SURGICAL TREATMENT OF ANEURYSM OF THE 
ANTERIOR CEREBRAL AND OF THE ANTERIOR 
COMMUNICATING ARTERIES DIAGNOSED BY 
ANGIOGRAPHY AND ELECTRO-
ENCEPHALOGRAPHY*

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A
LTHOUGH the subject of intracranial aneurysm has received too much attention in recent neurosurgical literature to consider it any longer “a lesion having such remote surgical bearings” (Cushing25), nevertheless it continues to pose many difficulties in diagnosis and treatment. Many of the diagnostic problems have been clarified by the stimulating clinical studies of Symonds,31 and of more recent investigators including Albright,1 Strauss, Globus and Ginsburg,49 Bramwell,2 McDonald and Korb,32 Richardson and Hyland,41 and Jefferson,22 and particularly by the development of intracerebral angiography by Moniz,35 Dott,8,18 Jefferson,23 Elvidge,11 Engeset,12 and others.

The problems related to the surgical therapy have been reviewed by Dott,9,10 Jefferson,24 Matas,24 Krayenbühl,26 Dandy,7 List and Hodges,30 Gross15 and Poppen,38 The role of carotid ligation in treatment has been discussed by Schorstein,47 Dandy,7 Olivecrona,36 Krayenbühl,27 Dott10 and Jefferson.24

Relatively few instances of successful surgical treatment of aneurysms located on the anterior cerebral or anterior communicating arteries have appeared in the literature. The present communication is concerned with 2 cases diagnosed by angiography and treated by direct surgical exposure. Follow-up now extends over 3 years.

REVIEW OF LITERATURE

Carotid Ligation. In the cases previously published, treatment has been carried out by carotid ligation or by direct exposure of the aneurysm or occasionally by a combination of these two methods. Most saccular aneu-
rysms for which carotid ligation has been performed have been situated on the carotid artery itself, but several authors mention anterior cerebral aneu-
rysms successfully treated by this method.

Krayenbühl28 has now ligatured either the common or internal carotid in 8 cases of bleeding anterior cerebral aneurysm. One patient recovered from a bitemporal hemianopsia caused by a large aneurysm of the anterior communicating artery.27 In another case of severe haemorrhage from an

aneurysm of the left anterior cerebral artery satisfactory recovery followed ligation of the left common and internal carotid arteries. Olivecrona reported 9 cases of carotid ligation for supraclinoid sacular aneurysm, and 2 of these, noted as anterior cerebral in location, were improved by carotid occlusion. In a patient who had had two attacks of haemorrhage, 2 weeks apart, with hemiparesis from an aneurysm at the junction of the right anterior cerebral and anterior communicating arteries, Walsh and Love ligated the right internal carotid artery. Except for two attacks of "syncope," neurological examination was reported to be normal at the end of 3 months.

Results in these cases suggest that carotid ligation may be effective in the prevention of recurrent bleeding from aneurysms not only of the carotid artery but also those of the main cerebral vessels. The local lesion, however, still remains and its behaviour over a long period is not predictable. A larger series of cases with longer periods of follow-up will be necessary before the value of this method can be properly assessed.

Intracranial Approach. The method of direct intracranial exposure, albeit hazardous, is more precise. Nine cases of anterior cerebral aneurysm treated successfully in this manner have been reported. In 2, the aneurysm was excised; in 1, trapped between clips, and in the remaining 6, pieces of muscle were placed around or inside the aneurysmal sac. The recoveries are perhaps the more remarkable considering that in only 2 cases was a definite diagnosis by angiography established, and, in addition, the sac ruptured during operative manipulation in 4 instances.

The earliest reported case (Dott) was that of a man aged 53 years with an aneurysm at the junction of the left anterior cerebral and the middle cerebral arteries. The sac ruptured during the operation. Haemorrhage was controlled by muscle tamponade. The patient was well 6 years following operation.

Tönnis demonstrated by arteriography an aneurysm of the anterior communicating artery. He exposed it by splitting the genu of the corpus callosum. Blood clot was removed and the sac surrounded with bits of muscle. The patient returned to work and was well a year later.

A leaking aneurysm of the right anterior cerebral artery in a boy aged 12 was removed by Cone in 1938 and reported by Russel. An encephalogram showed evidence of an expanding lesion in the right frontal region. An intracerebral haematoma was evacuated. This revealed an aneurysm, which was clipped and excised. The patient was in good health 7½ years later. This is the first recorded case of successful excision of an intracranial arterial aneurysm.

Dandy reported 2 successful results in 6 cases of anterior cerebral aneurysm. In his Case XXIV, a man of 49 years operated upon in 1941, the lesion, suspected of being a neoplasm, was a huge mass lying below and in front of the optic chiasm. It proved to be an aneurysm and was thought to arise from the anterior cerebral or the anterior communicating artery. The sac was opened, packed with muscle and cauterized. The left internal carotid artery
was partially occluded 7 days later with a band of fascia and totally occluded in another 10 days. The patient’s pre-operative aphasia, right motor weakness, loss of memory, personality changes and headaches disappeared.* He returned to work and was well 13 months after operation.

In Case XXV (Dandy⁶,⁷), a woman aged 45 years, clinical signs suggested the diagnosis of intracranial tumor. An aneurysm was disclosed arising from the anterior cerebral artery, and ruptured during manipulation. Haemorrhage was controlled by clipping the intracranial portion of the internal carotid artery. The aneurysm was excised and included resection of the upper half of the right optic nerve. The anterior portion of the softened left frontal lobe was removed at a later operation because of the development of drowsiness and postoperative seizures. The patient was well 13 months after operation. Klemme and Woolsey²⁵ described a case in which a hard non-pulsating mass under the left optic nerve yielded a gush of blood on curettage and a strip of muscle was inserted into the sac, which was considered to arise from the anterior cerebral artery. The patient recovered except for the pre-operative visual defect, and showed no evidence of recurrence for 4 months.

Jefferson²² mentions a case in which an anterior cerebral aneurysm was packed with muscle. Symptoms improved but fatal rupture occurred 18 months later. List and Hodges³⁰ successfully trapped by clips a small aneurysm of the left anterior cerebral artery. A case that might be mentioned is one of subchiasmal aneurysm which simulated clinically a pituitary tumour (McConnell¹³¹). The sac was opened and packed with muscle. The patient showed improvement of vision and remained without recurrence for several years.

**CASE REPORTS**

**Case 1. Neurosurg. No. 12809. Recurrent attacks of headache and vomiting for 2 years. Intracranial haemorrhage with sudden onset of severe headache, aphasia, stupor, and development of right hemiparesis. Aneurysm at junction of anterior communicating and left anterior cerebral arteries disclosed by thorotrast arteriography. Surgical exposure and clipping of aneurysm. Recovery. Follow-up for 3 ½ years.**

M.P., male, aged 20, was admitted to the Montreal Neurological Institute in a confused state on Sept. 24, 1945. For 2 years he had had attacks of severe frontal headache sometimes associated with vomiting. These recurred 5 times in a year and lasted some 12 hours; the patient would stop work, go home to a darkened room and sleep. He had been “nervous” since childhood, but during the last year he had been more restless and nervous. On the evening before admission his father found him in bed complaining of severe occipital headache. When his head was lifted from the pillow the patient screamed with pain. During the night he vomited, was incontinent and appeared confused and irritable. He had attempted to put on ski boots although it was much too early in the season. By the next morning he was

* In Table C (XXIV) it is stated that the partial ligation took place on the following day and complete ligation 7 days later. It also states that his condition was unchanged on discharge 3 weeks after 1st operation.
considerably worse, showed no response to questions and was in a condition of agitated stupor.

Examination. He was a youth of slight build who exhibited marked somnolence, but nevertheless was restless and agitated. There was evidence of meningeal irritation, bilateral Babinski signs and small pupils unreactive to light. He appeared to understand but gave no verbal response. Blood pressure was 110/40. CSF pressure was 350 mm.; the fluid was grossly bloody. There was little to suggest lateralization of the haemorrhage.

Over the next 2 weeks, in spite of clearing of the CSF, somnolence increased and was accompanied by a high fever, leucocyte count of 13,300/c.mm. and sedimentation rate of 46 at 1 hour, but no evidence of an infective process could be found clinically.

At the end of the 1st week a complete paralysis of the arm and weakness of the
leg on the right side developed. Right plantar was extensor; the left, equivocal. CSF, Wassermann and blood Kahn were negative. The prothrombin time was increased (62 sec.) and platelet counts reduced (180,000).

Electroencephalogram, Oct. 3, 1945, 4 days after onset of the right hemiparesis, showed large slow waves, fairly well restricted to the left frontotemporal region. A 2nd EEG, 2 weeks later, was similar. Rhythmic large slow waves from the left frontotemporal region at a rate coinciding with the pulse, probably represented a pulse artefact (Fig. 1, Pre-op.).

Angiography, Oct. 28, 1945. Thorotrast was injected into the exposed left common carotid artery in two successive amounts for lateral stereograms without changing syringe or needle and with the external carotid occluded by traction ligature.

The arteriogram showed a small aneurysmal dilatation 5 mm. in diameter which protruded (Fig. 2) from the superior surface of the anterior communicating artery.
The retention of thorotrast commonly seen within an aneurysm is shown on the phlebogram (Fig. 3) taken 4 sec. later.

Operation, Oct. 24, 1945. A left transfrontal osteoplastic craniotomy was made, the frontal lobe elevated, and the optic chiasm exposed. A loose ligature was placed around the left internal carotid artery intracranially as a safety measure. A lumbar puncture needle had been left in place in the spine in order to reduce brain volume during the procedure. The bifurcation of the internal carotid into the middle and anterior cerebral artery was identified. The junction of the anterior cerebral with the anterior communicating artery was visualized; the anterior was followed some distance as it passed forward to the right, and the anterior communicating artery was traced to approximately the point of its junction with the opposite right anterior cerebral. The middle cerebral artery was seen to be normal for a distance of 1–2 cm.

A small pea-sized yellowish aneurysm was discovered pointing posteriorly from

Fig. 3. Phlebogram film in Case 1, showing retention of thorotrast in the aneurysmal sac.
the anterior communicating artery near its junction with the left anterior cerebral artery, exactly as was depicted in the angiographic films. Adjacent there was a small black and yellow discoloured area with some pia-arachnoidal adhesions, the only trace of former rupture.

A tantalum clip was placed on the anterior cerebral artery proximal to its junction with the anterior communicating artery and two clips distal to it as the anterior cerebral swung across the inferior medial surface of the hemisphere. Tantalum clips were placed on either side of the sac on the anterior communicating artery and finally one clip was placed over the aneurysm itself, obliterating its neck. Altogether six tantalum clips were used in trapping the aneurysm (Fig. 4). Lack of evidence of local haemorrhage is not surprising in view of the complete disappearance of blood from the CSF specimens 2 weeks before operation. No bleeding occurred from the

FIG. 4. Postoperative roentgenograms show position of tantalum clips, Case 1.

aneurysm during manipulation so that it was fortunately not necessary to use the ligature around the carotid in the middle fossa.

Postoperative Notes. His course was one of progressive improvement. He spoke for the first time since his hospital admission on the 2nd postoperative day and was talking freely 2 weeks later. He had occasional incontinence for 3 weeks. In the EEG 13 days after operation (Fig. 1) the amount of slow wave discharge from the left frontotemporal region was somewhat less and there was absence of the “pulsation artefact” previously recorded. He left hospital 25 days after operation.

Subsequent Course. On Feb. 12, 1946, 3½ months postoperatively, the patient had no headache or dizziness. He could count rapidly in French and in English. Sense of smell was absent. His right leg appeared slightly stiff when he walked.

On June 4, 1946, there was merely a residual slight weakness of the right foot. Nervousness and fatigue were complained of and the patient did not go out due to lack of confidence in walking.

The EEG on July 16, 1946 (Fig. 1) demonstrated localized slow and sharp wave disturbance of moderate degree of severity, maximal in the left anterofrontal region, and in lesser amount from the left temporal region and from the homologous right side. The background activity from the parieto-occipital region was of good
amplitude and continuity, and fairly well regulated at about 10/sec. The persistence of these abnormalities was not surprising when one considers that the left anterior cerebral artery was occluded at operation. Neither the clinical signs nor the EEG were as severe as might have been expected in view of the serious consequences of this procedure that have been discussed (Dandy,7 Poppen).7

On Aug. 6, 1946, the patient still exhibited slight stiffness of the right leg. He lacked initiative, would not go out, apparently as a result of fear, and did not associate with any of his friends. On Sept. 30, 1946, he stated that he could not go back to work because he could not write well. He meant that he could not write as fast as before. Actually he could write very well.

The EEG on April 11, 1947 demonstrated a persistent area of very slow delta activity localized to the pole of the left frontal lobe. The delta activity was not widespread as previously, which suggested progressive improvement, but indicated a residual area of damaged cerebral tissue in the anterior pole of the left frontal lobe.

When tested in April 1947 sense of smell was intact. On May 3 the patient had an epileptic seizure with loss of consciousness and clonic movements in the extremities while asleep. This attack was followed by vomiting. When seen a month later and again in October 1947, he was still fearful of going out alone. His right hand was objectively normal but he still complained that he could not write as fast as before his illness. He walked well but his toe still rotated inward.

An EEG on Oct. 31, 1947 showed no further change and in particular no spike activity. An EEG on Feb. 5, 1948 showed slight improvement with residual slow wave focus in the left frontal pole. On July 20, 1948 EEG showed well regulated alpha rhythm with slow waves present but not prominent. Sharp waves in the left frontotemporal region suggested the presence of a potentially epileptogenic lesion.

When last seen in April 1949 the patient was very well. He exhibited the slightest residual internal rotation of the foot on walking. He was willing to work if he could be given a position without too much writing. His original occupation called for a great deal of close application.

Case 2. Neurosurg. No. 4938 and 6371. Sudden collapse from subarachnoid and intracerebral haemorrhage, localized clinically to left frontal region. Improvement. Discharge from hospital 1 month later with recurrence the same day and readmission. Diodrast angiography demonstrating aneurysm of right anterior cerebral artery. Excision of aneurysm and recovery. Clinical and EEG follow-up for 3 years.

History. R.O., aged 13, stated that he was struck on the head by a hockey stick, on April 8, 1946. He was taken to a local hospital where a lumbar puncture showed blood in the CSF. He became drowsy, irritable and incontinent.

Examination. On admission to the Montreal Neurological Institute 2 days later, patient exhibited stiffness of the neck, positive Kernig and positive Babinski sign, more complete on the right. The right pupil was larger than the left and there was paralysis of the left abducens nerve. Blood pressure was 130/90; pulse rate 60; temperature 100.8°F. Lumbar puncture showed grossly bloody fluid under normal pressure. The following morning weakness of the right leg developed, more noticeable in the distal groups of muscles, with a definite plantar extension on the right and questionable extension on the left. Pupils were normal but the left 6th nerve paralysis remained. The fundi showed fulness of the veins but no elevation of the discs. X-ray films of the skull were normal. Further lumbar punctures showed normal pressure and yielded yellow CSF.
EEG, recorded 2 days after onset of illness, showed severe generalized disturbance with slow waves and absence of alpha rhythm, but no evidence of a focal lesion (Fig. 5, Pre-op. 1).

Pneumoencephalogram on April 20 showed relative absence of subarachnoid markings over the frontal poles, especially on the left; dilated lateral and 3rd ventricles, and evidence that was interpreted as indicating that a subarachnoid haemorrhage in the lower left frontal region had communicated with the anterior horn of the left lateral ventricle (Fig. 6). An EEG repeated on April 30 (Fig. 5, Pre-op. 2), indicated improvement by an increase of wave frequency, but there remained severe delta wave disturbance, most marked in the right temporoparietal region and suggesting a deep-seated lesion. Normal alpha rhythm was not observed.

The boy showed progressive improvement with almost complete recovery except for the left abducens paralysis and at the end of 4 weeks was discharged from hosp-
tal with the diagnosis of subarachnoid haemorrhage probably due to ruptured intracranial aneurysm.

Readmission. On the day of his arrival home, May 6, 1946, he suffered a severe pain in the head while straining at stool and his right leg and arm started to shake. He was readmitted to hospital where he appeared drowsy and complained of severe headache. There was bilateral plantar extension. Pupils were sluggishly reactive to light and disc margins were hazy. The left 6th nerve paresis was still present and lumbar puncture gave bloody fluid. During the next 2 weeks he complained of headache and dizziness and moderate papilloedema developed. The Babinski sign on the left disappeared, the right persisted.

Angiogram, May 23, 1946. It may be recalled that the clinical findings and pneumogram indicated that the lesion was on the left while the EEG showed it to be on the right. On the left side the injection was made into the common carotid artery with the external carotid temporarily occluded. On the right side the external carotid was not occluded during the procedure. The radiopaque substance used was a 46 per cent solution of diodrast with a total dose of approximately 24 cc. for each side given in 2 amounts for stereoscopic purposes. During the injections the patient had momentary stimulation of respiration in spite of sedation. He was fortunately under rectal sodium pentothal anaesthesia, otherwise it is believed that he would have had a convulsion, which would be especially serious in this type of case. The angiogram on the right side demonstrated an aneurysm, about 1 cm. in diameter, communicating with the right anterior cerebral artery by means of at least two smaller branches (Fig. 7). The anterior cerebral arteries were stretched into a more open bow by the enlargement of the ventricles and the region of distribution was perhaps less well supplied with smaller branches. The aneurysmal sac retained the contrast medium (Fig. 8) for at least 7 sec. This aneurysm had apparently ruptured into the left frontal lobe and part one of the left lateral ventricle.

Operation. May 23, 1946. Right transfrontal craniotomy. The medial surfaces of the frontal lobes were adherent and yellowish fluid gushed up several times on elevat-
ing the right frontal pole. The ventricles were collapsed by ventricular puncture and by lumbar drainage of CSF. Following this the right frontal lobe was gradually separated from the falx and from the left frontal lobe, exposing at the base the rounded bluish aneurysm (Fig. 9).

**Fig. 7.** First film of arteriogram in Case 2 with diodrast 46 per cent, showing aneurysm of right anterior cerebral artery. Note small vessels running to the aneurysm.

**Fig. 8.** Phlebogram film in Case 2, showing retention of diodrast in aneurysmal sac.
Four small feeder vessels were seen running to the sac as depicted in the angiogram, but only one of these appeared to come from the right side. They were divided in turn between tantalum clips, permitting the aneurysm to be lifted up gently, exposing a rather large vessel running beneath and medially. After these manipulations and when we were prepared to make the final delivery, the friable sac blew out unexpectedly with violent haemorrhage which was not satisfactorily controlled until three clips had been placed on a large vessel identified in a calmer moment as the right anterior cerebral artery. The sac was then excised and a dry operative field obtained before closure. The aneurysm had lain immediately above the optic chiasm. At the end of the procedure the optic nerve was seen to be undamaged. Postoperative x-rays show the position of the tantalum clips (Fig. 10.)
Adhesions had prevented the safe placement of a loose ligature about the internal carotid artery within the skull and the ingenious method by Hamby for occluding the carotid circulation in the neck from the cranial operative field was unsatisfactory in our hands for controlling the haemorrhage.

**Pathological Note.** The aneurysmal sac measured 1.5 by 1.2 cm. (Fig. 11A and B). The laminated wall varied from 0.5 to 3 mm. in thickness and was made up of friable, poorly organized thrombus (Fig. 11C). No elastic or muscle fibres were seen except in remnants of vessel wall stretched onto the outer surface of the sac. The only supporting tissue present was occasional collagen. The small arterial vessels penetrated the outer layer of the wall as noted at operation and in the angiogram.

This arrangement supports the theory of Padget regarding the origin of congenital aneurysms from unresorbed embryonal vessels.

**Postoperative Notes.** For several weeks he showed lack of memory for recent daily happenings, but was bright and cheerful (Fig. 12). Incontinence cleared up when he began to get up and about the ward. In the EEG of June 20, 1946 (Fig. 5), 1 month after operation, there was an improvement of background activity but delta abnormality remained in considerable amount and was maximal in the right temporoparietal region. Lumbar puncture, June 19, 1946, showed clear fluid under normal pressure. On the day of discharge, June 28, neurological findings were essentially normal except for a slight left internal strabismus and an unsteadiness of gait apparently due to the rather long stay in bed.
Subsequent Course. The EEG of Aug. 5, 1946, 2½ months after operation (Fig. 5) continued to show generalized dysrhythmia with the greatest disturbance from the right hemisphere, with slow waves more localized to the frontotemporal region on the right but giving, on the whole, a record greatly improved as compared to that taken 1 month after operation. Neurological findings at this time were normal.

He showed progressive improvement and was in good physical health 11 months later. In the interval he returned to school and had been allowed to enter Grade 8 with his associates in September 1946, 4½ months after operation. His marks were generally poor except for passes in French and Science, and he failed his year.

He regularly defeated his parents at checkers and liked to play cards and chess. He was socially popular and happy but avoided athletics. Teachers were satisfied with his behaviour. When seen in 1947 the patient had continued to attend school.

His mental status was tested in some detail on several occasions by Dr. B. Silverman of the Mental Hygiene Institute. The boy's chief defect was poor memory and lack of initiative indicated by his failure to take interest in some former hobbies. He obtained poor results in tests designed to evaluate visual motor organization and new learning, but the results showed continual improvement, those of March 1947 being considerably better than those of September 1946.

The EEG of Oct. 25, 1947, showed a decrease of slow wave activity with an increase of rapid, more normal activity. Depression of alpha waves from the right occipital region as noted in a previous test was not seen. Residual general dysrhythmia was greater from right hemisphere, but with definite improvement.

When last heard from on June 29, 1949, it was stated that the patient had been generally backward at school. He had failed to pass from first year high school. He had been somewhat forgetful. On the other hand his general behaviour had been satisfactory and he was growing well. He had played baseball but cannot run fast. He rides a bicycle and goes swimming with other boys. He will probably be sent to a trade school. The EEG of July 6, 1949 suggested unresolved cerebral disturbance most marked in the right hemisphere.
Angiography. For vascular lesions of the brain especially we have preferred the method of surgical exposure, because of its reliability and safety. Moreover, ligations when necessary can be done simultaneously or through the same wound, and the radiopaque material can be directed to the external or internal division, or both at will.

There are no immediate untoward reactions to the injections of the carotid vessels with thorotrast, though one is naturally reluctant to use a radioactive substance. We never use more than 32 to 36 cc. in a selected case for bilateral injections, and generally never more than 24 cc. If the material should escape into the vascular sheath due to faulty injection, late scarring can be expected. With proper exposure and practice this should never occur.

Diodrast can be made as radiopaque as thorotrast but concentrations between 40 and 50 per cent must be used. We have used it and similar substances spasmodically since 1937. A concentration of approximately 45 per cent gave satisfactory visualization in Case 2 (Fig. 7). By percutaneous method we have been using 35 per cent diodrast which gives poor though adequate visualization but is still, though less, stimulating. Respiration may be accelerated and unless the patient is sedated twitchings or an epileptic seizure may result. Nevertheless it is a satisfaction to know that the material is rapidly excreted, so that repeat injections may be given if necessary. While rapid excretion of diodrast enables repeat injections to be made with some impunity, the potential danger of the medium cannot be discounted. It is felt that these iodine preparations may to some extent permeate through the vessel wall and to give rise to epileptic manifestations or in any event give rise to vascular spasm which may vary from case to case. One death is known to have occurred from the injection of this material and cases of hemiplegia following its injection are known to occur. Postmortem examination revealed a massive cerebral infarction. Why no organic chemist has linked the iodine atom to a more suitable molecule is difficult to understand.

Angiography makes possible an appropriately planned surgical attack on intracranial vascular lesions and in particular on saccular aneurysms. By this method the precise nature, localization, size and communications of the lesion may be determined. Pneumography, while of considerable value in the diagnostic work-up may, as in our Case 2 and in other published reports (Dandy,7 Russel,45), actually indicate the wrong side by giving the localization of the retained haemorrhage, which may extend for some distance from the actual source of bleeding. Furthermore, as in Case 2, the clinical signs, such as ocular nerve palsy (Walsh and Love53), or even unilateral blindness (Jefferson22) may also point to the wrong side.

Electroencephalography. The EEG was correct as to the side of the lesion in both of our cases. The clinical picture and the electrographic disturbance are generally greater than one would expect from the amount of haemorrhage and brain damage disclosed at operation, and this suggests that the aneu-
Aneurysm is capable of causing changes in the normal circulatory equilibrium with a resulting ischaemia of the cerebral tissue lying beyond the aneurysm. Such effects of intracranial aneurysm on the intracerebral circulation require further study. Woodhall (mentioned by Dandy) has previously noted 3 cases of aneurysm in which the side of the haemorrhage was indicated by EEG, and he assumed that the changes were due to a reduced blood supply to the brain.

Although the changes in the EEG record are not necessarily specific, they are similar in character and sequence to those described following experimental ischaemia due to the occlusion of the main arterial supply to the brain and to disturbance of the cerebral circulation after simple exposure of the surface of the brain.

In following the postoperative progress the EEG, a sensitive indicator of cerebral damage, continued to show disturbance at a stage when clinical examination revealed little abnormality, but when mental tests brought out deficiencies which were improving at a slower rate than the more gross clinical signs. The long period of recovery as indicated by the EEG is comparable to the results obtained in the study of head injuries (Jasper, Kershman and Elvidge), where dysrhythmias may persist after neurological signs have vanished.

**Surgical Treatment.** The various methods that have been used for the treatment of intracranial aneurysms have been recently reviewed by Dandy, Dott and Jefferson. In both cases reported here, tantalum clips were used to isolate the aneurysm because of the small size of the sac and its disposition as a circumscribed dilatation of the arterial wall. Six clips were placed at strategic points. It seems unlikely that they could be forced open by the pulsation of blood. In Case 2 the aneurysm was excised in toto, and it is quite likely that, unless an attempt had been made to remove the sac completely, the final large vessel would not have been disclosed and would have remained as a source of recurrent bleeding.

**The Use of Muscle.** One of the earliest records of the use of muscle in the treatment of aneurysm is that of a case of a lesion of the internal carotid reported from Cushing’s clinic by Sosman and Vogt, where the sac was opened, packed with muscle and sewn up. The fact that muscle was employed successfully in 6 of the cases previously published indicates its value. Nevertheless several unsuccessful experiences with muscle have been noted. Jefferson mentions an instance in which muscle was packed about an aneurysm but recurrent fatal haemorrhage took place 18 months later. At autopsy the aneurysm appeared completely thrombosed except on its upper surface where it had ruptured. In 3 similar cases Jefferson stated that 2 patients survived 4 and 5 years respectively, but in 1 of these the common carotid was tied 2 months later because of continuing pain which is “always a sign that the aneurysm is not securely thrombosed”. In the 3rd, muscle was packed also around the aneurysm but fatal haemorrhage took place 1 month later.
This method, then, has not been entirely reliable and its value must depend among other things upon the structure and disposition of the aneurysm itself. The absence of muscle and elastic tissue from aneurysmal sacs is a common observation (Forbus, Hermann and MacGregor). Certainly in our Case 2 reconsideration of the histology of the aneurysm (Fig. 11) throws doubt on the value of bolstering a thrombotic sac with muscle. Moreover, the pressure of arterial blood coursing through the aneurysm must approach the limits of pressure (60 to 80 mm. Hg.) that freshly placed muscle stamps can resist (Horsley).

Rupture of the Sac. Rupture of the aneurysm during operative manipulation is not uncommon, having occurred in 4 of the cases reviewed above, in our Case 2 and in instances of aneurysms located on other intracranial arteries. It is therefore worthwhile to plan some means of dealing with this eventuality. This may be done most readily by carotid occlusion, either by ligation in the neck, preliminary to direct intracranial approach, or by clipping the artery intracranially, or by placing a loose ligature about the artery in the middle fossa before attempting to deal with the aneurysm. We have since noted that this latter method was used effectively in an early case reported from Cushing’s clinic. The authors prefer now to place a loose ligature round the artery in the neck which can be occluded by an assistant. As already stated, the ingenious method proposed by Hamby for occluding the carotid circulation in the neck from the cranial operative field was attempted in our Case 2, but for technical reasons did not control the haemorrhage.

If permanent closure of the common or internal carotid artery is contemplated, some idea of the patient’s tolerance to the procedure should be gained before operation. The percutaneous compression test of Matas has been found reliable by some but has proved inconsistent in the experience of others. The present authors prefer to ligate the vessels in two stages under local anaesthetic. Angiography according to some has been of value in determining to what degree the opposite carotid is capable of contributing to the blood supply of the hemisphere on the ligatured side. This can be done by compressing the common carotid on the side of the lesion while injecting the contrast medium into the carotid on the opposite side (Dott), a technique similar to that used by Kristiansen to demonstrate simultaneously both middle cerebral arteries for the diagnosis of temporal lobe vascular lesions. An additional test procedure has been the investigation of the effects of temporary carotid occlusion on EEG records, as described by Rogers, Chusid and Mahoney, and in unpublished cases by ourselves. Although no case with serious complications after carotid ligation has as yet been available for EEG study, this method appeared to be a most promising one, particularly when its results could be combined with the information derived from angiography.*

* In a recent case hemiplegia developed 10 hours after 2nd stage ligation of the common carotid (external had been ligated at the 1st stage with partial ligation of common). This was not predicted by EEG studies. A further observation has now been made of a relatively normal EEG recorded during the development of a hemiplegia from carotid ligation.
We have recorded brain waves during selective closure of the internal carotid system, but our experience is not yet sufficiently clear to indicate that it is a reliable test.

SUMMARY

1. The literature of the surgical treatment of anterior cerebral aneurysms is reviewed and 2 new cases are reported in which the aneurysm was demonstrated by stereoscopic angiography and treated by direct operative approach, in one case by trapping and clipping an aneurysm of the anterior communicating artery and in the other by clipping and excision of an aneurysm of the right anterior cerebral artery.

2. The value of angiography, not only in confirming the diagnosis of intracranial aneurysm, but also in revealing information upon which to base a planned surgical treatment of the lesion is re-emphasized.

3. EEG was of aid in the diagnosis and follow-up in both cases and indicated residual cerebral damage even after 3 years.

4. Both patients showed satisfactory recovery although clips were placed in the one case on the left, and in the other on the right anterior cerebral artery.

5. Both patients showed clinical pictures, the severity of which was out of proportion to the amount of haemorrhage or cerebral damage disclosed at operation, and it is suggested that this was partly due to the aneurysm producing a mechanical disturbance in the blood supply to the cerebral tissue.

6. Some advantages of excision of the aneurysm as opposed to other methods of local treatment are pointed out.

7. From a consideration of the histology of the aneurysm in our Case 2 and of reports in the literature it is suggested that the use of muscle to bolster up an aneurysmal sac may not be reliable.

8. It is pointed out that rupture of an intracranial aneurysm occurs so frequently during operative manipulation that some method of dealing with this eventuality should be planned beforehand. We suggest further trials of the method of placing round the internal carotid artery intracranially a loose ligature which can be used in emergency. A loose ligature round the carotid artery in the neck under the control of an assistant probably offers the safest method.

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