cancer only. As differences in severity might explain variations in mortality rate, they confined their results to stage I only (cancer confined to breast tissue). They reported, as you explained, that the survival from stage I disease significantly decreased with increasing age. Unfortunately, only 8% of the oldest age group had a necropsy (as compared with 30% of the youngest). Thus they relied on clinical diagnosis of cause of death. It can often only be speculated that the presence of a stage I tumour in a woman aged 70-100 may or may not be the ultimate cause of death; and thus such a clinical diagnosis is liable to error.

Trials of aggressive chemotherapy in old women with early breast cancer might, you suggest, need to be considered. This step, not to be undertaken lightly, receives but little support from the evidence presented.

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¹ Mueller, C B, Ames, F, and Anderson, G D, *Surgery*, 1978, **83**, 123.

*.*We agree with Dr Silman that deaths due to competing risks become an increasingly significant factor the older the population at diagnosis of carcinoma of the breast. It is true that our leading article was guilty of a misquotation from Mueller's original paper concerning the 50% mortality time, which did in fact relate to deaths of all causes. Nevertheless, the article referred to clearly showed, by a life-table analysis of patients dying of breast cancer only, a more rapid death rate among the two older age groups than in the group aged 21-50. We accept that a difference in necropsy rate among the three subgroups analysed could in part have accounted for this finding, but one could equally well argue that the older the patient was at the time of death the less likely would carcinoma of the breast be implicated in the absence of necropsy, owing to the increasing likelihood of death from other causes influencing the judgment of whoever filled in the death certificate. Finally, we were not ourselves advocating trials of aggressive chemotherapy in older women with early breast cancer. Such an approach has already been advocated by Dr G Bonadonna's group in Milan and trials are under way; we were merely stating that this is the first evidence, albeit open to criticism, that lends support to the hypothesis that there are biological variations in breast cancer that may influence subsequent treatment in the postmenopausal women.—ED, *BMJ*.

Shingles: a belt of roses from Hell

SIR,—In their letter (3 February, p 346) on the leading article (6 January, p 5) I feel that Drs K D Crow and Julia P Ellis are unduly worried about the cost of specific antiviral treatment. This is because their calculations of the cost of topically applied idoxuridine are quite wrong. When the ingredients are bought from source the rough cost is about £50 per 100 ml of 35% (not 33%) idoxuridine in dimethylsulphoxide (DMSO). Idoxuridine costs £1.30 per gram when bought in bulk and the DMSO costs about £4 per 500 ml. The average cost of treating a patient is somewhere between £15

and £90, depending on the area. In my department we treat about 300 patients with zoster a year, which would not be possible if the cost ran to £1000 per patient as Drs Crow and Ellis suggest, for we suffer from financial restraints as much as any other region. The method of constant application is described by Juel-Jensen *et al.*¹ It is important to emphasise that the same piece of lint should be reused.

The paper by Merselis *et al.*² which Drs Crow and Ellis state is "notoriously bad," emanated from Hook's department at Cornell when he was there. Professor Hook is one of the two or three outstanding people in infectious disease in the United States and I have the deepest respect for what he says. We have compared notes and both of us have had really disastrous cases of herpes zoster provoked by high doses of steroids, even in non-immunosuppressed patients. I have no doubt that steroids may be useful, and I think Drs Crow and Ellis would do everybody a great favour if they published their results of treating hundreds of patients without side effects—that is, if they could publish the numbers, the duration of pain, the duration of treatment, and side effects.

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¹ Juel-Jensen, B E, *et al*, *British Medical Journal*, 1970, **4**, 776.

² Merselis, J G, Kaye, D, and Hook, E W, *Archives of Internal Medicine*, 1964, **113**, 679.

SIR,—We read with interest and appreciation the leading article of (6 January, p 5) but were somewhat disheartened by your emphasis on the destruction of the virus as being an essential factor in successful treatment. However, the letter from Dr F Ellis (17 February, p 490) commenting on the beneficial effects of irradiation of the posterior root ganglia, and his hypothesis about the reason for his success, prompts us to write in support of Dr Ellis's views, even though our series is pathetically small and may be judged insignificant. The suffering caused by the herpes virus, particularly at the stage of postherpetic neuralgia, is out of all proportion to the other aspects of the condition, and we believe that it would not be proper to withhold our information on statistical grounds.

Although our method of treatment of acute sensory zoster is very different from that of Dr Ellis, we think that the mechanism leading to our results is the same—that is, we seek to modify the inflammatory process in the afferent nerve cells during the acute phase of the disease. Our technique involves giving a single epidural injection containing cortisone and lignocaine at the segmental level indicated by the dermatomes involves and, in the case of trigeminal herpes, into the Gasserian ganglion. More precise details of concentrations, volumes, etc, will gladly be given on request.

Our results are encouraging in that, with 21 patients treated in the acute phase, we have had 100% success in (a) relieving the pain of the acute phase; (b) considerably shortening the duration of the acute phase; and (c) preventing the onset of postherpetic pain. So far we have not had any complications; presumably they, along with failures, come later. A colleague elsewhere in Holland (Dr M E Sluyter), who in fact suggested the method to one of us, has had similar results in about 100 cases.

We wish to emphasise that this method seeks to prevent and not to cure postherpetic

neuralgia, and must therefore be applied as applied as early as possible in the acute phase. We are not aware of any benefit it may have in established neuralgia. The experience of one of us in treating postherpetic neuralgia with epidural cortisone was not encouraging (100% failure in a small number of cases). However, we do have one patient, besides those treated in the acute phase, who was successfully treated nine weeks after the onset of the disease. We therefore find it difficult at the moment to say where the line should be drawn. We hope that others will be able to offer us some guidance here.

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*.*This correspondence is now closed.—ED, *BMJ*.

Prescription for a better British diet

SIR,—The recent paper by Dr R Passmore and others (24 February, p 527) sets out to provide a new approach to prescribing nutritionally acceptable diets for use by "caterers, dietitians, and housewives." In that respect the paper must be welcomed. However, a second and apparently equally important aim of this paper is to define a "better British diet," one likely to reduce the incidence of those diseases alluded to in the paper—namely, coronary heart disease, cancer, obesity, dental caries, and diseases of the large bowel. Recent correspondence in these columns suggests that the latter aim of the paper will receive most attention.

A survey of attitudes to diet-disease relations among members of the Nutrition Society has revealed alarming differences of opinion.¹ We feel that the credibility of the recommendations would have been considerably enhanced had the authors provided supporting data showing (a) the physiological basis for choosing the target diet, (b) the physiological changes anticipated by adopting the target diet, and (c) evidence that these physiological changes would reduce the incidence of the major diet-related diseases.

It is thus interesting to compare the proposed distribution of energy between carbohydrate, fat, and protein in the present and target diets with that which prevailed in 1956,² as in the table. The data do not include energy from alcohol.

Percentage distribution of dietary energy

	Carbohydrate	Fat	Protein
1972-7	48.7	40.0	11.3
% change represented by target diet	+ 6	- 9	+ 4
Target	51.8	36.4	11.8
1956	51.4	37.1	11.5
% change represented by target diet	+ 1	- 2	+ 3

Clearly, the authors feel that the diet which was consumed in 1956 is, for some undefined reason, one to be emulated. Yet, even in 1956 and for the ensuing decade death rates from coronary heart disease were at an unacceptably high level.³

Unlike the authors, we feel that the pursuit of particular trade and agricultural policies should not seriously impede the prescription of a better British diet. The food industry has shown considerable flexibility in providing foods which the public demand for nutritional reasons—for example, high-fibre breakfast cereals and polyunsaturated margarines. Perhaps the authors have tried to strike a compromise between what is physiologically desirable and economically feasible. However, their

recommendations, as at present stated, are at risk of being interpreted as the ideal, physiologically speaking.

Our further criticisms are as follows. Firstly, in recommending a reduction in the consumption of fats and oils to 36% of dietary energy, the authors ignore the suggestion of the joint committee of the British Cardiac Society and the Royal College of Physicians that the reduction in fat intake should take place at the expense of the saturated fat sources. Secondly, while an increased consumption of potatoes would provide a good source of dietary fibre and vitamin C, it is unlikely, as the authors imply, to provide a significant quantity of protein, with <5.0 g protein/day. Thirdly, we wonder on what basis the authors feel that it is unrealistic to recommend a substantial increase in the consumption of fish, pulses, and nuts, especially considering the increasing use of textured vegetable protein by the catering industry and the apparent increased popularity of pulses with housewives. An increased consumption of pulses and pulse products, if substituted for meat, would permit a decreased intake of fat, while maintaining protein intake constant.

Finally, however eminent the authors may be in the field of nutrition, we dispute their wisdom in prescribing a "better British diet." In view of the disagreement on matters relating diet to health, might not three equally eminent nutritionists propose a different better British diet in this or some other journal?

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¹ Brown, C L, Brown, A M, and Naismith, D J, *Proceedings of the Nutrition Society*, 1977, **36**, 96A.

² Ministry of Agriculture, Fisheries and Food, *Household Food Consumption and Expenditure*, 1956. London, HMSO, 1957.

³ Department of Health and Social Security, *Diet and Coronary Heart Disease*. Reports on Health and Social Subjects No 7. London, HMSO, 1974.

SIR,—Dr R Passmore and his colleagues are to be congratulated on their recommendations contained in their article "Prescription for a better British diet" (24 February, p 257). Three nutrition authorities have recommended changes to improve our national diet. These changes are moderate; they are designed to improve health and prevent disease; and they should not place too great a strain on agriculture and trade if they are spread over the next ten years.

The British proposals make no reference to comparable but more drastic changes recommended by the United States Senate Select Committee on Nutrition and Human Needs. US medical experts had given evidence to the committee that the current US diet was a major factor in coronary heart disease, obesity, hypertension, and other diseases. The US committee therefore recommended increasing consumption of starch derived from whole cereals and potatoes by 100%. British recommendations were an increase of grain products by 20% and potatoes by 15%. They made no recommendations concerning the replacement of white flour products by wholemeal, although the latter contains more protein, vitamins, minerals, and fibre. Other US recommendations were also very drastic: total fat reduced from 40% to 30% energy and sugar reduced from 24% to 15% energy. These recommendations for large dietary changes caused considerable concern in US agricultural and trade circles. Their justification certainly demands detailed study.

There is another striking difference between the British and US recommendations. The former made no mention of salt; the latter recommended that salt intake, which ranged from 6 to 18 g/day in the average US adult diet, should be reduced to 3 g/day; but this figure was increased in the second edition of *Dietary Goals for the United States* to 5 g/day. There is much interest in the US² but little in Britain³ concerning the role of high salt intakes as a causative factor in essential hypertension in salt-sensitive individuals.

It may prove difficult for people in Britain to study the evidence that led to these drastic recommendations to change the US national diet. It is contained in five reports all entitled *Diet Related to Killer Diseases*.⁴ It is also desirable to study any modification of these drastic recommendations in the second edition of *Dietary Goals for the United States*. I have made repeated efforts at several English bookshops to obtain a copy of this second edition but have failed and am informed that it is unobtainable.

Both the US and British recommendations agree that dietary fibre intakes should be increased and that this should prove beneficial. It has indeed proved difficult to review the expanding literature on dietary fibre; editorial articles and reviews are scanty and it was only in 1977 that the *Index Medicus* began to include the item "Dietary Fiber—see under Cellulose."

It behoves those who have put forward hypotheses that dietary fibre might be a causative factor in certain diseases of unknown aetiology to produce a bibliography by means of which others can examine the evidence and plan prospective studies. A bibliography of dietary fibre in human nutrition, with over 1000 citations, and with subject and author indexes, has been published recently.⁵

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¹ United States Senate Select Committee on Nutrition and Human Needs, *Dietary Goals for the United States*. Washington, US Government Printing Office, 1977.

² Meneely, G R, and Battarbee, H D, in *Nutrition Reviews: Knowledge in Nutrition*, 4th edn, p 259. Washington, Nutrition Foundation, 1976.

³ *Lancet*, 1978, **2**, 1136.

⁴ United States Senate Select Committee on Nutrition and Human Needs, *Diet Related to Killer Diseases*, vols 1-5. Washington, US Government Printing Office, 1977.

⁵ Trowell, H C, *Dietary Fibre in Human Nutrition: a Bibliography*. London, Libbet (80 Bondway, SW8), 1979.

Fats and atheroma: an inquest

SIR,—The role of prostacyclin in the prevention of platelet aggregation outlined by Dr J R Vane (2 February, p 484) accords well with inhibition of atheroma and thrombosis as viewed by Virchow and Rokitsansky.

There is another aspect of diet in the coagulation process. Chargaff¹ described coagulant and anticoagulant sphingomyelin, and I and my co-workers²⁻⁶ described sphingomyelin lipoprotein anticoagulant released from lung, liver, spleen, and kidney. We suggested⁷ that these probably played a role in inhibiting coronary clotting. The venous blood draining from these organs is delivered rapidly to the heart and so to the coronary circulation. Liver, spleen, and kidney are good sources of this anticoagulant. Brain perfusion⁸ on the contrary enhances the thrombin system. The hind legs and pelvis operate similarly. Diets containing the high-

density anticoagulant lipids probably operate to reduce the risk factor of cardiac infarction.^{9,10}

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¹ Chargaff, E J, *Journal of Biological Chemistry*, 1938, **125**, 677.

² Trethewie, E R, *Australian Journal of Experimental Biology*, 1945, **23**, 231.

³ Trethewie, E R, *Medical Journal of Australia*, 1945, **2**, 328.

⁴ Trethewie, E R, *Australian and New Zealand Journal of Surgery*, 1946, **15**, 286.

⁵ Trethewie, E R, Cleland, J B, and Pengelly, T, *Australian Journal of Experimental Biology*, 1947, **1**, 321.

⁶ Binet, F, Trethewie, E R, and Gaffney, F, *Australian Journal of Experimental Biology*, 1951, **29**, 233.

⁷ Trethewie, E R, *Medical Journal of Australia*, 1947, **1**, 129.

⁸ Trethewie, E R, *Australian Journal of Experimental Biology*, 1946, **24**, 185.

⁹ Berg, K, et al, *Lancet*, 1976, **1**, 499.

¹⁰ Rhoads, C, et al, *New England Journal of Medicine*, 1976, **294**, 293.

Prophylactic co-trimoxazole in biliary surgery

SIR,—We are pleased that Professor J McK Watts and others (6 January, p 58) agrees with the main conclusions of our study (12 August, p 462). We note that he deplores the fact that the wound sepsis rate after cholecystectomy appears to be higher in the United Kingdom than in the United States and Australia. To support this contention he quotes three prospective studies from the United Kingdom and three studies from the United States, two of which were retrospective. It is well recognised that retrospective studies seriously underestimate the incidence of wound sepsis.¹

As regards the mean duration of post-operative stay in hospital, there are obvious socioeconomic differences between Glasgow and Adelaide: many patients in Glasgow come from areas of social deprivation and require an additional period in hospital. Wound sepsis rarely declares itself within one week² and in our study only one of 12 patients developed wound sepsis before the seventh post-operative day, a point at which Professor Watts has already discharged his patients "whether or not their course was complicated."

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¹ Robson, M C, Bogart, J N, and Heggors, J P, *Surgery*, 1970, **68**, 471.

² Cunha, B A, Pyrtex, L J, and Quintiliani, R, *Lancet*, 1978, **1**, 207.

Chronic subdural haematoma

SIR,—In your leading article on chronic subdural haematoma (17 February, p 433) I was surprised that there was no mention of ultrasonography as a method of diagnosis for this condition. Skull radiographs are often of poor quality and the pineal gland is very often not localised. In the average provincial hospital ultrasound either by an A scale or by the more sophisticated midliner is a quick, easy, cheap, and accurate method of determining whether there is any shift of the midline. This causes no inconvenience to the patient and can be performed when his degree of restlessness makes satisfactory radiography impossible. If the patient is found to have a shift of