Table 1. *Incidence of spoke formation in schoolchildren in Tanganyika*

District	No. examined	Percentage of children with score*						
		0	1 or 2	3 or 4	5 or 6	7 or 8	9 or 10	11 or 12
Mwanza	551	21·6	17·9	22·5	17·6	10·2	7·2	3·0
Mvumi	869	14·8	16·2	32·7	23·6	9·4	2·9	0·3

* See page 79.

Though the mere presence or absence of spoke formation can be determined with considerable accuracy and observations are readily reproducible, the degree of change present can be estimated only in a very rough fashion, and the results shown in Table 1 are not suitable for detailed analysis. It does appear, however, that although the higher incidence of spoke formation was found in Mvumi ($\chi^2 = 10{\cdot}68$; $P \sim 0{\cdot}001$) there were more with the most marked degree of change in Mwanza.

At present it is impossible to say anything definite about the aetiology of these interesting lens changes. Further examinations are being carried out on children of other population groups, and although the work is incomplete it can be stated that European children of the same age and who have spent most of their life in Africa do not show the same changes. Histological and biochemical studies of the lenses of Africans which we have recently begun are in too early a stage to report upon. If this very high incidence of lens opacities at such an early age in the African is related to the eventual development of cataract in later years then it may constitute evidence in favour of the widely held belief that cataract occurs at an earlier age in the tropics than it does in some other parts of the world. Whether or not malnutrition is at work is an open question at the moment.

REFERENCES

Bagchi, K. (1959). *Indian J. med. Res.* **47**, 184.
Day, P. L., Langston, W. C. & O'Brien, C. S. (1931). *Amer. J. Ophthal.* **14**, 1005.
Hall, W. K., Bowles, L. L., Sydenstricker, V. P. & Schmidt, H. L. Jr. (1948). *J. Nutr.* **36**, 277.
McLaren, D. S. (1957). *Proc. Nutr. Soc.* **16**, xxiii.
McLaren, D. S. (1959). *Brit. J. Ophthal.* **43**, 78.
Pfeiffer, C. E. (1921). *v. Graefes Arch. Ophthal.* **107**, 71.
Totter, J. R. & Day, P. L. (1942). *J. Nutr.* **24**, 159.

Personal observations on some metabolic diseases of the eye

By F. C. RODGER, *University Laboratory of Physiology, Oxford*

The observations presented here were made by me as an oculist in tropical countries, dating back to 1945. These countries include Burma, India, Ceylon, Egypt, Ghana, Nigeria, the Cameroons and certain areas in central Africa under French control. The opportunity to witness a relationship between malnutrition and the eye has been unlimited as a result. The points now to be mentioned are all based on personal work—in some instances with collaborators.

The principle of a range of values in blood assays for vitamin A and carotenoids

Contrary to earlier opinions, individual blood values for vitamin A and the carotenoids appear to bear no relation to the health of the eye. Very low values may be associated with healthy eyes and non-defective rod thresholds (de Haas & Meulemans, 1938; Scrimshaw, Behar, Perez & Viteri, 1955; Sie, 1937; Rodger, Saiduzzafar, Grover & Fazal, unpublished). The statistical analysis of values for 162 subjects taken at random from a North Indian community, all with healthy eyes, revealed that the group mean values ranged from 58 to 108 i.u. vitamin A/100 ml plasma, and from 25 to 55 μg for the carotenoids. In the last-mentioned work (Rodger, Saiduzzafar *et al.*) the ranges of values for each of the ocular manifestations of vitamin A deficiency were compared. The ranges were as wide as in those with healthy eyes, but the important principle emerged that only when means are calculated may a relationship between a low blood value and the liability to any particular lesion of the eye emerge. The values for about 100 cases exhibiting Bitot's spots fell within the range of normal expectation. Cases with corneal xerosis revealed a very slight reduction in the vitamin A values. This is not surprising because the latter lesion commonly occurs as an isolated manifestation of avitaminosis A in a previously diseased eye, where there is interference with the limbal circulation, as in a chronic kerato-uveitis, or impaired corneal diffusion, as in trachoma, tuberculous keratitis, and similar conditions. In such circumstances, especially when the host is on the border-line of vitamin A deficiency, xerosis of the cornea develops, although the blood levels fall within our prescribed range of normal expectation. In this connexion I have found it possible to improve corneal translucency, and hence the vision, in cases of healed trachoma (trachoma grade IV) by giving massive vitamin A therapy.

When the blood levels for hemeralopia, xerophthalmia (that is, xerosis of the conjunctiva and cornea combined) and keratomalacia are plotted, consistently low values of vitamin A and the carotenoids and a small range are found, although there are a few inexplicable exceptions even here.

Bitot's spots

In view of the range of blood values already mentioned, of the total lack of a response in more than 100 cases to massive therapy, and of the complete absence of night-blindness in every one (as plotted on the Goldmann adaptometer), it is difficult to persist in classifying this lesion as a manifestation of avitaminosis A. There are two arguments usually put forward in this connexion. One is that the spot is a relic of a past crisis involving the vitamin A status in infancy or youth; but I have seen many cases in the children of rich and educated Indians and Africans, where such a history is absent. Moreover, in cases of acute xerophthalmia which I have treated, I have seen many instances of corneal scarring as a sequel, but never a Bitot's spot. The other argument is that the spot depends upon a prolonged chronic deficiency (Appelmans, Lebas & Missotten, 1956): again this hypothesis does not fit the facts as I personally have witnessed them. Despite these arguments, the

Bitot's spot is most commonly observed in regions where the vitamin A intake is in general known to be reduced, and is rare in vitamin A-rich communities; moreover, during an acute attack of xerophthalmia the Bitot's spot is superficially simulated. Though this lesion should no longer be classified as a true sign of vitamin A deficiency —there is and always has been overemphasis as to its importance—nevertheless, its origin deserves further attention. When the foam, which is the dominant sign of its presence, is wiped away it quickly re-forms; analysis of its chemical characteristics and origin should not be difficult. One important observation we made in North India was that beneath the foam the conjunctiva may be normal, or raised, gelatinous, opaque and wet, or thickened, wrinkled and dry. These appear to be progressive steps in the development of a Bitot's spot, yet none is consistently related to reduced blood values. It is important to note that the appearance of the foam precedes any change in the conjunctiva in the Bitot area. This is a new angle on a controversial theme (Oomen, 1958).

Acute diffuse xerophthalmia

This lesion is always associated with very low, frequently zero, blood values. Where dark adaptation is possible, the dark-adaptation curve is much reduced. The lesion is found in infants, small children and sometimes debilitated adults. The children correspond to what may be defined as Bloch's body type I, a marasmic child with a pot belly, or type II, in which the child looks healthy but on closer examination is seen to be somewhat puffy and fat (Bloch, 1924). The former type is more commonly associated with acute xerophthalmia, the latter with chronic. I need not stress here the probable relation of protein deficiency or carbohydrate excess to these body types, the former with type I, and both the dietetic anomalies with type II (Bietti, 1950; McLaren, 1958). Whether they affect the eye is another question.

The clinical appearance of xerophthalmia is well known. All stages in its progression are found. In the acute form, clear bubbles are seen lying horizontally across the Bitot area, frequently impinging on the cornea. These bubbles do not correspond to the foam seen with Bitot's spots but their position does. Even untreated, the child may get off with slightly scarred corneas, for an improvement in the diet can be expected as the seasons change; however, if the deficiency in vitamin A continues to wax and wane, mummification going on to the formation of an anterior staphyloma is probable. At the worst, the eyeballs will rupture at the height of the acute lesion and then either heal to form a badly scarred cornea, in which iris tissue is enclosed, or wither up (*phthisis bulbi*), or again progress to an anterior staphyloma. In a number of cases the final result is unequal, one eye being less affected than the other. The sad thing is that if the lesion is caught before the corneas begin to soften and rupture—by derivation this is a keratomalacia—the eyes can be cured with adequate vitamin A in a week, perhaps without even a scar being left. Acute diffuse xerophthalmia is the commonest ocular manifestation of avitaminosis A and in many parts of the world one of the major causes of blindness. We are apt to forget this fact (Rodger, 1959).

Keratomalacia and spontaneous iris prolapse

Keratomalacia may be the last stage in a progressive xerophthalmia, as I have just described. It results from a softening and thinning of the corneal stroma, a colliquative necrosis, although this is a term which, as a clinician, I would not like to see replacing the term keratomalacia. Epithelial thinning alone clearly cannot lead to rupture of the cornea. When the epithelium is involved hypoaesthesia exists; as a result this is a feature of all xerotic conditions. It is necessary to consider the probable relative effects of acute and chronic deficiency on the clinical appearance. In the acute, following a xerophthalmia, the cornea will either rupture over a wide but discrete area, usually where it is thinnest, that is centrally, or the entire cornea will be involved in the process. In chronic deficiency, I believe we get small discrete localized lesions followed by iris prolapse, a condition which for convenience I call spontaneous iris prolapse, although it has been called malnutritional keratitis (Blumenthal, 1950, 1954). This term is nosologically incorrect. I have classified these small prolapses as chronic keratomalacia for a long time. I have seen some of them develop from facets in a perfectly healthy-looking cornea, but never the actual rupture which occurs instantaneously; the small iris prolapse is re-covered by corneal epithelium in a matter of several hours. The mean blood values for a group of twenty-nine of these cases proved to be only 35 i.u. vitamin A and 39 μg carotenoids/100 ml plasma. Such a low blood value as 35 i.u. in a child not otherwise sick conceivably might in the end lead to such a weakening of the anterior coats of the growing eyeball that it will give way at its weakest points, namely the sulcus sclerae, just within the limbus, and the apex; the former is much the commoner. It must be remembered that the cornea grows very much more quickly than the rest of the eye during the first years of life, and that its expansion takes place from the limbus. Spontaneous iris prolapse I have seldom seen in children over the age of 5 years.

The lesion is not restricted to the Bantu race, as has been suggested. It is common in the African interior. I have also seen it on the West African coast, in India and in East Pakistan. It is usually found in the lower half of the cornea, that which is most exposed, and the fact that it may be associated with trachoma (which affects the upper half of the cornea) is a coincidence because active trachoma also affects children under 5 in these countries. I have the impression, however, that it may be more frequent in the Bantu, although its distribution is apparently world-wide. An investigation of socio-economic factors, such as the diets, in the different African tribes and in the Indian pradeshes (provinces) might help to solve the aetiology. As anyway we are unable to foretell how the eye is going to respond to a deficiency in vitamin A, I see no reason why we should not accept this conception of the pathogenesis of spontaneous iris prolapse, until more convincing evidence is presented that we are dealing with something new and strange.

Hemeralopia and its various aetiological factors

What from my personal observations is new, or confirmatory, in connexion with night-blindness? That men are more commonly affected than women; that most cases

have low blood values for vitamin A, but that the carotenoid values are frequently normal; that the position in time of the point α is unchanged in deficiency of vitamin A; that there are many examples of subjects with low, or zero, blood vitamin A values who reveal perfectly normal dark-adaptation curves, and although one may argue that this is possible because the retina preferentially retains its own vitamin (Pirie & van Heyningen, 1956), why does it not do so when the blood values are around 80 i.u./100 ml yet such a defective curve exists; that cone as well as rod function are both equally affected and the response to treatment, which may be as great as 10 000 times in 10 h, is equally quick and effective for both components of the curve; that periods of stress such as pregnancy, suckling and growth are not always significantly related? There is much food for thought here (Rodger, Saiduzzafar *et al.* unpublished).

It has been claimed recently from South India that an iron-deficiency anaemia, such as that induced by hookworm, can cause night-blindness (Rao, De & Rao, 1953; Patwardhan, 1958). No blood values were taken in these studies, assessment of the vitamin A status being based on the known dietary intake. It has been shown that exposure to bright light in hot countries in people who are debilitated can lower the rod threshold (Hecht, Hendley, Ross & Richmond, 1948); of course in a subjective test debilitation alone may lower the perceived threshold. (In the South Indian experiment increased intake of rice improved the rod threshold, apparently by improving the nutritional status.) Altitude, by inducing anoxia, is another way whereby night-blindness can be induced. These are far more practical considerations than those obtained by dragging in such conditions as hereditary tapeto-retinal disease, or tertiary lues. Despite these obvious difficulties, and ignorant of the South Indian study, my colleagues and I in North India (Rodger, Dhir & Hosain, unpublished) independently investigated a relationship between night-blindness and hookworm infestation. Our area was one of low altitude. Hookworm was common in the community. In half of the thirty-eight cases the haemoglobin level was reduced. Eight revealed markedly reduced dark-adaptation curves, the reduction being considerably greater than that caused by bright light. Four of these cases had haemoglobin levels under the values below which anaemia can be said to exist; in the other four they were above this level (WHO, 1959). Although the vitamin A levels in all eight cases were each below 100 i.u./100 ml, which is not high, nevertheless, the values individually and collectively could not be said to reveal a deficiency; the mean values for the thirty-eight cases infested with hookworm revealed no abnormality in the vitamin A levels but slight reduction in the carotenoid levels. In the control group of fifty-four subjects, paired as far as possible for sex, age and religion (on which the diets depend), the mean value for vitamin A was slightly higher and that for the carotenoids considerably higher. Many interesting observations were made in this study, which we hope shortly to publish.

Ocular onchocerciasis and vitamin A

There is a degenerative lesion of the choroid and retina in some onchocerca-infested patients which is not associated with the presence of microfilariae in the eye,

https://doi.org/10.1079/PNS19600020 Published online by Cambridge University Press

suggesting that the pathogenesis differs from that known to cause the other ocular lesions of this disease, namely inflammation around the dead bodies of the embryos. The presenting symptom of this degenerative lesion is night-blindness. On investigating a small group of ten such cases, I found the blood values of vitamin A much reduced—under 25 i.u./100 ml, lower than the values expected with a daily intake of 4000 i.u. and lower than the values (around 80 i.u.) in two control subjects. Moreover, four out of nine of these night-blind subjects, when given massive doses of vitamin A (Crookes Laboratories Ltd vitamin A capsules or Roche Products Ltd Arovit), exhibited a considerable recovery in vision by the end of a week, and night-blindness was cured in them all (Rodger, 1958). Now, I admit these are slender premises on which to conclude that the degenerative lesion of onchocerciasis is even partly dependent on vitamin A deficiency, which is as far as I went. Nevertheless, it is highly suggestive, especially as I have to date failed to recognize this lesion in the rain forests where the daily intake of vitamin A is 12 000 i.u. and onchocerciasis equally endemic. Moreover, I was able to carry out histological studies on five eyeballs exhibiting this lesion, and each one revealed changes in the retinal pigment epithelium, which corresponded to similar changes stated to have been found in the eyes of vitamin A-deficient monkeys (Ramalingaswami, Leach & Sriramachari, 1955). The lesion is more than a pigmentary one, however, so the pathogenesis is likely to involve some other factor or factors as well as vitamin A. That is my belief at the moment.

Beriberi and nutritional amblyopia

Without discussing the arguments concerning the origin of beriberi, any association of it with defective vision implicates thiamine deficiency as a causative factor in production of the ocular lesion. Since milling regulations were introduced into East Asia, both beriberi and nutritional amblyopia have decreased (FAO/WHO, 1958). Observers in P.o.W. camps have disagreed as to this relationship (Hobbs & Forbes, 1946): some claim to have cured nutritional amblyopia by administering thiamine (Houwer, 1946), and others say they failed to do so (Hazelton, 1946). Scrotal dermatitis, glossitis and angular stomatitis began in most camps about the 7th month, aching feet about the 10th, amblyopia about the 11th and wet beriberi later still, although an early transient wet beriberi has been described as occurring about the 3rd month. The Japanese, who have much experience of the disease, have reported loss of the ankle jerks in 120 out of 200 cases of nutritional amblyopia, a condition they called latent beriberi (Kagawa, 1938). One gets the impression that in the P.o.W. camps rather too much emphasis was placed on the classic signs of beriberi, nor must we forget the high death rate which may have concealed a more significant relationship. Moreover, the deficiencies were multiple and associated with an imbalanced diet and a severe reduction in calories, which collectively might alter the picture.

The most recent paper on the subject is a comprehensive study of American P.o.W.'s after repatriation from Korea (King & Passmore, 1955). The conclusion reached was that thiamine deficiency was the most significant factor. This had been

my conclusion 3 years earlier (Rodger, 1952), when I followed up the histories of 238 ex-P.o.W.'s from Japanese camps, each on the Ministry of Pensions list in the U.K. On the other hand, it was not the conclusion reached in the M.R.C. report on the subject (Smith & Woodruff, 1951). In the Korean cases electroretinograms revealed an ascending atrophy of the second visual neurone. A significant clinical feature is the nature of the scotomata (blind spots); in the early phase the scotoma is centrocaecal; it improves incompletely until a central scotoma alone remains.

Experiments with animals tend to support the view not only that thiamine deficiency is the main pathogenetic factor but also that the second visual neurone is affected (optic atrophy). They also make it clear that the deficiency has to be chronic. Acute cases of wet beriberi early on in prison life, as a result, were not likely to be associated with blindness, and in fact were not.

Nutritional amblyopia has been reproduced in rats and kittens made to suffer from beriberi (Swank & Prados, 1942). In my own small pair-controlled series of rats (Rodger, 1953), I found involvement not only of the second but of the third visual neurone, thereby explaining some of the atypical fields found in the P.o.W.'s. Although the cell bodies in the dorsal nucleus of the lateral geniculate bodies were markedly affected, I could find nothing significantly wrong with the ganglion cells of the retina, which was, I feel sure, due to technical difficulties in preparing rat retina, which is very vulnerable. Associated changes in the neuroglia of the rat visual pathway suggest that these cells—probably the oligodendroglia—may be involved in carbohydrate metabolism.

Thiamine and riboflavin deficiencies

A subacute deficiency of thiamine, it was noted, leading to death of rats in about 75 days, did not produce death in litter-mates when riboflavin was also excluded (Rodger, 1954). The structural changes in the visual paths of these animals were greater than in chronic thiamine deficiency alone. Prolonged riboflavin deficiency by itself produces no changes. One assumes that when riboflavin and thiamine deficiencies are combined, and life prolonged, the visual neurones are subjected to a more protracted breakdown in the carbohydrate metabolism with, as a result, greater structural damage. Why life is prolonged in these circumstances it is difficult to say; at the time I suggested that the metabolism might be made partly more dependent on fat. I also stressed at the time the probable close relationship existing between different members of the B-complex vitamins. There is much more evidence today in that direction (Dalgliesh, 1956). There can be little doubt that these animals were suffering from experimental beriberi, for in addition to the optic atrophies produced they had a measured bradycardia and a proven peripheral neuritis, although not in every rat, or in every nerve bundle, or in every cell in every dorsal nucleus. Up-country in West Africa, where thiamine is plentiful, beriberi and pellagra unknown, and only riboflavin, vitamin A and animal protein short, I found about half a dozen probable cases of nutritional amblyopia in a population of 12 million, exhaustively surveyed over 4 years. This observation supports my point that thiamine is the important factor.

As for the other recorded ocular manifestations of ariboflavinosis, apart from blepharitis (cataract being doubtful), I saw none, despite the belief of others (Irinoda & Sato, 1954) that it causes many lesions of the eye. A convincing diagnosis for every ocular lesion can be reached, however, without requiring ariboflavinosis as the explanation.

Amblyopia associated with other non-tropical diseases

Here is a subject urgently requiring confirmation and clarification. I would like to suggest that the amblyopias associated with tobacco–alcohol poisoning, pernicious anaemia and *hyperemesis gravidarum* (uncontrolled pregnancy sickness) are each due to thiamine deficiency. It is not a new suggestion, but there exists a divergence of opinion at the present moment. Such a deficiency would appear to depend either upon a diminished intake, defective absorption or the inability to stabilize thiamine in the stomach owing to the absence of hydrochloric acid (Rodger, 1951). Each of the types of case under discussion has been cured by the administration of thiamine (Carroll, 1944; Ungley, 1938; Ballantyne, 1941). Cases of tobacco–alcohol poisoning and pernicious anaemia have as the presenting scotoma a centro-caecal defect with dense nuclei especially in the central area. With treatment it may clear up completely or incompletely; if the latter, the residual defect is a central scotoma. With hyperemesis, the patient is too ill to permit scotometry in the early stages; when the subject has recovered, however, the residual blind spot is central. These observations on the whole correspond well to what we know of nutritional amblyopia in P.o.W. camps. Arguments based on the brief reference of Traquair (1949) to this subject can be faulted on the grounds of the fact that Traquair did not write at a time when amblyopia in P.o.W.'s had been fully described. In 1951, I described four cases of subacute combined degeneration of the cord each with amblyopia of this type (Rodger, 1951), an observation which at the time had been made only by a few others (Cohen, 1936; Turner, 1940); in two further cases (referred to me by the late Dr Charles Ungley) there was a history of tobacco amblyopia some years previously. In the same year I reported three recent cases diagnosed as tobacco–alcohol amblyopia, in each there being histamine-fast achlorhydria; similarly in sixteen old cases of *hyperemesis gravidarum*, 1 year after confinement, I found four in whom there was the expected residual peri- or para-central scotoma; in one there were homonymous quadrantic peri-central scotomata, which confirmed in a way my observations on the visual pathway of the rat in thiamine deficiency, namely that the third visual neurone was involved, or (alternatively) the optic tract affected. The same thread seems to run through all these conditions, and it is clearly one deserving more study. It has yet to be shown conclusively whether or not vitamin B_{12} deficiency leads to degeneration of the visual neurones, or only indirectly through thiamine.

Incidence of malnutritional blindness

Clearly in temperate zones the incidence of blindness from malnutrition, except in time of war, will be small. In developing countries, on the other hand, it will be large. Little is known that is definite. In the Guinea Coast territories I surveyed for

4 years, I found the incidence of malnutritional blindness, either direct or indirect, to be 5% of all blindness in each country. Thus in what was British West Africa there must be about 20 000 cases of this type. This figure refers to a people three-quarters of whom have attained a good level of physical development and nutrition. In Asia, I obtained the impression that the proportions are reversed: three-quarters of the people are malnourished. Thus in Asia I would expect to find a much higher incidence of malnutritional blindness. Such was my experience. If it is terrible to hear that there are 20 000 blinded for lack of a proper diet in four small West African territories, how much more terrible is it to contemplate the probability that this same figure may be found in only one Province of South India!

REFERENCES

Appelmans, P. J. M., Lebas, P. & Missotten, L. (1956). *Bull. Soc. belge Ophthal.* **113**, 327.
Ballantyne, A. J. (1941). *J. Obstet. Gynaec., Brit. Emp.*, **48**, 206.
Bietti, G. B. (1950). *Docum. ophthal.* **4**, 200.
Blumenthal, C. J. (1950). *S. Afr. med. J.* **24**, 191.
Blumenthal, C. J. (1954). *S. Afr. med. J.* **28**, 967.
Bloch, C. E. (1924). *Amer. J. Dis. Child.* **28**, 659.
Carroll, F. D. (1944). *Amer. J. Ophthal.* **27**, 713.
Cohen, H. (1936). *Lancet*, **231**, 1202.
Dalgliesh, E. E. (1956). *Brit. med. Bull.* **12**, 49.
de Haas, J. H. & Meulemans, O. (1938). *Lancet*, **234**, 1110.
FAO/WHO (1958). *Tech. Rep. Wld Hlth Org.* no. 149.
Hazelton, A. R. (1946). *J. R. Army med. Cps*, **86**, 171
Hecht, S., Hendley, C. D., Ross, S. & Richmond, P. M. (1948). *Amer. J. Ophthal.* **31**, 1573.
Hobbs, H. E. & Forbes, F. A. (1946). *Lancet*, **251**, 149.
Houwer, A. W. M. (1946). *Ophthalmologica*, **112**, 177.
Irinoda, K. & Sato, S. (1954). *Tohoku J. exp. Med.* **61**, 93.
Kagawa, S. (1938). *Jap. J. med. Sci.* **5**, 1.
King, J. H. & Passmore, J. W. (1955). *Amer. J. Ophthal.* **39**, 173.
McLaren, D. S. (1958). *Bull. Wld Hlth Org.* **19**, 303.
Oomen, H. A. P. C. (1958). *Fed. Proc.* **17**, Suppl. no. 2, p. 111.
Patwardhan, V. N. (1958). *Fed. Proc.* **17**, Suppl. no. 2, p. 110.
Pirie, A. & van Heyningen, R. (1956). *Biochemistry of the Eye*, p. 302. Oxford: Blackwell's Scientific Publications.
Rao, K. S., De, N. K. & Rao, D. S. (1953). *Indian J. med. Res.* **41**, 349.
Ramalingaswami, V., Leach, E. H. & Sriramachari, S. (1955). *Quart. J. exp. Physiol.* **40**, 337.
Rodger, F. C. (1951). The relationship of thiamine to the visual pathway. Thesis for the degree of Doctor of Medicine, University of Glasgow.
Rodger, F. C. (1952). *A.M.A. Arch. Ophthal.* **47**, 570.
Rodger, F. C. (1953). *Brit. J. Ophthal.* **37**, 11.
Rodger, F. C. (1954). *Brit. J. Ophthal.* **38**, 144.
Rodger, F. C. (1958). *Brit. J. Ophthal.* **42**, 21.
Rodger, F. C. (1959). *Blindness in West Africa*, Chapter 5. London: Lewis.
Scrimshaw, N. A., Behar, M., Perez, C. & Viteri, F. (1955). *Pediatrics, Springfield*, **16**, 378.
Sie, B. L. (1937). *Tijdschr. Geneesk. Ned.-Ind.* **77**, 3283.
Smith, D. A. & Woodruff, M. F. A. (1951). *Spec. Rep. Ser. med. Res. Coun., Lond.*, no. 274.
Swank, R. L. & Prados, M. (1942). *Arch. Neurol. Psychiat., Chicago*, **47**, 626.
Traquair, H. M. (1949). *Clinical Perimetry*, p. 158. London: Henry Kimpton.
Turner, J. W. A. (1940). *Brain*, **63**, 225.
Ungley, C. C. (1938). *Lancet*, **234**, 875.
WHO (1959). *Tech. Rep. Wld Hlth Org.* no. 182.