Considerations in early surgery on good-risk patients with ruptured intracranial aneurysms

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A retrospective analysis of 100 consecutive patients with proven ruptured intracranial aneurysms, classified as Botterell Grades I to III on admission, was carried out to evaluate the efficacy of early operation. Surgical and management mortality/morbidity rates were lower for cases in which a single hemorrhage was operated on within 48 hours than when surgery was delayed for 7 days or more. Surgical and management mortality/morbidity rates were worse in good-risk patients treated surgically between the 3rd and 7th days following a hemorrhage, reflecting the increased incidence of postoperative vasospasm and raised intracranial pressure encountered at surgery during this interval.

KEY WORDS • ruptured aneurysm • early operation • vasospasm • mortality • morbidity

There is no uniform agreement on the optimum time of surgical intervention in good-risk patients with ruptured intracranial aneurysms. Opinions have polarized in two directions. Some surgeons have chosen to operate early to prevent a fatal rebleed. These latter authors delay surgical repair to minimize the risk of postoperative vasospasm, presumably triggered by early surgery, and to allow resolution of the friable edematous condition of the brain that would be encountered during early surgery on poor-risk patients. Yet Samson, et al., observed no difference in the incidence of intraoperative complications or postoperative morbidity when they compared early operation with late intervention in 106 consecutive good-risk patients. Whereas neurologically damaged patients are likely to improve with a policy of delayed operation augmented by strenuous protocols to minimize rebleeding and ischemia, good-risk patients will only deteriorate from a policy of delay unless the overall management with such a protocol proves superior to a course of therapy that includes early operation.

Accordingly, in a search for guidelines to address this challenge, we have conducted a retrospective study of 100 consecutive reasonable-risk patients (classified as Botterell Grades I, II, and III on admission) with ruptured intracranial aneurysms, treated in a small unit by the two authors, in close collaboration.

Clinical Material and Methods

Case Material

The series included 100 consecutive patients with proven ruptured intracranial aneurysms classified on admission as Botterell Grades I, II, and III, who were managed between 1972 and 1980. Seventy-two patients were admitted within 24 hours, 21 between 2 and 7 days, and seven more than 7 days after their most recent subarachnoid hemorrhage (SAH). The clinical characteristics of the patients resembled those seen in the Cooperative Study.

Preoperative Management

The diagnosis of SAH was confirmed by lumbar puncture, and the responsible aneurysm was then demonstrated angiographically as soon as possible, within hours of admission (Table 1). Patients were uniformly managed with a protocol of bed rest in an acute-observation area. Mild sedation was provided with barbiturates (pentobarbital, 50 to 100 mg; phenobarbital, 30 mg) or chlorpromazine (Largactyl, 50 mg). Liberal analgesia with codeine, 30 to 60 mg, or...
Early aneurysm surgery

<table>
<thead>
<tr>
<th>Site &amp; No. of Aneurysms</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>internal carotid artery</td>
<td>36</td>
</tr>
<tr>
<td>anterior cerebral artery</td>
<td>30</td>
</tr>
<tr>
<td>anterior communicating artery</td>
<td></td>
</tr>
<tr>
<td>middle cerebral artery</td>
<td>20</td>
</tr>
<tr>
<td>posterior circulation</td>
<td>14</td>
</tr>
<tr>
<td>single aneurysm</td>
<td>85</td>
</tr>
<tr>
<td>multiple aneurysms</td>
<td>15</td>
</tr>
</tbody>
</table>

Demerol (meperidine), 50 to 100 mg, was considered essential to alleviate patient discomfort or restlessness. Mild hypotension was induced with reserpine, 0.25 mg, and systolic peaks above 140 mm Hg in known hypertensive patients or above 120 mm Hg in normotensive patients were reduced with hydralazine HCl, 10 to 20 mg. Normal fluid and electrolyte balance was maintained. Whenever ischemia and edema were considered present by virtue of neurological deficit, Decadron (dexamethasone, 2 to 4 mg) was administered. We prescribed Amicar (epsilon-amino-caproic acid (EACA), 24 gm/day orally) for the first few patients, but discontinued its use due to poor patient tolerance.

Operative Management

The time of operation was determined from Lougheed's criteria, according to aneurism site, severity of hemorrhage, and presence of vasospasm, until 1978, when we embarked on a policy of immediate aneurysm repair regardless of site or presence of vasospasm. Four patients had proximal ligation of a parent artery for multiple SAH, and one patient with a giant carotid artery aneurysm had progressive occlusion of the extracranial carotid artery. All other patients had their aneurysms repaired directly under the operating microscope. Intraoperative lumbar drainage was supplemented with osmotic diuresis (mannitol 20%, 0.25 to 0.5 gm/kg) to improve exposure and minimize brain retraction. Intraoperative hypotension (sodium nitroprusside) was routinely used during aneurysm dissection in the earlier 52 cases (prior to 1978), but only during the dissection of anatomically complex aneurysms thereafter.

Postoperative Management

Normal fluid and electrolyte balance was maintained postoperatively, and the preoperative corticosteroids were tapered off unless clinical deterioration due to angiographically proven vasospasm was noted. In this situation, measures additional to corticosteroid therapy and osmotic diuresis were used, including the removal of a bone flap with duraplasty (in three patients) and temporal lobectomy (in two patients). Intracarotid phenoxybenzamine was given without effect in one patient.

Analysis of Results

Results of management were tabulated at 3 months after the presenting hemorrhage, using the outcome categories of Jennett and Bond, as follows:

- Category I: Death
- Category II: Persistent vegetative state
- Category III: Severe disability (conscious but disabled)
- Category IV: Moderate disability (disabled but independent)
- Category V: Good recovery.

Patients classified in Botterell Grades I, II, and III on admission who failed to reach outcome Categories V, IV, and III, respectively, were considered worse. The operative mortality rate was based on the number of deaths within 30 days of surgery. The management mortality rate for any time interval was based on the sum of operative deaths attributed to that time interval, plus the total of nonoperative deaths accumulated during and preceding that interval. Combined management morbidity and mortality figures were based on the sum of patients who died or became worse after surgery during that time interval, plus the nonoperative patients who died during and preceding that time interval and the nonoperative survivors who remained worsened.

Results

Preoperative deterioration occurred in 36 patients, eight of whom died. This deterioration, more common in patients with multiple SAH's at admission, increased with time after admission and was uniform in all three grades (Table 2). Worsening persisted in 13 patients of whom four were excluded from surgery. Confirmed rebleeding was observed in 16 patients and suspected in another 12. Preoperative clinical vasospasm occurred in 13 patients. Of the 72 patients who were admitted to the hospital within 24 hours of SAH, 86% had maintained their admission grade or improved by 48 hours after admission, but this figure dropped to 50% after 1 week and 42% after 2 weeks. Only 88 of the initial 100 patients underwent surgery. This reflects a loss due to death or deterioration of 11 (18%) of the 62 patients managed prior to a policy of planned immediate operation and a loss of only one (3%) of 38 patients managed with planned immediate operation. Forty of the 88 patients undergoing surgery were operated on within 7 days of their most recent SAH.

At surgery, the condition of the brain correlated with the clinical grade of the patients and the interval from last SAH to surgery. In the 40 patients explored within 7 days of their most recent bleed, the brain was slack and easily retracted in all of the 11 good-risk patients (Grades I and II) undergoing operation within 48 hours of a single SAH. Although the brain was still found to be slack between the 3rd and 7th
day following a single bleed in seven of nine Grade I patients, it was noted to be tight during this interval in seven of nine Grade II patients with single bleeds. In all patients admitted with multiple bleeds and all patients admitted in Grade III, the brain was tight at all times during the 1st week after SAH. Hence, it appeared that within 48 hours of a single bleed, a slack brain could be anticipated in all good-risk patients presenting with a single bleed, but between the 3rd and 7th days after SAH the brain tended to be tight except in essentially asymptomatic Grade I patients who were admitted with a single bleed.

The results of overall management and surgery for Grade III patients subjected to early operation were poor (Table 3). The lower surgical mortality and the decline in management mortality/morbidity rates more than 7 days after SAH confirmed the benefits of delay in sick patients. In Grade I and II patients, both the surgical mortality/morbidity and the overall management mortality/morbidity rates favored operative intervention within 48 hours of a bleed, as opposed to a delay of 7 days or more. Similarly, in both Grade I and II patients, the surgical and management mortality/morbidity rates were worst between the 3rd and 7th days following SAH, reflecting the tension of the brains encountered at surgery.

Significant postoperative vasospasm was observed and treated in 15 of these patients: four Grade I patients, eight Grade II patients, and three Grade III patients. It occurred in seven of 14 patients presenting with multiple bleeds, and in five of eight patients with preoperative vasospasm. Significant spasm was noted in one of 13 patients who underwent surgery within 48 hours of SAH, but most commonly (seven of 23 patients) following surgical repair between the 3rd and 7th day and in seven of 47 patients with surgery more than 7 days after a bleed.

**Discussion**

The good-risk patient with a ruptured intracranial aneurysm risks deterioration with a policy of delayed surgical intervention. This progressive deterioration from rebleeding and vasospasm will afflict up to 30% of patients toward the end of the 1st week of their presenting SAH.1 The 32% rebleed rate reported by Sundt and Whisnant among their 310 cases of ruptured aneurysms contributed largely to the 18% of Grade I and II patients who died or deteriorated

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**TABLE 2**

Summary of preoperative clinical course

<table>
<thead>
<tr>
<th>Grade on Admission</th>
<th>Total Cases</th>
<th>Preop Deterioration</th>
<th>Resultant Clinical Grade</th>
<th>Grade at Operation</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Higher</td>
<td>Lower</td>
</tr>
<tr>
<td>I</td>
<td>39</td>
<td>14</td>
<td>0</td>
<td>6</td>
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<td>II</td>
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<td>III</td>
<td>13</td>
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<td>2</td>
<td>1</td>
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</table>

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**TABLE 3**

Surgical and management mortality and morbidity for patients undergoing intracranial repair of a ruptured aneurysm

<table>
<thead>
<tr>
<th>Grade on Admission</th>
<th>Time From Last SAH* (days)</th>
<th>No. of Cases</th>
<th>At Risk</th>
<th>No Surgery</th>
<th>Surgery</th>
<th>Surgery Mortality</th>
<th>Surgery Mortality + Morbidity</th>
<th>Management Mortality</th>
<th>Management Mortality + Morbidity</th>
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<tbody>
<tr>
<td></td>
<td>0-2</td>
<td>38</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>25%</td>
<td>0</td>
<td>25%</td>
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<tr>
<td></td>
<td>3-7</td>
<td>34</td>
<td>2</td>
<td>12</td>
<td>8%</td>
<td>33%</td>
<td>21%</td>
<td>43%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0-7</td>
<td>38</td>
<td>2</td>
<td>16</td>
<td>6%</td>
<td>31%</td>
<td>17%</td>
<td>39%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt; 7</td>
<td>20</td>
<td>3</td>
<td>17</td>
<td>6%</td>
<td>24%</td>
<td>27%</td>
<td>41%</td>
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</tr>
<tr>
<td>II</td>
<td>0-2</td>
<td>44</td>
<td>2</td>
<td>8</td>
<td>6%</td>
<td>13%</td>
<td>10%</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3-7</td>
<td>34</td>
<td>2</td>
<td>10</td>
<td>20%</td>
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<td>23%</td>
<td>54%</td>
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</tr>
<tr>
<td></td>
<td>0-7</td>
<td>44</td>
<td>4</td>
<td>18</td>
<td>11%</td>
<td>22%</td>
<td>14%</td>
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<td></td>
<td>&gt; 7</td>
<td>22</td>
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<td>21</td>
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<td>19%</td>
<td>15%</td>
<td>31%</td>
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<tr>
<td>III</td>
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<td>13</td>
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<td>1</td>
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<td>0</td>
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<td>50%</td>
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<tr>
<td></td>
<td>3-7</td>
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<td>33%</td>
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<tr>
<td></td>
<td>&gt; 7</td>
<td>7</td>
<td>0</td>
<td>7</td>
<td>14%</td>
<td>43%</td>
<td>22%</td>
<td>56%</td>
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</tr>
</tbody>
</table>

* SAH = subarachnoid hemorrhage.
Early aneurysm surgery

Preoperatively. Attempts to reduce the loss of patients prior to definitive surgical repair have focused on the use of antifibrinolytic agents and their combined use with controlled hypotension. Mullan and Dawley pointed out that EACA inhibits circulating enzymes in the blood and cerebrospinal fluid (CSF), but that the clot sealing the aneurysm defect contains sufficient enzymes to facilitate its own dissolution. Accordingly, the use of EACA could be expected to reduce the risk of a third hemorrhage but not necessarily a second hemorrhage. Nevertheless, although Shucart, et al., reported no obvious benefit from the use of EACA, dramatic reductions in rates of rebleeding have been reported, particularly when antifibrinolytic therapy was combined with controlled hypotension. Unfortunately, these protocols have not been without problems. It is estimated that it takes 3 to 5 days of therapy before adequate therapeutic levels will be obtained in the CSF. A number of complications, ranging from diarrhea, venous thrombosis, pulmonary embolism, renal failure, arteriopathies, increased vasospasm, and a higher likelihood of communicating hydrocephalus, have been reported. Since the completion of this study we have commenced the use of intravenous EACA for patients evaluated more than 48 hours after their first hemorrhage, and, as observed by Rowed, we have encountered unexpected technical difficulties at the time of surgery due to unusually tenacious adherence of the clot to the aneurysmal sac and its surroundings. Some of these difficulties may yet be overcome by the use of serum and CSF assays to enable better dosage regulation.

Preoperative deterioration from vasospasm, with resultant ischemic deficit, spares no clinical grade and may result in death or permanent incapacity. It is all the more disheartening when it occurs within 2 weeks of a hemorrhage in up to 30% of good-risk Grade I or II patients who are awaiting surgical repair; for, in these cases, vasospasm is seldom present within the first few days after a bleed, tending to appear toward the end of the 1st week or thereafter. Stornelli and French and Alcocock and Drake have cautioned that early surgery with or without preoperative spasm is more likely to precipitate postoperative vasospasm. However, Adams, et al., believed that pre- and postoperative vasospasm occur independently. Sano and Saito have suggested that a reduction in the incidence and complications of spasm may be achieved by clearing blood from the subarachnoid space at the time of early operation. However, our own experience suggests that the local blood around the aneurysm and its adjacent cisterns may well be washed out in this manner, but not the more remote blood that is widely dispersed throughout the subarachnoid space. Early surgery will not prevent the appearance of vasospasm, but it does facilitate a more aggressive approach to its treatment (such as the addition of low molecular weight dextran to protocols of fluid load and hypertension) once the ruptured aneurysm is repaired and protected from further hemorrhage.

Botterell, et al., and others have cautioned against surgery within a week of hemorrhage because of the friable, tight brain that was encountered. Not surprisingly, then, many authors have shown not only improved surgical mortality but also less morbidity by delaying surgery until the 2nd week. Yet, Hori and Suzuki reported encouraging results in their patients subjected to “peracute surgery” within 48 hours of a single bleed. They also stressed that “peracute surgery” should not be considered in patients presenting with multiple bleeds. We support this opinion by our own observation of a slack brain within 48 hours of a single bleed in Grade I and II patients, in contrast to the tight brain encountered in patients explored at any time within the 1st week of one of repeated bleeds. In the analysis of their 122 aneurysms repaired within the 1st week of rupture, Suzuki, et al., reported their highest success rate in cases repaired within 48 hours of a bleed and their worst results in cases explored on the 3rd day after SAH. We found the brains of our Grade II patients to be tight by the 3rd day post SAH even following a single bleed. No clear explanation is offered for the change in outcome between surgery in the first 48 hours following a bleed and surgery during the rest of the 1st week, except that it appears to reflect a possible biochemical and vasogenic reaction to the initial hemorrhage that is established by about 72 hours.

Our surgical and management mortality/morbidity rates suggest a poor outcome for surgery during the 1st week following a bleed; this reflects the poor results of surgery during the 3rd to 7th days after SAH as reported by Hori and Suzuki in their analysis of a series of anterior communicating artery aneurysms. After we had embarked for 2 years on a policy of early surgical repair regardless of grade or the presence or absence of vasospasm, our management mortality/morbidity rate dropped from 44% to 24%. Gillingham reported the lack of significant brain reaction and the lack of raised intracranial pressure within 48 hours of the first bleed of an aneurysm. Not surprisingly, we found surgical repair during the first 48 hours of the first bleed of an aneurysm no more difficult than surgery delayed for a week or more. It minimizes the risk of rebleeding, particularly in good-risk patients, and facilitates a more aggressive approach to the treatment of ischemic postoperative complications. However, if the patient presents with a history of multiple bleeds, or if surgical repair cannot be scheduled within 48 hours of a single bleed, we now advocate a policy of delay for up to a week or longer, using a vigorous preoperative protocol that includes controlled hypotension and antifibrinolysis. A thorough inquiry into the presenting symptoms is essential to spot any suggestion of prior minor bleeds. Although Hori and Suzuki also suggest that “peracute surgery” is applicable to Hunt’s Grades III...
and IV\textsuperscript{16} (the equivalent of Botterell's Grades II and III), our initial experience with these ill patients leads us to recommend a policy of delayed surgery. The better the patient's condition at admission following a single bleed, the more urgent the need to intervene early before the second day has elapsed after the bleed.

**Conclusions**

Early surgical intervention in good-risk patients (Botterell Grades I and II) with ruptured intracranial aneurysms appears to be a reasonable alternative to a policy of delayed operation in cases that present with a single bleed and can be repaired within 48 hours, because the brain will be slack and manageable using microsurgical technique. Patients presenting with multiple bleeds do poorly if explored early, for their brains are friable and edematous. We suggest that the 3rd day following an initial hemorrhage from a ruptured aneurysm is a critical time in terms of the brain's characteristics at surgery, and the results of early surgery at this time. The validity of this observation will require analysis of many more patients treated with early surgical repair.

**References**

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