Section of Neurology

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The Pathology, Diagnosis and Treatment of Intracranial Saccular Aneurysms

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THIS paper is based on a study of 120 aneurysms which occurred in 114 patients observed at the Neurosurgical Clinic in Stockholm since 1932 (Table I).

TABLE I.—TOTAL NUMBER OF PATIENTS AND ANEURYSMS

	Patients	Aneurysms
Int. carotid artery	43	44
Infraclinoid	9	
Supraclinoid	34 (35)	
Bifurcation of int. car. art.	5	6
Proximal part of ant. cerebral art.	8	8
Ant. comm. art.	23	23
Distal part of ant. cerebral art.	5	6
Middle cerebral art.	24	24
Basilar and vertebral artery	3	3
Multiple aneurysms with different localization:—		
Int. car. art. and bifurcation	2	4
Int. car. art. and middle cer. art.	1	2
	114	120

In all cases except 5 the aneurysm was verified either at autopsy, at operation or by angiography. The 5 unverified aneurysms occurred in patients exhibiting the typical picture of ophthalmoplegia associated with migraine or subarachnoid hæmorrhage. 5 might have been of arteriosclerotic nature while all the rest were congenital. Forbus (1930) attributed the development of congenital aneurysms to the persistent pressure against the weak point of bifurcation of an artery, where the media of the arterial wall is defective. An alternative theory was advocated by Dandy (1944), who held that these aneurysms have their favourite sites and are not always located at the point of bifurcation. On the basis of a study of the embryonic development of the cerebral arterial tree by Dorcas Hager Padget, he considers aneurysms to be the result of incomplete resolution of embryonic vessels. Particularly the location of an aneurysm of the internal carotid artery to a point proximal to its bifurcation is better understood if the embryonic features of the vessels of the brain are considered.

It is, of course, clear that the material from different clinics will vary according to special circumstances. Jefferson in 1947 reported 158 cases of aneurysm of which 100 were located to the carotid artery.

The material presented in Table I is probably also deficient compared with autopsy material. The series published by McDonald and Korb (1939) showed a much higher percentage (21%) of aneurysms in the basilar and vertebral arteries.

It is well known that the great majority of aneurysms produce no clinical symptoms before they rupture. This is true of aneurysms in all situations other than the cavernous sinus. The clinical features of this group have been gradually outlined, especially by Jefferson.

The relation of the aneurysm to cerebral structures may give rise to other symptoms which might point to a definite location. These symptoms may be caused by local pressure or intracerebral hæmorrhage, the importance of which, from the standpoint of diagnosis, treatment and prognosis, is quite clear.

In Jefferson's series of 158 cases, neurological signs, other than ocular or oculomotor, occurred in only 7 patients. As shown in Table II hemiparesis, with or without aphasia, was a fairly frequent symptom in the present series and may occur with aneurysms in any situation. On the other hand the sudden onset of subarachnoid hæmorrhage simultaneously with hemiplegia or hemiparesis definitely points to an aneurysm of the middle cerebral artery. This occurred in 15 of our 23 aneurysms at this location.

The precise location cannot, however, always be foreseen, and the presence of an aneurysm may not be clear from the clinical features alone. Actually, the sudden onset of intracranial symptoms should always arouse suspicion of an aneurysm.

TABLE II.—SYMPTOMATOLOGY

	Number of cases	Hæmorrhage	Hemiparesis (aphasia)	Ophthalmoplegia	Migraine	Visual field defects	Trigeminal pains	Epileptic fits
Carotid art.								
Infraclinoid ..	9	2		8	7	1	6	
Supraclinoid ..	34	23	12	14	8	5	5	4
Carotid art. and bifurcation ..	2	2	2			1		1
Carotid art. and middle cer. art. ..	1			1	1			
Bifurcation.. ..	5	3	3	1	2	1		1
Proximal part of ant. cer. art.	8	8	3	1		2		
Ant. comm. art. ..	23	21	10	4		2		1
Distal part of ant. cer. art.	5	5	4			1		2
Middle cer. art. ..	24	23	19	3		4		1
	111	87	53	32	18	17	11	10

Angiography.

Angiography is necessary for the diagnosis and exact location of aneurysms and should be employed in every case suspected of vascular lesion. Sometimes aneurysms do not fill with contrast substance and in these cases encephalography might be helpful in localizing the lesion. The angiogram sometimes only indicates the presence of an expanding lesion. Angiography is not free from risks and complications but these are very few and do not influence the indications for angiography in these cases.

Angiographic studies should not be confined to the diagnosis and location of an aneurysm. Further information is necessary regarding the relation of the aneurysm to adjacent arteries, the existence and size of a neck, the state of the local and peripheral cerebral circulation and the adequacy of the collateral circulation of the circle of Willis.

Operative Measures.

The principal method of operative treatment for the intracranial aneurysms has been ligation of the carotid artery. We know that following internal carotid occlusion the systolic pressure distal to the occlusion falls to 50% and the pulse pressure to 25% of their initial levels. Therefore this procedure is probably of therapeutic value in the treatment of these lesions, by slowing down the circulation and assisting in the formation of a thrombosis in the sac and closing of the rupture.

Though ligation of the carotid artery is now considered a relatively safe procedure it is not, however, without risks. Post-operative impairment of the cerebral circulation might occur and produce transient or permanent hemiplegia or even death. Schorstein (1940) stated the opinion that the mechanism chiefly responsible for these complications was pre-operative impairment of the local or general cerebral circulation caused by the lesion for which the ligature was performed. This view was supported by Olivecrona (1944) in a study of ligature of the carotid artery in different conditions. Not infrequently the angiogram demonstrates, in addition to a reduced velocity in the cerebral blood flow, local abnormalities in the vessels surrounding the aneurysm. These are probably caused by vasoconstriction. Dandy believed that the cause of late signs of cerebral involvement was mainly thrombosis and embolism. Cases are reported in which this may be true but the influence of vasomotor impulses must also be considered (Fig. 2A and B).

Several methods of ligation have been devised for eliminating the risks of ligature, but it has not definitely been proved that one method is superior to another. It can only be said that some patients who do not tolerate occlusion of the internal carotid artery are able to do so if the common carotid is ligated first. We have always used just a single linen thread around the vessel followed, in some cases, by periarterial sympathectomy (indicated in Table III by figures within parentheses).

Post-operative Results.

The immediate post-operative results are shown in Table III. In only one case was the ligature not tolerated, necessitating its immediate removal. The patient who died, a woman aged 53, was

TABLE III.—IMMEDIATE RESULTS OF CAROTID LIGATION IN 37 PATIENTS WITH INTRACRANIAL SACCULAR ARTERIAL ANEURYSM

	Ligatures	Dead	Not tolerated	Transient hemiparesis	Transient hemiparesis. Removal of ligature
Common carotid art..	21 (1)		1	1	
Common and int. car. art.	12				2
Int. carotid	17 (9)	1		3 (1)	2 (1)

admitted following an attack of ophthalmoplegia with trigeminal nerve pains and paræsthesias. Angiography demonstrated an aneurysm in the cavernous sinus and ligature of the internal carotid artery was performed. The ligature was well tolerated but a few days later the patient complained of an increase in pain. According to Jefferson this is always a sign of activity in the aneurysm. Ten days after the operation the patient suddenly became comatose and died. Autopsy disclosed rupture of the aneurysm. It is difficult to say whether death was due to the ligature or not. Perhaps it is better to say that the ligature was unable to prevent fatal rupture of the aneurysm. The logical procedure in this case would have been to turn down a flap and place a clip on the artery above the aneurysm as soon as there was an increase in the trigeminal nerve pains.

Hemiplegia was transitory in all cases except one, a woman aged 28, who had an aneurysm on the left middle cerebral artery. Ligation of the internal carotid artery was followed by right hemiplegia and aphasia three days later. The ligature was removed but improvement was slow. Four years later, however, only a slight hemiparesis was present and the aphasia was minimal. In some patients signs of cerebral anoxia disappeared spontaneously and in others after removal of the ligature. It is therefore difficult to draw any definite conclusions regarding the value of removal of the ligature.

Late Results.

Published reports of late results are very few. Of Jefferson's 19 cases who survived ligature only one is dead seven years after operation from an unknown cause. All the others are alive and well. Krayenbühl reported 35 patients submitted to carotid ligation with a follow-up study in 14, none of whom showed recurrence of subarachnoid hæmorrhage. In Poppen's series of 101 carotid ligations there were 8 late deaths, of which 2 were presumably due to inadequate ligation resulting in a new rupture of the aneurysm. Table IV shows the late results in 31 of the 37 cases of carotid ligation in this series.

The fate of those patients who were discharged from hospital without any specific treatment is shown in Table V. Here the death-rate was comparatively higher. It is difficult to draw any positive

TABLE IV.—LATE RESULTS OF CAROTID LIGATURE IN 31 PATIENTS AFTER 1–14 YEARS

	Number of patients
Good, full working capacity	17
Epilepsy	4
Full working capacity 2	
Mental changes 1	
Hemiparesis 1	
Mental disturbances	2
Dead from other disorders	2
Dead with signs of a new hæmorrhage	6
Verified rupture of the same aneurysm 2	
Probable rupture of the same aneurysm 2	
Verified rupture of another aneurysm 1	
Cerebral hæmorrhage 1	

TABLE V

Of 22 non-operated patients with saccular aneurysms verified by autopsy or angiography during 1935–1948 4 died in the hospital shortly after admission. Table V indicates the fate of the other 18.

	Number of patients
No reports	2
Dead from new bleeding (all within 3 years)	5
Dead from other disorders	1
Dead from unknown disorders	1
Alive up to 9 years after discharge	9
New attack of transient ophthalmoplegia 1	
Persistent ophthalmoplegia 1	
Disabled because of mental disorder 1	

conclusions from such small series; it can only be said that in some cases carotid ligation is not adequate to prevent recurrent rupture of the aneurysm. Clinical experience shows that it is probable that the risk of recurrent hæmorrhage is reduced by carotid ligation if we take in account the high rate of recurrent hæmorrhages, recently demonstrated by Hyland (1950) in his study of the prognosis of the subarachnoidal hæmorrhage.

Prognosis.

I have seen no reports of a comparative study of the prognosis in verified aneurysms of different locations. Should there be a difference it would, of course, influence the indications for surgical treatment. I can give no figures to illustrate the point but I feel sure we have all seen cases of aneurysms in different situations in which death was due to hæmorrhage. Hæmorrhage is also the predominant sign in aneurysms of all locations except those from the subclinoid part of the carotid artery.

Our attitude to these lesions has been rather conservative owing to the fact that ligature was considered to be a relatively safe method and the intracranial attack was always considered a hazardous procedure. During the years 1932 to July 1950 aneurysms have now and then been explored, especially those rising from the anterior communicating artery in cases in which we felt carotid ligation would be of no benefit. In some cases the aneurysm was a surprise finding at an operation performed for

suspected tumour. In his monograph published in 1944 Dandy has outlined various methods for the treatment of an aneurysm by intracranial attack. Tables VI and VII show the location of our aneurysms and the type of operation performed.

TABLE VI.—INTRACRANIAL EXPLORATIONS OF SACCULAR ANEURYSMS FROM 1932 TO JULY 1950

Localization	Number of patients	Dead
Carotid artery ..	5	1
Proximal part of ant. cer. artery ..	1	
Anterior communicating artery ..	5	2
Distal part of ant. cer. artery ..	2	
Middle cerebral artery	1	
Vertebral artery ..	1	
	15	3

TABLE VII.—METHOD

	Trapping				
	Intracranial	Between intracranial clip and ligature in the neck	Wrapping with muscle or gel foam	Clip on the neck or excision	Exploration
Carotid artery ..	1	2	1		1
Proximal part of ant. cer. art.				1	
Ant. communicating artery			3	1	1
Distal part of ant. cer. art.	1				1
Middle cer. art. ..				1	
Vertebral artery ..				1	
	2	2	4	4	3

In recent years a more active surgical treatment with intracranial exposure of the aneurysm has been advocated by some neurosurgeons. This attitude is based on studies of the prognosis in spontaneous subarachnoid hæmorrhage reported from various clinics during the last years.

In Sweden two years ago Ask-Upmark and Ingvar (1950) made a follow-up study of 138 cases of their own and 385 from the literature. They concluded that 60% of the cases of subarachnoid hæmorrhage died sooner or later from the lesion. Only 20% had a chance of making good recovery. The remaining 20% became invalids. Hyland in U.S.A. made a follow-up study in 1950 of 191 cases of subarachnoid hæmorrhage, of these 100 died within six months after the first attack; 70 had the fatal recurrence within two weeks of the initial attack.

This high percentage of early recurrence has also been stressed by Falconer and is of greatest importance in assessing indications for operation.

Spontaneous subarachnoid hæmorrhage does not, however, indicate an aneurysm and an aneurysm does not always give rise to subarachnoid hæmorrhage. But in 90% of cases subarachnoid hæmorrhage is caused by an aneurysm and if the infraclinoid aneurysms are excluded about 85–90% of all aneurysms cause subarachnoid hæmorrhage.

Other clinical signs that might influence the indications for operation are pain in the distribution of the trigeminal nerve and visual failure due to pressure on the optic nerve.

Several factors, chiefly the poor prognosis in subarachnoid hæmorrhage and the fact that carotid ligation does not prevent a new fatal rupture of the aneurysm, have made us change our attitude toward these lesions. In August 1950 it was decided that in the future an intracranial exposure would as far as possible be made in all cases of intracranial aneurysms admitted. Ligature of the neck of the aneurysm was considered the procedure of choice and would be used whenever possible.

Location of the Aneurysm.

Tables VIII and IX demonstrate the location of the aneurysms and the method used. Of these

TABLE VIII.—INTRACRANIAL EXPLORATIONS OF SACCULAR ANEURYSMS FROM AUGUST 1950 TO NOVEMBER 1951

Localization	Number of patients	Dead
Carotid artery	11	2
Bifurcation of internal car. artery	4	1
Proximal part of ant. cer. artery	1	
Ant. communicating artery	14	
Distal part of ant. cer. artery	4	
Middle cerebral artery ..	10	
	44	3

TABLE IX.—METHOD

	Ligature of the neck	Wrapping	Trapping	Exploration
Carotid artery	9		1	1
Bifurcation	4			
Proximal part of ant. cer. art.	1			
Ant. comm. artery ..	11	2		1
Distal part of ant. cer. artery	4			
Middle cerebral artery ..	5	4	1	
	34	6	2	2

44 cases 23 have been operated on by the author and 21 by Olivecrona to whom I express my gratitude for permission to use his material in this study.

In 34 cases the neck of the aneurysm was ligatured, in 6 a strip of muscle was wrapped around the vessel, in 2 the aneurysm was trapped between clips. In one of the latter bleeding from the carotid artery occurred when attempting to place a ligature around the neck, necessitating trapping the aneurysm between clips which was well tolerated. In the other case a small aneurysm was found in the peripheral branch of the middle cerebral artery. The aneurysm was excised.

There were 3 operative deaths. In one of the cases death was due to the development of an extradural clot diagnosed and operated upon too late following successful ligature of the neck of the aneurysm.

The second was the only case which did not regain consciousness after the first attack of hæmorrhage. Emergency operation was performed during artificial respiration following respiratory failure. An angiogram had revealed a large aneurysm of the carotid artery. A frontal flap was turned down. The intracranial pressure was found to be very high and the subarachnoid space filled with blood. The frontal lobe was resected, digital pressure was applied to the carotid artery in the neck and the intracranial part of the artery exposed. Severe bleeding from the aneurysm necessitated temporary occlusion of the carotid artery with a clip. It was now possible to place a ligature around the neck of the sac and the clip on the carotid artery was then removed. The patient, however, died a few hours later. At autopsy a large rupture of the sac was found.

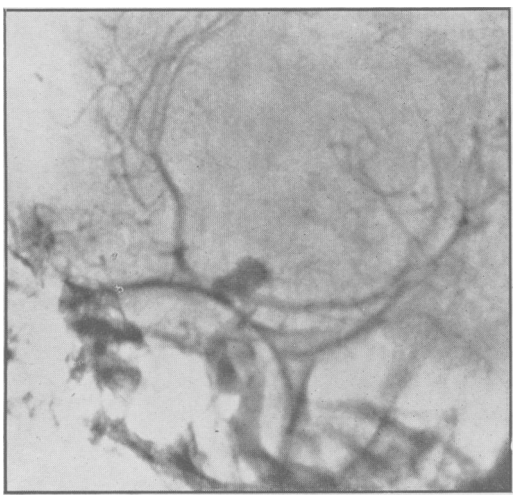

FIG. 1.—Aneurysm at the bifurcation of the left internal carotid artery.

The third case was a woman, aged 39, who had bilateral symmetrical aneurysms at the bifurcation of the internal carotid artery. She had lost consciousness after her first attack of subarachnoid hæmorrhage. On admission she was still drowsy, probably with signs of aphasia. She was observed for some days during which her condition fluctuated, one day more co-operative, another more somnolent. Nine days after the initial attack angiography of the left side showed an aneurysm at the bifurcation of the internal carotid artery (Fig. 1) and the following day an angiogram revealed an aneurysm of exactly the same location on the opposite side (Fig. 2A and B).

Immediately after this angiogram a left-sided hemiparesis developed and she became more drowsy. A stellate ganglion block was done and the hemiparesis cleared up. Close to the aneurysm on the right side the carotid artery as well as the anterior cerebral artery and the middle cerebral artery

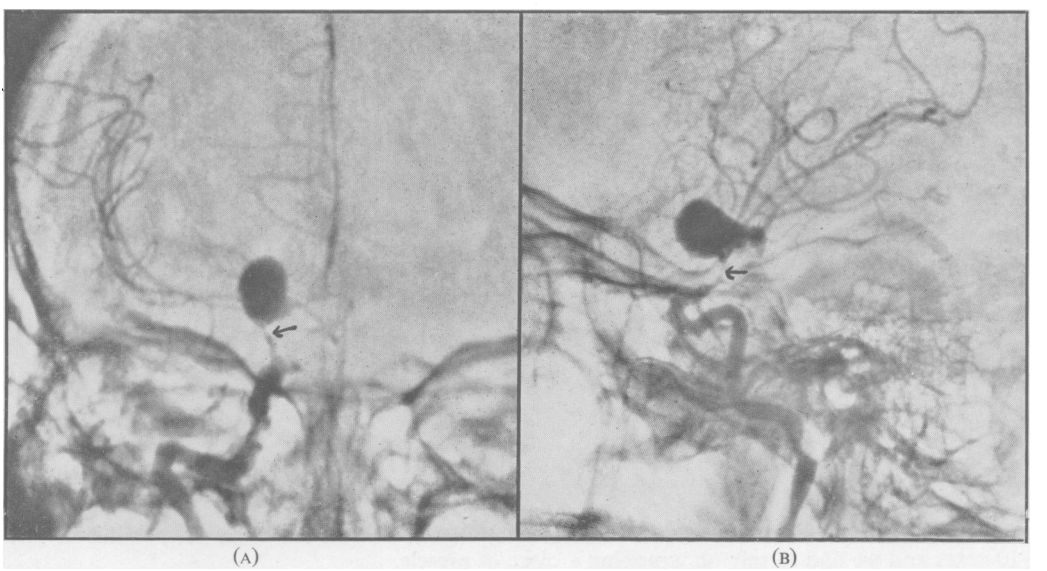

FIG. 2.—Aneurysm at the bifurcation of the right internal carotid artery. A, Frontal view. B, Lateral view. Note the vasoconstriction of the vessels close to the aneurysm, especially of the internal carotid artery just proximal to the aneurysm.

showed local vasoconstriction (Fig. 2A and B). At operation there was no difficulty in placing a ligature around the neck of the aneurysm, first on the right and then on the left side.

Shortly after the ligature was applied on the left side it was observed that the vessels changed their colour and size and became white and thin indicating pronounced vasoconstriction. At the same moment it was also noticed that the patient stopped breathing.

Presuming a reflex mechanism the ligature was removed and a clip was placed on the aneurysm slightly more distal to the main arteries. Stellate ganglion block and intravenous procaine were of no avail and the patient died the following day. Autopsy demonstrated that the ligatures were well placed around the neck of the aneurysms and the vessels had not been strangulated but were patent. No softening of the brain could be shown.

Other complications consisted of an extradural clot in one patient and post-operative brain œdema in another, both operated with success. 4 cases developed post-operative hemiparesis, one with complete recovery after stellate block, 2 with partial recovery and one became permanent. This last case, a woman of 46, was referred to the hospital after an attack of ophthalmoplegia and migraine. Angiogram demonstrated an aneurysm on the supraclinoid part of the right carotid artery (Fig. 3A).

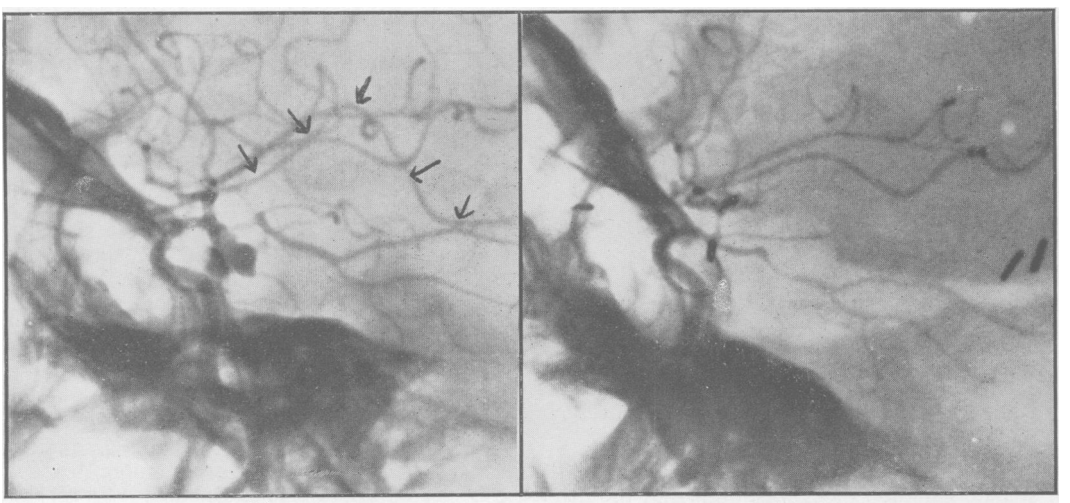

FIG. 3.—A, Aneurysm of the supraclinoid part of the internal carotid artery. Note the contrast filling of the posterior temporal artery. B, A clip is placed on the neck of the aneurysm. Note the absence of contrast in the posterior temporal artery.

At operation a clip was placed on the neck of the sac. In the afternoon the same day a slight left-sided hemiparesis was observed which increased during the following days to almost complete hemiplegia. There was no improvement in spite of the use of anticoagulants and sympathetic block. Angiogram three weeks later showed an occlusion of the posterior temporal artery in the Sylvian fissure (Fig. 3B).

In cases of aneurysm of the anterior communicating artery the study of the circulation of the circle of Willis by angiography is of utmost importance. In the present series the following variations were observed:

A. Filling of the aneurysm from both sides, 6 cases.

B. Filling of the aneurysm from only one side, 12 cases.

This latter group can be divided into two subgroups:

(1) In 3 cases filling of the aneurysm and anterior cerebral artery of the same side.

(2) In 9 cases filling of the aneurysm and both anterior cerebral arteries from one side. This group can also be divided into two subgroups: (*a*) no filling of the anterior cerebral artery from the opposite side, 7 cases; (*b*) filling of the anterior cerebral artery only from the opposite side, 2 cases.

We found that most aneurysms in this location, 10 out of 14, presented a neck suitable to ligation (Table IX). In one case only did the neck and the anterior communicating artery have to be ligated together.

In aneurysms arising from the middle cerebral artery ligation of the neck was not possible in 4 out of 10 cases and we had to rely on wrapping a piece of muscle.

In 3 cases post-operative angiography showed that the ligature was not successful. The clip was not well placed or had slipped.

Figs. 4 and 5 show angiograms of 2 cases before (A) and after (B) ligature.

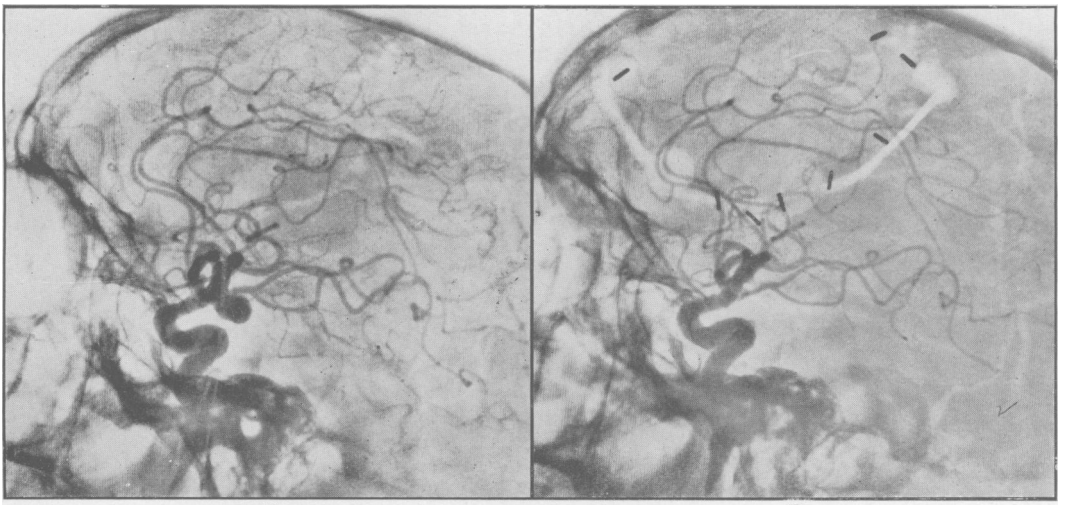

FIG. 4.—A, Aneurysm of the supraclinoid part of the internal carotid artery with a very wide neck. B, Angiogram after ligature of the neck of the aneurysm with linen thread.

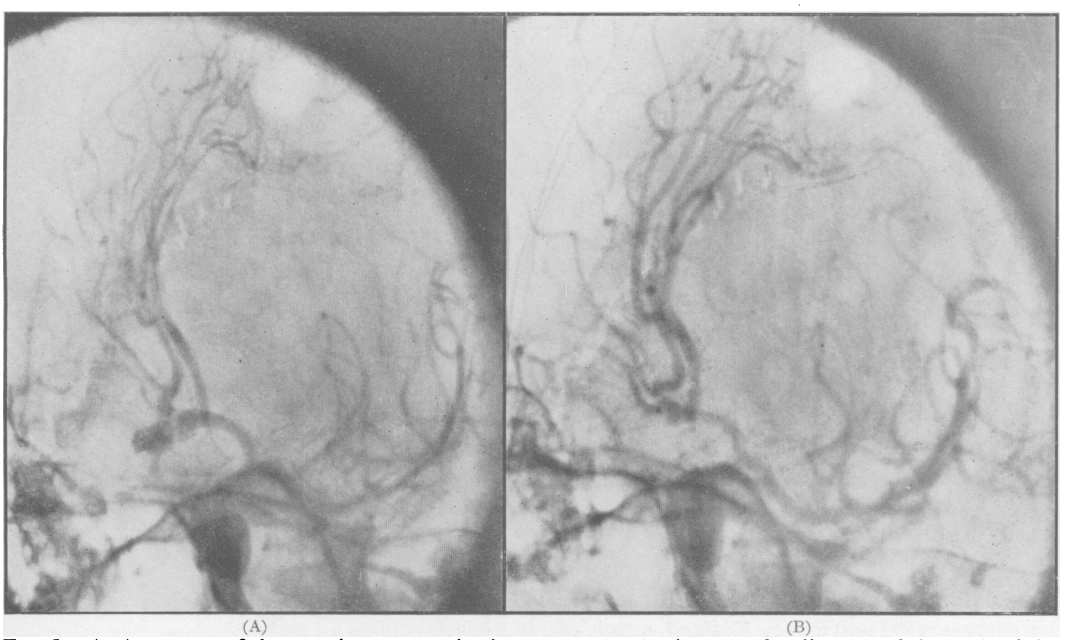

FIG. 5.—A, Aneurysm of the anterior communicating artery. B, Angiogram after ligature of the neck of the aneurysm, with blood flow through the vessel preserved.

Summary and Conclusions

When an aneurysm is suspected angiography should be used not only to establish the diagnosis and to show the size and position of the aneurysm but also to demonstrate the presence of a neck, the influence of the aneurysm on the intracranial blood flow and vessels, and the adequacy of a collateral circulation.

Recent studies, in particular by Hyland (1950), have clearly demonstrated the high mortality rate in cases of subarachnoid hæmorrhage. The high percentage of fatal early recurrences has been especially stressed.

Ligature of the carotid artery in the neck has in some cases not been adequate to prevent a new fatal rupture of the aneurysm.

In a series of 44 consecutive cases of aneurysms admitted to the neurosurgical clinic from August 1950 to November 1951 efforts have been made to treat these lesions by direct surgical attack rather than by carotid ligation or conservative measures.

In 34 cases it was possible to ligate the neck of the aneurysm, in 6 muscle was wrapped round the outside of the aneurysmal sac, in 2 the aneurysm was trapped between clips and in 2 no definitive treatment was possible.

There were only 3 post-operative deaths. Hemiplegia developed after operation in 4 cases; in 1 this was permanent, in 2 there was partial recovery and in the fourth complete recovery.

Based on these preliminary experiences direct surgical attack must nowadays be considered a fairly safe procedure in intracranial aneurysm of any location except those in the cavernous sinus. Carotid ligature is not necessary as a preliminary step in these cases.

This paper is one of a series presented to Professor Herbert Olivecrona on his 60th birthday by friends and pupils. The author wishes to express his deep gratitude for constant help and encouragement and for experience gained under his leadership.

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Mr. Murray Falconer: My experience of intracranial aneurysms has been chiefly with lesions associated with intracranial bleeding. About 75 to 80% of cases of subarachnoid hæmorrhage are due to aneurysms that mostly are small. I have had little experience of the large lesions which, unruptured, simulate a brain tumour. Recently I (1950 and 1951) reported my first 50 cases of bleeding aneurysm submitted to operation with 9 deaths. These cases had been diagnosed by performing carotid arteriography in a group of 69 patients with subarachnoid hæmorrhage admitted consecutively to a neurosurgical unit in New Zealand, mostly within a few days of an attack of bleeding. The selection of these patients was not my choice, but was made by various medical colleagues who, either because they were alarmed at the condition of their patient or because of previous experience of surgical help, transferred their patient to the unit, usually without a neurosurgeon having seen him beforehand. Half the patients were experiencing recurrent attacks of bleeding and several were unconscious. Consequently, as a group, their prospects with conservative treatment were particularly serious. It is therefore gratifying that, in the aneurysm series, 33 patients made excellent recoveries (66%), while 5 recovered with a slight disability (10%) and 3 recovered with a severe disability (6%)—a total recovery rate of 82%. In the non-aneurysm series, there were 4 cases of arteriovenous malformation, 1 case of primary intracerebral hæmorrhage, and 14 cases with normal arteriograms. 2 of these last cases died, making 11 deaths, or an overall mortality rate of 16%, for the entire group of 69 cases of subarachnoid hæmorrhage.

Prognosis of subarachnoid hæmorrhage treated conservatively.—At first mention, these mortality rates of 18 and of 16% for surgically treated cases may sound forbidding, but they are an improvement on the results of conservative management, figures relating to which are available in the papers of Taylor and Whitfield (1936), Richardson and Hyland (1941), Magee (1943), Hamby (1948), Hyland (1950), and Falconer (1951). These statistics show that in most general hospitals between 50 and 60%

of patients admitted with subarachnoid hæmorrhage die, while only a sixth to a third of patients leave in a satisfactory state of recovery. The ultimate prognosis is even graver, for Hyland in following up a group of patients discharged from hospital an average period of 6·1 years earlier found that 20% of them had subsequently died of recurrent hæmorrhage.

One reason for this high rate is the marked tendency to recurrent bleeding. Between a quarter and a third of patients die within a few days of their initial attack, while subsequently half the survivors develop a recurrent attack, even while they are still in hospital. The incidence of these recurrent attacks reaches its peak from one to three weeks after the first attack, but the possibility of recurrence still remains for months and even years. Some patients experience several attacks. With each recurrence, the mortality expectancy rises higher. Consequently if neurosurgical treatment is to be tried, it should be undertaken preferably at an early stage before recurrent bleeding gets under way.

Value of early carotid arteriography.—Bilateral carotid arteriography is necessary to establish both the nature of the bleeding lesion and its precise site and size. It is therefore the first stage in the surgical management of subarachnoid hæmorrhage. For choice we carry out the procedure as early as the second or third day, certainly within the first week after onset of symptoms. The percutaneous technique is satisfactory provided an expert does it, but the test should always be carried out on the threshold of an operating theatre lest fresh bleeding is provoked. Examination of both carotid arteries is necessary because in about 10% of cases multiple aneurysms are present. It is also useful to investigate the collateral circulation through the circle of Willis. Thus if the carotid artery on the side of the lesion is compressed and the opposite carotid artery is injected, one can often demonstrate that this artery will supply both cerebral hemispheres. When carotid arteriography has not disclosed a lesion, one often proceeds to vertebral arteriography a few days later.

In my experience arteriography will reveal an aneurysm in about 75 to 80% of cases of subarachnoid hæmorrhage, an arteriovenous malformation in about 8 to 10%, and usually no abnormality in the remainder. Often the presence of an associated intracerebral clot is indicated by localized displacement of cerebral arteries. Leaking intracranial aneurysms tend to occur at certain particular sites, and as these sites give characteristic angiographic patterns, the aneurysms may be classified on a regional basis as follows:

(1) On posterior aspect of intracranial internal carotid artery in relation to the posterior communicating artery, 38% of cases in my published series.
(2) At bifurcation of internal carotid artery, 8% of cases.
(3) On middle cerebral artery at first point of branching of artery within fissure of Sylvius, 20% of cases.
(4) On proximal portion of anterior cerebral artery, 4% of cases.
(5) On anterior cerebral artery in relation to the anterior communicating artery, 22% of cases.
(6) Distal course of anterior cerebral artery at point of branching of artery, 8% of cases.

Aneurysms can also occur on the vertebral-basilar arterial system, but they are not common, and my experience is still limited to a single case (Falconer, 1951). Aneurysms of the internal carotid artery within the cavernous sinus (infraclinoid aneurysms) have not in my experience caused subarachnoid bleeding.

Place of carotid ligation.—Once an aneurysm has been demonstrated, its surgical treatment depends largely on its situation. Two different methods of attack can be employed, a carotid ligation and an intracranial attack, and these can be used either singly or together. Carotid ligation is the sheet-anchor of treatment for aneurysms of the internal carotid artery itself, and for these lesions may be employed alone, but for aneurysms on the circle of Willis or on the distal cerebral arteries it is of much less value owing to the collateral circulation through the circle of Willis. Even with aneurysms of the internal carotid artery, carotid ligation alone may not be sufficient to prevent recurrent bleeding at a later date. This is suggested by the observation that, some months after ligation, the aneurysm often may still be demonstrated by performing arteriography of the opposite carotid artery, the aneurysm filling by reflux from the circle of Willis, although perhaps a little smaller than before carotid ligation.

Carotid ligation in the presence of subarachnoid bleeding carries an appreciable risk, as witness the figures of Hermann, Obrador and Dott (1937), Schorstein (1940), and Krayenbühl (1946), who between them reported mortality rates of from 25 to more than 50%. Death in many instances is largely due to the intracranial bleeding, but to this may be added an anoxic effect resulting from interference with the blood supply. Some workers, and I understand that Dr. Norlén is included among them, have tried to lessen the risk of hemiplegia by measuring the intra-arterial pressure distal to the trial occlusion of the carotid artery, and then proceeding to carotid ligation only if the distal arterial pressure is above a certain figure. I have not tried this method, but have relied on observing the effects of a trial period of occlusion of the artery for twenty to thirty minutes with a Crile's clamp, while it is exposed under local analgesia. My preference is to ligate the common carotid artery in two stages and then the internal carotid artery in a further stage. Even when this course is followed, a delayed hemiplegia will sometimes supervene.

In my published series 36 cases were treated by carotid ligation with 6 deaths; 20 of these cases, including 2 of the fatal cases, were also submitted to an intracranial procedure.

Place of an intracranial approach.—The intracranial approach allows of a more definitive treatment of an aneurysm, and although I do not think this approach should be applied as a routine, in most cases

it offers prospects of a more secure recovery to compensate for the added risks. In my published series an intracranial exposure, with or without preliminary carotid ligation, was undertaken in 34 cases with 5 deaths.

By means of a frontal craniotomy it is normally possible to expose an aneurysm of the internal carotid artery or of its anterior and middle cerebral branches without damaging or sacrificing any cerebral substance. Aneurysms of the middle cerebral artery are exposed by splitting open the anterior part of the fissure of Sylvius, and of the anterior cerebral artery by opening up the great longitudinal fissure. Often aneurysms in these two latter situations are associated with sizable intracerebral clots in the adjacent frontal and temporal lobes, and an essential part of the operative procedure is to suck out these clots which are acting as space-occupying lesions. On several occasions now, I have succeeded in reviving unconscious patients by so doing.

One of the risks in exposing an intracranial aneurysm is that it may burst while it is being dissected out. This has happened now and then, but I have always managed to control the bleeding with hammered muscle. One of the advantages of a preliminary carotid ligation is that, if the aneurysm should burst, the force of bleeding is less. Recently we have made some attempts at lowering the systemic blood pressure with the new sympathetic-paralysing drugs, but not without some difficulties.

Once the aneurysm is exposed, we can apply a variety of local measures, e.g. wrapping with muscle, clipping of its neck, trapping by occluding the parent artery on either side of it, diathermy cauterization of the sac, and even in certain favoured sites excision of the aneurysm itself. I have not yet tried Norlén's method of ligating the aneurysmal neck, which from his photographs seems very suitable for lesions with accessible necks. But it has its limitations, as when an aneurysm has a wide neck or is so closely surrounded by diverging arterial branches that it is not possible to place the ligature in position without including one or more of these branches. The method which, to date, I have found most generally practicable is that of wrapping pledgets of hammered muscle around the aneurysm. This method was first introduced by Professor Norman Dott (1933), and his patient subsequently lived for many years before dying of an unrelated illness. My observations confirm that this method is probably of permanent value.

Magnitude of problem of treating bleeding aneurysms.—Subarachnoid hæmorrhage and bleeding intracranial aneurysms are relatively common lesions. My experiences show that surgical measures can be applied to the great majority of cases of leaking intracranial aneurysm, and not just to a selected minority. I feel that in the years to come most cases of subarachnoid hæmorrhage will be admitted to our neurosurgical units for investigation and treatment. Judging from statistics derived from New Zealand, about a hundred cases of subarachnoid hæmorrhage occur per million of population each year, and about half of these die (Falconer, 1950).

If further and wider experience confirms the belief that these patients have better prospects with surgical treatment than with conservative management, the time will come when the number of patients with cerebral aneurysms seen in our neurosurgical centres will rival if not surpass the number of patients with brain tumour.

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Sir Geoffrey Jefferson: Any method of treatment stands or falls by its success or failure. We have now had in my own Service enough aneurysms (250) and have treated surgically enough of them (177) to have formed conclusions that give, at the least, a good working basis. The great majority have been treated by Hunterian ligature, i.e. by ligature of the common or internal carotid arteries or both in the neck. This has now been done in 142 cases and of these 12 died during the first six weeks, 6 of a second rupture within one month of ligature, 4 were *in extremis* and probably should not have been operated on at all, 1 died of rupture of a second unsuspected aneurysm on the vertebral artery, and 1 died of basal compression by a large aneurysm. In no case could the death be related to the ligature itself, the patients died in one way or another of their aneurysms, i.e. by intracerebral clots or lacerations caused by bleeding or by irreversible damage to the anterior hypothalamus and "visceral brain".

There were 8 late deaths, 3 known to have been from hæmorrhage. Of these 1 died six years later from multiple aneurysms on the other side, 2 died from a rupture of the original aneurysm two and six years respectively after ligature. One died from a vast suprasellar aneurysm in which the wrong carotid had been tied five years before: there was no leakage here, it was the mass that was eventually fatal. The rest died from causes unconnected with the aneurysm. Perusal of these figures shows that if we omit the leaks from concurrent aneurysms on the opposite carotid tree or vertebral artery

we have to record 8 cases in which the same aneurysm bled again in spite of the carotid ligature, but only in 2 so far, who survived the first month. 20 cases in all died from various causes after carotid ligature. To have alive 122 cases, some many years old now, indicates to me that the method is one that has, to put it soberly, considerable merits.

I have to ask myself whether all these cases are safe. I cannot say that. The presence of an aneurysm is a serious thing and no absolute forecast can be made of the future of many of them. But we do not yet know how secure those patients are who have had direct attacks made on them (35, of whom 5 died promptly). And we have to remember above all that a certain number of aneurysms are quite impossible problems for direct attack. It would take too long to illustrate this most important pathological point in detail but there is still one thing to which attention must be drawn, namely, the neck of aneurysm. This neck is too often a radiological appearance and not an anatomical reality. All or nearly all aneurysms are sessile and spring from a considerable segment of the length and the total circumference of the artery. Often, it must be that the surgeon who records clipping or tying the

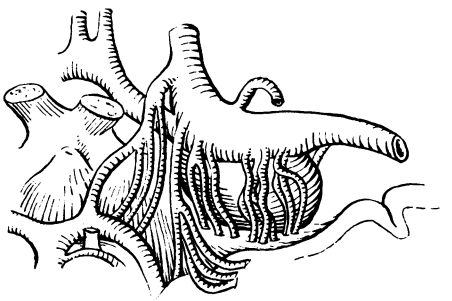

FIG. 1.

FIG. 1.—Middle cerebral aneurysm. Observe absence of "neck" and proximity of perforating arteries likely to be occluded by encircling ligature.

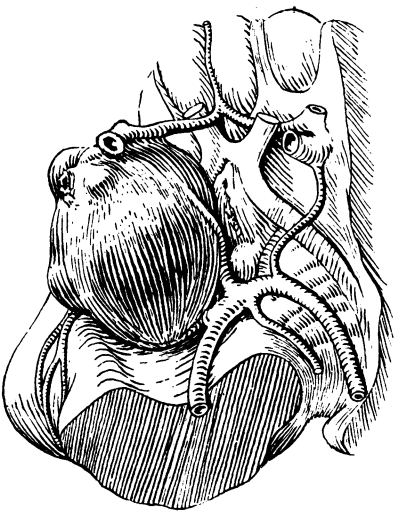

FIG. 2.

FIG. 2.—Aneurysm of internal carotid at origin of posterior communicating artery. Note wide base of origin—no "neck".

"neck" is constricting the aneurysm distal to its exact point of origin, as indeed Norlén has just shown in some of his post-operative angiograms. Whether this, none the less, confers immunity from further bleeding, time alone will show. It is for this and other reasons that one must demand an exact statement as to the real point of ligature in all future records. The aneurysms that cannot be treated by carotid ligature are those of the anterior cerebral where the aneurysm has been shown by bilateral angiography to fill equally well from both sides. The middle cerebral group occupies a middle position, for even though we may not be very satisfied with carotid ligature, everyone who has dissected them out knows that few can be dealt with locally by clipping, and that a ligature cannot be tied round them without including the branches of middle cerebral supply.

My colleague, Mr. Richard Johnson, will speak of the effects of ligature on the carotid circulation.

Mr. Richard Johnson: We know that proximal carotid ligation almost invariably reduces the size of aneurysms on the circle of Willis; the evidence is (1) clinical improvement in local signs, where present; and (2) visualization by angiography of an aneurysmal sac much reduced in size following ligation. There is also some evidence that ligation reduces the risk of subsequent leakage, although we have shown that it is not an absolute safeguard against further rupture, especially in the acute stage. About half the patients in this series had aneurysms which had leaked and in 29 cases the carotid was ligatured within a few hours of a severe hæmorrhage; 17 of these patients survived to be discharged from hospital and of these only 2 have since bled.

Ligation quite clearly has definite indications; it is sometimes an extremely valuable method of treatment in the very large aneurysms of the circle, which plainly offer no encouragement to a direct attack; and it is, furthermore, extremely effective in the treatment of infraclinoid aneurysms. Direct attack, on the other hand, is the method of choice for peripheral aneurysms which can be excised, or those which can be ligated (perhaps with the sacrifice of one or more relatively unimportant arterial branches). The best method of treating the small and moderately sized aneurysms of the circle itself (by far the largest and most important group) is not so apparent. Ligation may be of no value where there is free communication across the circle, or may be disastrous where the flow is inadequate, and yet our experience must mean that, between these extremes, it is for the most part effective. Exploration of the aneurysm, on the other hand, may be disappointing in revealing inoperability and carries a mortality and morbidity which at best is not very different from that of carotid ligation. Dr. Norlén's

impressive series demonstrates what advances in technical skill can do to render operable the great majority of basal aneurysms and it is doubtful if his results will ever be bettered. Eventually experience may determine clear indications for treatment, but at present it seems that for certain aneurysms, e.g. those in the region of the posterior communicating artery, carotid ligation and definitive surgery will have to stand trial side by side.

We attempted to solve some of the problems connected with the complications of ligature; the most serious is hemiplegia and in this series (150 cases) there were 11 instances, some transient, but 7 showing only that degree of recovery which one associates with a dense lesion. Some were slightly improved by removal of the ligature but there were no dramatic recoveries in the severe cases nor did sympathetic interruption affect them. Thrombosis or embolism in a main vessel would seem at first glance to be the most likely cause, but few observers have found these in fatal cases. Our evidence is that ischæmia, occurring either as a direct result of pressure drop in the vessels or as a result of a pressure fall allowing vessels stretched over an aneurysm to close down, is the cause in most instances.

For such reasons I commenced, some four years ago, to enquire in more detail what happened to the cerebral circulation when the carotid arteries were ligated in the neck. What effect did ligation have on the aneurysm (1) immediately, and (2) years later? What were the risks of carotid ligation, which carotid (common or internal) was it safer to tie, and which the more effective? These results were given in a Hunterian Lecture in 1950. In brief, the common carotid was ligated first, then, at various intervals, the internal was tied. At the second exposure an angiogram was made to show the sac and diodone was trickled into the common carotid and films taken to determine the direction of flow (demonstrated at the International Congress of Radiology, London, 1950). These investigations showed (1) that common carotid ligation reduced the size of an aneurysm, the fundus being partly thrombosed, and (2) that after an interval of six weeks or more the flow was from external to internal carotid, i.e. the external collaterals had opened up. Evidence obtained post mortem on 2 patients six years after ligation supported this: one after common ligation had bled from the same aneurysm; the other after internal ligation had bled from an aneurysm on the other side. It was apparent that after common ligation collaterals had opened up through the external and there was little difference in the calibre of the cavernous carotids of the two sides; the aneurysm, still large, had burst. After internal ligation, however, the carotid remained small up to its main branches and the aneurysm was shrivelled and fibrosed. Two years later we confirmed the work of Sweet and Bennett that common ligation reduced the mean carotid pressure by 40% to 60% and that although the external pressure was usually slightly higher than the internal, the difference was not great. We discovered, however, that in most cases after an interval of months, the external pressure rose as the collaterals opened up and that a further pressure drop could then be obtained by internal ligation. These results mean, in effect, that initially, common ligation is only slightly safer than internal ligation but that eventually internal ligation is more effective.

Sir Charles Symonds: Mr. Murray Falconer's conclusions concerning the mortality rate in patients with subarachnoid hæmorrhage who were not operated on were drawn from cases admitted to hospital and a considerable proportion of these patients were probably admitted because their symptoms were unusually severe. My experience suggested that many patients had subarachnoid hæmorrhages without being admitted to hospital at all. Therefore the prognosis for all cases of subarachnoid hæmorrhage not surgically treated was probably a good deal better than Mr. Murray Falconer's figures suggested.

Mr. D. W. C. Northfield: Dott and Adams McConnell were, I believe, the first to use muscle to pack around the aneurysm or in its cavity. I have operated upon a number of such cases, attempting a direct attack upon the aneurysm, in preference to carotid ligature. In those cases in which the aneurysm arises from the posterior aspect of the internal carotid artery close to the origin of the posterior communicating artery I have noted one point in its situation which I think is not usually perceived at necropsy. The sac may pass inferior to the sharp edge of the tentorium, and from the site of bleeding which has on occasion been encountered, it has seemed likely to me that spontaneous rupture may result from impingement of the sac on this sharp dural edge. In order to obliterate the sac, I have attempted to clip its neck; or have placed several clips across the sac itself. Where this has not been practical, I have surrounded the sac with a layer of muscle or of thin wisps of cotton-wool soaked in Thorotrast, hoping thereby to stimulate the formation of a barrier of firm scar. I have felt it important to prolong this packing over and around the parent vessel, much as a plumber "wipes" the T joint he makes with lead pipes. I have wondered whether ligating or clipping the neck may not still leave a weak area in the wall of the parent vessel, potentially another aneurysmal bulge. Reinforcing the aneurysm and binding it to its vessel of origin may possibly overcome this weakness. Reinforcement is also a method worth trying on aneurysms possessing several vital branches, provided one can adequately insinuate the material around it.

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