that followed had considerable protein restrictions too, up to as long as one year for certain foods.[4] Moreover, a prospective study of 1262 babies in New Zealand showed a positive relation between eczema and the number of different types of solid food given to infants in the first four months[5]: details of solid foods were not recorded beyond this age, but that does not lead to an assumption that these foods could not thereafter be culpable too. So it seems inappropriate to limit recommendations to breast feeding when restrictions to the weaning diet could be equally important.

As anyone concerned with infant feeding knows, not all mothers want to breast feed and not all mothers enjoy it. Furthermore, some mothers, while wishing to breast feed, find it difficult to maintain, and far more mothers than not have given it up (for various reasons) by the time their babies are 6 months old. How then can anyone lead the field in recommending, without a shred of evidence to support this, that all mothers of babies at atopic risk should continue breast feeding for one year?

All mothers want healthy children, mothers of atopic children perhaps even more than most. But they have a lot to contend with already without being subjected to an unsubstantiated "View-hollo" (and that is what it may become) to suckle their next child for one year, unless of course they want to. So please let us tell these mothers what there is to know, not what we may like to think.

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[1] Golding J, Butler NR, Taylor B. Breast-feeding and eczema/asthma. *Lancet* 1982;i:623.
[2] Gordon RR. Breast-feeding and eczema/asthma. *Lancet* 1982;i:699.
[3] Matthew DJ, Taylor B, Norman AP, Turner MW, Soothill JF. Prevention of eczema. *Lancet* 1977;i:321-4.
[4] Saarinem UM, Kajosaari M, Blackman A, Martti AS. Prolonged breast-feeding as prophylaxis for atopic disease. *Lancet* 1979;ii:163-6.
[5] Fergusson DM, Horwood LJ, Beautrais AL, Shannon FT, Taylor B. Eczema and infant diet. *Clin Allergy* 1981;**11**:325-41.

Leishman-Donovan bodies in the duodenal mucosa of a child with kala-azar

SIR,—The clinical features of the case reported by Dr D N Challacombe and others (5 March, p 789) were almost classic of kala-azar: anorexia, weight loss, miserable behaviour, intermittent fever, hepatosplenomegaly without lymphadenopathy, anaemia, leucopenia with monocytosis, and rise in total concentration of proteins and in IgG concentrations.

In the province of Bihar recently we had a massive epidemic of kala-azar. In 1977 there were 100 000 cases in Bihar,[1] out of 400 000 new cases occurring each year in the world.[2] In our series of 692 cases intermittent fever (98%), loss of weight (85%), anorexia (10%), splenomegaly (100%), hepatomegaly (98%), and anaemia (94%) were the main features. Lymphadenopathy was absent. Leucopenia, with monocytosis varying between 5% and 8%, was a constant finding. Total plasma concentrations of protein varied from 53 to 95 g/l with a rise in gammaglobulin.[3] Lymphadenopathy is a feature of African kala-azar and not of that found in the Mediterranean and India. Two patients in our series presented with diarrhoea which responded only to sodium stibogluconate. Parasitisation of the reticuloendothelial cells of the submucosa has been suggested by various workers. In our experience splenic aspiration gave more positive results for parasites than bone marrow aspiration. It was also our experience that we could show parasites in the aspirates of the bone marrow on repeat aspiration even when first aspiration performed a week earlier was negative.

I wonder why the splenic aspiration was not tried before peroral intestinal biopsy in the case of Dr Challacombe and others.

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[1] World Health Organisation. *A decade of health development in South-East Asia: WHO regional publication, South-East Asia series, No 7.* New Delhi: WHO Regional Office for South-East Asia, 1978.
[2] World Health Organisation. *Third annual report: UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases.* Geneva: WHO, 1980.
[3] Thakur CP, Kumar M, Pathak PK. Kala-azar hits again. *J Trop Med Hyg* 1981;**84**:271-6.

**We sent a copy of this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—We agree with Professor Thakur that splenic aspiration, although not without hazard, often shows Leishman-Donovan bodies. In our patient, however, lymphoma was initially considered to be the most likely diagnosis, and a small intestinal biopsy was performed only to exclude an enteropathy, as she was failing to thrive. The serendipitous finding of Leishman-Donovan bodies in the duodenal mucosa and our failure to perform a splenic aspiration may both possibly be excused on the grounds that kala-azar is not a common disorder in rural Somerset.

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Occupational medicine

SIR,—In my reply (3 September, p 686) to Professor Lee's timely article on occupational medicine I had hoped to focus attention on a real dilemma facing society and the profession. My principal theme was that at a time of crisis we must order our priorities and it seems to me, despite their contributions, that there is no real evidence to support the thesis that occupational health departments, as presently constituted within the NHS, do achieve the desired objective. Dr M Gatley (24 September, p 910) who is an occupational health practitioner in an adjacent district and was a member of a general practice which used the services of my hospital would, with a little elementary investigation, have discovered the answer to the question in his last paragraph. I am a radiologist of 20 years' standing, with a particular interest in occupational medicine; I have been involved in our occupational medicine department, since it started, both in a professional capacity and in a variety of roles in the administrative committee running the department, in fact for longer than the present incumbent, for whom I have great respect and who is also a part time general practitioner.

If Dr R J Rabett (24 September, p 910) would read my letter carefully, he would see that I am not suggesting that legal "requirements" of occupational health in the Health Service are less than those in industry. I was suggesting that they are likely to be less of a problem because of our unique structure, which incorporates medical expertise not readily available for industry, and I think it is true that our safety committees can and do enact the relevant codes of practice more rigorously and more efficiently than in industry at large because of this unique structure. This enables us to protect our staff, more easily from the hazards he catalogues.

To Dr Gatley, I am aware of the standard texts in occupational medicine and also of the distinction between community health and occupational medicine, and I was merely suggesting a closer liaison as a means of solving a staffing problem. This approach may not be ideal, but I suspect it might bear more fruit for both than the present approach.

Finally, I would have hoped that doctors in general would consider their basic education and accept that we are privileged and that from time to time we should use our intelligence to enter into general debate, not remain within our respective, sometimes narrow, specialties. I do not regard this as interference, and personally I welcome comment from any quarter. Fortunately, I find most of the colleagues with whom I work share this approach, and I am sure my practice has improved from their regular "interference."

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SIR,—Further to my letter (24 September, p 910) I quote from an article in the *Daily Telegraph*,[1] "More than 1000 children are to be screened after it was discovered that a young doctor with tuberculosis has worked in three children's hospitals in the last 18 months. . . . It was revealed that the doctor started work as a senior house officer without having medical tests."

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[1] Anonymous. Children's health alert over doctor with TB. *Daily Telegraph* 1983 Sept 23:19 (cols 4-5).

"Reciprocal" depression of the ST segment in acute myocardial infarction

SIR,—Dr Kevin Jennings and others (3 September, p 634) reported the adverse prognosis of patients with "reciprocal" ST segment depression during the acute phase of myocardial infarction. Over half of their patients later developed similar exercise induced changes, and 14 of 22 were shown to have stenosis of more than half of a coronary artery supplying the reciprocal area. They conclude that such changes on early electrocardiograms may identify a population which might benefit from late intervention. They provide adequate evidence that this phenomenon is related in some way to infarct size with an increased risk of late pulmonary oedema suggesting appreciable impairment of ventricular function. Some of their patients underwent cardiac catheterisation, but no