SURGICAL TREATMENT OF ANEURYSMS OF THE ANTERIOR COMMUNICATING ARTERY

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A study of the literature shows that there has been increasing interest in the surgical treatment of intracranial aneurysms, largely because of the extremely poor ultimate prognosis of conservative management in cases of subarachnoid hemorrhage from this source. With accumulating experience, the surgical mortality is improving and more authors are coming to the opinion that surgical attack of one sort or another is the method of choice in the approach to this problem. Interest has therefore been aroused

TABLE 1

<table>
<thead>
<tr>
<th>Procedure</th>
<th>No. of Cases</th>
<th>Op. Deaths</th>
<th>Subsequent Death from Subarachnoid Hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ligation of carotid artery</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Exploration only</td>
<td>2</td>
<td>0</td>
<td>1 (1½ yrs. later)</td>
</tr>
<tr>
<td>Muscle or gelfoam packed around aneurysm</td>
<td>4</td>
<td>0</td>
<td>1 (gelfoam case, 4 wks. later)</td>
</tr>
<tr>
<td>Trapping</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Ligature of aneurysmal neck with thread or clip (in 1 case aneurysm then excised)</td>
<td>15</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Total 24 2 2

as to the types of procedure effective in these cases and the specific indications for each in an effort to combine maximum result with minimum of hazard.

Because of their unique location on the major anterior channel for collateral circulation in the circle of Willis, we feel that aneurysms of the anterior communicating artery represent a special problem in the surgical treatment of intracranial aneurysms. In spite of their infrequent separation as a distinct category in the literature, they are not properly comparable to other intracranial aneurysms either in assessment for surgery or in choice of surgical procedure to be employed.

The present material comprises 26 cases of aneurysms originating from

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the anterior communicating artery and represents the entire unselected material of this classification from this clinic up to April 1952. Of these patients, 24 were subjected to surgery of one sort or another (Table 1). It is the purpose of this paper to review the problem of the surgical treatment of aneurysms located on the anterior communicating artery, and in light of our experiences and those of others, to assess which approach may be the most rational and at the same time most rewarding.

INCIDENCE

The incidence of intracranial aneurysms in the general population has been estimated up to about 1 per cent,\(^1,2\) and in almost all recorded series\(^1,3,4,11,13,23,28\) aneurysms of the anterior communicating artery represent somewhere between 20–25 per cent of them. The 26 aneurysms of the anterior communicating artery here recorded represent slightly over 20 per cent of all aneurysms recorded at the Serafiner clinic since 1932.

The importance of recognizing any specific problems presented by such a group is thus clear.

ETIOLOGY

Views as to the etiology of these “congenital” aneurysms have varied. Forbus\(^2\) felt that their development was caused by the absence of media at the bifurcation of the arteries which led to a weak point that gradually gave way under the constant pulsating pressure of the blood within at these points. In support of his theory was the high incidence of aneurysms at the points of bifurcation on the circle. It did not, however, explain those aneurysms that occurred not at all infrequently at a point along the course of a vessel and well away from its bifurcation, most notably those on the proximal part of the carotid and, more germane to our present discussion, at the midpoint of the anterior communicating artery.

On the basis of some very convincing investigations into the embryology of the circle of Willis, Dorcas Padget\(^4\) pointed out that the development of aneurysms could more logically be traced to weak points occurring at the sites of incomplete resolution of previously existing embryonic vessels. From the anterior communicating artery in the embryo, there arises a smaller median anterior cerebral artery. This normally disappears, but De Vries\(^6\) found it to be present in a large percentage of human embryos and called it the “median artery of the corpus callosum.” In adult man it may anomalously persist as a single anterior cerebral artery with usually a relative disappearance of the two normally well developed anterior cerebral arteries. In some vertebrates, this median artery normally persists. It is the incomplete resolution of this artery that may be the point of origin of the aneurysms of the anterior communicating artery. In any event, Padget’s investigations led Dandy to the belief that this embryological explanation was the more logical, and Bassett\(^2\) has recently re-emphasized the point of etiology as most likely “unresolved vestiges of a primordially normal circulatory system” as opposed to the theory of Forbus. The present authors
are inclined also to this view. Of our 26 cases, in only 9 was there any question that the aneurysm arose from the communicans trunk well away from the point of junction of the communicans with either anterior cerebral.

**SYMPTOMATOLOGY**

It is beyond the scope of this paper to discuss symptomatology in detail and our series is hardly large enough to warrant conclusions. There are, however, one or two points that have struck us as characteristic and they are worth brief mention. As with other intracranial aneurysms, the usual story is of no symptomatology whatsoever until the occurrence of a sudden subarachnoid bleeding with its familiar and typical signs. Of our 26 patients 24 had subarachnoid hemorrhage and the number of episodes varied from 1 to 3. Paralytic signs were surprisingly frequent, occurring in 10; 4 of these patients had partial paralysis (arm and/or face or leg only). We were unable to correlate the side of paralysis with the projection of the aneurysm and were surprised to find that only 3 of the 10 had intracerebral hematomas that could explain the paralysis. Richardson and Hyland had noted focal signs in 62 of their 118 cases which in most instances were caused by concomitant intracerebral hematoma. The presence of a hematoma had also been felt by us to be the most likely etiology of a hemiparesis. A more interesting and suggestive correlation is that of actual narrowing of a vessel or nonfilling of a specific vessel at arteriography. Ecker and Riemenschneider hypothesized that this narrowing of a vessel in cases of aneurysms

![Fig. 1. Aneurysm of the anterior communicating artery. Note spasm of the carotid artery, middle cerebral artery and anterior cerebral artery (arrows).](image-url)
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is in all probability caused by arterial spasm, and postoperative angiograms in our series have demonstrated disappearance of this narrowing, tending to confirm their opinion (Figs. 1 and 2). The direct operative visualization of the anterior cerebral artery as a thin white thread in some cases in this series has suggested severe spasm or congenital defect as a possible explanation.

Of the 10 patients exhibiting paralytic signs, 7 had either arterial spasm or nonfilling of a vessel and in each instance the vessel involved was the one that anatomically would give rise to the symptomatology observed (Table 2). Thus, the patients with involvement of face and arm or leg alone showed spasm of the anterior cerebral artery or nonfilling on the opposite side (see also Ethelberg\textsuperscript{10}) and the patients with complete hemiparesis showed spasm of the carotid on the opposite side. Of the 3 patients who had hemiparesis and no spasm or nonfilling, 1 had an intracerebral clot of the opposite frontal lobe which could have accounted for the symptomatology. The etiology of the other 2 is obscure.

We are all aware of the limitations of arteriographic evidence of spasm of a vessel, especially when 35 per cent umbradil (diodrast) rather than thorotrast is used and are also sure that absence of filling of a vessel (especially the anterior cerebral) on one examination cannot be used as an indication of its being in spasm, congenitally absent or thrombosed. However, the correlation of direct visualization of arterial spasm and/or nonfilling and the signs resulting indicate to us that this is more than coincidence. We would therefore suggest that the focal paralytic signs seen in our cases were caused by vasospasm secondary to the existence of the aneurysm, the spasm possibly being a protective mechanism after bleeding. It will be of
interest to observe if future larger series can confirm this hypothesis.

As may be seen in Table 2, there were 6 cases in which there was arterial spasm of varying degree without clinical hemiparesis, but we would interpret this as being related to the duration and degree of spasm necessary to cause functional damage to cerebral substance. Also, in 5 of the 8 cases of nonfilling there were no clinical signs. It is obvious that there are some difficulties in correctly interpreting the significance of spasm or nonfilling of a vessel. For the reasons outlined above, however, we feel that there is a cor-

<table>
<thead>
<tr>
<th>Case No.</th>
<th>True Vasospasm</th>
<th>Nonfilling</th>
<th>Paralytic Sign</th>
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</thead>
<tbody>
<tr>
<td>2</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>—</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>—</td>
<td>—</td>
<td>Slight lt. hemiparesis</td>
</tr>
<tr>
<td>5</td>
<td>Lt. carotid</td>
<td>—</td>
<td>Rt. hemiparesis</td>
</tr>
<tr>
<td>6</td>
<td>Rt. carotid</td>
<td>—</td>
<td>Lt. hemiparesis</td>
</tr>
<tr>
<td>7</td>
<td>Rt. carotid</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>—</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>Rt. carotid and middle cerebral</td>
<td>—</td>
<td>Lt. hemiparesis (temporary)</td>
</tr>
<tr>
<td>10</td>
<td>—</td>
<td>—</td>
<td>Lt. hemiparesis</td>
</tr>
<tr>
<td>11</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
<td>Lt. leg</td>
</tr>
<tr>
<td>12</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>14</td>
<td>—</td>
<td>Rt. ant. cerebral</td>
<td>Lt. arm and face</td>
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<tr>
<td>15</td>
<td>—</td>
<td>Rt. ant. cerebral</td>
<td>Lt. arm</td>
</tr>
<tr>
<td>16</td>
<td>Lt. ant. cerebral</td>
<td>—</td>
<td>Lt. arm</td>
</tr>
<tr>
<td>17</td>
<td>—</td>
<td>Rt. ant. cerebral</td>
<td>—</td>
</tr>
<tr>
<td>18</td>
<td>—</td>
<td>—</td>
<td>Rt. hemiparesis</td>
</tr>
<tr>
<td>19</td>
<td>—</td>
<td>—</td>
<td>Slight rt. hemiparesis and sl. aphasia</td>
</tr>
<tr>
<td>20</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>21</td>
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<td>—</td>
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<tr>
<td>22</td>
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<td>—</td>
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</tr>
<tr>
<td>24</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>8</td>
<td>10</td>
</tr>
</tbody>
</table>

relation of significance here, although actual proof of this must await further evidence.

We found absolutely no correlation between the site of vasospasm and the projection of the aneurysmal sac—that is to say an aneurysm projecting to the right was no more likely to produce spasm of the right anterior cerebral than one projecting to the left.

The sudden onset of blindness in one or the other eye was rather prominent as a finding, occurring in 7 (25.9 per cent) of our cases. This type of complaint seems rather characteristic for aneurysms in this location and we interpret the mechanism as one of sudden expansion of the aneurysmal sac with encroachment on the optic nerve.
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ARTERIOGRAPHY

The necessity for complete bilateral carotid angiography in all of these cases cannot be overemphasized. This has been stressed also by List and Hodges, Poppen, and Löfstedt. That angiography is not without its dangers is, of course, well accepted and the complications of the procedure have been much discussed, most recently by Dunsmore, Scoville and Whitcomb. Two of our patients were worse after angiography. If, however, one intends to practice surgery in these cases, it is a rather small risk to take compared to the vital information obtained. Firstly, the co-existence of multiple aneurysms is well known. Dandy found multiplicity in 15 per cent of his 108 patients, although Ask-Upmark and Ingvar have rightly stated that there is "usually" only one. We are aware that angiography does not always demonstrate the aneurysm or all of the aneurysms. However, the angiographic evidence of multiplicity will be a deciding factor in which procedure to employ or if any at all should be attempted. None of our cases of anterior communicating artery aneurysm showed multiplicity.

It is now generally conceded that angiography early after an episode of hemorrhage does not essentially increase the morbidity, and the shortness of the interval from the acute episode should not influence performance of this examination. Our 2 patients who were worse after angiography had it performed 1½ weeks and 8 weeks respectively after the last episode of bleeding.

Secondly, it is imperative to establish the exact state of the collateral circulation between the hemispheres and to exclude anomalies of the circle of Willis before deciding to attack the aneurysm. This is especially important if carotid surgery or trapping is contemplated and also aids in an emergency decision should uncontrollable hemorrhage from rupture at operation occur. Padget noted that in individuals with aneurysm, variations in the circle of Willis were about 2 times as frequent as in those without aneurysm. She felt that there was a high percentage of absent arteries in these cases. Jefferson believes that the circle of Willis is incomplete in about 8 per cent of normals. In Falconer's series, 5 out of 11 patients with aneurysm in this location had arteriographic evidence of anomalies of the circle of Willis "such that distal to the communicating artery, both anterior cerebials were supplied by one carotid artery only, suggesting a deficiency in the circle of Willis between the anterior communicating artery and the internal carotid artery of the opposite side." One of these patients died with bilateral frontal lobe infarction after carotid ligation.

That the aneurysm itself may not fill from both sides appears to be the usual occurrence (Fig. 3). Of 20 cases in which bilateral arteriography was done, there were only 2 in which the aneurysm filled from both sides, the marked predominance of unilateral filling being from the left carotid (12 out of 18). The significance of this last observation is not clear, but is rather striking.

Thirdly, it is by angiography alone that the size, configuration, extension,
and position of the aneurysm itself can be determined—information of great value in the planning of a surgical approach. Clinical signs alone may be misleading as to location of the aneurysm as shown in our cases of hemiparesis. Walsh and Love have noted ocular nerve palsy on the side opposite to an aneurysm and Jefferson noted unilateral blindness on the contralateral side. Elvidge and Feindel have mentioned the possibility of EEG helping in the laterization, but have also pointed out that clinical and pneumographic evidence may indicate the wrong side because of a hematoma, which is not necessarily on the same side as the lesion producing it. This ability of midline rupture to go to either side is exemplified when we consider that the anterior cerebral arteries may lie one above the other as they pass upwards between the hemispheres, and hence either may rupture into the brain substance of either side.

Of the 19 cases of this series in which arteriography disclosed a definite projection of the sac, the projection was downwards and forwards in most instances, as in Falconer's cases. We were unable, however, to correlate projection with symptomatology. The aneurysm was demonstrated in all 26 cases by angiography and was clearly stated to be anterior communicating in location by the roentgenologist prior to operation.

MANAGEMENT OF CASES

Results of Conservative Management. The prognosis of subarachnoid hemorrhage has been studied by several investigators. Ask-Upmark and Ingvar, in a study of 138 patients, found that about 32 per cent died in their first attack and about 32 per cent had recurrence of hemorrhage, the mortality being higher at the second bleeding. They have also made what we consider a most important observation as to time of death, i.e., that death
from one subarachnoid hemorrhage may be in the first 24 hours or as late as the 25th day, the average length of time being the 9th day after the episode. This is a factor worth considering in evaluating procrastination in active therapy, once decided upon. They concluded from their own experience and a review of the literature that a patient with subarachnoid hemorrhage has only 1 chance in 5 of a good recovery. He has 1 chance in 5 of being permanently crippled and 3 in 5 of dying sooner or later from his disease.

This gloomy outlook of high mortality, frequent recurrence and increasing mortality with recurrence has been confirmed by others.\(^4,13,24,33\) Hyland\(^16\) reported in 1950 that of 191 patients with subarachnoid hemorrhage 100 died within 6 months of the onset of the attack. Falconer\(^11\) has concluded from the literature that the mortality of conservative treatment of spontaneous subarachnoid hemorrhage is 50–60 per cent. He tabulated the figures of Taylor and Whitfield, Hamby, Hyland, Magee, and his own series to support this, and also showed the high incidence of recurrent attacks in patients \(while\ under\ observation\) (46 per cent of his and 51 per cent of Magee’s patients). Again, the time factor seems to us to speak against the advisability of procrastination.

It has been the hope of conservative management that “healing” would take place at bed rest with abstinence from straining, etc. or that the aneurysm itself would thrombose. The above figures would seem to belie this hope. Dandy\(^4\) noted that 2 of his series and several from the literature had \(almost\) filled with thrombus, but eventually ruptured from the small remaining lumen. He concluded that not more than 15–20 per cent of hemorrhages are cured permanently by thrombosis.

**Results of Surgical Management.** Although the results of surgical treatment of aneurysms are not yet brilliant, they are with experience constantly improving and, as compared with the results of conservative management, they are encouraging. We do not believe that a truly precise comparison between a series of patients with subarachnoid hemorrhage treated surgically and those handled by conservative therapy is possible at the present time. Falconer\(^11\) has also pointed out the difficulties in obtaining series that are reliable for such a comparison but is of the opinion that in his series of 50 surgically treated bleeding aneurysms the mortality would have been much higher than 18 per cent had they been conservatively treated.

From different statistics on the prognosis of subarachnoid hemorrhage it is quite obvious that the time factor is of utmost importance. Actually, we are faced with two problems. The first is how to treat these patients in the acute stage of hemorrhage. The second problem is what should be done in the free interval, i.e. how to prevent recurrent bleeding. The acute stage covers about the first 3 weeks and corresponds to the period of vascular spasm.\(^7\) It is the period of highest mortality.\(^1,28\) In any analysis that deals with this question, it is therefore necessary to consider the interval between the bleeding and the operation.
Any surgeon is naturally disinclined to operate on a critically ill comatose patient and wonders if it is not wiser to wait until the general condition improves before subjecting him to the additional stress of a major procedure. On the other hand, the high mortality in the first 24–48 hours of all cases as well as the frequent recurrence with death even in patients doing well under observation leads one to a consideration of whether these patients should not be operated upon as soon as possible after their episode of hemorrhage. The optimal time factor for operation has recently been considered in detail by one of us.26 As previously mentioned, early clinical symptoms may perhaps result from spasm of a vessel. We are convinced that in many cases the vasospasm is a factor in the coma following subarachnoid hemorrhage.

Surgical Procedures

Carotid ligation does not at this stage of experience seem to us a very rational procedure for aneurysms of the anterior communicating artery, in spite of what its effect may be on aneurysms of other locations. Ligation of one carotid can hardly be expected to reduce the blood flow more than temporarily in the communicating artery which under normal conditions potentially receives its flow from both sides. In the event that this were not so because of poor collateral circulation, ligation of one carotid would carry extreme hazard to the nutrition of the cerebral hemisphere on that side. However, Krayer61 reported recovery from a bitemporal hemianopsia after carotid ligation in a patient with anterior communicating artery aneurysm, and Walsh and Love32 ligated the right internal carotid in a patient who had had 2 episodes of bleeding from an aneurysm at the junction of the right anterior cerebral and the communicans with good results 3 months later.

On the other hand, Falconer11 tried carotid ligation alone in 3 patients with anterior communicating artery aneurysm and 2 died. One had a vascular anomaly of the circle of Willis and ligation resulted in infarcts of both frontal lobes. In the other the choice of the side of ligation was determined by the presence of an intracerebral hematoma on that side, which actually had arisen from the opposite anterior cerebral. Moreover, he admitted that occlusion of one carotid can only temporarily decrease the pressure in the communicating artery, especially if the aneurysm can be filled from either side, and concluded that a direct intracranial approach was the method of choice for aneurysms in this location. The attractions of carotid surgery have been that it is easy to do, there is less operative risk, and there may perhaps be benefit. The use of carotid surgery alone, however, precludes the evacuation of intracerebral hematoma, which we feel of importance for the most favorable outcome in any case. Four of our 22 intracranially explored patients had intracerebral clots and 3 of these 4 were the most critically ill of our series. The 4th was disoriented prior to evacuation of the clot. Two clots were frontal, 1 was callosal, and 1 hypothalamic.

Very few aneurysms of this location operated upon by direct surgical
attack have been reported. Dandy had only 4 in his group of anterior communicating and anterior cerebral artery aneurysms. Of Falconer's 11 cases a direct attack was undertaken in 8. Single cases of successful operation have been reported by Tönnis, Russel, and Elvidge and Feindel.

In discussing the surgical procedure in these cases it should be mentioned that since August 1950, when a more active attitude towards these lesions was instituted in this clinic, in practically all cases induced hypotension by spinal anesthesia or by hexamethonium has been used during the operation. We are of the opinion that this has been of extreme value.

Unless a left-sided hematoma is suspected, we have preferred to use a high right frontal flap. As has been pointed out by Falconer, some aneurysms of the anterior communicating artery project forwards and downwards and some upwards and backwards. The first type can be exposed by

![Image of an operation with an aneurysm and drawings indicating the approach.]

**Fig. 4.** In this photograph of an operation the aneurysm with the ligature on the neck is clearly seen localized above the chiasma and optic nerves. The drawings indicate the approach.
a subfrontal approach (Fig. 4), which was used in 6 cases. In the second type it is necessary to use a vertical approach down along the falx as described by Tönnis. This approach was used in 10 cases (Fig. 5). Occasionally exceptionally large veins running to the sagittal sinus may be encountered anteriorly, and these must be ligated. This may cause swelling of the frontal lobe and in 1 of our cases re-operation became necessary for resection of the edematous frontal lobe. In some cases, therefore, resection of the frontal pole has been performed at once to avoid the risk of subsequent edema, a factor which might tip the balance in a critically ill patient. This was done in 6 of our 22 intracranial procedures without persisting frontal lobe symptoms. One patient was euphoric for about 2 weeks and another slightly apathetic 5 months later. On the other hand, it was found that with ligation of veins
and retraction of the frontal lobes, 1 of our patients had marked symptoms of impaired function of the frontal lobes, especially of the memory functions. Whether a small resection of the right frontal pole causes less damage than ligation of parasagittal veins on the right side and retraction of frontal lobes has not yet been decided. This question is being investigated by our psychiatrist.

The procedure employed in relationship to the aneurysm itself again must depend upon the operative findings. It is our opinion that direct clipping of the neck of the aneurysm or ligating it with a thread has been found in some cases the procedure of choice, although naturally with an aneurysm firmly bound by adhesions this is more hazardous than packing about the sac with muscle and/or gelfoam, and the latter procedure may be the more prudent in some cases. The objection has been raised that dissection of the neck of the aneurysm carries a great risk of rupture during the procedure. This occurred in 2 of our 22 cases of intracranial operation and both patients survived. We have found the use of controlled hypotension by hexamethonium compounds of extreme value in the handling of bleeding in all cases of aneurysm and especially in those in which rupture occurred. Direct ligation or clipping of the neck was performed in 15 of our cases with only 1 fatality in a patient who was comatose and in shock at the onset of the procedure. Jefferson, in discussing a recent paper of Norlén, raised the question of how many aneurysms truly have a neck that is approachable and how many are merely arteriographic interpretations of a "neck" that does not exist. It is interesting to note that in this series the predominance was rather the other way—i.e., in 5 cases in which arteriography had disclosed no neck or only a very slight narrowing it was possible by careful operative dissection to free a neck and occlude it. In all other cases except 3, a neck was found and occluded when the arteriogram indicated one (Fig. 7). In 2 of these 3 cases gelfoam was packed about the sac since it was felt hazardous to attempt dissection because of dense adhesions. In the third, exploration did not reveal the aneurysm itself and no procedure was performed. It should be noted that in 1 of the cases in which gelfoam was packed about the sac, the patient died 4 weeks later from a recurrence of subarachnoid hemorrhage. In this case, gelfoam packing was obviously not protective.

The packing of muscle about the aneurysmal sac as early advised by
Dott does not strike us as entirely secure either, although Falconer\textsuperscript{11} seems to prefer it as a safer procedure, adding a gauze wrapping to his muscle bolster. He performed a direct approach in 8 out of the 11 cases of anterior communicating aneurysm with 6 good recoveries, 1 recovery with mental impairment (in which the anterior cerebral artery was clipped), and 1 death from recurrent bleeding. The only patient who showed recurrence of bleeding and died had had muscle wrapped about the aneurysm. Dandy\textsuperscript{4} was opposed to relying on muscle for support. Certainly, as Elvidge and Feindel\textsuperscript{9} have re-emphasized, a muscle protection can hardly be expected safely to resist the pressure in an aneurysm when one considers the work of Horsley,\textsuperscript{16} who placed the limit of safety of muscle tamponade at about 60–80 mm. Hg. Another factor that to us mitigates against the safety of muscle is the thinness of the aneurysmal sac itself. Forbus,\textsuperscript{12} Ectors,\textsuperscript{8} Hermann and Macgregor,\textsuperscript{14} and Elvidge and Feindel\textsuperscript{9} have all emphasized the lack of muscle and elastic tissue in the wall of the sac, the last pointing out that such a wall is rather hazardously supported by muscle packing alone. This doubt has not only been confirmed clinically by Falconer’s above-mentioned cases, but by Jefferson,\textsuperscript{17} who reported a case of anterior cerebral artery aneurysm packed with muscle, with symptomatic improvement but fatal rupture 18 months later. In another communication\textsuperscript{18} he referred to 2 other cases; in one, muscle packed around an aneurysm resulted in a fatal rupture 1 month later, and in the other, carotid ligation was performed 2 months after muscle packing because of continuing pain, which to him indicated activity of the aneurysm. Muscle packing about the aneurysmal sac was performed in only 2 of our cases; both patients have thus far had a satisfactory result. Of 2
patients who had gelfoam packing, 1 died from new bleeding 4 weeks later.

Complete resection of the aneurysmal sac is often advisable when it has enlarged to the point of causing local pressure symptoms. This procedure is, however, probably the most hazardous. Russell reported on a complete excision of an aneurysm in this region by Cone. This was done in 1 of our cases with good result. The danger and difficulty of the procedure in most instances are its main drawbacks.

Trapping of the aneurysm by occlusion of the communicating artery from the circulation is a satisfactory procedure provided that full angio-
graphic studies have demonstrated that no anomalies of the circle of Willis exist and that each carotid is able to take care of the blood flow to its own hemisphere without the aid of collateral circulation from the opposite side. Indeed, this procedure may then be safer and more certain, and it is particularly of value when no aneurysmal neck exists. It is, however, not without its dangers. In Elvidge and Feindel's case of anterior communicating aneurysm trapping was performed with 6 clips on the aneurysm, anterior communicating, and anterior cerebral. The resultant clinical defects, seizures, and evidence of frontal lobe damage in their case point out the in-
 advisability of sacrificing an anterior cerebral. Trapping was done in only 1 of the present series, a case in which the aneurysm represented a diffuse dilatation of the anterior communicating artery with no neck whatsoever. This patient was critically ill at operation and died the following day. This single case is not, of course, interpreted as indicative of the value of the procedure.

RESULTS

In our 26 cases of anterior communicating artery aneurysms (Table 1) 24 were surgically treated, with 2 operative fatalities, and 1 subsequent death from subarachnoid hemorrhage in a case in which gelfoam had been packed about the aneurysmal sac. One other patient died from subarachnoid hemorrhage 1½ years later in a case early in the clinic experience, in which an exploratory craniotomy but no surgical treatment of the aneurysm had been performed.

The time that has elapsed since operation is in most of these cases too short to permit any conclusions regarding the late results. Of the 15 patients who were treated by occlusion of the neck of the aneurysm, 14 survived the operation. None of these 14 patients showed any sign of recurrence of bleeding. Five returned to work and 2 of these had had impaired vision which improved considerably after the operation. Five left the hospital without any neurological signs and are well but have not yet started to work though they are supposed to do so. Of 2 patients with hemiplegia before operation, 1 is improving and 1 is unchanged. In 1 case, hemiplegia appeared some days after operation, probably as a result of thrombosis; some improvement has occurred, but the patient is still hemiparetic. In 1 case, as previously men-
tioned, there was pronounced postoperative impairment of memory func-
tion; some improvement has occurred.
We have tabulated the time interval from bleeding to operation, the condition of the patient at operation, and the operative mortality in Table 3. Our series is too small to draw any logical conclusion and is weighted as to time in that not more than 6 patients were operated on during the first 6 weeks after hemorrhage, because of problems of transportation and bed capacity. It is included here only to record the data so that others in the future may add them to their experience in attempting to answer this difficult question of when to operate and when to wait. The patients who survived for longer intervals were, as would be expected, in better condition and did well. How many patients might have been referred had they survived conservative management we cannot say (nor, in fairness can we say how many would have died from early surgical intervention). It is an important problem which needs clarification.

In summary then, it would be our conclusion that a direct intracranial approach with ligation of the neck of the aneurysm (when possible) is the

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**TABLE 3**

*Time interval from bleeding to operation and condition at operation compared with operative mortality in 22 cases of subarachnoid hemorrhage surgically treated*

<table>
<thead>
<tr>
<th>Interval from Bleeding to Operation</th>
<th>No. of Cases</th>
<th>Condition at Operation</th>
<th>Operative Risk*</th>
<th>Operative Death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Comatose</td>
<td>Subcomatose</td>
<td>Operative Risk</td>
</tr>
<tr>
<td>Up to 6 days</td>
<td>4</td>
<td>1</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>+</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>+</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1½–3 weeks</td>
<td>2</td>
<td>1</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td>1–3 months</td>
<td>9</td>
<td>1</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Over 3 months</td>
<td>7</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td></td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

* Classed 1 to 5, with 1 a perfect operative risk and 5 the poorest operative risk.
procedure of choice for aneurysms in this specific location. The other procedures, especially trapping, have their indications and contraindications, but on the whole we would echo the words of Dr. Rudolph Jaeger of Pennsylvania who in discussing a paper of Hamby remarked, "The aneurysm must be attacked directly and tied off. This is the only specific treatment for an intracranial aneurysm."

SUMMARY

1. Because of their location on the major vessel for collateral circulation in the anterior part of the circle of Willis, it is felt that aneurysms of the anterior communicating artery present certain specific problems in management different from other intracranial aneurysms.

2. A series of 26 cases of anterior communicating artery aneurysm is presented, this representing the entire unselected material of this category from this clinic up to April 1952.

3. With the experience in this series as a background, the problems of incidence, etiology, symptomatology, and management of this type of case are discussed. Special attention is drawn to vasospasm in relation to subarachnoid hemorrhage and its clinical importance.

4. The need for complete angiographic evaluation of all cases prior to surgical intervention is stressed.

5. Because of the poor outlook of conservative therapy, it is concluded that these aneurysms are best handled by a direct surgical approach. It is concluded that when possible a direct attack on this type of aneurysm with occlusion of its neck by clip or ligature is the procedure of choice. The optimum time for surgery following the hemorrhage is discussed.

6. In this series of 24 surgical cases only 2 could be considered as operative deaths. In addition there were 2 unselected deaths from subarachnoid hemorrhage ascribed to what we consider inadequate procedure.

REFERENCES


30. Sjögren, O. Personal communication.

