THE PRESENT POSITION IN THE SURGERY OF SUBARACHNOID HAEMORRHAGE

A Consideration of the Commoner Aneurysms

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I THINK it must be admitted that when surgeons first embarked upon the intracranial treatment of aneurysms enthusiasm cost as many lives as it saved. Knowing that aneurysmal sacs in at least some situations could be obliterated, and obsessed by the fear of repeated hæmorrhage, we operated on patients before they had sufficiently recovered from what is a grave illness, and while the techniques of operation were immature. We might profitably contrast this for a moment with the surgery of the stomach. For years the general surgeons muddled about with gastro-enterostomy, finding out what was possible before removing the stomach with its ulcer; and they then developed the technique of gastrectomy to an exact exercise on the 'cold' case before applying it to the 'hot' one—the emergency of perforation.

In dealing with perforated aneurysms we must follow their example and learn to walk before we can run.

Policy

A patient who, after subarachnoid hæmorrhage, has copious blood in his theca, or is stuporous, or has a dense hemiparesis is not fit for major surgery. Carotid angiography is best withheld until treatment is possible, for in the acute stage even skilled investigation may make the patient worse.

It follows that little is to be gained by moving such cases to a neurosurgical centre. Nevertheless, we often welcome them if our bed state allows, though they comprise by far the greater proportion of our fatalities.

Of patients surviving the first few days, about 50% untreated will bleed again and rather more than half of those will die. But it is well to think of these figures the other way round: from 30% to 50% of patients with aneurysms will not bleed again if left alone, and it is evident, therefore, that ruthless and indiscriminate prophylactic surgery is forbidden.

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Except for the occasional case that suffers cumulative hæmorrhages every one or two days (almost deserving of the appellation 'malignant subarachnoid hæmorrhage'), recurrence is said to be not common during the first week. The second, third and fourth weeks hold particular danger, but after two months the recurrence rate of untreated cases is reduced to about 20% (Ask-Upmark and Ingvar, 1950; Walton, 1956).

At this centre we aim to accept cases on the fifth day after hæmorrhage—but before this if the patient is well enough, and later if he is too ill to move. To delay the transfer of a fit patient into the second week or longer increases the risk of further hæmorrhage before prophylactic treatment can be completed, and for this reason it is best if transfer is intended, that the physician arrange for a summary to be telephoned to a neurosurgical unit immediately a fresh case is diagnosed. By this means provisional arrangements may be made to reserve a bed at an agreed time. Unfortunately neurosurgical centres suffer great pressure on their bed space and very occasionally, even with the best intentions, cases have to be deferred.

I am often asked such questions as 'Are you interested in a hypertensive patient of 65 who has had a subarachnoid hæmorrhage?'

Age. The age of a patient in years is to some extent inapplicable to a decision on transfer. I would rather operate on an active, skinny old lady who has a bright eye and does all her own shopping than a bull-necked man of 50 who has bronchitis every winter. But admittedly brittle-feeling, pipe-stem arteries must be taken into account.

The *blood pressure* is very frequently boosted by subarachnoid hæmorrhage, sometimes as high as 230/130 in a usually normotensive individual. Moderate boosting need not modify a plan of treatment for a given patient, and as to the increased risks at operation, the anaesthetist will say 'The blood pressure at operation is what I want it to be' (Barr, 1961). It is useful to know that a patient was previously hypertensive partly because of the arterial disease likely to accompany it, and

FIG. 1.—See text. The dotted line represents the midline of the head. Arrows point to the small posterior communicating artery.

partly so that one may know at what head of pressure that particular patient usually has his brain supplied with blood.

Time interval between haemorrhage and treatment.

The plan here has been to operate on patients as soon as they are considered fit for it. Some are fit in a few days; some not for weeks; some not at all. Preferable to the 'number of days since the haemorrhage' is a categorization such as that of McKissock and Walsh (1956), who report their most rewarding results in 'patients who had completely or partly recovered from the haemorrhage which caused admission to hospital, who were seen within eight weeks of the last bleed and who were not in danger from the initial haemorrhage'.

The aneurysms to be considered in this summary are those developing at the three commonest sites on the carotid tree: on the internal carotid artery itself above the cavernous sinus, on the middle cerebral artery, and on the anterior cerebral/anterior communicating junction.

Fig. 1 consists of tracings from four angiograms having the advantage of showing these three aneurysms existing on one side of one patient. The other carotid tree was normal. This patient was a 37-year-old man who had had a haemorrhage in March 1956, followed by temporary paresis of the right leg. Thirteen days later there was further haemorrhage, resulting in stupor, and Dr. Wylie Smith referred him for investigation. The lateral view (a) shows such a confusion of shadows that interpretation is impossible, except that there is a small 'posterior communicating' aneurysm. The antero-posterior view (b) elucidates these shadows into a large anterior communicating and a middle cerebral aneurysm. There was no evidence as to which of these aneurysms had bled, and carotid ligation was decided upon in the

hope of reducing the strain a little on all three. A test closure of the right common carotid produced an immediate hemiparesis, with recovery after it was opened. The clamp was then screwed down slowly over six days and the carotid then ligated without incident.

But I was still not happy about him and 10 months later he agreed to readmission for check angiography. (c) is an angiogram done on the same side by internal carotid puncture. The anterior communicating aneurysm has enlarged dangerously upwards and to the left. Otherwise there is not much change. The arteries on the right are still enormous and both anterior cerebrals fill; evidently the right carotid even now dominates the anterior communicating system, the flow being down the external carotid and up the internal. The last film (d) is a contralateral angiogram with compression of the right carotid above the ligature. It was obviously not safe to ligate the right internal carotid, so a clip was placed across the right anterior cerebral artery. I heard from him in December 1961 and he is very well, though he weighs 20 stone and gets out of breath. Of course, he is still in some danger, but I think this case illustrates well the place in surgical treatment of tiding a patient over.

About one in eight cases of proved intracranial aneurysms are multiple and it is impossible to categorize them or prove by mere figures the usefulness of surgery.

While each of the aneurysms at the three anatomical sites mentioned above constitutes an explosive threat to life, limb and intellect, they do so in different ways and vary greatly as to difficulties in treatment. Any report combining them into a single group is valueless.

Internal Carotid Aneurysms

Posterior Communicating Aneurysms

The so-called 'posterior communicating' aneurysms arise from the junction of the internal

FIG. 2.—A 'posterior communicating' aneurysm before and after carotid ligation. In this case no posterior communicating artery on that side fills.

carotid and posterior communicating arteries. They often rupture both into the carotid cistern and into the temporal lobe. Like all aneurysms of the intracranial carotid artery, the great majority can be rendered safe from further bleeding by carotid ligation in the neck (Schorstein, 1940; Jefferson, 1947; Black and German, 1953; Harris and Udvarhelyi, 1957; Poppen and Fager, 1960; McKissock, Richardson and Walsh, 1960).

But, at least, when they present a narrow origin, as that in Fig. 2, they are also peculiarly amenable to radical treatment, elevation of the temporal lobe and clipping of the neck itself being often an easy operation. Nevertheless, the occasional case will bleed and die at craniotomy, and carotid ligation would still be the method of choice were it not for the dreaded complication of hemiplegia—unforeseeable either by preliminary carotid compression tests or study of cross-flow on angiography. Some insurance is provided by gradual compression with a screw clamp, and it is probably never wise to ligate the artery outright. The check angiogram (*b*), Fig. 2, shows the result three months after common carotid ligation; the tiny remnant is certainly safe. This patient's third nerve was irreparably damaged at the time of the hæmorrhage.

The 'trapping' operation—placing of a clip on the carotid artery both below and above the neck of the aneurysm to isolate it—is unnecessary if the relevant posterior communicating artery does not fill angiographically, and dangerous if it does; death resulted in one of my cases because the forward filling posterior communicating artery after carotid ligation in the neck had no outflow except to the aneurysm when a clip was placed above it; the aneurysm was seen to swell up immediately and burst.

Table 1 gives a simple analysis of 50 such cases.

TABLE I
FIFTY CONSECUTIVE CASES OF 'POSTERIOR COMMUNICATING' ANEURYSM ADMITTED BEFORE THE END OF 1958

Not Due to Treatment	Due to Treatment	Considered Safe from Recurrence after Treatment
Deaths <i>a</i> 13	Deaths <i>c</i> 3	
Hemiparesis <i>b</i> 1	Hemiparesis <i>d</i> 3	<i>e</i> 34

e includes *d*. Five cases had craniotomy, 32 carotid ligation. Most of *a* were too ill for treatment, but included are four cases of recurrent bleeding after carotid ligation: two possibly and two certainly from the same aneurysm, one as long as six years after treatment. Two of *d* have a virtually complete hemiplegia. Of the three deaths *c*, one followed collapse on open check angiography, one had carotid ligation when too ill, and one succumbed after hæmorrhage at a 'trapping operation'. Most of *a* had hemiparesis.

Almost all cases of carotid ligation have had check angiograms a few months later to discover the size of the aneurysm, and it is chiefly on such evidence that the designation 'safe' is based, both in Tables 1 and 2.

Aneurysms elsewhere on the intracranial carotid artery are comparatively uncommon. As an example, Fig. 3 shows tracings of angiograms in a boy of six who was hemiplegic and aphasic after his third subarachnoid hæmorrhage within six weeks in 1955. We had to wait, on tenterhooks, for a further three and a half weeks before he was fit for angiography (*a*) Fig. 3). Even then it can be seen how spasm was maintained. Common carotid ligation made him no worse, and seven years later he is a very intelligent lad with no disability except, unfortunately, a right hemianopia.

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FIG. 3.—An ‘internal carotid/ophthalmic’ aneurysm before and after carotid ligation.

After check angiography ((b) Fig. 3) had shown reduction in size of the aneurysm, the internal carotid was ligated.

Table 2 gives some indication of the behaviour of these aneurysms in contrast to those in Table 1.

TABLE 2

FOURTEEN CONSECUTIVE CASES OF ANEURYSMS ELSEWHERE ON THE INTRACRANIAL CAROTID ARTERY ADMITTED BEFORE END OF 1958

Not Due to Treatment	Due to Treatment	Considered Safe from Recurrence after Treatment
Deaths <i>a</i> 1	Deaths <i>c</i> 0	
Hemiparesis <i>b</i> 3	Hemiparesis <i>d</i> 1	<i>e</i> 11

e includes *b* and *d*.

Middle Cerebral Aneurysms

These bleed primarily into the Sylvian fissure and more often into the temporal lobe than the frontal; they tend to cause hemiplegia and aphasia in survivors more commonly than do other aneurysms. In Fig. 4 the sac is represented as transparent.

Carotid ligation, though sometimes permissible, is not a reliable safeguard for middle cerebral aneurysms, and these are anxious cases. Extreme refinements of technique are necessary to avoid a post-operative hemiplegia; and if a dangerous case, which has already had a warning hemiparesis or shows extreme local spasm on angiography, can be tided over the first four or five weeks, it may even be wise to leave it alone. But if the sac

FIG. 4.—Sketch of a middle cerebral aneurysm. This is a typical appearance as seen at operation, with a broad base jealously guarded by the trifurcating branches, none of which may be sacrificed nor left in spasm.

is large—say, 1 cm. or more in diameter—the risk of craniotomy must be taken, for in my experience the walls of such sacs are often so attenuated as to be in places transparent and the blood may be watched whirling about inside—a state of affairs obviously incompatible with conservative treatment. These large ones are best dealt with by temporary occlusion of the middle cerebral artery under hypothermia and obliteration of the sac after opening it (Gibbs, 1957), a technique sometimes applicable to the smaller aneurysms also. Short of the ideal obliteration of the sac, reinforcement externally is commonly practised. Wrapping the aneurysm with muscle, described by Dott as long ago as 1933 and developed by Falconer (1951), is still considered an effective treatment, although the resulting natural fibrosis takes a few (probably three) weeks to mature. Realization of this led to

the introduction of acrylic investment (Dutton, 1959; Selverstone, 1961), which in turn has the at least theoretical disadvantage of enclosing pulsating structures in a rigid envelope.

Thirty-two of the 50 cases in Table 3 had craniotomies; only one had carotid ligation, and this has not been included in those considered safe.

TABLE 3

FIFTY CONSECUTIVE* CASES OF MIDDLE CEREBRAL ANEURYSM ADMITTED BEFORE END OF 1961

Not Due to Treatment	Due to Treatment	Considered Safe from Recurrence after Treatment
Deaths <i>a</i> 13	Deaths <i>c</i> 5	
Hemiparesis <i>b</i> 12 8 treated 4 treatment not advised	Hemiparesis Due to or Exaggerated by Treatment <i>d</i> 6	<i>e</i> 26

* Excluding four cases with no hemiparesis who refused treatment.

e includes *d* and eight of *b*.

This table shows the surgeon's dilemma. The more he attempts to extract from the ill cases in column 1, the more he will add to column 2. Obviously there has been very real cause for hesitating to treat these cases by craniotomy at all, as other workers have found (Small, 1957; Murphey, 1958). Yet, of the 13 conservatively treated cases *a*, no fewer than eight died of recurrent hæmorrhage either in hospital or during the follow-up period afterwards.

Of the five deaths *c* due to treatment, only one was neurologically normal the day before operation. Two cases died as the result of violent bleeding on exposure of the aneurysm. In retrospect, two of the five were certainly, and two possibly, operated upon earlier than was wise, with the reservation that any case which bleeds on gentle dissection at operation is the very one which needs it. Of the 13 cases in which the sac was obliterated, five are well, four had disability due to operation, two were no worse and two died. In two cases the proximal middle cerebral artery was clipped: one is well and one died of infarction. The degree of neurological deficit in *d* has not been elaborated. The fact that some of the 'hemiparesis' cases (which includes dysphasia if on the left and/or hemianopia) were mild is not very important; that such occur at all and were caused or exaggerated by treatment means that techniques have been at fault. Not until we can 'steal' aneurysms

FIG. 5.—Simplified plan of an anterior communicating aneurysm.

as the old surgeons used to steal the thyroid we be able to promise that an operation—possibly unnecessary operation—will not cripple the patient.

Aspiration of the intracerebral blood clot has occasionally been done, but has not been mentioned. It is part of conservative treatment just as the occasional tracheostomy and has nothing to do with treatment of the aneurysm.

Anterior Communicating Aneurysms

These are the most difficult problems. On rupture they tend to cause intellectual impairment out of proportion to other physical signs. Fig. 5 is a sketch, the sac again being represented as transparent to show the anatomy as the inexperienced surgeon hopes to find it at operation. In reality the sac is not only buried in one or both frontal lobes, but enveloped by an irregular network of tough arachnoid which must be stripped off to expose the base. Bleeding when this is done is likely to result in the death of the patient; but just as dangerous as this calamity is the necessary manipulation of the frontal lobes, already damaged by the initial hæmorrhage. Retraction of a tense brain may have condemned the patient to irreparable dementia even before the aneurysm is reached; and in my opinion any surgeon who consistently operates on these cases by direct attack on the sac itself is as foolish as his patients will be afterwards.

Most surgeons agree that the presently correct treatment consists of clipping the anterior cerebral artery most readily feeding the lesion, even though this does not afford complete protection from recurrence. I am sorry to say that, as far as anterior communicating aneurysms are concerned, my policy has been so bedevilled by vacillation and retrenchment that analysis would be of only negative value—the very state of affairs against which Logue appealed in 1956. In a determined attempt to assess the value of a particular operation, Logue clipped the anterior cerebral artery most readily filling the aneurysm in 37 cases with an operative mortality of 13.5%, a morbidity rate of 10% and a post-operative recurrence rate of 3% over an average follow-up period of 22 months. In a comparable series of cases treated conservatively the mortality was 44%. This operation, therefore, certainly has a place in treatment and is at present, I think, the best that can be offered. It was favoured as being relatively safe by Baumann and Bucy (1957), whose individual summaries of 22 cases treated by different methods gives an excellent over-all picture of the difficulties. Norlén and Barnum (1953) preferred direct occlusion of the base of the aneurysm, and of their 15 cases so treated only one died; though only five returned to work soon afterwards, recovery from frontal lobe damage may be prolonged over two years or more, and it is possible that long-term results may be more encouraging.

Intra-cerebral Haemorrhage from Ruptured Aneurysms

A small haemorrhage (say, under 1 in. in diameter) into the frontal or temporal lobe may go unnoticed and needs no treatment. Larger ones present as a classical apoplexy with noisy coma, and may themselves threaten life; but only too often the damage is done at the time of bleeding and its removal does not save the patient. A sudden intracerebral arterial haemorrhage is not only an expanding lesion; it disrupts the brain and destroys neurones. If a certain critical number of neurones is destroyed that patient will die, whether or not the clot is removed. In any case, a blood clot cannot be removed accurately with a brain needle through a burr-hole, so a more major operation is necessary. As far as aneurysms only are concerned, therefore, this is a dull subject and I do not propose to waste more time on it, except to say that refrigeration may sometimes be a better treatment than operation.

Recent Advances

(1) *Refrigeration*, though hardly recent, is of proved value in either expected or very early infarction, with or without raised intracranial

pressure. It is also worth while in decerebrate states after catastrophic bleeding—in some mysterious way a number of these patients stop dying when their rectal temperature has been reduced to between 30 and 32°C. Rapid cooling under anaesthesia is often indicated before craniotomy.

(2) *Controlled respiration* during craniotomy greatly reduces the tension of the brain so consistently present after recent subarachnoid haemorrhage. In young patients especially intravenous urea (Stubbs and Pennybacker, 1960) may be combined with it to advantage. I do not think it is going too far to hope that these aids may in the next few years revolutionize the major surgery of intracranial aneurysms by reducing column 2 to reasonable dimensions, say 5%. We shall then have learned to walk.

(3) During the last two years work has been going on at this centre designed to abolish the calamity of hemiplegia after carotid interruption, and the results at the moment are promising.

Conclusion

There have been many series of figures published attempting to solve a single question: Is surgery in subarachnoid haemorrhage worth while? For the benefit of the discerning physician who has glossed over the figures in the foregoing summary it may be said that the surgery of aneurysms has come to stay; but it needs improving, not analysing. Among all these statistics we tend to forget the patient, and yet I cannot exaggerate the importance of reassuring the patient who survives. Time and time again at the first follow-up clinic they ask 'Will it happen again?' If no convincing answer can be given, both patient and relatives worry and the family doctor forbids the housewife to do anything but dusting, and the man to play football or do gardening.

May I present three alternatives?

(1) Supposing 100 cases of subarachnoid haemorrhage are not investigated and are treated by medical measures only—how many survivors can the doctor in charge tell that they are safe from further bleeding? Answer: *none*.

(2) Supposing 100 cases are proved by angiography to have aneurysms, and are then treated conservatively—how many survivors can the doctor in charge tell that they are safe from further bleeding? Answer: *none*. He knows that some will not bleed, but does not know which.

(3) Supposing a series of 100 cases of proved aneurysm is treated, as you have seen, by conservative and surgical treatment at discretion—how many survivors can the doctor in charge tell that they are safe from further bleeding? Answer: with the exception of some cases of aneurysm

on the anterior communicating system, almost all those who have been operated upon.

It is a pleasure to record thanks to my colleagues, Mr. Geoffrey Knight and Mr. George Northcroft, who have allowed me to concentrate experience on these

cases, and who have now and again, I fancy, been kind enough to suppress a suspicion that they could do better to Dr. Leon Morris for imaginative and painstaking angiographic studies; Dr. Walter Hart for skilful anaesthesia in difficult cases; and not least to our nursing staff, whose devoted care has saved many lives.

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