states. Did any of Cliff and colleagues' patients have optic neuropathy, manifest by inability to distinguish between red and green (before vision to white is materially reduced) and by visual field changes with bilateral centrocecal scotoma? Besides tobacco smoke and alcohol many plants contain cyanide. The neurological manifestations of chronic cyanide toxicity in Nigerian patients included bilateral optic neuropathy and nerve deafness, myelopathy, and sensory ataxia. In patients with "West Indian amblyopia" optic neuropathy and pyramidal tract involvement with spasticity predominates. Raised plasma cyanocobalamin levels and raised plasma and urinary thiochycanate levels, products of cyanide detoxification, were found. This supports the contention that the dietary source of cyanide in cassava and in other vegetables contributes to the pathogenesis of neurological and ophthalmological disorders. Furthermore, the ophthalmic findings are consistent with the strict criteria we specified and adopted in the diagnosis of tobacco amblyopia visual fields.

Although neurotoxic effects of cyanide intoxication are more likely when nutrition is poor and in the indigenous populations of the tropics, they are also manifest in the Western hemisphere.

Furthermore, indiscriminate dumping of industrial cyanide waste may lead to contamination of food and water and so to chronic cyanide toxicity in people with a genetic or acquired error of cyanide or vitamin B12 metabolism.1 Cliff and co-workers did not discuss the treatment of patients with evidence of chronic cyanide intoxication. Cyanocobalamin, but not cyanocobalamin, is a powerful cyanide antagonist.10 Some patients with tobacco amblyopia did not respond to treatment because, though hydroxocobalamin had been prescribed, cyanocobalamin had been given. The diagnosis may then be questioned, treatment discontinued, and the patient consigned to a life of poor sight. In the future other cyanide antagonists, with the advantage of oral administration, may become available. All patients with tobacco or nutritional amblyopia, and optic atrophy, myelopathy, or neuropathy of obscure origin should be screened for vitamin B12 deficiency before embarking on any therapy other than hydroxocobalamin.

MERCURY IN PITUARY GLANDS OF DENTISTS

Sir,—Neuropathic studies have demonstrated significant correlation between the total mercury (Hg) content of the occipital cortex and the number of dental amalgam fillings.1 I have analysed the Hg content of pituitary glands in seven necropsy cases. Three patients were dentists, and I found surprisingly large amounts of Hg in the pituitary glands of the dentists compared with levels in occipital lobes (see table).

The four controls with no known occupational exposure to Hg (two had had double dental prostheses for some years) had low Hg concentrations in occipital lobes and pituitary (table). One control had many amalgam fillings and another had had many amalgam fillings removed during the last year of life (removal of amalgam generates high Hg vapour levels momentarily). A possible explanation for the difference between the Hg content of the pituitary gland and occipital lobe in the dentists is the difference in degree of penetration from the arteries. Furthermore, Hg from the vapour of dental amalgam may have been absorbed by the nasal mucosa and directly transported to the cranial cavity and pituitary. Fifty years ago the German chemist Alfred Stock described direct transport of Hg via mucosa from the nasal cavity to the brain, and Störtebeker1 discusses direct transport of Hg from nasal cavity to cranial cavity via the cranial venous system and the olfactory nerves.

These data suggest that patients with several amalgam fillings may have increased levels of Hg in their pituitary glands and that dentists should handle amalgam carefully.

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MAGNUS NYLANDER

DIETARY MANIPULATION IN RHEUMATOID ARTHRITIS

Sir,—Dr Darlington and colleagues (Feb 11, p 236) report significant changes in rheumatoid arthritis (RA) disease activity during a trial of dietary manipulation. The patients in this trial lost a significant amount of weight, indicating that they had reduced their calorie intake. Sköldstam and colleagues reported beneficial effects of calorie restriction in another trial of diet in RA1 and discussed two other studies with similar findings. Malnutrition is immunosuppressive, and this may be the mechanism through which calorie restriction affects RA. Because weight loss was similar in the good and poor response groups, one cannot conclude that the weight loss was unrelated to the changes observed during the study. RA response to immunosuppressive drugs varies greatly from patient to patient.

Darlington et al cite the little evidence that exists of the links that have been established between inflammatory arthritis and dietary constituents. The very high response rate in this study suggests that other factors such as calorie restriction may be involved. Future trials should include adequate calorie intake to maintain weight.

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