0148-396X/88/2306-0699$02.00/0
NEUROSURGERY
Vol. 23, No. 6, 1988
Printed in U.S.A.

Experimental and clinical reports

Early Aneurysm Surgery and Prophylactic Hypervolemic Hypertensive Therapy for the Treatment of Aneurysmal Subarachnoid Hemorrhage

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The prevailing sentiment of North American neurosurgeons is that there is no significant difference in overall morbidity between patients who are treated with early aneurysm surgery and those who are treated with delayed aneurysm surgery. This concept is based primarily on the high incidence of ischemic events after early intervention. Recent experience, however, indicates that prophylactic hypervolemic hypertensive therapy may be beneficial in reducing delayed ischemia after early aneurysm surgery. During the preceding 21 months, we have performed 125 operations for intracranial aneurysms. Fifty-six patients in this group presented less than 7 days after subarachnoid hemorrhage (SAH) (47 within 3 days) and were treated by a prospective protocol of urgent aneurysm surgery performed within 24 hours after presentation. In all cases, the aneurysm was clipped with the use of mannitol and spinal drainage for brain relaxation. All patients were then treated with prophylactic volume expansion therapy and induced hypertension with a central venous pressure or a Swan-Ganz catheter until the 14th day after SAH. Preoperatively, 17 patients were Hunt and Hess Grade I, 9 were Grade II, 28 were Grade III, and 2 were Grade IV. In this group of 56 patients at risk for delayed ischemia from vasospasm, 5 patients had significant intraoperative complications. Ten patients (18%) had delayed cerebral ischemia, totally reversible in 6 cases, with small infarcts in 3 cases, and with 1 death (2% mortality from delayed ischemia), there were 5 cases of shunted hydrocephalus, and 3 deaths from other complications. Overall, 41 patients (73%) returned to their premorbid occupations without neurological deficit. Four patients (7%) are independent with no neurological deficits, but have not returned to full-time employment. Four patients (7%) are independent, but have permanent deficits. Three patients (5%) are dependent on others for care, and 4 patients (7%) died. These data imply that delayed cerebral ischemia after SAH can be effectively minimized with prophylactic volume expansion therapy. Similar results have been reported for patients treated with calcium channel blocking agents. Given these techniques, perhaps the assumption that early operative intervention holds no advantage over delayed surgical treatment of an aneurysm rupture should be readdressed in a scientifically controlled fashion. (*Neurosurgery* 23:699–704, 1988)

Key words: Cerebral aneurysm, Delayed cerebral ischemia, Early aneurysm surgery, Subarachnoid hemorrhage, Timimg of aneurysm surgery, Vasospasm, Volume expansion therapy

The proper treatment of patients with a ruptured intracranial aneurysm continues to be an area of intense controversy (1, 47). The central issues seem to be the timing of surgical intervention and the treatment of delayed cerebral ischemia. Recently, a multicenter cooperative study on the timing of aneurysm surgery was completed, and the results have been presented in various formats (1, 9, 12). The outcome of the study indicates no relative benefit for patients with operations planned during the acute phases after subarachnoid hemorrhage (SAH) vs. those patients with operations planned during the delayed period. Rebleeding of the aneurysm accounted for morbidity in 6% of patients selected for early operation, and there was an additional 14% incidence of death or disability from delayed cerebral ischemia in this group of patients. These data indicate that 20% of patients selected for early operation experienced morbidity that can potentially be prevented.

Since the cooperative aneurysm study, several newer techniques have been investigated for the treatment of patients with aneurysmal SAH (55). Preliminary data suggested that ischemic complications and recidivous hemorrhage could be minimized after aneurysm rupture by urgent surgical intervention and prophylactic volume expansion (48). Further experience with these techniques has produced results that may indicate that early operative intervention has substantial merit compared with delayed aneurysm surgery, and perhaps this issue should be reopened.

SUBJECTS AND METHODS

Between July 1986 and March 1988, 125 operations were performed on patients with intracranial aneurysms. Because of the nature of our referral practice, the majority of these patients were not seen during the acute phases of SAH; however, 56 patients had bled within 7 days before admission to the Neurological Institute, and this group forms the basis of the present report. These patients were studied because they represented the group with the highest potential for morbidity from rebleeding and delayed ischemia.

All patients admitted within the 1st week after aneurysmal SAH are handled by standard protocol. Appropriate diagnostic testing is performed in an emergency fashion in all patients with the suspected diagnosis of ruptured intracranial aneurysm. Once the diagnosis has been confirmed and angiography has been completed, patients are taken to the operating room for definitive clipping of the aneurysm within 24 hours of admission. Before operative intervention, patients are monitored in the Neurological Intensive Care Unit, loaded with dexamethasone and phenytoin, and given maintenance doses of intravenous crystalloid solutions. Then a central venous catheter or a Swan-Ganz catheter is placed in all patients. The choice between these two monitoring devices is based on the patient's underlying hemodynamic status and age. Surgery is performed with maximal brain relaxation techniques. These methods include hyperventilation, lumbar spinal drainage or ventricular drainage of cerebrospinal fluid, and intravenous mannitol (1–2 g/kg before brain retraction). These techniques have been adequate to produce a slack brain that can be retracted easily. Standard microsurgical techniques were utilized for direct clipping of the aneurysm in all cases.

After the operation, all patients were treated with aggressive prophylactic volume expansion therapy and induced hypertension. Central venous pressure was artificially elevated to 10 to 12 cm H_2O with a combination of high volume crystalloid and periodic boluses of 5% albumin. When a Swan-Ganz catheter was in place, the pulmonary artery wedge pressure was maintained between 12 and 16 cm H_2O with similar intravenous infusions. Of course, the management of each patient was individualized depending on hemodynamic parameters, but the goal was to maintain all patients in a hypervolemic hypertensive state. If blood pressure did not spontaneously elevate to at least 10 mm Hg higher than the premorbid systolic pressures, pressor agents were utilized, including dopamine or phenylephrine hydrochloride as indicated.

In most instances, postoperative angiography was performed within 7 days after the operation. Patients were maintained on hypervolemic hypertensive therapy for a minimum of 10 days after the initial SAH and up to 14 days after SAH if angiographic vasospasm was observed on postoperative study. Patients who became symptomatic with delayed cerebral ischemia were treated with even more aggressive measures of increased volume and hypertension. In these cases, blood volume and hypertension were maximized until the neurological deficit resolved or it became evident that the deficit was fixed.

Before the operation, all patients were graded according to a modified Hunt and Hess grading scale (18). Each patient underwent daily neurological examinations, and more frequent assessments were made in patients with fluctuating signs. At 3 months, the patients were evaluated for outcome based on their ability to regain their premorbid occupations and live independently.

RESULTS

Fifty-six patients were entered into this protocol during a 21-month period. The aneurysms were distributed throughout the intracranial circulation, as shown in Table 1. Seventeen patients were Grade I, 9 patients were Grade II, 28 patients were Grade III, and 2 patients were Grade IV before operative intervention. Eighteen patients were admitted on the day of hemorrhage, 29 on Days 1 through 2 after SAH, and 9 on Days 3 through 6. One patient was operated on 5 days after admission (as described in the next section), and the other 55 patients were operated on within 24 hours of admission.

TABLE 1
Location of Intracranial Aneurysms in Early Operated Patients

Posterior communicating	22
Anterior communicating	10
Middle cerebral	8
Pericallosal	5
Carotid bifurcation	4
Anterior choroidal	2
Ophthalmic	2
Basilar apex	2
Posterior cerebral	1
Total	56

Surgical complications

The complications observed in this group of patients have been most instructive. One patient admitted on the day of SAH had an initial angiogram that did not demonstrate the aneurysm. Subsequent angiography, 3 days later, revealed a middle cerebral artery aneurysm; however, a series of nonmedical incidents caused the patient's operation to be delayed for 2 additional days. Hours before the planned operation, the aneurysm reruptured. Even though operation was performed in this acute setting, the patient fared poorly and died from the preoperative intracerebral hemorrhage.

One patient who had severe aortic stenosis was fluid-overloaded intraoperatively and had a respiratory arrest during the immediate postoperative period. Although he was successfully resuscitated, he eventually succumbed to medical complications. Postmortem examination showed that his aneurysm had been incompletely occluded.

The other two surgical complications involve the inadvertent occlusion of a significant intracranial artery. In a case of a large anterior communicating artery aneurysm, an intraoperative rupture resulted in the aneurysm clip being placed across one of the A_2 segments. This clip placement caused infarction in an anterior cerebral distribution. The patient, however, has made an excellent functional recovery. In another patient, the anterior choroidal artery was inadvertently occluded during a routine posterior communicating aneurysm operation, resulting in a capsular infarction and a contralateral hemiplegia. After the operation, a duodenal ulcer ruptured and required an emergency laparotomy. This patient eventually recovered from the medical complications and is recovering strength in the affected extremities.

Nonsurgical complications

The other complications in this series are not surgical. There were 10 cases (18%) of delayed cerebral ischemia. Six patients had transient neurological dysfunction related to vasospasm that resolved with more aggressive volume expansion and induced hypertension. Four patients developed computed tomography-documented infarctions as a result of delayed ischemia. Two of these patients have returned to their premorbid functional levels, one patient is disabled from bilateral frontal infarction, and one patient died from massive diffuse cerebral infarction (Table 2).

A 77-year-old woman with severe atherosclerotic disease suffered infarction in the posterior cerebral artery distribution, presumably related to uncal herniation on the day of SAH. She was successfully resuscitated and lives at home with a mild deficit.

There were five cases of postoperative hydrocephalus that required permanent ventriculoperitoneal shunts. All of these patients made a satisfactory recovery and have returned to premorbid functional levels.

TABLE 2
Delayed Ischemia Related to Vasospasm in 56 Early Operated Patients

Reversible deficits, no infarction	6 (~11%)
Infarction with minimal deficit	2 (~4%)
Infarction with significant deficit	1 (~2%)
Diffuse infarction and death	1 (~2%)
Total	10 (18%)

TABLE 3
Outcome in 56 Patients after SAH and Early Aneurysm Surgery[a]

Good outcome	
Return to premorbid occupation	41 (~73%)
Neurologically normal, not returned to premorbid occupation	4 (~7%)
Independent, mild neurological deficit	4 (~7%)
Total	49 (87.5%)
Poor outcome	
Dependent, significant deficit	3 (~5%)
Dead	4 (~7%)
Total	7 (12.5%)

[a] Follow-up of 1 to 21 months, mean of 8.2 months.

One complication was related to a central line catheter that inexplicably became disconnected. At the time, the patient was neurologically normal, but she bled through the central line catheter, producing hypotension, cardiac arrest, and death.

Outcome

Three-month follow-up data are available on 47 patients. At the time of this writing, the other 9 patients have been recovering for only 1 to 3 months after operation. Overall, excellent outcome and return to premorbid occupation without neurological deficit were observed in 41 patients (73%). Four patients (~7%) have not as yet returned to full employment, but are alive without neurological deficit. An additional 4 patients (~7%) are not able to return to their premorbid occupations, but live independently with mild neurological deficits. Therefore, 88% of the patients have had a satisfactory outcome, 3 patients (~5%) are alive but are significantly impaired, and 4 patients (~7%) are dead. These results are summarized in Table 3.

DISCUSSION

Fears about the technical difficulty of aneurysm surgery during the acute phases after SAH, especially in patients in poor neurological condition, have led to the prevailing practice of delayed aneurysm surgery in North America (1, 7, 12, 17, 21, 22, 26, 35, 36, 50, 51). Numerous reports from Europe, Japan, the U.S., and Canada, however, have documented large series of aneurysm patients operated on successfully during the acute phases after SAH (6, 9, 10, 17, 19, 20, 28, 29, 31, 33, 48, 52, 53). These studies, coupled with the results of the International Cooperative Study for the Timing of Aneurysm Surgery (1, 9, 12), indicate that there is no significant increase in surgical complications in patients operated on acutely after aneurysm rupture compared with delayed operation. With proper brain relaxation techniques, brain retraction in patients not in coma before operation is no more difficult than during the delayed period. It has certainly been our experience that early operative intervention is at worst no more difficult than delayed operation and may even facilitate aneurysm dissection.

The second misconception that has hindered the widespread acceptance of early aneurysm surgery is that acute surgical intervention in the face of a recently ruptured aneurysm greatly increases the risk of delayed cerebral ischemia. In fact, at neurosurgical centers where considerable experience has now been gained with the use of early aneurysm surgery, delayed cerebral ischemia in the early operated group seems to be no more frequent than the historical incidence of this complication (6, 9, 31). Again, the International Cooperative Study on the Timing of Aneurysm Surgery indicated that the incidence of delayed ischemic events leading to morbidity and mortality was exactly the same in patients with operations planned for the early period and patients with operations planned for the delayed period (12). Some reports that early aneurysm surgery might in itself reduce the incidence of delayed cerebral ischemia seem to be overly optimistic (33, 53). The successful completion of aneurysm repair allows effective prophylactic and therapeutic measures to be instituted to treat cerebral ischemia (48).

Volume expansion therapy utilized in a prophylactic fashion in this series seems to be successful in minimizing the risks of delayed ischemic events. Although this assumption has not been proven scientifically, the incidence of morbidity from delayed ischemic events in this study is at least as low as those reported in series of patients treated with calcium channel-blocking agents (2, 4, 7, 13, 30, 39, 45). The incidence of ischemia resulting in morbidity and mortality with either of these two techniques is far lower than that documented for untreated patients in numerous large series (11, 15, 16, 22, 24, 29, 32, 38, 40, 41, 44, 50, 51, 56). There does seem to be a real effect, and it may be that both approaches accomplish cerebral protection by similar mechanisms.

Although the exact mechanism of action of the calcium channel-blocking agents is unknown, it seems likely that the beneficial effects of the calcium channel-blocking agents are not related to reversal or prevention of cerebral vasospasm or angiographic narrowing of the major cerebral blood vessels (2, 3, 37, 39). It does seem likely, however, that the calcium channel-blocking agents have other possible modes of action. One mechanism may be the prevention of calcium influx into ischemic neurons and thereby the prevention of cell death in marginally ischemic zones (46).

A second beneficial effect of calcium channel-blocking agents may be their systemic vasodilatory properties. This phenomenon has required that patients treated with calcium channel-blocking agents be prophylactically volume-expanded and carefully monitored to prevent the development of decreased cardiac output and hypotension (2, 39). Decreased blood volume has been causally related to the development of delayed cerebral ischemia (49). A third possible action of these drugs may be the dilation of pial arteriolar collaterals (5, 14, 27) and inhibition of platelet aggregation (8), thereby improving blood flow in ischemic zones.

Although hypervolemic hypertensive therapy has no chemical neuronal protective effect, this therapy can be expected to augment intravascular volume, increase cardiac output, improve the rheology of blood flow in the microcirculation, and enhance collateralization (3, 25, 34, 42, 43, 47, 54). Therefore, calcium channel-blocking agents and prophylactic volume expansion may be expected to afford benefit to the same group of patients, i.e., those with mild to moderate cerebral vasospasm. Patients with severe and widespread vasospasm causing profound flow deficits in multiple vascular territories will probably not be helped by either form of therapy.

Certainly, as noted in this report and in the reports on calcium channel-blocking agents, patients continue to have ischemic difficulties despite the adequate institution of prophylactic therapy. A cooperative study is currently under way to determine the effectiveness of calcium channel-blocking agents in the treatment of delayed cerebral ischemia (13). It is likely that the design of the study will indicate whether there is any relative benefit to calcium channel-blocking agents over and above the benefits that can be derived simply from prophylactic volume expansion therapy.

Most importantly, there may currently be effective means to diminish the consequences and severity of delayed cerebral ischemia. Therefore, if delayed cerebral ischemia is minimized and rebleeding from the aneurysm is essentially eliminated as a complication by the use of urgent aneurysm surgery, then the theoretical advantage of early aneurysm surgery over delayed aneurysm surgery should become obvious. About 88% of patients who presented to our institution without coma after SAH eventually had a good outcome, and about 73% of patients returned to their premorbid occupations without neurological deficit. None of the complications in this group of patients can be related to the technique of early aneurysm surgery. Although these results are compelling, they cannot be accepted as substantiation of our bias toward early surgery and prophylactic volume expansion. These data do, however, suggest the potential value of renewed efforts to evaluate critically the relative merits of early vs. delayed aneurysm surgery in light of currently available techniques to minimize delayed cerebral ischemia.

Received for publication, May 17, 1988; accepted, June 28, 1988.
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COMMENTS

Solomon and coworkers have added yet another excellent contribution to the growing literature documenting the potential benefits of early surgery for ruptured intracranial aneurysms. After an aneurysmal subarachnoid hemorrhage, there are two major sources of morbidity and mortality facing the patient and concerning the surgeon: rebleeding and delayed ischemia secondary to cerebral vasospasm. The incidence of rebleeding is greatest immediately after the initial hemorrhage, and the incidence of vasospasm is highest between the 5th and 9th days after the ictus. It thus seems logical to proceed with early surgery. Such an approach is certainly the best means for eliminating rebleeding as a source of morbidity and mortality. Early surgery also facilitates the management of cerebral vasospasm by volume expansion and induced hypertension, maneuvers that are quite risky in the patient with an untreated aneurysm. The more widespread acceptance of early aneurysm surgery requires documentation that such an approach is not associated with an unacceptable increase in operative morbidity or mortality.

Unlike the authors, it is my belief that early aneurysm surgery is a technically more challenging procedure than delayed surgery. Despite the use of osmotic agents, hyperventilation, and cerebrospinal fluid drainage, it is my opinion that the brain is more friable and difficult to retract safely. Dissection must be performed through tenuous clot with some loss of anatomical detail and perhaps a greater likelihood of premature intraoperative rupture. Furthermore, after a subarachnoid hemorrhage, the autoregulatory mechanisms of the brain are altered, potentially increasing the risk of ischemic sequelae resulting from brain retraction and the induced hypotension often used for safer aneurysm dissection. I question whether the inadvertent occlusion of intracranial vessels during clip placement in two cases in the present series was related to the technique of early surgery. The authors think not.

Despite these distractors, there is a rapidly accumulating literature documenting a management morbidity equaling and often surpassing that achieved with delayed surgery. Sonesson et al. have recently shown that the psychological outcome in patients operated early or late is without significant differences (1). The results reported by Solomon et al. certainly support their contention that early aneurysm surgery can be performed with a significant degree of safety.

Although we are presently operating on the large majority of our aneurysmal subarachnoid hemorrhage patients early, this important decision is made on an individual basis rather than by following a strict protocol that does not take into account the large number of variables in these critically ill patients. Despite my personal bias toward the virtues of early surgery, I think that there are certain individuals who benefit from a delayed operation. Factors taken into consideration in the timing of surgery include but are not limited to: patient's neurological condition, medical condition, degree of blood in the subarachnoid cisterns, location of the aneurysm, and day of admission after subarachnoid hemorrhage. Of great importance is the individual surgeon's experience with the management of aneurysms. Whether the majority of neurosurgeons can achieve the excellent results with early surgery reported by such experienced surgeons as the present authors remains to be seen.

I entirely agree with these authors that their superb results should further stimulate interest in evaluating the merits of early vs. delayed aneurysm surgery.

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The incidence of delayed ischemia and the good results from the management of delayed ischemia in this study are certainly as good as in many series currently being reported with the use of calcium channel blockers.

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