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[1] gave the first clear description of the clinical features of spontaneous subarachnoid hæmorrhage 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 apoplexies 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.[2] In 1931, the next important clinical advance was made by Professor Edwin Bramwell's paper[3] 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. 234) 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,

* Read at a Meeting of the Medico-Chirurgical Society of Edinburgh, on 28th June 1933.

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 collapsed. He was unconscious, pale, and almost pulseless for half an hour and then gradually revived. For the next four days he was confused, noisy, and partially aphasic, 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 cerebro-spinal fluid and blood. In a few weeks all symptoms, except the formerly dilated left pupil, had 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 hæmorrhage, and such is the probable prognosis when the event has been survived for some months without further evidence of hæmorrhage.

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. 32) of 47. Four years before the fatal illness she had suffered from eclampsia, and during the next two years there 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. Papillœdema developed. Lumbar puncture at this time yielded fluid under high pressure, yellow from the former hæmorrhage and heavily loaded with fresh blood from the latter one. On the twelfth day there was another similar attack with similar sequelæ. On the sixteenth day a final and rapidly fatal hæmorrhage 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 hæmorrhage occupying the basal cisternæ and extending up the left Sylvian fissure. There was blood pigment staining in the minutest sulci over the entire cerebrum and cerebellum, in the leptomenix of the spinal cord and in the sheaths of the optic nerves. The most recent hæmorrhage, 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 hæmorrhage 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 attacks 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. 338) 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 œdema 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 hæmorrhage, 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 hæmorrhage 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 hæmorrhage or series of hæmorrhages 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 hæmorrhages 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 hæmorrhages 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 hæmorrhages 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 53. 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 hæmorrhage with the characteristic sequelæ 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 re-entered his house after a quiet evening stroll. He was recovering well, when, eight days after this attack, there was a further hæmorrhage. Again he made a good recovery. On the fourteenth day, while at stool, he had a third and more serious hæmorrhage 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 hæmorrhage, 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 œdematous 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 hæmorrhage 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 transformed 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. Papillœdema was noted at this time, and it persisted in a mild degree until her next attack.

The second major hæmorrhage 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 papillœdema with unusually massive subretinal hæmorrhages. 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 hæmorrhage, bilateral subtemporal decompression was carried out under local anæsthesia. 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 hæmorrhage 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 ligature 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 hæmorrhage 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 180. He complained of bad 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 except for cerebrospinal fluid. No tumour was found. The operative exploration did neither harm nor good, and the cause of his symptoms remained a mystery until his death eighteen months later. There had been no change in his symptoms, and he died from multiple cerebral softenings due to his arterial disease. At post-mortem the cause of his chiasmal and pituitary symptoms was found in an aneurysm arising from the under surface of the junction of the right anterior cerebral artery with the anterior communicating artery. This aneurysm had undergone spontaneous thrombosis, fibrosis, and cure. It was a solid nodule of fibrous tissue about 5 mm. diameter. Evidently when filled with fluid blood it had been large enough to cause the symptoms of tumour described. There was no evidence that the aneurysm had ever become ruptured.

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 hæmorrhage, 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 23 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 hæmorrhage 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 post-mortem 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,[4] 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 walls. 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 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.[5] In case No. 32, 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 hæmorrhage. 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 frequence 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 hæmorrhage with associated ophthalmoplegia and headache,

or of a major subarachnoid hæmorrhage, 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 hæmorrhage 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 hæmorrhages occur.

Conservative treatment by rest and morphia in the earlier days following a single subarachnoid hæmorrhage 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 meningeal 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.

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DISCUSSION.

Professor Drennan said—In the course of my work as a pathologist I have had opportunities of seeing a number of lesions of this type, some examples of which I recorded some years ago. Mr Dott mentioned that you may have more than one aneurysm: I can corroborate this statement, as I have seen as many as three or four at the bifurcations of different cerebral arteries. I think such a case would present great difficulty in handling, and one or more might easily be overlooked owing to their distance apart. As shown by Forbus and referred to by Mr Dott, the muscle coat is apt to be defective at the bifurcations of the cerebral vessels, and there is always the possibility of its giving way there. I think an additional factor in increasing that weakness is the occurrence of small arterial twigs at the angle of bifurcation of the larger vessel and at a point in line with the main blood flow. You have here a defective piece of mechanism—indeed bad plumbing—very likely to give way; and in the case of the older patient, arterio-sclerosis increases the possibility of the weak points giving way.

As regards the leaking, the points of leak can be seen when such aneurysms are examined serially. Fracture in the internal elastic lamina is often demonstrable and a patching up by fibrous tissue.

The outside of the aneurysm often has a knobby appearance due to its giving way at different points at different times, until there comes the stage when it gives way finally and death results. Sometimes the initial fracture is in quite the opposite direction from the final fatal rupture.

I should like to say one word in appreciation of Mr Dott's technique. Even at *post-mortem* examination with the brain removed, it is often difficult to demonstrate an aneurysm, especially if there has been repeated leaking, as it is embedded in layers of firm clot. If a small aneurysm, *e.g.* the size of a pea, is the cause of all the trouble, it must be extremely difficult to demonstrate it when working in the small area such as Mr Dott has at his disposal. There is one other point. What is the exact mechanism of this muscle wrapping? Is it merely a mechanical thing, and what is the end-result of it? Has Mr Dott had any opportunity of examining cases which have died from some other cause?

Dr Traquair said—We have learned to-night that another condition, hitherto regarded as obscure and fatal, is amenable to diagnosis and treatment. The oculist frequently sees cases of third-nerve palsy, and his main interest is in the diagnosis. Hitherto we have been taught to regard syphilis as the most common cause of uncomplicated third-nerve paralysis: from what we have learned to-night, we must also take aneurysm into consideration. I should like to ask Professor Bramwell and Mr Dott whether, in cases of third-nerve paralysis in which the third nerve alone is affected, the diagnosis is not likely to be syphilis. My experience had been that in such cases the blood Wassermann is often positive, while, in cases where another nerve, such as the fifth or sixth, is also affected, the Wassermann may be negative. Henceforward I shall think of aneurysms as a possible diagnosis, especially in young people.

In connection with the tumour type of case mentioned by Mr Dott, there is a case recorded by Dr Foster Moore, in which a correct diagnosis was made, during life, without arterial radiography, of an aneurysm underlying the optic nerve.

I should like to raise one point in regard to what Professor Bramwell said as to the frequency of permanent hemianopia in connection with migraine. I am intensely sceptical about this and should like to know a little more about the cases in which it has been found. Some cases have been recorded, but on close scrutiny they do not seem to me to be cases of migraine at all. Sometimes the term is a little loosely used and is applied to any kind of bad headache. I think one has to be careful in making a diagnosis of migraine.

Dr A. Rae Gilchrist said—I should like to ask Professor Bramwell and Mr Dott about the advisability of doing repeated lumbar punctures. I realise that the danger of cerebral compression by accumulation of blood is considerable, and that by withdrawing spinal fluid that pressure may be reduced. At the same time, I wonder if there is not

a possible danger that, simply by taking off fluid, one may not thereby reduce the pressure to such an extent that the aneurysm may start bleeding again. I know that in the case in which I had the pleasure of being associated with Mr Dott, when the first lumbar puncture was done, the patient was considerably relieved: but a few days later a recurrence took place; and it occurred to me that possibly the withdrawal of fluid (which we had carried out on more than one occasion before operation) might possibly have been a factor in promoting the second hæmorrhage. For treatment at the acute stage, when the headache is intense and the patient collapsed, one would like very much to have an indication if such a simple procedure as lumbar puncture is altogether free from danger.

Dr W. M'C. Harrowes said—I should like to know what exactly is meant by "ophthalmoplegic migraine" in the cases we have been hearing about to-night. I have seen patients suffering from migrainous attacks which did not present obvious features of an ophthalmic nature. Recently a long article by MacDonald Critchley on the subject of migraine appeared in the *British Medical Journal*, in which he made a division of certain types of migraine into several groups; but the thing that remained most clear in my mind after reading the article was that the only method of treatment for the unfortunate subject was to lie quietly in bed in a darkened room until the attack had passed off. One sees from time to time attacks of headache where the pain is so severe that the individual is completely incapable of performing any sort of work, and these attacks go on very often to severe vomiting. Ultimately recovery takes place in twenty-four hours without any particular eye symptoms appearing. Are these cases relatively or absolutely different from cases with frank ocular palsy?

My interest in those cases was focussed very strongly at a time when I was associated with Dr Wolff, now of Cornell (U.S.A.), and we had a case presented to us—a man of 35, who had sustained a severe injury to his head while working on the railway. This happened in Kentucky, and there an enthusiastic surgeon performed a decompression, leaving a very large deficiency in the left temporal area. The man came to the Johns Hopkins Hospital suffering from very severe headache of migrainous type. As these attacks did not respond to any treatment, Dr Wolff and I made an experiment. A tambour was fixed to the deficient area and we were able to register the pressure of the underlying brain on a drum. We found that when the headache was most severe the pressure on the soft mass underlying the deficiency was not high; but we discovered that the pressure of the mass rose very considerably if we attempted, in hypnosis, to make the patient re-live the accident and suggested that he was experiencing again the severe pain which had led up to the decompression being done. On suggesting that the pain had now gone, the

pressure diminished again. I regret that I am unable to remember the actual figures. What did not tally was the fact that when conscious and suffering from headache, the pressure was not high and this puzzled us. The connection between that observation and what has been said to-night regarding the influence of head injury seemed to me rather close, and I should be very interested to hear what Professor Bramwell and Mr Dott think of it.

Dr F. K. Kerr said—As a general practitioner, I find that migraine is a comparatively rare disease nowadays, less common than it used to be, and, with the panel patient's easy access to a doctor, I think the decrease is real and not just apparent. Probably there is a dietetic factor here, because people are much better fed nowadays.

I am interested in Mr Dott's statement that intracranial aneurysms have been found in patients with a normal or even subnormal blood pressure. I had always held that you could not have a real cerebral leakage or hæmorrhage unless the arterial systolic pressure is considerably above normal. The most amazing thing to me is that I have patients coming to me with a pressure of over 200, and they go on year after year without suffering any apparent consequences.

Dr J. Duncan White said—Mr Dott is to be congratulated on this case of intracranial aneurysm in which he made clear the diagnosis by arterial radiography, because it is the first of the kind in this country. He made the observation that the method of examination is perfectly harmless, but I think we do not know quite enough about it to be so dogmatic as yet. Between 50 and 70 c.c. of thorotrast are injected into the blood stream when one is demonstrating the liver or spleen, and the "dye" remains in the tissues for a period of two years. Thorium dioxide is radio-active, and there is a possibility of late results occurring in the tissues. Certainly Stewart of New York, who was investigating this matter, found that a month after 50 c.c. had been injected into the blood-stream, a spleen, removed at *post-mortem* examination, still had a photographic effect on a plate.

In addition to obtaining assistance from arterial radiography in the case of aneurysms, one would expect that in the diagnosis of large vascular tumours, the help given in localisation would be of considerable value. I should like to ask Mr Dott if he thinks there is any contra-indication to further arterial radiography of the opposite side at the same period, say the same day, so that we could have an idea as to whether the tumour was on the right or the left side.

Professor Bramwell, in reply, said—Aneurysms of the cerebral vessels are, as Mr Dott has indicated, not uncommon. A congenital weakness of the arterial wall, with in some cases superadded degenerative changes, is the most common cause. Syphilis very rarely plays an etiological rôle. Further, as Mr Dott has pointed out, these

aneurysms are usually of small size and rarely give rise to symptoms unless leakage or rupture occurs. An intracranial bruit was at one time believed to be a characteristic feature, but apart from arterio-venous aneurysms a bruit is very seldom heard. Beadles, who collected the records of 555 cases of intracranial aneurysm from the literature, found only two in which a murmur had been noted.

Spontaneous subarachnoid hæmorrhage, though often due to a ruptured aneurysm, is not diagnostic, for instances are reported in which death has resulted from a subarachnoid hæmorrhage and no aneurysm found after the most careful search. Conversely, the presence of blood or a xanthochromia in the cerebrospinal fluid is not necessary for the diagnosis of aneurysm. One of the cases reported by Mr Dott, to which I also referred in my paper, is of particular interest in connection with diagnosis. This patient complained of severe and persistent headache, particularly in the region of the left eye, after receiving a blow on the back of the head. A fortnight later she developed a left third-nerve paralysis. When examined she was found to have definite neck rigidity; Kernig's sign was absent, and there was no coloration of the cerebrospinal fluid. A probable diagnosis of a leaking aneurysm in the region of the intracranial termination of the internal carotid was arrived at. I asked Mr Dott to see the patient and suggested that, if he thought it justifiable, he should take an arterial radiogram. He has shown you a lantern slide in which the aneurysm is very clearly seen. This case is probably unique for, as Dr Duncan White has said, he knows of no case of congenital aneurysm of the cerebral arteries which has been demonstrated by this method. The case is of interest too from the medico-legal point of view, since the facts would appear to indicate clearly that a blow on the head had determined the rupture of the aneurysm.

So far as I know, this is the first occasion upon which the question of the treatment of a leaking aneurysm by direct exposure has been brought before a medical society, at any rate in this country. But the exposure of an aneurysm by the surgeon demands that the situation of the aneurysm should be accurately located. Arterio-radiography opens up new possibilities here, for an intracranial aneurysm can very seldom be localised with any degree of certainty on purely clinical grounds unless it happens to be situated in the region of the intracranial termination of the internal carotid and involves the third nerve. We require further facts in relation to the indications for operation in cases of leaking aneurysm for, as I have pointed out in my paper, there may be spontaneous arrest of the leakage with, it may be, permanent healing. Wilbrand and Saenger refer to two cases, with, however, no accompanying clinical record, in which a healed aneurysm was found *post mortem* lying in contact with the third nerve. Mr Dott has mentioned the case of a large aneurysm in which a decompression was carried out. I would ask him

whether he thinks that there is under such circumstances a risk of rupture. Dr Traquair has raised the question whether when a third-nerve paralysis occurs in elderly people it is usually attributable to syphilis. It is possible, I think, that in some of these cases the paralysis may be due to pressure on the third nerve by an intimately related posterior cerebral artery which is the seat of calcareous changes. I am inclined to agree with Dr Gilchrist that it is doubtful whether repeated lumbar puncture is indicated, as is recommended by some authorities, in cases of subarachnoid hæmorrhage unless there is evidence of increased intracranial pressure which is endangering life. Dr Harrowes's remarks upon the nature of migraine are interesting. The subject of migraine has been dealt with recently in an exhaustive critical digest by Riley. My impression is that while the etiological possibilities have been multiplied, we know little more of the etiology of migraine than we did twenty-five years ago.

Mr Norman M. Dott, in reply, said—Professor Drennan inquires as to the fate of implanted muscle used for the purpose of arresting hæmorrhage. I have had an opportunity of examining implanted muscle up to about six weeks after insertion. One finds the muscle in process of transformation by invasion of fibroblasts into fibrous tissue. In using muscle for the purpose of stopping bleeding in an aneurysm, I think that is all that happens. The muscle forms a basis on which blood clot can form—a kind of solid scaffold—and there is subsequent transformation both of the clot and the muscle into fibrous tissue. Certainly there is no question of muscle acting as a graft and retaining its muscular structure or function.

Dr Gilchrist raises the point as to the possible danger of reducing intracranial pressure by lumbar puncture on the ground that it might aggravate bleeding from a leaking aneurysm. I think that if the lumbar puncture is carried out carefully and the fluid removed slowly, it would have no significant effect in inducing or aggravating bleeding. Certainly, especially in the earlier stages of the condition, it relieves symptoms considerably and I think it is proper treatment.

Professor Bramwell inquires as to whether decompression might of itself tend to produce rupture of an aneurysm. I think this probably depends mainly on the size of the aneurysm. If the sac were large, I should think that on opening the skull and reducing the supporting pressure on its walls there would be very considerable danger of allowing further expansion leading to rupture. In the case of a small aneurysm, external pressure is obviously a much less important factor than internal pressure on the aneurysmal wall, and I do not think that opening the skull would have any significant effect.

Dr Harrowes's observations on conditions existing during attacks of migraine are extremely interesting. I am afraid I have nothing to add to speculations as to the cause of the pain.

Dr White's observations regarding the possible dangers of introducing thorotrast into the circulation certainly require consideration. He speaks of the large quantities used in demonstrating the liver and spleen—50 c.c. or more. In order to demonstrate the carotid artery and its branches, 7 or 8 c.c. is quite sufficient, so that we may at least say that cerebral arterial radiography is less dangerous than the commonly practised examination of the liver and spleen by this means.

Professor Bramwell criticises the use of the term "ophthalmoplegic migraine" as used in too wide a sense. I agree with him that the term should be restricted to cases having a periodicity resembling migraine and should not be applied to the symptoms induced by recurring leakages from a basal cerebral aneurysm.

Dr Traquair is sceptical concerning the production of permanent hemianopic defects by genuine migraine. I have two experiences concerning that matter. The two patients concerned had characteristic histories of migraine dating from their teens. They were aged about 35 when they came under my observation. They had suffered from apparently typical attacks of migraine, with the common unilateral homonymous hallucinations of light. When I saw them, one patient had a complete homonymous hemianopia and the other a superior quadrantic defect. I felt rather sceptical about a permanent hemianopia following migraine, and in both cases when the cranium was examined by means of a stethoscope a systolic bruit was heard. In each case the lesion was an arterial angioma, one in the temporal and the other in the occipital region. So far as my experience goes, I would endorse Dr Traquair's scepticism as to the occurrence of permanent hemianopia in true migraine.