developed overt diabetes mellitus within three weeks of receiving nifedipine retard 40 mg daily. All were already taking long term thiazides.

Excluding the above, 117 paired measurements of non-fasting blood glucose before and while receiving nifedipine showed a small but significant mean increase from 4.7 to 5 mmol/l (85 to 90 mg/100 ml) ($p < 0.05$). This effect was not exclusive to the cases treated with thiazides. Such observations do not prove cause and effect but taken in conjunction with other evidence should not be ignored. There are reports of hyperglycaemia with impaired insulin release and hyperglucagonaemia in normal subjects¹ and deterioration in glucose tolerance of diabetics with nifedipine,² although other studies have not detected changes.³⁻⁴ Calcium is important for insulin release, and catecholamine rises associated with nifedipine may be contributory.^{5,6}

Data are scant, short term, and lacking for patients with hypertension. Until more information is available the possible role of nifedipine in the development of diabetes or deterioration of glucose tolerance must be borne in mind and blood glucose routinely monitored.

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¹ Charles S, Ketelslegers JM, Buysschaert M, Lambert AE. Hyperglycaemic effect of nifedipine. *Br Med J* 1981;283:19-20.

² Guigliano D, Torella R, Cacciapuoti F, et al. Impairment of insulin secretion in man by nifedipine. *Europ J Clin Pharm* 1980;18:395-8.

³ Joffe BI, Lamprey JM, Shires R, et al. Lack of hormone effects of a single dose of nifedipine in healthy young men. *J Cardiovasc Pharmacol* 1983; 5:700-2.

⁴ Donnelly T, Harrower A. Effect of nifedipine on glucose tolerance and insulin secretion in diabetic and non diabetic patients. *Curr Med Res Op* 1980;6: 690-3.

⁵ Hellman B. The significance of calcium for glucose stimulation of insulin release. *Endocrinology* 1975; 97:392-6.

⁶ Murphy MB, Scriven AJ, Brown MJ, Causoen R, Dollery CT. The effects of nifedipine and hydralazine induced hypotension on sympathetic activity. *Europ J Clin Pharmacol* 1982;23:479-82.

SIR,—Dr Sudhir Kumar Bhatnagar and others (7 July, p 19) have observed a deterioration of glucose tolerance in two patients (one of whom was diabetic) taking nifedipine. They point out that more studies are needed to assess the potential diabetogenic effect of this drug.

In a three month prospective, randomised, double blind, placebo controlled study (to be published) we investigated the effect of nifedipine on glucose tolerance in non-insulin dependent diabetic patients. Sixteen well controlled diabetics (glycosylated haemoglobin $< 10\%$) with moderate untreated hypertension were divided in two groups—eight took nifedipine and eight placebo. After a 12 hour overnight fast all patients were submitted to an oral glucose tolerance test with 75 g glucose. They were then started on either 10 mg nifedipine three times a day or placebo. The

oral glucose tolerance test was repeated after three months of treatment. Blood samples were assayed for glucose (glucose oxidase method in venous whole blood).

No change in glucose tolerance was observed after treatment in these two groups of non-insulin dependent diabetic patients (table). We agree, however, with the conclusion of Dr Bhatnagar and others that plasma glucose concentration should be monitored in patients receiving nifedipine, especially when high doses of the drug are given.¹

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Unmet need in chronic disability

SIR,—Daphne Gloag in her report of the meeting of the Medical Disabilities Society pointed out that there should be one person in each district to organise and coordinate resources (28 July, p 211). I think this is good advice and can be done equally well by either a consultant in rehabilitation or a consultant rheumatologist, or where both are in post together by a joint approach to the National Health Service administration and the local community medical services. In my experience it is better for more than one voice to speak (provided they speak in unison) as notice is more likely to be taken.

As far as the organisation of services in the community is concerned, for the past 18 months in Cornwall we have had a community liaison nurse attached to the rheumatology unit, and she has followed through inpatients into the community after their discharge to act as a trouble shooter. As far as possible the services apply to outpatients as well, and it is surprising to see how many problems that it was thought had been solved are not because arrangements have broken down. We have found that the services of this nurse are invaluable. Health education is a very important part of her work, but equally important is coordinating the services of the general practitioner, the community nurse, the health visitor, the domiciliary occupational therapy service, the local plumber, and the home help service, etc. Indeed, we are now expanding this service as it has more than proved its worth.

Certainly as far as the Cornwall rheumatology department is concerned we can recommend this as a worthwhile development in improving the patient's quality of life in his own home. Such a nurse can give useful feedback to the general practitioner and to the consultant rheumatologist and can often alert services at an early stage when problems develop. Inevitably we have found problems in a rural area because of the considerable distances involved. We have problems too

finding enough finance and manpower to carry out a first class service.

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Appropriate technology: child health

SIR,—I was most interested to see that Dr G J Ebrahim is advocating a second dose of BCG at entry to school (2 June, p 1674). This is not the current policy of the Nepalese expanded programme of immunisation possibly because in this difficult terrain the World Health Organisation has advised it to target its mobile immunisation service to under 3 year olds.

I question, however, the grounds on which Dr Ebrahim bases the need for a second dose. Are there any conclusive studies to show that two doses are any more effective than one? And how can you interpret a second dose that reacts rapidly? Childhood tuberculosis is notoriously difficult to diagnose, especially without radiology, but we find that BCG given to a scar negative child that very quickly ulcerates (within 10 days) is unambiguous supportive evidence of active tuberculosis. If, however, the child already had a scar I would not know how to interpret a quick reaction to a second BCG. In our circumstances prior Mantoux testing has proved expensive and unhelpful since the children in question are almost invariably malnourished. But giving a much larger dose of antigenic material is it not likely that we would see many rapid reactions were we to pursue Dr Ebrahim's policy?

In the assessment of nutritional state he made no mention of the value of measuring weight for height. Auxiliary health workers can quickly learn how to use the Nabarro weight for height wall chart accurately¹: this gives an equally rapid and much more useful index of acute wasting than the arm circumference tape, is entirely independent of the child's age, and is not restricted to children between 12 months and 5 years. In the realm of tropical child health this is most certainly an example of appropriate technology.

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¹ Nabarro D, McNab S. A simple new technique for identifying thin children. *J Trop Med Hyg* 1980;83: 21-33.

* * Dr Ebrahim replies below.—Ed, *BMJ*.

SIR,—The expanded programme of immunisation sponsored by the World Health Organisation (WHO) is rightly concerned with achieving the target of 90% coverage with primary vaccination and does not mention booster doses even for the triple vaccine. The WHO expert committee on tuberculosis, however, recommends vaccination at school age irrespective of vaccination at birth.¹ Since then Dam and Hitze have reported that BCG vaccination at birth seemed invariably effective in those studies in which tuberculous disease appeared relatively soon after infection.²

In a recent study of Swedish children given BCG at birth 62% to 74% were shown to have no tuberculin reactivity at age 7.³ Similar studies in Tanzania and elsewhere show waning of tuberculin sensitivity with time.⁴ In countries with a high incidence of tuberculosis revaccination at school entry is a reasonable policy and this is practised in several east European countries.

Blood glucose concentrations in eight non-insulin dependent diabetics before and after taking nifedipine and in eight before and after taking placebo. Figures are mean (SEM)

	Before nifedipine	After nifedipine	Before placebo	After placebo
Fasting blood glucose concentration (mmol/l)	5.8 (0.2)	5.7 (0.3)	6.2 (0.5)	6.2 (0.3)
Peak blood glucose concentration (mmol/l)	15.4 (1.2)	13.4 (1.2)	14.8 (1.2)	14.6 (0.7)

Conversion: SI to traditional units—Glucose 1 mmol/l = 18 mg/100 ml.