West Indian amblyopia

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Summary

A series of 21 patients admitted to St Thomas' Hospital, Medical Ophthalmology Unit, with a diagnosis of West Indian or West African amblyopia is reported. Patients were investigated for haematological, biochemical, serological, and radiological abnormalities and particular attention was paid to dietary history. Patients admitted in recent years also underwent neurophysiological investigations. No definite correlation between visual loss and dietary or family history was found, and there was no evidence that the improvement in vision which occurred in just under half the patients on follow-up was related to treatment with hydroxocobalamin or multivitamins. Visual-evoked responses in 4 patients showed a prolonged latency suggesting optic nerve demyelination, while in only one case was the electro-oculogram definitely subnormal. These findings contrast with those in 'toxic' amblyopias and suggest that the syndrome of West Indian amblyopia may be due to bilateral optic nerve demyelination of unknown aetiology rather than the effect of toxic substances or nutritional deficiency on the retina.

Introduction

Visual loss affecting the populations of tropical areas, particularly the West Indies and Caribbean Islands, has been described since the mid 19th century. In more recent years, detailed studies in Jamaica (Degazon, 1956; Whitbourne, 1947) and Nigeria (Osuntokun and Osuntokun, 1971) have delineated a clinical entity consisting of bilateral visual loss with pallor of all or part of the optic discs; in both countries, these findings are also seen as part of a more generalized neurological disorder.

With the increase of emigration from these countries to the U.K. since the last World War, there have been 4 series of patients with this condition reported from British hospitals (Crews, 1963; Owen, 1966; Behrman, 1962; MacKenzie and Philips, 1968). These paid particular regard to possible contributory factors but it is still not certain whether the condition was a form of nutri-

tional or toxic amblyopia or whether some other unidentified factor was operating.

The authors report a further series of patients, all of whom were admitted with this condition to the Medical Ophthalmology Unit of the Royal Eye, Lambeth and St Thomas' Hospitals. This series includes patients from West Africa since they present a very similar clinical picture to those from the West Indies. In an attempt to determine whether there was evidence of underlying toxic factors or nutritional deficiency, they were investigated for haematological, biochemical, microbiological and radiological abnormalities (as in previous series) but also recently developed electrodiagnostic tests to assess retinal and optic nerve function. The authors also report a follow-up over a prolonged period to determine the natural history of this condition and the effect, if any, of various treatments.

Methods

The Medical Ophthalmology Unit was opened in 1965 and, in those 15 years, 21 patients of West Indian or West African origin were admitted to the Unit with a diagnosis of 'West Indian' or 'Tropical' amblyopia. Eight further patients were eliminated from the study as their history, clinical features and results of investigations indicated other possible causes for the visual loss, e.g. heavy alcohol or tobacco consumption, vitamin B₁₂ deficiency, iron-deficiency anaemia, diabetes, and pigmentary abnormalities of the retina. Another patient was eliminated from the study as her visual loss occurred during a severe febrile illness following childbirth.

Besides a detailed history of visual loss and other symptoms, the patients were asked about possible cassava and bush tea consumption. As well as a full ophthalmological examination (which included visual field charting by one or more of Bjerrum Screen, Friedmann Central Field Analyser, and Goldmann Perimeter) a general physical examination was carried out with particular regard to the nervous system. Patients had a full blood count and sickle test, and serum B₁₂ estimations. Routine biochemical profiles included tests of renal and hepatic function and protein levels. All patients were investigated

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TABLE 1. Findings at presentation of the 21 patients studied in this series

Case no.	Sex	Age at onset (years)	Age at presentation (years)	Years in U.K. at presentation	Corrected visual acuity		Visual fields		Optic discs		Treponemal serology in blood	Possible associated factors
					R	L	R	L	R	L		
1	M	8	9	5	6/24	6/36	C	C	TP	TP	Negative	Brother of case 2
2	M	10	12	5	6/24	6/24	Minimal constriction		TP	TP	Negative	Brother of case 1
3	M	32	42	5	6/24	6/24	C	C	TP	TP	Negative	Drank bush tea in West Indies
4	M	13	33	9	6/60	6/36	C	C	TP	TP	Negative	Drank bush tea in West Indies
5	M	16	28	Not known	6/18	6/12	Minimal constriction		TP	TP	Negative	Smallpox in childhood
6	M	28	29	2	5/60	4/60	C	C	TP	TP	Negative	Drank bush tea in West Indies
7	M	19	36	16	3/60	3/60	Generalized depression		TP	TP	Negative	Drank bush tea in West Indies
8	F	50	58	3	6/36	6/36	No scotoma		OA	OA	Negative	Drank bush tea; Hypertensive
9	M	18	33	2	6/24	6/60	C	C	TP	TP	Negative	Occasional bush tea in West Indies
10	F	48	50	9	6/36	6/36	Minimal C		TP	TP	Negative	Sickle cell trait
11	F	9	10	1-5	6/24	6/24	C	C	Marked cupping		Negative	
12	F	44	45	2-5	6/60	6/36	C	C	TP	TP	Negative	Yaws in childhood;
13	M	16	51	21	6/24	6/24	C	C	TP	TP	TPHA Positive 1 in 80	Ate cassava in West Indies
14	M	50	58	19	6/18	6/24	C	C	TP	TP	Negative	Ate cassava in West Indies; Hypertensive
15	M	35	35	16	6/18	6/12	C	CC	TP	TP	Negative	Sickle cell trait
16	F	59	64	17	6/36	6/24	C	C	OA	OA	Positive TPHA	Impaired glucose tolerance test
17	F	16	36	6	6/36	6/36	C	C	OA	OA	FTA Negative	Chickenpox at onset;
18	M	38	58	25	6/36	6/36	CC	CC	OA	OA	FTA	Hypertensive
19	F	42	62	20	3/60	6/60	C	C	TP	TP	Positive TPHA	Ate cassava in West Indies
20	M	36	36	20	6/9	6/9	C	C	Hyperaemic		FTA-IgM Negative	Hypertensive; Bilateral nerve deafness
21	M	18	41	10	3/60	3/60	C	C	OA	OA	Negative	

N = normal; C = central scotoma; CC = centro-caecal scotoma; TP = temporal pallor; OA = optic atrophy; TPHA = treponemal haemagglutinin
FTA = fluorescent treponemal antibody.

for treponemal serology and most patients had lumbar punctures with routine analysis of CSF. Routine radiological investigations were chest and skull X-rays with particular attention paid to the area of the pituitary. Patients investigated in recent years had electro-oculograms, electro-retinograms and visual-evoked responses (VER).

Results (Table 1)

The series comprised 14 males and 7 females, a male : female ratio of 2 : 1. With regard to age of onset, the male patients appeared to fall into 2 groups, about half noticing the onset of visual loss in their 'teens or earlier, while in the others the age of onset was generally in the 4th and 5th decades. Two females dated the onset at 9 and 16 years, others noted the start of symptoms in the 5th and 6th decades, one patient as late as 59 years. All came from the West Indies or Caribbean, except 2 who came from West Africa. In 11 patients, symptoms commenced after arriving in the U.K. whilst in 9 they commenced in the country of origin. In one case there was no record of the date of arrival in the U.K.

Possible associated factors

Seven patients admitted to having consumed cassava or bush teas in their country of origin, although the exact forms of these were not ascertained, but most of them emphasized that the quantities consumed were small. In one patient the onset of visual loss occurred at the time of an attack of chickenpox but no medical records of this episode were available.

Family history

The first 2 cases were of particular interest as they were brothers whose visual loss occurred within one year of each other. Their father had suffered a retinal

detachment but there was no other family history of visual loss. Of the other patients, only one knew of a relative suffering similar visual loss, although many of the patients had lost contact with most of their relatives after emigrating.

General examination

No abnormalities were found on physical examination in the majority of patients. Four patients were moderately hypertensive; these were all >40 years old when this incidence would not be considered unusual. Two patients had severe sensori-neural deafness. One of these had audiometry and auditory-evoked response (AER) tests, and the results were characteristic of the 'toxic' deafness found in people of African extraction.

Ophthalmic examination

In the majority of cases, the visual acuity was reduced to the same extent in both eyes. In about 50% of the cases, visual acuity was 6/24 or 6/36; a number of cases were more seriously affected but none worse than 3/60. Visual fields usually showed bilateral central scotomata although a few cases demonstrated centro-caecal scotomata, not necessarily in both eyes. Only 5 patients had classical optic atrophy with optic discs that were flat and pale. In most cases there was abnormal pallor of the temporal halves of the discs. A few patients, particularly those with longer histories, showed some narrowing of retinal vessels, but ophthalmic examination was otherwise normal.

Investigations

Haematology, biochemistry and radiology were normal except for 2 patients who were positive for sickle cell trait.

Treponemal serology. Four of the patients had positive treponemal haemagglutinin (TPHA) venereal

TABLE 2. Electrodiagnostic results on 4 patients in this series

Case no.	13 May 1977	13 Feb. 1979	17	18	20	Normal VER
EOG	—	Slightly subnormal	Normal	Subnormal	Slightly subnormal	
ERG	—	Subnormal	Slightly reduced	Subnormal	Subnormal	
VER	R 4.0 μ V 107 msec.	2.5 μ V 120 msec.	1.3 μ V 149 msec.	3 μ V broad 120–140 msec.	2.1 μ V 135 msec.	Amplitude of major positive peak over 7.5 μ V; Latency to the peak of major positive wave <115 msec; Interocular difference in peak latency <4 msec.
	L 3.8 μ V 110 msec.	2.8 μ V 170 msec.	1.5 μ V 137 msec.	3 μ V broad 120–140 msec.	2.0 μ V 137 msec.	
VER small	R 2.5 μ V 125 msec.	1.5 μ V 135 msec.	—	Abolished	1.6 μ V 155 msec.	
central pattern	L 2.0 μ V 125 msec.	1.5 μ V 135 msec.	—	Abolished	0 μ V	

VER = visual-evoked response.

disease, research laboratory (VDRL) or fluorescent treponemal antibody (FTA) tests in their blood, but there were no positive findings in CSF. One patient gave a history of yaws in childhood and one of several episodes of gonorrhoea although not, as far as he knew, syphilis; since this patient had a positive FTA-IgM test, he was considered to have active treponemal infection. There were no clinical stigmata of active primary, secondary or neuro-syphilis. The other 2 patients did not supply any relevant history.

Electrodiagnostic studies (Table 2). Four patients had electrodiagnostic studies comprising electro-oculograms (EOG), electro-retinograms (ERG), and VERs. Several other patients had these studies on older equipment for which control values had not been done; for this reason their results are not reported, as the findings are not comparable to those obtained on present-day equipment where the normal values are established.

In all cases, the VERs showed subnormal amplitude and delay, particularly obvious when a small central pattern was used. The ERG was reduced in all cases, although only slightly in one. The EOG was normal in one case, described as 'slightly subnormal' in 2, and unequivocally subnormal in one. One patient (Case 13) had repeat studies after a 22-month interval during which time his visual acuities had remained unchanged; the repeat VER showed a definite deterioration, in spite of the absence of any clinical change.

Follow-up treatment with hydroxocobalamin and multivitamins

Thirteen patients were followed-up for a period of 6 months or more, several of these were seen for > 2 years and one for 17 years.

The visual acuity of 2 patients deteriorated during the period of follow-up, one of these having received treatment with hydroxocobalamin and multivitamins; acuity improved in 6 patients, 3 of these having been treated. In the remaining 5, visual acuity remained the same and only one of these had received treatment. Apart from one patient who received a short course of oral steroids no forms of treatment were tried other than vitamins and hydroxycobalamin.

The most remarkable case was of a patient whose visual acuity improved from 6/60 to 6/5 in each eye over a period of 6 months without any treatment; as he returned to Nigeria at the end of this period it was not possible to follow him further.

Discussion

The clinical and ophthalmological features of the patients in this series correspond fairly well with others reported in the U.K. (Crews, 1963; Owen,

1966; Behrman, 1962; MacKenzie and Philips, 1968) and in their countries of origin (Degazon, 1956; Whitbourne, 1947; Osuntokun and Osuntokun, 1971; Moore, 1934; Clarke, 1936; Scott, 1918). The history of gradual deterioration of vision down to a stationary level with bitemporal optic disc pallor and central scotomata were common findings. Although some series (Whitbourne, 1947; Carroll, 1947) concentrated on visual loss in children, these were similar to the present cases where the age of onset was in the first 2 decades. In a larger series, Degazon (1956) found the highest incidence in the 3rd decade in men and the 5th decade in women, which is the same as in the present series but, unlike this and other series which showed a male preponderance, there was a roughly equal sex incidence.

Nutritional aspects

Although the syndrome of bilateral visual loss in tropical regions, commonly known as West Indian or West African amblyopia, has been extensively studied, its precise aetiology remains uncertain. Malnutrition or the consumption of cassava and bush tea have been thought to play a major part in the syndrome (Whitbourne, 1947; Osuntokun and Osuntokun, 1971; Moore, 1934; Clarke, 1936; Scott, 1918). Scott (1918) described a 'central neuritis of Jamaicans' in which other features, such as itching and burning of eyes and inflammatory lesions in the mouth, suggested that the visual loss was due to malnutrition. Whitbourne (1947) and Carroll (1947) also reported a high incidence of 'nutritional retrobulbar neuritis' in children who had many of the clinical features of vitamin B deficiency.

Following World War II, the idea that malnutrition was responsible for the visual loss was reinforced by studies on English survivors of prisoner of war camps. Spillane and Scott (1945) reported 112 cases, nearly all of whom had 'retrobulbar neuritis' and some had in addition nerve deafness and ataxia. Many of these patients also had evidence of pellagra and other nutritional deficiencies, and the severity of the symptoms was related to the length of time spent in the camp. Hobbs and Forbes (1946) found similar changes in prisoners of war from the Far East, but with less evidence of pellagra.

Degazon (1956) studied a large series in Kingston, Jamaica, and he excluded patients with a history of diabetes or heavy alcohol or tobacco consumption. Although the findings of profound visual loss, bitemporal disc pallor and central or para-central scotomata were similar to other series, he found little evidence of malnutrition, vitamin deficiencies or other coincidental illnesses. Several of his cases were relatively affluent private patients and, as their clinical features were similar to those of his other patients, he concluded that malnutrition

could not be a significant aetiological factor. In 10 of the present patients, visual loss was noticed after arrival in the U.K., in some cases 15 years after arrival; other U.K. studies have demonstrated a similar delay (Crews, 1963; Owen, 1966; Behrman, 1962; MacKenzie and Philips, 1968). There is no clinical evidence in any of these series of malnutrition, and a delayed effect from malnutrition in the home countries seems unlikely to cause such profound changes so many years later. If the syndrome were due to malnutrition, it would be expected that different patterns would emerge in those who had emigrated to the U.K. where the standard of nutrition is likely to be better even though migrants tend to be members of the lower socio-economic classes. It seems improbable, therefore, at least in the case of migrants to the U.K., that there is any simple direct relationship between malnutrition and visual loss. The evidence from prisoners of war (Spillane and Scott, 1945; Hobbs and Forbes, 1946) suggests that in those cases the visual loss is directly proportional to the degree and length of time of malnutrition.

Toxic factors

As long ago as 1934, Moore (1934) felt that optic atrophy in West Africa was due to cassava consumption and, in 1936, Clarke (1936) specifically considered that raised cyanide levels were responsible. Osuntokun and Osuntokun (1971) studied cassava and bush tea, both of which are cyanogenetic, i.e. they increase cyanide levels, especially when there is deficiency in vitamin B₁₂ and amino acids such as cystine, cysteine and glutathione which provide the sulphhydryl groups necessary for cyanide metabolism. Cyanide metabolism has been extensively studied in 'tobacco amblyopia' and Leber's optic atrophy and there is evidence particularly in the former of relatively inadequate cyanide breakdown which can be corrected by giving hydroxocobalamin and sulphhydryl supplying amino acids (Chisholm and Pettigrew, 1970; Phillips, Wang and Van Deburgh, 1970; Foulds *et al.*, 1970; Crews *et al.*, 1970). Cyanide can produce demyelination in experimental animals (Lessell, 1971) particularly in the optic nerves but a very high dose is needed which causes, in addition, widespread damage with a high fatality.

Osuntokun and Osuntokun (1971) demonstrated in patients with visual loss in the 'tropical neuropathy' syndrome, raised plasma thiocyanate and cyanide levels, raised urinary thiocyanate excretion together with reduced levels of hydroxocobalamin, cystine, cysteine and methionine. Seven of the present patients admitted to consuming cassava or bush tea in their native countries but considered that the quantities were minimal. Cassava and bush

tea are available in the U.K., but none of the patients admitted to consuming them. Owen (1966) studied 40 West Indians without visual impairment and found that 28 had drunk bush tea, several for many years.

In this series, there was no correlation with consumption of allegedly toxic foods as only one third of the patients admitted to doing so.

Neurological syndromes of West Indians and West Africans

In both Jamaica and West Africa, visual loss can occur as part of widespread neurological syndromes. The pathology of these cases is basically a chronic meningo-myelitis; nerve roots showed loss of myelin as did the optic and auditory nerves. Montgomery *et al.* (1964) studied 206 cases of Jamaican neuropathy; only 15% of their cases had an optic neuropathy and in these cases they described a background of 'poor nutrition'. In their series the majority of cases had positive treponemal tests in the blood, but only 6% were positive in the CSF. The incidence of the Jamaican neuropathy syndrome was much higher than that of neurosyphilis. None of the present patients had positive treponemal serology in the CSF and it seems improbable that treponemal infection (syphilis or yaws) is relevant in this condition.

Osuntokun and Osuntokun (1971) studied 308 Nigerian patients with tropical ataxic neuropathy, all of whom had bilateral optic atrophy together with sensori-neural deafness, panmyelinopathy and peripheral neuropathy; histology of the peripheral nerves showed areas of demyelination. The history of visual impairment and the appearance of the optic disc were similar to cases of tropical amblyopia, but central scotomata were only demonstrated in 16 out of 118 cases whose fields were plotted. They felt that the visual findings were different from the cases described in Jamaica.

Only 4 patients in the present series showed other neurological changes. Three patients, 2 of whom were West Indian and one West African, had sensori-neural deafness. Although the authors did not find generalized neurological abnormalities in their patients, the pathological findings in the other series mentioned may be useful in considering the underlying changes in the present patients. In both Jamaican and West African cases, demyelination was characteristically found in the optic nerves, and for this reason the electrodiagnostic findings are of relevance.

Electrodiagnostic findings (Table 2)

In all 4 patients in whom the VER was done, the most significant finding was a prolonged latency. This is a characteristic finding in optic nerve

demyelination and cannot be accounted for by purely retinal changes. There was also a subnormal amplitude of the VERs which suggest axonal as well as myelin involvement; the ERG in these cases was subnormal. Ikeda, Tremain and Sanders (1978) have suggested that the explanation for such cases is retrograde trans-neuronal 'degeneration'. In one of the patients in the present series the EOG was normal, in 2 reported as 'slightly subnormal', and in only one was it unequivocally subnormal. The reason for this was not clear but this could not account for the markedly delayed VER. In 'toxic' amblyopia, e.g. due to tobacco, the characteristic finding is a subnormal EOG, due to the 'toxic' effect on the retinal pigment epithelium, a consequent reduction in the ERG, and a VER with subnormal amplitude but *no delay* in the peak time; demyelination of the optic nerves, therefore, does not appear to play a part in these toxic amblyopias.

Genetic and racial factors

It was frequently difficult to obtain a reliable family history with immigrants as, so often, they have lost contact with a large part, if not all, of their family. Behrman (1962) considered that all external factors for this syndrome could be eliminated, and suggested a genetic aetiology, the mode of transmission producing visual loss in 1 in 16 of offspring. Other series in immigrants (Crews, 1963; Owen, 1966; MacKenzie and Philips, 1968) have not shown evidence of transmission in families and apart from 2 brothers and one other patient in the present series no positive family history was found. It is rare to find optic atrophy of unexplained aetiology in native whites since by definition this particular syndrome only occurs in the Negro race, and it is a circular argument to suggest racial susceptibility.

Results of treatment and follow-up

It has been thought previously that characteristically visual acuity reached a low point and thereafter remained static. Some patients, however, have shown improvement if treated early with B group vitamins and hydroxocobalamin, particularly those with definite evidence of malnutrition (Moore, 1934). In the 15 patients who were followed-up in this series, nearly 50% experienced an improvement in visual acuity but this did not appear to be related to treatment with vitamins, whereas 2 of the 3 patients whose vision had deteriorated had been so treated. These results seem to suggest that visual improvement or deterioration are independent of treatment with substances that would be expected to improve cyanide metabolism.

Conclusion

The particular value of studying patients who have immigrated to the U.K. is that these people have been removed from many of the environmental factors in their countries of origin which have been thought to play a part in aetiology. Crews (1963) pointed out that nutritional and personal habits common in the country of origin may be retained by individuals wherever they live, but there seems little doubt that emigration tends to raise the standard of living and to produce modifications of diet. In view of this, the finding of visual loss commencing many years after emigration makes a direct nutritional or 'toxic' cause unlikely.

In tobacco amblyopia, one of the most studied of the 'toxic' group, stopping smoking and administration of hydroxocobalamin has been shown to produce an improvement (Foulds *et al.*, 1970). In contrast, 3 of the 6 cases in this series which improved on follow-up did so without any apparent change in diet or administration of drugs.

The electrodiagnostic findings of delayed peak time of the VER do not accord with those found in toxic amblyopias (Ikeda *et al.*, 1978) attributed to defective cyanide metabolism and are against cassava and bush tea being responsible for tropical amblyopia.

A feature of peripheral and optic nerve pathology in patients with Jamaican and West African neuropathy is widespread demyelination, and this would certainly accord with the finding of delayed peak time of the VER. It is possible that the syndrome of West Indian and West African amblyopia may be due to bilateral optic nerve demyelination rather than a toxic effect on the retina.

Although the visual evoked responses are similar to those seen in optic neuritis (Ikeda *et al.*, 1978; Perkin and Rose, 1979) the clinical features (absence of pain, slow progression, and bilaterality) are against any relationship to this syndrome. Further study, with particular regard to electrodiagnostic and pathological findings, of such patients as well as those with Jamaican and West African neuropathy in the United Kingdom would be valuable.

Acknowledgments

We thank Dr Hisako Ikeda and her staff of the Vision Research Unit for carrying out the electrodiagnostic investigations, Miss Josephine Lace for her secretarial assistance, and our colleagues for referral of these patients.

References

- BEHRMAN, S. (1962) African race induced bilateral amblyopia among West Indian immigrants in the United Kingdom. *British Journal of Ophthalmology*, **46**, 554.
- CARROLL, F.J. (1947) Nutritional retrobulbar neuritis. *American Journal of Ophthalmology*, **30**, 172.
- CHISHOLM, I.A. & PETTIGREW, A.R. (1970) Biochemical

- observations in toxic optic neuropathy. *Transactions of the Ophthalmological Societies of the United Kingdom*, **90**, 827.
- CLARKE, A. (1936) Report on the effects of certain poisons contained in food plants of West Africa upon the health of the native races. *Journal of Tropical Medicine and Hygiene*, **38**, 269.
- CREWS, S.J. (1963) Bilateral amblyopia in West Indians. *Transactions of the Ophthalmological Societies of the United Kingdom*, **83**, 653.
- CREWS, S.J., JAMES, B., MARSTERS, J.D. & WEST, R.H. (1970) Drug and nutritional factors in optic neuropathy. *Transactions of the Ophthalmological Societies of the United Kingdom*, **90**, 773.
- DEGAZON, D.W. (1956) Tropical amblyopia in Jamaicans. *West Indian Medical Journal*, **5**, 223.
- FOULDS, W.S., CHISHOLM, I.A., BRONTE-STEWART, J. & REID, H.C.R. (1970) The investigation and therapy of the toxic amblyopias. *Transactions of the Ophthalmological Societies of the United Kingdom*, **90**, 739.
- HOBBS, A.E. & FORBES, F.A. (1946) Visual defects in prisoners-of-war from the Far East. *Lancet*, **ii**, 149.
- IKEDA, H., TREMAIN, K.E. & SANDERS, M.D. (1978) Neurophysiological investigation in optic nerve disease. Combined assessment of the visual evoked response and electroretinogram. *British Journal of Ophthalmology*, **62**, 227.
- LESSELL, S. (1971) Experimental cyanide optic neuropathy. *Archives of Ophthalmology*, **86**, 194.
- MACKENZIE, A.D. & PHILLIPS, C.I. (1968) West Indian amblyopia. *Brain*, **91**, 249.
- MONTGOMERY, R.D., CRUIKSHANK, E.K., ROBERTSON, W.D. & McMENEMY, W.H. (1964) Clinical and pathological observations on Jamaican neuropathy. *Brain*, **87**, 425.
- MOORE, D.G.F. (1934) Retrobulbar neuritis and partial optic atrophy as sequelae of avitaminosis. *Annals of Tropical Medicine and Parasitology*, **28**, 295.
- OSUNTOKUN, B.O. & OSUNTOKUN, O. (1971) Tropical amblyopia in Nigerians. *American Journal of Ophthalmology*, **72**, 708.
- OWEN, R.A. (1966) Defective vision in West Indian immigrants. *British Journal of Ophthalmology*, **50**, 561.
- PERKIN, G.D. & ROSE, F.C. (1979) *Optic Neuritis*, 1st Edn. Oxford University Press, Oxford.
- PHILLIPS, C.I., WANG, M.K. & VAN DEBURGH, P.F. (1970) Some observations on the mechanism of tobacco amblyopia and its treatment with sodium thiosulphate. *Transactions of the Ophthalmological Societies of the United Kingdom*, **90**, 809.
- SCOTT, H.H. (1918) An investigation into an acute outbreak of 'central neuritis'. *Annals of Tropical Medicine and Parasitology*, **12**, 109.
- SPILLANE, J. & SCOTT, G.I. (1945) Obscure neuropathy in the Middle East. *Lancet*, **ii**, 261.
- WHITBOURNE, D. (1947) Nutritional retrobulbar neuritis in children in Jamaica. *American Journal of Ophthalmology*, **30**, 161.