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

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Data from 722 consecutive cases with intracranial aneurysms were stored in a computer and later retrieved for analysis. Results and complications (including preoperative death and morbidity) of the surgical management of these patients were correlated with the Botterell grade of the patient in individuals with a recent subarachnoid hemorrhage (SAH), with the type of aneurysm, and with the timing of the surgical procedure. Patients with no SAH within 30 days prior to hospital admission were classified as “no SAH.” Approximately 30% of all patients had sustained more than one hemorrhage. Death and morbidity rates prior to surgery in good-grade patients with a recent SAH exceeded the risk of surgery itself. Rebleeding was the primary cause for death and morbidity in Grade 1 patients: 3% of Grade 1 patients died from a recurrent hemorrhage and 7% deteriorated to a lower grade. Deterioration from ischemia produced by vasospasm related or unrelated to rebleeding exceeded the risks of rebleeding in Grade 2 patients. There was an operative morbidity of 2% and mortality of 2% in patients who were classified as Grade 1 at the time of surgery, but an overall management morbidity of 3% and mortality of 6% in patients who were in Grade 1 at the time of hospital admission. Early surgery in Grade 1 patients was not associated with an increased incidence of delayed ischemia postoperatively. In Grade 2 patients, the operative morbidity and mortality was 7% and 4%, respectively, and the management morbidity and mortality 16% and 11%, respectively. Early surgery in this group was associated with a high frequency of postoperative delayed ischemia (particularly in patients with more than one SAH). Epsilon-aminocaproic acid appeared to protect against a rebleed, but was associated with a higher incidence of postoperative pulmonary emboli. Intraoperative complications were related both to the size of the aneurysm and to its location. Repair of multiple aneurysms did not adversely affect the result. The surgical approach, the importance of using a self-retaining brain retractor, and the technical complications in these cases are discussed.

KEY WORDS: aneurysm, subarachnoid hemorrhage, timing of surgery, vasospasm, neurological grade

It is the purpose of this report to correlate specific types of preoperative, operative, and postoperative complications of cerebral aneurysm surgery in one surgeon’s experience with the timing of surgical intervention, the location and size of the aneurysm, the neurological grade of the patient, and the methods of treatment. We have included surgically treated aneurysms that were acting as mass lesions or were not associated with a recent hemorrhage (within 30 days); these cases are included so as to contrast complications that were purely surgical and related to the technical problems of aneurysm repair to those that were, at least in part, attributable to a recent subarachnoid hemorrhage (SAH). Similarly, because our treatment at this institution for this illness is surgical repair of the aneurysm whenever possible, it is necessary to include deaths and complications prior to operation. Hopefully, this correlation will place in perspective the risks of early surgery, and serve as some standard for future comparison. During the period of this re-
port, variations in the surgical approach to some difficult lesions evolved to include increased use of bypass grafting and, accordingly, the series is heavily weighted toward the end of the period of the report with large and giant aneurysms. All aneurysms were operated on under the operating microscope.

Clinical Material and Methods

Case Material

This report includes two primary groups of surgical patients: those with a recent SAH (less than 30 days) from a ruptured intracranial aneurysm, and those without a recent SAH, in whom the aneurysm caused symptoms by its mass effect. This report includes only cases treated by one of the two neurovascular services at the Mayo Clinic from May, 1969, through December, 1981. All aneurysms were operated on by one surgeon or a member of the resident staff with that surgeon acting as the first assistant. Multiple aneurysms approachable through the same operative exposure were routinely repaired during the same operative procedure. However, patients undergoing totally separate procedures for repair of aneurysms (for instance, 1 month apart) in different vascular trees (13 patients) were considered separate cases and, therefore, the operative statistics are based on 644 cases. A total of 731 aneurysms were repaired in 644 operations: one aneurysm in 573 cases, two aneurysms in 58 cases, three aneurysms in 10 cases, and four aneurysms in three cases. Twenty-one additional aneurysms were not repaired.

Seventy-eight patients admitted with a recent SAH from a proven intracranial aneurysm died prior to surgery or deteriorated to a state that did not permit surgery. These patients (78 aneurysms and 78 cases) are included in the analysis of the group of patients with a recent bleed and, therefore, the entire series comprises 722 cases.

One hundred sixty-six or 29% of patients with a recent or old SAH had more than one SAH (5% with no SAH within 30 days at hospital admission, 30% who were in Botterell (modified, see below) Grade 1, 27% who were in Grade 2, 29% who were in Grade 3, and 10% who were in Grade 4). These same figures show, however, that 21% of Grade 1, 38% of Grade 2, 41% of Grade 3, and 22% of Grade 4 patients had had more than one bleed.

Data from each patient were collected during their hospitalization, and thereafter from follow-up visits or correspondence. This information was recorded on a standard abstract form and subsequently stored in a computer for data retrieval and analysis.

Management Protocol

Phenobarbital for sedation and codeine for analgesia were the major medications given before surgery. Antihypertensive agents were used cautiously, dexamethasone sparingly, and epsilon-aminocaproic acid (Amicar) at a dosage of 1 gm/hr later in the series in patients with a recent SAH.25 Postoperative ischemic complications were managed with blood volume expansion (usually whole blood or packed cells) and, when severe, with isoproterenol and lidocaine hydrochloride using a regimen previously described in detail.57 Fluids were restricted in patients with alteration in sensorium.

The timing of surgical intervention was, in general, determined by the patient's neurological function and the severity of meningeal signs (including fever). Early in the series an operation was customarily delayed for 7 to 9 days, or until the patient was free of acute meningeal complaints, whichever occurred earlier. A recurrent hemorrhage without neurological change was the primary indication for early operation before January, 1978.42 Thereafter, there was a gradual evolution toward earlier surgery in patients who were neurologically normal with only minimal headache. All aneurysms were repaired by intracranial clipping with the aid of the operating microscope through standard exposures, or (as was the case in some giant aneurysms) were treated with a combination of internal carotid artery (ICA) or vertebral artery ligation and some form of bypass procedure.40 Anastomoses included superficial temporal artery to middle cerebral artery (MCA) bypass or an interposition saphenous vein graft between the external carotid artery and the posterior cerebral artery or a major branch of the MCA.

For a general analysis of the results and complications of surgery, aneurysms were divided into six groups according to their location: ICA aneurysms, which included aneurysms arising from any point in the ICA between the siphon and the bifurcation of that vessel; anterior communicating artery (ACoA) aneurysms, which included any aneurysm arising at or near the ACoA; MCA aneurysms, including aneurysms arising at or near the bifurcation of the MCA; basilar artery caput aneurysms, which included aneurysms arising at the caput of the basilar artery; vertebrobasilar trunk aneurysms, which included aneurysms arising from the trunk of the vertebral or basilar artery and those arising at the origin of the posterior inferior cerebellar artery; and aneurysms associated with other arteries, which comprised aneurysms at more distal locations such as the distal anterior cerebral artery, posterior cerebral artery, or superior cerebellar artery.

Angiographic Spasm

Preoperative angiograms in Grade 1 and 2 patients completed within 9 days from the last SAH were evaluated retrospectively by one of us (S.K.) in an effort to correlate the appearance of spasm with the timing of arteriography and the occurrence of postoperative delayed ischemic complications. The majority of our patients underwent angiography elsewhere prior to referral or were referred after a period of

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conservative management. Thus, only 50 angiograms in Grade 1 patients and 29 in Grade 2 patients were available for analysis of the development of spasm. The group used to correlate types of angiographic spasm with postoperative ischemia was further restricted to patients who had angiography within 48 hours prior to surgery. Only 29 studies in Grade 1 patients and 22 in Grade 2 patients fulfilled the criteria for inclusion in this group.

**Neurological Grading**

We divided patients according to their neurological function, using the Botterell grading system (with modification). Four grades were used:

- **Grade 1:** With or without mild headache, alert and oriented, with no motor or sensory deficits.
- **Grade 2:** Severe headache and major meningeal signs, mild alteration in sensorium or focal deficits.
- **Grade 3:** Major alteration in sensorium or a major focal deficit.
- **Grade 4:** Semicomatose or comatose, with or without major lateralizing findings.

Patients operated on for mass effect from the aneurysm and without a recent SAH (within 30 days) were classified as "no SAH."

**Classification of Surgical Results**

Four categories were used for judging the results of surgery: 1) excellent: normal employment, with normal mentation and little or no neurological deficit; 2) good: neurological deficit but with normal mentation and employment; 3) poor: anything less than full activity (included patients with personality or mental change or a disabling focal deficit, or both); and 4) death. In determining surgical morbidity and mortality, we chose an arbitrary end point of 6 months from the time of operation, and accordingly any death within 6 months is reflected in the mortality figure.

**Complications**

**Preoperative Complications.** Preoperative complications were categorized primarily into two major types: rebleeding and deterioration from ischemia related to vasospasm.

**Operative and Postoperative Nonischemic Complications.** Any major complication during the operative procedure or directly attributable to a technical error in the operation itself, such as a postoperative hemorrhage from incomplete aneurysm repair, was included in this category.

**Postoperative Delayed Ischemia.** To qualify for inclusion in this category, the deficit had to appear at least 4 hours after the time of operation and when it was apparent that the patient had initially recovered from the operative procedure unchanged from his or her state prior to surgery. It was, furthermore, necessary to prove that the deficit was not related to a localized mass lesion, obstruction of flow through a major vessel, or vital perforating vessel occlusion.

We considered an ischemic or postoperative complication minor if it was associated with minimal changes in sensorium or mild motor or sensory symptoms on one side only. If the dominant hemisphere was involved, minor speech changes were often the only finding. Major deficits were associated with moderate to severe hemiparesis or profound changes in sensorium or both; they were frequently progressive; some led to death.

**Operative Approaches and Techniques**

Although all patients were operated on using recognized and standard techniques, some characteristics of the approaches used may be related to the prevalence or absence of certain complications in the series.

**Operative Approaches**

The pterional approach, diagrammatically illustrated in Fig. 1, evolved as the basic approach used for the repair of all aneurysms arising from the anterior circulation, except those of the distal anterior cerebral artery. Close attention must be given in this approach to the placement of the frontal burr holes so that they are as close as possible to the floor of the frontal fossa. There is considerable variation in the shape of skulls, so exact placement of the bone flap must be individualized. A considerable portion of the lesser wing of the sphenoid bone should be resected so that brain retraction can be minimized. The arachnoid in the Sylvian fissure is opened to permit movement of the frontal lobe without disturbing the temporal lobe and, therefore, avoiding the need to divide bridging veins coming off the tip of that lobe. Only a small number of instruments are required: a knurled-handled No. 11 blade knife holder, a small spatula, microvascular scissors, knot-tying forceps, and a "microvascular gimmick."

The anterior subtemporal or modified pterional approach differs from the pterional approach in that a greater amount of bone is resected below the sphenoid wing and the skull is positioned differently in a Mayfield-Kees headholder, so that the area in and around the caput of the basilar artery can be exposed along the floor of the middle fossa by retraction of the tip of the temporal lobe. Basically, this is merely a slight modification of Drake’s subtemporal approach.

The posterior fossa approach used is the one described by Drake.7 The procedure used for combined ICA ligation and extracranial to intracranial bypass has been described previously in detail.12 The bifrontal approach, no longer employed, was formerly used for repair of ACoA aneurysms. This approach was abandoned, not because of hemispheric or retraction complications, but because of the increased time re-
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**FIG. 1.** Schematic illustration of recommended positioning of the Yaşargil retractor. Note that the flexible arm is positioned in such a manner that it is supported by the scalp at Point A. The head of the bar of the fixation device which attaches the flexible arm to the operating table is placed relatively close to the operating table in order to avoid an excessive length of the flexible arm. The support rendered to the flexible arm at Point A gives considerable additional strength to the retractor which not only increases the life of the retractor but, more importantly, the precision of the instrument. The malleable retractor can be rotated about Point B for fine adjustments after the flexible arm has been positioned appropriately. The retractor not only retains the brain, but elevates it slightly, opening up the arachnoidal cisterns. See text for a more detailed description of retractor usage.

**FIG. 2.** Conventional use of the Yaşargil retractor. The retractor is shown totally suspended and free of additional support other than the point where it is attached to the fixation bar. Used in such a manner, the retractor is vulnerable to movement by the surgeon, and excessive tension is required on the flexible arm in order to retain the brain out of the field of surgery.

required for the procedure and the anosmia that invariably accompanied the exposure.

**Spinal Drainage and Mannitol**

For the pterional approach, controlled cerebrospinal fluid (CSF) drainage through a malleable spinal needle is used to provide the minimal space required for exploration of the ACoA, ICA, and MCA's. In most instances, it is only necessary to withdraw 60 to 70 ml of CSF to obtain adequate exposure to the area of the chiasm. In many instances of MCA aneurysm surgery, no CSF is withdrawn. For surgery of aneurysms arising at the caput of the basilar artery in which the anterior subtemporal approach has been used, total CSF drainage is used (120 ml or more) supplemented by 1 gm/kg of mannitol. This provides a major degree of brain relaxation so that the temporal tip can be retracted sufficiently to expose the area of the tentorium without creating a subcortical retraction hematoma.

Graded, measured CSF withdrawal has been preferred for anterior circulation aneurysms as it is well controlled and provides immediate brain relaxation at the time it is required and requested. Too much brain relaxation can rupture bridging veins and become a source of considerable trouble to the surgeon during and after the operative procedure. The hypervolemia that accompanies the use of mannitol or urea makes it more difficult to control the blood pressure and provide the desired degree of hypotension.

**Self-Retaining Brain Retractor**

It is mandatory that the Yaşargil self-retaining brain retractor be used accurately and with facility in this type of surgery. Recommended and conventional positions of the brain retractor are illustrated in Figs. 1 and 2. It is not necessary to have a major extension of the fixation bar beyond the tip of the operating table. The head of the bar, which is the fixation device for the flexible arm, should be positioned relatively close to the operating table. A malleable retractor, seated in fixation jaws at the distal tip of the flexible arm, is held in the approximate location in which it will be used during the operation. The universal joint affixing the bar to the operating table is loosened and the bar moved proximally and distally to a position where the flexible arm traverses the distance between the malleable retractor and the fixation head without distortion. The head of the bar is then loosened so that it can be rotated clockwise or counterclockwise to further adjust the flexible arm and determine the position where the untightened configuration of this arm follows the contour of the skull. This permits the establishment of a firm, reliable support for the ful-
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TABLE 1
Operative results in all patients who underwent aneurysm repair

<table>
<thead>
<tr>
<th>Preop Grade*</th>
<th>No. of Cases</th>
<th>Operative Result</th>
<th>Morbidity (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>no SAH</td>
<td>178</td>
<td>Excellent</td>
<td>150</td>
<td>12</td>
</tr>
<tr>
<td>1</td>
<td>232</td>
<td>Good</td>
<td>215</td>
<td>9</td>
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<tr>
<td>2</td>
<td>110</td>
<td>Poor</td>
<td>80</td>
<td>18</td>
</tr>
<tr>
<td>3</td>
<td>104</td>
<td>Dead</td>
<td>38</td>
<td>27</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td></td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>644</td>
<td></td>
<td>486</td>
<td>67</td>
</tr>
</tbody>
</table>

* Neurological grading by a modified Botterell classification, see text. SAH = subarachnoid hemorrhage.

TABLE 2
Results of preoperative management in patients with recent subarachnoid hemorrhage*

<table>
<thead>
<tr>
<th>Grade Prior to Surgery</th>
<th>No. of Died on Admission</th>
<th>No. Excellent</th>
<th>No. Good</th>
<th>No. Poor</th>
<th>No. Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>261</td>
<td>224</td>
<td>84</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>161</td>
<td>90</td>
<td>36</td>
<td>14</td>
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<tr>
<td>3</td>
<td>3</td>
<td>69</td>
<td>49</td>
<td>23</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>53</td>
<td>30</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>total</td>
<td>544</td>
<td>337</td>
<td>124</td>
<td>57</td>
<td>37</td>
</tr>
</tbody>
</table>

* Includes preoperative death and morbidity. Neurological grading by a modified Botterell classification, see text. SAH = subarachnoid hemorrhage.

TABLE 3
Overall management results in patients with recent SAH*

<table>
<thead>
<tr>
<th>Condition at Discharge</th>
<th>Grade Prior to Surgery</th>
<th>No. Excellent</th>
<th>No. Good</th>
<th>No. Poor</th>
<th>No. Dead</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>261</td>
<td>224</td>
<td>84</td>
<td>14</td>
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<tr>
<td>2</td>
<td>2</td>
<td>161</td>
<td>90</td>
<td>36</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>69</td>
<td>49</td>
<td>23</td>
<td>12</td>
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<tr>
<td>4</td>
<td>4</td>
<td>53</td>
<td>30</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>total</td>
<td>544</td>
<td>337</td>
<td>124</td>
<td>57</td>
<td>37</td>
</tr>
</tbody>
</table>

* Neurological grading by a modified Botterell classification, see text. SAH = subarachnoid hemorrhage.

Crum of the retractor. Thereafter, the optimum tension is created in the flexible arm to hold it in place. It is not necessary to have a great deal of tension in the flexible arm, and the malleable retractor can be held in position with relatively modest tension if the device is appropriately positioned. This increases the life of the retractor and, more importantly, the precision of the instrument.

It is important that the retractor be placed so that it has firm support on the scalp (Point A, Fig. 1). This allows rotation of the malleable retractor at Point B, the point where the retractor is attached to the flexible arm. This fulcrum is easily moved by loosening the flexible arm. A third advantage of placing the retractor in this position is safety: it is unlikely that a member of the operating team will bump the retractor when it is so placed and change its position at a critical point during the operation.

Clips

There is no single clip that is ideal for all aneurysms. Very often more than one clip was used to repair an aneurysm. The following clips were used: 99 Scoville, 280 McFadden, 55 Drake, 100 Yasargil, 12 Heifetz, 60 McFadden-Kees, 11 Sugita, 102 Sundt-Kees clip grafts, and 50 other clips. In general, for globular and giant aneurysms McFadden and Drake clips were used, and for smaller aneurysms the Yasargil clips, the new McFadden-Kees clips, and more recently, and with increasing frequency, the Sugita clips were used. Sundt-Kees clip grafts were used more often earlier in the series, and later only when bleeding occurred at the base of the aneurysm or when there was a tear in a major vessel.

Results and Complications

Patients Without Recent SAH

The operative results in 178 patients in whom symptoms (primarily mass effect) were unrelated to a recent SAH (the "no SAH" group) are summarized in Table 1. This group included 57 aneurysms larger than 25 mm in diameter (giant aneurysms) and 25 aneurysms between 15 and 25 mm in diameter (globular aneurysms).

Patients with Recent SAH

Results of preoperative management of patients with recent SAH are summarized in Table 2, which compares the grade of the patient prior to surgery to the grade of that patient at the time of hospital admission. The overall management results in this group, including deaths before surgery, are summarized in Table 3, and the operative results in those patients who survived to undergo surgery are summarized in Table 1.

Location of Aneurysms

The results of surgery according to the location of the primary aneurysm repaired are summarized in Table 4. These results are unrelated to aneurysm size and neurological grade of the patients. In this series, giant aneurysms were found at the following locations: four at the ACoA's, 42 at the ICA's, 22 at the MCA's, eight at the caput of the basilar arteries, two at the verteobasilar trunk, and four others. Globular aneurysms had a similar distribution: six at the ACoA's, 15 at the ICA's, four at the MCA's, seven at the caput of the basilar arteries, none at the verteobasilar trunk, and one other.

Complications

Preoperative Complications. Rebleeding prior to surgery in our hospital occurred in 24 Grade 1 patients.
Five were receiving Amicar (of a total of 57 Grade 1 patients treated with Amicar before surgery) and 19 were not (of a total of 204 Grade 1 patients not treated with Amicar before surgery). Thus, the rebleed rate in Grade 1 patients receiving Amicar was 9% and the rebleed rate in patients not receiving Amicar was 9%. However, there was a major difference in these two groups in that the mean time between SAH and hospital admission was 5.2 days in the Amicar group but 8.1 days in the group without Amicar, so the groups are not comparable.

Rebleeding occurred in 23 Grade 2 patients who were in our hospital awaiting surgery. Three were receiving Amicar (of a total of 35 Grade 2 patients treated with Amicar) and 20 were not (of a total of 126 Grade 2 patients not treated with Amicar). Thus, there was a rebleed rate of 9% in Grade 2 patients treated with Amicar and a rebleed rate of 16% in patients not treated with Amicar. The mean days between the last SAH and hospital admission was 5.5 days in both groups. There is a statistically significant difference in these two groups in favor of Amicar treatment (p < 0.05).

Ischemic complications with or without permanent deterioration and unrelated to a rebleed occurred in eight Grade 1 patients (3% of patients who were in Grade 1 on admission) and in 17 Grade 2 patients (10% of patients who were in Grade 2 on admission). Ten additional Grade 2 patients deteriorated in our hospital as a result of a rebleed that occurred before referral. Thus, in Grade 2 patients, deterioration from ischemia following hospital admission was a greater risk than a rebleed. Two Grade 2 patients treated with Amicar had major ischemic strokes that were sudden in onset, did not have the temporal profile of spasm-induced ischemia, and suggested a thrombotic major vessel occlusion.

Twenty-four of the Grade 1 patients deteriorated to Grade 2, and 16 Grade 2 patients to Grade 3 as the result of multiple causes that could not clearly be defined as the result of a rebleed or spasm. Deterioration in Grade 3 and 4 patients was usually attributable to multiple factors (such as communicating hydrocephalus, subdural or intracerebral hematoma, edema, etc.), and a primary cause was not usually identified; thus, categorization was difficult. The overall fate of these patients is summarized in Table 2.

**Intraoperative Complications.** Intraoperative complications related to the size and location of the aneurysm are summarized respectively in Tables 5 and 6. Four aneurysms ruptured during anesthesia induction or craniotomy and this led to two deaths and two poor results. Three of these patients were in Grade 1 and
Results and complications of aneurysm surgery

### TABLE 6
Intraoperative complications according to location of primary aneurysm

<table>
<thead>
<tr>
<th>Location of Symptomatic Aneurysm*</th>
<th>Total Cases</th>
<th>Bled During Uncontrolled Location of Induction or Bleeding Major Vessel Damage to Embolization</th>
<th>Occlusion</th>
<th>Perforating Vessels</th>
<th>Embolization from Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACoA</td>
<td>166</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ICA</td>
<td>249</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>MCA</td>
<td>113</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>BAC</td>
<td>63</td>
<td>1</td>
<td>0</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>VB trunk</td>
<td>32</td>
<td>†</td>
<td>†</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>other</td>
<td>21</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* ACoA = anterior communicating artery; ICA = internal carotid artery; MCA = middle cerebral artery; BAC = basilar artery caput; VB = vertebrobasilar.
† Same patient.

Bleeding during repair of the aneurysm occurred in 80 cases. This was unrelated to the location of the aneurysm but was more common in giant aneurysms. Only cases in which bleeding was uncontrolled or led to a neurological deficit are included in Tables 5 and 6. In general, bleeding during repair of the aneurysm was found to be unrelated to any postoperative morbidity if it was controlled and kept out of the subarachnoid space. It was also unrelated to the time of the last SAH.

Occlusion of major vessels was a complication related to the size of the aneurysm and, in general, predated the era of bypass surgery at this institution. Occlusion of perforating vessels was not only related to the size of the aneurysm but also to its location.

**Postoperative Non-Ischemic Complications.** Postoperative non-ischemic complications are summarized in Table 7 according to the operative approach used for repair of the aneurysm. Bleeding from the same aneurysm occurred in six cases and in two cases from a different aneurysm. Three of the lesions in which bleeding occurred from the aneurysm undergoing surgery were giant ICA aneurysms which had not bled previously and which were operated on because of mass effect from the aneurysm. Bleeding led to death in one patient when a Selverstone clamp had to be opened because of hemispheric ischemic symptomatology, and two patients bled after ICA ligation and saphenous vein bypass grafting between the external carotid artery and one of the two major divisional limbs of the MCA. Two globular aneurysms rebled following incomplete repair. One saccular aneurysm rebled, unequivocally as the result of clip slippage (a Heifetz clip).

Three patients underwent reoperation for treatment of subdural hematomas; two because of bleeding problems related to abnormalities in blood clotting known before surgery, and one from too much CSF drainage that had produced a subdural hematoma. One patient had an epidural hematoma. All four of these individuals made excellent recoveries.

Retraction hematomas occurred in seven cases, all early in the series. None were found in Grade 1 patients; however, there were three in Grade 2 patients: one 2 days after SAH and two more than 10 days after SAH. All three patients made excellent recoveries. There were two retraction hematomas in Grade 3 patients and two in Grade 4 patients; two made excellent recoveries and two died. All four of

### TABLE 7
Postoperative non-ischemic complications*

<table>
<thead>
<tr>
<th>Type of Surgery</th>
<th>Total Cases</th>
<th>Bleeding Same Aneurysm</th>
<th>Repaired Wrong Aneurysm</th>
<th>Epidural/ Subdural Hematoma</th>
<th>Retraction Hematoma</th>
<th>Seizures Focal</th>
<th>PLED's</th>
<th>Myocardial Infarction</th>
<th>Pulmonary Embolus</th>
<th>Korsakoff Psychosis</th>
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<tbody>
<tr>
<td>Pterional</td>
<td>409</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>9</td>
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<td>3</td>
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<tr>
<td>Bifrontal</td>
<td>89</td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Anterior subtemporal</td>
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<td>0</td>
<td>0</td>
<td>3</td>
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<td>4</td>
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* ICA = internal carotid artery; PLED's = paroxysmal lateralizing epileptiform discharges.

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these occurred in patients with red, angry brains which were operated on 7 to 10 days after SAH. Three of the seven hematomas developed in Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of the basilar artery. Death in these Grade 3 and 4 patients in whom deep retraction was required during modified pterional surgery for aneurysms at the caput of these patients was not considered to be related to the retraction hematomas.

Focal seizures occurred in 15 patients and paroxysmal lateralizing epileptiform discharges developed in seven patients (three patients also had generalized seizures upon emerging from anesthesia, but these were not related to postoperative seizures, and all three patients made excellent recoveries). Two patients with no SAH had focal seizures; these were related to a preoperative seizure focus and did not affect the ultimate recovery. However, three focal seizures occurred in Grade 1 patients, only one of whom eventually made an excellent recovery. The single Grade 2 patient with a focal seizure ultimately achieved a good result. The remaining focal seizures were found in Grade 3 patients and seemingly had no correlation with the degree of recovery finally achieved. Paroxysmal lateralizing epileptiform discharges occurred in three patients with no SAH, three Grade 3 patients, and one Grade 4 patient. These appeared to be unrelated to the level of recovery ultimately achieved. Seizures were most common in patients with medial temporal lobe retraction.

Three patients had myocardial infarctions; one of these was Grade 1, and two were Grade 2. One of these three patients was receiving Amicar (a Grade 2 patient).

Fourteen patients had unequivocal evidence of a major pulmonary embolism (all minor and question-
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Grade 2 patients studied 4 to 6 days after SAH, and in 30% of Grade 1 and 50% of Grade 2 patients 7 to 9 days post-SAH.

The type and severity of the spasm varied from case to case but could be conveniently classified as segmental or diffuse. Segmental spasm was limited to short sections of one or more vessels, and did not appear to slow the transit time of contrast media. Diffuse spasm extended over long segments of multiple vessels and slowed the transit time of the contrast material. Segmental spasm was found in 10 Grade 1 patients within 48 hours prior to operation, one of whom had a major but transient delayed ischemic event, and in nine Grade 2 patients, one of whom also had a major yet transient ischemic complication. Diffuse spasm was less common, being found in only two patients in each grade. It was associated with major postoperative delayed ischemia that culminated in two strokes and two deaths.

Communicating Hydrocephalus

Sixty-two patients (26 or 42% of whom had had more than one SAH) underwent ventriculoperitoneal shunting because of symptoms related to obstructive hydrocephalus. Four had not had a recent SAH, five were in Grade 1, 12 in Grade 2, 33 in Grade 3, and nine in Grade 4. Of these 62 patients, 13 ultimately achieved an excellent recovery and 15 a good recovery. Thus, 45% returned to employment or other productive activity.

Six of the 17 Grade 1 or 2 patients had been treated with Amicar preoperatively, which represents 7% of patients in these grades treated with Amicar. The remaining 11 patients had not been treated with Amicar (3% of those not receiving this drug). These differences are not statistically significant.

Mycotic Aneurysms

Six patients had an SAH from a mycotic aneurysm. Five of these had more than one bleed. Only two of these patients achieved a good recovery and in these two cases the aneurysm was excised.

Multiple Aneurysms

The 71 cases in which more than one aneurysm was repaired were in general poorer-grade candidates for surgery than the 573 cases in which one aneurysm was repaired. When patients were matched for grade and time of surgical intervention, the repair of more than one aneurysm did not appear to adversely affect the surgical result.

Discussion

Subarachnoid hemorrhage from an intracranial aneurysm results in a complex clinical picture often associated with many interrelated complications, including: cerebral edema, increased pressure from the volume of blood in the subarachnoid space or ventricle, chemical meningitis, obstructive hydrocephalus, an intracerebral hematoma, diffuse cerebral ischemia, and focal cerebral ischemia or infarction. To further confuse the clinical picture, there are differences in the patient's age and medical condition along with marked variations in aneurysm size, projection, and relationship to vital perforating vessels. All these variables are shadowed by the fear of a recurrent hemorrhage. It is, thus, difficult or impossible to formulate a precise protocol for the management of SAH from an intracranial aneurysm.

Analysis of Results

When analyzing the results of surgery in patients with an acute SAH, it is important to note that the patients were graded according to the Botterell classification with only minimal modification. Accordingly, our Grade 1 patients included those who would be Grade 1 and some patients who would be classified as Grade 2 using the Nishioka and Hunt and Hess systems. Similarly, our Grade 2 patients would correspond in general terms to Grade 3 patients using the other systems of classification.

One also should note differences in the grouping of patients in Tables 1 and 3. Thus, Table 1 relates only to the operative morbidity and mortality of a patient according to the grade of that patient at the time of surgery. Table 3 considers the result at hospital discharge according to the grade of the patient at the time of hospital admission, and includes death and morbidity during the preoperative period. Patients who were in Grade 1 or 2 at hospital admission and were then in Grade 1 at the time of surgery are included together in Table 1. However, a patient who was in Grade 1 on admission and then deteriorated to Grade 3 and underwent surgery as a Grade 3 patient would be included in the results of the Grade 3 group in Table 1, but in the Grade 1 group in Table 3. Nevertheless, recognizing these differences, the operative morbidity of 2% and mortality of 2% in Grade 1 patients must be seen in the perspective of an overall morbidity of 3% and mortality of 6% in patients who were in Grade 1 at the time of hospital admission. Similar observations can be made for the other grades of patients.

Preoperative Management

Approximately 30% of all patients admitted to our hospital had sustained more than one hemorrhage prior to hospital admission. The poorer-grade patients...
were more likely to have sustained multiple bleeds than were the better-grade patients. Many Grade 3 and 4 patients died before surgery from multiple factors related to the severity of their initial or recurrent hemorrhage. It is unlikely that surgery will favorably affect the outcome for these individuals.

Death and morbidity prior to surgery in good-grade patients with a recent SAH exceeded the risk of surgery itself. In Grade 1 patients, the primary cause of death or major morbidity was a recurrent bleed. In Grade 2 patients, deterioration from ischemia exceeded the risk of rebleeding following admission to our service. Death or deterioration before surgery in Grade 1 patients in the current series is higher than that previously reported. This can be attributed to earlier transfer by our referring physicians, with a resultant major increase in the proportion of patients being seen with a relatively recent bleed.

Operative Complications

As indicated in Tables 5 and 6, complications were related to both the size of the aneurysm and its location. Occlusion of or damage to a major vessel occurred primarily in large or giant aneurysms. Increased use of bypass grafting has lessened this risk, but by no means has it been eliminated.

Damage to perforating vessels remains a potentially major problem, and again was more frequent in large or giant aneurysms. In this regard, the tissue supplied by the anterior choroidal artery appears to be exquisitely sensitive to ischemia. To our knowledge, we have never occluded this vessel without creating a neurological deficit. Four of the nine patients with complications related to damage to perforating vessels underwent anterior choroidal artery occlusions. The ischemic tolerance for the tissue supplied by these perforating vessels undoubtedly varies from case to case. However, two of our patients recovered completely within 12 hours from the time the clip was repositioned and flow restored through the perforating vessel. In one patient the vessel had been occluded for 2 hours (anterior choroidal artery) and in the other the vessel had been occluded for 4 hours (large thalamoperforating vessel). We believe an anesthetic technique should be used for aneurysm surgery that permits prompt neurological evaluation at the conclusion of the procedure. Furthermore, a patient who, upon awaking from anesthesia, is found to have a significant neurological deficit not present prior to surgery should undergo immediate angiography or reinspection of the clip.

Postoperative bleeding from the aneurysm which had been repaired was usually attributable to the incomplete repair of the aneurysm and not to clip failure. We had only one case in which we were certain rebleeding had occurred as a result of clip slippage. Mycotic aneurysms have a tendency to rebleed after repair because of the infected character of the tissue, and we believe these aneurysms should be excised if possible. These are uncommon lesions and have a rather malignant natural history.

Retraction hematomas in this series were few in number. Those that did occur developed early in the series before skill with the use of the Yaşargil retractor had been acquired. Thereafter, the use of this retractor, along with spinal drainage and mannitol when required, seemingly prevented a complication of this sort.

Vasospasm and Delayed Ischemia

Vasospasm and the complications of ischemia and infarction related to it continued to be major problems in the management of patients with SAH. Angiographic spasm was not identified within the first 72 hours from the time of the initial SAH in any of our patients. These findings substantiate those of other workers and underscore the delayed temporal profile of this syndrome. Diffuse spasm leading to a reduction in the circulation time had a high correlation with a major deficit, but segmental spasm was seemingly more benign.

The key to successful management of vasospasm is early recognition and aggressive treatment in the initial stages of its evolution. The distinguishing feature of the ischemic syndrome that is attributed to progressive vasospasm preoperatively or postoperatively is the temporal profile of the neurological deficit with symptoms of either regional or general flow alterations, the former manifested by a focal deficit and the latter by an alteration in the level of consciousness. Characteristically, the evolution of the deficit is slow in comparison to the acute deficit seen with embolic occlusions, but there are exceptions.

It is probable that in all SAH patients there is a certain degree of spasm present that may or may not be symptomatic. Both laboratory and clinical studies have established the ability of the brain to function with a reduction in its blood flow to approximately 15 to 20 ml/100 gm/min without a major functional change. However, flows below 15 ml/100 gm/min result in rather rapid changes in function which parallel the relative severity of the reduction. Thus, a certain degree of angiographic spasm, even with a modest reduction in the circulation time of the contrast media, can be identified in patients without clinical symptoms of ischemia.

Vascular dilatation is an energy-dependent process. Up to the present time, no drug has been found to actively reverse the vasoconstriction of ischemic smooth muscle with damaged membranes and ionic transport systems, which were designed to function against a tremendous calcium gradient between the extra- and intracellular space. We have attempted to manage this complication by following measures designed to increase cerebral blood flow through changes in mean arterial perfusion pressure, cardiac output, and blood volume. There is little doubt that
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blood flow to ischemic regions can be increased by raising the mean perfusion pressure.\(^{46}\) The role of the cardiac output and blood volume is less certain, and may be related to their indirect effects on perfusion pressure.\(^{5,33}\) We have found no substitute for whole blood in the management of vasospasm. Blood volume expansion with plasma expanders is often unreliable as its agents do not remain within the vascular compartment. Overhydration in these patients runs a considerable risk of increasing cerebral edema which in effect decreases the intracranial perfusion pressure.

We believe that the following guidelines are sound for the management of vasospasm: 1) early recognition and institution of treatment as soon as a minor deficit is identified; 2) maintenance of a normal blood volume with whole blood or packed cells until a hematocrit of 40% to 45% is achieved; 3) maintenance of fluid restriction with a maximum limit of 1000 ml/24 hours; and 4) if all the above are ineffective, infusion of isoproterenol and lidocaine hydrochloride in an effort to increase not only the cardiac output but also the mean arterial perfusion pressure (the cardiac rate must be maintained under 100 beats/min).

Korsakoff Syndrome

The development of a Korsakoff syndrome in our experience was not a complication of vasospasm but rather a complication of structural damage to the anterior hypothalamus from the bleed itself, if present prior to surgery, or from surgical trauma, if noted only following surgery. The onset is acute and can be recognized immediately after surgery in a patient who is alert but confused, with major problems in recent memory which he attempts to mask by confabulation. Three of our patients exhibited this syndrome before surgery, but four additional cases occurred following operation for aneurysms of the ACoA complex. Only three of these seven patients ever achieved a full recovery with normal mentation and resumption of previous employment. This major complication may be related to damaged small vessels arising from the posterior aspect of the anterior communicating artery as suggested by Yaşargil and Smith\(^{44}\) and Perlmutter and Rhoton.\(^{29}\) We did note this complication more commonly when patients were operated on through a bifrontal craniotomy.

Seizures

The 3% incidence of this complication during the 1st week after surgery contrasts with that reported by Cabral, et al.,\(^4\) who found an incidence of approximately 6% within 1 week from surgery. Subsequently, they found a 28% incidence of a chronic seizure disorder. Although we have not arrived at a percentage for this complication in our follow-up group, we have been impressed that it is most uncommon. We have no explanation for these differences. We did note that the incidence of seizures within the first 2 weeks following surgery in patients operated on via the pterional approach was 2%, whereas the bifrontal approach carried a 6% risk, and the anterior subtemporal approach with retraction of the medial temporal lobe was associated with a 10% risk of seizures. Fortunately, these were usually isolated events, and chronic epilepsy was uncommon.

Timing of Surgery

The relatively small numbers of our cases operated on within 1 week from the time of the SAH makes it somewhat hazardous for us to proffer advice regarding the timing of aneurysm surgery. There are some excellent recent reviews and studies on this controversial subject.\(^{5,15,19,21,24,34,36,43,44,50}\) The decision pertaining to early surgery is dependent on a number of factors, for instance, aneurysm location and size, surgeon’s experience, availability of experienced anesthesia personnel, trained surgical nurses, and the emergency crew. Nevertheless, the greatest impact toward changing the morbidity and mortality of this illness would be derived from early surgery if this were not associated with too high a risk.\(^{29,47}\)

It currently appears to us that early surgery is justified in Grade 1 patients with an “uncomplicated” aneurysm in the anterior circulation. From our data, we have no recommendation to make regarding the time of surgery for Grade 2 patients. However, if early surgery is contemplated in these individuals, angiography on the day of surgery to identify the degree of spasm is probably helpful. The location of most posterior circulation aneurysms makes it wise to delay surgery in these cases until all effects of the acute hemorrhage have subsided. In most instances these lesions should probably be operated on in tertiary care facilities. The only indication for early surgery in Grade 3 patients is for a localized mass.

Amicar

Our data suggest that Amicar has a protective effect against a rebleed prior to surgery. This subject remains controversial, and our data are not conclusive as this was not a controlled study.\(^{16,23,35,37,30,38}\) Although others have not reported an increase in the incidence of thrombotic complications with the use of Amicar,\(^{25,30}\) this was not our experience. There was a statistically higher risk of postoperative pulmonary embolism in our patients who had received Amicar preoperatively in comparison to those who had not received this drug prior to surgery. We found no evidence that Amicar increased the incidence of communicating hydrocephalus.\(^{11}\)

Conclusions

The quality of life after an SAH is perhaps more important than the mortality rate. For this reason our categorization of results did not include a “fair” group. Any individual who is nonproductive and not able to function well in society is a poor result whether
or not he or she is fully ambulatory. In this regard, we have only rarely seen a patient who required intubation and respiratory resuscitative measures ever achieve a good result. It is our opinion that these measures should not be instituted without fully discussing the prognosis with the family.

There was an apparent progressive decrease in the frequency of the severe form of postoperative delayed ischemia throughout the period of this report, although more patients underwent early surgery later in the series and it is our experience that early surgical intervention increases the difficulty of the operation appreciably. In fact, this syndrome was recognized more frequently preoperatively than postoperatively in the last years of the study. Is this related to case selection, timing of surgery, changes in surgical technique, preoperative intuition, the routine administration of one unit more of blood at surgery than the estimated blood loss, or fortune? Throughout the period of the report, there was a reduction in the degree of brain retraction, the operating time, and the period in which hypotension was required. It is well known that major conducting vessels constrict in areas of ischemia. This has been referred to as a secondary vasospasm in contrast to the primary form that follows SAH, and presumably results from chronic (in contrast to acute) irritative effects of blood on the vessel wall or neurogenic mechanisms. Could it be that marginal ischemia from brain retraction, hypotension, or major vessel manipulation which, although not severe enough to be symptomatic by itself, augments and potentiates the primary form of vasospasm from an SAH and in this manner aggravates delayed ischemia? If this is the case, as we believe it is, then any report or analysis of aneurysm surgery is so inextricably entwined with surgical technique that generalizations such as ours can serve only as guidelines and not dicta.

Acknowledgments

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