Treatment of Right Hemispheric Cerebral Infarction by Hemicraniectomy

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An anecdotal series of nine patients (three men and six women with an average age of 57 years) presented with progressive neurologic deterioration while on medical therapy for large right hemispheric cerebral infarction. Clinical signs of uncal herniation (anisocoria or fixed and dilated pupils, and/or left hemiplegia with right decerebrate posturing) were present in seven of these nine patients. Computerized tomography of the head confirmed mass effect from cerebral edema. It was the clinical judgment of the treating neurologists and neurosurgeons that each of these nine patients would perish unless surgical decompression of the infarcted brain was performed. Accordingly, each was treated with right hemicraniectomy and dural augmentation. Six patients demonstrated neurologic improvement on the first postoperative day. One patient, with a postoperative diagnosis of lung cancer, died 1 month after surgery. The remaining eight patients are currently living with their families with a follow-up period ranging from 5 to 25 months. Patient outcome as evaluated by the Barthel Index indicates that three individuals are functioning with minimal assistance and that the remaining six patients are functionally dependent. After rehabilitative therapy, four patients returned for elective cranio-plasty. These results suggest that hemicraniectomy can be an effective lifesaving procedure for malignant cerebral edema after large hemispheric infarction. (Stroke 1990;21:874--881)

Stroke is the most prevalent disease involving the central nervous system. During the acute period following a cerebral infarction, current medical management is primarily supportive to prevent extension of the infarct or the development of cardiopulmonary complications. Neurologic decline is often attributed to surrounding edema during this acute infarction period. Fortunately, as the edema resolves, the patient frequently improves. However, massive unilateral hemispheric cerebral edema can develop from internal carotid or middle cerebral artery occlusion and can result in uncal herniation and death. Contemporary medical modalities involving steroids, hyperventilation, barbiturates, mannitol, or other antiedema agents are frequently ineffective in reducing acute cerebral edema following infarction. In those stroke victims with massive unilateral hemispheric edema, surgical decompression by hemicraniectomy may be an effective alternative when medical modalities fail. In this study we present an anecdotal series that describes the results of hemicraniectomy on nine consecutive patients presenting with progressive neurologic deterioration while on medical therapy for spontaneous large right hemispheric infarctions.

Subjects and Methods

Patients experiencing spontaneous acute cerebral ischemia and/or infarction were admitted to the medical, neurologic, or neurosurgical services at the University of Virginia, Charlottesville. These patients were followed by a stroke service comprised of a neurovascular neurologist, a neurosurgeon, and other health care professionals. Stroke was diagnosed by clinical history, physical examination, and head computed tomography (CT). Patients diagnosed with stroke were treated aggressively with adequate hydration and oxygenation. When necessary, stroke patients were observed in an intensive care unit setting, intubated, and ventilated. To avoid anticoagulation therapy, 325 mg aspirin was given daily to patients demonstrating findings of large cortical infarctions. If a stroke victim began to demonstrate neurologic decline, hyperventilation and antiedema agents, such as mannitol and steroids, were implemented. Cerebral hemorrhage and hydro-
cephalus were excluded as the etiology of neurologic decline in these patients by emergency repeat head CT. When unilateral cerebral edema as a result of right hemisphere infarction resulted in acute neurologic deterioration, patients were evaluated for right hemicraniectomy. When the clinical judgment of the physicians was that medical therapy was ineffective and a fatal outcome was imminent, then emergency right hemicraniectomy was performed. Surgical decompression of left hemisphere infarction was not performed in this initial series of patients because of concern for the severe disability and poor quality of life caused by aphasia and hemiplegia due to dominant hemisphere injury.

From August 1985 to April 1988, nine stroke victims (three men and six women with ages ranging from 41 to 68 years) with acute right cortical infarctions neurologically deteriorated while on medical therapy (Table 1). One of these patients (case 1) had deteriorated at his local hospital and was rapidly transferred to the University of Virginia hospital for treatment. In seven of these nine patients clinical signs of uncal herniation (anisocoria and/or right decerebrate posturing) accompanied neurologic deterioration. Head CT demonstrated large right middle cerebral distribution infarctions in all nine stroke victims. In addition, right anterior cerebral distribution infarction was demonstrated in five of these patients. The range of time elapsed between onset of symptoms of acute cerebral infarction and further neurologic deterioration was 18–168 hours with a median time of 72 hours. All nine patients were thought to be poorly responsive to aggressive medical modalities and were expected to expire without surgical decompression. Each of these nine stroke victims was treated for extensive unilateral cerebral edema by right hemicraniectomy.

The right cerebral hemisphere was surgically decompressed through a large hemicraniectomy (Figure 1). The bone flap was removed and placed in cold storage for future autologous cranioplasty. Additional bone was removed in the temporal region to the floor of the middle fossa. The dura was opened in a large cruciate incision involving the frontal, parietal, and temporal lobes. As the dura was opened, the pale infarcted brain typically began to herniate outward. An intracerebral hematoma was resected in one patient (case 9) with hemorrhagic infarction. Cortical resection of infarcted brain was not performed in the other eight patients. In all nine patients lyophilized cadaver dura was placed under the incised dura and secured with several sutures to allow the brain to herniate outward in a more controlled manner, as well as to prevent corti-

\[\text{FIGURE 1. Schematic representation of right hemicraniectomy procedure. Left panel: After right hemicraniectomy, the dura is opened by a large cruciate incision to allow the brain to expand outward. The large bone flap is stored in cold storage for future cranioplasty. Right panel: Lyophilized cadaver dura is then placed under the dura and secured with suture; the arrow indicates this placement.}\]
TABLE 2. Patient Perioperative Glasgow Coma Scale Scores

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<td>9</td>
<td>6T</td>
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GCS, Glasgow Coma Scale; T, intubated (unable to evaluate verbal response of GCS).

*Died from lung cancer 1 month after surgery.

Results

Preoperative GCS scores and GCS scores 1 day and 1 week after surgery are shown in Table 2. Six patients improved on the first postoperative day following right hemicraniectomy. Eight of the nine patients demonstrated neurologic improvement on the seventh postoperative day, and all nine patients survived the perioperative period. However, lung cancer was diagnosed in one patient (case 2) after surgical decompression had been performed; despite marked neurologic improvement (GCS score of 14), she died 1 month later from a respiratory arrest while undergoing pulmonary radiotherapy.

Patient outcome was evaluated in the eight surviving patients by the BI score (Table 4). The BI evaluation was performed 5–25 months after right hemicraniectomy. Four patients (cases 1, 3, 5, and 8) were functioning at a level of assisted independence. The remaining four patients required assistance from family members to accomplish typical tasks encountered in their activities of daily living. All eight patients were living at home with their families. Two patients (cases 3 and 8) demonstrated a mild left hemiparesis at the time of their BI evaluation; hemiplegia was found in the other six stroke victims. Family members of each of the eight patients indicated that emotional lability had persisted since cerebral infarction. Finally, subjective assessment of the surgical procedure and its outcome by the patient and a significant other was positive for all eight surviving stroke victims (Table 4). Four of these eight patients have returned for elective autologous bone cranioplasty.

During the same period, four other patients presented with clinical signs of uncal herniation while on medical therapy for large left hemispheric infarction. All four patients were hemiplegic on the right and...
densely aphasic. These patients did not undergo surgical decompression, and all four expired.

Of the nine patients who underwent hemicraniectomy, one (case 3) was a 48-year-old man who had developed left lower extremity weakness the night before admission. The following morning he had awakened with a left hemiparesis. At the time of admission he was lethargic, but arousable and oriented. He had a blood pressure of 160/95 mm Hg, pulse of 70/min, and a right carotid bruit on auscultation. Neurologic exam was remarkable for a GCS of 14, right gaze preference with a left homonymous hemianopsia, and a severe left hemiparesis. Admission head CT demonstrated early signs of infarction in the distribution of the right middle cerebral artery (Figure 2, upper left). He was observed in the neurologic intensive care unit and treated with frequent intravenous mannitol bolus injections. He became increasingly alert but had developed a left hemiplegia with neglect. However, on the third day
after admission, he deteriorated with obtundation, a right dilated pupil, and a GCS of 12. After intubation, the patient was hyperventilated, and an emergency head CT was performed. This demonstrated a large right hemisphere infarct with mass effect and edema (Figure 2, upper right). Right hemicraniectomy with dural augmentation was then performed. Immediately after surgery, he was extubated and placed on mannitol intravenous infusion of 5 mg/hr. His neurologic examination was unchanged on his first postoperative day, but head CT demonstrated a decrease in shift of midline structures (Figure 2, lower left). Over the ensuing days, he made marked improvement; a GCS score of 14 was recorded on the fourth day after surgery. The patient was discharged to home 17 days after hemicraniectomy, being alert, oriented, and hemiplegic on the left without neglect. When he was evaluated 9 months after surgical decompression, he lived at home, walked with a mild left hemiparesis, and required minimal assistance in performing his daily activities (BI score of 90).

Another of the hemicraniectomy patients (case 5) was a 58-year-old right-handed woman who presented on admission with a history of developing...
TABLE 4. Patient Barthel Index Score and Subjective Assessment After Hemicraniectomy

<table>
<thead>
<tr>
<th>Patient</th>
<th>Follow-up (mo)</th>
<th>BI score</th>
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<td>9</td>
<td>17</td>
<td>50</td>
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*BI, Barthel Index. A BI score of 100 indicates a patient who is able to perform without assistance in activities of daily living; a score of 60–95 indicates a patient who requires minimal assistance with daily activities; a score of <60 implies that the patient is functionally dependent.

For subjective assessment patients and their significant others were asked the following question at home after discharge from the hospital: "Considering everything that has happened since the original surgery following stroke (removal of bone), if you had to make the decision to have surgery again, what would your answer be? Please rate your answer on a scale of 1 (yes) to 10 (no)."

Discussion

Although this is a small anecdotal series, hemicraniectomy with dural augmentation appears to be an effective, lifesaving method of treating malignant cerebral edema secondary to stroke. The surgical decompressive procedure should be considered an additional mode of therapy when contemporary medical therapy fails to curtail cerebral edema in stroke victims. All patients treated by hemicraniectomy survived, but neurologic outcome was variable. Neurologic outcome following this procedure appears to be contingent on the location and extent of the infarction and may also depend on the timing of the surgery. Patients demonstrating neurologic decline while on medical therapy may benefit more from hemicraniectomy if surgery is performed before clinical signs of uncal herniation.

Cerebral swelling following infarction results from cytotoxic and vasogenic edema. Severe brain ischemia initially produces cytotoxic edema without apparent disruption of the blood-brain barrier. Early ischemia disturbs the regulatory mechanisms within cell membranes and results in the accumulation of intracellular fluid. During this acute period, intravascular injections of protein-bound dyes fail to extravasate into the area of ischemia, and the infarct typically is not visible on CT. However, as processes due to the infarction progress, protein-bound fluids are able to diffuse across a damaged blood-brain barrier and to produce vasogenic cerebral edema.

Severe brain edema following cerebral infarction is a known cause of death in the acute infarction period. Ng and Nimmannitya performed postmortem examinations of 353 consecutive cases of supratentorial cerebral infarction. Severe brain edema was observed in 45 of these patients. Acute brain edema associated with transtentorial herniation was attributed as the cause of death in 35 of these 45 stroke victims. A high mortality rate has also been described in stroke victims with large hemispheric infarctions who present with neurologic deterioration. Ropper and Shafman described 12 patients who deteriorated during their hospitalization for acute large cerebral infarction. All 12 patients became drowsy, nine developed asymmetric pupils or decerebrate posturing, and eight died. The cause of death in seven of the patients was attributed to cerebral edema; the other patient died as a result of systemic complications. As demonstrated by our nine patients, cranectomy with dural augmentation can effectively reduce mortality resulting from cerebral edema secondary to stroke.

Surgical decompression with removal of infarcted brain has previously been shown effective in selected cases with cerebellar infarction. Several case reports have also suggested that cranectomy may be an effective means of treating edema secondary to supratentorial infarctions. Rengachary et al described three patients with acute right cerebral infarction and uncinal herniation who were treated by hemicraniectomy. All three of their patients survived. Two of these patients were left with severe neurologic deficits including hemiplegia; the other patient, 15 years old, recovered with normal mentation and...
speech and a mild left hemiparesis. Young et al.11 also described a case of a 59-year-old woman who deteriorated 18 hours after presenting with a large right hemispheric infarction. An emergency right temporal craniectomy and lobectomy was performed. The patient remained comatose for 48 hours and then began to improve neurologically. After rehabilitation, she was alert, her speech was normal, and she could walk with assistance. She remained with a spastic left hemiparesis. Ojemann et al.9 have also reported that hemicraniectomy was effective in treating two patients with large cerebral infarctions who developed signs of brain stem compression.

Kondziolka and Fazl12 recently described the use of a more limited frontotemporal craniectomy in five patients with incipient cerebral herniation after cerebral infarction. Their patients were significantly younger than our patients (mean age 40 years, range 32–51 years). Their study included two patients with cerebral infarction in the setting of subarachnoid hemorrhage and aneurysm clipping, one patient with carotid artery dissection, and one with presumed embolic infarction from an anterior communicating artery aneurysm; the remaining patient had a more typical presentation of a middle cerebral artery distribution infarct. All of these patients were ambulatory at the time of follow-up, with neurologic deficits attributable to the original ischemic event.

Hemicraniectomy may be more effective for treatment of malignant edema related to stroke than for treatment of head trauma. Cerebral stroke results in a unilateral mass of necrotic brain and edema with a viable contralateral hemisphere and brain stem. Decompressive craniectomy over the infarcted area may prevent further ischemia and injury to these viable areas. Edema that produces elevated intracranial pressure has also been treated in severe head trauma by decompressive craniectomy; the results have varied.13–17 Severe head trauma is usually a diffuse process with irreversible bilateral cerebral and brain stem injury. Craniectomy for head trauma may not be as effective as in stroke because reduction of intracranial pressure and patient neurologic outcome may be limited in a diffusely damaged brain. Similarly, subarachnoid hemorrhage patients with ischemia due to diffuse vasospasm may not respond as effectively to decompressive craniectomy as do patients with spontaneous embolic or thrombotic stroke.

In the management of patients with acute cerebral infarction, physician objectives include the preservation of life, prevention of extending cerebral injury, and avoidance of systemic complications. Patient age, family support, and potential neurologic outcome must also be considered before aggressive management is undertaken. Hemicraniectomy for cerebral infarction with severe edema may preserve life and prevent extension of central nervous system injury. Although surgical decompression of patients with severe dominant hemisphere strokes may result in survival, we would anticipate an extremely poor quality of life. Patients who deteriorate while on medical therapy for nondominant hemispheric infarctions are probably more suitable for hemicraniectomy. However, outcome in these patients can be variable. Most patients will require extensive rehabilitative therapy and lifelong assistance. A few patients may demonstrate dramatic improvement and progress neurologically to a level where only minimal assistance is required. The variability in outcome is dependent on the location and extent of the infarction, the patient's age, and possibly the timing of surgery. Young stroke victims, with a supportive family and a potential for neurologic recovery, are probably more likely to have a favorable outcome. Therefore, when medical modalities fail to curtail cerebral edema in a young patient with nondominant hemispheric stroke, emergency hemicraniectomy should be considered.

In conclusion, it was the opinion of the treating physicians that nine patients with massive right hemispheric infarction would die imminently from brain swelling unless the infarcted brain was decompressed. Hemicraniectomy was performed, and all patients improved, although one died 1 month later from an unrelated condition. The neurologic function in the other eight was variable, but the quality of life was considered reasonable by the surviving patients and their families. Hemicraniectomy can be a lifesaving treatment for massive right hemispheric infarction and can provide a reasonable quality of life.

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References


**KEY WORDS** • brain edema • cerebral infarction • craniectomy
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