THE MANAGEMENT OF RUPTURED INTRACRANIAL ANEURYSM*

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JOHN HUNTER'S INTEREST in aneurysms was not only confined to those of the popliteal artery. I am fortunate in having a copy of one of his case records which describes a patient whom he saw in 1791. This was a unique case of a sixty-eight-year-old woman with bilateral intracranial aneurysms arising from the carotid arteries. There were symptoms and signs of fluctuating distension of these sacs with attacks of headache, diplopia, and dimness of vision. After her death in 1792 autopsy was performed by his brother, William Hunter, in the presence of Dr. Jenner and himself. He describes two large aneurysmal sacs about five-eighths of an inch in diameter, lying on either side of the sella turcica. They have been preserved in the Hunterian collection in the Museum. From his description and from inspection of the specimens, it would seem that they probably arose from the intracranial part of the internal carotid artery within the cavernous sinus.

Intracranial aneurysms occasionally present themselves as expanding intracranial lesions in this way, but this aspect of their natural history is not within the scope of my lecture today.

It was Symonds in 1923 who first drew attention to ruptured intracranial aneurysm as the commonest cause of spontaneous subarachnoid haemorrhage, a clinical syndrome described only a year before by Collier. From that time a vast literature grew up as the condition became increasingly recognized, and it was not long before surgical treatment, designed to prevent recurrence of bleeding from ruptured aneurysm, was being considered. In 1933 Dott reported two successful cases of operative treatment for ruptured intracranial aneurysm ; by direct exposure of the aneurysmal sac and muscle wrapping in one instance and by proximal or Hunterian ligation of the internal carotid artery in the other. Thereafter this field of work was elaborated by Tönnis, McConnell, Jefferson, Dandy and Dott, aided to a great extent by the increased use of cerebral angiography which had been discovered by Moniz in Lisbon in 1927. However, particular credit must go to Dandy who did so much to advance surgical treatment without the aid of angiography. In more recent years Falconer,

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Norlén, Olivecrona, Poppen, Hamby and many others have added to our knowledge of this fascinating field of medicine and surgery.

The frequency of ruptured aneurysm is now well recognized and its importance as a social problem cannot now be lightly disregarded, for the highest incidence lies in the age groups of thirty to fifty-five years, the contributors of our society. The gravity of the effects of aneurysmal rupture, and particularly that of recurrent bleeding, both as regards morbidity and mortality, constitutes a major challenge to the practitioner, physician and neurosurgeon alike.

The logical approach to the management of ruptured intracranial aneurysm is very dependent on a broad knowledge of the natural history and living pathology of the condition. Careful history taking, not only from the patient but also from his relatives, and the correlation of it with the results of clinical examination, cerebral angiograms, and operative findings form the basis of such knowledge.

Pathology

Aneurysmal sacs are largely confined to the circle of Willis itself, although a large group arise from the middle cerebral artery at its first primary branching (Fig. 1). When an aneurysm ruptures there is a sudden extravasation of blood from a breach in the fundus of the sac, into the subarachnoid space or brain or both, depending on the anatomical relationships of the sac and the severity of the haemorrhage. Approximately 50 per cent. rupture at some time or another into the brain substance and occasionally into the ventricular system as well (Hyland, 1950). Observations at operation would suggest that the trend of events is as follows. As the aneurysmal sac enlarges it becomes more closely apposed to overlying brain. The first relatively minor haemorrhage causes local meningeal reaction and softening of the adjacent brain substance. The sac distends further and its wall becomes progressively thinned, often patchily, with the formation of small blebs, loculi or daughter aneurysms. The sac soon ruptures again, this time with greater severity as the extravasated blood disrupts the adherent and softened brain and so forms a considerable intracerebral haematoma. This mode of behaviour is particularly true for aneurysms which arise from the anterior communicating and middle cerebral arteries as they lie in the median and Sylvian fissures. Other aneurysms, like those at the posterior communicating-carotid junction and at the termination of the basilar artery, lie free in the subarachnoid space within the basal cisterns, and therefore tend to bleed directly into the subarachnoid space. However, even then, if extravasation of blood is great, it may be forced through the basal fissures into the brain and sometimes into the cerebral ventricles. Occasionally blood is forced into the subdural space through a tear in the arachnoid, forming a subdural haematoma (Clarke, *et al.*, 1953).

It would appear from the fresh appraisal of many case histories, that a patient's first episode of subarachnoid bleeding is commonly of minor

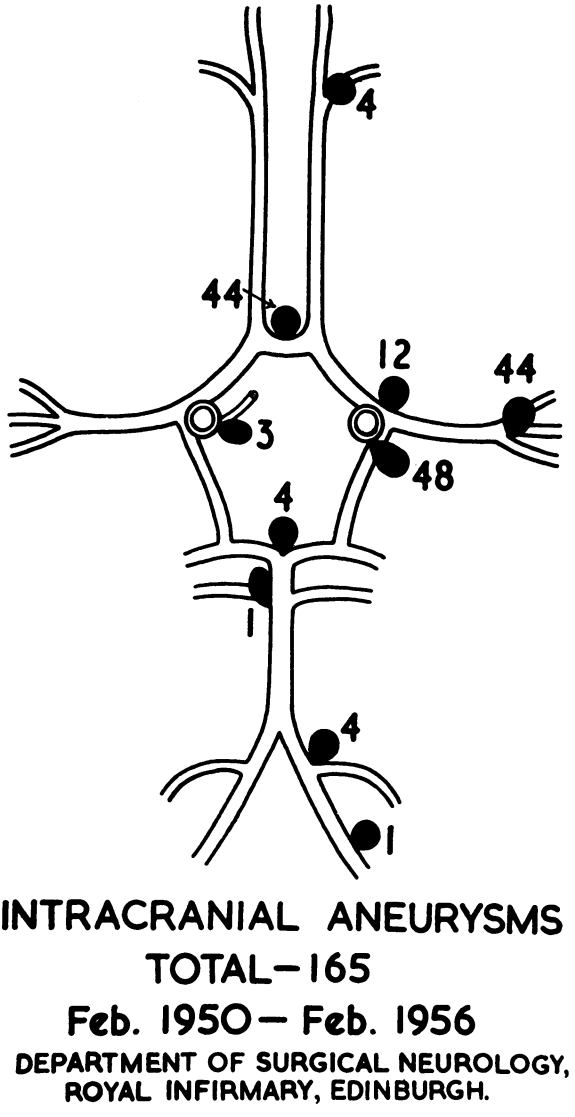


Fig. 1. Diagram of the circle of Willis showing the common situation of ruptured intracranial aneurysms as shown by angiography, operative exploration or autopsy.

severity ; a mere leak of blood from the sac. There is sudden severe pain in the neck which rapidly radiates upwards over the vertex of the head. It is then followed by generalized headache which clears up in a day or two. A diagnosis of fibrositis, influenza or stiff neck may be made by the patient, his relatives or his medical attendant. Fortunately the presence

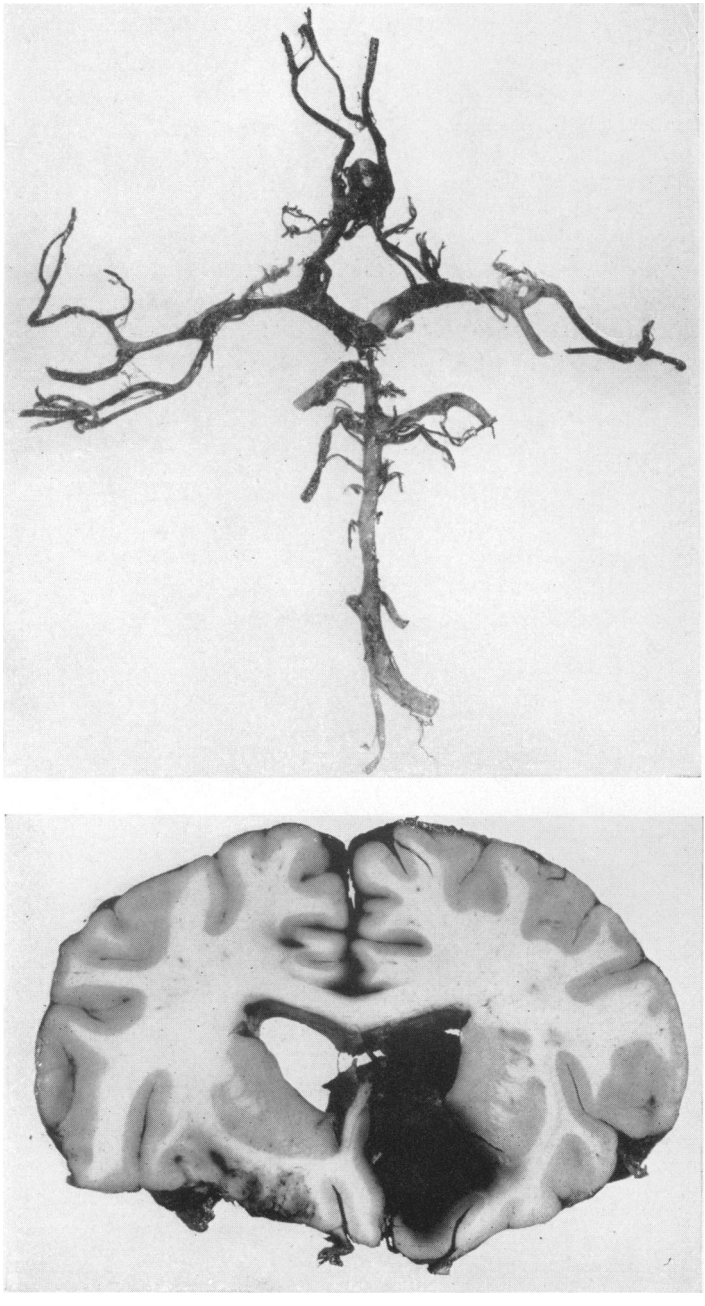


Fig. 2. Circle of Willis and coronal section of the brain showing an anterior communicating aneurysm which has ruptured upwards into the left postero-inferior frontal region, pre-optic region and into the ventricular system.

of neck stiffness, even of minor degree, and other signs of meningism, usually indicate the true diagnosis. The accurate clinical assessment of such an attack is of great importance, in view of the probability and gravity of a second haemorrhage within the next few days ; commonly between the second and the twenty-first day following the first haemorrhage. This second episode of bleeding, as we have seen, is often more severe than the first. The patient is unconscious for a period and is usually left with neurological deficits such as hemiplegia, visual field defects, language difficulties, apathy and negativism, dependent to some extent on the presence of an intracerebral haematoma. However, some patients die within minutes or hours of raised intracranial pressure from a massive intra-cerebral and intra-ventricular haemorrhage, and in a few patients this disastrous episode seems to represent the initial and only attack of bleeding, as opposed to the second or third (Figs 2 and 3).

Extravasation of blood into the basal cisterns and over the cerebral cortex within the subarachnoid space inevitably causes some obstruction of the cerebro-spinal fluid circulation, and this aspect of the living pathology is perhaps not fully appreciated. There follows a degree of distension of the ventricular system and to some extent the basal cisterns as well, for a variable period after the episode of bleeding. Indeed, at operation, during the early days after bleeding, this temporary distension of the ventricles is commonly found and can be usefully exploited. Continuous ventricular drainage during the definition of an aneurysmal sac markedly lowers intracranial tension, and allows good exposure with minimal retraction. Spinal drainage at operation produces this effect more slowly and is less easily controlled. Sometimes this early obstructive hydrocephalus may be so severe that the patient may become stuporose from this cause but is quickly relieved by recognition of the complication and the institution of spinal drainage (Dott, 1956). Later, in a few patients, within weeks of subarachnoid bleeding, there occurs an abnormal meningeal reaction to the presence of blood in the subarachnoid space. This often leads to permanent obstruction from arachnoid adhesions with the formation of a communicating hydrocephalus which demands treatment for its relief. In our experience, these patients have been successfully managed by the diversion of cerebrospinal fluid into the peritoneal cavity (Dott and Gillingham, 1958).

Another important factor besides the extravasation of blood in determining mortality and morbidity in aneurysmal rupture, is the effect of arterial spasm which appears to arise in the region of the neck of the sac at the time of rupture. Its cause is not certain for it is of variable duration and consequently elusive. Whether it arises from extravasation of blood into the arterial wall adjacent to the sac, from the tug on the parent vessel as the aneurysm ruptures (Johnson, 1954), or from the stretching of neighbouring vessels by accumulating clot outside them (Dott, 1953),

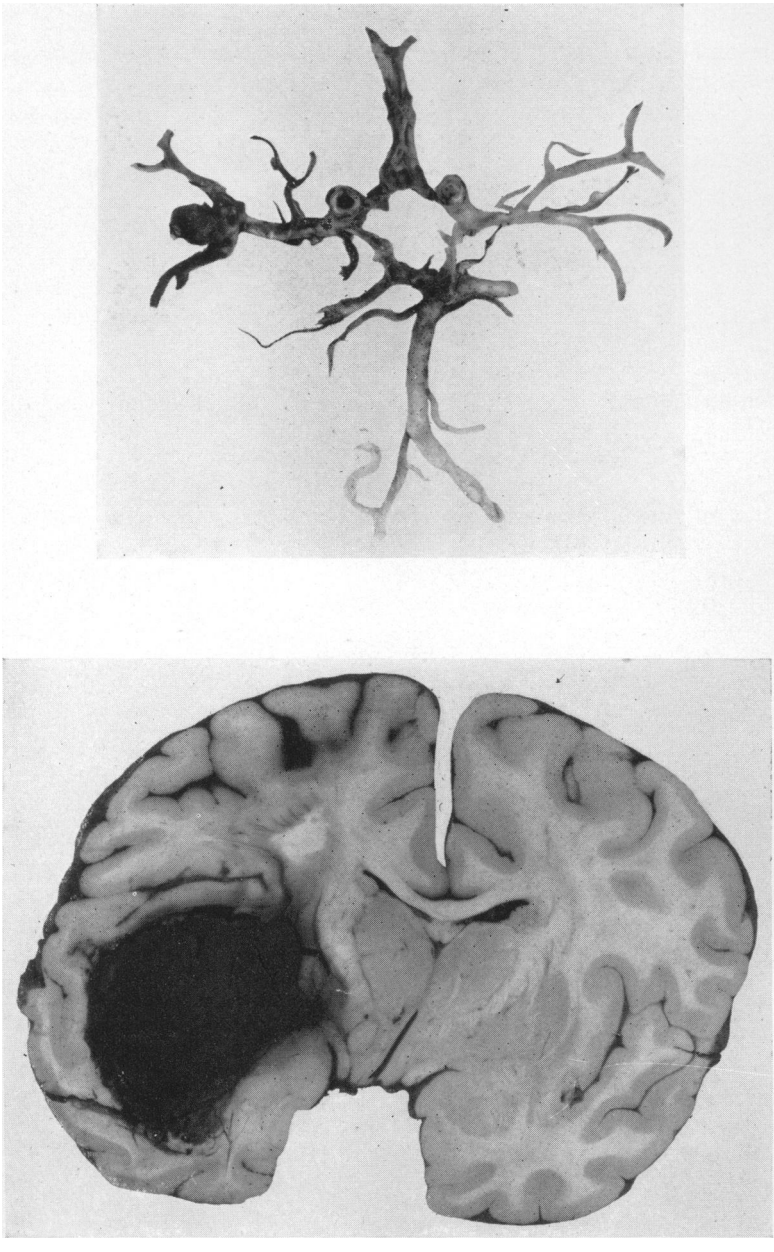


Fig. 3. Circle of Willis and coronal section of the brain showing an aneurysm of the middle cerebral artery which has ruptured downwards into the temporal lobe. Note the gross displacement of the midline cerebral structures and the herniation of the cingulate gyrus beneath the falx.

it is difficult to say. Certainly the degree of spasm would seem not to be related to the volume of extravasated blood outside the sac. In some patients spasm is intense and widespread, spreading proximal as well as distal to the aneurysm ; of long duration, and often quickly re-established by the irritating effects of the medium used in angiography, or by the operative manipulation of the vessel.

Case I

A woman of fifty-seven was admitted with a classical picture of spontaneous subarachnoid haemorrhage, verified by lumbar puncture. When seen six days after the episode, she was drowsy and showed a slight right hemiparesis, maximal in the face. Left carotid angiography failed to show a suspected middle cerebral aneurysm, and there was no spasm or significant displacement of vessels (Fig. 4*a*). She improved slowly and it was planned to repeat the left carotid angiogram in a few days. Unfortunately, four days after the first angiogram there was clinical evidence of further bleeding with a sudden depression in her level of consciousness, an increase of the right hemiparesis, and an increase of neck stiffness. Angiography carried out immediately showed a gross degree of spasm of the left middle cerebral artery, and a definite aneurysmal sac at the first primary branching. Spasm was most marked at the neck of the sac and extended widely into the peripheral branches and into the left anterior cerebral artery. Following angiography this patient deteriorated further. She became comatose and died six days later as a result of raised intracranial pressure from ischaemic swelling of the left cerebral hemisphere which arose from infarction following thrombosis of the fronto-temporal branch of the left middle cerebral artery (Fig. 4*b*, *c* and *d*). Autopsy showed only a minor extravasation of recent blood in the Sylvian fissure in the neighbourhood of the sac and no evidence of discolouration or frank blood elsewhere.

This patient illustrates the existence of spasm and of its probable relationship to rupture of an aneurysm with a minimal leakage of blood. It is possible that angiography further aggravated the spasm which ultimately precipitated the thrombosis of the vessel in the neighbourhood of the sac.

Case II

A young woman of thirty-three suffered a sudden attack of spontaneous subarachnoid haemorrhage, from which she showed apathy and slight drowsiness, but no neurological deficits. Bilateral carotid angiography showed a tiny aneurysm of the anterior communicating artery, associated with a gross degree of spasm of both anterior cerebral arteries in their basal and distal parts, more marked on the right side. Following angiography, which had probably greatly increased the spasm, she remained deeply comatose for several days and akinetic and mute for several months, slowly recovering but with considerable defects of initiative and perseverance. No surgical treatment was undertaken (Fig. 5*a* and *b*).

Case III

A nursing sister of thirty-nine was operated upon a few days following a relatively minor attack of spontaneous subarachnoid haemorrhage, an aneurysm of the right middle cerebral artery being demonstrated by angiography. Moderate spasm of the middle cerebral artery which extended into the anterior cerebral and internal carotid arteries, and into the peripheral branches of the middle cerebral artery was present. This spasm was demonstrated at operation three days after angiography but it was not severe. Just as a blunt hook was passed

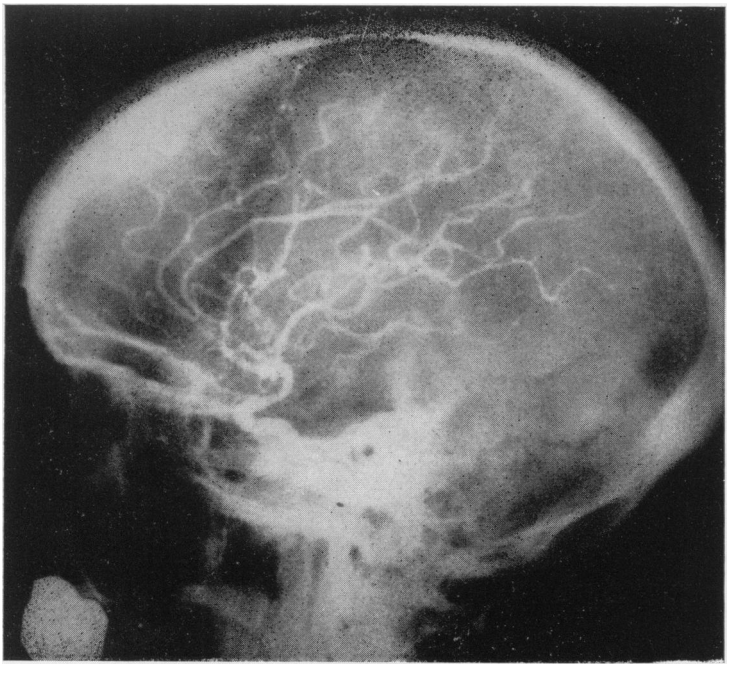


Fig. 4(*a*). Left carotid angiogram—apparently within normal limits.

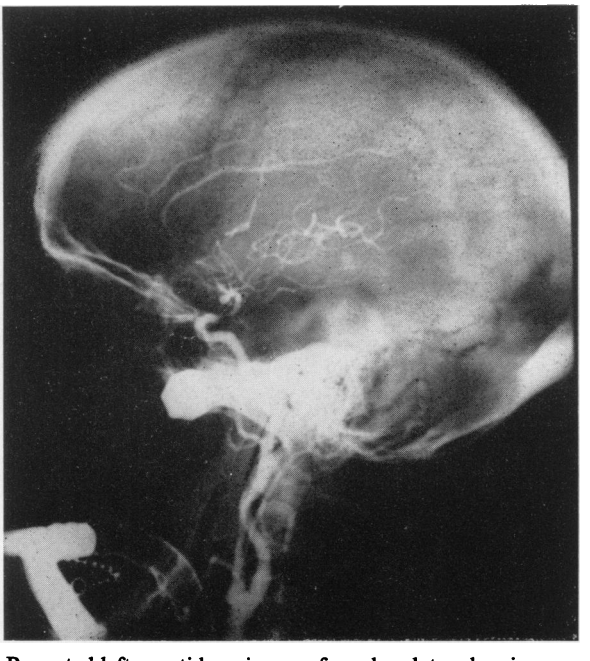


Fig. 4(*b*). Repeated left carotid angiogram four days later showing a severe degree of spasm of the left middle cerebral artery, maximal at the neck of an aneurysmal sac which arises at the first primary branching. Spasm also extends for some distance into the left anterior cerebral artery.

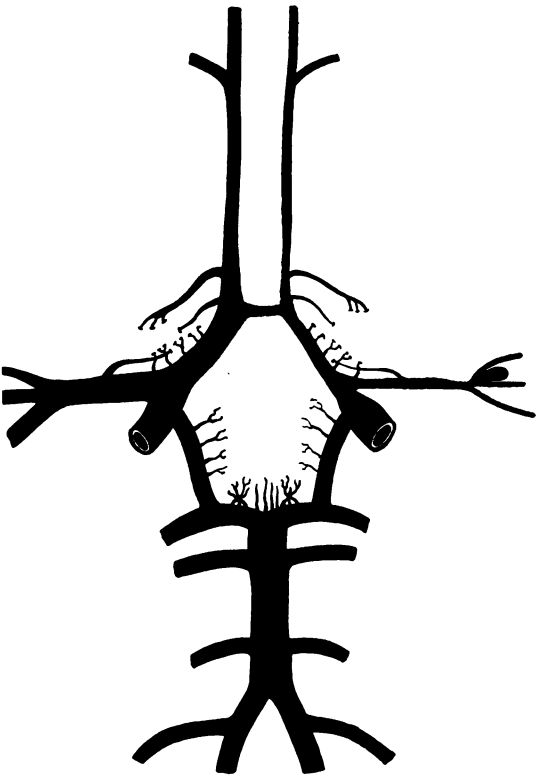


Fig. 4(*c*). Diagrammatic representation of the site of the sac and the associated spasm. Note the striate branches of the middle and anterior cerebral arteries which pass through the anterior perforated substance and which are involved in the spasm.

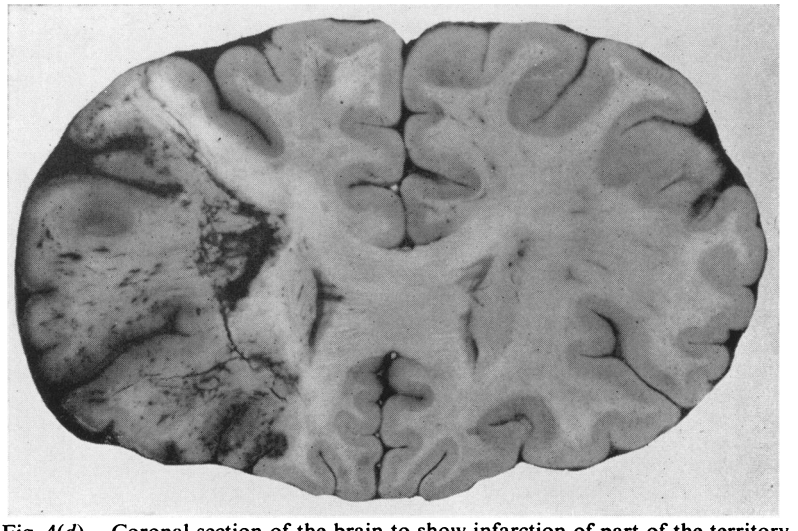


Fig. 4(*d*). Coronal section of the brain to show infarction of part of the territory of supply of the middle cerebral artery.

round the neck of the sac prior to placing a clip across it, the whole middle cerebral trunk and its peripheral branches were involved in a sudden intensification of the spasm, the vessels taking on the appearance of a white thread, and the aneurysm becoming much smaller. The prolonged application of 2·5 per cent. papaverine on linteen swabs to the vessel relieved spasm by approximately 25 per cent., and finally a silver clip was placed across the neck of the sac. Postoperatively this patient was slow to recover. She was comatose and hemiparetic for a few days and then apathetic, although ultimately she returned to part-time nursing, which she managed reasonably well. There was no recurrence of bleeding during a subsequent follow-up period of four years.

This is only one example of several instances of spasm occurring during manipulation of a vessel in the neighbourhood of aneurysmal sac, but since the use of prophylactic locally-applied papaverine to the vessels as soon as they were exposed it has not been seen.

Cerebral arterial spasm associated with aneurysmal rupture is not always widespread and intense, but often minimal and short-lived. In many patients one suspects an individual constitutional factor as a basis for the production of spasm. Its effect would seem to be that of protection —the aneurysm is greatly reduced in size and ceases to bleed, but if spasm is sustained, intense and widespread, it can be fatal or lead to permanent emotional, intellectual and neurological change of considerable severity. In this respect the situation of the midline anterior communicating aneurysm in relationship to the proximal anterior cerebral arteries of both sides, and their striate branches, is particularly vulnerable (Watson and Gillingham, 1953). These branches are concerned with the blood supply of the anterior part of the diencephalon and adjacent structures, areas of the brain concerned with the maintenance of consciousness and autonomic functions (Figs. 4 and 5). This is indeed borne out, as you will see, by the particular difficulties which arise in the management of the anterior communicating aneurysms and in the mortality and morbidity associated with this particular group of patients.

Case IV

A woman of forty-eight was left apathetic and in a Korsakow state following rupture of an anterior communicating aneurysm. Some six weeks after the episode she showed little improvement, and she remained facile and disorientated but without neurological deficit. Clipping of the neck of the sac was performed by the sub-frontal route to prevent a recurrence of bleeding. Operation was uneventful, and presented no difficulty. Postoperatively she was unchanged, but gradually over the next few weeks she slowly deteriorated, showing increased apathy and wasting, and she ultimately died. Autopsy showed no intra-cerebral or subarachnoid clot, and no macroscopic lesion. Satisfactory clipping of the neck of the sac was found to have been achieved without involvement of major or minor vessels. Histological sections of many parts of the brain, including the cerebral cortex, showed no significant abnormality, but there were definite ischaemic changes present in the head of the caudate nucleus and the pre-optic areas of both sides, the territory of supply of the striate or perforating branches of the anterior cerebral artery in its basal part. The absence of clot or macroscopic disruption of the pre-optic area suggested that her clinical state resulted from prolonged spasm of the basal anterior cerebral arteries at the time of aneurysmal rupture with ischaemia of their territories of supply.

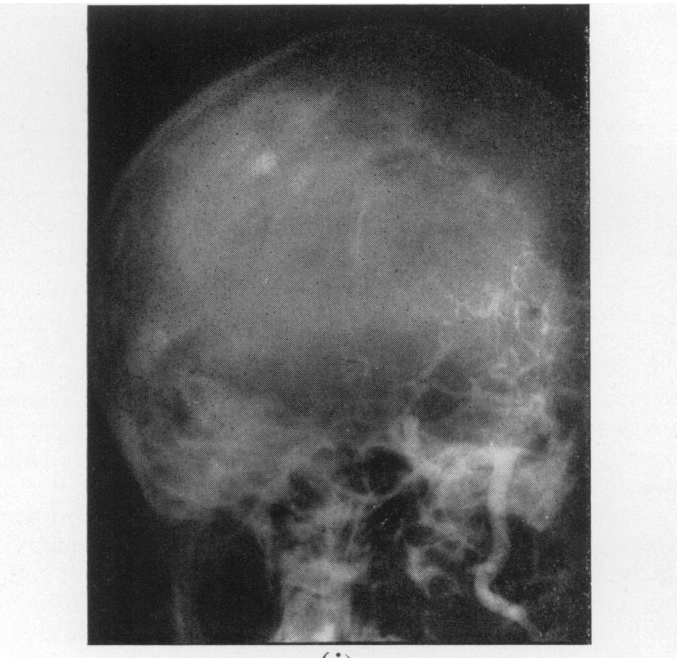
Occasionally spasm involves the distal as well as the basal parts of the anterior cerebral arteries with ischaemia of the corresponding territories of supply and resultant paraparesis of greater or less severity and duration which complicates the picture of spontaneous subarachnoid haemorrhage.

Recurrence of bleeding from an aneurysm is no doubt partly determined by the relaxation of this spasm and partly by imperfect healing of the sac by thrombosis, the greatest danger of such bleeding being between the seventh and fourteenth day after the first. So far we have found no reliable systemic antispasmodic and perhaps it is just as well for the mortality of recurrent bleeding within a few weeks of the first is in the region of 30 per cent. (Walton, 1956). However, we have found at operation that the local application of a 2.5 per cent. solution of papaverine by means of small linteen swabs to the vessel wall is an excellent prophylactic against the re-establishment of spasm during manipulation and does help to relax moderate spasm after application for ten minutes. Severe and widespread spasm does not seem, however, to respond even after prolonged application of up to forty-five minutes (Kinmonth, 1952). Sympathetic blocks in the neck or stellate ganglion block do not appear to relieve this type of spasm. Its relief by local application suggests a myogenic origin. A possible disadvantage of the use of locally applied papaverine is recurrence of spasm after its effect has worn off. We have suspected that such an event has occasionally occurred and in one patient a tiny polythene tube was placed within the Sylvian fissure close to the middle cerebral artery for the daily instillation of the drug. The value of this procedure was indefinite, for the pre-existing hemiparesis was unchanged. Lende of Montreal (1955) has demonstrated this type of recurrent spasm of the cerebral arteries which was initiated in his experiments by the trauma of manipulation. His results suggested that Rogitine was preferable to papaverine for the prolonged relief of spasm.

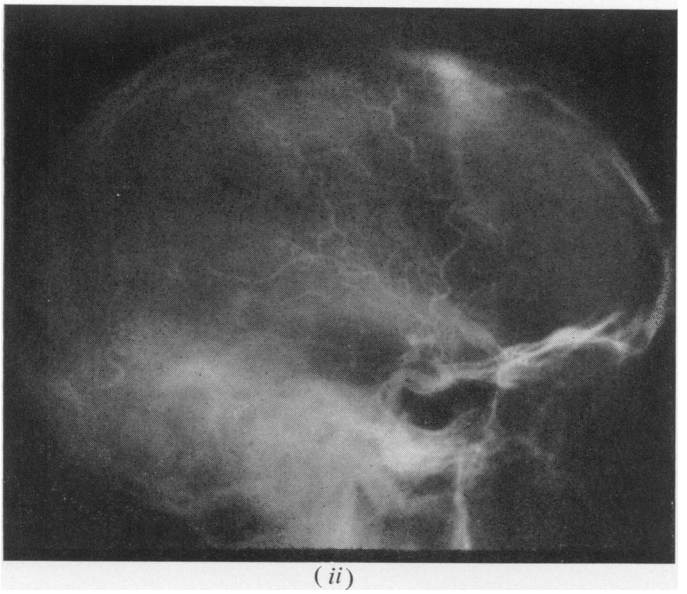
Diagnosis

The diagnosis of spontaneous subarachnoid haemorrhage is by careful clinical assessment and it is confirmed by lumbar puncture. Diffusely blood-stained fluid is obtained with xanthochromic discolouration of the supernatant fluid on centrifuging. There is a danger of confusing the diagnosis by clumsy procedure, blood from traumatised vessels being spilled into the C.S.F. A minor episode may thus be ignored with the possibility of a fatal issue from a further bleeding from ruptured aneurysm in a few days. This investigation should therefore be performed by skilled staff and preferably in hospital.

Ruptured aneurysm as a cause of spontaneous intracranial haemorrhage is the most likely diagnosis in the younger age groups. The catastrophic nature of the episode particularly in association with preceding and often unilateral headache, unconsciousness, meningism and neurological deficits adds confirmation. Other relatively common causes of subarachnoid



(*i*)



(*ii*)

Fig. 5(*a*). (*i*) A.P. and (*ii*) lateral left carotid angiogram showing a gross degree of spasm of the left carotid artery and its branches, maximal at the basal part of the left anterior cerebral artery. A small aneurysm of the anterior communicating artery is poorly outlined.

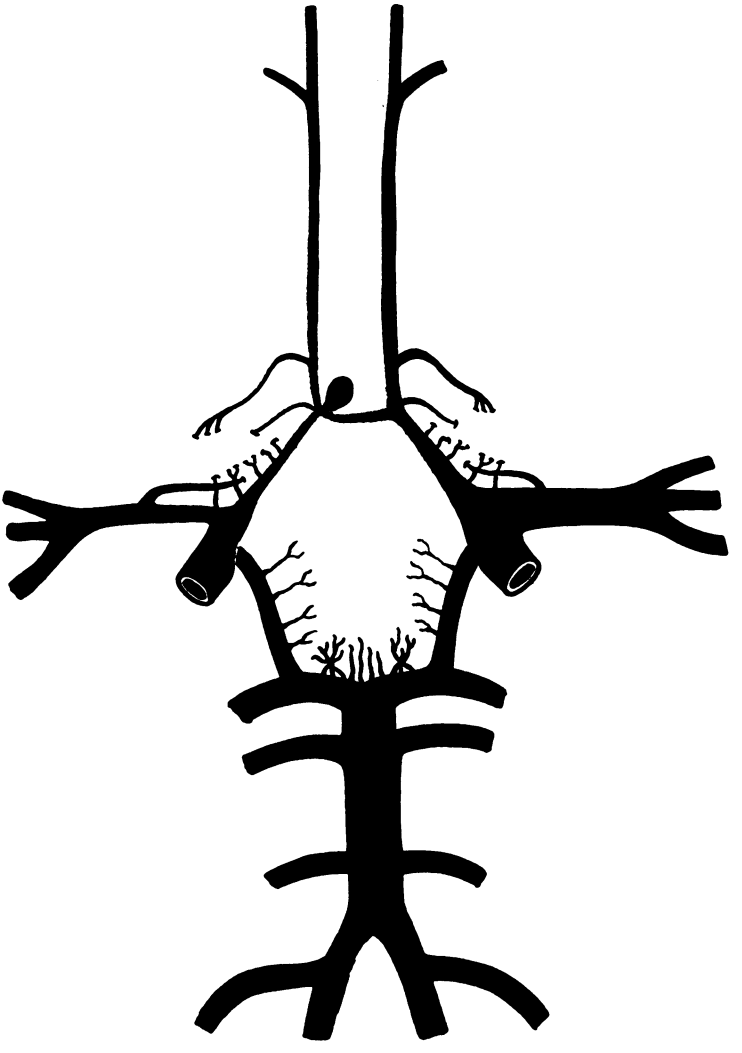


Fig. 5(*b*). Diagrammatic representation of the spasm and how it may extend bilaterally as well as into the ipsilateral middle cerebral and internal carotid arteries. Note how the striate branches of the anterior cerebral arteries are involved by the spasm on both sides.

haemorrhage are bleeding from an arteriovenous malformation and unsuspected intracranial tumour (Gillingham, 1952).

Plain X-ray of the skull is usually unhelpful. Aneurysmal sacs which calcify seldom rupture. The presence of enlarged diploic or meningeal channels are suggestive of arteriovenous malformation.

Confirmatory diagnosis of intracranial aneurysm is entirely dependent on cerebral angiography. This method of investigation was first developed by Moniz in Lisbon in 1925, and in Great Britain in 1929 by Dott and Jefferson. Rapid advances in percutaneous techniques followed in Scandinavia after the war, but mainly in the field of carotid angiography. Outlining of the vertebral system of vessels was less certain until the lateral percutaneous method was developed by Maslowski in Edinburgh in 1951 (Maslowski, 1955) (Fig. 6). Stereoscopy in addition has made angiography accurate for the precise definition of aneurysmal lesions and their connections.

Between March 1950 and December 1954, 228 patients were investigated for spontaneous intracranial haemorrhage, the majority of whom had bilateral carotid and vertebral angiograms if no lesion was found at first. Of these 120 were found to have intracranial aneurysms, twenty-five had arteriovenous malformations, and four had unsuspected tumours.

In spite of rapid progress in the development of angiography there is reason to believe that further advances are necessary in radiographic techniques, particularly in the outlining of the anterior communicating region. Of the 120 patients with aneurysms, five showed no lesion in their first angiograms. Each bled again within a few days or a few weeks. Four died of rupture of anterior communicating aneurysms, and one of rupture of a middle cerebral aneurysm. An aneurysm may not fill because of spasm or because of clot in the sac which later becomes excavated. When spasm is present and no sac apparent we have learned to repeat the investigation within a few days when the patient's clinical condition has improved, for often as spasm decreases the aneurysm can be demonstrated. Oblique films have shown an otherwise obscure sac in routine A.P. and lateral views.

Of these 228 patients, seventy-four (32.5 per cent.) with spontaneous intracranial haemorrhage failed to show any abnormality with careful angiographic studies. Whether they had minute aneurysms that were not outlined or whether thrombosis had occurred within the sac it is difficult to say. Nevertheless the prognosis in this group would seem to be very favourable. However, the number of negative angiograms has become somewhat less as investigation has been undertaken earlier and with greater accuracy.

In general, angiography does not aggravate the condition of the patient except where spasm has been severe and in these cases it has to be used with caution. Dangers lie in re-establishing spasm by the irritant effect of the dye, and in the production of marked vascular hypotension, as anaesthesia is induced (general anaesthesia was used in all cases). Severe and prolonged hypotension adds to the already existing ischaemic lesions of the diencephalon or other territories of supply of the involved vessels (Brown, 1955). Urographin 50 per cent. would appear to be more satisfactory than Uridone in this respect and at the same time provides improved definition (Donaldson, 1957).

If spasm is marked on the first film the procedure is abandoned until the clinical condition of the patient improves to suggest relaxation of spasm. A further cautious angiogram is then done and if spasm is no longer present, an attempt at full definition of the sac is undertaken.

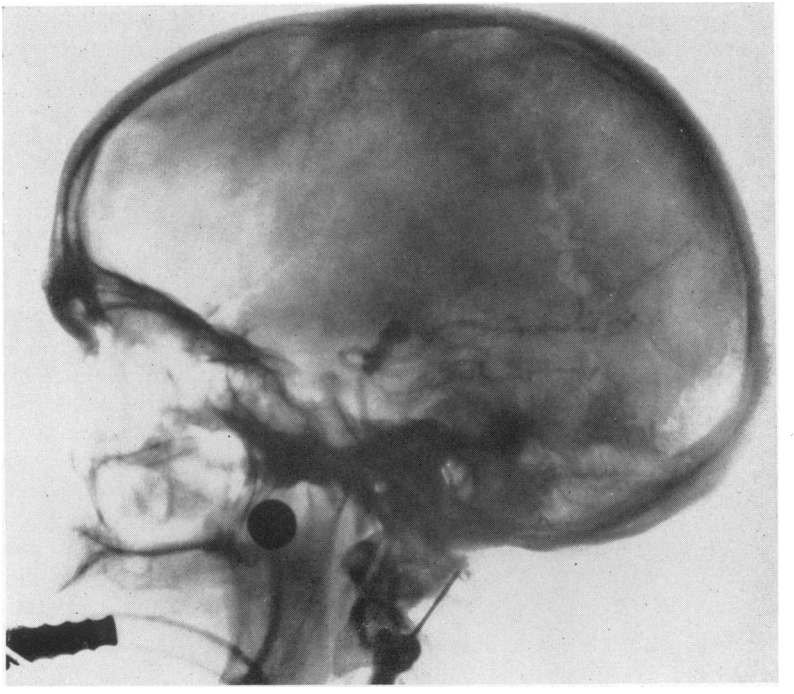


Fig. 6. Percutaneous vertebral angiography by the lateral route (Maslowski) showing a large bilocular aneurysm at the termination of the basilar artery which was approached by a right sub-temporal route. The aneurysm was wrapped in fascia.

Treatment

From pathological features we have considered, the logical approach to the problem of aneurysmal rupture is to prevent recurrent bleeding as soon as possible after the aneurysm has been localized and its connections defined by angiography ; if possible within the first week of bleeding. Nevertheless, experience has shown that the earliest intervention presents difficulties and dangers.

Direct exposure of the aneurysmal sac with a view to occlusion of its neck is the ideal surgical treatment, but there are certain hazards. Friable swollen brain which follows recurrent and severe episodes of bleeding from the sac tends to make the approach to the aneurysm traumatising. The sac itself, whilst being freed, may rupture with the attendant dangers

of an obscured operative field, accidental occlusion of important vessels, spasm, and severe reduction of blood flow to the territory of supply of the artery involved. Severe and prolonged bleeding from the aneurysm may further exsanguinate these territories of supply. Existing ischaemic lesions are very likely therefore to be accentuated and especially if planned hypotension and prolonged retraction are used. Fortunately these difficulties present a major problem only in the midline anterior communicating aneurysm which is situated so deeply beneath the frontal lobes above the sella turcica. Prolonged retraction of the frontal lobe necessarily causes traction on the important perforating striate branches of the anterior and middle cerebral arteries with the danger of spasm and further reduction of their blood flow (Figs. 5 and 6). Temporary contralateral hemiparesis and a slow return of consciousness over some hours in these patients after operation is probably dependent on this factor. Indeed, we have had the opportunity to demonstrate this complication of deep frontal lobe retraction in the open operation of coagulation of the Globus Pallidus for Parkinsonism (Guiot and Brion, 1953). The procedure is ideally carried out under local anaesthesia and as the anterior perforated substance is approached these striate vessels are seen to elongate as the frontal lobe is retracted upwards and backwards. Occasionally frank spasm of the anterior and middle cerebral arteries or their striate branches is seen, which can usually be promptly relieved with locally applied papaverine. There is coincidental raising of the systolic blood pressure and increase of the pulse pressure, lowering of the conscious level of the patient to light stupor and the appearance of a contralateral hemiparesis. The severity and continuance of these changes depends on the duration and extent of retraction. Withdrawal of the retractor almost completely reverses the changes produced within several minutes but they recur with further retraction irrespective of the site of compression by the tip of the retractor (Fig. 7).

The temptation to delay operative treatment until ideal conditions prevail, that is between four to eight weeks after bleeding, when spasm and its effects—hyperaemia and raised intracranial tension have subsided is, of course, great. A low mortality and morbidity can be achieved with operation at such a time (8 per cent. mortality in this series). However, many authors (Walton, 1956) have shown that the mortality of patients with ruptured aneurysms treated expectantly within the first eight weeks of their illness is in the region of 50 per cent., and that a further 20 per cent. will die during the following months or years from a further haemorrhage. About 15 per cent. (Walton, 1956) die within the first twenty-four hours of an initial overwhelming episode and it would seem unlikely that the surgeon will be able to help such patients. However, this figure may not represent the true estimation of the mortality attending the first haemorrhage. We have become increasingly aware of the significance of the first minor attack of subarachnoid bleeding which is so

often missed and only brought out with careful questioning of the patient and his relatives (Gillingham, 1954 ; Symonds, 1956). The second and sometimes severe episode is therefore often regarded as the first. Following such a minor incident—a mere leak of blood into the subarachnoid space —the patient is relatively undisturbed, and operating conditions within a few days of it are nearly ideal. Intracranial pressure is almost normal. The brain is often slack and free from hyperaemia, whilst the walls of the aneurysmal sac are still relatively strong and will tolerate manipulation

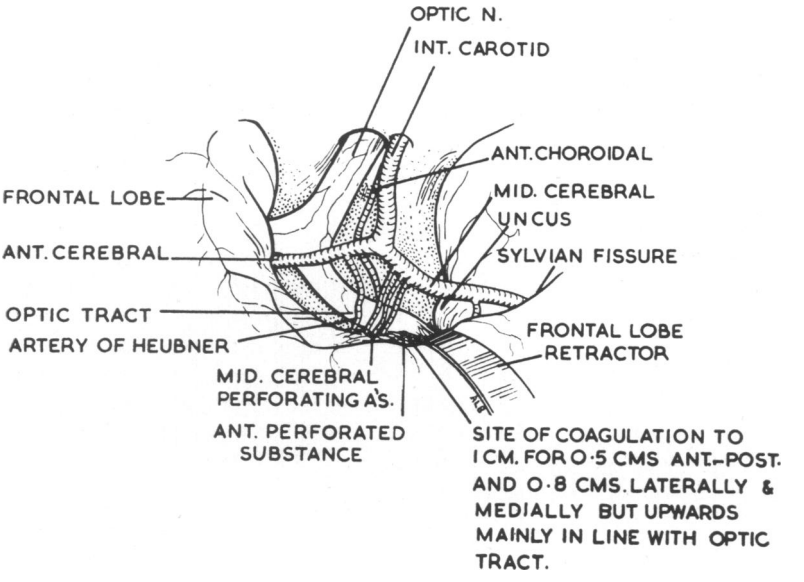


Fig. 7.

more easily. Mortality and morbidity from operative treatment in this group is low, whilst morbidity and mortality from operative treatment in patients with severe neurological deficits and a reduced level of consciousness is high. Another important factor in the true understanding of our problem is that morbidity and mortality of ruptured aneurysm is closely related to their situation on the circle of Willis and its branches.

Aneurysms of the anterior communicating artery

The anterior communicating aneurysm lies beneath the pre-optic region of the hypothalamus and in the midst of its blood supply. The combined effects of extravasation of blood into this area and ischaemia from spasm of the proximal parts of the anterior cerebral arteries and their branches which sometimes accompanies rupture of the sac, leads to disturbance of consciousness, apathy, disorientation, negativism and occasionally catatonia (Figs. 2 and 5). Severe lesions give rise to profound metabolic changes, wasting, stupor, finally coma and death.

Less severe lesions give rise to a state aptly described by Cairns (1941) as akinetic mutism. A direct operative approach in the early days after bleeding with a view to occlusion of the neck of the sac accentuates these lesions and we have therefore abandoned it in stuporose patients. Dott (1953) has advanced the theory that tension within an aneurysmal sac is maintained and its rupture often determined by the force of the arterial jet from the major arterial trunk upon its fundus. Angiographic studies show that aneurysms of the anterior communicating artery are often filled from one anterior cerebral artery and not the other. With the demonstration of an adequate cross-circulation through the anterior communicating artery, clip-occlusion of the appropriate anterior cerebral artery may therefore be carried out as a means of reducing the force of the arterial jet upon the sac. This method of treatment was first satisfactorily explored in 1944 by Dott, and this patient remains alive and well. Our more recent experiences have been equally favourable and coincide with those of Logue and McKissock (1956). This indeed would seem to be the method of choice at present for the early treatment of the anterior communicating aneurysm. The application of the clip to the anterior cerebral artery in most cases requires little retraction, but the site of application is a matter for care in order to avoid damage to the striate branches of the anterior cerebral artery. Placing the clip close to the aneurysm leaves these important arteries to be fed from the ipsilateral carotid artery, but in so doing the sac may be dislodged and rupture. If the cross-circulation is seen to be adequate on the angiogram it may be wiser to place the clip close to the bifurcation of the carotid artery. This aspect of the problem is under investigation at present (Fig. 8).

The type of clip used is important. I have the experience of a firmly closed Cushing type of silver clip being progressively dislodged after an interval of a month with fatal haemorrhage from the aneurysm because of the lack of corrugation on its inner surfaces. I have also watched three such silver clips placed separately across the body of a sac on the middle cerebral artery move in slow procession with each pulsation towards its fundus. The Olivecrona or similar pattern of clip grips more satisfactorily, being larger, flatter, and corrugated on its inner aspect. Some few patients unfortunately have an inadequate cross-circulation and in these clip-occlusion or ligation of the neck of the sac is the only adequate method of treatment.

As will be seen from the analysis of cases (Fig. 9), twenty-one of the eighty patients treated had anterior communicating aneurysms, many of these arising from the junction of one anterior cerebral artery and the anterior communicating artery. In some, the anterior communicating artery was no more than a " cross-roads " between one anterior cerebral artery and the other, the aneurysm arising at that point. None arose from the basal

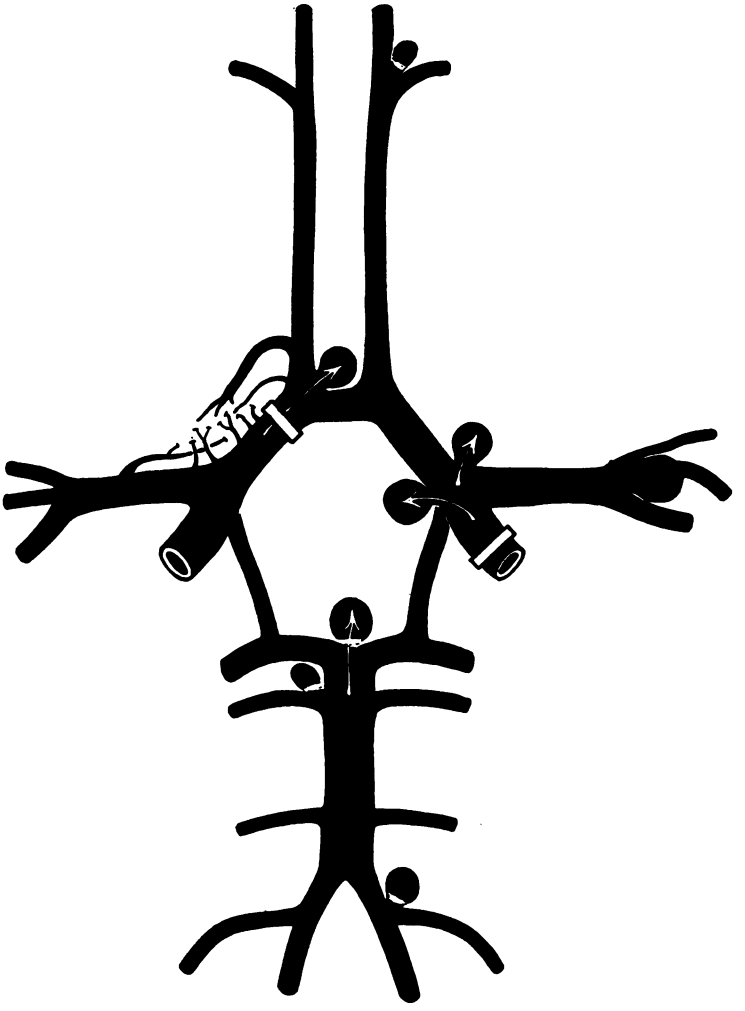


Fig. 8. Diagram of operative treatment of intracranial aneurysms. The clip on the internal carotid artery is purely diagrammatic, representing proximal ligation of the common carotid artery in the neck. Note the broad complex neck which complicates most middle cerebral aneurysms.

part of the anterior cerebral artery itself. Many of this group were operated upon in 1951 and 1952, when methods of treatment were being explored, usually within a day or two of bleeding, without selection and

usually by clip-occlusion of the neck of the sac using an inter-frontal or sub-frontal approach. At that time our comprehension of the living pathology of aneurysmal rupture was still immature. Most of the patients were in coma or stupor before operation. Mortality in this group was very high, but it is from our experience of these patients that we owe the evolution of a greater understanding of the problem. From 1953 this type of aneurysm was treated by clip-occlusion of the appropriate anterior cerebral artery, provided an adequate cross-circulation was demonstrated, with a remarkable lowering of mortality and morbidity. Operation carried out later than three weeks after bleeding resulted in a greatly reduced mortality. In this group of nine patients there were no

OPERATIVE TREATMENT OF RUPTURED INTRACRANIAL ANEURYSMS—TOTAL 80

Site of aneurysm	No. of cases	Cases treated in first three weeks	Alive and well	Severely dis-abled	Dead	Cases treated after three weeks	Alive and well	Severely dis-abled	Dead
*Carotid/ Post. Comm. Antr. Choroidal	18	15	12	2	1	3	2	1	0
Middle Cerebral	26	18	10	7	1	8	4	4	0
Intracnl. Carotid Bifurc.	7	4	1	2	1	3	3	0	0
Anterior Communic.	21	12	4	0	8	9	6	1	2
Anterior Cerebral	3	3	2	0	1	0	0	0	0
Vertebral-Basilar	5	3	0	0	3	2	1	1	0
TOTAL	80	55	29	11	15	25	16	7	2

* Two patients with aneurysms at the carotid/ophthalmic junction included.
Forty-five patients returned to their previous occupations (five slightly disabled).
Follow-up—Ten months to six years. Sixteen of sixty-three alive failed to report progress.

Fig. 9. Analysis of a consecutive series of eighty patients treated between February 1950 and February 1956.

immediate postoperative deaths. The two patients who died subsequently did so because of recurrent bleeding from the aneurysmal sac; in both, proximal occlusion of the appropriate anterior cerebral artery had been performed. In one case, death occurred one month after operation from displacement of the Cushing clip from the artery; in the other, death occurred two years after operation following a sudden attack of un-

consciousness, which was probably related to a recurrent haemorrhage. Unfortunately the patient was not seen and no autopsy was obtained to confirm this supposition. Nevertheless, in our experience, adequate clip-occlusion of the anterior cerebral artery would appear to be the most satisfactory method of treatment of the anterior communicating aneurysm in the early days after bleeding, in the absence of coma and when an adequate cross-circulation has been demonstrated.

Before leaving this important group of aneurysms it is, perhaps, important to observe that recovery from severe central basal lesions of the brain is more probable than was originally believed, provided that the initial hazards of coma, such as impaired airway, are overcome. In this and other respects these lesions are similar to those associated with severe closed head injury (Gillingham, 1952).

Aneurysms of the anterior cerebral artery

These sacs lie at the junction of the pericallosal and supramarginal branches of the anterior cerebral artery at the genu of the corpus callosum and are best approached along the medial aspect of the frontal lobe. Ligation or clip-occlusion of the neck of the sac is the method of treatment, and should be carried out as soon as possible after the first episode of bleeding, taking care to avoid kinking of the peripheral vessels. The otherwise excellent outlook for these patients is occasionally prejudiced by extravasation of blood downwards and backwards in the coronal plane, carrying out what is in fact a more or less posteriorly-placed leucotomy with all the untoward effects of it, namely, stupor, apathy, negativism, or in some cases coma and death (Meyer, 1954). The clinical picture is similar to that of rupture of an anterior communicating aneurysm.

All three cases in this series were treated early. Two are alive and well. One died a week after operation with deepening of preoperative coma, but this patient might have been saved by delaying operation until her level of consciousness had risen even at the risk of a recurrent haemorrhage from the sac. Autopsy showed no significant volume of intracerebral clot, but there was a good deal of destruction of the pre-optic area and the post-frontal region.

Aneurysms of the middle cerebral artery

The common site of origin of these aneurysms is at the first primary branching in the lateral half of the Sylvian fissure. The fundus of the sac is often adherent to the superior aspect of the temporal lobe but occasionally to the inferior aspect of the frontal lobe. Intracerebral clot is a frequent complication of rupture, but it is found more commonly within the temporal lobe than in the frontal lobe. Gentle evacuation of blood from the clot cavity by aspiration before opening the Sylvian fissure will give added access following reduction of intracranial tension

by ventricular drainage. Clot within the Sylvian fissure is often a guide to the aneurysmal sac, but it is a wise precaution to define the main trunk of the middle cerebral artery at the medial end of the Sylvian fissure before exposure of the sac is attempted. The early application of local papaverine to the exposed vessels is an essential step at an early stage to prevent accentuation or re-establishment of spasm. Every care must be taken to avoid dislodgement of the adherent fundus of the sac to prevent rupture. Minor bleeding can usually be controlled by the strategic application of a tiny piece of muscle. As soon as the neck and proximal half of the sac are displayed, a large, flat type of clip of the Olivecrona pattern is placed across the middle of the sac to prevent bleeding as the fundus is dislodged, and the sac and its connections are dissected free. Should major bleeding occur, temporary occlusion of the ipsilateral carotid artery in the neck by finger pressure is carried out by the anaesthetist. Reduction of bleeding then allows definition of the bleeding point by means of skilled suction. Proximal occlusion of the middle cerebral artery is probably unwise in view of the danger of injury to the striate branches of the middle cerebral artery or the intima of the main trunk of the vessel.

Usually the neck of the sac is broad and implicates one or two of the peripheral branches. Ligation or clipping of it often causes constriction of the peripheral branches and may lead to postoperative neurological, emotional or intellectual deficits from ischaemia due to kinking or thrombosis, and this practice has now been abandoned since 1954. Wrapping of the whole sac in sterile muslin gauze and then by a layer of temporal fascia surrounds it with a dense covering of fibrous tissue within a few weeks. The use of this method over the past three years has been entirely satisfatory with minimal postoperative deficit and no recurrence of bleeding. As will be seen from the analysis of cases, only one death occurred in this group of twenty-six patients, eighteen of whom were treated within twenty-one days of bleeding. This fatality was due to recurrent bleeding from a breach in an arteriosclerotic plaque on the middle cerebral artery, the site of the original haemorrhage. An aneurysm of the primary branching lateral to it appeared to be entirely intact and had apparently never ruptured.

In contrast to the group of anterior communicating aneurysms mortality in this group of cases is seen to be very low but morbidity relatively high (Fig. 9). Those severely disabled achieved their limb, visual and occasional language deficits, in the main, preoperatively from major recurrent haemorrhage. Two sustained permanent deficits from persistent spasm, which was induced by manipulation of the neck of the sac at operation, in the days before prophylactic locally-applied papaverine was used. This complication has not been observed since. Four others showed marked deficits following ligation or clipping of the

neck of the sac with kinking or occlusion of one or more of the peripheral branches of the middle cerebral artery.

Aneurysms of the intracranial carotid artery

This group, which includes aneurysms at the carotid bifurcation and at the junction of the posterior communicating and anterior choroidal arteries with the carotid artery, lends itself to two types of operative treatment. A detailed account of these aneurysms and their management in Edinburgh has been published elsewhere (Harris, *et al*, 1957). Proximal ligation of the ipsilateral common and internal carotid arteries in the neck in two stages, is the method of choice, provided that the cross-circulation has proved to be adequate by angiography and by carotid compression tests at the time of operation under local anaesthesia. However, it may well be that common carotid artery ligation is sufficient in many of these cases. In the absence of an adequate cross-circulation, gradual progressive occlusion of the common carotid artery in the neck over several hours or days, using the Dott occlusion clamp, may be achieved with success. Direct approach to aneurysms of the carotid bifurcation in the early days after bleeding, is extremely hazardous and proximal occlusion is much preferable to a direct approach. However, those aneurysms lying at the junction of the posterior communicating or anterior choroidal arteries with the internal carotid artery are less of a problem when a direct approach is required, and are best approached beneath the temporal lobe, through a vertical scalp incision and a relatively small bony decompression. The internal carotid artery is seen to pass directly upwards towards its bifurcation at the edge of the tentorium once the arachnoid is opened. The neck of the sac and the sac itself passes directly backwards and clip application is rendered easy providing retraction is gentle and the aneurysm is not ruptured at an early stage.

The effect of the sacrifice of one major artery to the brain of an ageing circulation has yet to be determined, but proximal ligation has much to offer. It removes many of the hazards of an intracranial operation. Major neurological deficits arising within the first forty-eight hours of carotid ligation are often dependent upon a hypotensive episode, but these are usually of a transient character if treatment is quickly directed towards the raising of the intracranial blood pressure. Ligation in two stages, first of the common carotid artery, and after a few weeks, of the internal carotid artery, lessens this risk. Hemiparesis or hemiplegia occurring towards the end of the first week is probably the result of a spreading thrombosis to the bifurcation of the internal carotid artery, with obstruction of the middle cerebral trunk. Fortunately, his event does not appear to be unduly common. The major contra-indication to carotid ligation in aneurysmal rupture is coma (Schorstein, 1940) as it is indeed to any type of operation for ruptured aneurysm. As a result of carotid occlusion some degree of cerebral ischaemia is always

added and the patient's condition deteriorates further. Expectant treatment is therefore the wise course to adopt, planned operation being carried out when the condition of the patient has improved sufficiently. Early operation may be necessary if clinical evidence and rising cranial pressure indicates the presence of an expanding clot, hydrocephalus or ischaemic swelling, and should be perhaps confined at that stage to the relief of intracranial pressure by decompression, ventricular or lumbar drainage or by evacuation of clot, leaving treatment of the aneurysm itself to a time when the patient's level of consciousness has improved to a satisfactory level.

Twenty-five patients with aneurysms of the carotid artery, have been treated in this series. Eighteen are alive, well and working, five are severely disabled, and two are dead. Of the five disabled patients, one showed considerable deficits from the original haemorrhage, and four developed deficits from operation complications, two from rupture of the aneurysm during operation, and two from occlusion of the carotid vessels in the neck, presumably from spreading thrombosis. One death occurred from rupture of a huge aneurysm at the junction of the carotid artery and its ophthalmic branch towards the end of continuous carotid occlusion over several days. Autopsy showed that the vessel had been virtually occluded completely, and it was felt that a possible explanation as to the cause of rupture following almost complete occlusion of the proximal artery, was that the aneurysm had shrunk down, pulling away from the overlying and supporting brain, thus tearing the friable parts of the wall of the sac. The other death occurred as a result of cerebral swelling from infarction after a difficult operative approach for an aneurysm of the bifurcation of the internal carotid artery.

Aneurysms of the vertebral-basilar system

The problem of operative treatment of these deeply-placed midline aneurysms of the basilar artery lies in their situation, at or below the level of the tentorium. Adequate occlusion of the neck of the sac because of impaired access is extremely difficult. Aneurysms at the termination of the basilar artery are particularly difficult to approach. Adequate access is achieved by means of subtemporal route, following the posterior cerebral artery medially to its junction with the posterior communicating artery. The posterior communicating artery is then divided between silver clips at this junction, and gently pushed aside. The posterior cerebral artery is followed further, medially and posteriorly. Soon another obstruction is encountered, namely, the leash of perforating vessels arising from the posterior cerebral artery, which passes upwards, and often lies between the operator and the aneurysmal sac. Wrapping of the sac with fascia was carried out in two cases, and clipping of the neck of the sac in two others. There was one early fatality (Fig. 9) and in this patient preoperative stupor was severe. The other fatality

occurred two years after operation from further rupture of the aneurysmal sac, the fascial wrapping having not been achieved adequately to cover the upper part of the fundus. During the two years of this patient's life he had been extremely well and had resumed his old occupation for most of the time.

Aneurysms of the vertebral-posterior inferior cerebellar junction would seem to be readily amenable to treatment, but the only one in this series presented at autopsy—the cause of the bleeding was thought to have occurred from a demonstrated carotid-posterior communicating aneurysm which had, however, remained intact, but which was treated by carotid ligation. Further fatal bleeding occurred from the unsuspected vertebral aneurysm several days following treatment for the other sac.

Multiple aneurysms

Dandy (1944) estimated that multiple aneurysms occurred in 15 per cent. of all cases. In our series from 1950 to 1954, it was 8 per cent., but it should be pointed out that if an aneurysm was found at a site which supported the clinical picture, no further angiography was performed. It may well be that the whole cerebral vascular tree should be outlined in each case, for we have the recent experience of bleeding from the second of two aneurysms within a few weeks of adequate treatment of the first. The problem is one of judgment, weighing up the hazards of angiography against the possible incidence of bleeding from an unknown aneurysm elsewhere. Sometimes it is extremely difficult to decide from the clinical picture which of two demonstrated aneurysms has bled. Electroencephalography sometimes helps to localise the site of bleeding. The presence of spasm and particularly of localised spasm on the angiograms is strongly suggestive of the site of aneurysmal rupture. The value of this sign has been confirmed operatively on several occasions. If there is still doubt, the operative approach can sometimes be designed to inspect the two sites. The presence of soft yellow clot in the neighbourhood of a sac strongly indicates that it has been the source of haemorrhage.

DISCUSSION

From these experiences and those of others it is apparent that the management of spontaneous subarachnoid haemorrhage from ruptured aneurysm is dependent on close liaison between the general practitioner, physician and neurosurgeon. The earliest diagnosis and reference of a patient to the neurosurgeon after the first, and usually minor, episode of bleeding, is eminently desirable. Indeed it is a matter of great urgency in the face of the high mortality and morbidity which accompany expectant treatment. Over 30 per cent. die of recurrent bleeding after the first twenty-four hours, and an additional 20 per cent. after the first three weeks have elapsed. When the patient is little disturbed by the attack,

the morbidity and mortality of operative treatment is correspondingly low. Mortality is especially high in the anterior communicating and basilar aneurysms, but the use of clip-occlusion of the proximal part of the appropriate anterior cerebral artery, as opposed to clip-occlusion of the neck of the aneurysmal sac has greatly reduced mortality in the group of anterior communicating aneurysms. Basilar aneurysms still present a problem largely because of difficulties of access.

The overall mortality for this personal series of eighty patients, treated between February 1950 and February 1956, is 21.25 per cent. For the twenty-five patients treated after three weeks bleeding, it is 8 per cent. Operative mortality for the fifty-five patients from all groups treated in the first three weeks bleeding is 27.3 per cent. During the first two and a half years of this period of study, no selection was made of patients for operation, and several were operated on whilst in coma, often as an emergency, after their second or third haemorrhage.

This overall figure of operative mortality of 21.25 per cent. in eighty cases of ruptured aneurysm compares favourably with approximately 50 per cent. which accompanies expectant treatment, and it can be lessened considerably by the earlier reference of patients and early operative treatment following the first episode of bleeding, by better judgment in the timing of angiography and operation, and by the type of operative procedure used.

When patients are admitted in coma or stupor it would seem wiser to delay angiography and operation until their level of consciousness improves to the state of being roused easily, in spite of the risk of further bleeding. If, in these patients, there is, however, evidence of an expanding clot, brain swelling, or communicating hydrocephalus, as shown by rising intracranial pressure, then operative treatment is indicated for the early relief of the condition.

Hypothermia (Botterell *et al,* 1956) was not used during operation for any of these patients. Controlled hypotension was used in a few, in the earlier cases by arteriotomy (Gardner, 1946) and more latterly by Arfonad. If we exclude the group of vertebral-basilar aneurysms, the operative mortality for the other seventy-five cases is 18.7 per cent. If we exclude the group of anterior communicating aneurysms as well, the operative mortality for the remaining fifty-four patients is 7.4 per cent. and hypothermia would seem unlikely to help the further lowering of this figure. It may be that hypothermia will be of special value in the management of the anterior communicating aneurysm, but a prolonged trial and follow-up of clip-occlusion of the appropriate anterior cerebral artery would appear to be profitable.

Since this lecture was given the opportunity has arisen for the further detailed analysis of forty-seven patients with spontaneous subarachnoid haemorrhage who were admitted for investigation from February 1956 to February 1958.

In fifteen, no cause for the haemorrhage was found after careful scrutiny of bilateral carotid and unilateral vertebral angiograms. Fourteen remain well and without significant morbidity but one is recovering from a residual hemiparesis which followed the episode of bleeding. This figure coincides remarkably with that previously obtained, 32.26 per cent. in this series and 32.5 per cent. in the original. In two cases, arteriovenous malformations were found, one in association with an aneurysm of the termination of the basilar artery. No tumours were discovered. Thirty-one patients were found to have aneurysms. Eight had posterior communicating/anterior choroidal-carotid aneurysms. All were treated by proximal ligation and all are alive and show no significant morbidity.

Six had anterior communicating aneurysms. Three were treated by proximal occlusion of the appropriate anterior cerebral artery and three by clip-occlusion of the neck of the sac because of an inadequate cross circulation. Five are alive and show no significant morbidity. There was one death from meningitis which complicated the use of spinal drainage in the early postoperative treatment of a communicating hydrocephalus.

Six had aneurysms of the middle cerebral artery. Five are well and without practical morbidity. One, a woman of sixty-four years, who was stuporous before operation, died on the fifth postoperative day. In this case, the operation was too early and she might have survived had she first regained consciousness more fully.

Seven had multiple aneurysms. Three are alive and well. Four died, one as a result of an ill-timed operation, the patient being stuporose, and one died from brain swelling following thrombosis of the middle cerebral artery, the site of a second aneurysm ; the first, an anterior communicating aneurysm, was treated by occlusion of the neck of the sac, ten days before. His early postoperative state was excellent until he developed an increasing hemiparesis and coma four days later. Two others died as a result of bleeding from known anterior communicating aneurysms a few weeks after operation for a middle cerebral aneurysm. In one case it was felt in retrospect that the middle cerebral aneurysm had never ruptured but in the other, the middle cerebral aneurysm had obviously ruptured first.

Four patients were not treated by operation. One died from recurrent haemorrhage five hours before planned operation and one died in coma a few hours after admission from his second haemorrhage. The two others survived but were treated expectantly. One was arteriosclerotic and the other had an arteriovenous malformation of the posterior fossa and a basilar aneurysm.

The overall mortality for this group of cases, managed by carefully judged expectant and operative treatment and without hypothermia, is 10.9 per cent., a significant figure in the light of the mortality of over 50 per cent. with expectant treatment. The operative mortality is approximately as before, namely, 22.2 per cent., but this disappointing figure is dependent upon a determined approach to the problem of multiple

aneurysms. If we exclude this group, the mortality for the other twenty cases (two deaths), which include anterior communicating, posterior communicating-carotid, and middle cerebral aneurysms, is 10 per cent. Both these two fatalities in retrospect were avoidable, namely, meningitis from spinal drainage, and death from an ill-timed operation, so that ultimately a lower figure can be anticipated in the future.

SUMMARY

This paper is concerned with the living pathology and management of ruptured intracranial aneurysm. It is based upon a personal experience of eighty consecutive operations* for spontaneous intracranial haemorrhage due to ruptured aneurysm from February 1950 to February 1956. A more recent and comprehensive series from February 1956 to February 1958, of forty-seven patients which has accumulated partly since this lecture was given, is briefly discussed.

There is now good evidence to support the view that patients who have suffered from their first, and often minor, episode of subarachnoid bleeding should be referred as a matter of urgency for neurosurgical investigation and treatment.

ACKNOWLEDGMENTS

I am greatly indebted to Professor Norman Dott, my mentor in this field of work. Dr. W. Young and Dr. A. da Rocha Melo gave considerable assistance in the analysis of the cases.

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*Sixteen patients in the earlier part of this series were managed together with Professor N. M. Dott, who carried out the major part of the operative treatment.

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PROVINCIAL MEETING IN CARDIFF
Programme of Events

Friday, 26th September

5.45 p.m.	Reception at Cardiff Castle at the kind invitation of the Lord Mayor and City Council of Cardiff. Tickets on application to the Secretary (limited to 150).
7.30 p.m. for 8.00 p.m.	Dinner for Fellows and Members at the New Continental Restaurant, Queen Street. Tickets (£2 2s. 0d. each, inclusive of wines) on application to the Secretary. A limited number of medical guests may be invited by those attending.

Saturday, 27th September

10.00 a.m. to 12.45 p.m.	Scientific exhibitions, films and operating sessions. at the Cardiff Royal Infirmary. Open to all members of the medical profession.
12.45 p.m.	Fork lunch at the kind invitation of the Board of Governors of the United Cardiff Hospitals, in the Cardiff Royal Infirmary.
2.15 p.m.	Annual Meeting of Fellows and Members in the Reardon-Smith Lecture Theatre, National Museum of Wales.
3.15 p.m.	Admission to the Honorary Fellowship of Sir Russell Brain, Bt.
3.30 p.m. to 4.30 p.m.	" The Cervical Spine "—Lecture by Sir Russell Brain, Bt., D.M., F.R.C.P., Hon. F.R.C.S. Open to all members of the medical profession.

NOTE.—Fast trains from Cardiff to London on Saturdays leave at 5.00 p.m. and 7.00 p.m.

APPOINTMENT OF FELLOWS AND MEMBERS TO CONSULTANT POSTS

F. E. CLYNICK, M.B., CH.B., F.F.A.R.C.S., Consultant Anaesthetist to St. Peter's, St. Paul's and St. Philip's hospitals.

The Editor is always glad to receive details of new appointments obtained by Fellows and Members, either through the Hospital Board or direct.