ANEURYSMS OF THE PERICALLOSAL ARTERY
A STUDY OF 14 CASES VERIFIED ANGIOGRAPHICALLY AND TREATED MAINLY BY DIRECT SURGICAL ATTACK
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(Received for publication March 4, 1959)

The pericallosal artery is the distal portion of the anterior cerebral artery, peripheral to the anterior communicating artery. From the anterior communicating artery it runs a few mm. away from its pair on the medial surface of the area adolfactoria forwards and upwards to the rostral part of the corpus callosum, from where it turns backwards around the genu of the corpus callosum. Then it continues its course on the upper surface of the corpus callosum to the splenium, where it turns downwards, and terminates in the posterior part of the third ventricle. A few cm. distal to the anterior communicating artery two branches arise from the pericallosal artery, viz. the prefrontal and frontopolar arteries. Just on the genu of the corpus callosum, where the pericallosal artery often makes a distinct bend, the callosomarginal artery takes its origin. This artery exhibits a wide variety in its calibre and course. Often it runs rectangularrly to the cingulate sulcus, where it continues backwards and divides into its terminal branches. The callosomarginal artery is not always coherent, but may consist of two or three separate stems, the anterior, middle and posterior internal frontal arteries. At the midportion of the corpus callosum, the paracentral artery and, somewhat more distally, the precuneal and parieto-occipital arteries arise from the pericallosal artery. In this study the ramification of the callosomarginal artery from the pericallosal artery is for practical purposes called the pericallosal bifurcation. When the callosomarginal artery is not a coherent stem, the pericallosal bifurcation is the starting point of the anterior internal frontal artery, just on the genu of the corpus callosum.

Hitherto, there have been few studies dealing with arterial aneurysm at the pericallosal bifurcation. In Dandy's7 and Hamby's10 monographs, respectively, there was no mention of this site. Neither Ethelberg9 nor Pluvien age16 seemed to know of it. One case was reported in 1953 by Castorina and Catalano.6 In an autopsy series consisting of 143 cases in which rupture of intracranial aneurysm was the cause of death, Wilson et al.18 found 5 pericallosal aneurysms. In the large series of McKissock et al.,12,14 13 cases of aneurysm of the distal portion of the anterior cerebral artery without more exact localization were reported. In 1956, Mount and Tavera15 reported one pericallosal aneurysm in a total of 43 aneurysms. In 1957, Allègre and Vigouroux2 stated that aneurysm of the distal part of the anterior cerebral...
artery was less frequent, but its location at the pericallosal bifurcation was found to be constant. These authors had no personal cases, however.

PRESENT MATERIAL

The present series comprises 14 patients treated in the years 1936–1958 in the Neurosurgical Department of the Finnish Red Cross Hospital, Helsinki. All cases of intracranial arterial aneurysms admitted during this period are shown in Table 1. The aneurysms of the internal carotid artery and its bifurcation have been studied by af Björkesten,3 those of the middle cerebral artery by af Björkesten and Troupp,4 and those of the anterior communicating artery by Snellman and co-workers.17 As can be seen in Table 1, perical-

TABLE 1

Arterial aneurysms treated in neurosurgical department of Finnish Red Cross Hospital, Helsinki, 1936–1958

<table>
<thead>
<tr>
<th>Site of Aneurysm</th>
<th>No. of Patients</th>
<th>Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal carotid artery</td>
<td>109</td>
<td>34</td>
</tr>
<tr>
<td>Anterior communicating artery</td>
<td>88</td>
<td>27.5</td>
</tr>
<tr>
<td>Middle cerebral artery</td>
<td>87</td>
<td>27.5</td>
</tr>
<tr>
<td>Anterior cerebral artery</td>
<td>18</td>
<td>5.5</td>
</tr>
<tr>
<td>Pericallosal artery</td>
<td>14</td>
<td>4.5</td>
</tr>
<tr>
<td>Vertebral system</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>320</td>
<td>100%</td>
</tr>
</tbody>
</table>

losal aneurysms constitute 4.5 per cent of the intracranial arterial nonfistulous aneurysms. There were 5 females and 9 males. The age of the patients on admission varied from 20 to 57 years, the average being 40 years. Further details regarding the individual cases are summarized in Table 2.

INCIDENCE OF SUBARACHNOID HEMORRHAGE

Eight patients had had only one subarachnoid hemorrhage before admission, the interval from the bleeding to admission being from 1 day to 9 months. In 5 cases there had been two attacks. In one of these the first hemorrhage, which was not verified, occurred when the patient was 25 years old, and the second when he was 45, 6 weeks prior to admission to the neurosurgical department. In Case 14 there was subarachnoid bleeding 1 month previously, followed by another on the day of admission. In 1 case three hemorrhages had occurred prior to admission. In every case the interval between the hemorrhages and admission can be seen in Table 2. The severity of the subarachnoid bleeding seemed not to have increased with the second hemorrhage. In Case 9, in which three hemorrhages had occurred, the patient was in very poor condition after the third attack.
ANEURYSMS OF PERICALLOSAL ARTERY

CLINICAL SYMPTOMS

In all cases but one there had been a typical picture of subarachnoid hemorrhage. The exceptional case was that of a man aged 48, who suddenly felt dizziness followed by weakness of the left lower limb, but no headache. He went of his own accord to the local municipal hospital, where the spinal fluid was found to be bloody. Left carotid angiography revealed an aneurysm of the left pericallosal artery (Case 5).

In 7 cases there had been unconsciousness, mostly of short duration, from a few minutes to 3 hours. In 6 cases there had been mental deterioration after the attack. In 4 cases papilledema was noted, and in 1 case the sense of smell was found to be lost.

Pyramidal tract signs were found in 10 patients. In 4 cases they consisted of transient paresis of the leg and Babinski’s sign on the side contralateral to the aneurysm. Two patients showed bilateral Babinski’s sign, and 1 patient with aneurysm of both pericallosal arteries had Babinski’s sign on the right side only. In 1 case there was slight paresis of the lower limb on the same side as the aneurysm. One patient had slight paresis of the right upper limb and expressive aphasia. Carotid angiography revealed an aneurysm of the left pericallosal artery, and a little hematoma in the interhemispheric fissure. The 10th patient with pyramidal signs had right hemiparesis and aphasia. Two aneurysms were found, one of the left pericallosal artery, the other of the left internal carotid artery.

In 4 cases no neurological abnormality was found in spite of oxycephaly in 1 case (Case 13). This patient also had coarctation of the aorta. In another case systolic murmur in the heart was heard. When the patient was sent to the medical department for cardiological examination, he refused further treatment and left the hospital.

ANGIOGRAPHIC EXAMINATION

Percutaneous carotid angiography was performed bilaterally. Vertebral angiography was never performed. The contrast medium used was Triurol (Leo) or Urographin (Schering). Six patients had an arterial aneurysm at the left pericallosal bifurcation (Fig. 1). Once an aneurysm was found at the right pericallosal bifurcation. In the remaining 7 cases multiple aneurysms were found. In Case 3 three arterial aneurysms were disclosed: one of them was at the left pericallosal bifurcation; the other two were bilateral and symmetrical on the middle cerebral artery. In Case 4, three aneurysms were also found: one at each pericallosal bifurcation, and the third on the left middle cerebral artery. In Case 6, an aneurysm was seen bilaterally on each pericallosal artery. In Case 8 there was an aneurysm on the left pericallosal artery and another on the right middle cerebral artery. In Case 10, a saccular aneurysm was found on the left pericallosal artery; there was also a small arteriovenous malformation just behind the left internal carotid bifurcation. In Case 12 there were two aneurysms, one on the left pericallosal artery and
### TABLE 2

*Summary of 14 cases of pericallosal aneurysm*

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>No. of Hemorrhages</th>
<th>Interval Between Bleeding(s) and Admission</th>
<th>Verified Hemorrhage</th>
<th>Neurological Signs</th>
<th>Site of Aneurysm(s)</th>
<th>Treatment</th>
<th>Operative Complications</th>
<th>Control Angiography</th>
<th>Follow-Up Time, and Late Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 T.R.</td>
<td>F</td>
<td>21</td>
<td>1</td>
<td>9 mos.</td>
<td>—</td>
<td>Mental confusion, papilledema, paresis rt. leg</td>
<td>Lt. peric.</td>
<td>Aneurysm clipped</td>
<td>None</td>
<td>Not done</td>
<td>5 yrs. Well</td>
</tr>
<tr>
<td>3 E.J.</td>
<td>M</td>
<td>50</td>
<td>1</td>
<td>1 day</td>
<td>+</td>
<td>—</td>
<td>Lt. peric. &amp; both middle cerebals</td>
<td>Conservative</td>
<td>—</td>
<td>—</td>
<td>30 mos. Died from rupture rt. middle cerebral aneurysm</td>
</tr>
<tr>
<td>4 A.T.</td>
<td>M</td>
<td>45</td>
<td>2</td>
<td>20 yrs. 6 wks.</td>
<td>+</td>
<td>Mental confusion, papilledema, loss sense of smell, bilat. Babinski</td>
<td>Both peric. &amp; lt. middle cerebral</td>
<td>Rt. peric. aneurysm ligated</td>
<td>None</td>
<td>Aneurysm not filling</td>
<td>27 mos. Well</td>
</tr>
<tr>
<td>5 N.L.</td>
<td>M</td>
<td>48</td>
<td>1</td>
<td>3 wks.</td>
<td>+</td>
<td>Paresis lt. leg</td>
<td>Lt. peric.</td>
<td>Aneurysm clipped</td>
<td>None</td>
<td>Proximal part filling</td>
<td>30 mos. Well</td>
</tr>
<tr>
<td>6 P.M.</td>
<td>M</td>
<td>33</td>
<td>1</td>
<td>1 mo.</td>
<td>+</td>
<td>Paresis rt. leg</td>
<td>Both peric.</td>
<td>Rt. aneurysm clipped; lt. ligated</td>
<td>None</td>
<td>Not done</td>
<td>25 mos. Well</td>
</tr>
<tr>
<td>Case</td>
<td>Age</td>
<td>Sex</td>
<td>Time</td>
<td>Symptom</td>
<td>Treatment</td>
<td>Outcome</td>
<td>Initial Findings</td>
<td>Further Findings</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>------</td>
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<td>-----------------</td>
<td>------------------</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>T.J.</td>
<td>48</td>
<td>F</td>
<td>3 mos.</td>
<td>Mental confusion, papilledema, rt. hemiparesis</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, papilledema</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K.H.</td>
<td>57</td>
<td>M</td>
<td>2 mos.</td>
<td>Mental confusion, bilateral Babinski</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, bilateral Babinski</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.S.</td>
<td>47</td>
<td>F</td>
<td>3 mos.</td>
<td>Mental confusion, papilledema, rt. facial paresis, l. Babinski</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, papilledema, rt. facial paresis, l. Babinski</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.K.</td>
<td>57</td>
<td>M</td>
<td>1</td>
<td>Mental confusion, papilledema</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, papilledema</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.N.</td>
<td>57</td>
<td>M</td>
<td>1</td>
<td>R. Babinski</td>
<td>Conservative</td>
<td>Died</td>
<td>R. Babinski</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.P.</td>
<td>40</td>
<td>F</td>
<td>1</td>
<td>R. hemiparesis</td>
<td>Conservative</td>
<td>Died</td>
<td>R. hemiparesis</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.L.</td>
<td>27</td>
<td>M</td>
<td>1</td>
<td>Mental confusion, papilledema, l. arm</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, papilledema, l. arm</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.S.</td>
<td>20</td>
<td>F</td>
<td>1</td>
<td>Mental confusion, papilledema, l. arm</td>
<td>Conservative</td>
<td>Died</td>
<td>Mental confusion, papilledema, l. arm</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
the other on the left internal carotid artery. In Case 13 an aneurysm was seen at the left pericallosal bifurcation, and another on the right callosomarginal artery, about 2 cm. distal to the pericallosal bifurcation.

Thus 14 patients showed a total of 22 saccular arterial aneurysms and 1 arteriovenous malformation. There were 16 pericallosal aneurysms, and in addition 1 aneurysm of the callosomarginal artery. Of the 16 pericallosal aneurysms, 13 were on the left side, and only 3 on the right.

The pericallosal aneurysm in every case was located at the pericallosal bifurcation on the genu of the corpus callosum, where the pericallosal artery often makes a sharp bend. Before this study was started, the senior author suggested that there might be an extra junction between the two pericallosal arteries just at the pericallosal bifurcation. This junction might be a remnant persisting from early embryonic life, when the rostral part of the carotid system consisted of an arterial network. Normally such a connection disappears during development, but under certain circumstances it might persist, or its remnants might be the etiological cause of saccular aneurysm at this site.
When the angiograms were examined, a communication between the two pericallosal bifurcations was found in 3 cases (Cases 5, 6, and 11). Only one arterial stem was visible proximal to the pericallosal bifurcation, i.e. between the anterior communicating artery and the aneurysm, but distal to the pericallosal bifurcation, the pericallosal and callosomarginal arteries were filled bilaterally. At operation, no special attention was paid to this junction, but once (Case 5) an arterial communication between the base of the aneurysm and the contralateral pericallosal bifurcation was noticed.

**PATIENTS NOT OPERATED ON**

Four patients were not operated on (Cases 3, 8, 12, and 13). In Case 3, the patient, with three separate arterial aneurysms, refused to undergo operation. He died 2½ years later from rupture of the aneurysm of the right middle cerebral artery. Case 8, a man aged 57, was in extremely poor condition. Because he had two aneurysms, operative treatment was considered contraindicated. He was discharged to the medical department, where he died 4 weeks later from recurrent subarachnoid hemorrhage. The third patient not operated on (Case 12) had an aneurysm on the left pericallosal artery and another on the left internal carotid artery. Because of systolic heart murmur he was sent to the medical department for cardiological examination. There he refused to continue treatment and left the hospital. Twelve months later he was working at full capacity as an agriculturist. No new subarachnoid bleeding had occurred. The fourth patient (Case 13) was a man aged 27. Since birth he had had a distinct oxycephalic deformity of the skull caused by premature fusion of the coronal sutures. On March 27, 1957, he had a subarachnoid hemorrhage. When examined on Jan. 3, 1958 at the neurosurgical department, in addition to the two aneurysms (left pericallosal and right callosomarginal) a coarctation of the aorta was diagnosed. He was sent to the department of thoracic surgery where, on June 25, 1958, he was operated on for the coarctation. His blood pressure thereafter showed a fall from 220 to 150 systolically. He was re-examined on Jan. 8, 1959 in the neurosurgical department. Angiography on both sides showed no change in the aneurysms. As 22 months had elapsed since the rupture, and the arterial hypertension had been corrected, surgical treatment of the aneurysms was considered unwise. He has continued to be free from recurrent hemorrhage.

**PATIENTS OPERATED ON**

Direct surgical attack on the aneurysm was made in 10 cases. Frontal craniotomy under intratracheal anesthesia, and controlled hypotension with Arfonad in a few cases, was performed. In 8 cases the aneurysm was approached through the interhemispheric fissure. When necessary, some superior cerebral veins were dissected to make the exposure better. The pericallosal and callosomarginal artery could easily be visualized and followed forwards as far as the pericallosal bifurcation. In 3 cases, the surface of the genu of the corpus callosum had to be dissected before the aneurysm could be
treated. Three times the aneurysm lay inside the cerebral cortex, and twice it was firmly adherent to the falx. In Case 4 the aneurysm was approached by the subfrontal route from the internal carotid bifurcation along the anterior cerebral artery up to the pericallosal bifurcation. This approach is very difficult and has since been abandoned. In Case 9 an intracerebral frontal hematoma was found which had perforated the cerebral cortex to the subdural space. As soon as the clots were removed from the cavity of the hematoma, the aneurysm was visible hanging on the medial wall of the cavity. A silver clip was easily applied across its neck.

In 5 cases the neck of the aneurysm was ligated with linen thread, and in 4 cases a silver clip was applied across the stalk of the aneurysm. Once the aneurysm was extirpated because it was entangled within the branches of the pericallosal and callosomarginal arteries. After ligation of the aneurysmal stalk, the sac was in some cases wrapped with muscle. In Case 14, a silver clip was applied across the aneurysmal stalk during the first operation. Control angiography showed that the clip had slipped from its place and the aneurysm was filled. At the second intervention, the neck of the aneurysm was tied with linen thread, after which the aneurysm did not fill at control angiography.

In Case 4, in which three saccular aneurysms were revealed by angiography, the right pericallosal aneurysm was considered to be ruptured, and only this was ligated. In Case 6, in which an aneurysm was visible at both pericallosal bifurcations, both could be treated during the same operation.

There was 1 operative death (Case 7). The patient was a 46-year-old housewife, who in 1931 had suffered from pyelonephritis. Since then the blood pressure had been elevated. She had had two subarachnoid hemorrhages 3 and 2 months prior to admission on Sept. 22, 1956. Before the operation she was in good condition. The aneurysm on the left pericallosal artery was easily reached through the interhemispheric route and ligated with linen thread. Hypotension was not used. No damage to the brain was noticed, nor was there any bleeding. When the aneurysm was treated, acute heart failure with arrhythmia and tachycardia set in, and the patient died half an hour after operation despite adequate medication. Autopsy showed collagenous alterations in the heart, lungs, liver and kidneys similar to lupus erythematosus disseminatus. In the brain nothing was found that could have explained the death.

In Case 1 a slight transient paresis of the contralateral lower limb developed after operation. Control angiography showed that the aneurysm was not filling. The patient was doing extremely well until 22 months later, when headache suddenly developed, and she became unconscious and died. Autopsy was not performed. It is evident that there was a new subarachnoid bleeding, the origin of which remains unknown, however.

In Case 10, in which the aneurysm at the left pericallosal bifurcation was extirpated and the left pericallosal and callosomarginal arteries were clipped, a severe right-sided hemiparesis and aphasia developed. One year later, the
aphasia had almost disappeared, as had the paresis of the right arm, but the right leg was quite paralyzed. The patient was totally incapable of work.

In the remaining 7 cases, recovery was excellent. The follow-up time in 6 of them varied from 15 months to 5 years, and all patients were doing extremely well. The seventh of them has been operated on recently and shows no disability. Control angiography was performed on 8 of the 10 patients operated on. In all of them the aneurysm had disappeared, in Case 14 after the second intervention, as already stated.

The prognosis for all 14 patients, 4 of whom were treated conservatively, and 10 through direct surgical attack, is presented in Table 3. The follow-up time in all but 1 case is over 15 months.

TABLE 3
Prognosis for all 14 patients with pericallosal aneurysm

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No. of Cases</th>
<th>Operative Deaths</th>
<th>Late Deaths</th>
<th>Disabled</th>
<th>Well</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conservative</td>
<td>4</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Aneurysm clipped</td>
<td>4</td>
<td>—</td>
<td>1*</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>Aneurysm ligated</td>
<td>5</td>
<td>1†</td>
<td>—</td>
<td>—</td>
<td>4</td>
</tr>
<tr>
<td>Aneurysm extirpated</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>—</td>
</tr>
</tbody>
</table>

* Doing well until 30 months later, then death from nonverified subarachnoid hemorrhage.
† Death caused by lupus erythematosus disseminatus.

COMMENTS

In this series from the Finnish Red Cross Hospital, pericallosal aneurysms constitute 4.5 per cent of all intracranial arterial aneurysms. The percentage in the material reported by McKissock et al.19,14 is 3.0. It is very interesting to note that the percentage of aneurysms of the pericallosal artery in the autopsy material reported by Wilson et al.18 is the same as that reported by us. It may be assumed that fatality from this aneurysm is approximately the same as from those at other sites.

As already suggested by Allègre and Vigouroux,2 the aneurysm in the pericallosal artery is located mainly at the bifurcation of the pericallosal and callosomarginal arteries. This is so in all 14 cases reported in this paper. The distance between the internal carotid bifurcation and the aneurysm in lateral projections of the angiogram measures from 3.6 to 5.0 cm., the average distance being 3.9 cm. The aneurysm is always situated on the genu of the corpus callosum. In most cases it arises from the normal bifurcation of the pericallosal and callosomarginal artery. If the callosomarginal artery is not coherent, the aneurysm lies at the bifurcation of the pericallosal and anterior internal frontal artery. In every case, however, the aneurysmal bifurcation lies on the genu of the corpus callosum, where the pericallosal artery not infrequently makes an abrupt bend backwards.

At operation the aneurysm is easily found when it is approached through
the interhemispheric fissure. In most cases the neck of the aneurysm can be ligated with linen thread, and we consider this to be the method of choice. It has been described by af Björkesten in the study dealing with the aneurysms of the internal carotid artery. A silver clip placed across the stalk of the aneurysm may slip, and ligature of the pericallosal artery, at least in the dominant hemisphere, is dangerous, as could be seen in one of our cases. If ligature of the aneurysmal stalk is not possible, e.g. when the aneurysm is sessile, or entangled within the branches of the pericallosal and callosomarginal arteries, the aneurysmal sac can be compressed with a broad silver clip and, in addition, wrapped with muscle.

It seems probable that the risk of recurrent rupture of the aneurysm lessens the longer the elapse of time since the initial bleeding. Höök has recently stated that for 92 patients who survived the first 8 weeks after the initial hemorrhage, the risk of recurrent fatal bleeding during an observation period of 7 years was 20 per cent. Hence, as long as we can keep the mortality of the operatively treated patients below this level, operation seems to be justifiable. In the present material there was 1 operative death among the 10 patients operated on. This fatal outcome unfortunately was caused by an undiagnosed lupus erythematosus disseminatus. Another patient, who died presumably from subarachnoid bleeding 2½ years after the evidently successful operation, may have had an undiagnosed aneurysm in the basivertebral system. In 1 case a severe disability developed after ligature of the dominant pericallosal artery. In the conservatively treated group, 2 out of the 4 are alive and doing well 2 years after the first subarachnoid hemorrhage. It must be borne in mind that the patients on whom no surgery was done had multiple aneurysms, and some were in poor condition, so that the two groups cannot be compared with each other. We believe, however, that the best prognosis can in most cases be obtained by direct surgical attack in view of the relative facility with which pericallosal aneurysm can be operated upon.

Because of the long waiting list to our neurosurgical department in Finland, the majority of patients had survived the actual danger of subarachnoid hemorrhage, and they belonged to McKissock’s category C, and were good subjects for operation.

The patients with pericallosal aneurysm very often seem to have multiple aneurysms. Seven patients only, i.e. 50 per cent, showed a single aneurysm. Not infrequently there is an aneurysm at each pericallosal bifurcation which clearly implies that a developmental anomaly plays a role in the etiology of the disease. Other concomitant malformations, such as craniostenosis, kyphoscoliosis, coarctation of the aorta, and an undefined systolic heart murmur were encountered in this series. The frequency of multiple aneurysms stresses the importance of bilateral carotid and possibly also of vertebral angiography, as soon as a pericallosal aneurysm has been found.

There are a number of neurological signs that should lead to suspicion of pericallosal aneurysm when a subarachnoid hemorrhage has been diagnosed. Pyramidal tract signs are frequent, 10/14, and 4 times a unilateral paresis
was encountered in the lower limb opposite to the side of the aneurysm. This
paresis was always of short duration, and had wholly disappeared before
operation. Mental confusion was found in half the patients, and papilledema
in one-third.

It is a well known fact that the anterior communicating artery and its
vicinity normally exhibit a wide range of anatomical variations.1,5,8 Adachi1
has demonstrated a case in which the trunecus of the anterior communicating
artery was 3 cm. long. The two pericallosal arteries arose from its distal end.
We are not aware whether an extra junction between the pericallosal arteries
distal to the anterior communicating artery has been found previously. In
the present material such a communication has been demonstrated in 3 cases
angiographically, and one of these has been verified at operation, though no
special attention was paid to this detail. We have called this junction the
“supreme anterior communicating artery.” It lies between the two perical-
losal bifurcations. It is probably a remnant of the primitive embryological
arterial system. A further patho-anatomical study dealing with this problem
has been started by one of us.

SUMMARY

Fourteen patients with saccular aneurysm on the pericallosal artery are
reported. In 4 cases conservative treatment alone was given; 2 of the pa-
tients were alive and well 2 years after the first subarachnoid hemorrhage. In
10 cases direct surgical attack was made on the aneurysm. In 9 of them the
neck of the aneurysm was ligated with linen thread, or a silver clip was ap-
plied across the stalk of the sac. Once the whole aneurysm was extirpated. Of
the 10 patients operated upon, 7 recovered completely, and 1 was severely
handicapped. There was 1 operative death, probably caused by collagenous
disease with cardiac involvement. Another of the surgically treated patients
died 2 1/2 years after the evidently successful operation, presumably from a re-
currence of subarachnoid hemorrhage.

The clinical signs, angiographic examinations, and patho-anatomical as-
pects have been described.

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