EXPERIENCES IN THE TREATMENT OF RUPTURED INTRACRANIAL ANEURYSMS

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Since the inauguration of the neurosurgical department six and a half years ago, 162 cases of verified spontaneous subarachnoid haemorrhage have been admitted to the Royal Victoria Hospital and surgical aid was sought in 117 of these patients. Our material does not include any infraclinoid aneurysms but relates only to those saccular aneurysms which, situated intradurally, have ruptured into the subarachnoid pathways or cerebral substance, or, in a few cases, into the subdural space.

Angiography had not been done or had failed on 13 of the 117 cases. In 26 instances in which it was performed successfully, no aneurysm was revealed but it is probable, even when bilateral carotid angiography proved to be negative, that some aneurysms arising from the vertebral system escaped detection, as this approach was only employed in seven of these cases. Only one example was found of a saccular aneurysm of the vertebro-basilar system; it was on the posterior cerebral artery at the junction with the posterior communicating artery and was not operated on.

In 16 patients in whom an aneurysm had been demonstrated by angiography, operation was not undertaken for various reasons. Thus, refusal to have any further treatment accounted for five cases. In five others it was because they were critically ill at the time of angiography, death taking place in four cases as a direct result of the procedure, and in the fifth case the aneurysm rupturing fatally a few hours later. Of the remaining six patients in whom no operation was performed, two were in coma and hemiplegic, and as such considered too ill, but four made a good initial recovery. Two of these, one with a middle cerebral and the other with an internal carotid aneurysm, left hospital in good health. Although one died two months later of further

haemorrhage, the other has continued in good health up to the present time, nearly six years later. The third patient had a severe hemiplegia and gross intellectual deterioration, for which reason operation was not recommended. The remaining patient, whose aneurysm was on the upper limb of the carotid syphon, was hypertensive and reacted severely each time carotid compression was tried. It was considered inadvisable to proceed further with ligation of vessels in the neck.

Ruptured Intracranial Aneurysm Treated by Operation

It has been our usual practice to employ ligation of the carotid vessels in the neck for aneurysms on the supraclinoid portion of the internal carotid, and to have recourse to an intracranial procedure for those situated on the anterior part of the circle of Willis and its branches, and for those on the middle cerebral artery. This policy was adopted partly because of the risks attending carotid ligation, but also on the assumption that although such occlusions would result in permanent reduction in intra-arterial pressure and expansile thrust of the blood as high as the internal carotid bifurcation they would not be likely to have more than a temporary effect on pressures in the circle of Willis and its branches. However, the work of Bakay and Sweet (1953) and others in regard to direct measurement of intra-arterial pressure distal to the point of occlusion of the carotid vessels in the neck appears to indicate that our reasoning in the latter respect may have been incorrect.

Operative treatment was undertaken in 62 cases, and of these 13 died, a death rate of 21% (Table I). Four of the patients who did not survive were deeply comatose when first seen and interference was limited to evacuation of large haematomata. There can be little doubt that they had already suffered overwhelming cerebral damage. Another patient, who had been admitted in coma after a third lumbar puncture, had a subtemporal decompression with

* Prepared by Dr. R. S. Allison (Belfast) and Dr. John F. Mullen (Chicago) from notes used by the late Cecil Calvert for a paper which he delivered to the Society of British Neurological Surgeons in 1954 at Belfast. Unfortunately it has not been possible to trace any notes referring to the detailed interpretation of Tables I, II, III, and IV which Mr. Calvert gave during the course of his address.

exploration for clot a few hours afterwards, but died the same day. Post-mortem examination revealed necrosis of the tips of both cerebellar tonsils.

Fifty-seven patients each had a definitive surgical procedure and amongst these there were eight deaths—a mortality rate of 14%. Two died following carotid ligation for an aneurysm on the upper limb of the carotid syphon; one of them had made a good recovery from the immediate effects of the subarachnoid haemorrhage, but, although carotid occlusion was employed with all the usual safeguards, he passed into coma 12 hours after operation and died the same day. The other case was a woman aged 42, who had been admitted shortly after a third haemorrhage which had occurred on the fourteenth day of her illness. She was much confused and drowsy and had a complete third nerve palsy with meningeal signs. Angiography had to be deferred twice during the succeeding 10 days on account of two further haemorrhages. Eventually, although she was still acutely ill, contrast injections were made and on the same day the lumen of the common carotid artery was reduced to about a half. This procedure was well tolerated and three days later occlusion of the common carotid was completed. But after a lapse of a further 24 hours she quickly became comatose and died within six hours, despite removal of the ligature. As necropsy was not permitted, I do not know whether the fatal issue was determined by cerebral ischaemia or further haemorrhage.

In the other six patients who died after operation, a direct attack was made on the aneurysm. Three were very ill at the time. One, an elderly doctor with an anterior communicating aneurysm, had had an initial haemorrhage of moderate severity and three subsequent severe haemorrhages at intervals of four or five days, each one resulting in coma. He rallied temporarily after evacuation of clot, first in one and then in the other frontal lobe. But directly after the fourth relapse, in desperation, we turned a bifrontal flap and evacuated large recurrent clots from both frontal lobes. The aneurysm was then isolated, wrapped in muscle and a ligature tied around its neck. He died five days later and was found to have extensive oedema of both frontal lobes. The second case had severe subarachnoid bleeding from a middle cerebral aneurysm and there was a large intracerebral temporal haematoma. An anaesthetic was not required nor did the vein bleed when an intravenous drip was set up. A bone flap was reflected, the clot evacuated and the aneurysm wrapped in wool. For the first two days after operation it looked as if the patient might pull through, but coma then supervened fairly quickly and she died three days later. At necropsy it was

found that there had been no recurrence of bleeding, but the middle cerebral artery was completely thrombosed. The third case was that of a woman operated upon (while in coma with a flaccid hemiplegia) 18 hours after a second haemorrhage on the sixth day after the initial rupture. Intracranial tension was marked but relieved by evacuation of a large clot in the frontal lobe, so that the aneurysm was isolated without great difficulty. The sac was adherent to the opposite anterior cerebral artery, and as it was not possible to isolate the parent artery proximal to the aneurysm, the sac was enclosed in muslin net and fibrin foam. Forty-eight hours later the patient had much improved, was moving all limbs and able to answer simple questions; but then she fairly quickly relapsed and died two days later. Necropsy revealed fresh clot in the cavity in the frontal lobe, which communicated with the ventricle.

Of the remaining three patients who died after a definitive operation, all were deteriorating following a second haemorrhage, and operation was undertaken earlier than one would have wished because it was felt that further delay might be hazardous. One patient had a middle cerebral aneurysm with a small haematoma, which was evacuated, situated far medially in the upper part of the frontal lobe. Although careful search was made for the aneurysm, it was not found. The contrast shadow in the angiograms had not been entirely convincing, and the wound was closed in the belief that there was probably an aneurysm of the anterior communicating artery. Death occurred two days later without consciousness being regained. Necropsy showed a small aneurysm on the middle cerebral artery which had bled again post-operatively. The second case was also one of a middle cerebral aneurysm; two clips had first been applied to obliterate the sac, but when a third clip was tightened on the neck, it unfortunately tore off close to the parent artery. Bleeding was controlled by application of muscle to the artery and after a delay of 20 minutes, during which the field remained dry, the wound was closed. However, sudden torrential bleeding recurred three hours post-operatively and led to death in about 10 minutes. The third case was a woman of 22 with an aneurysm extending upwards from the internal carotid bifurcation. Operation was undertaken five days after a second haemorrhage, when the brain was still rather tense and exposure difficult. The sac unfortunately ruptured just as it was coming into view, and bleeding was so profuse that the blood pressure became unrecordable within a few minutes. Haemorrhage having by then considerably lessened, it was possible to apply several clips so as to obliterate the aneurysm. There was still some oozing from the point of confluence of the aneurysm and the

anterior cerebral artery, and a clip was placed on the latter completely controlling the bleeding. Post-operatively the circulatory condition improved but consciousness was never regained; there was a contralateral flaccid hemiplegia and she died five days later in spite of bilateral subtemporal decompression. Necropsy showed that there had been no recurrence of bleeding and death was presumed due to ischaemia from occlusion of Heubner's artery.

To summarize results, of the 26 cases of supraclinoid internal carotid aneurysm treated by ligation in the neck, there were as described two deaths. Four of the remaining 24 cases developed hemiplegia after operation from which they made a good recovery. A number of other patients had various neurological defects before operation, such as partial or complete third nerve palsy, hemiparesis, impaired vision from subhyaloid haemorrhage, or optic atrophy. In all instances in which there was hemiparesis, recovery of function ensued, but three patients remain blind in one eye and another is nearly so. In five there is still residual weakness of the oculomotor mechanism. One patient died a year after carotid ligation from cancer of the lung. The patients with oculomotor and visual defects are still in good health and doing full work, except for one middle-aged woman with

arterial hypertension. Twenty of the patients who were operated upon have no residual disability, and in no case surviving carotid ligation has there been recurrent rupture of the aneurysm.

Amongst the 31 patients in whom a direct attack was made on the aneurysm, there were six deaths. Of the latter, as recounted above, three were very ill, but the other three, although with raised intracranial tension, were no more than drowsy, but nevertheless deteriorating at the time of operation. These patients might have recovered if rupture of the aneurysm could have been avoided at the time it was being dealt with, and in the third, if the lesion had not eluded detection. In two of the very ill patients, death was probably inevitable, but in the third, the fatality must be explained by the fact that wrapping the sac with muslin net and fibrin foam did not give adequate protection. Of the 25 patients who survived direct treatment to the aneurysms, all are still alive—at varying periods from a few months up to six years later. A little over a third of these had more or less severe dysphasia and/or hemiparesis pre-operatively, but in all except two, hemiplegia has cleared. The exceptions are still severely handicapped by dysfunction of the paretic arm. Dysphasic disabilities have proved more

TABLE I
62 CASES OF RUPTURED INTRACRANIAL ANEURYSMS TREATED BY OPERATION

Site of Aneurysm	No.* of Cases	Post- Operative	Deaths		Alive Six Months to Five Years Later		
			Later from		No Disability	Slight Disability	Light Work Only
			Fresh Rupture	Other Cause			
Supraclinoid internal carotid	30	3 (10%)	—	1	20	5	1
Middle cerebral	16	6 (37%)	—	—	7	3	—
Anterior communicating	11	2 (18%)	—	—	7	1	1
Anterior cerebral	3	1 (33%)	—	—	1	—	1
Bifurcation internal carotid	2	1 (50%)	—	—	—	—	1
Total	62	13 (21%)	—	1	35	9	4

* 9 cases in coma.

TABLE II
30 OPERATIVE CASES OF SUPRACLINOID ANEURYSM

Procedure	No. of Cases	Time of Operation			Died		Post- Operative Complications	Alive Months-Years	
		1st Week	2nd Week	Later	Post- Oper- atively	Later		No Disability	Neural Defect
Exploration for clot	1	1	—	—	1	—	—	—	—
Direct attack, sac wrapped in muscle	3	—	—	3	—	—	—	2	1
Ligation of common carotid	8	3	2	3	1	—	2*	7	—
Ligation of internal carotid	6	—	1	5	1	—	—	5	—
Comm. followed by internal ligation	12	—	2	10	—	1 (cancer)	2*	6	5**
Total	30	4	5	21	3	1	4	20	6

No operation—4 cases:

- 1 refused operation.
- 1 compression not tolerated.
- 1 comatose.
- 1 operation not advised.

* Temporary hemiplegia.

** Partial III Rd.

TABLE III
24 CASES OF MIDDLE CEREBRAL ANEURYSM
(16 cases operated on and 8 cases unoperated)

6 Died		2 Alive	
While in Hospital	From Later Recurrence	Well	With Neural Defects
4 Soon after angiography	2	1 (8 months)	1 Poor insight (9 months)

OPERATION RESULTS IN 16 CASES

Procedure	No. of Cases	Pre-Operative Condition	Time of Operation			Died	Alive	
			1st Week	2nd Week	Later		Well	Neural Defect
Evacuation of clot only	4	4 in coma	4	—	—	4	—	—
Evacuation of clot, wrapping with muscle	3	1 in coma, 2 Hemiplegic	1	—	2	1	—	2 Dysphasia
Neck of sac clipped	2	1 Hemiplegic	1	—	1	1	1	—
Wrapping with muscle and/or muslin	7	7 Hemiparetic or Dysphasic	—	2	5	—	6	1 Dysphasia
Definitive operations	12	—	2	2	8	2	7	3

TABLE IV
11 OPERATIVE CASES OF ANTERIOR COMMUNICATING—A.I. ANEURYSMS

Procedure	No. of Cases	Time of Operation			Died	Alive	
		Within 4 Days	Within 3 Weeks	Later		No Disability	Neural Defect
Anterior cerebral—A.I. clipped	9	1 (in coma)	2	5	1	4, 3-5 yr. 1, 1 yr. 1, recent	1, impaired vision 3 yr. 1, weak R. leg
Wrapped in muscle and pericranium	1	—	—	1	—	1, 1½ yr.	—
Ligation neck of sac	1	1 (in coma)	—	—	1	—	—
Total	11	2	2	6	2	7	2

No operation (3)—2 refused ; 1 in coma.

refractory, and although all have much less disturbance of speech than they had, two men doing the major part of their farm work have still fairly severe dyslexia. Of nine patients who had the first part of the anterior cerebral artery clipped for aneurysms on the anterior communicating and second parts of the anterior cerebral artery respectively, one has a minimal weakness of the opposite foot and the other, a hypertensive man, aged 53, is unable to work because of intellectual deterioration. Altogether 17 of the 31 patients in whom the aneurysm was dealt with directly have no residual disability and are at their ordinary work. The results are summarized in Tables I, II, III, and IV.

Discussion

In a patient acutely ill after initial rupture of an aneurysm the first problem to decide is how soon operative measures can be instituted with the object of protecting him against a second haemorrhage. When death results in the first 48 hours, the initial haemorrhage has usually been of such magnitude that neither medical nor surgical measures are of any avail. In such severe cases surviving the first

two or three days, the cerebral circulation is only precariously balanced against the high intracranial tension. To reduce the blood flow to one hemisphere by carotid ligation is obviously unwise because the systemic blood pressure, elevated at the insistence of the cardiovascular centre, is scarcely more than sufficient to maintain adequate oxygenation of the brain. Equally unwise is it to inflict further trauma on an already dangerously swollen brain by forcing access to the aneurysm, the rent in which is insecurely plugged by fibrinous clot. This is usually only too readily dislodged and such an event will almost certainly determine a fatality in critically ill patients. There is thus an interim period following the initial rupture, varying in length from case to case, during which intracranial mechanisms are gradually recovering and when a watchful conservation is the wisest course.

As soon as vital processes show signs of stabilizing, an electro-encephalogram should be done, for not only are intracerebral clots lateralized thereby, but the side of the aneurysm may be indicated when neither history nor clinical findings afford a clue. In order to apply operative measures logically when

the opportune moment has arrived, it is necessary to have precise information about the aneurysm itself, and as to whether there is a complicating clot or some anomaly of the circle of Willis. This information can be furnished only by angiography, but we do not employ the investigation until severe signs of generalized cerebral dysfunction are on the wane, for as already mentioned we have had four deaths directly attributable to diodone injections in critically ill patients.

In some severely ill patients improvement occurs up to a certain stage but is then not further maintained, slow retrogression setting in. In some such cases intracerebral clot is chiefly responsible for raised intracranial tension and careful evacuation of the haematoma through a large burr hole by means of a sucker is of value. In other cases, such as a middle cerebral aneurysm complicated by a large temporal lobe clot, reflection of a small lateral bone flap and evacuation of extravasated blood may even facilitate direct treatment of the aneurysm itself.

When signs indicate that the cerebral circulation is no longer labouring under duress, when, for example, the systemic blood pressure has returned to normal for at least a few days and clouding of consciousness has considerably diminished, then the decision has to be made whether carotid occlusion or direct attack on the aneurysm is the safer course. There is also the question as to which of these procedures may be applied earlier with reasonable safety—an important matter regarding which there is little information available in the literature. There are so many variables to be considered that decision is very difficult. For example, one must evaluate the site of the lesion and which is its parent artery, the possibility of complicating clot or some anomaly of the circle of Willis, whether the patient is still acutely ill with swollen brain and parlous cerebral circulation or has sufficiently recovered from the immediate effects of the haemorrhage so as to make operation feasible. Not the least of the many difficulties is lack of precise knowledge as to the permanence of the protection afforded either by occlusion of the carotid arteries in the neck or by the various local devices that may be employed in dealing with the sac. My own limited experience would not warrant an attempt to formulate guiding principles in making a choice between carotid ligature and direct attack, or in deciding the earliest stage at which either of these procedures may be employed with reasonable safety, but I would be most interested to know the view of others with wider experience in this connexion.

The only certain method of excluding risk of recurrence would appear to be excision of the sac

along with that portion of the parent artery from which the aneurysm springs, or to clip or ligate the latter immediately proximal and distal to the aneurysm, when the blood supply of the parent artery is dispensable. That clipping or ligating the neck of an aneurysm gives certain protection is probably not true, for there still remains the pathological area in the wall of the parent artery. As regards the respective merits of occlusion of the carotid arteries in the neck and the local measure of wrapping the sac with muscle or other materials, probably long follow-up studies are not yet sufficiently numerous to permit of firm conclusions.

Carotid ligation has the advantage over direct attack of being technically much simpler and one can apply various tests which will afford some (although by no means infallible) guides as to its safety. Whether one can make the procedure safer by direct recordings of intra-arterial pressure and so regulating the degree of carotid occlusion as to avoid precipitous drops in systemic and pulse pressures I do not know. However, in a recent case, that of a woman with an aneurysm on the upper limb of the carotid syphon giving rise to residual dysphasia, but no other signs, five weeks after rupture, the lumen of the left common carotid was reduced by about one-third, leaving the internal carotid pressure at 76% of the normal. Nevertheless, three hours later the procedure was followed by signs of severe cerebral ischaemia.

There remains the question whether carotid ligation in the neck will afford as lasting protection as local measures for aneurysms situated above the internal carotid bifurcation. For those on the anterior communicating artery it seems improbable that it could. For those on the middle and anterior cerebral arteries it is doubtful, for we do not know whether intra-arterial pressure may not build up again in some cases to such an extent as eventually to threaten rupture of the aneurysm.

As regards the local measures which may be employed during a direct attack, I must confess I have but rarely come upon an aneurysm with a neck that one felt could be clipped or ligated safely. As a rule I have resorted to wrapping the sac with muscle or muslin net and muscle. What degree of permanent security such a procedure affords our follow-up is much too short to indicate, but so far we have had only one case so treated in which fresh haemorrhage occurred two days post-operatively, and none in which the sac later gave way. As regards anterior communicating aneurysms, I feel that the method we have employed in nine cases is open to criticism. Thus when the aneurysm only filled by injection from one side, we have clipped the first part of the corresponding anterior cerebral

artery as close to the neck of the sac as possible in the hope of getting rid of the "jet" effect of blood within the aneurysm. However, there has not been any recurrence of bleeding in any of these patients and all are alive and well five and a half years later, except a boy, aged 9, who has still slight weakness of the contralateral foot after three years, and a hypertensive man, aged 53, with an aneurysm on the second part of the left anterior cerebral artery. In his case clipping of the first part of this vessel resulted not only in increased dysphasia but also in temporary severe right-sided hemiparesis. You will recall also the case already cited in which an aneurysm at the internal bifurcation ruptured during exposure, and in which clips were applied both to the sac and the first part of the anterior cerebral artery. This patient had a contralateral hemiparesis post-operatively and died.

In conclusion, I admit it is possible that our results might have been better had we employed carotid occlusion for some of the aneurysms at or

above the internal carotid bifurcation, instead of waiting until the patient's condition appeared propitious for direct attack, for in some of these bleeding recurred during the delay. However, I am not entirely convinced that of the indirect and direct methods of attack, carotid occlusion can be applied with reasonable safety any earlier after the acute episode, or that it certainly affords as enduring protection as direct measures. I think it is worth while continuing with investigations as to the permanency of falls in arterial pressure caused by carotid occlusion in the hope that the information obtained thereby may lead to earlier and safer application of measures designed to prevent recurrent bleeding, not only in the treatment of aneurysms of the supraclinoid portion of the internal carotid, but also at its bifurcation and on the middle cerebral artery.

REFERENCE

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