CORRESPONDENCE

XEROPHTHALMIA AND PROTEIN MALNUTRITION

To the Editorial Committee of the BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS—There are two points with regard to the study of Dr. Kuming and Dr. Politzer on xerophthalmia in Johannesburg (*Brit. J. Ophthalmol.* (1967), **51**, 649) on which I believe the record should be set straight. It is not correct to quote me, as the authors do on page 659, as saying that Bitôt's spots are not a sign of vitamin A deficiency. Sometimes they are, but at other times their aetiology is not clear (McLaren, 1966). On page 660 the authors say that Rodger and his co-workers and myself state that keratomalacia can be cured or arrested by vitamin A. I think I speak for both of us when I say that we have always been careful to indicate that the very real response of this condition takes place within certain fixed limits determined by the nature and extent of the pathological process.

The almost invariable association of the severe eye lesions of xerophthalmia with manifestations of protein-calorie malnutrition in young children has led Dr. Kuming and Dr. Politzer, as it has others, to the erroneous conclusion that "protein malnutrition was the cause of xerophthalmia, and that xerophthalmia was, in this series, a sign of protein malnutrition". All the evidence of many years of careful animal experimentation is against this, as are the results of recent studies of liver and serum levels of vitamin A in malnourished children with and without the eye lesions (McLaren, 1966; McLaren, Shirajian, Tchalian, and Khoury, 1965).

Clean iris prolapse ("discrete colliquative keratopathy") remains an aetiological problem and the present paper sheds no fresh light. None of the cases illustrated resembles those pictured by Blumenthal or seen by me in East Africa. At present I am more inclined than previously to accept that we are dealing with one and the same condition, but we must have biochemical evidence to back this up (McLaren, 1967). It is very unfortunate that Dr. Kuming specifically chose to exclude patients with measles. It has been suggested that D.C.K. is nothing but measles keratitis (Pratt-Johnson, 1959) and there may be some truth in this. However, the relationship between xerophthalmia and measles is widely recognized in Asia (Scragg, J., and Rubidge, 1960; Oomen, McLaren, and Escapini, 1964). Recent evidence from other parts of Africa has shown this combination to be the cause of previously ill-understood childhood blindness problems (Awdry and Cobb, 1964; MacManus, 1967).

Yours faithfully,

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To the Editorial Committee, BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS—Regarding Bitôt's spots, when this article was written in 1965 Prof. McLaren's comments in 1966 had not yet been published. He did however state (McLaren, 1963) "that one cannot jump to the conclusion that vitamin A deficiency is present if Bitôt's spots are present", a view which he modified later (McLaren, 1966): "there is no doubt that they can be an integral part of the ocular manifestation of vitamin A deficiency". In our experience we have seen this Type I spot occurring in normal children and we cannot differentiate the types on the clinical grounds advanced by Prof. McLaren.

Again Prof. McLaren did indeed say in 1961 that "the arrest of colliquative necrosis in keratomalacia is one of the most dramatic therapeutic tests in medicine" (McLaren, 1961). Pathologically it is characterized by cell death and this process is irreversible. Once colliquative necrosis has occurred the cornea cannot be restored to its former clarity except perhaps by a later graft.

We specifically chose to exclude cases of measles because we were trying to establish what the lesions of protein malnutrition were and we found these to be identical to those attributed to vitamin A deficiency only. An important aspect concerning measles was made clear in Pratt-Johnson's original paper (Wessels and Pratt-Johnson, 1956) where he pointed out that the ocular complications of measles were rare in Caucasians but common in the group that he was studying; i.e. local environmental factors are extremely important such as trachoma, secondary infection, and malnutrition. Measles causes severe lesions only in a cornea severely embarrassed by existing dietary deficiencies; and measles, herpes simplex, or secondary bacterial infection are all equally potent factors in causing keratomalacia. Incidentally, none of the photographs illustrating Pratt-Johnson's paper bears any resemblance to discrete colliquative keratopathy. Just as Prof. McLaren believes that the response of keratomalacia to vitamin A takes place within certain fixed limits determined by the nature and extent of the pathological processes, we believe that the nature and extent of these same pathological processes determine whether localized or generalized keratomalacia occurs.

I have recently had the opportunity of discussing this problem with Dr. L. Joos van de Walle, who worked in the Luapula River Valley in what was then Northern Rhodesia in 1962. He has informed me that there is much malnutrition in this area and that all his cases of measles keratitis occurred in malnourished children.

The basic difference between Prof. McLaren's approach and ours is on the interpretation of the role of protein deficiency. In our series we found many cases with ocular lesions with normal vitamin A values and *vice versa*. In Prof. McLaren's paper (McLaren, Shirajian, Tchalian, and Khoury, 1965), the liver values of his various groups are very similar and the vitamin E levels are even more significant than those of vitamin A.

While Prof. McLaren believes that "xerophthalmia, associated as it is with some degree of protein malnutrition, and frequently accompanied by various infectious diseases, also causes considerable mortality" (McLaren and others, 1965), we believe that it is the protein malnutrition and the associated diseases which carry with them the considerable mortality.

Yours faithfully,

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