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Original Article

Specialized Neurocritical Care, Severity Grade, and Outcome of Patients With Aneurysmal Subarachnoid Hemorrhage

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Abstract

Introduction: To evaluate the impact of specialized neurocritical care on the population admitted to a neurovascular center and on the outcome of patients with severe aneurysmal subarachnoid hemorrhage (aSAH).

Methods: After exclusion of patients treated with endovascular techniques, between 1999 and 2003, 198 patients with aSAH treated with early aneurysm clipping were analysed. In 1999, a new standardized protocol for intensive care treatment was established in the Department of Neurosurgery, University Hospital Zurich. The results were compared to the earlier time period (1993–1994) immediately after introduction of early aneurysm clipping.

Results: Out of 198 patients with aSAH, 90 patients (45.5%) suffered from mild aSAH World Federation of Neurosurgical Societies (WFNS) grade 1 and 2, 41 (27.3%) from aSAH WFNS grade 3, 36 (18.2%) from grade 4, and 57 (28.8%) from grade 5. From 1999 to 2003, significantly more patients with severe aSAH WFNS grade 4 and 5 underwent (further) treatment (93 out of 198 patients; 47.0%) compared to the former time-period after introduction of early surgery (23 out of 150 patients; 15.3%) ($p < 0.0001$). In the early series, 10 out of 23 patients (43.5%) with WFNS 4 recovered with good outcome Glasgow Outcome Score 4 and 5, whereas in the later series 23 out of 36 (63.9%) with WFNS grade 4 survived in a good functional state. Before 1999, all patients with WFNS grade 5 died or survived in a vegetative state. From 1999 to 2003, 20 out of 57 patients (35.1%) with aSAH WFNS grade 5 survived with good outcome.

Conclusions: The availability of extended specialized neurocritical care seems to induce a change within the patient population towards a higher severity grade. Patients with high-grade aSAH might benefit most from highly specialized neurocritical care treatment.

Key Words: Aneurysmal subarachnoid hemorrhage; severity grade; neurocritical care treatment; brain edema; intracranial pressure; cerebral vasospasm.

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Introduction

Aspects in neurosurgical and neurocritical care treatment changed in the periods before 1993, between 1993 and 1999, and between 1999 and 2003. Before 1993, patients with aneurysmal subarachnoid hemorrhage (aSAH) World Federation of Neurosurgical Societies (WFNS) grade 3 and higher underwent no surgery during the acute stage *(1)*. In 1993, early aneurysm surgery was established in the Department of Neurosurgery, University Hospital Zurich *(2)*. Patients with WFNS grade 5, however, were excluded from neurosurgical treatment. In 1999, a standardized protocol for emergency and specialized neurocritical care treatment was introduced,

whereas the technical aspects in microneurosurgery didn't change. The purpose of this report is to evaluate the impact of specialized neurocritical care treatment on the patient population admitted to a specialized neurovascular center and on the outcome of patients with high-grade aSAH. Therefore, after exclusion of patients treated with endovascular techniques, the results from a patient series with aSAH treated with early aneurysm clipping between 1999 and 2003 (immediately after implementation of a structured neurocritical care treatment) are presented and compared to a former published series of patients, treated between 1993 and 1994 (immediately after implementation of early aneurysm surgery) *(2)*.

Material and Methods

The study was approved as a part of the project E-015/99 by the Ethics Committee of the University of Zurich. Since January 1999, special neurocritical care personnel have been available at the Department of Neurosurgery, University Hospital Zurich. Nursing and medical staff were educated, and guidelines and standards were implemented in generalized and specific aspects of neurocritical care. Protocols for treatment of elevated intracranial pressure (ICP) and detection and treatment of cerebral vasospasm (CVS) were established. For this study, data from patients treated between January 1999 and December 2003 with aneurysm clipping within 3 days after symptom onset were analyzed. 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 *(1)*. Surgical techniques didn't change between 1993 and 2003. The evaluation included only patients with aneurysm clipping within 3 days after aSAH. All patients were treated with nimodipine for 21 days and kept flat in bed as long as cerebral autoregulation, examined with transcranial Doppler (TCD), was defective. Dexamethasone was given perioperatively. Prophylactic antiepileptic treatment with valproate was initiated in patients with aSAH and Hunt & Hess grade 3 and higher *(3–6)*. In patients with aSAH Hunt & Hess grade 1 to 3, sedation was stopped immediately after surgery.

Treatment of Elevated ICP

In patients with poor-grade aSAH, emergency treatment of elevated ICP consisted of sedation with intubation, osmotherapy (Mannitol 20% and hypertonic NaCl-hydroxyethyl-starch solution), mild hyperventilation, and thiopental boli in a dose of 5–10 mg/kg body weight (BW) *(7)*. Patients with occlusive hydrocephalus were treated immediately with a ventricular probe for cerebrospinal fluid (CSF) drainage. If, in patients with most-severe aSAH WFNS grade 5, pupil disturbances normalized because of the previously mentioned treatment, the treatment was continued and craniotomy and aneurysm clipping were performed based on emergency diagnostic evaluation with angiography or computed tomographic (CT) angiography. After early aneurysm surgery, patients with poor-grade aSAH and severe brain edema remained sedated. A ventricular catheter (NMT Neuroscience, Frankfurt, Germany) was inserted to provide continuous ICP monitoring and drainage of CSF, if necessary. If a ventricular catheter could not be placed in the ventricular system because of massive brain edema, a subdural (NMT Neuroscience) or an intraparenchymatous (Codman microsensor, Johnson & Johnson, Raynham) ICP probe was inserted. With elevated ICP (>20 mmHg) treatment with intermittent CSF drainage, osmotherapy, mild hyperventilation (target $PaCO_2$ values adapted to jugular bulb oximetry), and *tris*-hydroxy-methyl-aminomethane (THAM) buffer was initiated *(8)*. Patients with persistant ICP values greater than 20 mmHg were eligible for treatment with barbiturate coma combined with mild hypothermia. Medical therapy, barbiturate coma, and hypothermia treatment were performed according to a standardized algorithm for treatment of elevated ICP *(9)*. Barbiturate coma was induced at the same time as induction of hypothermia and was adapted to a burst suppression pattern in continuous electroencephalogram (EEG) monitoring. Cooling of the patients (target brain temperature 33°C) was accomplished by using cooling blankets (Bair Hugger, Augustine Medical, Saint Prarie, MN, and Blanketrol, CSZ, Cincinnatti, OH) or endovascular cooling catheters (Cool Line Catheter and Coolgard System; Alsius Corporation, Irvine, CA) *(10)*. All patients were treated under extended monitoring of cerebral hemodynamics (monitoring of jugular bulb oxygen saturation, cerebral blood flow [CBF], and/or intraparenchymatous oxygen partial pressure) *(11–13)*. If, after aneurysm surgery, patients developed increasing brain edema and elevated ICP was resistant to the previously mentioned conservative treatment, secondary decompressive hemicraniectomy combined with duraplasty was performed *(14)*.

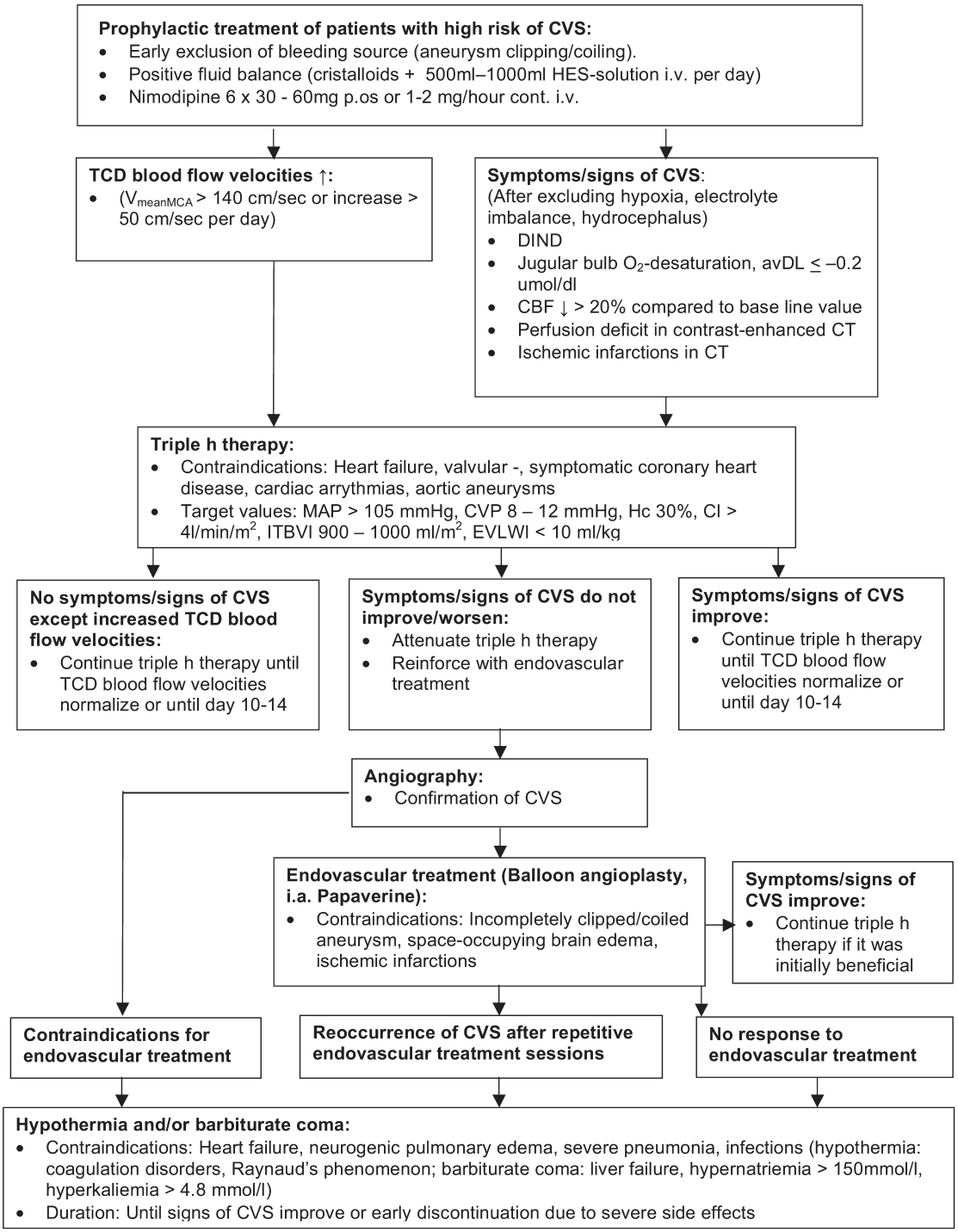
Detection of CVS

TCD blood flow measurements were performed daily. In conscious patients, symptomatic CVS was defined by the occurrence of delayed ischemic neurological deficit (DIND) (decrease in consciousness, new focal neurological deficits). After hypoxia, electrolyte imbalance and hydrocephalus were excluded on the basis of a further CT scan, and symptomatic CVS was suspected. CVS in all patients was confirmed with digital subtraction angiography. The role of TCD in predicting symptomatic CVS is limited *(15)*. Therefore, if the neurological state could not be assessed properly (e.g., because of sedation or poor neurological grade) patients were additionally monitored with jugular bulb oxymetry, lactate measurements from the jugular bulb *(16)*, daily CBF measurements (near-infrared spectroscopy indocyanine green dye dilution method) *(13)*, and/or perfusion CT examinations *(17)*. If jugular bulb O_2 desaturation occurred, arteriovenous difference of lactate (avDL) was –0.2 µmol/dL or less, CBF values decreased more than 20%, and/or differences in territorial or hemispheric transit times occurred in perfusion CT, symptomatic CVS was suspected and digital subtraction angiography was performed. Likewise, all patients with new ischemic infarctions, compared to the postoperative CT examination, were classified as suffering from CVS.

Treatment of CVS

Benefits of hypertensive hypervolemic hemodilution (Triple H) therapy have never been unequivocally demonstrated by randomized controlled studies *(6,18)*. In early studies,

Table 1 CVS Treatment Protocol



CVS, cerebral vasospasm; DIND, delayed ischemic neurological deficit; CBF, cerebral blood flow; EVLWI, extravascular lung water index; TCD, transcranial Doppler.

patients with volume expansion showed less frequent DINDs and better outcome compared to control patients who were kept dehydrated *(19)*, whereas in more recent studies, no effect of hypervolemia on either CBF or DINDs was found if control patients were already receiving more than 3000 mL fluids per day *(20)*. Therefore, our treatment protocol didn't foresee a prophylactic Triple H therapy for our patients. Before the patients developed signs of CVS, the daily fluid balance was aimed to be positive, adjusting the fluid intake by intravenous infusion of crystalloids and hydroxyethyl-starch solution (HES; 1000 mL/day). If signs of CVS occurred, patients were treated according to a multimodal structured treatment protocol based on the results of clinical studies and personal experience (Table 1) (*6,9,11,18,21–29*).

Triple H Therapy

Triple H therapy was induced if TCD mean blood flow velocities increased (mean middle cerebral artery blood flow velocities > 140 cm/second or increase >50 cm/second within 24 hours). If significant differences were present in perfusion CT, jugular bulb O_2 desaturations occurred in case of increased avDL, decreased CBF, and/or if the patient developed symptomatic CVS. Contraindications to initiate Triple H therapy were heart failure, valvular heart disease, symptomatic coronary heart disease, cardiac arrhythmias, and aortic aneurysms. Triple H therapy was executed using a new system to monitor systemic hemodynamics (PICCO Pulsion Medical Systems, Munich) *(30)*. Thermodilution measurements with calibration of cardiac index (CI) and determination of global enddiastolic volume index, intrathoracic blood volume index (ITBVI) and extravascular lung water index (EVLWI) were performed every 6 hours. In addition to the conventional parameters (mean arterial pressure [MAP] > 105 mmHg, central venous pressure (CVP) 8–12 mmHg, and hematocrit 28–32%), Triple H therapy was adapted to the following target values: CI greater than 4 L/(minute • m^2), ITBVI 900–1000 mL/m^2, and EVLWI less than 10 mL/kg. Triple H therapy was induced by administration of crystalloid and colloid infusions. Dobutamine and norepinephrine were adjusted to maximize cardiac function. Excessive natriuresis and diuresis (osmolarity in urine > osmolarity in serum, sodium in serum < 140 mmol/L) in combination with a negative fluid balance and signs of intravascular volume depletion were inhibited with fludrocortisone 0.2 mg/day *(31,32)*. Excessive water diuresis (osmolarity in urine < osmolarity in serum, sodium in serum > 140 mmol/L) was treated with desmopressine 1–4 × 2 µg i.v. per day.

Endovascular Treatment

If patients with DIND did not improve or worsened despite Triple H therapy, digital subtraction angiography and endovascular treatment with percutaneous balloon angioplasty and/or superselective papaverine infusion (total dose of 300 mg) into the vasospastic vessels were performed *(22,33)*. Contraindications for endovascular treatment were the presence of incompletely clipped aneurysms, ischemic infarctions, or a space-occupying brain edema in CT.

Barbiturate Coma, Hypothermia

Symptomatic CVS (resistant to the previously mentioned treatment or reoccurring after two to three spasmolysis sessions) was treated with barbiturate coma and/or hypothermia (target brain temperature 33°C). Barbiturate coma with thiopental (loading dose of 5–10 mg/kg BW, followed by continuous infusion) was induced at the same time as induction of hypothermia and was adapted to a burst suppression pattern in continuous EEG monitoring *(7)*. Patients were excluded if they initially suffered from congestive heart failure, neurogenic pulmonary edema, severe aspiration pneumonia, other infections, or Raynaud's phenomenon. If specific contraindications for thiopental were present, such as liver failure, hyperkaliemia (serum potassium > 4.8 mmol/L), or hypernatriemia (serum sodium > 150 mmol/L), only hypothermia was performed. Barbiturate coma and hypothermia were continued until signs of CVS decreased. Barbiturate coma and hypothermia were terminated earlier if signs of severe infection, cardiovascular instability (arrhythmias, signs of myocardial ischemia, or in case of norepinephrine, dose > 50 µg/minute), liver failure (barbiturate coma), severe electrolyte disturbances (barbiturate coma), or coagulation disorders (hypothermia) were observed.

Outcome Measurements

Neurological outcome was assessed after 3 and 12 months in the outpatient clinic by a neurologist using the Glasgow Outcome Score (GOS) *(34)*. Those patients who did not show up for control were contacted and asked about their functional status.

Statistical Analysis

Statistical calculations were carried out using the Mann-Whitney test (patient characteristics) and two-tailed Fisher's exact test (severity grade and outcome of patients).

Results

Between January 1999 and December 2003, 210 patients with aSAH were treated within 3 days. A group of 12 patients were treated with aneurysm coiling and therefore excluded from the analysis. The remaining 198 patients treated with microsurgical techniques were analyzed. Patient characteristics and the distribution of aneurysms are given in Table 2. There were no statistical significant differences between the two

Table 2
Patient Characteristics and Localization of Aneurysms

	Series 1993–1994 (n = 150) (2)	*Series 1999–2003 (n = 198)*
Age; mean (SD)	49.5 (12.5)	52.2 (13.3)
Gender		
Male	46 (30.7%)	54 (27.3%)
Female	104 (69.3%)	144 (72.7%)
Timing of surgery		
Day 0	103 (68.7%)	45 (22.7%)
Day 1		107 (54.0%)
Day 2		31 (15.7%)
Day 3		15 (7.6%)
Localization of ruptured aneurysms		
ICA (-OphA, -PcomA, -AchorA, -bifurcation)	44	52
AcomA	60	72
Distal ACA	2	11
MCA	35	43
BA (head, -SCA, trunk)	6	12
VA-PICA	3	7
C1	0	1

n, number; SD, standard deviation; GCS, Glasgow Coma Scale; ICA, internal carotid artery; OphA, ophthalmic artery; PcomA, posterior communicating artery; AchorA, anterior choreoidal artery; AcomA, anterior communicating artery; ACA, anterior cerebral artery; MCA, middle cerebral artery; BA, basilar artery; SCA, superior cerebellar artery; VA, vertebral artery; PICA, posterior inferior cerebellar artery; C1, cervical trunk artery.

Table 3
Severity of aSAH

	Series 1999–2003 (n = 198)
Hunt & Hess Grading	
H & H Grade 1	14 (7.1%)
H & H Grade 2	65 (32.8%)
H & H Grade 3	43 (21.7%)
H & H Grade 4	47 (23.7%)
H & H Grade 5	29 (14.7%)
WFNS Grading	
WFNS Grade 1	50 (25.2%)
WFNS Grade 2	40 (20.2%)
WFNS Grade 3	15 (7.6%)
WFNS Grade 4	36 (18.2%)
WFNS Grade 5	57 (28.8%)
Fisher Grading	
Fisher Grade 1	4 (2.0%)
Fisher Grade 2	32 (16.2%)
Fisher Grade 3	72 (36.4%)
Fisher Grade 4	90 (45.4%)

aSAH, aneurysmal subarachnoid hemorrhage; *n*, number; H & H, Hunt and Hess; WFNS, World Federation of Neurological Surgeons.

patient series according to age, sex, and localization of ruptured aneurysms. Table 3 shows the severity of aSAH. According to the Hunt & Hess grading scale, 76 patients (38.4%) suffered from severe grades 4 and 5; whereas according to the WFNS grading scale, 93 (47%) patients suffered from severe grades 4 and 5; and the Fisher grading scale defined 162 (81.8%) as suffering from grades 3 and 4. Figure 1 gives the severity of aSAH of the patient series from 1999 to 2003, compared to the former series (1993 to 1994) *(2)*. The percentage of severe aSAH WFNS grade 4 increased from 15.3 to 18.2% and the percentage of patients with WFNS grade 5 undergoing aneurysm clipping and special neurocritical care increased from 0 to 28.8%. During the period from 1999 to 2003, significantly more patients with most severe aSAH WFNS grade 4 and 5 underwent treatment (93 from 198 patients; 47.0%) compared to 23 out of 150 patients; 15.3% ($p < 0.0001$) during the earlier period (1993–1994). Table 4 gives the characteristics of treatment. With the high proportion of patients with poor-grade aSAH, the incidence of severe brain edema with elevated ICP despite osmotherapy, THAM, and mild hyperventilation reached a relatively high 15.7%. All of these patients were treated with hypothermia combined with barbiturate coma or (because of contraindications to barbiturates) with hypothermia only. Because of refractory intracranial hypertension, 16 patients (8.1%) were treated with secondary decompressive surgery.

CVS occurred in 39.9% of the cases. Out of 79 patients with symptomatic CVS, 78 were treated with Triple H therapy and 33 with balloon angioplasty and/or intraarterial papaverine instillation. A group of 23 patients with CVS refractory to Triple H therapy and spasmolysis were treated with hypothermia combined with barbiturate coma or (because of contraindications to barbiturates) with hypothermia only. In the patient series of 1993 to 1994, two patients with CVS refractory to Triple H therapy and spasmolysis were treated with barbiturate coma *(2)*. No hypothermia treatment was performed during the earlier period.

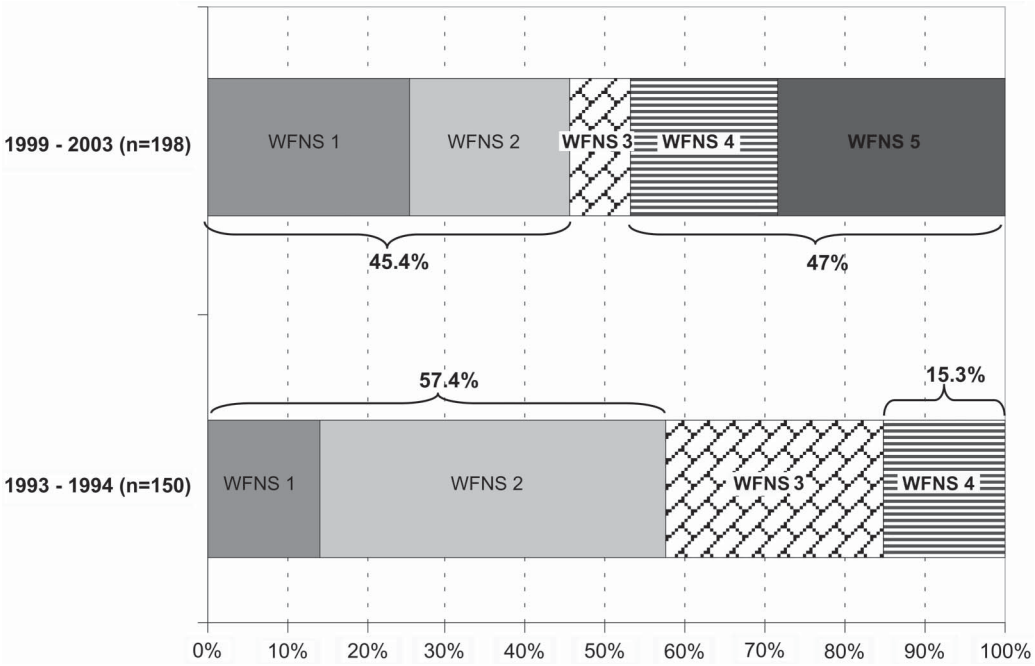


Fig. 1. Severity of aneurysmal subarachnoid hermorrhage. Comparison between patient series 1993–1994 (after introduction of early surgery) *(2)* and 1999–2003 (after introduction of structured neruointensive care). In the time period 1993–1994 *(2)* patients with WFNS grade 5 aSAH were not treated with aneurysm surgery. The percentage of patients with poor grade aSAH WFNS 4 and 5 increased from 15.3% (in the earlier series) to 47% (in the later series), whereas the percentage of patients with WFNS 1 and 2 aSAH decreased from 57.4 to 45.4%.

In 36.9% of the cases, shunt dependency from malresorptive hydrocephalus occurred. All but four out of 73 patients treated with ventriculoperitoneal shunt suffered from Fisher grades 3 and 4. Their mean age (59.7 ± 10.1 years) tended to be higher compared to the whole population.

The results of the patient series 1999 to 2003 are given in Table 5. Compared to the earlier series, the overall outcome didn't change. Between 1999 and 2003, 131 patients (66.1%) survived with good functional outcome GOS 4 and 5, respectively, after 3 months and 142 (71.7%) after 12 months. Whereas between 1993 and 1994, 112 out of 150 (75%) survived with GOS 4 and 5 after 3 months *(2)*. Table 6 shows the outcome of the patient subgroup with poor-grade aSAH WFNS 4 and 5 in the series of 1999 to 2003. The analysis of the patient series from 1993 to 1994 does not include any patients with WFNS grade 5. Special treatment not being available before 1999, patients with WFNS grade 5 died or survived in a vegetative state. Therefore, only the outcome of patients with severe aSAH WFNS grade 4 can be compared between the two series. In the early series of 1993 to 1994, 10 out of 23 patients (43.5%) with WFNS 4 aSAH recovered with good functional outcome *(2)*, whereas in the series between 1999 and 2003, 23 out of 36 patients (63.9%) with WFNS grade 4 survived in a good functional state after 3 months (without statistical significance). In the later series (from 1999 to 2003), 57 patients (28.8%) suffered from most-severe aSAH WFNS grade 5. Of these, 20 (35.1%) survived with good functional outcome after 12 months (21.1% with GOS 5 and 14% with GOS 4).

Table 4
Characteristics of Treatment

	Series 1999–2003 (n = 198)
Incidence of ICP > 15 mmHg despite osmotherapy, THAM, and mild hyperventilation	31 (15.7%)
Treated with hypothermia combined with barbiturate coma	29 (14.6%)
Treated with hypothermia only	2 (1.0%)
Treated with secondary decompressive surgery	16 (8.1%)
Incidence of symptomatic CVS	79 (39.9%)
Treated with Triple H therapy	78 (39.4%)
Treated with spasmolysis	33 (16.7%)
Once	26 (13.1%)
Twice	5 (2.5%)
Three times	2 (1.0%)
Treated with hypothermia combined with barbiturate coma	23 (11.6%)
Treated with hypothermia only	3 (1.5%)
Incidence of malresorptive hydrocephalus treated with VP shunt	73 (36.9%)

n, number; ICP, intracranial pressure; THAM, trishydroxyaminomethan buffer; CVS, cerebral vasospsm; Triple H, hypertensive hypervolemic hemodilution; VP, ventriculoperitoneal.

Table 5
Outcome of Patients

	Series 1999–2003 (n = 198)	
Glasgow Outcome Scale	3 months after aSAH	12 months after aSAH
GOS 5	88 (44.4%)	115 (58.1%)
GOS 4	43 (21.7%)	27 (13.6%)
GOS 3	36 (18.2%)	28 (14.2%)
GOS 2	13 (6.6%)	1 (0.5%)
GOS 1	18 (9.1%)	27 (13.6%)
Died of CVS		7 (3.5%)
Permanent additional deficits caused by CVS		17 (8.6%)

n, number; aSAH, aneurysmal subarachnoid hemorrhage; GOS, Glasgow Outcome Scale; CVS, cerebral vasospasm.

Discussion

In recent years, increased experience in neurosurgical techniques and the possibility of endovascular coiling made it possible to address patients with most severe aSAH and to exclude aneurysms in the acute stage of "angry brain" *(2,35,36)*. These patients, however, suffer from a high incidence of specific complications such as severe brain edema with refractory ICP elevations and symptomatic CVS. In the later series, 47% of the patients suffered from severe aSAH WFNS 4 and 5. Because of the high percentage of patients with Fisher grade 3 and 4 bleedings, the incidence of symptomatic CVS reached a high 40%. With 36.9% compared to 9%, the number of cases of shunt dependency from malresorptive hydrocephalus was higher in the later series than in the early series *(2)*. However, surgical techniques (extensively opening of the lamina terminals and membrane of Liliequist, avoiding the use of continuous drainage if possible) did not change in the meantime. The higher shunt dependency in the present series might be explained by the higher percentage of patients with severe aSAH and the more frequently used ventricular catheters in order to monitor ICP and to perform and control CSF drainage *(37)*.

Therefore, to obtain good results (especially in patients with high-grade aSAH), not only the prevention of rebleeding by performing early surgery as well as avoiding technical operative complications, but also standardized protocols for specialized neurocritical care are of great importance. The main

Table 6
Outcome of Patients Suffering From Poor-Grade aSAH WFNS 4 and 5

	Series 1999–2003 (n = 92)	
Glasgow Outcome Scale	3 months after aSAH	12 months after aSAH
GOS 5	24 (26.1%)	36 (39.1%)
GOS 4	20 (21.7%)	14 (15.2%)
GOS 3	22 (23.9%)	21 (22.8%)
GOS 2	12 (13.1%)	1 (1.1%)
GOS 1	14 (15.2%)	20 (21.8%)

aSAH, aneurysmal subarachnoid hemorrhage; WFNS, World Federation of Neurological Surgeons; n, number; GOS, Glasgow Outcome Scale.

features of a structured neurocritical care treatment approach are: (1) emergency therapy of the acute ICP crisis and cardiovascular stabilization before surgery (neurorescucitation); (2) postoperative treatment of brain edema and elevated ICP; and (3) prophylaxis, detection, and treatment of possible CVS and 4, and treatment of medical complications.

Comparisons with historical patient series, not adjusting for patient and treatment characteristics, have major limitations. This report, however, is focused on the evaluation of the impact of specialized neurocritical care treatment. It is beyond the scope of the study to examine the influence of aneurysm coiling on the outcome of the patients. Therefore, patients treated with coiling were excluded from the analysis. Principles of microsurgical techniques didn't change between 1993 and 2003. During both periods, more than 90% of the operations were performed by the second author (Y.Y.) applying the standard microsurgical technique described by Yasargil *(1)*. Therefore, the study shows clearly some trends and may serve as a guide for future planning with regards to resources in neurocritical care.

Over the years, a change in the patient population towards a higher severity grade occurred in our specialized neurovascular center. On the one hand, the shift within the admitted patient population may be caused by changes in national health policy aspects. On the other hand, it may be owing to the fact that the availability of extended resources for neurorescucitation, early aneurysm surgery, and specialized neurocritical care becomes common knowledge among referring institutions and may lead to different admission policies.

In the period between 1993 and 1994, patients with WFNS grade 5 didn't undergo special treatment *(2)*. Therefore, in the early years, patients with WFNS grade 5 died or survived in a vegetative state. In 1999, the previously listed standards for emergency neurorescucitation and postoperative neurocritical care were established in our neurocritical care unit. If, in patients with aSAH WFNS grade 5, pupil disturbances normalized after emergency treatment, they consequently underwent early aneurysm clipping and, after surgery, were treated against severe brain edema and CVS in the neurocritical care unit. In recent years, in more than one-third of these patients with aSAH WFNS grade 5 a good functional outcome could be obtained, and in the group with most severe aSAH WFNS grade 4 and 5, even more than 50% of the cases showed a good functional outcome. Furthermore, the proportion of patients with aSAH WFNS grade 4 surviving in a good functional state after 3 months tended to increase from 43.5% in the earlier series to 63.9% in the later series. This might suggest that patients with most-severe aSAH, suffering from massive brain edema and prolonged ICP elevations, as well as those with intractable CVS may benefit most from highly specialized neurocritical treatment. Indeed, recent studies demonstrated that patients with severe head injury, ischemic stroke, and intracerebral hemorrhage benefit from specialized neurocritical care and stroke units *(38–40)*. Specialized knowledge of elaborate diagnostic procedures is particularly required in patients with aSAH. Complex treatment strategies applied in these patients (e.g., long-term hypothermia) call for experienced medical and nursing staff, especially trained in neurocritical care topics. Not only do the expensive technical equipment for specialized neuromonitoring, neuroimaging, and extended monitoring of cerebral and systemic hemodynamics have to be available, but most of all optimal interdisciplinary collaboration between neurosurgeons, neuoradiologists, and neurointensivists is required *(41)*. In many neurocenters, specialized neurocritical care units serve as the focal point for these combined efforts *(42,43)*.

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