THE NATURAL HISTORY OF AMAUROSIS FUGAX

BY

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IN 1952 Fisher called attention to the association of transient monocular visual loss (amaurosis fugax) and contralateral hemiplegia in patients with stenosis or occlusion of the internal carotid artery and described seven personal cases. He also made an exhaustive survey of the literature of the preceding one hundred years and found 150 cases of transient monocular blindness. In this material he discerned seven groups of cases: amaurosis fugax occurring in older patients with atherosclerosis; in younger people without apparent cause; in association with migraine, Raynaud's phenomenon and heart disease; reflex amaurosis fugax and finally a miscellaneous group. The majority of cases appeared to be due to retinal ischæmia and, on the basis of the then available evidence, Fisher formed the opinion that the intermittent ischæmia was the result of vasospasm.

Seven years later Fisher (1959) was able to examine the retina of a patient during a transient attack of monocular blindness and observed the passage of white bodies through the retinal arteries. These bodies impacted for a time at arterial bifurcations, then fragmented and moved on, their disappearance coinciding with the recovery of vision. Fisher formed the conclusion that they were emboli and that amaurosis fugax could be due to the temporary interruption of the retinal circulation during the passage of emboli through the vessels.

Hollenhorst (1960) found that amaurosis fugax was a symptom in no less than 50 of 124 cases of carotid artery disease that he examined. He also reported (1961) seeing bright, refractile, yellow plaques in the retinal arterioles of 27 in a series of 235 patients described as having disease of the carotid arterial system; some of the plaques were observed to move

distally and others to fragment. In five of the patients who underwent carotid endarterectomy further plaques appeared in the retina during the operation, showers of plaques appearing in three of these cases. Hollenhorst also concluded that these plaques were emboli.

Since these initial descriptions a number of authors, including Ross Russell (1961) Ashby, Oakley, Lorentz and Scott (1963) and Balla, Howat and Walton (1964), have observed the presence of white or yellow refractile bodies in the retinal arteries in patients who had suffered attacks of amaurosis fugax. Moreover, the composition of these bodies has been established at autopsy and has been found to consist of agglutinations of platelets in one instance (McBrien, Bradley and Ashton, 1963), cholesterol esters in another (David, Klintworth, Friedberg and Dillon, 1963), and globules of neutral fat in a third (Cogan, Kuwabara and Moser, 1964). There is good evidence that the source of these emboli is commonly an atheromatous lesion at the origin of the internal carotid artery (Ross Russell, 1963, Gunning, Pickering, Robb-Smith and Ross Russell, 1964).

The passage of emboli through the retinal arterioles is not always associated with transient loss of vision, depending upon the size of vessel involved and the degree to which it is occluded. Skovborg and Lauritzen (1965) were fortunate to observe and photograph the retina during an attack of amaurosis fugax in such a case and Ross Russell (1963) observed impacted emboli in the retinal arterioles in patients who gave no history of visual disturbance. Furthermore, it is not in every case of amaurosis fugax that emboli can be seen, for Gerstenfeld (1964) and Dyll, Margolis and David (1966) have examined and photographed the fundus in two cases in which there was pallor of the retina and attenuation of the arterioles, followed by segmentation of the column of blood within the arterioles and darkening of the veins which in turn gave place to hyperæmia of the retina and dilatation of the veins; no emboli were observed throughout the attacks.

Amaurosis fugax can occur in conditions other than carotid artery disease, as was mentioned by Fisher in his original communication (1952) and stressed by Cogan (1961). The latter reported the occurrence of amaurosis fugax with and without papillœdema. It may also occur occasionally in migraine, as a premonitory symptom in central retinal artery occlusion and in giant-cell arteritis. The majority of cases, however, are associated with atherosclerosis. Hollenhorst (1966) reported 208 cases in which he observed bright refractile plaques in the retina, and stated that only 5 per cent were free from evidence of atherosclerosis. Sixty-three per cent of his patients had experienced a stroke or a transient ischæmic cerebrovascular attack within 10 days of the plaques being observed and, although Hollenhorst had not been able to make a systematic follow-up study of his patients, he knew of 23 who had died, the

majority from cardiovascular disease. It was the need for further information about the natural history of amaurosis fugax, and its association with other diseases, which prompted the present study.

MATERIAL

The series contained 80 patients who had experienced at least one attack of transient loss of vision in one or other eye. A transient loss of vision was defined for the purpose of this study as one that did not persist for more than four hours; in fact the duration of the visual loss was usually not more than a few minutes, but on occasion a patient complained that the vision was "not quite right" for some time after the period of complete loss.

The patients had been referred to one or other of us in the neurological clinic of an eye hospital, the neurological clinic of a general hospital, at a neurological hospital, or in private practice. Many had continued to be seen at regular intervals after the initial referral, but a special enquiry was made of all for the purpose of this study; in five instances we were unable to ascertain the ultimate outcome.

Because our particular interest was in the significance of transient loss of vision as a forerunner of either persisting visual loss, or hemiplegia, the patients were divided into three groups. Group I comprised the 67 patients who during the period of follow-up did not develop persisting visual loss or hemiplegia; Group II consisted of 9 patients who subsequently lost vision permanently in one eye; Group III contained five patients who went on to develop a contralateral hemiplegia. One male patient who developed both permanent visual loss and hemiplegia has been included in both Groups II and III, hence the total of 81.

RESULTS

The age and sex distribution of the series is given in Table I; attacks were uncommon in men below the age of 50 years but not in women, 45 per cent of the female patients being less than 50 years old. There was no significant age or sex difference between the three groups.

The duration of the history

The duration of the history up to the time of the study, the death of the patient, or the time at which he was lost sight of is given in Table II. The occurrence of permanent visual loss or hemiplegia was not a function of a more prolonged period of observation as the mean duration of follow-up for Groups I, II and III was 55, 51 and 24 months respectively.

TABLE I.—DISTRIBUTION OF PATIENTS ACCORDING TO AGE AND SEX

Age in years	*Males*	*Females*	*Total*
20—	—	1	1
30—	1	1	2
40—	6	9	15
50—	22	5	27
60—	22	5	27
70—	5	1	6
80—	1	1	2
Total	57	23	80

TABLE II.—TIME FROM FIRST ATTACK OF AMAUROSIS FUGAX TO DEATH OR FOLLOW-UP

Time	*Amaurosis fugax only*	*Patients who had Permanent visual loss*	*Hemiplegia*	*Total*
3 months	5	—	—	5
3 months or more	3	—	1	4
6 months or more	4	2	—	6
1 year or more	8	—	2	10
2 years or more	10	2	—	12
3 years or more	12	3	2	17
5 years or more	9	1	—	10
7 years or more	10	—	—	10
10 years or more	6	1	—	7
Total	67	9	5	81*

*One patient developed both permanent visual loss and hemiplegia.

Description of the attacks

Most commonly the patients described visual loss of sudden onset involving the entire field; the loss was usually complete, the description "completely black" being most often applied. Vision recovered after an interval of seconds or minutes, recovery taking somewhat longer than loss. Several variants of this typical story were encountered. Sometimes the visual loss was described as grey rather than black; this type of loss did not occur consistently in any one patient, attacks of "grey loss" being interspersed with others in which everything went black. Occasionally, the vision was described as becomming blurred, these attacks also being interspersed with episodes of more severe visual impairment; patients who complained only of attacks of blurred vision were not included because of the difficulty of being certain of the nosology of their attacks.

The visual loss did not always involve the whole field simultaneously. One patient, for example, experienced nine attacks all of which began with the appearance of a black spot in the centre of her right visual field, the visual loss then extending to the entire field within thirty seconds. Other patients described the development of the visual loss as like a shutter coming from above downwards or from below upwards, the development of the loss being completed in not more than thirty seconds. The visual loss did not always extend to the entire field; attacks consisting throughout of scotomata and altitudinal and segmental areas of loss were described.

TABLE III.—DURATION OF THE ATTACKS

Time	*Amaurosis fugax only*	*Patients who had Permanent visual loss*	*Hemiplegia*	*Total*
5 mins. or less	29	4	3	36
10 mins. or less	11	2	—	13
15 mins. or less	3	1	—	4
30 mins. or less	8	1	2	11
1 hour or less	8	1	—	9
2 hours or less	8	—	—	8
Total	67	9	5	81

Duration of the attacks

The duration of the attacks is given in Table III. It was sometimes difficult to determine the duration, especially in those patients in whom the amaurosis was followed by a period in which the vision was felt to be "not quite right". The total period of altered vision was used in preparing the table. In the case of those attacks lasting one or two hours there were usually two phases, an initial period of complete loss of vision followed by a more prolonged period of altered vision. The duration of the attacks of amaurosis in 6 of the 9 patients who subsequently developed permanent visual loss was 10 minutes or less, there being no suggestion that more prolonged attacks were more likely to be followed by permanent visual loss. Similarly the attacks of amaurosis lasted 5 minutes or less in 3 of the 5 patients who subsequently developed a hemiplegia.

Number of attacks

The total number of attacks experienced by the patients is given in Table IV. Most commonly patients experienced not more than 10 to 20 attacks. Patients who experienced large numbers of attacks were very uncommon, there being only two examples in the series.

TABLE IV.—TOTAL NO. OF ATTACKS OF AMAUROSIS FUGAX

No. of attacks	Amaurosis fugax only	Patients who had Permanent visual loss	Hemiplegia	Total
1	21	2	1	24
2	9	2	1	12
3	4	2	—	6
4	7	—	—	7
5–9	6	1	1	8
10–19	6	1	2	9
20–	6	—	—	6
uncertain	8	1	—	9
Total	67	9	5	81

Case Report C.V. No. 602. This was a man of 75 years who, at the time he was first seen, gave a six month history of attacks of complete loss of vision in the left eye each of five minutes' duration. These attacks had gradually been increasing in frequency. The fundi, which showed some degree of silver-wiring of the arteries, were never observed during an attack. The patient had a harsh, well-localised systolic bruit over the bifurcation of the left common carotid artery. A diagnosis of carotid stenosis was made but, because of his age, angiography was not performed nor anticoagulants given. He was followed over a period of ten months during which time the attacks continued to increase in frequency, several occurring each week. Finally, following one attack, vision did not recover; the bruit which had been present up to that time could no longer be heard. It was presumed that the putative stenosis had become an occlusion.

Case Report C.V. No. 962. The second patient was a woman of 58 years who over the course of six weeks experienced on average three attacks per day of loss of vision in the right eye, each of ten minutes' duration. They then ceased spontaneously and over a period of follow-up of fifteen months she had no recurrence.

TABLE V.—DURATION OF OCCURRENCE OF ATTACKS OF AMAUROSIS FUGAX

Time	Amaurosis fugax only	Patients who had Permanent visual loss	Hemiplegia	Total
Single attack	17	2	1	20
1 week or less	5	2	—	7
1 month or less	12	2	1	15
3 months or less	5	2	1	8
1 year or less	13	1	—	14
3 years or less	9	—	1	10
5 years or less	2	—	—	2
10 years or less	4	—	1	5
Total	67	9	5	81

Duration of occurrence of attacks

The length of time during which the patients experienced attacks was so variable that the tabulation of the data in Table V cannot convey an adequate picture of the pattern; this table has therefore been supplemented by some illustrative case histories.

One end of the spectrum is illustrated by the following case report.

Case Report C.V. No. 968. A woman of 44 years had a single attack of loss of vision in the left eye for two hours one morning. She was examined the same afternoon when a white body was seen at a bifurcation in the left superior nasal retinal artery. Three days later this was no longer visible. She had no bruits, no cardiac lesion and her blood pressure was 150/90. She was followed for six and a half years and has had no further trouble.

A contrast is provided by the next case.

Case Report C.V. No. 944. A man of 47 years had experienced three attacks of loss of vision in the right eye two years, one year and ten days before being seen, each lasting twenty minutes. He had no carotid bruit, the fundi were normal and his blood pressure was 125/90. He was followed for seven years and continued to have attacks but never more than three per year. His acuity remained at 6/9 in both eyes.

These two patients experienced isolated attacks, but others had several attacks within a short period.

Case Report C.V. No. 472. A man of 47 years had three episodes in one day, one the next day and a further attack on the third day; in each of these he lost the sight of the right eye for about a minute. He had no bruits, blood pressure was 125/70 and four-vessel angiography showed no evidence of a lesion. He had no further trouble for four years when he experienced one further attack. Over the next year up to the time of the study he was free from symptoms.

More numerous attacks occurring over a restricted period are illustrated by the following cases.

Case Report C.V. No. 973. A woman of 44 had up to a dozen attacks of loss of vision in the right eye daily for eleven consecutive days, with pain over the eye for most of this time. Each attack of amaurosis lasted "many seconds". She had a localized bruit over the right carotid artery in the neck and her blood pressure was 140/90. The attacks ceased spontaneously and she had no more in the next six years.

A second example is provided by the patient already described (Case Report C.V. No. 962) who had 3 attacks per day over a period of six weeks.

The retinal arteries

The retinal arteries were reported as presenting normal ophthalmoscopic appearances in 39 of the cases, Grade I changes (Keith-Wagener classification) in 26, Grade II in 8 and Grade III in only 1 case. The condition of

the arteries was not specifically mentioned in 7 cases but as other ophthalmoscopic findings were recorded, it can be presumed the arteries were normal or at most showed only Grade I changes. There was no difference between the groups in the degree of change in the retinal arteries.

Emboli were seen in the retinæ in five of the cases, details being given in Table VI. It will be seen that the attacks of amaurosis tended to be longer than average.

TABLE VI.—DETAILS OF FIVE CASES IN WHICH EMBOLI WERE SEEN

C.V. No.	*Age Sex*	*B.P.*	*Carotid bruit*	*No. of attacks*	*Duration of attacks*	*Angiography*	*Appearance of emboli*
765	68M	165/80	No	6	15 mins.	Carotid stenosis	White
710	63M	130/70	Yes	2	20 mins.	Innominate stenosis	Yellow refractile
906	55M	220/130	No	2	1 hour	Normal	Yellow refractile
941	58M	160/110	Yes	1	2 hours	Normal	Yellow refractile
968	44F	150/90	No	1	2 hours	Not done	White

Blood pressure

The distribution of the diastolic blood pressure is given in Table VII. The frequency of higher blood pressures was rather less than in most patients with cerebrovascular disease where about a third of patients may have a diastolic pressure of 110 mm. Hg. or above (Hill, Marshall and Shaw, 1960), compared with only a quarter of the present series. There was no difference in the distribution between the three groups.

TABLE VII.—DISTRIBUTION OF DIASTOLIC BLOOD PRESSURE

Diastolic B.P. in mm. Hg.	*Males*	*Females*	*Total*
60—	1	—	1
70—	4	3	7
80—	14	2	16
90—	13	10	23
100—	13	1	14
110—	3	1	4
120—	1	1	2
130—140	3	2	5
170—	1	—	1
Total	53	20	73*

*Seven no record of blood pressure.

Carotid bruit

The occurrence of bruits over the carotid artery in the neck is listed in Table VIII. Seventeen of the 80 patients had bruits, 16 of these being on the side of the amaurosis, one on the contralateral side. They were no more common in those who developed permanent loss of vision or hemiplegia than in the rest.

TABLE VIII.—THE OCCURRENCE OF CAROTID BRUITS IN THE NECK

	Amaurosis fugax only	*Patients who had Permanent visual loss*	*Hemiplegia*	*Total*
No bruit	54	7	3	64
Bruit ipsilateral to amaurosis	12	2	2	16
Bruit contralateral to amaurosis	1	—	—	1
Total	67	9	5	81

Results of angiography

Angiography was carried out in 27 of the 80 patients, the results being presented in Table IX. As the series was not part of a prospective study, the criteria on which patients were selected for angiography cannot be defined. The physician clearly thought there may have been a lesion of the carotid artery amenable to surgery, but in retrospect it is difficult to determine, and even more so to tabulate, the reasons for this expectation. The findings at angiography cannot therefore be taken to indicate the incidence of angiographic lesions in an unselected series of cases of amaurosis fugax. Sixteen of the 27 patients (55 per cent) showed stenosis or occlusion of the carotid artery, which is much higher than would be found, for example, in an unselected series of acute strokes where the incidence is about 17·0 per cent (Bull, Marshall and Shaw, 1960).

TABLE IX.—RESULTS OF ANGIOGRAPHY IN 27 PATIENTS

	Amaurosis fugax only	*Patients who had Permanent visual loss*	*Hemiplegia*	*Total*
Normal angiogram	10	—	1	11
Carotid stenosis	7	2	2	11
Carotid occlusion	4	—	1	5
Total	21	2	4	27

Cause of death

Ten of the male patients and nine of the females had died by the time of follow-up. Three of these deaths were due to myocardial infarction, two to cardiac failure, one to cerebral infarction, three to carcinoma, and one from complications following removal of a meningioma. The history of the last patient is worth recording.

Case Report C.V. No. 938. A man of 74 years experienced loss of vision in the right eye for one hour in January 1961. He was seen eight days after this incident at which time he was found to have heavy glycosuria, a blood pressure of 170/100, bilateral cataracts, no carotid bruits and no other abnormal neurological signs. Treatment for his diabetes mellitus was started with diet and Tolbutamide. He had no further trouble until July 1961, when he noticed the gradual onset of weakness of the left hand and dragging of the left leg over a few days. Bilateral carotid angiography was carried out; this showed no evidence of carotid stenosis but the mid-line vessels were displaced 5 mm. to the left and the right pericallosal artery was depressed. A parasagittal meningioma was successfully removed by Mr. Wylie McKissock; the patient died a month later of bronchopneumonia; there was no autopsy.

Patients who developed permanent visual loss

The characteristics of the amaurosis in the patients who developed permanent visual loss have been grouped in Table X to see if an overall pattern can be discerned. Only two of the patients had a carotid angiogram performed (Cases C.V. Nos. 793, 970) but both showed a carotid stenosis in the neck and one of these patients (C.V. No. 793) developed a hemiplegia one week later.

TABLE X.—DETAILS OF NINE CASES IN WHICH PERMANENT LOSS OF VISION OCCURRED

C.V. No.	*Age Sex*	*B.P.*	*Duration of attacks*	*Attack Persistent*	*Time from first attack*	*Carotid bruit*
971	67M	190/110	5 mins.	3rd	3½ hours	0
999	60M	160/100	30 mins.	2nd	3 months	0
793	54M	160/95	5 mins.	5th	5 weeks	0
602	75M	115/80	5 mins.	50+	14 months	+
998	33M	120/80	1 hour	2nd	1 day	0
997	57M	300/170	minutes	3rd	1 day	0
996	61M	140/70	10 mins.	after several	5 months	0
1000	68M	130/70	10 mins.	2nd	1 year	+
970	42F	260/130	6 mins.	3rd	10 days	+

The onset of the permanent visual loss was quite unpredictable as is illustrated by these two case reports.

Case Report C.V. No. 970. A woman of 42 years had one attack of amaurosis fugax in the left eye lasting for six minutes, a second lasting fifteen minutes a few days later and finally a third with no recovery of her sight ten days after the first attack. Her blood pressure was 260/130 and she had a left carotid bruit. An angiogram subsequent to the permanent visual loss showed a left carotid stenosis which was removed by endarterectomy. She had no further trouble during a follow-up period extending over six years.

Even more abrupt was the story of a man of 67 years.

Case Report C.V. No. 971. This man had his first attack of loss of vision in the right eye at 11 a.m.; this persisted for five minutes. At 1 p.m. he had a second attack of the same duration; at 2.30 p.m. he had a third attack from which he did not recover his sight. His blood pressure was 190/110 and he had no bruits. He was followed over two and a half years without any further symptoms appearing.

In contrast is the case already reported (C.V. No. 602) of the man who had over 50 attacks over a period of 14 months before suffering permanent visual loss.

Patients who developed hemiplegia

Five of the patients (3 males, 2 females) developed a persisting hemiplegia; details of these patients are given in Table XI. Illustrative case histories were:

TABLE XI.—DETAILS OF FIVE CASES IN WHICH HEMIPLEGIA DEVELOPED

C.V. No.	*Age*	*Sex*	*B.P.*	*Duration of attacks*	*No. of attacks before hemiplegia*	*Time from first attack*	*Preceding transient hemiplegia*	*Carotid bruit*	*Angiog.*
793	54	M	160/95	5 mins.	5	6 weeks	Yes	0	C.S.
940	71	M	250/120	5 mins.	10	17 days	Yes	+	—
676	69	M	140/90	10 mins.	2	18 months	No	+	C.O.
512	70	F	195/100	4 mins.	12	6 months	Yes	0	C.S.
823	54	F	230/120	30 mins.	1	6 months	No	0	—

C.S. internal carotid stenosis. C.O. internal carotid occlusion.

Case Report C.V. No. 940. A man of 71 years experienced 10 attacks of loss of vision in the right eye for five minutes or so over a period of fourteen days. On the fourteenth day he experienced weakness and numbness of the left upper limb for ten minutes. He was admitted to hospital on the seventeenth day, at which time he had a localized carotid bruit on the right and slow atrial fibrillation. The same evening he suddenly developed a dense left hemiplegia and the bruit was no longer audible.

This patient had a number of attacks of amaurosis before his hemiplegia but another man of 69 years (Case Report C.V. No. 676) had one attack of visual loss lasting 30 minutes on the right. Six months later he had a second attack lasting 5 minutes; he had a right carotid bruit. One year later he developed a left hemiparesis.

The effects of anticoagulant therapy

Sixteen of the patients received anticoagulant therapy at some stage, the results of this treatment being given in Table XII. This was in no sense a controlled trial but, as can be seen, 4 of the 16 patients had further attacks whilst on treatment whereas only 10 of the remaining 64 patients experienced further attacks over a much longer period of observation.

TABLE XII.—RESULTS OF ANTICOAGULANT THERAPY

	Treated	*Not treated*	*Total*
Further attacks of amaurosis fugax	4	10	14
No further attacks	12	54	66
Total	16	64	80

There was however one experience which suggested that anticoagulants were of benefit in one patient at least.

Case Report C.V. No. 580. A man of 67 years had experienced attacks of loss of vision in the left eye each lasting thirty minutes at approximately monthly intervals over the preceding five years. His blood pressure was 150/90, he had no carotid bruit and no history of transient hemiparesis. A left carotid angiogram showed stenosis of the artery in the neck and also in the siphon. As this lesion was beyond the reach of the surgeon, he was treated with anticoagulants for a year in which time he had no more attacks. The anticoagulants were then stopped and he had six attacks within four weeks, so they were resumed for a further five years by which time he was 73 years old. During this time he had no further attacks of amaurosis and no ischæmic episodes of any other kind. The anticoagulants were then withdrawn again and in the next three months he had no trouble.

DISCUSSION

One of the problems of a natural history study is to ensure that the series of patients is representative. There can only be complete assurance on this point when, either cases are notified by all the general practitioners in a geographical area, or a field survey involving strenuous efforts to secure a high level of ascertainment is made (Brewis, Poskanzer, Rolland and Miller, 1966). These approaches are not practicable for conditions which are relatively rare and which do not excite the interest and concern of the community. Resort has then to be made to hospital records with the danger of non-representative sampling.

This source of error was minimized in this study of amaurosis fugax by drawing the cases from four sources which seemed likely to cover the various modes of presentation, namely an eye hospital, a neurological

hospital, a general hospital and private practice. The necessity of doing this was shown by the fact that there was not a single instance of associated transient hemiparesis among the cases referred to the eye hospital, all of these patients having been referred to the neurological or general hospital. A series of patients obtained from any one of these sources would have given a misleading picture of the total spectrum of amaurosis fugax.

There were a number of features which clearly reflected the relationship of amaurosis fugax to vascular disease, particularly of the carotid artery. The presence of bruits in the neck in 20 per cent of the cases, the occurrence of transient or permanent hemiparesis in 10 of the 80 cases, the finding of stenosis or occlusion of the carotid artery at angiography in 16 out of 27 cases, and the fact that there were two and a half as many men as women in the series—atherosclerosis being commoner among men than among women—all pointed to this.

There was one feature at variance with this, namely the fact that 11 of the 23 female patients were under the age of 50 years. Arteriosclerosis of sufficient severity to be manifest clinically is uncommon below this age, especially in women. This raises the question as to whether their attacks were due to some other cause. Migraine is often put forward as an explanation for attacks of this kind, though often on tenuous grounds. Only two of these patients had headache associated with their amaurosis, none had experienced more typical attacks of migraine in the past, and all lacked a positive family history. Of the two that experienced associated pain, one described in Case Report C.V. No. 973, had more or less continuous pain for 11 days during which time she was experiencing up to a dozen attacks of amaurosis a day, each lasting many seconds. The pain and attacks of amaurosis ceased and did not recur during a period of six years. This history is most unlike that encountered in migraine. The second patient was the one (Case Report C.V. No. 968) in whom an embolus was seen in the retina shortly after her attack of amaurosis.

Formerly, rheumatic endocarditis could reasonably have been suggested as a possible factor in this type of case, but the decreasing frequency of this condition in the population, and the absence of any history or evidence of rheumatic endocarditis in these patients makes this suggestion untenable. It is clear that amaurosis fugax in younger women is a phenomenon deserving of further study.

The mechanism of many of the attacks of amaurosis is undoubtedly embolic, as the passage of bodies of various kinds through the retinal arteries has now been seen often enough in the attacks to place the matter beyond doubt. A common source of these emboli is the atheromatous

lesion which can frequently be found at the origin of the internal carotid artery in the neck (Gunning, Pickering, Robb-Smith and Ross Russell, 1964). That this is not the only source is shown by those cases in which angiography fails to reveal a lesion at this site. The possibility that minimal lesions too slight to be distinguished by angiography, but sufficient to provide a nidus for the formation of thrombus with subsequent embolization, cannot be excluded. Lesions at other sites in the vascular tree must also be considered; for example, angiographically demonstrable lesions in the innominate artery are by no means rare.

Another possible source of emboli is within the ophthalmic or central retinal arteries. These arteries are of much smaller calibre than the carotid, and so are more liable to be affected by the medial hypertrophy associated with hypertension. It was thought before the study was begun that amaurosis fugax might occur in two types of case; one was the normotensive patient with carotid artery disease, the other the hypertensive patient with disease of smaller arteries. In the event, such a distinction did not emerge, but as there were so few patients with high blood pressure this hypothesis is deserving of further study.

Although the association between amaurosis fugax and disease of the carotid artery was clearly established, no clues emerged as to which patients were likely to develop the more serious complications of permanent visual loss or hemiplegia. Neither the duration, total number, frequency, or mode of development of the attacks provided any indication as to which patients were most at risk. As Tables X and XI show, monocular blindness and hemiplegia followed short and long attacks of amaurosis, few and many, a history of only hours and one of many months. Such clues as were present were provided, not by features of the amaurosis fugax itself, but by associated features; patients experiencing transient hemiparesis and those with bruits were more liable to develop permanent weakness than others. These features did not help with forecasting permanent visual loss, which remained entirely unpredictable.

It therefore seems wise to examine many of these patients by angiography so as to detect and remove stenoses before serious damage occurs. The facts that 16 per cent of the series developed one, or other, or both, of the serious complications of monocular blindness or hemiplegia, and that the number of lesions found at angiography was high, indicates the wisdom of this course. The empirical administration of anticoagulants does not seem justified on the basis of the, admittedly inadequate, evidence provided by this study.

Whether endarterectomy has a marked influence upon the prognosis of transient ischæmic attacks in the retina or brain is still the subject of study by controlled trials (Bauer, Meyer, Gotham and Gilroy, 1966). Clinical

impression favours this procedure and until further evidence is available it would seem to be the appropriate treatment for those patients with amaurosis fugax in whom stenosis of the internal carotid artery has been demonstrated.

SUMMARY

Eighty cases of amaurosis fugax have been studied with regard to the frequency and duration of their attacks and the presence of associated evidence of vascular disease such as hypertension and carotid bruits. Sixteen of the 27 patients in whom angiography was performed had stenosis or occlusion of the carotid artery. Nine patients in the entire series developed permanent visual loss and five hemiplegia during the period of follow-up.

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