effects of fibre depleted foods. These include dental disease, excessive energy intake, and rapid absorption of carbohydrate with consequent hyperinsulinaemia.

There is now experimental evidence that fibre depleted foods, at least those containing sugars, do enhance energy intake and insulin responses by comparison with their "natural" fibre rich counterparts,[3-6] or with chemical sweeteners,[7] and that they lead to weight gain and to undesirable changes in the lipid composition of blood[6] and bile.[8] These experimental findings harmonise with epidemiological data linking obesity, type II diabetes, hyperlipidaemia, and gall stones with the Western diet.

Cleave's last publication about the dangers of sugar was entitled "Overconsumption, now the most dangerous cause of disease in westernised countries.[9]" Foods and drinks containing fibre depleted sugar are not the only reason for people overnourishing themselves. Thanks to their palatability, cheapness, and universal availability allied to their ease and speed of consumption and their rapid and complete absorption they are uniquely capable of causing obesity and all the other consequences of overnutrition. Our bodies are adapted to taking in carbohydrate in small packages. To remove the packaging before eating the contents is to open Pandora's box. That too is the fibre hypothesis.

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[1] Cleave TL. The neglect of natural principles in current medical practice. *J R Nav Med Serv* 1956;**42**:55-83.
[2] Heaton KW. T. L. Cleave and the fibre story. *J Roy Nav Med Serv* 1980;**66**:5-10.
[3] Haber GB, Heaton KW, Murphy D, Burroughs L. Depletion and disruption of dietary fibre. Effects on satiety, plasma-glucose, and serum-insulin. *Lancet* 1977;ii:679-82.
[4] Bolton RP, Heaton KW, Burroughs LF. The role of dietary fiber in satiety, glucose, and insulin: studies with fruit and fruit juice. *Am J Clin Nutr* 1981;**34**:211-7.
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[7] Porikos KP, Hesser MF, van Itallie TB. Caloric regulation in normal weight men maintained on a palatable diet of conventional foods. *Physiol Behav* 1982;**29**:293-300.
[8] Thornton JR, Emmett PM, Heaton KW. Diet and gall stones: effects of refined and unrefined carbohydrate diets on bile cholesterol saturation and bile acid metabolism. *Gut* 1983;**24**:2-6.
[9] Cleave TL. Overconsumption, now the most dangerous cause of disease in westernised countries. *Public Health* 1977;**91**:127-31.

SIR,—Dr Rodney Taylor gives inadequate references to the importance and the scope of the work of the late Surgeon Captain T L Cleave. Cleave did not, as Dr Taylor states, "propound the fibre hypothesis"; what he did do was to give comprehensive evidence in support of his simple thesis that when carbohydrate bearing crops—be they cereals, roots, or sugar cane—are refined the resulting products if eaten in quantity cause widespread disease.[1] Lack of fibre is only one of the faults which stem from carbohydrate refining. Of equal or more importance is the fault of overconsumption occurring with greater intensity in populations having a high intake of sucrose. According to Cleave overconsumption not lack of fibre per se is the cause of ischaemic heart disease, diabetes, and obesity.[1] There is thus little point in consuming bran without drastically reducing or eliminating the intake of sugar.

It is a pity that Dr Taylor did not make a clear distinction between prevention and treatment. The results of using bran to treat the symptoms of established disease are likely to be uncertain. Bowels hypertrophied and fibrosed from years of chronic constipation complicated by bouts of diverticulitis are not likely to be made normal by any amount of fibre in any form.

There would be no need of bran tomorrow if throughout the land low extraction white flour was replaced by 100% wholewheat and if the mixed high fibre diet which he advocates was not heavily laced with sugar.

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[1] Cleave TL. *The saccharine disease*. Bristol: John Wright and Sons, 1974.

SIR,—Dr Rodney H Taylor perpetuates by omission a longstanding misattribution of the fibre hypothesis. The true originator was Mr N S Painter. The hypothesis follows logically from his MS thesis in 1962,[1] and he developed it in his subsequent papers[2 5] long before the publications cited by Dr Taylor.

In 1964 he wrote: "A low residue diet leads to narrowing of the colon which renders segmentation more efficient so that pressures are produced more frequently . . . this may account for the progressive nature of diverticulosis."[3] In 1967 he followed a discussion of the effect on the colon of the diet of West Indians with "our 'civilised' diet may be responsible for the prevalence of this particular disease [diverticular disease of the colon] in countries in the Western hemisphere."[4] In a paper read early in 1969 at the Anglo American proctology conference in London he was specific: "Diverticulosis is a deficiency due to a lack of natural fibre in our diet."[5]

In what must be one of the first (if not the first) clinical prospective study of bran in diverticular disease published in 1972 he wrote "The bran trial started in 1967"[6]—well before any of the work cited by Dr Taylor. All this and more will be found in Mr Painter's own book,[7] which according to at least one reviewer, "will become a classic."

And yet so many other workers seem to get the credit in review articles. The time has come to put the record straight.

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[1] Painter NS. *Diverticular disease of the colon*. London: University of London, 1962 (MS thesis.)
[2] Painter NS. Diverticular disease of the colon. *Br Med J* 1968;ii:475-9.
[3] Painter NS. Aetiology of diverticular disease of the colon with special reference to the action of certain drugs on the behaviour of the colon. *Ann R Coll Surg Engl* 1964;**34**:98-119.
[4] Painter NS. Diverticular disease of the colon—fact and speculation. *Am J Dig Dis* 1967;**12**:222-7.
[5] Painter NS. Pressures in the colon related to diverticular disease. *Proc R Soc Med* 1970;**63**:144-5.
[6] Painter NS, Almeida AZ, Colebourne KW. Unprocessed bran in the treatment of diverticular disease of the colon. *Br Med J* 1972;i:137-40.
[7] Painter NS. *Diverticular disease of the colon*. London: Heinemann, 1975.

*** Dr Taylor replies below.—ED, *BMJ*.

SIR,—As Dr Trowell and Mr Burkitt say, the literature on dietary fibre is vast. Only now is it becoming properly documented and classified. Some excellent studies have received little attention whereas many poorly designed, inadequately controlled ones have had a great deal. No brief review of this enormous topic can be comprehensive and give both a historical perspective, and a balanced overview of current opinion, and satisfy all viewpoints. The main purpose of my leading article was to draw attention to those gastrointestinal disorders in which dietary fibre had an accepted and proved role, either in prevention or treatment, and to those other conditions for which evidence is, and may always be, lacking. Dr Trowell and Mr Burkitt also emphasise the wider issue that the coincidence of a particular diet and a pattern of disease does not mean that diet is the only environmental factor involved or that there is any direct causal relation.

The contribution of Surgeon Captain T L Cleave to the study of dietary fibre was enormous and pioneering, as Dr Heaton and Dr Yellowlees indicate. He extended his original observations on the benefits of bran in treating constipation in Royal Navy ratings to develop the concept of the intrinsic dangers of fibre depleted foods, and particularly their overconsumption, in the causation of many Western diseases. In gastrointestinal terms the effects of a diet of low fibre content may be more immediate than the remoter metabolic consequences of overconsumption of fibre depleted, energy rich foods. No dietary constituent, however, can be considered in isolation. A decrease in dietary fibre not only increases the proportions of major nutrients but also alters their form, their rate of digestion, absorption, and metabolism, and their transit through the gut as well as the environmental conditions within the intestinal lumen. Some or all of these factors may operate in altering the incidence of the diseases implicated in the fibre hypothesis.

The distinction mentioned by Dr Yellowlees between prevention and treatment of disease when discussing dietary fibre is subtle and often arbitrary. Though some diseases reach a point of irreversibility, some evidence is accumulating that dietary change can alter the course of certain conditions, such as hyperlipidaemias, and thus be a treatment with a longer term preventive role. On these grounds a case can be argued for an increase in our dietary fibre intake or, better, the consumption of a less fibre depleted diet. The long term justification for this, however, still cannot be proved for most diseases.

The extensive work on colonic diverticular disease over 20 years by N S Painter, of which we are reminded by Mr Godfrey, is an exceptional example of the successful search for evidence of an association between dietary fibre intake and a common Western disease. For many other diseases dietary habits and environmental conditions change more rapidly than experimental or epidemiological data can be accumulated.

When proof is lacking advocacy of a less fibre depleted diet must remain a matter of philosophy or conviction.

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Diabetogenic effects of nifedipine

SIR,—We want to add to the observations of Professor Sudhir Kumar Bhatnagar and others (7 July, p 19). Between December 1979 and January 1984 235 patients in our hypertension clinic were prescribed nifedipine in doses ranging from 20 to 80 mg daily. Among these, three patients not previously hyperglycaemic

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 subjects[1] and deterioration in glucose tolerance of diabetics with nifedipine,[2] although other studies have not detected changes.[2-4] 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, Cacciapuotti F, *et al.* Impairment of insulin secretion in man by nifedipine. *Europ J Clin Pharm* 1980;**18**:395-8.
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[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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[1] Charles S, Ketelslegers JM, Buysschaert M, Lambert AE. Hyperglycaemic effect of nifedipine. *Br Med J* 1981;**283**:19-20.

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.

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 accurately[1]: 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.