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Predicting outcome in poor-grade patients with subarachnoid hemorrhage: a retrospective review of 159 aggressively managed cases

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To determine what factors predict outcome, the authors retrospectively reviewed the management of all 159 poor-grade patients admitted to Harborview Medical Center at the University of Washington who suffered aneurysmal subarachnoid hemorrhage between 1983 and 1993. Favorable outcome (assessed by the Glasgow Outcome Scale) occurred in 53.9% of Hunt and Hess Grade IV, and 24.1% of Grade V patients. Outcome was largely determined by the initial hemorrhage and subsequent development of intractable intracranial hypertension or cerebral infarction. Using multivariate analysis, the authors developed three models to predict outcome. It was found that predicting outcome based only on clinical and diagnostic criteria present at admission may have resulted in withholding treatment from 30% of the patients who subsequently experienced favorable outcomes. It is concluded that aggressive management including surgical aneurysm obliteration can benefit patients with poor neurological grades and should not be denied solely on the basis of the neurological condition on admission.

KEY WORDS • aneurysm • clinical grade • outcome • subarachnoid hemorrhage

PATIENTS categorized as poor clinical grade (Hunt and Hess[18] Grades IV and V) comprise between 20% and 40% of patients admitted to the hospital after subarachnoid hemorrhage (SAH).[22,29,30,42,43,52,55,59,60,62,63] The majority of these patients subsequently die if left untreated.[2,4,37,42,54,56,63] Many neurosurgeons, however, remain reluctant to treat these patients or treat only a select few.[4,34,37,42,52–54,56,58,59,63]

The criteria used to decide which poor-grade patients with SAH are irreparably damaged and consequently excluded from treatment are not clear. A selective SAH treatment policy requires accurate outcome predictions that ideally should be based primarily on admission data. During the last 10 years all patients presenting to our institution after aneurysm rupture were managed according to a standard policy.[25,29] We retrospectively analyzed our experience during the past decade in managing 159 patients with a poor clinical grade (Hunt and Hess Grades IV or V) after aneurysmal SAH to define the determinants of patient outcome.

Clinical Material and Methods

Patient Population

Between September 1983 and August 1993, 543 patients admitted to Harborview Medical Center at the University of Washington underwent treatment for cerebral aneurysms. Harborview Medical Center is the Level 1 acute care facility for Seattle and the surrounding area. Population-based studies demonstrate that 97% of patients suffering SAH in the Seattle area are hospitalized.[31] All patients presenting to the emergency room were considered surgical candidates irrespective of their clinical status. Patients with no brainstem reflexes or function, however, were not considered for treatment. We evaluated all patients retrospectively with the following inclusion criteria: diagnosis of SAH, and poor clinical grade, defined as Hunt and Hess[18] Grade IV or V at admission. Changes in grade were not made for systemic disease or the presence of vasospasm. Subarachnoid hemorrhage was confirmed by computerized tomography (CT) and the presence of an aneurysm was diagnosed by four-vessel angiography or, in moribund patients, by contrast-enhanced CT scan.[26,40] Information was obtained from patient charts, operative reports, review of radiological investigations including CT scans and angiographic films, clinic visits, and telephone interviews.

We reviewed all hospital records and radiographic studies of all poor-grade aneurysm patients and abstracted more than 500 data entries from the preoperative, operative and postoperative periods. We categorized these data into 89 variables as illustrated in Table 1. These factors included pre-SAH information, admission clinical status, admission laboratory studies, admission CT and angiographic findings, preoperative clinical course, surgical findings, postoperative radiographic studies, and postop-

TABLE 1
*Variables evaluated for prognostic significance in 159 poor-grade patients with SAH**

Category	No. of Variables	Factor
patient characteristics	6	age, sex, smoking, hypertension, cardiac disease, vascular disease
admission clinical status	16	Hunt & Hess grade,† Glasgow Coma Scale total & motor score, systolic blood pressure, intubated, meningeal signs, pupils, focal motor weakness, cardiac & pulmonary abnormalities, EKG, hematological & coagulation profile, fibrin degradation products, serum sodium, blood glucose
admission CT findings	9	amount of SAH, presence & size of ICH, midline shift, ventricular size, IVH, SDH, low density, herniation
admission angiogram findings	4	aneurysm size & location, atherosclerosis, vasospasm
preop clinical course	4	rebleed, clinical course, ventriculostomy, ICP
surgery	9	timing, immediate surgery without angiography, cerebral swelling, aneurysm rupture, use of hypotension, temporary clips, clip applications, technique of aneurysm obliteration, condition of brain at closure
postop radiographic studies	9	aneurysm obliteration, angiographic spasm, vascular injury, extent of ICH evacuation, new ICH, low-density changes on CT, ventricular size, effusion, SPECT perfusion defect
postop course	32	new postop cranial nerve palsy or hemiparesis, day follow commands, secondary cerebral insults (hypotension, increased ICP, hypoxia, hyperglycemia),‡ maximum ICP & response to mannitol, seizures, surgical complications (ICH, wound dehiscence, CSF leak, reclip aneurysm), medical complications (DVT, pulmonary embolus, cardiac, pulmonary, renal, hepatic), infection (CNS, wound, pneumonia, UTI, line sepsis, bacteremia, enterocolitis), angiographic spasm, symptomatic spasm & treatment, shunt for hydrocephalus, tracheotomy

* CNS = central nervous system; CSF = cerebrospinal fluid; CT = computerized tomography; DVT = deep vein thrombosis; EKG = electrocardiogram; ICH = intracerebral hemorrhage; ICP = intracranial pressure; IVH = intraventricular hemorrhage; SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; SPECT = single-photon emission CT; UTI = urinary tract infection.

† Clinical status graded according to Hunt and Hess.[18]

‡ Intensive care records were analyzed to determine the incidence of secondary cerebral insults, which are defined as follows: 1) hypotension: systolic blood pressure < 80 mm Hg for at least 15 min; 2) increased ICP: > 20 mm Hg for at least 15 min; 3) hypoxia: $PaO_2 < 70$ mm Hg on two consecutive arterial samples; and 4) hyperglycemia: blood glucose > 200 mg/dl on two consecutive venous samples.

erative hospital course. The complete data instrument is available by request.

Management Protocol

All patients were managed according to a standardized policy that included aggressive prehospital and preoperative resuscitation, early surgery, aggressive prevention and treatment of intracranial hypertension and vasospasm, and comprehensive intensive care. Further details of this management protocol are described elsewhere.[25,29]

Preoperative Care. Aggressive resuscitation, including intubation, ventilation, and mannitol administration, was used before hospitalization and continued in the emergency room. A member of the Neurosurgery Service evaluated all patients in the emergency room. When the patient was stable immediate unenhanced head CT and CT infusion scans were obtained.[40] In neurologically deteriorating patients demonstrating aneurysmal intracerebral hemorrhage (ICH) and brainstem compression, the patient was taken directly to the operating room for hematoma evacuation and aneurysm clipping based on the infusion scan alone; these patients underwent surgery within 8 hours of ictus.[26] The remaining patients were transferred to the Neurosurgery Intensive Care Unit (ICU) and routine intracranial pressure (ICP) and invasive hemodynamic monitoring, including an arterial line and Swan–Ganz catheter, were started. A ventriculostomy was performed in patients demonstrating severe ventricular enlargement or intraventricular hemorrhage (IVH). Four-vessel angiography was performed once normal ICP, normovolemia, and adequate oxygenation were achieved. All patients received phenytoin to therapeutic levels, dexamethasone, and since 1987, nimodipine was routinely administered.

Operative Technique. Surgical obliteration of the aneurysm was achieved as soon as possible in the 24 hours following admission. A craniotomy that was significantly larger than the standard aneurysmal approach was usually performed, particularly if an ICH was present.[28] Brain relaxation was achieved by a combination of osmotic diuresis (mannitol 1–2 mg/kg and furosemide 20–40 mg) and judicious cerebrospinal fluid (CSF) drainage. Hyperventilation was not routinely used. Induced systemic hypotension was avoided; rather judicious temporary clip application was used to decrease the risk of aneurysm rupture and to facilitate aneurysm dissection. Standard microvascular techniques and magnification were used in all cases to obliterate the aneurysm. A duraplasty was performed using temporalis fascia and the bone flap was not replaced if cerebral swelling persisted during closure.

Postoperative Care. Immediate postoperative and subsequent follow-up CT scans were obtained in all patients. Baseline single-photon emission computerized tomography scans and four-vessel angiography were obtained in the majority of patients within 24 hours of surgery. Patients were treated in the Neurosurgery ICU using ICP and invasive hemodynamic monitoring. An ICP greater than 20 mm Hg was aggressively treated by medical and surgi-

TABLE 2

Clinical presentation and findings at admission for 159 poor-grade patients with subarachnoid hemorrhage

Feature	No. (%)
presentation	
sentinel bleed	34 (21.4)
headache	107 (67.3)
seizure	39 (24.5)
transient loss of consciousness	17 (10.7)
coma	138 (86.8)
finding	
neck stiffness	60 (37.7)
dilated pupil or pupils	71 (44.6)
cranial nerve abnormality	89 (56.0)
mild motor weakness	30 (18.9)
severe motor weakness	22 (13.8)
prehospital intubation	133 (83.6)
Hunt & Hess Grade IV	76 (47.8)
Hunt & Hess Grade V	83 (52.2)

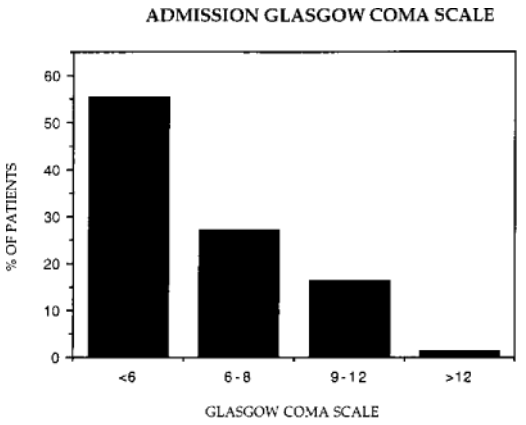

FIG. 1. Bar graph showing Glasgow Coma Scale (GCS) scores on admission; 82.4% of patients demonstrated a GCS less than or equal to 8.

cal techniques. Volume expansion and hemodilution was initiated prophylactically in all patients. According to our treatment protocol since 1987, each patient underwent daily transcranial Doppler examination to diagnose vasospasm, and the diagnosis was confirmed by angiography. Patients who developed symptomatic vasospasm were treated with induced hypertension, and since 1990, angioplasty has been routinely used when other therapies were ineffective.

Patient Outcome

Outcome was assessed at 6 months according to the Glasgow Outcome Scale (GOS).[21] Patients with a GOS score of good or moderately disabled live independently, caring for all their needs, and participate in a normal social life; for statistical comparison, these patients were classified as having a favorable outcome. Patients who died or were not capable of living independently were classified as having an unfavorable outcome.

Statistical Analysis

Data analyses for this report include several bivariate techniques selected for their appropriateness to the distribution of the data. Data are summarized as the mean ± the standard deviation, or as the median if samples depart substantially from normal distributions. The Mann–Whitney U-test was used to evaluate differences between samples whose distributions failed to meet the assumptions underlying difference of means test. Spearman rank-order correlations were used to relate ordered variables to one another. The Student t-test was used to test for differences between groups whose distributions were consistent with normally distributed populations. Where appropriate, Pearson chi-square was used to test for sample differences on categorical variables. The Fisher's exact test was substituted when one or more cells had expected frequencies less than 5. All univariate and bivariate statistical analyses were performed using commercially available software (SPSS/PC+, Version 4.0; Marija J. Norusis/SPSS, Inc., Chicago, IL).

Multivariate model construction began with specification of a pool of potential outcome predictors. To be included, a variable met two requirements: variability in outcome over its range of probability values and an unadjusted association with outcome, based on the techniques described above, that was significant at or beyond the 0.05 level without correction for multiple tests. In addition to the total pool, two smaller pools were identified, one including only measures obtained preoperatively and one including pre- and intraoperative measures. Using these pools three separate multiplicative models were constructed using stepwise logistic regression. Each resulting model was subjected to a series of tests for first-order statistical interactions. The final three models retained only those predictors and any significant interaction terms that after adjustment of covariates were significant at or beyond the 0.05 level. Probability values and confidence intervals for the final models are based on likelihood ratio tests. Logistic regression modeling was performed using commercially available software (EGRET; Statistics and Epidemiology Research Corp., Seattle, WA).

Results

Clinical Data

Over a 10-year period 159 patients, including 53 males and 106 females (median age 54 years) were admitted to the Harborview Medical Center in poor clinical grade (Hunt and Hess Grade IV or V) after an aneurysm rupture. Presentation and clinical characteristics are listed in Table 2. The admission Glasgow Coma Scale (GCS) score[61] is illustrated in Fig. 1. Twenty-nine patients (18.2%) demonstrated clinical evidence of progressive brainstem herniation. Overall 45 patients (28.3%) demonstrated some clinical improvement, whereas 26 patients (16.4%) deteriorated before surgery. Aneurysm rebleeding confirmed by CT scan was observed in 24 patients (15.1%). No patient could be reclassified in good clinical grade before surgery. Five patients died before surgery could be attempted; however, these patients are included in outcome analysis. The remaining 154 patients underwent surgical aneurysm obliteration; 137 (89%), including 29 who underwent immediate surgery (< 8 hours after SAH[26]),

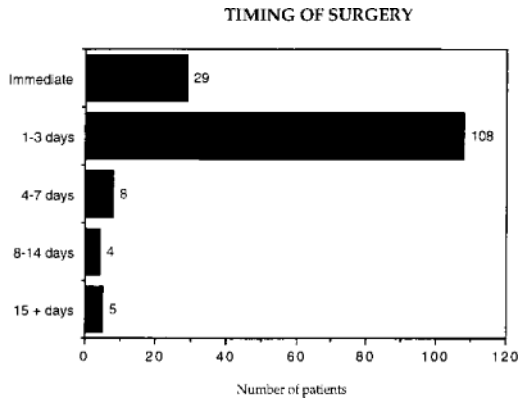

FIG. 2. Bar graph showing timing of surgery in 154 patients with subarachnoid hemorrhage (SAH) resulting in a poor clinical grade. Five patients died before surgery could be attempted. Aneurysm obliteration was achieved within 3 days of SAH in 88.9% of patients. Day 1 = day of hemorrhage.

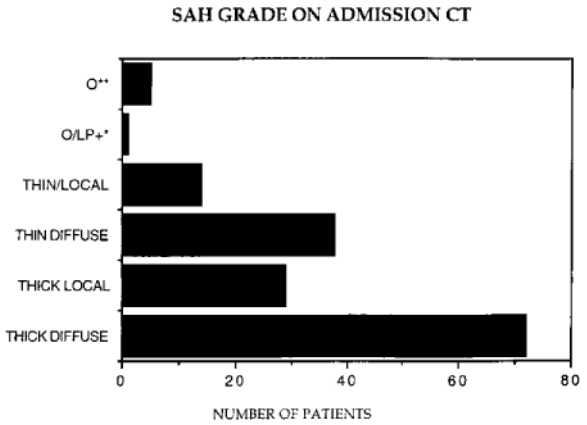

FIG. 3. Bar graph showing subarachnoid hemorrhage (SAH) grade on admission computerized tomography (CT) scans. O** = SAH not visible on admission CT, only intracerebral or intraventricular hemorrhage present. O/LP+* = SAH not visible on admission CT, diagnosed by lumbar puncture (LP).

underwent operation within 72 hours of aneurysm rupture (Fig. 2). Overall 119 (77.3%) patients underwent surgery within 24 hours of admission, 18 (11.7%) between 24 and 48 hours, and eight (5.2%) between 48 and 72 hours after admission to our institution.

Radiographic Data

The preoperative CT characteristics are summarized in Table 3 and Fig. 3. Intracerebral hemorrhages occurred in 70 patients (44%); midline shift (median 10 mm) was observed in 52 (74%) of these patients. Median maximum ICH diameter was 40 mm. The ICH diameter was significantly greater in the presence of CT evidence of herniation than in its absence (Mann–Whitney $z = -5.1$, $p < 0.0001$).

Preoperative four-vessel angiography was performed in all patients, except the 29 (18.2%) moribund patients who underwent immediate operation for aneurysmal ICH based on CT infusion scan alone.[18,24] The distribution of ruptured aneurysms is summarized in Table 4. Mean aneurysm size was 10.5 ± 6.0 mm. Two aneurysms were larger than 25 mm in diameter. Multiple aneurysms were observed in 41 patients (25.7%). Vasospasm and intracranial atherosclerosis were demonstrated on admission angiogram in 26 (16.4%) and 25 (15.7%) patients, respectively.

Patient Outcome

Overall, 61 patients (38.3%) had achieved a favorable outcome at 6-month follow-up review. The outcome according to grade is shown in Table 5; favorable outcomes (good and moderately disabled according to the GOS) were observed in 41 Grade IV patients (53.9%) and 20 Grade V patients (24.1%). Over the decade, we did not observe any improvement in the percentage of patients who achieved a favorable outcome. Sixty-nine patients (43.4%) died; the cause of death is listed in Table 6.

Duration of Neurosurgical Care

The median duration of neurosurgical care for all Hunt and Hess Grade IV and V patients was 23 days. Overall, the 69 poor-grade patients who died remained hospitalized a median of 5 days. By the same time 68.8% of the 90 survivors had regained consciousness and were able to follow commands.

TABLE 3

Computerized tomography features in 159 poor-grade patients with subarachnoid hemorrhage

Feature	No. (%)
subarachnoid hemorrhage	153 (96.2)
mild or moderate intraventricular hemorrhage	93 (58.4)
severe intraventricular hemorrhage	31 (19.5)
mild or moderate ventricular enlargement	84 (52.8)
severe ventricular enlargement	9 (5.7)
intracerebral hemorrhage	
<3 cm	25 (15.7)
≥3 cm	45 (28.3)
low-density changes	7 (4.4)
herniation (compressed or obliterated perimesencephalic cisterns)	48 (30.2)

TABLE 4

*Location of ruptured aneurysms in 159 poor-grade patients with subarachnoid hemorrhage**

Location	No. (%)
pericallosal	4 (2.5)
anterior cerebral or communicating artery	37 (23.3)
middle cerebral artery bifurcation or trunk	41 (25.8)
posterior communicating artery	32 (20.1)
internal carotid & carotid ophthalmic artery	19 (11.9)
posterior circulation	26 (16.4)

* Aneurysm distribution is based on preoperative four-vessel angiography in 130 patients or computerized tomography infusion scans that were obtained in 29 moribund patients who underwent immediate operation for aneurysmal intracerebral hemorrhage without angiography.

TABLE 5

*Admission clinical grade and outcome in 159 poor-grade patients with subarachnoid hemorrhage**

Admission Clinical Grade‡	No. of Patients	Outcome (%)†				
		Good	Moderately Disabled	Severely Disabled	Vegetative	Dead
Grade IV	76	18.4	35.5	19.7	1.3	25.0
Grade V	83	4.8	19.3	10.8	4.8	60.2

* Clinical grade at admission according to Hunt and Hess,[18] not corrected for systemic disease or vasospasm.

† Outcome was assessed at 6 months according to the Glasgow Outcome Scale.[21]

‡ Grade V at admission correlated with a worse outcome ($\chi^2 = 15$, $p = 0.0001$).

Factors Associated With Outcome

To determine which factors were associated with outcome, we examined all 89 factors outlined in Table 1 by bivariate analysis. The significant results are summarized in Table 7.

Advanced age was associated with poor outcome ($p = 0.02$, Fig. 4). The median age for patients who achieved a favorable outcome was 50 years, whereas the median age for patients with an unfavorable outcome was 55 years. A similar tendency for older patients to experience less favorable outcomes was observed when patients were stratified according to clinical grade; however, this difference was not statistically significant. The most statistically significant preoperative patient characteristics associated with outcome included the admission GCS score and the Hunt and Hess grade (Table 6; $p = 0.0001$). A decrease in either total ($p = 0.0006$) or motor ($p = 0.001$) GCS score correlated with a poor outcome. In contrast to the level of consciousness, clinical findings such as pupillary abnormalities and hemiparesis on admission did not correlate with outcome. Diagnostic studies such as blood glucose in Grade V patients only ($p = 0.02$) and fibrin degradation products in Grade IV and V patients ($p = 0.01$) were associated with outcome; elevation of either variable was correlated with an unfavorable outcome.

Among the various CT and angiographic features assessed, only the presence of IVH ($p = 0.0003$), ventricular enlargement ($p = 0.04$), or low-density CT changes ($p = 0.03$) were associated with outcome; all predicted an unfavorable outcome. In the preoperative course only a failure to improve clinically ($p = 0.04$) was associated with an unfavorable outcome.

Three surgical factors demonstrated an association with unfavorable outcome: 1) intraoperative aneurysm rupture ($p = 0.02$); 2) the condition of the brain at closure ($p = 0.003$); and 3) parenchymal resection necessary to expose the aneurysm in 38 patients ($p = 0.05$). The timing of surgery, including immediate surgery without angiography, did not correlate with outcome. Surgical complications, including a postoperative ICH requiring surgical evacuation or reclipping of the aneurysm, were similarly not associated with outcome.

The development of a new postoperative pupillary abnormality correlated with a poor outcome ($p = 0.02$), whereas the early ability to follow commands correlated with good recovery or moderate disability at 6-month follow-up review ($p < 0.0001$). The development of multiple postoperative medical complications ($p = 0.046$) was associated with an unfavorable outcome. Among secondary insults that occurred during the hospital course only an episode of hypotension ($p = 0.017$) predicted a poor outcome. The ICP was monitored in all patients in the immediate postoperative period. Although an episode

TABLE 6

Cause of death in 69 poor-grade patients with subarachnoid hemorrhage

Cause of Death	No. (%)*
initial hemorrhage	46 (28.9)
rebleed	8 (5.0)
vasospasm	1 (0.6)
increased intracranial pressure	1 (0.6)
stroke	1 (0.6)
myocardial infarction	2 (1.3)
respiratory failure	5 (3.1)
sepsis	2 (1.3)
undetermined	3 (1.9)

* Percent of entire series of 159 patients.

TABLE 7

*Variables associated with outcome in 159 poor-grade patient with SAH after uni- or bivariate analysis**

Factor	Significance
pre-SAH condition	
age	Mann–Whitney, $z = -2.4$, $p = 0.02$
admission clinical status	
Hunt & Hess grade	$\chi^2 = 14.95$, df = 1, $p = 0.0001$
GCS, motor score	Mann–Whitney, $z = -3.2$, $p = 0.0014$
GCS, total score	Mann–Whitney, $z = -3.5$, $p = 0.0006$
blood glucose, Grade V only	Mann–Whitney, $z = -2.3$, $p = 0.02$
fibrin degradation products	Mann–Whitney, $z = -2.6$, $p = 0.01$
admission CT findings	
IVH	Mann–Whitney, $z = -3.6$, $p = 0.0003$
ventricular enlargement	Mann–Whitney, $z = -2.1$, $p = 0.04$
low-density changes	$\chi^2 = 6.6$, df = 1, $p = 0.03$
admission angiogram	NS
preop course	
clinical course	Mann–Whitney, $z = -2.06$, $p = 0.04$
surgery	
parenchyma resected during exposure	$\chi^2 = 3.9$, df = 1, $p = 0.05$
aneurysm rupture	$\chi^2 = 5.7$, df = 1, $p = 0.02$
brain condition at closure	Mann–Whitney, $z = -2.9$, $p = 0.003$
postop radiographic studies	
effusion	$\chi^2 = 4.4$, df = 1, $p = 0.035$
low-density changes on CT	$\chi^2 = 22.9$, df = 1, $p < 0.0001$
postop course	
new pupillary abnormality	Mann–Whitney, $z = -2.3$, $p = 0.02$
day follow commands	Mann–Whitney, $z = -8.0$, $p < 0.0001$
hypotension	$\chi^2 = 5.7$, df = 1, $p = 0.017$
number of medical complications	Mann–Whitney, $z = -1.9$, $p = 0.046$
no ICP response to mannitol therapy	Mann–Whitney, $z = -2.6$, $p = 0.008$

* CT = computerized tomography; GCS = Glasgow Coma Scale; ICP = intracranial pressure; IVH = intraventricular hemorrhage; NS = not significant; SAH = subarachnoid hemorrhage.

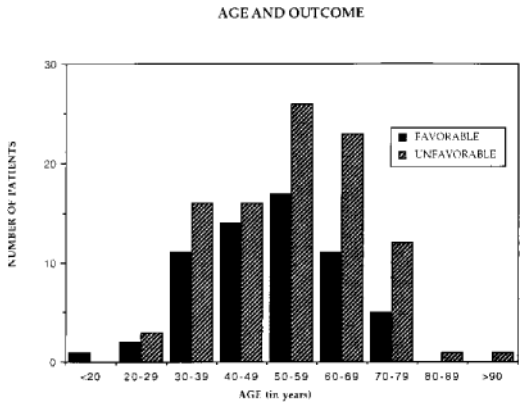

FIG. 4. Bar graph correlating age and outcome. Advanced age was associated with poor outcome using bivariate analysis ($p = 0.02$). However, age was not associated with outcome when stratified according to clinical grade and after multivariate analysis.

of raised ICP or median maximum ICP did not correlate with outcome, lack of ICP response after mannitol administration was significantly associated with unfavorable outcome ($p = 0.008$). All patients underwent at least one follow-up CT scan. An area of low density observed on the follow-up CT scan predicted a poor outcome ($p < 0.0001$).

Multivariate Analysis and Outcome Prediction

To determine which variables most correlated with outcome, three prediction models were constructed using variables found to have a significant association with outcome in uni- and bivariate analysis (Table 7). The results of multivariate analysis are listed in Table 8.

Preoperative Model. This paradigm used clinical, diagnostic and radiographic factors observed at admission and characteristics of the patient's preoperative clinical course. Of the variables tested, low density observed on admission CT demonstrated the strongest association with outcome. Other variables that were associated with unfavorable outcome included Hunt and Hess Grade V, elevated blood glucose, fibrin degradation products, severity of IVH on admission CT, and no clinical improvement. The preoperative model correctly predicted 77.4% of outcomes, including 70.5% of favorable and 81.6% of unfavorable outcomes.

Pre- and Intraoperative Model. This analysis included factors present at admission, characteristics of the preoperative course, and surgical factors that demonstrated significant unadjusted associations with outcome (Table 7). Low density on admission CT continued to exhibit the strongest association with an unfavorable outcome. The combination of admission, preoperative, and operative variables slightly increased the accuracy of correctly predicting outcome: 78.6% of all outcomes including 72.1% of favorable and 82.7% of unfavorable outcomes were correctly predicted.

Pre-, Intra-, and Postoperative Model. This comprehensive model added postoperative factors and included all predictors of outcome for which there were no missing data, which were entered using a stepwise procedure. The day on which patients first followed commands was excluded from this analysis because a value would be assumed for patients who died without having followed commands. Six factors that made significant prediction were retained from the 19 potential predictors (Table 7). The preoperative variables of clinical grade, blood glucose, and IVH on admission CT were replaced by medical complications and the development of new low-density CT changes during the hospital course (Table 9). The comprehensive model correctly predicted 82.4% of all outcomes, including 70.5% of favorable and 89.8% of unfavorable outcomes.

In summary, our models correctly predicted between 77% and 82% of all patient outcomes. Favorable outcome, however, was correctly predicted in only 70.5% of patients whether preoperative factors alone or all pre-, intra-, and postoperative factors were included.

TABLE 8

*Multivariate analysis of variables demonstrating potential prognostic significance in 159 poor-grade patients with SAH**

Predictor	Odds Ratio	95% CI	p Value
preop model			
Hunt & Hess Grade V	2.59	1.18–5.71	0.017
blood glucose	2.42	1.03–5.69	0.038
fibrin degradation products	7.41	1.93–28.47	<0.001
severity of IVH on admission CT	1.88	1.24–2.85	0.002
low density on admission CT	15.59	1.66–144.2	0.002
no clinical improvement	3.23	1.35–7.77	0.007
pre- & intraop model			
Hunt & Hess Grade V	2.34	1.03–5.33	0.042
blood glucose	3.11	1.25–7.73	0.011
fibrin degradation products	9.08	2.22–37.11	<0.001
severity of IVH on admission CT	1.86	1.2–2.87	0.004
low density on admission CT	11.80	1.29–107.9	0.007
no clinical improvement	3.05	1.26–7.41	0.011
intraop aneurysm rupture	3.51	1.22–10.07	0.016
pre-, intra-, & postop model			
fibrin degradation products	12.64	3.0–53.24	<0.001
low density on admission CT	14.67	1.32–162.8	0.007
no preop clinical improvement	3.86	1.39–10.71	0.007
intraop aneurysm rupture	13.07	2.82–60.72	<0.001
multiple medical complications	6.26	2.14–18.3	<0.001
new low density on postop CT	8.01	3.15–20.34	<0.001

* All variables that predicted outcome from uni- or bivariate analysis (Table 7), for which there were no missing data, were included in a multiplicative model without interactions, using logistic regression techniques. The day on which patients first followed commands was excluded from multivariate analysis because a value would be assumed for patients who died and never followed commands. Abbreviations: CI = confidence interval based on Walds' statistic; CT = computerized tomography; IVH = intraventricular hemorrhage; SAH = subarachnoid hemorrhage.

Discussion

This retrospective report describes our experience with the management of 159 unselected patients who were classified at admission as Hunt and Hess Grade IV or V after aneurysm rupture. All patients with poor-grade aneurysmal SAH admitted to our institution during the last 10 years were evaluated and are included in this analysis, including 29 moribund patients demonstrating

TABLE 9

*Outcome in poor-grade (Hunt and Hess IV and V) patients after aneurysm rupture**

Authors & Year	No. of Patients (%)‡	Age (yrs)	Management§	Outcome (%)†		
				Favorable	Poor	Dead
Hunt & Hess, 1968	47 (17.1)	NR	delayed surgery until Grade I or II	NR	NR	78.7
Adams, *et al*., 1981	61 (26)	NR	delayed surgery; antifibrinolytics	18	24.6	57.4
Testa, *et al*., 1985	80 (36)	mean 51.4, range 14–73	delayed surgery until Grade I, II, or III; limited ICU care	3.8	8.8	87.4
Freckmann, *et al*., 1987	20 (6.3)	NR	delayed surgery unless ICH present; routine CCB, HV	5	20	75
Hijdra, *et al*., 1987	42 (15.9)	est; 28% >60	delayed surgery until Grade I or II; some patients received antifibrinolytics, excluded patients >65 yrs old	5	23	71
Ohno, *et al*., 1988	32 (34.7)	14 patients >70	delayed surgery unless ICH present	15.6	15.6	68
average				9.5	18.4	72.9
Chyatte, *et al*., 1988	80 (32.8)	NR	selective early surgery (26%)	25	29	46
Inagawa, *et al*., 1988	157 (24.8)	44% >60	selective early surgery; surgery deferred in 66.8% patients	9.5	15.2	75.2
Petruk, *et al*., 1988	108 (NR)	est; 54	multicenter randomized trial of CCB; no standard management	25	21.3	53.7
Sevrain, *et al*., 1990	66 (24.4)	mean 47.2, range 20–74	early surgery except patients with large ICH and abnormal pupils	19.6	12.2	68.2
Medlock, *et al*., 1992	41 (36)	mean 53.1	early surgery; routine HV, no CCB	7	5	88
Miyaoka, *et al*., 1993	370 (22.8)	NR	multicenter; selective early surgery (28%)	20.8	15.7	63.5
average				17.8	16.4	65.8
Bailes, *et al*., 1990	54 (23.3)	mean 56	EVD; selective aggressive; routine HV	42.6	7.4	50
Seifert, *et al*., 1990	74 (17.3)	14 patients >60	EVD for hydrocephalus; selective aggressive	20.2	12.2	67.4
Nowak, *et al*., 1994	109 (39.4)	NR	EVD; selective aggressive; routine CCB	21.1	36.7	42.2
Steudel, *et al*., 1994	116 (20.2)	est; 49.5	EVD for hydrocephalus; selective aggressive	35.3	7.8	56.9
Ungersbock, *et al*., 1994	48 (24.5)	mean 53.1, range 31–77	EVD; selective aggressive; routine CCB	21.3	36.2	42.5
average				28.1	20.1	51.8
Le Roux, *et al*., 1996 (present series)	159 (36.5)	median 54	aggressive management of all patients	38.4	18.2	43.4

* CCB = calcium channel blocker antagonists; est = estimated from limited data; EVD = extraventricular drainage; GOS = Glasgow Outcome Scale; HV = hypervolemia; ICH = intracerebral hemorrhage; ICP = intracranial pressure; ICU = intensive care unit; NR = not reported.

† Favorable = independent, including GOS scores of good and moderately disabled; poor = dependent, including GOS scores of severely disabled and vegetative.

‡ The number of patients presenting in poor clinical grade after aneurysm rupture. This number is given in parentheses as a percentage of all patients in all clinical grades treated at the same institution(s).

§ Selective aggressive = emergency evacuation of ICH, early surgery in patients demonstrating clinical improvement or controllable ICP after ventricular drainage; not all patients are intubated and ventilated. Aggressive management is described in *Clinical Material and Methods*.

clinical evidence of brainstem herniation. No patient was excluded. A standardized aggressive medical and surgical treatment protocol centered on early aneurysm obliteration was used to treat all patients; overall 38.3% of Hunt and Hess Grades IV and V patients experienced a favorable outcome. Although outcome was largely determined by the initial hemorrhage and its immediate pathophysiological consequences, attempting to select patients for treatment may have resulted in care being withheld from nearly one-third of the patients who subsequently experienced a favorable outcome.

Aggressive Management of Poor-Grade SAH Patients

Poor clinical grade is unquestionably associated with unfavorable outcome following SAH; what is the optimum management of these patients? Although less relevant than a randomized study, comparison of the results obtained in this series with those reported from centers using a less aggressive approach to poor-grade SAH patients suggests that outcome is favorably influenced by an aggressive approach, including rapid resuscitation and control of ICP, early surgery, comprehensive intensive care and prophylaxis against delayed ischemia (Table 9). More than 90% of untreated poor-grade patients die.[4,17,37,52,56] When treatment is delayed until clinical improvement is observed or select patients undergo early surgery using a less aggressive approach, favorable outcomes are observed, on average, in 9% and 18% of patients, respectively (see Table 9).[6,13,17,20,22,33,37,43,47,54,56,62] Our overall outcome results also compare favorably to those reported when only selected poor-grade patients, who improve after ventricular drainage, are managed aggressively; using this ventricular drainage strategy 28% of patients, on average, experience a favorable outcome (Table 9).[4,42,54,59,63]

Ventricular drainage and aggressive management of the selected patients who demonstrate clinical improvement is a common strategy used to manage poor-grade patients.[4,42,59] This approach is based on the premise that ventricular drainage will control increased ICP and attenuate the deleterious effects of SAH. We agree that rapid ICP control is necessary; however, in our patients ICP was similar whether or not they received ventriculostomy. In addition, after SAH ventricular drainage only reduces ICP briefly and is followed by elevated ICP when the CSF buffer becomes exhausted.[41] There are other potential disadvantages of ventricular drainage that may limit its

therapeutic application in poor-grade patients. First, ventricular drainage is associated with a significant increase in aneurysm rebleeding and infection.[45,48,49,64] Second, catheters may be difficult to insert when severe edema or shift is present, or they may drain poorly when there is severe IVH. Third, an expectant strategy using ventriculostomy alone leaves the ruptured aneurysm unprotected, potentially precluding vasospasm treatment by hyperdynamic therapy or angioplasty.[39] Finally, clinical improvement with ventricular drainage is not always associated with a favorable outcome.[38,42,49,59,64] Furthermore, many patients who do not improve with ventricular drainage undergo surgery with satisfactory results.[42,59] We did not observe an association between outcome and the use of ventriculostomy. Therefore, although ventriculostomy may be effective in some patients, it should be viewed primarily as a temporary measure to stabilize the patient and should not delay definitive surgery or management. In addition, the response to ventricular drainage alone should not determine which poor-grade patients receive definitive care.

The impact of aggressive management for patients with poor-grade SAH is more impressive when natural history data are considered. For example, in the present series 24.1% of Grade V patients experienced a favorable outcome. By contrast, natural history data and population based studies indicate that only 5% of Grade V patients are expected to survive when admitted to the hospital within 3 days of SAH.[2,5] Despite what appears to be an improvement in outcome in this series compared to earlier studies, we did not note an improvement in outcome over the decade. This is in contrast to the statistically significant increase in the number of patients with good-grade SAH (Hunt and Hess Grades I–III) treated at our institution who experienced a favorable outcome over the same time period.[25]

An aggressive approach for patients with poor-grade SAH is often questioned because of concerns such as 1) increased risk of surgical complications; 2) an increase in the number of survivors in poor condition; and 3) an unnecessary use of neurosurgical resources. Our results suggest that these concerns may be less valid than previously thought. First, when compared to 282 good-grade patients treated during the same review period, poor-grade patients demonstrated a similar risk of surgical complications such as intraoperative aneurysm rupture, failure to occlude the aneurysm, or cerebral contusion.[27] However, poor-grade patients had a higher rate of intraoperative brain swelling, which was correlated with the presence of ICH. Second, survivors in poor condition represented less than 20% of the outcome observed in this series. A similar number of survivors in poor condition is observed whether an aggressive approach is applied to selected poor-grade patients or a less aggressive approach is used (Table 9).[4,6,30,37,47,54,56,59] Data from population-based studies and the Cooperative Study also suggest that more active treatment for SAH including early surgery is associated with a significant increase in the number of functional survivors, whereas mortality remains similar.[12,15] Third, whereas many variables influence utilization of resources, one variable, median length of time spent in neurosurgical care, was not statistically different for all patients with poor- (23 days) or good-grade SAH (282 patients; 19 days) patients treated at our institution during the same time period. In addition, although the mean cost of treating cerebral aneurysms in patients harboring unruptured aneurysms and of Grades I through IV following SAH correlates with clinical grade, the mean cost of treating Grade V patients is similar to that for Grade II patients.[10] In part, these findings were influenced by the prevalence of early mortality in poor-grade patients; those who died generally did so within a short time of aneurysm rupture, similar to the observations of Bailes, *et al*.,[4] and Steudel, *et al*.,[59] indicating that most neurosurgical attention is provided to those patients who are likely to experience a favorable outcome.

Elderly Patients

An association between advanced age and poor outcome was observed; should the elderly patient in poor clinical condition after aneurysm rupture be aggressively treated? Whereas many studies suggest advanced age is associated with poor outcome after SAH, other studies demonstrate that old and young people in the same clinical condition experience a similar outcome.[22,31,44,50] There are several important considerations when evaluating the association between age and outcome: 1) patients older than 65 years are frequently excluded from admission or active treatment,[55,64] with this exclusion exerting a considerable influence on outcome; 2) older patients are more frequently in poor clinical grade,[43,66] which has a greater impact on outcome than age;[14] and 3) other variables such as hypertension or atherosclerosis are more frequent in elderly patients, and these factors taken independently may have an adverse effect on outcome.[22] In our series, bivariate analysis demonstrated an association between advanced age and poor outcome. However, age was not associated with outcome when patients were stratified according to clinical grade. In addition, following multivariate analysis, age was replaced by other clinical and radiographic variables. Similarly, when we analyzed outcome in patients with good-grade SAH treated during the same review period, advanced age was replaced by clinical grade, IVH, and atherosclerosis on admission angiogram when all pre-, intra- and postoperative variables were subjected to multivariate analysis.[25] Consequently we believe that withholding or delaying treatment solely on the grounds of advanced age may not always be justified.

Timing of Surgery

There is little available information on the optimum time for poor-grade patients to undergo surgery. Several nonrandomized clinical series[26,46,65] and a single randomized study[16] demonstrate a tendency for patients with aneurysmal ICH to experience a more favorable outcome when emergency surgical hematoma evacuation and simultaneous aneurysm obliteration is achieved. Similarly, in the absence of an ICH, clinical series demonstrate a tendency for poor-grade patients undergoing early surgery to experience a more favorable outcome than patients undergoing delayed surgery after aneurysm rupture (Table 9).[9,15,37,54,63] However, neurosurgeons have generally delayed surgery in poor-grade patients to avoid technical difficulties and surgical complications.[32] These expec-

tations may not be valid because several studies comparing patient cohorts or historical controls demonstrate that, whereas cerebral swelling is more frequent during early surgery, the incidence and severity of technical difficulties, surgical complications, or surgical morbidity is similar to patients of all grades undergoing delayed surgery.[4,6–8,23,36]

The results of this series do not provide evidence favoring either early or delayed surgery; however, there are several theoretical advantages of early surgery in poor-grade patients. First, mass lesions and intracranial hypertension are common in poor-grade patients.[29,46,65] Delayed surgery may, therefore, subject the patient to potentially reversible insults.[11] Second, cerebral blood flow (CBF) is reduced after SAH. This reduction in CBF is significantly greater in poor-grade than good-grade patients and progresses in severity from the day of aneurysm rupture;[35] early surgery may therefore be preferable because CBF is least reduced. Third, rebleeding is more frequent in poor-grade than good-grade patients.[3,19,51,54] Finally, the incidence of vasospasm is greater in poor-grade patients; optimum therapy is best performed after aneurysm occlusion.[28,40,57] Whereas none of the studies described above are conclusive, when taken together they indicate that early surgical obliteration of ruptured aneurysms may provide the poor-grade patient the most reasonable chance for a favorable outcome.

Outcome Prediction

The association of poor outcome and poor clinical grade post-SAH is well described.[2,7,18,22,50,52] What admission features, however, specifically predict outcome in these patients in poor clinical condition? Our results suggest that when using an aggressive management strategy, admission clinical criteria, including evidence of brainstem herniation,[26] may be insufficient to reliably predict outcome. Diagnostic tests easily performed in most laboratories, such as blood glucose[24] or fibrin degradation products, may offer useful information but are relatively nonspecific. Additional prognostic information can be provided by CT scans obtained at admission, particularly if there is evidence of infarction[4] or severe IVH.[38] In contrast to previous studies,[4,56,65] we did not observe an association between ICH and outcome; in part this may be related to our aggressive surgical approach to these patients.

To decide which poor-grade patients should be aggressively managed, we constructed a simple predictive model based on admission and preoperative factors. Two points are important in interpreting the results of this multivariate analysis. First, the model only examined variables that demonstrated a direct association with outcome; nonsignificant variables that may have been associated with these ultimate predictors were not included. Second, the estimates of several odds ratios were imprecise, with wide confidence intervals. Bearing these caveats in mind, if we had attempted to select patients for aggressive management based on admission clinical characteristics, radiographic criteria, and early preoperative observation, including ventricular drainage and ICP monitoring, we may have failed to provide definitive treatment to nearly 30% of the 61 patients who ultimately achieved a favorable outcome. Similar levels of predictive inaccuracy using stepwise multiple regression techniques or discriminant functional analysis have been observed in studies of aneurysmal ICH[65] or Grades III to V patients,[9] and prospective studies comparing high risk and low-risk patients of all grades.[14] Clinical series describing ventricular drainage for poor-grade patients have also found that clinical and radiological evaluation performed at admission provides inadequate prognostic information.[4,59] Together these findings suggest that outcome following SAH in patients with poor neurological grades may not be reliably predicted at admission. However, a bivariate analysis of this series indicates that outcome is largely determined by the initial hemorrhage and subsequent development of intracranial hypertension. Therefore, a short time frame exists in which the deleterious effects of severe SAH are potentially reversible.[11] Consequently we believe that aggressive management, including early surgery, can benefit poor-grade patients and should not be denied or delayed on the basis of admission clinical or radiological findings alone.

When should care be withheld from patients in poor clinical grade? A decision to withhold care must be based on an accurate outcome prediction that may not be possible at admission. However, once management is initiated, observation appears to provide additional prognostic information. In this series most poor-grade patients who survived and experienced a favorable outcome were able to follow commands within 5 days of aneurysm rupture. By contrast, those who died generally did so within the same time frame. Management can also be guided by the progression of neurological abnormalities, failure to improve postsurgery, the development of intractable intracranial hypertension, or low-density changes on follow-up CT scans. These postoperative data can be used to determine if therapy should be pursued or discontinued. This protocol of initial aggressive management in all poor-grade patients with SAH and appropriate withholding of care based on continued evaluation provides these patients with their most reasonable chance of a favorable outcome without excessive demands on physician or hospital resources.

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References

1. Adams HP Jr, Kassell NF, Torner JC, et al: Early management of aneurysmal subarachnoid hemorrhage. A report of the Cooperative Aneurysm Study. **J Neurosurg 54:**141–145, 1981
2. Alvord EC, Loeser JD, Bailey WL, et al: Subarachnoid hemorrhage due to ruptured aneurysms. A simple method of estimating prognosis. **Arch Neurol 27:**273–284, 1972
3. Aoyagi N, Hayakawa I: Analysis of 223 ruptured intracranial aneurysms with special reference to rerupture. **Surg Neurol 21:**445–452, 1984
4. Bailes JE, Spetzler RF, Hadley MN, et al: Management morbidity and mortality of poor-grade aneurysm patients. **J Neurosurg 72:**559–566, 1990
5. Bonita R, Thomson S: Subarachnoid hemorrhage: epidemiology, diagnosis, management, and outcome. **Stroke 16:**591–594, 1985
6. Chyatte D, Fode NC, Sundt TM Jr: Early versus late intracranial

aneurysm surgery in subarachnoid hemorrhage. **J Neurosurg 69:**326–331, 1988
7. Deruty R, Mottolese C, Pelissou-Guyotat I, et al: Management of the ruptured intracranial aneurysm—early surgery, late surgery, or modulated surgery? Personal experience based upon 468 patients admitted in two periods (1972–1984 and 1985–1989). **Acta Neurochir 113:**1–10, 1991
8. Disney L, Weir B, Grace M: Factors influencing the outcome of aneurysm rupture in poor grade patients: a prospective series. **Neurosurgery 23:**1–9, 1988
9. Disney L, Weir B, Petruk K: Effect on management mortality of a deliberate policy of early operation on supratentorial aneurysms. **Neurosurgery 20:**695–701, 1987
10. Elliott JP, Le Roux PD, Ransom G, et al: Aneurysm grade on admission and length of hospital stay and cost. **J Neurosurg 85:** (In press, 1996)
11. Fisher CM, Ojemann RG: Bilateral decompressive craniectomy for worsening coma in acute subarachnoid hemorrhage. Observations in support of the procedure. **Surg Neurol 41:** 65–74, 1994
12. Fogelholm R, Hernesniemi J, Vapalahti M: Impact of early surgery on outcome after aneurysmal subarachnoid hemorrhage. A population-based study. **Stroke 24:**1649–1654, 1993
13. Freckmann N, Noll M, Winkler D, et al: Does the timing of aneurysm surgery neglect the real problems of subarachnoid haemorrhage? **Acta Neurochir 89:**91–99, 1987
14. Gerber CJ, Lang DA, Neil-Dwyer G, et al: A simple scoring system for accurate prediction of outcome within four days of a subarachnoid haemorrhage. **Acta Neurochir 122:**11–22, 1993
15. Haley EC Jr, Kassell NF, Torner JC, et al: The International Cooperative Study on the Timing of Aneurysm Surgery: the North American experience. **Stroke 23:**205–214, 1992
16. Heiskanen O, Poranen A, Kuurne T, et al: Acute surgery for intracerebral haematomas caused by rupture of an intracranial arterial aneurysm. A prospective randomized study. **Acta Neurochir 90:**81–83, 1988
17. Hijdra A, Braakman R, van Gijn J, et al: Aneurysmal subarachnoid hemorrhage. Complications and outcome in a hospital population. **Stroke 18:**1061–1067, 1987
18. Hunt WE, Hess RM: Surgical risk as related to time of intervention in the repair of intracranial aneurysms. **J Neurosurg 28:**14–20, 1968
19. Inagawa T, Kamiya K, Ogasawara H, et al: Rebleeding of ruptured intracranial aneurysms in the acute stage. **Surg Neurol 28:**93–99, 1987
20. Inagawa T, Takahashi M, Aoki H, et al: Aneurysmal subarachnoid hemorrhage in Izumo City and Shimane Prefecture of Japan. Outcome. **Stroke 19:**176–180, 1988
21. Jennett B, Bond M: Assessment of outcome after severe brain damage. A practical scale. **Lancet 1:**480–484, 1975
22. Kassell NF, Torner JC, Haley EC Jr, et al: The International Cooperative Study on the Timing of Aneurysm Surgery. Part 1: Overall management results. **J Neurosurg 73:**18–36, 1990
23. Kassell NF, Torner JC, Jane JA, et al: The International Cooperative Study on the Timing of Aneurysm Surgery. Part 2: Surgical results. **J Neurosurg 73:**37–47, 1990
24. Lanzino G, Kassell NF, Germanson T, et al: Plasma glucose levels and outcome after aneurysmal subarachnoid hemorrhage. **J Neurosurg 79:**885–891, 1993
25. Le Roux P, Elliott JP, Downey L, et al: Improved outcome after rupture of anterior circulation aneurysms: a retrospective 10-year review of 224 good-grade patients. **J Neurosurg 83:** 394–402, 1995
26. Le Roux PD, Dailey AT, Newell DW, et al: Emergent aneurysm clipping without angiography in the moribund patient with intracerebral hemorrhage: the use of infusion computed tomography scans. **Neurosurgery 33:**189–197, 1993
27. Le Roux PD, Elliott JP, Newell DW, et al: The incidence of surgical complications is similar in good and poor grade patients undergoing repair of ruptured anterior circulation aneurysms: a retrospective review of 355 patients. **Neurosurgery 38:**887–895, 1996
28. Le Roux PD, Mayberg MR: Management of vasospasm: angioplasty, in Ratcheson RA, Wirth FP (eds): **Ruptured Cerebral Aneurysms: Perioperative Management.** Baltimore: Williams & Wilkins, 1994, pp 155–167
29. Le Roux PD, Winn HR: The poor grade aneurysm patient, in Salcman M (ed): **Current Techniques in Neurosurgery.** Philadelphia: Current Medicine, 1993, pp 10.1–10.28
30. Ljunggren B, Säveland H, Brandt L, et al: Early operation and overall outcome in aneurysmal subarachnoid hemorrhage. **J Neurosurg 62:**547–551, 1985
31. Longstreth WT Jr, Nelson LM, Koepsell TD, et al: Clinical course of spontaneous subarachnoid hemorrhage: a population-based study in King County, Washington. **Neurology 43:** 712–718, 1993
32. Marsh H, Maurice-Williams RS, Lindsay KW: Differences in the management of ruptured intracranial aneurysms: a survey of practice amongst British neurosurgeons. **J Neurol Neurosurg Psychiatry 50:**965–970, 1987
33. Medlock MD, Dulebohn SC, Elwood PW: Prophylactic hypervolemia without calcium channel blockers in early aneurysm surgery. **Neurosurgery 30:**12–16, 1992
34. Mercier P, Alhayek G, Rizk T, et al: Are the calcium antagonists really useful in cerebral aneurysmal surgery? A retrospective study. **Neurosurgery 34:**30–37, 1994
35. Meyer CHA, Lowe D, Meyer M, et al: Progressive change in cerebral blood flow during the first three weeks after subarachnoid hemorrhage. **Neurosurgery 12:**58–76, 1983
36. Milhorat TH, Krautheim M: Results of early and delayed operations for ruptured intracranial aneurysms in two series of 100 consecutive patients. **Surg Neurol 26:**123–128, 1986
37. Miyaoka M, Sato K, Ishii S: A clinical study of the relationship of timing to outcome of surgery for ruptured cerebral aneurysms. A retrospective analysis of 1622 cases. **J Neurosurg 79:**373–378, 1993
38. Mohr G, Ferguson G, Khan M, et al: Intraventricular hemorrhage from ruptured aneurysm. Retrospective analysis of 91 cases. **J Neurosurg 58:**482–487, 1983
39. Newell DW, Eskridge JM, Mayberg MR, et al: Angioplasty for the treatment of symptomatic vasopasm following subarachnoid hemorrhage. **J Neurosurg 71:**654–660, 1989
40. Newell DW, LeRoux PD, Dacey RG Jr, et al: CT infusion scanning for the detection of cerebral aneurysms. **J Neurosurg 71:**175–179, 1989
41. Nornes H, Magnaes B: Intracranial pressure in patients with ruptured saccular aneurysm. **J Neurosurg 36:**537–547, 1972
42. Nowak G, Schwachenwald R, Arnold H: Early management in poor grade aneurysm patients. **Acta Neurochir 126:**33–37, 1994
43. Ohno K, Suzuki R, Masaoka H, et al: A review of 102 consecutive patients with intracranial aneurysms in a community hospital in Japan. **Acta Neurochir 94:**23–27, 1988
44. O'Sullivan MG, Dorward N, Whittle IR, et al: Management and long-term outcome following subarachnoid hemorrhage and intracranial aneurysm surgery in elderly patients: an audit of 199 consecutive cases. **Br J Neurosurg 8:**23–30, 1994
45. Paré L, Delfino R, Leblanc R: The relationship of ventricular drainage to aneurysmal rebleeding. **J Neurosurg 76:**422–427, 1992
46. Pasqualin A, Bazzan A, Cavazzani P, et al: Intracranial hematomas following aneurysmal rupture: experience with 309 cases. **Surg Neurol 25:**6–17, 1986
47. Petruk KC, West M, Mohr G, et al: Nimodipine treatment in poor-grade aneurysm patients. Results of a multicenter double-blind placebo-controlled trial. **J Neurosurg 68:**505–517, 1988
48. Raimondi AJ, Torres H: Acute hydrocephalus as a complication of subarachnoid hemorrhage. **Surg Neurol 1:**23–26, 1973

49. Rajshekhar V, Harbaugh RE: Results of routine ventriculostomy with external ventricular drainage for acute hydrocephalus following subarachnoid haemorrhage. **Acta Neurochir 115:** 8–14, 1992
50. Rosenørn J, Eskesen V, Schmidt K, et al: Clinical features and outcome in 1076 patients with ruptured intracranial saccular aneurysms: a prospective consecutive study. **Br J Neurosurg 1:**33–45, 1987
51. Rosenørn J, Eskesen V, Schmidt K, et al: The risk of rebleeding from ruptured intracranial aneurysms. **J Neurosurg 67:** 329–332, 1987
52. Säveland H, Hillman J, Brandt L, et al: Overall outcome in aneurysmal subarachnoid hemorrhage. A prospective study from neurosurgical units in Sweden during a 1-year period. **J Neurosurg 76:**729–734, 1992
53. Schramm J, Cedzich C: Outcome and management of intraoperative aneurysm rupture. **Surg Neurol 40:**26–30, 1993
54. Seifert V, Trost HA, Stolke D: Management morbidity and mortality in grade IV and V patients with aneurysmal subarachnoid haemorrhage. **Acta Neurochir 103:**5–10, 1990
55. Seiler RW, Reulen HJ, Huber P, et al: Outcome of aneurysmal subarachnoid hemorrhage in a hospital population: a prospective study including early operation intravenous nimodipine, and transcranial Doppler ultrasound. **Neurosurgery 23:** 598–604, 1988
56. Sevrain L, Rabenhenoina C, Hattab N, et al: Les anévrismes à expression clinique grave d'emblée (grades IV et V de Hunt et Hess). Une série de 66 cas. **Neurochirurgie 36:**287–296, 1990
57. Solomon RA, Onesti ST, Klebanoff L: Relationship between the timing of aneurysm surgery and the development of delayed cerebral ischemia. **J Neurosurg 75:**56–61, 1991
58. Spetzger U, Gilsbach JM: Results of early aneurysm surgery in poor-grade patients. **Neurol Res 16:**27–30, 1994
59. Steudel WI, Reif J, Voges M: Modulated surgery in the management of ruptured intracranial aneurysm in poor grade patients. **Neurol Res 16:**49–53, 1994
60. Sundt TM Jr, Kobayashi S, Fode NC, et al: Results and complications of surgical management of 809 intracranial aneurysms in 722 cases. Related and unrelated to grade of patient, type of aneurysm, and timing of surgery. **J Neurosurg 56:** 753–765, 1982
61. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. A practical scale. **Lancet 2:**81–84, 1974
62. Testa C, Andreoli A, Arista A, et al: Overall results in 304 consecutive patients with acute spontaneous subarachnoid hemorrhage. **Surg Neurol 24:**377–385, 1985
63. Ungersböck K, Böcher-Schwarz H, Ulrich P, et al: Aneurysm surgery of patients in poor grade condition. Indications and experience. **Neurol Res 16:**31–34, 1994
64. Van Gijn J, Hijdra A, Wijdicks EFM, et al: Acute hydrocephalus after aneurysmal subarachnoid hemorrhage. **J Neurosurg 63:**355–362, 1985
65. Wheelock B, Weir B, Watts R, et al: Timing of surgery for intracerebral hematomas due to aneurysm rupture. **J Neurosurg 58:**476–481, 1983
66. Yano T, Inagawa T, Kamiya K, et al: Comparative study of aged patients with ruptured intracranial aneurysms. **Jpn J Stroke 8:**231–236, 1986

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