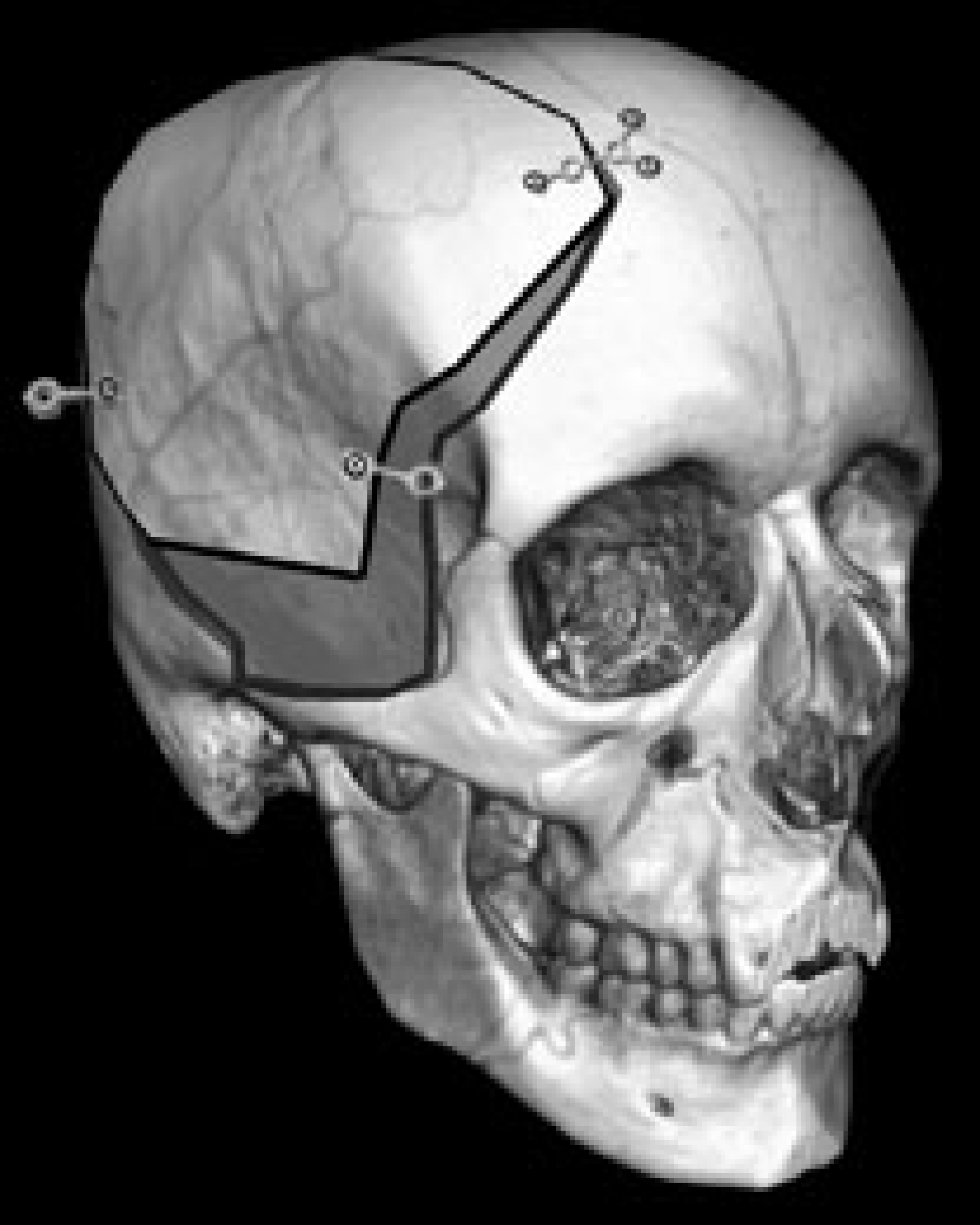

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Neurosurg Focus 26 (6):E1, 2009

Introduction: Decompressive craniectomy for trauma and cerebrovascular disease

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Elevated intracranial pressure is one of the most common causes of death and disability following severe traumatic brain injury and ischemic stroke. Unfortunately, there have been no new medical treatments for cerebral edema and elevated intracranial pressure in more than 80 years. Decompressive craniectomy may be an appropriate surgical option in the face of elevated intracranial pressure that is refractory to medical treatment. When performed correctly, this procedure can reduce intracranial pressure and prevent cerebral herniation and death. The last decade has seen a renewed interest in the use of decompressive craniectomy, but many questions remain regarding patient selection, timing of surgery, surgical technique, timing of cranioplasty, and complications.

This issue of *Neurosurgical Focus* serves to summarize some of the latest findings and presents new data in decompressive craniectomy. In the first article, Danish et al. report their use of meta-analysis to examine outcome and quality of life in patients after decompressive craniectomy and their finding of improved outcome compared to studies published 30 years ago. This is followed by 3 articles that describe the successful use of decompressive craniectomy in patients with cerebral ischemia secondary to cerebrovascular disease. These studies suggest a wider indication for this procedure beyond ischemic stroke and begin to tackle important questions regarding the use of this surgical technique in the elderly. From a technical perspective, Kenning et al., compare a "hinge" craniotomy to the standard decompressive craniectomy. Their paper highlights the need to study more technical aspects of this procedure. Stiver, importantly, reviews the complications of decompressive craniectomy in traumatic brain injury, and Aarabi et al. describe the interesting phenomena of subdural hygromas following this procedure. The timing of cranioplasty following successful recovery from decompressive craniectomy is controversial. Gooch et al. explore this issue in a single institution with interesting results. Finally, Cabraja and colleagues report their experience and long-term results using titanium implants for correcting large cranioplasty defects.

Together these articles provide timely and much needed information regarding decompressive craniectomy for trauma and cerebrovascular disease. I hope that you will find this issue of *Neurosurgical Focus* interesting and that it will serve to stimulate future studies.

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Quality of life after hemicraniectomy for traumatic brain injury in adults

A review of the literature

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Decompressive hemicraniectomy is well accepted for the surgical treatment of intractable intracranial hypertension in cases in which medical management fails. Although it is performed as a life-saving procedure when death is imminent from intracranial hypertension, little is known about the functional outcomes for these patients on long-term follow-up. In this study, the authors performed a systematic review of the literature to examine neurological outcome after hemicraniectomy. A literature search revealed 29 studies that reported outcomes using GOS scores. The GOS scores were transformed to utility values for quality of life using a conversion method based on decision analysis modeling. Based on the literature, 1422 cases were analyzed. The average 6-month-postoperative mortality rate was 28.2%. The mean QOL value among survivors was 0.592, which corresponds roughly to a GOS score of 4. Although more studies are needed for validation of long-term neurological outcome after hemicraniectomy, the assumption that most patients remain in a vegetative state after this intervention is clearly incorrect. *(DOI: 10.3171/2009.3.FOCUS945)*

Key Words • **traumatic brain injury** • **hemicraniectomy** • **outcome**

Head injury is a major cause of morbidity and mortality worldwide. Trauma itself is the leading cause of death in the first 4 decades of life, with traumatic brain injury being implicated in at least half the cases.[13] One of the fundamental pathophysiological processes after traumatic brain injury is the development and propagation of an escalating cycle of brain swelling and an increase in ICP. The goals of the clinical management of severe head injury consist of interrupting this cycle by controlling ICP and maintaining cerebral perfusion pressure and cerebral blood flow to avoid brain ischemia. This management strategy has been developed as a result of reported strong correlations between uncontrollable high ICP and high rates of morbidity and mortality. The relationship between high ICP and poor outcome has been demonstrated consistently in both single-center and multicenter studies, and the ability to bring elevated ICP under control has long been considered a requirement for improving outcome of patients with severe head injuries.[8,17,20,23] In an effort to reduce ICP, hemicraniectomy has evolved as a surgical option that has recently seen a "re-birth." Logically, it seems that opening a tight skull would reduce ICP, improve blood flow, and reduce swelling, leading to reduced morbidity and mortality. This concept has yet to be proved, however, with respect to improvement in clinical outcome.[30]

Those who are skeptical of the procedure raise several questions. Does the craniectomy quantitatively control raised ICP? Does brain herniating through the defect escalate the problems? What prognostic information can we give the families of those for whom the procedure is being proposed? Do the results justify the treatment? The last question is the focus of this review. Because of the lack of prospective, randomized trials, debate exists over the clinical outcome expected for patients undergoing the procedure. Especially in centers where craniectomy has not gained acceptance, there is a notion that patients whose head injuries are severe enough to mandate hemicraniectomy persist with severe disability or in a vegetative state, rendering the procedure futile and wasteful. Furthermore, because most studies use GOS scores to report outcomes, results from different studies cannot be combined and simply averaged. Finally, we must ask if the growing experience with hemicraniectomy over the years has led to better outcomes.

The present work is a review of the literature with respect to outcomes following hemicraniectomy. By con-

Abbreviations used in this paper: GOS = Glasgow Outcome Scale; ICP = intracranial pressure; QOL = quality of life.

TABLE 1: Total number of patients in each GOS category 6 months postoperatively

GOS Score	No. of Patients
5	383
4	283
3	232
2	110
1	414

verting GOS scores to utility values for QOL, we provide an average outcome for patients undergoing hemicraniectomy that is derived from the literature and is statistically sound.

Methods

A structured search of the English-language literature was performed to obtain the necessary data. A Medline search of all entries containing the subject heading "craniocerebral trauma" along with any combinations of the words "hemicraniectomy," decompression" (or "–ive"), or "craniectomy" was examined. The list was supplemented by reviewing the bibliographies of selected papers and using the "Related Articles" feature of PubMed. We decided to limit the analysis to series of at least 5 surgically treated cases, published between 1997 and December 2008 and containing GOS scores obtained at least 6 months after initial treatment. Series restricted to children (< 20 years old) were not included; when identified separately, pediatric cases were removed from series containing both adults and children.

We converted GOS scores to measures of utility or QOL using the formula published by Aoki et al.[3] The authors queried 135 healthcare professionals and elicited utilities for each of the GOS scores using a standard-gamble method. By convention,[10] the utility of a normal state of health (equivalent to a GOS score of 5) is 1, and that of death (GOS score, 1) is zero. The utilities or QOL values of intermediate GOS scores 0.63, 0.26, and 0.08 for GOS scores of 4, 3, and 2, respectively. These numbers represent values of QOL proportional to the respective GOS scores. For each study, we calculated mean utility and variance from the distribution of GOS scores. We also calculated the mean year of patient accrual.

To calculate pooled means for patient survival and the QOL values of survivors, we used a random effects meta-analytical model applied to prevalence data. We used the "metan" function of Stata (StataCorp LP). We used the same techniques to calculate pooled mean percentage of favorable outcomes (GOS scores 4 or 5). To check for temporal trends, we regressed mean QOL values of each case series against the mean year of patient accrual, using the "metareg" function. We considered a probability value of < 0.05 significant.

Results

Our Medline search yielded 269 English-language articles dealing either with hemicraniectomy or bifrontal decompressive craniectomy for severe traumatic brain injury. Of these, the majority of reports dealt with laboratory research, aspects of the surgery rather than general results. Other publications were reviews, editorials, or duplicated previous reports.

We found 29 articles reporting 1422 cases involving adult patients in whom decompressions were performed.[1,2,4–7,9,11,14–16,18,19,21,22,25,27–29,31–40] The mean years of patient accrual were between 1987 and 2005. Total numbers in each GOS category are shown in Table 1. Among survivors, the median GOS score was 4. For the pooled data, the average 6-month postoperative mortality rate was 28.2% (range 23.5–33%), and the mean QOL value among survivors was 0.592 (range 0.526–0.658). This QOL value is quite close to that reported for a GOS score of moderate disability.[3] The main outcomes are illustrated in Fig. 1. Overall, the pooled mean utility of decompressive craniectomy was 0.426 (95% CI 0.362–0.490). This outcome value is the mean of values for surviving patients and those who died. Favorable outcomes (GOS scores of 4 or 5) were achieved by 63.3% of survivors (95% CI 59.9–66.7%). For the entire series, including nonsurvivors, 46% of outcomes were favorable (95% CI 34.5–57.5%).

There did not appear to be any trend toward improvement of surgical outcomes between 1987 and 2005. Meta-regression showed that there were no significant trends over time with respect to either mortality or QOL among survivors ($p = 0.429$ and 0.891, respectively). This is also evident in Fig. 1, in which case series are presented from the above-described group of articles, in order of publication date.

Discussion

We reviewed more than 1400 reported cases of decompressive craniectomy performed in adults for the treatment of severe traumatic brain injury and uncontrollable intracranial hypertension. The pooled mean 6-month-postoperative mortality rate for the series was approximately 28%, and the pooled mean QOL value among the survivors was almost 60% of the value of a normal state of health. This score is very close to the QOL associated with moderate disability score (GOS score of 4).[3] Favorable outcomes (GOS scores of 4 or 5) were reported for 46% of the series when all patients were considered, and 63% of survivors. Our data also indicate that neither mortality rate nor QOL have improved since the late 1980s, when the procedure regained popularity and saw renewed application to the treatment of ICP.

The findings from this study challenge the occasional charge that hemicraniectomy shifts the outcome from death to a persistent vegetative state and severe disability. Recent outcome studies of protocols including decompressive craniectomy have also not supported this concept.[21,26] The mean QOL value of survivors after hemicraniectomy when all series were combined in our study places the average survivor in the moderate disability category. Based on our findings, it is reasonable to counsel families that many patients undergoing hemicraniectomy for uncontrollable ICP will be able to function independently.

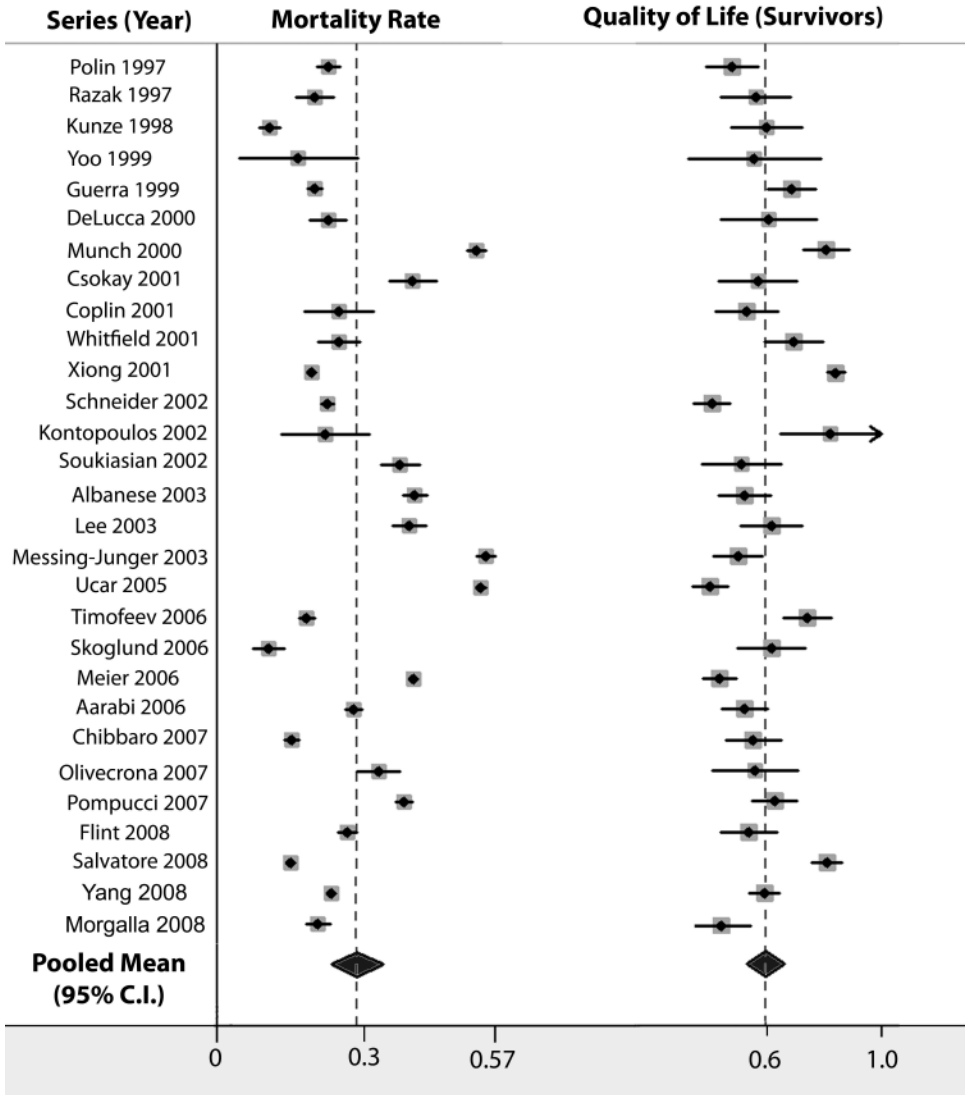

FIG. 1. Summary of mortality rates and QOL after hemicraniectomy. Twin Forest plots illustrating outcomes of decompressive craniectomy for severe traumatic brain injury in adults. Mortality rate is plotted on the *left*, QOL on the *right*. The first author and year of publication of each quoted report are listed to the *far left*. The *center of each square* represents the mean of that study, the *horizontal bar* its 95% CI. The *size of each box* represents the weight of the study, and is inversely proportional to its variance. The pooled estimates are represented by the *diamonds* below. For each *diamond*, the center represents the pooled mean, the horizontal extent equals its 95% CI.

There are a number of limitations in our study. The populations from which our data are drawn are heterogeneous in terms of patient characteristics, indications for surgery, and decompression techniques used. This heterogeneity limits the precision of our estimates. We cannot adjust for a number of clinical factors known to be associated with outcome, such as age,[1,11,15,21,27,28] timing of surgery,[15,24,27] admission GCS score, preoperative pupillary examination, and other features.[12,21] We chose QOL as an outcome measure over GOS scores because of its almost universal use in quantitative studies of medical outcomes[10] and because of its conformance to mathematical calculations, such as determining mean values and confidence intervals. The GOS is an interval scale of neurological function; calculating mean GOS scores and other mathematical manipulations are meaningless. Converting a score to a parametric value of QOL has, at most, limited validity but at least allows valid mathematical and statistical calculations. Some might also question the conversion process used by Aoki and associates,[3] in which preferences were those of health care professionals rather than the community. In addition the complication profile queried by Aoki et al. was drawn from aneurysm surgery rather than from hemicraniectomy. Some of the many complications of hemicraniectomy are transient and not reflected in the GOS scores at 6 months.[39]

Nevertheless, the data may prove useful in providing prognostic guidance when counseling family members about a decompression procedure. We cannot conclude that decompressive craniectomy is superior to medical

care alone, because we did not compare outcomes. In addition, a recent Cochrane Review suggested that evidence is lacking.[30] Any definitive conclusions about treatment comparisons must await the completion of the ongoing DECRAN and Rescue ICP trials. In the interim, the findings from this study should lend confidence to practitioners who use the procedure in the treatment of intractable ICP.

Conclusions

Decompressive hemicraniectomy is a well-known surgical option for uncontrollable intracranial hypertension. Although it lacks evidence from randomized trials, pooled outcomes from the current literature show that patients who undergo hemicraniectomy for severe traumatic brain injury can achieve a reasonably good QOL. In the future, the results of ongoing randomized trials should reveal the patient population in which this intervention can make the largest impact.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Assessment of outcome following decompressive craniectomy for malignant middle cerebral artery infarction in patients older than 60 years of age

A review

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Object. Decompressive surgery can be life saving after malignant cerebral infarction. However, severe residual disability occurs in a significant number of surviving patients. Most discussion about the benefits of surgery is based on studies performed in patients who are ≤ 60 years of age. Less is known about the benefits of the procedure in the elderly population. The authors undertook a review of the literature on decompressive craniectomy for malignant cerebral infarction and compared the mortality and outcome data published in patients older and younger than 60 years of age. The authors discuss their analysis, with specific reference to the limitations of the studies analyzed, the outcome measures used, and the special considerations required when discussing stroke recovery in the elderly.

Methods. Studies on decompressive craniectomy for malignant middle cerebral artery infarction reported in the English literature were analyzed. A cutoff point for age of > 60 or ≤ 60 years was set, and the study population was segregated. No studies specifically analyzed patients > 60 years old. A total of 19 studies was identified, 10 of which included patients who were > 60 years of age. A comparison between the 2 age groups was made within the 10 studies and also among all the patients in the 19 studies. Mortality rates and outcome scores were assessed for each study, and a Barthel Index (BI) score of < 60 or a modified Rankin Scale (mRS) score of > 3 was considered to represent a poor outcome. Rates were compared using the Fisher exact test, and p values < 0.05 were considered statistically significant.

Results. Nineteen studies were found, which included 273 patients undergoing decompressive craniectomy for malignant cerebral infarcts. Ten of these studies included 73 patients (26.7%) who were > 60 years of age. The mean follow-up times ranged from 5.75 to 12.3 months in the > 60-years group and 4.2 to 28 months in the ≤ 60-years group. The mortality rate was significantly higher, at 51.3% in the > 60-years group (37 of 72 patients) compared with 20.8% (41 of 197 patients) in the ≤ 60-years group ($p < 0.0001$). Similarly, patients who survived in the > 60-years group had significantly higher rates of poor outcomes, at 81.8% (27 of 33), compared with 33.1% (47 of 142) in the ≤ 60-year-old group ($p < 0.0001$). The BI was the most commonly used primary outcome measure (15 out of 19 studies), followed by the mRS score, which was used in 4 studies.

Conclusions. The mortality rate and functional outcome, as measured by the BI and mRS, were significantly worse in patients > 60 years of age following decompressive craniectomy for malignant infarction. Age is an important factor to consider in patient selection for surgery. However, cautious interpretation of the results is required because the outcome scores that were used only measure physical disability, whereas other factors, including psychosocial, financial, and caregiver burden, should be considered in addition to age alone. *(DOI: 10.3171/2009.3.FOCUS0958)*

Key Words • decompressive craniectomy • infarction • stroke • age • elderly population

Among cases of supratentorial infarction, 10–15% involve the entire MCA territory.[1,14,22,45] Despite optimal medical therapy, the mortality rate approaches 80%. This type of extensive stroke has been termed malignant MCA infarction and is accompanied by severe brain edema, leading to raised ICP and subsequent brain herniation.[11] A vicious cycle develops as the resulting ischemic insult leads to further edema, and thus to increases in ICP and reduction of regional cerebral blood flow.[32] Medical treatment has not been shown to be effective.[16]

Several experimental studies[7,9] have shown the benefit of decompressive craniectomy in rats after MCA occlusion. In these studies, craniectomy resulted in im-

Abbreviations used in this paper: BI = Barthel Index; ICP = intracranial pressure; MCA = middle cerebral artery; mRS = modified Rankin Scale.

TABLE 1: Literature review of decompressive craniectomy for malignant MCA infarction in patients > 60 years of age*

Authors & Year	No. of Patients	No. of Lesions		Patients w/ Early Op (<24 hrs)	Mean Time to FU (mos)	No. w/ Good Outcome/ No. Who Survived	No. of Deaths/ No. in Study	Main Outcome Score
		Rt MCA	Lt MCA					
Carter et al., 1997	3	3	0	1	12	1/3	0/3	BI
Walz et al., 2002	4	3	1	1	NA	NA	4/4	BI
Leonhardt et al., 2002	9	9	0	1/9	12	0/6	2/8†	BI
Holtkamp et al., 2001	7	5	2	3/7	7	0/3	4/7	BI
Delashaw et al., 1990	3	3	0	1/3	15	1/3	0/3	BI
Rieke et al., 1995	6	6	0	3/6	11	2/3	3/6	BI
Koh et al., 2000	2	1	1	NA	NA	NA	0/2	BI
Harscher et al., 2006	14	13	1	5/14	12.3	1/3	11/14	BI
Kilincer et al., 2005	13	8	5	4/13	6	0/4	9/13	mRS
Yao et al., 2005	12	9	3	0	5.75	1/8	4/12	BI

* FU = follow-up; NA = not available.
† One patient dropped out of this study.

proved outcome, and, if treated early enough, reduced infarction size. In 1956, Scarcella[36] first described the surgical removal of a large bone flap (frontotemporal-parietal) with duraplasty ipsilateral to the infarction to reduce ICP and prevent brain herniation. Since then, there have been several case series and reports indicating improved survival and functional outcome. Recently, 3 randomized controlled trials were performed to study the effects of decompressive craniectomies after malignant stroke.[15,20,40] The pooled analysis of these trials confirmed the suggestions from nonrandomized studies that decompressive craniectomy undertaken within 48 hours of stroke onset reduces mortality rates and increases the number of patients with a favorable functional outcome after malignant hemispheric infarction.[39]

However, fundamental questions remain unanswered. The importance of preoperative neurological status, hemispheric dominance, timing of surgery after onset of stroke, and patient age are a few factors that may affect outcome. The pooled results from the 3 recent controlled trials included only patients ≤ 60 years of age. In their multivariate regression analysis studies, Gupta et al.[10] and Chen et al.[3] showed that age was the only variable found to be significant in the outcome of decompressive hemicraniectomy after malignant MCA infarction. Hence, despite the positive outcome demonstrated by these controlled trials, it is controversial how applicable the results are to patients > 60 years of age. In addition, outcome measures may produce different results depending on patient age, and these results must be interpreted cautiously. We review the studies in which patients > 60 years of age who underwent decompressive craniectomies for hemispheric infarction were analyzed.

Methods

A PubMed search for studies reported in the English language after 1970 was performed using the key words "craniectomy" or "hemicraniectomy" in several combinations with the key words "stroke," "ischemia," and "malignant middle cerebral artery infarction." We included the studies in which information was available for individual patients. Mortality rates and outcome scores were assessed for each study group. Because the recent controlled trials all included patients < 60 years old, we specified the cutoff point for age as > 60 or ≤ 60 years. We considered a BI score of < 60 or an mRS score of > 3 to represent poor outcome, as suggested elsewhere.[37] GraphPad Instat version 3.05 (GraphPad Software, Inc.) was used for statistical analysis. Rates were compared using the Fisher exact test, and p values < 0.05 were considered statistically significant.

Results

Nineteen studies were identified in the English literature, and 10 included patients > 60 years of age. There were 273 patients for whom individual information on age, side of infarction, outcome scores, and survival were available. Seventy-three patients (26.7%) were > 60 years of age. The right/left hemisphere infarction ratio was 60:13 in the > 60-year-old group, and it was 148:51 in the ≤ 60-year-old group. Early surgery (< 24 hours postadmission) was performed in 19 (26.8%) of 71 patients in the > 60-year-old group and in 60 (36.6%) of 164 in the ≤ 60-year-old group. The mean follow-up times ranged from 5.75 to 12.3 months in the > 60-year-old group and 4.2 to 28 months in the ≤ 60-year-old group (Tables 1 and 2).

The mortality rates in the > 60-year-old group were significantly higher, at 37 (51.3%) of 72, compared with 41 (20.8%) of 197 in the ≤ 60-year-old group ($p < 0.0001$, Fig. 1). Similarly, among patients who survived, poor outcomes were significantly higher in the > 60-year-old

TABLE 2: Literature review of decompressive craniectomy for malignant MCA infarction in patients ≤ 60 years of age

Authors & Year	No. of Patients	No. of Lesions		Patients w/ Early Op (<24 hrs)	Mean Time to FU (mos)	No. w/ Good Outcome/No. Who Survived	No. of Deaths/ No. in Study	Main Outcome Score
		Rt MCA	Lt MCA					
Carter et al., 1997	11	11	0	4/11	12	7/8	3/11	BI
Walz et al., 2002	14	7	7	8/14	14	6/12	2/14	BI
Leonhardt et al., 2002	17	17	0	10/17	12	11/12	4/16*	BI
Holtkamp et al., 2001	5	4	1	1/5	4.2	1/5	0/5	BI
Delashaw et al., 1990	6	6	0	2/6	12.5	3/5	1/6	BI
Rieke et al., 1995	26	20	6	5/26	13	14/18	8/26	BI
Koh et al., 2000	5	3	2	NA	NA	NA	1/5	BI
Rengachary et al., 1981	3	3	0	0/3	21	1/3	0/3	BI
Kalia & Yonas, 1993	4	2	2	1/4	17	3/4	0/4	BI
Young et al., 1982	1	1	0	0/1	9	0	0/1	BI
Ivamoto et al., 1974	1	1	0	0/1	7	1	0/1	BI
Kondziolka & Fazl, 1988	4	3	1	2/4	20	4/4	0/4	BI
Cheung et al., 2005	3	3	0	2/3	16	2/3	0/3	BI
Harscher et al., 2006	16	9	7	3/16	28	6/13	3/16	BI
Kilincer et al., 2005	19	7	12	2/19	6	7/12	7/19	mRS
Yao et al., 2005	13	8	5	0	6.6	11/12	1/13	BI
Vahedi et al., 2007 (DECIMAL)	20	NA	NA	20/20	12	10/15	5/20	mRS
Juttler et al., 2007 (DESTINY)	17	8	9	NA	10	7/14	3/17	mRS
Hofmeijer et al., 2006 (HAMLET)	14	NA	NA	NA	10	1/11	3/14	mRS

* One patient dropped out of this study.

group, at 27 (81.8%) of 33 compared with 47 (33.1%) of 142 in the ≤ 60-year-old group ($p < 0.0001$, Fig. 2).

We also analyzed patient data derived only from the 10 studies that included both age groups to minimize bias and confounding factors when analyzing all 19 studies. Again, the mortality rate was significantly higher in the > 60-year-old group (51.3%) compared with 23% (30 of 130) in the ≤ 60-year-old group ($p < 0.0001$, Fig. 3). Similarly, patients who survived in the > 60-year-old group had significantly higher rates of poor outcomes—81.8% compared with 32% (31 of 97) in the ≤ 60 group ($p < 0.0172$, Fig. 4).

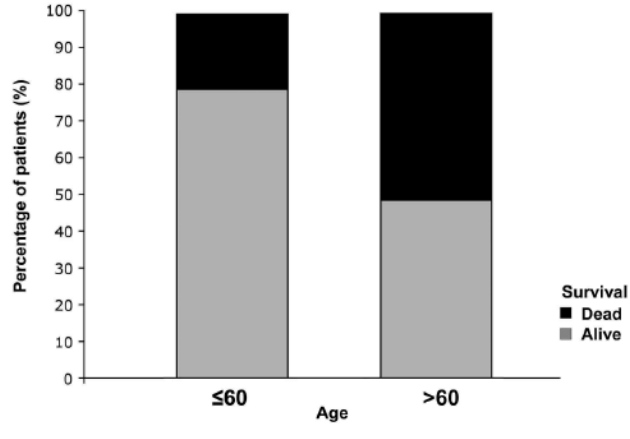

Fig. 1. Bar graph showing the mortality rates in patients after decompressive craniectomy for malignant MCA infarction in all studies.

Although a higher proportion (45%) of patients > 60 years old who had surgery within 24 hours of stroke onset showed good outcome, compared with 14% in those who underwent surgery after 24 hours, the results were not statistically significant.

In 15 of 19 studies the BI was primarily used to measure outcome, whereas in 4 studies the mRS was used.

Discussion

Decompressive surgery with duraplasty has been performed in patients with malignant brain edema caused by infarction or trauma. The aim of the procedure is to decrease the mass effect of the edema, preventing brain herniation, secondary injury to the brain, and death. The benefits of early surgery after malignant infarction have been shown in experimental studies in rats, and encouraging results both in mortality rates and functional outcome have been shown by several authors worldwide. However, the importance of age in functional outcome, especially in the older population, has been less well established.

Recently, 3 European randomized controlled trials (the French DECIMAL trial, the German DESTINY tri-

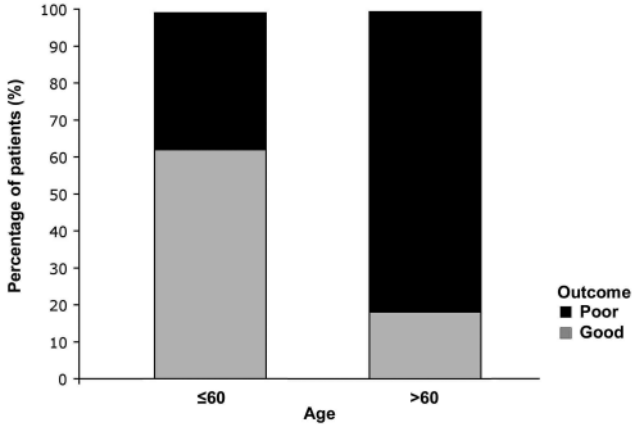

Fig. 2. Bar graph showing the outcomes of patients after decompressive craniectomy for malignant MCA infarction in all studies.

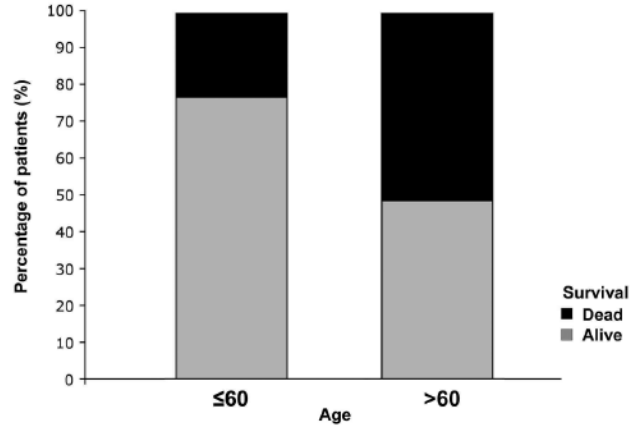

Fig. 3. Bar graph showing the mortality rates in patients after decompressive craniectomy for malignant MCA infarction in studies with both age groups included.

al, and the Dutch HAMLET trial) published their pooled analysis of 93 patients, all < 61 years of age. This showed that hemicraniectomy reduced the mortality rate by 50% at 1 year when compared with best medical treatment. This confirms the results from previous nonrandomized studies and case reports analyzed in this review. The question arises whether similar results would be expected in an older patient population, because the incidence of stroke increases with age, and large hemispheric strokes also occur in patients > 60 years of age.

In their retrospective analysis, Yao et al.[46] specifically compared results between younger (< 60 years old) and elderly patients (≥ 60 years old) after decompressive craniectomy, and showed a mortality rate of 7.7 and 33.3%, respectively. The 10 studies we analyzed, which included patients > 60 years of age, showed that the mortality rate was also significantly higher, at 51.3%, compared with the younger population. The latter had a mortality rate of 20.8%, which was similar to the rate of 22% reported in the pooled analysis. Decisions for surgery based on survival benefits are therefore less clear-cut in patients in the group older than 60 years, even though the mortality rate is 80% without surgery.[1,11]

Despite the comparable survival and mortality rates after decompressive surgery in patients > 60 years of age, information on the risk of death is relatively straightforward to convey to relatives. However, discussion of surgery after a large stroke is not usually based on survivability, but more importantly on functional outcome. One of the main difficulties for families and physicians is to ascertain whether the patient will have acceptable residual disability and a good quality of life. This difficulty is compounded by the fact that family and patient perceptions of quality of life vary. Moreover, the inability to predict with certainty the degree of residual disability and the absence of the patient's participation in the decision-making process add to the difficulty.

In the pooled European randomized controlled trials analysis of patients < 61 years of age it was demonstrated that more patients in the surgery group (43 vs 21%) survived with moderate disability (mRS score of ≤ 3: able to walk without assistance), but there were also more patients (31 vs 2%) who survived with moderately severe disability (mRS Score 4). The number of patients with residual severe disability (mRS Score 5) was not increased following decompressive craniectomy and remained ~ 5%. Our analysis of all 19 studies discussed here showed poor outcome following surgery in 33.1% of patients < 60 years of age, which is similar to the 35% shown in the pooled controlled studies. However, poor outcome was significantly higher in patients in the > 60 group, at 81.8%. Despite the statistical limitations of this analysis, these results are discouraging, making it difficult to recommend decompressive surgery for malignant stroke in most patients > 60 years of age.

In previous reports age has been suggested as a key factor in determining who will benefit from decompressive surgery after malignant cerebral infarct,[10,38] and some investigators have reported poorer outcomes in elderly patients.[46] Increasing age has also been shown to be an important factor in poor recovery after stroke.[26] This may be due to the diminished capacity for neuroplasticity in elderly patients.[29] In addition, preexisting disability and severe comorbid conditions are also important factors that increase poor outcome, and both are more prominent in elderly patients.

However, a cutoff age of 60 years is clearly arbitrary, and surgical decisions must be made on an individual basis. Moreover, there is much controversy over whether age per se is as important a predictor of outcome as other factors, such as admission functional status, cognitive status, and social situation, including the availability of resources or employment. Indeed, many would advocate that advanced age should not be regarded as a limiting factor to rehabilitation after stroke.[6,26,28]

We have shown here the differences in mortality rates and outcomes between the > 60-year-old and ≤ 60-year-old patients following decompressive surgery for malignant stroke. We have also alluded to the fact that outcome is a more complex measure than mortality rate. "Good outcome" in these studies was measured primarily by the BI and the mRS, indicating a level of disability or "activity" levels as defined by the International Classification of Functioning.[44] However, what disability means and how

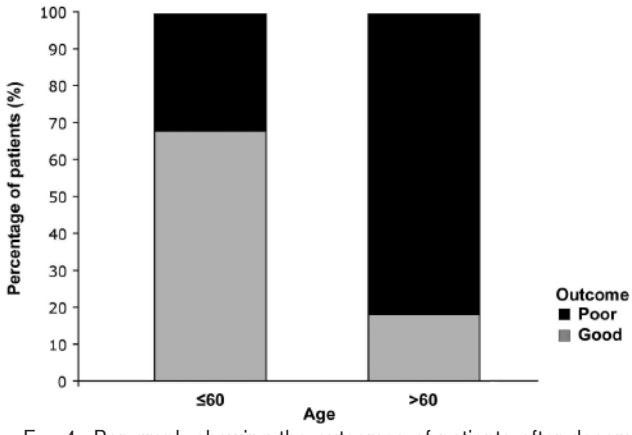

FIG. 4. Bar graph showing the outcomes of patients after decompressive craniectomy for malignant MCA infarction in studies with both age groups included.

it is ultimately measured are debatable. The BI and mRS, although widely used as basic standard measures, have gross limitations when determining activity level. The BI correlates highly with motor ability and physical disability,[8,41,42] leaving cognitive and psychological consequences common to stroke as predictors of a poor outcome potentially neglected.[13,19,31] Furthermore, the BI and mRS have been shown to be inconsistent indicators of independence due to the lack of consensus between measures,[37,38] which calls into question the clinical and research utility of the BI and mRS to measure perceptions of disability, independence, and participation.

Predicting outcomes for older adults after stroke has revealed complex and contextual factors that could not be assessed using the BI or mRS; these included depression, cognitive functioning, and demographic and economic factors.[30] The ability to perform activities of daily living declines with age,[34] putting potentially elderly caregivers and stroke survivors at greater risk because of the task of caring and of strain on the caregiver. Therefore, the presence of strong family support and the impact on the caregiver may be at least as important as age per se when considering decompressive surgery following malignant stroke.

There are several limitations in our review. The data are mostly obtained from previous case series and retrospective studies, and thus the standard meta-analysis techniques could not be applied. There is heterogeneity in the population analyzed, which limits the scope of this study. In addition, the patients did not have a uniform approach in terms of follow-up and management. We also need to take into account that there is publication bias favoring good outcomes in the literature. Despite all these limitations, this review has highlighted the difference in mortality rates and outcome scores between the > 60-year-old and ≤ 60-year-old patients following decompressive surgery for malignant stroke, and cautious interpretation of these results is required.

Any definitive conclusion will require a randomized controlled study to assess the benefits of decompressive surgery specifically in elderly patients. The study would similarly compare surgery against best medical treatment, with mortality rates and clinical outcome as primary end points. The outcome measure should use a stroke- and age-sensitive assessment tool such as the Stroke Impact Scale and the Assessment of Motor and Process Skill. These tools are more rigorous than both the BI and the mRS score. The Assessment of Motor Process Skills requires formal training, and both measures are less susceptible to scorer variability. Moreover, they can be used to measure the severity of disability and participation restriction after stroke in a way pertinent to the patient's age and lifestyle.

Elderly patients are also more likely to have multiple factors that can contribute to outcome following a stroke. Analysis of occupation and economic factors, cognitive level, level of activities of daily living, and family support status are a few factors that would be of particular interest to assess in this population in any future studies.

Conclusions

Decompressive craniectomy is a life-saving procedure after malignant brain infarction, but the selection of patients for decompressive surgery remains controversial. Age may be a key factor in helping guide surgical decisions because elderly patients have a significantly higher mortality rate and do worse after decompressive surgery. However, factors other than age should be considered, and the treatment decision should be individualized. The BI and mRS systems are crude measures of physical domains of disabilities only, and patients with severe neurological deficits with decreased quality of life cannot be evaluated thoroughly by them. The psychosocial and financial burden of care, which may not be related solely to physical disability, should also be taken into account when determining outcome in the elderly.

Disclosure

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Decompressive craniectomy in subarachnoid hemorrhage

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Object. The aim of this study was to analyze decompressive craniectomy (DC) in the setting of subarachnoid hemorrhage (SAH) with bleeding, infarction, or brain swelling as the underlying pathology in a large cohort of consecutive patients.

Methods. Decompressive craniectomy was performed in 79 of 939 patients with SAH. Patients were stratified according to the indication for DC: 1) primary brain swelling without or 2) with additional intracerebral hematoma, 3) secondary brain swelling without rebleeding or infarcts, and 4) secondary brain swelling with infarcts or 5) with rebleeding. Outcome was assessed according to the modified Rankin Scale (mRS) at 6 months (mRS Score 0–3 favorable vs 4–6 unfavorable).

Results. Overall, 61 (77.2%) of 79 patients who did and 292 (34%) of the 860 patients who did not undergo DC had a poor clinical grade on admission (World Federation of Neurosurgical Societies Grade IV–V, $p < 0.0001$). A favorable outcome was attained in 21 (26.6%) of 79 patients who had undergone DC. In a comparison of favorable outcomes in patients with primary (28.0%) or secondary DC (25.5%), no difference could be found ($p = 0.8$). Subgroup analysis with respect to the underlying indication for DC (brain swelling vs bleeding vs infarction) revealed no difference in the rate of favorable outcomes. On multivariate analysis, acute hydrocephalus ($p = 0.009$) and clinical signs of herniation ($p = 0.02$) were significantly associated with an unfavorable outcome.

Conclusions. Based on the data in this study the authors concluded that primary as well as secondary craniectomy might be warranted, regardless of the underlying etiology (hemorrhage, infarction, or brain swelling) and admission clinical grade of the patient. The time from the onset of intractable intracranial pressure to DC seems to be crucial for a favorable outcome, even when a DC is performed late in the disease course after SAH. *(DOI: 10.3171/2009.3.FOCUS0954)*

Key Words • decompressive craniectomy • intracranial aneurysm • subarachnoid hemorrhage • intracerebral hemorrhage • brain swelling

Decompressive craniectomy lowers elevated ICP in patients with an intractable increase in pressure following brain trauma or cerebral infarction and improves outcome in patients with large territorial infarctions of the MCA.[4,14,16]

In patients with aneurysmal SAH, brain swelling can occur very early after the ictus ("primary") and later in the course of the disease as the result of complications associated with SAH ("secondary"; for example, cerebral infarction or bleeding). Regardless of its origin, brain swelling is known to worsen outcomes following SAH.[3,7] Its medical treatment is highly significant and often effective, although it can also be associated with severe side effects. Because of the unknown functional recovery, especially in poor-clinical-grade patients with SAH and associated elevated ICP, the indication for aggressive surgical treatment options such as DC is controversial.[5,6,13] Recent literature dealing with DC has included patients with ICH and SAH only[6] or had excluded patients treated with endovascular coiling.[5] There are sparse data concerning the etiology of the mass effect that leads to DC—such as a space-occupying infarct, an ICH, or brain swelling without bleeding or infarction—which might per se significantly affect outcome.[8,13]

The purpose of the present analysis was to update another recent study in providing comprehensive clinical material[9] and to analyze outcomes in a larger cohort of consecutive patients with primary and secondary DC, stratified according to the different underlying pathologies—that is, bleeding, infarction, or brain swelling—to find predictors that may guide treatment.

Parts of the clinical material have been published in a predecessor study.[9]

Methods

We studied 939 consecutive patients with aneurysmal SAH confirmed on CT or lumbar puncture and angiography between June 1999 and June 2008. During this

Abbreviations used in this paper: ACoA = anterior communicating artery; DC = decompressive craniectomy; DHC = decompressive hemicraniectomy; DW = diffusion weighted; ICA = internal carotid artery; ICH = intracerebral hematoma; ICP = intracranial pressure; MCA = middle cerebral artery; mRS = modified Rankin Scale; PW = perfusion weighted; SAH = subarachnoid hemorrhage; WFNS = World Federation of Neurosurgical Societies.

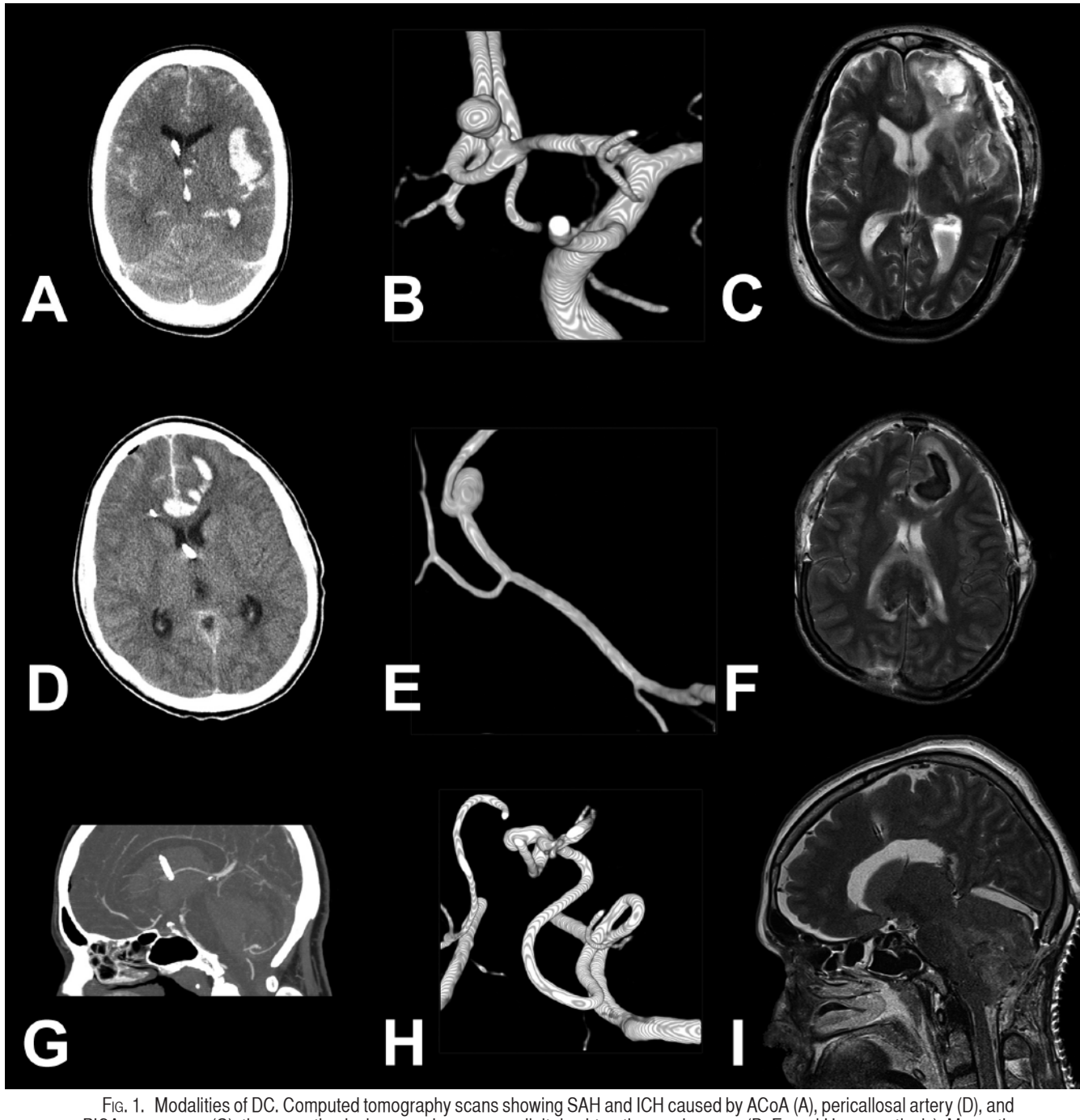

Fig. 1. Modalities of DC. Computed tomography scans showing SAH and ICH caused by ACoA (A), pericallosal artery (D), and PICA aneurysms (G); the causative lesions can be seen on digital subtraction angiograms (B, E, and H, respectively). Magnetic resonance images obtained after DHC (C), bifrontal craniectomy (F), and suboccipital craniectomy (I) for intractably elevated ICP, demonstrating the extent of injury caused by SAH and the decompression achieved by DC.

period, 79 patients underwent DC as a treatment for elevated ICP. Information, including patient characteristics on admission and during the treatment course, treatment modality, size and location of the ruptured aneurysm, radiological features, presence and size of the ICH, presence of infarction, rebleeding, and the specific indication for DC, was prospectively entered into an SPSS database (SPSS, version 15, SAS Institute, Inc.). The treatment decision (coiling or clipping) was based on an interdisciplinary approach in each case. We followed an early surgery strategy whenever possible (within 24 hours of the onset of SAH) in patients of all clinical grades except in those who were hemodynamically unstable or moribund. Acute hydrocephalus was treated with external CSF diversion. Osmotherapy and mild hyperventilation were used for the medical treatment of elevated ICP. Our emphasis was to establish high normal euvolemia, and in cases of symptomatic cerebral vasospasm hemodynamic therapy was

TABLE 1: Subgroups of 79 patients treated with DC

DC Groups				
Primary		Secondary		
1	2	3	4	5
brain swelling w/o ICH	brain swelling w/ ICH	brain swelling w/o infarction or rebleeding (SAH)	brain swelling w/ signs of infarction	brain swelling w/ signs of rebleeding(SAH)

instituted.[12] Patients with impeding cerebral ischemia and proximal vasospasm were treated with balloon angioplasty[2] guided by PW and DW MR imaging.

Surgical Technique

In patients with persistent and medically refractory ICP values > 20 mm Hg, DC was performed by removing a large bone flap at the involved site. During DHC, a large frontotemporoparietooccipital flap extending from the supraorbital rim to just behind the lambdoid suture and from the temporal base to close (1.5 cm) to the midline was removed. The temporal bone was removed osteoclastically down to the base of the middle cerebral fossa. The size of the craniectomy was at least 15 cm in diameter. During bifrontal craniectomy, a large bone flap extending from the roots of the zygoma bilaterally and from ~ 1 cm parallel and superior to the orbital rim to just about the coronal suture was removed in 1 piece. Craniectomy of the posterior cranial fossa was performed via a midline approach, with the removal of a bone flap extending from the lower margin of the transverse sinus to the foramen magnum. The lateral extensions were close (~ 1 cm) to the expected margins of the sigmoid sinus. During each craniectomy the dura mater was opened widely, and the opening was extended to the sinus and bone margins in a stellate fashion (Fig. 1).

Patients were stratified into 5 groups (Table 1). In the primary DC groups the bone flap was not reinserted but instead was enlarged after surgical aneurysm obliteration in patients with signs of massive brain swelling without (Group 1) or with (Group 2) an ICH. Additionally, external ventricular drains or ICP (Spiegelberg) or brain tissue PO_2 probes (Raumedic) were inserted in selected patients. Persons with elevated ICP (> 20 mm Hg) despite medical therapy underwent secondary DC. All of those undergoing secondary DC had already been treated for the ruptured aneurysm. Indications for secondary DC were intractable elevated ICP (> 20 mm Hg) without radiological signs of rebleeding (SAH) or infarction (Group 3) and elevated ICP with signs of infarction (Group 4) or rebleeding (repeat SAH in patients with coagulopathy, Group 5).

In all patients in Group 5, rebleeding consisted of SAH, not ICH. Rebleeding was confirmed on CT as a larger SAH volume than before. All of these patients had coagulopathy—for example, warfarin before aneurysm rupture due to cardiac arrhythmia, artificial cardiac valve, or deep vein thrombosis.

Apart from close neurological monitoring, routine surveillance included daily transcranial Doppler measurements of red blood cell flow velocities and, in selected cases, multimodal monitoring of parenchymal brain tissue PO_2, regional cerebral blood flow (thermodilution microprobe), interstitial metabolites (microdialysis), and PW/DW MR imaging. Computed tomography was routinely performed 1) 24–48 hours after aneurysm clip application or coil obliteration to assess procedural complications, 2) on Day 14–21 to diagnose vasospastic infarctions and assess the necessity of a ventriculoperitoneal shunt, and 3) at various time points whenever neurological deteriorations occurred. Magnetic resonance imaging, including PW/DW imaging,[2] was performed on Day 7, and cerebral angiography between Days 7 and 10.

Sufficient fluid was administered to maintain a high normal euvolemic status. All patients received nimodipine from the day of admission. In the case of hyponatremia, fludrocortisone was added to the therapy. Desmopressin was used to control excessive diuresis. In cases of symptomatic vasospasm hypervolemia was instituted, and hypertension was induced with catecholamines.[12] When hypertensive-hypervolemic-hemodilution therapy failed to improve delayed ischemic neurological deficit symptoms, patients with focal vasospasm were selected to undergo angioplasty.[2] Outcome was assessed according to the mRS 6 months after treatment. Patients were divided into good-grade (WFNS Grade I–III) versus poor-grade (WFNS Grade IV–V) groups on admission and stratified into favorable (mRS Score 0–3) versus unfavorable (mRS Score 4–6) outcomes.

Statistical analysis was performed for all patients who underwent DC. An unpaired t-test was used for parametric statistics. Categorical variables were analyzed in contingency tables using the Fisher exact test. Results with a $p < 0.05$ were considered statistically significant. In a second step, multivariate analysis was performed to find independent predictors of outcome at 6 months after discharge by using a binary logistic regression analysis and to find the confounding effects between potentially independent predictors. Variables with significant probability values on univariate analysis were considered potentially independent variables on multivariate analysis. A backward stepwise method was used to construct multivariate logistic regression models with the inclusion criterion of $p < 0.05$. All calculations were made with standard commercial software (SPSS Institute, Inc.).

Results

Patient Characteristics

Patient characteristics, including age, sex, clinical

TABLE 2: Summary of patient characteristics on admission*

Characteristic	No. (%)			
	w/o DC	w/ DC	OR (95% CI)	p Value†
no. of patients	860	79		
mean age in yrs	54.0 ± 14.2	51.0 ± 12.3		0.07
mean WFNS grade	II	V		<0.0001
cases w/ poor WFNS grade (IV–V)	292 (34)	61 (77.2)	6.6 (3.8–11.4)	<0.0001
female sex	510 (59)	59 (74.7)	2.0 (1.2–3.4)	0.007
cases w/ ICH <50 cm^3	84 (9.8)	15 (19)	2.1 (1.2–4.0)	0.02
cases w/ ICH >50 cm^3	46 (5.3)	22 (27.8)	6.8 (3.8–12.1)	<0.0001
Fisher grade	3	3		0.9
mean time from ictus to aneurysm obliteration in hrs	83.3 ± 11	51.2 ± 18		<0.0001
cases w/ rebleeding before treatment	66 (7.7)	14 (17.7)	2.6 (1.4–4.9)	0.005
cases w/ acute hydrocephalus	474 (55.1)	43 (54.4)		0.9
cases of clipping	278 (32.3)	53 (67.1)	4.2 (2.6–7.0)	<0.0001
mean aneurysm size in mm	6 ± 4.2	11 ± 7	5 (3.9–6.0)	<0.0001
cases of shunt placement after 6 mos	121 (14.1)	20 (25.3)	2.1 (1.2–3.6)	0.01
mean mRS score	2	5		<0.0001
cases w/ unfavorable outcome (mRS 4–6)	238 (27.7)	59 (74.7)	7.7 (4.5–13.1)	<0.0001

* Values are presented as the means ± SDs, unless indicated otherwise.
† Fisher exact test.

status on admission, and angiographic, and CT imaging findings are shown in Table 2. Of 939 patients, 353 (37.6%) presented with a poor clinical grade. Sixty-one of the poor-grade patients (17.3%) underwent DC. Among patients admitted in a good clinical grade, DC was performed in 18 (3.1%) of 586 patients ($p < 0.0001$, OR = 6.6, 95% CI 3.8–11.4).

Age did not differ between patients with (51 ± 12.3 years) or without (54.0 ± 14.2 years) DC ($p = 0.07$). The rate of women was higher in the group treated with DC compared with the group not treated with DC (74.7 vs 59%, $p = 0.007$, OR = 2.0, 95% CI 1.2–3.4). Microsurgical clipping was performed in 67.1% of patients treated with DC compared with 32.3% patients not treated with DC ($p < 0.0001$, OR = 4.2, 95% CI 2.6–7.0). Four hundred seventy-four (55.1%) of 860 patients who did not undergo DC and 43 (54.4%) of 79 patients who did undergo DC presented with acute hydrocephalus ($p = 0.9$).

Aneurysm Size and Site

In patients treated with DC the most frequent site for a ruptured aneurysm was the MCA (55.0%) and the ICA (18.3%). Anterior communicating artery aneurysms caused SAH in 15.0% of patients who underwent DC (Table 3). In the group of patients that did not undergo DC, aneurysms were more frequently located at the ACoA (33.4%, $p = 0.003$, OR = 2.8, 95% CI 1.3–5.9) and less frequently at the MCA (17.0%, $p < 0.0001$, OR = 5.9, 95% CI 3.4–10.3). There was no difference between groups the DC groups in terms of bleeding at the ICA (23.2 vs 18.3%). Patients treated with DC had significantly larger aneurysms (mean: 11 vs 6 mm, $p < 0.0001$, OR = 5, 95% CI 3.9–6.0; Table 2).

Decompressive Craniectomy Group

Of the 79 patients treated with DC, 61 (77.2%) presented in a poor grade. Decompressive hemicraniectomy was performed as a primary procedure in 32 patients (40.5%) and as a secondary procedure in 47 (59.5%). According to the indication for DC, in only 3 patients (3.8%) with clear signs of brain swelling during aneurysm obliteration, the bone flap was not reinserted, but the craniotomy site was enlarged instead (Group 1: primary DC without ICH). Twenty-nine patients (36.7%) underwent primary DC because of brain swelling together with an ICH at presentation (Group 2). Sixteen patients (20.3%) were treated with secondary DC due to intractable ICP without infarcts (Group 3). In 23 patients (29.1%) secondary DC was performed due to space-occupying infarcts (Group 4), and 8 patients (10.1%) underwent secondary DC after rebleeding caused by coagulopathy (SAH, Group 5).

Decompressive Craniectomy Modality

Depending on the site of the space-occupying lesion, a right hemispheric DHC was performed in 36 cases and a left hemispheric DHC in another 36 cases. A bifrontal craniectomy was performed in 6 cases and a DC of the posterior fossa in 1 case. In comparing the rate of favorable outcomes in patients undergoing DHC (25.0%) and bifrontal DC (16.7%), no difference could be found ($p = 1.0$).

Appropriate Time for DC

In all patients who underwent a primary DC, decompression was performed on the day of the SAH. The mean time to surgery was 4.5 ± 5.7 hours. In patients who underwent secondary DC, decompression was performed

TABLE 3: Aneurysm site in 79 cases*

Aneurysm Site	w/o DC (% cases)	w/ DC (% cases)	OR (95% CI)	p Value†
ant circulation				
ACoA	33.4	15.0	2.8 (1.3–5.9)	0.003
ACA	4.4	6.7		0.3
ICA	23.2	18.3		0.4
MCA	17.0	55.0	5.9 (3.4–10.3)	<0.0001
pst circulation	22.0	5.0		0.2

* ACA = anterior cerebral artery.
† Fisher exact test.

3.3 ± 3.1 days (79.2 ± 73 hours) after SAH. The mean time to aneurysm obliteration was 26 ± 25.8 hours after ictus in patients who underwent secondary DC. Hence, the time to aneurysm treatment was significantly shorter in the group of patients that underwent primary DC compared with the group that underwent secondary DC ($p < 0.001$).

Treatment Outcome

Treatment outcome was stratified according to the 5 DC groups (Table 4). Overall, 21 (26.6%) of 79 patients achieved a favorable outcome. No patients in Group 1 and 9 (31.0%) of 29 patients in Group 2 attained favorable outcomes. Six (37.5%) of 16 patients in Group 3, 5 (21.7%) of 23 in Group 4, and 1 (12.5%) of 8 in Group 5 had favorable outcomes (Fig. 2). In comparing the rate of favorable outcomes in patients treated with primary (28.0%) and secondary DC (25.5%), no difference could be found ($p = 0.8$).

There was no difference in favorable outcomes between patients in Groups 2 and 5 (brain swelling with ICH or repeat SAH) and those in Group 4 (brain swelling with infarction; $p = 0.8$). Furthermore, no difference in favorable outcomes could be found in a comparison of patients in Groups 1 and 3 (brain swelling without additional lesion on CT), and those in Groups 2, 4, and 5 (brain swelling with ICH, repeat SAH, or infarcts; $p = 0.6$).

The mortality rates 6 months after treatment were 40% in patients who had undergone primary DC and 21.7% in those who had undergone secondary DC ($p = 0.3$).

In poor-grade patients with a favorable outcome, the time to aneurysm obliteration was 3.9 ± 1.8 hours compared with 15.2 ± 13.9 hours in poor-grade patients with an unfavorable outcome ($p = 0.01$). Eight of the 15 poor-grade patients with a favorable outcome had an additional ICH and underwent primary DC (53.3%, Group 2), 3 patients underwent secondary DC without signs of infarctions (20%, Group 3), and 4 patients underwent DC due to infarction (26.7%, Group 4).

Overall, 42 of the 79 patients had clinical signs of tentorial herniation (mydriasis). Eight (19%) of the 42 patients with and 11 (18.9%) of 37 patients ($p = 0.3$) without signs of cerebral herniation attained a favorable outcome.

Cranioplasty was performed in surviving patients with DHC and bifrontal craniectomy. The mean time from ictus to cranioplasty was 90 ± 47 days.

Multivariate Analysis

Using a backward stepwise method in a binary logistic regression model, the multivariate relationships were analyzed in patients with SAH and DC for the variable outcome at 6 months after discharge. Of the variables that influenced outcome 6 months after discharge on univariate analysis in patients with SAH who had undergone DC, acute hydrocephalus ($p = 0.009$, OR 5.8, 95% CI 1.5–21.9) remained, and clinical signs of cerebral herniation ($p =$

TABLE 4: Summary of patient characteristics according to DC group

Parameter	DC Group (No. [%])				
	1	2	3	4	5
no. of patients	3	29	16	23	8
mean age in yrs	43 ± 6	52.0 ± 14	48.5 ± 9	51.0 ± 11.5	52.0 ± 11.6
mean WFNS grade	4	5	4	4	5
cases w/ poor WFNS grade (IV–V)	1	28 (96.5)	10 (62.5)	16 (69.6)	6
female sex	2	22 (75.9)	13 (81.3)	18 (78.3)	6
cases w/ acute hydrocephalus	1	8 (27.6)	11 (68.8)	16 (69.6)	7
cases w/ clipping	2	22 (75.9)	7 (43.8)	16 (69.6)	6
cases w/ angiographic vasospasm (≥60%)	0	8	5	23	8
cases w/ shunt placement at 6 mos after treatment	2	3 (10.3)	8 (50)	7 (30.4)	0
mean mRS score at 6 mos after treatment	6	5	4	5	6
cases w/ unfavorable outcome (mRS 4–6)	3	20 (69)	10 (62.5)	18 (78.3)	7
cases w/ unilat dilated pupil	1	12 (41.4)	3 (18.8)	15 (65.2)	1
cases w/ bilat dilated pupils	0	4 (13.8)	2 (12.5)	2 (8.7)	2
cases w/o dilated pupils	2	13 (44.8)	11 (68.7)	6 (26.1)	5

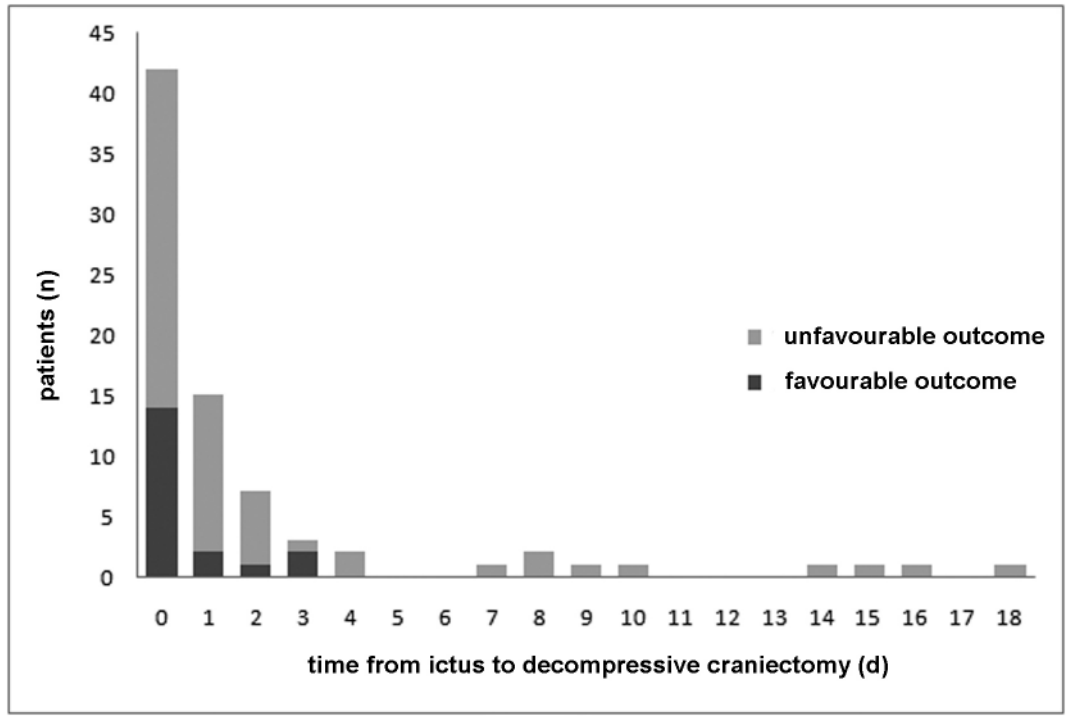

Fig. 2. Bar graph showing the timing and outcome of DHC. d = day; n = number.

0.02, OR 3.1, 95% CI 1.1–8.1) became significant in the multivariate regression model (Nagelkerke $R^2 = 0.33$).

Discussion

The role of DC in patients with refractory elevated ICP following traumatic brain injury or cerebral infarction is beneficial.[1,4,11,14,17] Decompressive craniectomy leads to a 2-step reduction in elevated ICP after bone flap removal and dural opening as well as improvement in tissue perfusion and oxygenation.[10] However, authors of only a few studies have dealt with DC in patients with aneurysmal SAH. To the best of our knowledge, we have presented the largest series of patients with aneurysmal SAH who underwent DC. To improve treatment decision making, we placed special emphasis on the clinical settings that indicated primary or secondary DC; therefore, we further stratified our patients according to their underlying pathology, for example, bleeding, infarction, or brain swelling.

In this study of 939 consecutive patients the data accumulation was prospective, whereas the analysis was retrospective. Outcome in the 79 patients who had undergone DC was favorable in 21 (26.6%), despite the presence of space-occupying lesions (ICH or infarct), signs of clinical herniation (mydriasis in 53.2% of the cases), and a population of 77.2% with poor-grade SAH (WFNS IV–V) on admission. There was no difference in the rate of favorable outcomes between patients who underwent primary (29%) and secondary DC (26.1%; $p = 0.8$). Our results indicated that DC may be warranted regardless of whether the patient suffers from bleeding, infarction, or brain swelling.

According to the data of Schirmer et al.,[13] 5 (31%) of 16 patients were treated with primary and 11 (69%) with secondary DC. A favorable outcome was attained in 4 (80%) of the 5 patients who had undergone primary and 3 (27%) of the 11 patients who had undergone secondary DC. Four of the 5 patients with primary DC had an additional ICH on admission. The patients with ICH in that study had a smaller hematoma volume (< 50 cm^3) in contrast to a rate of 27.8% of patients with hematomas larger than 50 cm^3 in our current series, which may have contributed to a better outcome. In addition, Schirmer et al. reported no patients with a Fisher Grade 3 SAH on admission, which translated into a lower risk of vasospasm and consecutive infarctions. Indeed, these authors described no patients who underwent DC for space-occupying infarcts. Our population consisted of 69.6% patients with Fisher Grade 3 SAHs, and 29.1% underwent DC because of infarcts; nevertheless, 21.7% of them had a favorable outcome. In other words, we showed that a relevant number of even those patients with high-grade SAHs as well as additional large, space-occupying ICHs and those with Fisher Grade 3 bleeding and infarcts can have favorable outcomes.

Buschmann et al.[5] have treated 38 patients with SAH by using DC for intractable ICP and have found 52.6% favorable outcomes after 12 months. According to their published data, 76.2% of the patients treated with primary DC had Fisher Grade 4 SAHs, but no information about ICH size was given. Sixty percent of the patients who had undergone secondary DC for a postoperative epidural or subdural hematoma attained a favorable outcome. This result might explain the good outcome in their study group given the extraaxial nature of the lesion and the possibil-

ity of a quick ICP reduction following hematoma evacuation. No DC was performed as a result of epi- or subdural hematomas in the current study. Moreover, 84.3% of patients had a poor clinical grade on admission, and 83.3% of those without and 16.7% of those with infarctions attained a favorable outcome. Among the subgroup of patients without infarctions, 80% had Fisher Grade 4 SAHs. Again, no data on ICHs or intraventricular hematomas size were given, which would be important for a comparison of outcomes.

In our series the subgroup that underwent secondary DC for space-occupying infarctions attained a comparable or slightly better rate of 21.7% favorable outcomes. Careful decision making is needed in this group of patients with per se poor prognoses due to large infarctions. D'Ambrosio et al.[6] have analyzed 12 patients with ICHs on admission and found that 33.3% had favorable outcomes, a rate somewhat higher than our 31.0%. Note, however, that D'Ambrosio and colleagues' series included only 8.3% large ICHs (> 50 cm^3) versus 27.8% in our study population.

Altogether, it is not surprising that the outcome in severely ill patients undergoing DC in smaller series varies, especially because of different patient characteristics on admission (for example, WFNS grade, Fisher grade, and accompanying ICH). It is intriguing that outcome in the current study is comparable among the DC groups, regardless of the different underlying pathologies leading to DC (ICH, brain swelling, or infarction). An overall favorable outcome of 25.3% in the subset of critically ill patients with life-threatening conditions, in which a general conservative approach is usually accepted because of an expected poor prognosis, in our opinion is encouraging and warrants aggressive therapy in the future.

We believe that DC is useful in lowering intractably elevated ICP regardless of the aneurysm location (Fig. 1). The predominance of patients with MCA aneurysms undergoing DC in our series might have been attributable to the higher rate of large ICHs caused by MCA aneurysms compared with aneurysms of other locations as previously described.[8]

On multivariate analysis, we found that early hydrocephalus ($p = 0.009$) and clinical signs of herniation—for example, mydriasis ($p = 0.02$)—were associated with an unfavorable outcome. The association between cerebral herniation and poor outcome is not surprising and has been addressed by other authors.[5,13] Smith et al.[15] have even proposed the use of a prophylactic DC for the treatment of patients with poor-grade SAH and an additional ICH. Although the optimal time point for DC must still be defined, an early craniectomy seems to be beneficial, leading to favorable outcomes in 25.3% of cases. This result is corroborated by the significantly poorer outcome in a heterogeneous group of patients with signs of cerebral herniation due to different underlying pathologies (brain swelling, infarct, and ICH). The result of univariate analyses showing no effect of mydriasis on outcome was somewhat surprising. However, the multivariate analysis, with its better control for confounding factors, showed that the presence of mydriasis indeed was 1 factor that significantly influenced outcome. The correlation between acute hydrocephalus and poor outcome has been addressed elsewhere.[18] Mortality rates as well as cerebral infarction rates are higher in patients with acute hydrocephalus, as compared with the rates in patients without, as described by van Gijn et al.[18] Therefore, it is not surprising that on multivariate analyses in the current series, acute hydrocephalus was 1 of the factors determining outcome.

Study Limitations

The present study has several limitations. The data analysis was performed retrospectively. Patients were not randomized for the treatment or control groups. Even though the patient series in the current study represents the largest thus far to suffer from aneurysmal SAH and to be treated with DC, our statistical analysis was still handicapped.

Altogether, we provide data showing 1) that the underlying pathology does not seem to limit outcome after DC, and 2) that the time from the initial SAH to DC is not relevant. Instead, the time from the onset of elevated ICP—whether due to bleeding, infarction, or brain swelling—seems to be crucial for patient outcome. This conclusion is corroborated by the finding that cerebral herniation significantly affects outcome.

Conclusions

According to our data, DC is a valid option in the treatment of patients with aneurysmal SAH and intractable ICP. Decompressive craniectomy can be indicated early or late in the course after SAH if performed immediately after the onset of intractable ICP.

A favorable outcome can be attained in one-quarter of these patients. Even in the subgroup of patients with infarcts or large hematomas for which the prognosis seems limited, DC might be warranted. Nevertheless, careful decision making is needed for each patient, especially when signs of cerebral herniation have persisted for a long time. It is important for clinical decision making that DC may be indicated regardless of the underlying pathophysiology—bleeding, infarction, or brain swelling—and the admission grade of the patients.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or findings specified in this paper.

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Decompressive surgery in malignant dural sinus thrombosis: report of 3 cases and review of the literature

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Cerebral venous and dural sinus thrombosis (CVDST) is a rare cause of stroke in young and middle-aged adults. When the clinical course is complicated by uncontrollable intracranial hypertension and brainstem compression due to edema or cerebral hemorrhage, the prognosis is poor. The authors evaluated the therapeutic role of surgical decompression in patients with clinical signs of impending herniation. Cerebral venous and dural sinus thrombosis complicated by impending brain herniation a very rare, life-threatening but potentially treatable clinical condition.

Three patients with pupillary signs of transtentorial herniation due to brain edema and hemorrhage caused by CVDST (superior sagittal sinus in 1 patient and transverse and sigmoid sinus in 2 patients) were treated surgically. The intervention consisted of clot removal, infarcted tissue resection, and frontotemporoparietooccipital craniectomy with duraplasty. According to the Glasgow Outcome Scale, 2 patients were classified as having good recovery and 1, moderate disability. The results of neuropsychological assessment were normal in 2 patients and demonstrated a partial neuropsychological deficit (neglect) in the other.

Surgery may be indicated in selected patients with CVDST whose condition is deteriorating because of intractable intracranial hypertension and impending brain herniation. *(DOI: 10.3171/2009.3.FOCUS0910)*

Key Words • cerebral venous thrombosis • craniectomy • stroke • intracranial pressure • dural sinus • hemorrhagic infarct

Cerebral vein and dural sinus thrombosis predominates in young and middle-aged adults. Despite improvements in diagnosis and treatment, more than 15% of patients remain permanently disabled or die.[7,9] Recently, the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) has shown that the most common mechanism of acute death is transtentorial herniation due to hemorrhagic lesions or diffuse brain edema.[1,2] In these circumstances, decompressive surgery (DS) may be the treatment to choose as it promptly creates space for swelling cerebral tissue and may also improve perfusion of collateral leptomeningeal vessels.

The ISCVT suggested the importance of investigating DS with randomized trials.[7] However, the rarity of the pathology (5 cases per million),[1] and the fact that only a minority of patients with CVDST suffer from impending brain herniation necessitating DS, means that such a study is unlikely to succeed, even with long-term international collaborative efforts.

We report the cases of 3 patients with pupillary signs of impending brain herniation secondary to CVDST who underwent DS on an emergency basis, and we discuss the rationale and the clinical results of the procedure in relation to the physiopathology of CVDST.

Abbreviations used in this paper: CVDST = cerebral venous and dural sinus thrombosis; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; ISCVT = International Study on Cerebral Vein and Dural Sinus Thrombosis.

Methods

Source of Data

We reviewed a prospectively collected database of the patients admitted for cerebrovascular pathological conditions to the Neurosurgical Intensive Care Unit of the Ospedali Riuniti of Bergamo from January 2006 to November 2008. A patient was eligible if a diagnosis of CVDST was confirmed by angiography, CT venography, MR imaging, or autopsy. Exclusion criteria were: age < 18 years, cerebral lesions other than those related to CVDST, and posttraumatic, postinfective, or iatrogenic CVDSTs.

Causal Treatment (Anticoagulation Therapy)

Causal treatment was started at the diagnosis of CVDST and consisted of low-molecular-weight heparin (Nadroparin) in a dose of approximately 180 anti–factor Xa units/kg per 24 hours or dose-adjusted intravenous heparin with an at least doubled activated partial thromboplastin time. Cerebral hemorrhage was not a contraindication against causal treatment. Patients in critical condition who could become potential surgical candidates were preferentially treated with unfractionated heparin because it has the advantage of being promptly antago-

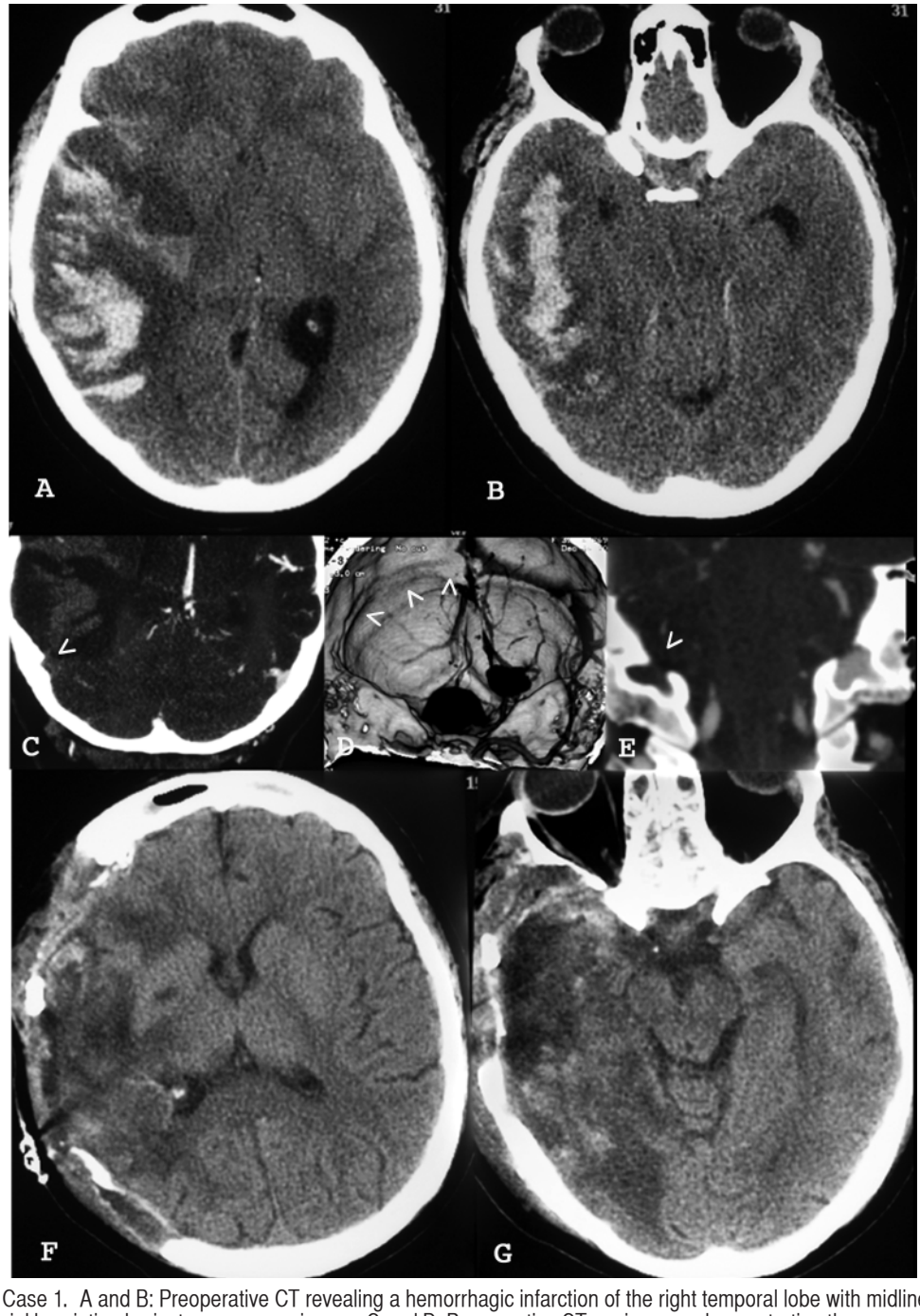

FIG. 1. Case 1. A and B: Preoperative CT revealing a hemorrhagic infarction of the right temporal lobe with midline shift and transtentorial herniation brainstem compression. C and D: Preoperative CT angiograms demonstrating the non-opacification of the transverse sinus on the right side. The *arrowheads* indicate the transverse sinus sulcus. E: Preoperative CT angiogram in frontal projection showing the non-opacification of the jugular bulb on the right side; the jugular foramens are symmetrical. The *arrowheads* indicate the jugular foramen. F and G: Postoperative CT obtained after the second operation.

nized. In the other patients, low–molecular-weight heparins were preferred because they appear to be as effective as unfractionated heparin, they cause fewer hemorrhagic complications, and they can be given at fixed doses without constant laboratory monitoring.[4,14] The anticoagulation therapy was never interrupted. It was maintained with a half dosage for the first 24–72 hours after surgery (during the period of major postoperative hemorrhage risk).[8] The temporary reduction of dosage is in accordance with the guidelines of both the European Federation of Neurological Societies and the ISCVT treatment protocol, which suggest the temporary interruption of anticoagulation therapy in the event of invasive procedures.[4,6]

Decompressive Surgery

Indications for DS included: progressive neurologi-

cal deterioration unresponsive to causal treatment and conventional intensive care (osmotherapy, moderate hyperventilation with a pCO_2 of 30–35 mm Hg and hypothermia with a target temperature of 33–35°C) and CT signs of space-occupying lesions with midline shift or obliteration of the basal cisterns and pupillary signs of transtentorial herniation. Written consent for surgery was obtained from the patient or his or her next of kin. Decompressive surgery was defined in terms of external and internal decompression. External decompression was defined as a hemicraniectomy with duraplasty (diameter ≥ 12 cm), and internal decompression consisted of resecting the infarcted brain tissue and draining cerebral hematomas. External decompression was primarily indicated in the event of diffuse hematomas infiltrating edematous, potentially still viable, eloquent brain, whereas internal decompression was primarily indicated in the event of localized hematomas in infarcted noneloquent brain parenchyma. Internal decompression was performed under microscopic magnification. During the operation the patients were rehydrated with isotonic crystalloids with care taken to maintain euvolemia and a serum osmolality of 300 mOsm/L. The target hematocrit was kept at about 30% to reduce blood viscosity.[6] Colloids and inotropes were used to keep the mean arterial blood pressure level above 90 mm Hg. Postoperative ICP monitoring was performed in all patients who underwent DS. In the event of postoperative ICP elevation a CT scan was performed and the indications for repeated surgery were ruled out. Those patients who were not candidates for repeated surgery were eligible for barbiturate coma with the aim of achieving electroencephalographic burst suppression.

Case Reports

Case 1

History, Examination, and Diagnosis. This 32-year-old right-handed woman was transferred to our institution from another hospital where she had presented in deep coma after a 12-hour history of headache. At examination, the patient was comatose with no motor response to pain and bilaterally fixed and maximally dilated pupils. A CT scan of the brain revealed a hemorrhagic infarct of the temporal lobe on the right side, brain edema, and brainstem compression (Figs. 1A and B). A CT angiography study demonstrated nonvisualization of the transverse and sigmoid sinuses on the right side (Fig. 1C, D, and E). Intravenous mannitol and hyperventilation were ineffective.

Operation and Postoperative Course. Emergency decompressive surgery was planned, and a large frontotemporoparietal hemicraniectomy was performed. The sylvian veins were congested and the vein of Labbé appeared thrombotic. An intraparenchymal superficial clot was removed, while the infarcted cerebral tissue was only partially resected with the rationale of sparing edematous, potentially still viable brain. At the end of the operation the brain appeared slack, well perfused, and pulsating, and the ICP was 5 mm Hg. The postoperative CT scan showed complete clot removal and reduction of the midline shift. On Day 3, the ICP increased up to 25 mm Hg and a CT scan revealed an increased mass effect due to edema and bleeding of the infarcted temporal lobe. Hypothermia and barbiturate coma were effective in lowering the ICP to 15–18 mm Hg until Day 5, when the ICP rose to 30 mm Hg and the right pupil became maximally dilated and unreactive to light. The patient was returned to the operating room, where the craniotomy was enlarged at the site of the prior parietooccipital craniotomy and the temporal squama was extensively drilled up to the cranial base. The bone bleeding was abnormally intense, suggesting compensatory venous drainage through the emissary veins. The theoretical hypothesis that craniotomy may affect the osseous compensatory circuits was not a contraindication to DS because of the multiplicity of the emissary veins and the wide distribution of the diploic venous network.[12] The drilling was extended from the superior orbital fissure to the posterior root of the zygomatic arch with the aim of maximizing the decompression and obtaining an unobstructed view of the temporal lobe from the lateral, basal, and medial surfaces. This exposure was considered necessary to control possible bleeding from the congested and hypertrophic sylvian, temporobasal, and polar veins that were compensating for the thrombosed vein of Labbé. The dura mater was opened. The brain appeared diffusely swollen and the sylvian veins as well as the anterior temporal veins appeared red and congested. The infarcted tissue of the second and third temporal gyri was resected with extreme care taken to preserve all the veins and spare the mesial temporal structures that were not involved in the infarction. Once the infarcted tissue had been resected, the temporal and sylvian veins normalized and the brain swelling rapidly reduced. Intraoperatively, the ambient cistern and the brainstem appeared decompressed from the uncus. The postoperative CT scan showed complete clot removal, decompression of the ambient cistern, and absence of midline shift (Fig. 1F and G).

Outcome. At the 6-month follow-up examination the patient's GOS score indicated moderate disability because of partial neglect and a mild monoparesis of the left arm. She suffered from seizures, which were controlled with phenobarbital.

Case 2

History, Examination, and Diagnosis. This 51-year-old left-handed man was admitted to another hospital because of headache, confusion, and mild right-sided hemiparesis. A CT scan showed an intracerebral hematoma and brain edema of the left parietal lobe with midline shift. He was transferred to our institution, where digital subtraction angiography demonstrated slow circulation, stagnation of the contrast medium in the frontoparietal ascending veins, and no opacification of the superior sagittal sinus, suggestive of thrombosis (Fig. 2B and C). On Day 4, the right-sided hemiparesis worsened and a CT scan revealed the enlargement of the hematoma and worsening of the edema and the mass effect (Fig. 2A). Osmotics were ineffective and the following day he became

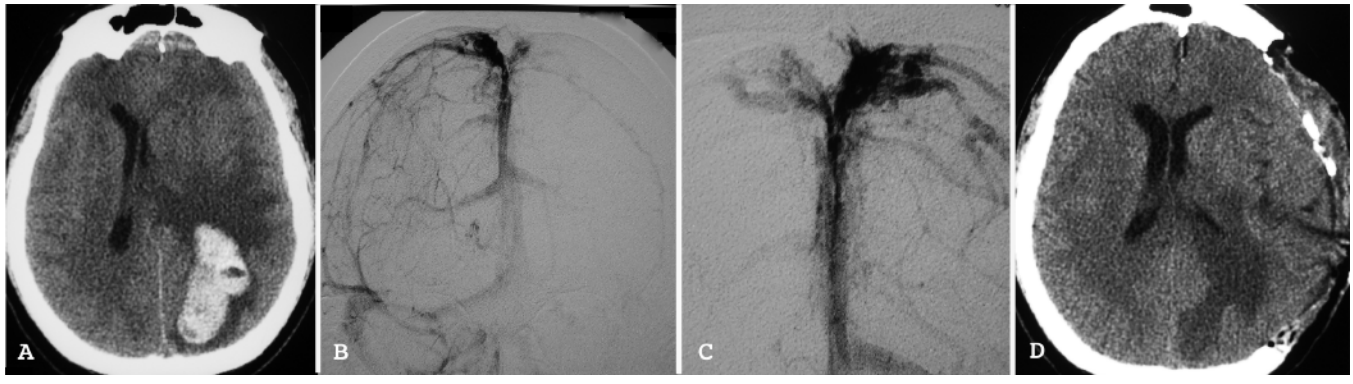

FIG. 2. Case 2. A: Preoperative CT revealing a hemorrhagic infarction of the left parietal lobe with brain swelling and midline shift. B and C: Late-phase right (B) and left (C) internal carotid artery digital subtraction angiograms in frontal projection, demonstrating stagnation of the contrast medium in the frontoparietal ascending veins and no opacification of the superior sagittal sinus. D: Postoperative CT scan of the brain.

comatose with an abnormal flexion response to pain and a dilated pupil on the left side.

Operation and Postoperative Course. A large frontotemporoparietooccipital hemicraniectomy was performed. The bone bleeding was abnormally intense. The dura mater was opened. The brain appeared swollen and the cortical veins draining into the sagittal sinus were congested and red. The clot was evacuated together with the bleeding, congested, infarcted tissue surrounding the hematoma. The ICP remained ≤ 15 mm Hg. On Day 7, sedation was discontinued and the patient began to respond to commands. The postoperative CT scan showed complete clot removal and reduction of the midline shift (Fig. 2D).

Outcome. After 3 months the bone flap was repositioned. The patient returned to his normal daily life; his GOS score indicated good recovery and the neuropsychological evaluation was normal. He suffered from seizures, which were controlled with carbamazepine.

Case 3

History, Examination, and Diagnosis. This 39-year-old left-handed man was admitted to the emergency department because of headache, vomiting and a reduced level of consciousness (GCS score 12). A CT scan showed a hemorrhagic infarct of the temporal lobe on the right side, brain swelling, and compression of the ambient cistern (Fig. 3A). An MR imaging study demonstrated the absence of a flow signal into the transverse and sigmoid sinuses on the right side (Fig. 3B). Diffusion-weighted images revealed predominant cytotoxic edema. During the next 3 hours the patient's condition deteriorated further and, on reevalution, he was comatose (GCS score 8) with dilation of the pupil on the right side. After resuscitation with tracheal intubation, artificial hyperventilation, and mannitol, the pupillary diameter normalized.

Operation and Postoperative Course. Emergency decompressive surgery was planned and a large frontotemporoparietal hemicraniectomy, with drilling of the temporal bone, was performed. The temporal lobe was swollen, pale, and nonpulsating. The vein of Labbé was thrombosed while the sylvian and anterior temporal veins were prominent and congested. The hemorrhagic infarct was resected (middle and inferior temporal gyrus) with extreme care taken to preserve all the veins and to spare the mesial temporal structures. Postoperative CT showed complete clot removal and reduction of the temporal lobe swelling (Fig. 3C).

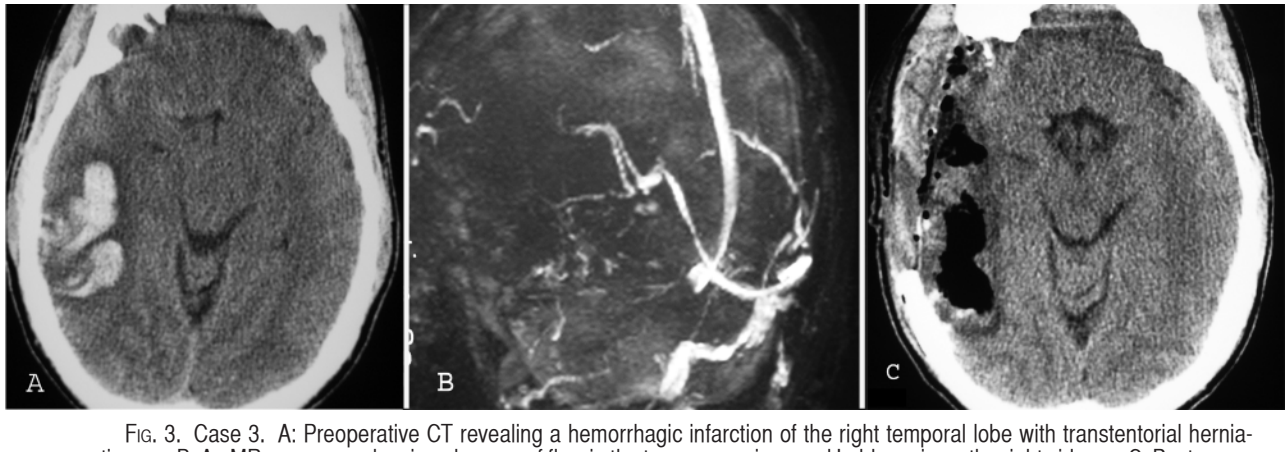

FIG. 3. Case 3. A: Preoperative CT revealing a hemorrhagic infarction of the right temporal lobe with transtentorial herniation. B: An MR venogram showing absence of flow in the transverse sinus and Labbe vein on the right side. C: Postoperative CT of the brain.

TABLE 1: Summary of published clinical series and case-reports on decompression for CVDST*

Authors & Year	GCS Score	Pupillary Signs	Dural Sinus	Decompression	Outcome (GOS)
Stefini et al., 1999	4	bilaterally dilated	transverse & sigmoid	ED	GR
	6	bilaterally dilated	transverse & sigmoid	ED	GR
	5	bilaterally dilated	transverse & sigmoid	ED & drainage of a superficial clot	SD
Weber & Spring, 2004	<8	unilaterally dilated	transverse & sigmoid	ED	MD
Keller et al., 2005	9	normal	superior sagittal	ED & ID	GR
	6	normal	frontal cortical veins	ED & ID	GR
	13	normal	transverse & sigmoid	ED & ID	MD
	13	normal	transverse & sigmoid	ED	MD
present series	3	bilaterally dilated	transverse & sigmoid	ED & ID	MD
	5	unilaterally dilated	superior sagittal	&	GR
	8	unilaterally dilated	transverse & sigmoid	ED and ID	GR

* Based on MEDLINE and EMBASE search from 1970 to 2008. Abbreviations: ED = external decompression (defined as craniectomy and duraplasty); GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; GR = Good Recovery; MD = Moderate Disability; ID = internal decompression (defined as hematoma drainage and resection of necrotic tissue); SD = Severe Disability.

Outcome. After 1 month the patient's GOS score indicated good recovery. He did not suffer from seizures. The neuropsychological assessment was normal.

Discussion

The clinical history of these 3 patients supports the hypothesis that emergency DS may be life-saving in patients with transtentorial herniation due to CVDST and that both the functional and the neuropsychological outcome may be favorable, even in deeply comatose patients with ominous pupillary signs. No intracranial bleeding complications directly related to the DS occurred and no patient developed postoperative hydrocephalus.

Consistent with the rarity of the pathology, a MEDLINE and EMBASE search of the literature from January 1970 to October 2008 using 6 key words (cerebral, dural sinus, vein, thrombosis, surgery, decompression) revealed only 3 surgical reports on patients (a total of 8) who underwent DS for CVDST.[11,17,20] Four of these 8 patients had pupillary signs of brain herniation[17,20] (Table 1).

Our unexpectedly favorable results were consistent with those reported in the literature. Including our 3 patients, 6 of the 7 patients who were moribund with pupillary signs were independent in daily living after discharge from the rehabilitation units.[17,20] Furthermore, Keller and colleagues[11] observed a good functional outcome (moderate disability, 2 cases; good recovery, 2 cases) in all 4 of their patients who underwent DS before developing clinical signs of herniation (Table 1). The 2 patients who were categorized as having moderate disability suffered from slight focal signs probably related to the eloquence of the cortex involved in the hemorrhagic infarction.[11]

Although the analysis of case reports and small case series may be affected by publication bias and a formal statistical investigation of confounding variables and procedure-related risk factors is impossible, the reported clinical results are worthy of attention for at least 2 reasons: 1) according to the ISCVT analyses, all patients had one or more of the main independent predictors of death (intracranial hemorrhage, clinical worsening, GCS score < 9, new focal sign);[7] and 2) the most frequent CVDST mechanism of death is transtentorial herniation, and 7 of the 11 patients already had related clinical signs.[2]

Explanations of how DS may affect the physiopathology of CVDST can only be speculative. Cerebral venous and dural sinus thrombosis causes a wide spectrum of parenchymal changes, ranging from vasogenic and cytotoxic edema to intracerebral hematomas.[3,5] These complications may lead to a compartmentalized intracranial hypertension that secondarily compresses intraparenchymal veins, with a subsequent worsening of edema and swelling. Furthermore, in CVDST the regional cerebral blood flow may be reduced because of paradoxical arteriolar vasoconstriction.[5,19] A condition that, based on experimental data, has features reminiscent of ischemic penumbra.[18] Decompressive surgery creates space for the swelling brain and, by reducing intracranial hypertension and brainstem compression, may break the vicious cycle by relieving arteriolar constriction and restoring flow in compressed venous collaterals.

In view of the rarity of patients with malignant CVDSTs necessitating emergency DS, the surgical experience is limited and accepted guidelines on how to perform the decompression are lacking. The review shows that there is no unanimity as to what constitutes the preferable surgical strategy. Different authors have adopted distinct approaches: pure external decompression (in 5 cases) or internal decompression with or without removal of the bone flap (in 6 cases) (Table 1). A statistical comparison of the results obtained with the 2 strategies is beyond the limits of small series because of limited power and clinical, timing, and risk-factor heterogeneity among the studies.

In our experience, we tried to define criteria a priori to guide surgical decision making. In particular, we considered the site and characteristics of the clot and the eloquence of the brain, with the aim of tailoring surgery and

reducing its burden in a situation where cytotoxic edema may coexist with viable brain.[5,13] In practice, the peculiarities of the cerebral lesions (for example, site, clot dimension, depth), together with the clinical course and the supposed hemispheric dominance, guided our decisions toward internal decompression with removal of the bone flap in all cases. In the light of the favorable neuropsychological outcomes, this experience appears reasonable and is in accordance with that of other authors.[11]

On the other hand, other case reports have shown almost equally favorable results with pure external decompressions.[17,20] Recently, MR imaging studies have demonstrated the existence of parenchymal changes (vasogenic edema) that could be completely reversible, and such studies may be advocated in support of a less invasive surgical approach.[16] From the analysis of the cases in which patients were treated by means of external decompression, the importance of surgical timing emerges. Stefini et al.[17] observed that the ICP can be better controlled when the operation is promptly performed. The importance of timing may be indirectly supported by the recent trials investigating external decompression for patients with space-occupying arterial infarcts.[10]

In conclusion, although the rarity of the condition precludes any statistically based evidence, the functional recovery obtained in a situation in which the prognosis would otherwise have been grim[15] appears clinically relevant. Decompressive surgery may offer the last chance of life and a favorable functional outcome in selected patients with CVDST whose condition is deteriorating because of impending brain herniation that is unresponsive to causal therapy and intensive care. In the future, updates on the pathophysiology of CVDST and new developments in imaging technologies will provide a better and more comprehensive insight into the specific therapeutic requirements of each patient. A patient-specific pathophysiologically based approach could enable the clinician to choose the appropriate surgical strategy and timing with more accuracy and, probably, greater effectiveness.

Disclosure

The authors report no conflict of interest concerning the material or methods used in this study or the findings specified in this paper.

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A comparison of hinge craniotomy and decompressive craniectomy for the treatment of malignant intracranial hypertension: early clinical and radiographic analysis

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Object. Hinge craniotomy (HC) has recently been described as an alternative to decompressive craniectomy (DC). Although HC may obviate the need for cranial reconstruction, an analysis comparing HC to DC has not yet been published.

Methods. A retrospective review was conducted of 50 patients who underwent cranial decompression (20 with HC, 30 with DC). Baseline demographics, neurological examination results, and underlying pathology were reviewed. Clinical outcome was assessed by length of ventilatory support, length of intensive care unit stay, and survival at discharge. Control of intracranial hypertension was assessed by average daily intracranial pressure (ICP) for the duration of ICP monitoring and an ICP therapeutic intensity index. Radiographic outcomes were assessed by comparing preoperative and postoperative CT scans for: 1) Rotterdam score; 2) postoperative volume of cerebral expansion; 3) presence of uncal herniation; 4) intracerebral hemorrhage; and 5) extraaxial hematoma. Postoperative CT scans were analyzed for the size of the craniotomy/craniectomy and magnitude of extracranial herniation.

Results. No significant differences were identified in baseline demographics, neurological examination results, or Rotterdam score between the HC and DC groups. Both HC and DC resulted in adequate control of ICP, as reflected in the average ICP for each group of patients (HC = 12.0 ± 5.6 mm Hg, DC = 12.7 ± 4.4 mm Hg; $p > 0.05$) at the same average therapeutic intensity index (HC = 1.2 ± 0.3, DC = 1.2 ± 0.4; $p > 0.05$). The need for reoperation (3 [15%] of 20 patients in the HC group, 3 [10%] of 30 patients in the DC group; $p > 0.05$), hospital survival (15 [75%] of 20 in the HC group, 21 [70%] of 30 in the DC group; $p > 0.05$), and mean duration of both mechanical ventilation (9.0 ± 7.2 days in the HC group, 11.7 ± 12.0 days in the DC group; $p > 0.05$) and intensive care unit stay (11.6 ± 7.7 days in the HC group, 15.6 ± 15.3 days in the DC group; $p > 0.05$) were similar. The difference in operative time for the two procedures was not statistically significant (130.4 ± 71.9 minutes in the HC group, 124.9 ± 63.3 minutes in the DC group; $p > 0.05$). The size of the cranial defect was comparable between the 2 groups. Postoperative imaging characteristics, including Rotterdam score, also did not differ significantly. Although a smaller volume of cerebral expansion was associated with HC (77.5 ± 54.1 ml) than DC (105.1 ± 65.1 ml), this difference was not statistically significant.

Conclusions. Hinge craniotomy appears to be at least as good as DC in providing postoperative ICP control and results in equivalent early clinical outcomes. *(DOI: 10.3171/2009.4.FOCUS0960)*

Key Words • **decompressive craniectomy** • **hinge craniotomy** • **cerebral decompression** • **intracranial hypertension**

Cranial decompression for the treatment of medically refractory intracranial hypertension was first reported by Kocher in 1901 and Cushing in 1908.[4,16] In the century following its introduction, its role remained controversial. Recently, many have reported a significant reduction in ICP and even a positive effect on outcome using this technique.[3,9,20] Currently, despite the paucity of randomized trials, DC has become an accepted and effective therapy for patients with malignant intracranial hypertension.

Like any other surgical procedure, there is morbidity associated with DC.[1,2,8,11,14] With a large bone defect, there is a potential risk to the exposed underlying brain, which may require the use of protective headgear. Delayed postoperative seizures have been reported in 37% and hydrocephalus in 40% of patients after DC.[10,14] Neurological worsening has also been described after craniectomy with improvement following cranioplasty.[19] In addition, as many as 17% of patients will have chronic, debilitating headaches that only improve with replacement of the cranial plate.[5] Cranioplasty requires a second operation performed under general anesthesia and exposes the pa-

Abbreviations used in this paper: BMI = body mass index; DC = decompressive craniectomy; EDH = epidural hematoma; GCS = Glasgow Coma Scale; HC = hinge craniotomy; ICH = intracerebral hemorrhage; ICP = intracranial pressure; ICU = intensive care unit; INR = international normalized ratio; IVH = intraventricular hemorrhage; MCA = middle cerebral artery; PT = prothrombin time; PTT = partial thromboplastin time; SDH = subdural hemorrhage; TII = therapeutic intensity index.

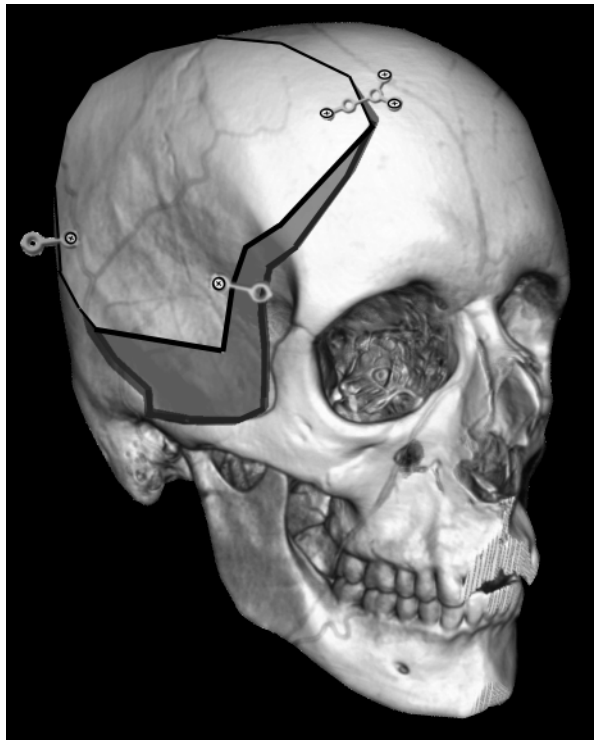

FIG. 1. Illustration of the technique of HC. A single Y-shaped plate secures the bone flap to the surrounding skull, allowing for elevation of the flap with underlying cerebral edema. Peripheral plates prevent bone flap subsidence following resolution of the edema.

tient to additional potential complications. Cranioplasty has a significant infection risk with many patients requiring removal of the autologous cranial plate or prosthetic with subsequent revision.[7,10,14,15] In addition, resorption of the bone plate can be a problem, especially in the pediatric population.[1,7,10]

The ideal procedure for cranial decompression would be one that combines the effectiveness of DC but limits the aforementioned difficulties with which it is associated. In 2007, three investigative groups reported on the use of HC.[6,12,17] This procedure involves resecuring the cranial plate in a noncircumferential fashion to the underlying skull at the time of the initial decompression, allowing for cerebral expansion through the cranial defect. By allowing the native plate to remain in place, there is a limited cosmetic defect once the cerebral edema resolves and the need for delayed cranioplasty is limited. Despite these potential advantages, the utility of HC for the relief of elevated ICP is "yet to be determined" and may be only appropriate for "borderline" cases.[12] To date, there does not exist a published comparison of DC and HC in the treatment of malignant intracranial hypertension.

Methods

Patient Characteristics

We conducted a retrospective chart review of 50 patients undergoing cerebral decompression for treatment of malignant intracranial hypertension related to mass lesions. These patients were all treated at a single institution (Albany Medical Center) by 9 different neurosurgeons over a 25-month period (November 2006 to December 2008).

TABLE 1: Intracranial pressure TII*

Score	Therapy
1	head elevation and sedation
2	hyperosmotics (such as mannitol and hypertonic saline)
3	acute hyperventilation
4	paralytics
5	barbiturate coma

* For each day of ICP monitoring, the maximal therapy administered for ICP control was determined and the patient was assigned one of the corresponding values (1–5) for that day.

Surgical Decision-Making

In the current patient series, the decision to perform either an HC or DC was left to the discretion of the attending neurosurgeon. Since January 2007 the senior author (J.W.G.) has uniformly applied the technique of HC whenever it was believed that a patient would benefit from cerebral decompression. The sole exception to this policy was a patient who was hypotensive and hypoxic with multiple traumatic injuries, who died within hours after presenting with an acute SDH and massive cerebral swelling; in this patient, it was difficult to close the scalp. As local experience with the technique expanded, other attending neurosurgeons have adopted it. In the current patient series the senior author performed 13 (65%) of the 20 HCs. The remaining 7 HCs (35%) were performed at the discretion of the individual attending neurosurgeon.

In general, patients with trauma and intracranial hy-

TABLE 2: Rotterdam score calculation*

Category	Score†
basal cisterns	
normal	0
compressed	1
absent	2
midline shift	
≤5 mm	0
>5 mm	1
epidural mass lesion	
present	0
absent	1
intraventricular or subarachnoid blood	
absent	0
present	1
adjustment	+1

* From Maas et al., 2007.
† Scores based on CT findings.

TABLE 3: Summary of patients undergoing HC*

Cause	Age (yrs), Sex	Motor GCS Score	Mechanism	CT Finding	MLS (mm)	Rotterdam Score	Survival
trauma							
	26, M	2	nonpenetrating GSW	SDH, GSW	8.6	4	yes
	55, F	2	fall	SDH	22	6	no
	71, M	4	MVA	SDH	18	2	yes
	51, M	4	fall	SDH, EDH, ICH	14	2	yes
	56, M	3	high-speed blunt object	SDH, EDH, open fracture	15	4	yes
	36, M	2	MVA	SDH	3	3	yes
	25, M	6	MCC	SDH	7	2	yes
	60, M	6	fall	SDH, ICH	7	4	yes
	31, M	4	MVA	SDH, ICH	10	2	yes
	39, M	4	MCC	ICH	14	4	yes
	77, F	5	fall	SDH, ICH	13	3	no
	39, M	5	assault	SDH, ICH	8	3	yes
infarct							
	72, M	2	rt M1 occlusion	hypodensity of entire MCA distribution	16	2	no
	44, F	6	rt M1 occlusion	hypodensity >2/3 MCA distribution	5	2	no
	52, M	5	rt ICA occlusion	hypodensity entire MCA distribution	5	3	yes
	43, F	5	lt M1 occlusion	hypodensity >2/3 MCA distribution	12	4	yes
ICH							
	66, M	5	hemorrhagic conversion of lt MCA infarct	118 ml lt parietal ICH	12.4	3	no
	69, F	5	lobar, suspected amyloid angiopathy	44 ml rt temporal ICH	9	3	yes
	45, F	2	hypertensive hemorrhage	60 ml rt basal ganglia ICH w/IVH	10	3	yes
	53, M	5	hypertensive hemorrhage	78 ml lt basal ganglia	11	4	yes

* GSW = gunshot wound; ICA = internal carotid artery; MCC = motorcycle collision; MLS = midline shift; MVA = motor vehicle accident.

pertension demonstrating clinical evidence of cerebral herniation with a radiographically confirmed intracranial mass lesion were taken directly to the operating room for decompression. In addition, mass effects with ≥ 5-mm midline shift were treated with immediate surgery. In transport to the operating room, these individuals may have received medical management (such as hyperventilation therapy and osmotic therapy) for intracranial hypertension.

Patients suffering an MCA infarct involving a large percentage of the vascular distribution were treated at variable time points based largely on the clinical judgment of the treating neurosurgeon. These patients received cerebral decompression either "prophylactically," after demonstration of medically refractory intracranial hypertension as recorded by ICP monitoring, or after development of a cerebral herniation syndrome.

Surgical Technique

Incision and Cranial Exposure. Hinge craniotomies were performed utilizing the technique described by Schmidt et al.,[17] Ko and Segan,[12] and Goettler and Tucci.[6] Briefly, the patient was taken to the operating room where general anesthesia was induced. The patient was placed either supine with the head turned or in the lateral decubitus position to allow the sagittal suture to be as close to parallel as possible with the floor. The head and neck were then elevated above the heart to maximize venous return. The scalp was then shaved, prepared, and draped in the usual fashion. A standard skin incision in the shape of a (reverse) question mark was marked beginning 1 centimeter in front of the tragus, extending above and behind the ear to approximately the posterior mastoid line and then curving forward to within 1 centimeter from the midline to end at or near the hairline. The scalp was incised and reflected forward off the underlying temporalis fascia and muscle. The root of the temporozygomatic process was identified and the temporalis fascia and muscle were then divided using monopolar cautery. The temporalis muscle was mobilized off the underlying skull using a periosteal elevator. The exposed skull was then inspected to ensure that the pterion and the temporal bone had been adequately exposed.

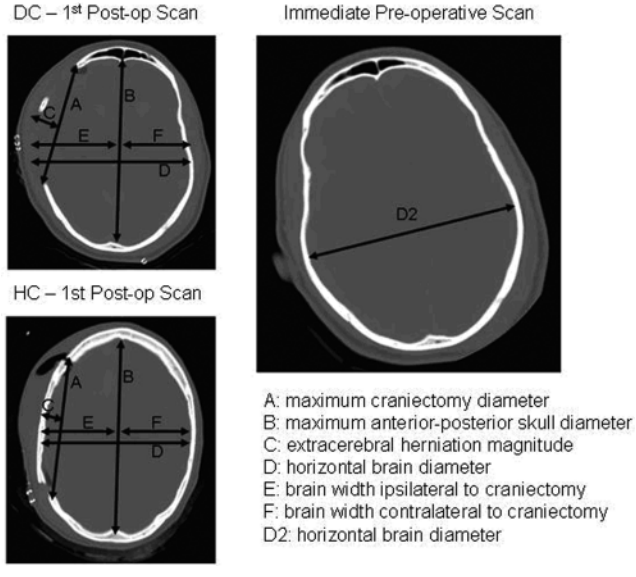

FIG. 2. Diagrams showing CT morphometrics for each group. A demonstration of the measurements taken from the first postoperative CT scan for patients in the DC *(top left)* and HC *(bottom left)* groups is shown. The measurements that were obtained are listed on the bottom right, along with corresponding indicators. At the same approximate level on the last preoperative CT scan *(right)* the maximal preoperative horizontal brain diameter was obtained (D2).

Craniotomy and Temporal Craniectomy. Bur holes were fashioned at the pterion (exposing frontal and temporal dura), the temporal region, the posterior parietal region, and the frontal region, with each bur hole placed as close to the scalp incision as possible. The dura was stripped from each bur hole using a dissector. A large bone flap was fashioned and elevated off the underlying dura. A temporal craniectomy was begun by using a large rongeur, and the temporal bone was resected to the floor of the middle fossa. The temporal craniectomy was completed by using a large (5-mm), rough, diamond drill bit to ensure adequate bone hemostasis. The sphenoid wing could also be drilled to ensure an adequate hinge effect. The bone edges were inspected and any violation of the mastoid air cells or frontal sinus was then appropriately addressed using bone wax or a local pericranial graft.

Dural Opening and Evacuation of Hematoma. In the specific case of SDH the dura was often fenestrated first to allow for evacuation of the SDH while the underlying brain was gradually rather than abruptly decompressed. The dural leaves are connected to form a roughly cruciate pattern. In the case of stroke or intraparenchymal hematoma the dura was opened in a cruciate fashion. Large intraparenchymal hematomas were then evacuated through a corticotomy using suction, irrigation, and ring forceps. Intradural hemostasis was obtained using bipolar cautery and packing and was then confirmed by return of clear irrigation.

Dural Closure and Bone Plate Placement. After intradural hemostasis had been confirmed, the dural leaves were laid over the surface of the brain. Duragen (Integra LifeSciences Corp.) was placed between any dural defects. The bone edges were inspected and epidural tack-up sutures were placed for epidural hemostasis. If a DC was performed, at this juncture, the bone plate was packaged for storage in the tissue bank for replacement at a later date. If an HC was performed, 3 miniplates were fastened: a Y-shaped plate just posterior to the coronal suture, and two 2-hole plates, 1 at the sphenoid wing and a second in the posterior temporal region both below the temporalis muscle and fascia. The Y-shaped plate was secured to the surrounding skull while the 2-hole plates acted as buttress plates to prevent future settling (Fig. 1).

Soft Tissue Closure. The temporalis muscle but not the fascia was then reapproximated with sutures, thus allowing for greater cerebral expansion. Care was taken to ensure that the buttress plates were placed underneath the temporalis muscle so that the scalp was protected from the underlying hardware. The galea could then be scored with monopolar cautery, and the galea of the surrounding scalp could be undermined to encourage further scalp expansion. The scalp was then closed in two layers.

Clinical Analysis

Patient Demographics. Preoperative demographics reviewed for each patient included: patient age, BMI, sex, indication for decompression, laterality of decompression, preoperative pupillary examination, motor GCS score, preoperative use of anticoagulation or antiplatelet agents, coagulation studies (PT, PTT, and INR), and need for correction of coagulopathies.

Postoperative Results. Several postoperative variables were analyzed, including: 1) postoperative ICP control, which was measured by the average daily ICP for each of the first 5 postoperative days and average overall ICP for the duration of monitoring; 2) ICP TII, determined for each day of ICP measurement based on maximal therapy required for ICP control (Table 1); 3) operative time of cerebral decompression procedure; 4) need for reoperation; 5) duration of mechanical ventilation and ICU stay; and 6) hospital survival.

Radiographic Analysis

For each patient undergoing cranial decompression, both the immediate preoperative and postoperative cranial CT scans were reviewed and classified based on the following characteristics: midline shift, status of basal cisterns (present, compressed, effaced), and the presence of uncal herniation, midbrain torsion (as assessed by deformation of the cerebral peduncle and midbrain tegmentum), IVH, subarachnoid hemorrhage, SDH, and/or EDH. In addition, for each CT scan, a Rotterdam score was calculated (Table 2).[13]

Volumetric Analysis and CT Morphometrics. BrainLAB iPlan software (BrainLAB) was used to calculate the volumetric cerebral volume and ICH volume of both the preoperative and postoperative cranial CT scans for each patient. The volume of cerebral expansion and hematoma volume change was then calculated according to the following formulas: 1) volume of cerebral expansion

TABLE 4: Summary of patients undergoing DC*

Category	Age (yrs), Sex	Motor GCS Score	Mechanism	CT Finding	MLS (mm)	Rotterdam Score	Survival
trauma							
	59, M	1	fall	SDH	15	6	no
	41, F	5	fall	SDH	16	5	yes
	10, M	3	pedestrian vs automobile	SDH, EDH	6	6	yes
	46, M	4	MVA	SDH, ICH	6	6	no
	23, M	5	MCC	SDH, ICH	7	4	yes
	22, M	4	MVA	SDH	9	5	yes
	30, M	5	assault	SDH, EDH	8	5	yes
	36, F	5	pedestrian vs automobile	SDH, ICH	7	3	yes
	41, F	5	fall	SDH, ICH	10	4	yes
	48, M	2	fall	SDH	18	5	yes
	27, M	4	fall	SDH, ICH	8	6	yes
	47, M	4	MCC	SDH, ICH	0	5	no
	16, M	5	fall	SDH	5	5	yes
	75, M	6	fall	SDH	8	4	no
	20, M	2	assault	EDH	11	3	yes
	28, M	2	assault	SDH	22	6	no
	66, M	1	ATV rollover	SDH, ICH	4	5	no
	48, M	4	fall	SDH	13	4	yes
infarct							
	65, F	6	rt MCA embolus	hypodensity > 2/3 MCA distribution	6	3	yes
	42, M	4	lt ICA dissection	36 ml ICH within previous MCA infarct	10	4	yes
	63, M	5	rt M1 occlusion	hypodensity > 2/3 MCA distribution	11	4	yes
	30, M	4	rt M1 thrombus	hypodensity > 2/3 MCA distribution	11	4	yes
	57, M	5	rt ICA dissection, rt M1 thrombus	hypodensity > 2/3 MCA distribution	2	2	no
	79, M	6	rt M1 embolus	hypodensity > 2/3 MCA distribution	9	3	yes
ICH							
	22, M	6	postop ICH into tumor bed	41 ml of rt frontal ICH	17	5	yes
	42, F	1	hypertensive hemorrhage	diffuse IVH w/hydrocephalus	0	3	yes
	57, M	5	hypertensive hemorrhage	96 ml rt thalamic ICH	17	5	no
	69, M	6	anticoagulation-induced	75 ml rt thalamic ICH	10	5	no
	58, F	6	hemorrhagic conversion rt MCA infarct	22 ml rt frontal ICH	10	4	yes
	48, M	5	hypertensive hemorrhage	67 ml lt basal ganglia ICH w/IVH	6	4	yes

* ATV = all terrain vehicle.

= postoperative volumetric cerebral volume – preoperative volumetric cerebral volume and 2) hematoma volume change = postoperative volumetric ICH volume – preoperative volumetric ICH volume

Utilizing the technique detailed by Flint et al.,[5] a series of CT scan measurements was used to standardize the size of cranial decompression between patients (Fig. 2).

Statistical Analysis

Each of the clinical and radiographic characteristics was compared between the HC and DC groups. The independent 2-sample t-test was used for comparison of variables of normal distribution. For nonparametric analysis, the Wilcoxon-Mann-Whitney test was used. Statistical significance was defined as $p < 0.05$.

TABLE 5: Patient demographics according to group*

Variable	HC	DC
no. of patients	20	30
M:F ratio	14:6	24:6
mean patient age	50.5 ± 15.5	43.8 ± 18.4
mean BMI	26.8 ± 4.9	25.9 ± 4.3
surgical indication		
trauma	12 (60)	18 (60)
infarct	4 (20)	6 (20)
ICH	4 (20)	6 (20)
side of decompression		
rt	13 (65)	23 (77)
lt	7 (35)	7 (23)
pupillary examination result		
equal/reactive	11 (55)	13 (43)
anisocoric	7 (35)	14 (47)
fixed/dilated	2 (10)	2 (7)
unable to assess	0	1 (3)
mean motor GCS score	4.1 ± 1.4	4.2 ± 1.6
no. w/use of anticoagulation/antiplatelet agents	6 (30)	7 (23)
mean PT (sec)	16.5 ± 19.8	12.2 ± 3.9
mean PTT (sec)	26.9 ± 8.4	24.9 ± 3.4
mean INR	1.6 ± 1.9	1.2 ± 0.5
required correction for coagulopathy	3 (15)	3 (10)

* All values are number of patients (%) unless otherwise indicated. None of the variables were statistically significant between groups.

Results

A total of 50 patients were reviewed, 20 who received an HC and 30 who received a DC. The indication for cerebral decompression was trauma (12 patients in the HC group, 18 patients in the DC group), ischemic stroke (4 patients in the HC group, 6 patients in the DC group), and ICH (4 patients in the HC group, 6 patients in the DC group). Within the category of trauma, the primary indication for surgery was evacuation of a mass lesion in 26 patients (87%) and posttraumatic swelling in 4 patients (13%; Tables 3 and 4).

Demographics

There were no statistically significant differences ($p < 0.05$) among any of the preoperative demographic variables: patient age, BMI, sex, surgical indication, side of decompression, pupillary examination result, motor GCS score, use of anticoagulation/antiplatelet agents, coagulation studies, and need for correction of coagulopathy (Table 5).

Postoperative Results

There was no statistically significant difference ($p < 0.05$) in either postoperative ICP control or ICP TII between groups (Tables 6 and 7; Figs. 3 and 4). Both groups demonstrated adequate ICP control as evidenced by the average ICP for the duration of monitoring (12.0 ± 5.6 mm Hg in the HC group vs 12.7 ± 4.4 mm Hg in the DC group) at a low average TII (1.2 ± 0.3 in the HC group vs 1.2 ± 0.4 in the DC group). All patients received head elevation and sedation as a daily therapeutic measure with only a few requiring more intensive treatment. There was no significant difference between the 2 groups in terms of operative time, need for reoperation, duration of mechanical ventilation, ICU stay, or hospital survival (Table 8).

TABLE 6: Intracranial pressure control summary*

ICP Measurement Time Point	HC Group ICP (mm Hg)	DC Group ICP (mm Hg)
length of monitoring	12.0 ± 5.6	12.7 ± 4.4
POD 1	10.1 ± 5.9	12.1 ± 6.7
POD 2	13.4 ± 8.2	12.4 ± 5.1
POD 3	11.3 ± 3.5	13.1 ± 5.9
POD 4	11.9 ± 3.2	13.1 ± 5.5
POD 5	12.1 ± 2.6	15.0 ± 6.3

* All values are means ± SDs. None of the values were statistically significant between groups. Abbreviation: POD = postoperative day.

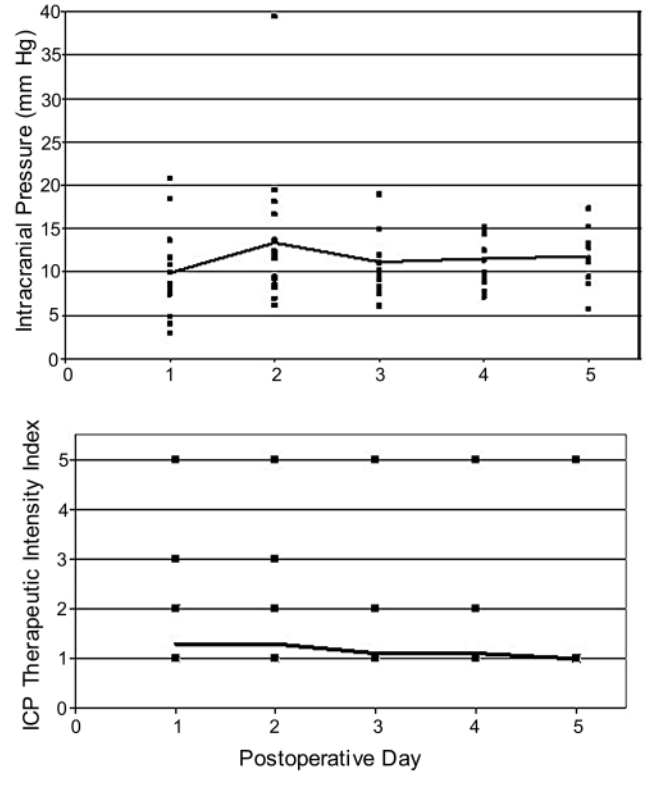

FIG. 3. Line graphs of the average daily ICP recordings *(upper)* and ICP TII *(lower)* for each of the patients in the HC group with ICP monitoring during the first 5 postoperative days. The solid line represents the overall average for the groups in each graph.

TABLE 7: Intracranial pressure TII summary*

TII Measurement Time Point	HC Group TII	DC Group TII
average	1.2 ± 0.3	1.2 ± 0.4
POD 1	1.3 ± 0.5	1.1 ± 0.3
POD 2	1.3 ± 0.6	1.1 ± 0.5
POD 3	1.1 ± 0.3	1.1 ± 0.3
POD 4	1.1 ± 0.3	1.0 ± 0.2
POD 5	1.0 ± 0.0	1.3 ± 0.8

* All values are means ± SDs. None of the values were statistically significant between groups.

Radiographic Analysis

Analyzing the immediate preoperative and postoperative cranial CT scans for both groups revealed no differences that were statistically significant (Table 9). Both groups had comparable baseline radiographic study results and achieved comparable postoperative results. The imaging in 15 of the patients revealed a reversal in the direction of the midline shift between the preoperative and postoperative scans (Fig. 5). A larger proportion of these patients were in the DC group (11 [37%] of 30) than the HC group (4 [20%] of 20). This subset of the DC group also experienced a greater overall preoperative to postoperative change in midline shift than the HC subset (14.8 ± 7.7 mm in the HC group vs 16.0 ± 6.4 mm in the DC group) although this difference was not statistically significant.

Volumetric Analysis and CT Morphometrics

Although a smaller volume of cerebral expansion was associated with the HC group (77.5 ± 54.1 ml) than the DC group (105.1 ± 65.1 ml), this difference was also not statistically significant (Table 9). Analysis of morphometric characteristics of the cranial CT scans for both the HC and DC groups revealed similar results (Table 10). The maximum craniectomy diameter was comparable between the two groups even when standardized for different cranial sizes. The extracerebral herniation magnitude varied but not to a statistically significant degree. However, when extracerebral herniation was expressed as a ratio with the maximum craniectomy diameter, creating the extracerebral herniation index, the difference between the HC and DC groups was statistically significant (0.16 ± 0.05 in the HC group vs 0.21 ± 0.06 in the DC group; $p = 0.007$).

Discussion

The use of HC for cerebral decompression was introduced in 2007 by three investigative groups.[6,12,17] These studies were primarily technical reports describing the nuances of the procedure (such as enlarging the subgaleal space, plating of the autologous flap, and performing a galeatomy). Although adequate postoperative ICP control was reportedly obtained, no objective data were provided to demonstrate this assertion. Overall, the survival of the patients treated appeared satisfactory. Despite the early promising results with this technique, a comparative analysis of HC and DC has not yet been published.

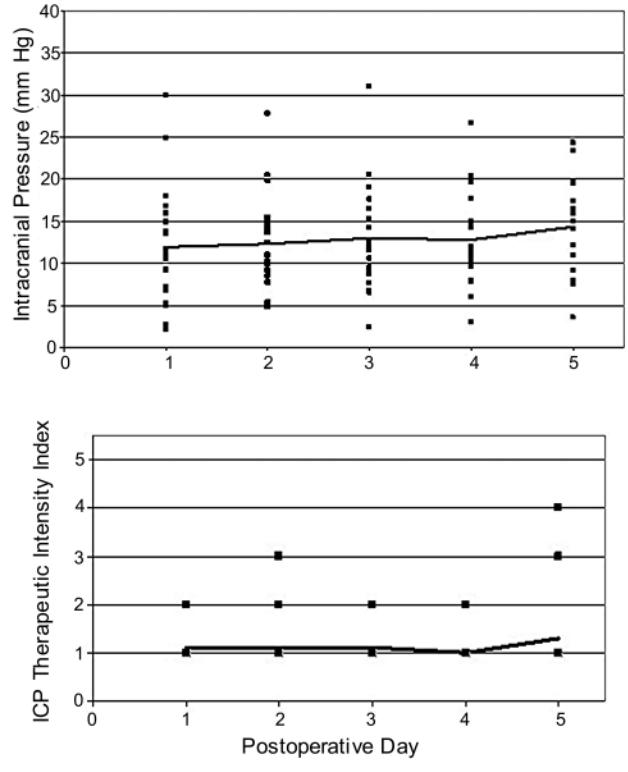

Figure 4. Line graphs of the average daily ICP recordings *(upper)* and ICP TII *(lower)* for each of the patients in the DC group with ICP monitoring during the first 5 postoperative days. The solid line represents the overall average for the groups in each graph.

Our review of the unilateral cranial decompressions at our institution over a 25-month period appears to demonstrate that HC is likely comparable to DC. Between the two subgroups of patients, the preoperative clinical and radiographic characteristics were very similar. This similarity helped to ensure that the results from the HC and DC groups could be reliably compared. No significant end points were statistically different, including radiographic and survival analysis.

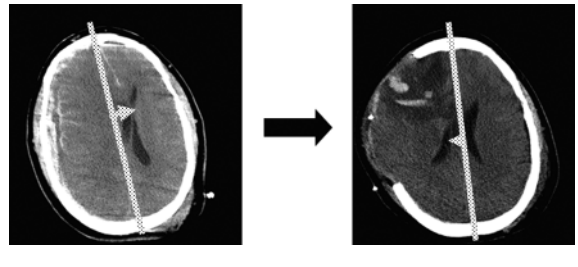

FIG. 5. Computed tomography scans showing postoperative reversal of midline shift. A preoperative CT scan *(left)* demonstrates a right acute SDH with right-to-left midline shift *(arrow)*. A postoperative CT scan *(right)* obtained after DC shows extracranial herniation and midline shift from left to right *(arrow)*.

TABLE 8: Postoperative clinical results*

Variable	HC	DC
mean operative time (min)	130.4 ± 71.9	124.9 ± 63.3
no. of patients needing reoperations (%)	3/20 (15)	3/30 (10)
mean duration of mechanical ventilation (days)	9.0 ± 7.2	11.7 ± 12.0
mean ICU stay (days)	11.6 ± 7.7	15.6 ± 15.3
no. of patients w/ hospital survival (%)	15/20 (75)	21/30 (70)

* None of the variables were statistically significant between groups.

Effect of HC on Postoperative ICP

The most important finding of this study was that the two procedures achieved equivalent ICP control, limiting the need for aggressive medical therapy. This was true despite a smaller volume of cerebral expansion in association with HC. It appears that both HC and DC provide adequate space for critical brain swelling.

In addition, we encountered a pair of surprising observations in our review. The first observation was the number of patients who experienced a reversal in the direction of the midline shift between the preoperative and postoperative scans. This shift occurred in a greater number and proportion of patients in the DC group, and these sets of images also showed a greater magnitude in the overall preoperative to postoperative change of midline shift when compared with the HC group.

The second observation was the difference between HC (0.16 ± 0.05) and DC (0.21 ± 0.06) in the extracerebral herniation index, which was the only statistically significant value in our CT analysis. The extracerebral herniation index compared the magnitude of extracerebral herniation with the maximum diameter of the craniectomy in ratio form. A higher index occurs with a significant degree of extracerebral herniation through the cranial defect. Conversely, a smaller index would indicate less extracerebral herniation in comparison with the defect. Despite a comparable size of cranial decompression between HC and DC (as evidenced by our morphometric analysis) and similar postoperative ICPs at a similar TII, this index was smaller in the HC group.

These two findings of change in the direction of midline shift and the extracerebral herniation index may suggest that DC results in a greater degree of postoperative brain deformation when compared with HC. We speculate that this change may be a result of the rapid cerebral decompression that occurs with DC and the theoretical risk of increased edema with greater cerebral expansion.[18] These features of DC may place the patient at a greater risk for venous congestion and potential infarction with hemorrhagic conversion along the boundaries of the cranial defect.

In addition, although the difference was not statistically significant, our analysis revealed a greater degree of postoperative parenchymal contusion enlargement with DC. This enlargement may reflect blossoming of the contusions secondary to unconstrained brain expansion, which may portend a worse neurological outcome as previously demonstrated by Flint et al.[5] By leaving the native bone plate in place during HC, there is a possibility that these effects may be avoided while still achieving the desired ICP control.

Study Limitations

The limitations of this study include the heterogene-

TABLE 9: Preoperative and postoperative radiographic results according to group*

	HC		DC	
Variable	Preop	Postop	Preop	Postop
mean MLS (mm)	11.0 ± 4.7	6.4 ± 4.4	9.4 ± 5.2	5.5 ± 4.6
cisterns				
effaced	9 (45)	1 (2)	8 (27)	4 (13)
compressed	8 (40)	2 (10)	12 (40)	4 (13)
present	3 (15)	17 (85)	10 (33)	22 (73)
uncal herniation	13 (65)	2 (10)	18 (60)	5 (17)
midbrain torsion	4 (20)	4 (20)	2 (7)	2 (7)
IVH	6 (30)	7 (35)	8 (27)	10 (33)
SAH	12 (60)	9 (45)	17 (57)	14 (47)
SDH	12 (60)	14 (70)	17(57)	15 (50)
EDH	2 (10)	1 (5)	3 (10)	2 (7)
mean Rotterdam score	4.8 ± 1.1	3.2 ± 1.0	4.4 ± 1.1	3.4 ± 1.3
mean VCE (ml)		77.5 ± 54.1		105.1 ± 65.1
mean hematoma volume (ml)	12.3 ± 27.3	21.9 ± 27.7	4.4 ± 6.0	22.5 ± 37.5
mean hematoma volume change (ml)		8.9 ± 17.2		14.7 ± 31.9

* All values are number of patients (%) unless otherwise indicated. None of the variables were statistically significant between groups. Abbreviation: VCE = volume of cerebral expansion.

TABLE 10: Computed tomography morphometric results*

CT Morphometric	HC Group	DC Group	p Value
max craniectomy or craniotomy diameter (mm)	124.8 ± 20.7	124.0 ± 10.8	NS
ratio†	0.79 ± 0.14	0.80 ± 0.10	NS
ECH magnitude (mm)	21.2 ± 9.5	26.0 ± 7.7	NS
ECH index‡	0.16 ± 0.05	0.21 ± 0.06	0.007
% lateral brain expansion§	107.4 ± 3.9	107.7 ± 6.2	NS

* ECH = extracerebral herniation; NS = not significant.
† ratio = max craniectomy or craniotomy diameter/max anteroposterior skull diameter.
‡ ECH index = ECH magnitude/max craniectomy or craniotomy diameter.
§ % lateral brain expansion = (postop horizontal brain diameter – immediate preop horizontal brain diameter) × 100.

ity of the patient population, its retrospective nonrandomized nature, limited size, and lack of long-term outcomes. The operative decision-making was not uniform as the type of decompression performed was at the discretion of the treating neurosurgeon. In the majority of patients, no preoperative ICP data are available because the patients were taken directly to the operating room after showing evidence of a clinical herniation syndrome or a mass lesion producing a significant midline shift. The measurement of metabolic parameters (such as microdialysis and cerebral oxygenation) was not used because the patients treated predated the use of these techniques at our institution. Finally, despite its routine use in neurooncology, the technique of assessing cerebral volume with the BrainLAB software has not yet been validated. Future studies will involve validation of this technique as well as long-term analysis of these patients, including neurological outcomes and assessment of adequate osteosynthesis.

This is the first study to evaluate the cerebral decompression techniques of HC and DC through a review of postoperative ICP control in the context of a TII. We have demonstrated that, in comparison with DC and in patients with comparable underlying pathology, the early ICP and survival goals are equivalent and acceptable in HC. These findings, in combination with the potential benefits of avoiding both the immediate morbidity of DC (such as seizures, protective headgear, and syndrome of the trephined) and the eventual cranioplasty, make HC an attractive option for cerebral decompression and one that warrants further study.

Conclusions

Our results suggest that HC may be widely applicable for cerebral decompression rather than just in borderline cases as previously suggested. Hinge craniotomy appears to be at least as good as DC in providing postoperative ICP control and results in equivalent early clinical outcomes. Long-term outcome analysis as well as a randomized, prospective study is still required.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Complications of decompressive craniectomy for traumatic brain injury

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Decompressive craniectomy is widely used to treat intracranial hypertension following traumatic brain injury (TBI). Two randomized trials are currently underway to further evaluate the effectiveness of decompressive craniectomy for TBI. Complications of this procedure have major ramifications on the risk-benefit balance in decision-making during evaluation of potential surgical candidates. To further evaluate the complications of decompressive craniectomy, a review of the literature was performed following a detailed search of PubMed between 1980 and 2009. The author restricted her study to literature pertaining to decompressive craniectomy for patients with TBI. An understanding of the pathophysiological events that accompany removal of a large piece of skull bone provides a foundation for understanding many of the complications associated with decompressive craniectomy. The author determined that decompressive craniectomy is not a simple, straightforward operation without adverse effects. Rather, numerous complications may arise, and they do so in a sequential fashion at specific time points following surgical decompression. Expansion of contusions, new subdural and epidural hematomas contralateral to the decompressed hemisphere, and external cerebral herniation typify the early perioperative complications of decompressive craniectomy for TBI. Within the 1st week following decompression, CSF circulation derangements manifest commonly as subdural hygromas. Paradoxical herniation following lumbar puncture in the setting of a large skull defect is a rare, potentially fatal complication that can be prevented and treated if recognized early. During the later phases of recovery, patients may develop a new cognitive, neurological, or psychological deficit termed syndrome of the trephined. In the longer term, a persistent vegetative state is the most devastating of outcomes of decompressive craniectomy. The risk of complications following decompressive craniectomy is weighed against the life-threatening circumstances under which this surgery is performed. Ongoing trials will define whether this balance supports surgical decompression as a first-line treatment for TBI. *(DOI: 10.3171/2009.4.FOCUS0965)*

Key Words • **traumatic brain injury** • **intracranial hypertension** • **surgical decompression** • **cranioplasty** • **complication** • **trephine**

Two randomized, multicenter prospective trials, RESCUEicp and DECRA, are currently in progress to determine whether decompressive craniectomy improves outcome in the management of TBI.[14,37] Complication rates of this procedure will be a major determinant of whether decompressive craniectomy proves superior to medical treatment for patients with intracranial hypertension after TBI. These procedures are performed for serious life-threatening intracranial hypertension, and mortality rates following decompressive craniectomy for severe TBI are high. During a 5-year experience with 170 consecutive decompressive hemicraniectomies for severe TBI at our institution, 50% of patients died.[82] Many of the complications of decompressive craniectomy arise from the normal pathophysiological changes that occur in ICP, CSF circulation, and CBF following removal of a large area of skull bone.

The rationale behind decompressive craniectomy is to convert an injury within a closed box, with a fixed volume and limited reserve, into an open system with increased capacity to accommodate mass.[64] After bone removal, there is an increase in brain compliance and a shift of the pressure volume curve to the right.[29,33] The RAP index, determined as a correlation between fluctuations in ICP with heart rate against the mean ICP averaged over repetitive time points, is a surrogate for cerebral compliance.[87] Improvements in RAP values as well as direct measurements using Spiegelberg compliance monitors have demonstrated improvements in cerebral

Abbreviations used in this paper: CBF = cerebral blood flow; EDH = epidural hematoma; GCS = Glasgow Coma Scale; ICP = intracranial pressure; RAP = pressure-volume compensatory reserve; SDH = subdural hematoma; TBI = traumatic brain injury.

TABLE 1: Literature summary of complications after decompressive craniectomy for TBI

Authors & Year	No. of Patients	Subdural Hygroma	Contusion/ Hematoma Progression	Intra-cranial Infection	New Contralat Extraaxial Hematoma	Hydro-cephalus	Bone Flap Re-sorption	Reoperation (excluding cranioplasty)	Comment
Aarabi et al., 2006	50	25 (50%)	8 (16%)	wound infection (1 [2%])		5 (10%)	6 (12%)		6 (12%) developed parenchymal lucency possibly related to ischemia; bone flap infection after cranioplasty (in 3)
Chibbaro and Tacconi, 2007	48		3 (6%)	1 (2%)		1 (2%)			
Flint et al., 2008	40		23 (58%)		11 (28%)				12/40 (30%) new or worsened IVH
Gaab et al., 1990	37 (18 unilat, 19 bilat)	13 (22%)				5 (13%) shunt-treated			
Guerra et al., 1999	57 (31 unilat, 26 bilat)	15 (26%)				8 (14%)			1 (2%) wound infection after cranioplasty
Huang et al., 2008	38	10 (26%)	2 (5%)	2 (5%)		3 (8%)		3 (8%)	
Jiang et al., 2005	241	38 (16%)		8 (3%)					incisional CSF fistula (3%); epilepsy (3%)
Polin et al., 1997	35		2 (6%) (1 required reop)	1 (3%)		10 (29%) shunt-treated	2 (6%)	1 (3%) reop for delayed expansion of a temporal contusion requiring lobectomy	1 CSF leak treated w/ lumbar drain
Yang et al., 2003	68	18 (26%)		4 (6%)		20 (29%)			
Yang et al., 2008	108				7 (6%) w/ reop	9 (8%) shunt-treated		19 (18%): contralat intracranial hematoma (7 [6%]), subdural effusion (3 [3%]), hydrocephalus (9 [8%])	

compliance following decompressive craniectomy.[2,87] As a rescue therapy, decompressive craniectomy has been shown to reliably abort progressive increases in ICP.[59,89] Decompressive craniectomy also brings about changes in CBF and CSF dynamics.[7,20,24,96,97] Cine MR imaging studies have demonstrated impaired CSF flow across the craniovertebral junction at the skull base, which improves following cranioplasty repair of the skull defect.[20] Decompressive craniectomy also induces a hyperemic response as evidenced by ^{99m}Tc SPECT, ^{31}P MR imaging, and ^{133}Xe CT studies demonstrating augmentation of CBF as well as cerebral metabolism.[96,101] Physiological monitoring using CBF, transcranial Doppler, and brain tissue oxygen probes has demonstrated increased CBF and brain tissue oxygen following decompressive craniectomy.[7,39,80] Cerebrovascular reactivity is also impaired for the first 24 hours, trending toward a return to intact autoregulation within the first 72 hours following decompressive craniectomy. Measurements of PRx, the correlation coefficient of ICP versus arterial blood pressure over repetitive time intervals, have shown increases indicative of loss of cerebrovascular reactivity after decompression.[89,94] Loss of autoregulation in the early period following decompressive craniectomy may derive from a state of maximal vasodilation and hyperemia following relief of prolonged severe compression.[4,71] These alterations in compliance, CBF and autoregulation, and CSF circulation are the basis for many of the complications that arise following decompressive craniectomy.

Overview and Risks for Complications

Complications following decompressive craniectomy occur at predictable time points following the surgery. They can often be anticipated and in many cases prevent-

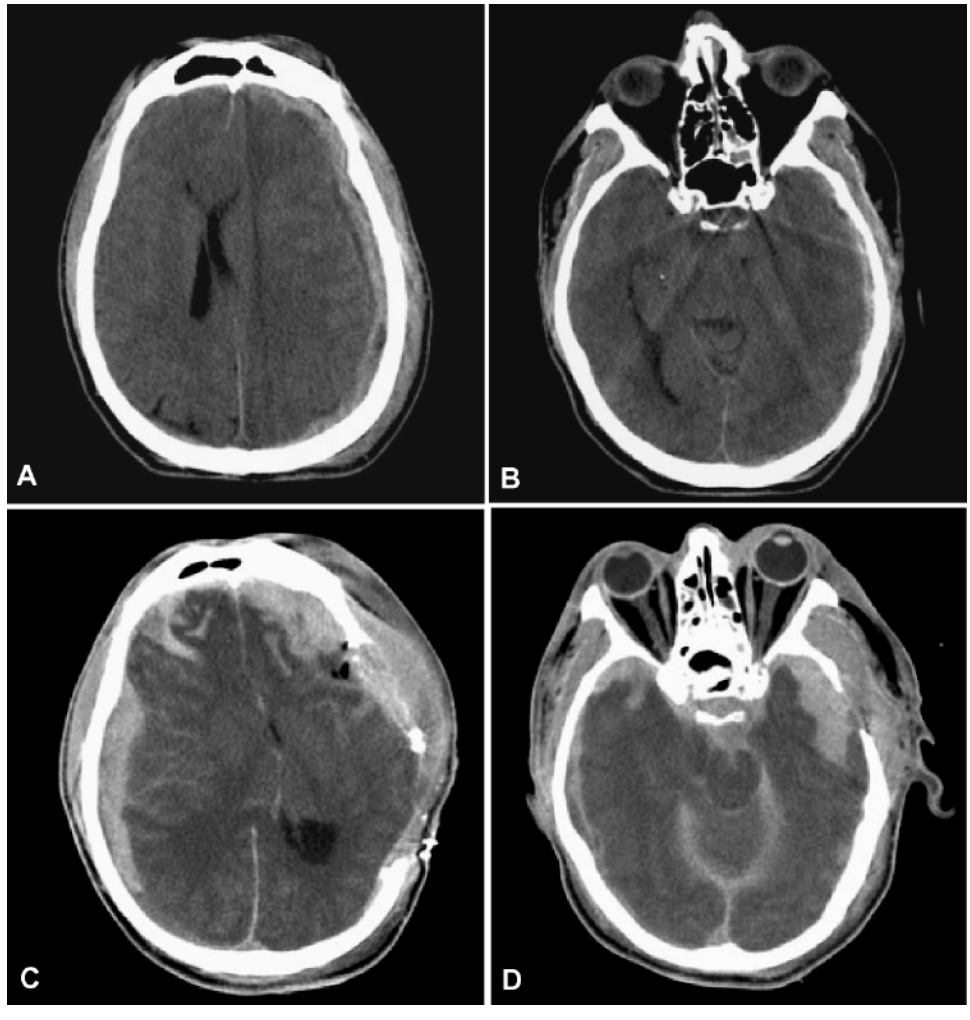


FIG. 1. Coagulopathy. Preoperative noncontrast CT scans of an SDH in a patient on a regimen of warfarin (A and B). Despite reversal of the coagulopathy before surgery, marked hemorrhagic blossoming occurred as evidenced by postoperative noncontrast images (C and D). Note in panel C the development of an extraaxial SDH contralateral to the decompressed hemisphere.

ed. Bleeding may be initiated during bone decompression and this, as well as development of new or expanded contusions, may present with malignant swelling or elevations in ICP in the first few hours or days after decompression. In a decompressed skull, even an ICP of 20 mm Hg may be of concern. External cerebral herniation frequently evolves over the first few days following decompressive surgery and can be problematic if the bone decompression is not sufficiently large to allow the brain to expand without constriction. In the early postoperative period (< 1 week), manifestations of deranged CSF flow become evident with evolution of subdural hygromas. Fevers are common during this time, and the need for lumbar puncture for CSF sampling needs to be weighed carefully against the risks of inducing a paradoxical herniation syndrome. Wound infections are common. In the late postoperative period, unique complications of decompressive craniectomy include those of syndrome of the trephined.

Patients with severe TBI are at increased risk of developing complications after decompressive craniectomy (Table 1).[99] In patients having sustained TBI, Yang et al.[99] reported that the frequency of complications was 62% for GCS Scores 3–5, 39% for GCS Scores 6–9, and 36% for GCS scores > 9. Older patients (> 60 years) also tended to have more complications, but the differences compared with younger patients were not statistically significant.[99] Patients taking aspirin, Plavix, and warfarin are at particularly high risk for severe or nonsurvivable complications following decompressive craniectomy (Fig. 1).

Complications

Perioperative Complications

Blossoming of Contusions. Hemorrhagic expansion of contusions is inherent in the injury process and has been demonstrated on serial CT scanning in patients with TBI.[10,58,79] Hemorrhagic contusion expansion was observed in 42% of 142 TBI patients with median GCS scores of 8,[58] 59% of 37 patients in a coma,[77] 48% of 104 children with TBI,[28] and 47% of 141 patients with TBI presenting with traumatic subarachnoid hemorrhage on their CT

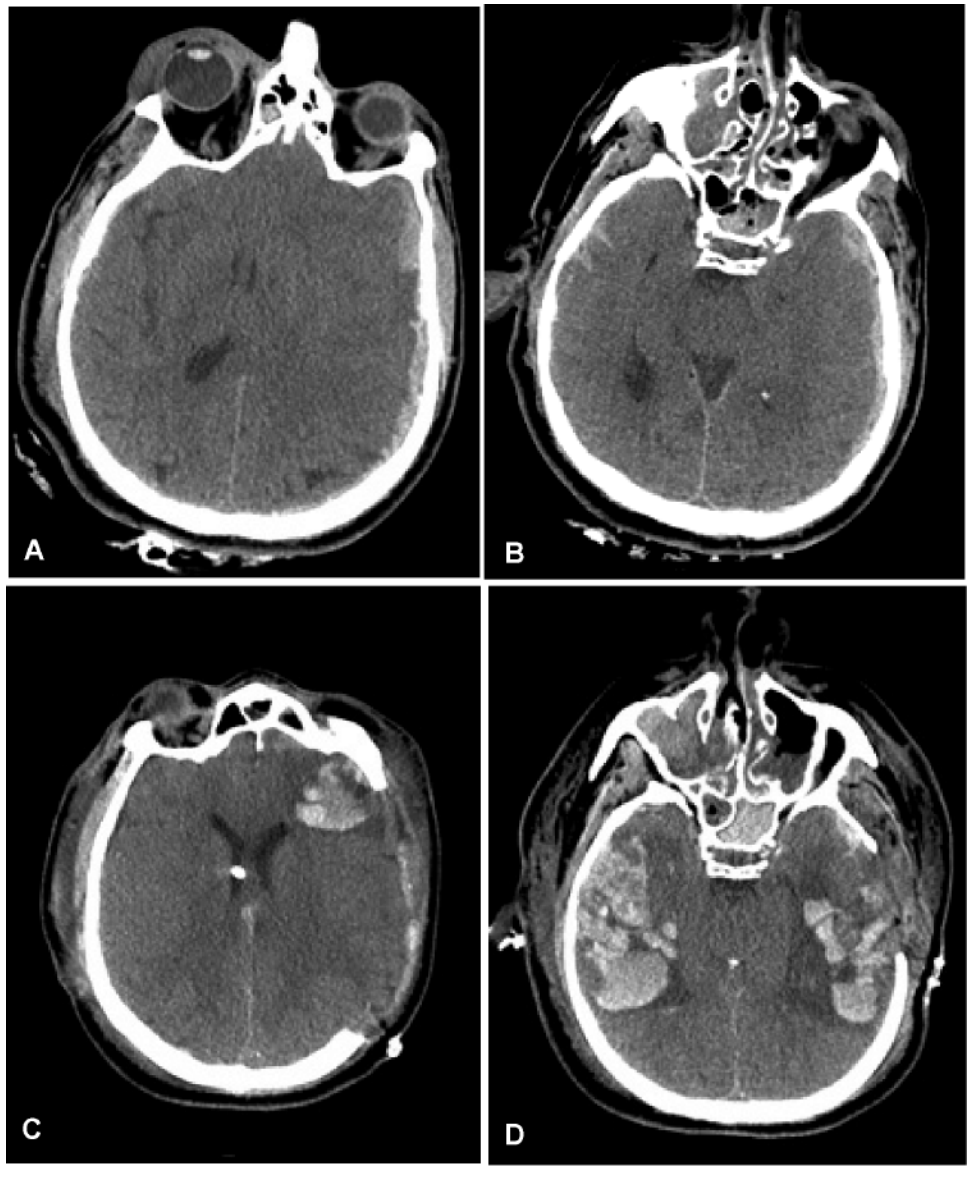


Fig. 2. Contusion blossoming. Preoperative (A and B) and postoperative (C and D) noncontrast CT images demonstrating massive temporal contusions following decompressive craniectomy. Note the relative absence of parenchymal lesion burden in the temporal lobes on the preoperative images.

scan.[12] Experimental animal models of controlled cortical impact have demonstrated that craniectomy, compared with craniotomy, reduces contusion volume by 40%.[102] In contrast, relief of the tamponade effect with bone removal in patients with severe TBI may facilitate growth and expansion of contusions following decompressive craniectomy.[23,49] In a CT imaging study of contusions pre– and post–decompressive craniectomy, Flint et al.[23] found a remarkably high incidence of new or expanded hemorrhagic contusions following decompressive surgery (Fig. 2). In that study, the authors defined nonhemorrhagic contusions as areas of hypodensity on CT scans with focal stippling of blood but without a hematoma that could be measured. By contrast, hemorrhagic contusions were defined as contusions associated with a measurable hematoma. Following decompressive craniectomy, new or expanded hemorrhagic contusions were observed in 23 (58%) of 40 patients. The evolving contusion was ipsilateral to the decompressed hemisphere in 82% of cases. Severity of the head injury as judged by the Rotterdam score[48] of the initial CT scan correlated with the risk of hemorrhagic contusion expansion. Patients with Rotterdam scores of 5 or 6 had an 80% chance of expansion of their hemorrhagic contusions following decompressive craniectomy. The frequency and the total volume increase of the hemorrhagic contusion correlated with the Rotterdam score. In addition, there was a 30% (12 of 40) incidence of new or worsened IVH and a 28% (11 of 40) incidence of new or worsened extraaxial subdural or epidural hemorrhage. Hemorrhagic contusion volume was observed to correlate with outcome. Using a cutoff of 20 ml determined from a receiver operator curve analysis, 8 (57%) of 14 patients

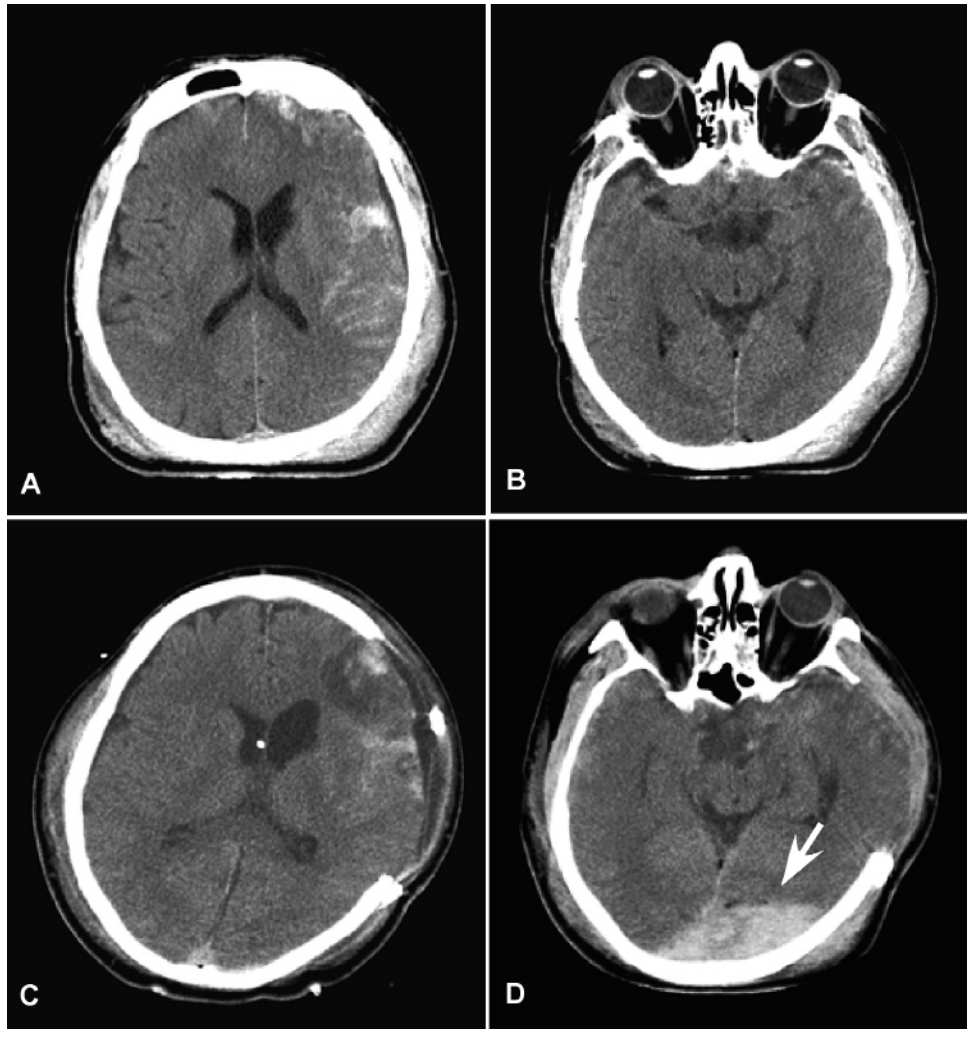
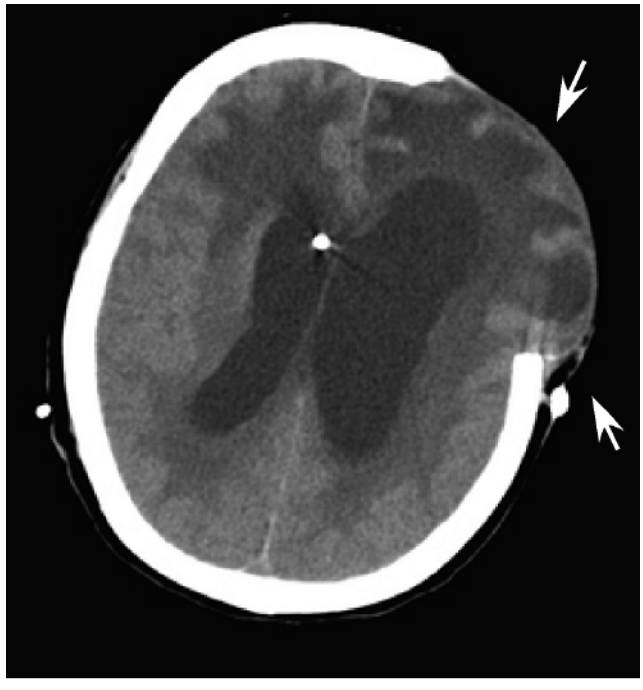


Fig. 3. Evolution of remote extraaxial hemorrhage. A and B: Preoperative noncontrast CT scans obtained in a patient with traumatic subarachnoid hemorrhage and a small SDH who underwent decompressive craniectomy. C and D: Postoperative noncontrast CT scans showing evolution of an occipital EDH *(arrow)*, remote from the site of surgery. The patient underwent an occipital craniotomy and evacuation of the EDH. At surgery a linear, nondisplaced skull fracture was found overlying the occipital EDH. A retrospective review of this patient's CT scan suggested that the fracture was parallel to the cuts of the CT image and the density of the occipital bone had masked detection of the fracture on the scout CT image.

with an expanded hemorrhagic contusion volume > 20 ml died within 6 months, whereas only 4 (15%) of 26 patients with contusion volumes < 20 ml ($p = 0.011$) died.[23]

Evolution of Contralateral Mass Lesion. Surgical craniectomy decompression for TBI may incite a new mass lesion, contralateral or remote to the decompressed hemisphere[13,52,63] (Fig. 3). Reduction in ICP after decompression likely plays an important role.[8,43,86] Piepmeier and Wagner,[63] however, have pointed out that if tamponade relief underlies contralateral bleeding, then one would expect delayed EDH lesions more frequently. High Rotterdam scores of initial CT scans have been observed to correlate with the development of contralateral extraaxial hemorrhages.[23] Su et al.[84] observed 14 new contralateral EDHs following surgical evacuation of acute SDHs. In 10 of these patients, the EDH manifested with intraoperative brain swelling and external cerebral herniation during the acute subdural evacuation, necessitating that the bone flap not be replaced. Yang et al.[99] reported a 7% incidence of new contralateral hematomas. Over half occurred within the first 24 hours after decompressive surgery. Detection of a fracture on CT scanning should increase awareness of this potential intraoperative or postoperative complication. In the study by Su et al.,[84] a fracture was found on CT scans in 10 (71%) of 14 patients with new contralateral EDHs. A skull fracture was identified at surgery in each of the 12 patients treated by reoperation for evacuation of the EDH. The EDH localized to the site of the fracture in all cases.[84]

Theoretically patients may be at higher risk for developing a contralateral EDH following decompressive craniectomy than following craniotomy. Decompressive surgery may relieve the tamponade effect on a contralateral bleeding site and predispose the patient to an EDH.[8,86] In general, the severity of the underlying TBI most strongly determines prognosis. Prompt awareness



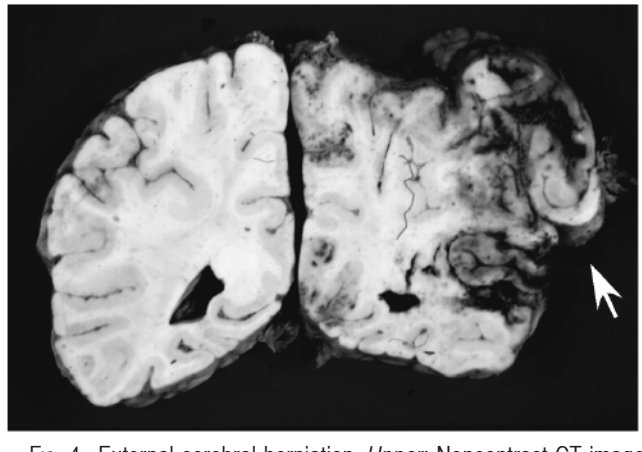


FIG. 4. External cerebral herniation. *Upper:* Noncontrast CT image demonstrating external cerebral herniation *(arrows)* through a small craniectomy. *Lower:* Photograph of a gross specimen obtained at autopsy in a different patient demonstrating external cerebral herniation *(arrow)* and venous infarction within the herniated brain tissue.

and evacuation of the delayed EDH may not impair recovery as long as the mass effect has not progressed to the stage of impairing brainstem function.[84]

External Cerebral Herniation. Expansion of the brain with external cerebral herniation through the craniectomy defect is often observed in the early period after decompression (Fig. 4). There is no consensus on how to measure external cerebral herniation. In the study by Yang et al.,[99] herniation through the craniectomy defect was measured at the middle of the cranial defect. Herniation was defined as brain tissue in the center of the bone defect > 1.5 cm above the plane where the outer table of the cranium would normally lie.[99] Using this measure, external cerebral herniation was observed in 28 (26%) of 108 decompressive craniectomies. In contrast, Flint et al.[23] measured extracranial cerebral herniation as the diameter of brain extending beyond a straight line drawn between the outer table edges of the craniectomy defect. In their series of decompressive craniectomies, the mean bone opening diameter was 13.9 ± 1.2 cm, and the mean diameter of external cerebral herniation was 2.1 ± 0.9 cm.[23]

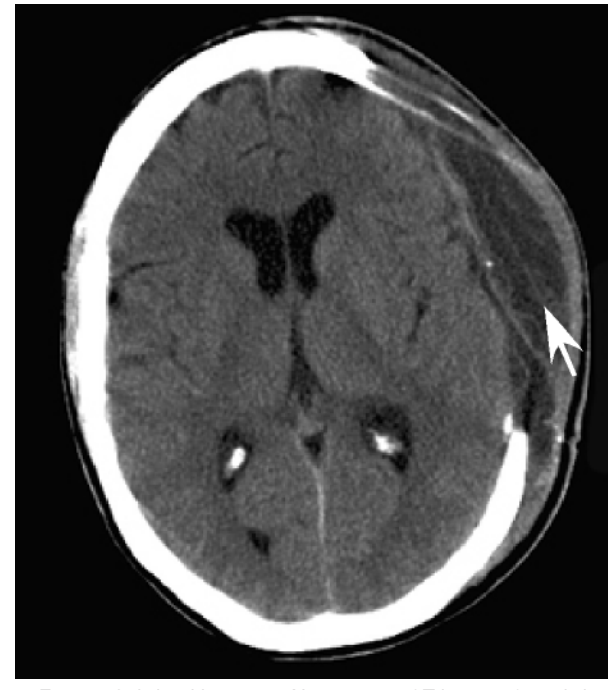


FIG. 5. Subdural hygroma. Noncontrast CT image of a subdural hygroma *(arrow)* following decompressive craniectomy.

Increased brain swelling is common in the 1st week following decompressive craniectomy. The brain swelling may correspond to hyperperfusion, as detected by CT perfusion imaging.[96] In addition, loss of resistance in brain tissue lacking a protective skull invokes a higher hydrostatic pressure gradient that may permit transcapillary leakage of edema fluid.[59] Brain edema following decompressive craniectomy has been modeled in animal studies of injury using Evans blue dye extravasation.[15,25,69] Cooper et al.[15] found a 7-fold increase in edema formation as determined by Evans blue dye extravasation following craniectomy. Evidence of increased brain edema following craniectomy has not been verified in humans.[70]

Potential adverse effects of external cerebral herniation include compression of cortical veins within the herniated segment of brain and subsequent venous infarction of the herniated tissue. Small bone openings can lead to a mushroom cap–like appearance to brain that has herniated through the open defect (Fig. 4).[59] Large bone decompression allows the brain to expand outward without constriction and minimizes the risk of venous ischemia. A technique of inserting "vascular cushions" adjacent to large draining veins at the craniectomy margin has been reported to reduce the risk of venous ischemia.[17]

Postoperative Complications Within 30 Days

Subdural Effusions or Hygromas. Decompressive

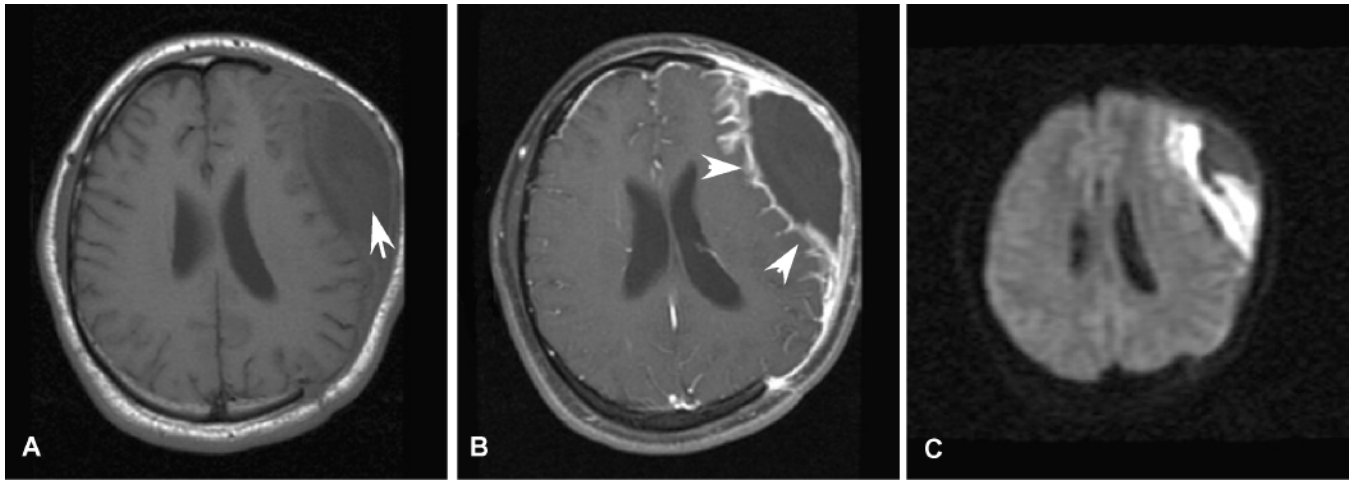


Fig. 6. Extraaxial empyema. A: A T1-weighted MR image showing a subdural collection *(arrow)*. B: A T1-weighted Gd-enhanced MR image demonstrating rim enhancement *(arrowheads)* of the collection. C: Diffusion weighted image with bright signal that is diagnostic of a subdural empyema.

craniectomy alters the dynamics of CSF circulation.[9,100] This may exacerbate the occurrence of subdural hygromas and hydrocephalus. Subdural hygromas develop early after decompressive surgery (Fig. 5). In the study by Aarabi et al.,[1] subdural hygromas developed in 25 (50%) of 50 patients after a mean of 8 days following decompressive craniectomy. Hygromas are generally ipsilateral to the skull defect with volumes ranging from 10 to 120 ml (mean 51 ml).[1] While most authors favor a mechanism of altered CSF dynamics to account for the occurrence of hygromas, others have suggested that increased cerebral perfusion pressure that accompanies decompressive craniectomy may play a role.[45]

Duraplasty at the time of decompression has been observed to lower the incidence of subdural effusions.[100] Wrapping the cranium after the peak time of cerebral swelling has also been suggested to help prevent effusions.[99] In the early experience of decompressive craniectomy, Guerra et al.[32] reported that 10 (67%) of 15 patients who developed hygromas responded to puncture treatment and only 5 (33%) required a ventriculo- or lumboperitoneal shunt. In this report, 8 (14%) of the 57 patients developed hydrocephalus. At our institution we avoid tapping into CSF hygromas and have found that most resolve spontaneously without need for intervention. Yang et al.[99] reported a 21% incidence of subdural hygromas in their series and noted that 20 of 23 effusions resolved spontaneously without neurological deficits. Aarabi et al.[1] have also shown that subdural hygromas following decompressive craniectomy regress spontaneously over weeks to months. In their series, no patient required decompression or shunt treatment of a hygroma collection.[1] Two of the 25 patients, however, went on to develop delayed hydrocephalus.

Paradoxical Herniation. Paradoxical herniation with compression of the brainstem and neurological deterioration may present in a delayed fashion after a lumbar puncture in patients with decompressive craniectomy.[93] Vilela[93] described a case of a patient with a decompressive craniectomy whose condition suddenly deteriorated 38 days after a lumbar puncture to rule out meningitis. The precipitous deterioration was attributed to upright positioning with mobilization of the patient as she started to participate in rehabilitation therapy. Mobilization into the upright position may have invoked a higher cranial-spinal pressure gradient and perhaps even opened up a preexisting small dural hole from the lumbar puncture site. Neurological deterioration after lumbar puncture or lumbar CSF drainage in the setting of large cranial defects has also been observed following decompressive craniectomy for stroke.[21,61] In the early period of recovery from traumatic injury following decompressive craniectomy, symptoms of paradoxical herniation may be masked and even mistaken for neurological damage from the trauma.

The concept that a negative pressure gradient between the cranial and spinal compartments, provoked by a spinal CSF leak, can precipitate downward herniation, even in the absence of raised ICP, has been carefully documented by many groups.[5,6,42,72,73] With a large skull defect, a lumbar puncture exacerbates the negative pressure gradient between the atmosphere and the cranium.[93] Based on this, we have adopted a high threshold for performing lumbar puncture in patients with a decompressive craniectomy. Treatment for symptomatic herniation following lumbar puncture with a skull defect is the antithesis of that used for herniation in the setting of raised ICP, hence the term paradoxical herniation. Patients with paradoxical herniation should be treated with intravenous fluids, Trendelenburg positioning, and clamping of CSF drainage; hyperosmolar therapy should be discontinued, and early attention given to insertion of a blood patch.[61,76,93] Cranioplasty may act to reduce the negative pressure gradient and should also be considered in the management of paradoxical herniation.[76,93]

Delayed Complications After 1 Month

Wound Healing and Infection. There are several factors associated with decompressive craniectomy that

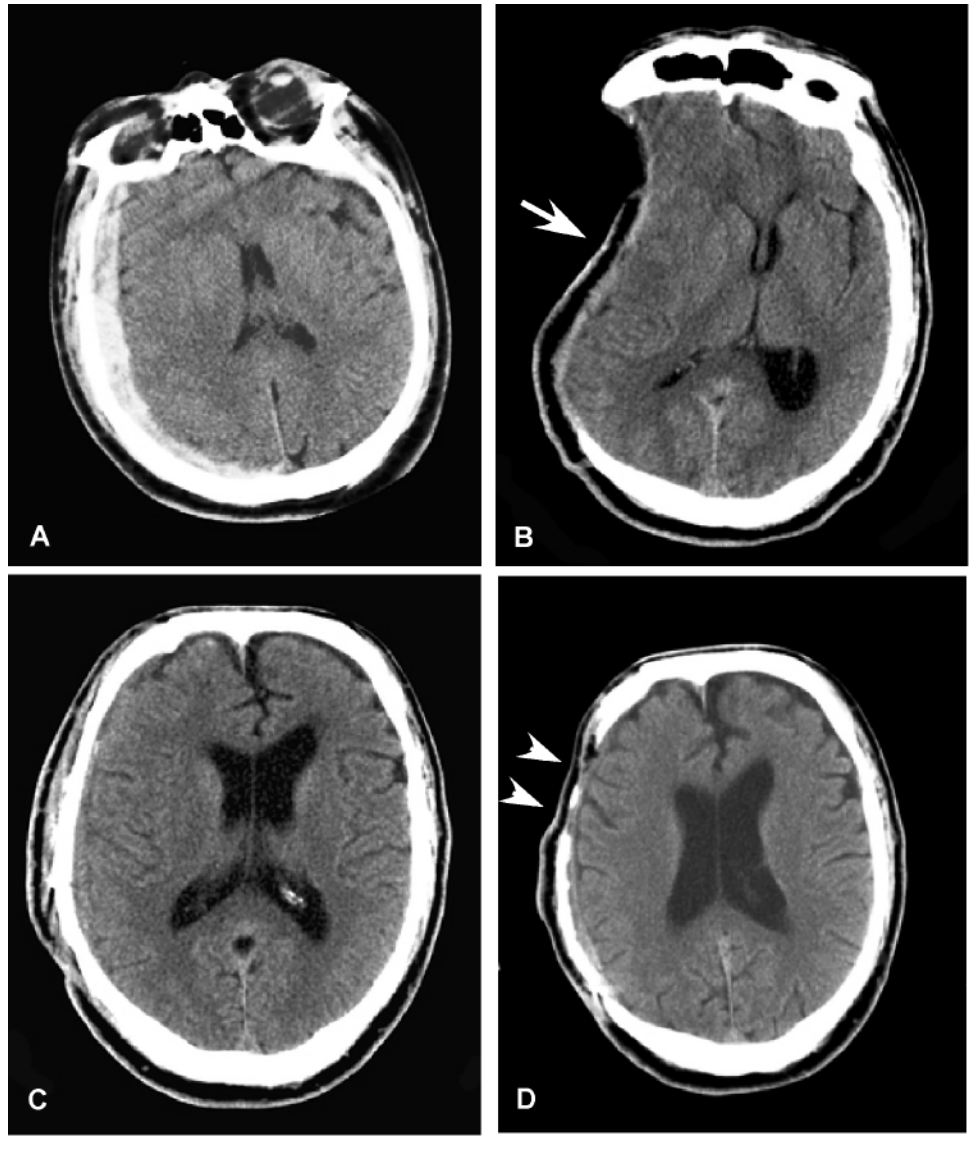


Fig. 7. Sinking scalp flap. A: Preoperative noncontrast CT scan demonstrating a right acute SDH, which was evacuated and the bone left off as a prophylactic measure against subsequent intracranial hypertension. B: A CT scan without contrast obtained several months later demonstrates a sunken parenchymal contour *(arrow)* with ~ 1.5 cm of midline shift. C: After autologous cranioplasty repair of the skull defect, a CT scan showed restoration of both the sunken brain contour and the midline shift. D: Within 2 months following cranioplasty, noncontrast CT imaging began to show signs of partial resorption *(arrowheads)* of the replaced bone flap.

should lead one to expect a higher rate of infection than with standard craniotomy for general neurosurgical procedures. The incision varies but the typical, large, reverse question mark incision with a long scalp pedicle on a comparatively small base predisposes to wound breakdown along the parietal and posterior temporal limbs farthest along the flap. Bone removal is needed low in the temporal fossa to decompress the basal cisterns. To expose the scalp and temporalis muscle down to the level of the zygoma, the incision is carried to 1 cm below the zygoma anterior to the tragus. The urgency to decompress may not facilitate careful dissection and preservation of the superficial temporal artery. Sacrifice of the artery may impair perfusion of the scalp pedicle and negatively impact wound healing. The dura is not closed primarily. Duraplasty using a dura substitute is associated with an increased risk of infection.[50] If the dura is left open without duraplasty, a foreign synthetic material should be laid over the brain surface to prevent adherence of the scalp to the underlying brain. With the large bone flaps used for decompression, opening into the frontal sinus or air cells low in the middle fossa risks delayed meningitis and subdural empyema, especially if a duraplasty is not performed. Last, surgical fields in trauma surgery are often clean contaminated. The bone, soft tissues, and brain are easily contaminated by penetrating injury.

In the intensive care unit, fevers are common. We avoid tapping subdural collections and prefer to use contrast enhancement on CT and MR imaging together with diffusion weighted MR imaging to diagnose infected

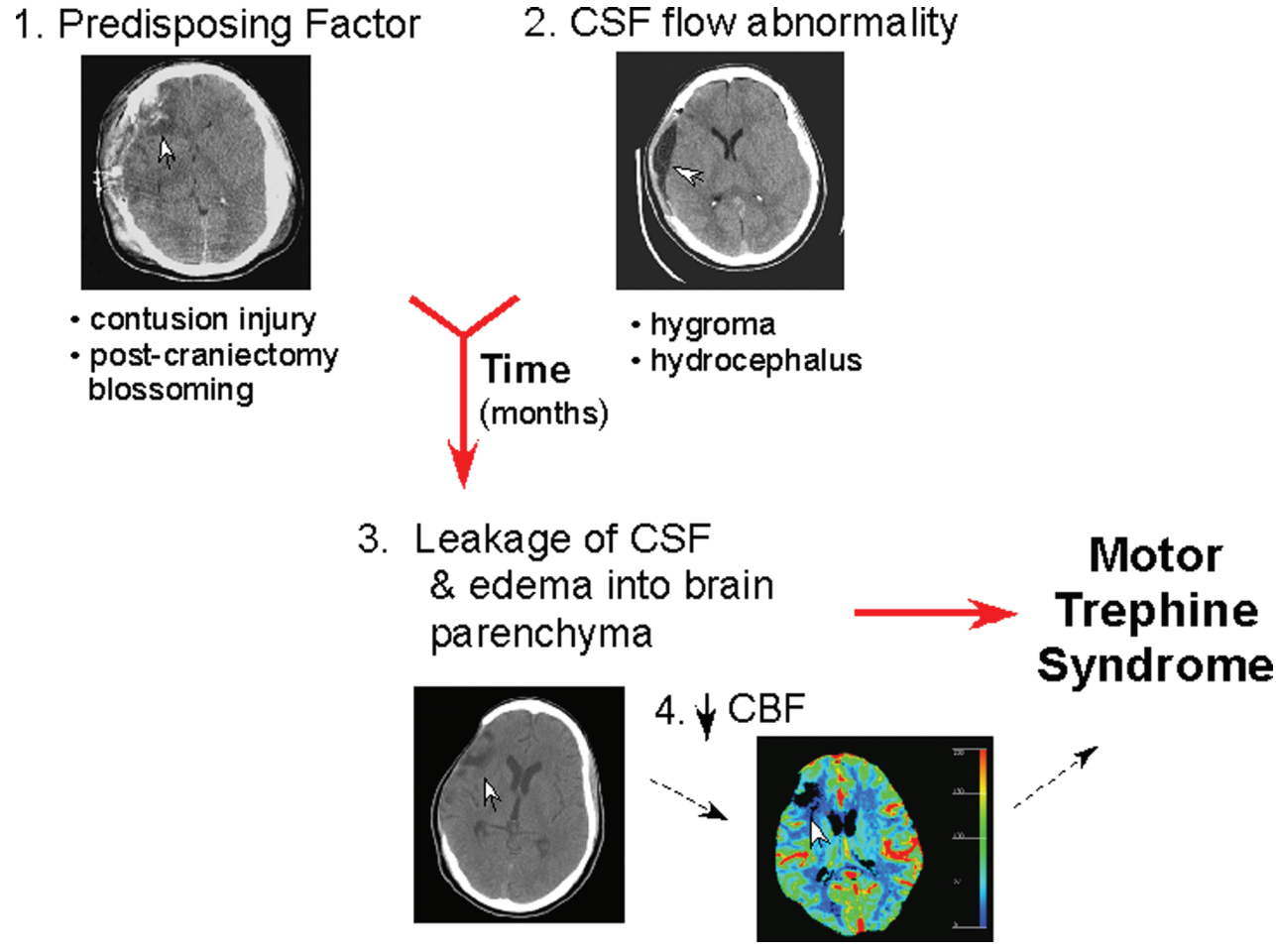


Fig. 8. Mechanistic schematic for motor trephine syndrome. The presence of prior intraparenchymal contusions as well as CSF flow abnormalities are requisite factors; neither alone is sufficient to induce the syndrome. 1) Contusions may be caused by the injury itself or blossoming following decompression *(arrow)*. 2) The CSF hygromas *(arrow)* are present on early postoperative CT imaging in 90% of patients who develop motor trephine syndrome. Over time, dysfunction of CSF flow persists and reserve mechanisms decompensate. Long intervals to cranioplasty repair increase the likelihood of developing the syndrome. 3) The CSF and edema transgress into brain parenchyma underlying the skull defect, manifested by areas of hypoattenuation on CT imaging *(arrow)*. Previous intraparenchymal contusion injury, decreased resistance to fluid accumulation in the absence of bone enclosure, and long intervals to cranioplasty facilitate transgression of CSF and edema into brain parenchyma underlying the craniectomy defect. 4) The CBF flow by CT perfusion imaging is diminished in areas of hypoattenuation *(arrow)* corresponding to CSF and edema leakage. Transgression of CSF and edema, acting through or in concert with diminished CBF leads to motor trephine syndrome. Reprinted from Stiver SI et al: **Acta Neurosurg Suppl 102:**273–277, 2008.

subdural collections (Fig. 6). In many cases, a superficial wound infection aids in the diagnosis of the underlying intracranial problem.

Decompressive craniectomy necessitates a second cranioplasty surgery to replace the bone. Repeat surgery inherently increases risks of infection. In our patient population, we and others have observed high rates of infection following early cranioplasty repair and prefer to wait a minimum of 3 months and generally longer, before replacing the bone.[90] Storage of the bone for prolonged periods in a freezer also increases the risk of infection.[60,67,90] To minimize risks of infection through opening of communications to the frontal sinus, we do not remove scar tissue from the area of the frontal sinus during exposure of the craniectomy margins for replacement of the bone flap.

Adhesions between the brain and scalp may be a source of neurological injury during subsequent exposure of the brain for the cranioplasty. If a duraplasty is not performed, it is important that the Gelfilm (Upjohn) or another dural substitute cover all aspects of exposed brain to prevent adhesion between the brain surface and the overlying scalp. The disadvantage of Gelfilm is that it appears to take several months for the pseudodura layer to form and completely seal a barrier layer overlying the brain. Duraplasty up front may enable an earlier cranioplasty repair. A recent report has advocated the use of DuraGen (Integra LifeSciences Corp.) that is laid over the brain without suture closure. Over time the Duragen is replaced by ingrowth of connective tissue that has properties similar to dura.[34]

Decompressive craniectomy is not an esthetic operation. The patient's scalp contours are disfigured for a period of months. In many patients, asymmetric cerebral

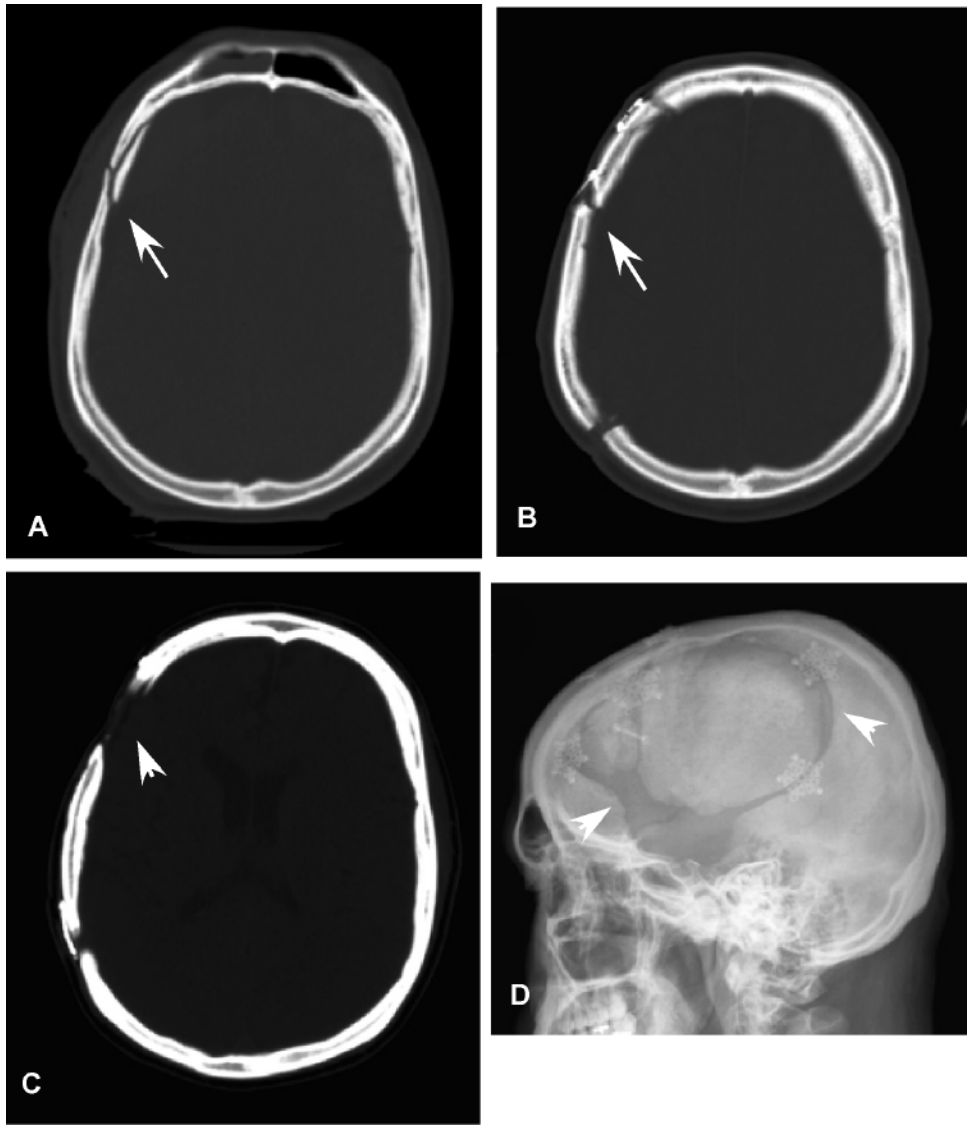


FIG. 9. Bone resorption. A: Noncontrast CT image of a frontal skull fracture *(arrow)* in a patient who underwent decompressive craniectomy. B: At 4 months following the decompressive surgery, an autologous cranioplasty repair was performed with plate reconstruction of his skull fracture *(arrow)*. C and D: Noncontrast CT scan (C) and lateral plain skull film (D) obtained 2 months after the cranioplasty demonstrating extensive bone resorption of the fractured segment *(arrowheads)*. The patient underwent a second cranioplasty with a synthetic bone flap.

swelling in the early phases is followed later by markedly sunken scalp contours ipsilateral to the bone decompression (Fig. 7). Cerebral atrophy secondary to brain injury can exacerbate the degree of concavity. The temporalis muscle is detached and atrophies, which may limit the excursion of mouth opening and cause difficulty chewing. Masticatory function, however, does not seem to be significantly impaired. In 1 study, even after the temporalis muscle was excised, only 2 (25%) of 8 patients experienced lateral deviation of the mandible on opening their mouths or had difficulties with eating.[62]

Hydrocephalus. Hydrocephalus and syndrome of the trephined are the most frequent complications of decompressive craniectomy beyond 1 month. Decompressive craniectomy has been identified as a risk factor for CSF alterations and development of posttraumatic hydrocepalus.[53] Hydrocephalus has been associated with poorer outcome following TBI.[51] Relatively few patients require ventriculoperitoneal shunt treatment before the bone flap has been replaced. Ventriculoperitoneal shunt treatment of hydrocephalus in the setting of a large cranial defect may also risk neurological deterioration consistent with a paradoxical herniation phenomenon.[47]

Syndrome of the Trephined. Syndrome of the trephined is a frequent, delayed complication of decompressive craniectomy.[30] Common symptoms include headaches, dizziness, irritability, concentration difficulty, memory problems, and mood disturbances, which typically arise weeks to months following decompressive craniectomy. The diagnosis is often overlooked, as many of these symptoms are also common sequelae to postconcussion and

posttraumatic stress syndromes that accompany TBI.

Less commonly, syndrome of the trephined can present with delayed onset of new neurological deficits. We have observed a small cohort of patients who developed motor weakness of the contralateral hand and upper extremity months after their decompressive surgery.[82] We termed this motor trephine syndrome in reference to Grant's initial description of syndrome of the trephined.[81,82] These motor deficits evolve in an insidious and progressive manner in a limb that was otherwise normal and not affected by the injury. From a series of 170 consecutive patients with TBI who were treated with decompressive craniectomy, we studied 38 patients with long-term follow-up after cranioplasty repair.[82] We found 10 patients (26%) with motor trephine syndrome who developed delayed upper-extremity weakness a mean of 5 months following decompressive craniectomy. All experienced marked, rapid improvement of their weakness within a few days of cranioplasty repair with subsequent full motor recovery.

The pathophysiological mechanisms underlying syndrome of the trephined have been a subject of debated theories.[19] Changes in atmospheric pressure,[83] altered CSF circulation,[20,24] and changes in CBF[68,85] have all been proposed to explain the pathophysiology underlying the syndrome. In early studies, a sunken scalp was noted in many patients with syndrome of the trephined (Fig. 7).[27,98] Recently we discovered a high incidence of CT scanning abnormalities in patients who developed motor trephine syndrome.[82] Patients who developed motor trephine syndrome were significantly more likely to have demonstrated CSF subdural hygromas as well as frontal and temporal contusions on their early postoperative CT scans. At later time points, CT scans demonstrated areas of hypodensity consistent with evolving interstitial edema in the brain parenchyma.[22] This form of extracellular edema was attributed to transependymal flow and accumulation of water and CSF in previously contused areas of parenchyma underlying the skull defect. The CT perfusion imaging studies in 2 patients demonstrated reduced CBF in these areas of edematous change. Rapid improvements in CBF and resolution of these hypodense areas on CT occurred within days of cranioplasty repair, simultaneous with dramatic recovery of motor function.

We have observed that motor trephine syndrome develops in the setting of decompressive craniectomy as a result of 2 predisposing requisite factors (Fig. 8).[81,82] First, a contusion or other parenchymal injury imparts a lower threshold for fluid shifts into brain tissue. In the absence of an overlying skull, reduced resistance promotes fluid shifts into areas underlying the skull defect. Second, decompressive craniectomy impairs normal CSF circulation. Over time, impairments of CSF flow, decompensate beyond the phases of subdural hygroma collections and hydrocephalus.[1,19,20] Decompensated CSF flow manifests at late time points with leakage of CSF and edema fluid into vulnerable areas of injured parenchyma underlying the skull defect. This process is akin to the development of transependymal edema as a late response in normal-pressure hydrocephalus.[40] Microcirculatory compromise ensues within the edematous parenchyma, thereby impairing CBF. The combination of decreased cerebral perfusion and interstitial edema initiates a delayed, slowly progressive motor deficit. Cranioplasty repair quickly normalizes interstitial pressure, CSF flow, transependymal fluid shifts, and CBF leading to rapid recovery of motor function.

Patients with decompressive craniectomy should have serial neurological assessments for syndrome of the trephined and complaints of weakness, particularly in activities such as writing and buttoning, which involve the small muscles of the hand. Motor testing of the contralateral upper extremity should focus attention on the grip and metacarpal extensor strength. The motor trephine syndrome deficits are reversible, and physiotherapy should be involved to prevent development of contractures that could impair long-term function. Early cranioplasty may be considered, noting that the risks of infection are high with early replacement of the autologous bone flap.[98,99] However, Liang et al.[46] have recently shown that titanium mesh cranioplasty can be performed as early as 5–8 weeks after decompressive craniectomy without untoward infectious complications.

Bone Resorption. In decompressive craniectomy, bone resorption of free bone flaps is common and may approach an incidence as high as 50% in long-term follow-up (Figs. 7 and 9).[31,38,66] Skull fractures identified at the time of the original decompression should raise concern for possible bone resorption following cranioplasty (Fig. 9). Multiple fragments should lead to consideration of an upfront synthetic cranioplasty repair. During cranioplasty repair, tight approximation of the free flap to a lengthy surface of the native craniectomy margin reduces bone resorption. In our experience, rigid plate and screw fixation that permits minimal movement of the flap also appears to be important. A modified craniectomy in which the bone flap is not removed but rather is hinged has been proposed.[74] In 25 patients, hinge craniotomy was observed to provide adequate decompression of ICP with only 1 infection, 1 cranial deformity, no complications of bone resorption, and no cases of syndrome of the trephined.[74] Osteoclast inhibitors may also be used to prevent bone resorption in the future.[92]

Persistent Vegetative State. Decompressive craniectomy is very effective in ameliorating raised ICP as a life-saving measure. While decompressive craniectomy reduces mortality, it may fail to rescue neurological function from devastating injury incurred by either the primary impact or secondary damage that evolves during the early resuscitation period. Risks of survival with an outcome of a persistent vegetative state after decompressive craniectomy have been reported to range upwards of 15 to 20% in many series (Table 2). Concerns have been raised that decompressive craniectomy facilitates survival, in a persistent vegetative or minimally conscious state, for patients who would otherwise have died of cerebral herniation from raised ICP.[88] Preoperative GCS scores < 6, brainstem dysfunction, older age, and longer time to decompression have been reported to be associated with a higher risk of persistent vegetative outcome, but further study is needed to precisely delineate the role of these

TABLE 2: Literature summary of outcome following decompressive craniectomy for TBI*

Authors & Year	No. of Patients	Indication†	Follow-Up Time	No. of Patients (%)			
				Favorable	Severe Disability	Persistent Vegetative	Mortality
Aarabi et al., 2006	50	primary (20%) & secondary (80%)	3 mos	20 (40)	9 (18)	7 (14)	14 (28)
Albanese et al., 2003	40	primary (68%) & secondary (32%) DC	12 mos	10 (25)	13 (33)	included w/ severe disability	17 (43)
Chibbaro & Tacconi, 2007	48	secondary DC	mean 14 mos (range 6–25 mos)	25 (55)	6 (12)	9 (20)	6 (12)
De Luca et al., 2000	22	primary & secondary DC	not specified	9 (41)	4 (18)	5 (23)	4 (18)
Gaab et al., 1990	37	secondary DC	12 mos	14 (38)	12 (32)	3 (8)	5 (13)
Guerra et al., 1999	57	primary (68%) & secondary (32%) DC	12 mos	33 (58)	6 (11)	5 (9)	11 (19)
Howard et al., 2008	40	primary (60%) & secondary (40%) DC	3–26 mos (mean 11 mos)	12/18 survivors (67%), 12/40 (30%) overall	6/40 (15)	included w/ severe disability	22/40 (55)
Huang et al., 2008	38	primary DC	>6 mos	29 (76)	2 (5)	2 (5)	5 (13)
Jiang et al., 2005	241	primary DC	6 mos	96 (40)	71 (30)	9 (4)	63 (26)
Kunze et al., 1998	28	secondary DC	12 mos	15 (56)	5 (18)	4 (14)	3 (11)
Meier et al., 2006	117	primary (63%) & secondary (37%)	not specified	30 (26)	24 (20)	16 (14)	47 (40)
Morgalla et al., 2008	33	primary (27%) & secondary (73%) DC	3 yrs	13 (40)	8 (24)	7(20)	7 (20)
Munch et al., 2000	49	primary (63%) & secondary (37%) DC	6 mos	20%	33%	14%	33%
Olivecrona et al., 2007	21	secondary DC	not specified	15 (71)	2 (10)	1 (5)	3 (14)
Polin et al., 1997	35	secondary bifrontal DC, pediatric	at discharge (mean 63 days, range 10–220 days)	13 (37)	11 (31)	3 (9)	8 (23)
Schneider et al., 2002	62	secondary DC	6 mos	18 (29)	30 (48)	0 (0)	14 (23)
Skoglund et al., 2006	19	primary (11%) & secondary (89%) DC	min 12 mos (range 1–6 yrs)	13 (68)	3 (16)	1 (5)	2 (11)
Timofeev et al., 2006	49	secondary DC	6 mos	30 (61)	10 (21)	0 (0)	9 (18)
Whitfield et al., 2001	26	secondary DC	>6 mos (mean 10 mos)	18 (69)	2 (8)	0 (0)	6 (23)

* DC = decompressive craniectomy.

† Primary decompressive craniectomies were performed for up-front surgical decompression. Secondary delayed decompressive craniectomies were performed for raised ICP refractory to medical management.

factors in the risk-benefit analysis guiding operative decisions.[3,91]

Complication Avoidance

In treating patients who have sustained TBI, the neurosurgeon needs to carefully judge those who will benefit from immediate decompressive craniectomy and those who are best treated by initial monitoring and medical treatment of intracranial hypertension. Careful patient selection for decompressive craniectomy is needed to guard against a heroic intervention that increases survival at the expense of a persistent vegetative outcome. At surgery, a wide bone decompression should be performed and the dura opened. To minimize risk of wound dehiscence and infection, care should be made to preserve the superficial temporal artery and to ensure a scalp flap with a wide base-to-pedicle ratio. In addition, support beneath the myocutaneous scalp flap as it is reflected forward prevents acute angulation and impaired perfusion of the scalp pedicle. The frontal bone decompression should avoid opening into the frontal sinus. Augmentation of the craniectomy with a duraplasty has been suggested as a mechanism to prevent or limit external cerebral herniation.[99] Others have devised vascular cushions tunneled along the vessels at the bone edge to protect the veins from obstructive pressure.[16,17] The dura should be opened in a manner such that large veins at the bone edge are not situated at the apex of the dural slits.[17] Techniques of a lat-

tice duraplasty have also been suggested to limit external cerebral herniation.[55]

The presence of a skull fracture contralateral to the craniectomy should prompt consideration for an immediate or very early postoperative CT scan to rule out development of a mass lesion underlying the fracture.[84] If possible, tapping and shunt treatment of CSF subdural hygromas should be avoided. They almost always resolve over time on their own. Lumbar puncture carries a risk of paradoxical herniation at the time of puncture and even months later.

Hydrocephalus is a delayed complication of decompressive craniectomy. Few decompressed skulls require shunt treatment. Ventriculoperitoneal shunt treatment in patients with a skull defect can also risk paradoxical herniation. In many cases cranioplasty repair of the skull defect may normalize the impaired CSF flow and obviate the need for a shunt. The patient's neurological examination should be followed closely with particular attention for signs and symptoms of syndrome of the trephined.

Conclusions

Decompressive craniectomy is viewed by many as a simple surgical procedure, yet complications may be as common as those associated with complex skull base surgery. High mortality rates relate primarily to the severity of the trauma for which the operation is performed. Factors important in patient selection need to be further studied and refined to ensure the highest chances of favorable outcome. Many complications of decompressive craniectomy are inherent in the pathophysiological processes that ensue following removal of a large portion of skull bone. Other complications, including high rates of infection, derive from the technical aspects of the surgery including large scalp incisions, need for bone storage, and the challenges of sterility accompanying the nature of trauma surgery.

Different types of complications occur in discrete temporal windows. Changes in interstitial pressures and brain compliance, CSF circulation, CBF, and cerebral vasoreactivity and autoregulation play a role in early postoperative complications of contusion blossoming, external cerebral herniation, and formation of contralateral extraaxial hematomas. During the first weeks following decompression, subdural hygromas arise secondary to alterations in brain compliance and CSF circulation. Rare paradoxical herniation syndromes highlight the abnormal pressure dynamics across the cranial-spinal compartments following decompression.

In the late phases following decompression, syndrome of the trephined may lead to cognitive, psychological, and neurological deficits. Patients may develop a motor trephine deficit, typically a new, delayed-onset motor deficit of the contralateral upper extremity. We postulate that these motor trephine deficits arise from development of interstitial edema in areas of contused and injured brain underlying the skull defect. Motor trephine syndrome deficits are rapidly and completely reversed following cranioplasty repair. Further studies are needed to advance our understanding of the pathophysiological processes following decompressive craniectomy to obviate complications and provide patients with TBI with the best possible outcomes from this surgery.

Disclaimer

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Dynamics of subdural hygroma following decompressive craniectomy: a comparative study

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Object. This retrospective comparative cohort study was aimed at discovering the risk factors associated with subdural hygroma (SDG) following decompressive craniectomy (DC) to relieve intracranial hypertension in severe head injury.

Methods. Sixty-eight of 104 patients who had undergone DC during a 48-month period and survived > 30 days were eligible for this study. To assess the dynamics of subdural fluid collections, the authors compared CT scanning data from and the characteristics of 39 patients who had SDGs with the data in 29 patients who did not have hygromas. Variables significant in the appearance, evolution, and resolution of this complication were analyzed in a 36-week longitudinal study.

Results. The earliest imaging evidence of SDG was seen during the 1st week after DC. The SDG volume peaked between Weeks 3 and 4 post-DC and was gradually resolved by the 17th week. Among the mechanisms of injury, motor vehicle accidents were most often linked to the development of an SDG after DC ($p < 0.0007$), and falls were least often associated ($p < 0.005$). Moreover, patients with diffuse brain injury were more prone to this complication ($p < 0.0299$) than those with an evacuated mass ($p < 0.0001$). There were no statistically significant differences between patients with and without hygromas in terms of age, sex, Glasgow Coma Scale score, intraventricular and subarachnoid hemorrhage, levels of intracranial pressure and cerebral perfusion pressure, timing of decompression, and the need for CSF diversion. More than 90% of the SDGs were ipsilateral to the side of the craniectomy, and 3 (8%) of 39 SDGs showed evidence of internal bleeding at ~ 8 weeks postinjury. Surgical evacuation was needed in 4 patients with SDGs.

Conclusions. High dynamic accidents and patients with diffuse injury were more prone to SDGs. Close to 8% of SDGs converted themselves into subdural hematomas at ~ 2 months postinjury. Although SDGs developed in 39 (~ 60%) of 68 post-DC patients, surgical evacuation was needed in only 4. *(DOI: 10.3171/2009.3.FOCUS0947)*

Key Words • **decompressive craniectomy** • **subdural hygroma** • **head injury** • **intracranial pressure**

Autopsy verification of subdural fluid collections following severe head injury was first documented in 1733 by Professor Thomas Schwencke.[20] In 1894 Mayo cared for a 12-year-old boy suffering from headache, dysphasia, and right hemiparesis following trauma.[15] Trephination of the skull on the left side revealed a liquid similar to CSF, whose evacuation relieved the symptoms and led to functional recovery.[9,15] In 1924 Naffziger[49] discovered a tear in the arachnoid membrane, permitting egress of the CSF into the subdural space, which would then become a mass. In 1945 Walter Dandy[16,17] coined the term "subdural hygroma" for a benign collection of CSF in the subdural space. In the 1980s, after the introduction of CT, SDGs became the subject of intense imaging investigations.[4,6,15,22,28,33,64,65] Detailed electron microscopy studies of the arachnoid-dura interface by Haines et al.[25] and Schachenmayr and Friede[63] combined with documentation of CSF collections in the subdural space on contrast cisternography have helped us to better understand the pathogenesis of SDGs.[27,46] The inflammatory response in the SDG cavity, as indicated by cytokine activation, results in neomembrane formation rich in porous and fragile capillaries. The rupture of these capillaries, together with stress-induced tearing of

Abbreviations used in this paper: ATP = adenosine-5′-triphosphate; CPP = cerebral perfusion pressure; DC = decompressive craniectomy; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; MVA = motor vehicle accident; SDG = subdural hygroma; SDH = subdural hematoma.

TABLE 1: Summary of characteristics in 68 patients with or without SDG following DC for severe head injury*

Variable	No. (%)			p Value
	Total	w/ SDG	w/o SDG	
no. of patients	68	39	29	
male sex	50 (73.5)	27 (69.2)	23 (79)	NS
mean age (yrs)	27.8	24.9	29	NS
mechanism of injury				
MVA	45 (66)	31 (79.5)	14 (48.3)	0.0007
fall	13 (19)	3 (7.7)	10 (34.5)	0.005
assault	7 (10.3)	3 (7.7)	4 (13.8)	NS
other	3 (4.4)	2 (5.1)	1 (3.4)	NS
clinical characteristics				
mean admission GCS score	6.8	6.8	6.9	NS
mean admission GCS motor score	4.1	4.2	4	NS
patients w/APR	21	11	10	NS
mean injury severity score	30.5	31.4	29	NS
injury severity (no. of patients w/)				
>5-mm shift on CT	29 (42.6)	14 (35.9)	15 (51.7)	0.1918
compressed or obliterated BCs	53 (77.9)	33 (84.6)	20 (69)	0.1253
intraventricular hematoma	21 (30.9)	13 (33.3)	8 (27.6)	0.6108
subarachnoid hemorrhage	45 (66.2)	24 (61.5)	21 (72.4)	0.3458
diffuse injury	45 (66.2)	30 (76.9)	15 (51.7)	0.0299
evacuated mass	23 (33.8)	9 (23.1)	24 (82.8)	0.0001
treatment				
patients w/ early surgery†	31 (45.6)	16 (41)	15 (52)	0.3810
patients w/ delayed surgery‡	37 (54.4)	23 (59)	14 (48)	0.3810
ICP before DC (mm Hg)	22.6	22.2	23.1	NS
ICP after DC (mm Hg)	13.9	13.6	14.3	NS
CPP before DC (mm Hg)	69	68	69	NS
CPP after DC (mm Hg)	74.6	75	74	NS
patients needing CSF diversion	13	6 (46)§	7 (54)§	NS
GOS score				
1–3	30 (44)	15 (50)	15 (50)	NS
4–5	38 (55.9)	24 (63)	14 (37)	0.2760

* APR = abnormal pupillary response to light; BC = basal cistern; NS = not significant.
† Within 48 hours of injury.
‡ More than 48 hours after injury.
§ Percentages based on 13 patients total.

parasagittal veins, converts a benign-looking SDG into a potentially problematic chronic SDH.[19,21,37,48,55,56] In the studies by Koizumi et al.[37] and St John and Dila,[64] 32 of 63 SDGs were bilateral, 12 were on the right side, and 19 were on the left side, indicating no predilection for any particular side of the brain.

In recent years, DC has been used more often as a means of reducing intracranial hypertension unresponsive to maximum medical treatment.[1,2,10,11,23,29,47,58,68,70,74] Selecting DC as a second-tier option in the management of severe head injury mandates attention to a frequent complication of this operative procedure, namely the SDG.[1,2,34,41,76]

In this investigation, we studied the clinical and imaging characteristics of 39 patients with SDGs and compared them with those of 29 patients without to better understand the dynamics of the initial appearance, increase

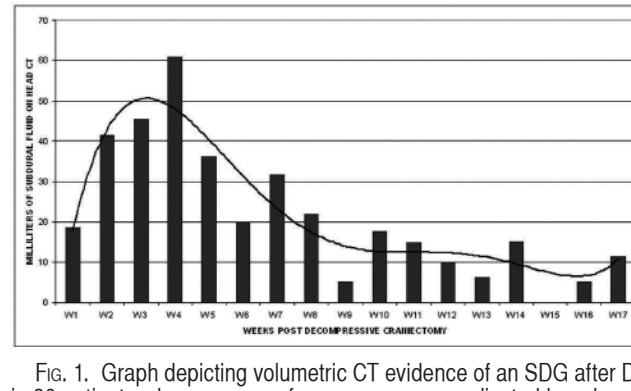

FIG. 1. Graph depicting volumetric CT evidence of an SDG after DC in 39 patients whose course of recovery was complicated by a hygroma. Fluid collection in the subdural space started from Week 1 post-DC, peaked at Week 4, and gradually declined and disappeared around Week 17. The volume of each SDG was calculated using the methods of Gebel et al. and Sucu et al. W = week.

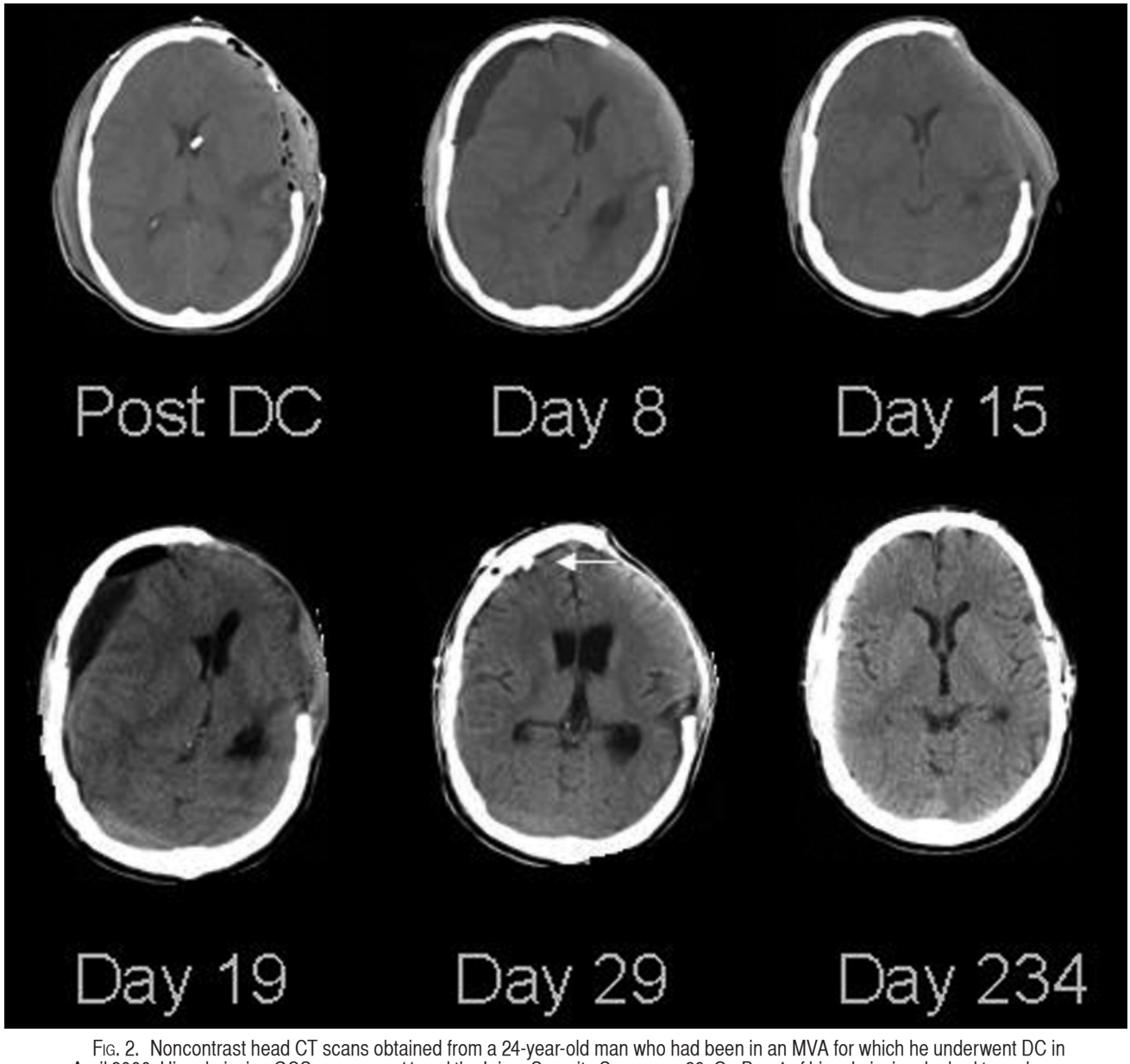

FIG. 2. Noncontrast head CT scans obtained from a 24-year-old man who had been in an MVA for which he underwent DC in April 2000. His admission GCS score was 11 and the Injury Severity Score was 29. On Day 4 of his admission, he had to undergo urgent exploration because of neurological worsening. His postoperative clinical course was uneventful; however, on Day 8 after DC he underwent another CT study because of a decline in his mental status; the CT scan indicated evidence of an SDG with significant shift of the midline structures to the left. He underwent 2 attempts to evacuate the SDG by the insertion of a drainage system through a twist drill bur hole at bedside in the intensive care unit (*arrow*, Day 29). Eight months after the DC, he had an autologous cranioplasty. He remained neurologically intact 47 months after discharge from the institution.

in volume, and resolution of this seemingly benign-looking complication following DC.[1–3,66,76,77]

Methods

The institutional review board of the University of Maryland School of Medicine approved this study.

Patient Population

During a 48-month period between March 2000 and March 2004, 104 of 967 patients with severe head injury underwent DC. The clinical characteristics as well as the prehospital, initial medical, maximum medical, and surgical treatments in these patients have been reported elsewhere.[2,3] Included in the present analysis were 39 patients in whom an SDG developed postoperatively and 29 patients without an SDG. Excluded were those patients who died shortly after DC (36 patients). Demographics, clinical and imaging profiles, and outcomes in these 2 groups of patients are listed in Table 1.

Timeline of SDG Evolution

We monitored the postoperative head CTs of the

TABLE 2: Composition of subdural fluid in 5 patients with SDGs

Case No.	Red Blood Cells (/mm^3)	White Blood Cells (/mm^3)	Protein (mg/dl)	Glucose (mg/dl)
1	40,000	726	408	48
2	22,000	17	556	78
3	13,000	55	246	88
4	8,950	50	118	61
5	1,990	17	60	100

group with SDGs for 36 weeks and measured the volume of hygroma fluid using the methods of Gebel et al.[24] and Sucu et al.[67] The mean volume of SDG fluid was ~ 10 ml during the 1st week, rose to 60 ml during the 4th week, and then gradually decreased and ultimately disappeared around the 17th week post-DC (Fig. 1).

Lateralization of SDGs

Thirty-six (92%) of 39 SDGs were ipsilateral to the site of DC and 3 were contralateral. Significant shifts of the midline structures were noticed in 1 (2.8%) of the 36 patients with an ipsilateral SDG and in 2 (67%) of 3 patients with contralateral hygromas (Fig. 2).

Hydrocephalus With or Without SDG

Six (15%) of 39 patients with SDGs had evidence of hydrocephalus during Weeks 5–8, 17, and 22 post-DC and underwent placement of a shunting device (Fig. 3). In 4 of these patients autologous cranioplasty was performed 1, 2, 5, and 26 days after CSF diversion, and in 2 patients a shunting device was inserted 35 and 155 days after cranioplasty for late-onset hydrocephalus. Among the patients without SDGs, hydrocephalus developed in 7 (24%). Criteria for the diagnosis of hydrocephalus were CT evidence of dilation of the temporal horn tips, globu-

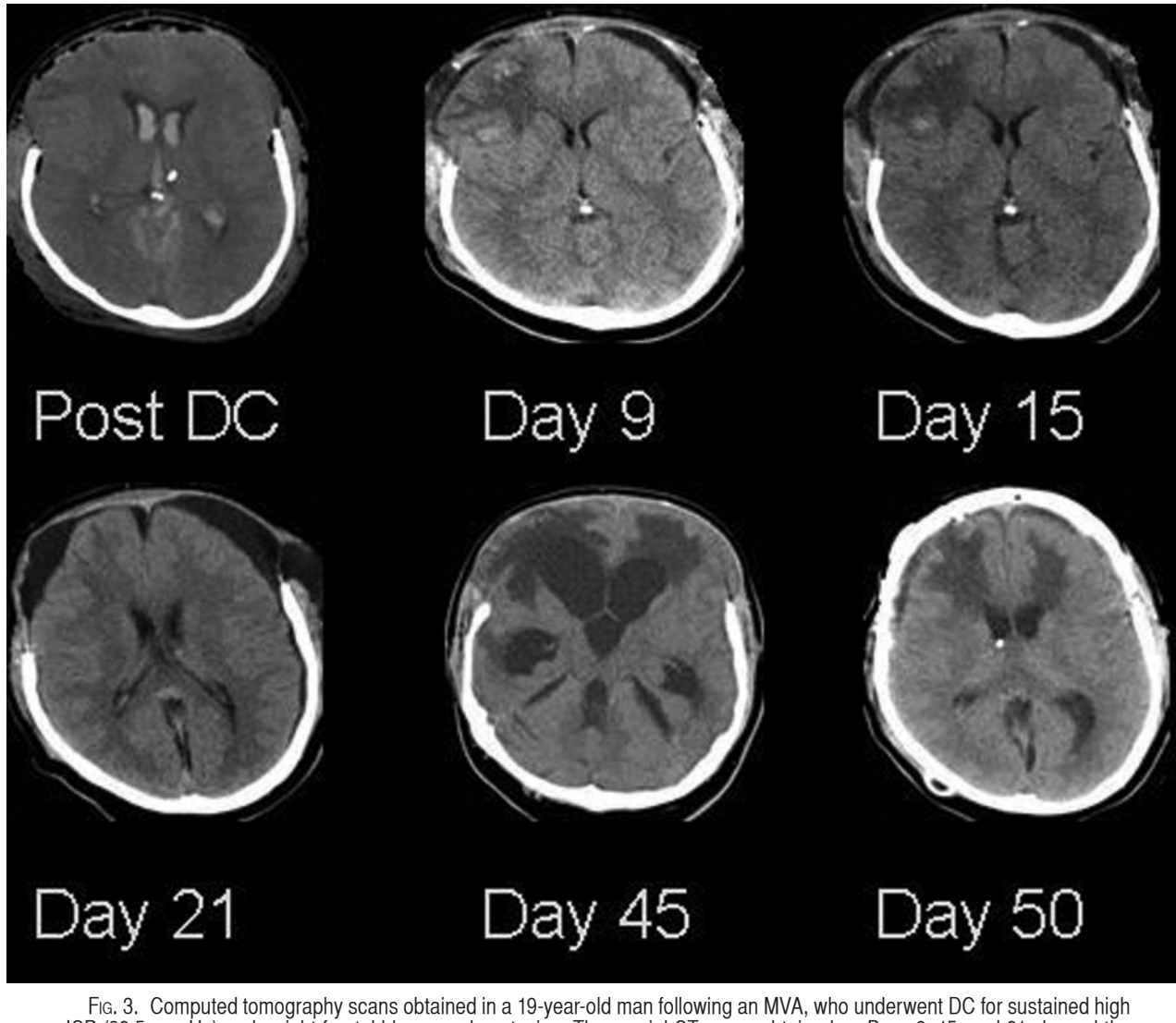

FIG. 3. Computed tomography scans obtained in a 19-year-old man following an MVA, who underwent DC for sustained high ICP (22.5 mm Hg) and a right frontal blossomed contusion. Three axial CT scans obtained on Days 9, 15, and 21 showed the gradual appearance of an SDG. The hygroma was absent on a CT scan obtained 24 days later (Day 45), although there was evidence of hydrocephalus. A shunting device was inserted, and autologous cranioplasty was performed 2 days later. Three months after his accident the patient remained severely disabled (GOS Score 3).

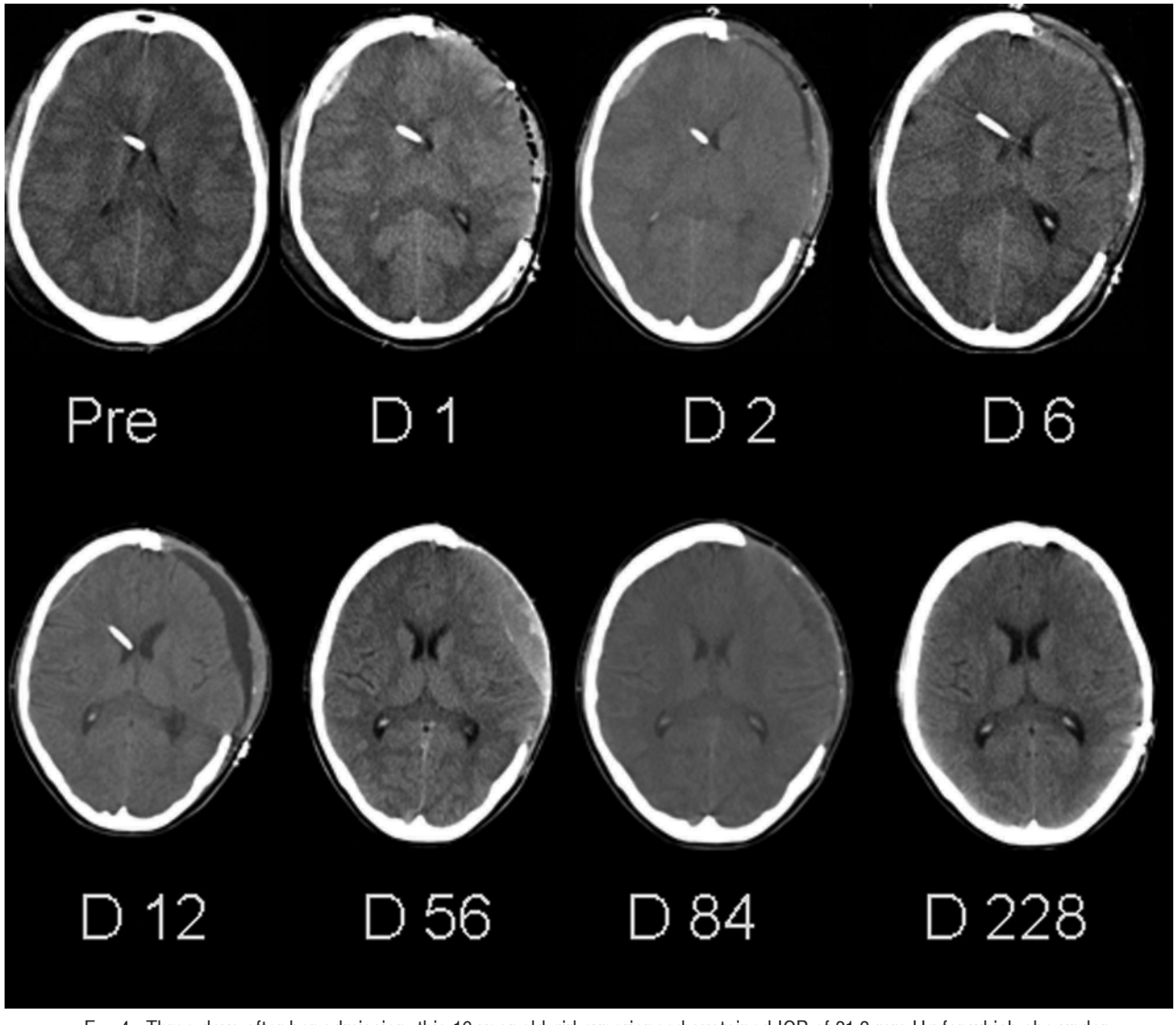

FIG. 4. Three days after her admission, this 16-year-old girl experienced sustained ICP of 31.8 mm Hg for which she underwent DC. A follow-up CT scan indicated the first evidence of an SDG on Day 2 (D2) after DC. Although her condition was clinically stable, a head CT obtained on Day 56 (D56) showed evidence of internal bleeding. The SDH gradually disappeared, and she had cranioplasty on Day 228. Thirty-one months after the injury she was asymptomatic with a GOS score of 5.

lar deformation of the third ventricle, enlargement of the lateral ventricles, or evidence of transependymal absorption of CSF. Clinical evidence of a changed mental status or the arrest or slow progression of rehabilitation milestones, as judged by rehabilitation providers, also triggered new CT studies to confirm or disprove the presence of hydrocephalus.

Composition of SDG Fluid

We checked the composition and cellular content of the SDGs in 5 patients. This fluid was usually bloody and its protein content was high (Table 2). The fluid was collected either in the operating room or at the bedside under aseptic conditions. The volume of the collected fluid was anywhere from 10 to 50 ml. One sample cultured enterococci twice after bur hole evacuation of a contralateral SDG on Days 8 and 19 following DC (Fig. 2). The patient in this case was treated with antibiotics. Tapping the SDG collection never resulted in complete resolution of the hygroma.

Conversion to SDHs

The unexpected appearance of bleeding in an SDG cavity was noted in 3 patients: in 1 on Day 55 of the post-DC period and in 2 on Day 57. One SDG with internal bleeding had to be drained (Fig. 4).

Surgical Intervention

Four patients (10%) underwent surgical drainage of an SDG—by twist drill in 1 patient, bur hole in 1, and percutaneous insertion of a draining system in 2.

Statistical Analysis

Statistical analysis was performed using JMP soft-

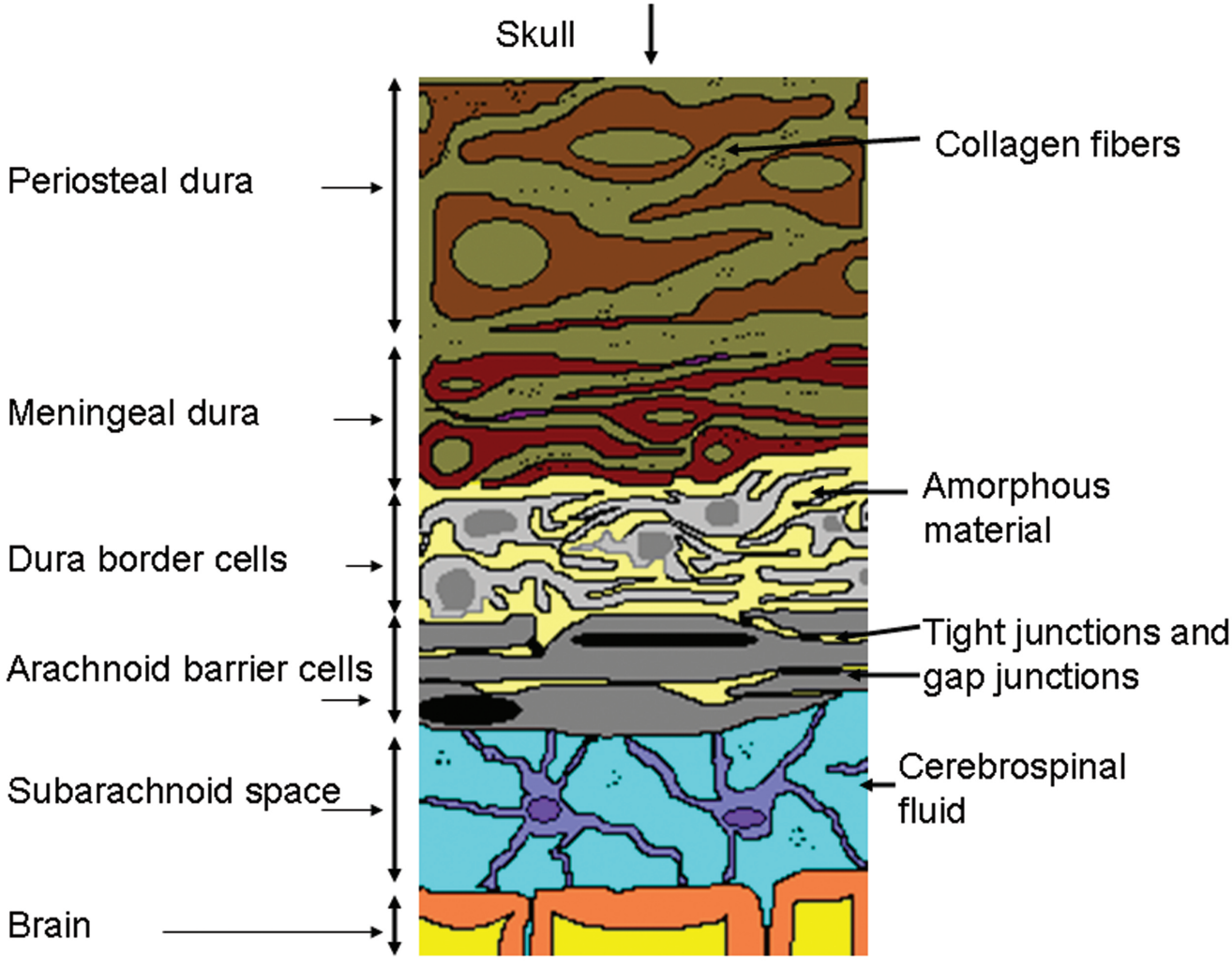

FIG. 5. Schematic representation of dural border cells as observed by electron microscope.[25]

ware (versions 7 and 8, SAS Institute). Two-sided probability values were calculated using the likelihood ratio chi-square for the categorical variables in the 2 groups of patients with or without hygroma. Unpaired t-tests were performed for continuous variables to test for differences in the means between patients with and without hygroma.

Results

Patient and Injury Factors

Patients with SDGs were close to 4 years younger than those without, but this difference was not statistically significant. Motor vehicle accidents, which were responsible for 66% of the injuries, were significantly associated with SDG following DC. Among the 45 patients who had been in an MVA, an SDG developed in 31 (69%) and did not in 14 (31%); this difference was significant ($p = 0.0007$). In contrast, a fall was the injury mechanism in 10 of 29 patients without a hygroma versus 3 of 39 patients with an SDG, which represented a statistically significant difference between the 2 patient groups ($p = 0.005$).

Clinical Injury Severity

There were no significant differences between the 2 patient groups with respect to GCS score, GCS motor score, abnormal pupillary response to light, or injury severity score.

Imaging Profile

Diffuse injury[44] occurred in 30 of 39 patients with an SDG compared with 15 of 29 patients without hygromas; this difference was significant ($p = 0.0299$). A compendium of CT characteristics was considered as evidence of diffuse injury: the presence of a mass < 25 ml, effacement of gyral patterns, disappearance of the third ventricle, and variable compression or obliteration of the basal cisterns.

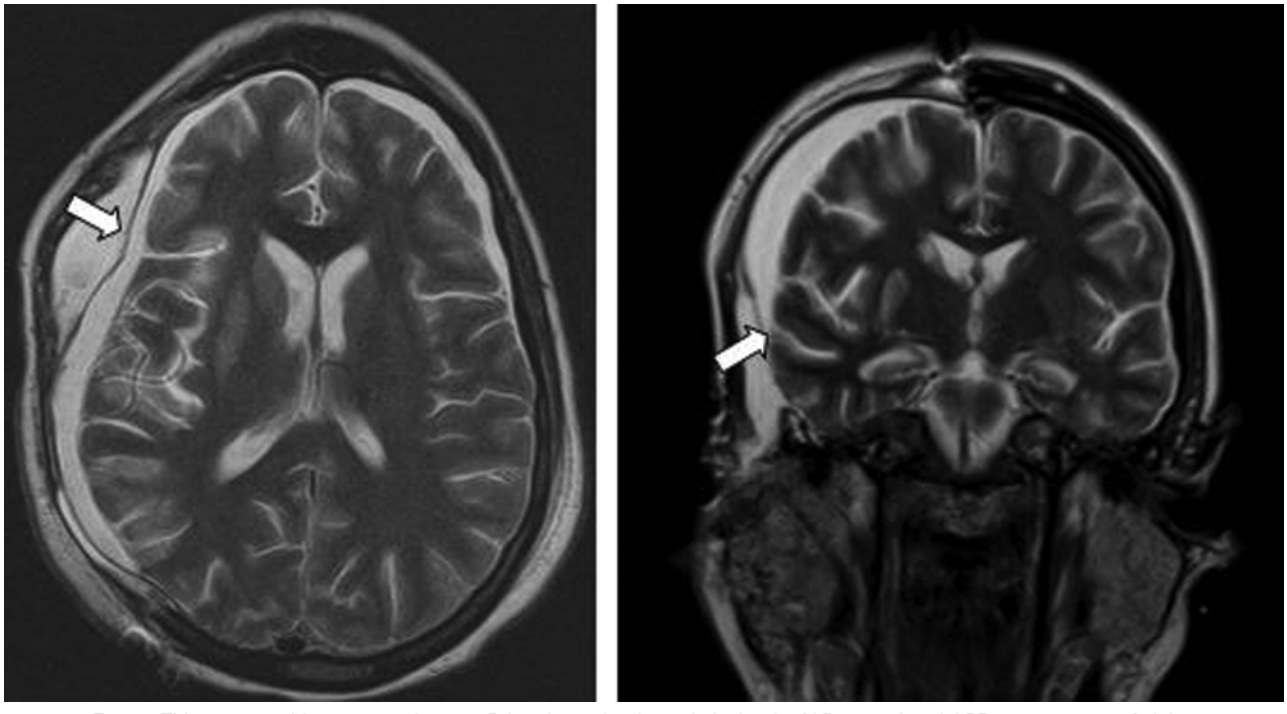

FIG. 6. This 19-year-old woman underwent DC 5 days after her admission for ICP control and CPP management. Axial *(left)* and coronal *(right)* T2-weighed MR images obtained 14 days after a traumatic brain injury, indicating an SDG collection over and under the expansive duraplasty *(arrows)*.

Furthermore, 82.7% of patients with no hygroma—compared with 23.1% of those with an SDG—had an evacuated mass ($p = 0.0001$).[44] A shift of the midline structures > 5 mm on preoperative CT, compressed or obliterated basal cisterns, and the presence or absence of an intraventricular or subarachnoid hemorrhage were not significant variables in the development of an SDG.

Medical and Surgical Management

Although Lang et al.[38] have reported a relationship between increased CPP and the appearance of an SDG, we could not verify that finding in our study. In addition, the timing of a DC did not play a role in the development of an SDG. Hydrocephalus occurred in 13 patients and required CSF diversion; 6 of these patients had a hygroma whereas 7 did not (Table 1).

Patient Outcome

Although an unfavorable outcome (GOS Score 1–3) was split between the 2 patient groups, there seemed to be an indication that patients with an SDG had a better outcome (GOS Score 4 or 5) than those without. Almost 67% of the patients with an SDG fared well compared with < 40% of patients without an SDG who fared well; however, this difference was not a statistically significant finding.

Discussion

Dynamics of CSF Physiology

The CSF located in the ventricular system and subarachnoid space constitutes ~ 150 ml of the 1600 ml defined as the volume contained in the intracranial vault. It is a continually produced, absorbed, and circulated fluid providing both mechanical and chemical protection for the brain through the regulation of the CNS chemical environment, the removal of metabolic waste in a role analogous to the peripheral lymphatics, the support/buoyancy of the brain, and the maintenance of ICP in accordance with the Monro-Kellie doctrine.[26,59,69] The CSF is formed in a diurnal fashion at a rate of ~ 35 ml/hour or 500 ml/day with maximal production occurring at around 2:00 a.m. and minimal production at around 6:00 p.m.[52] Located in the lateral, third, and fourth ventricles, the choroid plexus is responsible for ~ 60–70% of the daily CSF production, with the remaining fluid generated through a number of mechanisms including the movement of water across parenchymal capillaries, across the ependyma, and via the generation of water from the oxidative metabolism of glucose.[18,26,59,72]

Choroid Plexus Anatomy

The choroid plexus, responsible for the majority of CSF production, consists of a superficial layer made of a single sheet of epithelial cells, supported by a core of connective tissue interspersed with blood vessels of various diameters from small arteries to capillaries to small veins. The epithelial lining of the choroid plexus is a single sheet of polarized cuboidal to columnar cells with a smooth basal side, a more structurally complex luminal side containing numerous microvilli intended to increase the luminal surface area, and tight junctions composed of occludin and claudin found laterally between adjacent cells, all contributing to the formation of the blood-CSF

barrier.[59] The blood supply originates from the choroidal arteries, travels through a fenestrated network of capillaries, and ultimately drains into the vein of Galen via the internal cerebral veins at a rate of ~ 3 ml/g/min, 10 times that of the brain parenchyma.

Choroidal Production of CSF

In the simplest terms, the production of CSF at the choroid plexus is the result of the active secretion of Na, Cl, and bicarbonate ions in exchange for K ions across the luminal surface of the choroidal epithelium to create an osmotic gradient that allows movement of water from the vasculature into the subarachnoid spaces. The primary driving force of CSF secretion is an Na^+/K^+-ATPase transporter located on the luminal surface, which actively secretes 3 molecules of Na^+ in exchange for 2 molecules of K^+ at the expense of a molecule of ATP hydrolyzed.[45,60,78] Additional transporters including the ion channel NKCCl, the K^+/Cl^- cotransporter (KCC), and several K^+-specific channels (Kir7.1, Kv1.1, and Kv1.3) are involved in generating an ionic gradient across the luminal surface.

In contrast to luminal transport mechanisms, basolateral ion movement is not as well understood. Several channels have been proposed to have a role in the movement of Na^+ and Cl^- into cells from the basolateral surface, including Na^+/H^+ exchangers and an electroneutral Cl^-/HCO_3^- exchanger, but the work to clarify these processes is ongoing.[42,59] Intracellularly, the H^+ and HCO_3^- needed for these channels are generated by carbonic anhydrase.

The movement of water along the osmotic gradient generated across the CSF barrier, a relatively water-impermeable structure, is facilitated by AQP1, a member of the aquaporin family of water channels that is expressed at both the basolateral and luminal cell surfaces.[51,52,54]

Cerebrospinal Fluid Circulation

The flow of CSF originates at its sites of production within the ventricles, with the majority thought to be within the lateral ventricles. Cerebrospinal fluid continues through the foramina of Monro into the third ventricle, through the sylvian aqueduct into the fourth ventricle, and subsequently into the cisterna magna via the foramina of Luschka and Magendie. From the cisterna magna, CSF flows caudally into the spinal subarachnoid spaces, rostrally into the basal cisternal spaces (prepontine, interpeduncular, ambient, and suprasellar cisterns), and dorsally into the subarachnoid spaces over the cortical convexities and cerebellum. Movement of the CSF via these pathways is thought to occur primarily through bulk flow with multiple contributory factors generating a current including respiratory variation, pulsatility imparted by the cardiac pressures transduced through the circle of Willis within the basal cisterns, ciliary movement at the surface of the choroid, and production of new CSF itself.[12,13,30]

Absorption of CSF

Under normal conditions, CSF is maintained at a relatively stable volume that is balanced by the difference between fluid production, as described above, and the return of fluid to the venous circulation. The traditional view of CSF circulation and cycling has been that the major, perhaps sole, site of CSF absorption and return to the venous system is the arachnoid granulation. In actuality, CSF outflow involves both the arachnoid granulations and lymphatic drainage in what has been referred to as the "dual system of CSF drainage."[32] Arachnoid granulations or villi, also called "pacchionian bodies," are small protrusions of the arachnoid membrane through the dura into the venous sinuses. These structures have also been demonstrated in the arachnoid angles of spinal nerves. A hydrostatic pressure gradient in which subarachnoid pressure exceeds venous sinus pressure forces the return of CSF to the venous system.[14] Interestingly, in the reverse situation, when venous pressures exceed ICP (for example, venous sinus thrombosis), CSF return is inhibited via the arachnoid villi, but reverse flow is not seen.[32] Electron microscopy studies have shown that the arachnoid cells of these granulations form large vacuoles that appear to simultaneously communicate with the luminal and subarachnoid portions, and thus allowing the movement of water.[69] As mentioned above, CSF resorption is believed to be facilitated through a lymphatic pathway. It is true that no lymphatic system exists within the CNS, but experimental data from both animals and humans have shown that tracer molecules injected directly into the brain can be found in the perivascular areas of cranial nerves and ultimately within cervical and spinal lymph nodes. Additionally, experimental occlusion of lymphatics draining the head in several animal models has led to increased ICP, suggesting an important role for lymphatic drainage.[7,8,30,31,35,50]

Incidence of SDGs

In patients with a closed-head injury who have not undergone a DC, the reported incidence of SDG varies from 5 to 21%.[5,22,33,39,40,53] Ohno and colleagues[53] have reported 43 SDGs (6%) in 715 patients, and in the Kaufman et al. study[33] the incidence of an SDG was 38 (4%) in 881 patients with a closed-head injury. Born and associates[5] have described 16 patients (15%) with SDGs among 109 with blunt traumatic brain injury. The incidence of an SDG following DC for severe head injury is not well documented in the literature.[1–3,34,66,76,77] In a 2009 study Yang et al.[75] demonstrated 11 cases (6.5%) of contralateral SDGs among 169 patients who had undergone DC for severe head injury. In an earlier study[76] these same authors documented 23 cases (21.3%) of SDGs in 108 patients who had undergone DC for severe head injury. In that study 11 patients had SDG collections ipsilateral and 9 had hygromas contralateral to the side of the DC; 3 of the 23 SDG collections were interhemispheric. Contralateral hygromas have also been documented in case reports by Kilincer et al.[34] and Su et al.[66] In the present study we saw that SDGs developed in 39 (57.4%) of 68 patients who had survived > 1 month after traumatic brain injury and DC.[1–3]

Pathogenesis and Evolution of SDG

Traumatic Shearing Stress. Detailed electron microscopy studies by Haines et al.[25] and Schachenmayr

and Friede[63] have indicated that there is no dead space between the dura and arachnoid layers. The interface between the dura mater and subarachnoid space is made of an interface layer composed of an arachnoid barrier layer and a dural border cell layer (Fig. 5 *upper*). Whereas a significant number of collagen fibers bond the cellular structure of periosteal and meningeal dura, the dural border cell layer contains very little collagen, and instead, cellular elements are loosely held together by an amorphous material prone to disruption by physical stress.[25,63] Protecting the subarachnoid space and its trabeculae is the arachnoid barrier cell layer in which the cellular elements are tightly packed together by desmosomes, tight junctions, and gap junctions.[25,63] Shearing stress generated by kinetic energy can tear arachnoid barrier cells and damage the integrity of the arachnoid-dura interface layer.[25] In our study patients who had been in MVAs were significantly more prone to SDGs ($p < 0.0007$) than were those who had fallen. It is quite likely that the former patients had a significantly more damaged arachnoid-dura interface layer during sudden exposure to a highly dynamic kinetic energy. Supporting this notion are the reported cases of SDGs contralateral to the side of the DC.[66,75,76] In our series of 39 cases with SDGs, 3 hygromas were contralateral to the side of DC (Fig. 2).

Iatrogenic Surgical Disruption of the Arachnoid-Dura Interface. Another mechanism contributing to the egress of CSF into the subdural space and the formation of an SDG in the context of a DC is iatrogenic damage to the arachnoid-dura interface layer by the neurosurgeon during expansive duraplasty (Fig. 5 *lower*). Up to 90% of hygromas in our study were ipsilateral to the side of craniectomy. This finding is contrary to the laterality of SDGs in closed-head injury, which is almost equally distributed on the right and left sides. Koizumi et al.[36] and St John and Dila[64] have documented 63 cases of SDGs: 32 bilateral, 12 on the right side, and 19 on the left side.

Evolution of an SDG. The accumulation of CSF in the subdural space in closed-head injury, as reported by Koizumi et al.,[37] peaks at 20–30 days. The evolution of an SDG following DC has never been systematically evaluated. In our analysis an SDG could be seen even on the 2nd postoperative day (Fig. 4). When studied in the aggregate, the fluid collections increased in size for up to 4 weeks post-DC and gradually tapered, but lingered for up to 4 months after surgery (Fig. 1). Once in the subdural space, an SDG may exit the suture line of an expansive duraplasty and collect underneath the skin (Fig. 6).

Hydrocephalus. An added complexity to the pathogenesis of these hygromas includes the appearance of hydrocephalus after the complete disappearance of SDGs in 3 patients, pointing to possible difficulties with the dynamics of CSF circulation in patients with DC (Fig. 3). In our study hydrocephalus and the need for surgical CSF diversion was noted at Weeks 5–8, 17, and 22 after DC in 6 patients.

Hemorrhagic Conversion. Subdural hygroma fluid noted in severe head injury and DC is usually xanthochromic, contains a varied number of red blood cells, and is high in protein.[15,39,40,61,62,65,71,73] Hygroma fluid aspirated in 5 patients at the Shock Trauma Center contained high numbers of red blood cells and high protein levels (Table 2). Evidence indicates that this high-protein, highly xanthochromic fluid induces an inflammatory response in the subdural space, predisposing to neomembrane formation with fragile and leaky capillaries.[21,61,62] The maturation of a hygroma neomembrane, containing fragile and leaky capillaries superimposed on stretched parasagittal veins,[55] may make hygromas susceptible to internal bleeding and foster the conversion of a benign SDG into a chronic SDH.[19,21,37,39,43,48,53,56,57] Between 6 and 32% of SDGs convert into chronic SDHs.[21] In the Liu et al. series[43] conversion happened from Days 22–100 after traumatic brain injury. In the Ohno et al. study[53] 20 of 43 patients with hygromas eventually showed evidence of bleeding inside the collection. In the Koizumi et al. series[37] hygromas in 4 patients converted to SDHs on Days 50, 70, 71, and 72 after their trauma. The hygromas in 3 patients in our study converted into chronic SDHs on Days 55, 57, and 57 post-DC (Fig. 4).

Management and Outcome

The visualization of SDGs on imaging studies does not suggest immediate intervention. Contralateral SDGs are more likely to induce a global decline in cognition and consciousness. Yang et al.[75] have reported on 11 patients with contralateral SDGs among 169 who had undergone DC; the hygroma spontaneously resolved in 7 patients and had to be drained in 4. Kilincer et al.[34] have described a 56-year-old man who had undergone a DC for malignant stroke and in whom a contralateral SDG later developed. The SDG in this patient was evacuated first by drainage and subsequently by subdural-peritoneal shunting. Similarly, Su et al.[66] have described a 63-year-old man with a posttraumatic evacuated mass who had undergone a DC and whose level of consciousness globally declined due to a contralateral SDG and significant shift of the midline structures. This patient responded well to simple drainage of the hygroma and was discharged symptom free 1 week later. There seems to be a tendency for contralateral SDGs to cause more symptoms.[34,66,75] In our study 4 patients underwent drainage of the SDG. Three (8%) of 39 patients with hygromas had evidence of mass effect, that is, a shift in the midline structures > 5 mm: 1 of 36 patients with an ipsilateral and 2 of 3 patients with contralateral SDGs. All 3 patients needed subdural drains to reverse the existing shift (1 bur hole, 1 twist drill, and 1 percutaneous insertion of a draining system; Fig. 2). Two of 3 patients whose images had demonstrated a shift of midline structures (5.9 and 6.8 mm) had unchanged GCS scores following drainage of the subdural fluid: GCS Score 8 (Motor Score 3) in 1 patient and GCS Score 14 in the other. In 2 patients with clinical neurological worsening, the GCS score improved after drainage of the SDGs. One patient had a sudden decrease in his GCS motor score (from 6 to 5), which improved after the insertion of a percutaneous drain for an ipsilateral hygroma. Dysautonomic changes in another patient's vital signs occurred (heart rate increased to 115 bpm and temperature increased to 102°F), and he became obtunded. Computed tomography studies in this patient

revealed a 20.2-mm shift due to a contralateral hygroma. This patient underwent bur hole evacuation of this SDG twice. His neurological status improved immediately after surgery (Fig. 2). The overall prognosis for an SDG was better in patients with diffuse injury than in patients with an evacuated mass, which may be simply a reflection of the fact that the prognosis of DC in our 2 published series was better in patients with diffuse injuries than in those with evacuated masses and possibly had nothing to do with the presence or absence of an SDG.[2,3]

Conclusions

An SDG developed in almost 57% of the patients who had undergone DC and survived the 1st month of hospitalization.[2,3] Although 3 patients harbored a contralateral SDG, 36 (92%) of 39 patients had an SDG ipsilateral to the side of DC.

Based on electron microscopy studies,[25,63] necropsy findings,[15,49] and the entrance of contrast material into the hygroma cavity after contrast cisternography,[46] it is hypothesized that injury to the arachnoid-dura interface layer is the central pathology in SDGs. Spillage of the CSF into the subdural space peaked at 3–4 weeks after DC, and this pathological process gradually ceased from 14–17 weeks postoperatively. There is evidence that the proximity of the hygroma fluid with the dura upregulates excitatory cytokine synthesis and neomembrane formation. It is likely that expansion of the hygroma fluid will put undue stress on the walls of parasagittal veins, resulting in fresh bleeding into the hygroma cavity.[55] In addition, bleeding from the neomembrane capillaries may contribute to the possible conversion of a benign-looking hygroma into a chronic SDH.

In our experience, high dynamic accidents and diffuse traumatic brain injuries were risk factors for an SDG following DC.

The majority of hygromas—regardless of their size or internal bleeding—gradually disappeared without surgical management. Contralateral hygromas needed more aggressive treatment because of their tendency to cause midline shift. A variety of surgical management strategies have been used for the evacuation of SDGs including bur hole evacuation, bedside subdural drains, and craniotomy.[37,40,64,65,75,76]

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Complications of cranioplasty following decompressive craniectomy: analysis of 62 cases

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Object. Decompressive craniectomy is a potentially life-saving procedure used in the treatment of medically refractory intracranial hypertension, most commonly in the setting of trauma or cerebral infarction. Once performed, surviving patients are obligated to undergo a second procedure for cranial reconstruction. The complications following cranial reconstruction are not well described in the literature and may very well be underreported. A review of the complications would suggest measures to improve the care of these patients.

Methods. A retrospective chart review was undertaken of all patients who had undergone cranioplasty during a 7-year period. Demographic data, indications for craniectomy, as well as preoperative, intraoperative, and postoperative parameters following cranioplasty, were recorded. Perioperative and postoperative complications were also recorded. Patients were classified as having no complications, any complications, and complications requiring reoperation. The groups were compared to identify risk factors predictive of poor outcomes.

Results. The authors identified 62 patients who had undergone cranioplasty. The immediate postoperative complication rate was 34%. Of these, 46 patients did not require reoperation and 16 did. Of those requiring reoperation, 7 were due to infection, 2 from wound breakdown, 2 from intracranial hemorrhage, 3 from bone resorption, and 1 from a sunken cranioplasty, and 1 patient's cranioplasty procedure was prematurely ended due to intraoperative hypotension and bradycardia. The only factor statistically associated with need for reoperation was the presence of a bifrontal cranial defect (bifrontal: 8 [67%] of 12, requiring reoperation; unilateral: 8 [16%] of 49 requiring reoperation; $p < 0.01$)

Conclusions. Cranioplasty following decompressive craniectomy is associated with a high complication rate. Patients undergoing a bifrontal craniectomy are at significantly increased risk for postcranioplasty complications, including the need for reoperation. *(DOI: 10.3171/2009.3.FOCUS0962)*

Key Words • cranioplasty • craniectomy • cranial reconstruction • cerebral decompression • intracranial hypertension

Decompressive craniectomy is a potentially life-saving procedure used in the treatment of medically refractory intracranial hypertension, most commonly in the setting of trauma or large-vessel infarct and less frequently in the settings of aneurysmal subarachnoid hemorrhage, intraoperative brain swelling, and encephalitis.[21,59] Decompressive craniectomy, however, remains controversial. Its efficacy is currently being investigated with respect to survival and quality of life in multicenter, prospective, randomized trials in the setting of traumatic brain injury[22] and middle cerebral artery infarction.[27,66]

Once patients undergo decompressive craniectomy, those who survive are obligated to undergo a second procedure for surgical cranial reconstruction, that is, cranioplasty. Much of the modern literature regarding cranioplasty following decompressive craniectomy is based on case series that emphasize the technical aspects of the procedure such as the use of materials,[2,3,9,10,12,14,26,30,33,35,50–52,54,55,60,63,68,70,71] the use of techniques to store the bone flap prior to reconstruction,[16,19,24,25,43,48,49,72] the timing of surgical intervention,[6,37] or other specific modifications to either the craniectomy or cranioplasty procedure, which may influence the cranioplasty.[20,28,34,36,38,41,47,67] There are relatively few modern-day large clinical series describing the clinical outcomes and perioperative complications of cranioplasties in the setting of nonpenetrating traumatic brain injury and large vessel infarction.[40,42] Complications after cranial reconstruction, often viewed as a straightforward neurosurgical procedure, may very well be underreported. Furthermore, traditional neurosurgical dictums regarding certain aspects of cranioplasty such as timing of surgery may not be appropriate in the modern era of neurosurgical care. A review of the complications would suggest measures to improve the care of these patients.

In the current study, our goal was to provide a complete review of all perioperative complications, defined as any potentially adverse event within 30 days of surgery, as well as identify any risk factors that may be associated with the need for reoperation after a primary cranioplasty.

Methods

The study was approved by the institutional review board of Albany Medical Center, a Level 1 trauma, tertiary care, teaching hospital. After approval, the billing and discharge databases of the neurosurgical service were reviewed to identify all patients who had undergone cranioplasty following a decompressive craniectomy during a 7-year period (January 1, 2002–December 31, 2008). This series only included patients who underwent craniectomies for cerebral swelling and excluded those undergoing craniectomy for meningioma resection and craniosynostosis. Once identified, the available hospital charts and clinic records were reviewed retrospectively to abstract relevant data.

Abstracted data included age at time of cranioplasty (years), sex (male or female), medical comorbidities (hypertension, diabetes, and tobacco use), indications for craniectomy (trauma, stroke, infection, and intraoperative swelling), laterality of craniectomy (bilateral, unilateral, or bifrontal), time between craniectomy and cranioplasty (days), type of prosthesis if used (titanium, methylmethacrylate, or porex), storage of bone flap if used (subcutaneous or tissue bank), operative time (minutes), identification of intraoperative CSF leak (yes or no), estimated blood loss (ml), intraoperative fluid administration (ml), length of stay after cranioplasty (days), and disposition before and after the cranioplasty (home, hospital, or inpatient nursing facility).

Any potentially adverse medical or surgical events identified within 30 days of surgery were recorded as early complications. Late complications were unsatisfactory events directly related to the cranioplasty occurring > 30 days postoperation. Patients were classified as having no complication, any complication, and complication requiring reoperation. Specifically, our 2 outcomes of interest were complications after cranioplasty and the need for reoperation after cranioplasty. Both variables were dichotomized, and all patient- and surgery-related factors were assessed as risk factors for each of the 2 outcomes of interest via bivariate analysis. Chi-square analysis and the Fisher exact test were used to assess the association between the categorical risk factors and the outcomes. The Wilcoxon rank-sum test was used to assess if the distribution of the continuous variables was different among those who did and those who did not have the outcomes of interest. To assess complication rates associated with time to cranioplasty, patients were divided into quartiles, and ORs were calculated. All tests were 2-tailed. All associations were assessed at a level of 0.05 of statistical significance. Statistical analysis was performed using STATA 10 software (StataCorp LP).

Results

We identified 109 patients who had undergone a cranioplasty following cerebral decompression via craniectomy. Of these, 23 charts were incomplete for the purpose of this study, and 24 patients were lost to follow-up in the early postoperative period. As a result, 62 patients were identified who had undergone a decompressive craniectomy and subsequent cranioplasty with a minimum of 4 months of follow-up. Even among this population, some variables could not be reliably extracted from the medical records including medical comorbidities, disposition before and after cranioplasty, and intraoperative CSF leakage. The baseline patient characteristics and perioperative factors are shown in Table 1. The majority of the patients in this review were male (34 [55%]), and the most common indication for craniectomy was trauma (41 [66%]). Other reasons for decompressive craniectomy included stroke, infection, and intraoperative swelling. Most craniectomies were unilateral (49 [79%]). In addition, 12 patients (19%) underwent a bifrontal craniectomy, and 1 (2%) underwent a bilateral decompression. Regarding the cranioplasty procedure, the overwhelming majority of repairs used autologous bone (57 [92%]), and all autologous bone was stored in a tissue bank. Titanium and methylmethacrylate repairs were performed much less frequently (2 [3%] and 3 [5%] cases, respectively).

TABLE 1: Demographic and operative details in 62 patients undergoing craniectomy and cranioplasty

Characteristic	No. of Patients (%)*
no. of patients	62
male	34 (55)
female	28 (45)
craniectomy	
mean age (yrs)	31.5 ± 2.4
indication	
trauma	41 (66)
stroke	15 (24)
infection	2 (3)
intraop swelling	4 (6)
type	
unilat	49 (79)
bifrontal	12 (19)
bilat	1 (2)
cranioplasty	
mean age (yrs)	31.9 ± 2.4
type of prosthesis	
autologous	57 (92)
titanium	2 (3)
methylmethacrylate	3 (5)
mean no. of days btwn craniectomy & cranioplasty	133 ± 18.2
mean op room time (min)	173 ± 10
mean estimated blood loss (ml)	238 ± 28
mean intraop intravenous fluids (ml)	1881 ± 138
mean length of stay (days)	11 ± 2.1

* Unless otherwise indicated, mean values are presented as the means ± SEMs.

Surgical Complications

Perioperative and postoperative complications are summarized in Table 2. Of the 62 patients undergoing

TABLE 2: Complications in 62 patients undergoing craniectomy and cranioplasty*

Complication	No. of Patients (%)		
	Early (≤30 days postop)	Late (>30 days postop)	No. Requiring Reop
wound	3 (4.8)	6 (9.7)	9
infection	2 (3.2)	5 (8.1)	7
dehiscence	1 (1.6)	1 (1.6)	2
hematoma	2 (3.2)	0	2
epidural	1 (1.6)	0	1
subdural	1 (1.6)	0	1
bone resorption	0	4 (6.5)	3
sunken bone plate	0	1 (1.6)	1
intraop hemodynamic instability	1 (1.6)	0	1
status epilepticus	1 (1.6)	0	0
hydrocephalus	1 (1.6)	0	0
deep vein thrombosis	2 (3.2)	0	0
upper extremity	1 (1.6)	0	0
lower extremity	1 (1.6)	0	0
	0	0	0
total	10 (16.1)	11 (17.7)	16/21 (76.2)

* Of 62 patients, 21 (33.8%) had some complication and 16 (25.8%) required a reoperation.

cranioplasty, 41 (66%) experienced no complications and 21 (34%) experienced at least 1 complication. Complications included infection (7), wound dehiscence (2), epidural hematoma (1), subdural hematoma (1), bone resorption (4) which was diagnosed clinically and confirmed on CT scanning, sunken bone plate (1), status epilepticus (1), hydrocephalus (1), deep vein thromboses (2), and intraoperative bradycardia and hypotension necessitating a premature end to the cranioplasty procedure (1). Ten patients (16.1%) had early complications and 11 (17.7%) had late complications. After statistical analysis, the only measured variable found to be significantly associated with complication after cranioplasty was a bifrontal defect compared with a unilateral defect (bifrontal: 8 [67%] of 12, with complications; unilateral: 13 [27%] of 49, with complications; $p < 0.05$). Suffering a postoperative complication resulted in a longer duration of inpatient hospitalization, but this difference was not statistically significant (uncomplicated: mean 5.0 days vs any complication: mean 7.4 days, $p > 0.05$).

Complications Requiring Reoperation

Complications requiring reoperation are summarized in Table 2. Sixteen patients (26%) had complications that required an additional surgery to address their primary cranioplasty site. Such complications included infection, wound dehiscence, intracranial hemorrhage, bone resorption, and sunken bone plate. In 1 patient, the initial procedure was aborted midoperation due to hemodynamic instability. After statistical analysis, the only variable found to be significantly associated with a need for reoperation was the presence of a bifrontal cranial defect (bifrontal: 8 [67%] of 12 requiring reoperation; unilateral: 8 [16%] of 49 requiring reoperation; $p < 0.01$) (Table 3).

TABLE 3: Data regarding reoperation and laterality

Characteristic	No. of Patients (%)	
	No Reop	Reop
no. of patients	46 (74)	16 (26)
laterality of cranioplasty		
unilat	41 (84)	8 (16)
bifrontal	4 (33)	8 (67)
bilat	1 (100)	0 (0)
no. of days btwn craniectomy & cranioplasty*	129 ± 22	143 ± 34

* Presented as the means ± SEMs.

Time to Cranioplasty

The 62 patients who underwent cranioplasty were divided into 4 groups, each containing approximately the same number of patients, based on duration of time between craniectomy and cranioplasty. Odds ratios calculating the likelihood of complications and complications requiring reoperation with respect to timing of cranioplasty are presented in Table 4. For complications and complications requiring reoperation, ORs were highest in the 100- to 136-day group (OR 1.67 and 3, respectively).

Discussion

Cranioplasty following decompressive craniectomy is a conceptually intuitive procedure from the perspective of safety and cosmesis. More recent reports have suggested that the procedure may help optimize neurological recovery, both physiologically and/or clinically.[1,4,11,13,15,18,23,31,32,44–46,57,58,61,64,65,69] However, there is no specific technique or material that has consistently stood alone as superior, and postoperative complication rates vary widely.[62] In the modern era, most reports in the literature regarding cranioplasty have focused on technical aspects of the procedure and have not emphasized overall surgical complications.[2,3,9,10,12,14,20,26,28,30,33–36,38,41,50–52,54,55,60,63,67,68,70,71] Because decompressive craniectomy is now being reevaluated in large, prospective, randomized trials,[22,27,66] an analysis of complications for cranioplasty is particularly important. As almost all patients surviving a decompressive craniectomy will require cranioplasty, the complications of this second operative intervention should be acknowledged.

Complications Following Cranioplasty

The current report suggests that cranioplasty following decompressive craniectomy is associated with a high complication rate (33.8%). Of those patients who did experience complications, 5 (24%) of the 21 did not require

TABLE 4: Data regarding time between craniectomy and craniotomy and ensuing complications

No. of Days Btwn Craniectomy & Cranioplasty	No. of Patients	Complications		Complications Requiring Reoperation	
		No. (%)	OR	No. (%)	OR
0–48	16	6 (38)	0	4 (25)	0
49–99	15	3 (20)	0.42	0 (0)	—
100–136	16	8 (50)	1.67	8 (50)	3
≥137	15	4 (27)	0.61	4 (27)	1.1

a subsequent operation. However, these complications could not all be classified as "minor." Specifically, one such patient was found to be in status epilepticus immediately postoperatively. Of the patients with complications, 76% required another operation to address their previous cranioplasty. Such complications included infection, bone resorption, wound dehiscence, sunken bone flap, hematoma, and intraoperative hemodynamic instability necessitating a premature end to the primary cranioplasty procedure. Of note, our infection rate (11.3%) appears to be similar to that previously reported in large series of patients undergoing cranioplasty.[7,29,40,42]

Complications Requiring Reoperation Following Cranioplasty

In the current analysis, complications requiring reoperation occurred in 16 patients (26%) and in 76% of those who had any complication (16 of 21). This overall reoperative complication rate is surprisingly high, considering the perceived straightforward nature of this procedure.

In our series, all patients who experienced wound complications as defined by infection and wound dehiscence required reoperation. Three of these patients initially presented with wound dehiscence without gross evidence of infection. All 3 were treated with primary closure. Two of the patients received scalp tissue expanders prior to this procedure. The third patient underwent uneventful revision of his scalp flap, but bacterial cultures taken from the operating room eventually revealed methicillin-resistant *Staphylococcus aureus*. He was subsequently treated with intravenous antibiotics. None of these patients was treated by removal of the bone plate. Our review demonstrated that patients undergoing a bifrontal cranioplasty were significantly more likely to have complications. The complications in this group included infection (in 4 patients), wound dehiscence (in 1), sunken bone plate (in 1), resorption (in 1), and hemodynamic instability during cranioplasty (in 1). All complications for these patients led to reoperation. Several factors may have contributed to this observation, including a longer incision in the case of a bifrontal defect, less available temporalis muscle to provide soft-tissue coverage, possible violation of the frontal sinus, and perhaps a longer operative time. Of these, violation of the frontal sinus has previously been described as a definitive risk factor for infection after cranioplasty.[5,39] Unfortunately, because of the retrospective nature of this study we were unable to reliably quantify any of these potentially contributing variables.

Time to Cranioplasty

According to traditional neurosurgical dictum, a shorter time from craniectomy to cranioplasty is associated with poor outcome.[17,53,62] While often recommended in neurosurgical texts, the basis of this recommendation is not well cited. The rationale for this waiting period is most likely based on the large series of patients reported by Rish and colleagues[56] in 1979. This group found that cranioplasties taking place 1–6 months after craniectomy had the highest complication rate (7.9%) and those performed 12–18 months after craniectomy had the lowest complication rate (4.5%). The purported advantage of this waiting period includes avoidance of operating on a potentially contaminated wound.[56] However, because this study included only patients with penetrating head injuries, the results may not apply to patients who have undergone decompressive craniectomy in the setting of nonpenetrating injury. Recently Carvi et al.[6] and Liang et al.[37] have suggested that cranioplasty following decompressive craniectomy for blunt injury can be performed sooner than previously suggested. The possible advantages of performing cranioplasty in a more timely fashion may include easier dissection of tissue planes, as well as prevention of negative postcraniectomy sequelae including posttraumatic hydrocephalus,[8] syndrome of the trephined,[31,46] or other neurological complications.[32,61,64]

Our analysis in regard to time between craniectomy and cranioplasty revealed a higher risk of both postoperative complications and need for reoperation in those patients treated between 100 and 136 days. It is difficult to rectify this point clinically, and our small number of events in each group limits the strength of any conclusions to be made regarding these data. Most significant may be the fact that those patients treated early (0–48 days) or those treated late (≥ 137 days) did not have a significantly increased risk. This finding, along with the reports by Carvi et al.[6] and Liang et al.,[37] may negate previous dictum suggesting cranioplasty needing to be performed during a certain time point postcraniectomy. Again, this conclusion is limited and requires further analysis in a prospective study.

Critique of the Current Study

The current study is a retrospective analysis of the complications of cranioplasty following decompressive craniectomy. Accordingly, the study suffers from all the anticipated deficiencies of a retrospective analysis including loss of patient information, poor follow-up, inconsis-

tent operative indications for craniectomy, and inconsistent techniques for cranioplasty. These deficiencies may lead to an inaccurate estimation of the true complication rate.

In an effort to accurately estimate the complication rate, any potentially adverse event in this series was identified as a complication. This method may have falsely inflated the observed complication rate. On the other hand, in a review of cranioplasties in 75 children, Blum et al.,[5] did not identify any complications until 2.5 years postoperatively. Given that our patients were included with only a minimal follow-up of 4 months, it is possible we may actually be underreporting the total number of complications. Despite these limitations, the current study is important as it highlights the fact that a significant number of complications do occur following cranioplasty, including complications that may necessitate reoperation.

Conclusions

Patients undergoing decompressive craniectomy are obligated to undergo a second procedure for cranial reconstruction. This second surgery has a remarkably high rate of complications. Additionally, patients undergoing bifrontal craniectomies are at a significantly increased risk for postcranioplasty complications including the need for an additional operation. The time between craniectomy and cranioplasty does not appear to be associated with complications; however, our data are too limited to make definitive conclusions as to this point. Prospective studies are needed to further evaluate cranioplasty complications.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Long-term results following titanium cranioplasty of large skull defects

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Object. Decompressive craniectomy is an established procedure to lower intracranial pressure. Therefore, cranioplasty remains a necessity in neurosurgery as well. If the patient's own bone flap is not available, the surgeon can choose between various alloplast grafts. A review of the literature proves that 4–13.8% of polymethylmethacrylate plates and 2.6–10% of hydroxyapatite-based implants require replacement. In this retrospective study of large skull defects, the authors compared computer-assisted design/computer-assisted modeled (CAD/CAM) titanium implants for cranioplasty with other frequently used materials described in literature.

Methods. Twenty-six patients underwent cranioplasty with CAD/CAM titanium implants (mean diameter 112 mm). With the aid of visual analog scales, the patients' pain and cosmesis were evaluated 6–12 years (mean 8.1 years) after insertion of the implants.

Results. None of the implants had to be removed. Of all patients, 68% declared their outcomes as excellent, 24% as good, 0.8% as fair, and 0% as poor. There was no resulting pain in 84% of the patients, and 88% were satisfied with the cosmetic result, noting > 75 mm on the visual analog scale of cosmesis. All patients would have chosen cranioplasty again, stating an improvement in their quality of life by the calvarial reconstruction. Nevertheless, follow-up images obtained in 4 patients undergoing removal of meningiomas was only suboptimal.

Conclusions. With the aid of CAD technology, all currently used alloplastic materials are suited even for large skull defect cranioplasty. Analysis of the authors' data and the literature shows that cranioplasty with CAD/CAM titanium implants provides the lowest rate of complications, reasonable costs, and acceptable postoperative imaging. Polymethylmethacrylate is suited for primary cranioplasty or for long-term follow-up imaging of tumors. Titanium implants seem to be the material of choice for secondary cranioplasty of large skull defects resulting from decompressive craniectomy after trauma or infarction. Expensive HA-based ceramics show no obvious advantage over titanium or PMMA. *(DOI: 10.3171/2009.3.FOCUS091)*

Key Words • craniectomy • decompressive surgery • calvarial reconstruction

Despite being in use for a long time, decompressive craniectomy remains an established procedure to lower intracranial pressure due to malignant brain swelling,[19,30] and it is still undergoing technical improvements.[32] Therefore, cranioplasty will remain a necessity in neurosurgery as well.

The surgical correction of skull defects has 2 main purposes: protection of the brain and a satisfying cosmetic result. The implanted material has to be durable and provide a low repulsion rate by the host.[14] Because of immunological compatibility, the reimplantation of the patient's own bone flap is usually the treatment of choice. Nevertheless, age, difficulties with storage, timing of surgery, and anatomical conditions can lead to the loss of the bone flap in a large number of cases.[16,28] Various techniques are currently being discussed to rescue infected bone flaps or improve their preservability,[1,4,20,24] but as of yet none of these offers results superior to the ones achievable with nonbioresorbable materials. Recent developments are looking toward osteoconductive bioresorbable materials, tissue engineering,[36,37,40,41] osteoinduction by growth factors, and gene therapy,[12,17,31] but despite promising experimental results in animals and preliminary studies, these new technologies still have to prove their worth in large-scale long-term clinical settings.

Until then the surgeon has the choice between various cranioplasty techniques. Computer-assisted design and modeling of cranioplastic materials has improved the cosmetic outcome as well as minimized the procedure time needed for plate insertion.[7,21,39] Numerous materials have shown high biocompatibility and clinical reliability,[32] such as PMMA, titanium, numerous ceramics such as HA, carbon materials such as CFRP, and others (Table

Abbreviations used in this paper: CAD = computer-assisted design; CAM = computer-assisted modeled; CFRP = carbon fiber reinforced polymer; HA = hydroxyapatite; PMMA = polymethylmethacrylate; VASC = visual analog scale for cosmesis; VASPI = VAS for pain intensity.

TABLE 1: Review of the literature regarding clinical studies dealing with alloplast graft cranioplasty

Authors & Year	Study Design	Material	No. of Patients	Mean Follow-Up Duration (mos)	Complication
Joffe et al., 1999	prospective	titanium	148	12	1 infection (0.6%)
Blake et al., 1990	retrospective	titanium	20	52	no removals (0%)
Eufinger et al., 1998	retrospective	titanium	22	24	1 infection (4.5%)
Kamyszek et al., 2001	retrospective	titanium	76	8	2 impaired healings (2.6%)
Matsuno et al., 2006	retrospective	titanium	77	64	2 infections (2.6%)
		PMMA	58		8 infections (13.8%)
		autogenous bone	54		14 infections (25.9%)
		ceramics (Alumina + HA)	17		1 infection (5.9%)
van Gool, 1985	retrospective	PMMA	45	39	2 removals (4.4%)
Moreira-Gonzalez et al., 2003	retrospective	autogenous bone	312	39	22 infections/exposure (7%)
		PMMA	75		10 infections/exposure (13.3%)
		HA	58		13 infections/exposure (22.4%)
Kriegel et al., 2007	retrospective	PMMA	36	44	removal of 2 implants (4.5%)
		autologous bone graft (Tutoplast)	25	15	removal of 2 implants (8%)
Marchac & Greensmith, 2008	retrospective	PMMA	32	8.2*	4 removals (12.5%)
Friedmann et al., 200013	prospective	HA	38	24	3 infections (7.9%)
Costantino et al., 2000	retrospective	HA	21	15	no removal (0%)
Verheggen & Merten, 2001	retrospective	HA	11	6	1 impaired healing (9%)
Durham et al., 2003	retrospective	HA	9	11	2 infections (22.2%)
Eppley et al., 2003	retrospective	HA	62	24	3 infections (5%)
Poetker et al., 2004	retrospective	HA	76	13	2 infections (2.63%)
Saringer et al., 2002	retrospective	CFRP	29	39	0 infections
Sanus et al., 2008	prospective	acrylic resin (Cortoss)	13	24.3	0 removals or infections
Scolozzi et al., 2007	case report	PEEK	1	12	0 removal

* This follow-up duration was reported in years.

1). Nevertheless, it is unclear which material provides the best overall result.

The CAD/CAM titanium plates offer an excellent choice for cranioplasty based on their strength, low infection rate, biocompatibility, handling characteristics, and suitability for postoperative imaging techniques,[5,21–23] but they are often avoided because of their high costs. However, it may be advisable to take into account that the long-term suitability of this material could compensate its higher production costs. Furthermore, operative time, infection, and revision rates as additional cost factors have to be calculated as well. There are few long-term studies of cranioplastics in the literature. In this long-term follow-up study, we examined 25 patients with large calvarial defects who underwent cranioplasty with titanium plates. We observed these patients for up to 12 years after the procedure, and we reviewed the existing literature focusing especially on complications, removal rates, and long-term follow-up results of cranioplasties.

Methods

Between 1983 and 2002, 241 patients underwent craniectomy for various reasons in our department. The bone flap was reinserted in 149 cases. Since 1996, CAD/CAM titanium plates (Fig. 1) have been inserted for a variety of large skull defects (Table 2).

Between 1996 and 2002, 26 patients (15 men and 11 women) underwent cranioplasty with CAD/CAM titanium implants (Cranio Construct; Bochum GmbH) due to a posttraumatic nonrestorable large skull defect, lysis, or infection of the bone flap after severe head trauma (13 patients); cerebral infarction (5 patients); meningioma (4 patients); and dysplasia, bone erosion as a result of a growing arachnoidal cyst, herpes encephalitis, and brain abscess (1 patient each). In 6 of these patients 2 plates were implanted. One patient underwent bilateral cranioplasty with titanium (Fig. 2). Four patients underwent bifrontal cranioplasty (Figs. 3 and 4).

The titanium implant was inserted after exposure of the margins of the skull defect without opening the dura mater. If necessary, plate insertion was supported by hyperventilation or lumbar CSF drainage for several minutes. Central dural tenting sutures were placed routinely. Between 1996 and 1998, the fastening of the plates was performed with titanium miniplates from various companies. Since 1998, CranioFix titanium clamps (Aesculap AG) have been used to fasten the plates. A wound drain was placed for 3 days in all cases.

The size of the skull defects and plates ranged from

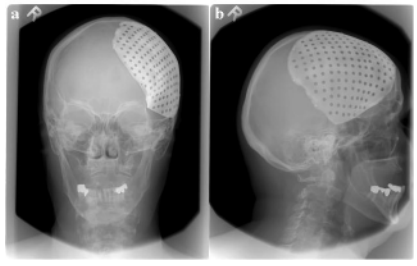

Fig. 1. Anteroposterior (a) and lateral (b) radiographs obtained in a patient following frontotemporoparietal craniectomy for decompression.

65 to 155 mm (mean 112 mm). The patients' age at operation ranged from 6 to 63 years (mean 35.6 years), and the follow-up period ranged from 6 to 12 years (mean 8.1 years). The span of time between the removal of the bone flap and the insertion of the CAD/CAM titanium plate ranged from 2 to 14 months (mean 6.5 months).

The follow-up examination included a questionnaire covering subjectively experienced pain and satisfaction with the cosmetic result based on two 100-mm-long VASs: the VASPI and VASC. Overall satisfaction with cranioplasty as a whole was evaluated using the Odom criteria. Furthermore, eventual changes in the quality of life after cranioplasty were evaluated, and the patients were asked if they would have chosen cranioplasty again. Postoperative CT scanning was routinely performed.

One patient died of a heart attack 6 years after plate insertion and was lost to the last follow-up. At the 1-year follow-up, this patient reported no specific complaints, but he did not fill out the questionnaire. Therefore, a total of 25 patients were evaluated.

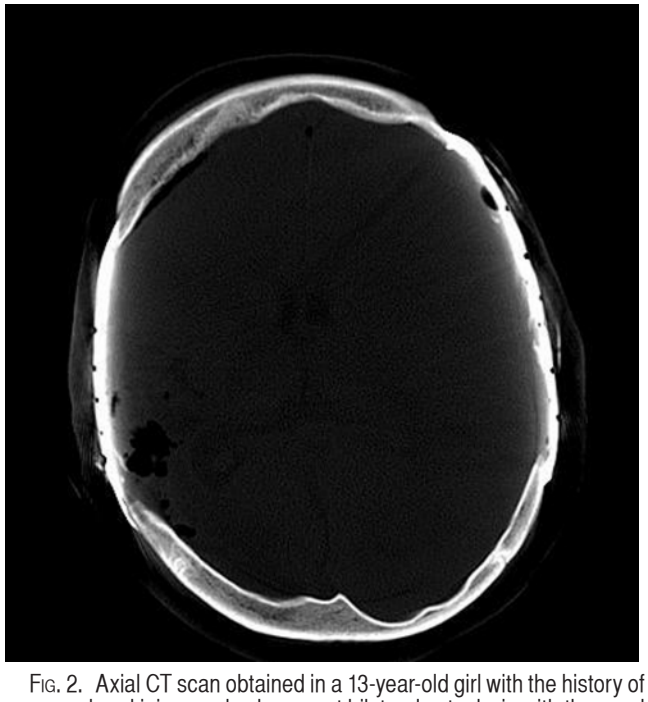

Fig. 2. Axial CT scan obtained in a 13-year-old girl with the history of a severe head injury and subsequent bilateral osteolysis with the need of bilateral alloplastic cranioplasty.

TABLE 2: Localization, side, and reasons of autologous bone flap loss in 26 patients

Localization	No. of Patients
frontal	
frontotemporoparietal	16
bifrontal	4
frontal	3
frontoparietal	1
temporal	2
side	
lt	10
rt	11
bifrontal	4
bilat	1
reason for bone flap loss	
infection	14
lysis	8
nonrestorable trauma	2
bone erosion	1
dysplasia	1

Results

Analysis of the Odom criteria showed that 68% of the patients (17 of 25) noted excellent, 24% (6 of 25) good, 0.8% (2 of 25) fair, and 0% poor results. Overall, 84% (21 of 25) did not suffer any pain, and 88% (22 of 25) were satisfied with the cosmetic result of titanium cranioplasty with a score of > 75 mm on the VASC (Fig. 5).

Only 2 female patients, one with a history of a severe head injury and the other with a left brain infarction suffered pain periodically with an intensity of 48 and 61 mm on the VASPI, respectively. The patient with the left brain infarction was dissatisfied with the cosmetic result and scored 43 mm on the VASC.

Another female patient with a history of left brain infarction noted a suboptimal cosmetic result and scored 51 mm on the VASC, but suffered no pain. One patient scored 71 mm on the VASC. Clinical evaluation and imaging did not reveal any objective reasons for impaired cosmetic results such as asymmetry, swelling, or ill fitting of the plates in these cases. All patients would have chosen to undergo cranioplasty again, noting a considerable improvement in their quality of life following calvarial reconstruction.

Despite artifacts, follow-up imaging of the 4 patients undergoing removal of meningiomas was possible. Nevertheless, the quality of follow-up images in these patients was regarded as suboptimal. Imaging quality was acceptable in the follow-up of the other cases. Extraaxial collections without midline shift were seen on postoperative CT scans in 4 cases, but no surgical intervention was required in the clinically unaffected patients.

The operation time ranged from 60 to 219 minutes (mean 118 minutes). The costs for the titanium plates, including instances in which 2 plates were used, ranged from €2500 to €5050 (mean €3733).

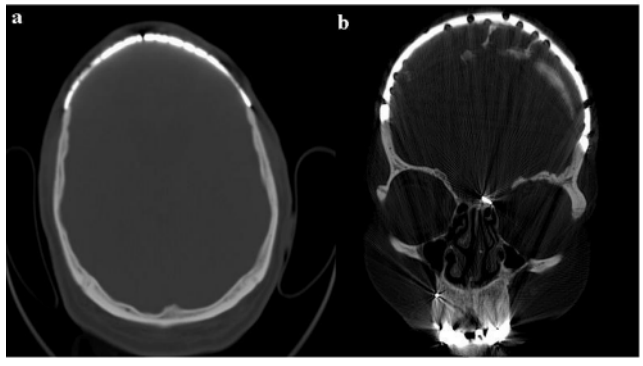

Fig. 3. Axial (a) and coronal (b) CT reconstructions obtained in a 62-year-old patient with the history of a severe head injury and frontobasal fractures with a subsequent infection of the bifrontal bone flap.

None of the implanted titanium plates had to be removed. Due to a postoperative hypertrophy of the temporal muscle and a resulting asymmetry of the face 4 months after insertion of the titanium plate, a reduction of the temporalis muscle was performed in 1 case. In this case, a transient palsy of the frontal ramus of the facial nerve occurred postoperatively.

Discussion

We presented a long-term follow-up study of 26 patients after placement of CAD/CAM titanium for cranioplasty. None of the plates had to be removed, and almost 90% of the patients were satisfied with the cosmetic result and overall outcome. The cosmetic and overall outcomes of our patients did not differ substantially from other studies dealing with CAD/CAM implants, but we focused on long-term results, patients' satisfaction, and quality of life. Cranioplasty is the surgical correction of skull defects and has 2 main purposes: protection of the brain and a satisfying cosmetic result. Furthermore, cranioplasty affects cerebral metabolism positively and may facilitate patient rehabilitation.[45,46]

Because of issues of immunocompatibility, a patient's own bone flap is considered the material of choice for a cranioplasty, and we support this point of view. If the bone flap is lost to osteolysis or infection, autologous bone from other parts of the body is rarely used for calvarial reconstruction because of donor site morbidity and shaping problems. In these cases alloplastic implants are more frequently used. The decision for one of the many available materials often depends on the surgeon's preference and experience as well as costs and availability of modeling techniques. The most frequently used cranioplasty materials are PMMA, HA, and titanium.

Because of its good biocompatibility and low costs, PMMA is the most frequently used alloplastic material and is still regarded as the material of choice by many authors.[26,29] Nonetheless, depending on anatomical conditions as well as the size and shape of the skull defect, intraoperative modeling can be time-consuming and difficult. The disadvantage of inappropriate modeling especially in large skull defects and sensitive cosmetic regions has been solved by the CAD technique, which can be used for PMMA with good results. The CAD/CAM PMMA implants are an acceptable choice even in poorer regions of the world.[9,18] Recently, bioactive composite materials consisting of acrylic resins gained access into calvarial reconstruction with good results.[38]

Hydroxyapatite is probably the most frequently used ceramic and is increasingly used in reconstructive surgery. It is the principal component of bone, has the advantage of osteoconductivity, and allows osteointegration.[6,25] Despite high biocompatibility, inflammatory reactions have been described in numerous studies conducted in

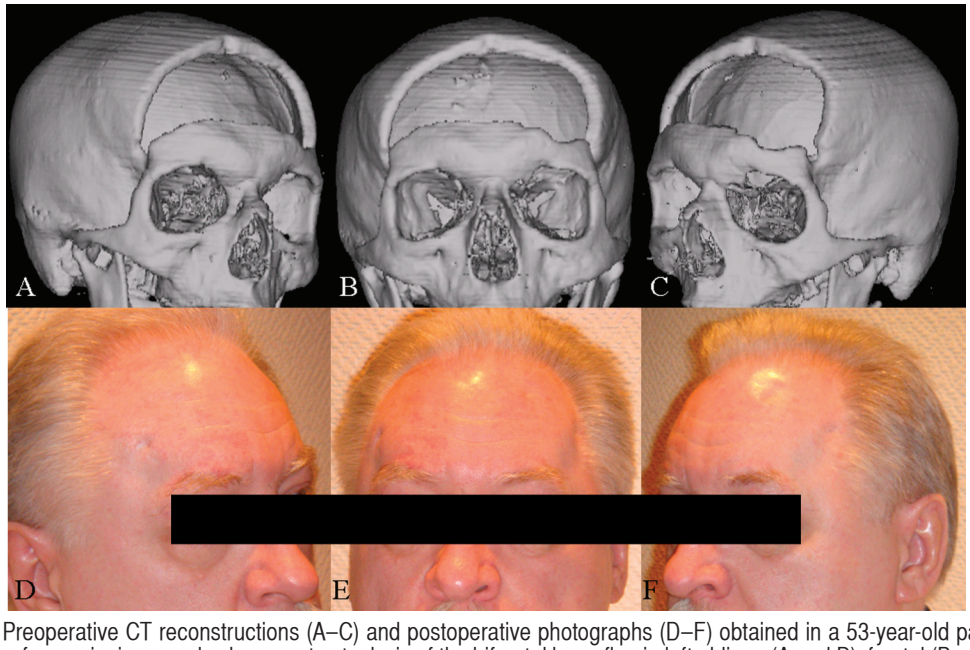

Fig. 4. Preoperative CT reconstructions (A–C) and postoperative photographs (D–F) obtained in a 53-year-old patient with the history of a meningioma and subsequent osteolysis of the bifrontal bone flap in left oblique (A and D), frontal (B and E), and right oblique (C and F) views.

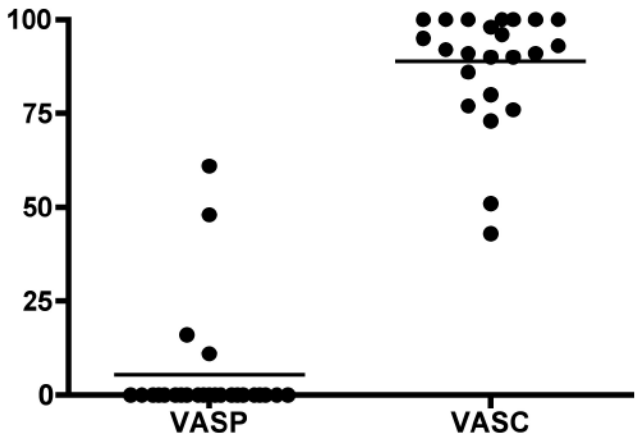

Fig. 5. Graph showing the clinical results according to the VASPI and VASC.

the early postoperative period and the few months after surgery. Thus, some authors have regarded HA as a contraindication for craniofacial reconstruction and pediatric populations in special settings.[6,13,15,27,29] Costantino et al.[6] managed to attain a 0% infection/removal rate, but their small collective comprised mostly patients with small skull defects that occurred after the suboccipital lateral approach for vestibular schwannomas. Furthermore, the costs of HA and other ceramic CAD/CAM implants exceed even CAD/CAM titanium implants.[8]

Carbon and PEEK are other biocompatible materials that provide high strength and radiolucency for postoperative imaging. They have been put to use particularly in orthopedic and spinal surgery with good clinical and radiographic results.[3] In a series of 29 patients, not a single CFRP plate had to be removed.[39] The CAD/CAM PEEK implants for cranioplasty are gaining access in calvarial reconstruction,[42] but larger case studies have not yet been reported in the literature.

Bioactive materials are gaining greater importance and exhibit superior characteristics to classic allografts in biomechanical studies and small clinical short-term settings,[35,38] but they still have to stand the test of time. Osteoconductive bioresorbable materials, tissue engineering,[36,37,40,41] osteoinduction by growth factors, and gene therapy[12,17,31] are only recently gaining access in larger clinical settings. While there are studies in which authors have preferred PMMA or HA as the optimal material for cranioplasty, titanium plates offer a good choice for cranioplasty based on their strength, biocompatibility, handling characteristics, and suitability for postoperative imaging techniques.[5,21–23]

While materials such as PMMA and HA show biocompatible and osteoconductive characteristics without significant toxic and immunogenic properties, ~ 4–13.8% of PMMA plates[26,43] and 2.6–10% of HA-based implants need replacing.[10,11,44] The best HA series with a complication rate of < 2.6% involved only cranioplasties < 6 cm following a retrosigmoid approach in most cases.[34] In a small series of 9 patients undergoing large skull defect reconstruction, 22% of HA-based ceramics had to be removed because of infections.[8] The infection rate of titanium implants including large skull defects ranges from 0 to 4.5%.[2,11,21,23] An often-cited reason for the removal of HA and PMMA implants is the proximity to the sinuses, a problem that exists for cranioplasty with titanium as well. In our own study, the frontal sinus was involved in 6 cases.

The costs of large ceramic plates amount to ~ $7000,[8] while the costs of large titanium CAD/CAM implants range from €2050 to 5000[11] and are thus comparable in cost to the CAD/CAM implants used in our series. As patients requiring alloplastic cranioplasty have often already undergone multiple operative procedures, one has to question whether titanium CAD/CAM cranioplasty is actually more expensive than PMMA cranioplasty. In our view, this statement does not take into account the more frequent revision surgery due to higher complication rates of PMMA and the resulting costs. Furthermore, results from clinical series with HA cranioplasty show higher complication rates and higher production costs than titanium cranioplasty.

The suboptimal results of imaging quality due to titanium artifacts for follow-up of the meningiomas in our study cannot be denied. Nevertheless, imaging quality was acceptable in the follow-up of the other cases.

Conclusions

With the aid of CAD technology, all of the currently used alloplastic materials are good choices even for large skull defect cranioplasty. Cranioplasty with CAD/CAM titanium plates is suitable for calvarial reconstruction of all sizes, providing the lowest complication rate, reasonable costs when regarding complications and removal rates over long-term periods, and the possibility of acceptable postoperative imaging. Analyzing our data and the literature, we have come to the conclusion that due to its costs and availability, PMMA is suitable for patients requiring primary cranioplasty or long-term follow-up imaging of tumors. Titanium implants seem to be the material of choice in cases of secondary cranioplasty of large skull defects resulting from decompressive craniectomy after trauma or infarction. Expensive ceramics that are gaining larger access into reconstructive calvarial surgery show no obvious advantage over titanium or PMMA.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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