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mineral filter aids been used in the past few decades in sugar refining in Sweden? And do workers in such factories have an increased incidence of gastrointestinal tumours and lung carcinomas?

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D. WYNN PARRY

STANDARDS FOR ASBESTOS EXPOSURE

SIR,—Your Round the World correspondent (Dec 3, p 1298), reporting on the US emergency standard for asbestos exposure, states that attempts to modify current health standards "have been resisted by industry". This is not true. For some years now the US asbestos industry, through the Asbestos Information Association of North America (AIA/NA), has been trying to persuade the Occupational Safety and Health Administration to hold public hearings so that the need for a revised standard could be properly investigated. The Administration took no action but has now suddenly (as your correspondent points out) imposed an emergency temporary standard which carries with it implications of an extreme danger to the workforce in need of an urgent remedy, which is quite at variance with the facts. US courts subsequently suspended this emergency temporary standard, pending further evidence.

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NEVILLE STACK,
Director General

IS MIGRAINE FOOD ALLERGY?

SIR,—I was impressed with the results and scientific rigour of the trial reported by Dr Egger and colleagues (Oct 15, p 865). Nevertheless, one point troubles me. Of 88 children who completed the oligoantigenic diet, only 40 were randomised into the double-blind trial. 28 children were excluded for a variety of reasons, including "reacted to placebo tin". Why were these children given a placebo tin? My understanding was that children were offered the placebo tin only after randomisation to the double-blind trial. Children offered the active food had significantly more headaches than did children offered the placebo tin; however, if children who had reacted to the placebo tin were systematically removed from the study it is easy to understand why the placebo effect would be so much less than that of the presumed active food. If any number of children who reacted to the placebo food were removed from the final analysis, the conclusions of this study reached by Egger et al become suspect.

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WILLIAM FELDMAN

**This letter has been shown to Dr Egger and his colleagues, whose reply to it and to earlier comments follows.—ED. L.

SIR,—Professor Feldman has drawn attention to an imperfection in the text of our paper, which resulted from editorial compression and from removal of the accompanying table, which gave the reasons for the exclusions. Most refusals were because the parents were unwilling, despite previous agreement, in view of the remarkable improvement in their children; others because children did not react to foods for which we had tins; and only one because he reacted to some of the ingredients of both placebo tins, when these were introduced openly, one by one, in the reintroduction phase.

We reject the suggestion of Dr Wilkinson and Dr Blau (Nov 5, p 1082) that our patients did not have migraine. As stated, patients were selected for severity and frequency and so the proportion of patients with complicated migraine was high; seizures in 15% is about twice the rate reported in an unselected series (6·5%).[1] The recommendation that research be done on "straightforward cases of migraine" is unsound. Such demanding treatment would be indicated only in children with frequent severe attacks, and relapse on reintroduction of the food would be detectable only in those responding quickly, but the selection was obviously not a bad thing since those with complicated migraine responded as well as those with simple migraine; this also suggests that the existing view that these are all one disease is correct.

COMPLETE DATA ON PATIENT RECRUITMENT

	No
Entered study	99
Completed oligoantigenic diet	88
Responded	82
Relapsed on open provocation	74
Entered trial	46
Completed trial	40
Did not enter trial because:	
Unwilling	11
No appropriate tin	9
Reacted to constituent of placebo tin	1
Ready after trial was complete	7
Withdrawal from trial:	
Accidental break of diet	2
Refused tins	4

Dr Cook and Dr Joseph (Nov 26, p 1256) fail to understand the principle of the oligoantigenic diet (few foods). Any of the foods can provoke allergy, so it is not surprising that some patients needed a second oligoantigenic diet, and this is not evidence of failure of compliance. Of course some of the symptoms during the reintroduction phase may have been fortuitous, but the trial shows that most were not. They seem to have misphrased their letter; our patients had frequent headaches, not one every 6 months. The results were assessed by development of headache, and by preference (ie, any symptom) not by only one related symptom. Since this is the first double-blind controlled trial of the food allergy hypothesis of migraine, there can be no "internationally accepted practice". Experimental designs appropriate for the study of drugs may well be quite different. The suggestion that we should have ruled out deficiencies in certain enzymes prejudges whether such deficiencies are causes or effects of the disease. Since 90% of the children responded to the diet, such differences are clearly unimportant. Those who look for "substantiation" of an allergic hypothesis by IgE antibody or skin tests will often be disappointed.

In answer to Dr Peatfield (Nov 5, p 1082), we would point out that we are about to start a study of the mechanisms involved, but we doubt whether these will answer the allergy-idiosyncrasy questions; both may be true, sometimes together. Like Congdon and Forsythe[2] and Moffett et al,[3] we are not impressed by the published data on oral tyramine provocation but, if it does work, such a response might be similar to those we observed to physical stimuli in being secondary to food allergy. The important point for the patients is that diet can benefit far more of them if it is based on the allergy hypothesis. We agree that a comparison of speed of effect of high and low tartrazine foods would be interesting.

In reply to Dr Stephenson (Nov 26, p 1257), the seizures were typical of partial or generalised epilepsy according to the international classification. All patients had persistently abnormal EEGs. One had been given antiepileptic drugs (by Dr Stephenson himself). The patients who fainted were not included in the epilepsy group. We are preparing a more detailed account of patients with epilepsy responding to diet. We did not include a question on travel sickness in our questionnaire, but only one on migraine attacks provoked by travel.

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1. Lennox WG, Lennox MA. Epilepsy and related disorders. London: Churchill, 1960.

2. Congdon PJ, Forsythe W. Migraine in childhood, a study of 300 children. *Devel Med Child Neurol* 1979, 21: 209–16.

3. Moffett A, Swash M, Scott DF. Effect of tyramine in migraine: a double blind study. *J Neurol Neurosurg Psychiatry* 1972; 35: 496–99.