ANGIOMATOUS MALFORMATIONS OF THE BRAIN: THEIR NATURE AND PROGNOSIS

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by

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THE SUBJECT I have chosen happens to be appropriate at least to John Hunter's great interest in the vascular system ; although it is his brother, William, whom we more particularly associate with arteriovenous aneurysms—with the traumatic variety at any rate.

I must first try to indicate the lesions that I am going to consider. They are the so-called cerebral angiomas ; but the multiplicity of names given to them is evidence of the confusion that has existed in the past as to their precise nature. Purely descriptive terms given to them include : *racemose, serpentine, cirsoid, plexiform,* and *cavernous angiomas* (or *haemangiomas*). Attempts to identify a predominant type of vessel have led to : *arterial, venous* or *capillary angioma,* and to *telangiectasis.* The difficulties involved are reflected in the terms : *varix arteriale, varix aneurysmaticus* and *varicose angioma.* Belief in their congenital origin has given us the word *hamartoma,* and *congenital angioma* ; and picturesque analogy has produced *haemorrhoids of the pia mater. Arteriovenous aneurysm* is now a more popular term than *arteriovenous angioma, anastomotic angioma* or *aneurysm by anastomosis.* I prefer the less confusing, although cumbersome term, *angiomatous malformation,* and it is my personal belief that these are almost all, if not quite all, *arteriovenous malformations* at some level or other of the cerebral vascular system.

Now these lesions are *areas of circulatory perversion* (and it is well to stress now their dynamic aspect), and they are without any evidence of associated neoplasia, such as occurs in the *haemangioblastomas,* which I have not included in this series ; for most people regard them as a distinct group, and true neoplasms, although Willis (1948) does not believe that this is justified. It may serve to emphasise this distinction that I have not included any cerebellar angiomatous malformations in this series ; not because I do not think that they occur (Logue and Monckton (1954), for example, have recently described some) but because I have not been quite satisfied with the data in the few cases available.

These malformations appear to be commoner in the brain than elsewhere. I should say, rather, that they are constantly being discovered nowadays by cerebral angiography, and at least three series of 50 or so have now been collected ; whereas, in a Hunterian Lecture last year, Robertson's series of 40 occurring in the four limbs is, I believe, the largest reported ; and they too were arteriovenous lesions.

Material

My original object was to discover the prognosis of the patients suffering from this condition, and it was necessary to find cases in which the diagnosis had been verified as long ago as possible. I have therefore been fortunate to have had access to the careful and comprehensive case records of the late Sir Hugh Cairns, both while he was at the London Hospital from 1927, and after he went to Oxford in 1938. I should like, at this stage, to acknowledge fully all those who were associated with him in connection with these patients; also Dr. Philip Sheldon, whose opinion on angiography I have found invaluable, and those other physicians and surgeons at Oxford and elsewhere who have allowed me access to their cases and their records; and, in particular, Mr. Joe Pennybacker, who has given me much help. The drawings (Figs. 1 and 8) are by Miss Audrey Arnott.

58 cases have been collected, in which the lesions have been verified either at operation, by angiography, or at autopsy.

THE NATURE OF THESE MALFORMATIONS

In view of the confusion that has existed in the past as to the nature of these structures, I have felt that it might be useful to devote a main part of this lecture to an attempt to clarify this aspect of the subject; for it is a recurrent topic.

There is now an increasing belief, derived from the findings at angiography, that at least the vast majority of these lesions contain abnormal arteriovenous connections. If we now consider in what other ways one may try to find out definitely whether this is so, we will appreciate the difficulties in precise diagnosis that were present before the advent of angiography. Three main approaches that were available may be considered briefly:—

1. The Clinical Approach

Dr. Percival Bailey (1948), in the latest edition of his book on intracranial tumours, states that the diagnosis of the "arterial angiomas" (as he and Cushing (1928) called the arteriovenous malformations) is based on the combination of two things—a *cranial bruit* and *increased extracranial vascularity*. This belief has been perpetuated in most textbooks, but these signs are quite unreliable.

In fact, a bruit is not heard in more than 50 per cent. of those cases where the lesion is shown by angiography to be arteriovenous. This proportion, the highest yet recorded, was noted in MacKenzie's (1953) recent series of 50 cases from the National Hospital, London. In the present series, only 22 per cent. of those in whom it was sought had a bruit.

Increased extracranial vascularity is an even less reliable criterion. It occurred only twice in this series, and apparently not at all in MacKenzie's. It indicates either an associated anomaly of extracranial vessels, or is a late secondary effect from a large arteriovenous shunt. It is an uncommon feature of the intracranial malformations.

2. The Operation Appearances

There is no mistaking the *obvious* case of an arteriovenous anomaly when it is seen on the surface of the brain at operation, such as that in Fig. 1. But relatively few are so obvious, and by that I mean that they are not conclusively recognised to be arteriovenous; and Sir Percy Sargent, in 1930, said, " It is very difficult to tell by inspection alone whether the vessels of which they are composed should be called arteries or veins."

The relevant clues to the arteriovenous nature of the lesion may be hidden among huge secondarily dilated veins, and in the past this has led to the belief that they were essentially " venous angiomas." The main

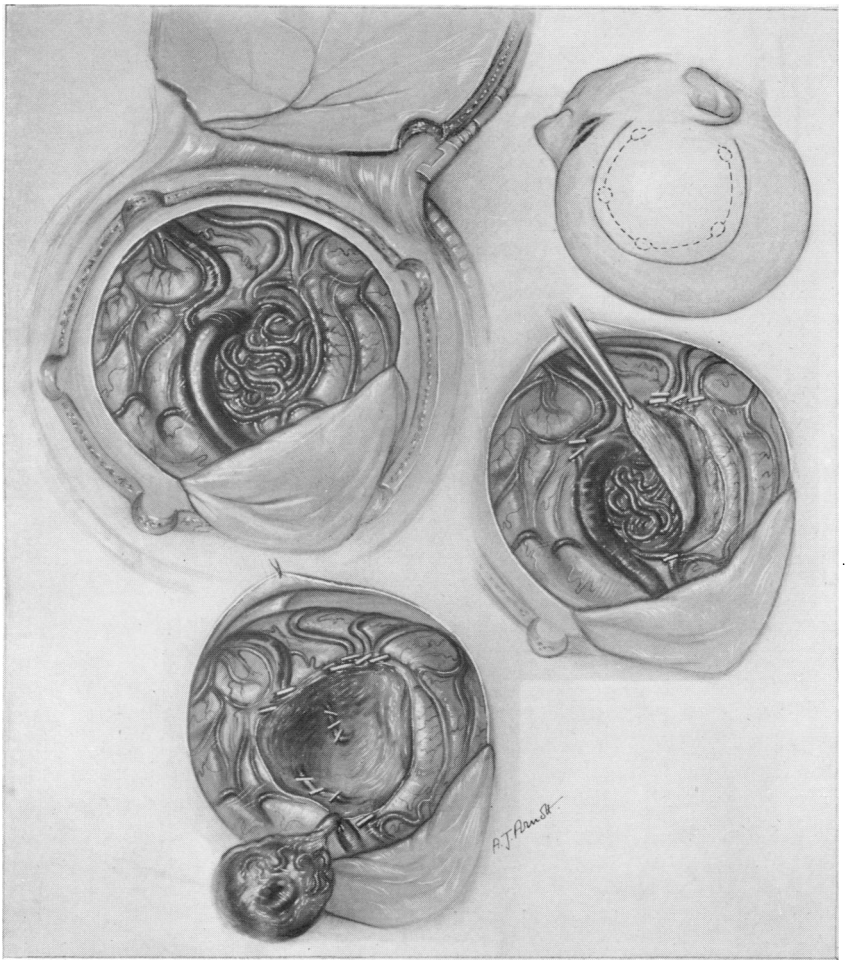


Fig. 1. Excision of an arteriovenous malformation.

draining vein carrying the shunted " arterial " blood may pass deeply or be otherwise concealed. The *colour* of the blood itself may be of little help, because of varying degrees of oxygenation under operative conditions, and because of the effect of the thickness of the vessel walls. *Pulsation* has also been stressed as an important sign, but often neither arteries nor veins show any visible pulsation—in this series it was noted in only one of nine cases where the opportunity existed for careful study.

3. The Histo-Pathological Approach

Neither has this method settled the true nature of these structures, except to distinguish them from the haemangioblastomas. An angiogram will catch the diodone in the veins during the " arterial phase "—an abnormal event which is perhaps an unsurpassed demonstration of a phase in living pathology, but impossible to recall in the morbid anatomical state. It might be possible to demonstrate the arteriovenous connections either by cutting serial sections, or by careful dissection perhaps facilitated by injection with " neoprene latex " (Trueta *et al.,* 1947) but I am not aware that this has been done in these brain malformations.

In microscopic sections, one sees a mass of blood vessels, many of which may be single vessels cut several times in their sinuous courses. Some appear to be arteries, some veins, some indeterminate ; and many are often degenerate, hyperplastic or otherwise atypical. This is not surprising, because, if a certain degree of arteriovenous shunting has been reached, the arteries in which the pressure is abnormally low have become more like veins ; while the veins under high pressure have adapted themselves and become more like arteries (similar changes may be found in a vein graft in an artery). The problem becomes greater when one realises that some of the vessels seen may be the arteriovenous connections themselves. Are these to be regarded as arteries—or veins ? And how does one identify them ?

This is, of course, largely negative evidence, and we are therefore brought back to the most reliable method of demonstrating arteriovenous fistulae ; and that is the angiogram. This method showed that all of MacKenzie's 50 cases were arteriovenous ; and so were all of the 38 of the present series in which the details of the participating vessels were shown with sufficient clarity for one to be certain. Where, then, are all those other angiomas, particularly the venous ones that continue to receive distinct descriptions of their clinical features in the textbooks ? I think, apparently in common with others, that one may have considerable doubts as to their existence at all. We know that most of them are, in fact, arteriovenous, but we do not yet know definitely that the rest are not.

Now you may ask, what is the importance of this great preoccupation with the *arteriovenous* part of these structures ? The importance lies in the peculiar properties of abnormal arteriovenous fistulae in general ;

and if I may remind you of our knowledge of the latter and compare it with our information about these angiomatous malformations, the result will, I think, be rewarding.

The foundations on which we base our ideas of the circulation have been slowly laid as a result of careful experiments. Such experiments are essential, because only the simplest biological occurrences are predictable as a result of the laws of pure physics. Thus, the complicated effects of the presence in the circulation of an abnormal arteriovenous shunt were carefully and thoroughly elucidated by experiment 30 years ago by Emile Holman (Holman, 1937). Although it is common knowledge among surgeons concerned with peripheral vascular disorders elsewhere in the body, little attention seems to have been paid to this work by writers on the cerebral lesions, with the exceptions, particularly, of Bronson Ray (1941) ; and of Dandy (1928), whose insight into the true nature of these malformations was considerable. The important facts may be considered briefly in relation to the particular lesions under discussion.

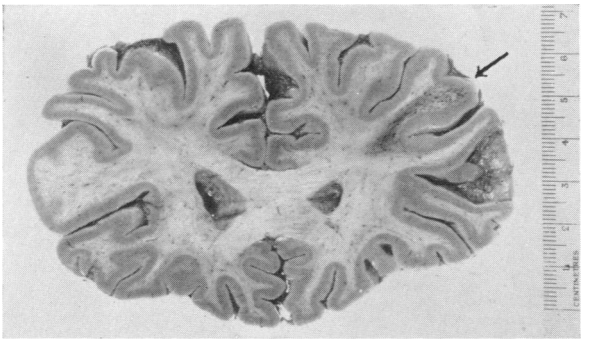


Fig. 2. A right frontal telangiectasis found incidentally at autopsy.

If the abnormal arteriovenous shunt is very small, it may be insufficient to divert a significant quantity of arterial blood away from the capillary bed for which it is destined. This " leak in the peripheral resistance " is too small to require compensatory measures. This may be important in explaining why some of these arteriovenous malformations may be congenital and yet remain inactive for a long time. The telangiectases (which may be regarded as abnormal arteriovenous shunts at capillary or pre-capillary level) may remain inactive throughout life and be discovered fortuitously at autopsy ; such as one, shown in Fig. 2, in the right frontal lobe of a woman of 86 who died from bronchopneumonia and uraemia after suffering from paralysis agitans for three years. Indeed, Dandy recognised that some of the fistulae might be so small as to be not definitely established as abnormal, and these are unlikely to be seen even in an angiogram. (I think that this may also be the case with Sturge-Weber disease, where the angiomatous malformation is primarily in the

leptomeninges, and which may be the cranial counterpart of those rare, diffuse, small-vessel malformations of the limbs, with cutaneous "birth-marks." In these (according to Robertson) angiography is similarly unrewarding.)

Now if one of these small, abnormal vessels in an arteriovenous malformation ruptures, then a larger lesion may be formed in the way that McKissock (1950) has suggested, whereby an arteriovenous sac is formed, or an aneurysmal varix. This may then result in a wider shunt and progression of the lesion as a result of the secondary changes that I shall describe. But first I will show an example of this occurrence (Figs. 3 and 4) :—

A man, aged 22, had a severe intracranial haemorrhage. A left carotid angiogram four days later (Fig. 3) showed a small arteriovenous malformation with a small aneurysmal sac in the region of the left posterior cerebral artery. There was also some evidence of a posterior temporal clot, but he recovered spontaneously and seven weeks later a further angiogram (Fig. 4) showed enlargement of the lesion, of its feeding artery, its venous drainage, and of the aneurysmal sac.

These changes are the same as those that occur once an experimental arteriovenous fistula exceeds a critical size (Fig. 5) ; for it then offers a

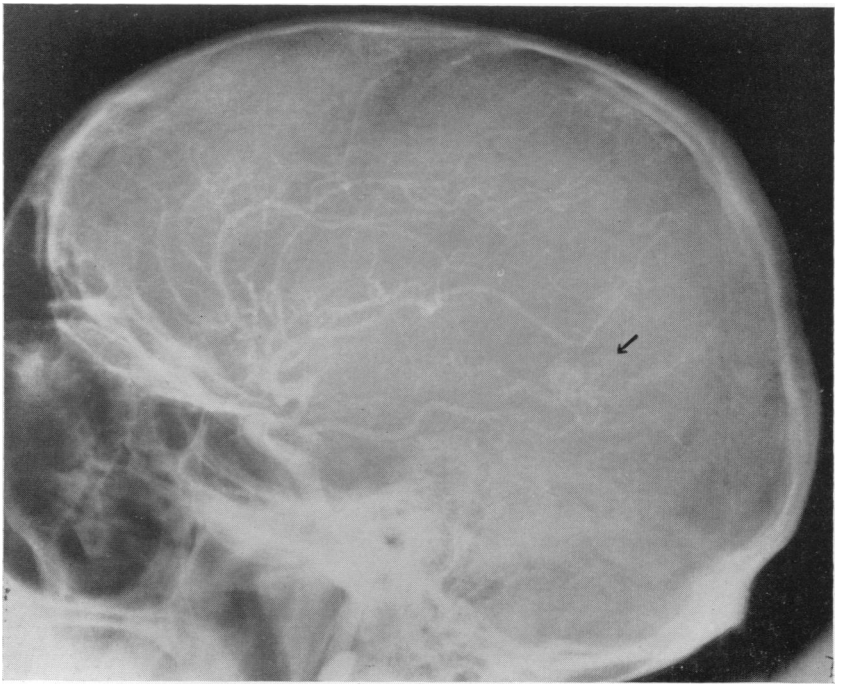


Fig. 3. A small arteriovenous malformation with an aneurysmal sac (indicated by arrow).

pathway of less resistance than that leading to the capillary bed distally, and changes occur in the participating vessels. With the increase in local venous pressure due to the arterial influx, the draining vein or veins and their radicles become dilated and usually tortuous. The feeding artery follows suit (it may not be dilated in the early stages), and this allows more blood to be brought to this " parasite on the circulation," rather than to the proper capillary bed ; and collateral channels may also develop. Presumably then, either equilibrium is reached or, if the shunting channel or channels are capable of being stretched, further changes take place, or a vessel again ruptures and haemorrhage occurs, with the possibility of the cycle being repeated. This seems a much more likely happening than the postulate of Cushing and Bailey (1928), and of Northfield (1940), that some of these lesions were once venous angiomas that have somehow become " arterialised".

Certain general circulatory changes may also occur, such as an increase in blood volume, a lowered diastolic blood pressure, a bradycardiac response on carotid compression, and extensive retrograde arterial dilatation down to and including the heart itself ; but these are not met with unless large intracranial shunts exist, and seem to be uncommon. The degree of any of these secondary effects depends not only on the size of the fistula, but also upon its *duration,* and its *location* in the arterial tree.

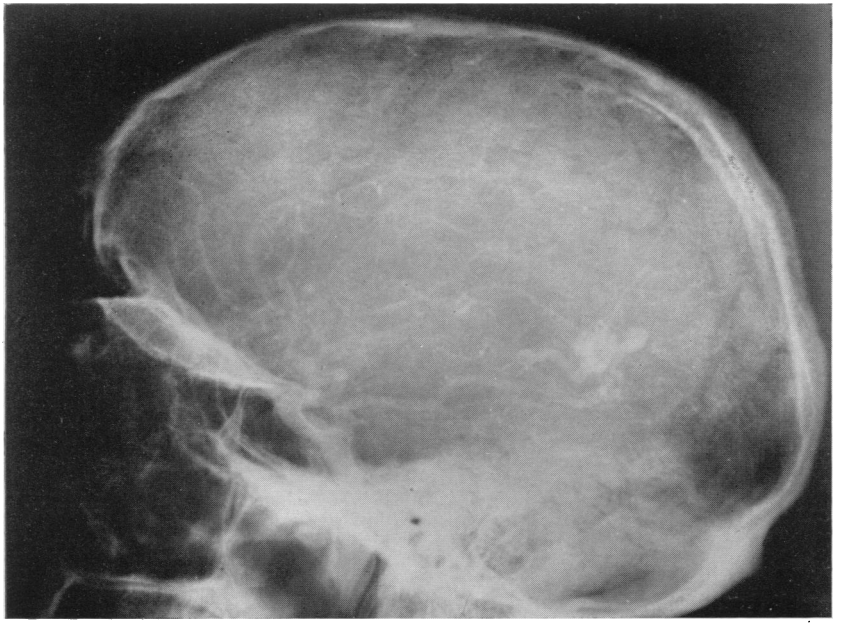
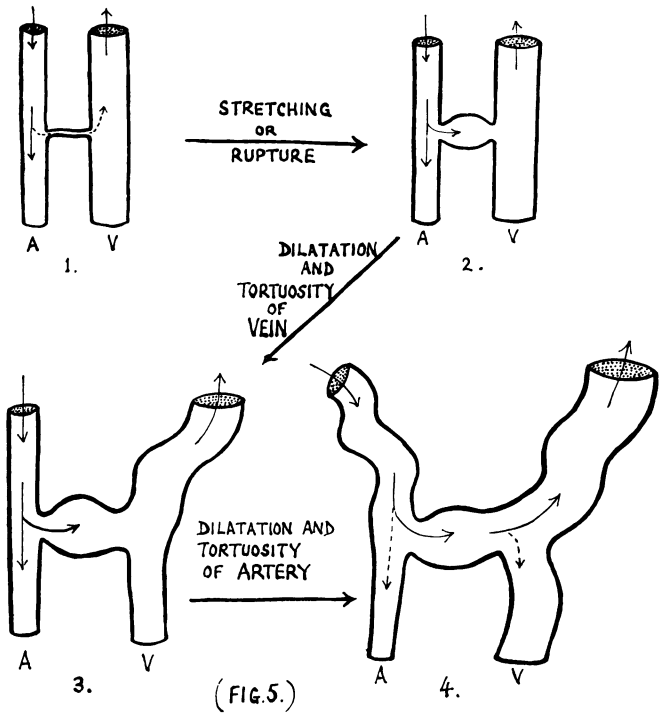


Fig. 4. The same as in Fig. 3, but seven weeks after a haemorrhage. Note the increase in size.



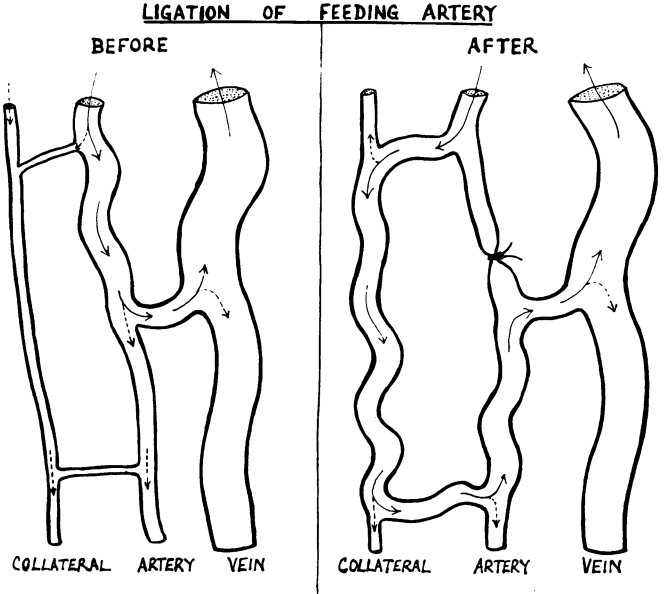
(FIG.5.)

Thus, it is the abnormal arteriovenous connections that are the important—indeed, the essential—lesion, and they must be eradicated if the process is not to recur. The huge, secondarily dilated and tortuous veins, especially, and the feeding arteries that are so impressive in an angiogram or at operation are an *effect* rather than a basic part of the primary lesion, and they give bulk to what might otherwise be a relatively small structure. It is known from post-operative angiograms in this series and from those of Norlén (1949) that the surrounding vessels return to normal if the shunts are excised ; and indeed the perverted circulation of an animal behaves likewise soon after closure of an experimental arteriovenous fistula.

At this stage we should just consider the question of ligation of the feeding artery, or arteries. This has sometimes been done with the intention of at least reducing the blood through the lesion, and has been reported upon favourably on occasions ; but it seems to have no sound basis, and is unlikely to have a lasting effect. It was found to be ineffective by Olivecrona and Riives (1948) ; and, after all, the shunting mechanism remains the same size and will invite arterial blood from somewhere on the " easy terms " of a low resistance. This can only be at the expense of normal brain and via the collateral supply, unless, of course, further compensatory changes occur, in which case the situation will get back to

much the same as it was before (Fig. 6). It is fortunate that the brain is so generously endowed with anastomotic safeguards, for the corresponding ligation operation in, for example, a leg lesion will often result in gangrene.

Now there is nothing really original in all this, but it is I think valuable to review this earlier knowledge obtained by animal experiment and to marry it to the newer knowledge that has now been gained from angiography. This can, moreover, be done with greater confidence if one has had the opportunity to consider a large series of cases which tend to confirm the conclusions that have been reached.



(FIG. 6.)

PROGNOSIS

I now propose to consider this series of cases rather more specifically in relation to the subject of prognosis—an aspect that has received scanty attention in the literature, because of the relatively short time that has elapsed since the certain diagnosis of these lesions has become at all frequent.

What is the problem ? It is, briefly, whether or not these people are going to die young from cerebral haemorrhage ; and whether those that remain are going to be incapacitated by epilepsy, paralysis, or mental change ; or whether they are going to be able to lead long and useful lives. I have only some of the answers to these and to many other questions that arise.

I will first mention some points of clinical interest that have a bearing on this particular problem. In the first place, there is the relatively

early age at which symptoms and complications usually occur—that is, if they are going to occur at all. In this series, as in others, there is a particularly heavy incidence of trouble in the second decade (45 per cent.). The average age of onset of symptoms in general has been 23, the extremes of age being 3 and 64.

It has sometimes been difficult to be certain about the time of onset of symptoms ; and this has been particularly so where *headache* was the symptom. The relevance of perhaps infrequent headache occurring in childhood has also been hard to assess. This headache (often migraine-like, as stressed by others) seems to have been definite as a first symptom in 18 per cent. of the present series, and it is a prognostic point perhaps that it has been followed by a relatively long survival period : all 10 patients who started with headache have lived more than 10 years, eight of the 10 for more than 20, and five for more than 30 years, and one for 54 years.

Haemorrhage is the most feared complication. It occurred in 82 per cent. of these cases ; and, as the presenting symptom, it had the high incidence of 58 per cent. But it seems to be more benign than that occurring from " berry " aneurysms. It tends also to happen at an earlier age : the average age in this series was 25, and 71 per cent. of the patients were under 30 ; whereas for " berry " aneurysms, according to Falconer's (1954) figures, the average age is around 45, and 80 per cent. are *over* 30 at the time of the haemorrhage. However, the relative incidence of haemorrhage from the two types of lesion in the different age groups is not yet clear ; so that the age at the time of the haemorrhage is not necessarily of help in the differential diagnosis. There is also an interesting group of 11 children in whom the initial haemorrhage occurred between the ages of 11 and 13. (From this, and from similar cases recorded recently by Professor Dorothy Russell (1954), it would seem that the text-books might lay greater stress on these lesions as a cause of cerebral apoplexy in childhood.) There is a greater tendency for the smaller malformations to bleed : 18 out of 19 of those arbitrarily classified here as " small " ruptured and bled at some time. This also has been commented upon by other writers. There is a good example shown in Fig. 7—a small-vessel lesion (verified also at operation) in which there is no obviously enlarged feeding artery, but enlarged draining veins are stretched by an intra-cerebral clot.

There have been some long survivals among the 29 patients who have had a single episode of haemorrhage : one has lived 13 years, and others have had 18, 25 and even 39 years of freedom from recurrence of haemorrhage.

One of the six patients who has survived two haemorrhages is worth mentioning in order to illustrate the often surprising natural history of these lesions when left to themselves. Her first haemorrhage (from a malformation in the right Sylvian fissure) was at the age of 13, and it

resulted in a left hemiparesis which prevented her from being accepted for a nurse's training. However, she was able to do auxiliary nursing duties efficiently for 20 years, after which she had a second haemorrhage at the age of 39. She has recovered from this and is back at her nursing duties, but with a slightly increased hemiparesis. She has had no fits at any time.

Four patients have had at least six haemorrhages each, two surviving for 17 and 22 years since the initial one. Another has had at least 12 haemorrhages in 25 years, and is still able to get around on a bicycle; he has had no fits.

With regard to the important problem of *epilepsy* in this condition, I do not propose to go into any detail, as this has been done already, notably by MacKenzie. However, I should like to emphasise from the experience of the present series that there may be extreme variability in both the severity and the frequency of attacks. Cushing and Bailey originally noted that long periods of remission may occur. In five cases in this series the fits have become less frequent and severe as time has gone on, but in some this has been associated with an increase in a hemiparesis. All this is clearly of prognostic importance and makes any evaluation of operative or any other treatment (where this has been undertaken with the intention of influencing the fits) unconvincing unless follow-up periods of, I should say, at least 10 years are available. (In parenthesis

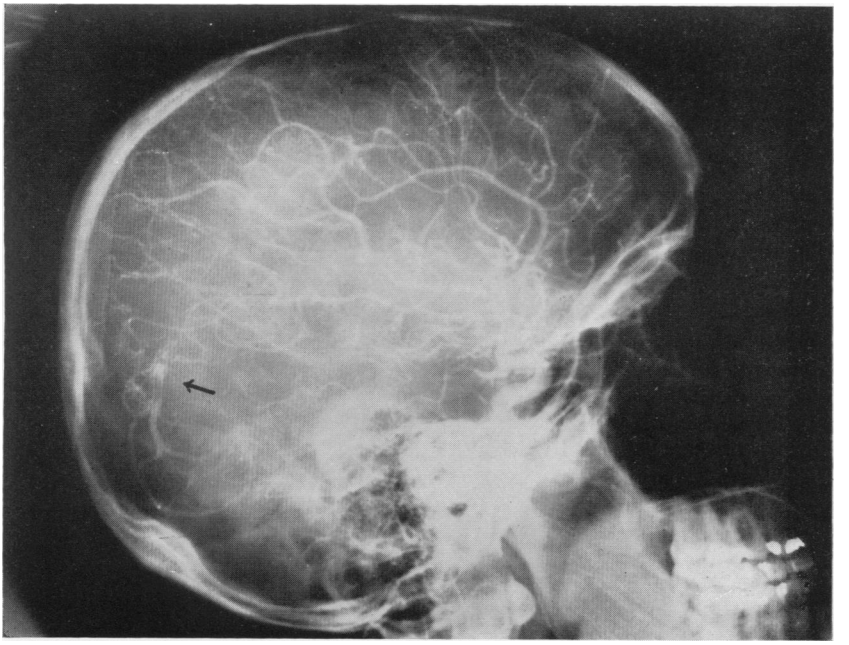


Fig. 7. A very small arteriovenous malformation (indicated by arrow) with a large intracerebral clot.

here, I should mention also that there are five patients who have had fits which started after their operations, and who had had none previously. In another, a useful aura, consisting of a feeling of self-consciousness, was abolished after carotid ligation; and the patient was therefore worse off than before.)

Some degree of *mental disturbance* has been emphasised by Olivecrona and Riives as occurring in 50 per cent. of their series of 42 patients; but details are not given. In MacKenzie's 50 cases, it was noted to be of a mild degree only in 10 per cent., but, again, details were not given. In the present series, I have found mental changes difficult to evaluate. In the first place, the children with these lesions were almost as often bright and intelligent as backward (in a proportion of six to seven). And out of eight adults who might be said to show mild mental disturbances three were subject to vague attacks of " depression." In one, these preceded his epileptic fits and were presumably part of them; and in the other two the episodes seemed to differ in no way from the not uncommon bouts of " depression " that may occur in anybody. Another man had always been highly-strung, and another always rather an inadequate personality. Thus, there is nothing specific about these minor disturbances, but they must be noted, for these are, we believe, congenital lesions. But in three patients whose symptoms have been present for 30, 36 and 54 years, a slight, general slowing-up in intellectual performance has been noted—in all three after about the age of 50. This was not necessarily obvious to the occasional examiner, but had been noticed by others closer to the patient; and it was as if the brain had aged a little prematurely: " not quite so good in the office", " getting a bit ' soft ' "—that sort of thing.

Of those severely affected mentally (there were only four), one was a man who acquired a gross internal hydrocephalus after six episodes of intraventricular haemorrhage. Another man became severly demoralised by fits, a left hemiplegia, and by the fear of a second haemorrhage, which eventually occurred and put an end to a miserable existence. Two others had severe and long-standing personality disorders. Now these last three patients must naturally be noted, but, at the same time, other cases may be found in this series comparable entirely as regards duration of disease and anatomical site and size of the lesion; and in them no such dementia has occurred. Marked mental change is, therefore, by no means inevitable in this condition, and is associated only with a small minority (seven per cent.) in this series.

Survival and Disability

And now let us turn to the question of prognosis in relation to disability. Crude figures for disability in the survivors are of little value, as the follow-up period has in most cases been too short. However, if we consider (as in Table 1) the disability in relation to the survival period

since the onset of symptoms, some useful information may be obtained. (There are three patients in whom I have been unable to ascertain the degree of disablement.)

Disability has been assessed arbitrarily as follows: *nil,* in which there is no interference with work or everyday life; *slight,* where there is mild interference by, say, hemiparesis or sporadic fits; *moderate,* where not much work is possible, but some useful activities can be carried out; and *severe,* where the patient is bed-ridden or otherwise severely incapacitated.

TABLE 1
SURVIVAL (SINCE ONSET OF SYMPTOMS) AND DISABILITY

Survival (years)	No. of cases	Disability				Deaths
		nil	slight	mod.	sev.	
Less than 5	22	13	4	2	2	*4*
5-10	12	3	6	1	1	*4*
10-20	6	1	2	2	1	—
20-30	7	*1*	*3*	2	1	1
30-40	5	—	*2*	2	—	1 (from other causes)
40-50	2	*1*	*1*	—	—	—
50-60	1	—	—	—	1	—
Lesions found incidentally	3	3	—	—	—	2 (from other causes)
TOTALS	58	22	18	9	6	12

Those 22 cases that have been followed for less than five years tell little; but it is noteworthy that eight out of nine deaths (if we exclude those in whom the malformations were not the cause of death) occurred within 10 years of the onset of symptoms; and 27 per cent. have survived for more than 20 years, more than half of these having only slight disability or none at all.

Deaths

With regard to the *deaths,* there were, as you see, 12, but three of these patients did not die as a result of their angiomatous malformations. Seven died from acute intracerebral haemorrhage. Two were boys of 11, both of whom had won scholarships. In each, the episode was unheralded and they lived for 15 and 48 hours only. Professor Dorothy Russell was able to find the causative lesion in each; but this immediately invites the question that Professor Russell has herself raised recently, how often does this occur and the cause is not discovered? An example of this happening is seen in another of the deaths in this series: a man died at the age of 27 from his first haemorrhage six years after the onset of focal fits. These had led to a craniotomy at which a left temporal malformation was disclosed but not disturbed; and you might think from the drawing in Fig. 8 that this would have been difficult to miss later. Nevertheless, at the autopsy, an experienced pathologist, who did not know of the operative findings, assumed that a tumour had been removed

earlier, and could find no evidence of its recurrence or any abnormality of the blood vessels. Conversely, death may occur from other causes, and malformations be discovered which have been unsuspected during life, as in the old lady of 86 already mentioned (Fig. 2); and in another patient, a Greek man of 53, who died from the effects of an associated glioma in the right frontal lobe—the malformation, a rather diffuse, but also a small-vessel one, being in the left occipital region. Or, death in a case known to have a malformation may be from a probably unassociated trouble, as in one man who died at 66 from bronchopneumonia a few

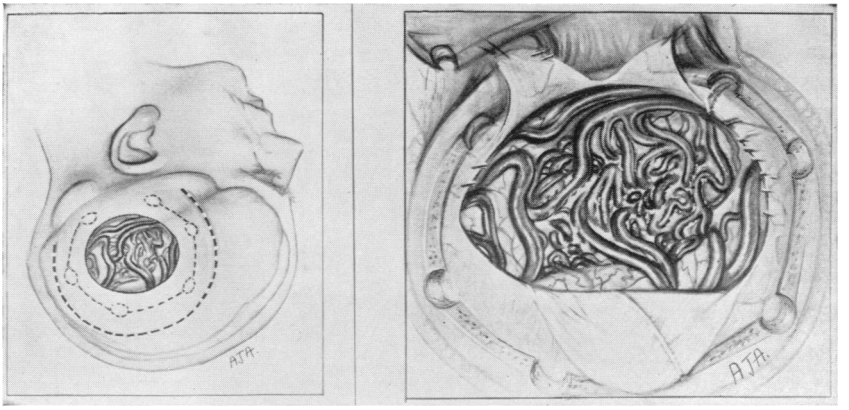


Fig. 8. Operation appearance of angiomatous malformation, later destroyed by a haemorrhage and not found at autopsy.

days after an operation for a perforated duodenal ulcer. 15 years earlier he had had a haemorrhage from a right Sylvian angiomatous malformation for which he received deep X-ray therapy; and for many years before that he had suffered from "bilious headaches." Unfortunately, there was no *post-mortem* examination of the brain.

Two patients died from their second haemorrhages: one, a girl of 18, two years after the first; and the other, a man of 29, six years after his first. Neither had had operations or radiotherapy. One man died from the last of several haemorrhages three years after a left parietal malformation had been exposed at operation, a few vessels tied, and radiotherapy given subsequently. Another, a man of 27, had a fatal haemorrhage during air encephalography which was being performed to discover the cause of intermittent attacks of left-sided weakness and paraesthesiae which he had had for six years. The malformation in this case was a basal one, involving the right anterior choroidal artery. Another, a man of 60 (referred to already) died from the effects of six episodes of intraventricular haemorrhage occurring over the space of four years. There was considerable internal hydrocephalus and dementia, and excision of the lesion latterly produced no benefit.

Lastly, there was a man who had had an episode of raised intracranial pressure at the age of 29, and another one 14 years later, when an angiomatous malformation invading the third ventricle from the region of the right thalamus was exposed at operation, but left untouched after biopsy. He survived, greatly disabled by a left hemiplegia and hemianaesthesia, for a further 15 years before he died in the last of several attacks of coma. Nowadays he might have had benefit from Torkildsen's operation; and it is of interest that in only one other of these patients who died from their malformations is radical operative treatment likely to have been possible without risking a severe disablement.

TABLE 2

TREATMENT, SURVIVAL AND DISABILITY

Method of of Treatment (after diagnosis)	No. of cases	Survival after starting treatment (years)				Final Disability				Deaths
		0-5	5-10	10-20	20-30	nil	slight	mod.	sev.	
Symptomatic	38	28	7	3	—	14	11	8	3	7
Radio-therapy	10	2	1	3	4	2	3	2	2	1
Radical Excision	7	6	1	—	—	3	3	—	1	1

Prognosis in Relation to Treatment

The final aspect I wish to consider is that outlined in Table 2, which attempts to assess the influence of treatment on the survival and disability of these people. The survival period in each group starts after the diagnosis has been made, so that the symptomatic treatment group (some of which had previously received medical treatment, e.g., for undiagnosed epilepsy) shall correspond more closely to those receiving radiotherapy or radical surgery, in whom the diagnosis has, of course, been made. Symptomatic treatment has usually meant anticonvulsant drugs, but I have included also minor surgical assaults on the lesion, such as vessel ligation or clot removal, the malformation still remaining afterwards.

Unfortunately, the figures for the results of the three main methods of treatment are not comparable—one must, of course, have both large numbers of patients and long "follow-ups"; and here, where there is one there is not the other. In the early days, the majority received radiotherapy, but follow-up periods for the other methods are in comparison too short, particularly those for radical excision. Nevertheless, I would draw your attention to the radiotherapy group; and although there are only 10 cases it is difficult, I think, not to be a little impressed by the long-term results of this method, which is now unfashionable. Only one is dead (he lived for less than five years after treatment), but four of the 10

have survived for more than 20 years since their treatment and half have only slight, or no, disability. Two might even be presumed cured (one cannot, of course, tell in such a drawn-out disease) : one, a man of 24, had a large intracerebral clot evacuated by Mr. Cairns in 1928, when a small " racemose angioma " was left undisturbed in the wall of the haematoma cavity. Deep X-ray therapy was given post-operatively, and there have been no further symptoms referable to this lesion, and no increase in his right hemiparesis in the 26 years that have elapsed. Another patient had a right temporal craniotomy by Sir Percy Sargent at which a " haemangioma " was exposed. Mr. Stanford Cade at that time (in 1931) gave her radium by means of a sorbo helmet, and she has been well during the subsequent 22 years.

Now these results with radiotherapy, for what they are worth statistically, were produced mostly by methods that were in vogue 20 years ago—all but Sir Stanford Cade's case received X-rays at 250 kilovolts. Greater accuracy should be possible now, and it would, on this suggestive evidence, seem wrong to deny any chance that this method of treatment might give to a patient with a lesion that is for any reason considered unsuitable for radical excision.

To sum up, then : these malformations do not necessarily cause early death or disability, but if death from haemorrhage is to occur, it is most likely within 10 years of the onset of symptoms. Useful lives may, however, be led for very many years after symptoms and complications have occurred, and this despite a usually early onset of symptoms, often in childhood. Headache as a presenting symptom augurs well ; and haemorrhage (occurring particularly from the smaller lesions) is earlier and more benign than that from a " berry " aneurysm ; it may be survived many times. The epilepsy associated with these malformations may be particularly capricious. Mental disturbance was not a marked feature in this series, and it was not clear to what extent it should be attributed to the lesion. Finally, a plea is made for the reconsideration of radiotherapy, particularly for those cases unsuitable for excision.

In dividing this lecture into two parts, I had hoped to avoid boring you with a large number of tables and figures, which I personally feel are best kept to the minimum in a lecture. Nevertheless I have had in mind the advice of an Oxford mathematician given nearly 70 years ago, and which I will quote in concluding. It runs, " . . . if you want to inspire confidence, give plenty of statistics. It does not matter that they should be accurate, or even intelligible, so long as there is enough of them. . . . Circulate some abstruse tables of figures particularly if printed in lines and columns, so that ordinary readers can make nothing of them, and all is changed at once. ' Oh, go on, go on ! ', they cry, satiated with facts." The author of that quotation was the Rev. C. L. Dodgson, better known as Lewis Carroll.

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