Hemicraniectomy and Durotomy for Malignant Middle Cerebral Artery Infarction

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KEYWORDS

- Stroke • Hemispheric infarction • Cerebral herniation
- Craniectomy • Durotomy • Syndrome of the trephine
- MCA infarction (middle cerebral artery)
- ICA infarction (internal carotid artery)

Large middle cerebral artery (MCA) infarction accounts for 10% to 15% of all supratentorial infarction, with a mortality of 50% to 80%.[1–4] Clinical manifestations reflect the hemisphere involved and possible associated anterior cerebral artery (ACA) or posterior cerebral artery (PCA) infarctions. Signs and symptoms include hemiplegia, hemianesthesia, hemianopia, aphasia (mainly in left, dominant hemispheric infarctions), hemineglect (typically in right, nondominant hemispheric infarction), forced gaze deviation, possible head deviation, and progressive deterioration in the level of consciousness. In general, these large MCA ischemic strokes are associated with poor prognosis.[1,3–8]

Kasner describes 201 patients who had large MCA strokes, 94 (47%) of whom died from massive cerebral edema, 12 (6%) died from nonneurologic causes, and 95 (47%) survived to day 30.[4] Risk factors for early mortality included history of arterial hypertension, history of heart failure, elevated white blood cell count greater than 50%, MCA hypodensity, and additional involvement of other arterial territories. Although the presenting level of consciousness, National Institutes of Health Stroke Scale (NIHSS) score, early nausea and vomiting, and serum glucose levels were associated with neurologic death in univariate analyses, they were not significant factors in multivariate analyses.[4] Hacke and colleagues also report that occlusion of the ICA or MCA and poor collateral flow were risk factors for poor outcome.[1]

Radiologic parameters of poor outcome include hypodensity of greater than 50% of the MCA territory by CT scan or lesion volume greater than 145 cm^3 on diffusion-weighted MRI.[4,8–11] Mori and coworkers state that infarction volumes of greater than 240 cm^3 predict a poor outcome in more than three quarters of all cases of malignant cerebral edema.[9] Other investigators also identify major CT hypodensity in the MCA territory as a significant risk factor.[8,10] Additional predictors of poor outcome by CT of significance include midline shift infarctions involving more than just the MCA territory.[12] Diffusion-weighted magnetic resonance (MR) lesions, however, may not be predictive early in the course of an infarction, and MR

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perfusion-weighted lesions or findings by MR angiography of MCA occlusion may be more sensitive predictors.[13,14] Adjusted diffusion coefficient changes of greater than 82 mL were the most accurate MRI prognostic factors in another study, although clinical NIHSS scores were more sensitive than any single MRI predictor.[14] Serena and colleagues note that clinical and radiologic factors are not sufficiently sensitive or specific for predicting which patients who have MCA infarction actually deteriorate clinically and suggest using various biomarkers as predictors of MCA infarction.[15] Among compounds investigated were matrix metalloproteinase-9, cellular fibronectin, interleukins 6 and 10, tumor necrosis factor, and amino acids, such as glutamate, glycine, and γ-aminobutyric acid. Of these markers, Serena and colleagues note that the sensitivity and specificity of plasma cellular fibronectin (at a cutoff of >16.6 μg/mL) was highest at 90% and 100%, respectively, with a positive and negative predictive value of 100% and 90%, respectively.[15] Cellular fibronectin is a component of the basal lamina whose presence in plasma signifies disruption of the blood-brain barrier. S-100B is an alternate marker of blood-brain barrier disruption that has been studied. This astroglial protein is released into the bloodstream after acute stroke and reaches a maximum 2 to 4 days post stroke. At 12 hours, the S-100B values were significantly higher in patients who had MCA stroke and who had a malignant course, and by 24 hours, plasma values greater than 1.03 μg/L were associated with a 94% sensitivity and 83% specificity for a malignant course.[16]

Cerebral edema reaches its maximum approximately 96 hours post stroke and is the major cause of death in large strokes. In the context of malignant MCA territory infarction, however, deterioration may progress much more rapidly.[4,6–9] Several investigators suggest that clinical deterioration from hemispheric infarction is to the result of enlargement of the ischemic swollen tissue rather than global increases in intracranial pressure (ICP) and decreases in cerebral perfusion pressure.[3,4,17] A recent multicenter retrospective chart review of 53 patients not treated surgically reports that by 48 hours, two thirds of patients had clinical deterioration and 47% of the patients died most typically on day 3 after the stroke.[6] Approximately 13% of proximal MCA infarctions are associated with severe edema and herniation, with 7% of patients dying from brain swelling in the first week post stroke.[18] Furthermore, although from a mechanistic standpoint, the simplest explanation for the benefit of decompressive hemicraniectomy is preventing compression of normal brain, improved cerebral perfusion also may account for some of the subsequent improvement observed after hemicraniectomy. Doerfler and colleagues show that early craniectomy in a rat model of MCA stroke is associated with lesser mortality and improved behavioral outcomes, and Engelhorn and coworkers note that in an experimental rat model of MCA stroke, early reperfusion therapy (by release of the MCA occlusion suture) and decompressive craniectomy were found to improve cerebral perfusion (assessed by MRI), although a reperfusion strategy was more effective than surgery and combined treatment did not provide additional benefit.[19,20] Based on these data, hemicraniectomy is proposed as treatment for brain swelling post stroke.

MEDICAL MANAGEMENT

As with all stroke patients, initial management of patients who have large hemispheric infarction should follow standard guidelines, including assessment of hemodynamic and respiratory stability.[21,22] Patients who have large infarctions are at particular risk for early aspiration, and although immediate endotracheal intubation is not mandatory, any sign of neurologic or respiratory compromise should prompt early intervention. Early imaging using CT or MRI to identify signs of early cerebral ischemia also is essential with possible herniation (and to rule out other causes of mass effect, such as tumor, abscess, or blood). When available, perfusion and diffusion-weighted MRI and gradient-echo imaging may be of particular usefulness in distinguishing acute ischemic strokes and hemorrhages. For those patients who have only small diffusion-weighted lesions, despite large perfusion-weighted defects, thrombolytic therapy may preclude progression to large hemispheric infarction.

An important general principle in the care of patients who have an acute ischemic stroke is admission to a stroke unit, neurointensive care unit, or other monitored setting. Additionally, in all strokes, avoidance of arterial hypotension is critical to maintaining adequate cerebral perfusion to preserve normal brain tissue, and aggressive treatment of hyperglycemia, hyperthermia, and prevention of medical complications, including cardiac dysrhythmias, aspiration pneumonia, urinary tract infections, and deep venous thrombosis, along with early mobilization, applies as much to large hemispheric infarction as to any other stroke.

Medical interventions to reduce ICP, including hyperventilation, hypothermia, barbiturates, ventricular drainage, and osmotic diuresis (eg, with mannitol), are used to treat elevated ICP in ischemic stroke. None of these therapies are shown to improve outcome, however. Corticosteroids, used in the management of vasogenic cerebral

edema associated with brain tumors, do not increase poststroke survival.[23] Mannitol also is used to decrease cerebral edema and infarction post stroke and is used widely in acute stroke for the control of malignant cerebral edema. Despite the wide use of this agent, there are no strong randomized studies to support the use of mannitol and its administration is based on clinical observation and animal studies.[24] A systematic review of the literature from 1966 to 2002 shows no benefit for any of the medical therapies used routinely (discussed previously) in humans, and animal studies produce conflicting results.[13] There are no randomized clinical trials supporting either medical or surgical intervention for large space-occupying hemispheric infarctions, although the investigators cite two nonrandomized series, by Rieke and colleagues and Schwab and colleagues, respectively, as promising support for surgical intervention.[2,13,25] The review notes that the assumption of elevated ICP as the dominant cause of deterioration may not be true and cites one study that fails to show ICP monitoring may help in guiding therapy.[26]

HEMICRANIECTOMY PROCEDURE

Decompressive surgery for cerebral edema dates back to the early twentieth century and studies of surgery for MCA infarction date back to the 1930s.[27,28] In a crude sense, the concept may date back to furthest antiquity when trephine procedures were performed to relieve ill humors. In the modern era, surgical decompression was described by Kocher for traumatic brain injury patients and Harvey Cushing described surgical decompression for tumor-related edema. Heros and Morcos credit Donaghy and coworkers, in 1974, as reporting the first case in the English literature specifically on the treatment of cerebral edema related to hemispheric infarction.[29] The rationale for surgery, derived from the basic principles of the Monroe-Kellie doctrine, is to open up the box, thereby allowing outward expansion of the edematous infarcted brain tissue and, thereby, preventing lateral or downward cerebral herniation.

The surgical procedure involves removal of the skull, durotomy, and duroplasty to accommodate further swelling (**Fig. 1**). After a minimum of 3 months, the stored cranium then is replaced. Typically, removal of necrotic tissue or partial lobectomy is deferred except in circumstances of severe temporal lobe swelling. Demchuk describes the minimal adequate decompression as continuing to the following bony boundaries: anterior, frontal to the midpupillary line; posterior, approximately 4 cm to the external auditory canal; superior, to the saggittal sinus; and inferior, to the floor of the middle cranial fossa with a cruciate or circumferential durotomy over the entire region of bony decompression.[30] Recommended management of hemicraniectomy cases includes recognition of high-risk patients, including patients who have high NIHSS scores (>15 for right and >20 for left hemisphere lesions), early CT signs of greater than 50% MCA territory involvement, and high comorbidities. These patients should be rescanned early, within 6 to 12 hours after the initial scan, and consideration of hemicraniectomy may be appropriate if there are early signs of complete MCA or MCA plus ACA or PCA infarctions with mass effect. Otherwise, monitoring for altered levels of consciousness or anisocoria is warranted, and an immediate CT scan should be obtained if there is any change in neurologic status. Additional evidence of compartmental right-to-left shift greater than 1 cm then may be an indicator for hemicraniectomy.[30] Typically, the bone segment from the craniotomy is stored either in a frozen preservative solution or in a surgical pouch within a patient's abdominal wall. Alternatively, synthetic cranioplasty material can be used. As patients recover and swelling subsides, typically in 3 months (range, usually 1.5–6 months), the bone flap can be replaced in a subsequent procedure.[31]

OUTCOME FROM HEMICRANIECTOMY

There have been many series in the past decade that confirm not only improvements in overall mortality but also improved functional outcomes. Critical elements involve patient selection criteria and timing of intervention. Factors, including the role of hemicraniectomy for older patients and those who have dominant hemispheric stroke, influence case selection in the various nonrandomized series. Extent of the procedure and how much, if any, brain tissue to remove also are areas of debate. It is reported that simply by performing a craniotomy, there is a 15% decrease in intracranial pressures and when accompanied by durotomy, there is a significant 70% decrease in ICP.[27] Elevated ICP alone, however, is not a clear indictor for intervention.[32] Typically, early signs of particular changes in level of consciousness or dilation of one or both pupils possibly warrant intervention. But, by the time severe brainstem signs appear, hope for salvage is exceedingly poor and patients are not probable candidates for intervention.[32,33] The Hemicraniectomy and Durotomy on Deterioration from Infarction Related Swelling Trial (HeADDFIRST) in the United States remains unpublished at this time except in abstract form.

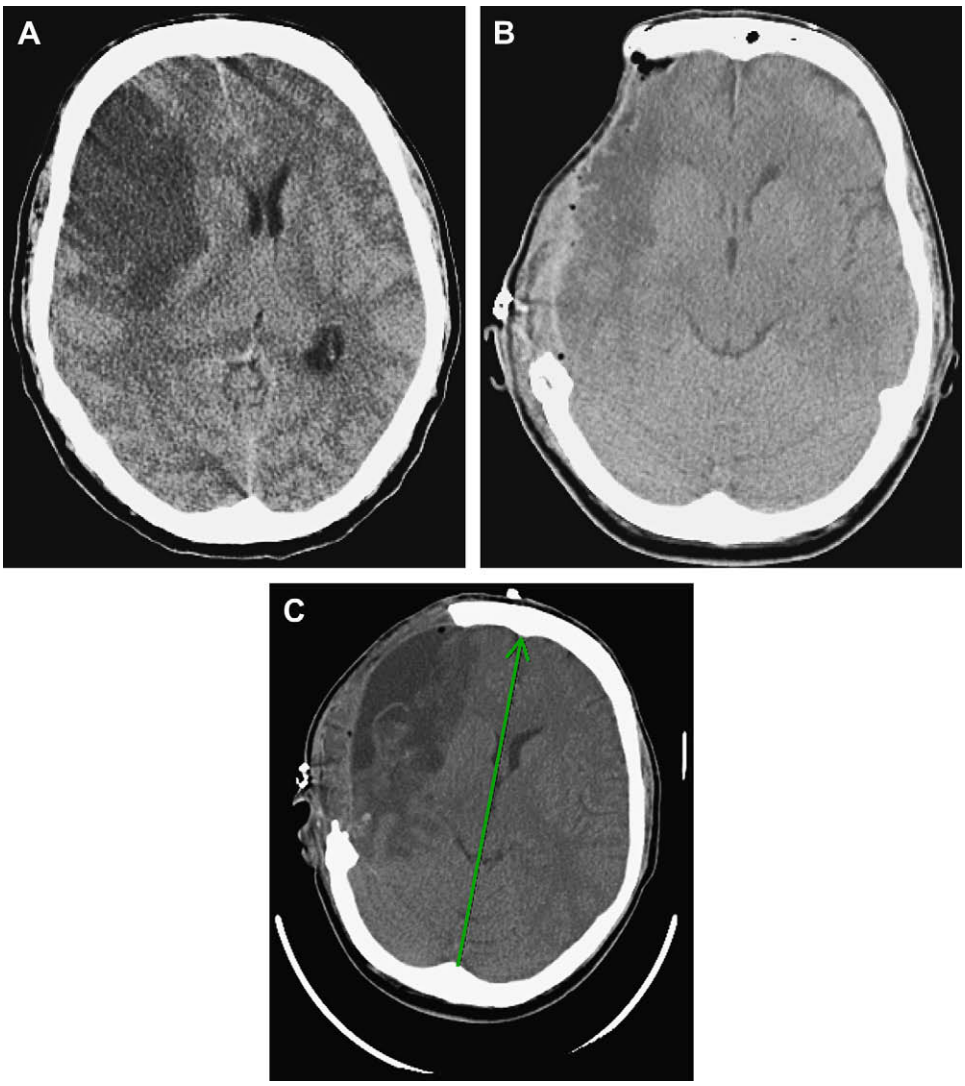

Fig. 1. (*A*) CT scan of a 50-year-old, right-handed man who had right internal carotid artery occlusion and who presented with acute onset of dysarthria, right gaze preference, left facial droop, left hemiplegia, and left hemianesthesia. Over the next 2 days, he had a decline in the level of consciousness. The preoperative CT demonstrates a right frontal infarction with subfalcine herniation. (*B*) Postoperative scan from the day after craniotomy. The patient had been taken urgently to the operating room, where he had a large frontal-temporal-parietal-occipital craniotomy, and the bone over the temporal tip was removed completely, decompressing the temporal tip. The dura then was opened circumferentially. The brain came out under pressure. At this point, a right temporal and right frontal lobectomy was performed, removing approximately 6 cm of the temporal lobe and approximately 8 cm of the frontal lobe. This allowed significant reduction and mass effect. Thereafter, synthetic dural material was laid over the top of the brain with the dura having been tented back circumferentially over Oxycel for hemostasis. (*C*) CT scan 5 days after craniotomy showing outward swelling of the brain with resolution of the right to left ventricular shift.

In this series, 26 patients were randomized and there was a statistically non-significant reduction in mortality from 46% with medical therapy to 27% in the surgically treated group.[34,35] Randomized clinical trials in Europe were recently published and give some perspective as to patient outcomes in selected patients.[36–41] Three European trials were completed: Hmicraniectomy after middle cerebral artery infarction with life-threatening edema trial (HAMLET, the Netherlands), decompressive surgery for the treatment of malignant infarct of the middle cerebral artery (DESTINY; German), and decompressive craniectomy in malignant middle cerebral artery infarcts

(DECIMAL, France).[38–40] The DESTINY and the DECIMAL trials ended early when significant outcome differences were shown. These studies, by intent, had similar designs and primary outcomes and so the data were subsequently presented in a pooled analysis.[41]

These were patients with large strokes (NIH Stroke Scale Scores >15 and imaging that showed infarct volumes >50 percent of the MCA territory) who were enrolled within 48 hours of symptoms onset. Exclusion criteria included those with either life expectancy of less than 3 years or poor premorbid functional status, patients with bilateral dilated pupils at time of enrollment, hemorrhagic transformation of stroke, or other areas of infarction outside of the affected MCA territory. The modified Rankin scale (mRS) was the primary outcome measure and the analysis dichotomized patients into favorable (mRS 0–4) and unfavorable groups (mRS 5–6). As an aside, typically patients with mRS 0–2 are usually considered to have a good outcome and mRS 5–6 represents those who are dead or comatose. There were 93 subjects in the pooled analysis (52 surgical and 41 non-surgical patients). At on year, 32/41 (78%) of patients in the non-surgical arm and 13/52 (25%) of the patients in the surgical arm had an unfavorable outcome. Of considerable import, there was no increase in the number of patients with severe disability, as opposed to death, in the surgical versus the non-surgical groups. Unfortunately there was limited data about whether there were particular limitations for those with left versus right hemisphere procedures and early versus late hemicraniectomy was not addressed adequately in these studies. Only five observational studies with comparative data were identified in the most recent *Cochrane Database of Systemic Reviews*, including one trial of moderate hypothermia versus hemicraniectomy for malignant MCA infarctions in which surgery was shown to be associated with lower mortality rates and lesser immediate complications.[36,42] At this time, data regarding critical elements of patient selection, timing of intervention, and patient outcomes after the procedure are based mainly on observational series.

In 1981, Rengachary and colleagues reported an initial case series of three patients who had reasonable clinical outcome, but it was not until the early 1990s that enthusiasm for hemicraniectomy in ischemic stroke began to take off.[43] In 1990, Delashaw and coworkers reported a case series of nine patients who had right hemispheric infarction; all patients survived the surgery, and three of the eight patients followed from 5 to 25 months were functioning with minimal assistance.[44] Subsequently, Rieke and coworkers described the results of decompressive craniotomy in one nonrandomized prospective trial of 32 patients who had a reported mortality rate of 35% compared with 21 nonoperative controls who had an 80% mortality rate.[25] Six of the 32 patients who were treated surgically were rated as having a good outcome compared with none of the 21 nonsurgical patients. Another case series of 14 patients who had nondominant hemispheric infarction reported death from non-neurologic causes in three of the patients; seven of the 11 survivors were walking 1 year after the procedure.[45] One major caveat was that in both of these and in other series, patients typically were younger and had fewer comorbidities.

Woertgen and colleagues reviewed data from 48 patients (26 men, mean age 48 years) with outcomes measured using the Glasgow Outcome Score (GOS) and Barthel Index (BI).[46] Mortality was 26%.[41] Younger age correlated with mortality (44.5 versus 60.3 years; GOS 1; $P < .0006$). Craniectomy with dural patch also correlated with mortality (58 versus 14%; GOS 1; $P < .005$). In this study, quality of life did not differ between patients who had left- or right-sided lesions or between patients who did or did not have aphasia. The investigators stated that in retrospect, 83% of patients ostensibly would have agreed to have the procedure done in the future. In another series, Foerch and colleagues also noted that older age, more severe neurologic deficit on admission and longer duration of intensive care and mechanical ventilation were associated with worse prognosis; functional outcome was impaired significantly, although survival was improved.[47]

In a predominantly male series of 19 patients who had a mean range of 46.5 years, with 53% having a dominant hemispheric stroke, Pranesh and coworkers report a relatively good outcome, especially in younger patients who had a mean NIHSS score of 20.5 before surgery and a mean NIHSS score of 10.5 after surgery, with a poor Rankin score in 21% of cases and only one death in the series. Outcome was better for patients under age 50, and language also was improved for cases of dominant stroke.[5]

Timing of intervention may play a role in determining outcome. Schwab and coworkers report a survival rate of 73%, and none of the survivors were wheelchair bound.[2] All survivors who had dominant hemispheric stroke (11/46) had improvement in aphasia and a severe Rankin score was present in only 13% of cases. In particular, early intervention within 24 hours of symptom onset, based only on clinical findings and CT findings before herniation signs developed, was associated with improved mortality and less intensive

care time, but the disability scores, as measured by BI were no different at 3 months between early and delayed interventions.[2] Similarly, Mori and colleagues compared three groups retrospectively: (1) those patients treated conservatively; (2) those patients treated after clinical and radiologic signs of herniation; and (3) those patients treated before clinical and radiologic signs of herniation. The investigators suggest there was a statistically significant improvement in mortality and clinical outcome scores (measured by BI and GOS) for the early surgery group.[48] For surviving patients, there was no difference in functional outcome between the medically treated and the late surgery groups, although mortality was better in the latter group. To date, however, no prospective data are available to compare early hemicraniectomy and duroplasty within 24 hours of infarction versus later intervention. Because the resulting edema from MCA infarction may cause extension of the infarction into surrounding ischemic penumbra tissue or cause secondary damage to otherwise normal tissue from compression, a cogent argument has been made that early intervention is reasonable. As discussed previously, however, prediction of which MCA infarctions will progress to malignant edema is an uncertain science and selection bias may reflect the positive outcomes reported.[28]

There also are no data comparing hemicraniectomy and duroplasty with or without anterior temporal lobectomy.[3,18] Conceivably, the extent of the surgical resection also may lead to better outcome. An aggressive surgical approach with craniectomy, resection of infarcted tissue, and duroplasty is proposed as providing a more favorable result.[18]

In regard to functional outcomes, retrospective series suggest that the outcomes were related mainly to comorbidities. Harscher and coworkers report a series of 30 patients, five of whom died within the first week after signs of herniation, and nine additional patients died within the ensuing 1 to 12 months.[49] Of the 16 surviving patients, long-term follow-up was available for a mean of 2.1 ± 1.5 years; 11 of the patients were at home and five were institutionalized. The mean BI was 60 and the mean Rankin score was 3.1 for the noninstitutionalized patients. There was no difference in outcome by involved hemisphere, although age, as expected, played a significant role in outcome, and increased numbers of comorbid conditions also were associated with worse functional outcome as measured by the BI. Additionally, Walz and colleagues report on their small series of 12 surviving patients, followed out 7 to 26 months post stroke. Out of 18 who underwent surgery, six died within 6 months of the procedure.[50] Survivors were significantly younger than those who died and had a mean Rankin score of 3.3 and mean BI of 61.1. Those patients who were under 45 years of age had a significantly better outcome and two patients actually returned to work. Although all five of the survivors who had a left hemispheric stroke had some degree of aphasia (ranging from slight Broca's aphasia to global aphasia), the investigators suggest that quality-of-life scores were not related clearly to hemispheric involvement. Similar results were observed by Kastrau and colleagues, who noted significant recovery in aphasia in 13 of 14 patients less than 50 years old and who underwent hemicraniectomy for dominant hemispheric stroke.[51]

Curry and coworkers also report a series with 1-year outcome of 38 patients who had large hemispheric infarction status post decompressive surgery, 32 of whom survived more than 1 year. Available follow-up on 20 patients again demonstrates that age was the major factor for better outcome, and the average BI in this series was similar to previous studies, with a mean BI of 67.[52] This observation was confirmed by Holtkamp and coworkers, who note, in a series of 12 patients, ages 55 to 75, that hemicraniectomy in the elderly was indeed associated with improved survival but functional outcome was poor.[53] In the Holtkamp study, and other series, outcomes were not as positive, Thus, Kilincer and colleagues also report poor functional outcome in their series, with most patients having a poor Rankin score, with mortality at 6 months in this series of 32 patients approaching 50%.[54] Older age, low preoperative Glasgow Coma Scale score, greater than 10-mm preoperative midline shift, anisocoria, rapid clinical deterioration, and presence of an internal carotid artery infarction were poor outcome predictors. Finally, in a series of 18 patients, there was a good (BI > 90) or fairly good (BI 75–90) outcome in only half of the patients, and formal neuropsychologic testing in 14 cases status post hemicraniectomy showed profound attention deficits in all patients and impaired visuospatial and visuoconstructive deficits in those patients who had lower educational attainment. Again, older age was an independent predictor of poor outcome.[55] Subsequently, in 2007 two additional series were published. Chen and colleagues described their experience with 60 patients in China, mean age of 62.7, who underwent hemicraniectomy. In the series, 38/60 were right MCA infarections and 22/60 were left MCA infarctions. 20/60 (33%) had evidence of brainstem compression. In this series there was a favorable outcome in 29 patients (66%) with a 16% mortality. The factors

associated with higher mortality were age greater than 59 years, involvement of more than one vascular territory, pre-operative signs of herniation before surgery, and surgery after 24 hours of symptom onset.[56] Pillai and colleagues reported their experience in India of a series of somewhat younger patients (n = 26; mean age 48.3). They performed 14 right and 12 left hemicraniectomies of which (35%) had signs of brainstem compression prior to surgery. In this series there was a 28% mortality rate and 15/126 (60%) had good outcomes. Again, older age was associated with a less favorable outcome.[57] In a summary review of 13 studies, many of which are cited in this article, representing data from a total 138 patients, Gupta and colleagues confirm that the outcome after hemicraniectomy remains problematic.[58] Only 7% (10/138) of patients were functionally independent and 35% were mild to moderately disabled. Furthermore, 80% of those older than age 50 were dead or severely disabled compared with 32% of those age 50 or under (statistically significant P value of $< .00001$). Timing of surgery, dominant versus nondominant hemispheric involvement, signs of herniation before surgery and involvement of other vascular territories in addition to the MCA territory did not have a significant affect on outcome. It is not surprising, therefore, that Leonhardt and coworkers remark that 4 of the 18 patients in their series would not have consented again to the procedure because of poor postoperative quality of life.[58]

HEMICRANIECTOMY AND THE SYNDROME OF THE TREPHINED

A subset of patients who have undergone craniectomy for malignant MCA infarction (or other causes of malignant cerebral edema) and who have improved or stabilized undergo a secondary loss of neurologic function as the craniotomy site sinks and becomes concave. The patients may present with symptoms, including headache, vertigo, tinnitus, fatigue, irritability, loss of concentration, memory loss, depression, dysphagia, increasing paresis, new-onset seizures, intolerance to vibration, and local pain over the defect site.[59,60] Over the decades, several names have been given to this process, including syndrome of the trephined, sunken flap syndrome, postcraniotomy syndrome, and post-traumatic syndrome.[61–63] The mechanism of this process—a consequence of atmospheric pressure applied directly to the brain—first was postulated by Gardner in 1945.[64] Since that time, several mechanisms have been investigated, especially in the positron emission tomography and MRI era, which have demonstrated hemodynamic, hydrodynamic, and metabolic causes.[63,65–68] The synthesis of these findings has led to the following understanding of the syndrome of the trephined, in which the sunken flap leads to a decrease in the cerebrospinal fluid space, especially the arachnoid space surrounding the vessels. This collapsed space restricts arterial inflow and, more importantly, venous outflow, which then leads to a decrease in overall cerebral blood flow (CBF). This decrease in CBF is reflected by a decrease in oxygen extraction and metabolic activity, thereby leading to a decrease in neurologic function. Closure of the cranial defect results in reversal of this pathologic cascade, leading to improved cognition and neurologic function.

SUMMARY

Decompressive surgery with hemicraniectomy and durotomy for malignant MCA infarction remains a salvage procedure but can be associated with reasonable clinical outcomes in highly selected patients. This selection of patients appropriate for intervention is of the utmost importance, but exact criteria remain to be defined; older age and increased numbers of associated medical comorbidities seem to define a group of patients who would not derive long term benefit, however. The determination as to whether or not surgery is equally beneficial for dominant or nondominant hemispheric infarction is hampered by lack of good comparative data, but selected case series suggest that some patients who have dominant hemispheric infarction achieve a reasonable degree of independence. Although a well-defined principle of stroke practice is that ''time is brain,'' there are no clear data as to when intervention should be done, as there are some patients who have large MCA infarction and who may not progress to cerebral herniation. Clinicians managing the growing population of patient status post hemicraniectomy should also be aware of this process of the syndrome of the trephined and the potential for resolution that may prompt earlier cranial reconstruction. At present, the decision to proceed with this aggressive intervention of hemicraniectomy and durotomy for large ischemic infarction remains a case-by-case individualized approach, based on patient and family preferences and clinicians' subjective perspective as to patients' potential for clinical recovery.

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