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Patients in Poor Neurological Condition after Subarachnoid Hemorrhage: Early Management and Long-Term Outcome

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Summary

We report management and outcome data on 118 patients that presented to our emergency room over a 4 year interval (1990–1994) in poor neurological condition after subarachnoid hemorrhage. All patients were treated following a strict protocol. After initial evaluation, patients underwent a head computerized tomography (CT) scan to try to understand the mechanism of coma. If CT did not show destruction of vital brain areas, a ventriculostomy was inserted and ICP measured. If ICP was less than 20 mm Hg, or if standard treatment of increased ICP was able to lower the ICP to a value less than 20 mmHg, patients were evaluated with cerebral angiogram to determine the location of the ruptured aneurysm. The lesion was then treated by craniotomy for aneurysm clipping or endovascular obliteration. Postoperative monitoring for vasospasm with clinical exam and transcranial doppler studies was performed routinely. If vasospasm developed, this was managed aggressively with hypertensive, hypervolemic and hemodilutional therapy and, at times, endovascular treatment with angioplasty or papaverine. Outcome was measured at 1 year or more after treatment. Among patients who met criteria for aneurysm treatment, 47% had excellent or good neurologic outcome. There was a 30% mortality rate in these patients. In patients with high ICP, poor brainstem function or destruction of vital brain areas on CT, comfort measures only were offered and almost all died. It is concluded that an approach of early aneurysm obliteration and aggressive medical and endovascular management of vasospasm is warranted in patients in poor neurological conditions after subarachnoid hemorrhage.

Keywords: Aneurysm; subarachnoid hemorrhage; coma; outcome.

Introduction

Early surgery for patients in good neurologic condition after subarachnoid hemorrhage (Hunt and Hess grade I–III) [22] is a generally accepted treatment worldwide that has led to the improvement of perioperative management and subsequently to a satisfying outcome in a large majority of patients [31]. The results of substantial series of aneurysm patients in Hunt and Hess neurological grades I–III lie in the 70 to 90% good neurological recovery range, with a mortality rate from 1.7 to 8% [1, 3, 14, 31, 32, 51].

On the other hand, patients in poor neurological condition (Hunt and Hess grade IV and V) after aneurysmal subarachnoid hemorrhage pose a significant therapeutic challenge. In the past there has been a fairly nihilistic approach in terms of potential for outcome of these patients [2, 21, 22, 49]. Surgery was considered technically difficult in this group of patients because of cerebral edema and associated increased ICP. In retrospect some patients suffered potentially reversible but deleterious insults such as acute hydrocephalus or resectable intracranial hematoma [25, 47, 50, 58–61]. In addition, clinical series reported prior to the routine use of early surgery may have included many patients who had two or more hemorrhages which could have been avoided with early surgery. Each hemorrhage after the first is an event known to be associated with an increase in morbidity and mortality [33].

More recently it as been shown that some poor grade aneurysm patients can achieve a good outcome with active surgical and medical treatment [5, 44]. Bailes *et al.* were among the first to propose a treatment plan of early surgery for selected patients in poor neurological condition [5]. They reported the results of 35 grade IV and V patients chosen for active treatment with a mortality rate of 23% and a good outcome in 54%. Following this report, other groups have reported similar outcomes of poor grade patients

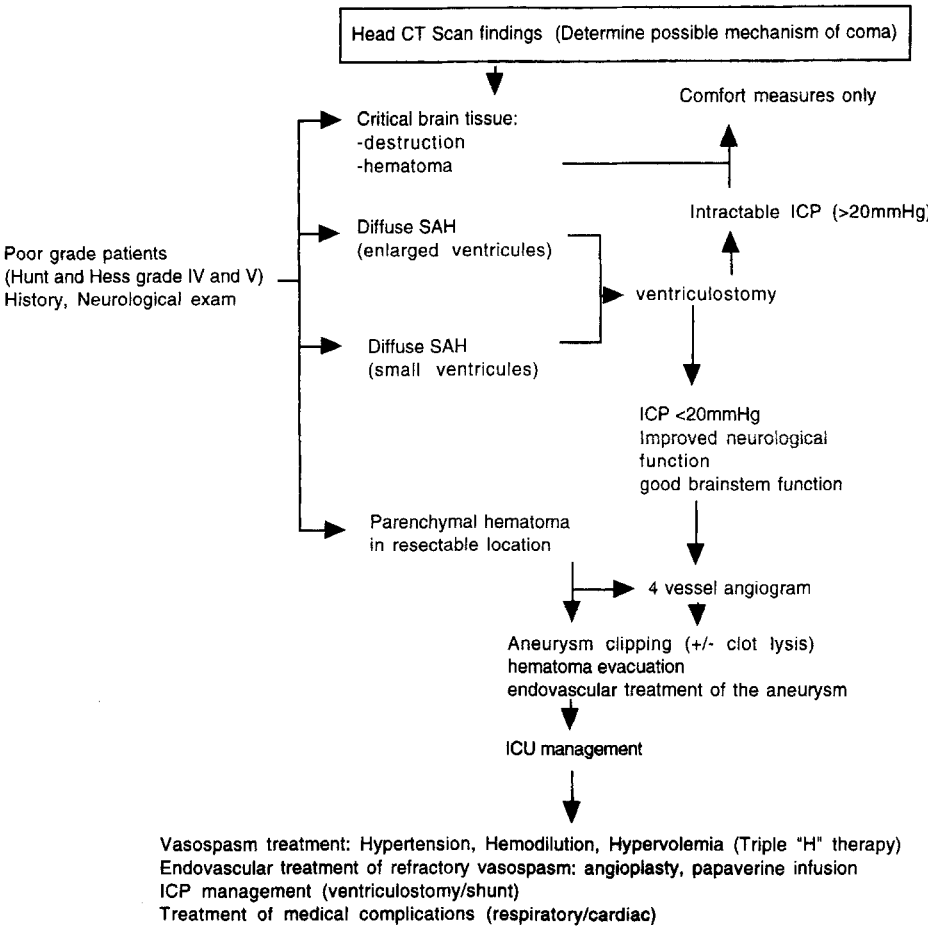

Fig. 1. Management algorithm for patients in poor neurologic condition. The CT scan findings were used to determine the proposed mechanism of coma andto direct subsequent treatment

[44, 53, 64]. These improved outcomes were obtained by combing early surgery with intensive medical management of vasospasm and elevated ICP. The follow-up of this group of patients seemed to indicate that early surgery in poor grade patients did nor result in a greater number of poor outcomes but rather, increased the overall number of good survivors.

We now report our experience with patients in poor neurological condition after subarachnoid hemorrhage. In our evaluation of these patients we have taken an approach where we attempt to identify the underlying mechanism of coma. If there is a reversible cause of coma, then patients are treated early for their aneurysm with either surgical or endovascular techniques and subsequently managed aggressively in the intensive care unit. Results and long-term (1 year or more) outcomes using this strategy are presented.

Methods and Patients

Over a four year interval, from 1990 to 1994, we managed 118 patients who were Hunt and Hess grade IV and V (in stupor or coma) at the time of aneurysm treatment. Patients who improved to Hunt and Hess grade III, II or I after ventricular drainage were excluded from this analysis. All patients had a CT scan, performed within an hour of triage in the emergency room or immediately reviewed if the patient was transferred from an outside facility, documenting SAH. After review of the patient's clinical condition and after having obtained a CT scan, some attempt was made to define the overall mechanism of induced poor clinical condition.

After the initial neurologic evaluation an arterial blood pressure monitoring catheter and a venous central line were inserted. Blood pressure was controlled aggressively and anticonvulsant medication given. Oral or nasotracheal intubation was carried out as needed. All patients except those who showed CT scan evidence of destruction of critical areas such as left frontotemporal lobe, thalamus, midbrain or pons had a ventriculostomy placed in order to measure the intracranial pressure. In patients with significant tissue destruction, no further aggressive treatment was recommended. Comfort measures were offered and patients were allowed to expire or meet formal brain death criteria.

If after placement of the ventriculostomy the ICP could be controlled below 20 cm of water and if the patients condition was stable or improved, angiography was performed. Following definition of the aneurysm location and size, the lesion was treated operatively or by endovascular techniques within 2 to 24 hours from admission. If the patient's poor clinical condition was judged to be the result of a surgically resectable intraparenchymal hemorrhage or intraventricular hemorrhage, our general principle was to pro-

ceed with surgery to clip the aneurysm and remove the hematoma without further delay. In 3 cases, once the aneurysm had been clipped, intracisternal TPA was used.

Following aneurysmal obliteration, patients were managed in the neurosurgical intensive care unit for intensive postoperative monitoring. Patients were routinely maintained with mild hypertension, hypervolemia and hemodilution in anticipation of cerebral vasospasm. Daily Dual Trranscranial Doppler (TCDs) measurements and clinical observations were used to detect the development of vasospasm. Once symptomatic vasospasm was detected by TCDs and/or clinical exam, aggressive treatment with hypertension, hypervolemia and hemodilution was immediately instituted using intravenous beta-adrenergic or alpha agonists (dopamine or neosynephrine), colloid solutions (5% albumin) and if needed, phlebotomy to maintain the hematocrit between 30 and 32%. If an ischemic deficit appeared or progressed despite maximal medical therapy, intraarterial papaverine and angioplasty were used. ICP and cerebral perfusion pressure (CPP) were continously monitored and any increase of ICP or worsening of CPP was promptly treated with CSF drainage, blood pressure increase, mannitol or the institution of a barbiturata coma. Constant surveillance was maintained to evaluate the development of medical complications including cardiac, pulmonary, renal and infection [52]. Figure 1 shows our management protocol for patients in poor neurologic condition on presentation.

Once patients had passed the period of predicted vasospasm and were medically and neurologically stable, invasive monitoring was discontinued and they were transferred to a general hospital ward for further recovery. Discharge planning depended on the clinical status of the patients and included a rehabilitation unit if necessary. Outcome was graded at 3 months and subsequently up to 4 1/2 years after treatment. Long-term follow-up was obtained by personal interviews with patients and families following discharge. Of the 79 patients who underwent treatment, the median length of follow-up was 2.5 years and all 79 patients had more than one year follow-up. Outcome was assessed as follows: excellent, no neurologic deficit, returns to work or previous lifestyle; good, mild deficit, returns to premorbid work or lifestyle; fair, neurologic deficit, lives at home, unable to return to work or previous lifestyle; poor, requires nursing care in institution or at home; and dead.

Results

Of the 118 patients who were in Humt and Hess grade IV and V at the time of presentation, 69 were in grade IV and 49 were in grade V. The mean age of all patients was 57.5 (between 20 and 89 years old). Of the 69 grade IV patients, 59 were selected for evaluation with angiography and subsequent obliteration of the aneurysm. Of these patients, 56 had initial insertion of a ventriculostomy with controlled ICP. Patients that improved from grade V to grade IV are included in the analysis of grade IV patients. 48 patients were subsequently managed with surgery and 11 with endovascular techniques. Of the 49 patients in grade V condition, 15 were treated with surgery and 3 with an endovascular procedure, after having had a ventriculostomy placed and ICP controlled. No difference was found in ICP values between patients in grade IV and V. ICP values could not be predicted by the initial clinical presentation or CT findings.

Table 1. *Total Management Outcome for Poor Grade Aneurysm Patients Treated Over a Five Year Interval with Follow-up 1 Year or More*

	n	%
Excellent	27	22.8
Good	10	8.5
Fair	11	9.3
Poor	8	6.8
Dead	62	52.5
Total	118	100

Table 2. *Total Management Outcome in 118 Patients Evaluated for Possible Treatment*

	Acute aneurysm obliteration and medical management		Comfort measures	
	n	%	n	%
Excellent	27[a]	34		
Good	10	13		
Fair	11	14		
Poor	7	9	1	3
Dead	24	30	38	97
Total	79	100	39	100

[a] 2 patients had a negative angiogram and were therefore treated with ventricular drainage only.

Using the outcome measures described under the methods section, the outcome at one year or more is shown in Table 1. As can be seen, 31% of patients that presented with Hunt and Hess grade IV or V had excellent (22.8%) or good (8.5%) outcomes. Death occurred in 52.5% of all the patients. More importantly among patients treated aggressively with surgery or an endovascular procedure and ICU care, 47% had excellent (34%) or good (13%) outcome and 30% died (Table 2). There was no difference in outcome for patients selected for aggressive treament that pressented in grade IV or V (Table 3). Average length of stay was 3 1/2 weeks.

Patients selected for treatment had a mean age of 54.6 (between 20 and 88 years old) and the male to female ratio was 1 : 1.5. The treated aneurysm was located in the anterior circulation in 58 patients, while

Table 3. *Outcome of Patients in Grade IV or V Clinical Condition Prior to Treatment*

Grade IV	Acute aneurysm obliteration and medical management		Comfort measures	
	n	%	n	%
Excellent	21[a]	34		
Good	8	13		
Fair	9	15		
Poor	5	8	1	13
Dead	18	30	7	88
Total	61	100	8	100
Grade V	**Acute aneurysm obliteration and medical management**		**Comfort measures**	
	n	%	n	%
Excellent	6	33		
Good	2	11		
Fair	2	11		
Poor	2	11		
Dead	6	34	31	100
Total	18	100	31	100

[a] 2 patients had a negative angiogram and were therefore treated with ventricular drainage only.

Table 4. *Aneurysm Location in Treated Patients*

	Grade IV	GradeV
Surgical Treatment		
anterior communicating	21	5
internal carotid	3	1
middle cerebral	9	4
posterior communicating	9	1
basilar tip	3	
posterior inferior cerebellar	1	2
anterior inferior cerebellar	1	1
posterior cerebral	1	
superior Cerebellar		1
Subtotal	48	15
Endovascular Treatment		
basilar tip	2	1
vertebral	2	
posterior inferior cerebellar	2	
superior cerebellar	1	
posterior communicating	3	1
Anterior communicating	1	1
Subtotal	11	3
Total	59	18

Table 5 a. *Patients with 2 or More Hemorrhages Prior to Presentation*

	n	%
Grade IV	35/69	50
Grade V	28/49	57

Table 5 b. *Incidence of Vasospasm in Patients Managed with Early Aneurysm Obliteration*

	n	%
Grade IV	41/61	67
Grade V	13/18	72

19 patients had posterior circulation lesions. Ten of the patients had multiple aneurysms (Table 4). In the treated group intraparenchymal hematoma was found in 16 patients in Hunt and Hess grade IV and in 6 patients in grade V. Intraventricular hemorrhage was seen in 32 out of 59 patients in grade IV and in 11 out of 18 patients in grade V. Obstructive hydrocephalus was present in 24 patients in grade IV and in 8 patients in grade V. In 8 patients in grade IV and 2 in grade V both obstructive hydrocephalus and significant intraparenchymal hematoma were present. Two or more subarachnoid hemorrhages were identified in 31 patients in the grade IV group (51%) and in 7 in the grade V group (38%) (Table 5). Of patients managed aggressively, vasospasm was detected (TCDs, clinical evidence or angiography) in 41 patients in grade IV (67%) and in 13 grade V patients (72%) (Table 5). Four patients were treated endovascularly for severe vasospasm.

Of the 24 patients who died after aggressive treatment was offered, 18 presented in grade IV and 6 in grade V. Cause of death included vasospasm with infarction and herniation in 11 patients (8 in grade IV and 3 in grade V), sepsis in 3 patients, renal failure in 1 patient and elective withdrawal of care in 9 patients (7 in grade IV and 2 in grade V) in a persistent vegetative state (Table 6).

Nearly 100% of patients not chosen for aggressive treatment died, there was one survivor that was discharged in poor neurological condition. The mean age of patients treated with comfort care only was 60.5 years (between 33 and 89 years old). 18 patients did not meet criteria for angiogram after admission.

Table 6. *Causes of Death in Patients Who Underwent Treatment*

Grade IV	18/61
Vasospasm/infarction	8
Sepsis	2
Renal failure	1
Vegetative state; late withdrawal of care	7
Grade V	**6/18**
Vasospasm/infarction	3
Sepsis	1
Vegetative state; late withdrawal of care	2

Table 7. *Reason for Comfort Measures Early in Management (No Treatment of the Aneurysm)*

	Grade IV
ICP greater than 20	4
Poor brainstem function with no improvement after ventriculostomy	2
Both	2
Total patients	8
	Grade V
ICP greater than 20	14
Poor brainstem function with no improvement after ventriculostomy	5
Old age or poor medical conditions prior to SAH	3
Head CT showing vital brain destruction	9
Total patients	31

Angiogram was performed in 21 patients that were in the non treated group. There were 16 anterior circulation aneurysms and 5 posterior circulation lesions. Ventriculostomy only was used in 9 of the 10 patients that presented in Hunt and Hess grade IV not selected for surgical or endovascular treatment.

Two patients had no aneurysm or arteriovenous malformation demonstrated on angiography. Both of these patients were in grade IV condition and both went on to make an excellent neurological recovery. Among the other 8 patients in grade IV condition who did not have treatment of their aneurysm, 4 had ICP greater than 20 mmHg, 2 had poor or no evidence of brainstem function that did not improve after placement of a ventriculostomy and 2 had both ICP elevation and poor brainstem function. Of patients in grade V condition, 31 did not meet criteria for aggressive aneurysm treatment. In 22 of these 31 patients, ventriculostomy was inserted and no further treatment was offered. In 14 of the 22 patients were ventriculostomy was used, yet no further therapy used, ICP was non controllable (between 30 and 100 mmHg) and did not improve wtih standard therapy. In five patients, there was no clinical evidence of neurological function and no improvement with ventricular drainage. Of these five patients, one had 3 generalized tonic-clonic seizures associated with the initial hemorrhage and another patient had a cardiac arrest at the time of his bleed. Despite immediate CPR, the patient presented to the emergency room with no cerebral function. In 3 patients it was decided not to offer any aggressive therapy based on age of patient, general poor medical condition prior to hemorrhage or preexisting wishes of the patient in the form of a living will. In 9 patients, ventriculostomy was not used. These patients had head CT findings consistent with catastrophic brain destruction and therefore no further treatment other then comfort care was offered (Table 7).

Discussion

In 1990 Bailes *et al.* were one of the first groups to propose a treatment plan of early surgery for selected patients in poor neurological condition after SAH [5]. They reported the outcome results of 35 grade IV and V patients selected for active treatment with a mortality rate of 23% and a good outcome in 54%. These results were later duplicated by other groups [44]. Our own data is similar in that the patients who were chosen for surgery or endovascular treatment had a mortality rate of 30% with a good outcome achieved in 47%. It is important to note in these observations that a patient's clinical condition was evaluated immediately prior to treatment so that patients that improved to a good Hunt and Hess grade (grade III, II or I) after placement of a ventriculostomy were excluded from the present analysis.

In our evaluation of patients with subarachnoid hemorrhage in Hunt and Hess grades IV and V, we have taken an approach of trying to identify the underlying mechanism of coma based on history, neurologic examination and findings on the initial head CT scan. The CT scan may the best way to evaluate the possible etiology of coma and therefore be used to guide the therapeuric approach. If there is a presumed

reversible cause of coma, then patients are treated early for their aneurysm with either surgical or endovascular techniques. Substently, patients are managed in the intensive care unit with early detection and treatment of vasospasm as well as complete respiratory and cardiovascular support.

The possible mechanisms of coma after SAH are multiple and frequently several mechanisms may be present in a single patient. One of the most frequent presumed causes of coma after SAH is persistent increased ICP. The sudden rupture of an intracerebral vessel with introduction of blood into the subarachnoid space has been shown to increase ICP [62, 63]. This may result in reduction of cerebral perfusion pressure with a reduction of cerebral blood flow and, over time, to the development of cerebral edema. Diminished cerebral perfusion pressure has been documented in the acute phase (seconds to minutes) of SAH in patients with increased ICP [30, 36, 40]. It has also been shown that cerebral blood flow on any single day after SAH is lower in patients in poor grade when compared with good grade patients [36]. In many of our patients there was mild or no ventricular enlargement on CT. Uncontrollable ICP was discovered upon insertion of a ventriculostomy. We believe that in poor grade patients, increased ICP is present to some degree and should be measured. If possible this elevated ICP should be treated early in the overall management of the patient by placing a ventriculostomy. Intraparenchymal hemorrhage with resultant increase in ICP and mass effect may occur as a complication of aneurysmal rupture and be the cause of coma. In theory the prognosis of patients with a ruptured aneurysm and large intracerebral hematoma may be affected by early hematoma evacuation as shown by isolated case reports [4, 8]. If a hemorrhage has occurred in an area of the brain where clot removal would not be expected to create significant neurologic deficit, such as the frontal of temporal lobes, emergent removal of the hematoma combined with obliteration of the aneurysm should be performed in an attempt to improve the outcome. As seen in the Results section, 30% of our patients presented with intraparenchymal hematoma and rapid evacuation in association with aneurysmal clipping was felt to contribute to a better finale outcome.

Acute hydrocephalus is a known complication of SAH and a frequent cause of coma. Dramatic improvement of consciousness has been observed following immediate drainage of cerebrospinal fluid [28, 46]. Commonly, acute hydrocephalus may develop as a result of dense blood in the basal cisterns or it may be due to direct intraventricular hemorrhage with interference of CSF pathways at the foramen of Monroe or at the level of the fourth ventricle with obstruction of the foramen of Luschka and Magendie. The incidence of acute hydrocephalus following SAH has been reported to be from 12% to 63% [6, 32, 37, 65]. Acute hydrocephalus may present as relatively abrupt onset of lethargy, stupor or coma occurring after the initial rupture and is often seen on the admission cranial CT scan in patients in poor neurologic condition. Urgent placement of unilateral or bilateral ventriculostomies can be beneficial in reversing the process and in rapidly improving the neurological condition of the patient. In the present series, patients were assigned a clinical grade immediately prior to endovascular or surgical treatment and after ventricular drainage (if used).

A very important cause of poor neurologic condition after an initial subarachnoid hemorrhage is a subsequent aneurysmal hemorrhage. Aneurysmal rebleeding is a devastating complication of SAH. A second hemorrhage increases mortality to approximately 70% [33]. In the Cooperative Aneurysm Study, rebleeding was maximal (4%) in the first 24 hours after SAH, and thereafter was approximately 1.2% per day over the next 2 weeks [26]. As many as 20% of patients who do not have surgical treatment rebleed in the first 2 weeks, 50% within 6 months, and thereafter 3% per year [66]. Rehemorrhage carries a significantly higher morbidity and mortality then the initial bleed [27]. In our experience rehemorrhage is a common event in patients presenting in poor neurologic condition with 48% of patients presenting with a second or more hemorrhage.

Other causes of coma following subarachnoid hemorrhage may be due to secondary insults such hypoxia, hypotension, seizures and cardiac aythmias. These events may be difficult to document given the fact they can occur in the first few minutes or hours after hemorrhage. In additon, what is initially described as a "seizure" may be the neurological changes observed with hemorrhage like patient collapse. These events do not represent true epileptiform activity. In our series we were able to document true seizure activity in two patients. Following SAH, seizures are reported to occur with an incidence of 4% to 25% [9, 17, 20, 43, 56]. Seizure can occur immediately after the initial hemorrhage (within seconds) and explain a transient loss of consiousness (post ictal phase) or in a later stage. SAH is also frequently

accompanied by electrocardiographic disturbances, including ventricular and supraventricular arrhythmias, prolongation of the QT interval and inverted T waves. Arrhythmias occur in up to 90% of patients and are most common during the first 48 hours following SAH [12, 34]. The sudden loss of consciousness that can occur at the onset of the SAH may in part be due to an acute arrhythmia. This mechanism could well produce an additional event of transient global brain ischemia with resultant poor tissue perfusion.

In conclusion, based on our experience as well as others, it appears that there is a population of patients who are in poor neurologic condition prior to treatment who have the potential to make a meaningful recovery by one year. The hospitalizations for these patients are often long and recovery is slow. If identifiable mechanisms of coma can be recognized, however, there is the possibility that early treatment of the aneurysm and aggressive ICU management of the consequences of subarachnoid hemorrhage may lead to recovery. We believe that our protocol of aggressive management based on clinical evaluation combined with the head CT appearance and ICP management will select patients with the greatest potential for good recovery regardless of the initial neurological condition. Although other groups have used the criteria of an ICP of 30 mmHg of water to proceed with angiography and subsequent craniotomy [5], we have chosen 20 mmHg of water as the cutoff to proceed with further treatment. The few patients we have tried to manage with initial ICPs between 20 and 30 cm of water have done poorly or have died.

We conclude that an aggressive approach based on selection criteria is warranted in poor grade patients. Clearly others factors such as age, general medical condition, aneurysm size and location may influence the final outcome [10]. Such variables must be considered in conjunction with the entire clinical situation, including patients pre-existing wishes (living will) or their family members wishes when making the final decision regarding aggressive surgical and ICU management of subarachnoid hemorrhage patients who present in poor clinical condition.

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Comments

This manuscript provides some guidelines for management of patients who come into the hospital with subarachnoid hemorrhage that are below Grade III, Hunt and Hess. Many neurosurgeons, particularly in the United States, don't operate on patients in that condition because they assume the outcome will be poor. This paper provides some rationale for an aggressive approach to all patients except those who are moribund. This study comes from a good institution by reliable investigators. The strength of the paper is the criteria used for surgery in patients who often are denied that treatment based on a relatively nonobjective view.

J. T. Hoff

This is a well-written paper that addresses an important therapeutic problem for all neurosurgeons and for vascular neurosurgeons in particular. This same topic has recently been discussed by other groups (see J Neurosurg 85: 39–49, 1996).

The authors reviewed 118 patients who presented in poor neurological condition after subarachnoid hemorrhage. Patients were considered eligible for aggressive management based upon clinical evaluation, computed tomography (CT) studies, and intracranial pressure (ICP).

The information presented here is very significant.

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