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Decompressive Hemicraniectomy for the Treatment of Intractable Intracranial Hypertension After Aneurysmal Subarachnoid Hemorrhage

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Background and Purpose—Decompressive hemicraniectomy and duroplasty (DHCD) can improve survival in patients with severe cerebral edema. We present our clinical experience with DHCD for the treatment of refractory elevated intracranial pressure (ICP) in patients with aneurysmal subarachnoid hemorrhage (aSAH).

Methods—DHCD was performed in 16 patients (11 female; median age, 49.5 years) with aSAH (11 Hunt-Hess grade 4 to 5) for sustained ICP >250 mm H_2O refractory to maximal medical treatment and cerebrospinal fluid drainage at a median of 2 days from admission. Half of the patients were treated with endovascular coiling and the other half with surgical clipping.

Results—DHCD (mean flap size, 8536 mm^2) reduced ICP from 350±157 to 147±124 mm H_2O. Eleven patients survived (69%), and at latest follow-up (median, 450 days), 7 (64%) had a modified Rankin score of 0 to 3 and 4 (36%) a score of 4 to 5. Peak herniated brain volume was inversely associated with good outcome ($P<0.005$). Early craniectomy performed within 48 hours after the aSAH was associated with better outcome: 6 of 8 patients had good outcomes (75%) compared with 1 of 8 patients in whom late decompression was performed ($P<0.01$). Midline shift, Hunt-Hess grade, presence of hemorrhage, hematoma volume, craniectomy area, peak ICP, and relative ICP reduction were not associated with outcome in this patient population.

Conclusions—DHCD is a useful adjunct modality for management of refractory intracranial hypertension in patients with high-grade aSAH, even in the absence of large intraparenchymal hemorrhage. In our series, long-term outcome was better in patients who underwent early intervention. **(*Stroke*. 2007;38:987-992.)**

Key Words: cerebral edema ■ intracranial aneurysm ■ intracranial hypertension ■ subarachnoid hemorrhage ■ surgical decompression

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Hemicraniectomy has been used both prophylactically and as a salvage method for patients with cerebral edema refractory to medical management.[1–5] Although the common denominator has been the treatment of elevated intracranial pressure (ICP), the clinical indication has been variable and has included infarction, traumatic hemorrhage, nontraumatic hemorrhage, and infection.[1] Most of the recent literature on hemicraniectomy has addressed its use in the setting of massive hemispheric stroke.[2–5]

A number of recent studies have explored the role of hemicraniectomy in the setting of aneurysmal subarachnoid hemorrhage (aSAH)[6–9] associated with large intracerebral hemorrhage (ICH) in patients undergoing surgical clipping of the ruptured aneurysm. In the current report, we present our experience with decompressive hemicraniectomy (DHC) used in the setting of intractable ICP in aSAH patients with little or no ICH who underwent either endovascular coiling or surgical clipping.

Patients and Methods

Patient Selection

All aSAH patients presenting to the neurovascular service between July 2001 and May 2006 were admitted to the neurosurgical intensive care unit. External frontal ventriculostomies were inserted at the time of admission. Patients underwent either endovascular coiling or surgical clipping for treatment of the ruptured aneurysm within 12 to 24 hours. After aneurysm treatment, ICPs consistently >250 mm H_2O that did not respond to maximal medical therapy led to DHC and duroplasty (DHCD) in all of the patients presented here. Before DHCD was considered, malfunction of the ventriculostomy was addressed, including placement of a contralateral ventriculostomy.

Decompressive Hemicraniectomy and Duroplasty

After 3-point fixation of the head, a large, frontotemporal-parietal bone flap with smooth borders was turned by use of a high-speed craniotome. The dura was opened in a stellate fashion to minimize peripheral dural tension. The dura was then expanded with thin sheets of Alloderm dural graft material (LifeCell Inc). This enabled closure of the expanded duroplasty over the entire cortical surface in

Received August 17, 2006; accepted October 10, 2006.

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Stroke **is available at http://www.strokeaha.org** **DOI: 10.1161/01.STR.0000257962.58269.e2**

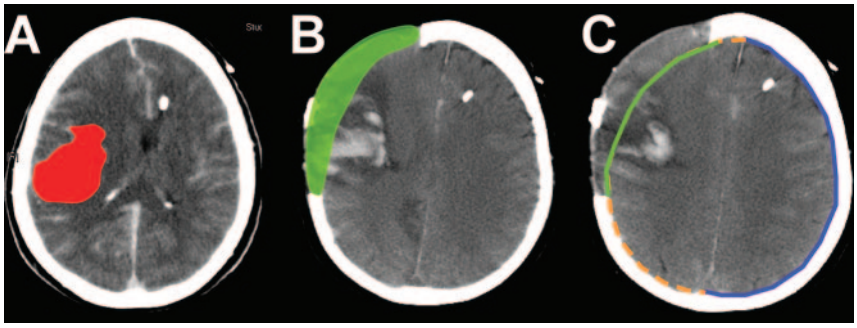


Figure 1. Schematic overview of the method used to derive ICH volume, HBV, and bone flap surface area. On every CT slice, ICH volume (A), HBV (B), and bone flap surface (C) are defined. See Patients and Methods for details.

a semiwatertight fashion. A subgaleal Jackson-Pratt drain was used, and the bone flap was either stored in a subcutaneous abdominal pocket or sent for storage at the bone bank (−80°C). After resolution of cerebral swelling and before initial discharge from the hospital (in all except patient 13), the bone flap was replaced by reopening the surgical incision and separating the galea from the duroplasty layer and then securing the bone flap in position with a titanium plating system.

Clinical Data

Clinical admission and outcome data were prospectively recorded in a central database. ICP and vital signs were recorded from the intensive care unit flowcharts. During long-term follow-up visits, patients were assessed according to the modified Rankin scale.[10] Modified Rankin scores were grouped into 2 tiers, for scores 0 to 3 (good outcome) and scores 4 to 6 (bad outcome). Data analysis was performed with JMP software (version 5.0; SAS).

Volumetric Imaging Analysis

Intraparenchymal hematoma (IPH) volumes were estimated on 5-mm serial axial computed tomographic (CT) cuts of the head with an open-source software package (Osirix, available at http://homepage.mac.com/rossetantoine/OsiriX)[11] running on a Macintosh Quad-G5 computer (Apple Inc). Polygonal regions of interest were defined on each axial section around the ICH, the herniated brain volume (HBV), and along the craniectomy defect. Volume computations of the polyhedron defined by the regions of interest for the ICH and the HBV and of the area of the craniectomy defect were performed by the software (Figure 1).

Results

Between July 2001 and May 2006, 16 patients were admitted with aSAH, that went on to undergo DHCD. Demographic information is presented in Table 1. The median age was 48.8 years, ranging between 28 and 70 years, with 11 female (69%) patients.

The majority of patients presented with Hunt-Hess grades 4 to 5 (11 of 16 patients, 69%). The most frequent aneurysm location was at the anterior communicating artery (5 patients, 31%; Table 1). Eight patients underwent surgical craniotomy and clipping of the aneurysm, and 8 patients were treated with endovascular coil embolization. Patients underwent DHCD to relieve intractable ICP at a median of 2 days (mean, 3.2 days) after presentation. CT examination of the head just before DHCD revealed a midline shift of 7.9±5.4 mm (mean±SD), ranging between 1 and 19 mm (Table 2).

Four patients presented with significant intraventricular hemorrhage (IVH) (25%); 12 patients had ICH (75%), with an average hematoma volume of 10.4±14.4 cm^3 and ranging

TABLE 1. Demographic Features of the Patients

No.	Age/Sex	Hunt-Hess Grade	Aneurysm Location	Treatment	Time to Craniectomy, d	Time to Cranioplasty, d	Length of Stay, d	VP Shunt at Discharge	Follow-Up, d	Modified Rankin Score
1	34/M	5	ACOM	Coil	3	24	62	Yes	62	4
2	54/F	2	ACOM/MCA	Clip	4	22	36	No	652	0
3	62/F	5	MCA/ACOM	Coil	0	42	58	Yes	978	3
4	34/F	3	ICA	Clip	4	...	19	...	...	6
5	64/F	4	MCA	Clip	18	21	76	Yes	930	4
6	70/F	4	MCA	Clip	1	37	43	Yes	39	4
7	28/F	5	ICA	Coil	0	...	3	...	...	6
8	58/F	4	PCOM	Coil	4	...	4	...	...	6
9	43/F	3	ACOM	Clip	0	20	25	No	450	0
10	57/M	5	MCA	Coil	3	...	8	...	...	6
11	48/M	2	ACOM	Coil	0.5	31	43	No	1175	0
12	43/F	4	ACOM	Coil	5	...	9	...	...	6
13	51/M	4	MCA	Clip	1	42	26	No	194	2
14	36/M	5	ACOM	Coil	0	26	38	No	233	0
15	57/F	4	ACHOA/MCA	Clip	1	27	36	Yes	681	3
16	41/F	1	ICA	Clip	6	44	59	Yes	59	4

VP indicates ventriculoperitoneal; ACOM, anterior communicating artery; MCA, middle cerebral artery; ICA, internal cerebral artery; PCOM, posterior communicating artery; ACHOA, anterior choroidal artery.

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TABLE 2. Features of the Initial Radiographic Examination, Hematoma Volumes, HBVs, and Radiographic Imaging Findings on Serial Examinations

No.	Fisher Grade	IVH/ICH	Hematoma Volume, cm^3	Midline Shift, mm	Side of Craniectomy	Craniectomy area, mm^2	Immediate HBV, cm^3	Peak HBV, cm^3	Time to Peak HBV, Days After Craniectomy
1	4	Yes/No	...	6	Left	12 927	197.6	209.8	8
2	4	No/Yes	4.4	7	Right	9023	117.9	124.4	8
3	4	No/Yes	34.3	11	Right	7159	26.9	105.4	13
4	2	No/Yes	2.9	6	Left	8295	105	130.5	3
5	4	No/Yes	5.3	16	Left	6336	48.2	98	5
6	4	No/Yes	42.9	19	Right	8403	60.6	119.6	12
7	4	Yes/Yes	12.6	12	Left	6764	76.9	126.7	1
8	4	No/Yes	3.0	1	Right	6644	74.5	132.2	1
9	2	No/No	...	4	Left	8175	108	81.4	9
10	4	No/Yes	34.4	9	Left	9846	142.1	187.3	2
11	4	No/Yes	19	7	Right	8800	93	81	2
12	4	Yes/Yes	2.8	2	Left	8009	129.4	154.2	1
13	2	No/No	...	15	Right	8500	92	104	5
14	4	No/Yes	6.0	5	Right	8591	93.8	119	3
15	2	Yes/Yes	3.1	5	Right	7958	76.1	87.9	6
16	1	No/No	...	1	Right	11 155	131.3	188.2	16

from 2.8 to 42.9 cm^3. Three patients presented with a combination of both (19%). The radiographic appearance of the aSAH was classified as Fisher grade 1 to 2 in 5 patients (31%) and grade 4 in 11 patients (69%). Only 5 patients (31%) had an ICH volume >6 cm^3.

DHCD was performed on the left side in 7 patients (44%); in patients who had undergone previous craniotomy for clipping, DHCD was performed on the ipsilateral side by extension of the preexisting craniotomy. In 8 patients who had undergone coil embolization of the aneurysm, DHCD was performed over the hemisphere with the greater amount of localized swelling (in 4 of 8 [50%] patients, on the left side). DHCD was performed on the right side in 9 of 16 cases (56%). The mean craniectomy area was 8536 ± 1682 mm^2,

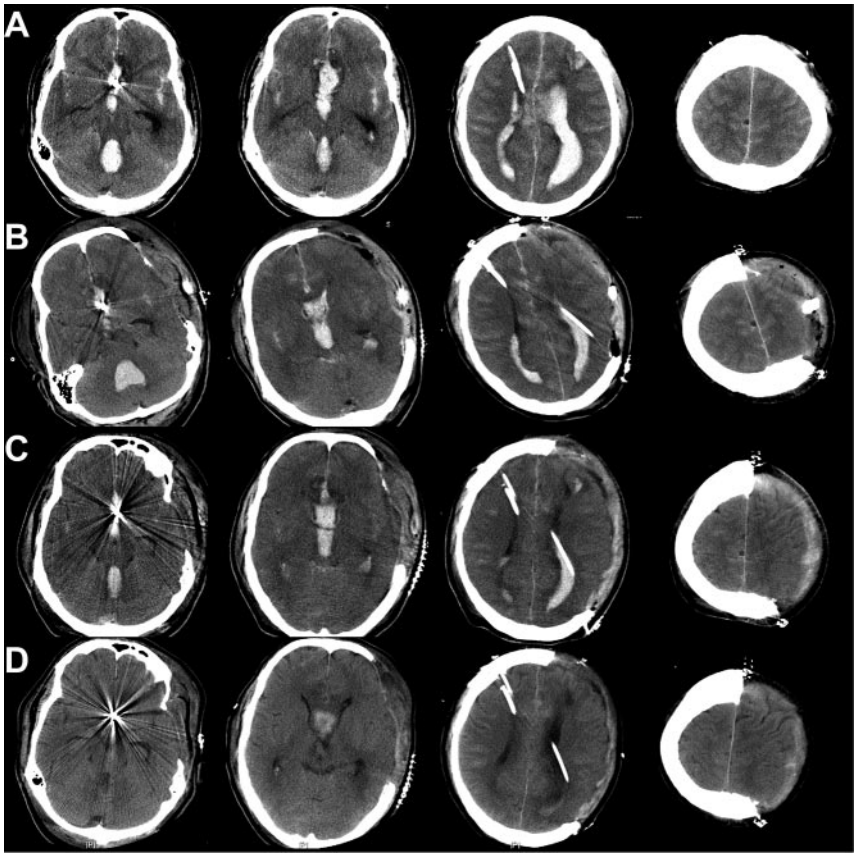


Figure 2. Images in a 34-year-old man (patient 1) who presented with Hunt-Hess grade 5 aSAH resulting from a ruptured anterior communicating artery aneurysm that was successfully treated with endovascular coil embolization. A large, intraventricular clot made effective cerebrospinal fluid drainage difficult, despite repeat intraventricular thrombolysis. Axial CT imaging (A) at initial presentation, (B) before DHCD, (C) immediately after DHCD, and (D) a later scan on day 8 showing peak swelling.

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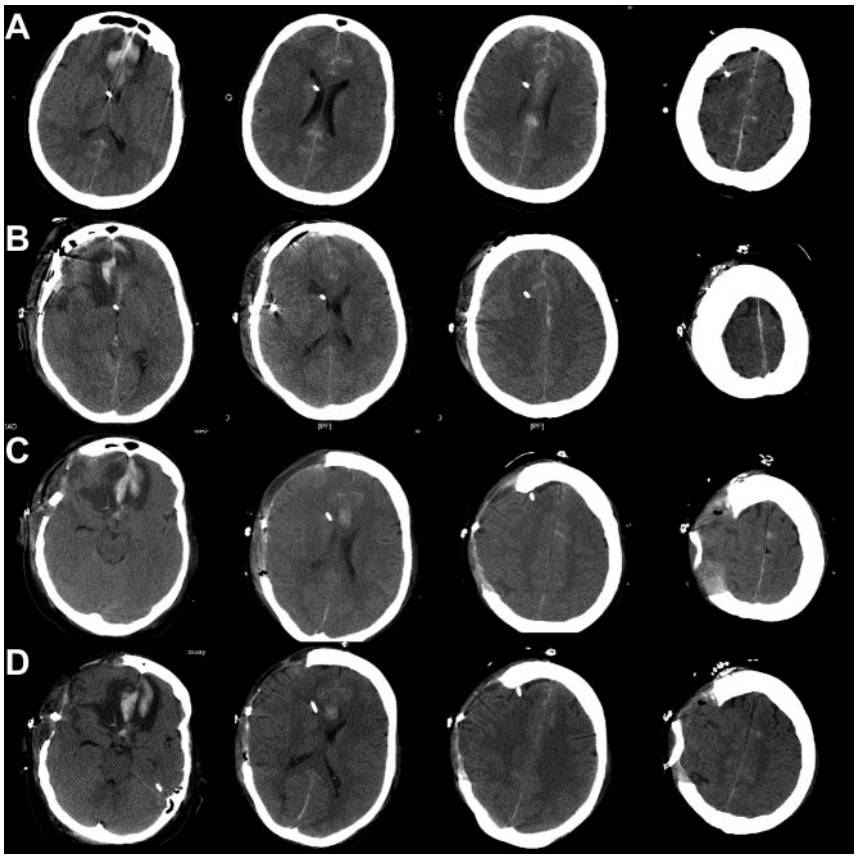


Figure 3. Images in a 54-year-old woman (patient 2) who presented with a Hunt-Hess grade 2 aSAH resulting from a ruptured anterior communicating artery aneurysm that was surgically clipped. Axial CT imaging (A) at initial presentation, (B) before DHCD, (C) immediately after DHCD on day 4, and (D) on day 8 showing peak swelling.

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ranging from 6336 to 12927 mm^2. Serial images obtained in 2 patients who underwent DHCD for intractable ICP in the absence of large IPHs are shown in Figures 2 and 3.

ICP peaked at 350±157 mm H_2O just before the DHCD and decreased significantly to a mean of 147±124 mm H_2O after the procedure ($P<0.001$; Figure 4, A and B). ICPs were recorded in 5 patients after replacement of the bone flap (mean, 85.9±33 mm H_2O), and these were not significantly different from the time-averaged ICPs before the replacement procedure (see Figure 4B).

HBV averaged 98.3±40.8 cm^3, ranging from 26.9 to 197.6 cm^3 immediately after decompression. In all patients in this series, cerebral edema continued to progress, with peak swelling reached 1 to 13 days after DHCD (median, 5 days) at a mean peak HBV of 128.1±38.8 cm^3 (range, 81 to 209.9 cm^3). Creation of a larger bone flap allowed a significantly larger brain volume to swell and herniate through the craniectomy defect both immediately after DHCD ($P<0.0001$) and during peak swelling ($P<0.002$).

Five patients (31%) died during their initial hospital stay, with a length of stay between 3 and 19 days. All patients who survived 20 days were eventually discharged to rehabilitation. Peak HBV was smaller in patients with a good outcome (7 patients; average volume, 100.4 cm^3) compared with an HBV of 149.6 cm^3 in 7 patients with bad outcomes ($P<0.005$). Early DHCD (within 48 hours after admission) was performed in 8 patients (56%), 4 of these after coil embolization and 6 having had a significant ICH >6 cm^3. Seven of the 8 patients (88%) in whom early DHCD was performed were discharged from the hospital. In contrast, 4 of 8 patients (50%) in whom late DHCD was performed had an ultimately fatal outcome (P=NS). Decompression on the nondominant right side was associated with reduced mortality (8 of 9 patients [89%] survived compared with 3 of 7 patients [43%] who were decompressed on the left side; $P<0.05$). After failure to wean from external ventricular drainage, 6 of the 11 patients discharged from the hospital (55%) required placement of a permanent ventriculoperitoneal shunt before discharge.

At long-term clinical follow-up (median, 450 days; range, 39 to 1175 days), 5 of 11 patients reached a modified Rankin score of 0 to 2 (45%); the remaining 55% (6 of 11 patients) had a modified Rankin score of 3 to 4 (Table 1). Early DHCD was associated with better outcome: 6 of 8 patients (75%) had good modified Rankin score outcomes compared with 1 of 8 patients in whom the decompression was performed after 48 hours ($P<0.01$).

Midline shift at the time of presentation ($P=0.92$), Hunt-Hess grade ($P=0.31$), Fisher grade ($P=0.22$), presence of IVH ($P=0.37$), presence of IPH ($P=0.77$) or the combination of IVH and IPH ($P=0.68$), hematoma volume ($P=0.89$), craniectomy area ($P=0.64$), peak ICP before decompression ($P=0.88$), and relative ICP reduction ($P=0.79$) were not statistically associated with outcome.

Discussion

Recognized as a potentially useful intervention to relieve intracranial hypertension after exceeding maximal cerebrospinal fluid drainage by Kocher in 1901,[12] early reports on decompressive craniectomy were not very encouraging.

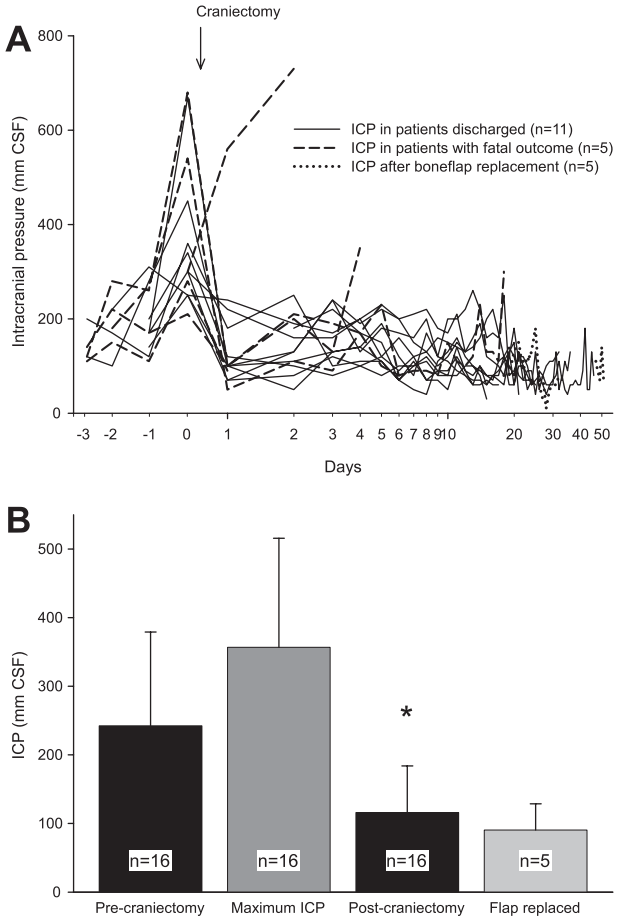


Figure 4. A, ICP recordings of patients in the series. Time shown is relative to the DHCD date; note the logarithmic time scale after DHCD. B, Mean ICP in the time between admission and DHCD, peak ICP that prompted performance of the DHCD, and mean ICPs after DHCD (significant vs peak ICP) and after replacement of the bone flap.

There is still no class I evidence to support or discourage the use of decompressive craniectomy for the treatment of refractory intracranial hypertension. Class II and III evidence exists to show that hemicraniectomy has proven a useful procedure in several diseases. The majority of the recent literature has focused on its utility in ischemic stroke, particularly holohemispheric stroke.[13–18] Its potential beneficial role in the setting of trauma has also been studied extensively.[19,20]

Studies have shown that in high-grade SAH patients without evidence of irreversible brain injury, rapid and continued effective ICP management is associated with improved outcomes.[21] Although the beneficial effects of decompressive craniectomy on the maintenance and recovery of brain function in the setting of refractory brain swelling remain poorly defined, cerebral perfusion pressure, cerebral tissue oxygenation, and reduction of mechanical compression of the basal cisterns have all been hypothesized to benefit from the intervention.[22,23] Fisher and Ojemann[24] argued for the favorable effects of rapid decompression in patients with SAH by describing a feed-forward cycle of increased ICPs that cause brain injury. In the setting of ischemic stroke, DHC has been shown to be a highly effective way to treat ICH, particularly when combined with extensive durotomy.[25] The volume gained after decompressive craniectomy was calculated to be between 15.9 and 247.4 cm^3, with a median volume of 73.6 cm^3,[23] a value similar to HBV in our series of between 26.9 and 197.6 cm^3 (median, 122 cm^3) at the time of peak swelling. Two series have examined whether the ICP reduction seen in ischemic stroke after DHCD could also be achieved in SAH, with the potential for improved patient outcomes.[7,9] The first examined the role of prophylactic hemicraniectomy performed at the time of surgical clipping of ruptured middle cerebral artery aneurysms. All 8 patients included in that study had a Hunt-Hess grade of 4 or 5, along with a large sylvian hematoma. The study confirmed the effectiveness of hemicraniectomy in reducing ICP and showed that hemicraniectomy may lead to improved outcome in a carefully selected subgroup of patients.[7] The second study also focused on surgically clipped aneurysm patients with high-grade Hunt-Hess 4 and 5, perisylvian or temporal lobe clots, and clinical signs of brainstem compression. The procedure was not performed prophylactically. Although the study showed a statistically nonsignificant increase in short-term survival, it did not detect any improvement in overall patient quality of life.[9]

The current study sought to evaluate the utility of DHCD in managing refractory intracranial hypertension in patients with aSAH, regardless of treatment modality or presence of ICH. The majority of our patients (11 of 16, 69%) had only a small IPH measuring <6 cm^3 or no IPH. We found that DHCD performed in the first 48 hours after aSAH had a beneficial effect on outcome: 75% of patients (6 of 8) who underwent early DHCD fared better at long-term follow-up (modified Rankin score, 0 to 3) compared with only 12.5% of the group of patients in whom DHCD was performed after 48 hours (1 of 8 patients). The period of 48 hours in many cases precedes onset of vasospasm, but a longer latency period may pose the additional diagnostic challenge of distinguishing between the cause of a patient's decline that may stem from the development of symptomatic intracranial hypertension or the onset of vasospasm. In a population of patients with posttraumatic cerebral edema, younger patients who underwent decompression in <48 hours, though having had a maximal ICP of 40 mm Hg, 60% had better outcomes compared with 18% in a matched control group, suggesting a beneficial role of early decompression.[19]

Delayed cerebral ischemia from vasospasm contributes a major part of the morbidity and mortality in patients with aSAH.[26,27] Hypertensive, hypervolemic, and hemodilutional therapy is a widely accepted therapy for vasospasm secondary to aSAH, but such treatment may be hindered in its effectiveness by significant medical comorbidities, including pulmonary edema, myocardial ischemia, hyponatremia, renal medullary washout, indwelling catheter–related complications, cerebral hemorrhage, and cerebral edema.[26–28] DHCD may allow hypertensive/hypervolemic/hemodilutional therapy to be used to its full potential, allowing for cerebral edema, that would, in the absence of decompression, cause unacceptably elevated ICPs with deleterious effect. In addition, decompressive craniectomy may also enable the normalization of arterial P_{CO_2} with its beneficial effect on counteracting vasoconstriction without risking concomitant

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intractable ICP. Although hemicraniectomy reduced immediate mortality,[3,9] the decrease was not found to be uniformly significant, because the overall experienced quality of life of the surviving patients in that study was poor.[9] We assessed long-term outcome with the modified Rankin scale and found the results to be encouraging in this cohort of high-grade patients. Seven of 11 patients (63%) presenting with high-grade SAH (Hunt-Hess grade 4 to 5) survived their initial hospital stay, with 4 patients (of 7, 57%) reaching a good outcome (modified Rankin score 0 to 3) at long-term follow-up, suggesting that in some patients, a considerable good quality of life may be reached after rehabilitation. This compares favorably with other studies,[7,9] even though the majority of our patients did not have a large ICH.

Summary

Refractory intracranial hypertension secondary to aSAH poses a difficult clinical management problem, with osmotic dehydration and hyperventilation conflicting with the optimal therapy of delayed ischemia from vasospasm. In the current series of patients undergoing DHCD for refractory ICP after aSAH, the data suggest reduced overall mortality for decompression on the nondominant side and better long-term outcomes in patients in whom early decompression was performed within 48 hours after aneurysm rupture.

Acknowledgments

We are grateful for the clinical assistance of Joyce Pugatch-Scally MS, APRN.

Disclosures

None.

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