spontaneous remission of the nephrotic syndrome after a course of 6 months in a Sephard Jewish boy affected with renal amyloidosis associated with familial Mediterranean fever. The patient then remained free of any renal symptom, including proteinuria, for the next 5½ years until he experienced a dramatic relapse with a full-blown nephrotic syndrome and severe renal failure.¹ A renal biopsy performed after 3½ years of remission showed the persistence of amyloid deposits with a distribution and intensity similar to those seen when the first biopsy was performed, when the nephrotic syndrome was present.

The importance of such spontaneous remissions must be stressed in view of several recent articles ascribing to various drugs the disappearance of the nephrotic syndrome in renal amyloidosis.²⁻⁵

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¹ Méry, J-P, and Mostefa, S, *Annals of Internal Medicine*, 1975, **83**, 581.

² Cohen, H J, et al, *Annals of Internal Medicine*, 1975, **82**, 466.

³ Horne, M K, *Annals of Internal Medicine*, 1975, **83**, 281.

⁴ Zemer, D, et al, *New England Journal of Medicine*, 1976, **294**, 170.

⁵ Ravid, M, Robson, M, and Kedar, I, *Annals of Internal Medicine*, 1977, **87**, 568.

Snakes and snake bite

SIR,—I was interested in the review by Dr David A Warrell of the book *Snakes and Snakebite* by John Visser and David Chapman (13 May, p 1269). As I have a special interest in this subject I would like to make one or two comments on the review which may be pertinent.

Dr Warrell has already made a number of observations about the book and its contents. However, the implication of the "futility of local interference at the site of the bite" is that pressure-relieving incisions play no part in the treatment of snake bite. I believe it has been amply demonstrated in several disciplines of medicine that pressure-relieving fascial incisions can prevent necrosis. A particularly good example of this is the anterior tibial compartment syndromes. The use of tourniquets is only controversial in as far as the dangers in the hands of lay people are concerned. If they are not applied within a few minutes of the bite they are unlikely to do any good at any time.

Dr Warrell correctly points out that puff adder bites can cause significant delayed hypotension and concealed blood loss, but his suggestion that the antivenom can help in preventing local necrosis appears unfounded in view of the fact that the antivenom does not contain any local cytotoxin-effective antibodies. The point to be emphasised is that antiserum need never be given for puff adder bites except in controlled circumstances and after admission to hospital. In my own experience the incidence of reaction to intravenous antivenom is low except in snake handlers who have previously been bitten and treated. Neither the authors nor the reviewer make any mention of prevention of serum sensitivity by the use of high-dosage cortisone. Although adrenaline is highly effective in the treatment of serum sensitivity, it is in itself a dangerous drug which should not be handled by lay persons, and if treatment with antivenom is always preceded by a bulk injection of a pharmaco-

logical dose of cortisone antiserum sensitivity can be expected to disappear completely; this has been our experience. Finally I would like to re-emphasise Dr Warrell's point that the doses of antivenom administered in most hospitals are quite inadequate and should not be used routinely.

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SIR,—It would be interesting to know the evidence on which Dr D A Warrell (13 May, p 1269) makes the assertion that venom from a spitting cobra spat into the eye "frequently causes corneal ulceration, and may be absorbed . . . causing hypopyon and anterior uveitis."

There is no doubt that in travellers' tales the severity of such incidents has been repeatedly asserted, but my own limited experience¹ suggests that such sequelae are rare and that the consequences of such an event are relatively mild and unlikely to occasion permanent damage. Duke-Elder's comprehensive *System of Ophthalmology*² makes only limited reference to this particular problem. This again would tend to confirm one's impression that this particular snake's venom in the eye is perhaps no worse a mishap than accidental contamination with, say, neat washing-up liquid or one of the quaternary ammonium compounds—painful and discomfiting but not the major event suggested by the quotation.

It is a little difficult to envisage in a case of a simple "spit" a more effective treatment than local lavage and analgesia. One surely would be very reluctant in such a case to add the potential risk of necrosis by use of antivenom.

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¹ Gilkes, M J, *British Journal of Ophthalmology*, 1959, **43**, 638.

² *System of Ophthalmology*, ed S Duke-Elder, vol 14, part 2, p 1206. London, Kimpton, 1972.

Alcohol and the blood

SIR,—Your leading article (10 June, p 1504) provides a fine historical review of the blood changes that may be related to excess alcohol consumption. However, it gives little information about which of these abnormalities are common and which are rare, and no guidance in their use for the detection of alcoholism. Most of the studies referred to were made on "skid-row" alcoholics, who are not typical of patients with alcohol-related diseases seen in the UK.

The majority of our alcoholic patients with macrocytosis are well nourished,^{1,2} are in regular employment, and have normal vitamin B₁₂ and folate stores. Unger and Johnson³ found that 3% of an insurance company's employees were macrocytic on routine blood count and, of 17 examined, 16 were probably consuming excessive amounts of alcohol. A low serum folate concentration was found in only one subject, and although the remainder were treated with large doses of first folic acid and then B₁₂ their mean cell volume (MCV) did not fall. In addition, there have been several other recent reports on macrocytosis of alcoholism from the UK and France which give a similar picture.⁴⁻⁸

Further evidence that the rise in MCV may

be unrelated to folate deficiency has been provided by the deoxyuridine (dU) suppression test,⁹ which is a sensitive biochemical test for folate or B₁₂ deficiency performed on short-term bone-marrow culture. All eight of the alcoholic patients with macrocytosis studied by Wickramasinghe and Longland¹⁰ gave normal results in this test, and their erythrocyte folate and serum B₁₂ levels were subsequently found to be normal. This implies that the alcohol effect could not be attributed to an interference with B₁₂ or folate metabolism and we believe that alcohol has some other direct effect on the developing erythroblast. When folate deficiency does occur in alcoholics it is almost always due to associated nutritional folate deficiency, a feature universally present in the "skid-row" alcoholic but seen in only one-third of our patients, who are more likely to be spirit than beer drinkers.

Finally we would suggest that British practitioners are unlikely to see many alcoholic patients with sideroblastic anaemia, thrombocytopenia, or Zieve's syndrome but will often see patients with a normal haemoglobin concentration, a normoblastic bone marrow, normal B₁₂ and folate levels, and a raised MCV that returns to normal after three or four months of abstinence from alcohol.

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¹ Wu, A, Chanarin, I, and Levi, A J, *Lancet*, 1974, **1**, 829.

² Wu, A, et al, *British Journal of Haematology*, 1975, **29**, 469.

³ Unger, K W, and Johnson, D, *American Journal of the Medical Sciences*, 1974, **267**, 281.

⁴ Buffet, C, *Revue de l'Alcoolisme*, 1976, **22**, 15.

⁵ Wright, S G, and Ree, G H, *Lancet*, 1978, **1**, 49.

⁶ Khaund, R R, *Lancet*, 1978, **1**, 327.

⁷ Davidson, R J L, and Hamilton, P J, *Journal of Clinical Pathology*, 1978, **31**, 493.

⁸ Whitehead, T P, Clarke, C A, and Whitfield, A G W, *Lancet*, 1978, **1**, 978.

⁹ Metz, J, et al, *British Journal of Haematology*, 1968, **14**, 575.

¹⁰ Wickramasinghe, S N, and Longland, J E, *Acta Haematologica*, 1974, **52**, 14.

Nutrition and the brain

SIR,—Your leading article (17 June, p 1569) presents an informative analysis of the present state of knowledge and uncertainty as to the relationship between nutrition and cerebral development. The general approach is factually based, with a brief review of some of the relevant data. In sharp contrast is your curt dismissal of "unscrupulous" attempts to link malnutrition to "mental retardation in its usual sense."

Severer degrees of mental handicap (profound, severe, and moderate) occur at about 4 per 1000 in populations aged 10-16 years in Britain, as shown by a number of careful surveys. While it seems improbable that such severe mental handicap can be specifically or commonly due to malnutrition in a relatively developed country, the sources which you quote, and others, still leave open the possibility in cases of prolonged maternal or severe infant malnutrition elsewhere. However, the majority of the mentally retarded singled out for help from the special services in Britain or the USA are the mildly retarded, as defined by the International Classification of Diseases or the American