

Table 2 Prospective and randomized controlled trials on DC for traumatic brain injury

Study	Studytype	*N*	Age/Sex	Inclusion criteria	Fu length	Measures	Outcome
Guerra et al. [9]	Prospective Cohort	57	Cutoff at less than 50 years (exceptions were made)	Diffuse edema on CT after TBI, subgroups with or without hematoma	12 months	GOS	58% with moderate deficits, 19% mortality
Taylor et al. [16]	Single Center RCT	27	1.8–15 years	Age 1.8–15 years, ICP > 20 mmHg in first 24 h or evidence of herniation	6 months	GOS and Health State Utility Index after 6 months	57% favorable outcome in DC group versus 14% in medical group (NS)
RESCUEicp (ISRCTN66202560)	Multicenter RCT	Target number 600	10–65 years	Severe TBI, ICP > 25 mmHg for 4–6 h despite optimal medical therapy (protocol)	6 months	Favorable (GOSE 1–3) and unfavorable (GOSE 4–8) outcome, SF-36 after 6 and 24 months	Ongoing
DECRA (ISRCTN61037228)	Multicenter RCT	Target number 210	15–60 years	Severe diffuse TBI, no mass lesions, ICP > 20 mmHg for > 15 minutes in first 72 h after injury	6 months	Favorable (GOSE 1–4) and unfavorable (GOSE 5–8) outcome after 6 months	Ongoing

edema without a focal lesion, and 17 patients for whom DC was a second procedure used after edema developed subsequent to surgical evacuation of a space-occupying hematoma [9]. No significant difference in outcome between the two groups was found, with good outcomes (GOS score 4–5) in 58% and 65%, respectively. Overall mortality was 19%. Age was not shown to be predictive of outcome, but patients older than 30 years of age were initially excluded from the study. During the study the exclusion age was raised to 50 years following initially promising results. The GCS score on post-trauma day one was a statistically significant predictor of outcome [9].

The presentation of intracranial hypertension (ICP > 20 mmHg) is seen in many neurological conditions and poses a challenging management problem. In the majority of cases this elevation can be controlled by conservative treatment which may include a combination of the following: 30-degree elevation of the head, mannitol therapy, hypertonic fluid infusion, brief hyperventilation, and mild hypothermia. When ICP levels remain persistently high, the treatment options may also include CSF drainage, barbiturate coma, and, when all else fails, decompressive craniectomy [61]. However, this set of treatments and especially their order of preference and timing lack an evidence-based support; there is no Class I data describing the effectiveness of any of the second-line treatments mentioned [62]. As a result of this gap in the literature, there has been a recent call for reevaluation of the treatment of intractable ICP and the role of surgical decompression in particular [8, 22, 30, 62]. Guerra et al., in one of the largest prospective studies of DC use to date, discussed the merits of DC when compared to hyperventilation, barbiturate coma, and hypothermia. The authors argued that the surgical approach was safer, with a lower mortality and fewer complications than any of the less invasive treatments [9]. The subsequent recommendation was that DC should be moved to first among the second tier treatments for intractable ICP [9].

In support of these findings, Munch et al. [63] found that an initial GCS < 8 or age > 50 years predicted poor outcome. Early decompression within 4.5 h of injury was more likely to result in good outcome. This retrospective study included 49 patients and the authors found the 33% mortality and 14% persistent vegetative state compared to 22% good outcomes; these results were similar to historical controls from the Traumatic Coma Data Bank and, accordingly, not encouraging [63].

DC for Older Patients with TBI

Older age has been associated with poor functional outcome after DC treatment for malignant MCA infarction and is recommended as an exclusionary criteria for DC in this pathology by many authors [37, 38, 53, 56, 63]. This can be explained by reduced brain plasticity, an increased rate of underlying disease, and a general disability to cope with the stress of surgery. However, the exact age cut-off remains unclear with some authors advocating 60 years [18, 54], and others predicting poor outcomes for those over 50 or 55 years of age [37, 38]. The debate over age in TBI patients is not as widely documented. A German group with multiple reports on the subject of DC for TBI began their investigations in the 1970's, excluding patients over 30 years of age, and gradually raised the age cutoff over the next 20 years to 50 years because their initial findings were so promising [9, 64]. The general consensus is that better outcomes and survival are possible with younger patients; age itself, regardless of the clinical status of the patient, has been called into question as a basis for the surgical decision for TBI patients.

In a recently published study of TBI patients, the small number of older patients represented in the literature and the lack of consistent statistical analysis were cited as undermining the use of age as a selection criteria [65]. The authors examined this problem with a retrospective analysis of 55 DC patients at their institution where age was not used as a pre-surgical exclusionary factor, citing "cultural reasons." 20 out of the total 55 patients treated with DC were over 65 years old, with the oldest being 94 years old. Although the group of patients above 65 years of age were shown to have significantly worse outcomes than those in the younger age groups ($P = 0.006$), the authors argue that age alone is not a good prognostic factor for the outcome after DC, but should be weighed with the presenting neurological state before a surgical decision is made [65]. Initial GCS was a statistically significant independent predictor of outcome ($P = 0.001$), with poor outcomes seen in 22 of 30 patients who presented with an initial GCS of 3–5. All patients over 65 years with GCS scores of 3–5 (11 patients) had poor outcomes, but 4 patients over 65 with GCS > 5 had favorable outcomes.

Timing of DC in TBI

"Early" surgical intervention is linked to better outcomes in many of the recently published case series of DC for TBI [1, 16, 22] and in animal models [41, 66]. However, a comprehensive definition of the term "early" is not in use, especially in light of the many pathologies underlying cerebral hypertension and the fact that hypertension may not present until days after the primary event.

Albanese et al. reported worse outcomes and a mortality of 52% in their early-DC group as opposed to 23% in the late group. The early group was defined as those operated on within 24 h of admission to the hospital and the late group were patients that required DC after ICP became medically unmanageable (ICP > 35 mm Hg despite maximum medical treatment) 2–6 days after admission [67]. In the early group 8 out of the 27 patients demonstrated signs of brainstem dysfunction and herniation shown by the lack of pupillary response in their first neurological exam and 7 of these 8 patients died [67]. The authors concluded that DC should be excluded for patients presenting with brainstem dysfunction upon their first neurological exam as the procedure holds little hope for improvement from this stage. Furthermore, the exclusion of patients with initial deterioration, would significantly improve the outcome of DC cases in general, as appropriate candidates with minimal pre-surgery brain damage are better identified [67].

One case report of a child with severe TBI demonstrated a remarkable recovery after delayed deterioration and DC 8 days post injury, underlining that close monitoring is key in patients with TBI and may reveal delayed deterioration early enough to make DC successful [68].

Other studies where DC was performed in the context of malignant MCA infarction agree with this approach, and argue that time alone is less predictive of a good outcome than the initial neurological state of the patient [21, 54].

Penetrating Blast Injury

Armonda and Ecklund retrospectively reviewed their experience with the incidence of vasospasm secondary to blast-related neurotrauma in the Iraq war and concluded that there was a statistically significant difference between GCS scores in the field for those who received hemicraniectomies versus those who received either craniotomy or no intervention. At discharge, however, the GCS score was not statistically different between the three populations, suggesting that, although patients who had hemicraniectomy were neurologically worse initially, they improved to a status that was statistically indistinguishable from the non-craniectomy group. The mean GCS at discharge for the craniectomy group was 11, compared to 14 for both the non-surgical and craniotomy groups [69].

Conclusions for Traumatic Brain Injury

A Cochrane review of the topic concluded that there is no evidence to support the routine use of DC in TBI for the treatment of medically refractory elevated ICP [70]. In contrast to this, and largely based on the results from nonrandomized trials, the American Brain Trauma Foundation guidelines mention bifrontal DC within 48 h of injury as a treatment option in patients with diffuse, medically refractory, post-traumatic cerebral edema, and resultant increased ICP [71]. With no clear consensus, Class I evidence from the ongoing randomized trials (Table 2) is very much needed. Nonetheless, DC should be recognized as a treatment option in individual cases with severe TBI.

Subarachnoid Hemorrhage

The use of DC to treat patients with elevated ICP secondary to SAH has received less attention than TBI or MCA infarction. Elevated ICP can be seen in SAH patients with or without a space occupying hematoma but there have been encouraging reports of DC use for both.

Smith and colleagues [72] demonstrated the use of DC as a prophylactic step in the setting of poor-grade patients with aneurysmal SAH carried out during the primary evacuation of large sylvian fissure hematomas and clipping of the aneurysm. The study included 8 patients prospectively selected for DC by the presence of a sylvian fissure hematoma of at least 25 cc clot volume (mean 121 cc) ipsilateral to an MCA aneurysm. The authors reported that the procedure added only 20–25 min to the originally planned evacuation and the ICP immediately fell below 20 mmHg for all 8 patients [72].

In a recent report the authors showed that even in cases of SAH without large intracranial hematoma, DC led to significant reduction of elevated medically unresponsive ICP [73]. 16 patients were treated with DC for refractory ICP after primary treatment for aneurysmal SAH by either surgical clipping or endovascular coiling. Mortality was 31% and 7 of the 11 survivors had modified Rankin scores of 0 to 3. DC within 48 h from admission led to good outcomes in 6 out of the 8 patients, significantly better than 1 out of 8 patients that underwent later DC [73].

Other authors have not been enthusiastic about DC in the context of aneurysmal SAH. In a case-controlled study of DC for SAH, d'Ambrosio et al. found a non-significant increase in short-term survival in 12 patients treated with DC, compared to a control group of 10 patients treated conservatively after the initial aneurysm treatment [74]. An overall poor quality of life, as measured on multiple functional and emotional tests was experienced by the surviving members of the DC group and the authors were skeptical of the benefits that DC could offer.

Buschmann and colleagues reported their experience with using DC as treatment for intracranial hypertension in 38 patients of a total of 193 patients with aneurysmal SAH after either brain swelling during the initial surgical clipping of the aneurysm (primary DC) or as secondary

treatment after development of subsequent intractable ICP. Overall, more than half of the patients had a good outcome and the subgroup of patients that developed ICP in the absence of mass occupying intracranial hemorrhage or infarction had a good outcome in 83%, followed by 60% in patients with hemorrhage but no infarction. In contrast only 17% of the patients with ICP due to infarctions had a good outcome, prompting the authors to call for a restrictive indication for DC in patients with SAH [75].

In conclusion, any recommendation for the role of DC in SAH is currently hampered by the lack of randomized data on the subject. DC for SAH should be considered as an option, however, more data on the short- and longterm outcome of these patients is needed to refine the indication.

Role of Improved Technology as an Adjunct to DC

Technological improvements in physiologic monitoring have only recently been used to refine the selection of patients for DC [4, 75]. ICP, cerebral perfusion pressure, and brain tissue oxygenation ($PtiO_2$) can now all be constantly monitored, whereas many of the studies discussed above did not utilize these tools. In the above mentioned series of patients with SAH by Buschman et al. the authors attributed the use of ICP monitors for the improved mortality rate compared to other similar studies which did not utilize this technology [75]. Some investigators also recommend that $PtiO_2$ monitoring of the cerebral tissue should be a critical factor in determining the timing of DC surgery [5, 75, 76]. Clinical signs of brainstem compression or uncal herniation will possibly not present until after there has already been a prolonged period of intracranial hypertension and low $PtiO_2$ [76]. DC at this stage with likely permanent ischemic damage will have an inherently worse chance of a good outcome. Strege et al. demonstrated in a retrospective analysis of 26 patients with either SAH or TBI that clinical deterioration was always preceded by pathological monitoring trends with decreasing $PtiO_2$ and increasing ICP. In patients with SAH a decrease of the $PtiO_2$ was a first warning sign that occurred earlier than ICP changes or neurological decline [76]. Cho et al. advocated the use of diffusion-weighted magnetic resonance imaging to identify malignant MCA infarction accurately in all cases compared with 33% accuracy using computed tomography [6].

Complications of DC

Direct surgical risks have been reported anecdotally but not emphasized as a contraindication for the procedure. Primary surgical complications are rare, but secondary infection or homeostatic reaction to the procedure have been more frequently reported. Hygroma was the most common complication found by Guerra and colleagues, seen in 15 out of 57 patients (26%), and also by Aarabi et al. who found half of their patients (25/50) developing hygroma [9, 77]. Kilincer et al. reported a contralateral subdural effusion after DC for an SAH patient and suggested that this could be a complication more specifically related to the concomitant SAH treatment [78]. Albanese et al. reported a high 22% incidence of meningitis which was not seen in other studies [67]. Pillai et al., reported a 37% incidence of delayed post-operative seizures after 6–9 months [21]. Kan et al. also reported seizure development in 20% of 6 pediatric DC patients as well as shunt-dependent hydrocephalus in 40% [20]. Bone resorption after cranioplasty has also been seen in a number of cases [19, 20, 77]. The risks associated with the surgical procedure of DC are generally considered to be low in relation to the morbidity and mortality of the disease process for which it is applied.

Conclusion

Throughout the medical literature there is much anecdotal evidence to suggest a larger role for DC as an effective and safe treatment for cerebral hypertension secondary to multiple pathologies. However, there is a lack of definitive evidence to support a clear recommendation for its use. Selection criteria with regard to timing, age, clinical symptoms, and relevant testing values are still under review. In our opinion, an individualized approach to the surgical intervention is mandatory, and should include both a consideration of the underlying pathological process and the ease of the surgeon with the particular variants of DC, but should in all instances include a durotomy to increase the potential space for the brain to swell. Currently strong data supports that young patients with malignant MCA infarction appear to benefit from DC, resulting in reduced mortality. This recommendation becomes less clear when SAH or TBI are the underlying pathologies. Further publication of data from the ongoing randomized trials for DC may ultimately identify the subgroup of patients that benefits the most and produce a consensus on the subject, since the majority of retrospective data is too heterogeneous to be readily compared, and the current debate surrounding this controversial procedure is yet to be resolved. Future studies should address in a systematic fashion variables such as lesion type, size, location, and surrounding edema. The necessary imaging modalities to evaluate both lesion and edema, whether or not quantitative analysis is necessary and numerical cutoffs for certain lesion types will also need to be defined. Current studies attempt to include patient gender

and age in the analysis, but severity of illness and preexisting conditions should also be considered. Larger studies with sufficient statistical power to allow for the analysis of meaningful subgroups will undoubtedly aid in the identification of the patients that will benefit from DC.

References

1. Polin RS, Shaffrey ME, Bogaev CA, et al. Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. Neurosurgery. 1997;41(1):84–92; discussion 92–4.
2. Fisher CM, Ojemann RG. Bilateral decompressive craniectomy for worsening coma in acute subarachnoid hemorrhage. Observations in support of the procedure. Surg Neurol. 1994;41(1):65–74.
3. Jaeger M, Soehle M, Meixensberger J. Effects of decompressive craniectomy on brain tissue oxygen in patients with intracranial hypertension. J Neurol Neurosurg Psychiatry. 2003;74(4):513–5.
4. Stiefel MF, Heuer GG, Smith MJ, et al. Cerebral oxygenation following decompressive hemicraniectomy for the treatment of refractory intracranial hypertension. J Neurosurg. 2004;101(2):241–7.
5. Reithmeier T, Lohr M, Pakos P, Ketter G, Ernestus RI. Relevance of ICP and ptiO(2) for indication and timing of decompressive craniectomy in patients with malignant brain edema. Acta Neurochir (Wien). 2005;147(9):947–52.
6. Cho DY, Chen TC, Lee HC. Ultra-early decompressive craniectomy for malignant middle cerebral artery infarction. Surg Neurol. 2003;60(3):227–32; discussion 232–3.
7. Schwab S, Steiner T, Aschoff A, et al. Early hemicraniectomy in patients with complete middle cerebral artery infarction. Stroke. 1998;29(9):1888–93.
8. Kontopoulos V, Foroglou N, Patsalas J, et al. Decompressive craniectomy for the management of patients with refractory hypertension: should it be reconsidered? Acta Neurochir (Wien). 2002;144(8):791–6.
9. Guerra WK, Gaab MR, Dietz H, Mueller JU, Piek J, Fritsch MJ. Surgical decompression for traumatic brain swelling: indications and results. J Neurosurg. 1999;90(2):187–96.
10. Arnott R, Finger S. Trepanation: history, discovery, theory. 2003: Taylor francis (UK).
11. Clower WT, Finger S. Discovering trepanation: the contribution of Paul Broca. Neurosurgery. 2001;49(6):1417–25; discussion 1425–6.
12. Rawlings CE 3rd, Rossitch E Jr. The history of trephination in Africa with a discussion of its current status and continuing practice. Surg Neurol. 1994;41(6):507–13.
13. Al-Rodhan NR, Fox JL. Al-Zahrawi and Arabian neurosurgery, 936–1013 AD. Surg Neurol. 1986;26(1):92–5.
14. Kocher T. editor. Die Therapie des Hirndruckes. In: Holder A, editor. Hirnerschutterung, Hirndruck und chirurgische Eingriffe bie Hirnkrankheiten. Vienna; 1901. p. 262–266.
15. Cushing H. The establishment of cerebral hernia as a decompressive measure for innaccessible brain tumors; with the description of intramuscular methods of making the bone defect in temporal and occipital regions. Surg Gynecol Obstet. 1905;1:297–314.
16. Taylor A, Butt W, Rosenfeld J, et al. A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Childs Nerv Syst. 2001;17(3):154–62.
17. Jourdan C, Convert J, Mottolese C, Bachour E, Gharbi S, Artru F. Evaluation of the clinical benefit of decompression hemicraniectomy in intracranial hypertension not controlled by medical treatment. Neurochirurgie. 1993;39(5):304–10.
18. Chen CC, Cho DY, Tsai SC. Outcome of and prognostic factors for decompressive hemicraniectomy in malignant middle cerebral artery infarction. J Clin Neurosci. 2007;14(4):317–21.
19. Grant GA, Jolley M, Ellenbogen RG, Roberts TS, Gruss JR, Loeser JD. Failure of autologous bone-assisted cranioplasty following decompressive craniectomy in children and adolescents. J Neurosurg. 2004;100(2 Suppl Pediatrics):163–8.
20. Kan P, Amini A, Hansen K, et al. Outcomes after decompressive craniectomy for severe traumatic brain injury in children. J Neurosurg. 2006;105(5 Suppl):337–42.
21. Pillai A, Menon SK, Kumar S, Rajeev K, Kumar A, Panikar D. Decompressive hemicraniectomy in malignant middle cerebral artery infarction: an analysis of long-term outcome and factors in patient selection. J Neurosurg. 2007;106(1):59–65.
22. Ruf B, Heckmann M, Schroth I, et al. Early decompressive craniectomy and duraplasty for refractory intracranial hypertension in children: results of a pilot study. Crit Care. 2003;7(6):R133–8.
23. Liang W, Xiaofeng Y, Weiguo L, et al. Cranioplasty of large cranial defect at an early stage after decompressive craniectomy performed for severe head trauma. J Craniofac Surg. 2007;18(3):526–32.
24. Carvi YNMN, Hollerhage HG. Early combined cranioplasty and programmable shunt in patients with skull bone defects and CSF-circulation disorders. Neurol Res. 2006;28(2):139–44.
25. Wada Y, Kubo T, Asano T, Senda N, Isono M, Kobayashi H. Fulminant subdural empyema treated with a wide decompressive craniectomy and continuous irrigation—case report. Neurol Med Chir (Tokyo). 2002;42(9):414–6.
26. Schwab S, Junger E, Spranger M, et al. Craniectomy: an aggressive treatment approach in severe encephalitis. Neurology. 1997;48(2):412–7.
27. Agrawal D, Hussain N. Decompressive craniectomy in cerebral toxoplasmosis. Eur J Clin Microbiol Infect Dis. 2005;24(11):772–3.
28. Ausman JI, Rogers C, Sharp HL. Decompressive craniectomy for the encephalopathy of Reye's syndrome. Surg Neurol. 1976;6(2):97–9.
29. Mitchell P, Gregson BA, Vindlacheruvu RR, Mendelow AD. Surgical options in ICH including decompressive craniectomy. J Neurol Sci. 2007;261(1–2):89–98.
30. Ziai WC, Port JD, Cowan JA, Garonzik IM, Bhardwaj A, Rigamonti D. Decompressive craniectomy for intractable cerebral edema: experience of a single center. J Neurosurg Anesthesiol. 2003;15(1):25–32.
31. Uhl E, Kreth FW, Elias B, et al. Outcome and prognostic factors of hemicraniectomy for space occupying cerebral infarction. J Neurol Neurosurg Psychiatry. 2004;75(2):270–4.
32. Carter BS, Ogilvy CS, Candia GJ, Rosas HD, Buonanno F. One-year outcome after decompressive surgery for massive nondominant hemispheric infarction. Neurosurgery. 1997;40(6):1168–75; discussion 1175–6.
33. Koh MS, Goh KY, Tung MY, Chan C. Is decompressive craniectomy for acute cerebral infarction of any benefit? Surg Neurol. 2000;53(3):225–30.
34. Kunze E, Meixensberger J, Janka M, Sorensen N, Roosen K. Decompressive craniectomy in patients with uncontrollable intracranial hypertension. Acta Neurochir Suppl. 1998;71:16–8.
35. Pranesh MB, Dinesh Nayak S, Mathew V, et al. Hemicraniectomy for large middle cerebral artery territory infarction: outcome in 19 patients. J Neurol Neurosurg Psychiatry. 2003;74(6):800–2.

36. Delashaw JB, Broaddus WC, Kassell NF, et al. Treatment of right hemispheric cerebral infarction by hemicraniectomy. Stroke. 1990;21(6):874–81.
37. Erban P, Woertgen C, Luerding R, Bogdahn U, Schlachetzki F, Horn M. Long-term outcome after hemicraniectomy for space occupying right hemispheric MCA infarction. Clin Neurol Neurosurg. 2006;108(4):384–7.
38. Holtkamp M, Buchheim K, Unterberg A, et al. Hemicraniectomy in elderly patients with space occupying media infarction: improved survival but poor functional outcome. J Neurol Neurosurg Psychiatry. 2001;70(2):226–8.
39. Woertgen C, Erban P, Rothoerl RD, Bein T, Horn M, Brawanski A. Quality of life after decompressive craniectomy in patients suffering from supratentorial brain ischemia. Acta Neurochir (Wien). 2004;146(7):691–5.
40. Walz B, Zimmermann C, Bottger S, Haberl RL. Prognosis of patients after hemicraniectomy in malignant middle cerebral artery infarction. J Neurol. 2002;249(9):1183–90.
41. Doerfler A, Schwab S, Hoffmann TT, Engelhorn T, Forsting M. Combination of decompressive craniectomy and mild hypothermia ameliorates infarction volume after permanent focal ischemia in rats. Stroke. 2001;32(11):2675–81.
42. Doerfler A, Forsting M, Reith W, et al. Decompressive craniectomy in a rat model of "malignant" cerebral hemispheric stroke: experimental support for an aggressive therapeutic approach. J Neurosurg. 1996;85(5):853–9.
43. Forsting M, Reith W, Schabitz WR, et al. Decompressive craniectomy for cerebral infarction. An experimental study in rats. Stroke. 1995;26(2):259–64.
44. Juttler E, Schwab S, Schmiedek P, et al. Decompressive surgery for the treatment of malignant infarction of the middle cerebral artery (DESTINY): a randomized, controlled trial. Stroke. 2007;38(9):2518–25.
45. Vahedi K, Vicaut E, Mateo J, et al. Sequential-design, multicenter, randomized, controlled trial of early decompressive craniectomy in malignant middle cerebral artery infarction (DECIMAL Trial). Stroke. 2007;38(9):2506–17.
46. Hofmeijer J, Amelink GJ, Algra A, et al. Hemicraniectomy after middle cerebral artery infarction with life-threatening Edema trial (HAMLET). Protocol for a randomised controlled trial of decompressive surgery in space-occupying hemispheric infarction. Trials. 2006;7:29.
47. Vahedi K, Hofmeijer J, Juettler E, et al. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol. 2007;6(3):215–22.
48. Hutchinson P, Timofeev I, Kirkpatrick P. Surgery for brain edema. Neurosurg Focus. 2007;22(5):E14.
49. Mori K, Nakao Y, Yamamoto T, Maeda M. Early external decompressive craniectomy with duroplasty improves functional recovery in patients with massive hemispheric embolic infarction: timing and indication of decompressive surgery for malignant cerebral infarction. Surg Neurol. 2004;62(5):420–9; discussion 429–30.
50. Oppenheim C, Samson Y, Manai R, et al. Prediction of malignant middle cerebral artery infarction by diffusion-weighted imaging. Stroke. 2000;31(9):2175–81.
51. Gupta R, Connolly ES, Mayer S, Elkind MS. Hemicraniectomy for massive middle cerebral artery territory infarction: a systematic review. Stroke. 2004;35(2):539–43.
52. Brown MM. Surgical decompression of patients with large middle cerebral artery infarcts is effective: not proven. Stroke. 2003;34(9):2305–6.
53. Leonhardt G, Wilhelm H, Doerfler A et al. Clinical outcome and neuropsychological deficits after right decompressive hemicraniectomy in MCA infarction. J Neurol. 2002;249(10):1433–40.
54. Foerch C, Lang JM, Krause J, et al. Functional impairment, disability, and quality of life outcome after decompressive hemicraniectomy in malignant middle cerebral artery infarction. J Neurosurg. 2004;101(2):248–54.
55. Georgiadis D, Schwarz S, Aschoff A, Schwab S. Hemicraniectomy and moderate hypothermia in patients with severe ischemic stroke. Stroke. 2002;33(6):1584–8.
56. Rabinstein AA, Mueller-Kronast N, Maramattom BV, et al. Factors predicting prognosis after decompressive hemicraniectomy for hemispheric infarction. Neurology. 2006;67(5):891–3.
57. Sandalcioglu IE, Schoch B, Rauhut F. Hemicraniectomy for large middle cerebral artery territory infarction: do these patients really benefit from this procedure? J Neurol Neurosurg Psychiatry. 2003;74(11):1600; author reply 1600.
58. Kastrau F, Wolter M, Huber W, Block F. Recovery from aphasia after hemicraniectomy for infarction of the speech-dominant hemisphere. Stroke. 2005;36(4):825–9.
59. Kilincer C, Asil T, Utku U, et al. Factors affecting the outcome of decompressive craniectomy for large hemispheric infarctions: a prospective cohort study. Acta Neurochir (Wien). 2005;147(6): 587–94; discussion 594.
60. Els T, Oehm E, Voigt S, Klisch J, Hetzel A, Kassubek J. Safety and therapeutical benefit of hemicraniectomy combined with mild hypothermia in comparison with hemicraniectomy alone in patients with malignant ischemic stroke. Cerebrovasc Dis. 2006;21(1–2):79–85.
61. Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care.Critical pathway for the treatment of established intracranial hypertension. J Neurotrauma. 2000;17:537–8.
62. Adamides AA, Winter CD, Lewis PM, Cooper DJ, Kossmann T, Rosenfeld JV. Current controversies in the management of patients with severe traumatic brain injury. ANZ J Surg. 2006;76(3):163–74.
63. Munch E, Horn P, Schurer L, Piepgras A, Paul T, Schmiedek P. Management of severe traumatic brain injury by decompressive craniectomy. Neurosurgery. 2000;47(2):315–22; discussion 322–3.
64. Gaab MR, Rittierodt M, Lorenz M, Heissler HE. Traumatic brain swelling and operative decompression: a prospective investigation. Acta Neurochir Suppl (Wien). 1990;51:326–8.
65. Pompucci A, De Bonis P, Pettorini B, Petrella G, Di Chirico A, Anile C. Decompressive craniectomy for traumatic brain injury: patient age and outcome. J Neurotrauma. 2007;24(7):1182–8.
66. Harrington ML, Bagley RS, Moore MP, Tyler JW. Effect of craniectomy, durotomy, and wound closure on intracranial pressure in healthy cats. Am J Vet Res. 1996;57(11):1659–61.
67. Albanese J, Leone M, Alliez JR, et al. Decompressive craniectomy for severe traumatic brain injury: Evaluation of the effects at 1 year. Crit Care Med. 2003;31(10):2535–8.
68. Reithmeier T, Speder B, Pakos P, et al. Delayed bilateral craniectomy for treatment of traumatic brain swelling in children: case report and review of the literature. Childs Nerv Syst. 2005;21(3):249–53; discussion 254.
69. Armonda RA, Bell RS, Vo AH, et al. Wartime traumatic cerebral vasospasm: recent review of combat casualties. Neurosurgery. 2006;59(6):1215–25; discussion 1225.
70. Sahuquillo J, Arikan F. Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. Cochrane Database Syst Rev. 2006;(1):CD003983.
71. Bullock R, Chesnut RM, Clifton G, et al. Guidelines for the management of severe head injury. Brain Trauma Foundation. Eur J Emerg Med. 1996;3(2):109–27.
72. Smith ER, Carter BS, Ogilvy CS. Proposed use of prophylactic decompressive craniectomy in poor-grade aneurysmal subarachnoid hemorrhage patients presenting with associated large sylvian hematomas. Neurosurgery. 2002;51(1):117–24; discussion 124.

73. Schirmer CM, Hoit DA, Malek AM. Decompressive hemicraniectomy for the treatment of intractable intracranial hypertension after aneurysmal subarachnoid hemorrhage. Stroke. 2007;38(3):987–92.
74. D'Ambrosio AL, Sughrue ME, Yorgason JG, et al. Decompressive hemicraniectomy for poor-grade aneurysmal subarachnoid hemorrhage patients with associated intracerebral hemorrhage: clinical outcome and quality of life assessment. Neurosurgery. 2005;56(1):12–9; dicussion 19–20.
75. Buschmann U, Yonekawa Y, Fortunati M, Cesnulis E, Keller E. Decompressive hemicraniectomy in patients with subarachnoid hemorrhage and intractable intracranial hypertension. Acta Neurochir (Wien). 2007;149(1):59–65.
76. Strege RJ, Lang EW, Stark AM, et al. Cerebral edema leading to decompressive craniectomy: an assessment of the preceding clinical and neuromonitoring trends. Neurol Res. 2003;25(5):510–5.
77. Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. J Neurosurg. 2006;104(4):469–79.
78. Kilincer C, Simsek O, Hamamcioglu MK, Hicdonmez T, Cobanoglu S. Contralateral subdural effusion after aneurysm surgery and decompressive craniectomy: case report and review of the literature. Clin Neurol Neurosurg. 2005;107(5):412–6.