December and all were in the City Hospital alone. Five of these patients died within the next two months. During the same period there were three patients in whom the initial antibody titre was high (256 or greater) and who died within a few days of the onset of symptoms. An additional four patients (one asymptomatic) were subsequently found to have high initial titres that remained raised, and of these only one survived a further nine months, by which time his respiratory syncytial virus titre had fallen to a quarter of its original level. Isolation of the virus was achieved in only one case.

During the same study we detected small epidemics of influenza A and B and in comparison the cases of respiratory syncytial virus tended to have a more prolonged illness; wheezing was a frequent feature, while sore throat was not. None of the cases with only viral infection had either clinical or radiological evidence of pneumonia (this contrasts with 87 out of 192 reported to the Public Health Laboratory Service (26 November, p 1618), though two patients who had proved additional bacterial infection did have signs and radiological evidence of consolidation.

This outbreak of respiratory syncytial virus infection was confined to one hospital and all the proved cases occurred within a four week period. A repeat prospective study carried out over the winter of 1982-3 showed only one case of respiratory syncytial virus infection (this patient died) and 10 cases of proved influenza A.

We conclude that respiratory syncytial virus infections in the elderly are associated with appreciable morbidity and mortality.

ANN CAPEWELL

Longmore Hospital,
Edinburgh EH9 1SJ

J M INGLIS
J WILLIAMSON

City Hospital,
Edinburgh EH10 5SB

Range of mycobacterial disease

SIR,—Although we do not doubt the interpretation of the case reported by Dr J H Shribman and others (26 November, p 1593), we question the increasing acceptance of the view to which they subscribe—namely, that there is in tuberculosis a clinical and immunological range comparable with that of leprosy. The evidence of such a range in tuberculosis is so far mainly immunological, and though this may be of some importance one doubts that it is fundamental, or that it constitutes a sound basis for classification, unless clinical and histological counterparts can be shown. The range of leprosy was worked out clinically and histologically.[1] The only attempt so far to show a comparable histological range in tuberculosis has shown instead that the vast majority of clinically overt cases are characterised by a fairly consistent histological response[2] (further work is in progress). Only in a few are there signs corresponding to the high or low immune responses of leprosy. As regards clinical manifestations many reports are silent, and anergic cases cannot be recognised except by skin testing.[3]

One apparent similarity between the two diseases, the haematogenous dissemination of miliary tuberculosis and lepromatous leprosy, needs to be treated with caution. Lepromatous leprosy is disseminated often almost from the inception of the infection. Miliary tuberculosis arises from the discharge of a large focus into a blood vessel, and though a low immune state is a predisposing factor the event is partly a matter of chance. Miliary tubercles, therefore, do not present a uniformly low immune histological response. Rich describes "hard" epithelioid cell tubercles with few bacilli and "soft" necrotic ones with many bacilli.[4]

All infectious diseases must be subject to some modification dependent on host responses. For a disease caused by a single species of organism the range of leprosy is perhaps uniquely broad, being the consequence of remarkably low immunogenicity in an organism with an immunologically privileged accommodation with nerve Schwann cells.[2]

D S RIDLEY
MARIAN J RIDLEY

Hospital for Tropical Diseases,
London NW1 0PE

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Allergy to cows' milk presenting as chronic constipation

SIR,—As the parent of a child with a quite severe allergy to cows' milk, I read with considerable interest the report by Dr K C Chin and others (26 November, p 1593).

My third daughter, unlike her siblings, had feeding problems from birth with the usual label of "colic" applied to these difficulties. At about the 18th month of her life she developed constipation, which subsequently progressed to faecal incontinence. After numerous medical consultations (including a barium enema examination) we were gently upbraided for allowing our child to become so constipated, and various powerful purgatives and enemas were prescribed, with no effect.

At that time she was lethargic, appeared to have hearing difficulties, had a watery discharge from her nose, cervical lymphadenopathy, and her abdomen was distended and tympanitic. Her stools were liquid and offensive.

We stumbled on the diagnosis purely by chance and thereafter curtailed not only milk but any food product which contained cows' milk. Her symptoms soon resolved and within a week she passed a normal stool. Since then she has remained well, except when she succumbs to the temptation of ice cream or chocolate.

JAMES MCGRATH

London W1N 1AH

Calcium antagonists in hypertension

SIR,—Dr A M Heagerty and others (12 November, p 1405) report the lack of effect of treatment with nifedipine on abnormal leucocyte sodium transport in hypertension. From this they have concluded that a link between calcium and sodium transport is improbable and that sodium transport is therefore irrelevant to the aetiology of the condition. They suggest that the abnormality of sodium transport characteristic of the leucocytes of hypertensive patients may be "an innocent bystander in the cellular changes underlying hypertension." We cannot accept that the failure to show an effect of nifedipine on sodium transport in leucocytes justifies such a conclusion.

In a similar study using the calcium antagonist verapamil we have reported an improvement in leucocyte sodium transport accompanying a reduction in blood pressure.[1] Comparable effects on sodium transport were also obtained by exposing normal cells to therapeutic concentrations of verapamil in vitro. This suggests either that a link between sodium and calcium transport does exist or that verapamil has an effect on sodium transport independent of its effects on intracellular calcium. The failure of Dr Heagerty and others to show a similar effect using nifedipine may be due to two substantial differences between these drugs. Firstly, there is evidence that nifedipine (unlike verapamil) penetrates poorly into cells,[2] and its effects may therefore be dependent on a continued presence in the extracellular fluid, a condition that would not have been satisfied in the studies of Dr Heagerty and others.

Secondly, nifedipine is intensely sensitive to photodegradation, and Dr Heagerty and his colleagues do not mention any precautions taken to shield the cell suspension from light. If the cells were exposed to daylight or ordinary artificial light during the experiment then a spurious negative result could have been obtained as a result of destruction of the drug during preparation of the leucocyte. For these reasons it appears unjustified to conclude that nifedipine has no effect on leucocyte sodium transport.

H H GRAY
L POSTON
P J HILTON

St Thomas's Hospital,
London SE1 7EH

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***The authors reply below.—ED, *BMJ*.

SIR,—We are grateful for the comments of Dr Gray and his colleagues. To reiterate the conclusions of our paper: we observed no relation between initial changes in leucocyte handling in hypertensive patients and the fall in blood pressure produced by the calcium channel blocker nifedipine. Thus there is a dissociation between calcium-mediated blood pressure changes on the one hand and abnormalities of leucocyte handling on the other. The failure of nifedipine to influence sodium handling was a secondary observation. We would not wish to deny that changes in intracellular calcium can influence, for instance, membrane sodium permeability in some situations.[1]

We are particularly interested to note that in this context verapamil in vitro influences sodium transport in leucocytes. This might suggest that changes in sodium transport induced by verapamil are secondary to a primary abnormality in calcium handling and not, as Dr Gray and his colleagues suggested in the abstract they cite, a consequence of a reduction in circulating concentrations of a