occur in renal disease (McNicol *et al.*, 1965; Wardle *et al.*, 1970; Prentice *et al.*, 1972), although Pandolfi *et al.* (1970) showed that the activator content of renal vessels is independent of the degree of uraemia.

Raised levels of urine F.R.-antigen were present in 34 of 38 patients but did not correlate with the type of renal disease, serum F.R.-antigen level, or severity of the renal failure. We found, as did Clarkson *et al.* (1971) that there were wide variations in the amounts of urine F.R.-antigen from day to day in the same patient. The lack of correlation between the serum and urine F.R.-antigen levels, and the presence in 14 patients of raised urine F.R.-antigen together with normal serum levels supports previous evidence that urine F.R.-antigen appears as a result of local fibrinogen or fibrin degradation in the kidney (Humair *et al.*, 1969). On the other hand, correlation between the amounts of F.R.-antigen and of protein in the urine suggests that part of the urine F.R.-antigen concentration is derived by glomerular filtration. Although raised urinary F.R.-antigen has been described previously in a variety of renal diseases (Rayner *et al.*, 1969; Clarkson *et al.*, 1970b), the wide range of results obtained in diagnostic groups precludes any use as a diagnostic aid, except possibly in the detection of incipient renal transplant rejection (Braun and Merrill, 1968; Carlsson *et al.*, 1970; Clarkson *et al.*, 1970a). Additionally, serial assays may be of use as a prognostic guide (Clarkson *et al.*, 1971).

An important problem is whether F.R.-antigen is an almost irrelevant by-product of basic renal disease which will continue on its way uninfluenced by therapeutic measures, or whether the immediate mechanism which produces F.R.-antigen is central to the pathogenesis of the disease. It might be that heparin, for example, could control some of the manifestations of renal disease without in any way influencing the fundamental downhill progression. Although initial results of anticoagulant therapy in glomerulonephritis in experimental animals (Vassalli and McCluskey, 1964; Halpern *et al.*, 1965) and in man (Kincaid-Smith *et al.*, 1970) have been encouraging, further information, preferably based on a controlled clinical trials, is urgently needed.

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MEDICAL MEMORANDA

Quinine Amblyopia Treated with Stellate Ganglion Block

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Quinine overdosage is uncommon nowadays in medical practice.

The syndrome of cinchonism is, however, well described, the most dangerous component of which is loss of vision. We report a case of quinine amblyopia which responded dramatically to stellate ganglion block and the findings of almost a year's follow-up.

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Case History

An unemployed 16-year-old girl took about 15 300 mg tablets of quinine sulphate (4.5 g) on the evening of 20 September 1970. They had been prescribed for her mother's night cramps and were swallowed as a suicide gesture after a family row. She vomited several times that night and found by the following morning she was virtually blind though otherwise perfectly well. She was admitted to St. Mary's Hospital, London, at noon on 21 September.

On examination she was fully conscious, with regular pulse and normal blood pressure. The only abnormal physical findings were ocular. She was only just able to distinguish light from dark; the pupils were widely dilated and just reacted to light. Her fundi appeared entirely normal.

MANAGEMENT

Forced diuresis was begun after stomach washout. Initial investigations showed a normal haematological picture, urea, and electrolytes. The serum quinine level was 7.2 mg/l. Inhalation of amyl nitrite produced flushing of the conjunctivae but no subjective visual improvement or fundal changes. Three hours after admission bilateral stellate ganglion block was performed (Dr. K. Herdman). A dose of 10 ml of 5% Marcain was injected each side.

Subjective improvements in vision occurred in the left eye within two to three minutes, coinciding with a conjunctival flush and Horner's syndrome on that side. Acuity improved sufficiently for her to count fingers at 1 metre. Blockade of the right side was initially unsuccessful, producing neither a Horner's syndrome nor visual improvement, but a second attempt three hours later produced changes as dramatic as on the left side.

By the second day a bilateral Horner's syndrome was still apparent. Distance visual acuity was not quantitatively tested then, but the patient's near vision was N6 binocularly. However, a definite peripheral field constriction could be shown on confrontation, and was confirmed later by perimetry. Pupils were still dilated although reacting to light and the fundi were unchanged at this stage. All other systems remained normal.

Serial E.C.G.'s showed intermittent nodal rhythm occurring on the second day, reverting to normal after one week.

OCULAR FINDINGS AND FOLLOW-UP

Three days after admission (four days after swallowing the quinine tablets) the pupils were normal size and reacting briskly to light. Visual acuity right 6/9 (unaided), left 6/6 (unaided). The fundi showed normal vessels and discs, but macular oedema was observed for the first time in both eyes. The visual fields showed severe constriction peripherally.

One month after admission the visual fields remained constricted peripherally (see Fig.), with visual acuity remaining right 6/9 and left 6/6 (unaided). The macular oedema persisted but the retinal vessels and discs remained normal.

Visual fields at one month

(A), three months

(B), six months

(C) and 10 months

(D) after admission.

Two months after admission the visual fields had improved slightly and acuity remained right 6/9 and left 6/6 (corrected). The left disc showed slight pallor for the first time. The right disc appeared normal.

Three months after admission there was further slight improvement in visual fields, especially temporally, in both eyes (see Fig.). The right disc was normal but the left disc showed increased uniform pallor. The retinal vessels remained normal.

Six months after admission the visual fields showed some regression from three months previously (see Fig.), while the visual acuity remained right 6/9 and left 6/6 (corrected). For the first time slight temporal pallor of the right disc was present, the left disc remained uniformly pale, and the retinal vessels remained normal.

Ten months after admission the visual fields were unchanged from four months previously (see Fig.) and showed the peripheral field loss, especially nasally. Visual acuity was right 6/9 and left 6/6 (corrected), while the retinal vessels remained normal in appearance and the discs showed optic atrophy, more pronounced on the left side.

Comment

This patient was aged 16 years and 2 months, and was thus one of the youngest patients ever recorded with quinine amblyopia. She took the quinine tablets on impulse, probably at a time of

indecision for her, as she was being encouraged to live away from home by her girl friend. The psychiatrist who saw her while she was an inpatient believed that she took the quinine tablets as an act of desperation and indecision which reflected her immature personality. The quinine tablets were in a bottle kept in the kitchen and had been prescribed for her mother for night cramps.

The exact mechanisms of the visual loss, visual field constriction, and fundus signs in quinine amblyopia remain largely unknown. The most recent publications on the subject support the view that the ocular effects of quinine are due to a direct toxic effect on the ganglion cells of the retina. However, De Jean *et al.* (1958) and Perner and Saskin (1942) thought that quinine acted directly on retinal arterioles, producing vasoconstriction and consequent retinal damage and visual loss. In support of this view they pointed to the rapid improvement in vision after using vasodilators (retrobulbar nicotinic acid (Nicyl) and intravenous acetylcholine and sodium nitrite).

Certainly in this case there was a rapid improvement of vision after stellate ganglion block, and this does suggest that the effects of quinine can partly be overcome by increasing the blood flow to the retina. Stellate ganglion block in the treatment of quinine amblyopia was first used by Redslob *et al.* (1946) and later by Glick and Mumford (1955), each group of authors reporting individual cases with good recovery of vision. Bricknell *et al.* (1967) reported a case where the patient received 19 separate stellate ganglion blocks in seven days. We strongly

recommend the use of stellate ganglion block in the emergency treatment of quinine amblyopia, as we believe that vasodilatation can favourably influence the direct toxic effect quinine has on the retinal ganglion cells.

It should be noted that we did not consider that electroretinography or fluorescein angiography were justifiable procedures in this particular patient in view of her emotional state at the time of her hospital admission or at subsequent follow-up.

We would like to express our thanks to Dr. P. Sanderson, assistant director of the medical unit, under whose care the patient was admitted, for his help and agreement to report the details of this case. The serum quinine levels were estimated by Dr. B. Houghton. Miss G. Whitehead provided secretarial help.

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