Neurosurg. Rev.
16 (1993) 15–25

Outcome of patients with aneurysmal and presumed aneurysmal bleeding. A hospital study based on 100 consecutive cases in a neurological clinic

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Abstract

One hundred patients with spontaneous subarachnoid hemorrhage due to aneurysm or presumed aneurysm consecutively admitted to a neurological clinic and subjected to CCT during the first 72 hours were examined retrospectively. The outcome after two months as defined by the Glasgow Outcome Scale (GOS) was relatively good: 23% of the patients suffered management mortality (GOS I) (postoperative lethality 8%), 3% showed GOS-Grade II, 14% grade III, 17% grade IV, and 43% grade V. The extent of intracranial hemorrhage correlated well with the initial Hunt-Hess Grade which, in turn, had a strong influence on case fatality and the degree of disability. Lethal factors were: 1. massive subarachnoid hemorrhage together with a massive ventricular hemorrhage ($p < 0.001$), 2. massive subarachnoid hemorrhage together with an intracerebral hematoma > 20 ml ($p < 0.05$). Case fatality was lower when angiography was negative. In our study rebleeding (12%) and delayed cerebral ischemia (DCI) (18%) were less frequent and the lethality due to acute hydrocephalus (5%) and delayed cerebral ischemia (5%) was less pronounced than in comparable studies. The degree of disability (GOS) was directly related to the amount of intracranial blood, to the development of acute or chronic hydrocephalus, delayed cerebral ischemia and rebleeding. DCI occurred in 60% of patients with marked hydrocephalus. Rebleeding was more frequent in patients with acute hydrocephalus. Hydrocephalus, DCI, and rebleeding were associated with a poorer initial grade on the Hunt and Hess Scale.

Keywords: Complications, outcome, subarachnoid hemorrhage.

1 Introduction

The reported outcome of aneurysmal and presumed aneurysmal subarachnoid hemorrhage (SAH) has varied widely: in a number of studies the case fatality ranged from 52% to 65% and correspondingly a large number of patients were reported to be seriously disabled [24, 35]. Furthermore a great number of cases of undetected lethal subarachnoid hemorrhage has often been suspected [36]. In contrast, management-mortality in the Cooperative Study, in which, however, some criteria for exclusion were applied, amounted to only 27% [28]. In a Swedish study management mortality was 25%, plus 12% pronounced dead on arrival at a hospital or a forensic institution [44]. Based on those patients who were initially admitted to our Department of Neurology we investigated whether the prognosis of aneurysmal or presumed aneurysmal subarachnoid hemorrhage has improved in recent years [26] and which factors influence prognosis.

2 Subjects and methods

The study includes all 100 cases of spontaneous subarachnoid hemorrhage that were admitted to the Department of Neurology of the University of Giessen between 1983 and mid 1988. Prerequisites for inclusion in the study were a typical history, a neurological examination, and a CT scan performed within the first 72

hours after the initial clinical event. If CT scan was negative lumbar puncture was performed. Patients were included regardless of whether they were considered for operation or not. Early operation has been performed since 1986, the clinical state permitting. All patients were treated with calcium antagonists. Frequent CCT follow-ups were performed in all patients; transcranial Doppler follow-ups, postoperative hypervolemic hemodilution and controlled hypertension since 1986.

Two categories of subarachnoid hemorrhage were established: 1. mild subarachnoid hemorrhage: no blood visible in CT scan, a limited amount of blood localized at the convexity, the insular cistern or interhemispheric fissure, circumscript blood in the cisterna praepontis and/or the cisterna interpeduncularis or a limited bleeding in one of the other basal cisterns. 2. massive subarachnoid hemorrhage (MSAH): extensive bleeding around the convexity, the insular cisterns, the interhemispheric fissure or in the basal cisterns as well as massive supra- and infratentorial subarachnoid hemorrhage.

The extent of intraventricular bleeding was also subdivided into two categories: 1. no or little ventricular hemorrhage; limited bleeding in localized areas of the supratentorial ventricles. 2. massive intraventricular hemorrhage (MIVH); larger collections of blood in the supratentorial ventricles or in the fourth ventricle; hemorrhage in all four ventricles.

Intracerebral hematomas (ICH) were subdivided into two categories: 1. 20 ml and less. 2. more than 20 ml.

Acute hydrocephalus was diagnosed when ventricular enlargement took place within the first week, whereas chronic hydrocephalus was diagnosed when ventricular enlargement was present after the first week. In either case the original neuroradiological reports were used. Rebleeding was diagnosed when a CT scan or lumbar puncture performed subsequent to clinical deterioration revealed the presence of renewed bleeding. Delayed cerebral ischemia (DCI) was diagnosed when a hypodense area was observed on a CT scan or when new localizing cerebral symptoms, combined with an EEG focus, could not be explained by other complications. As transcranial Doppler sonography (TCD) was only performed after 1986, vasospasms diagnosed only by means of TCD were not evaluated. Therapy, however, had been influenced by TCD since 1986. Vasospasms detected on angiography were evaluated. In order to permit early operation, angiography was performed during the first three days, the clinical state permitting.

On admission, grading was carried out according to the Hunt and Hess Scale and the final report was coded according to the GOS [27]. The mean period of observation was approximately two months. The Chi-Square-Test and, if possible, the exact test of Fisher were used for statistical evaluation.

3 Results

The median of age was 54 years, with a range of 20 to 82 years. Fourty-nine patients were treated surgically and 51 conservatively. Angiography was performed in 90 patients. In 9 cases the patients' condition precluded angiography, one patient refused angiography. Angiography was positive in 68 cases (anterior communicating artery 30, middle cerebral artery 22, multiple 7, vertebrobasilar 6, internal carotid artery 3) and negative in 22 cases. In 8 cases with a negative angiogram there was blood only in the cisterna praepontis or the cisterna interpeduncularis. In 13 cases no blood was detectable in the CT scan so that the subarachnoid hemorrhage could only be diagnosed by means of lumbar puncture. The angiogram was positive in 8 and negative in 5 of these patients (Figure 1).

Twenty-three patients died (GOS I). Of these 4 died postoperatively (cerebral infarction (2), cerebral edema (1), pulmonary embolism (1)) and 8 died within the first 48 hours following the subarachnoid hemorrhage. Nineteen patients died without having reached a condition suitable for surgery.

Seventeen patients remained severely disabled (4 GOS II, 13 GOS III). Eight of these had been treated surgically and 9 conservatively. On discharge 60 patients (37 operated, 23

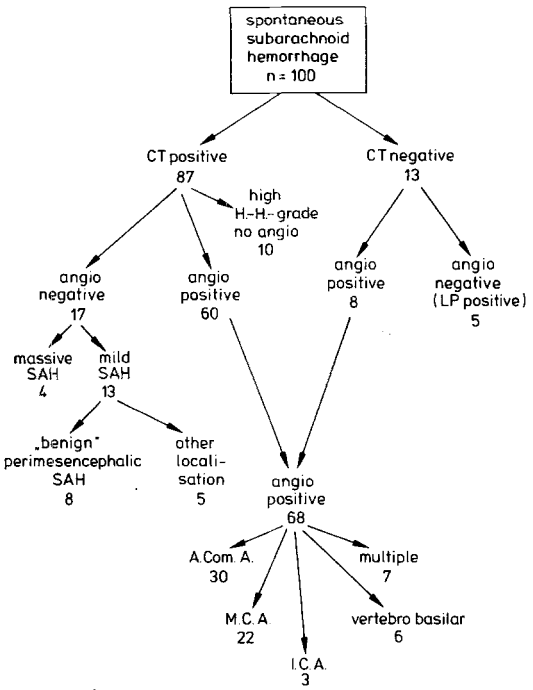

Figure 1. Flow chart of CT and angiographic findings in 100 patients with spontaneous SAH. Abbreviations: AComA anterior communicating artery, MCA middle cerebral artery, ICA internal carotid artery.

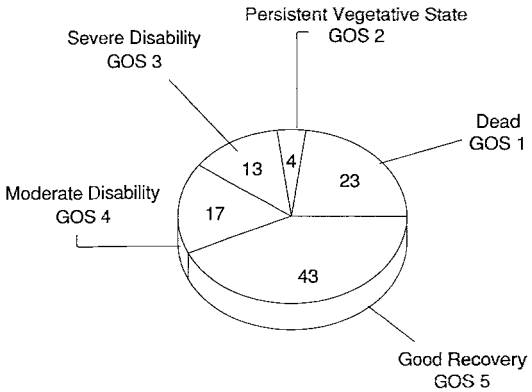

Figure 2. Glasgow Outcome Scale of 100 patients with spontaneous SAH on discharge. Mean hospital stay of surviving patients 2 months (SD 0.90).

Table I. Hunt-Hess Grading/Glasgow Outcome-scale

H.-H.	I, II n = 45	III n = 32	IV n = 13	V n = 10
GOS 1	4 (9%)	6 (19%)	4 (31%)	9 (90%)
2, 3	1 (2%)	10 (31%)	5 (38%)	1 (10%)
4, 5	40 (89%)	16 (50%)	4 (3%)	0

$p < 0.001$

treated conservatively) showed no or only slight disability (17 GOS IV, 43 GOS V) (Figure 2). Mean hospital stay of the surviving patients was 2 months (SD 0.9). Case fatality among patients with negative angiogram was lower than fatality of patients with proven aneurysm ($p < 0.005$). The degree of disability among patients with negative and positive angiogram did not vary. The localization of the aneurysms had no influence on case fatality. Case fatality and the degree of disability (GOS-System) correlated closely with the result of the initial neurological examination (HH) ($p < 0.001$; Table I).

The following factors taken separately, i.e., ignoring the interference of the various radiological parameters, were related to increased case fatality ratio: 1. massive subarachnoid hemorrhage ($p < 0.005$). 2. massive intraventricular hemorrhage ($p < 0.005$) 3. intracerebral hematoma of more than 20 ml ($p < 0.001$). In the presence of MSAH case fatality ratio rose from 3% to 17% ($p < 0.05$). MIVH and ICH > 20 ml, which only rarely occurred alone, killed, in combination, 2 of 3 patients. The combination of MSAH and ICH > 20 ml led to a case fatality ratio of 57% ($p < 0.05$) and MSAH combined with a MIVH showed a case fatality ratio of 55% ($p < 0.001$). The prognosis of the combination of MSAH and blood in the 4th ventricle was particularly bad (case fatality 55%; GOS IV + V 20%). Thus the combination of 2 or 3 of the lethal factors was basically responsible for the difference in case fatality ratio due to MSAH (17%) and the management case fatality (23%) of the study population (Table II).

Table II. Relationship of the findings in the initial CT and case fatality ratio and morbidity

MSAH n = 53	MVH n = 27	ICH > 20 ccm n = 12	GOS 1 n = 23	GOS 2, 3 n = 17	GOS 4, 5 n = 60	Σ n = 100
−	−	−	1 (3%)	2	34***	37
+	−	−	5 (17%)**	8	16	29
−	+	−	0	00	2	2
−	−	+	0	7***	2	2
+	+	−	11 (55%)***	0	2	20
+	−	+	4 (57%)**	0	3	7
−	+	+	2 (67%)		1	3

** $p < 0.05$, *** $p < 0.001$

The following factors had no influence on case fatality ratio: acute hydrocephalus, delayed cerebral ischemia, former subarachnoid hemorrhage, hypertension, rebleeding during treatment in the hospital, age > 65 years.

The following complications were associated with a higher degree of disability: 1. acute ($p < 0.001$) and chronic ($p < 0.001$) hydrocephalus 2. DCI ($p < 0.001$) 3. rebleeding during hospitalization ($p < 0.05$) (Table III).

A higher occurrence of the following symptoms was observed in patients with an initial Hunt and Hess grade between III and V: 1. massive subarachnoid hemorrhage ($p < 0.05$), 2. massive intraventricular hemorrhage ($p < 0.05$), 3. intracerebral hematoma ($p < 0.001$). In addition these patients suffered the following complications more frequently: 1. delayed cerebral ischemia ($p < 0.05$), 2. acute hydrocephalus ($p < 0.001$), 3. rebleeding ($p < 0.05$) (Table IV).

During hospitalization 18 patients suffered delayed cerebral ischemia; 17 of these had vasospasms on angiography. DCI developed in patients with 1. massive subarachnoid hemorrhage ($p < 0.001$), 2. chronic hydrocephalus ($p < 0.001$).

In 20 patients acute hydrocephalus developed. In 12 of these patients the ventricular enlargement was only moderate. In the remaining 8 cases of significant ventricular enlargement a ventricular shunt was implanted. Two of these 8 patients died after rebleeding. Three of them suffered from delayed cerebral infarction. Acute hydrocephalus developed more often in patients with: 1. massive intraventricular hemorrhage ($p < 0.001$), 2. massive subarachnoid hemorrhage ($p < 0.001$). Chronic hydrocephalus was diagnosed in 11 cases and was judged to be significant in 5 patients. These patients suffered more frequently from: 1. massive subarachnoid hemorrhage ($p < 0.05$), 2. massive intraventricular hemorrhage ($p < 0.05$), 3. acute hydrocephalus ($p < 0.001$), 4. DCI ($p < 0.001$) (Table IV). A ventricular shunt was implanted in all 13 cases of significant ventricular enlargement whether acute or chronic. Eight of these 13 patients suffered from DCI prior to or after shunting. Statistically patients

Table III. Relationship of the three major complications to the degree of disability

		GOS 2, 3 n = 17	GOS 4, 5 n = 60	
Hydrocephalus	− acute	9 (53%)	6 (10%)	$p < 0.001$
	− chronic	9 (53%)	1 (2%)	$p < 0.001$
DCI		9 (53%)	4 (7%)	$p < 0.001$
rebleeding		5 (29%)	5 (8%)	$p < 0.05$

Table IV. Correlations of factors and symptoms

Factor/ symptoms		n	Delayed cerebral ischemia n = 18	Acute hydroc. n = 20	Chronic hydroc. n = 11*	Rebleeding n = 12	HH III–V n = 55
ICH		23	5/23 22%	5/23 22%	2/23 75 9%	3/23 13%	22/23 96%
no ICH		77	17/77 17%	15/77 19%	9/75 12%	9/77 12%	33/77 43%
							$p < 0.001$
Massive IVH		27	5/27 19%	12/27 44%	6/27 22%	4/27 15%	23/27 85%
no or moderate IVH		73	13/73 18%	8/73 11%	5/71 7%	8/73 11%	32/73 44%
				$p < 0.001$	$p < 0.05$		$p < 0.05$
Massive SAH		53	17/53 31%	18/53 33%	9/51 18%	9/53 16%	43/53 81%
no or moderate SAH		47	1/47 2%	2/47 4%	2/47 4%	3/47 6%	12/47 26%
			$p < 0.001$	$p < 0.001$	$p < 0.05$		$p < 0.05$
DCI	yes	18		5/18 28%	7/18 39%	2/18 11%	14/18 78%
	no	82		15/82 18%	4/80 5%	10/82 12%	41/82 50%
					$p < 0.001$		$p < 0.05$
Acute	yes	20			8/18 44%	6/20 30%	19/20 95%
hydroc.	no	80			3/80 4%	6/80 7%	36/80 52%
					$p < 0.001$	$p < 0.001$	$p < 0.001$
Chronic	yes	11				4/11 36%	9/11 82%
hydroc.	no*	87				8/87 9%	46/87 53%
						$p < 0.01$	
Rebleeding	yes	12					10/12 83%
	no	88					45/88 51%
							$p < 0.05$

* in two cases no follow-up CT available

with a shunted hydrocephalus suffered from DCI more frequently than patients without shunted hydrocephalus ($p < 0.005$) or patients without hydrocephalus ($p < 0.001$).

Rebleeding occurred in 12 cases and was more frequent in patients with acute ($p < 0.001$) and chronic ($p < 0.01$) hydrocephalus (Table IV). Only 4 patients suffered rebleeding ($p = 0.5$) after 13 ventricular shunt implantations.

4 Discussion

We wanted our study to include the whole spectrum of spontaneous subarachnoid hemorrhage. Both patients with proven aneurysm and those in whom an angiography could not be performed because of bad clinical state and those with a negative angiogram were included. Among the patients with a negative angiogram 4 had a massive SAH whereas 8 had a so-called benign perimesencephalic SAH, which by some authors is not considered to be of aneurysmal origin [15, 52]. We also included patients with a characteristic history of sudden onset headache with negative CT scan and positive lumbar puncture. An aneurysm was proven in 8 of these, the remaining 5 may also have had perimesencephalic SAH. An artifically blood-stained CSF due to a traumatic lumbar puncture in patients with thunderclap headache [54] may have been a pitfall in some of these cases. The proportion of SAH's with a negative angiogram, well known to have a better prognosis [13], was 20%, which compares to other studies [40].

The reported outcomes of SAH with presumed or proven aneurysm has varied widely. This is practically due to varying inclusion criteria used in the various studies (Table V). In the International Cooperative Study on Timing of Aneurysma Surgery [28], which covered 3500 patients, angiographically proven aneurysm was a prerequisite and only patients with a first bleed were considered. Exclusion criteria were large intracerebral hematomas, serious internal complications, and treatment with anticoagulants. In some studies an aneurysm had to be proven by CT scan or by angiogram [35]. The Dutch study of HIJDRA et al. [24] included, besides patients with angiographically proven aneurysms, those patients with a characteristic history and a typical CT in whom an angiography could not be performed. "Benign" perimesencephalic SAH, however, was excluded. In a Swedish population study carried out in forensic and pathoanatomic institutes SAH was diagnosed in 15% of patients who had not reached a clinic alive after an ictus [44]. The study of the Mayo Clinic [26] included presumed aneurysms in patients with a positive lumbar tap and a SAH on CT scan. A major argument to include patients without angiographically proven aneurysms is supplied by SUZUKI et al. [46] who reduced their number of negative angiograms by 20% by repeating angiograms over a period of 44 months. In many cases of so-called SAH without proven aneurysm it could thus be shown that initially there is a thrombosed aneurysm.

Compared with many previous studies [3, 8, 10, 24, 35, 39, 42] our patients had a better outcome. The better outcome in our study, which covered a large spectrum of SAH patients, appears to be due to the fact that our patients (1983–1988) were treated with recently developed strategies including calcium antagonists and early operation whereas the patients in older studies (Table V) were mostly

Table V. Outcome and inclusion criteria of different studies

Author	Case fetality	"Good outcome"	Inclusion criteria
MOHR and KASE 1978–1980 [35] n = 60	65%	13%	Aneurysm proven by angiography or CT.
HIJDRA et al. 1977–1983 [24] n = 264	52%	26%	Proven aneurysm. If angiography not feasible only CT. Exclusion of "benign perimesencephalic SAH".
KASSEL et al. Cooperative study 1981–1983 [28] n > 3500	27%	56%	Proven aneurysm. Only first SAH. Day 1 to 3. No large ICH. No anticoagulants. No major internal complications.
SÄVELAND et al. 1983 [44] n = 78	37% (15% died before reaching the hospital)	41%	Population study. Proven aneurysm.
INGALL et al. Mayo Klinik 1975–1984 [26] n = 49	42%	36%	Population study. Proven or presumed aneurysm (lumbar puncture, CT-scan).
SCHÜTZ et al. 1983–1988 n = 100	23%	43%	Hospital based study. Proven or presumed aneurysm (lumbar puncture, CT-scan). Day 1 to 3. Perimessencephalic SAH included.

treated with antifibinolytics and fluid restriction. Postoperative lethality was only half of that observed in the study of HIJDRA et al. (1977–1983) [24]. Although the distribution of the patients' grades on the initial Hunt-Hess Scale was very similar (Hunt-Hess grades I and II 46% and 45%, grade III 27% and 32%, grade IV 15% and 13%, and Hunt-Hess-grade V 12% and 10% $p = 0.78$), 52% of the patients died in the Dutch study before the end of the third month and only 29% recovered well (GOS V). In our study with a mean follow-up of two months only 23% of our patients died and 43% recovered well (GOS V). Although the possibility cannot be ruled out that our study covered the favorable and HIJDRA's group the unfavourable part of the spectrum of the disease, it is, however, more likely that we observed a further reduction of mortality following subarachnoid hemorrhage as recently described at the Mayo-Clinic [26]. In this population study there was a decline in lethality from 57% in the period 1945–1974 to 42% in 1975–1984. Our prognosis data correspond best with the result of the study by SÄVELAND et al. [45]. In this study, carried out in 1983 in a Swedish district, the outcome was recorded 12 months after admission to hospital. Mortality was 25%, and 41% of the patients recovered well.

Our study confirms the hypothesis that the prognosis primarily depends on the extent of the intracranial hemorrhage [17]. Statistically case fatality ratio was related to the extent of the subarachnoid hemorrhage, the extent of the ventricular hemorrhage and on the size of the intracerebral hematoma. In the presence of MSAH case fatality ratio rose from 3% to 17%. Massive subarachnoid hemorrhage together with massive ventricular hemorrhage had a case fatality ratio of 55%. A massive subarachnoid hemorrhage together with a hematoma greater than 20 ml had a case fatality ratio of 57%. Thus, the combination of two or three of these factors was responsible for the total management case fatality ratio of 23%. A good prognosis correlated with a negative angiogram. These findings confirm a number of other studies: [1, 8, 9, 10, 22, 39, 41].

As expected the extent of the intracranial hemorrhage had a strong influence on the initial Hunt-Hess grade which, in turn, correlated with the case fatality ratio and the degree of disability (GOS grade). Interestingly, fewer of our patients with Hunt-Hess grade III to IV died than did patients surveyed in the Canadian Nimodipine Study Group (grade III 19% compared to our 23%, grade IV 31% compared to our 44%) [10].

In our survey 20% of the patients suffered from acute hydrocephalus, a proportion also reported in other studies [13, 14, 18, 34]. Predispositions for ventricular enlargement are hypertension, old age, and mainly subarachnoid and intraventricular hemorrhage [11, 13, 16, 21, 25, 26, 47, 50]. Accordingly 90% of our patients with acute hydrocephalus were diagnosed as having a massive subarachnoid hemorrhage and 60% of these patients showed substantial ventricular hemorrhage and/or tamponade of the 4th ventricle and aquaeduct. Ventricular shunting, which was performed in all cases of significant ventricular enlargement, was apparently often successful, since case fatality ratio of patients was down to 25% compared to other studies that reported 39% [16] and 59% [14]. Patients with acute hydrocephalus often (40%) developed chronic hydrocephalus and more often retained severe disability (95% GOS II + III).

Eighteen percent of our patients suffered delayed cerebral ischemia, which is less than that reported in other studies (27% to 32% in more recent studies [23, 24] and up to 76% in older studies [38]). Possible reasons for this improvement are the treatment with calcium antagonists, the avoidance of fluid restriction, and avoidance of antifibrinolytic therapy [28, 31, 51]. In this context it is relevant that we diagnosed a delayed cerebral infarction only when the CT scan displayed a hypodensity or when focal symptoms and an EEG focus could not be otherwise explained. Planned studies which evaluate ICD findings will probably show different figures. As in the study by BROTT and MANDYBUR [15], who recorded that delayed cerebral ischemia had occurred with equal frequency among survivors and deceased, in our

study delayed cerebral ischemia was not identified as a lethal factor. Three of the 5 patients with delayed cerebral ischemia who died, suffered from a massive subarachnoid hemorrhage and in 1 of these 3 there was a combination of massive subarachnoid hemorrhage, massive intraventricular hemorrhage and a large intracerebral hematoma. The 2 other patients died as preexisting cerebral infarctions increased postoperatively. However, patients with delayed cerebral ischemia often remained severely disabled. It was confirmed that the probability of delayed cerebral ischemia is increased by massive subarachnoid hemorrhage [2, 12, 19, 28, 30, 33, 37, 38, 43] since 75% of the patient with delayed cerebral ischemia initially had a massive subarachnoid hemorrhage. In contrast to HASAN et al. [18] we found no correlation between delayed cerebral ischemia and massive intraventricular hemorrhage.

In our study 60% of the patients with hydrocephalus (acute or chronic) requiring treatment had delayed cerebral ischemia, which confirms previous observations [6, 14, 18, 25]. Although delayed cerebral ischemia occurred more frequently in patients with severe hydrocephalus, cerebral infarctions occurred with the same frequency prior to and after ventricular shunting. Thus, it seems unlikely that the operative procedure caused further infarctions. The high number of cerebral infarctions in patients with marked hydrocephalus is probably due to reduced regional cerebral blood flow [33, 56] or to hyponatremia due to a hypothalamic dysfunction in dilatation of the 3rd ventricle [53, 55]. Furthermore, cerebral infarctions are considered to be a consequence of fluid restriction after aneurysm rupture [18], which was not practiced in our study. Finally, there may be a chance concurrence of two major complications in high-risk patients with overall high initial Hunt–Hess grades.

A rebleeding rate of 12% is also lower than in other studies (19% to 30%) [24, 25, 32, 36]. The proportion of lethal rebleedings, 25%, was at the same level as in other studies [45]. Statistically, rebleeding was not a lethal factor in our study but patients with rebleeding remained more severely disabled. Since antifibrinolytic therapy was no longer applied during our study, the most likely explanation for the less frequent occurrence of this complications is the early operation time. Rebleeding was not related to the extent of initial subarachnoid hemorrhage, of ventricular hemorrhage, and of intracerebral hematoma. It occurred, however, more frequently in patients with acute and chronic hydrocephalus. In contrast to the findings of the study by HASAN et al. [18] rebleeding did occur independently of ventricular shunting. Since rebleeding occurred more often in patients with an initial Hunt–Hess grade III to V, a coincidental existence of both complications in patients with an unfavourable initial Hunt–Hess grade cannot be ruled out [4, 20, 24, 32, 49].

Our results leave us with the impression that the prognosis of aneurysmal hemorrhage or presumed aneurysmal hemorrhage has improved in recent years due to the application of new diagnostic and therapeutic strategies, i.e., early operation, early detection of hydrocephalus by CT follow-ups, treatment of vasospasm by calcium antagonists, monitoring of vasospasm by transcranial Doppler and therapy of postoperative vasospasm by hypervolemic hemodilution and controlled hypertension.

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Submitted October 14, 1991. Accepted November 22, 1991.

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