Section of Neurology

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Intracranial Aneurysms

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Ruptured Intracranial Aneurysms

[*Abridged*]

The natural life history of a once ruptured aneurysm is nothing short of bleak. The figures (Pakarinen 1967, Sahs *et al.* 1966) serve only to corroborate what has been called the rule of five, i.e., if 5 patients with primary subarachnoid hæmorrhage from rupture of an aneurysm are left untreated, at the end of a year only one will be alive and well, another will be disabled and 3 will be dead. It follows that surgical treatment must be considered in almost every case for, as Adams (1967) stated: 'If there is any place where surgery has its place, it is to keep someone from bleeding to death from a burst vessel.'

A matter of importance is the fact that over half of initial bleeding is not catastrophic but rather of minor severity so that it is often not recognized (Gillingham 1958, Tomlinson 1959). The accurate assessment of this 'warning leak' is of great importance in view of the probability and gravity of a second haemorrhage within the next few days or weeks. What is the harm in doing a lumbar puncture on a patient with sudden unexplained headache if the index of suspicion is high?

The basis of this presentation is a personal experience with over 350 intracranial operations for ruptured aneurysm.

Treatment

Two problems are to be faced in the care of a patient with subarachnoid hæmorrhage: first, to help the brain hurt by the hæmorrhage and, secondly, to prevent the disaster of recurrent bleeding.

Unfortunately, surgeons have been able to do little to help the swollen hæmorrhagic brain with its spastic arteries except for the removal of clot. Brain œdema may respond temporarily, or occasionally have its trend reversed with the use of mannitol or steroids. However, the frequency of a significant amount of clot is not generally recognized for it occurs in over half the cases with severe hæmorrhage. The majority, therefore, deserve consideration of operative treatment on this count alone, to save life or function by the evacuation of clot, providing the hæmorrhage has not put the patient in an irretrievable state. Another factor to be considered is that of acute hydrocephalus from flooding of the subarachnoid spaces with blood (Ellington & Margolis 1969). Raimondi (1969, personal communication) has suggested that acute hydrocephalus may often be the cause of coma in the critical states following bleeding. He has carried out ventricular shunts on 18 such patients; following this 12 awakened fairly promptly and subsequently became candidates for definitive operation on the aneurysm.

In the treatment of the sick brain the physician should take into account the diencephalic lesions and their consequences: hyperthermia, gastric hæmorrhage, water and electrolyte disturbance, and such secondary effects as pulmonary œdema. Attention to these factors may play a significant role in the reduction of the mortality (Barnett 1968). The physician in the neurosurgical unit must also prevent recurrent bleeding until the patient is safe to operate upon. Reduction of the head of pressure in the sac seems an obvious answer. Unfortunately, artificial hypotension is difficult to induce and maintain in the recumbent patient. Slosberg (1965) has shown that it is possible, but in his unit it has required the almost constant attendance of a dedicated team of nurses and physicians. The other solution is to prevent or delay the dissolution of the clot sealing the rent in the sac so that healing may occur. To this end, the antifibrinolysin epsilo-aminocaproic

acid (EACA) has been used (24 g/day) (Mullan & Dawley 1968, Norten & Thulin 1969) but unfortunately there are no good controlled studies as yet. Mullan (1969, personal communication), from his experimental work, feels that this drug may delay lysis of the clot for a week or more, thus buying time for the patient.

There is at present little that can be done for the arterial spasm which is almost peculiar to rupture of an aneurysm; it is said to occur in 30–40% of these patients (Kagstrom *et al.* 1966). The importance of spasm lies in two spheres: (1) The impairment of the circulation and its probable relation to local and even widespread infarctions and (2) the enforced delay of operations, at least for most surgeons who do not dare to touch a spastic vessel within a few days of bleeding. Even in the absence of spasm, operations within a few days of bleeding seem to promote spasm in a distressingly high proportion of cases (Allcock & Drake 1965). Not uncommonly the patient is well for a few days or even a week after operation before developing the swollen infarcted brain which usually leaves survivors with severe deficits. Most surgeons prefer to wait six or seven days when severe post-operative spasm is unlikely to occur; hence the importance of keeping the patient safe from rebleeding for a week or so.

Prevention of the devastating effects of a major hæmorrhage from an aneurysm must be the goal. At present the only way is for the family physician to recognize the warning or minor initial leak which probably occurs in over half the cases and see to it that the case comes to surgical attention before the brain is torn asunder.

Prevention of recurrent bleeding is only certain by surgical means. It was suggested a few years ago that there was little reason for operating upon intracranial aneurysms as the risk of surgical treatment was about the same as doing nothing. However, the factor of security, which is so difficult to measure, was not taken into account. Surely the danger of rebleeding lingers for ever; one has only to visualize the bright red blood swirling around in this evil, transparent and fragile sac as seen at operation. Even were the chances equal, who would turn down an operation which would give a future secure from the dread of recurrent hæmorrhage and the knowledge that excitement, anger or even constipation would pose no threat. Sleep itself is no longer innocent for in the REM phase substantial elevations of blood pressure may occur.

Two principles have been used in the surgical attempts to prevent recurrent bleeding from aneurysms, the indirect approach aimed at reducing the head of pressure within the sac and the direct approach to obliterate the aneurysm.

The oldest indirect method, Hunterian ligature of the carotid artery in the neck, was more widely

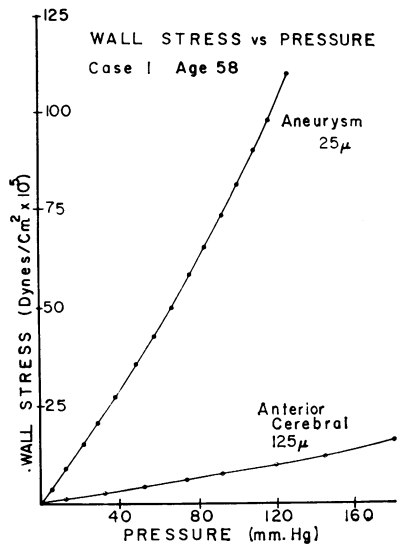

Fig 1 *Comparison of stress with increasing pressures on wall of an aneurysm (thickness 25 μ) and anterior cerebral artery in fresh unfixed state. The rigidity of the aneurysm is apparent while the elastica of the artery takes up the pressure easily (courtesy of G G Ferguson)*

used while intracranial methods were being developed. I have had little experience with this method as a primary procedure for ruptured saccular aneurysms. It is not ideal in that there is a higher incidence of recurrent bleeding than with intracranial methods and the brain is forever deprived of an important source of arterial irrigation. Its effectiveness is probably restricted to those aneurysms arising from the internal carotid artery itself rather than those on its branches. Even with the gradually occluding clamps, the incidence of cerebral infarction and death or serious morbidity is over 10% (McKissock *et al.* 1960, Odom 1969). Nevertheless, in a restudy of Odom's cases after one year, reduction in size or occlusion of the aneurysmal sac was demonstrated in 63 of 66 cases, averaging a 25% reduction. The incidence of rebleeding was 9% during closure of the clamp but only 4% after the artery had been occluded.

Logue (1969) has been an advocate of Dott's proximal occlusion of the dominant anterior cerebral artery filling a ruptured anterior communicating aneurysm. He has now operated on over 100 cases followed up for more than 10 years; the overall mortality was 26% but it was only 9% in good risk patients. Recurrent bleeding has occurred only in 7% and the quality of survival has been excellent, with 90% working.

At the present time, most surgeons are engaged in a concentrated effort to obliterate intracranial aneurysms by occlusion of the neck or reinforcement of the wall through an intracranial approach. Ingenious methods have been developed

Table 1
Results of treatment in 163 Grade 1 patients

	0–3 days				4–6 days				7–14 days				>14 days			
	E	G	P	D	E	G	P	D	E	G	P	D	E	G	P	D
1952–5, normotension, normothermia:																
ICA													1			
MCA									1		1					
ACA					2								1			1
1956–62, hypothermia: ●																
ICA	3		2		3				5	1			4			
MCA													1			
ACA		1		1									4			
VBA				1	1				1		1		1			1
1963–70, deep hypotension:																
ICA				2	6			1	18			2	9	2		
MCA					1				4		2	1	3			1
ACA	1		1		1	1			16	2			13	1		
VBA			1						10			1	22	1	1	1
Total	4	1	4	4	14	1		1	55	3	4	4	59	4	1	4
Mortality	31%				7%				6%				6%			
Success rate	38%				93%				91%				94%			

E, excellent. G, good (non-disabling deficit).
P, poor (disabling deficit). D, dead
ICA, MCA, ACA, VBA: internal carotid, middle cerebral anterior cerebral and vertebral-basilar aneurysms
● Hypothermia at 28°C except for 10 cases operated upon under deep hypothermia (9–15°C) with cardiopulmonary bypass

to produce thrombosis within an aneurysmal sac such as the injection of horse hair (Gallagher 1964), the insertion of tiny cadmium wires to alter the electrical potential (Mullan *et al.* 1965) and the introduction, stereotactically, of iron filings held in place by a magnet for 4 or 5 days until thrombosis is complete (Alksne 1969). However, most aneurysms can be obliterated by the placement of a clip or ligature on the neck. Ligatures are very satisfactory for broad necks or those awkwardly placed. A figure-of-eight ligature is particularly useful for a broad-based bilocular aneurysm.

The major technical advances which have furthered the course of aneurysm surgery include the use of magnification of vision (with loups or the operating microscope), which is now indispensable, the deeper levels of artificial hypotension (Aitken & Drake 1970, unpublished) and the brain-shrinking agents.

The fragile nature of aneurysms and the fear of inadvertent rupture often made the surgeon reluctant to pursue accurate complete dissection of the sac or prompted him to use more indirect methods such as ligation of the parent artery at a distance. However, as seen in Fig 1, the lowering of arterial pressure causes a tremendous decrease in the stress on the wall of the aneurysm as compared with a normal artery. At 40 mmHg mean arterial pressure as measured with a cannula in the radial artery, the aneurysm is soft and pliant so that under magnification its origins and connexions can be dissected out clearly and tiny vital perforating arteries separated from the neck. The clip or ligature can be applied accurately or multiple trial applications are possible to be certain of the best position. In straightforward cases the pressure need only be reduced for a few minutes but in more complicated situations, mean arterial pressures of 40–50 mmHg have been tolerated for over 2 hours without known residual effect except in one of over 200 cases; this case was a hypertensive atherosclerotic Grade 3 patient who died suddenly as a vertebral aneurysm was about to be clipped, although she had been at 40 mmHg for only 20 minutes. The other important feature of deep hypotension is that it allows the surgeon to explore doubtful cases with reasonable safety.

The accuracy of occlusion of aneurysms deserves some emphasis. In 1967, the dire late consequences of incomplete obliteration of aneurysms was reported, for over a period of years rebleeding will occur in 50% of cases where a large portion of the aneurysm remains (Drake & Vanderlinden 1967). It is now felt necessary to operate again if a significant portion of the sac remains.

Results of Treatment

The overall mortality of intracranial operation for ruptured aneurysm in 367 cases was 21% (77 deaths). This figure has little meaning, however, since it fails to take into account the condition of the patient, the site of the aneurysm, the interval to operation and the surgical adjuncts used.

In Tables 1–4, the results are shown according to the grading (Botterell *et al.* 1956) of the patients, the interval and the site of the aneurysm in three periods. During the first period, 1952–5, the patients were operated upon without adjuncts at normal body temperature and without hypotension except for the occasional compression of

Table 2
Results of treatment in 72 Grade 2 patients

	0–3 days				4–6 days				7–14 days				>14 days			
	E	G	P	D	E	G	P	D	E	G	P	D	E	G	P	D
1952–5, normotension, normothermia:																
ICA														1	1	1
MCA	1				1								1			
ACA											1					
1956–62, hypothermia:																
ICA	3			3	1									1		
MCA		1	1						1			1	1			
ACA	3	2	1				1	1	1	1				1		
VBA																1
1963–70, deep hypotension:																
ICA	2			2					6	2			1		1	
MCA	1				1				2				1			1
ACA		2					1		2	3	1	1	2			
VBA					1				5		1		1			
Total	10	5	7		4	0	1	2	17	6	3	2	7	3	2	3
Mortality	32%				29%				7%				20%			
Success rate	45%				57%				82%				67%			

Table 3
Results of treatment in 69 Grade 3 patients

	0–3 days				*4–6 days*				*7–14 days*				*>14 days*			
	E	*G*	*P*	*D*	*E*	*G*	*P*	*D*	*E*	*G*	*P*	*D*	*E*	*G*	*P*	*D*
1952–5, normotension, normothermia:																
MCA	1							1			1			1		1
1956–62, hypothermia:																
ICA	1	2	1	3	1									2		
MCA	1				1											
ACA			1					1								1
VBA	1	2	1	1									1			
1963–70, deep hypotension:																
ICA		1	2	1					1	1		2		1	2	1
MCA	1	2	2					1	1	1	3			1	3	
ACA			1	1				2	2	1	1	1			1	
VBA									2		1			1	2	
Total	5	7	8	6	2	0	0	5	6	3	6	3	1	6	8	3
Mortality	23%				71%				17%				17%			
Success rate	46%				29%				50%				39%			

the carotid by the anæsthetist's thumb just prior to clipping or after the sac was torn. The small number of good risk cases probably reflects the reluctance of the physicians at that time to refer patients for surgical treatment unless they were seriously ill. The second period, 1956–62, corresponds to the universal use of moderate hypothermia (28–30°C) on our unit. However, it gradually became apparent that hypothermia did not solve the problems of early operation, although the results with later operation were satisfactory, and was not, therefore, necessary. Since abandoning hypothermia in 1962, deep hypotension at normothermia and magnified vision have been the major adjuncts utilized.

As seen in Tables 1–4, the mortality from operation under a week from bleeding was never less than 23% except in the Grade 1 cases operated upon during the last half of the week. It rose precipitously with the worsening condition of the brain so that it was 45% in Grade 4 patients and 79% in the moribund Grade 5 patients (the percentage figures are given to the nearest whole number).

An interesting and rewarding change in the results took place in the Grade 1 patients operated upon four days or later after bleeding: there was 6–7% mortality in the 128 good risk cases who were mentally alert at the time of operation. The less recovery the brain had made during the week or longer, the worse the results, running from 7–20% in the still drowsy confused Grade 2 patients to nearly 75% mortality in the stuporous Grade 4 patients with severe focal deficits. Furthermore, over half the 23 deaths after one week were due to potentially avoidable causes: 4 slipped clips, 1 stenosing ligature, 2 pulmonary emboli, 1 meningitis, 2 bleeding diatheses with low molecular weight dextran and 2 after bursting of the aneurysm during scalp incision under light anæsthesia.

Of equal importance to the mortality is the quality of survival, that is the number of patients emerging from operation as excellent or good results, able to return in security to their life with no or non-disabling defects. This 'success rate' is determined by excluding the unsuccessful results, those who were classified as fair or poor and those who died.

Operations under a week in poor risk cases are barely rewarding; one-third to one-half the cases died and less than one-half were successful results. This is especially true where the cause of the poor grade was brain swelling and infarction, whereas the majority of the good results in these groups were patients who survived because of the removal of clot. There is no useful survival in the Grade 5 cases and they should not be operated upon. Even after a week there is barely an improvement when the patients are still graded 3 or worse. Although the mortality was only 17%, there was a high morbidity indicating that when, after a week, there is continuing severe disturbance of brain function, the results are likely to be poor in about one-half of the cases.

The situation is quite different in the Grade 1 patients, even those who were operated upon during the last half of the first week. Whereas in the first 3 days the mortality and success rate are poor, 31% dead and only 38% good, there was only one death in 16 Grade 1 cases operated upon on the 4th, 5th or 6th days and the success rate was excellent (93%). These results are continued for cases (Grade 1) operated upon after a week.

Until this review of our cases, it was felt strongly that operation on good risk patients should be delayed for a week after bleeding when the incidence of severe post-operative spasm was

Table 4
Results of treatment in 50 Grade 4 and 14 Grade 5 patients

	Grade 4 patients												*Grade 5 patients*			
	0–3 days				*7–14 days*				*>14 days*				*0–3 days*			
	E	*G*	*P*	*D*	*E*	*G*	*P*	*D*	*E*	*G*	*P*	*D*	*E*	*G*	*P*	*D*
1952–5, normotension, normothermia:																
ICA			1	1				1								1
MCA	1	2	2													1
ACA		1	2	6												3
1956–62, hypothermia:																
ICA	1	1		5				1			1					1
MCA	2	3	1	1			1									1
ACA			1	3												2
VBA				1												
1963–70, deep hypotension:																
ICA				1											3	1
MCA		3	4	2												
ACA								1								
VBA																1
Total	4	10	11	20	0	0	1	3	0	0	1	0	0	0	3	11
Mortality	45%												79%			
Success rate	31%												0%			

less. However, these satisfactory figures on the 4th to 6th day operations, albeit small, suggest that the interval may be shortened slightly providing there is no arterial spasm. In this regard, there is a significant number of cases where arterial spasm is not seen at first but where deterioration occurs toward the end of the week with severe spasm but without rebleeding. An operation on the 4th to 6th day in such a case would be disastrous. There may be a place for repeat angiography prior to such a venture.

The essentials of successful surgical treatment, therefore, are to operate when the brain can be made slack, preferably a week or more after bleeding, so as to avoid arterial spasm; to have the aneurysm defused with deep hypotension, soft and workable so that gentle, bold and accurate dissection may be carried out under magnification; this to be followed by an accurate occlusion of the sac by a clip or ligature, or, if this is impossible or unwise, by reinforcement of the wall with gauze or plastic, and finally post-operative angiography.

Although we are having increasing success in obliterating aneurysms in reasonably well patients, nearly 1 in 10 patients is lost from rebleeding in our unit during the delay to a safe period for operation. Further, a similar number deteriorate to a lower grade before operation, usually from rebleeding, which diminishes the chances of a successful procedure. It is still considered that this combined mortality of nearly 15% and the morbidity figures are very much better than would occur if all patients were operated upon immediately.

The Future

If it is true that the majority of patients have an initial warning leak, then it is an urgent matter that these episodes be recognized for what they are by physicians and that a state of emergency exists. Further, since the surgeon is forced to delay operation for a few days because of the danger of post-operative spasm, the patient is left at great risk from rebleeding. It is hoped that there will be an answer to arterial spasm soon. In the meantime what is needed is a means to keep the patient safe from rebleeding for a week or two.

There is a great need for special units, 'subarachnoid hæmorrhage care units', to be developed regionally, where, after immediate transfer following recognition of a hæmorrhage, intense medical effort might keep the patient hypotensive. Hopefully, an antifibrinolysin may prove of value in preventing the dissolution of the clot sealing the rent in the sac. Such measures may allow the surgeon to operate for the most part on brains that have not been torn apart by recurrent hæmorrhage. We now have the surgical techniques to deal with the majority of saccular aneurysms. The state of the art is such that operations on good risk patients should carry a mortality in the neighbourhood of 5%. The quality of survival is also excellent for over 90% are able to return to a normal life. In other words, it should be expected that the good risk patient with a ruptured aneurysm would do well after craniotomy just as it would be anticipated that the patient with a bleeding peptic ulcer would do well after gastrectomy.

Perhaps the scope of these units should be broadened to harbour all those with impending or threatened strokes from other causes, for it has been said of stroke that there is nothing in the field of medicine to equal the disability and loss of human dignity.

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Ruptured Cerebral Aneurysms

The rational treatment of ruptured cerebral aneurysms is by operation. The techniques are based on the experience of the individual surgeon developing his methods over the years. Given a fairly basic technique, the results will be related increasingly to methods of selection of patients.

Case Material

Of 230 patients with intracranial hæmorrhage referred since January 1967, 138 had ruptured cerebral aneurysms of whom 105 (77%) were operated upon (Table 1). It is apparent that there