Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex

DAVID PERLMUTTER, AND ALBERT L. RHOTON, JR., M.D.

Neurological Surgery, University of Florida Health Center, Gainesville, Florida

The microvascular relationships important to surgery of aneurysms in the anterior communicating region were defined in 50 cadaver brains. The recurrent artery of Heubner was frequently exposed before the A-1 segment in defining the neck on anterior cerebral aneurysms because it commonly courses anterior to A-1. It arose from the A-2 segment of the anterior cerebral artery (ACA) in 78% and most commonly terminated in the area of the anterior perforated substance, and lateral to it in the Sylvian fissure. The anterior communicating artery (ACoA) frequently gave rise to perforating arteries which terminated in the superior surface of the optic chiasm and above the chiasm in the anterior hypothalamus. This finding contrasts with previous reports that no perforating branches arise from the communicating artery. The proximal half of the A-1 segment was a richer source of perforating arteries than the distal half. The A-1 branches most commonly terminated in the anterior perforated substance, the optic chiasm, and the region of the optic tract. The ACoA increased in size as the difference in the diameter between the right and left A-1 segments increased. Frequent variants such as double or triple ACoA's, triple A-2 segments, and duplication of the A-1 segments were encountered. The clinical consequences of occlusion of the recurrent artery and of the perforators from the ACoA and medial and lateral segment of A-1 are reviewed.

KEY WORDS • microsurgical anatomy • anterior cerebral artery • recurrent artery of Heubner • circle of Willis • intracranial aneurysm

Of aneurysms arising in the anterior half of the circle of Willis, those originating on the anterior communicating artery (ACoA) are regarded as the most complex. This complexity is caused by frequently associated variants of normal anatomy, the multiple directions in which the aneurysm may point, difficulties in fully visualizing the normal ACoA and adjacent vessels on angiography, and incomplete angiographic filling of the area due to spasm. In addition, arteriosclerotic tortuosity and elongation may distort normal anatomy. The risks of disregarding the smaller arteries and arterioles in this region include altered personality, memory deficits, hemiplegia, aphasia, visual field defects, and even death. Magnification now makes it possible to visualize and preserve adjacent small vessels in aneurysm dissection. The purpose of this study was to define the microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent arterial complex and its smaller branches.

Method and Material

In 50 adult brains removed at autopsy the circle of Willis was examined under 3× to 40× magnification with special attention
directed to the anterior cerebral artery (ACA), the ACoA, the recurrent artery of Heubner, and the small arteries arising from the basal portions of the anterior cerebral complex. The segment between the internal carotid and the ACoA is referred to as the A-1 segment, and the segment just distal to the ACoA as the A-2 segment. Only the A-1 segment and the first 5 mm of the A-2 segment were examined. The sites of termination of the basal branches of A-1, the proximal 5 mm of A-2, the ACoA, and each branch of the recurrent artery of Heubner were recorded. The sites of termination of up to 40 small arterial branches were plotted in some brains.

Results

Anterior Cerebral and Anterior Communicating Arteries

Each ACA arises as the medial component of the carotid artery bifurcation and courses over the superior surface of the optic chiasm or nerves to join the ACoA (Fig. 1 A to I). The junction of the ACoA with the right and left A-1 segment was usually above the chiasm (70%) rather than above the optic nerves (30%); of those above the optic nerves, 23% were above the half nearest the chiasm and 7% above the distal half (Figs. 2 A and B). The shorter A-1 segments were stretched tightly over the chiasm; the longer ones passed anteriorly over the optic nerves. The arteries with a more forward course were often tortuous and elongated with some coursing forward and resting on the tuberculum sellae or planum sphenoidale (Fig. 1 I). The A-1 segments varied in length from 7.2 to 18.0 mm (average 12.7 mm). The length of the ACoA was usually between 2 and 3 mm: it varied from 0.3 to 7.0 mm (average 2.6 mm). The longer ACoA’s were curved, kinked, or tortuous.

A normal ACA-ACoA complex has been defined as one in which a communicating artery connects A-1 segments of equal size, and both A-1 segments and the ACoA are of sufficient size to allow circulation between the two carotid arteries and through the circle of Willis. In this study, the average diameter of the ACoA was approximately 1 mm less than the average diameter of the A-1 segment. The diameter of the A-1 segments varied from 0.9 to 4.0 mm (average 2.6 mm). The ACoA diameters varied from 0.2 to 3.4 mm (average 1.5 mm), and were equal to or larger than the smaller A-1 segment in only 26% of the brains examined (Figs. 2 and 3).

Abnormalities of the circle of Willis usually consist of a stringlike or absent component (Fig. 2). The diameter below which a vessel would be called hypoplastic has not been defined previously but a diameter below 1.5 mm is much smaller than average and use of this figure yielded an incidence approximating those of other studies. In this study, 10% of the brains examined had an A-1 segment of 1.5 mm or less in diameter and only one of 50 brains had an A-1 segment with a diameter of 1.0 mm or less. In 44% of the brains examined, the diameter of the ACoA was 1.5 mm or smaller and in 16% it was 1.0 mm or smaller. Alpers, et al. found stringlike components in 28% of 350 circles of Willis; the A-1 segments were a favorite site for such hypoplasia. Riggs and Rupp found hypoplasia of the primary stem of one of the paired ACA’s in 7% of 1647 circles of Willis and an ACoA so small that it would restrict circulation between the carotids in 6%.

The correlation between abnormalities of the circle of Willis and cerebrovascular disease is great; it is most outstanding in the anterior portion of the circle where hypoplasia of an A-1 segment has a high rate of association with anterior communicating aneurysms. Stehbens stated that hypoplasia of the A-1 segment was the only anatomical variant that correlated with the location of cerebral aneurysm. Wilson, et al. found that 85% of 40 aneurysms of the ACoA were associated with hypoplasia of the first portion of one ACA and were located at a site of focal increase of resistance. Whether one believes aneurysms are caused by congenital defects such as vestiges of the primitive embryonic vessels, or media defects common at bifurcations of cerebral vessels, or by postnatal changes such as progressive fragmentation of the elastic layer occurring at bifurcations, the obvious hemodynamic implications of this observation are of interest. It appears that a malformation in structure, such as asymmetry of the anterior circle of Willis, by producing local alterations in intravascular hemodynamics, may provide a mechanical basis for the development of aneurysms. Sahs and Meyers noted that hemodynamic factors may exert unusual stress on certain apical regions; others reasoned that
FIG. 1 (continued). Three anterior views of A-1 and proximal A-2 segment of the ACA, ACoA, and recurrent arteries. Gyrus rectus, olfactory tract, and frontal lobe above; optic nerves and chiasm below. Arterioles to optic nerves, chiasm and tracts, and lamina terminalis arise from the ACA and ACoA. D. A-1 segments are connected by double communicating artery. Right recurrent artery arises from A-2 and courses above A-1. Left arises from A-1 and passes anterior to A-1. Multiple arterioles pass to optic chiasm and tract. E. A-1 segments are connected by double communicating artery. Two recurrent arteries arise on right; one arises proximal and one distal to the communicating. Left recurrent artery arises from posterior aspect of A-1. Spray of arterioles passes from communicating to optic chiasm. F. Multichanneled ACoA gives rise to multiple arterioles to the optic nerves and chiasm and lamina terminalis. Double recurrent artery on right. Left recurrent gives rise to a large branch which passes below gyrus rectus to frontal tip.
Anterior cerebral-anterior communicating anatomy

Fig. 1 (continued). Three anterior views of A-1 and proximal A-2 segments of the ACA, ACoA, and recurrent arteries. Gyrus rectus, olfactory tract, and frontal lobe above; optic nerves and chiasm below. Arterioles to optic nerves, chiasm and tracts, and lamina terminalis arise from the ACA and ACoA. G. Left A-1 segment is split into a double channel. Both A-2 segments arise predominantly from right A-1. Left recurrent artery arises from one of the two A-1 segments on left. Right recurrent artery arises from A-2. H. Triple A-2 segments arise from communicating artery area. Left recurrent artery arises from A-1 segment; right from junction of A-1 and A-2 segments. I. Tortuous A-1 segments loop forward to area of tuberculum sellae. Left recurrent artery arises from A-2 segment and right from A-1.
D. Perlmutter and A. L. Rhoton, Jr.

FIG. 2. Left: Tortuous anterior cerebral-anterior communicating complex loops anterior to optic chiasm between optic nerves. Large right A-1 supplies both A-2 segments. Proximal A-2 on left gives rise to a branch to gyrus rectus medial to olfactory nerve (CNI). Right: Arteries lifted away from optic chiasm and nerves show small left A-1.

Aneurysms favor the anterior communicating location because the anterior wall of the ACoA receives the greatest force of the A-1 pulse wave. Most aneurysms in this area project anteriorly.

It has been observed that there is a direct correlation between the difference in size of the right and left A-1 segments and the size of the ACoA. As the difference in diameter between the A-1 segments increases, so does the size of the ACoA. Thus, a large ACoA was often associated with a significant difference in diameter between the right and left A-1 segments. This is understandable from a functional point of view because, with a small or hypoplastic A-1 segment, more collateral circulation flows across the ACoA to make up the deficit. A difference in diameter of 0.5 mm or more between the right and left A-1 segment was found in 50% of the brains examined and a difference of 1 mm or more was found in 12%. The average ACoA diameter was 1.2 mm in the group of brains in which the difference in diameter between the right and left A-1 segments was 0.5 mm or less; it was 2.5 mm if the difference was greater than 0.5 mm. This correlation between the size of the A-1 segments permits a rough estimate of the size of the ACoA even though the artery is not visualized because it is the most difficult part of the circle of Willis to define on cerebral angiography.

Another difficulty in angiographically defining the ACoA is that it may not be oriented in a strictly transverse plane, as one would expect from many descriptions. The length of the ACoA was found to be oriented in an oblique or straight anteroposterior plane if one ACA passed between the hemispheres behind the other. The ACA's were side by side as they passed between the cerebral hemispheres in only 18%; the left was anterior to the right in 48% and the right anterior to the left in 34% (Figs. 4 and 5). These variations may explain why angiography in the oblique positions may outline the artery if it is not seen on conventional lateral and anteroposterior views.

The ACoA usually has a round appearance but it may appear flat because of a broad connection with both ACA's or even triangular with a large base on the ACA and a threadlike connection on the other. The diameters of the right and left junctions of the ACoA with the A-1 segments were equal in 74%, greater on the right in 14%, and greater on the left in 12%.

One ACoA was present in 60%, two in 30%, and three in 10%. Double ACoA's could

FIG. 3. A-1 segments and communicating artery are of equal size. Left recurrent artery and a branch passing to frontal tip arise from A-1. Right recurrent artery arises at level of the communicating artery.
Anterior cerebral-anterior communicating anatomy

Fig. 4. Multiple small arteries passing to superior surface of optic chiasm arise from ACA's. Broad communicating artery is split into multiple channels. Right A-2 passes into longitudinal fissure anterior to left A-2.

take a variety of forms; one was simply a hole in the middle of a broad or triangular artery separating arteries several millimeters apart (Figs. 1, 4–8). The double or triple arteries could have approximately the same size or vary markedly in diameter. A common pattern was for one to be large and the others relatively small. Critchley stated that doubling or tripling of the ACoA is not uncommon; it occurred in 15 of Windle's 200 cases. Critchley stated that there is no intercommunication between ACA's beyond the anterior communicating area; however, we found one communication between the A-2 segments after they had passed superiorly between the cerebral hemispheres. We found an ACoA in every case although Critchley noted that it was occasionally absent. This discrepancy may be explained by our use of magnification and Critchley's reliance on the naked eye; an artery as small as 0.2 mm, as was seen in one of our cases, might not have been noted with the naked eye.

An infrequent finding was duplication of a portion of A-1. It occurred on one side in two brains, and did not occur on both sides in any case (Figs. 1 G and 9). Another interesting anomaly consisting of a third or median ACA arising from the ACoA was found one time in our 50 cases (Figs. 1 H and 10). This vessel, also known at times as the arteria termatica of Wilder, has been reported in 1.5% to 10% of brains. The median artery courses upward and backward over the dorsum of the corpus callosum. According to Blackburn, it frequently divides opposite the paracentral lobule and gives branches to the paracentral lobules of both sides. In such cases, the

Fig. 5. Small ACoA connects right and left A-1 segments of nearly equal diameter. Larger communicating arterial channel is connected to left A-1 segment by a rudimentary channel. Large left recurrent artery arises from A-1. Multiple arterioles supply dorsal surface of chiasm. Left A-2 passes anterior to right A-2 as A-2 segments enter the longitudinal fissure.
ACA's proper are usually small and supply the anteromedial surfaces of the hemispheres.

**Recurrent Artery of Heubner**

The recurrent branch of the ACA, first described by Heubner in 1874, is unique among arteries in that it doubles back on its parent vessel and accompanies it and the middle cerebral artery a variable distance before entering the brain (Figs. 1, 3-11). It was the largest artery arising from the A-1 segments or the proximal 0.5 mm of A-2 in 88% of the brains examined. It was smaller in diameter than the largest basal perforating artery arising from A-1 in 6% and equal to the largest one in 6%. Heubner's artery varied in diameter from 0.2 mm to 2.9 mm (average 1.0 mm). The average diameter of the A-1 segment was 2.6 mm. It was as large as the adjacent A-1 segment in only one case.

There is a great degree of inconsistency in the descriptions of the origin of the recurrent artery. Critchley noted that it may emerge from the superior surface of A-1 at some point between the most medial and lateral portion of the anterior perforated substance, and that it may occasionally arise from the internal carotid at its bifurcation, from the middle cerebral, or even from the ACoA. Ostrowski, et al., found it to arise just proximal to the ACoA in 26 of 28 brains and distal to the ACoA in the other two. Ahmed and Ahmed described it as usually having its origin from the ACA at the level of the ACoA. In our study, it was found to have its origin from the A-2 segment in 78% (Figs. 1, 4, 6, 7, 10, 11), from the A-1 segment in 14% (Figs. 1, 3, 5, 8, 9), and at the level of the ACoA in 8% (Figs. 1 and 3). In 52% it arose within 2 mm of the ACoA, in 80% within 3 mm, and in 95% within 4 mm.

---

**FIG. 6.** A-1 segments are connected by two communicating arteries. Right recurrent artery passes lateral to upper surface of carotid bifurcation and enters Sylvian fissure above the middle cerebral artery.

**FIG. 7.** Right and left A-1 are of nearly equal diameter. Double communicating artery: one is small and the other rudimentary. Left recurrent artery passes above A-1. Right recurrent artery courses superior and posterior to A-1.

D. Perlmutter and A. L. Rhoton, Jr.
Anterior cerebral-anterior communicating anatomy

The recurrent artery arising furthest from the ACoA arose 9.8 mm proximal to the ACoA. The recurrent artery was absent on one side in one brain, but another had a double left Heubner's. Ahmed and Ahmed found the vessel to be absent on one side of the circle of Willis in two of 12 cases, to have a double origin in one case, and to consist of twin trunks in two.

In our study, 60% of the recurrent arteries coursed anterior to the A-1 segment and would have been seen upon elevating the frontal lobe prior to visualizing the A-1 segment (Figs. 8, 10, 11). Forty percent coursed predominantly superior to the A-1 segment, between it and the anterior perforated substance (Figs. 1, 3, 4, 5, 6). Some of the recurrent arteries passing superiorly looped posterior to A-1 (Figs. 6 and 7). Ahmed and Ahmed described the artery as passing in a position closely bound to the superolateral aspect of the A-1 segment in its proximal course and above the internal carotid bifurcation and the proximal middle cerebral artery in its lateral course (Fig. 6).

The recurrent artery has been described as terminating as a single stem or as one or two branches which enter the anterior perforated substance above the carotid bifurcation. Our examination with magnification revealed much more branching. It entered the brain as a single trunk without branching in 14%, but it had as many as 12 branches in one case. The average number of recurrent artery branches was 4.2. Of the total number of branches from 99 recurrent arteries (artery absent on one side of one brain), 39% terminated in the anterior perforated substance, and 41% in the Sylvian fissure lateral to the anterior perforated substance and lateral to the origin of the ACA from the internal carotid artery. The remaining branches

<table>
<thead>
<tr>
<th>Site of Termination</th>
<th>Artery of Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>(optic chiasm, tract, nerve, and suprachiasmatic area)</td>
<td>A-1 (%)</td>
</tr>
<tr>
<td>optic chiasm, tract, nerve, and suprachiasmatic area</td>
<td>42</td>
</tr>
<tr>
<td>anterior perforated substance</td>
<td>41</td>
</tr>
<tr>
<td>Sylvian fissure</td>
<td>5</td>
</tr>
<tr>
<td>frontal lobe</td>
<td>10</td>
</tr>
<tr>
<td>other</td>
<td>2</td>
</tr>
<tr>
<td>total</td>
<td>100</td>
</tr>
</tbody>
</table>

TABLE I
Termination of branches by percent of branches arising from each artery.
D. Perlmutter and A. L. Rhoton, Jr.

FIG. 9. Both A-1 segments and ACoA are of approximately equal size. Right A-1 segment, medial half, splits into two channels and recurrent artery arises from one of these.

passed to the inferior surface of the frontal lobe (20%) and terminated in descending order of frequency; 13% entered the inferior frontal area lateral to the olfactory sulcus, 5% the olfactory sulcus, and 2% the gyrus rectus (Table 1).

The branches of Heubner's artery have been described as supplying the anterior part of the caudate nucleus, the anterior third of the putamen, the tip of the outer segment of the globus pallidus, and the anterior limb of the internal capsule. Injection of the recurrent artery with a gelatin-india ink mixture gave consistent staining of the head of the caudate nucleus, the anterior-inferior portion of the anterior limb of the internal capsule, and the anterior portion of the globus pallidus. Constant but few vessels were found in the putamen and anterior hypothalamus.8 The hypothalamic filling was less than when the A-1 segment alone was injected. The injection of Heubner's artery gave constant staining to a variable degree of the uncinate fasciculus and the olfactory regions.

In the treatment of anterior communicating aneurysms, great care must be taken to avoid unnecessary manipulation of Heubner's artery; occlusion may cause hemiparesis with brachial predominance due to compromise of that branch supplying the anterior limb of the internal capsule, and aphasia if the artery is on the dominant side. Involvement of this supply will also cause paralysis of the face and tongue. Critchley8 presented a case in which occlusion of Heubner's artery on the dominant side was demonstrated and the area of infarction was studied pathologically.

There was softening in the head of the putamen, the caudate nucleus, and the anterior half of the forelimb of the internal capsule. It was concluded that the recurrent artery occlusion was responsible for aphasia, severe weakness in the upper extremity, and slight paralysis of the face, palate, and tongue.

**Basal Perforating Branches**

The A-1 and A-2 segments and the ACoA were the site of small arterial branches to the anterior perforated substance, subfrontal area, the dorsal surface of the optic chiasm, suprachiasmic area, hypothalamus, and the Sylvian fissure. From two to 15 basal arterioles (average of eight), exclusive of Heubner's artery, arose from each A-1 segment (Figs. 1, 4, 11). The lateral half of A-1 was a richer source of branches than the medial half; the lateral half gave rise to as few as one and as many as 11 (average 5.3); the number from the medial half ranged from none to six (average 2.5). Of the total number of A-1 perforating arteries counted in 50 brains, 68% arose from the lateral half and 32% from the medial half. Dunker and Harris7 also found that the lateral half of A-1 gave rise to more perforating branches than the medial half. This contrasts with Barry's report9 that it is extremely unusual for any of the perforating vessels to arise from the initial 5 mm of A-1. He advocated placing a clip on the ACA immediately after its take-off from the internal carotid artery for the surgical treatment of anterior cerebral-anterior communicating aneurysms, stating that this prevents the clip from being placed across the recurrent artery.
of Heubner or another small perforating artery. Care must be taken to prevent clipping of Heubner’s artery even at this lateral point because it is often adjacent and attached to the A-1 segment by arachnoidal bands even at the level of the carotid bifurcation (Fig. 6).

Of the total number of basal arteries arising from the A-1 segment, 54% arose from the superior surface, 9% from the inferior, 32% from the posterior, and 5% from the anterior. The diameter of the arteries arising from A-1 segments ranged from 0.1 to 1.0 mm (average 0.2 mm). The A-1 branches terminated in the anterior perforated substance (41%), the dorsal surface of the optic chiasm or the suprachiasmatic portion of the hypothalamus (29%), the optic tract (11%), dorsal surface of the optic nerve (2%), the Sylvian fissure (5%), between the cerebral hemispheres (2%), and other sites on the inferior surface of the frontal lobe (10%). The striking difference in termination of A-1 branches as compared to those from the recurrent artery is the lack of recurrent artery branches to the dorsal surface of the optic nerves and chiasm and the anterior hypothalamus, and the greater number of recurrent branches entering the Sylvian fissure. Approximately 40% of both A-1 and Heubner’s branches terminated in the anterior perforated substance, but almost no Heubner’s branches entered the area around the optic chiasm and tract although 40% of those from A-1 terminated there. Forty-one percent of the recurrent artery branches entered the Sylvian fissures as compared to 5% of those from A-1 (Table 1).

These findings correlated well with the findings of Ostrowski, et al., that injection of a gelatin-india ink mixture into only the A-1 segment, excluding it from Heubner’s artery and the A-2 segment, most consistently filled the chiasm and anterior third ventricle and hypothalamus area but only inconsistently filled the caudate and globus pallidus. Injection of Heubner’s artery only, by contrast, revealed a rich supply to the caudate and adjacent internal capsule but much less filling of the hypothalamus than when the A-1 segment was injected. Recently Dunker and Harris, using similar techniques, demonstrated an A-1 supply to the internal capsule and rostral thalamus in addition to that demonstrated by Ostrowski.

Critchley noted that involvement of the hypothalamic branches that arise mainly from A-1 without implication of the recurrent artery may result in psychiatric symptoms without paralysis. Ostrowski, et al., noted that feelings of anxiety and fear, generalized weakness with weak spells, and symptoms referable to disordered mentation, such as dizziness, agitation, and hypokinesis were common. Lesions in the anterior hypothalamic region irrigated by the anterior cerebral circulation have not caused alterations of the conscious or waking state, although emotional changes, personality disorders, and intellectual deficits may be ascribed to occlusions of the arterial supply to this area.

Dandy believed that ligation of the left ACA produced a permanent loss of consciousness. Both Dandy and Poppen referred to the potential hazards that may attend ligation of the ACA distal to the ACoA. Others feel that loss of consciousness should be attributed to a compromise of blood flow in the region of the ascending reticular pathways in the posterior portion of the hypothalamus outside the anterior cerebral supply rather than to the direct involvement of the area supplied by the ACA.

The frequent inclusion of recurrent artery ischemia when the A-1 segment branches are involved adds a hemiparesis with brachial predominance to the deficit. This contrasts with the crural weakness of distal ACA occlusion. With proximal occlusion of the ACA, the ACA distal to the ACoA is often bilaterally supplied. Webster, et al., noted that it may be possible to differentiate proximal and distal occlusion clinically but that neither can be diagnosed with certainty before death except by angiography.
FIG. 11. A-1 segments are of equal diameter. Communicating artery is smaller than A-1 segments. Communicating artery gives rise to small arterioles passing to optic chiasm and lamina terminalis. Tortuous left recurrent artery passes anterior to A-1. Right recurrent artery passes posterior to A-1 segment.

The ACoA was also a site of arterial branches to the dorsal surface of the optic chiasm and suprachiasmatic area (Figs. 1 E and F). The number of perforating branches arising from the ACoA ranged from none to four (average 1.6). The anterior communicating branches terminated in the suprachiasmatic area (51%), dorsal surface of the optic chiasm (21%), anterior perforated substance (15%), frontal lobe (5%), and other areas (8%). In order of greatest frequency, the anterior communicating perforating branches arose from the superior (54%), posterior (36%), anterior (7%), and inferior (3%) aspects of the artery. Critchley found no perforating arteries from the anterior communicating area, but recently Dunker and Harris found no fewer than three branches arising from the ACoA and showed by dye infusion that they perfuse the fornix, corpus callosum, septal region, and anterior cingulum. Krayenbühl, et al., state that the use of magnification for surgery permits identification and preservation of the small perforating arteries arising from the ACoA.

The number of basal branches arising from the first 5 mm of the A-2 segment ranged from none to four (average 1.2). The great majority arose from the lateral (46%) and superior (43%) aspects of the artery. The inferior surface was the source of the other 11%. The majority of the A-2 branches terminated in the gyrus rectus (29%) and inferior frontal area (31%). Other common sites were the anterior perforated substance (15%), and the dorsal optic chiasm and suprachiasmatic area (12%). In dissecting the more proximal areas, 13% of the branches in this area were damaged and their termination could not be defined (Table 1).

Discussion

Controversy has arisen about the most advantageous approach to the anterior communicating area. Each approach, whether it be the gyrus rectus approach of VanderArk, et al., the bifrontal approach of Pool, the frontal approach with frontal lobe resection of French and Ortiz-Suarez, or the pterional approach of Yaşargil, et al., has the common feature of placing the bone flap as low as possible to minimize retraction and resection of the brain. Routine gyrus rectus removal is not necessary if the aneurysm is exposed in the subarachnoid cistern above the chiasm. If resection is required, it is kept to a minimum; no more than the posterior 1.5 cm of the gyrus rectus usually gives full visualization of both A-1 and proximal A-2 segments, and the recurrent and anterior communicating arteries.

The first artery seen upon frontal lobe elevation may be the recurrent artery. If A-1 is hypoplastic, the recurrent artery on that side may be nearly as large as the A-1 segment and might even be confused with it. The recurrent artery may lie in any direction from the A-1 segment but if followed, usually joins the A-2 segment just distal to the ACoA. We have seen it adherent to the wall of aneurysms. Dunker and Harris noted that the investing adventitia of A-1 may so obscure Heubner's artery that inadvertent occlusion by a clip may easily occur even under the microscope.

It should be remembered that small arteries arise from the ACoA. Passage of a Sundt-clip graft completely around the ACoA could occlude these anterior communicating branches and cause hypothalamic and frontal symptoms. We have seen that occlusion of these perforators by clip grafts can cause severe recent memory deficits. A bladed clip applied in such a way that all areas between the blades of the clip are clearly visualized so as not to include small surrounding vessels is recommended.

The dome of the aneurysm should be judiciously avoided at the onset of the dissection with primary attention directed to the parent artery or arteries and the aneurysm.
Anterior cerebral-anterior communicating anatomy

The majority of the aneurysms point anteriorly and inferiorly. The approaches of French and Ortiz-Suarez and Pool lead to more frequent dissection of the dome before the base is identified. An approach along the pterion facilitates exposure of the base before the fundus. Yaşargil approaches all from the right side. We use the right side except if a left frontal hematoma is present, if the fundus of the aneurysm projects toward the right, or if the left ACA is dominant and the right is hypoplastic. It is important to have control of the dominant ACA.

The majority of anterior cerebral-anterior communicating aneurysms occur in association with dominance of one A-1 and hypoplasia of the other. It is important to remember that even hypoplastic A-1 segments have a lumen which should be preserved, and which can lead to bleeding from a damaged fundus even if only the dominant A-1 is temporarily clipped.

Durity and Logue, and Tindall, et al., advocate proximal clipping of the A-1 segment feeding the aneurysm in selected cases. Barry placed the clip on the proximal ACA because this keeps the surgical intervention as far as possible from the aneurysm and prevents the clip from being placed across the recurrent artery of Heubner or another of the small perforating arteries. Barry states it is extremely unusual for any of these small vessels to arise from the initial 5 mm of the artery. This contrasts with our findings and those of Dunker and Harris that the majority of perforating arteries arise from the lateral half of the A-1 segment.

Prior to the use of magnification, there was a tendency to keep aneurysm dissection to a minimum because of the hazard of rupture. The use of magnification has permitted increased accuracy of dissection of the aneurysm neck and more frequent preservation of the recurrent and other perforating arteries. When using magnification, we feel that placement of a clip on an inadequately exposed aneurysm with resultant occlusion of perianeurysmal perforating arterioles is a greater risk than the hazard of rupture with microsurgical dissection. If rupture of an aneurysm does occur, it can usually be controlled by placing a small cotton pledget over the bleeding point and continuing accurate dissection after lowering mean blood pressure by 10 or 15 mm Hg.

References


This work was supported in part by the Heart Association of Broward County and National Institutes of Health Grant NS 10978-02.
Address reprint requests to: Albert L. Rhoton, Jr., M.D., Neurosurgery, University of Florida Health Center, Gainesville, Florida 32610.