Surgery for brain edema

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ARGUABLY THE MOST fundamental pathophysiological processes following brain injury are brain edema, increased ICP, reduced cerebral blood flow, inadequate oxygen delivery, energy failure, and further edema. These processes are common to a number of neurosurgical conditions, including TBI, SAH, intracerebral hemorrhage, and cerebral infarction. One of the goals of treatment is to interrupt this vicious cycle by controlling swelling and maintaining an adequate blood and oxygen supply to meet the needs of the injured brain. Early intervention with simple treatment measures (administration of oxygen and intravenous fluids) to correct hypoxia and hypotension is essential. Following this initial stage of resuscitation, ongoing specialist treatment is designed to minimize the occurrence of secondary insults, which contribute to brain edema. One of the greatest advances in neurointensive care is the implementation of protocols to standardize treatment in patients with cerebral edema. There is now good evidence that such protocols have resulted in an improvement in neurological outcome. The steps of protocol-driven management differ among centers (type and order of intervention), but the overall concept of increasing the intensity of treatment until targets (for example, ICP and cerebral perfusion pressure) are reached is a common theme. Therapies include medical measures such as maintaining oxygenation, inducing hypothermia or mild hyperventilation, and administering mannitol, hypertonic saline, and barbiturates. Drainage of CSF by ventriculostomy is also an effective means of controlling brain swelling in many patients. Whereas these therapies will allow treatment targets to be reached in most patients, there is a cohort with pathophysologies including TBI, SAH, and ischemic stroke in whom the vicious cycle of brain edema will continue to propagate, culminating in an increase in cell injury and death. Such patients may be candidates for brain edema surgery; that is, decompressive procedures to assist in the control of brain swelling. In this review we will therefore focus on decompressive craniectomy as a therapy in controlling brain edema in patients with TBI, poor-grade SAH, and ischemic stroke.

KEY WORDS • brain edema • cerebral infarction • decompressive craniectomy • head injury • intracranial pressure • stroke • subarachnoid hemorrhage

Abbreviations used in this paper: BI = Barthel Index; CI = confidence interval; CSF = cerebrospinal fluid; CT = computed tomography; DECRA trial = decompressive craniectomy trial; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; mRS = modified Rankin Scale; RESCUEicp = Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure; SAH = subarachnoid hemorrhage; TBI = traumatic brain injury.

Decompressive Craniectomy

The concept of decompressive craniectomy is by no means novel; it can be defined as the removal of a large area of skull to increase the potential volume of the cranial cavity (Fig. 1). At the beginning of the last century, Kocher12 asserted that “if there is no CSF pressure, but brain...
pressure exists, then pressure relief must be achieved by opening the skull.” Since then, decompressive craniectomy has been in and out of vogue with the recognition that, although the procedure is theoretically attractive, a number of fundamental questions remain as to whether or not it should be performed. Such questions include the following. First, does decompressive craniectomy control raised ICP? Second, does decompressive craniectomy propagate brain herniation (and therefore edema at the bone margins)? Third, do the results justify the treatment? Fourth, what is the complication rate? Across a spectrum of pathological entities, there is concern that the operation is performed unnecessarily in patients who have a good prognosis with medical treatment alone and that decompressive craniectomy can save lives by controlling brain edema but could shift outcome to vegetative state and severe disability. What is different about this procedure now, compared with several years ago, however, is that it is being performed in the context of modern intensive care as part of protocol-driven therapy and is being evaluated in randomized controlled trials.

Surgical Technique

There are many variations in the technique of decompressive craniectomy. Factors that should be carefully considered include the involved hemispheres (unilateral or bilateral), disease location (frontal, temporal, parietal, or occipital), size, and dural opening (dura left intact, dura left open, or dural graft patch).

In deciding which hemisphere is involved, one of the fundamental determinants is the presence of midline shift. In patients with TBI, diffuse brain edema, and no midline shift, bilateral craniectomy is advocated. This procedure can be performed in several ways. We advocate a bifrontal craniectomy from the floor of the anterior cranial fossa (avoiding the frontal air sinus) to the coronal suture posteriorly and to the pterion laterally. Although a bridge of bone in the midline can be left over the superior sagittal sinus, in our experience removing the bone completely (Fig. 2) enables one to perform a wide U-shaped dural opening based on the superior sagittal sinus. Ligation and division of the sinus and the falx at its most anterior extent relieve constriction. In patients with TBI, unilateral hemisphere swelling, and midline shift, a large, question mark–shaped unilateral craniectomy is suitable. This technique is also appropriate in patients with hemisphere swelling due to ischemic stroke and SAH. The size of the craniectomy is of critical importance. Small craniectomies risk brain herniation with venous infarction and increased edema at the bone margins. Ashcroft et al. have modelled the potential gain in volume depending on the size of the craniectomy.

Aiming for a minimum diameter of 10 cm enables a potential gain in cranial volume of 50 ml. In the past, craniectomies were performed without dural opening, but it is now recognized that the dura must be opened to achieve decompression. Whether the scalp is closed over the widely opened dura mater and lined with a sheet of Surgicel or whether a dural graft large enough to accommodate the expanding brain is performed is debatable. In our experience, however, we have found that leaving the dura open, resting on top of the brain and lined with a sheet of Surgicel, shortens the procedure, is not associated with the potential complication of CSF leakage, and provides a satisfactory plane for the subsequent cranioplasty. Csokay and colleagues have described the creation of a vascular tunnel to prevent venous engorgement by avoiding compression of the cortical veins at the edge of the flap. In our experience, however, if the craniectomy is large, the tunnel is not necessary.

**Fig. 1.** Computed tomography scans demonstrating the appearance of large bifrontal (A) and unilateral (B) decompressive craniectomies. An H<sub>2</sub>O positron emission tomography scan (C) revealing preservation of cerebral blood flow in widely decompressed frontal regions after bifrontal craniectomy.

**Fig. 2.** Intraoperative photograph obtained during a bifrontal decompressive craniectomy, depicting the brain’s appearance prior to dural incision.
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### TABLE 1
Summary of randomized controlled trials on decompressive craniectomy in patients with TBI*

<table>
<thead>
<tr>
<th>Study Name/Description</th>
<th>Principal Investigator/Steering Center</th>
<th>Principal Inclusion Criteria</th>
<th>Study Design</th>
<th>Primary Outcome Measure</th>
<th>Sample Size</th>
<th>Progress/Results</th>
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<tr>
<td>RESCUEicp</td>
<td>PJ. Hutchinson, P.J. Kirkpatrick/University of Cambridge Academic Dept. of Neurosurgery, UK, &amp; European Brain Injury Consortium</td>
<td>severe TBI; ICP refractory to optimal, protocol-driven conservative therapy (excluding barbiturates); that is, ICP &gt;25 mm Hg for 4–6 hrs</td>
<td>international multicenter RCT</td>
<td>favorable vs unfavorable outcome based on dichotomized extended GOS (GOSE 1–3 vs GOSE 4–8) &amp; SF-36 QOL questionnaire at 6 mos &amp; 2 yrs after injury</td>
<td>600 patients</td>
<td>10% difference in outcome, ongoing recruitment</td>
</tr>
<tr>
<td>DECRA trial</td>
<td>D.J. Cooper, J. Rosenfeld/Alfred Hospital, Melbourne, Australia</td>
<td>severe diffuse TBI (no mass lesions), 72 hrs after injury, refractory ICP (&gt;20 mm Hg for &gt;15 min)</td>
<td>multicenter RCT, Australia/New Zealand</td>
<td>favorable vs unfavorable outcome based on dichotomized extended GOS (GOSE 1–4 vs 5–8) at 6 mos after injury</td>
<td>210 patients</td>
<td>ongoing recruitment</td>
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* GOSE = extended GOS; QOL = quality of life; RCT = randomized controlled trial; SF-36 = 36-Item Short Form Health Survey.

### Traumatic Brain Injury

For many years, decompressive craniectomy has been applied in patients with TBI. Within the past 10 years, the modern era of neurointensive care, there have been several observational studies. In one of the first analyses, authors from Charlottesville compared the results in patients undergoing decompressive craniectomy with historical control data from the National Traumatic Coma Databank. They reported good outcomes in 37% of the patients as opposed to 16% in controls, with better results if decompression was performed within 48 hours of injury and if ICP at the time of decompression was less than 40 mm Hg. From Wurzburg, Kunze et al. reported a favorable outcome rate of 56% and a mortality rate of 11%, with later decompressions averaging 68 hours postinjury in 28 patients. Similar results have been reported from Griefs bridge, described in 2001, and a second cohort of 49 patients yielded a favorable outcome in 61%.

In summary, the literature demonstrates a wide range of clinical outcomes, with no clear consensus regarding the indications for surgery. What are the current recommendations for the role of decompressive craniectomy in TBI? In Europe, Sahuquillo and Arikan have published a Cochrane review. These authors have concluded that there is no evidence to support the routine use of decompressive craniectomy in adults with severe TBI and medically refractory elevated ICP. In the pediatric population, decompressive craniectomy reduces the risk of death and unfavorable outcome. However, the results of nonrandomized trials and controlled trials with historical controls involving adults have sug-
gested that decompressive craniectomy may be a useful option when maximal medical treatment has failed to regulate ICP. The American Brain Trauma Foundation guidelines state that bifrontal decompressive craniectomy within 48 hours of injury is a treatment option in patients with diffuse, medically refractory, posttraumatic cerebral edema and resultant intracranial hypertension. With no clear consensus, proposals for randomized studies have been offered to obtain Class I evidence (Table 1). In 2001, a small randomized study originating from the Royal Children’s Hospital in Melbourne was published. Patients were randomized to standard treatment alone or with decompression. Those in the standard treatment group had a mean ICP reduction of 3.7 mm Hg and a favorable outcome (normal or mild disability) in 14%; patients in the standard treatment plus decompression (performed at 19 hours postinjury) group had a mean ICP reduction of 8.9 mm Hg and a favorable outcome rate of 54%. This difference did not quite reach significance. There were several problems with this study—sample size, outcome evaluation, and prolonged duration (7 years)—and the authors recognized these limitations. Two multicenter prospective randomized studies are ongoing: the RESCUEicp study and the DECRA study.

The RESCUEicp is a multicenter randomized trial in which decompressive craniectomy will be compared with medical management coordinated by the University of Cambridge, UK, and the European Brain Injury Consortium. Patients (50 for the pilot phase and 600 for the main study) with TBI and elevated ICP (> 25 mm Hg) refractory to initial treatment measures are eligible for the study. Patients are randomized to one of two arms: continuation of optimal medical management (including barbiturates) and surgery (decompressive craniectomy). Outcome will be assessed using the extended GOS and 36-Item Short Form Health Survey at 6 months posttreatment, with additional surrogate end points (ICP control and duration of stay in the intensive therapy unit). The pilot phase of the study has been completed and has demonstrated that randomizing patients with TBI to decompressive craniectomy as opposed to optimal medical management is feasible. Whether this operation is effective and safe remains to be seen. The main phase of the study continues, and interested centers are welcome to join the trial.

Subarachnoid Hemorrhage

In contrast to TBI, there are substantially fewer published studies on the experience of decompressive craniectomy as a treatment for brain edema following SAH. Ziai and colleagues reported on their experience with decompressive craniectomy for intractable cerebral edema in four patients following aneurysmal SAH: one patient died and three had a severe disability (poor results). Recently, data from two other studies have been published. Schirmer and associates have described the results of decompressive hemicraniectomy in the treatment of refractory elevated ICP in 16 patients with aneurysmal SAH. Half of the patients were treated with endovascular coil embolization and the other half with surgical clip application. Sixty-nine percent of patients survived, and at the follow-up (median 450 days) 64% of them had an mRS score of 0 to 3 and 36% a score of 4 to 5. Early craniectomy performed within 48 hours after SAH was associated with a better outcome. These authors concluded that decompressive hemicraniectomy is a useful adjunctive modality in the management of refractory intracranial hypertension in patients with poor-grade aneurysmal SAH, even in the absence of extensive intraparenchymal hemorrhage. Buschmann and colleagues have reported the results of 38 patients following decompressive hemicraniectomy after early aneurysm clipping. They divided the indications for decompressive craniectomy into four groups: 1) signs of brain swelling during aneurysm surgery; 2) ICP elevation and epidural, subdural, or intracerebral hematoma after aneurysm surgery; 3) brain edema and elevated ICP without radiological signs of infarction; and 4) brain edema and elevated ICP with neuroimaging-demonstrated signs of infarction. The pooled data from all 38 patients showed a favorable outcome (GOS Scores 4 and 5) in 53% of patients, severe disability in 26% (GOS Score 3), and death in 21%. After 12 months, a good functional outcome was seen in 52% of the cases in Group 1, in 60% in Group 2, in 83% in Group 3, and in 17% in Group 4. These authors concluded that in more than half of the patients with intractable intracranial hypertension after aneurysmal SAH, a good functional outcome could be achieved after decompressive craniectomy; that patients with progressive brain edema but no radiological signs of infarction and those with hematoma may benefit the most; and that the indication for decompressive craniectomy should be set restrictively if secondary infarction is present.

Surgical Decompression for Stroke

Despite recent advances in the prevention and treatment of ischemic stroke, there is still a proportion of patients in whom large cerebral infarcts develop as a result of a major vessel occlusion. Rapidly progressing, malignant cerebral edema is commonly associated with this condition, and usually manifests as clinical and radiological deterioration on Days 2 to 5 (Fig. 3). Escalating swelling and mass effect compromise cerebral perfusion, oxygenation, and metabolism and can eventually lead to refractory intracranial hypertension with increasing shifting of intracranial contents and transtentorial herniation, the leading cause of death in these patients. Despite optimal medical therapy and intensive care support, death from space-occupying cerebral infarction remains in the range of 70 to 80%, with little change in recent years. In survivors, severe residual disability is common. Not surprisingly, decompressive craniectomy has been performed as an empiric lifesaving measure for a long time in an attempt to protect the brain from the damaging effects of propagating edema. More recently, the physiological rationale for this approach has been backed by evidence from animal studies. In experimental models of extensive ischemic stroke, decompressive craniectomy improved cerebral perfusion, survival, and neurological outcome. It was also shown to reduce the volume of the infarction particularly when performed early after vessel occlusion. These beneficial effects are likely to be linked to increases in collateral circulation and reductions in tissue edema, together with improvements in
oxygenation and energy metabolism in ischemic penumbra. Cerebral tissue monitoring in patients undergoing craniectomy for ischemic stroke has demonstrated that decompression leads to a reduction in ICP and an increase in brain tissue oxygen levels. Imaging and clinical study data suggest that late restoration of function is possible in the affected cerebral hemisphere, making decompression a preferred option over unguided “strokectomy.”

The effect of decompressive craniectomy on neurological outcome is the crucial question in the assessment of its clinical utility. During the last three decades, more than 20 centers have published retrospective or nonrandomized prospective case studies on outcomes in patients undergoing decompressive craniectomy for ischemic cerebral edema. Data from all groups revealed improved survival following surgical decompression, with a mean mortality rate of 20 to 30% (range 8–50%) compared with 70 to 80% among patients treated conservatively. Nonetheless, as in other applications of decompressive craniectomy, concerns about the functional outcome in surviving patients remain. Because it is unrealistic to expect complete functional recovery following extensive ischemic stroke, most authors regard moderate or moderate-to-severe disability (mRS ≤ 4 or BI ≤ 60; that is, relative independence in daily activities) as a favorable outcome, and severe disability, vegetative state, or death as an unfavorable outcome. From this perspective, many authors have reported that a substantial proportion of surviving patients can attain a favorable outcome after decompression in other series, however, presented outcomes have been worse. This discrepancy has fueled interest in isolating the criteria that help to identify patients who are most likely to benefit from decompressive craniectomy. An association between poor neurological outcome and patient age was found in several studies, with some data demonstrating a correlation between postdecompression BI and age. The timing of decompressive craniectomy has also been suggested as an important predicting factor, with more patients surviving with favorable outcomes when decompressive craniectomy was performed very early or early after the onset of symptoms and before the development of mass effect and neurological features of herniation. Low initial Glasgow Coma Scale scores, involvement of additional vascular territories and the dominant...
and failure to open the dura mater as part of the surgical technique may also be independent predictors of a worse outcome. Given the fact that most surviving patients have some residual neurological loss, the patient and family’s perception of the craniectomy and resulting outcome is especially important. In study by Woertgen et al., 83% of patients or their relatives indicated that they would agree to surgery in the future. Erban and associates found that patient approval of surgery was lower (61%) and depression in survivors was common.

The failure of case series and nonrandomized trials to provide a definitive answer on the benefits of decompressive craniectomy in ischemic brain edema was highlighted in recent systematic reviews and triggered the initiation of several prospective, randomized, controlled trials.

### TABLE 2

Summary of randomized controlled trials on decompressive craniectomy in ischemic stroke with brain edema**

<table>
<thead>
<tr>
<th>Study Name/Description</th>
<th>Principal Investigator/Steering Center</th>
<th>Principal Inclusion Criteria</th>
<th>Study Design</th>
<th>Primary Outcome Measure</th>
<th>Sample Size</th>
<th>Progress/Results</th>
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<tr>
<td>HeaDDfirst (Hemian- &amp; Durotomy Upon Deterioration From Infarction-Related Swelling Trial)</td>
<td>J.I. Frank/Dept. of Neurology, University of Chicago</td>
<td>clinical &amp; radiological deterioration w/in 96 hrs of stroke onset</td>
<td>multi-center RCT, North America</td>
<td>death, functional outcome, QOL, patient perceptions, &amp; acute healthcare use at 21, 90, &amp; 180 days after stroke onset</td>
<td>75 proposed</td>
<td>study stopped at 26 (11 medical, 15 surgical); 21-day mortality rate, 45.5% in medical, 26.7% in surgical group; awaiting publication of definitive results</td>
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<tr>
<td>HeMMI (Hemian- craniectomy for Malignant MCA Infacts)</td>
<td>R.D. Jamora/University of the Philippines, Manila</td>
<td>MCA stroke, clinical deterioration w/in 72 hrs of symptom onset</td>
<td>single-center RCT</td>
<td>mRS score &amp; BI at discharge, 2 wks, &amp; 1, 3, &amp; 6 mos</td>
<td>56 patients</td>
<td>15 patients recruited in 2004; ongoing recruitment (?); no recent update available</td>
</tr>
<tr>
<td>DECIMAL (Decompressive Cranietomy in Malignant MCA Infacts)</td>
<td>K. Vahedi &amp; M.G. Bousser/Service de Neurologie Hospital Lariboisiere, Paris, France</td>
<td>radiological criteria of malignant MCA infarct w/in 24 hrs of stroke symptom onset</td>
<td>multi-center RCT, France</td>
<td>mRS score &lt;4 at 6 mos</td>
<td>60 patients</td>
<td>recruitment stopped after inclusion of 38 patients due to slow enrollment &amp; significant difference in mortality rate favoring surgery (see pooled analysis results below)</td>
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<tr>
<td>DESTINY (Decompressive Surgery for the Treatment of Malignant Infarction of the MCA)</td>
<td>S. Schwab, E. Jüttler/Dept. of Neurology, University of Heidelberg, Germany</td>
<td>radiological criteria of malignant MCA infarct w/in 24 hrs of stroke symptom onset</td>
<td>multi-center RCT, Germany</td>
<td>mRS score &lt;4 at 6 mos</td>
<td>60 patients</td>
<td>recruitment stopped because of significant benefit of surgery on 30-day mortality rate &amp; revised sample size projection indicated that 188 patients would be needed to show significant difference</td>
</tr>
<tr>
<td>HAMLET (Hemicraniectomy After MCA infarction with Life-threatening Edema Trial DECIMAL, DESTINY, &amp; HAMLET pooled analysis)</td>
<td>J. Hofmeijer, University Medical Centre, Utrecht, The Netherlands</td>
<td>clinical &amp; radiological criteria of malignant MCA infarct w/in 96 hrs of stroke symptom onset data from 3 trials only for patients recruited in the first 48 hrs after stroke symptom onset</td>
<td>multi-center RCT The Netherlands pre-planned pooled data analysis from 3 ongoing RCTs</td>
<td>mRS score &lt;4 at 1 yr dichotomized data favorable (mRS Score 0–4) &amp; unfavorable (mRS Score 5 &amp; death)</td>
<td>93 patients</td>
<td>proportion favorable outcomes (mRS score ≤4) higher in decompressive surgery group (75 vs 24%, PARR 51%, 95% CI 34–69), as was mRS score ≤3 (43 vs 21%, PARR 23%, 95% CI 5–41), and survival (78 vs 29%, PARR 50%, 95% CI 33–67); effect of surgery highly consistent across 3 trials</td>
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* MCA = middle cerebral artery; PARR = pooled absolute risk reduction.
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in which investigators will compare decompressive craniectomy and medical treatment with the best conservative therapy alone. A summary of the protocol, progress, and findings from these trials is presented in Table 2. Currently, only the combined results of the DECIMAL, DESTINY, and HAMLET trials have been published in the peer-reviewed literature. These multicenter trials were started independently in France, Germany, and The Netherlands, respectively. Because of their similar design and outcome measures, however, the steering committees planned a pooled analysis of the data from these trials while they were still ongoing to increase the chances of obtaining sufficient data and to minimize the number of patients involved in individual trials. This pooled analysis included 93 patients younger than 55 to 60 years who underwent decompressive craniectomy within 48 hours from the onset of stroke. The primary outcome measure for the pooled analysis was the proportion of patients with an mRS score of 4 or lower in the surgical and medical treatment groups at 1 year posttherapy. Survival and an mRS score of 3 or lower were secondary end points. The results have suggested that significantly more patients in the surgical group than in the control group had an mRS score of 4 or lower (75% compared with 24%; pooled absolute risk reduction 51%, 95% CI 34–69), an mRS score ≤ 3 (43% compared with 21%; pooled absolute risk reduction 23%, 95% CI 5–41), and survived (78% compared with 29%; pooled absolute risk reduction 50%, 95% CI 33–67). This effect was highly consistent across the three trials and did not change on adjustment for baseline differences. This study represents the best evidence available to date (and possibly ever) on the role of surgical decompression in malignant brain swelling associated with extensive cerebral infarction. As acknowledged by the investigators, however, these data cannot be translated automatically into the widespread use of decompressive surgery in all eligible patients: first, because the findings of an improved outcome cannot be extrapolated to older patients and those who undergo surgery within the first 48 hours after the onset of stroke; and second, although surgery doubles the number of patients who require minor support (mRS score ≤ 3), it increases 10-fold the number of patients with a moderately severe disability (mRS score ≤ 4) that requires almost continuous assistance. This fact must be carefully considered and discussed with the next of kin in each individual case before proceeding to surgery. More information is also required on the longer-term and neuropsychological effects of decompression, rate of surgical complications, and patient’s viewpoint on the procedure.

Authors of several studies have considered the role of decompressive craniectomy in the treatment of the hemorrhagic form of stroke, spontaneous intracranial hemorrhage, which is also associated with mass effect and elevated ICP. Data from all of these retrospective case series have revealed decreased mortality rates, and some suggest the possibility of an improved functional outcome after decompressive craniectomy. Prospective, randomized evidence is required to support these findings.

Other Indications

 Decompressive craniectomy has also been applied in the treatment of brain edema in a variety of other conditions. Reported indications, described predominantly in single cases or small case series, include meningitis, acute encephalitis, toxoplasmosis, acute disseminated encephalomyelitis, encephalopathy due to Reye syndrome, subdural empyema, and cerebral venous and dural sinus thrombosis.

Conclusions

Decompressive craniectomy as a surgical treatment for brain edema has been performed for many years and for several different pathophysiologies, including TBI, SAH, and ischemic stroke. The medical literature contains a wealth of information on decompressive craniectomy, almost exclusively observational series from individual centers. The role of this procedure remains unclear, however. We hope that randomized studies will provide Class I evidence that will aid the decision-making process in treating patients with refractory intracranial hypertension and brain edema.

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