Decompressive craniectomy for space-occupying supratentorial infarction: rationale, indications, and outcome

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A subset of patients with ischemic cerebrovascular stroke suffer a progressive deterioration secondary to massive cerebral ischemia, edema, and increased intracranial pressure (ICP). The evolution is often fatal. In these patients, a decompressive craniectomy converts the closed, rigid cranial vault into an "open box." The result is a dramatic decrease in ICP and a reversal of the clinical and radiological signs of herniation. For these reasons, decompressive craniectomy has been increasingly proposed as a life-saving measure in patients with large, space-occupying hemispheric infarction. The authors review the rationale, indications, and clinical experience with this procedure, which has been performed in patients who have had supratentorial ischemic stroke.

KEY WORDS • craniectomy • ischemic stroke • decompressive surgery

Recent successes with intravenous³² and intraarterial¹⁴ thrombolytic therapy have resulted in an increased awareness of stroke as a medical emergency.³ Thus, increasing numbers of patients are being evaluated in the early hours following the ictal event. In the process of gaining more experience in the early management of patients with acute ischemic stroke, it has become clear that in a number of these patients a progressive and often fatal deterioration secondary to mass effect from the edematous, infarcted tissue occurs. An increasing body of experimental and clinical evidence suggests that some of these patients may benefit from undergoing a decompressive craniectomy. However, the timing and indications for this potential life-saving procedure are still debated. In the present article we review the rationale, current indications, technique, and outcome of decompressive craniectomy for space-occupying supratentorial infarction.

HISTORICAL BACKGROUND

Decompressive craniectomy procedures have been used to relieve increased ICP and cerebral edema caused by a variety of pathological events. In a classic article in

Abbreviations used in this paper: CT = computerized tomography; CVA = cerebrovascular accident; ICA = internal carotid artery; ICP = intracranial pressure; MCA = middle cerebral artery.

1905, Cushing reported the use of this procedure to relieve the pressure caused by the growth of an intracranial tumor.⁷ Since then, surgical decompression has been reported as a treatment option for traumatic head injury,^{16,18,29,34} subdural hematoma,^{6,35} edema resulting from vasospasm secondary to subarachnoid hemorrhage,¹² encephalitis,^{26,42} intracerebral hematoma,⁹ cerebral venous and dural sinus thrombosis,⁴⁶ cerebellar infarction,^{5,20,30,31,38} and supratentorial cerebral ischemia.^{4,8,21,22,27,28,37,39,44,51}

In the 1950s and 1960s, an increasing number of reports were published in which the authors described cases of massive cerebral ischemia accompanied by acute and severe brain swelling.^{1,25,45,47} These cases were often fatal, with the edema caused by the infarct producing a "pseudotumor" increase in pressure within the cranial vault. In 1968, Greenwood¹⁷ used surgical intervention in the treatment of such cases, which decreased the mortality rate to below 50%. In Greenwood's series of nine patients with acute infarction involving the MCA or ICA, decompressive hemicraniectomy as well as resection of the necrotic parenchyma were performed.¹⁷ Six of these patients survived, although three suffered postoperatively from severe disability.¹⁷ In their classic report in 1971, Kjellberg and Prieto²⁷ described a bifrontal decompressive craniectomy procedure for the treatment of a massive infarction. In that case, however, the patient did not survive. In 1981, Rengachary and coworkers³⁷ reported the first cases in which straightforward craniectomy was undertaken, without re-

TABLE 1

*Summary of clinical studies in which decompressive craniectomy was completed in patients with massive supratentorial infarct**

Authors & Year	No. of Cases	Time to Surgery	Tissue Re-section	Mean Patient Age (yrs)	Outcome
Greenwood, 1968	9	1–21 days	yes	49.8	6 survivors: 3 w/ severe disability, 3 w/ moderate & acceptable neurological deficit
Kjellberg & Prieto, 1971	1†	not given	no	not given	death
Ivamoto, et al., 1974	1	3 days	yes	49	mild disability; depression
Rengachary, et al., 1981	3	2–5 days	no	31	2 w/ persistent hemiplegia (1 severely disabled); one w/ only a mild hemisensory deficit
Young, et al., 1982	1	2 days	yes	59	hemiparesis
Kondziolka & Fazl, 1988	3	1–5 days	no	43.7	3 w/ independent function & mild deficits
Delashaw, et al., 1990	9	< 1–7 days	no	57	1 died of nonneurological causes; 8 survived (BI scores: 4 60–95, 4 < 60) 5 patients would undergo op again w/out hesitation; 3 would undergo again w/ some reservation
Jourdan, et al., 1993	7	2–3 days	no	43.8‡	5 survivors: all independent, 4 w/ residual hemiparesis interfering w/ ambulation, 2 w/ residual aphasia but normal comprehension; depression present in 2
Rieke, et al., 1995	32	1–5 days (mean 39 hrs)	no	48.8	21 survivors; mean BI = 62.6: (15 ≥ 60, 1 excellent, 5 ≤ 60); of 6 survivors w/ dominant hemisphere infarct, 5 w/ incomplete aphasia; mean ICU stay 13.3 days
Carter, et al., 1997	14	< 1–19 days	yes	49.2	3 died of nonneurological causes; mean BI = 63 (8 > 60, 3 < 60); mood disturbances were significant; all had problems w/ social reintegration; 6 would undergo op again, 3 maybe, 2 would not
Schwab, et al., 1998	31	< 1 day (mean 21 hrs)	no	50.3	26 survivors; mean BI = 68.8 (range 60–90) mean ICU stay 7.4 days
Yoo, et al., 1999	2§	not given	no	29	good outcome on Glasgow Outcome Scale in both

* Cases associated with concomitant disease such as an intracranial aneurysm were excluded. Abbreviations: BI = Barthel Index; ICU = intensive care unit.

† A bifrontal craniectomy was performed.

‡ The age of one patient was not reported.

§ A bilateral craniectomy was performed.

removal of necrotic brain tissue. Since that study, at least an additional 99 cases of hemicraniectomy and two of bilateral craniectomy have been reported in the treatment of massive cerebral ischemia (Table 1).

“MALIGNANT” CEREBRAL INFARCTION

Ischemic cerebral infarction is associated with a high rate of morbidity and mortality. Mortality rates are highest when lesions involve the trunk of one or more of the main cerebral vessels. In fact, occlusion of either the distal ICA or proximal MCA trunk has been characterized as a “malignant” stroke in both clinical¹⁹ and animal^{10,11} studies.

Of all cases with supratentorial infarctions in which an autopsy is performed, 13% are shown to suffer from severe brain swelling after an infarction involving the entire distribution of the ICA or MCA.³³ Severe cerebral edema can lead to herniation of cerebral structures through the tentorium or falx, as well as the brainstem structures through the foramen magnum. In fact, transtentorial herniation has been cited as the probable cause of death in many of these cases of malignant stroke. Bounds, et al.,² have reviewed 100 autopsy cases of patients in whom an infarction involving the ICA distribution had been diagnosed. Thirty-one patients died of tentorial herniation, which was the only neurological cause of death in all the cases reviewed.²

The prognosis for patients who suffer a “malignant” CVA is poor, with death occurring usually within the first 4 to 5 days. In this subset of patients, a mortality rate of 78% was observed by Hacke, et al.¹⁹ All deaths were at-

tributed to transtentorial herniation, which occurred within 2 to 7 days (median 4 days).¹⁹ Similarly, Schwab and coworkers⁴¹ have found that 81% of patients with malignant CVA died, and all deaths occurred within 5 days and were caused by herniation. Given the poor prognosis in these patients, it is of critical importance to recognize imaging or clinical characteristics suggestive of such a progressive and rapid deterioration.

In patients who suffer a malignant CVA the clinical course is generally predictable. Presenting symptoms may include the sudden onset of hemiplegia, homonymous hemianopsia, forced eye and head deviation toward the lesion side, and aphasia.^{19,33,37,40} Precipitous coma and pupillary dilation usually occur together following the initial symptoms.^{4,8,33,37,51} In the absence of further intervention, death occurs.

To establish objective criteria for aggressive intervention, many investigators have measured ICP once significant clinical deterioration is apparent. In an early study the authors showed that patients in whom ICP values were greater than 15 mm Hg did not survive the malignant infarct.⁴⁰ In subsequent studies other authors have shown that a fatal outcome occurred in most cases when the level was greater than 30 mm Hg¹⁹ or 35 mm Hg.⁴¹

In addition to clinical findings, neuroimaging criteria can help to identify those patients at particular risk for a malignant infarction in the early phase of their stroke. In patients with malignant CVA, a large area of parenchymal hypodensity in the MCA territory is often visualized on the admission CT scans.^{39,43,44} With progressive clinical deterioration, CT-demonstrated signs may also include

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mass effect, effacement of the basal cisterns, compression of the ventricular system, a shift of midline structures,⁴⁹ and herniation of tissue through the falx, foramen magnum, or tentorium.³³

RATIONALE FOR DECOMPRESSIVE CRANIECTOMY AND EXPERIMENTAL STUDIES

Cerebral ischemia results in edema formation in and around the ischemic area. The larger the area of the infarction, the greater the extent of edema. In the case of malignant CVA, the entire vascular distribution of the MCA, and possibly the anterior cerebral artery, is compromised. A severe edematous response ensues throughout a large area.^{4,33} Edema is responsible for the parenchymal hypodensity that is demonstrated on CT scanning.^{8,37,39,43}

Progressive brain edema and the exacerbating effect it has on increasing ICP can cause the area of damaged brain to extend. Within the confined cranial vault, the edematous tissue places pressure against surrounding normal parenchyma. This is evidenced by the changes seen on CT scanning. Intracranial hypertension results in decreased cerebral perfusion pressure and therefore decreasing blood supply throughout the cerebrum. Because of the increase in mechanical pressure and ICP, other major cerebral vessels may be compressed by the expanding tissue, against dural edges or against the skull. The result is secondary ischemia and a further expansion of the infarcted area.³

Although originally proposed as a life-saving procedure, increasing experimental and anecdotal clinical evidence indicates that an early decompressive craniectomy can limit the extension of the infarcted area. From a mechanical perspective hemicraniectomy provides an immediate opening in the otherwise closed cranial vault. Therefore, compression of normal tissue and the penumbra by the infarct area is prevented or limited.⁴⁴ The additional space created allows the tissue to expand through the bone defect, away from midline structures,²⁴ so that CT-demonstrated changes normally observed when surgery is not performed (that is, midline shift, decreased ventricular size, and herniation) are minimized or completely resolved postoperatively.^{4,8,22,28,37}

Because the cranial vault has essentially been expanded during surgery, there is an immediate decrease of ICP. Jourdan, et al.,²² have found that initial ICP values of 25 to 60 mm Hg decreased by 15% once the bone flap was removed, and by 70% once the dura was opened, resulting in the normalization of the ICP after surgery. Similar findings were demonstrated in a more recent study in which the authors reported performing a bilateral craniectomy.⁵⁰ In two patients with ischemic CVA whose initial ICP values were 54.8 mm Hg and 20 mm Hg, respectively, removal of the bone flap caused a decrease in ICP to 35.5 mm Hg and 10 mm Hg, and opening of the dura caused a reduction to 4.4 mm Hg and 3 mm Hg, respectively.⁵⁰ In the immediate postoperative period, the ICP values were recorded as 4.4 mm Hg and 10.2 mm Hg, respectively.⁵⁰ A decrease in ICP allows for an increase in cerebral perfusion pressure, aiding blood flow to the ischemic penumbra³⁹ and optimizing circulation to the damaged area through collateral vessels.

Because hemicraniectomy alone may improve blood flow to the penumbra, surgical resection of the infarcted tissue should not be conducted in these patients. Although such resection or "strokectomy" has been associated with postoperative improvements in some cases,²³ it is impossible to differentiate at surgery between ischemic tissue (potentially salvageable) and necrotic tissue (irreversibly compromised).³⁶ Being poorly delineated from necrotic tissue, the penumbra may possibly be damaged or removed upon resection of the infarct.

Timing and Indications for Decompressive Craniectomy

Historically, hemicraniectomy has been used as a last resort to prevent impending death after all medical therapies have been attempted. The surgical procedure certainly preserves life, as evidenced by decreased mortality rates when compared with patients who undergo medical therapy alone.^{4,28,37,39,44} In many of the reported cases, the symptoms of a frank herniation syndrome—fixed, dilated pupils; precipitous coma; cardiorespiratory difficulties; and decerebrate posturing³³—were used to indicate the need for decompressive surgery.^{8,17,21,37}

Patients suffering malignant CVA receive antiedema medical treatment, such as steroids, mannitol, and hyperventilation, before consideration is given to a decompressive craniectomy. Usually, an initial reversal of symptoms, such as the degree of pupillary dilation, occurs with aggressive medical treatment. After its initial effectiveness, however, additional medical therapeutic efforts often fail to control or prevent herniation. In the case of massive cerebral ischemia, the effectiveness of such medical therapy is severely limited, at best, as evidenced by the high mortality rates observed in the absence of surgical intervention. As Rengachary, et al.,³⁷ have noted in the case of stroke, which is typically not treated surgically, physicians may wait too long to intervene surgically. Once the pupils are fixed and a deep coma has indicated an irreversible decline of cerebral function, surgery should not be performed.

Evaluation of experimental findings suggests that aggressive, early surgical decompressive surgery for the treatment of massive cerebral ischemia may limit the extension of the infarction and reduce morbidity.^{10,13} Forsting, et al.,¹³ have demonstrated that craniectomy can decrease the infarct volume and improve neurological outcome in a rat model of MCA occlusion when surgery is completed early (1 hour postictus). Doerfler, et al.,¹⁰ have found similar results in the same model when surgery was completed 4 hours postictus. In the 4-hour treatment group, outcome and infarct volume were significantly better as compared with those observed in control animals and animals surgically treated at 12, 24, and 36 hours postictus.¹⁰ Animals treated at these later time periods improved, but no significant differences were reported among these three groups and the control group.¹⁰ The results of recent clinical studies support this notion.⁴⁴ When patients who suffer malignant CVA were surgically treated on average 21 hours postictus, there was a greater decrease in mortality rate and length of stay in the intensive care unit as compared with patients who underwent surgery an average 39 hours postictus.⁴³ There was also a trend of improved Barthel Index scores demonstrated at follow up for patients in the earlier surgical group.⁴⁴

Several factors need to be considered to optimize both the timing and the indication for decompressive craniectomy.

Intracranial Pressure Monitoring

To determine when surgery should be performed, ICP monitoring has been recommended as a guide to surgical timing.⁵¹ Carter, et al.,⁴ have used an ICP measurement of greater than 25 mm Hg, and Rieke, et al.,³⁸ a measurement of greater than 30 mm Hg, despite attempts at medical therapy, as an indicator for surgical intervention. Increased ICP measurements are preceded by the constellation of clinical signs and symptoms constituting the "malignant CVA syndrome;" thus, the usefulness of ICP monitoring in these cases has been questioned.⁴¹

Neuroimaging Studies

Neuroimaging is fast becoming the most promising prognostic tool with which to judge the appropriateness of surgery in the subset of patients with malignant CVA. Extensive MCA infarction with edema in greater than 50% of the MCA territory can be identified early after the ictal event on CT scans, and it is observed on the initial CT scan in approximately 69% of the reviewed cases.^{19,44} Parenchymal hypodensity in greater than 50% of the MCA territory is highly indicative of a progressive clinical course, leading to severe morbidity or death.⁴⁸ With current, newer CT scanners, parenchymal hypodensity can be seen and followed soon after symptom onset.

A midline shift of the cerebral structures is another phenomenon of increasing unilateral cerebral edema that can be identified on CT scanning. Rieke, et al.,³⁹ have found that the amount of midline shift was significantly different between survivors and nonsurvivors of malignant CVA. In a recent study conducted to examine only the prognostic value of midline shift, it was suggested that at 32 hours after the occurrence of cerebral infarction, a shift of the third ventricle greater than 4 mm was indicative of a fatal outcome.¹⁵ Regardless of its potential for prognostic significance, however, midline shift is not visualized as early on CT scanning as is parenchymal hypodensity.⁴⁸

Early changes demonstrated on CT scans are also an indicator of the viability of collateral circulation. In their study von Kummer, et al.,⁴⁸ performed cerebral angiography in patients in whom stroke was demonstrated early with CT scanning. Comparing the angiographic findings with those obtained using CT scanning, the authors observed that parenchymal hypodensity in greater than 50% of the MCA territory was predictive of poor collateral circulation, as evidenced by the angiographic study.⁴⁸ These findings are important, because in patients with adequate collateral circulation, decompressive hemicraniectomy may not be necessary. Continued refinements of newer imaging techniques such as diffusion/perfusion magnetic resonance will lead to an earlier identification of those patients more likely to benefit from early decompressive craniectomy.

Patient Age

The age of patients undergoing surgical intervention reported in the reviewed series ranged from 11 to 70 years

of age. Based on data provided in the literature, it was impossible to determine if a certain age range of patients benefits more from surgical decompression. Most investigators, however, noted that they are more aggressive in performing hemicraniectomy in young patients in whom CVA has occurred^{4,8,17} and that the young seemed to benefit more from the procedure.^{4,39} Carter and coworkers⁴ specifically divided their patients by age in those younger than and older than 50 years of age. They found that five of five patients under the age of 50 years made a good functional outcome (Barthel Index scores > 60 [100 = independent, 60–95 minimum assistance, and < 60 = dependent]), whereas only in three of six patients over 50 years of age was this observed at follow-up examination.⁴

In theory and in practice, it would seem that younger patients with ischemic stroke would benefit from early decompressive surgery for the following reasons. 1) Their brains are less atrophied, allowing less room for edematous expansion within the cranial vault.^{4,28,37} Individuals aged 50 years and younger have been identified as benefiting more from bilateral decompressive craniectomy in cases of subarachnoid hemorrhage because of their unatrophied brains, as compared with those over 50 years of age.¹² 2) The ventricular system in younger persons is smaller than in older persons.^{28,37} 3) It has been proposed that the edematous response to ischemia is greater in younger individuals.^{28,37}

Dominant Hemisphere Infarction

As a rule, investigators in the past did not undertake surgery in patients with dominant hemisphere infarctions. The loss of communicative abilities and a plegic dominant upper extremity were judged to be too damaging.¹⁷ Analysis of recent evidence suggests that considering a dominant hemisphere infarction to be a contraindication to surgery may be too harsh a criterion. A recent study included seven patients who experienced massive dominant hemisphere CVA and who underwent hemicraniectomy.³⁹ One of those patients died. Good functional outcome was achieved in the remaining six patients (Barthel Index scores ranging from 60–100). All surviving patients were able to understand and communicate despite some aphasia, and one patient suffered no aphasic deficits. In the most recent series involving five hemicraniectomy-treated patients who suffered dominant hemisphere CVA, no patients were diagnosed as being globally aphasic at follow up.⁴⁴ Three patients sustained communication deficits that were so minor as to allow them to return to work. Functionally, the patients with the dominant hemisphere infarct who underwent hemicraniectomy were not significantly different from those patients who underwent craniectomy after CVA in the nondominant hemisphere. Therefore, surgery can be considered in patients with dominant hemisphere infarction, especially if some residual language function is present at admission. This delicate issue, however, warrants further investigation.

DECOMPRESSIVE CRANIECTOMY: TECHNIQUE, RESULTS, AND COMPLICATIONS

Operative Technique

Hemicraniectomy for supratentorial infarction usually

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involves aggressive bone removal to alleviate better the symptoms of malignant cerebral edema. The need for a radical approach (in terms of extension of bone removal) was recognized by Kerr²⁴ in the event of severe posttraumatic cerebral edema. His observation was echoed in the case of massive cerebral ischemia by Rieke and coworkers³⁹ who found that a few of their initial surgically treated patients harbored a bone defect that was too small, not providing adequate space for decompression and resulting in brain herniation through the skull opening. Prolapse of the edematous brain through the edges of the craniectomy defect, with possible exacerbation of brain damage is one of the possible limitations of decompressive craniectomy. In the case of cerebral infarction, however, this phenomenon does not result in significant increased cerebral damage or venous stasis, because most likely the protruding tissue is already necrotic.³⁷

In the event of massive cerebral ischemia, the frontal, temporal, and parietal bones overlying the infarcted hemisphere are removed.^{4,8,17,37} The dura is incised and reflected. A dural expansion graft of pericranium,⁴ lyophilized cadaver dura,^{8,39} homologous temporal fascia,³⁹ or Silastic³⁷ is loosely sutured to the dura edges to prevent cortical adhesions. The dura is fixed to the craniectomy edges to prevent or limit epidural bleeding,³⁹ and the temporal muscle and skin flap are reapproximated and sutured or stapled into place. The bone flap may be frozen^{8,37} or preserved in a subcutaneous pocket overlying the abdomen.⁴ Cranioplasty is then performed at a later date, when functional recovery has stabilized. Artificial flaps have also been used to close the bone defect.^{22,39}

RESULTS OF HEMICRANIECTOMY

Although increasing numbers of studies have reported encouraging results after decompressive craniectomy for ischemic stroke, these studies are mostly limited to case series without a control group. A summary of some of these studies is shown in Table 1. Rieke and coworkers³⁹ have recently reported the results of a prospective, non-randomized, single-institution, control study involving 32 patients selected for surgical treatment and 21 patients with space-occupying cerebral infarction treated medically. Entry criteria into the control group included extensive left hemispheric infarction presenting with global aphasia on admission (14 patients), additional severe medical complications (two patients), and lack of informed consent from the relatives (five patients). Although other clinical characteristics did not differ significantly between the two groups, patients in the control group were older (mean age 58.4 years) than patients who underwent surgical treatment (mean age 48.8 years). In this study, the mortality rate was 76.2% in the control group and 34.4% in the surgically treated group. Functional outcome, as reflected by the Barthel Index scores obtained 4 to 36 months after surgery, was consistent: excellent level of activity in one patient, minimal assistance required by 15 patients, and a severe disability in five patients. In the control group, four of the only five survivors had global aphasia.

Because decompressive craniectomy can be a life-saving procedure in patients who will most likely be left with a significant neurological deficit, the operation has important ethical and psychological implications. Because of

their altered level of consciousness, patients cannot directly provide consent. In such cases, informed consent has to be obtained from the relatives. This occurs in a stressful situation, and a difficult question is posed to the family regarding the life of their loved one. Psychological disturbances in this patient population were addressed by Carter and coworkers.⁴ In their series of 14 patients who underwent decompressive craniectomy after right-sided hemisphere ischemic stroke, they found mood disturbance to be significant in all survivors. Four of 11 patients suffered severe depressive symptoms, whereas in the remaining seven mild to moderate impairment was demonstrated. When asked if they would have chosen to undergo the operation given the choice, six patients answered affirmatively, three were uncertain, and two would have declined the operation. Additional studies will have to be mounted to analyze in more detail these implications.

SURGERY-RELATED COMPLICATIONS

Few complications have been reported in the literature when hemicraniectomy has been completed after cerebral infarction. Although postoperative epidural and subdural hemorrhage³⁹ as well as hygromas⁴⁴ have occurred in a few cases, none of these complications led to additional neurological deficits.

CONCLUSIONS

Malignant cerebral ischemia occurs in a significant number of patients who undergo emergency evaluation for ischemic stroke. The mortality rate in these patients is very high. Fatal outcome is usually related to progressive, severe cerebral edema with brain herniation and compression of critical brainstem structures. This patient population can be identified by early clinical and neuroimaging characteristics. In some of these patients, decompressive craniectomy appears to be a life-saving procedure. If craniectomy is performed early, especially in young patients, a satisfactory functional outcome can be achieved in a significant proportion of cases. Questions persist regarding the indications for such a procedure in patients with dominant infarctions. Anecdotal clinical experience, however, demonstrates that even in such patients, an acceptable functional outcome can be achieved after surgery if some preservation of speech is present at the time of intervention.

Future studies should better define the indications for and the timing of the procedure. In these studies the authors should also investigate the possible ethical implications and the overall level of satisfaction in survivors whose lives have been preserved by craniectomy but who are often left with significant neurological impairment. Ideally, a prospective, randomized, multiinstitutional trial would provide the definitive answers to these important questions.

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