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Clinical Article

Decompressive hemicraniectomy in patients with subarachnoid hemorrhage and intractable intracranial hypertension

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Summary

Background and purpose. To evaluate the outcome of patients with aneurysmal subarachnoid hemorrhage (aSAH) developing intractable intracranial hypertension and treated by decompressive hemicraniectomy (DHC).

Methods. Of 193 patients with aSAH 38 patients were treated with DHC after early aneurysm clipping. Indications for DHC were 1. Signs of brain swelling during aneurysm surgery (group 1: primary DHC). 2. Intracranial pressure- (ICP)-elevation and epidural, subdural or intracerebral hematoma after aneurysm surgery (group 2: secondary DHC due to hematoma) 3. Brain edema and elevated ICP without radiological signs of infarction (group 3: secondary DHC without infarction). 4. Brain edema and elevated ICP with radiological signs of infarction (group 4: secondary DHC with infarction).

Results. Thirty-one patients (81.6%) suffered from high grade aSAH Hunt & Hess 4–5. 21 belonged to group 1, five to group 2, six to group 3 and six to group 4. Of a total of 38 patients a good functional outcome according to Glasgow Outcome Score (GOS 4 & 5) could be reached in 52.6% of the cases. 26.3% survived severely disabled (GOS 3), no case suffered from a vegetative state (GOS 2) but 21.1% died (GOS 1). After 12 months good functional outcome could be achieved in 52.4% of the cases in group 1, in 60% in group 2, in 83.3% in group 3 and in 16.7% in group 4.

Conclusions. In more than half of the patients with intractable intracranial hypertension after aSAH a good functional outcome could be achieved after DHC. Patients with progressive brain edema without radiological signs of infarction and those with hematoma may benefit most. The indication for DHC should be set restrictively if secondary infarcts are manifest.

Keywords: Decompressive hemicraniectomy; subarachnoid haemorrhage; brain edema; cerebral vasospasm; intracerebral hematoma.

Introduction

Patients with high grade aneurysmal subarachnoidal hemorrhage (aSAH) often show a poor outcome [2, 4, 6, 18, 27]. In recent years more experience in microneurosurgical techniques, endovascular coiling and extended resources in neuro-intensive care have made possible the treatment of patients with most severe aSAH and to exclude aneurysms from the circulation in the acute stage of a traumatised brain [10, 15, 18, 31]. These patients, however, suffer from a high incidence of specific complications such as severe brain edema with refractory elevations of intracranial pressure (ICP) and symptomatic cerebral vasospasm (CVS).

Recent literature dealing with DHC after hemispheric infarction and traumatic brain injury show encouraging results, although the patient selection, timing of surgery and functional recovery are discussed controversially [1, 5, 23, 24, 26]. So far, only a few small case series of patients treated by decompressive hemicraniectomy (DHC) after severe aSAH have been described [7, 9, 16, 27, 28].

The purpose of this report is to present the results of the up to date largest patient series treated by DHC after aSAH and early aneurysm clipping and to discuss the indications for DHC.

Methods

Patient population

The study was approved as a part of the project E-015/99 by the Ethics Committee of the University of Zurich. Between January 1999 and December 2003, 193 patients with aSAH were admitted to the Department of Neurosurgery, University Hospital Zurich and treated by aneurysm clipping within three days. During this period 38 patients were treated by DHC due to brain swelling and elevated ICP resistant to conventional treatment. Patients treated with endovascular coiling were excluded from the analysis.

Structured treatment

Aneurysm clipping was performed according to the standard microsurgical technique described by Yasargil [30]. Emergency treatment of elevated ICP consisted in sedation with intubation, osmotherapy (Mannitol 20% and hypertonic NaCl-hydroxyethyl-starch solution (Hyper-Haes)), mild hyperventilation and thiopental boli in a dose of 10 mg/kgBW. Craniotomy and aneurysm clipping were performed within the next hours, after emergency diagnostic evaluation with angiography or CT angiography. Patients with poor-grade aSAH and severe brain edema remained sedated (fentanyl infusion 2–8 µg/kg/h and midazolam 0.1–0.4 mg/kg/h) after surgery. A ventricular catheter (NMT Neuroscience, Frankfurt, Germany), a subdural (NMT Neuroscience, Frankfurt, Germany) or an intraparenchymatous (Codman microsensor, Johnson & Johnson, Raynham) ICP probe were inserted. All patients with aSAH were treated with nimodipine given intravenously. Dexamethasone was given peri-operatively.

Treatment of elevated ICP

If ICP (>20 mmHg) was elevated, treatment with intermittent cerebrospinal fluid (CSF) drainage, osmotherapy and mild hyperventilation was initiated. Patients with persistant ICP-values >20 mmHg were eligible for treatment with barbiturate coma combined with mild hypothermia [11]. Cooling of the patients (target brain temperature 33 °C) was accomplished by endovascular cooling catheters (Cool Line Catheter and Coolgard System; Alsius Corporation, Irvine, CA, USA). All patients were treated under extended monitoring of cerebral hemodynamics (monitoring of jugular bulb oxygen saturation, cerebral blood flow monitoring with near infrared spectroscopy indocyanine green dye dilution and/or intraparenchymatous oxygen partial pressure ($ptiO_2$)) [8, 20, 21].

Treatment of cerebral vasospasm

Transcranial Doppler (TCD) blood flow measurements were performed daily. Triple-H therapy was induced if signs of CVS were manifest, e.g. increased TCD blood flow velocities, and/or development of delayed ischemic neurological deficits (DIND) due to CVS. Excessive natriuresis and diuresis were inhibited with fludrocortisone 0.2 mg/day. If patients with DIND did not improve or worsened, angiography and treatment with percutaneous angioplasty and/or superselective papaverine infusion (total dose of 300 mg) into the vasospastic vessels was performed. Symptomatic CVS, resistant to conventional treatment or re-occurring after angioplasty and papaverine, were treated with barbiturate coma and/or hypothermia.

Decompressive craniectomy

1. DHC was performed in patients after aSAH with signs of brain swelling occurring during or immediately after craniotomy and aneurysm surgery (angry brain). The bone flap was not replaced and duraplasty was performed if necessary (group 1: primary DHC). The average diameter of craniectomy was 9 cm due to enlarged skin and bone opening in high grade SAH patients suspected of brain edema. 2. Patients with ICP-elevations (>20 mmHg) and space occupying epidural, subdural or intracerebral hematomas after aneurysm surgery were treated with decompressive craniotomy combined with hematoma evacuation (group 2: secondary DHC due to hematoma). 3. If, after aneurysm surgery, patients developed increasing brain edema and elevated ICP (>20 mmHg) resistant to conventional treatment but without radiological signs of infarctions, secondary DHC in combination with duraplasty was performed (group 3: secondary DHC without infarctions). 4. The fourth group treated by DHC were patients with ICP values >20 mmHg, the CT scans revealing infarctions (secondary DHC with infarctions). In case of secondary decompression a hemicraniectomy with removal of frontal, temporal and parietal bones (diameter in average 14–15 cm) was performed. Pericranium, homologous temporal fascia or Silastic (polydimethylsiloxane) were used for duraplasty [32]. The bone flap was frozen and stored at −80 °C. Replacement of the bone flap depended on reduction of edema and was performed within three months after craniectomy.

Outcome measurements

Neurological outcome was assessed after 12 months in the outpatient clinic by a neurologist using the Glasgow Outcome Score (GOS) [17], GOS 1 denominating death, GOS 2 vegetative state, GOS 3 severe disability, GOS 4 moderate disability and GOS 5 mild or no disability.

Statistical analysis

To compare the neurological outcome between patient groups Fisher's exact test, and the time of DHC after aSAH unpaired t-test were applied.

Results

Of 193 patients treated by early aneurysm surgery, 38 (19.7%) were treated by DHC. The patient and outcome characteristics are summarized in Tables 1 and 3 . 31 patients (81.6%) suffered from high grade aSAH Hunt & Hess 4–5. The distribution of the bleeding aneurysms is given in Table 2. Multiple aneurysms were found in 39.5% of the cases. 24 external ventricular drainages were inserted. In group 1 (primary DHC) 11 of 21 patients and in group 2–4 (secondary DHC) 12 of 17 patients needed a ventriculo-peritoneal shunt during hospitalisation. No severe side effects associated with DHC like wound infections or hematomas occurred.

Grouped according to the indications for DHC the results are as follows: In 21 patients (55.3%) with signs of brain swelling during aneurysm surgery (angry brain) the bone flap was not replaced (group 1: primary DHC). Eight additional patients (21.1%) were treated by primary and secondary surgical decompression. For analysis they are attributed to group 2–4. Group 2 (secondary DHC due to hematoma) included five patients (13.2%) with epidural, subdural or intracerebral hematoma treated by secondary DHC and hematoma evacuation. Group 3 (secondary DHC without infarctions) included six patients (15.8%) developing brain edema and intractable elevated ICP after aneurysm surgery. Group 4 (secondary DHC with infarctions) consisted of six patients (15.8%) with ICP values >20 mmHg caused by ischemic brain edema, the CT scans revealing infarctions.

In eight patients, not having had the bone flap replaced after aneurysm clipping, the craniotomy was enlarged and duraplasty was perfomed in a second operation (primary and secondary DHC). In two of these patients ICP increased >20 mmHg due to primary intracerebral hema-

Table 1. *Patients' characteristics*

	All patients ($N=38$)	Group 1 ($N=21$)	Group 2 ($N=5$)	Group 3 ($N=6$)	Group 4 ($N=6$)
Age, years mean ± SD	50.1 ± 11.8	49.4 ± 12.5	53.8 ± 14.4	46.3 ± 10.4	53.8 ± 8.2
Gender N (%)					
Male	11 (28.9)	7 (33.3)	0 (0)	3 (50)	1 (16.7)
Female	27 (71.1)	14 (66.7)	5 (100)	3 (50)	5 (83.3)
Hunt & Hess N (%)					
5	13 (34.2)	9 (42.9)	2 (40)	1 (16.7)	1 (16.7)
4	18 (47.4)	9 (42.9)	1 (20)	4 (66.6)	4 (66.6)
3	2 (5.2)	1 (4.7)	0 (0)	1 (16.7)	0 (0)
2	5 (13.2)	2 (9.5)	2 (40)	0 (0)	1 (16.7)
1	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
WFNS N (%)					
5	28 (73.8)	18 (85.7)	2 (40)	4 (66.6)	4 (66.6)
4	4 (10.5)	1 (4.8)	1 (20)	1 (16.7)	1 (16.7)
3	1 (2.6)	0 (0)	0 (0)	1 (16.7)	0 (0)
2	4 (10.5)	2 (9.5)	1 (20)	0 (0)	1 (16.7)
1	1 (2.6)	0 (0)	1 (20)	0 (0)	0 (0)
Fisher grade N (%)					
4	30 (79)	16 (76.2)	3 (60)	6 (100)	5 (83.3)
3	7 (18.4)	5 (23.8)	1 (20)	0 (0)	1 (16.7)
2	1 (2.6)	0 (0)	1 (20)	0 (0)	0 (0)
1	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

N Number of patients; *SD* standard deviation; *WFNS* World Federation of Neurosurgical Societies.

Table 2. *Localization of bleeding aneurysm*

	All patients ($N=38$)	Group 1 ($N=21$)	Group 2 ($N=5$)	Group 3 ($N=6$)	Group 4 ($N=6$)
Anterior communication artery N (%)	8 (21.1)	6 (28.6)	0 (0)	0 (0)	2 (33.3)
Middle cerebral artery N (%)	19 (50)	8 (38.1)	3 (60)	5 (83.3)	3 (50)
Pericallosal artery N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Internal carotid artery N (%)	5 (13.2)	4 (19.1)	0 (0)	0 (0)	0 (0)
Ophthalmic artery N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Posterior communicating artery N (%)	3 (7.9)	2 (9.5)	1 (20)	1 (16.7)	0 (0)
Anterior choroid artery N (%)	1 (2.6)	0 (0)	0 (0)	0 (0)	0 (0)
Basilar artery N (%)	1 (2.6)	0 (0)	0 (0)	0 (0)	1 (0)
Posterior inferior cerebellar artery N (%)	1 (2.6)	1 (4.8)	1 (20)	0 (0)	0 (0)
Multiple aneurysms N (%)	15 (39.5)	7 (33)	2 (40)	3 (50)	3 (0)

N Number of patients.

Table 3. *Patient outcome after 12 months*

	All patients ($N=38$)	Group 1 ($N=21$)	Group 2 ($N=5$)	Group 3 ($N=6$)	Group 4 ($N=6$)
GOS 5, N (%)	10 (26.3)	7 (33.3)	0 (0)	2 (33.3)	1 (16.7)
GOS 4, N (%)	10 (26.3)	4 (19.05)	3 (60)	3 (50)	0 (0)
GOS 3, N (%)	10 (26.3)	4 (19.05)	1 (20)	1 (16.7)	4 (66.6)
GOS 2, N (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
GOS 1, N (%)	8 (21.1)	6 (28.6)	1 (20)	0 (0)	1 (16.7)

GOS Glasgow outcome score; *N* number of patients.

toma (both with ruptured aneurysms of the middle cerebral artery) and the primary craniotomy was enlarged in combination with hematoma evacuation. Four patients, all with most severe aSAH Hunt and Hess grade 4–5, were treated by primary and secondary decompression due to increasing brain edema and intractable intracranial hypertension and two patients due to ischemic brain edema with infarctions. Secondary DHC was performed usually on day 4 (range 1–17 days) after bleeding.

Symptomatic CVS was detected in 13 (33.2%) of the 38 patients. Irreversible DIND occurred in nine patients (23.7%).

Table 4. *Patient, treatment characteristics and outcome for every single patient*

Patient number, sex, age	Severity grade of SAH			Type of aneurysm		Day of clipping after bleeding	Treatment of elevated ICP			Decompressive Hemicraniectomy		DIND	GOS after 12 months
	Hunt & Hess grade	WFNS grade	Fisher grade	Ruptured	Unruptured		Barbiturate coma	Hypo-thermia (33 °C)	VD	Primary craniectomy	Secondary craniectomy (day after bleeding, reason)		
1, F, 71	4	4	4	MCA r	MCA l	2	−	−	+	+	+ (5, ICH)		4
2, F, 57	4	5	3	MCA r		1	−	−	−	+	−		5
3, F, 50	5	5	4	MCA r	MCA l	1	−	−	−	+	−		4
4, M, 62	4	5	4	AcomA		0	−	−	+	+	+ (14 + 17, CVS + edema + infarction)	+	3
5, F, 37	5	5	4	MCA l	MCA r	1	−	−	−	+	−		3
6, M, 46	4	5	4	ICA/OA l		1	−	−	+	+	−		4
7, F, 47	2	2	3	AcomA		1	−	−	−	+	−		5
8, F, 59	4	4	4	AcomA		1	+	+	+	−	+ (3, edema + early infarction)		3
9, F, 68	2	2	3	PcomA l		0	−	−	+	−	+ (1, EDH + SDH)		4
10, F, 46	4	5	4	MCA r	MCA l	1	+	+	−	+	+ (3, edema)		4
11, M, 52	5	5	4	ICA		2	−	−	+	+	−		3
12, M, 65	5	5	4	MCA r		3	+	+	−	+	−		1
13, F, 52	5	5	4	PcomA r		1	+	+	+	+	−		1
14, M, 35	4	5	4	AcomA		0	−	−	−	+	−		5
15, M, 31	4	4	4	MCA l		1	+	+	−	+	+ (4, CVS + edema)	+	4
16, M, 35	5	5	4	MCA r		+	+	+	+	+	−	+	1
17, F, 58	4	4	4	MCA r		0	+	+	+	−	+ (2 + 5, CVS + edema + infarction)		3
18, F, 44	5	5	4	MCA r		0	+	+	−	−	+ (1, EDH)		4
19, F, 61	5	5	4	MCA r		1	−	−	+	+	−	+	1
20, F, 48	4	5	4	OA r		0	−	−	+	+	−		5
21, F, 45	4	5	4	MCAr		0	+	−	−	+	+ (3, edema + early infarction)		5
22, F, 44	5	5	4	AcomA		0	+	+	+	+	−		3
23, F, 59	3	3	4	MCA l		0	+	+	+	−	+ (7, CVS + edema)		4
24, F, 61	4	5	4	MCA r		0	−	−	+	+	−		1
25, F, 58	2	2	3	BA/SCA r	MCA r	0	+	+	+	−	+ (8, CVS + edema + infarction)	+	3
26, F, 57	4	5	4	MCA r		0	−	+	+	+	+ (1, edema)	+	3
27, F, 42	2	1	2	PICA l		1	+	+	+	−	+ (4, SDH)		1
28, M, 41	4	5	4	MCA r,	VA l	0	+	+	−	+	+ (5, CVS + edema)		5
29, F, 63	4	5	3	Achorant r	ICA bifurcation r	0	−	−	+	+	−		4
30, M, 49	4	5	4	AcomA		0	+	+	+	+	−		5
31, M, 44	5	5	4	ICA l		1	+	+	+	−	+ (3, edema)		5
32, F, 32	2	2	3	AcomA		1	+	+	−	+	−		5
33, F, 44	5	5	4	MCA l,	MCA l	0	+	+	+	+	+ (1, ICH)	+	3
34, F, 75	4	5	4	PcomA r		0	−	−	+	+	−		3
35, M, 54	4	5	4	MCA l		0	+	+	+	+	−		1
36, F, 53	3	4	3	ICA l	AcomA, MCA r	1	−	−	−	+	−		4
37, F, 45	5	5	4	MCA l	BA tip ± PCA l	2	+	+	+	−	+ (4, CVS + edema + infarction)	+	1
38, F, 22	5	5	4	AcomA		1	−	−	−	+	−	+	5

SAH Subarachnoid hemorrhage; *ICP* intracranial pressure; *DIND* delayed ischemic neurologic deficit; *GOS* Glasgow outcome scale; *WFNS* World Federation of Neurosurgical Societies; *VD* ventricular drainage; *r* right; *l* left; *MCA* middle cerebral artery; *AcomA* anterior communicating artery; *PcomA* posterior communicating artery; *ICA* internal carotid artery; *PCA* posterior cerebral artery; *OA* ophthalmic artery; *BA* basilar artery; *SCA* superior cerebellar artery; *VA* vertebral artery; *ICH* intracerebral hemorrhage; *CVS* cerebral vasospasm; *EDH* epidural hemorrhage; *SDH* subdural hemorrhage.

The outcome of all patients and the patient groups after 12 months is shown in Table 3. A good functional outcome (GOS 4 and 5) was achieved in 20 of 38 patients (52.6%). 10 patients survived severely disabled (26.3%) and eight patients died (21%). In group 1 (primary DHC) 11 of 21 patients (52.3%) survived with good functional outcome, four patients survived severely disabled (19.05%) and six patients died (28.6%). In group 2 (secondary DHC due to hematoma) three of five patients survived with good functional outcome (60%), one patient survived severely disabled (20%) and one patient died (20%). In group 3 (secondary DHC without infarctions) five of six patients survived with good functional outcome (83.3%), one patient survived severely disabled (16.7%). In group 4 (secondary DHC with infarctions) of six patients only one survived with good functional outcome (16.7%), whereas four patients survived severely disabled (66.7%) and one patient died (16.7%). In patients who had to undergo secondary DHC due to hematoma, secondary DHC was performed in average 2.4 ± 1.9 days after aSAH. Patients with brain edema without infarctions were usually treated earlier by secondary DHC (after 3.8 ± 2 days) compared to those with brain edema and infarctions (after 7.0 ± 5.6 days ($p = 0.11$)). Patients who recovered with good functional outcome (GOS 4 and 5) tended to have been treated earlier by secondary DHC (within 3.6 ± 1.6 days after aSAH) than those who died or survived with severe or moderate disability (GOS 1–3), (within 5.9 ± 5.5 days ($p = 0.12$)).

Discussion

Despite recent advances in microsurgical techniques and neurocritical care, patients with severe aneurysmal aSAH often suffer from bad outcome [2, 3, 22, 29]. Besides the initial injury and the occurrence of CVS, delayed brain swelling with ICP elevations influences the outcome decisively [2, 14, 22, 27]. The acute aSAH is accompanied by increasing ICP and a subsequent increase of cerebrovascular resistance with a reduction of cerebral blood flow (CBF) leading to a vicious cycle of anoxia and more extensive edema and ischemia [2, 9, 27].

Recent literature shows promising results applying DHC in patients with severe brain edema and refractory ICP elevations after hemispheric infarction and traumatic brain injury [1, 4, 23, 24, 26]. A large DHC combined with dural opening and duraplasty leads to an impressive intraoperative two step reduction of raised ICP values [16, 27]. This normalisation of ICP values is associated with an immediate, significant improvement of tissue perfusion and oxygenation [16, 27]. However, only a few publications deal with DHC in patients with aSAH, severe brain edema and intracranial hypertension [7, 9, 16, 27]. Fisher *et al.* published a case report of a woman being successfully treated by bilateral DHC to fight worsening of acute SAH [9]. Smith *et al.* treated eight patients with prophylactic DHC after severe SAH from middle cerebral artery (MCA) aneurysms with associated large Sylvian fissure hematomas [27]. Five patients recovered with favourable outcome. Jaeger *et al.* studied three patients suffering from severe cerebral edema and intracranial hypertension after aSAH [16]. Although mean ICP decreased and simultaneously measured $ptiO_2$ rapidly increased with DHC, the patients' clinical status remained poor [16]. In a most recent study D'Ambrosio *et al.* analyzed a group of 12 patients with poor-grade aSAH, perisylvian or temporal lobe hemorrhage and clinical signs of brain stem compression [7]. Eight patients had a hemicraniectomy during first surgery and four initially underwent aneurysm clipping and subsequently returned to the operating theatre for secondary DHC due to worsening of neurological status. Compared to 10 patients treated with clot evacuation, a 12-months follow-up established that three (43%) of seven survivors in the group studied remained severely disabled, compared to two (33%) of the six patients in the control group. The authors concluded that if DHC is performed in patients with poor-grade aSAH and intracerebral hemorrhage the overall quality of life experienced by survivors is poor. The 12 months follow-up of our patient series established that 10 (33%) of 30 survivors and in the hematoma group one (25%) of four survivors remained severely disabled (GOS 3). None survived in a vegetative state (GOS 2). The groups treated by DHC in the two studies may not be compared directly due to different bleeding characteristics and overall management. However, one reason for the better outcome concerning morbidity in the present series might be that due to ICP-monitoring DHC in our patients was performed early whereas D'Ambrosio et al. intervened only after clinical signs of brainstem compression were manifest.

In our patient series the outcome of the patients differed according to the indication for DHC. Secondary DHC in patients with elevated ICP due to ischemic brain edema with radiological infarctions lead only in one of six patients to a favourable outcome, whereas three of five patients with epidural, subdural or intracerebral hematoma and five of six patients with brain edema

without infarctions survived with good functional outcome. This discrepancy in outcome between the patient groups might have two reasons: 1. The etiology of brain edema might affect the outcome substantially. Space-occupying intracerebral hematoma can be removed surgically and intracranial hypertension can be treated rapidly. Russegger *et al.* investigated patients with poor grade aSAH suffering from large aneurysmal intracerebral hematomas [25]. After evacuation of the hematoma, clipping of the aneurysm and DHC a good outcome was achieved in 9 of 19 patients. On the other hand, treatment options of ischemic brain edema are less effective. Hacke *et al.* showed that with conventional treatment of elevated ICP up to 80% of patients with complete MCA territory infarction die from transtentorial herniation [12]. The successful treatment of early ischemia in the reversible state, with spasmolysis in the presence of CVS or with neuroprotective treatment strategies, may have the greatest influence on the outcome. Definite tissue infarction, inducing the ischemic cascade to run off completely and leading to malignant cytotoxic brain edema, must be prevented in any case. This might explain the difference in outcome between group 3 (secondary DHC without infarctions) and group 4 (secondary DHC with infarctions). 2. Although statistically not significant because of the small sample size, patients who recovered with good functional outcome tended to be treated by secondary DHC earlier than those who died or survived with severe or moderate disability. This might emphazise the necessity to determine the correct timing for DHC in patients with severe aSAH and increasing ICP. In patients with primary craniectomy the decision was made very early due to the intra-operative aspect of brain swelling (angry brain). Smith *et al.* propose the use of prophylactic large fronto-temporo-parietal DHC in poor-grade aSAH patients presenting with associated large Sylvian hematomas after ruptured aneurysms of the MCA [27]. The advantage of primary DHC is the immediate ICP reduction, prevention and control of ICP increase occurring during the first days. Furthermore, primary DHC minimizes the risk of a second operation during the most vulnerable period when the patient is threatened by diffuse brain swelling and CVS. In addition, to differ between edema and CVS as the reason for deterioration might be difficult during the course [27] of the illness. A further argument in favour of primary DHC and/or early secondary DHC might be the fact that brain damage caused by ICP elevations and consecutive edema can occur before ICP changes are detected [16, 27]. Evidence for the usefulness of combined ICP, $ptiO_2$ monitoring or jugular bulb oximetry in order to detect cerebral edema compromising cerebral perfusion and to set the optimal time point for secondary craniectomy is given [13, 28]. This strategy was adopted in our patients of group 2–4. In a recent retrospective study Strege *et al.* analysed the course of illness of 26 patients treated by secondary DHC due to extensive cerebral edema after aSAH or severe head injury [28]. They showed that pathological trends in monitoring ICP and $PtiO_2$ always preceded clinical deterioration. Furthermore, the combination of ICP and PtiO2 monitoring was superior to solely ICP-guided management.

The present study has several limitations. The differences in outcome between the four groups reflect the heterogeneity of the study population, especially regarding the indications of primary versus secondary decompression. Although the indications for DHC were prospectively defined, data analysis was performed retrospectively without a control group. Further the location of the aneurysm might have had influence on the outcome. However numbers were not sufficient to draw reliable conclusions. Even though the present patient series treated by DHC after aSAH is the largest currently available in literature, it is too small to have statistical impact and to give guidelines concerning indications and timing of DHC.

In conclusion, our preliminary data show that in more than half of a selected patient population with intractable intracranial hypertension after aSAH good functional outcome can be achieved after DHC. Patients with progressive brain edema without radiological signs of infarctions and those with hematoma and consecutive ICP elevations might benefit most from DHC. On the other hand, patients manifesting severe infarctions on CT scans should be excluded from this aggressive treatment option in future. DHC may be indicated if performed early in a very carefully selected subset of patients with refractory intracranial hypertension after aSAH. Further prospective studies with standardized treatment protocols and clear indications for DHC will be needed to identify select subsets of patients who benefit most regarding survival rate and long-term quality of life.

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