Significant medical advancements usually are based on preliminary observations and experiments made by various investigators over a period of many years. Frequently, though, the final steps in the process are not made until the introduction of a key idea or technique. An example of this is the development of the surgical treatment of intracranial arterial aneurysms. Procedures for the treatment of other arterial aneurysms were known for centuries, but because aneurysms of the intracranial arteries were hidden within the skull, these lesions were rarely diagnosed before the introduction of carotid arteriography in 1927. Since then, intracranial aneurysms have been diagnosed frequently, and a variety of operative procedures have been devised for their treatment.

Basic principles in the treatment of arterial aneurysms have been advanced since the time of the ancients. At first, attempts were made to empty or excise the aneurysmal sac after ligation of the artery, but during the Middle Ages conservative therapy by compression became popular. With the introduction of the tourniquet in 1674, treatment by arterial ligation again came into vogue.\textsuperscript{28,29} Arterial ligation for hemostasis was also practiced early in the history of surgery.\textsuperscript{19} Exactly when the common carotid artery was first ligated is not clear because of the incomplete medical records of those times. This procedure has been credited to Ambrose Paré and to several surgeons of the late eighteenth and early nineteenth centuries.\textsuperscript{18,22,33}

On November 1, 1805, Astley Cooper performed the first ligation of the common carotid artery for an aneurysm of that artery.\textsuperscript{5} Hemiplegia developed on the eighth postoperative day and the patient died on the twenty-first day. Cooper's second operation of this type, on June 22, 1808, was successful.\textsuperscript{5,7,22,35}

After it had been shown that the common carotid artery could be ligated for hemorrhage or aneurysm, the operation was applied injudiciously in the treatment of a variety of other conditions: epilepsy, exophthalmos, tumors of the head and neck, pain in the head, psychosis, etc.\textsuperscript{4,33} The resulting operative mortality rates were so high that surgeons were stimulated to invent devices for the gradual occlusion of the artery.\textsuperscript{33} George W. Crile\textsuperscript{8} introduced a spring-end screw clamp, and William S. Halsted\textsuperscript{16} recommended aluminum bands for this purpose. Rudolph Matas also emphasized the importance of testing and increasing the collateral circulation by the temporary occlusion of the carotid artery before its ligation.\textsuperscript{22,24,26,33,34}

The history of man's experience with intracranial aneurysms, like his experience with carotid ligation, can also be traced back at least to the eighteenth century. Although it is difficult to be certain about previous observations, these lesions were definitely encountered at postmortem examination by Francisci Biumi in 1763 and John Hunter in 1792.\textsuperscript{3} Surprisingly, aneurysms were not described by Fallopius, Casserius, Vesling, Wepfer, or Willis, whose studies during the sixteenth and seventeenth centuries established the anatomy of the circle of Willis.\textsuperscript{3,14,20,27,30} By 1851, about 40 well authenticated cases of cerebral aneurysm had been reported, and in 1861 Jonathan Hutchinson became the first to clinically diagnose a saccular, nontraumatic intracranial aneurysm.\textsuperscript{3,21,22}

Despite many later retrospective studies of the signs and symptoms associated with intracranial aneurysms, they were rarely diagnosed during life until the advent of
carotid arteriography. Surgeons therefore had few encounters with these lesions.

The following four operations are illustrative of the few attempts at surgical correction of intracranial aneurysms prior to 1933. At the turn of the present century, Victor Horsley accidentally found an aneurysm in the middle cranial fossa arising from the right internal carotid artery, and successfully treated it by ligating the common carotid artery in the neck. In 1924, Wilfred Trotter performed the first planned operation for an intracranial aneurysm diagnosed preoperatively when he ligated the right internal and external carotid arteries for a traumatic aneurysm of the intracranial portion of the internal carotid artery. Four years later a saccular aneurysm of the left middle cerebral artery in another patient was correctly diagnosed preoperatively, and treated by Walter Dandy by partial occlusion of the internal carotid artery in the neck. Finally, in 1931 the first planned intracranial operation for aneurysm was performed when Norman Dott wrapped muscle around an aneurysm at the bifurcation of the left anterior and middle cerebral arteries.

Then in 1933 Dott became the first to operate on an aneurysm previously demonstrated by carotid arteriography. With the use of this diagnostic technique, a new era in the treatment of intracranial aneurysms was initiated. Many of the modern intracranial procedures for the correction of these aneurysms were developed during the following five years.

"The first intracranial operation involving the actual excision of an aneurysm was reported by German to the Harvey Cushing Society in 1881. German excised a posterior cerebral aneurysm with recovery."

The difficulty in ascribing credit to the first person to do a particular procedure is illustrated by the discussion which followed German's report to the Harvey Cushing Society. McKenzie mentioned excising an aneurysm; Wilkins described opening an aneurysm by error; Poppen stated that he preferred to surround an aneurysm with strips of muscle; Lyerly described a case in which he ligated the artery and removed the sac; and Fay related removing an aneurysm under the impression that it was a meningioma.

Two other significant procedures for the treatment of intracranial aneurysms also were developed during those few years. Both can be attributed to Walter Dandy. In 1936 he began to treat aneurysms of the internal carotid artery in or near the cavernous sinus by ligating this artery in the neck and intracranially, thus trapping the aneurysm between the proximal and distal points of ligation. Two years later Dandy also introduced the important technique of directly occluding the neck of a saccular aneurysm with a silver clip.

The publications describing these two procedures mark the end of the period when several new techniques were rapidly introduced for the treatment of intracranial aneurysms. They also mark the beginning of the present comparative evaluation of these techniques in large numbers of patients.

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**INTRACRANIAL ANEURYSMS:**

**CEREBRAL ARTERIO-RADIOGRAPHY: SURGICAL TREATMENT.**

*By Norman M. Dott, F.R.C.S. Ed.*

The subject of aneurysms of the basal cerebral arteries has a long and sustained association with Edinburgh Medicine, with this Society, and with the *Edinburgh Medical Journal*. In 1886 the late Sir Byrom Bramwell gave the first clear description of the clinical features of spontaneous subarachnoid hemorrhage and its association with rupture of an aneurysm of one of the basal cerebral arteries. He gave this clinical syndrome a place of its own among the various forms of apoplexy or strokes. He made important further contributions to the subject during the next decade. In his earlier years in Edinburgh, from 1911 onwards, Professor Drennan was collecting a series of cases, to which he added later in New Zealand, and produced his important contribution to the clinical and pathological aspects of the subject in 1921. In 1931, the next important clinical advance was made by Professor Edwin Bramwell’s paper in which he defines the association of basal cerebral aneurysms with recurrent oculomotor paralysis. Thus the subject has been carried forward in Edinburgh, and I have now the honour to bring before you some further observations, more especially on accurate diagnostic definition by means of arterial radiography, and on some experiences and suggestions as to practical surgical treatment. I have a personal experience of seventeen cases of aneurysms of the basal cerebral arteries, and shall proceed to mention some of them which illustrate matter of interest.

**Case I.**—An apparently healthy man (No. 324) of 36 developed quite suddenly, in 1920, an almost complete left oculomotor paralysis. Its sudden onset was accompanied by a sharp pain behind the left eye and some left frontal headache persisted for a few days. In the course of a few weeks the ptosis and external strabismus had gone, but some enlargement of the left pupil remained permanently. He remained perfectly well until ten years later when he had an apoplectic seizure. This struck him while sitting quietly in his office. He suddenly felt “queer in the head,” sound seemed to fade away, an intense left frontal pain developed, and the right leg and foot felt temporarily numbed and weak. He was able to walk to his club a few hundred yards away. There he suddenly cried out with extreme pain in his head and

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the next four days he was confused, noisy, and partially aphasis and he complained of severe headache and pain right down the spine. The temperature was moderately elevated, the neck rigid and retracted, the Kernig sign positive, and the pulse slow and full. During the next week these symptoms improved. A lumbar puncture on the twelfth day after the apoplexy yielded strongly yellow fluid in which some remaining crenated red blood cells and active phagocytosis of blood pigment by endothelial cells were observed. In addition a strongly positive Wassermann reaction was obtained from the cerebrospinal fluid and blood. In a few weeks all symptoms, except the formerly dilated left pupil, disappeared. Anti-syphilitic treatment was given and the patient continues to enjoy good health, now four years after his apoplectic seizure.

In the light of experience with other similar cases it is easy to reconstruct the events in this one. He had an aneurysm of the left internal carotid or middle cerebral artery near the circle of Willis, in the development of which syphilis may have been a factor. In 1920 there was a slight leakage from the aneurysm which ceased spontaneously, but local effusion from which caused pain and oculomotor paresis. As the clot was absorbed and organised these symptoms subsided. Not until ten years later did the second and more serious leakage occur. As the blood escaped he experienced pain, and as it spread up via the left Sylvian fissure, he recognised numbness and weakness of the right limbs. There was a lull for a few minutes and then a larger effusion occurred causing apoplexy. This was followed by a period in which meningeal irritation combined with cerebral compression were the prominent symptoms, both being due to the presence of the effused blood in the subarachnoid space, and subsiding as this was absorbed. Firm clot had formed around and almost certainly within the aneurysm; this has been partly absorbed and partly organised. He may be spontaneously permanently cured by these processes, and in any event it is unlikely that he will have further trouble from this thrombosed aneurysm for many years. Such, then, is the clinical picture of spontaneous subarachnoid haemorrhage, and such is the probable prognosis when the event has been survived for some months without further evidence of haemorrhage.

The next case I wish to refer to illustrates a similar clinical picture, but a different and no less characteristic course of events.

**Case II.**—The patient was a married lady (No. 92) of 47. Four years before the fatal illness she had suffered from eclampsia, and during the next two years they were recurrences of raised blood pressure and signs of cardiac overload. However, for the two years preceding her death she had remained well and able for such activities as tennis, and the systolic blood pressure averaged about 150. Sixteen days before her death, after she had retired to bed, she experienced a sudden severe pain in her head which rapidly radiated to the left frontal region, down the back of her neck and spine and legs. She vomited and felt as if she would die. She was unable to summon assistance and probably lost consciousness for a considerable period. During the next five days, headache, backache and neck stiffness were present, the pulse was slow and the temperature slightly elevated. On the tenth day before death she had another attack, more abrupt and more severe than the former. It occurred at the breakfast-table; she gave a sudden cry of pain, collapsed, and was unconscious for two hours. During this time she was pale and almost pulseless. As she slowly recovered, signs of cerebral compression and of meningeal irritation were again present, and this time a definite degree of right hemiparesis and aphasia suggested that the effused blood was mainly implicating the left cerebral hemisphere. Papilloedema developed. Lumbar puncture at this time yielded fluid under high pressure, yellow from the former haemorrhage and heavily loaded with fresh blood from the latter one. On the twelfth day there was another similar attack with similar sequelae. On the sixteenth day a final and rapidly fatal haemorrhage occurred.

A post-mortem examination was obtained. There was slight cardiac hypertrophy, but the arteries generally, including those of the brain, appeared healthy. There was a large clotted subarachnoid haemorrhage occupying the basal cisternae and extending up the left Sylvian fissure. There was blood pigment staining in the midnests sulci over the entire cerebrum and cerebellum, in the leptomeninges of the spinal cord and in the sheaths of the optic nerves. The most recent haemorrhage, being confined by surrounding clot from the previous effusions, had forced its way along the path of the choroidal arteries and ruptured into the temporal horn of the left lateral ventricle, which was filled with recent blood clot. Clot had apparently impacted in the foramen of Monro and had prevented the effusion from extending into the third ventricle. The responsible lesion was found in a ruptured saccular aneurysm arising from the lateral aspect of the junction between the left internal carotid and unusually large posterior communicating arteries. Two smaller and unruptured saccular aneurysms were present, symmetrically situated on the upper aspect of the junction between internal carotid and posterior communicating arteries on each side. The larger ruptured aneurysm was about 5 mm. diameter, pedunculated, with a neck of attachment about 1 mm. The rupture was opposite this attachment, and in the form of a semi-detachment of the fundus of the sac. Around and within the sac was definite ante-mortem clot of considerable standing, and the recent haemorrhage had pushed its way along one side of this. Elsewhere it was firmly adherent to the internal and external surfaces of the aneurysm. The two smaller aneurysms were sessile and their diameter was about 1–5 mm.

In this case I would emphasise the previous eclampsia, the sudden unprovoked onset of bleeding, the diagnosis from the character of the clinical picture, its confirmation by the finding of blood in the cerebrospinal fluid and the evidence of the approximate site of the aneurysm by the left-sided frontal pain and right hemiparesis and aphasia. It is especially important to note the recurrent at-
tacks at intervals of ten to two days leading up to the fatal result of the fourth attack. The attempt at spontaneous healing by thrombosis is also noteworthy.

A third case (No. 388) illustrates a very similar train of events and I shall describe it in less detail.

Case III.—The patient was a single lady of 56. She had not previously complained of associated symptoms but her systolic blood pressure was known to be in the neighbourhood of 190. Seventeen days before her death, while speaking at the telephone, she was struck with an intense pain in her head and immediately collapsed. In an hour she recovered and exhibited the characteristic signs of cerebral compression and meningeal irritation. Lumbar puncture three days later showed a spinal fluid pressure of 320 mm. of water, the fluid was yellow and contained crenated red blood cells. Gradually the patient improved, but on the fifteenth day she again suddenly collapsed. Again she made a partial recovery, but signs of cerebral compression and oedema were progressive and she died from this cause two days later. In this case also there was fairly clear evidence from the site of headache, from a degree of hemiparesis, and from cranial nerve involvements that the source of bleeding was to the right of the midline.

A post-mortem examination showed gross generalised arteriosclerosis, with extensive atheromatous disease of the cerebral arteries especially. There was an extensive subarachnoid hemorrhage, filling every crevice of the subarachnoid spaces over the cerebrum, cerebellum, and in the spinal theca. The greatest mass of clotted blood was in the basal cisterns and in the right Sylvian fissure. The responsible lesion was found in a single small saccular aneurysm arising from the antero-inferior aspect of the junction of the right middle cerebral artery with its first large branch in the base of the right Sylvian fissure. The aneurysm was 1.5 mm. in diameter and was pedunculated in form, being attached to the artery by a neck about 0.5 mm. in diameter. The rupture affected the fundus of the sac which was semi-detached. Within and around the sac was ante-mortem clot of some standing, which was firmly adherent to the sac except along one side where the recent hemorrhage had detached it.

In this case there was a previous high blood pressure from arterio-sclerosis. The general characters and diagnostic features of the attacks are similar to the preceding case. Again recurrent attacks at short intervals proved rapidly fatal. There was again evidence of an attempt at spontaneous thrombosis within and around the ruptured sac.

From observation of a number of cases with single attacks and spontaneous recovery and return to health, and of a number with recurrent bleedings at intervals of days or weeks which ended fatally, we began to appreciate the sinister significance of a recurrence and the possibility of satisfactory and indefinite survival in its absence. Also from post-mortem observations we saw how a leakage from one of these small aneurysms induces thrombosis within and around the sac, and inferred that if a hemorrhage or series of hemorrhages is not fatal it is likely to result in a fairly secure healing of the aneurysm by thrombosis and organisation into a solid mass. Thus we decided that if another patient should have recurrent hemorrhages and there was evidence of the site of the aneurysm we should make some attempt to reinforce Nature's attempt at healing. We were accustomed to deal successfully with quite formidable intracranial hemorrhages during operations by applying to the bleeding point a fragment of fresh muscle which formed a secure scaffolding for the clot, and became organised into fibrous tissue with it. Why not expose a bleeding aneurysm and deal with it after this fashion? It is surprising how few of these hemorrhages from aneurysms on the large basal arteries are immediately fatal; the majority give sufficient warning to allow one to formulate a plan of treatment. A majority of these patients, moreover, are comparatively young, and many are perfectly healthy apart from this one small defect on a cerebral artery.

We had not long to wait in order to put these speculations to the test of practice, for, ten days after the death of the last recorded case, another presented, and began to run a similar course.

Case IV.—The patient was a healthy active man (No. 345) of 58. For several years he had suffered from recurrent left frontal headaches with simultaneous drooping of the left eyelid. These symptoms are recognisable in the light of subsequent events as due to slight recurrent leakage from his basal aneurysm. Then he had a typical attack of spontaneous subarachnoid hemorrhage with the characteristic sequelae of meningeal irritation and cerebral compression. Lumbar puncture showed blood in the cerebrospinal fluid. The blood pressure was only 130, and the attack occurred as he entered his house after a quiet evening stroll. He was recovering well, when, eight days after this attack, there was a further hemorrhage. Again he made a good recovery. On the fourteenth day, while at stool, he had a third and more serious hemorrhage with collapse for some hours, and then recovery with a residual left oculomotor paresis and some degree of aphasia. These signs indicated the site of the aneurysm on the left side of the circle of Willis, and of its effusion of blood up the left Sylvian fissure. From former experiences we felt certain that the illness would end fatally from further bleeding, and decided to operate in the hope of averting this.

Accordingly, on the sixteenth day of illness, after three progressively severe attacks of hemorrhage, the aneurysm was exposed by operation on 22. 4. 31. A left frontal approach was employed, and it was a difficult matter to elevate the tense and oedematous brain, and identify the basal structures which were blood-stained and largely embedded in clot. The left optic nerve was found, and the internal carotid artery was defined at its outer side. This vessel was closely followed upwards, outwards and backwards to its bifurcation into the middle and anterior cerebral arteries. As this point was being cleared of tenacious clot, a formidable arterial hemorrhage filled the wound. With the aid of suction apparatus held close to the bleeding point, we were able to see the aneurysm. It sprang from the upper aspect of
the bifurcation junction: it was about 3 mm. in diameter; blood spurted freely from its semi-detached fundus. Meanwhile a colleague was obtaining fresh muscle from the patient's leg. A small fragment of muscle was accurately applied to the bleeding point and held firmly in place so that it checked the bleeding and compressed the thin-walled aneurysmal sac. Thus it was steadily maintained for twelve minutes. As the retaining instrument was then cautiously withdrawn, no further bleeding occurred. The vessel was further cleared and thin strips of muscle were prepared and wound around it until a thick collar of muscle embedded the aneurysm and adjacent arterial trunks. A quantity of clot was removed from the left Sylvian fissure, and a small subtemporal decompression provided to relieve the considerable intracranial tension. It is now over two years since the operation. The patient has so fully recovered that he is able for the responsible legal and social duties on which he was formerly engaged, and he is able to indulge in shooting, mountaineering, etc. His old headaches have quite disappeared, and no trace of oculomotor paresis or aphasia remains. We believe that his aneurysm is tranformed into a solid nodule of fibrous tissue, and that the weak spot at his arterial junction is surrounded by a heavy collar of fibrous tissue organised from the muscle and clot.

The indications for carotid ligation in basal cerebral aneurysm will be considered later, but I should mention here that carotid ligation could not have benefited this man, for his aneurysm was situated on the collateral arterial channel via anterior communicating, anterior cerebral, and middle cerebral vessels, necessary to the adequate blood supply of his left cerebral hemisphere in the event of left carotid ligation. Thus the aneurysm would have remained with an active arterial circulation passing its mouth, and a normal blood pressure acting upon its walls. Nothing but an operation aiming at preserving and patching up the artery could meet the case.

In a second case (No. 562) we were successful in surgical treatment, by employing proximal carotid ligation.

Case V.—The patient is a healthy woman of 26 years, a hospital nurse. Her systolic blood pressure is about 118. During two years before the more serious illness to be described she had had several attacks of severe left frontal headache with vomiting and elevation of temperature lasting for several days. The attacks are probably ascribable to slight premonitory leakage of blood from her basal cerebral aneurysm. Suddenly, while on duty, and without any particular physical exertion, she experienced pain in the head “as if something had given way.” She felt faint and collapsed. She recovered, but was in bed with severe headache for two weeks. Papilloedema was noted at this time, and it persisted in a mild degree until her next attack.

The second major hemorrhage occurred (rather exceptionally) four months after the first. It took place while she was standing in a shop. She was able to telephone for assistance, but then lost consciousness. She remained thus, pale and apparently dying, with a pulse rate of 50, and with Cheyne-Stokes' breathing for twelve hours. Then she began to revive, and improved during the next four days. There was a left oculomotor paralysis, intense headaches, and gross papilloedema with unusually massive subretinal hemorrhages. It now appeared likely that she would recover from the attack, but it was apparent that unless intracranial pressure were relieved, she would become blind. We decided to carry out a decompressive operation so that if she recovered she might retain some vision.

Accordingly, on the fifth day after the hemorrhage, bilateral subtemporal decompression was carried out under local anaesthesia. On the left side I was surprised by encountering a large subdural clot. It was removed with marked relief of pressure. The pia arachnoid was quite free of blood. Evidently then the hemorrhage had occurred into the subdural space, and not as usual into the subarachnoid space. It was inferred that the point of bleeding must be situated on the left internal carotid artery between the point where it penetrates the dura and the place of its entering within the subarachnoid space. This is just where the artery bends back under the optic nerve and gives off its ophthalmic branch. This aneurysm then was situated proximal to the anastomosing circle of Willis. Proximal ligation of the internal carotid artery in the neck would leave the carotid stagnant from the point of ligation up to the circle of Willis, and would almost certainly induce thrombosis in this segment of the vessel.

The decompression gave relief, but on the sixteenth day of the illness there was a sudden recurrence of headache, and the decompressions bulged ominously. Clearly there was further bleeding in progress. Accordingly, the left internal carotid artery was tied in the neck. The evidences of hemorrhage subsided. On the third day after ligation there was a transient motor aphasia, which clearly indicated that a small embolus had lodged in one of the anterior branches of the left middle cerebral artery. The intravascular clot which furnished the embolus must have extended upwards at least to the junction of the posterior communicating artery. Thus we inferred that the part of the carotid artery carrying the aneurysm had become thrombosed, and that the dependent aneurysm must also be thrombosed and permanently cured. Fortunately, vision recovered unexpectedly well, and the patient is able to continue her work as a hospital nurse.

The point in this case is that by the unexpected discovery of a subdural clot we were able to infer the exact site of the aneurysm. The site was suitable for treatment by proximal ligation, and this treatment was apparently successful.

I would now refer to two cases in which the aneurysms simulated intracranial tumour.

Case VI.—The first concerned a man (No. 181) of 58 years, who exhibited a marked degree of arteriosclerosis and a systolic blood pressure of 190. He complained of loss of vision and headaches. He had a right homonymous upper quadrantic hemianopia, which was incongruous, and indicated a lesion at the junction of the left optic tract with the chiasm. He had a grossly enlarged sella turcica and evidence of long-standing hypopituitarism. The natural assumption was a tumour of the pituitary neighbourhood. At operation the optic nerves and chiasm appeared normal, and the large sella was empty.
Arterial radiography has now come to our aid. Our earliest attempts at cerebral arterial radiography were made in 19~7, when we used outlined on the X-ray film. Its size, connections, seen, together with the cerebral arteries, perfectly By this means an intracranial aneurysm can be the ligation been carried out immediately the rupture would not have occurred. The cause of spontaneous thrombosis in this case was, no doubt, intimal degeneration of the sac wall from arteriosclerosis.

This case shows, then, that a basal aneurysm large enough to cause tumour symptoms may become spontaneously obliterated, and permanently healed. The cause of spontaneous thrombosis in this case was, no doubt, intimal degeneration of the sac wall from arteriosclerosis.

Case VII.—The second tumour-like aneurysm I wish to refer to from the point of view of diagnosis and treatment. She was a married woman (No. 484), of 36 years, and was evidently a case of tumour in the pituitary neighbourhood. The left eye was blind from compression of its nerve, and the right visual field indicated that the optic chiasm was becoming involved at its junction with the left nerve. X-ray showed a shallow, wide depression in the bone between the two anterior clinoid processes. At operation a tumour was found underlying the left optic nerve, and presenting in the interval between the two nerves. Its bluish colour, even, rounded contour, and tense consistence, made us suspect aneurysm, and this was verified by aspirating arterial blood from it with a very fine needle. No direct treatment was attempted. We had in mind to tie the left internal carotid in the neck, but anticipated no harm from leaving this over for a few days. After an excellent primary recovery, this patient died suddenly twenty hours after operation from rupture of the sac. Evidently the removal of surrounding support occasioned by opening the skull had allowed this large sac to expand and burst.

In this case I wish to point out that although we could locate the swelling by perimetry and X-ray, we had no means of knowing that it was an aneurysm. Further, after learning at operation that it was an aneurysm suitable for treatment by proximal ligation, we made the error of not carrying this measure into effect at once, with disastrous consequences. I have no doubt that had the ligation been carried out immediately the rupture would not have occurred.

Arterial radiography has now come to our aid. By this means an intracranial aneurysm can be seen, together with the cerebral arteries, perfectly outlined on the X-ray film. Its size, connections, and relations can be seen as clearly as if it were exposed. Our earliest attempts at cerebral arterial radiography were made in 1927, when we used sodium iodide as the opaque medium. More recently we have employed “Thorotrast”—a colloidal suspension of thorium dioxide. It seems to be quite harmless when injected into the internal carotid artery, and it gives very clear definition of the cerebral arteries. By this means any deviations of the vessels occasioned by distortion from adjacent tumours can be seen, and any vascular anomaly such as aneurysm or arterial angioma is clearly depicted. This method, then, has put into our hands a means of defining whether a basal intracranial tumour is an aneurysm or some other swelling. Similarly, in a case of suspected aneurysm giving signs of spontaneous subarachnoid hemorrhage, an aneurysm may be detected and accurately located, and treatment planned accordingly. My next case exemplifies this eventuality.

Case VIII.—The patient is a young married woman (No. 635) of 28 years. Her average systolic blood pressure is about 112. She had previously enjoyed good health. Five weeks prior to admission to hospital she had struck the vertex of her head forcibly against the mantelshelf in raising herself from bending over the fire. She experienced no immediate inconvenience beyond the pain of the blow, but an hour later she developed a severe left frontal headache, and she vomited. The headache gradually subsided in a period of about three weeks, and just about this time she developed a left oculomotor paralysis—external squint, ptosis and dilated pupil. The paralysis was at first incomplete, but increased progressively. At this time we saw her, and suspected an aneurysm of the intracranial portion of the left internal carotid artery. As the oculomotor paralysis continued to increase we suspected either that the aneurysmal sac was rapidly enlarging or that it was leaking and clot was accumulating around it progressively. In either event a serious rupture was an imminent danger, and if we could only determine its exact site, this might be averted by proximal ligation or muscle wrapping. We, therefore, made an arterial radiogram of the left internal carotid artery, and its branches. This showed a round aneurysmal sac, about 7 mm. in diameter, attached by a narrow neck to the inferior aspect of the junction of the left internal carotid artery with its posterior communicating branch. From this point the aneurysm projected in a backward and downward direction. It certainly was not large enough to press injuriously on the oculomotor nerve by its own volume, and the symptoms must, therefore, have been due to accumulating clot from progressive leakage of blood. With knowledge of this alarming state of affairs and of the site of the aneurysm just proximal to the circle of Willis, proximal ligation was decided on, and the left internal carotid artery was tied in the neck forthwith. The patient made an excellent recovery, and soon returned to her home. The oculomotor paresis gradually recovered. The treatment was carried out on 24th March 1933, and the patient remains entirely well to the date of writing.

It will be observed from the foregoing remarks that intracranial aneurysms may present three different clinical aspects. There are the ocular
paretic type, the apoplectic type, and the tumour-like type. The ocular paretic varieties are usually characterised by an incomplete oculomotor paresis accompanied by homolateral frontal headache, and are due to small effusions of blood by limited leakage from an aneurysm near the circle of Willis. The apoplectic type is characterised by a more or less sudden "stroke"—with partial or complete loss of consciousness for a period, and subsequent signs of cerebral compression and meningeal irritation, with or without cranial nerve palsies and focal cerebral signs. These symptoms are provoked by a more extensive effusion of blood into the subarachnoid space or cerebral substance or both. There is blood in the cerebrospinal fluid obtained from lumbar puncture. The tumour-like variety is characterised by signs of compression of adjacent structures. The optic nerves and chiasm, the clinoid processes and adjacent bone are commonly involved.

In differential diagnosis the ocular paretic type offers little difficulty for the oculomotor nerve is not involved just in this way and with associated headache by any other disease process. Carcinomatous invasions of the base of the skull from the nasopharynx may simulate, but they involve the abducent before the oculomotor nerve. In the apoplectic cases the mode of onset and symptoms during recovery, and the fact that the patient is usually young and healthy, will serve to differentiate from cerebral thrombosis, intracerebral hemorrhage and meningitis. The diagnosis is confirmed by finding gross blood in the fluid from lumbar puncture. An accurate localising diagnosis in both these types may be made from arterial radiography. In the tumour-like aneurysms the clinical diagnosis cannot be carried beyond the inference of a progressive swelling in a certain situation. In such a case the diagnosis that the swelling is an aneurysm can be made only by operative exploration or by arterial radiography. We have been employing arterial radiography in the diagnosis of doubtful tumours about the cerebral base, and, in addition to defining aneurysms, we have been able to gain information concerning tumours of other kinds by their effects in distorting the adjacent cerebral arteries.

The etiology of saccular aneurysms of or near the circle of Willis has been the subject of much speculation and dispute. There exists reliable evidence to show that aneurysms may develop here as elsewhere in consequence of infective emboli or by reason of adjacent pyogenic, tuberculous, or syphilitic inflammatory processes. These are instances of local disease damaging a presumably healthy vessel wall. They are rare eventualities. Aneurysms of the basal cerebral arteries, on the contrary, are relatively common. An average quotation of their incidence from available literature is about one in seven hundred consecutive postmortem examinations, and in many instances the lesion has been symptomless and is not connected with the cause of death. They are much commoner in the absence of arteriosclerosis and of syphilis than in their presence. The average age for rupture of these aneurysms is about thirty-two years; instances of rupture at six and nine years of age are on record. It is evident enough that the primary factors both in formation and in rupture of an aneurysm are the pressure of the blood and a local weakness of the vessel wall.

Recent researches, and especially those of Forbus, furnish us with an adequate explanation of the factor of local defect of the vessel wall. In the development of arteries the larger trunks acquire a muscular coat, while their smaller branches remain for a time as simple endothelial tubes. Later the branches acquire muscular coats, not as outgrowths from those of the larger trunks, but as independent developments. At the line of junction of branch with trunk the new and the old muscle coats meet and become fused, but the joint or fusion may be imperfect. In apparently normal arteries small developmental gaps are demonstrable along these lines. They constitute quite definite weak points of developmental origin in the vessel wall. In this connection it is significant that all the saccular aneurysms under discussion are found to arise along the line of juncture of arteries with their branches and never from the arterial walls between junctures. Cases are on record in which the "plumbing" of arterial joints has been generally effective, and in which small aneurysms were present at arterial junctions in many parts of the body. This is, however, quite rare; it is the basal cerebral arteries in particular which are affected with considerable frequency. Much remains to be learned about the cerebral arteries and their circulation, but it is true that they are peculiarly thin-walled, and that they are specially protected from the force of the pulse by obviously designed flexures or "baffles" on the main vertebral and carotid trunks just as they enter the skull. Why they are thin-walled and specially protected we do not know but only that they are so. Being much thinner in their walls than arteries elsewhere in the body, it is easy to understand why the developmental junction defects in the muscle coats should more readily give way in the cerebral arteries while they remain insignificant on the thicker-walled arteries elsewhere.

Lastly we come to the factor of the pressure of the blood in blowing out the arterial wall through such a defect to form a saccular aneurysm. It is evident from the clinical cases that this may be caused by a normal blood pressure, as in the cases occurring in children, and in most of the younger and some of the older adult subjects. There is, however, no doubt that an abnormally high
arising from the junctions at the origin of the gross subarachnoid hemorrhage. The aneurysms recurrent ophthalmoplegia and with spontaneous rather than as tumours, and are associated with election. These aneurysms are apt to manifest first considerable branch from the middle cerebral cerebral arteries, and the points of origin of the formation is in relation to the termination of the carotid artery on one or other side. There arterial blood pressure may cause an arterial junction to give way which would have remained intact under a normal pressure. This is evidenced by the definite association of basal cerebral aneurysm with stenosis of the isthmus of the aortic arch. In such a case (No. 619) I have observed a blood pressure of 190 in the right arm and associated carotid arteries, while in the left arm and leg receiving blood distal to the stenosis it was 120. This man had had several attacks of sudden collapse associated with temporary oculomotor paralysis, headache, and neck rigidity, which left no doubt of the presence of an aneurysm on or close to the intracranial portion of his left internal carotid artery. The association is sufficiently frequent to make it clear that the abnormally high pressure thrown on the cerebral arteries by the stenosis is the essential cause of the formation of aneurysms in these cases. In case No. 38, quoted above, who can doubt that it was the raised blood pressure of eclampsia which caused her multiple aneurysms to bulge through junctions which would have withstood normal pressures? Again in case No. 345 the third attack of bleeding was obviously precipitated by the rise in arterial pressure due to straining at stool. It seems clear, then, that there is a developmental basis of weakness in the arterial walls at their junctions, and that this is accentuated in the thin-walled cerebral arteries. The developmental gap in the muscle coat may amount to a developmental defect or anomaly in such a degree that the vessel gives way, and an aneurysm forms under normal arterial blood pressure. In severe defects an aneurysm may form and burst even in childhood. In lesser defects the event may be delayed even until old age. On the other hand the arterial junctions may be normal, and capable of withstanding normal blood pressure, yet they remain the weak points in the vessel wall, containing minute gaps in the muscle coat which will give way and form aneurysms under conditions associated with abnormally high pressures in the cerebral arteries.

The commonest site of saccular aneurysmal formation is in relation to the termination of the internal carotid artery on one or other side. There is a preponderance in favour of the left side. These aneurysms are always at arterial junctions, and the junctions at the posterior communicating branch, the bifurcation into middle and anterior cerebral arteries, and the points of origin of the first considerable branch from the middle cerebral artery in the base of the Sylvian fissure are sites of election. These aneurysms are apt to manifest themselves by minor or major effusions of blood rather than as tumours, and are associated with recurrent ophthalmoplegia and with spontaneous gross subarachnoid hemorrhage. The aneurysms arising from the junctions at the origin of the ophthalmic branch from the internal carotid artery and at the joining of anterior cerebral and anterior communicating arteries are rather less frequent in incidence and tend to manifest themselves as tumefactions rather than by the occurrence of bleeding from them. Of still lesser frequency are aneurysms arising at the junctions of vertebral and basilar arteries and at the bifurcation of the basilar into the posterior cerebral arteries. Aneurysms can occur at any arterial junction of the larger cerebral and cerebellar arteries.

In the practical treatment of these lesions a conservative line may be adopted, or proximal ligation of a carotid artery, or application of muscle fragments directly to the aneurysm. Each method has its indications. In the event of a minor hemorrhage with associated ophthalmoplegia and headache, or of a major subarachnoid hemorrhage, the presence of an aneurysm as its cause should be verified and the exact site of the aneurysm identified by arterial radiography on the internal carotid artery. If the aneurysm is found to be proximal to the circle of Willis (i.e., the posterior communicating branch), proximal ligation of the internal carotid artery in the neck should be practised. The procedure is not without risk of cerebral complications, but the advantage of diminishing the pressure on the aneurysm outweighs these risks. Similarly, in the event of an aneurysm being defined in this situation by radiography or operative exploration in cases exhibiting a basal tumour syndrome, proximal ligation should be employed. In the cases of operative exploration the ligation should follow immediately in order to minimise the danger of rupture.

If in a case of minor or major hemorrhage from an aneurysm, the aneurysm is defined by clinical facts or arterial radiography as distal to or actually on the circle of Willis, I should advise conservative treatment in the first instance. Proximal ligation is obviously no use in such cases by reason of the relation of the anastomotic blood supply to the aneurysm. The only alternative to conservative treatment is direct operative exposure and application of muscle, which is necessarily a hazardous and difficult procedure. Experience teaches us that a considerable proportion of first bleedings settle down and remain quiescent for many years and perhaps permanently. It is felt that the patient's interests will be served best by relying on those chances of spontaneous healing rather than undergoing the risks incidental to direct operative exposure in the first instance. In the event of a repetition of bleeding, however, especially at an interval of a few days or weeks, the prognosis becomes extremely grave, the probability of spontaneous healing extremely low, and the risks of direct operation are then justified. At operation there is no question of ligation of a main arterial trunk distal to the circle of Willis.
The functional loss from this would be far too severe. The aim is to form a secure scaffolding for clot and fibrosis around the aneurysm by application of fragments of muscle, while the artery remains patent and intact.

In the case of an aneurysm giving a tumour syndrome and found to be distal to the circle of Willis, conservative measures should be adopted unless repeated hemorrhages occur.

Conservative treatment by rest and morphia in the earlier days following a single subarachnoid hemorrhage may be supplemented by lumbar puncture. I do not think that this is likely to promote further bleeding if the fluid is removed slowly, and so as not to reduce pressure below normal. By this means considerable quantities of irritating blood may be removed, and the symptoms of cerebral compression and meningal irritation thereby relieved, and convalescence shortened. After recovery such patients should be warned against such activities as are likely to raise arterial blood pressure considerably, but with this reservation that they should be encouraged to live normal and active lives. No doubt they carry a potential source of danger in their heads, but, after all, any one of us may have such a latent lesion.


INTRACRANIAL ANEURYSM OF THE INTERNAL CAROTID ARTERY
CURED BY OPERATION*
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Case Report.—A rather frail, small, sallow man, age 43, applied at the Johns Hopkins Dispensary February 16, 1937, because of complete paralysis in the distribution of the right oculomotor (third) nerve. The family history was negative. His general health was good until last year when his stomach “went bad” from drinking. He was hospitalized from July to September, 1936, for this gastric disorder which was pronounced “ulcer.” He has been a very heavy drinker for the past 18 months.

Present History.—Six days ago he was awakened by a severe pain in the right frontal region. During the afternoon there was a very severe shooting pain in the right eye, but it lasted only a moment. He slept poorly that night because of the pain. On the following morning diplopia was first noted and in the evening the right eyelid drooped. The eye was completely closed the next morning. The pain became less severe but two days later became greatly intensified and prevented his sleeping. Since then the pain has been present but less severe. Examination at that time showed a complete paralysis of the right, third cranial nerve (Fig. 1). There were no other positive findings. The eye grounds, visual fields and reflexes were normal. A diagnosis of aneurysm along the circle of Willis was made. A roentgenologic examination of the head revealed no abnormality. The patient returned to the dispensary from time to time until March 19, 1937—nearly five weeks after the onset of his trouble—when Dr. Frank Ford referred him to me with the thought that a surgical effort might be worth while. There had been no improvement in the local condition in the interim.

The following findings were reported by Dr. Frank Walsh, of the Ophthalmological Department, March 12, 1937:

“The upper lid is completely closed and can only be moved slightly by the frontalis muscle (Fig. 1). The globe is abducted to 45° and only moves laterally and slightly down when it rotates inward (Fig. 1). The pupil is 4½ Mm. in diameter and one-fourth larger than the left. It reacts slightly to light, directly and consensually. Visual acuity 20/40 right and 20/25 left. Visual fields normal. Fourth and sixth nerves are functioning.” The Wassermann reaction was negative.

Operation.—March 23, 1937: A small hypophyseal approach was made on the right side, using the concealed incision. There was marked cortical atrophy, evidenced by the pools of fluid in the subarachnoid spaces (doubtless the result of his heavy drinking). The removal of this fluid and that from the cisterns chiasmatis gave ample room for exposure of the chiasmal re-


Fig. 1.—Photograph of patient taken before operation. Note the ptosis on the right, and the extreme pull of the eyeball outward due to paralysis of the 3rd nerve.

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tion upon retraction of the frontal and temporal lobes. A pea-sized aneurysm projected from the outer wall of the internal carotid artery and adjacent to the entry of the posterior communicating artery (Fig. 4). The aneurysm, however, did not involve this vessel, but arose from the internal carotid by a narrow neck beyond which it expanded to the size of a pea; therefore, it was quite a small aneurysm. Laterally it bridged the adjacent cerebral space and firmly attached itself to the free border of the dura which projects mesially from the middle cranial fossa; it spread out beneath the dura forming quite a broad attachment. At this site the covering of the aneurysm changed from the normal grayish-white, shiny covering, similar to that of the carotid artery, to a deep red color. Moreover, the surface was irregular, three or four tiny nodules projecting along the margin of the cavernous sinus. This change represented the false aneurysmal sac resulting from rupture of the aneurysmal sac. The third nerve passed obliquely backward in its normal course and was attached to the aneurysm at only one point—where it entered the cavernous sinus. Since it was quite evident that the red color of the aneurysmal wall indicated a reduction in its thickness, no attempt was made to dissect the attachment to the wall of the cavernous sinus. There was no evidence of subarachnoid bleeding; doubtless the growth into the wall of the cavernous sinus prevented this. Forceps placed upon the thick aneurysmal wall pulsated forcibly.

The small neck of the aneurysm afforded an easy surgical attack. An ordinary flat silver clip was placed over the neck of the sac and tightly compressed, obliterating it completely (Fig. 4). The clip was flush with the wall of the carotid artery (Figs. 5 and 6). The sac, lateral to the silver clip, was then picked up with the forceps and thrombosed by the electrocautery. It shriveled to a thin shred of tissue. It is worthy of note that the aneurysm became much softer after the silver clip had been applied; it also ceased to pulsate.

**Postoperative Course.**—Aside from an attack of delirium tremens which lasted three days, patient made an uneventful recovery and left the hospital April 5, 1937—two weeks after the operation. At that time there was a definite improvement in the function of the extra-ocular muscles (Fig. 2).

On April 8, 1937 (three days later), Doctor Walsh reports: (1) Improvement in the ptosis; (2) slight upward movement; and (3) the lateral movements of the eyeball are close to normal. The pupillary reaction is still a little less than the left. Seven months later there was complete return of all functions referable to the third nerve (Fig. 3).

Perhaps ten years ago I saw, with Dr. Fuller Albright, an aneurysm situated in a somewhat similar position, localized because of the paralysis of the third nerve and pain in the eye. An attempt was made to cure it by ligation of the internal carotid artery in the neck, but the patient died of cerebral softening as a result, probably from an extending thrombus. Such an indirect attack surely had little chance of curing the aneurysm but there then seemed no other rational effort indicated. The present case is a sequel to this unsuccessful attempt. The precise point of origin of this aneurysm could not be predetermined; it might have arisen from the carotid or the posterior communicating artery; the latter was our impression at the time of operation. If it had arisen from the posterior communicating artery it was hoped that a silver clip could be placed upon the artery on each side of the aneurysm if there was not a satisfactory neck by which the aneurysm could be attached directly.

A number of publications have appeared in recent years indicating that aneurysms of the circle of Willis are quite common. It is from them that most of the subarachnoid hemorrhages arise. Unfortunately, in most instances there are no localizing signs by which the position of the aneurysm or, indeed, the size of the aneurysm can be estimated. Those with paralysis of the third
nerve, as in our case, are exceptional. Sands makes the statement that 47 per cent of those along the posterior communicating artery produce signs referable to the third nerve. Certainly those with palsies of the third nerve may be given the chance of surgical cure. On the other hand, there is no assurance that the aneurysm after its disclosure may be amenable to surgical attack—the aneurysm may be too large, or it may be placed too far posteriorly on the posterior communicating artery. Under the latter condition perhaps a single clip anterior to the aneurysm might be effective; or the aneurysm, if arising from the carotid, may be less favored by a narrow neck by which it can be isolated and cured by the application of a silver clip. The present effort is but a beginning or a suggestion that an aneurysm at the circle of Willis is not entirely hopeless. A word may be added concerning the cauterization of the aneurysm by which it is shriveled to a small shred. The silver clip, of course, added the same sense of security against an extending thrombus, which, I should think, would be quite likely if the cautery were used alone, but perhaps no more probable than by a spontaneous thrombosis, which may conceivably occur. At least I should be fearful of such an outcome without the intervening clip to prevent its spread. Should the outlook be hopeless one would, of course, be justified in this attempt, and it is not inconceivable that even then a cure might result from thrombosis of the aneurysm without extension into the main arterial trunk.

In general, the indications for operation on aneurysms at the circle of Willis and causing only
subarachnoid hemorrhage, are none too clear. Certainly without a knowledge of the side of the circle of Willis upon which the aneurysm is located there would be no justification in exploring either side in search of the lesion. When a patient has had a subdural hemorrhage and has recovered, one is loath to suggest an operation, which certainly would be classed as hazardous, because another hemorrhage may never occur; at least many go for years with no further trouble, although this is not the usual story. During a subarachnoid hemorrhage and the immediate period thereafter one would not dare operate because the intracranial room needed for operation would be occupied by blood—and one needs all the room obtainable for the operation. For cases with a third nerve palsy the indications are clear enough. And where subarachnoid hemorrhages are recurring and the eventual outlook seems hopeless I should feel inclined to advise operative attack if there is even a suggestion that the aneurysm may be on one side. Arteriography may here become an

Fig. 5.—Roentgenogram showing the silver clip at the sella turcica. It also shows the size and position of the bone flap.

Fig. 6.—Roentgenogram of the base of the skull showing the position of the silver clip placed upon the neck of the aneurysm.
important means of locating one of these aneurysms around the circle of Willis. Then, too, the frequency of multiple aneurysms, and under such circumstances the difficulty of locating the one that is at fault, make the problem of therapy an even more difficult one.

So far as I know, this is the first attempt to cure an aneurysm at the circle of Willis by direct attack upon the aneurysm.

REFERENCES


