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SURGERY IN SPONTANEOUS SUBARACHNOID HAEMORRHAGE

OPERATIVE TREATMENT OF ANEURYSMS ON THE ANTERIOR CEREBRAL AND ANTERIOR COMMUNICATING ARTERY

BY

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The natural history of spontaneous subarachnoid haemorrhage from its various causes (of which aneurysms form some 80%) and the mortality to be expected from it under conservative treatment have been clarified for us in recent years as a result of a number of reports concerning large series of patients (Hyland and Richardson, 1941; Magee, 1943; Wolf *et al.*, 1945; Hamby, 1948; Ask-Upmark and Ingvar, 1950; Hyland, 1950; Walton, 1952, 1955), but the same clarity unfortunately does not pertain to the surgical aspects of this condition, in particular to that of direct intracranial attack on aneurysmal haemorrhage. Despite the recent appearance of a large number of surgical publications it still remains a fair statement to say that there has been nothing written which dispels the widely held impression that to operate in the acute phase, within three weeks of the bleed, results in a prohibitive mortality, and that to delay surgery until the quiescent phase, after three weeks, results in a lower operative mortality but saves very few lives from aneurysmal haemorrhage.

This surgical stagnation stems from a number of reasons; partly of course from the inadequacy of the surgery practised, but partly also from the method of reporting operative series. For the dominating factor in aneurysmal haemorrhage, and that which largely determines the operative mortality, is the time interval between the bleed and the operation. In the majority of surgical reports this interval is not mentioned, so that it is not possible to assess the gravity of the patient's condition at the time of operation, nor to compare one surgical series with another, nor, more important, to compare any operative series with the results of conservative treatment.

It is the omission of this information which also explains the wide variation in operative mortality (from 50% to as low as 3.6%) in what otherwise appear to be similar aneurysmal series. Another factor which adds to the confusion is that in any surgical series a number of different types of operative procedure are used, all of which vary in their individual hazards and risks, such as ligation of the internal or common carotid artery in the neck; radical ligation or clipping of the neck of the sac; or surrounding the sac wall with a coat of hammered muscle to support it from the outside, a procedure which surely must have more effect on the surgeon's peace of mind than on the aneurysm. Another factor which must enter into consideration and which is not accurately known at the present time is, Do aneurysms in all situations behave similarly? Do "subarachnoid" aneurysms on the large internal carotid trunk carry the same natural mortality as the "intracerebral" aneurysms on the smaller-calibre vessels—the anterior and middle cerebral—or is the variation so great that a surgical mortality which is acceptable for one group may be excessive for another?

It seems that before we can assess what can be done to prevent mortality from a condition which includes so many variable factors as does aneurysmal haemorrhage, it is imperative to study, as separate groups, aneurysms in each particular site, and to ascertain the mortality and morbidity of a series conservatively treated and then compare them with a similar group treated by one type of operation, with especial regard for, and reference to, that crucial factor the time interval between the bleed and the operation.

This paper concerns an attempt to do this in that group of aneurysms situated on and about the anterior communicating artery and its junction with the anterior cerebrals, a group which forms between 25 and 30% of supraclinoid aneurysms and which bears a sinister surgical reputation.

The series consists of 73 patients with ruptured aneurysms in this situation: 36 were treated conservatively by the usual methods, of which bed rest for six weeks was the essential feature, and form the control group; and 37 were subjected to operation.

Conservatively Treated Group

It is notoriously difficult to obtain a truly unselected series in this condition; for selection, natural selection, starts with the patient outside hospital—the most severe cases going straight to the mortuary and the mildest cases not coming to hospital at all—and continues at each step through hospital. The best method seems to be to set out the reasons for adopting conservative treatment in these 36 cases and let the reader judge for himself their representative nature; Table I gives the reasons for conservative treatment.

TABLE I.—*Reasons for Adopting Conservative Treatment in 36 Control Cases*

Group	No. of Cases	Reasons
1	12	Admitted after a single haemorrhage and had no symptoms
2	15	Deemed too ill for surgery owing to further subarachnoid haemorrhage(s), as a result of procrastination in referring patient for investigation and/or delay with investigation
3	4	Had an anomalous arrangement of the anterior part of the circle of Willis demonstrated by arteriography; or an unsatisfactory Matas test
4	5	Had sustained severe neurological damage, usually as a result of a late thrombosis

All the 73 cases were admitted to one of two hospitals, in which the conditions of admission did not vary over the years. Two-thirds of the control group were seen in the years 1949–52 during the stage of surgical disillusionment (which followed the phase of enthusiasm prevalent in the early 1940s) in which any excuse was grasped at to avoid operation, and in fact only seven cases outside the control group in these four years were subjected to direct intracranial surgery.

Of the operation group, on the other hand, three-quarters of the patients were treated in the years 1952–4 inclusive, so that, although overlapping to some extent, the two series were in the main not concurrent.

The series is not a very large one but is representative, and conclusions can be drawn from it.

Mortality of Conservatively Treated Group.—Of these 36 patients, 16 died from the effect of haemorrhage, a mortality of 44.4%. A striking feature, however, is that only one patient died from the effects of the initial haemorrhage—some 6% of the total deaths, compared with the figure of 25–30% given for subarachnoid haemorrhage generally. One died from the effects of a late thrombosis, a condition to which reference will be made later, and the other 14 patients died from recurrent haemorrhages. It would therefore seem that among hospital cases an anterior cerebral aneurysm usually takes two or more bleeds to kill. This is indeed a fortunate dispensation, because it means that the vast majority of these cases are available for preventive surgery.

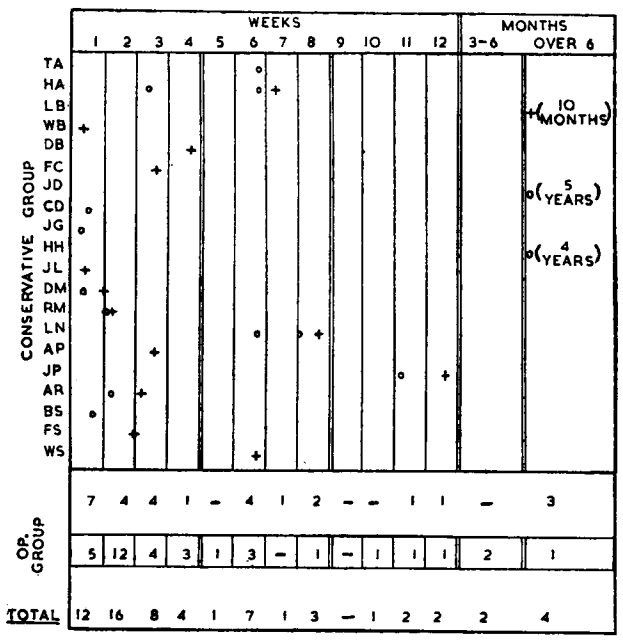

FIG. 1.—Of the conservatively treated group of patients 20 bled again, with 14 deaths. The circles indicate recurrent haemorrhages and the crosses recurrent haemorrhages which resulted in death. Of the group treated by surgery 25 had further haemorrhages. In the conservatively treated group it will be seen that at the end of four weeks from the initial bleed 9 deaths have occurred out of a total of 14 from recurrent haemorrhage, so that at this stage out of 25 survivors 5 more are destined to die—a risk of 20%. After eight weeks a total of 12 out of 14 deaths have occurred, so that now out of 22 survivors only two more are due to die, a risk of 1 in 11. Note that there are only three late recurrent haemorrhages (after six months), at ten months, four years, and five years respectively, with one death.

Recurrent Bleeding

Fig. 1 shows the incidence of recurrent haemorrhage, and in 20 of the patients the aneurysm bled again. In order to obtain as large a series as possible the operative group is also included, and of these 25 had recurrent haemorrhages prior to operation. It would seem that the peak for recurrence of this total of 45 patients is in the second week, in agreement with most statistics, and there is a smaller risk in the third, with a secondary peak in the sixth week, but there are a formidable number in the first week. Even so, it is a striking fact that in none of these 45 cases did a recurrent haemorrhage occur within the first two days after the initial (or warning) leak.

As a counsel of perfection for the management of these patients, therefore, we must strive to admit, investigate, and operate on them within the first two days following the first haemorrhage in order to prevent the maximum number of haemorrhages and save the largest number of lives—as well, of course, as preventing the augmenting morbidity and the agony of repeated subarachnoid haemorrhage, for the saving of life in this condition is not our sole concern.

If we now turn our attention to the deaths from recurrent haemorrhage in the control group we see from Fig. 1 that at the end of four weeks from the initial haemorrhage the risk of dying from a further bleed has dropped to 20%. At the end of eight weeks the chances have dropped to as low as 9%.

No operative mortality has been reported up to the present time low enough, in general, to justify surgery at this stage, after the lapse of eight weeks, and, in the event of recourse to operation, out of every 11 operations performed 10 will be unnecessary anyhow.

It is my practice at the present time, if a patient is seen eight weeks or later after haemorrhage from an anterior cerebral aneurysm, and appears to be well, not to advise any form of surgical treatment, for nature offers the lesser risk.

Late Haemorrhage

The incidence of late recurrent haemorrhage—that is to say, after six months—is remarkably low. There are only three cases, with one death, so that late haemorrhage in this particular group of aneurysms is a small hazard. The 20 survivors have now been followed up for a period of from nine years to seventeen months, an average of forty-eight months.

The characteristics of this group of anterior communicating/anterior cerebral aneurysms are now made clear, and it is seen that they differ in some respects from the average picture of aneurysms generally, although the total mortality is roughly the same. Thus there is a low death rate from the initial haemorrhage. Two or more bleeds are required to kill the patient, and there is usually an interval of at least two days between the first and subsequent bleeds. There is a high recurrent haemorrhage rate in the first two weeks, and some 88% of the deaths have occurred within the first eight weeks. Late bleeds, after the expiration of six months, are not an important consideration.

Morbidity

Of the 20 survivors, 4 showed serious neurological damage: one remained demented and hemiplegic, an inmate of a mental hospital until death two years later from coronary disease; one showed permanent memory defect and inability to work; the two others had poor memory, hemiparesis, and dysphasia, but were capable of doing

unskilled work. Five had lesser neurological symptoms which cleared up completely, and 11 had no persistent signs; all these have returned to full work, although often with some complaint of headache or giddiness.

The striking feature in this group of anterior cerebral aneurysms is not so much the residual neurological damage that may occur, but the remarkable degree of recovery that can take place, with the passage of time, in patients gravely crippled, both mentally and physically.

Symptomatology

It is not possible to go into the detailed symptomatology of these lesions—this forms a fascinating study in itself—but there are two points which need brief mention. Although in well over half the cases coma was the first sign, among those who did not lose consciousness initially were some 15 patients in whom the site of the aneurysm was suggested by the early symptoms; in seven these consisted of weakness of both legs and in eight patients blurring of vision or transient blindness, complaints which it is generally agreed are suggestive of an anterior communicating situation.

The other feature concerned the condition of late thrombosis, which occurred spontaneously between the seventh and the fifteenth day after the initial or subsequent bleeds and produced a varied symptomatology ranging from a mild hemiparesis to a complete capsular syndrome of hemiplegia, hemihypaesthesia, and hemianopsia often associated with dysphasia, and with some deterioration of conscious level. The symptoms came on, sometimes dramatically, sometimes spread over a number of hours, without, however, any signs of intracranial haemorrhage. There was no headache or stiff neck, and on lumbar puncture a yellow fluid without blood was withdrawn. This occurred in 6 of the 73 cases, an incidence of about 8%. It is important to recognize the complication, which seems probably to be due to thrombosis of some of the perforating vessels, and not to confuse it with a further bleed, for it is inadvisable to be stampeded into operating at this stage in the presence of actual infarcted brain.

Recovery from the neurological damage in these patients was less complete and slower than that occurring at the time of the ictus, and, as already mentioned, in this series one died from this complication, which appeared at the fifteenth day.

Patho-physiology

Some aspects of the changes that take place after aneurysm rupture need to be considered, although still to some extent speculative, for they not only play a dominating part in the production of physical signs, but also determine to a large extent the mortality that may result from surgical treatment in the acute stage.

When the aneurysmal sac ruptures, usually at the fundus (and not the neck as is sometimes stated), blood at high arterial pressure erupts into the subarachnoid space or brain tissue for a short time.

This active bleeding phase lasts probably only a matter of seconds and only a small quantity of blood escapes, in any event so far as anterior cerebral aneurysms are concerned. It has been estimated on theoretical grounds by Lindsay (1950) that the escape of 3 ml. of blood can give rise to a count of 100,000 red cells in the lumbar C.S.F., which is quite heavy blood-staining, and 3 ml. can be shed in a very short time, as any surgeon who has been unfortunate enough to burst one of these aneurysms at operation will agree. Now, the immediate response to this rupture is for the vessels in continuity with the aneurysm and for a varying distance around it to go into intense spasm, and this spasm slows up, perhaps for a time even arrests completely, the blood flow through the sac. With the resultant relaxation of tension on it the sac wall can shrink a little; the tear will narrow and become sealed with clot. The spastic vessels now tend to relax, probably gradually and at different rates in different individuals, and blood under increasing pressure flows through the sac again. If the seal of clot is strong enough there is no further haemorrhage; if not it gives way and the whole process is repeated.

Now this spasm, although the crucial factor in stopping haemorrhage and saving life, is harmful in other ways, for it must render the brain ischaemic in its territory of supply, and on the intensity and duration of this initial spasm will depend to a large degree the extent and severity of the neurological signs, and, although the spasm may relax quite quickly (but usually not completely), recovery from the acute ischaemic damage produced in the first few minutes after the bleed may take much longer—hours, days, or weeks—and may, in fact, never be complete.

Blood disrupting the brain, so far as anterior cerebral aneurysms are concerned, is a less potent factor in producing neurological signs, but assumes more importance with successive bleeds and is the common terminal incident.

It will be seen that the aneurysm itself acts as a detonator, emitting blood for a few seconds, with the production of longer-lasting spasm, the neurological results of which persist longer still.

It seems that this vascular spasm may be responsible not only for the arrest of haemorrhage but also for (1) death (in those in whom at necropsy no intracerebral clot is found); (2) the initial coma; (3) to a large extent, the neurological damage; and (4) partly for the intense headache.

The precise mechanism responsible for the occurrence of spasm is still the subject of pure conjecture. It may be due to irritation of the vessel from outside by blood or clot in contact with its wall, to stretching or traction on the vessel by clot, or possibly reflexly from the tearing of the intima.

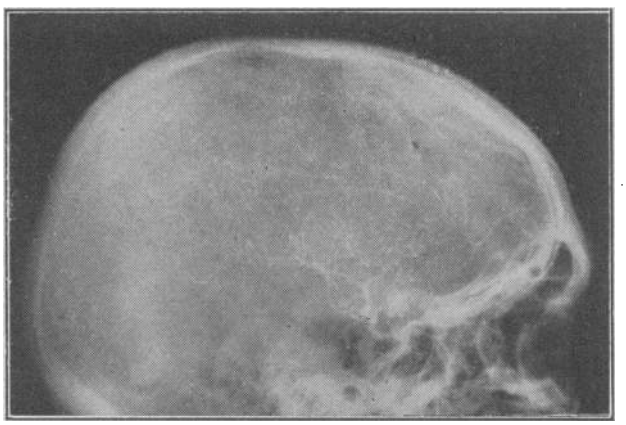

FIG. 2.—Lateral arteriogram taken on the seventh day after haemorrhage, which reveals an irregular sac with severe spasm of the proximal and distal anterior cerebral artery. The commencement of the first main branch distal to the aneurysm is also involved in spasm, and it would seem reasonable to assume that the smaller branches, the perforating vessels supplying the hypothalamus, basal ganglia, etc., which arise closer to the sac, and which cannot be demonstrated arteriographically, are in similar spasm.

The important spasm is not so much that which affects the main arterial branches and which can be demonstrated arteriographically but that which occurs in the small perforating vessels that cannot be seen and which produces ischaemia in the hypothalamus, basal ganglia, and the internal capsule (Fig. 2).

It is the effect of this spasm in these important areas of brain in the vicinity of the anterior cerebral aneurysm, aided to some extent by intracerebral bleeding when it occurs, that is responsible for the high mortality from direct surgical attack in the acute phase. The retraction and removal of brain in this vital area during the exposure of the aneurysm and the intricate anatomy of the anterior communicating and anterior cerebral junction must add greatly to the ischaemic damage already present and in some cases must render sublethal damage lethal, and, however skilled we may become in defining and clipping the necks of aneurysms in this situation, it is this unavoidable damage

to the ischaemic vital areas from retraction which must remain the eventual limiting factor and the cause of the high mortality.

The conclusion is this : If we are to operate in the acute phase on aneurysms in this situation we must influence the aneurysm, and prevent its rupture, at a distance from this ischaemic area. Carotid ligation, however, appears to be too remote, for out of a series of nine cases so treated there were two recurrent bleeds, both fatal, and this result has, with other considerations, led to the adoption of the operation to be described.

Surgical Principles

Three principles of treatment to be considered in the surgery of these aneurysms, which are illustrated in the dramatic *x*-ray picture (Fig. 3), concern (1) the removal of the haematoma, (2) treatment of vascular spasm, and (3) prevention of further haemorrhage. Now, the intracerebral haematomas associated with aneurysms in this situation do

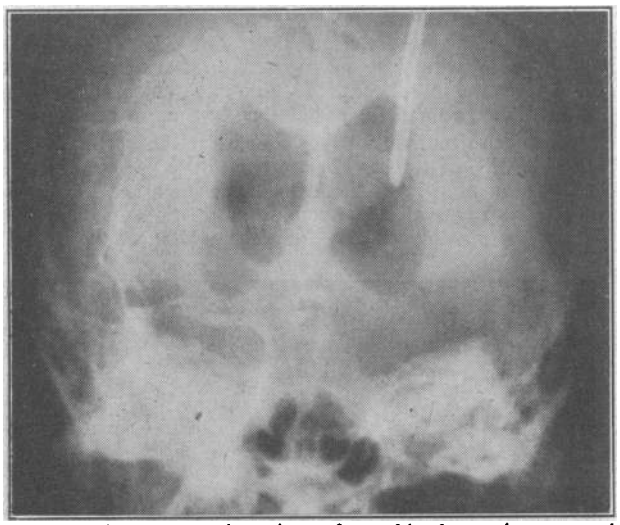

FIG. 3.—Antero-posterior view of combined arteriogram and ventriculogram which shows (1) blood clot in the brain substance and ventricles, (2) spasm of the anterior cerebral artery, and (3) an irregular aneurysm on the anterior communicating artery. (A catheter has been introduced through a burr-hole in an attempt to remove the ventricular clot.)

not act as expanding or compressing lesions (temporal lobe clots from middle cerebral aneurysms are in a different category), and their removal has little effect on the gravity of the patient's condition. It is the erupting fluid blood that does the harm, not the subsequent clot.

If one could relieve the vascular spasm in the earliest phases after the haemorrhage there is little doubt that it would influence the patient's condition, but secondary dangers would arise as a result of a full blood pressure playing too early on a weakened sac wall, with the possibility of further leaking. In any event, at the present time no treatment has any consistent effect in relieving spasm. Personal experience of papaverine, procaine block of the cervical sympathetic, and the use of a general anaesthetic has shown them to be ineffective.

We are therefore left with the third principle—prevention of further haemorrhage—as the only goal of our surgical endeavour ; and if we accept this fact and realize that nothing we can do will modify the effects of the haemorrhage that has already occurred, then two corollaries follow : (1), which has already been emphasized, we must obtain the cases as soon after the initial bleed as possible in order to be able to prevent further haemorrhage, and (2) we will operate in the acute phase (knowing that our surgery is purely prophylactic) only when it is evident that the patient has not been destroyed by the bleed under observation and shows evidence of a reasonable chance of recovery. For this reason surgery is not performed when the patient is in coma (when further operative trauma could only make him worse), but is deferred until there is some sign of sustained improvement in his condition ; this will usually be apparent within a matter of a few hours of the haemorrhage, but may be delayed up to one or at the most two days.

Observation and management of the patient are carried out on the threshold of the operating theatre—a phrase familiar with other types of surgical emergency—and it is during this stage that surgical judgment plays its important part. This management is epitomized in the following case.

A man of 25 had his first (non-coma-producing) subarachnoid haemorrhage 31 days prior to admission to hospital. Arteriography demonstrated a large anterior cerebral aneurysm filling from the right side and projecting upwards. Operation was decided upon, but just before it was performed the patient suddenly became deeply unconscious and remained so for 10 minutes. He regained consciousness, and then became deeply comatose again and stayed in this state, responding to painful stimuli only by slight extension movements of the arms. Craniotomy at this stage, with the vessels in tight spasm and the brain ischaemic, was deferred; the only surgical treatment consisted in making burr-holes under local analgesia to ascertain the presence of an intraventricular clot. Moderately blood-stained C.S.F. was found but no clot. Routine nursing measures were instituted and he was carefully observed. He began to improve, and just 12 hours after the ictus he became accessible, although grossly confused, and a left hemiparesis became less marked. It was now evident that he would probably recover from this particular bleed, and that spasm had relaxed enough to render it reasonably safe to operate to prevent further bleeding.

Surgery was carried out at once; the right anterior cerebral artery was clipped shortly after its origin, and a small clot which happened to lie in the path of the operative approach was evacuated. He made a slow and steady recovery, his confusion clearing up in three weeks, and he returned to his former occupation 10 weeks after the operation.

This case illustrates the surgical principles well. To have operated while the patient was still in coma immediately after the two haemorrhages could only have added to the damage already done by spasm and intracerebral haemorrhage, and would certainly have destroyed him. As soon as the main effect of the intense spasm had passed off and it was evident that the patient was going to recover from the bleed, surgery, to prevent another haemorrhage, was carried out without further delay.

In addition to coma there are two other contraindications to surgery for this particular group of aneurysms. (1) Extreme neurological damage as evidenced by mutism and double hemiplegias. Experience has shown that these patients invariably die from chest infection or bedsores. (2) Anomalies of the anterior part of the circle of Willis, so that both anterior cerebral arteries arise from one internal carotid trunk, with the result that occlusion of the anterior cerebral is not feasible. This complication was present in three patients investigated during the compilation of this series of 37 ; they were treated conservatively and have had no further haemorrhage to date.

Operation

The operation that has been used in every one of these cases consists of occlusion by a clip of that anterior cerebral artery responsible, mainly or entirely, for the filling of the aneurysmal sac as judged by arteriography.

The artery is clipped shortly after its origin from the internal carotid trunk by a lateral approach along the lesser wing of the sphenoid bone by opening up the Sylvian fissure, and without any exposure or disturbance of the aneurysm, or of the clot, or of the brain in its vicinity. The occlusion is designed to cut down the " thrust " of the blood pressure, or, perhaps more important, the pulse pressure, on the sac. The opposite anterior cerebral trunk then takes over the supply of both frontal lobes via the anterior communicating artery, and will of course still supply the aneurysm but with less effect, and the fact that the operation does have considerable influence on the tension in the aneurysmal sac is

shown by a number of arteriographic studies, by post-mortem findings, and, more important, by the subsequent course of the patients.

Heubner's artery or other perforating vessels are not often seen arising from the first 5 mm. of the artery. If, however, a branch is seen the clip is placed across the main trunk beyond it so that it is supplied directly from its own side and not indirectly by reflux from the opposite side. Any disturbance of the ischaemic brain tissue anterior to the chiasm is rigorously avoided. The operation is often difficult with a swollen soft brain, but tapping of the ventricle and release of C.S.F. will usually reduce the tension enough to open the dura. In some of the early cases continuous lumbar drainage of the C.S.F. was carried out by means of a flexible lumbar-puncture needle. Hypotensive drugs were not employed as a routine, although they were always available in case any haemorrhage accidentally started from the aneurysm before the artery was clipped.

Arteriography

As the essential preliminary investigation arteriography is carried out as soon after the bleed as possible. It is usually performed under local analgesia, but if the patient is at all uncooperative a general anaesthetic is administered in order to make certain of obtaining all the necessary information at the first arteriographic attempt. The period of delay while waiting to perform a second arteriogram may well witness a further and fatal haemorrhage. Bilateral studies are carried out to show other vascular anomalies or multiple aneurysms (which occurred in two cases in this series), the presence of clots, and the cross-flow, which is demonstrated by injecting one side and compressing the other carotid manually, during the injection and for 20–30 seconds beforehand. Antero-posterior, lateral, and oblique views give all the information necessary.

The two important points to demonstrate are (1) by which anterior cerebral artery the aneurysm is fed, and (2) the extent of the cross-flow. This may vary from a profuse flow far out to the middle cerebral artery to filling merely of the opposite anterior cerebral (this latter having already been shown to fill from its own side); but this filling is the minimum that can be accepted, and the artery is never clipped unless it can be demonstrated. The aneurysms differed considerably in shape and size, being round, oval, elongated, or, after a bleed, often irregular, varying in this series from 3 mm. in diameter to 17 by 9 mm. Some were bilocular, some were sessile, and some had necks. They projected in various ways—upwards and backwards, upwards, upwards and forwards, forwards, downwards and forwards, and downwards (Fig. 4).

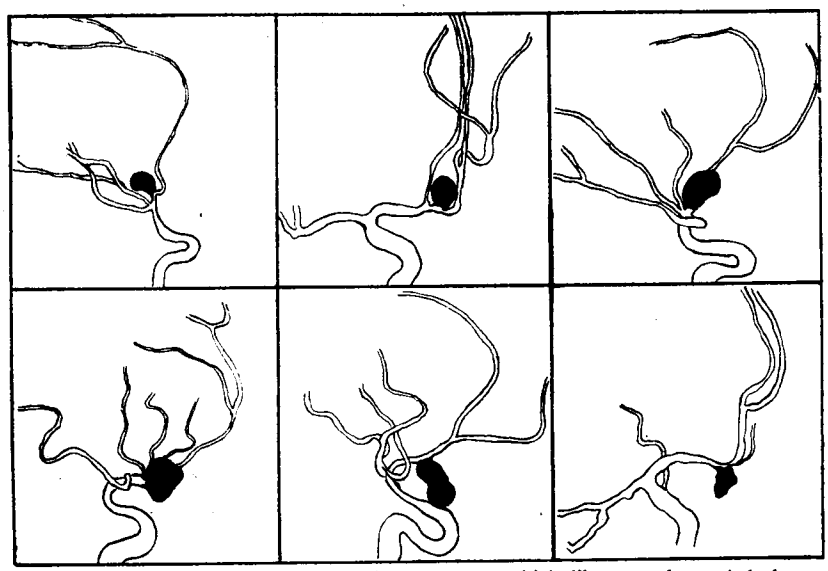

FIG. 4.—Tracings of arteriograms of six aneurysms which illustrate the varied shapes and sizes and the different directions in which they may project.

In 20 patients the sac filled from the left anterior cerebral artery and in 14 from the right; in only 3 did the aneurysm fill spontaneously from both sides, but even in these it filled better from one side than the other, and it was not difficult to decide which was the artery to clip.

Post-operative Arteriographic Findings.—Post-operative arteriograms were carried out on all except two of the surviving patients as a routine measure some two weeks after the operation. The usual appearances were these: the anterior cerebral artery on the side of the operation stopped

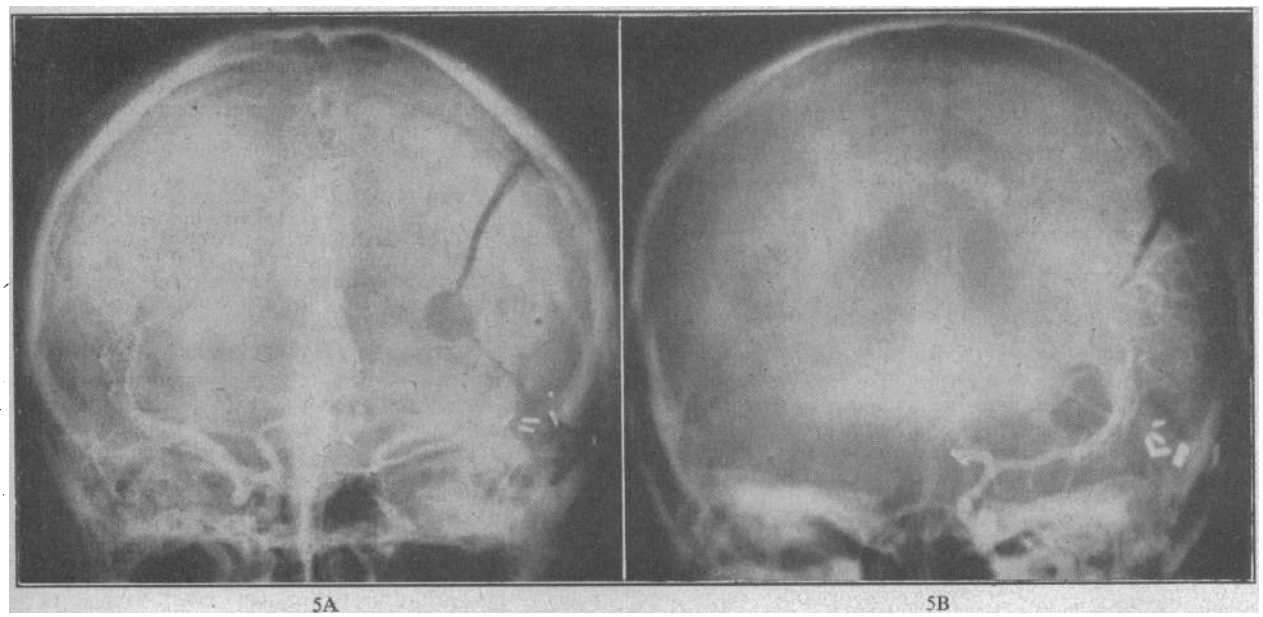

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FIG. 5.—Post-operative arteriographic appearances. The left anterior cerebral artery stops at the clip. Both distal anterior cerebral arteries fill from the right side and the oval aneurysmal sac can be faintly seen near the midline. (There is a clot in the right frontal lobe displacing the vessels across the midline.)

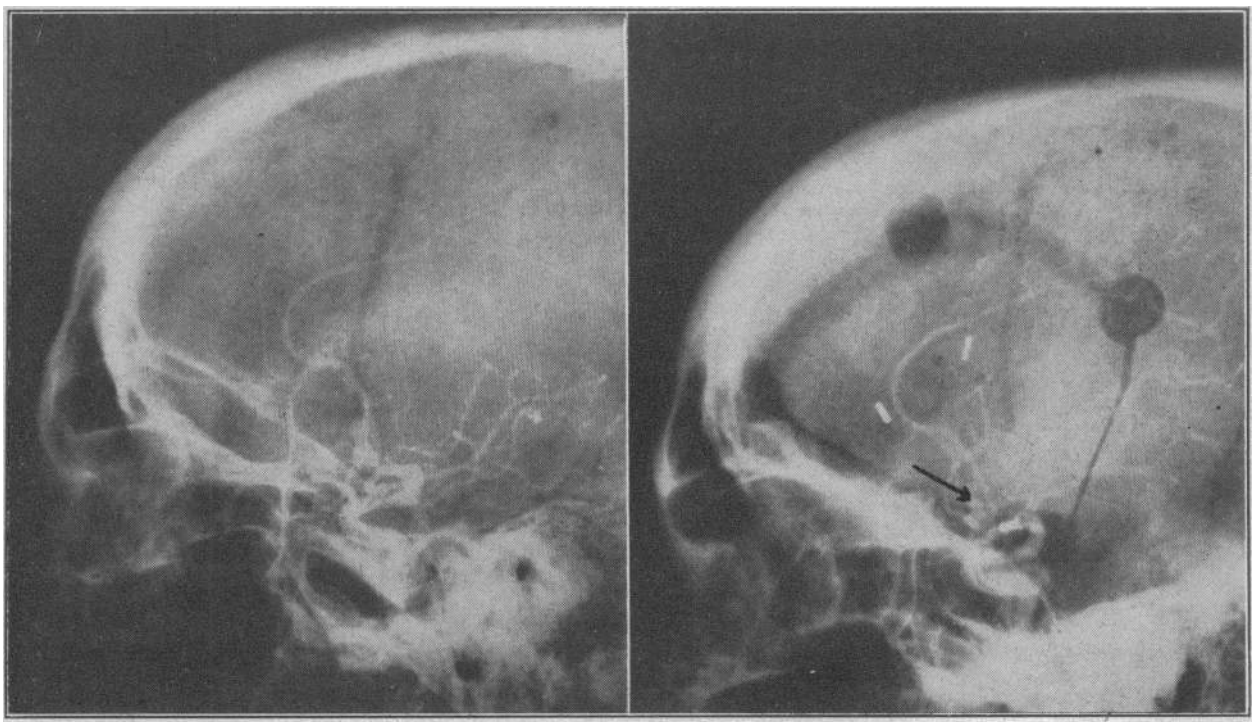

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FIG. 6.—Post-operative arteriogram on the right shows the reduction in size of the bilocular aneurysm (marked with an arrow) compared with the pre-operative appearances.

at or a few millimetres short of the clip; both anterior cerebrals filled from the opposite side (Fig. 5); and the spasm had disappeared. The aneurysm in some cases no longer filled, but it could usually be seen. It was often smaller (Fig. 6) or filled less densely or had a lag of dye indicating that the circulation through it had slowed up. In some cases it was unchanged.

There were two technical faults in these 37 operations, both demonstrated by the post-operative arteriograms. In one the clip on the anterior cerebral had not been squeezed tight and some blood was getting beyond it, and in the other the clip had been applied not to the anterior cerebral but to a very large fronto-polar artery given off more proximal than usual. In both cases the anterior cerebral was subsequently firmly clipped and was so demonstrated by a further arteriogram.

Results

A series of 37 consecutive operations were carried out over a period of four and a half years, from June, 1950, to January, 1955, the average length of follow-up being 22 months (to July, 1955). These 37 patients, aged from 23 to 62, are compared in Table II with the control group. A

TABLE II

	Control Group	Operation Group
Average age	45·6 years	43·9 years
Total No. of haemorrhages	67	78
Total No. coma-producing	45 (67·2%)	42 (54%)
Severe neurological damage	17 cases	17 cases
No neurological damage	11 cases	10 cases

cause for comment is the large number of recurrent haemorrhages in both groups, higher in fact in the operation series owing to two patients who had seven and five haemorrhages respectively prior to operation.

Among these 37 cases there were 5 operative deaths, a mortality rate of 13.5%, less than one-third of that of the control group (44.4%). These deaths are now briefly reviewed, for other factors are concerned in them apart from the operation.

Case 1.—A woman of 46 had her second haemorrhage while in hospital; it produced deep coma with cessation of respiration. An endotracheal tube was passed and operation carried out under artificial respiration. A large frontal lobe clot was removed and the anterior cerebral artery clipped. The patient's condition was quite unchanged by the intervention; artificial respiration was continued, and the heart stopped 24 hours later. The futility of operating while the patient is in coma is emphasized in this case, although it is an extreme example of a patient destroyed by her haemorrhage.

Case 2.—A man of 32 had a second haemorrhage which produced semi-coma and a paralysis of the right arm and both legs. A subdural clot was removed and the relevant anterior cerebral artery was clipped. The patient was unchanged by the operation and died from chest infection 15 days later.

Case 3.—A woman of 51 had a second haemorrhage which produced mutism with double hemiplegias. A frontal lobe clot was removed and the anterior cerebral artery clipped. Death ensued two days later.

Case 4.—A man of 56 had two series of arteriograms carried out as the first was not entirely successful in demonstrating the degree of cross-flow. He remained drowsy post-operatively and then deteriorated, developed a quadriplegia, and died one week later. At necropsy the anterior cerebral system showed no abnormality apart from the clip, but both internal carotid arteries were almost occluded by clot at the site of the arteriographic punctures and there was an area of embolic infarction in the left parietal region.

Case 5.—A man of 60 had two coma-producing bleeds, one nine days and the second 24 hours before operation, which proceeded uneventfully. He did not come round from the anaesthetic, developed a hemiplegia, then a quadriplegia, and died three weeks later. At necropsy there was some softening in the left caudate head and the tip of the frontal pole.

Time Interval from Haemorrhage to Operation

As emphasized earlier, this time interval is the all-important factor, and is shown in Table III. The feature to be noted is that 17, or nearly half, of the cases were operated upon within one week of a haemorrhage and 31 out of 37 within four weeks. Even so, too many cases were operated upon late owing to procrastination at some stage, many of them in fact being referred for investigation only under the stimulus of a second haemorrhage.

Of the six patients operated upon within twenty-four hours of the haemorrhage, two died, one of them being the patient who was operated upon under coma and artificial respiration. Three further deaths occurred among the cases operated upon in the first week, one of them being the

TABLE III.—*Time Interval from Bleed to Operation in 37 Surgically Treated Cases*

Time of Operation	Total No. of Cases
Within 24 hours of haemorrhage	6
„ 7 days „ „	17
„ 14 „ „ „	19
„ 21 „ „ „	25
„ 28 „ „ „	31
In the 5th week after haemorrhage	2
„ „ 6th „ „ „	2
„ „ 8th „ „ „	1
„ „ 10th „ „ „	1

death resulting from arteriography. Of the remaining 20 cases operated on after the first week, all survived.

Recurrent Haemorrhage

Experience has shown that this operation of clipping of the anterior cerebral artery is reliable in preventing further haemorrhage, but, in common with many other surgical operations, it does not guarantee a cure, and in this series of 32 survivors from the operation there was one recurrent, and fatal, bleed (an incidence of some 3%). This occurred on the eighth post-operative day in a patient who was a known hypertensive of some five years' duration and whose arteriogram, carried out within twenty-four hours of a bleed, revealed no evidence of spasm—an ominous indication that the natural mechanism for control of haemorrhage was in abeyance. There were no haemorrhages in the other cases.

There is one further death to report—that of a man aged 58 who had two bleeds, producing a crippling Korsakoff syndrome. He died suddenly three and a half months after operation, while still in hospital from what was thought to be pulmonary embolism. At necropsy there was no evidence of any recent change in the brain, and it did not seem to be a "cerebral" death. On the other hand, a definite cause for his demise was not found in the other systems.

Operative Morbidity

In three patients a hemiplegia was produced as a result of operation. Two of these had gross neurological damage pre-operatively; the third was quite well except for some degree of mental confusion. The neurological damage seemed to be due to a thrombosis, and occurred respectively on the second, third, and tenth post-operative days. In one of the cases arteriography showed that thrombosis had occurred in the remaining anterior cerebral trunk. So that in 31 cases that survived there were three instances of ischaemic or thrombotic damage after operation, an incidence of about 10%. This is a complication, however, that will have to be accepted with any operation in which we tamper with the lumen of an artery supplying the brain. For instance, ligation of the carotid carries a hemiplegia rate of about 8% as shown by the statistics of Poppen (1951) and Jefferson and Johnson (1952); and, similarly, intracranial ligation of the neck of the aneurysm will not escape the risk, for the intima must inevitably be damaged, and in Norlén and Barnum's (1953) fine series there was one late hemiplegia in 14 cases, an incidence of 7%.

What sort of people, then, are the survivors? The majority have sustained two or even more subarachnoid haemorrhages, have had ischaemic areas, often with clots, within the brain, and have been subjected to a major craniotomy in the acute stage of haemorrhage with a redistribution of their cerebral circulation as a result of the clip. It is not possible in the space available to go into detail regarding the intellectual or personality change, or residual symptoms, and for the sake of brevity a single criterion of their well-being has been used—that of their ability to carry out their original work.

Of the 30 survivors to the present time, 22 are doing, full-time, their original work or work requiring comparable skill. Two more are doing, full-time, less skilled work. So that 24 are leading reasonably full working lives. Six are incapable of working, but three cases are still improving and have a good chance of eventually being capable of some sort of unskilled occupation. Three patients have permanent severe intellectual and/or physical disability.

This represents, of course, only one, although perhaps the most important, facet of their recovery. A number of patients still complain of headache and giddiness and other more minor symptoms.

Conclusion

Of a group of 36 cases conservatively treated 20 survived, of whom 4 (20%) had a severe and persistent neurological disability.

Of a group of 37 cases treated by one particular type of operation 30 survived. There was an operative mortality of 13.5%, a post-operative recurrent haemorrhage rate of 3%, and a morbidity rate of 10%.

It is not, of course, claimed that this particular operation is the final answer to anterior cerebral aneurysms, but as our knowledge stands at the present time it would seem that the operation has a good deal to offer to the patient in the acute phase of aneurysmal haemorrhage, at any rate up to the age of 60.

It is suggested, however, that the principles outlined in this report—those of dealing with aneurysms in separate groups according to location, to ascertain the natural history and mortality of each group, and then of comparing a series treated by one type of operation—will stand a greater chance of adding to our knowledge of the part surgery can play in the treatment than the haphazard and misleading method of reporting that has in general prevailed hitherto.

In conclusion, the comment must be made that any publication concerned with the surgical treatment of ruptured intracranial aneurysms which does not give the time interval between the bleed and the operation can be of no value as a guide to the surgical reduction of the mortality from aneurysmal haemorrhage, whatever its other merits may be.

Summary

Of 73 patients with aneurysms situated on and about the junction of the anterior cerebral and anterior communicating artery, 36 formed a control group conservatively treated, and 37 were subjected to one particular type of operation, that of proximal clipping of the anterior cerebral artery supplying the sac. The control group showed a mortality of 44.4% and the operation group 13.5%.

Other factors influencing the outcome, such as vascular spasm, intracerebral clots, and the time interval from bleed to operation, are discussed.

ADDENDUM.—Since the acceptance of this article for publication a further death has occurred, eight and a half months after operation. This was the result of slow deterioration in a patient who, pre-operatively, showed severe neurological damage which was made worse by operation.

It is a pleasure to record my indebtedness to my neurosurgical colleague, Mr. Wylie McKissock, who made a number of these cases available to me, and to my neurological colleagues at the Maida Vale Hospital and St. George's Hospital who also referred patients. My thanks are also due to Dr. James Bull and Dr. David Sutton and their respective radiological departments for the arteriograms, and to Mr. Prickett and the photographic department of the National Hospital, Queen Square, for the reproductions.

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